Expression of the *Arabidopsis* jasmonate signalling repressor *JAZ1/TIFY10A* is stimulated by auxin

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Plant hormones have pivotal roles in almost every aspect of plant development. Over the past decades, physiological and genetic studies have revealed that hormone action in plants is determined by complex interactions between hormonal signalling pathways. Evidence is accumulating for the existence of crosstalk between the auxin and jasmonate (JA) signalling pathways. Recently, the JASMONATE ZIM-domain (JAZ) proteins have been identified as the long-sought repressors of JA signalling. Here, we show that expression of *JAZ1/TIFY10A* is not solely inducible by JA, but that it is also an early auxin-responsive gene. Furthermore, we could show that the auxin-inducible expression of *JAZ1/TIFY10A* is independent of the JA signalling pathway but is controlled by the auxin/indole-3-acetic acid-auxin response transcription factor signalling pathway. Our results provide evidence for the existence of at least two different input signals regarding *JAZ1/TIFY10A* expression and thus support the idea of an intimate molecular interplay between auxin and JA signalling.

Keywords: JAZ; MeJA; NLS; TIFY; ARF

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INTRODUCTION

Plant hormones are implicated in every aspect of plant growth and development, as well as in the response to environmental cues. Both positive and negative regulators are involved in hormonal signal-transduction pathways to optimize the efficiency of the stimulus (Ulmasov *et al*, 1999; Ikeda *et al*, 2001; Sasaki *et al*, 2003). These regulators are often targeted for degradation by the ubiquitin–proteasome pathway, which is orchestrated by

Skp1–Cullin–F-box (SCF)-type E3 ubiquitin ligases. The SCF transport inhibitor response 1 (SCF^{TIR1}) complex has a central role in the signalling of the plant hormone auxin by targeting auxin/indole-3-acetic acid (Aux/IAA) proteins on auxin response (Dharmasiri *et al*, 2005; Kepinski & Leyser, 2005). Aux/IAAs are short-lived nuclear proteins that dimerize auxin-response transcription factors (ARFs). The proteasome-mediated degradation of Aux/IAAs results in the release of ARFs, which subsequently can interact with DNA motifs, designated auxin -responsive elements, to activate or repress auxin target genes (Ulmasov *et al*, 1999). Similar SCF-mediated regulation has been shown for gibberellic acid, ethylene and jasmonates—jasmonate (JA) and other oxylipin derivatives (for a review, see Yu *et al*, 2007)—of which the jasmonates' signalling pathway is most reminiscent of that of auxin. JASMONATE ZIM-domain (JAZ) proteins belonging to the larger family of the TIFY proteins (Vanholme *et al*, 2007) act as repressors of JA signalling (Chini *et al*, 2007; Thines *et al*, 2007). Perception of bioactive JAs by the F-box protein CORONATINE INSENSITIVE 1 (COI1) causes degradation of JAZ proteins through the ubiquitin–proteasome pathway, which, in turn, activates the expression of JA-responsive genes.

Besides the overall similarity between both pathways, physiological and genetic studies have suggested a complex and little understood crosstalk (Kazan & Manners, 2008). For example, treating *Arabidopsis* plants for 48 h with methyljasmonate (MeJA) resulted in a significant increase of free IAA levels (Dombrecht *et al*, 2007). Conversely, ARF6 and ARF8 have been shown to promote JA production in *Arabidopsis* flowers (Nagpal *et al*, 2005). In this report, we provide new insight into this close molecular crosstalk by showing that *JAZ1/TIFY10A* is an auxin-responsive gene. Furthermore, the auxin-inducible expression of *JAZ1/TIFY10A* is controlled by ARFs and does not depend on the JA signalling pathway.

RESULTS AND DISCUSSION

Identification of *JAZ1/TIFY10A*

When studying the interaction between plants and plant-parasitic nematodes, we previously identified the Att0728 line by a promoter trap experiment (Barthels *et al*, 1997). Besides the reported β -glucuronidase (GUS) activity in the developing feeding

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Fig 1 | Tissue-specific and hormone-inducible *JAZ1/TIFY10A* expression. (A–D) *GUS* expression in the Att0728 promoter trap (A,B) and TIFY10A::GUS (C,D) lines during LR initiation (A,C) and at the base of the LR after emergence (B,D). (E) Reverse transcription–PCR gel blot showing the transcript level of *At1g19180*, *At1g19170* and *ACTIN2* in C24 WT and the homozygous Att0728 line. (F–K) TIFY10A::GUS expression in mature root (F–H) and primary root meristem (I–K) on mock (F,I), auxin (10 μ M NAA, 2 h; G,J) and jasmonate (25 μ M MeJA, 2 h; H,K) treatment. GUS, β -glucuronidase; LR, lateral root; WT, wild type.

Fig 2 | Subcellular localization of JAZ1/TIFY10A–GFP in tobacco BY2 cells. (A) Schematic representation of JAZ1/TIFY10A protein regions fused with GFP (amino acids are indicated in parentheses). (B) GFP signal in BY2 cells (inset, free GFP). (C) Close-up of nuclear GFP localizations. Arrows indicate nuclear bodies. Scale bars, 10 μ m. BY, Bright Yellow; GFP, green fluorescent protein; Jas, jasmonate-associated; NTD, amino-terminal domain.

sites of plant-parasitic nematodes (supplementary Fig S1 online; Barthels *et al*, 1997), GUS staining was also observed during lateral root initiation and at the base of lateral root primordia (Fig 1A,B). By using inverse PCR, we show here that the Att0728 line carries a transfer DNA (T-DNA) insertion in the promoter region of the *At1g19180* gene 1.4 kb upstream from the predicted coding region (supplementary Fig S2 online). The Att0728 line showed a highly reduced *At1g19180* expression level compared with that of the corresponding wild type (Fig 1E). Furthermore, a 1.4-kb *At1g19180* promoter fragment fused to the *GUS* gene showed essentially the same GUS pattern as the Att0728 line; that is, during lateral root initiation and at the base of lateral roots (Fig 1C,D). We therefore concluded that *At1g19180* was trapped and that its promoter controlled the expression of the GUS-containing T-DNA in the Att0728 line.

JAZ1/TIFY10A is localized in nuclear bodies

The *At1g19180* gene product was initially catalogued as TIFY10A, a member of the plant-specific TIFY family (Vanholme *et al*, 2007). Shortly thereafter a subset of TIFY proteins was classified as JAZ proteins (Chini *et al*, 2007; Thines *et al*, 2007), leading to the double nomenclature JAZ1/TIFY10A. Previously, a nuclear localization has been shown for several TIFY proteins (Nishii *et al*, 2000; Chini *et al*, 2007; Thines *et al*, 2007; Yan *et al*, 2007), but a nuclear localization signal (NLS) had hitherto not been defined. In an effort to identify the essential region for nuclear import, we set up a deletion screen for JAZ1/TIFY10A in tobacco (*Nicotiana tabacum*) Bright Yellow 2 cells (Fig 2). The full-length construct as well as deletion constructs $\Delta 1$, which lack the amino-terminal domain, and $\Delta 3$, which has only the carboxy-terminal region, all showed a strong nuclear localization. However, with $\Delta 3$, green fluorescent protein (GFP) fluorescence was observed in the

nucleolus, indicating that the NLS is still present but that essential nucleoplasmic retention signals are missing, leading to the accumulation of the basic protein in the nucleolus (Meng *et al*, 2007). Deletion construct $\Delta 2$, which lacks the N-terminal and C-terminal regions, was localized both in the nucleus and in the cytoplasm, a pattern also observed when expressing free GFP. These results indicate that the C-terminal region is responsible for the nuclear localization of JAZ1/TIFY10A. This region contains a stretch of basic amino acids conserved among the JAZ proteins. Although NLSs are characterized by basic amino acids, this basic motif alone was not sufficient to function as an NLS. Neither $\Delta 4$ nor $\Delta 5$ directed GFP specifically to the nucleus (Fig 2). By using construct $\Delta 6$, we could show that five additional non-basic amino acids restored the NLS. This observation is consistent with earlier reports (Boulikas, 1994; Sekimoto *et al*, 2004) and reveals that the function of the basic core of an NLS is influenced by its flanking residues. Recently, by using both yeast two-hybrid and protein pull-down experiments the C-terminal region (designated as the Jas motif; Yan *et al*, 2007) has been shown to interact with COI1 in a JA-Ile-dependent manner (Katsir *et al*, 2008; Melotto *et al*, 2008). Together with our findings a dual role for the Jas motif can be put forward: one role in protein–protein interactions and one role in subcellular protein sorting.

In planta expression of truncated versions of *JAZ1/TIFY10A*, *JAZ3/TIFY6B* and *JAZ10/TIFY9* lacking the Jas motif showed JA insensitivity, phenocopying *coi1* mutants (Chini *et al*, 2007; Thines *et al*, 2007; Yan *et al*, 2007). These results indicate a role for the N-terminus and/or tify domain in regulating the expression of JA-responsive genes. An interesting observation from our deletion screen was that the full-length JAZ1/TIFY10A protein and construct $\Delta 1$ were localized in the nucleus as discrete speckles, often referred to as nuclear bodies (Fig 2). As the speckled pattern was not observed with constructs lacking the tify domain, it is tempting to speculate that this domain is involved in this patterning, suggesting that it can be designated as a nucleoplasmic retention signal. Recently, Chung & Howe (2009) showed that the tify motif mediates homomeric and heteromeric interactions between JAZ proteins, which is in agreement with the model of Meng *et al* (2007) that suggests that protein–protein interactions are necessary for the localization of proteins to the nucleoplasm.

Knocking down *TIFY10A* enhances sensitivity to MeJA

Recently, *JAZ1/TIFY10A*, together with its closest homologues, has been shown to act as a repressor of JA signalling, and constitutive expression of a truncated JAZ1 derivative creating dominant-negative mutants has been shown to promote JA insensitivity (Thines *et al*, 2007). The lack of knockout mutants for *JAZ1/TIFY10A* prevented the study of the endogenous full-length protein. We used the Att0728-tagged line to tackle this problem. In addition, artificial microRNA (amiRNA) lines were constructed (supplementary Fig S3 online). The *JAZ1/TIFY10A* knockdown lines were grown on several MeJA concentrations and primary root growth and lateral root density were analysed. These characteristics have been extensively used to screen for JA-insensitive mutants (Chini *et al*, 2007; Thines *et al*, 2007; Yan *et al*, 2007). Both Att0728 and amiRNATIFY10A plants showed a stronger response to MeJA compared with their corresponding controls: primary root growth was more inhibited (Fig 3A),

Fig 3 | *JAZ1/TIFY10A* mutant phenotypes. (A) Root length of *TIFY10* knockdown and knockout plants compared with their respective controls grown for 9 days on MeJA-containing medium. (B) Number of lateral roots per centimetre root length of Col-0 WT and amiRNATIFY10A seedlings grown for 9 days on NAA and MeJA-containing medium compared with mock-treated plants. (C) qRT-PCR analysis of the expression of *AOS*, *LOX3* and *MYC2* in Col-0 WT and amiRNATIFY10A plants. Averaged transcript levels are plotted relative to the WT control. Error bars indicate standard error; asterisks in (A,B) mark significant differences as compared with the respective controls (* $P < 0.05$; ** $P < 0.01$). MeJA, methyljasmonate; NAA, naphthalene acetic acid; qRT, quantitative real time; WT, wild type.

whereas the number of lateral roots increased significantly (Fig 3B). In addition, the level of transcripts of three molecular JA markers was higher in *JAZ1/TIFY10* knockdown lines (Fig 3C). These results indeed show that *JAZ1/TIFY10A* is one of the JAZ repressors involved in JA signalling.

The observation that full knockout lines of *JAZ2/TIFY10B*, *JAZ5/TIFY11A*, *JAZ7/TIFY5B* and *JAZ9/TIFY7* did not alter JA signalling suggested functional redundancy among the JAZ proteins (Thines *et al*, 2007). To investigate whether *JAZ1/TIFY10A* would be functionally redundant with its closest homologue *JAZ2/TIFY10B*, we generated double mutants between *Att0728* and a *tify10b* full knockout line (supplementary Fig S3

Fig 4 | *JAZ1/TIFY10A* expression is induced by auxin. (A) Time-course experiment of *JAZ1/TIFY10A* expression in auxin-treated seedlings (10 μM NAA). (B) *JAZ1/TIFY10A*, *Aux/IAA1*, *Aux/IAA5* and *Aux/IAA19* expression in WT seedlings treated for 30 min with increasing auxin concentrations. (C) Expression of *JAZ1/TIFY10A* in WT, *arf6*, *arf8* and *arf6arf8* backgrounds on mock, NAA (10 μM 30 min), MeJA (25 μM 30 min) and NAA + MeJA treatments (* $P < 0.01$; ** $P < 0.001$). (D) *JAZ1/TIFY10A* expression in mock and auxin-treated WT, *HS::axr3* and *HS::shy2* backgrounds. MeJA, methyljasmonate; NAA, naphthalene acetic acid; WT, wild type.

online). Surprisingly, no extra response could be seen when comparing *Att0728* and *tify10b* × *Att0728* (Fig 3A), indicating that *TIFY10A* and *TIFY10B* are not redundant and that further sensitivity to MeJA is probably countered by other JAZ proteins.

***JAZ1/TIFY10A* interconnects JA and auxin signalling**

A detailed analysis of the *TIFY10A* promoter showed the presence of several AREs (supplementary Fig S2 online). Owing to the presence of these elements, transcription can be activated within minutes on application of auxin (Katagiri et al, 1989; Singh et al, 1990; Ulmasov et al, 1995). Furthermore, data from AtGenExpress (Goda et al, 2008), in which the transcriptional response of seedlings to seven phytohormones was recorded, showed an induced *JAZ1/TIFY10A* expression on a 30-min auxin treatment and suggested that *JAZ1/TIFY10A* is the only early auxin-inducible JAZ gene (supplementary Fig S4 online). Auxin-inducible expression of *JAZ1* was also observed by Vanneste et al (2005; supplementary Fig S5 online), but not by Nemhauser et al (2004) and Peng et al (2009). As auxin induction of *JAZ1* was not consistent in various microarray experiments, expression of the *JAZ1/TIFY10A* gene was tested for its response to changes in auxin level (by using 10 μM naphthalene acetic acid (NAA)) in a time-course experiment using quantitative real-time polymerase chain reaction (qRT-PCR). This analysis indeed showed a very early and transient upregulation of the *JAZ1/TIFY10A* transcript (Fig 4A). To investigate whether lower, more physiologically

relevant auxin concentrations could regulate *JAZ1* expression, we carried out an auxin dose-response study. Upregulation of *JAZ1* in seedlings treated for 30 min with NAA could be observed from a concentration of 1 μM onwards, similar to the behaviour of *Aux/IAA* genes (Fig 4B). The auxin-induced promoter activity was confirmed using the *TIFY10A::GUS* lines. In contrast to non-treated *TIFY10A::GUS* lines, strong GUS staining could be observed in the vascular cylinder (Fig 1F,G,I,J; supplementary Fig S6 online). Interestingly, the auxin response differed from the MeJA response. By using the latter hormone, upregulation of *JAZ1/TIFY10A* could be observed in almost all root cells (Fig 1H,K).

Given the fast upregulation of the *JAZ1/TIFY10A* transcript on auxin treatment, we considered that the *Aux/IAA*–ARF signalling pathway might regulate *JAZ1/TIFY10A* expression. Previously, ARF6 and ARF8 were reported to promote JA production in flowers (Nagpal et al, 2005). Therefore, we checked the expression of *JAZ1/TIFY10A* in *arf6* and *arf8* single mutants as well as in the *arf6arf8* sesquimutant (*arf6arf6 arf8/ARF8*). In both *arf6* and *arf8*, the auxin-induced expression was highly reduced and, interestingly, no *JAZ1/TIFY10A* auxin response could be observed in the sesquimutant (Fig 4C), suggesting that ARF6 and ARF8 both mediate the auxin-induced expression of *JAZ1/TIFY10A*. In none of the *arf* mutant backgrounds was the MeJA-induced expression of *JAZ1/TIFY10A* affected (Fig 4C). In addition, expression of *JAZ1/TIFY10A* was analysed in the *aux/iaa* mutants *HS::axr3/iaa17* and *HS::shy2/iaa3* on auxin treatment. In both

Fig 5 | Auxin-induced expression of *JAZ1/TIFY10A* is independent of MeJA signalling. (A) Visualization of *JAZ1/TIFY10A*-GFP on MeJA (25 µM) and NAA (10 µM) treatment. (B) Expression of *JAZ1/TIFY10A* in Col-0 WT, *coi1-16* and *myc2* backgrounds on mock, NAA (10 µM), MeJA (25 µM) and NAA + MeJA treatments (*P < 0.01; **P < 0.001). MeJA, methyljasmonate; NAA, naphthalene acetic acid; WT, wild type.

mutant backgrounds, the auxin-induced *JAZ1/TIFY10A* expression was abolished on heat-shock treatment (Fig 4D).

To exclude the possibility that the auxin response of *JAZ1/TIFY10A* is because of a stimulation of MeJA signalling, we generated transgenic plants producing a *JAZ1/TIFY10A*-GFP fusion protein driven by the 35S promoter and checked its stability in the presence of NAA and MeJA. As expected, the GFP signal rapidly disappeared when transferred to MeJA-containing medium; however, when seedlings were treated with NAA, no degradation of *JAZ1/TIFY10A*-GFP could be observed (Fig 5A). These results indicate that auxin does not influence the stability of *JAZ1/TIFY10A* and suggest that auxin and JA act differently with regard to the transcriptional upregulation of *JAZ1/TIFY10A*. Next, we checked the expression of *JAZ1/TIFY10A* in the MeJA signalling mutants *coi1-16* and *myc2* on treatment with NAA and MeJA. In the *coi1-16* background, the MeJA response was highly reduced compared with wild type, whereas the auxin-dependent induction of *JAZ1/TIFY10A* was unaffected (Fig 5B). Similar results were obtained using the *myc2* mutant: a reduced

MeJA induction and unaffected auxin-induced expression. These findings strongly suggest that auxin-dictated *JAZ1/TIFY10A* expression is independent of the MeJA signalling pathway. To investigate whether the auxin-inducible *JAZ1/TIFY10A* expression is important physiologically, we analysed lateral root outgrowth in response to NAA. In two independent experiments, the number of emerged lateral roots of the amiRNATIFY10A seedlings was found to be slightly but significantly increased relative to wild type in the presence of 0.1 µM NAA (Fig 3B). *JAZ1/TIFY10A* knockdown mutants thus show an increased lateral root formation on treatment with MeJA as with auxin. *JAZ1/TIFY10* is known to bind to and inhibit the activity of MYC2, which, interestingly, was reported as a positive regulator of lateral root formation (Yadav et al, 2005). Together with the observed lateral root-specific *JAZ1/TIFY10A* expression pattern, these results reveal that a modulation of *JAZ1/TIFY10A* expression levels might be used by both MeJA and auxin to control lateral root formation.

The results presented here suggest that the auxin and JA signalling pathways are interconnected through *JAZ1/TIFY10A* (supplementary Fig S7 online). Furthermore, our results can be used to explain some of the observations of Chung et al (2008), in which the expression of three *JAZ* genes was determined in cycloheximide-treated *coi1* mutants. The expression of *JAZ7* and *JAZ10* could not be induced by cycloheximide in *coi1*, showing that SCF^{COI} also contributes to JAZ turnover in the absence of exogenous MeJA. By contrast, the transcript level of *JAZ1/TIFY10A* was upregulated by cycloheximide (Chung et al, 2008). This observation can now be explained by the action of ARF proteins, which are derepressed in the presence of cycloheximide and further supports the fact that auxin-induced expression of *JAZ1/TIFY10A* is independent of JA signalling and seemingly specific to *JAZ1/TIFY10A*.

METHODS

Hormone treatments, RNA extraction and qRT-PCR analysis. For the NAA time-course experiment, 10-day-old seedlings were transferred to mock MS medium or MS medium supplemented with 10 µM NAA (Sigma-Aldrich, St Louis, MO, USA) for 0, 15, 30, 60, 120, 240 and 360 min. For the NAA dose response, plants were incubated for 30 min on medium supplemented with 0, 0.01, 0.1, 1 or 10 µM NAA. For all other hormone treatments, 10-day-old plants were transferred to a medium containing 25 µM MeJA and 10 µM NAA, or 10 µM NAA and 25 µM MeJA, all for 30 min. For heat-shock experiments, 10-day-old plants were incubated for 2 h at 37 °C. Afterwards, plants were incubated for 30 min at 21 °C. Plants were then incubated for 30 min on auxin-containing (NAA 10 µM) or mock medium. After all treatments, plants were ground and total RNA was isolated with TriZol (Invitrogen, Merelbeke, Belgium) according to the manufacturer's instructions. Poly(dT) complementary DNA was prepared from 2 µg total RNA with Superscript III reverse transcriptase (Invitrogen) and quantified on a LightCycler 480 apparatus (Roche Diagnostics, Mannheim, Germany) with the SYBR Green I Master kit (Roche Diagnostics) according to the manufacturer's instructions. Target quantifications were carried out with specific primer pairs designed with the Beacon Designer 4.0 (Premier Biosoft International, Palo Alto, CA, USA). All individual reactions were done in triplicate. Data were analysed with qBase (Hellems et al, 2007). Expression levels were normalized to those of *EEF1α4* and *CDKA*, which showed no

clear systematic changes in C_i value. The primers used to quantify gene expression levels are listed in supplementary Table S1 online. A t -test was carried out manually using the results obtained from qBase.

Root growth analysis. To measure root growth, seedlings were grown vertically on the media mentioned. Nine days after germination, the number of lateral roots was counted using a binocular microscope and the plates were scanned on a colour copier CLC-iR C3200 (Canon, Tokyo, Japan). Root growth was measured using the software programme IMAGE J (<http://rsb.info.nih.gov/ij/>). The data were statistically analysed in SPSS (Chicago, IL, USA; version 16.0) using the Levene test for the analysis of homogeneity of variance and a one-way ANOVA (using Tukey's test with a significance level $P < 0.05$ or $P < 0.01$) for comparison of means.

Supplementary information is available at *EMBO reports* online (<http://www.emboreports.org>).

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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