

SHORT COMMUNICATION

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JUB1 suppresses *Pseudomonas syringae*-induced defense responses through accumulation of DELLA proteins

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ABSTRACT

Phytohormones act in concert to coordinate plant growth and the response to environmental cues. Gibberellins (GAs) are growth-promoting hormones that recently emerged as modulators of plant immune signaling. By regulating the stability of DELLA proteins, GAs intersect with the signaling pathways of the classical primary defense hormones, salicylic acid (SA) and jasmonic acid (JA), thereby altering the final outcome of the immune response. DELLA proteins confer resistance to necrotrophic pathogens by potentiating JA signaling and raise the susceptibility to biotrophic pathogens by attenuating the SA pathway. Here, we show that JUB1, a core element of the GA - brassinosteroid (BR) - DELLA regulatory module, functions as a negative regulator of defense responses against *Pseudomonas syringae* pv. *tomato* DC3000 (*Pst* DC3000) and mediates the crosstalk between growth and immunity.

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Arabidopsis; defense; DELLA proteins; gibberellin; jasmonic acid; pathogens; salicylic acid; transcription factor

Introduction

The final outcome of a plant-pathogen interaction depends on the competence of the host to recognize the pathogen and trigger an appropriate, rapid defense response. Crosstalk between the different hormone signaling pathways plays an essential role in fine-tuning the plant's response against pathogens. Several studies demonstrated the mutually antagonistic interaction of jasmonic acid (JA) and salicylic acid (SA) in the response to pathogens.¹⁻³ JA-mediated signaling elevates the resistance to necrotrophic pathogens such as *Botrytis cinerea* or *Alternaria brassicicola*, whereas the SA pathway is associated with enhanced resistance to (hemi)biotrophs such as *Pseudomonas syringae*.⁴⁻⁷ Thus, elevated resistance to necrotrophs is often correlated with an increased susceptibility to (hemi)biotrophs, and *vice versa*.⁸ Emerging evidence indicates that gibberellic acids (GAs), which are essential growth regulators, also play a central role in the plant's response to pathogen attack. The role of GAs in the regulation of growth and disease response is attributed to the degradation of DELLA proteins, the major suppressors of GA signaling.⁹ It has been shown that DELLA proteins can influence disease outcome by modulating the balance between SA and JA signaling.^{10,11} In *Arabidopsis thaliana*, DELLA proteins amplify JA-dependent defense signaling, while they antagonize SA-mediated defense, thereby promoting resistance to necrotrophic pathogens but increasing susceptibility to biotrophic pathogens.^{10,11}


It has been demonstrated that DELLA proteins interact with jasmonate ZIM domain (JAZ) proteins known as repressors of JA

signaling.^{12,13} In the absence of GA, interaction with DELLA proteins releases the inhibitory effect of JAZ on JA-responsive transcription factors (TFs) such as MYC2, a basic helix-loop-helix (bHLH) TF, thus resulting in the activation of JA-responsive gene expression.¹⁴⁻¹⁶ DELLA-enhanced expression of JA-responsive defense genes, but suppression of SA signaling genes contributes to an enhanced resistance to necrotrophic pathogens and increased susceptibility to biotrophic pathogens.¹⁰

We have recently discovered a novel function of the NAC transcription factor JUNGBRUNNEN1 (JUB1) in balancing growth and stress responses in *Arabidopsis*.¹⁷ We have shown that JUB1 exerts its cellular function partly in a DELLA-dependent manner via the synergistic regulation of brassinosteroid (BR) and GA biosynthesis/signaling coordinated through direct repression of the key GA and BR biosynthesis genes *GA3ox1* and *DWF4*, respectively, and activation of *DELLAs*.¹⁷ Repression of GA and BR biosynthesis by JUB1 results in the accumulation and activation of DELLA proteins in *JUB1* overexpressor (*JUB1-OX*) plants. We showed that the repressive effect of high *JUB1* expression can be fully relieved by concomitant overexpression of *GA3ox1* (*GA3ox1-OX*) and *DWF4* (*DWF4-OX*) in the *JUB1-OX* background (*Triple-OX* lines): the GA and BR deficiency as well as the enhanced abiotic stress tolerance phenotype of *JUB1-OX* plants was reversed to normal in the *Triple-OX* plants, showing the critical contribution of the GA and BR biosynthesis genes to the phenotypes observed in *JUB1-OX* plants.

In the current study, we identify JUB1 as a negative regulator of the defense response against the bacterial (hemi)

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biotrophic pathogen *Pseudomonas syringae* pv. *tomato* DC3000 (*Pst* DC3000). We demonstrate that suppression of the *Pst* DC3000-induced defense response in *JUB1-OX* plants is mainly attributed to the accumulation of DELLA proteins, resulting in potentiation of JA signaling and therefore promotion of the susceptibility to *Pst* DC3000. Our data thus identify *JUB1* as a transcriptional regulator acting at the interface of growth regulation (via affecting GA and BR synthesis/signaling) and the response to pathogens.

Results

Activation of *JUB1* expression by biotrophic pathogens

Publicly available gene expression data (Arabidopsis eFP browser; www.bar.utoronto.ca/efp/cgi-bin/efpWeb.cgi) revealed that *JUB1* expression is induced in response to infection with the biotrophic pathogens *Pseudomonas syringae* pv. *tomato* DC3000 (*Pst* DC3000) and *P. syringae* strain *avrRPM1*. Saga et al. (2012) also showed that expression of *JUB1* is induced upon infection with *Pst* DC3000, and *P. syringae* strain *avrRPT2*, suggesting a role of *JUB1* in regulating plant immune responses.¹⁸

Here, we confirmed induction of *JUB1* by *Pst* DC3000 in Arabidopsis wild-type Col-0 (WT) plants sprayed with *Pst* DC3000 (10^8 cfu/mL), by quantitative RT-PCR (qRT-PCR). *JUB1* expression is highly induced 6 and 24 hours post inoculation (hpi) (Fig. 1A). To analyze the spatial expression of *JUB1*

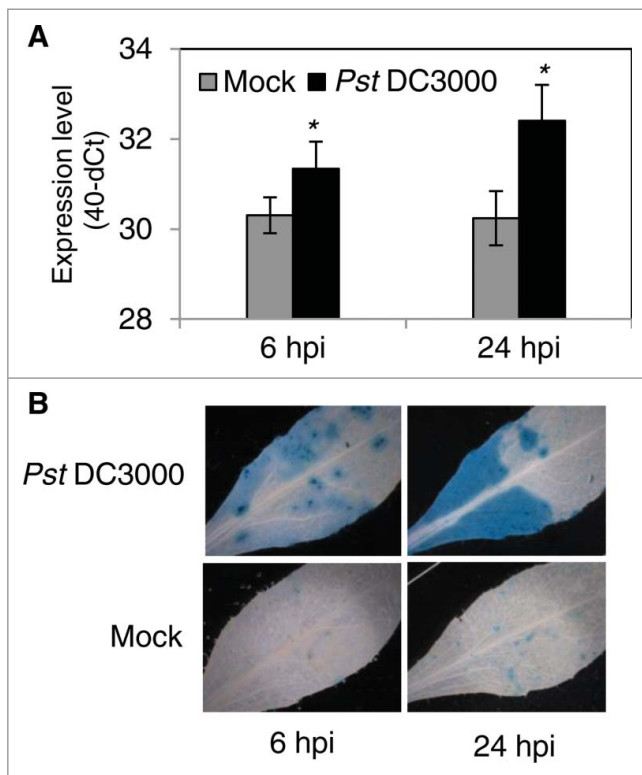


Figure 1. Induction of *JUB1* upon infection with *Pst* DC3000. (A) Expression level of *JUB1* measured by qRT-PCR in 5-week-old WT plants sprayed with *Pst* DC3000 compared to mock (10 mM MgCl₂) treated plants, 6 and 24 hpi. Means \pm SD ($n = 3$). Asterisks indicate a significant difference from Mock ($*p < 0.05$; Student's *t*-test). (B) Histochemical analysis of GUS expression in rosette leaves of 5-week-old *ProJUB1:GUS* plants, 6 and 24 h after pressure infiltration with *Pst* DC3000 or MgCl₂ (mock).

in response to *Pst* DC3000 inoculation, we used transgenic plants expressing GUS under control of the *JUB1* promoter (*ProJUB1:GUS*).¹⁹ Analysis of GUS expression was carried out in leaves infiltrated with *Pst* DC3000 (10^6 cfu/mL) or MgCl₂ (mock) 6 and 24 hpi. GUS activity slightly increased at 6 hpi, but strongly enhanced at 24 hpi in leaves infiltrated with *Pst* DC3000 compared to mock-treated leaves (Fig. 1B), confirming activation of the *JUB1* promoter by *Pst* DC3000 treatment.

JUB1 functions as a negative regulator of the defense against *Pst* DC3000

Our previous study reported that transgenic Arabidopsis plants overexpressing the *JUB1* gene (*JUB1-OX*) showed reduced growth but improved tolerance to different abiotic stresses,^{17,19,20} suggesting its involvement in the crosstalk between plant growth and stress response. We found that the stress- and growth-related phenotypes observed in *JUB1-OX* plants are mainly associated with the negative regulatory impact of *JUB1* on the expression of key enzymes of GA and BR biosynthesis (*GA3ox1* and *DWF4*, respectively) which ultimately results in accumulation and activation of DELLA proteins. To determine whether altered expression of *JUB1* can also affect disease responses to *Pst* DC3000 and whether this is linked to the *JUB1* regulation of growth via DELLA proteins we inoculated *JUB1* transgenic plants (*JUB1-OX*, *jub1-1* knockdown mutant,¹⁹ *Triple-OX*) as well as WT plants with *Pst* DC3000 by both, pressure infiltration and surface inoculation, and monitored the progress of infection over time (Fig. 2). Pressure infiltration of leaves with *Pst* DC3000 (1×10^6 cfu/mL) revealed differential defense responses in *JUB1* transgenic plants. While an increased susceptibility was observed in *JUB1-OX* plants, *jub1-1* and *Triple-OX* plants developed less disease symptoms compared to WT (Fig. 2A). Consistent with these results more bacterial growth was observed in *JUB1-OX* plants at 3 d post inoculation (dpi) compared to WT, but *jub1-1* exhibited a bacterial titer that was lower than that of both, WT and *Triple-OX* plants. The number of bacteria present in *Triple-OX* plants was similar to that of WT (Fig. 2B). Next, we assessed the phenotype of *JUB1* transgenics upon surface inoculation with *Pst* DC3000, which manifests the efficiency of bacteria to enter the plant tissues through the stomatal pores and to cause infection. To this end, plants were spray-inoculated with a bacterial suspension (1×10^8 cfu/mL). Development of disease symptoms was assessed at 5 dpi as disease severity index. While *JUB1-OX* plants developed severe necrotic lesions, *jub1-1* plants exhibited less disease symptoms than both, *Triple-OX* and wild-type plants that show a similar response toward the treatment (Fig. 2C, D). Recovery of the *JUB1-OX* stress response in *Triple-OX* plants confirms that the negative regulation of GA and BR pathways by *JUB1* contributes to the biotic stress response against *Pst* DC3000. Taken together, our data indicate that *JUB1* negatively regulates the defense response against *Pst* DC3000 in conjunction with hormonal networks.

JUB1 regulates defense responses to *Pst* DC3000 mainly through the accumulation of DELLA proteins

To evaluate the role of *JUB1* for the regulation of defense response genes, we profiled the expression of several defense

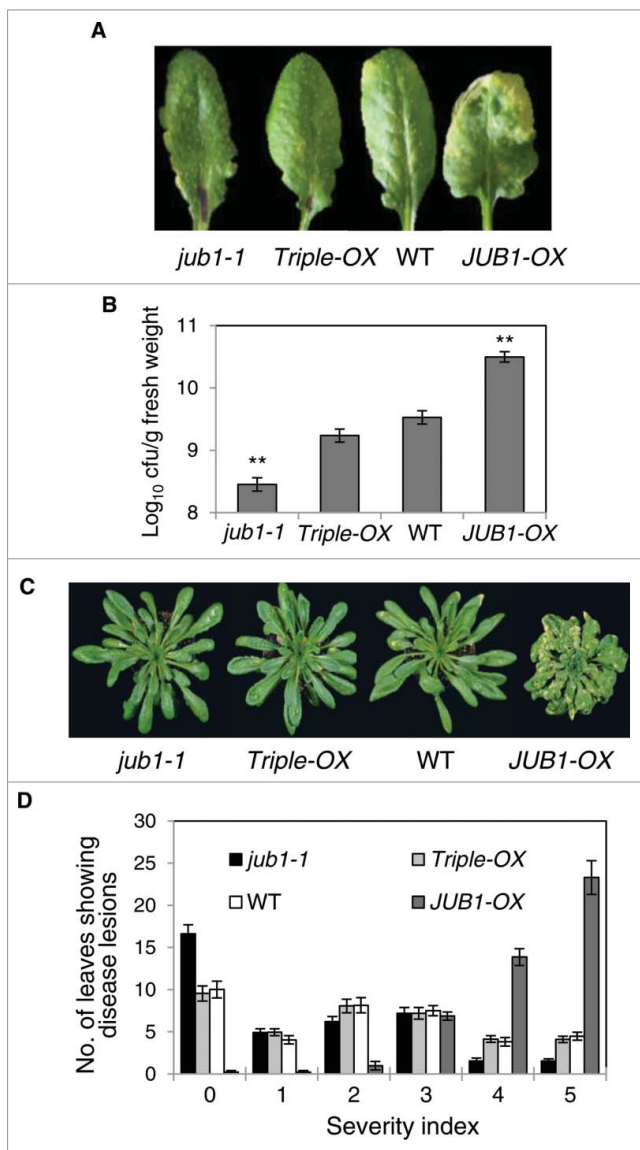


Figure 2. Defense responses observed in *JUB1-OX* are fully restored in *Triple-OX* plants. (A) Rosette leaves of 5-week-old plants pressure infiltrated with *Pst* DC3000 at 2 dpi. The experiments were repeated 3 times and similar results were obtained. (B) *In planta* growth of *Pst* DC3000 bacteria in *JUB1* transgenics and WT, assayed 3 dpi. Data are from 2 experiments with at least 8 plants per genotype in each. Mean growth \pm SD. Asterisks indicate a significant difference from WT (** $p < 0.01$; Student's *t*-test). (C) Defense response of plants sprayed with *Pst* DC3000 at 5 dpi. The experiment was repeated 4 times with similar results. (D) Disease severity index (according to lesion diameters; 0, small chlorotic lesions; 5, large lesions) assessed in plants shown in (C). Data are from 3 experiments with at least 8 plants per genotype in each. Means are shown \pm SD.

responsive genes in 5-week-old *JUB1* transgenic and WT plants sprayed with *Pst* DC3000 at 6 hpi using qRT-PCR. We identified 11 genes differentially expressed between *JUB1* transgenics and WT, including 5 JA- and 6 SA-responsive genes. Expression of the JA-responsive genes *PDF1.2A*, *THI2.1*, *THI2.2*, *ANAC019*, and *ERF1* was enhanced in *JUB1-OX* plants, while expression of these genes was highly or moderately decreased in *jub1-1* and *Triple-OX* plants, except for *THI2.2*, which was not altered (Fig. 3). Elevated expression of JA-responsive genes in *JUB1-OX* plants upon *Pst* DC3000 treatment may indicate the activation of JA-mediated defense responses resulting in the promotion of susceptibility toward *Pst* DC3000. Expression

of the SA-defense marker gene *PR1* was repressed in *JUB1-OX* plants, but induced in *jub1-1* plants, whereas *Triple-OX* plants displayed a WT level of *PR1* expression. Expression of *TGA2* and *TGA5*, which encode TFs that interact with NPR1 and activate *PR1*,²¹ was induced in both *jub1-1* and *Triple-OX* plants, whereas the expression of both genes was close to WT levels in *JUB1-OX* plants. Moreover, expression of *NIMIN2* and *NIMIN3*, negative regulators of the SA-induced defense response that act through their differential interaction with NPR1,²² is induced in *JUB1-OX* plants but repressed or unchanged in *jub1-1* and *Triple-OX* plants. Furthermore, *UGT74F1* transcript level was higher in *jub1-1* and *Triple-OX* plants, while it was slightly decreased in *JUB1-OX* plants. *UGT74F1* encodes an UDP-glucosyl transferase involved in the glycosylation of SA to SA 2-*O*- β -D-glucose (SAG); the *ugt47f1* mutant shows an increased susceptibility to *Pst* DC3000.²³ The expression profile of these genes in *JUB1* transgenic plants clearly reflects an attenuated SA defense pathway, which is either due to direct regulation by *JUB1* or an indirect effect exerted by DELLAs (through direct repression or by activation of JA signaling). To understand this regulation, we analyzed the promoters of the genes differentially expressed between *JUB1* transgenic and WT plants. The absence of *JUB1* binding sites in the promoters of differentially expressed genes suggests their indirect regulation by *JUB1*. Previously we showed that the level of DELLA proteins is enhanced in *JUB1-OX* plants compared to *jub1-1*, *Triple-OX* and WT plants.¹⁷ DELLA accumulation by *JUB1* occurs via suppression of GA biosynthesis and direct transcriptional activation of *DELLA* genes. Indirect regulation of defense responsive genes by *JUB1* further highlights a role for DELLA proteins in the regulation of *JUB1* stress responses against pathogens and the modulation of JA-SA antagonistic crosstalk.

Discussion

Here, we report that the transcription factor *JUB1* regulates disease responses to *Pst* DC3000 likely through modulation of DELLA proteins. *JUB1* expression is induced by the biotrophic pathogen *Pseudomonas syringae* pv. *tomato* DC3000 (*Pst* DC3000) (Fig. 1; eFP browser). Overexpression of *JUB1* results in attenuated resistance to *Pst* DC3000, while the *jub1-1* knock-down mutant and *Triple-OX* plants (which concomitantly overexpress *JUB1*, *GA3ox1* and *DWF4*¹⁷) exhibit reduced disease symptoms and support less bacterial proliferation when surface-inoculated or pressure-infiltrated with *Pst* DC3000 (Fig. 2). Thus, *JUB1* represents a negative regulator of plant defense against *Pst* DC3000. Expression profiling of several SA- and JA-responsive defense genes in *JUB1* transgenics and WT plants upon *Pst* DC3000 treatment displayed differential regulation of 11 genes by *JUB1* (Fig. 3).

Transcript abundance of JA-related defense genes such as *PDF1.2*, *THI2.1*, *ANAC019* and *ERF1* was enhanced in *JUB1-OX* plants, but attenuated in both, *jub1-1* and *Triple-OX* plants. In contrast, expression of SA-marker gene *PR1* was reduced in *JUB1-OX* plants, while it was increased in *jub1-1* plants. Accordingly, transcript levels of *TGA2* and *TGA5*, the positive transcriptional regulators of *PR1*, were enhanced in *jub1-1* and *Triple-OX* plants. Moreover, transcript levels of *NIMIN2* and *NIMIN3*,

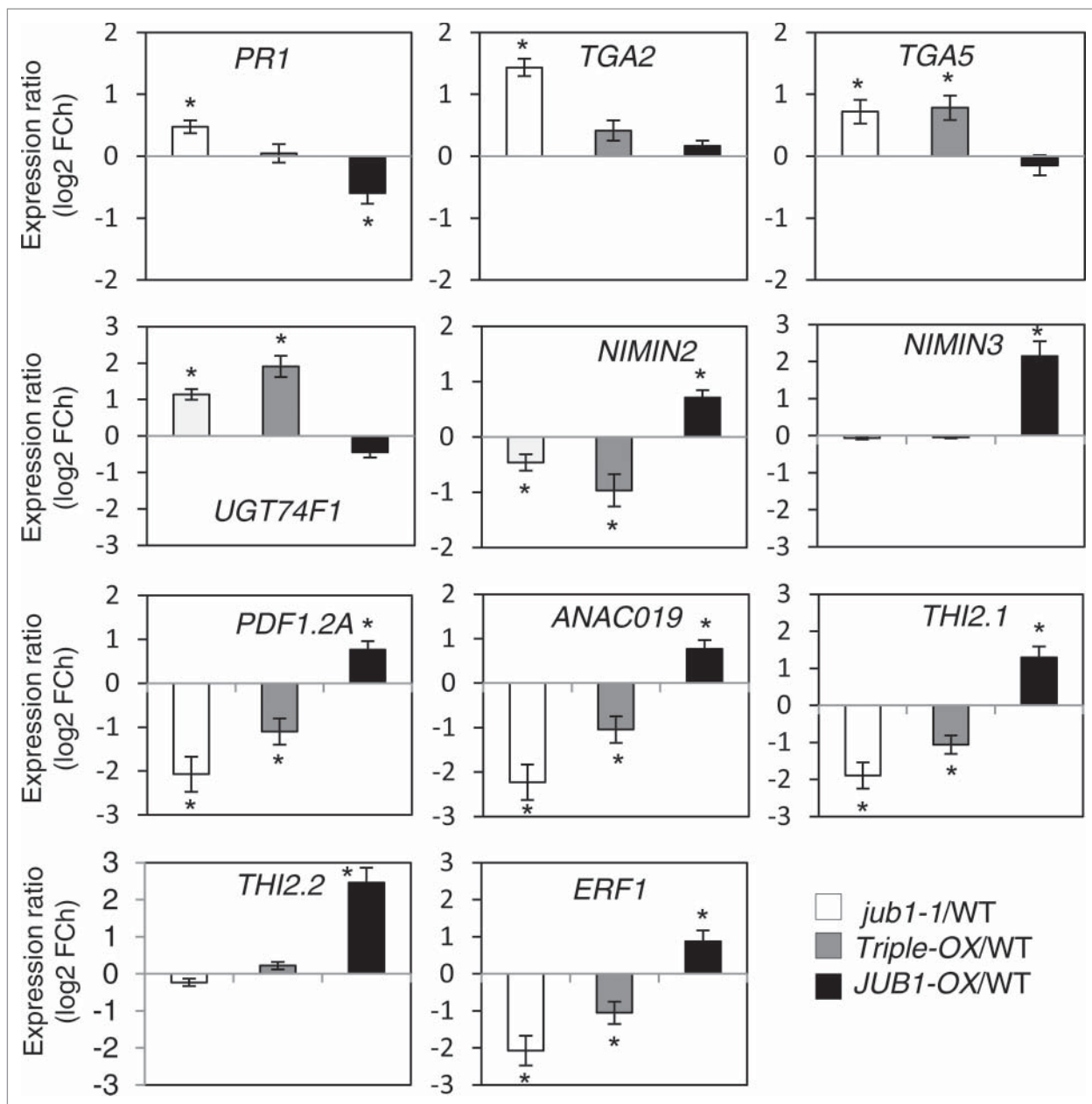


Figure 3. SA- and JA-responsive defense genes differentially expressed in *JUB1* transgenic plants compared to WT. Gene expression was quantified by qRT-PCR in rosettes of 5-week-old *JUB1* transgenics compared to WT, 6 h after spraying with *Pst* DC3000. FCh, fold change. Means \pm SD ($n = 3$). Asterisks indicate a significant difference from WT ($*p < 0.05$; Student's *t*-test).

which encode the negative regulators of *PR1*, were enhanced in *JUB1*-OX plants, but repressed in *jub1-1* and *Triple OX* plants. Such patterns of gene expression indicate that *JUB1* acts as a negative regulator of the SA pathway in response to *Pst* DC3000 infection. The SA-mediated pathway is crucial for the establishment of an efficient defense response against *Pst* DC3000. SA exists in free or conjugated forms, which involves glycosylation or methylation.²⁴ Induced expression of *UGT74F1*, a UDP-glucosyl transferase that is involved in the glycosylation of SA, was observed in both, *jub1-1* and *Triple-OX* plants, while its transcript level was decreased in *JUB1*-OX plants. Recently, Boachon *et al.* (2014)²³ showed that the *ugt74f1* mutant displays an increased susceptibility to *Pst* DC3000.

Although *Pst* DC3000 elevates both, JA and SA levels, the balance between these hormonal pathways is critical for

establishing the output of the plant-pathogen interaction.²² Transcript changes observed in *JUB1* transgenics clearly demonstrate that the JA pathway is favored over SA signaling leading to increased *Pst* DC3000 susceptibility in *JUB1*-OX plants. The JA-mediated defense pathway is crucial for the plant's response to necrotrophic pathogens. A previous study by Saga *et al.* (2012)¹⁸ showed that *jub1* mutant lines were highly susceptible to infection by the necrotrophic fungus *Alternaria brassicicola* in part due to a defect in camalexin biosynthesis. Activation of JA-related defense genes may be a further mechanism through which *JUB1* exerts the resistance against necrotrophs.

Recently, an antagonistic interaction of JA and GA was observed in the regulation of hypocotyl elongation, flowering, root growth as well as the defense response against

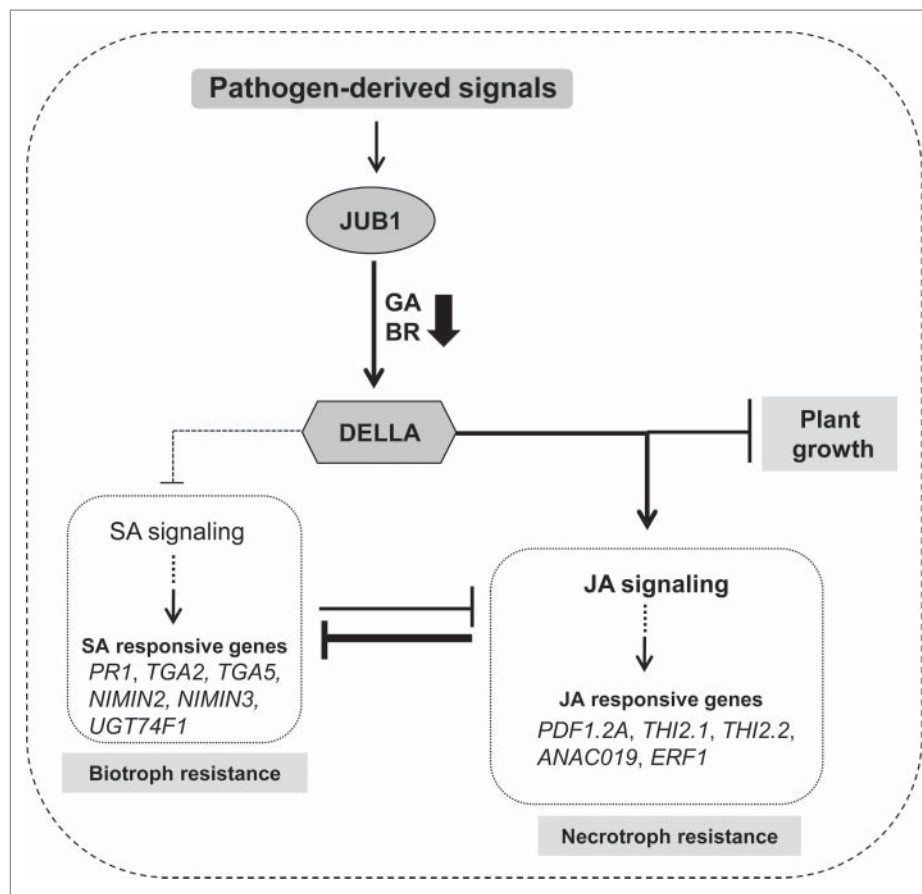


Figure 4. Model showing the function of JUB1 during pathogen attack. A working model showing the JUB1-mediated defense response mechanism; *JUB1* expression is induced by various pathogen-derived signals, and it mediates the plant's response to pathogen attack through accumulation of DELLA proteins resulting in activated JA defense signaling, but an attenuated SA defense response leading to enhanced susceptibility to *Pst* DC3000. This study also emphasizes the role of JUB1 for the regulation of plant growth and defense crosstalk.

hemibiotrophic and necrotrophic pathogens.^{8,11–13,25} These reports suggest a role for both, GA and JA in the regulation of the tradeoff between plant growth and stress response where the interaction between DELLAs and JAZ proteins determines the output. Activation of JA signaling requires the release of JA-activated TFs such as MYC2 from the JAZ-mediated repression.¹⁴ Interaction of DELLAs with JAZ proteins leads to release of repression on JA-activated TFs, resulting in an activated JA response.¹² Recently, we reported JUB1 as a core component of a regulatory module that triggers the accumulation of DELLA proteins.¹⁷ Activated JA signaling in *JUB1* overexpressors upon *Pst* DC3000 infection can be attributed to increased levels of DELLA proteins in these plants,¹⁷ while reduced levels of DELLA proteins in *jub1-1* and *Triple-OX* plants may result in attenuated JA signaling upon *Pst* DC3000 infection. Interestingly, Wild et al. (2012)⁸ reported that the *rgl3-5* mutation enhances susceptibility to *Botrytis cinerea* but resistance to *Pseudomonas syringae*. Like in *jub1-1* plants, *ERF1* and *PDF1.2* transcript levels were lower in infected *rgl3-5* plants than in wild-type plants. JAZ proteins interact with various transcription factors, including EIN3, a positive regulator of *ERF1* and *PDF1.2*.²⁶ The competitive binding of DELLA proteins to JAZ proteins releases repression on EIN3 (or other JAZ-interacting factors). Consistently, overexpression of *RGL3* enhances expression of EIN3-regulated genes,

including *ERF1* and *PDF1.2*, in transgenic Arabidopsis plants.^{27–29} In addition, JA-mediated induction of *PDF1.2* was reduced in the *rgl3-5* mutant in comparison to wild-type plants,^{27–29} whereas strong and earlier induction of *PR1* and *PR2* expression was reported in the *quadruple della* mutant compared to WT plants.¹¹ These findings indicate that DELLAs play a critical role in modulating the defense response to *Pst* DC3000 by activating JA responses through their differential interaction with JAZ proteins and repressing the SA defense pathway.

Collectively, our study indicates that JUB1 participates in the SA-JA antagonism by favoring JA signaling over the SA pathway, mainly due to the accumulation of DELLA proteins (Fig. 4), leading to attenuated resistance against *P. syringae* pv. *tomato* DC3000, but enhanced resistance to necrotrophic fungi such as *Alternaria brassicicola*.

Material and methods

Plant materials and growth conditions

The *Pro_{JUB1}:GUS*, *jub1-1* (SALK ID 036474), *JUB1-OX* and *Triple-OX* lines were reported previously.^{17,19} Seeds of *Arabidopsis thaliana* were germinated on half-strength Murashige and Skoog (MS) agar medium containing 1% (w/v) sucrose. Ten-day-old seedlings

grown on sterile media were transferred to pots containing soil and were grown at 22°C under an 8-h light/16-h dark cycle.

Treatments

For pathogen treatment, *Pst* DC3000 was grown on antibiotics-supplemented King's B medium at 28°C for 2 days.³⁰ *Pst* DC3000 infection was carried out according to Percheip et al. (2006).³¹ Briefly, bacterial cells were collected by centrifugation (2,500 g), and resuspended in 10 mM MgCl₂. Pressure infiltration of 5-week-old plants was performed using a needleless syringe with *Pst* DC3000 (OD_{600nm} = 0.002; 10⁶ cfu/mL). Bacterial growth (cfu/g fresh weight⁻¹) was measured at 3 dpi as described previously.³¹ For surface inoculation, 5-week-old plants were sprayed with a suspension of *Pst* DC3000 (OD_{600 nm} = 0.2; 10⁸ cfu/mL) containing 0.02% (v/v) Silwet L-77 and kept under a plastic cover. The disease severity index was scored according to Kover et al. (2005).³²

Gene expression analysis

Procedures for total RNA extraction, synthesis of cDNA, and qRT-PCR using SYBR Green (Life Technologies) as well as sequences of gene-specific primers were reported previously.³³ Real-time qPCR was performed using an ABI PRISM 7900 HT sequence detection system (Applied Biosystems Applied). Expression levels were normalized against the expression level of *ACTIN2*. The AGI codes and primer sequences of defense responsive genes used in this study are provided in Table S1.

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Disclosure of potential conflicts of interest

No potential conflicts of interest were disclosed.

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