Biochemical and Biophysical Research Communications 511 (2019) 931–934

Contents lists available at ScienceDirect

Biochemical and Biophysical Research Communications

journal homepage: www.elsevier.com/locate/ybbrc

AtMYB44 suppresses transcription of the late embryogenesis abundant protein gene *AtLEA4-5*

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ARTICLE INFO

Article history:
Received 25 February 2019
Accepted 1 March 2019
Available online 7 March 2019

Keywords:
Arabidopsis
AtMYB44
Histone acetylation
AtLEA4-5
Osmotic stress

ABSTRACT

AtLEA4-5 is a member of the group 4 late embryogenesis abundant (LEA) proteins, which are involved in the tolerance of water deficit in *Arabidopsis thaliana*. Chromatin immunoprecipitation assays revealed that the transcription factor AtMYB44 bound directly to the *AtLEA4-5* gene promoter region under normal conditions, but was eliminated in response to osmotic stress (mannitol treatment). A quantitative reverse transcription PCR assay revealed that transcription of the *AtLEA4-5* gene was induced in response to either salt (salinity) or mannitol (osmosis) treatment. The abiotic stress-induced increase in *AtLEA4-5* transcripts was reduced in *35S:AtMYB44* transgenic plants, indicating that the transcription factor AtMYB44 represses gene transcription. More RNA polymerase II stalled at the transcription start site (TSS) of the *AtLEA4-5* gene loci under osmotic stress, but the increment was reduced in the *35S:AtMYB44* plants. Histones are evicted from the promoter region under osmotic stress; however, histone eviction was hampered in the *35S:AtMYB44* plants. Under osmotic stress, the acetylated histones remaining at the TSS region was significantly lower in the *35S:AtMYB44* plants compared with wild-type plants. These results indicate that AtMYB44 suppresses polymerase-mediated transcription of the *AtLEA4-5*.

1. Introduction

The accumulation of late embryogenesis abundant (LEA) proteins is one strategy plants use to confront water-deficit stress [1]. Initially, LEA proteins accumulate at the late stages of seed development and play a role in desiccation tolerance [2]. Many *LEA* genes are induced by abscisic acid (ABA) and various abiotic stresses, such as drought, salinity, and coldness [3–5].

Battaglia et al. [6] classified LEA proteins into seven groups based on the presence of distinct motifs conserved in each group. There are three Arabidopsis group 4 LEA proteins: AtLEA4-1, AtLEA4-2, and AtLEA4-5. Cuevas-Velazquez et al. [7] showed that AtLEA4-2 and AtLEA4-5 are intrinsically disordered proteins when fully hydrated, but form α-helical structures and exert chaperone-like activity under water deficit conditions. The conformational plasticity of AtLEA4 proteins contributes to preserving the activity of their target proteins to overcome the water-deprived environment [8].

In this study, we observed that the Arabidopsis R2R3 MYB transcription factor AtMYB44 acts as a repressor to control the expression of the *AtLEA4-5* gene. Several lines of independent study have reported that AtMYB44 regulates the ABA-mediated tolerance to abiotic stresses such as drought and salinity [9,10]. Transgenic Arabidopsis, soybean, and rice plants transformed with the *AtMYB44* gene showed strong tolerance to abiotic stresses [9,11,12]. Upon ABA treatment, the transgenic Arabidopsis showed enhanced stomatal closure and reduced water loss from the leaves [9]. Recently, we reported that AtMYB44 represses its target genes, including *AtMYB44* [13] and the clade A type 2C protein phosphatase (PP2C) genes *ABI1*, *ABI2*, and *HAI1* [14]. AtMYB44 interacts physically with a Topless-related (TPR) corepressor, and suppresses the transcription of its target genes by promoting histone deacetylation at the gene loci [14]. Our current data indicates that AtMYB44 binds to the *AtLEA4-5* promoter region and suppresses polymerase-mediated transcription of the gene.

2. Materials and methods

2.1. Plant materials and treatments

The Arabidopsis *AtMYB44*-overexpressing transgenic lines *35S:AtMYB44 #21* and *35S:AtMYB44:GFP #28–3* [9] were used in

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https://doi.org/10.1016/j.bbrc.2019.03.006
0006-291X/

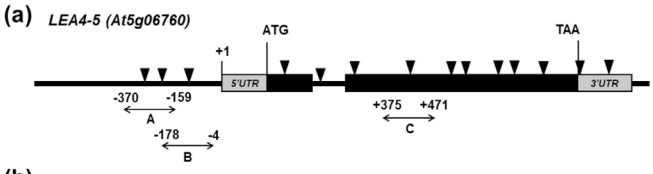
this study. Seeds (T_4) were surface-sterilized with sodium hypochlorite solution and vernalized at 4 °C for 3 days. The seeds were germinated on half-strength Murashige and Skoog (1/2 × MS) agar (1.2%, w/v) medium supplemented with 2% (w/v) sucrose. The plantlets were grown in a growth chamber at 22–24 °C, under 100 μmol photons $m^{-2} s^{-1}$ on a 16-h-light/8-h-dark cycle. Two-week-old plants were transferred to 1/2 × MS liquid medium supplemented without (control) or with 250 mM NaCl (for salt stress) or 300 mM mannitol (for osmotic stress), and grown for an additional 6 h.

2.2. Quantitative reverse transcription polymerase chain reaction (qRT-PCR)

Total RNA was extracted from the plantlets using the Spectrum™ Plant Total RNA kit (Sigma–Aldrich) and first-strand cDNA was synthesized using the RevertAid First Strand cDNA Synthesis Kit (Thermo Fisher Scientific). Quantitative PCR (qPCR) was performed with the Mx3005P qPCR system (Agilent Technologies), using SolGent™ 2 × Real-Time Smart Mix (SolGent) and specific primer sets (Fig. 1a and b). *ACTIN2* (At3g18780) was used as an internal control. Real-time PCR thermal cycling and the calculation of relative transcript levels were performed as described in our previous study [15]. For each experiment, three independent replicates of the qRT-PCR assays were performed and the data were analyzed using Duncan's test [16] at the 95% confidence level.

2.3. Chromatin immunoprecipitation (ChIP) assay

The EpiQuik™ Plant ChIP kit (EpiGentek) was used for chromatin immunoprecipitation. The antibodies used for the ChIP assays were anti-RNA polymerase II (RNAPII) CTD repeat YSPTSPS (4H8) (Abcam, ab5408) (2 μg/reaction), anti-acetyl-histone H3 (H3ac; Merck Millipore, 06–599) (2 μg/reaction), and anti-green fluorescent protein (GFP; Abcam, ab290) (5 μg/reaction). For a negative control, normal rabbit IgG (Merck Millipore, 12–370) (1 μg/reaction) was used. The precipitated DNA fragments were quantified by qPCR using specific primer sets (Fig. 1a and b). The relative RNAPII, total histone (H3), and H3ac levels were calculated by normalizing the percent input of the tested DNA region to the percent input of *ACTIN2*. To determine the AtMYB44–GFP binding signal, the *Gypsy-like retrotransposon* gene (At4g07700) was used as non-specific binding control. The percent input (%IP/IN was calculated as described in our previous study [14]. For each experiment, at least three independent ChIP assays were performed and analyzed using Duncan's test [16] at the 95% confidence level.



(b)

Name	Forward primer (5'-3')	Reverse primer (5'-3')
ACTIN2	GATCTCCAAGGCCGAGTATGAT	CCCATTCATAAAACCCCAGC
LEA4-5-A	CTCTGAAGATTCGACGAGTTC	ATGTTGACGCGTGTTAGGCT
LEA4-5-B	AGCCTAACACGCGTCAACAT	TCGCGTTCGTCACGATAGTT
LEA4-5-C	AAGAGAGAAACGCGTCAGCA	GTCCAGTGGTCGAGTGAGTG
Gypsy	ACTGGTTGCTAGCTGGGAGA	CCAGTGTTGGTTCTCCTTGG

Fig. 1. AtMYB44 binding sites on the *AtLEA4-5* gene promoter. (a) Schematic diagram of the AtMYB44 binding sites at the *LEA4-5* locus examined. Specific primer sets were designed to target to these regions (A, B, and C) and used for ChIP-qPCR assays. (b) Nucleotide sequences of the primers used in this study. *ACTIN2* (At3g18780) was used as an internal control in qPCR assays. *Gypsy-like retrotansposon* gene (At4g07700) was used as non-specific binding control in ChIP-qPCR assays of AtMYB44–GFP binding signal.

3. Results and discussion

3.1. AtMYB44 binds to the LEA4-5 promoter but is released under osmotic stress

Song et al. [17] performed a large-scale ChIP-sequencing assay to identify genome-wide targets of ABA-related transcription factors, and reported that *AtLEA4-5* is a direct target of AtMYB44. The *AtLEA4-5* promoter (~1.5 kb) contains three AACnG AtMYB44-binding sites [18] (Fig. 1a). Two DNA-primer sets designed from the promoter sequence and one from the gene body region were used in the qRT-PCR and ChIP-qPCR experiments (Fig. 1b).

A ChIP-qPCR assay with *35S:AtMYB44-GFP* transgenic plants [9] using a GFP antibody revealed that AtMYB44–GFP fusion proteins bound directly to the *AtLEA4-5* promoter region (Fig. 2a), but were released in response to mannitol treatment (Fig. 2b). Previously, we observed similar results with a ChIP-qPCR assay of the *AtMYB44* promoter itself [13] and PP2C gene (*ABI1*, *ABI2*, and *HAI1*) promoters [14]. These results suggest that the *AtLEA4-5* gene is one of the targets of AtMYB44.

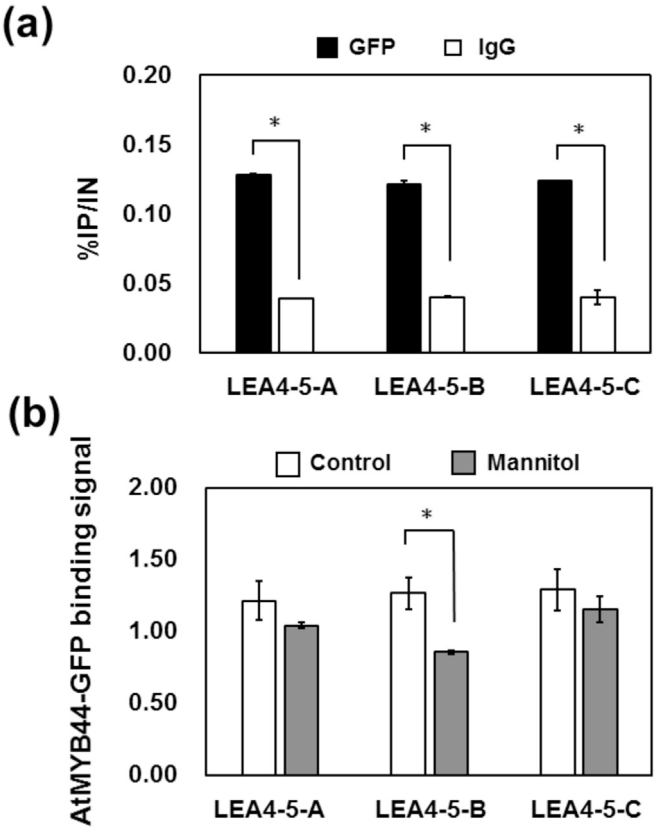


Fig. 2. AtMYB44 binding to the *AtLEA4-5* gene promoter. (a) ChIP-qPCR assay of AtMYB44 binding to the *AtLEA4-5* gene promoter. Two-week-old *AtMYB44-GFP* transgenic plants [9] grown on 1/2 × MS agar medium (2% sucrose) were transferred to fresh medium supplemented with or without (control) 300 mM mannitol for 6 h prior to ChIP assays using antibodies against GFP. IgG was included as a negative control. The AtMYB44–GFP binding signal was calculated as percent input (IP/IN%). Data are reported as mean ± standard error of values obtained from three independent experiments performed in triplicate. Columns marked with an asterisk (*) differ significantly ($P < 0.05$). (b) Alteration of AtMYB44-GFP binding to the *AtLEA4-5* gene promoter in response to mannitol treatment. Three independent experiments were performed in triplicate. Columns marked with an asterisk differ significantly ($P < 0.05$). Bars represent the standard error.

3.2. AtMYB44 suppresses AtLEA4-5 transcription

Two-week-old Arabidopsis seedlings were treated with 250 mM NaCl or 300 mM mannitol to mimic abiotic stresses, as previously described [15,19–21]. qRT-PCR using the primer set LEA4-5-C designed from the gene body sequence (Fig. 1a and b) revealed that either treatment increased transcription of the *AtLEA4-5* gene in wild-type plants (Fig. 3a), consistent with other studies [3,22]. However, the increased *AtLEA4-5* transcript levels were significantly lower in the *35S:AtMYB44* transgenic plants compared with wild-type plants. Therefore, AtMYB44 suppresses *AtLEA4-5* expression in response to abiotic stresses.

Olvera-Carrillo et al. [3] reported that *35S:AtLEA4-5* transgenic plants showed improved tolerance to severe water-deficit in terms of the relative water content, biomass accumulation, and bud maintenance compared with wild-type plants. A reduction in the accumulated levels of AtLEA4-5 proteins resulted in plants that were more sensitive to water-limiting conditions. In comparison, *35S:AtMYB44* transgenic Arabidopsis had higher survival rates than did wild-type plants under water deprivation [9]. The rate of water loss from detached shoots of *35S:AtMYB44* plants was lower than that from wild-type plants. ABA treatment also resulted in a higher rate of stomatal closure. The similar phenotypes of transgenic Arabidopsis plants transformed with either AtLEA4-5 or AtMYB44 is a discrepancy. The roles of AtMYB44 and AtLEA4-5 proteins and their relationships in the tolerance response to osmotic stress await further physiological studies.

Some differences were found in the germination experiments. Seeds of *35S:AtLEA4-5* transgenic plants did not show a significantly improved ability to germinate under high salinity or osmolarity imposed by mannitol compared with wild-type seeds [3]. By contrast, ABA inhibited the germination of *35S:AtMYB44* plants more severely than that of wild-type plants, indicating ABA-hypersensitivity of the transgenic plants [9].

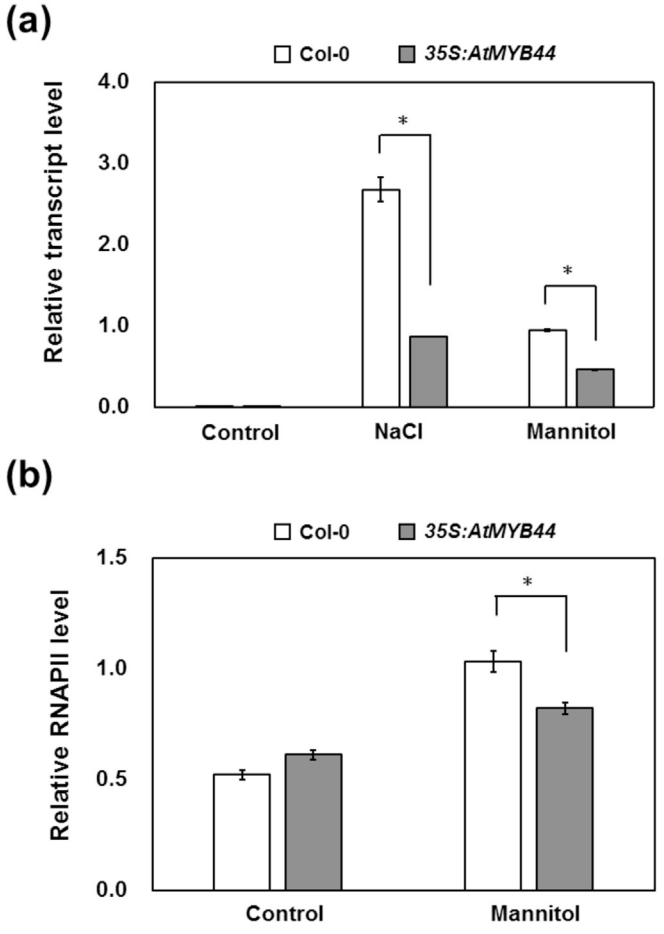


Fig. 3. Transcription of *AtLEA4-5* under abiotic stress. (a) Level of AtLEA4-5 transcripts in response to salt or mannitol treatment. Two-week-old wild-type (Col-0) and *35S:AtMYB44* plants growing on 1/2 × MS medium were transferred to fresh 1/2 × MS medium supplemented without (control) and with 250 mM NaCl or 300 mM mannitol for 6 h and used for total RNA extraction or ChIP assays. Total RNA was used for first-strand cDNA synthesis and qRT-PCR using specific primers (Fig. 1b). The internal control was *ACTIN2*. (b) Level of RNAPII stalled at the *AtLEA4-5* TSS proximal region. Antibodies against RNAPII were used for ChIP-qPCR assays. Real-time PCR was performed using specific primers for region B of *AtLEA4-5* (Fig. 1a and b). The ChIP signal was normalized to the input DNA and an internal control (*ACTIN2*). Three independent experiments were performed in triplicate. Columns marked with an asterisk differ significantly ($P < 0.05$). Bars represent the standard error.

3.3. RNA polymerase-mediated transcription initiation is reduced by AtMYB44

For gene transcription in eukaryotes, RNAPII binds to the gene promoter sequence with the help of activators. In the signal-integration step, the polymerase pauses proximal to the transcription start site (TSS) before active elongation begins [23].

A ChIP-qPCR analysis using the LEA4-5-B primer set (Fig. 1a and b) revealed that mannitol treatment increased the levels of RNAPII stalled proximal to the TSS of the *AtLEA4-5* gene (Fig. 3b). This demonstrates that osmotic stress accelerates *AtLEA4-5* expression by transcriptional, but not post-transcriptional, regulation. However, the increment of stalled RNAPII levels was significantly lower in the *35S:AtMYB44* transgenic plants compared with wild-type plants. This indicates that AtMYB44 interferes with the stalled RNAPII in the *LEA4-5* gene region and suppresses gene transcription under osmotic stress.

3.4. AtMYB44 alters H3 acetylation at the LEA4-5 gene

In the chromatin of eukaryotic cells, genomic DNA is wrapped around a histone octamer consisting of H2A, H2B, H3, and H4, to form a nucleosome. In gene transcription mediated by RNA polymerase, promoter activity is modulated by dynamic competition between nucleosomes and transcription factors. Before RNAPII accesses this position, the chromatin structure must be modified to facilitate the accessibility of DNA for RNAPII. Therefore, gene transcription is accompanied by nucleosome eviction or replacement with other nucleosomes with different compositions [24].

The ChIP-qPCR assay with H3 antibody revealed that nucleosomes were evicted from the AtLEA4-5 promoter regions in response to mannitol treatment (Fig. 4a). After the release of AtMYB44 and nucleosomes, the promoter region may be opened and consequently occupied by other osmotic stress-responsive activators that induce *AtLEA4-5* gene transcription.

Gene activation is associated with histone acetylation (H3ac or H4ac) at specific lysine residues [25,26]. Since AtMYB44 directly suppresses the polymerase-mediated transcription of *AtLEA4-5* (Fig. 3b), we tested whether AtMYB44 functions are also involved in the histone modifications at the target locus. Like the transcript and RNAPII levels, the increment in H3ac levels in *35S:AtMYB44* was lower than that in wild-type plants grown under normal or osmotic stress (Fig. 4b). These results indicate that AtMYB44 represses *AtLEA4-5* transcription in association with decreasing histone acetylation at this gene locus.

Previously, we reported that AtMYB44 forms a complex with TPR corepressors and recruits histone deacetylase(s) to suppress PP2C gene transcription under normal conditions [14]. Like PP2C genes, the histone deacetylases recruited by TPR corepressors are

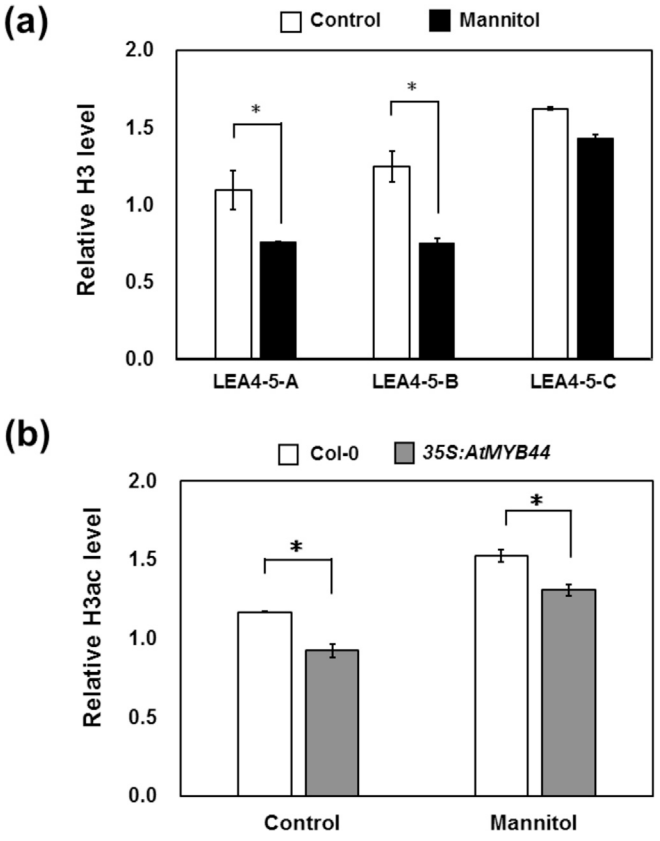


Fig. 4. Histone eviction and acetylation at the *AtLEA4-5* locus. Two-week-old wild-type (Col-0) and *35S:AtMYB44* plants growing on 1/2 × MSA medium were transferred to 1/2 × MS medium supplemented without (control) and with 300 mM mannitol for 6 h and used for ChIP-qPCR assays. (a) Relative histone levels. Antibodies against H3ac were used for the ChIP assays. Real-time PCR was performed using specific primers for region B of *AtLEA4-5* (Fig. 1a and b). The internal control was *ACTIN2*. (b) Level of acetylated histones in the proximal TSS region. Antibodies against H3ac were used for the ChIP assays. Real-time PCR was performed using specific primers for region B of *AtLEA4-5* (Fig. 1a and b). The ChIP signal was normalized to the input DNA and an internal control (*ACTIN2*). Three independent experiments were performed in triplicate. Columns marked with an asterisk differ significantly ($P < 0.05$). Bars represent the standard error.

eliminated from the *AtLEA4-5* chromatin in response to osmotic stress, resulting in gene transcription. Alternatively, or additionally, activator proteins bind to open promoters generated by histone eviction and the replaced repressors may recruit histone acetyltransferases to the gene loci [27,28].

Conflicts of interest

We have no conflicts of interest to declare.

Acknowledgments

This work was supported by the National Research Foundation of Korea (grant number 2016R1A2B4012248).

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