Rewiring Plant Salt Stress Resilience through *SAGT1*-Dependent Glycoprotein Stabilization

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Climate change–induced environmental stresses such as high temperature, drought, and salt accumulation threaten global crop productivity. To investigate plant adaptation mechanisms, we conducted a dose-dependent salt stress assay in *Arabidopsis thaliana* (0, 50, 100 mM NaCl). Salt-treated seedlings displayed growth inhibition and tissue bleaching, confirming effective stress induction. Transcriptomic analysis revealed upregulation of known salt-responsive genes (*SOS1*, NHX1) and identified *SAGT1*, a previously uncharacterized gene, as the most strongly induced. *SAGT1* was predicted to encode a glycosyltransferase. CRISPR-Cas9 knockout of *SAGT1* impaired salt tolerance despite normal expression of *SOS1* and NHX1 transcripts, suggesting a post-transcriptional defect. Pull-down and Western blot assays confirmed that *SAGT1* is required for *SOS1* glycosylation, which is essential for its stability and function. This study identifies *SAGT1* as a novel regulator of salt stress adaptation via glycosylation-mediated post-translational control. By highlighting protein quality control as a key component of stress resilience, our findings expand the current paradigm of plant abiotic stress responses. *SAGT1* and its downstream pathway offer a promising target for engineering stress-resilient crops in the face of accelerating climate change.

Keywords: Salt tolerance, *SAGT1*, glycosylation, climate change, Tunicamycin

Introduction

Since the mid-20th century, global climate change—largely driven by the accumulation of greenhouse gases—has resulted in a steady increase in average global temperatures, alongside a rise in extreme weather events such as heatwaves, droughts, and intense rainfall[1]. According to the Intergovernmental Panel on Climate Change (IPCC) Fifth Assessment Report, the frequency and duration of extreme heat events are expected to continue increasing, posing significant threats to both natural ecosystems and agricultural productivity[2]. In particular, high temperatures during the reproductive phase of plants have been shown to drastically reduce crop yields[3], and repeated cycles of heat and humidity can lead to extensive damage in a wide range of plant species[4].

These environmental challenges have become a major concern for food security, especially in tropical and temperate regions where key staple crops such as wheat, rice, and maize are grown. Elevated temperatures can exacerbate salt accumulation in the soil through increased evapotranspiration, further compounding abiotic stress on plants[5–7]. Salt stress is one of the most detrimental abiotic stresses affecting plant growth and productivity, particularly in regions where salinity in soil is prevalent[8,9].

To better understand how plants respond to salt stress under climate change–related conditions, we conducted a dose-dependent analysis of salt stress in *Arabidopsis thaliana*. Seedlings were grown on media supplemented with increasing concentrations of NaCl (0, 50, and 100 mM) to simulate mild to severe salt stress. Phenotypic observations revealed progressive growth inhibition, chlorosis, and bleaching with increasing salinity. Based on these morphological responses, we performed RNA sequencing to identify salt-responsive genes and dissect the underlying molecular mechanisms of salt adaptation.

Our transcriptomic analysis highlighted several canonical salt-tolerance genes such as *SOS1* and NHX1, along with a previously uncharacterized gene, *SAGT1*, which showed the highest fold-change under high-salt conditions. Given that *SAGT1* encodes a putative glycosyltransferase[10], we hypothesize that *SAGT1* a critical role in regulating the glycosylation and stability of the *SOS1* transporter, thereby modulating salt stress tolerance in *Arabidopsis thaliana*.

Previous studies have established that the SOS pathway, particularly the Na^+/H^+ antiporter *SOS1*, is central to plant salt tolerance[11]. However, despite extensive work on transcriptional regulation of *SOS1*, much less is known about post-translational mechanisms that govern its stability and activity. Glycosyltransferases have recently emerged as important modulators of membrane protein quality control and abiotic stress adaptation in plants[12]. This raises the possibility that a salt-inducible glycosyltransferase such as *SAGT1* could directly influence *SOS1* function through protein glycosylation.

To investigate this, we generated *SAGT1* knockout lines using CRISPR-Cas9 and examined their responses to salt stress at phenotypic, transcript, and protein levels. Our findings suggest a novel regulatory role for *SAGT1* in modulating *SOS1* glycosylation and highlight the potential of glycosylation-targeted strategies for enhancing plant stress tolerance.

Specific Aims

- To investigate the expression patterns of *SAGT1* under salt stress in *Arabidopsis thaliana*.
- To assess the functional impact of *SAGT1* knockout on *SOS1* glycosylation and salt tolerance.
- To explore the mechanistic link between *SAGT1*-mediated glycosylation and *SOS1* stability using biochemical assays and genetic complementation.

Materials and Methods

Plant Material and Growth Conditions

Arabidopsis thaliana seeds (Col-0) were surface-sterilized and germinated on Murashige and Skoog (MS) medium. The plants were grown under a 16-hour light/8-hour dark cycle at 22C. After germination, seedlings were transferred to MS medium supplemented with 1% sucrose, 0.8% agar, and adjusted to pH 5.7. To induce salt stress, seedlings were grown on MS media with increasing concentrations of NaCl (0, 50, and 100 mM) for a duration of 10 days.

Experimental Design, Replication, and Randomization

All salt-stress assays were conducted in three independent biological replicates, each consisting of at least 15 seedlings per treatment group. For each replicate, seedlings were germinated and grown on separate plates to ensure biological independence. The values presented in figures ($n = 3$) indicate biological replicates, not technical repeats. Seedlings were randomly assigned to NaCl treatment conditions (0, 50, or 100 mM) across plates to minimize positional or plate effects. Phenotypic measurements (e.g., primary root length, chlorosis scoring) were performed by two independent investigators in a blinded manner, such that the treatment identity of each sample was concealed until after measurements were completed.

RNA and Protein Extraction

Total RNA and proteins were extracted from seedlings grown under salt stress conditions. For RNA extraction, samples were homogenized in TRIzol reagent (Thermo Fisher Scientific), and RNA was purified using the RNeasy Mini Kit (Qiagen). For protein extraction, seedlings were homogenized in protein extraction buffer containing 50 mM Tris-HCl (pH 7.5), 1 mM EDTA, 150 mM NaCl, and 1% SDS. Protein concentrations were determined using the BCA assay.

Quantitative RT-PCR

Quantitative RT-PCR was performed to measure the expression of *SAGT1* under salt stress. cDNA was synthesized from 1 μg of total RNA using the iScript cDNA Synthesis Kit (Bio-Rad). qPCR was carried out using SYBR Green PCR Master Mix (Bio-Rad) with specific primers for each gene. UBQ10 was used as a reference gene for normalization.

Gene name	Sequences (5' $\rightarrow$ 3')
*SAGT1*_Forward	CTCGTGCCCAACTACAATCG
*SAGT1*_Reverse	CTCGTCCTTCATCGCCTTTG
*SOS1*_Forward	TGGTGTTACTTGCTGTCCCT
*SOS1*_Reverse	TGGAAAACAACAATCGCCGT
NHX1_Forward	CACAGATGTACGCGGGAATG
NHX1_Reverse	ACGTATACTGTCAGGCCGAG
UBQ10_Forward	TGGTGGTTTGTGTTTTGGGG
UBQ10_Reverse	GAGTCGAGTCACTTTGCAGG

Gene Expression Analysis and RNA-seq

For quantitative RT-PCR, total RNA was extracted using TRIzol reagent (Thermo Fisher Scientific), followed by DNase I treatment and cDNA synthesis with the iScript cDNA Synthesis Kit (Bio-Rad). Gene expression was quantified using SYBR Green Master Mix (Bio-Rad) on a CFX96 Real-Time PCR System (Bio-Rad). Ubiquitin10 (UBQ10) were used as reference genes for normalization, and all reactions were performed in triplicate. Relative expression levels were calculated using the $\Delta\Delta$Ct method.

For transcriptome analysis, RNA-seq libraries were prepared with the Illumina TruSeq Stranded mRNA Library Prep Kit, and sequencing was performed on the Illumina HiSeq 4000 platform, generating approximately 30 million paired-end reads (2×150 bp) per sample. Raw reads were quality-checked with FastQC and trimmed using Trimmomatic. Clean reads were aligned to the *Arabidopsis thaliana* TAIR10 genome using HISAT2 with default parameters. Gene expression counts were obtained with featureCounts, and differential expression analysis was performed using DESeq2 with thresholds of $|\log_2 \text{ fold change}| \geq 1$ and FDR < 0.05. Gene Ontology enrichment analysis was conducted using the AgriGO v2.0 toolkit.

CRISPR-Cas9-Mediated Knockout of *SAGT1*

CRISPR-Cas9-mediated knockout of *SAGT1* was performed by designing guide RNAs (gRNAs) targeting the first exon of the

gene to induce frameshift mutations. Two independent gRNAs were selected based on minimal predicted off-target effects using CRISPOR. The gRNAs were cloned into the pCas9 binary vector, which was subsequently introduced into *Arabidopsis thaliana* Col-0 plants via *Agrobacterium tumefaciens*-mediated floral dip.

Transformed plants were selected on MS medium containing the appropriate antibiotic, and T1 seedlings were screened for successful integration. Sanger sequencing of the target locus was performed to confirm the presence of indels, and three independent knockout lines (*SAGT1*-1, *SAGT1*-2) were established.

For each line, three independent biological replicates were used for downstream phenotypic, RT-qPCR, and protein analyses, with each replicate consisting of $\geq$15 seedlings per treatment condition. To minimize positional effects, seedlings were randomly assigned to treatment plates, and all measurements were performed in a blinded manner. Potential off-target sites predicted in silico were PCR-amplified and sequenced, and no unintended edits were detected in the analyzed loci.

Western Blot Analysis

Western blotting was used to assess protein levels of *SOS1* in wild-type and *SAGT1* knockout lines under salt stress. Seedlings were treated with 100 mM NaCl for 10 days to induce stress. Total protein was extracted from rosettes using a protein extraction buffer containing 50 mM Tris-HCl (pH 7.5), 150 mM NaCl, 1% SDS, and protease inhibitors. Protein concentrations were determined using the BCA protein assay (Thermo Fisher).

Proteins (30 μg per lane) were separated by SDS-PAGE using a 10% polyacrylamide gel, and transferred to PVDF membranes (Immobilon-P, Millipore) using a semi-dry transfer system at 15 V for 1 hour. Membranes were blocked in 5% non-fat dry milk in TBS-T (Tris-buffered saline with 0.1% Tween-20) for 1 hour at room temperature.

Membranes were probed with custom-produced antibodies against *SOS1* (dilution 1:1000) and Actin (dilution 1:5000, used as a loading control). The *SOS1* antibody was kindly provided by Dr. Kim. Its specificity had been confirmed by testing against wild-type and *SOS1* knockout *Arabidopsis thaliana* lines (data not shown).

After overnight incubation at 4C, membranes were washed and incubated with horseradish peroxidase-conjugated secondary antibodies (1:5000 dilution, Thermo Fisher) for 1 hour at room temperature. Immunoreactive bands were visualized using chemiluminescent detection (Pierce ECL, Thermo Fisher) and quantified using a Bio-Rad Chemidoc imaging system. Band intensities were analyzed using ImageJ software, and normalized to UBQ10 levels for comparison.

Pull-Down Assay

To investigate the glycosylation of *SOS1*, pull-down assays were performed with a glycosylation-specific lectin to capture glycosylated proteins from total protein extracts. The eluted proteins were analyzed by Western blot to determine *SOS1* glycosylation levels.

Tunicamycin Treatment

To restore *SOS1* glycosylation in *SAGT1* knockout plants, seedlings were treated with tunicamycin. Plants were grown on media supplemented with tunicamycin, and protein levels of *SOS1* and NHX1 were analyzed by Western blot.

Results

Dose-Dependent Salt Stress Response in *Arabidopsis thaliana*

To assess the phenotypic effects of salt stress on *Arabidopsis thaliana*, seedlings were grown under three different NaCl concentrations (0, 50, and 100 mM). As salt concentration increased, clear signs of growth inhibition were observed, including reduced root elongation, chlorosis, and overall developmental delay (Fig. 1B and C). At 100 mM NaCl, plants exhibited severe bleaching and a decrease in primary root length (Fig. 1D). A two-way ANOVA revealed a main effect of salt treatment ($F(2,12) = 45.3$, $P < 0.0001$, $\eta^2 = 0.79$). Post hoc Tukey's tests indicated that root length at 100 mM NaCl was significantly shorter than both 0 mM ($P = 0.0021$) and 50 mM ($P = 0.0045$). Data represent mean $\pm$ SEM of three biological replicates, each with $\geq$15 seedlings. These stress-induced morphological changes were used to validate the effectiveness of the salt treatment and to select samples for transcriptomic analysis (Fig. 1A).

Salt-Induced Upregulation of *SOS1* and NHX1 at the Transcript Levels in *Arabidopsis thaliana*

To validate the transcriptomic findings and assess salt-responsive gene expression at the transcriptional and translational levels, total RNA and protein were extracted from *Arabidopsis thaliana* seedlings treated with 0, 50, and 100 mM NaCl (Fig. 2A). Consistently, qRT-PCR showed elevated levels of *SOS1* and NHX1 proteins under higher salt concentrations, confirming transcriptional upregulation is accompanied by transcript-level enhancement (Fig. 2B). These results support the role of these ion transporters in salt tolerance and validate their upregulation in response to increasing salt stress.

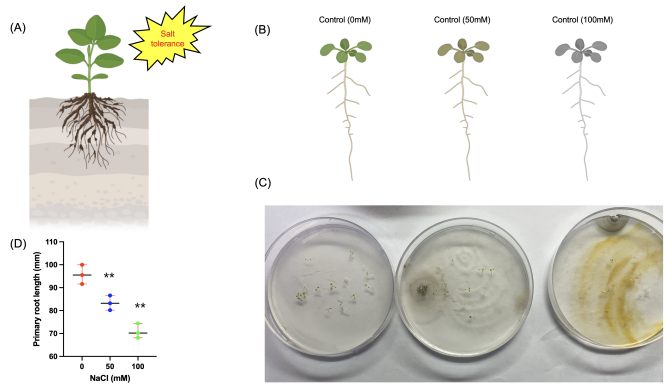

Fig. 1 Salt stress–induced phenotypic changes in *Arabidopsis thaliana*. (A) Schematic of *Arabidopsis* exhibiting enhanced salt tolerance. (B) Illustrations of seedlings grown under 0, 50, and 100 mM NaCl, showing reduced growth and leaf discoloration with increasing salt concentrations. (C) Photographs of seedlings on NaCl-treated media, confirming growth inhibition and bleaching under high salt conditions. (D) Primary root length of 0, 50, and 100 mM NaCl was measured at day 10 after seeding (mean ± SEM, $n = 3$, $P < 0.05$, two-way ANOVA).

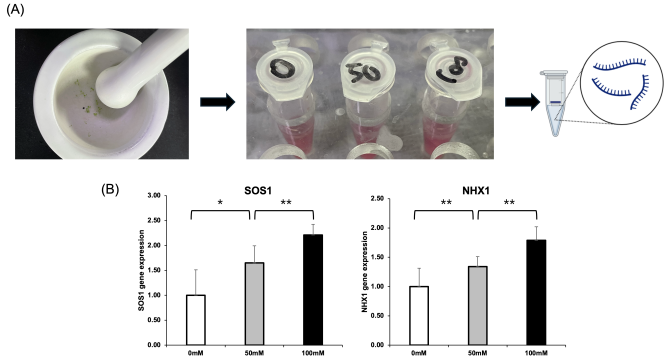

Fig. 2 Expression Analysis of *SOS1* and NHX1 under Increasing Salt Stress. (A) Workflow of RNA and protein extraction from *Arabidopsis* seedlings treated with 0, 50, and 100 mM NaCl. (B) Quantitative RT-PCR analysis shows a serial induction in *SOS1*, NHX1 transcript levels (mean ± SD, $***p < 0.001$).

Transcriptomic identification of *SAGT1* as a novel salt-inducible gene in *Arabidopsis thaliana*

RNA sequencing was performed to identify salt-responsive genes in *Arabidopsis thaliana* under control (0 mM) and high-salt (100 mM NaCl) conditions. Pathway enrichment analysis revealed a significant upregulation of canonical salt stress–associated pathways, including ion transport, osmotic stress response, and abscisic acid signaling (Fig. 3A). Differential expression analysis further identified a set of known salt tolerance–related genes, including *SOS1*, NHX1, P5CS1, and HKT1, all of which were significantly upregulated. Notably, a previously uncharacterized gene, *SAGT1*, exhibited the most robust induction, with over 5-fold higher expression in 100 mM NaCl compared to the control (Fig. 3B). Western blot analysis confirmed that *SAGT1*'s expression level surpassed those of canonical salt tolerance genes, suggesting its potential role as a novel regulator of salt stress adaptation (Fig. 3C).

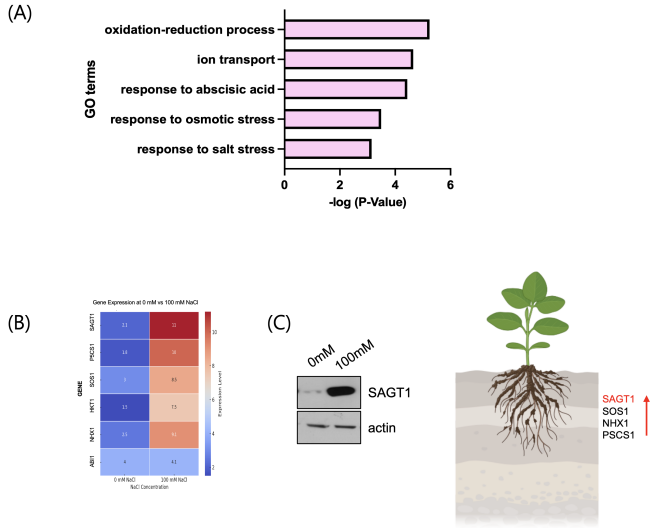

Fig. 3 RNA-seq–based identification of salt-responsive genes. (A) Gene Ontology (GO) enrichment analysis of differentially expressed genes under salt stress. (B) Top upregulated genes under 100 mM NaCl compared to 0 mM control, highlighting *SAGT1* as the most strongly induced. (C) Immunoblot analysis confirms increased *SAGT1* protein abundance in 100 mM NaCl *Arabidopsis*. Actin serves as a loading control (left). Schematic illustration summarizing *SAGT1* upregulation in roots under salt stress (right).

CRISPR-Cas9 Knockout of *SAGT1* Impairs Salt Tolerance in *Arabidopsis thaliana*

To functionally validate the role of *SAGT1* in salt stress tolerance, we generated CRISPR-Cas9–mediated *SAGT1* knock-out lines in *Arabidopsis thaliana*. Transformed lines were selected on antibiotic-containing media, and mutations were confirmed by sequencing (Fig. 4A). Sanger sequencing confirmed a 10-bp deletion in the first exon of *SAGT1*, resulting in a frameshift mutation (Fig. 4B). qRT-PCR analysis confirmed that *SAGT1* was 60% downregulated upon knockout. Immunoblot analyses further confirmed loss of *SAGT1* transcript and protein expression in the knockout lines (Figs. 4C and D). These results demonstrate that *SAGT1* is essential for maintaining salt tolerance and plant viability under high-salt conditions.

Investigation of *SAGT1*'s Role in *SOS1* Glycosylation

SAGT1 is a protein belonging to the glycosyltransferase family 9 and is hypothesized to play a role in regulating glycosyla-

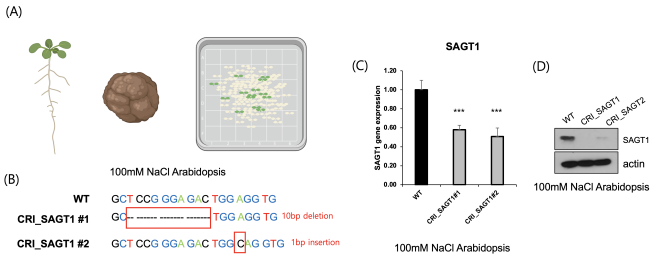

Fig. 4 CRISPR-Cas9–mediated knockout of *SAGT1* impairs salt tolerance in *Arabidopsis thaliana*. (A) Schematic overview of the CRISPR-Cas9–mediated generation of *SAGT1* knockout lines in *Arabidopsis thaliana* via *Agrobacterium tumefaciens*–mediated floral dip transformation. (B) Sanger sequencing reveals a 10-bp deletion in the first exon of *SAGT1* in CRI_*SAGT1* lines, resulting in a frameshift mutation. (C) Quantitative RT-PCR analysis shows a significant reduction in *SAGT1* transcript levels in CRI_*SAGT1* mutants relative to WT (mean ± SD, $***p < 0.001$). (D) Immunoblot analysis confirms decreased *SAGT1* protein abundance in CRI_*SAGT1* lines. Actin serves as a loading control.

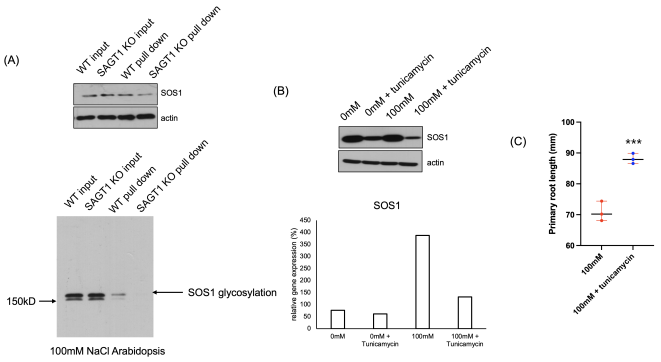

Fig. 5 *SAGT1* is required for efficient *SOS1* glycosylation. (A) Pull-down assay showing reduced glycosylated *SOS1* levels in *SAGT1* knockout compared to WT *Arabidopsis thaliana*. (B) Western blot analysis of *SOS1* proteins (top). Bar graph showing *SOS1* expression (bottom). Actin serves as a loading control. (C) Primary root length of tunicamycin 1 μM treated or not treated 100 mM NaCl was measured at day 10 after seeding (mean ± SEM, $n = 3$, $P < 0.05$, two-way ANOVA).

tion. The SOS pathway, particularly the Na^+/H^+ antiporter *SOS1*, is well established as a central regulator of salt stress tolerance in plants [11]. While previous studies focused mainly on transcriptional regulation of *SOS1* [13,14], glycosyltransferases have been reported to modulate membrane protein stability and activity in plants, suggesting a possible role in post-translational regulation [5].

In this study, we aimed to investigate whether *SAGT1* facilitates the glycosylation of ion transporters such as *SOS1*, thereby aiding their membrane trafficking, protein stability, and functional activation.

We hypothesized that if *SAGT1* assists in the glycosylation of *SOS1*, the functionality of *SOS1* would be fully activated. To test this, we performed a pull-down assay and found that in *SAGT1* knock-out plants, *SOS1* glycosylation was significantly impaired (Fig. 5A). This result suggests that *SAGT1* plays a crucial role in *SOS1* glycosylation, and its deficiency negatively impacts *SOS1* functionality.

After confirming that *SAGT1* is essential for *SOS1* glycosylation, we aimed to inhibit this glycosylation defect by treating 100 mM NaCl *Arabidopsis* with tunicamycin 1 μM. We hypothesized that restoring *SOS1* glycosylation in *SAGT1*-KO would improve salt tolerance. Following treatment, we assessed the phenotypic recovery of the plants and performed Western blot analyses to confirm the restoration of functional activity of *SOS1* and NHX1 (Fig. 5B and C).

Discussion

In recent years, due to the rapidly changing climate and diverse growing environments, complex stress situations in which biotic and environmental stresses act simultaneously are becoming increasingly common in real farms. In particular, environmental stresses such as high temperature, drought, and salt accumulation have a significant impact on crop growth and yield [15,16], requiring an understanding of the complex adaptive mechanisms of plants to cope with them [17,18].

In this study, we investigated the molecular and physiological responses of plants to salt stress using *Arabidopsis thaliana* as a model. Through progressive salt treatment experiments with 0, 50, and 100 mM NaCl, we observed clear growth inhibition, chlorophyll loss, and tissue bleaching in a concentration-dependent manner (Fig. 1). These phenotypes validated the salt stress model and enabled us to extract high-quality samples for transcriptomic analysis.

RNA sequencing revealed a consistent upregulation of canonical salt-responsive genes such as *SOS1* and NHX1. Notably, *SAGT1*, a previously uncharacterized gene, showed the most robust induction under high-salt conditions. Previous studies found that *SAGT1* belongs to the glycosyltransferase family [10], implying a post-transcriptional regulatory defect.

Through pull-down and Western blot assays, we demonstrated that *SAGT1* is involved in the glycosylation of *SOS1*, a process essential for its protein stability and functional activation (Fig. 5A). Furthermore, treatment with a tunicamycin partially inhibited the glycosylation in *SAGT1*-KO plants, leading to the restoration of *SOS1* and NHX1 protein levels and improved phenotypic resilience under salt stress (Fig. 5B and C).

This study is the first to report that *SAGT1* regulates salt stress adaptation via glycosylation-mediated post-translational control. While previous research has largely focused on transcriptional or hormonal signaling pathways in salt stress, our results reveal

a novel mechanism at the protein quality-control level that is crucial for functional transporter stability and downstream stress mitigation. These findings introduce glycosylation as a key regulatory node that can be targeted to engineer stress-resilient plants.

However, there are several limitations to this study. First, the precise glycan structures modified by *SAGT1* remain unidentified, and further mass spectrometry–based glycoproteomic analysis will be required to fully elucidate the molecular mechanism. Second, although we observed glycosylation-dependent effects in *Arabidopsis thaliana*, it remains unclear whether *SAGT1* orthologs exist and function similarly in major crop species such as rice or wheat. Third, while our CRISPR-Cas9 knockout approach strongly implicates *SAGT1* in *SOS1* glycosylation and salt stress responses, we cannot completely exclude off-target effects or secondary stress responses unrelated to glycosylation as contributors to the observed phenotypes. Finally, the tunicamycin treatment used to inhibit N-linked glycosylation is not optimized for field applications, and further work will be necessary to improve its stability, uptake, and cost-effectiveness for practical agricultural use.

Despite these limitations, our findings present a powerful conceptual advance: that targeted manipulation of glycosylation machinery can modulate membrane protein function to improve abiotic stress tolerance in plants. This opens the door to a new class of genetic and chemical strategies for crop improvement, particularly under extreme environmental conditions predicted to increase due to climate change.

Given the increasing global urgency for sustainable agriculture and food security, *SAGT1* and its downstream pathway may represent a potentially interesting target for further investigation in developing climate-adaptive crops. However, as our study was conducted in *Arabidopsis thaliana*, further research is needed in crop species to determine whether similar mechanisms operate and can be harnessed effectively. This work broadens our understanding of plant stress biology and highlights the value of integrating molecular genetics with translational approaches, while acknowledging the current limitations and the preliminary nature of its applications to agriculture.

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