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Molecules and Cells

Non-Specific Phytohormonal Induction of AtMYB44 and Suppression of Jasmonate-Responsive Gene Activation in *Arabidopsis thaliana*

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The *Arabidopsis thaliana* transcription factor gene *AtMYB44* was induced within 10 min by treatment with methyl jasmonate (MeJA). Wound-induced expression of the gene was observed in local leaves, but not in distal leaves, illustrating jasmonate-independent induction at wound sites. *AtMYB44* expression was not abolished in *Arabidopsis* mutants insensitive to jasmonate (*coi1*), ethylene (*etr1*), or abscisic acid (*abi3-1*) when treated with the corresponding hormones. Moreover, various growth hormones and sugars also induced rapid *AtMYB44* transcript accumulation. Thus, *AtMYB44* gene activation appears to not be induced by any specific hormone. MeJA-induced activation of jasmonate-responsive genes such as *JR2*, *VSP*, *LOXII*, and *AOS* was attenuated in transgenic *Arabidopsis* plants overexpressing the gene (*35S:AtMYB44*), but significantly enhanced in *atmyb44* knockout mutants. The *35S:MYB44* and *atmyb44* plants did not show defectiveness in MeJA-induced primary root growth inhibition, indicating that the differences in jasmonate-responsive gene expression observed was not due to alterations in the jasmonate signaling pathway. *35S:AtMYB44* seedlings exhibited slightly elevated chlorophyll levels and less jasmonate-induced anthocyanin accumulation, demonstrating suppression of jasmonate-mediated responses and enhancement of ABA-mediated responses. These observations support the hypothesis of mutual antagonistic actions between jasmonate- and abscisic acid-mediated signaling pathways.

INTRODUCTION

Plants are continually exposed to a variety of developmental and environmental signals throughout their life cycle. Phytohormones such as jasmonates, abscisic acid (ABA), ethylene, and salicylic acid are important plant signal transducers that mediate gene activation for proper cellular responses. Jasmonates, including jasmonic acid and methyl jasmonate (MeJA), are a family of compounds that regulate reproductive developmental processes such as flower development, pollen maturation, and senescence. In addition, jasmonates act as local or systemic signal transducers that activate the expression of defense genes in response to wounds or pathogen infection (Cheong and Choi, 2003; 2007; Creelman and Rao, 2002; Farmer et al., 2003). ABA regulates many aspects of plant development, including the synthesis of seed storage proteins and lipids and the promotion of seed desiccation tolerance and dormancy (Finkelstein et al., 2002). In addition, it mediates tolerance to abiotic stresses such as drought, high salinity, and low temperature (Xiong et al., 2002).

The phytohormone-mediated signaling pathways influence each other through crosstalk, forming a complex network. The best-studied examples of these interactions are the signaling pathways induced by jasmonates and ethylene, which interact to concurrently activate a group of defense genes (Penninckx et al., 1998; Wang et al., 2002). Interaction between jasmonates and salicylic acid may be antagonistic (Kunkel and Brooks, 2002; Lorenzo and Solano, 2005) or synergistic (Grant and Lamb, 2006). In *Arabidopsis thaliana*, the interaction between salicylic acid-mediated systemic acquired resistance and the ABA-mediated abiotic stress response was shown to be antagonistic (Yasuda et al., 2008). Mutually antagonistic interactions between the ABA and jasmonate signaling pathways was observed under conditions of water stress (Moons et al., 1997) and in the expression of defense genes (Anderson et al., 2004).

Transcription factors are the common elements that integrate the crosstalk between these hormonal signaling pathways (Fujita et al., 2006). For example, the transcription factor ERF1 integrates signals from the jasmonate and ethylene signaling

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pathways in defense responses (Berrocal-Lobo et al., 2002; Lorenzo et al., 2003). In *Arabidopsis*, AtMYC2 (JIN1) represses expression of defense genes while activating wound-response genes (Rojo et al., 1999), thus opposing the actions of ERF1 (Lorenzo et al., 2004). The transcription factor that integrates the jasmonate- and ABA-mediated responses is not yet identified. A possible candidate, AtMYC2, a positive regulator of ABA signaling, was shown to suppress jasmonate/ethylene signaling (Anderson et al., 2004).

We report that the *Arabidopsis* transcription factor AtMYB44, which was reported as a positive regulator of ABA signaling (Jung et al., 2008), suppresses jasmonate-responsive gene activation. *AtMYB44* was initially identified as a jasmonate-inducible gene using a microarray (Jung et al., 2007a; 2007b). Subsequently, we observed that *AtMYB44* transcript accumulation is also induced by ABA and ethylene (Jung et al., 2008). Transgenic *Arabidopsis* overexpressing the gene exhibited enhanced drought/salt stress tolerance, by suppressing the expression of genes encoding a group of Ser/Thr protein phosphatase 2Cs (PP2Cs) that were described as negative regulators of ABA signaling.

In the present study, we observed that MeJA-induced jasmonate-responsive gene activation was diminished in transgenic plants overexpressing *AtMYB44*. *AtMYB44*-overexpressing plants exhibited phenotypes consistent with the suppression of jasmonate-mediated responses and enhancement of ABA-mediated responses. These data support the hypothesis of mutual antagonistic actions between jasmonate- and abscisic acid-mediated signaling pathways. However, we found evidence that AtMYB44 does not integrate jasmonate-mediated signaling.

MATERIALS AND METHODS

Plant materials

The *Arabidopsis thaliana* ecotype Columbia (Col-0) was used throughout this study. Independent T4 homozygote lines of transgenic *Arabidopsis* plants constitutively expressing *AtMYB44* (*35S:AtMYB44*) containing one copy (line T-21) or two copies (lines T-17 and T-18) of the transgene (Jung et al., 2008) were used in this study. Seeds of the *atmyb44* T-DNA insertion line (SALK_039074) were obtained from The *Arabidopsis* Information Resource (TAIR).

Seed surfaces were sterilized by soaking in 70% (v/v) ethanol for 15 min and then in 100% ethanol for 5 min. The seeds were placed on autoclaved filter paper and dried in a laminar-flow clean bench. They were then vernalized at 4°C for 4 days after sowing on half-strength Murashige-Skoog (MS) medium (Sigma-Aldrich Corp., USA) solidified in sterile transparent polypropylene dishes. The seeds were incubated in a growth chamber maintained at 21 to 23°C and 60% relative humidity under a 16-h light /8-h dark cycle using white light at 500 $\mu mol/m^2/s$. For adult plants, 2-week-old seedlings were transferred to soil and grown in the same growth chamber.

Chemical treatments

All chemicals for chemical treatments were purchased from the Sigma-Aldrich Corp. A hormone or sugar solution (10 ml) was applied to the MS agar medium in which 2-week-old *Arabidopsis* seedlings were growing. During the treatments, the Petri dishes (87 mm D) were sealed with Parafilm. After treatment, the solution was drained, and the seedlings were harvested and rapidly frozen in liquid nitrogen. When treated to adult plants, whole plant was sprayed with 50 μM MeJA (dissolved in 0.1% ethanol) and harvested 6 h after the treatment.

Wounding

Wounds were made to rosette leaves of 5-week-old *Arabidopsis* plants by pinching with a forceps. The treated leaves (local) were enclosed in a plastic bag to isolate them from distal leaves (systemic), and the plants were kept in an open area.

Northern blot analysis

Northern blot analysis was performed with total RNA extracted from frozen, ground samples using the phenol/SDS/LiCl method (Carpenter and Simon, 1998). Total RNA (5 μg) was separated on 1.3%-agarose formaldehyde gels and transferred to GeneScreen Plus hybridization transfer membranes (Perkin-Elmer, USA). The blots were hybridized with probes and washed at 65°C under stringent conditions. The cDNA probes used in blotting, including *AtMYB44* cDNA (EST 119B8), were expressed sequence-tag (EST) clones obtained from TAIR.

Determination of chlorophyll and anthocyanin contents

Chlorophylls were extracted from 2-week-old *Arabidopsis* seedlings with 95% ethanol at 80°C for 20 min. The amount of chlorophyll was determined spectrophotometrically according to the methods of Lichtenthaler (1987).

Anthocyanin was extracted and determined spectrophotometrically, as described by Mancinelli (1990). Briefly, 50 to 80 12-day-old seedlings (approximately 0.2 g) were extracted with acidified (1% HCl) methanol. The amount of anthocyanin was calculated as A_{530}-0.33A_{657} per gram sample, as described by Kim et al. (2003).

RESULTS

Induction of AtMYB44 gene transcription

Northern blot analysis revealed that *AtMYB44* gene transcripts accumulated within 10 min after *Arabidopsis* rosette leaves were treated with 100 μM methyl jasmonate (Fig. 1A). Gene transcript accumulation was observed (30 min) after the leaves were treated with 0.1 μM MeJA (Fig. 1B).

AtMYB44 expression was not abolished in *Arabidopsis* mutants insensitive to jasmonate (*coi1*), ethylene (*etr1*), or abscisic acid (*abi3-1*) when they were treated with the corresponding hormones (Fig. 2). Northern blots showed that various growth hormones, such as auxin, gibberellin, cytokinin (BAP), and brassinosteroid also induced rapid (within 30 min) *AtMYB44* transcript accumulation (Fig. 3A).

AtMYB44 transcript accumulation was observed when *Arabidopsis* rosette leaves were treated with 200 mM of a variety of sugars, including sucrose, glucose, fructose, mannose, galactose, and trehalose (Fig. 3B). Moreover, same molar concentration of mannitol and sorbitol also induced the gene transcript accumulation, indicating that *AtMYB44* expression can be induced by osmotic pressure. Treatment with 6% (167 mM) sucrose induced *AtMYB44* gene transcript accumulation within 30 min (Fig. 3C).

AtMYB44 gene transcript accumulation was observed at the wound sites on the mechanically damaged leaves (Fig. 4). *AtMYB44* expression was not observed in the distal undamaged leaves, whereas a systemic wound-inducible marker gene *Jasmonate-Responsive 2* (*JR2*) was induced.

Responses of *35S:AtMYB44* plants to jasmonate

Transgenic *Arabidopsis* plants *35S:AtMYB44* (Jung et al., 2008) constitutively expressing *AtMYB44* were examined for jasmonate responsiveness. Northern blots revealed that the expression levels of well-known jasmonate-responsive genes such as *JR2*, *VSP*, *LOXII*, and *AOS* (Wasternack and Hause,

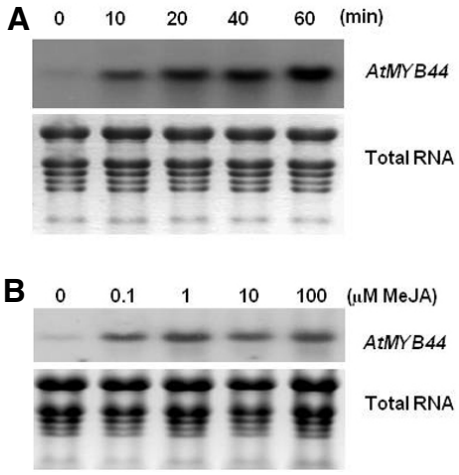

Fig. 1. Northern blot analysis of *AtMYB44* expression in *Arabidopsis* plants. (A) Induction of *AtMYB44* by jasmonate. Methyl jasmonate (MeJA, 100 μM) was applied to the surface of a solid MS agar medium in which 2-week-old *Arabidopsis* seedlings were growing. Total RNA was extracted from plants harvested at the indicated lengths of time after each treatment. (B) Induction of *AtMYB44* expression by various concentrations of MeJA. Total RNA was extracted from plants after treatment with the indicated concentrations of MeJA for 30 min. Equal RNA loadings were confirmed using ethidium bromide-stained gels.

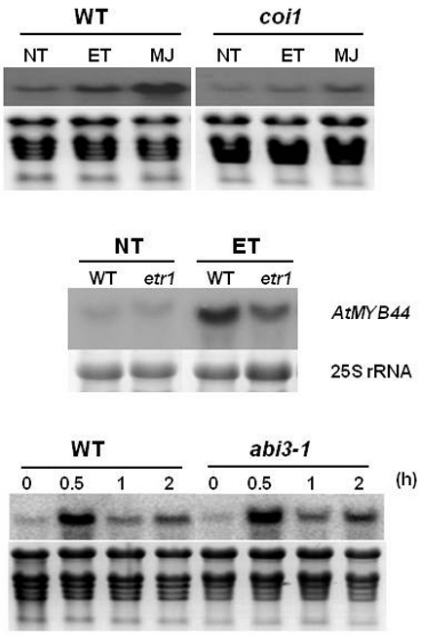

Fig. 2. Northern blot analysis of *AtMYB44* induction in *Arabidopsis* mutants. Two-week-old seedlings of wild-type (WT) plants and mutants insensitive to jasmonate (*coi1*), ethylene (*etr1*), or abscisic acid (*abi3-1*) were treated with their corresponding hormones, i.e., 100 μM methyl jasmonate (MJ), 50 μM ethephon (ET), or 100 μM abscisic acid (ABA), respectively. Equal RNA loadings were confirmed using ethidium bromide-stained gels or by 25S rRNA detection.

2002) increased in all types of 5-week-old plants treated with 50 μM MeJA (Fig. 5). However, compared to wild-type plants, the degree of inducement was significantly less in *35S:AtMYB44* plants but greater in *atmyb44* knockout mutant plants.

To examine MeJA inhibition of root growth, seeds were placed on half-strength MS agar medium containing MeJA. After 7 days, the growth of the primary roots of all types of plants examined was inhibited by MeJA: those of wild-type, *35S:AtMYB44*, and *atmyb44* plants were shortened by 62-69% in the presence of 10 μM MeJA and 75-83% in 50 μM MeJA (data not shown).

Phenotypes of *35S:AtMYB44* seedlings

Young leaves of *35S:AtMYB44* seedlings exhibited a slightly more intense green color than wild-type seedlings. Chlorophylls were extracted from 2-week-old seedlings with ethanol, and their concentrations were determined. The level of chlorophyll accumulation was approximately 16% and 20% higher in *35S:AtMYB44* seedlings (1,320-1,460 μg chlorophyll/g fresh weight) than in wild-type (1,165 μg) or *atmyb44* (1,107 μg) seedlings, respectively (Table 1).

Less anthocyanin accumulated in the hypocotyls of 3-day-old *35S:AtMYB44* plants than in those of wild-type or *atmyb44* knockout plants (Fig. 6A). The lower anthocyanin accumulation in the hypocotyls of *35S:AtMYB44* plants was not overcome by the presence of 10 μM jasmonic acid in the medium. In addition, when determined spectrophotometrically for 12-day-old seedlings, *35S:AtMYB44* (T-18 line) plants contained less anthocyanins (absorbance value = 0.46) than did wild-type (0.69), *atmyb44* knockout (0.95), or the jasmonate-insensitive mutant *coi1* (0.58) plants (Fig. 6B). In the presence of 10 μM jasmonic acid or MeJA in the medium, the anthocyanin content was increased significantly in wild-type (1.64 and 2.11, respectively), *35S:AtMYB44* (0.84 and 1.12), and *atmyb44* knockout (1.62 and 2.23) plants, but not in the *coi1* mutant (0.65 and 0.69).

DISCUSSION

The *Arabidopsis* transcription factor gene *AtMYB44* (At5g67300) was identified as a jasmonate-responsive gene in our previous microarray-based screening experiment (Jung et al., 2007a). We subsequently reported that gene expression was induced by external treatment with ABA, MeJA, or ethylene (Jung et al., 2008). Phytohormonal induction experiments in the present study revealed that a significant level of *AtMYB44* gene transcript accumulated after treating *Arabidopsis* rosette leaves with low concentrations (0.1 μM) of MeJA, and within 10 min after treatment with 100 μM MeJA (Fig. 1). *AtMYB44* expression was not abolished in mutants insensitive to jasmonate (*coi1*), ethylene (*etr1*), or abscisic acid (*abi3*) (Fig. 2). Moreover, gene expression was also induced by various growth hormones, sugars, and osmotica (Fig. 3).

This result is consistent with previous expression profiles showing that the *AtMYB44* gene is expressed with basal level under normal condition and upregulated by a variety of hormone treatments, environmental conditions, and microbial infections (Kranz et al., 1998; Yanhui et al., 2006). We also previously reported that the gene was activated under various abiotic stresses such as dehydration, low temperature, and salinity (Jung et al., 2008). Therefore, it appears that *AtMYB44* expression is not specifically induced by the action of a particular phytohormone. The mechanism leading to the non-specific hormonal induction of *AtMYB44* expression has yet to be identified.

AtMYB44 expression was detected in wounded local leaves (Fig. 4). However, systemic wound-induction of the gene was not observed in distal leaves, where that of the jasmonate response-marker gene *JR2* (Rojo et al., 1999) was obvious. This

Table 1. Chlorophyll content of *35S:AtMYB44* transgenic and *atmyb44* knockout *Arabidopsis* plants

Plant[a]	Chlorophyll A[b,c]	Chlorophyll B[b,c]	Total chlorophyll[b,c]
Wild-type	866.7 ± 16.4	298.5 ± 3.0	1165.1 ± 16.0
35S:AtMYB44 T-17	1030.7 ± 31.5	359.7 ± 18.3	1390.4 ± 48.9
35S:AtMYB44 T-18	984.0 ± 34.3	336.7 ± 11.0	1320.9 ± 42.7
35S:AtMYB44 T-21	1074.3 ± 85.1	388.3 ± 67.0	1463.0 ± 152.7
atmyb44	830.3 ± 39.5	276.8 ± 16.5	1107.4 ± 46.1

[a]Two-week-old *Arabidopsis* seedlings grown on MS agar medium.
[b]μg chlorophyll/ g fresh weight
[c]Mean ± SD of chlorophyll content (N = 12)

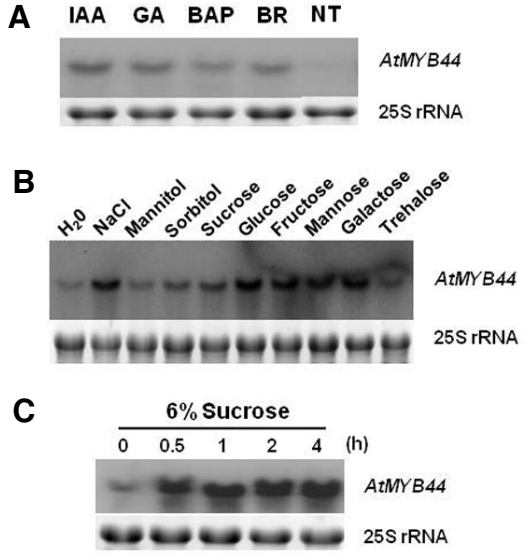

Fig. 3. Northern blot analysis of *AtMYB44* induction by growth hormones and sugars. (A) *AtMYB44* induction by growth hormones. Two-week-old *Arabidopsis* seedlings were treated with 100 μM auxin (IAA), 100 μM gibberellin (GA), 100 μM cytokinin (6-benzylaminopurine; BAP), or 1 μM brassinosteroid (BR), respectively, and harvested 30 min after each treatment. (B) *AtMYB44* induction by various sugars. *Arabidopsis* was treated with 200 mM of each sugar for 1 h. NaCl (200 mM) was used as a positive control. Equal RNA loading was confirmed through 25S rRNA detection. (C) Time course of *AtMYB44* induction by 6% sucrose. Equal RNA loading was confirmed by 25S rRNA detection.

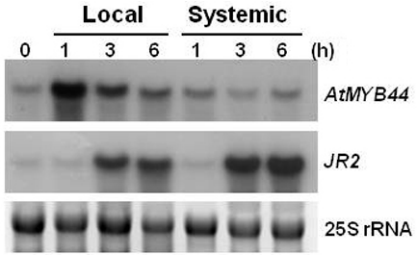

Fig. 4. Northern blot analysis of *AtMYB44* induction by wounding. Rosette leaves of 5-week-old *Arabidopsis* plants were wounded by pinching with a forceps. The damaged leaves (local) were enclosed in a plastic bag to isolate them from undamaged distal leaves (systemic), and the plants were kept in an open area. Equal RNA loading was confirmed by 25S rRNA detection.

result indicates that wound-induced *AtMYB44* expression at the wound site is a jasmonate-independent process, as observed in *choline kinase* (*CK*) and *wound-responsive 3* (*WR3*) genes (León et al., 1998; Rojo et al., 1999; Titarenko et al., 1997). It was reported that cell wall-derived oligosaccharides in mechanically damaged tissues induce the expression of a specific set of wound-responsive genes, while repressing jasmonate-responsive genes activated in systemic tissues (Rojo et al., 1999). Therefore, it appears that *AtMYB44* activation does not involve jasmonate as a mediator and acts as a suppressor of jasmonate-responses.

Indeed, Northern blots revealed that the MeJA-induced activation of well-known jasmonate-responsive genes such as *JR2*, *VSP*, *LOXII*, and *AOS* (Wasternack and Hause, 2002) was suppressed in *35S:AtMYB44* plants but significantly enhanced in *atmyb44* knockout plants (Fig. 5). *JR2* is a wound-inducible jasmonate-responsive gene (Rojo et al., 1999; Titarenko et al., 1997). *LOXII* and *AOS* encode jasmonate biosynthesis enzymes (Cheong and Choi, 2007). *VSP* is a jasmonate-responsive vegetative storage protein gene expressed during development and in response to jasmonate (Mason and Mullet, 1990). Thus, it appears that AtMYB44 acts as a negative regulator in jasmonate-mediated developmental processes.

Consistent with the above supposition, we observed that the level of chlorophyll accumulation was elevated in *35S:AtMYB44* seedlings compared to wild-type seedlings (Table 1), suggesting that a jasmonate-related response was down-regulated. It was reported that jasmonates induce chlorophyllase biosynthesis to catalyze chlorophyll degradation (Kariola et al., 2005; Tsuchiya et al., 1999). Furthermore, our previous microarray analyses revealed that genes encoding chlorophyll a/b-binding (CAB) proteins were significantly down-regulated in MeJA-treated (Jung et al., 2007a) and MeJA-overproducing (Jung et al., 2007b) plants. Thus, the enhanced accumulation of chlorophyll in *35S:AtMYB44* plants implies a negative effect of *AtMYB44* overexpression on jasmonate-responsive secondary metabolism.

Significantly less anthocyanin accumulated in *35S:AtMYB44* plants than in wild-type or *atmyb44* knockout plants (Fig. 6). The anthocyanin content of the latter plants was increased significantly in the presence of jasmonic acid or MeJA, while the increment in the anthocyanin content of *35S:AtMYB44* plants was only 40% or 60% in medium containing jasmonic acid or MeJA, respectively, when compared with wild-type plants. Therefore, the jasmonate-induced accumulation of anthocyanin was suppressed in the plants overexpressing *AtMYB44*.

Anthocyanins are water-soluble pigments synthesized through the phenylpropanoid and flavonoid pathways, and they have been implicated as ABA-related pathways (Holton and Cornish, 1995). Treatment with ABA causes anthocyanins to accumulate in the leaves of rice seedlings (Hung et al., 2007).

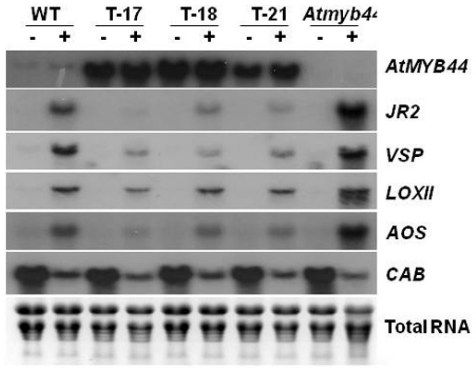

Fig. 5. Northern blot analysis of *35S:AtMYB44* transgenic *Arabidopsis* plants. *AtMYB44* cDNA was fused to the cauliflower mosaic virus 35S (CaMV35S) promoter and transformed into *Arabidopsis* to create the *35S:AtMYB44* T-17, T-18, and T-21 transgenic lines (Jung et al., 2008). The *atmyb44* knockout line (SALK_039074) was obtained from the SALK collection. Five-week-old adult plants were sprayed with (+) or without (-) 50 μM MeJA and their RNA was extracted 6 h later. The cDNA probes used were EST clones obtained from TAIR. Expression of the MeJA-suppressing *CAB* gene, which encodes the chlorophyll a/b-binding protein, was analyzed as a control. Equal RNA loadings were confirmed using ethidium bromide-stained gels.

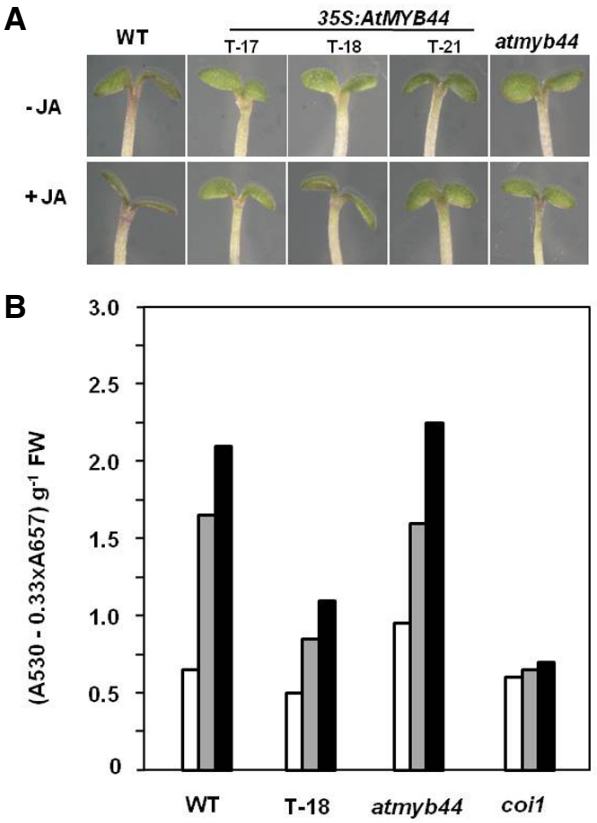

Fig. 6. Accumulation of anthocyanin. (A) Anthocyanin accumulation in the hypocotyls of 3-day-old *Arabidosis* seedlings. *Arabidopsis* seeds were germinated and grown on MS medium without (- JA) or 10 μM jasmonic acid (+ JA). Tissue samples were visualized using an Axiophot microscope (Carl Zeiss, Germany) coupled to a CCD camera. (B) Anthocyanin content of 12-day-old seedlings. Anthocyanin was extracted from 50-80 whole seedlings (approximately 0.2 g) and determined spectrophotometrically. *Arabidopsis* seeds were germinated and grown on MS medium without jasmonates (white bars), with 10 μM jasmonic acid (gray bars), or with 10 μM MeJA (black bars). The data represent average values obtained from two separate experiments that did not differ qualitatively.

In addition, treatment with MeJA causes a substantial decrease in chlorophyll and a significant accumulation of anthocyanin in *Arabidopsis* (Jung, 2004). The anthocyanin content of the jasmonate-insensitive mutant *coi1* was not increased in jasmonate-containing medium (Fig. 6B), demonstrating the effect of jasmonate on anthocyanin accumulation. Thus, the lower level of anthocyanin in *35S:AtMYB44* plants can be attributed to a negative effect of *AtMYB44* overexpression on the MeJA-responsive signaling pathway and a positive effect on the ABA-responsive pathway.

Primary root growth of *Arabidopsis* seedlings is inhibited when seedlings are grown on agar medium containing 0.1 μM MeJA (Staswick et al., 1992). Mutants showing decreased sensitivity to MeJA inhibition of root elongation, including *jar1* (Staswick et al., 1992), are thought to have a defective jasmonate signaling pathway. JAR1 catalyzes the formation of the jasmonoyl-isoleucine (JA-Ile) conjugate (Staswick and Tiryaki, 2004). The JA-Ile conjugate promotes physical interactions between COI1 and JAZ1, promoting the binding of SCF^{COI1} ubiquitin ligase to the JAZ1 repressor protein as well as its subsequent degradation; this in turn negatively regulates the key transcriptional activator of jasmonate responses, MYC2 (Chini et al., 2007; Thines et al., 2007). However, *35S:MYB44* and *atmyb44* mutants did not show defective or enhanced primary root growth inhibition (data not shown). This result suggests that the suppression of MeJA-responsive gene expression observed in *35S:AtMYB44* plants was not due to alterations in the jasmonate signaling pathway, and that AtMYB44 does not integrate jasmonate-mediated signaling.

We previously reported that AtMYB44 transcription factor plays a role in the ABA-mediated signaling pathway (Jung et al., 2008). The expression of PP2C genes including those encoding ABI1, ABI2, AtPP2CA, HAB1, and HAB2 (which have been described as negative regulators of ABA signaling) was reduced in salt-treated *35S:AtMYB44* plants. In contrast, the present study shows that AtMYB44 is also a negative regulator of jasmonate-responsive gene expression. The MeJA-induced suppression of jasmonate-responsive gene activation in the *35S:AtMYB44* transgenic plants might be due to the indirect antagonistic effects of ABA-responsive signaling activated by AtMYB44 overexpression. Taken together, our data support the idea that jasmonates and ABA act antagonistically to regulate the expression of stress-inducible genes (Anderson et al., 2004; Jung et al., 2007a; 2007b; Moons et al., 1997).

Our previous microarray analysis showed that in the absence of salt or jasmonate treatment, the overall gene expression patterns of *35S:AtMYB44* plants are not significantly different from those of wild-type plants (Jung et al., 2008). Thus, AtMYB44 overproduction in itself does not appear to be sufficient to suppress the expression of a group of specific target genes. Rather, this transcription factor may function either through stimulus-induced structural modification or by working cooperatively with other transcription factors. Further studies to identify the target genes of the AtMYB44 transcription factor, its binding sites on promoters, and its interacting proteins should clearly define the role(s) of AtMYB44 in the antagonistic interaction between jasmonate and ABA in gene activation processes.

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