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More than growth: Phytohormone-regulated transcription factors controlling plant immunity, plant development and plant architecture

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Abstract

Activation of immunity by exogenous signals or mutations leading to autoimmunity has long been associated with decreased plant growth, known as the growth-defense tradeoff. Originally thought to be a redirection of metabolic resources towards defense and away from growth, recent studies have demonstrated that growth and defense can be uncoupled, indicating that metabolic regulation is not solely responsible for the growth-defense tradeoff. Immunity activation has effects on plant development beyond the reduction of plant biomass, including changes in plant architecture. Phytohormone signaling pathways, and crosstalk between these pathways, are responsible for regulating plant growth and development, and plant defense responses. Here we review the hormonal regulation of transcription factors that play roles in both defense and development, with a focus on their effects on plant architecture, and suggest the targeting of these transcription factors to increase plant immunity and change plant growth and form for enhancement of agronomical traits.

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Introduction

When plant immunity is activated by pathogen perception, treatment with chemical activators of defense, or mutations inducing autoimmunity, a negative effect on plant growth is often observed [1,2]. This phenomenon, known as the growth-defense tradeoff, is observed in model plants such as *Arabidopsis thaliana* (hereafter, Arabidopsis), as well as crop plant species. Such suppression of growth is detrimental to overall plant yield and is a barrier for the use of mutations leading to constitutive immunity in plant breeding programs [3].

Early studies on the characterization of plants with activated states of immunity led to the hypothesis that the growth-defense tradeoff was attributed to a redirection of the plant's metabolism to defense, at the expense of growth. Support for such a hypothesis includes evidence of decreased rates of photosynthesis [4,5], and changes in plant primary and specialized metabolisms after defense activation [6]. However, metabolism redirection for the production of defense compounds is not the entire cause of this suppression of growth by immunity (reviewed in [7]). Recent studies have shown that growth and defense can be uncoupled, resulting in plants with increased defense without severe compromises to plant growth [8–10].

In general, the growth-defense tradeoff has been mostly documented at the level of overall plant growth, i.e., by observations of reduction of biomass and yield [2,11]. However, defense-activated plants can also display changes in plant development, such as ectopic meristem development [12], changes in apical dominance and internode length [13], as well as changes in flowering time [14,15]. Therefore, immunity activation modulates more than growth (defined as changes in cell division and expansion) to involve changes in development (which encompass changes in patterns of cell division and expansion) resulting in altered organ shape and plant architecture. Plant architecture is of paramount importance to agriculture. Reduced plant stature, but also leaf length or width, or even patterns of branching and leaf angle, can have a substantial effect on light capture and photosynthesis, changing overall plant performance. In addition, such traits also directly influence the ability of plants to grow under the high-density planting of agronomical settings, and their suitability for mechanical harvest. Similarly, altered root architecture, with a reduction in root length, or the number or structure of root hairs and lateral roots,

prevent plants from properly obtaining water and capturing essential nutrients from the soil for growth. And patterns of the vasculature, in both roots and shoots, are essential not only for the transport of water, minerals, and of photosynthates from source to sink tissues, but also for mechanical strength and resistance to lodging.

Phytohormone signaling pathways, and their extensive crosstalk, help plants integrate signals from the environment and determine whether developmental programs begin, continue or arrest [16]. As such, phytohormones are obvious mediators of this growth-defense tradeoff. After attack by biotrophic or necrotrophic pathogens, plants respond by increasing the levels of the phytohormones salicylic acid (SA) or jasmonic acid (JA), respectively. Once over a threshold, high levels of SA or JA can result in negative impacts on plant growth, through their interplays with other phytohormonal pathways [1]. In this review, we focus on phytohormone-regulated transcriptional factors mediating plant immunity and plant development, and their resulting effects on the architecture of plants. Understanding of these transcriptional mediators and the growth, immune, and developmental processes they control, together with engineering of their expression and activity, can lead to plants not only with increased resistance to pathogens and increased biomass, but also with altered plant architecture, thus influencing several traits of agronomical importance.

Phytohormone-regulated transcription factors affecting plant immunity and shoot development

Transcription factors (TFs) are responsible for transducing signals into gene expression, and transcriptional regulation plays a major role in the processes of plant development and defense activation. As such, several TFs have been identified as regulators of both plant development and immunity. In most cases, these TFs are transcriptionally regulated in opposing ways by phytohormones involved in defense or growth. While primary TFs of phytohormonal pathways are major regulators of these processes, changes in their function lead to vast pleiotropic effects, making them less than optimal targets for manipulation to achieve favorable outcomes of resistance to pathogens and increased plant yield. On the other hand, manipulation of non-primary phytohormone-regulated TFs allows for more precise regulation of the processes of immunity and development. Here we highlight select non-primary phytohormone-regulated TFs whose regulation and function in both immune responses and plant development have been recently elucidated (Figure 1 and Table 1), with consequences to plant architecture.

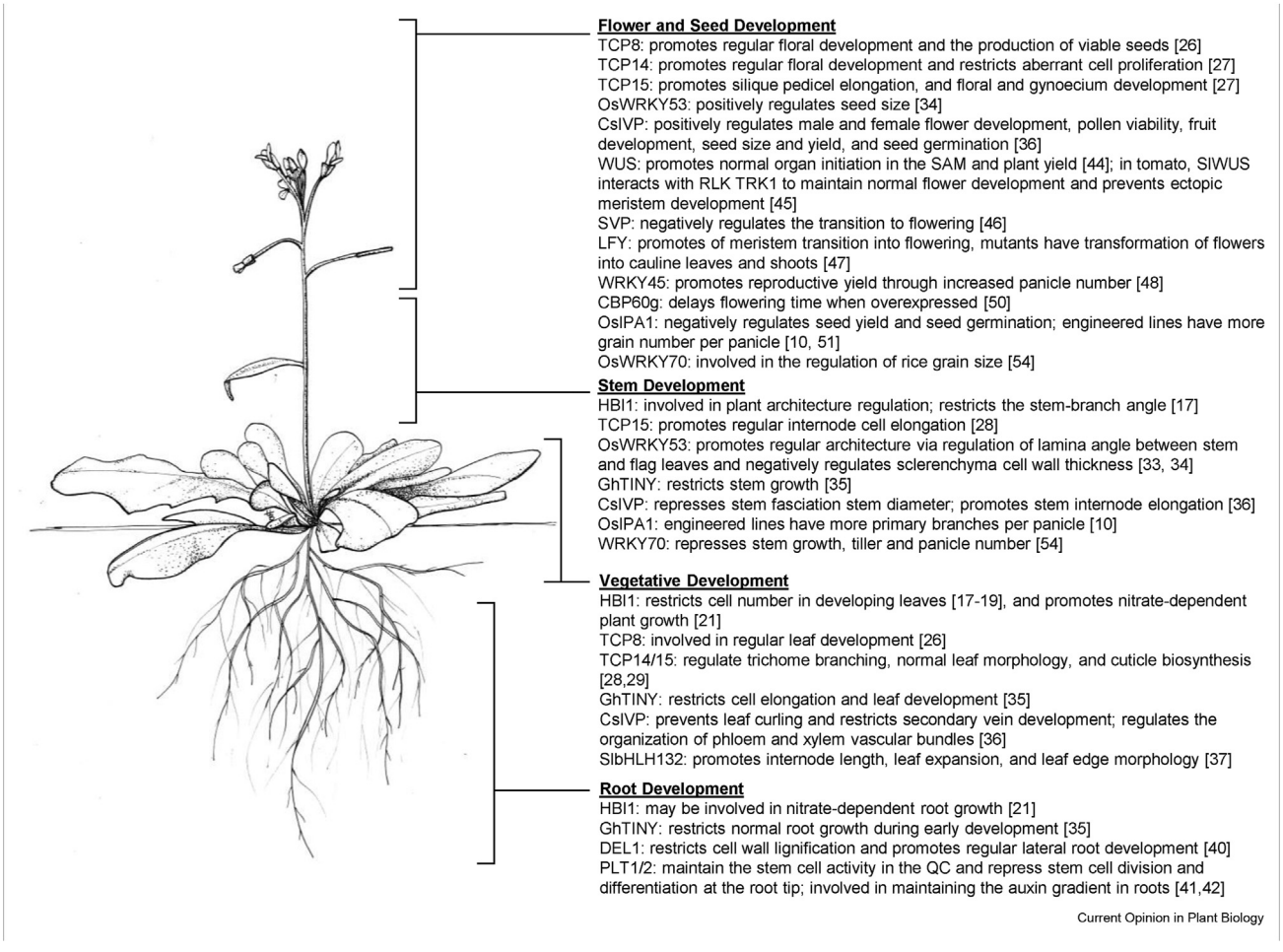
Among phytohormonal pathways, signaling of the phytohormone brassinosteroid (BR) is tightly intertwined with plant growth and immunity. The basic helix-loop-helix (bHLH) HOMOLOG OF BRASSINOSTEROID ENHANCED EXPRESSION 2 INTERACTING WITH IBH 1 (HBI1) was one of the first TFs identified regulating plant immunity and growth [17]. BR signaling and perception of microbial patterns (Pattern Triggered Immunity, or PTI) oppositely regulate the expression of *HBI1* [17]. Induction of *HBI1* expression by BR directly changes the expression of genes involved in reactive oxygen species (ROS) homeostasis, as seen by the up-regulation of *RbohC* resulting in leaf cell expansion, and repression of *RbohA*, a critical component of bacterial resistance response [17]. Not surprisingly, overexpression of *HBI1* is associated with increased rosette, leaf, and cell area, and *HBI*-overexpressing plants are more susceptible to infection by the bacterial pathogen *Pseudomonas syringae* pv. *tomato* (*Pst*) [17,18]. However, plants overexpressing *HBI1* also display a change in plant architecture, with a reduction in the angle of stem-branch junctions [18,19], a phenotype that is associated with BR signaling [20]. The mediation of plant growth by HBI1 may be linked to the regulation of nitrate signaling and ROS homeostasis [21]. Nitrate-induced plant growth is reduced in a quadruple mutant of *hbi1* and its three closest homologues in Arabidopsis, while overexpression of *HBI1* results in the opposite phenotype with increased shoot and root growth under nitrate [21]. In wild-type plants, HBI1-induced expression of antioxidant genes reduces the inhibitory effects of ROS on nitrate signaling, promoting plant growth [21]. Whether nitrate signaling is also involved in the HBI1-mediated regulation of root architecture is unknown, but of note, nitrate is a major regulator of root architecture, through mechanisms that also involve BR signaling [22].

The TEOSINTE BRANCHED1/CYCLOIDEA/PCF (TCP) is a TF family involved in the regulation of plant development and immunity. TCPs mediate plant immunity through the direct binding of TCP8 and TCP9 to the promoter of the SA biosynthesis gene *ISOCHORISMATE SYNTHASE 1* (*ICS1*), driving *ICS1* expression during pathogen infection, resulting in increased SA levels necessary for defense responses [23]. TCP8, TCP14, and TCP15 also play a role in PTI, through regulation of the gene encoding the pattern recognition receptor (PRR) EF-TU RECEPTOR (EFR), and *tcp8,14,15* mutants are deficient in *EFR*-dependent PTI [24]. For their role in plant growth, TCPs have been linked to BR biosynthesis [25]; however, the contribution of individual TCPs in plant development differs. Overexpression of *TCP8* results in delayed flowering compared to wild type [26]. Because *tcp8* mutants do not display obvious morphological changes, a *35S::TCP8-EAR* transcriptional repressor line was used to evaluate the role of TCP8 in plant development [26]. *35S::TCP8-EAR* plants have severe developmental defects including arrested growth, and

surviving seedlings have abnormal development with dark green leaves, hyponastic cotyledons, and shorter primary roots [26]. Seedlings that reach the reproductive stage are considerably smaller than the wild type and have three outer fused floral whorls, resulting in exposed, irregular gynoecia that fail to develop viable seeds [26]. Lines overexpressing *TCP15* also have irregular flower development, including fused carpel defects, increased number of seeds per silique, and reduced auxin content, with TCP15 acting to balance cytokinin and auxin responses to regulate gynoecium development [27]. *tcp15-3* has a significant reduction in silique pedicel length and inflorescence height resulting from reduced internode length, and increased trichome branching in leaves and stems [28,29]. Pedicel and internode length are further reduced in *tcp14-4, tcp15-3* double mutants [28]. Furthermore, *tcp14-4, tcp15-3* has reduced expression of cuticle biosynthesis genes, leading to a more permeable cuticle layer [29], potentially contributing to increased plant susceptibility. The dominant repressor *pTCP14:TCP14-SRDX* plants have a larger reduction in stem height and pedicel length compared to the double mutant, and show leaf development defects, including a broader leaf base, reduced leaf tip elongation, and increased trichome branching, as well as severe defects in floral development, including ectopic tissue proliferation on the carpel valve boundaries and flower receptacle [28]. Of interest is the fact that TCPs are targeted by effectors from a variety of pathogens, highlighting their importance to plant immunity [30].

WRKY TFs make up a large family of TFs in plants that contain the WRKY (Trp-Arg-Lys-Tyr) domain and are widely associated with responses to pathogens; however, their role in the regulation of plant development is less known. The expression of the gene encoding the rice TF OsWRKY53 is induced by fungal elicitors, and

Figure 1



Effects of transcription factors associated with regulation of plant immunity on plant developmental processes, and their consequences to plant architecture. Several plant transcription factors (TF) play roles in plant immunity and development. Altered TF activity leads to changes not only in plant immunity, but also in plant developmental processes in various plant tissues, changing overall plant architecture, with consequences to plant yield. SAM: shoot apical meristem; RLK: receptor-like kinase; QC: quiescent center. Scientific illustration of *Arabidopsis thaliana* by Hannah M. Berry.

Table 1

Immunity role of phytohormone-regulated transcription factors with a role in plant development and architecture.

Transcription Factor	Regulating Hormone	Effect on Plant Defense	Reference
HBI1	BR	Differentially regulates *NOX* and *POX* gene expression to regulate apoplastic ROS Represses expression of *RbohA* to suppress immunity to bacterial pathogens Negatively regulates *POX* in response to PTI to restrict ROS accumulation	[17–19]
TCP8	SA	Directly binds to the promoter of *ICS1* to promote SA biosynthesis during pathogen infection Promotes PTI by driving the expression of pattern recognition receptor EFR Promotes ETI responses	[23,24]
TCP14/TCP15	SA	Promotes PTI by driving the expression of pattern recognition receptor EFR Promotes ETI responses	[24]
OsWRKY53	SA, BR, JA	Transcription is induced during pathogen infection Acts as a transcriptional activator to enhance defense responses Is a negative regulator of JA and JA-Ile accumulation in response to herbivory Promotes *PR* gene expression and resistance to *M. oryzae* Promotes susceptibility to *Xoo*	[32,33]
GhTINY2	SA, JA	Expression is induced by SA and JA Promotes *WRKY51* expression leading to increased SA accumulation and immunity Promotes resistance to *V. dahliae*	[35]
CsIVP	SA, Auxin	Promotes susceptibility of cucumber to powdery mildew Negatively regulates SA accumulation via a strong interaction with SA repressor CsNIMIN1	[36]
SlbHLH132	SA, JA	Expression is moderately upregulated by SA or JA treatment Expression is specifically induced by the effector XopD during *X. euvesicatoria* infection Promotes ETI responses and resistance to *X. euvesicatoria*	[37]
DEL1	SA	Acts as a transcriptional repressor of the SA transporter *EDS5* Restricts SA accumulation in growing tissues Promotes plant susceptibility to powdery mildew and root-knot nematodes	[39,40]
PLT1/PLT2	SA, JA, Auxin	SA-induced ROS down-regulates expression, thus promoting pathogen resistance Transcriptionally regulated by binding of MYC2 to its promoter	[41,42]
WUS	CK, Auxin, JA	WUS prevents viral infection of pluripotent cells in the SAM WUS may act to prevent viral infection by repressing *MTases* and global protein synthesis Tomato SlWUS interacts with and is phosphorylated by RLK TRK1 during chitin-triggered PTI	[44,45]
SVP	Unknown	Promotes SA biosynthesis during ARR to *Pst* Promotes plant immunity to bacterial pathogens	[46]
LFY	Unknown	Promotes resistance to *Pst* in cauline leaves	[47]
WRKY45	SA, CK	Transgenic line *pOsUbi7::WRKY45* results in further increase of disease resistance	[48,49]
CBP60g	SA	Promotes defense to *Pst* under higher temperatures when overexpressed	[50]
OsIPA1	SA	In the phosphorylated state, induces *WRKY45* expression to promote immunity Promotes resistance to *M. oryzae* Reduces GA-mediated plant susceptibility and promotes resistance to *Xoo* *pHEN1::IPA1* enhances plant disease resistance	[11,51]
WRKY70	SA	In the unphosphorylated state, WRKY70 represses the expression of defense-related genes Is phosphorylated in response to SA treatment or pathogen infection leading to its ubiquitination and degradation, and reduction of defense gene expression	[52–54]

BR: brassinosteroids; SA: salicylic acid; JA: jasmonic acid; CK: cytokinin; GA: gibberellic acid; NOX: NAPDH oxidase; POX: peroxidase; ROS: reactive oxygen species; PTI: pattern-triggered immunity; ETI: effector-triggered immunity; MTase: S-adenosyl-L-methionine-dependent methyltransferase; SAM: shoot apical meristem; ARR: age-related resistance.

overexpression of *OsWRKY53* leads to increased expression of several genes encoding proteins involved in defense, such as PATHOGENESIS-RELATED (PR) proteins, chitinases, and peroxidases, and also to increased resistance to the fungal pathogen *Magnaporthe oryzae* [31,32]. However, overexpression of *OsWRKY53* leads to thinner sclerenchyma cell walls and increased susceptibility to the bacterial pathogen *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) [33]. Notably, overexpression of *OsWRKY53* also has a positive effect in rice reproductive development, leading to increased seed size and increased lamina angle of the flag leaf, while *oswrky53* plants have erect leaves and exhibit many BR-deficient phenotypes including reduced plant and seed size [34]. BR reduces the expression of *OsWRKY53* but increases OsWRKY53 protein stability, indicating a fine-tuning mechanism for WRKY53 regulation [32].

An APETALA2/ETHYLENE RESPONSIVE FACTOR (AP2/ERF) TF in cotton, GhTINY2, mediates SA-dependent defense response and suppresses BR-dependent growth responses [35]. Infection by the fungus *Verticillium dahliae* or treatment with SA induces expression of *GhTINY2*, which directly activates expression of *WRKY51*, leading to increased SA accumulation and signaling and increased defense responses [35]. Overexpression of *GhTINY2* in Arabidopsis results in increased resistance to *V. dahliae*, however, these lines have stunted growth, with reduced petiole elongation, shortened hypocotyl cell length, and fewer rosette leaves compared to wild type [36]. Similarly, overexpression of *GhTINY2* in cotton results in increased disease resistance, due to the upregulation of genes associated with defense responses, including SA biosynthesis and signaling genes, and decreased plant growth, in part due to reduced expression of genes associated with BR-dependent signaling [35]. In Arabidopsis lines overexpressing *GhTINY2*, repression of BR-mediated growth occurs by direct interaction between GhTINY2 and the Arabidopsis bHLH TF BRASSINAZOLE RESISTANT 1 (BZR1), resulting in suppression of BZR1-mediated gene expression of BR-regulated genes [35].

In cucumber, the *IRREGULAR VASCULAR PATTERNING* (*CsIVP*) gene encodes a bHLH transcription factor that regulates vascular development and is also involved in plant immunity [36]. RNAi lines specifically silencing *CsIVP* show defects in vascular differentiation and reduced plant growth. CsIVP directly binds to the promoters of several vascular developmental regulators and auxin-related genes, and *CsIVP* RNAi lines show increased auxin content. This is also accompanied by an increased expression of SA-regulated genes, including the SA-marker *PR-1*, and decreased susceptibility to downy mildew. Furthermore, the CsIVP protein interacts with CsNIMIN1, a cucumber orthologue of the Arabidopsis NIMIN1 protein involved in SA signaling, consolidating that an alteration in a developmental program affects SA signaling, and consequently, plant immunity. And in tomato, the expression of another bHLH TF, bHLH132, is up-regulated after SA or JA treatment and infection by the bacterial pathogen *Xanthomonas euvesicatoria* containing the effector XopD [37]. Overexpression of *bHLH132* leads to increased resistance to *X. euvesicatoria*, but also to narrower leaves with sharp, serrated edges, while silenced lines have stunted growth, shorter internodes, smaller leaves, and also show increased disease susceptibility [37].

Phytohormone-regulated transcription factors affecting plant immunity and root development

While most of the TFs regulating plant development and immunity described have roles in the plant shoot, roots are also the site of pathogen attack and defense, and immunity activation can also have developmental effects on roots [38]. The atypical transcription factor dimerization partner (DP)-E2F-like 1 (DEL1) is expressed in actively dividing cells to promote endoreduplication, and acts as a transcriptional repressor of the SA transporter ENHANCED DISEASE SUSCEPTIBILITY 5 (EDS5), restricting SA accumulation in actively growing shoots, as well as roots [39,40]. The knockout mutant *del1-1* is more resistant to infection by powdery mildew, resulting from increased total SA, but has reduced rosette and leaf size compared to wild type [40]. Additionally, *del1-1* mutant plants show ectopic patterns of lignification of root cells, which become more resistant to root-knot nematodes, and display an altered pattern of lateral root initiation [40].

Evidence of immunity activation regulating meristematic activity in roots is also supported by the AP2-domain transcription factors PLETHORA (PLT) 1 and 2, which mediate an auxin gradient that is essential for proper root apical meristem (RAM) patterning and root growth. The expression of *PLT1* and *PLT2* is repressed by JA, through direct binding of the main JA transcriptional regulator, the bHLH transcription factor MYC2, to their promoters. Repression of *PLT1* and *PLT2* promotes the division of the cells of quiescent center (QC) in the RAM and deregulated differentiation of the columella stem cells during root development [41]. Similarly, accumulation of SA also down-regulates *PLT1* and *PLT2* expression, and the expression of a QC marker, the homeodomain transcription factor WUSCHEL-RELATED 5 HOMEOBOX (WOX5), leads to increased ratios of QC cell division and decreased root growth [42]. This action of SA in the RAM is likely mediated by ROS production, is dependent on the main regulator of SA signaling NPR1 (NONEXPRESSOR OF PATHOGENESIS RELATED PROTEINS 1), and is associated with increased pathogen resistance [42].

Phytohormone-regulated transcription factors affecting plant immunity and meristem function and flowering

WUSCHEL (WUS) is a homeodomain transcription factor required for stem cell maintenance in the shoot apical meristem (SAM). Proper spatial expression of *WUS* in the SAM is regulated by phytohormones and is essential for the regulation of cell proliferation and differentiation in the SAM, resulting in primordia initiation [43]. The SAM has widely been known to be free from disease, so much so that culture of meristems is a known way to make plants virus-free. A role for WUS in maintaining a virus-free SAM was recently identified [44]. During infection of Arabidopsis with Cucumber Mosaic Virus (CMV), WUS functions to prevent viral infection in the cells comprising the SAM where WUS is expressed (WUS expression domain). Genetic and transgenic alteration of the WUS expression domain allows for CMV proliferation in WUS-free cells and the ability of the virus to take over the SAM. Gene expression profiling of the WUS domain identified genes encoding *S*-adenosyl-L-methionine-dependent methyl transferases (MTases), some of which had their expression induced by CMV but repressed by WUS, through direct binding of WUS to their promoters [44]. Because MTases are essential for global protein synthesis, the hypothesis put forward is that WUS represses global protein synthesis to prevent the viral takeover of the SAM, thus preventing infection. Whether this function of WUS is regulated by plant hormones, and whether WUS also modulates immune signaling in the SAM, remains untested. The correlation between total protein synthesis and pathogen proliferation provides a link between plant metabolism and growth to immunity, via a central transcriptional regulator of plant development.

Another important connection between meristem regulation and immunity has been uncovered in tomato, where a receptor-like kinase (RLK) was recently identified to regulate resistance to necrotrophic fungi, as well as plant development [45]. TRK1, an RLK from the RLK-VII subfamily, interacts with SlLYK1, the tomato orthologue of Arabidopsis CERK1 involved in fungal chitin perception that results in PTI activation. *TRK1* RNAi plants display decreased defense responses upon chitin perception and increased susceptibility to *B. cinerea*, by mechanisms that involve suppression of JA signaling, through the transcriptional and post-transcriptional regulation of MYC2 by TRK1. Pointing again to an intersection of plant immunity and development, TRK1 is required for chitin-induced growth suppression, and was also shown to physically interact and phosphorylate the TF SlWUS, the tomato orthologue of Arabidopsis WUS, and SlCLV1. As expected, given this interaction of TRK1 with key meristem regulators, *TRK1* RNAi lines also show altered reproductive developmental patterns, displaying ectopic meristems, and flowers with increased number of flower organs, including carpels, and altered WUS expression domains. Thus, phosphorylation of the transcriptional factor SlWUS by this RLK receptor TRK1 controls immunity and flower development. Of further interest is the fact that the function of TRK1 is spatially regulated, functioning to promote chitin-induced PTI in leaves, while suppressing it in meristematic tissue, likely through regulation of the TF SlWUS [45].

Finally, two major transcriptional regulators of flowering also have roles in response to pathogens. The MADS-domain transcription factor SHORT VEGETATIVE PHASE (SVP) is directly associated with a developmentally-regulated type of immunity, known as Age-Related Resistance (ARR), which is expressed in plants at the late adult vegetative and reproductive transition [46]. SVP is a major negative regulator of flowering, acting both in the SAM and in leaves. While SVP is not itself transcriptionally regulated by defense phytohormones, it mediates the transcriptional regulation of SA biosynthetic genes for direct defense against biotrophs, while acting at the same time as regulator of flowering initiation [46]. And a major regulator of the meristem transition into flowering and of floral organ patterning, the plant-specific helix-turn-helix transcription factor LEAFY (LFY), binds to the promoters and regulates the expression of several genes involved not only in developmental processes and phytohormone signaling, but also in defense against pathogens [47]. In addition to the impaired meristem transition observed in *lfy* mutants, characterized by the transformation of flowers into cauline leaves and shoots, these mutants also show decreased susceptibility to *Pst* in both rosette leaves and cauline leaves during reproductive development. Therefore, the transcriptional regulator of meristem transition LFY, integrates extrinsic cues of immunity activation to control of meristem function.

Engineering transcription factors for regulation of plant immunity and plant architecture

Given the complexity of phytohormonal signaling networks, and the extensive crosstalk amongst them, engineering hormonal networks for specific outcomes is a challenging endeavor. Thus, relying on regulators with specific roles, such as TFs, can be an attractive alternative to engineer hormonal-regulated processes. The identification of phytohormone-regulated transcription factors involved in the control of plant immunity and development opens the door to the manipulation of their expression and activity for specific outcomes of defense and development, with consequences to plant architecture and therefore yield.

One of the strategies successfully used has been the optimization of transcriptional levels of such regulatory

TFs. WRKY45 is a well-characterized rice TF induced by SA [48]. Overexpression of *WRKY45* leads to robust defense to *M. oryzae*, mediated by SA and cytokinin synergism [49], and to *Xoo*, however, these lines have decreased height and reduced number of flowers [48]. In an effort to optimize defense and growth, candidate promoters were identified and used to drive the expression of *WRKY45* [48]. Transgenic lines using the rice *UBIQUITIN7* promoter driving *WRKY45* resulted in increased disease resistance to both types of pathogens, and plant height, number of panicles, and seed number that were comparable to wild-type plants, thus eliminating tradeoffs due to increased immunity [48]. Regulation of levels of protein translation by the use of upstream open reading frames (uORF) of the transcription factor *TL1*-BINDING FACTOR 1 ($uORFs_{TBF1}$) has been used to drive expression of defense regulators resulting in increased immune responses without compromising plant yield [9]. This $uORF_{TBF1}$ approach was recently used to offset a developmental tradeoff in flowering observed in plants overexpressing the transcription factor CAM-BINDING PROTEIN 60-LIKE G (CBP60g), which is an essential transcriptional regulator of SA biosynthetic genes in response to pathogens [50].

In addition to manipulation of transcriptional and translational expression, post-translational modifications can also have a role in regulating TF activity, and therefore can be used to change TF function. In rice, a member of the SQUAMOSA PROMOTER BINDING PROTEIN-LIKE (SPL) family of TFs, Ideal Plant Architecture 1 (IPA1), is regulated at the post-translational level, with effects on reproductive yield and plant immunity, depending on its phosphorylation state [51]. In the non-phosphorylated state, IPA1 promotes vegetative growth and seed set [51]. Upon pathogen perception, IPA1 is phosphorylated, resulting in altered DNA binding specificity and IPA1-induced expression of *WRKY45* and resistance to *M. oryzae* [51]. Lines overexpressing *IPA1* are more resistant to *Xoo*, however, these transgenic plants are dwarfed, have delayed germination associated with reduced sensitivity to gibberellic acid, and have significantly reduced seed set compared to wild type [10]. Because overexpression of *IPA1* resulted in increased disease resistance with negative impacts to plant growth, the promoter of the RNA methyltransferase homolog of Arabidopsis HUA ENHANCER 1 (HEN1), containing a binding site for the *Xoo* TAL effector Tal9a, was used to drive expression of *IPA1* [10]. This resulted in a further increase in disease resistance to *Xoo* but also enhanced yield-related phenotypes, including the increased number of primary branches per panicle and increased grain number [10].

Another example of post-translational modification of a TF controlling development and immunity is the Arabidopsis TF WRKY70. WRKY70 acts as an activator of defense responses upon SA treatment or pathogen infection [52] and as a transcriptional repressor of defense genes under naive conditions [53]. The phosphorylation state of WRKY70 is responsible for its opposing roles in plant growth and immunity [54]. WRKY70 is phosphorylated in response to SA treatment or pathogen infection and promotes defense-related gene expression [53]. After accumulation of phosphorylated WRKY70 promotes defense responses, WRKY70 is ubiquitinated by the E3 ligase CHY ZINC FINGER AND RING PROTEIN 1 (CHYR1), leading to its degradation and reduction of defense-related gene expression [54]. Unphosphorylated WRKY70, on the other hand, acts as a transcriptional repressor of defense gene expression, allowing for normal plant growth [54]. In the absence of pathogen signals, lines overexpressing *WRKY70* display reduced stature, reduced tiller and panicle number, and leaf angle phenotypes associated with altered BR signaling [55].

Finally, synthetic biology approaches have also been recently used to alter hormone-regulated gene expression, such as the use of transgene or viral-mediated sgRNA and Cas9-based technologies to create transcription activators and repressors with programmable binding domains responsive to specific phytohormones. Such approaches have been successful in changing patterns of root branching and phyllotaxy and overall plant growth [56,57]. Similar concepts could be useful to manipulate phytohormone-regulated TFs in the context of plant immunity.

Conclusions

Beyond the normally characterized parameters of plant biomass, immunity activation also changes plant development, with consequences to plant architecture. The severity of these developmental alterations can vary after immunity is triggered by a pathogen attack, depending on the pathogen and pathogen inoculum [58], or can be induced by mutations that lead to constitutive immunity, or through activation with chemicals. Because plant architecture can have a substantial impact in plant productivity, it is critical to consider these developmental effects, along with the typical biomass phenotypes, when discussing tradeoffs of immunity.

In the past decade, several TFs regulating plant immunity but also with roles in plant development have been identified (highlighted here). It is likely that other TFs also play a role in the integration of immunity and plant development, whose roles in these processes have yet to be uncovered or may have been overlooked due to incomplete characterization of resulting phenotypes. As expected, many of these TFs have their expression regulated by phytohormones, underscoring the central role of these molecules in regulating both processes. Of note, many of the phenotypic outcomes from

manipulation of these TFs result from experiments using overexpression approaches, which may not reflect the native temporal and/or spatial regulation of these TFs, and therefore their inherent function. However, these experiments also demonstrate that the altered expression of these TFs may be used to change plant architecture in the context of immunity for better growth and yield, even in tissues in which their expression, or even pathogen infection, is not normally present.

Proper balance of immunity and development requires perception of specific pathogen and growth signals, followed by transcriptional reprogramming mediated by TFs, culminating in plant physiological changes. Engineering of transcription and translation of TFs, along with uncovering of post-translational modifications that change their regulation and DNA binding specificity, may be used to optimize plant architecture in active states of immunity, and consequently, plant productivity. While the contribution of the TFs mentioned here to immunity and plant development may have been defined, the identity of the upstream regulators that control their function and activation are mostly unknown. Similarly, the discovery of the transcriptional targets of these TFs, under native or overexpression conditions, is of paramount importance, as it may allow for more precise manipulation of the resulting traits. Furthermore, whether orthologues of these TFs function similarly in other plant species is still an open question. Unveiling the connection between the perception of growth and pathogen signals, and the resulting transcriptional reprograming, will be an important step to understand and manipulate developmental changes upon immunity activation, to control and optimize plant productivity.

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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 paper.

Data availability

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- • of special interest
- •• of outstanding interest

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