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Review article

Review: Losing JAZ4 for growth and defense

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ABSTRACT

JAZ proteins are involved in the regulation of the jasmonate signaling pathway, which is responsible for various physiological processes, such as defense response, adaptation to abiotic stress, growth, and development in Arabidopsis. The conserved domains of JAZ proteins can serve as binding sites for a broad array of regulatory proteins and the diversity of these protein-protein pairings result in a variety of functional outcomes. Plant growth and defense are two physiological processes that can conflict with each other, resulting in undesirable plant trade-offs. Recent observations have revealed a distinguishing feature of JAZ4; it acts as negative regulator of both plant immunity and growth and development. We suggest that these complex biological processes can be decoupled at the JAZ4 regulatory node, due to prominent expression of JAZ4 in specific tissues and organs. This spatial separation of actions could explain the increased disease resistance and size of the plant root and shoot in the absence of JAZ4. At the tissue level, JAZ4 could play a role in crosstalk between hormones such as ethylene and auxin to control organ differentiation. Deciphering biding of JAZ4 to specific regulators in different tissues and the downstream responses is key to unraveling molecular mechanisms toward developing new crop improvement strategies.

1. Introduction

Plant metabolic processes are tightly regulated by hormone signaling pathways that have been extensively studied in recent decades in efforts to decipher their mechanisms of action. In 2007, the JAZ (JASMONATE ZIM DOMAIN) co-receptor for hormone jasmonoyl-*L*-isoleucine (JA-Ile) was discovered (Chini et al., 2007; Thines et al., 2007; Yan et al., 2007), and, two years later, COI1 (CORONATINE INSENSITIVE 1) was biochemically proven to be a JA-Ile receptor (Yan et al., 2009). The crystal structure of the co-receptor complex was defined shortly after (Sheard et al., 2010). These seminal studies revealed that a co-receptor complex is formed by the proteins COI1 and JAZs, where JA-Ile serves as a "molecular glue" bringing them together. COI1 is the F-box component of the Skp1/Cullin1/F-box protein ubiquitin E3 ligase (SCF^{COI1}), while JAZ belongs to a family of transcriptional repressor proteins. After JA-Ile recognition, the JAZ protein undergoes proteolytic degradation by the 26 S proteasome, causing transcriptional activation of JA-responsive genes (Pauwels and Goossens, 2011; Wager and Browse, 2012; Zhang et al., 2017) such as *MYC2* and *JAZ* as part of feedback loop mechanisms (Chung et al., 2008; Moreno et al., 2013). However, variations in this JA/COI1/MYC2 canonical pathway exist. For instance, other bioactive forms of JA-Ile, such as its stereoisomer (3 R,7 S)−12-Hydroxy-JA-Ile, also bind to COI-JAZ9 and partially induce the expression of *MYC2* and *JAZ1/9*, but not *JAZ8/10* (Saito et al., 2023). In addition, COI1 functions independently of JA-Ile, contributing to susceptibility of Arabidopsis to *Verticillium longisporum* (Ulrich et al., 2021).

It is widely accepted that the JA-Ile signaling pathway is regulated by members of the JAZ family (Zhai et al., 2015; Chini et al., 2016; Liu et al., 2021) and that each plant species, as early as the moss *Physcomitrella patens*, have multiple *JAZ* genes (Garrido-Bigotes et al., 2019). In the model plant Arabidopsis, 13 *JAZ* genes are expressed, which encode for 24 proteins (TAIR database) as multiple splice variants exist (Chung et al., 2010). These proteins have two conserved domains, TIFY and Jas, across all members of the family, except for JAZ13 that does not have a TIFY domain (Thireault et al., 2015). The TIFY domain promotes JAZ-NINJA (Pauwels et al., 2010) and JAZ-JAZ interactions (Chung and Howe, 2009), and the Jas domain constitutes a highly conserved signature of 26 amino acids required for COI1-JAZ and MYC2-JAZ

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interactions. However, some members of the JAZ family, such as JAZ7 and JAZ8, have a non-functional Jas degron sequence that weakly binds to COI1 preventing their degradation by the 26 S proteasome (Shyu et al., 2012). Furthermore, the N-terminus of JAZ1/10 contain a binding site for MYC2 (Withers et al., 2012; Moreno et al., 2013) and other JAZ proteins (JAZ5/6/7/8/13) contain additional motifs, such as EAR (ethylene-responsive element-associated amphiphilic repression; Ohta et al., 2001), which is the binding site for the TOPLESS co-repressor (Kagale et al., 2010; Shyu et al., 2012). Thus, the domain structure within the JAZ protein family can vary, forming JAZ subgroups with specific modes of transcriptional repression.

Domains in the JAZ proteins serve as binding sites for specific proteins (Chini et al., 2016, Garrido-Bigotes et al., 2019, Liu et al., 2021), creating differential modes of action for each JAZ. Several transcription factors (TFs) and repressor proteins have been experimentally validated as JAZ binding partners with proven functions (reviewed by Wager and Browse, 2012, Chini et al., 2016, and Howe et al., 2018). Variant pairing between JAZ proteins and regulators offers functional specificity within the JA signaling pathway. However, redundancies are also evident as in cases where multiple JAZ proteins can bind to the same regulatory proteins through conserved domains. Overall, the redundancy in JAZ protein function relies on the conservation of the TIFY and Jas domains among all family members (Garrido-Bigotes et al., 2019), while specificity might be achieved by induction of transcriptional expression, alternative splicing, spatiotemporal hormonal thresholds, and JA-other hormonal crosstalk (Chini et al., 2016). Further studies may elucidate other strategies to explain all the functional diversity of the JAZ proteins.

2. Connections between JAZ proteins and plant growth and defense tradeoffs

Historically, plant growth and defense have been regarded as two counteracting biological processes that each vie for limited plant resources (He et al., 2022). A predominant premise has been that plants under stress redirect resources to plant defense, resulting in penalties for certain physiological processes related to plant growth (Huot et al., 2014; Campos et al., 2016; Major et al., 2017; Guo et al., 2022). Illustrating this assumption, high-order *JAZ* mutants, such as *jazQ* (*jaz1/3/4/9/10*) and *jazD* (*jaz1–7/9/10/13)* that reduce the broad repressive functionality of JAZ proteins, keep the plant's immune response in an active state which results in growth reduction even in the absence of stress (Major et al., 2017, Guo et al., 2018, Guo et al., 2022). However, depending on the combination of *JAZ* mutant alleles, such as plants with *jazQ*, *jazD*, or phylogenetically related *jaz* mutations (Liu et al., 2021, Zhang et al., 2022) have contrasting reactions (i.e., resistance, susceptibility, or no reaction) to insect herbivores and *Botrytis cinerea*, highlighting the functions of each individual, or each combination of, JAZ proteins in immune responses to pathogens of various lifestyles.

The function of JA signaling in resource allocation to growth or defense processes was discovered by altering environmental conditions to favor either enhanced plant growth or enhanced defense. The scenario, in which JA and light stabilize MYC2/3/4 proteins through the activation of phytochrome B and make plants more resistant to pathogens was elegantly demonstrated by Chico et al. (2014). Under shade, JAZ proteins (JAZ1/5/7/9/10/11/12) are more stable, exerting additional repression on an already limited pool of MYC proteins and rendering plants more susceptible to herbivores and necrotrophs.

The examples above affirm undesirable growth-defense trade-offs explained by activation or repression of JA signaling. If one signaling pathway controls both growth and defense, then how can these processes be decoupled to achieve desirable plant fitness under stress? It has been proposed that this antagonism may be broken by rewiring hormonal pathways at key regulatory nodes, such as light response. For instance, the *jazQ*-associated growth penalty in response to enhanced JA signaling was shown to be fully rescued in the *phyB* mutant background, a mutant that lacks a functional phytochrome B (phyB) photoreceptor acting upstream of gibberellin (GA) signaling (Campos et al., 2016). However, a *phyB* mutation does not rescue the growth penalty observed in *jazD* (Major et al., 2020), suggesting that these phenotypes could be a consequence of epistatic interactions among different JAZ proteins, and that individual JAZ function might be dependent on cell and/or tissue type in the Arabidopsis rosette. Additional studies are required to dissect these complex interactions.

It has been recently hypothesized that typical growth-defense tradeoffs may happen independently of a resource ceiling and that network re-wiring may not be the only strategy to decouple growth and defense (He et al., 2022). These authors propose that removing susceptibility genes from the plant genome and searching for natural allelic variations in a wild population are reasonable approaches for improving plant growth and fitness in less-than-optimum conditions (He et al., 2022). JAZ proteins are notoriously known as negative regulators of plant immunity (Chico et al., 2008; Campos et al., 2014; Chini et al., 2016). Thus, the encoding genes are potential susceptibility genes. However, the large redundancy observed in the JAZ family usually masks the function of individual proteins in single mutant plants (de Torres Zabala et al., 2016; Gimenez-Ibanez et al., 2017; Major et al., 2017; Liu et al., 2021). Nonetheless, some altered phenotypes have been reported on a few loss-of-function single mutants (Shyu, 2016; Oblessuc et al., 2020; DeMott et al., 2021; Liu et al., 2021) providing opportunities to study specific JAZ regulatory nodes.

3. JAZ4 roles in plant defense and plant growth

3.1. JAZ4 acts on plant defenses within the canonical JA signaling pathway

Of JAZ proteins of Arabidopsis, JAZ4 seems to play a prominent role in whole plant physiology, which is evident by the multiple altered phenotypes of the *jaz4–1* loss-of-function mutant (Hu et al., 2013; Jiang et al., 2014; Oblessuc et al., 2020, DeMott et al., 2021). Owing to its extremely low expression in leaves, the *JAZ4* transcript has not been reliably detected by any RNA-sequencing experiment reported in the literature, but only by highly sensitive gene expression assays, such as RT-qPCR (Oblessuc et al., 2020). As such, the *JAZ4* gene was initially identified by Jas and TIFY domain searches in the Arabidopsis genome (Thines et al., 2007). *JAZ4* expression, assessed by RT-qPCR or Northen blot analyses, is not induced by any elicitors tested thus far, such as insect feeding or wounding (Chung et al., 2008), infection by *Pseudomonas syringae* pv. *tomato* (*Pst*) DC3000 (Demianski et al., 2012), or treatments with MeJA (Chini et al., 2007; Wu et al., 2020) and coronatine (Oblessuc et al., 2020). Like other members of the JAZ protein family, however, JAZ4 acts as a transcriptional repressor of JA pathway through the canonical COI1-JAZ-MYC2 signaling module (Oblessuc et al., 2020). For instance, *jaz4–1* mutant plants have increased susceptibility to *Pst* DC3000, while wild-type Col-0 plants expressing the dominant negative form of JAZ4 (i.e., *JAZ4ΔJas*) are resistant to this pathogen (Oblessuc et al., 2020), which is consistent with the role of other JAZ proteins in JA signaling (de Torres Zabala et al., 2016; Gimenez-Ibanez et al., 2017; Major et al., 2017; Liu et al., 2021).

While up-regulation of the JA pathway increases susceptibility to the hemibiotrophic, coronatine-producing pathogen *Pst* DC3000 (Cui et al., 2002), it confers enhanced resistance to necrotrophs, such as *Botrytis cinerea* (Thomma et al., 1998), and insect herbivory, such as *Trichoplusi ni* and *Spodoptera exigua* feeding on Arabidopsis (Walling, 2000; Major et al., 2017; Liu et al., 2021). Thus, the loss of JAZ proteins should enhance resistance to necrotrophs and pests. We used well-characterized plant genetic material to confirm that JAZ4 plays a role in plant responses to both *Pst* DC3000 (Oblessuc et al., 2020) and insects (Fig. 1a). We placed *T. ni* larvae on four-week-old rosette leaves and let them feed for six days to measure larval weight following a standard protocol (Cui

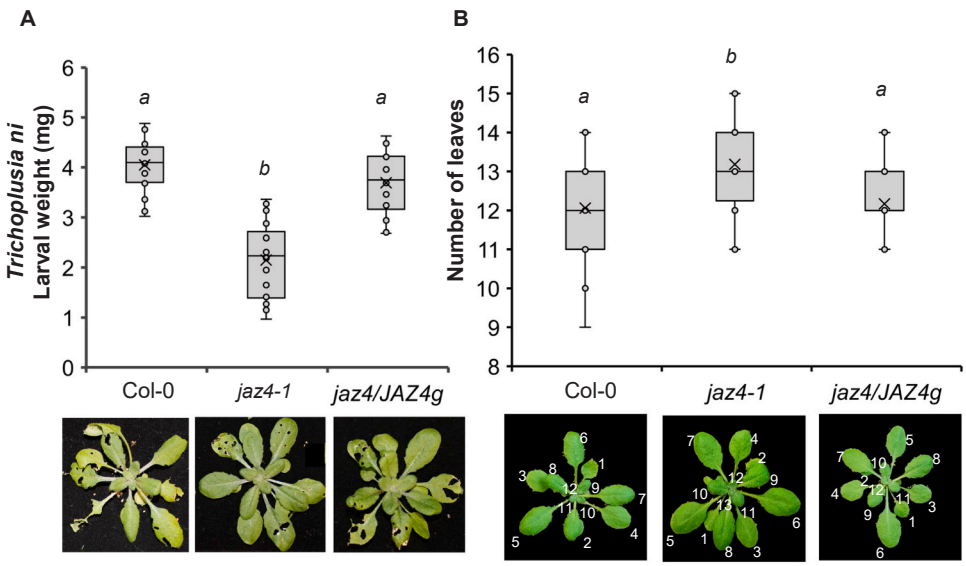

Fig. 1. Loss of JAZ4 increases both resistance to *Trichoplusia ni* and number of leaves in Arabidopsis. (A) *Trichoplusia ni* larvae were placed on one leaf per plant and larval weight was measured 6 days post-placement, n = 18 larvae. Images at the bottom represent leaf damage on each genotype at the time of sampling to measure larval weight. (B) Number of leaves (n = 48 plants) and representative photographs of rosettes (bottom) taken from 24-day old plants. Plot markers and center lines show the means and medians, respectively; box limits indicate the 25th and 75th percentiles and whiskers extend to minimum and maximum data points. Statistical analysis was performed using ANOVA, and significance was determined using Games-Howell and REGWQ post-hoc tests. Different letters on top of the graphs indicate statistically significant differences among the means ($p < 0.05$).

et al., 2002). We observed that *jaz4–1* rosettes are highly resistant to larval feeding when compared to Col-0 and the complemented *jaz4/-JAZ4g* lines (Fig. 1a). The larvae nearly decimated Col-0 and complemented leaves, while they unsuccessfully attempted to feed on *jaz4–1* leaves (Fig. 1a photographs). Similarly, simultaneous knock-out of the closely related genes *JAZ3/4/9* shows an additive effect on the plant's resistance to the insect herbivores *S. exigua* and *S. frugiperda*. This response is associated with MYC2-dependent activation of the gene *JASMONIC ACID RESPONSIVE 2* (Figure S10 in Liu et al., 2021). Additionally, *jaz3/4/7/8/9* mutations in the *coi1–1* background partially restore plant resistance upon *B. cinerea* infection (Figure 6c in Liu et al., 2021), suggesting a synergistic effect of the encoded proteins that is dependent on JA-Ile perception through COI1-JAZ binding.

3.2. *JAZ4 may act on plant growth and development independently of MYC2*

Although the JAZ4-dependent plant response to biotic stressors has a clear connection with the JA signaling pathway, as discussed above, the JAZ4 control of growth and development-related traits is not always associated with the JA/COI1/MYC2 module. This is not particularly surprising as JAZ4 affects many physiological processes, including

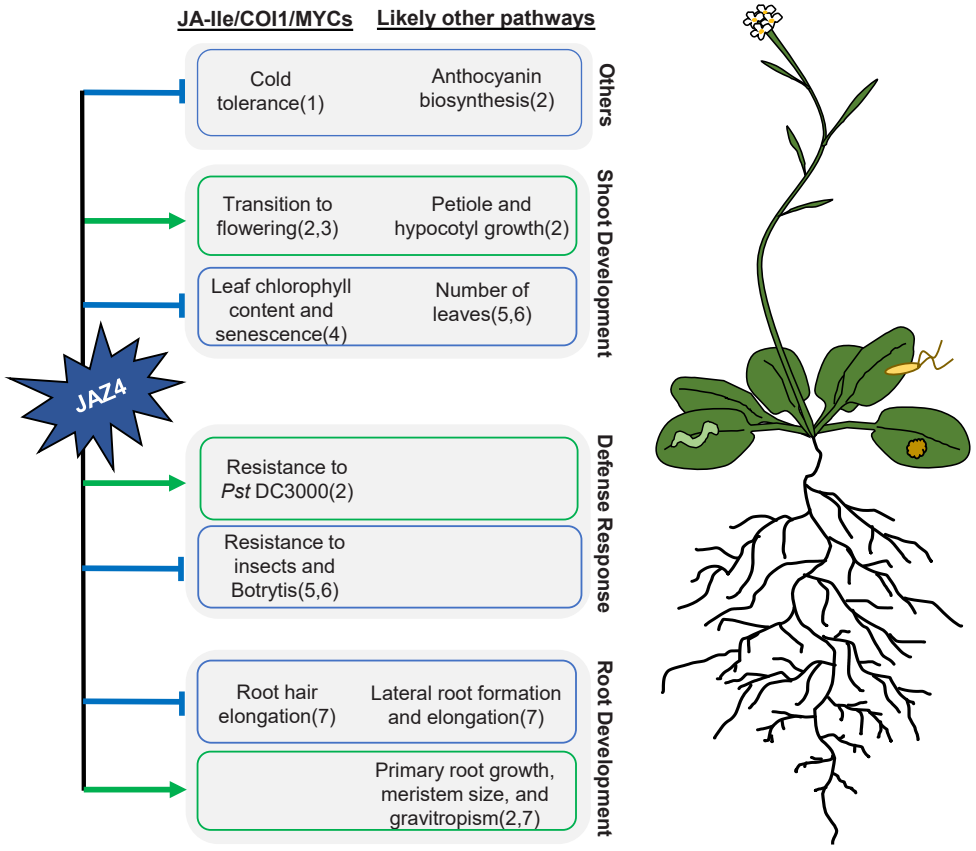

Fig. 2. Biological processes controlled by JAZ4. Activation (green line) or inhibition (blue line) of observed phenotypes are indicated by arrows or bars, respectively. Traits on the left have been associated with the canonical JA-Ile/COI1/MYCs pathway, whereas there is no evidence for traits listed on the right being part of this signaling module. Thus, JAZ4 controls those biological processes possibly via alternative signaling nodes. (1) Hu et al., 2013, (2) Oblessuc et al., 2020, (3) Zhai et al., 2015, (4) Jiang et al., 2014, (5) Liu et al., 2021, (6) Fig. 1, (7) DeMott et al., 2021. JA-Ile/COI1/MYC-dependent processes were determined through phenotypic characterization after MeJA or coronatine application, protein-protein interaction assays, and mutant lines for the canonical JA signaling pathway. Traits linked to other pathways were determined through phenotypic characterization of dominant negative lines (i.e., *JAZ4ΔJas*), such as hormone application, and protein-protein interaction assays with transcription factors other than MYCs.

chlorophyll content and senescence (Jiang et al., 2104), tolerance to cold (Hu et al., 2013), transition to flowering (Oblessuc et al., 2020), and leaf number (Fig. 1b; Liu et al., 2021), as well as hypocotyl and petiole elongation, anthocyanin accumulation, root development that includes root hair elongation, gravitropic response, lateral root formation, and root meristem size (Oblessuc et al., 2020; DeMott et al., 2021) (Fig. 2). Thus, it is expected that JAZ4 can interact with several regulatory proteins other than the MYCs to control these seemingly unrelated metabolic processes. Some of these JAZ4-TF pairs have already been described and are highlighted below.

Thus far, four JAZ4-TF interactions that regulate JA-dependent growth have been elucidated through genetic and biochemical approaches. JAZ4 binding to INDUCER OF CBF EXPRESSION 1 (ICE1) results in repression of this TF, attenuating freezing tolerance via the ICE–C-REPEAT BINDING FACTOR/DRE BINDING FACTOR 1 (CBF/DREB1) cascade in Arabidopsis. Under cold stress, JA-induced degradation of JAZ4 enhances freeze tolerance (Hu et al., 2013). JAZ4 competes with INDOLE-3-ACETIC ACID INDUCIBLE 29 (IAA29) to bind the zinc finger domain of WRKY57, a negative regulator of senescence, in which JAZ4 represses and IAA29 activates WRKY57. Interestingly, this binding competition explains the antagonism between JA and auxin in promoting and repressing leaf senescence, respectively (Jiang et al., 2014). JAZ4 binds to TOE1/2 (TARGET OF EAT1 and 2), a repressor of flowering via repression of FT (FLOWERING LOCUS T) (Zhai et al., 2015) and *jaz4–1* and *JAZ4ΔJas*-expressing plants show late and early flowering, respectively (Oblessuc et al., 2020), suggesting that the JAZ4-TOE1/2 interaction regulates the transition to reproductive phase via JA-COI1 pathways. Finally, JAZ4 also binds to TFs PHR1 and PHL2/3, controlling the expression of genes associated with anthocyanin accumulation and root growth inhibition (He et al., 2023). Corroborating the function of JAZ4 in repressing the action of these TFs, the *jaz4–1* mutant shows increased anthocyanin in the leaves and plants expressing the dominant negative *JAZ4ΔJas* construct have longer primary roots in comparison to the wild-type plant (Oblessuc et al., 2020).

Although several biological processes are controlled by JAZ4 (Fig. 2), the underlying molecular bases are only beginning to be discovered. Nonetheless, JAZ4 has been shown to interact with many proteins in yeast assays and through other methods (BioGRID; Oughtred et al., 2021) (Table 1), and it will be interesting to learn how these interactions could control hormonal actions in the plant.

3.3. JAZ4 spatial expression is linked to its function in new organ development

Although JAZ-TF interactions are extremely important in linking hormonal signaling with downstream biological responses, the time and place where these interactions occur is also crucial for the plant to express specific phenotypes (highlighted by Jin and Zhu, 2017). Recently, it has been shown that the *JAZ4* transcript accumulates differentially in different organs and tissues at different times of development, and amounts of the splice variants *JAZ4.1* and *JAZ4.2* are variable (DeMott et al., 2021). The total *JAZ4* transcript levels are elevated in flowers, siliques, and roots of mature plants; however, the spatial distribution of the transcripts varies within each organ. Specifically, *JAZ4* is more abundant in the root apex of seedlings, particularly the more stable *JAZ4.2* transcript, indicating that JAZ4 is associated with root growth and development (DeMott et al., 2021). The biological relevance of *JAZ4* gene expression is supported by the fact that overexpression of *JAZ4ΔJas* results in constitutively longer primary root than that of in the wild type plant (Oblessuc et al., 2020). Furthermore, the *jaz4–1* loss-of-function mutant has increased root branching and lateral root length and these phenotypes are rescued to the wild type level in the respective complemented lines (DeMott et al., 2021). Interestingly, a detailed analysis of the auxin and ethylene signaling pathways placed JAZ4 as a positive regulator of auxin biosynthesis and transport in the root apex, while in the root elongation zone, JAZ4 may act as a negative regulator of ethylene (DeMott et al., 2021). The precise molecular mechanism is yet to be elucidated.

The connection between JAZ4 and new organ development is additionally supported by the observation that the *jaz4–1* mutant line has an increased number of leaves in comparison to wild-type Col-0 (Fig. 1b; Liu et al., 2021). This process seems to be independent of, or parallel to,

Table 1
Known interactors of the JAZ4 protein compiled based on data available on the BioGRID platform (Oughtred et al., 2021), the Bio-Analytic Resource (BAR) for Plant Biology (https://bar.utoronto.ca/), and published literature. AGI = Arabidopsis Genome Identifier; PCA = Protein-Fragment Complementation Assay; Y2H = Yeast-Two-Hybrid; ACW = Affinity Capture-Western, RC = Reconstituted Complex.

Interactor AGI	Interactor code	Interaction assay	JAZ binding domains	Reference	Related processes
AT2G46510	AIB	Y2H	Jas (JAZ8/11)	Song et al., 2013	ABA signaling
AT2G39940	COI1	Y2H	Jas	Oblessuc et al., 2020	JA-Ile receptor
AT5G15850	COL1	Y2H	not determined	Wanamaker et al., 2017	Flowering
AT3G26744	ICE1	ACW; PCA; Y2H	Jas (JAZ1)	Hu et al., 2013	Cold stress, seed dormancy
AT1G12860	ICE2/SCRM2	ACW; PCA; Y2H	Jas (JAZ1)	Hu et al., 2013	Freezing stress
AT1G01260	JAM2	Y2H	Jas (JAZ8/11)	Song et al., 2013	JA biosynthesis
AT1G19180	JAZ1	Y2H	TIFY	Chung and Howe, 2009	Transcriptional repressor
AT5G13220	JAZ10	Y2H	TIFY	Chung and Howe, 2009	Transcriptional repressor
AT3G43440	JAZ11	Y2H	TIFY	Chung and Howe, 2009	Transcriptional repressor
AT3G17860	JAZ3	Y2H	TIFY	Chini et al., 2009	Transcriptional repressor
AT1G48500	JAZ4.1	Y2H	TIFY	Chung and Howe, 2009	Transcriptional repressor
AT1G48500	JAZ4.2	Y2H	not determined	DeMott et al., 2021	Transcriptional repressor
AT1G30135	JAZ8	Y2H	TIFY	Chung and Howe, 2009	Transcriptional repressor
AT1G70700	JAZ9	Y2H	TIFY	Chini et al., 2009	Transcriptional repressor
AT1G32640	MYC2	RC	TIFY	Chini et al., 2009	Transcriptional repressor
AT5G46760	MYC3	Y2H	Jas	Oblessuc et al., 2020	JA-signaling transcription factor
AT4G17880	MYC4	RC; Y2H	not determined	Fernandez-Calvo et al., 2011	JA-signaling transcription factor
AT1G08970	NF-YC9	Y2H	not determined	Wanamaker et al., 2017	GA-ABA seed germination
AT4G28910	NINJA	Y2H	TIFY	Pauwels et al., 2015	Transcriptional repressor
AT3G24120	PHL2	Y2H	not determined	He et al., 2023	Phosphate starvation
AT4G13640	PHL3	Y2H	not determined	He et al., 2023	Unknown
AT4G28610	PHR1	Y2H	not determined	He et al., 2023	Phosphate metabolism
AT5G37260	RVE2/CIR1	Y2H	not determined	Wanamaker et al., 2017	Circadian regulation
AT2G42200	SPL9	Y2H	TIFY (JAZ1/3/4/6/10/11)	Mao et al., 2017	Vegetative to reproductive transition
AT2G28550	TOE1/RAP2.7	Y2H	N-terminus (JAZ1)	Zhai et al., 2015	Flowering, innate immunity
AT5G60120	TOE2	Y2H	N-terminus (JAZ2)	Zhai et al., 2015	Flowering, innate immunity
AT1G69310	WRKY57	PCA; RC; Y2H	not determined	Jiang et al., 2014	Drought tolerance
AT3G19070		Y2H	not determined	Wanamaker et al., 2017	Unknown

the JA-COI1-MYC2 module, as Col-0 and *coi1–1* have a similar number of leaves and stacked *jaz4/3/9* mutations promote increased leaf number in both Col-0 and *coi1–1* backgrounds (Liu et al., 2021). These findings suggest that JAZ4 might be also connected with auxin distribution at the shoot apex (Braybrook and Kuhlemeier, 2010), inhibiting leaf formation. Whether the JAZ4 protein is restricted to or preferentially accumulated at the shoot apex remains to be determined.

3.4. Potential mechanisms for enhanced growth and defense via JAZ4 signaling

The studies highlighted above suggest that the role of JAZ4 in growth and development depends on the time and place that JAZ4 is expressed at the cell and tissue levels. The spatial separation of JAZ4 action could modulate different hormonal signaling pathways due to its interactions with co-expressing regulators (Table 1 as an example). This potential mechanism might explain why the *jaz4–1* mutant shows enhanced defense against insects (Fig. 1a) as well as enhanced growth of the shoot (Fig. 1b; Liu et al., 2021) and root (DeMott et al., 2021). These discoveries lead us to consider JAZ4 functions within the context of whole plant physiology and to hypothesize about the mechanistic connections between signaling pathways that depend on specific environmental signals (*e.g.*, biotic stress) and biological scenarios (*e.g.*, organ development) that are separated in time and space (Fig. 3).

In leaves challenged with necrotrophic pathogens or generalist insects, JAZ4 would primarily work through the well-established role of JAZ proteins as a co-receptor and a repressor of JA-Ile signaling, as explained above. This transient and strong plant response would decrease the abundance of JAZ4, mainly on the leaf blade, to bring about plant resistance to necrotrophs and insects or susceptibility to coronatine-producing bacteria and other JA-mimicking pathogens. The significant reduction of *JAZ4* expression in guard cells when plants are exposed to high humidity is additional evidence that JAZ4 levels may decrease with the activation of the JA-Ile pathway (Panchal et al., 2016). Thus, it is unlikely that JAZ4 participates in negative feedback loops to dampen the JA-signaling, eloquently explained by Howe et al. (2018). However, once JA biosynthesis quickly returns to the basal level through various mechanisms, the cell would be replenished with JAZ4.

In a scenario where the plant is undergoing normal growth and development, JAZ4 function in growth and development would be

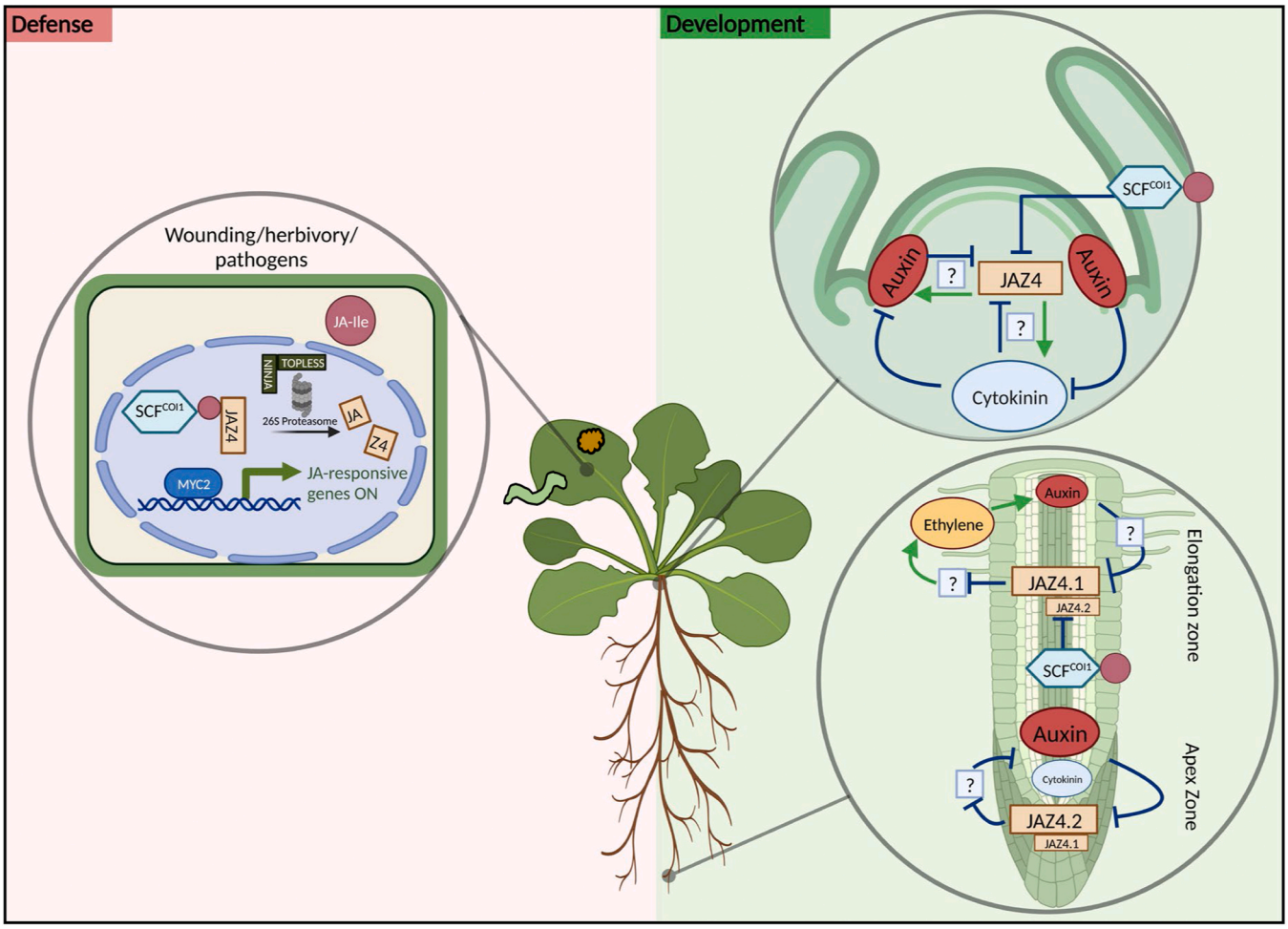

Fig. 3. A model for the role of JAZ4 in growth and defense in different plant tissues and organs. In leaves (left), JAZ4 acts as a negative regulator in defense functions against chewing/wounding insects and necrotrophic pathogens through the canonical JA-Ile/COI1/MYC2 pathway. In the plant apexes, the prominent expression of JAZ4 in the root (and possibly shoot) apex promotes its regulation of meristem activities and organ development in connection with auxin, cytokinin, and ethylene crosstalk, forming local regulatory loops. In the shoot apex, JAZ4 might regulate auxin-cytokinin balance through undiscovered regulators, which can control leaf formation. This function may also be regulated by the JA-Ile/COI1 complex to maintain JAZ4 levels, as the *coi1–2* mutant displays a significantly reduced number of leaves (Zhai et al., 2015). In the root, JAZ4 splice variants may be responsible for root growth and development. In the root apex, JAZ4.2 could repress an unidentified positive regulator of auxin and/or cytokinin regulator, restricting root growth. In the root elongation zone, the most abundant splice variant, JAZ4.1, could act as a negative regulator of ethylene hormonal signaling to induce root elongation. JAZ4 protein abundance might be also regulated by JA-Ile/COI1/MYC2 since the overall *JAZ4* transcript pool is reduced (DeMott et al., 2021) and MYC2/3/4 are preferentially expressed in the root elongation zone (Fernandez-Calvo et al., 2011). Drawing created with BioRender.

prominent wherever it is expressed at the highest level. For instance, *JAZ4* is preferentially expressed, and the JAZ4 protein potentially accumulated, at the root apex, positively controlling auxin biosynthesis and signaling in this zone (DeMott et al., 2021). How exactly this *JAZ4* expression is maintained in specific tissues remains elusive; nonetheless, it suggests that JAZ4 participates in a localized regulatory loop to promote and maintain the meristem. For instance, it is well known that biosynthesis of auxin and cytokinin is important for root growth and differentiation (Nishimura et al., 2004; Zhao, 2008). Specifically, these hormones control each other's biosynthesis and signaling to promote organ development from the meristems (reviewed by Jones and Ljung, 2011). One can imagine that JAZ4 could repress an auxin- and/or cytokinin-associated regulator, and the different levels of auxin and/or cytokinin would negatively regulate JAZ4, thereby imposing some restriction at the root apex. A similar scenario would be possible at the root elongation zone, where the *JAZ4.1* transcript is more abundant and connects auxin with ethylene signaling (Fig. 3; DeMott et al., 2021). We cannot rule out the possibility that the core JA-Ile/COI1/MYC2 module could also be involved in reducing the overall pool of *JAZ4* transcripts, as COI1 is required for root inhibition (Adams and Turner, 2010; Raya-Gonzalez et al., 2012) and, through the analyses of promoter reporter lines, MYC2/3/4 have been shown to be highly expressed at the root elongation and maturation zones (Fernández-Calvo et al., 2011; Gasperini et al., 2015). There is also evidence that the JA repression of primary root growth could be independent of auxin signaling (Raya--Gonzalez et al., 2012).

It is reasonable to assume that a similar process would happen at the shoot apex, where auxin and cytokinin work together to promote leaf formation (Barton, 2010). The number of leaves would be fine-tuned by auxin levels through the action of JAZ4, in which JAZ4 would preferentially bind to a yet-to-be-discovered regulator in that region to repress leaf formation. In the absence of JAZ4, this regulation is lost and additional leaves are formed (Fig. 1b; Liu et al., 2021). This process could also be dependent on JA-Ile perception by COI1-JAZ4, as the *coi1–2* mutant shows a significantly reduced number of leaves (Zhai et al., 2015). To the best of our knowledge, there is no evidence that MYC2 is involved in leaf formation as changes in leaf number have not been described for *myc2* mutants.

At these apical locations, JAZ4 would be a hub that connects hormonal pathways controlling plant immune responses and organ differentiation, such as lateral root initiation, root elongation, transition to flowering, and leaf formation (Fig. 3). The scenarios above need to be validated as many pieces of the puzzle are still missing, and future studies are required to uncover how signaling of JAZ4-controlled processes are fine-tuned at specific tissues.

4. Opportunities to explore JAZ4 regulatory nodes for precise metabolic engineering

A main unresolved question regarding the negative regulation of immune response and growth by JAZ4 is "*are these processes mechanistically disconnected in time and/or space?*" At the moment of this writing, there is no concrete evidence for this being the case, and additional experimentation to determine the extent of JAZ4-dependent growth restriction under stress is required. Nonetheless, mounting evidence suggests that the answer to this question is "yes". If JAZ4 negatively regulates growth and defense through preferential spatiotemporal binding to regulators of distinct signaling pathways, then plants could continue to grow under stress in the absence of JAZ4. As plant growth and defense are quantitative traits, it is possible that the loss of JAZ4 function will not fully restore growth under stress. Nonetheless, one could devise metabolic engineering strategies to achieve disease resistance without excessive growth penalties. Furthermore, JAZ3/4/9 seem to have an additive effect on many growth and defense phenotypes (Liu et al., 2021), which could be great targets to modulate the traits at the desired level. As the JAZ family is conserved in the plant kingdom (Howe et al., 2018; Garrido-Bigotes al., 2019), similar approaches could be employed for improving crop plants growing under biotic stress. For instance, increased shoot area along with increased resistance to necrotrophs may be relevant for horticultural crops (e.g., lettuce, spinach, and other leafy vegetables) that are normally grown under high fungal disease pressure (Mostafidi et al., 2020; Gao et al., 2022). Similarly, increased root branching in tomato is a highly desirable agronomic trait, which is normally achieved by applying auxin to transplants (Guan et al., 2019). Finding a JAZ4 ortholog in tomato would enable targeted engineering strategies for increased root branching.

The uncoupling of undesirable tradeoffs through engineering JAZ proteins has been already proposed. A proof-of-concept study showed that expressing the dominant negative form of JAZ2, SlJAZ2ΔJas, in tomato plants spatially decouples the typical JA-SA tradeoff in plant resistance to hemibiotrophs and necrotrophs (Ortigosa et al., 2019). These plants have a strong stomatal immunity to the hemibiotroph *Pst* DC3000 without compromising leaf resistance to the necrotroph *B. cinera*. However, it remains to be determined whether this transgenic approach disturbs desirable agronomic traits and plant fitness in the field. Different stresses may activate different signaling pathways, thus it would be important to establish how exactly JAZ proteins regulate the pathways to control various aspects of plant biology.

To completely explore the distinguishing features of JAZ4 and take advantage of the spatiotemporal separation of its actions (Fig. 3), it is crucial to learn more about the underlying molecular mechanisms. Determining the function of each domain in the JAZ4 protein, validating the protein-protein interactions that control each phenotype, and uncovering the downstream responses could provide exciting opportunities for precise metabolic engineering of desired traits to increase crop productivity under less than optimum conditions.

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All authors contributed to the writing of this manuscript.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this review.

Data availability

Data will be made available on request.

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