Title

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Journal

Plant Signaling & Behavior, 12(12)

ISSN

1559-2316

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Publication Date

2017-12-02

DOI

10.1080/15592324.2017.1403708

Peer reviewed

Plant Signaling & Behavior

ISSN: (Print) 1559-2324 (Online) Journal homepage: https://www.tandfonline.com/loi/kpsb20

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To cite this article: Maysaya Thitisaksakul, Shaoyun Dong & Diane M. Beckles (2017) How rice Glycogen Synthase Kinase-like 5 (OsGSK5) integrates salinity stress response to source-sink adaptation: A proposed model, Plant Signaling & Behavior, 12:12, e1403708, DOI: 10.1080/15592324.2017.1403708

To link to this article: https://doi.org/10.1080/15592324.2017.1403708

Accepted author version posted online: 13 Nov 2017.
Published online: 08 Dec 2017.

PLANT SIGNALING & BEHAVIOR
2017, VOL. 12, NO. 12, e1403708 (4 pages)
https://doi.org/10.1080/15592324.2017.1403708

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ARTICLE ADDENDUM

How rice Glycogen Synthase Kinase-like 5 (OsGSK5) integrates salinity stress response to source-sink adaptation: A proposed model

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ABSTRACT

We have previously shown that overexpression of *GSK3-like kinase 5* in rice (*OsGSK5*) was associated with higher starch accumulation and better growth under severe salinity stress. Short-term $^{14}CO_2$ feeding experiments suggested that OsGSK5 promoted higher flux to starch accumulation in the roots under this condition and that this mechanism may help to underscore the better growth characteristics observed. Here, we expand upon this hypothesis and consider (1) how OsGSK5 action could fit into a signaling model that integrates salinity stress to changes in starch metabolism, and (2) how this would facilitate whole plant physiological adaptations in source-to-sink partitioning. We also discuss additional functions of OsGSK5, necessary to support this adaptive mechanism.

ARTICLE HISTORY
Received 5 October 2017
Revised 29 October 2017
Accepted 3 November 2017

KEYWORDS
Glycogen Synthase Kinase 3-like kinase 5; starch metabolism; salinity stress; source-sink relations

Introduction

Glycogen synthase kinase 3-like genes are found in diverse organisms but the family has expanded in plants[1,2] such that in rice there are nine isoforms[3] with distinct, but presumably some overlapping roles. Based on previous study by Kempa et al. (2007),[4] we hypothesized that the MSK4 equivalent, OsGSK5, could integrate carbohydrate metabolism and salinity stress response in plants.[5] OsGSK5 was highly expressed in root, was responsive to the ionic effect conditioned by high salt, and was upregulated under extended darkness that led to root carbon starvation.[5] We created an *OsGSK5* overexpressing line (OE), which showed enhanced growth under highly saline conditions via modulation of source-sink relations.[5] Here, we present models illustrating (1) how OsGSK5 possibly links salt stress responses to starch regulation, and (2) how such links may lead to whole-plant source-sink adaptation to salinity.

OsGSK5: Linking salt stress signals and carbohydrate regulation in rice?

If our hypothesis is correct, then OsGSK5 acts at the nexus of salinity stress signaling and carbohydrate metabolism. Abscisic acid (ABA) and Brassinosteroids (BR) are known hormonal mediators of salinity stress[6] and *OsGSK5* possesses ABA-cis regulatory elements[5] and the transcript is ABA- and BR- inducible,[7] making it likely to be a downstream component of these pathways (Fig. 1). OsGSK5, in turn, could act upstream of enzymes or proteins that determine starch accumulation, regulating their activity through phosphorylation (Fig. 1).

Our data indicated that after exposure to high salt, carbon flux from the leaf to the root was accelerated in the OE, with greater allocation of carbon into the starch fraction of the roots.[5] How this process is mediated remains elusive.[4] The *OsGSK5* was expressed under a constitutive promoter, and presumably, high levels of protein were present in the cell prior to salt exposure.[5] Imposition of salinity stress could have led to posttranslational activation of OsGSK5 in the root, the organ in which native *OsGSK5* is expressed, to promote higher starch biosynthesis. There is support for this hypothesis. MSK4 kinase activity was directly stimulated by high salt,[4] and because of the sequence similarity with OsGSK5 it is plausible that OsGSK5 could also be regulated in this way.[5] In addition, the contribution of the native *OsGSK5* to the adaptive mechanism in the OE cannot be discounted, since this gene is transcriptionally activated by high salinity.[5] Additional protein so produced could increase carbon partitioning to root starch.

It is worth exploring whether OsGSK5 is involved in any known carbohydrate-modulated stress response systems. Sucrose-non-fermenting 1-related kinase1 (SnRK1) is a well-characterized 'energy sensing protein kinase' in plants that links sugar and stress signals.[8-11] It works together with trehalose-6-phosphate (T6P) to adjust assimilate distribution between sink and source under stress.[12] Carbon supply and utilization at the whole plant level is rebalanced, in part, by regulating starch metabolism.[13-16] The similarities in the role of OsGSK5 and the T6P/SnRK1 pathway under stress, make it interesting to consider a potential connection.

Although our work focused on starch, OsGSK5 may contribute to the detoxification of harmful Reactive Oxygen Species (ROS) (Fig. 1). The *Arabidopsis* ASKα, a related GSK3-like protein, phosphorylated Glucose-6-Phosphate

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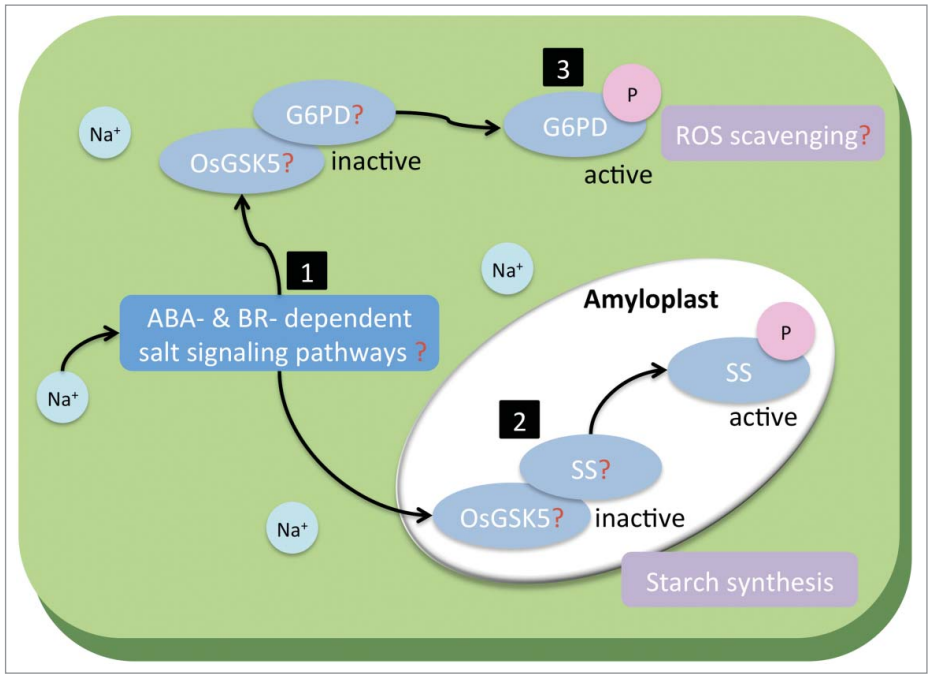

Figure 1. A model hypothesizing the role of OsGSK5 in integrating salt stress responses to starch metabolism. Upon perception of salt, an ABA- and/or BR-dependent salt signaling pathway relays the signal to OsGSK5 by enhancing either its expression level and/or its activity (Step 1). OsGSK5, presumably localized to the amyloplast, then phosphorylates SS and increases its starch synthesis activity (Step 2). It is widely accepted that phosphorylation is an important mechanism in the regulation of starch metabolism.[21,22] Another potential phosphorylation target of OsGSK5 may be cytosolic G6PD, resulting in its ROS detoxification activity (Step 3). For clarity, other subcellular compartments are not shown. Processes with questions marks are unproven or speculative.

Dehydrogenase (G6PD), the activity of which is necessary for maintaining plant cellular redox homeostasis.[17] Transgenic lines overexpressing *ASKα* had more efficient ROS scavenging ability, which facilitated enhanced survival of the modified lines under salinity stress.[17]

Ectopic expression of *OsGSK5* enhances rice physiological adaptation to salt stress via source-sink modification

We showed that transcript abundance of *OsGSK5* correlated with a better physiological response of transgenic rice to salinity stress.[5] A combination of steady-state measurements and ^{14}C-labeling experiments pointed to higher sugar export from the OE leaf, coupled with greater starch accumulation in the root, as the predominant changes underscoring this better adaptation to salinity compared to the control[5] (Fig. 2). Increased sugar flux to the root may have (i) reduced interference of photosynthesis in the OE by relieving sugar build-up in source leaves, and (ii) the amassed starch may have contributed positively to root growth via improved plant gravitational response (Fig. 2). Salinity stress accelerates the degradation of starch statoliths, which negatively alters root growth in *Arabidopsis*,[18] but this problem would be minimized in the OE line. Furthermore, higher starch in the OE roots and leaves could enhance the entrapment of Na^+ within the starch granules (Fig. 2). Kanai et al. (2007) showed that reeds (*Phragmites australis*) grown under 100 mM salt stress adapted by sequestering Na^+ within starch granules in the parenchyma cells of the shoot base.[19] Starch as a potential chelating agent to reduce cellular ionic toxicity is worth investigating.

The model we present does not take into account other factors that may explain the improved phenotype of the OE under salinity stress. First, how source-sink relations change during the diurnal cycle was not assessed. The OE accumulated more leaf starch and should have a greater carbon reserve for nighttime use, which would be a more effective buffer against stress-induced carbon starvation. Second, *OsGSK5* overexpression positively influenced panicle initiation under salinity stress,[5] but it is not known if this was due to changes in carbohydrates in reproductive structures or other regulatory effects of the kinase, since some GSK isoforms can regulate floral initiation.[20] Third, changes in other metabolic pathways could have contributed to the salt-adaptation. ^{14}C-labeling showed that the 3-fold increase of flux to starch in the root was matched by a 3-fold decrease in the flux to amino acids,[5] which suggests that the OE may have also accelerated protein utilization for energy under stress. Finally, the impact of starch chelation of Na^+ and improved gravitropic response was not experimentally determined.

In conclusion, plant GSKs have emerging functions in regulating plant response to environmental stress. Identifying how OsGSK5 is integrated in salt-responsive signal transduction pathways would present a fascinating view of how crop plants reconfigure their metabolism to better tolerate unfavorable environments.

Disclosure of potential conflicts of interest

No potential conflicts of interest were disclosed.

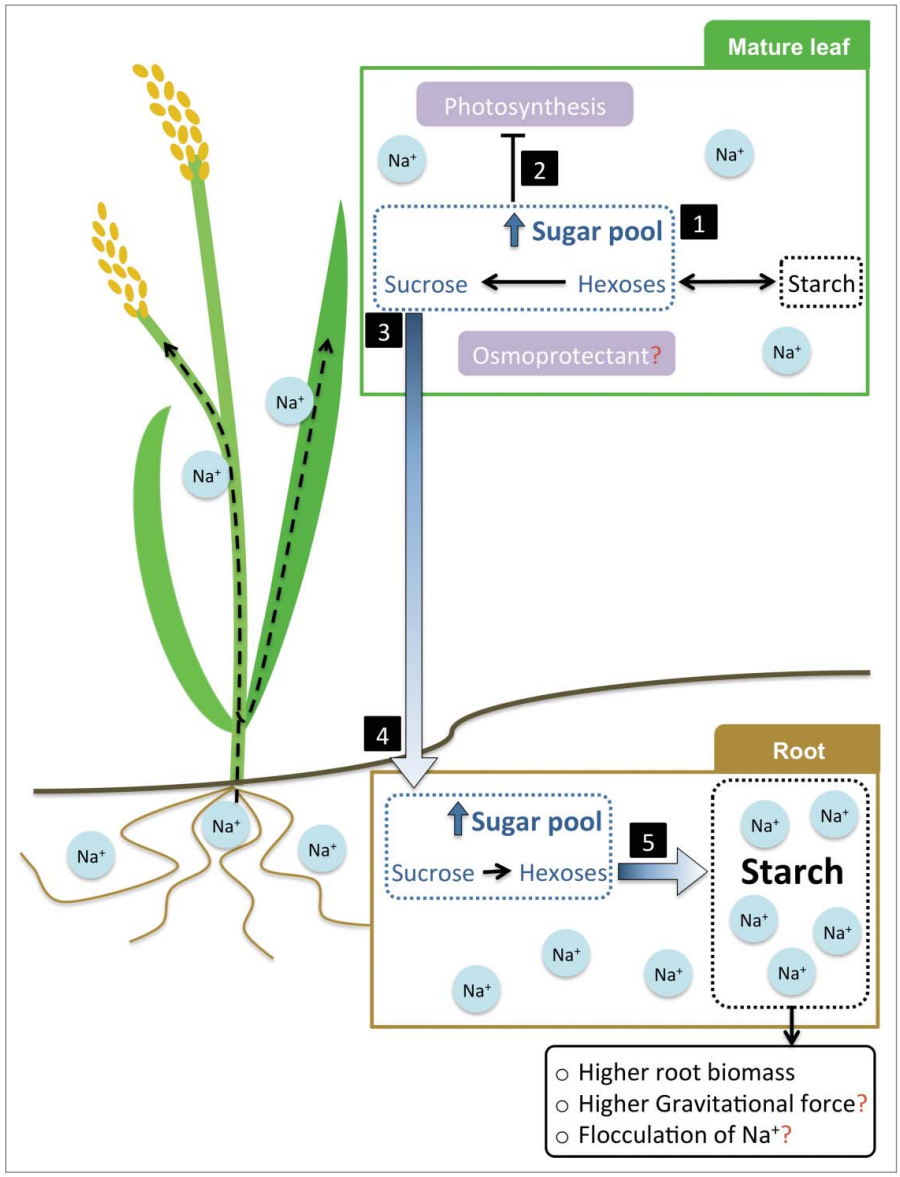

Figure 2. A model explaining putative adaptive alteration in source-sink regulation in the OsGSK5 line after a short-term salt shock. Increases in cellular ion concentration are sensed in source tissues and sugars accumulate, presumably to serve as compatible solutes. This is coupled with reductions in starch content in the control (Steps 1, 2, and 3). In the overexpressor (OE) line, there was no change in leaf starch, but ^{14}C data showed both a higher flux to the sugar pool and increased export from the source to the root with increased storage of starch in that organ (Steps 4 and 5). We hypothesize that higher sugar export potentially reduces feedback interference with photosynthesis. This effect was further enhanced by the higher biosynthesis of root starch, which may steepen the gradient between the source and sink thus facilitating optimal sugar levels. The accumulation of root starch may also improve gravitational response and could flocculate salts, reducing cellular ion concentration and metabolic imbalances. The relative sizes of the sugar and starch pools approximate to those assayed. Processes with questions marks are unproven or speculative.

Acknowledgments

MT thanks the Royal Thai Government and Henry A. Jastro Graduate research award (UC Davis), which supported her study on OsGSK5. SD acknowledges the China Scholarship Council, UC Davis Horticulture & Agronomy Graduate Group Scholarship, and the Henry A. Jastro Graduate Research Award for Ph.D. funding. The France Berkeley Fund and Hatch Project CA-D-PLS-2164-H provided supplemental funding to DMB for this work. We also thank Dr. Kwanjeera Wanichthanarak for her help with the graphical illustration.

Abbreviations

ABA	Abscisic Acid
BR	Brassinosteroids
GSK	Glycogen Synthase Kinase
G6PD	Glucose-6-Phosphate Dehydrogenase
MSK	Medicago Glycogen Synthase Kinase
OE	OsGSK5 Overexpressing Line
ROS	Reactive Oxygen Species
SnRK1	Sucrose-non-fermenting 1-related kinase1
T6P	Trehalose-6-Phosphate

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