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REVIEW

Brassinosteroids: Multi-Dimensional Regulators of Plant Growth, Development, and Stress Responses

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Short title: Multiple Roles of Brassinosteroids

One-sentence summary: Brassinosteroids are a class of plant steroid hormones that are versatile regulators of growth, development, and responses to stresses such as extreme temperatures and drought.

ABSTRACT

Brassinosteroids (BRs) are a group of polyhydroxylated plant steroid hormones that are crucial for many aspects of a plant's life. BRs were originally characterized for their function in cell elongation, but it is becoming clear that they play major roles in plant growth, development and responses to several stresses such as extreme temperatures and drought. A BR signaling pathway from cell surface receptors to central transcription factors has been well characterized. Here, we summarize recent progress towards understanding the BR pathway, including BR perception and the molecular mechanisms of BR signaling. Next, we discuss the roles of BRs in development and stress responses. Finally, we show how knowledge of the BR pathway is being applied to manipulate the growth and stress responses of crops. These studies highlight the complex regulation of BR signaling, multiple points of crosstalk between BRs and other hormones or stress responses, and the finely tuned spatiotemporal regulation of BR signaling.

INTRODUCTION

The plant steroid hormone brassinosteroids (BRs) play important roles in plant growth and development, regulating diverse processes such as cell elongation, cell division, photomorphogenesis, xylem differentiation, and reproduction as well as both abiotic and biotic stress responses. The most active BR, brassinolide (BL), was purified from more than 200 kg of rapeseed (*Brassica napus*) pollen and its structure determined by X-ray analysis (Grove et al., 1979). The growth-promoting effect of crude lipid extract from rapeseed pollen was observed nine years earlier in a classic bean second-internode bioassay (Mitchell et al., 1970). In 1996, several independent studies involving the isolation of BR-insensitive and -deficient mutants of the model plant Arabidopsis (*Arabidopsis thaliana*) clearly established BRs as important endogenous growth regulators (Clouse et al., 1996; Li et al., 1996; Szekeres et al., 1996). Loss-of-function BR mutants displayed similar pleiotropic phenotypes including severe dwarfism, a dark-green color, and a de-etiolation phenotype when grown in darkness. In the case of BR-deficient mutants, these phenotypes could be rescued to that of the wild type by the external application of BRs. Molecular studies of BR mutants in Arabidopsis have led to the identification of BR receptors (Clouse et al., 1996; Li and Chory, 1997). Unlike animal steroid hormones, which bind nuclear receptors to directly modulate gene expression, BR receptors are plasma membrane (PM)-localized receptor kinases (Li and Chory, 1997; Cano-Delgado et al., 2004). In the past two decades, tremendous progress has been made in understanding the signal transduction pathways from cell surface receptors to the nucleus where thousands of genes are modulated in response to BRs to confer various biological responses (Figure 1 and Table 1) (Kim and Wang, 2010; Clouse, 2011; Guo et al., 2013; Dejonghe et al., 2014; Nolan et al., 2017a). In this review, we intend to capture the latest developments in the field by highlighting recent publications in the context of BR research over the past 25 years. We discuss the latest studies in BR perception, signaling, development, and stress responses. Finally, we provide an overview of BR functions and potential applications in crops.

BR PERCEPTION

BRs are perceived at the cell surface. The site of their biosynthesis is probably restricted to the endoplasmic reticulum (ER), as BR biosynthesis enzymes in Arabidopsis have been localized to the ER (Kim et al., 2006; Northey et al., 2016). From the ER, BRs are further transported into the apoplast, where they directly bind to the PM-localized receptors, BR INSENSITIVE1 (BRI1) (Friedrichsen et al., 2000; He et al., 2000) and its homologs, BRI1-LIKE1 (BRL1) and BRL3 (Cano-Delgado et al., 2004; Kinoshita et al., 2005).

The BRI1 ectodomain confers BR binding

BRI1, BRL1, and BRL3 belong to the leucine-rich repeat (LRR) receptor kinase (RK) family of proteins (Li and Chory, 1997; Cano-Delgado et al., 2004). BRI1 consists of an ectodomain that includes an N-terminal signal peptide, 25 LRRs, and a 70-amino acid island domain inserted between LRR21 and LRR22, followed by a single transmembrane domain and a cytoplasmic part including the juxtamembrane, kinase, and C-terminal domains (Vert et al., 2005). Despite sequence similarity to animal Toll-like receptors (TLRs) (Choe et al., 2005), structural studies demonstrated that the BRI1 ectodomain does not adopt the anticipated TLR-like horseshoe structure but forms a right-handed superhelix composed of 25 LRRs (Hothorn et al., 2011; She et al., 2011). The island domain then folds back into the interior of the superhelix to create a surface pocket for binding of the BR hormone (Hothorn et al., 2011; She et al., 2011). Historically, it was first concluded that the ectodomain of BRI1 perceives BRs based on a study involving a chimeric receptor consisting of a fusion of the ectodomain of BRI1 to the kinase domain of Xa21, a rice (*Oryza sativa*) disease resistance receptor. Exogenous application of BL to rice cells expressing the BRI1-Xa21 chimeric receptor triggered defense responses (He et al., 2000). Furthermore, it was shown that immunoprecipitated BRI1 conferred BR-binding and that a fully functional ectodomain was required for this binding (Wang et al., 2001). To identify the BR binding region, a series of truncated versions of the BRI1 ectodomain were generated and tested for binding to biotin-tagged photoaffinity castasterone, a synthetic precursor of BL. This analysis revealed that the minimal BR-binding domain of BRI1 consists of 94 amino acids comprising the island domain and the carboxy-terminal flanking LRR22 (Kinoshita et al., 2005).

Residues that are important for BR-binding have been revealed by solving the crystal structures of BRI1 ectodomain in complex with BL (Hothorn et al., 2011; She et al., 2011). LRRs 23-25 provide the hydrophobic surface for the binding of A-D rings of BL, while LRR21, LRR22, and the island domain form a pocket for the binding of the alkyl chain of BL (Hothorn et al., 2011; She et al., 2011). These findings were corroborated by homology modeling with BRL2, a homolog of BRI1 that does not bind BRs (Cano-Delgado et al., 2004; Kinoshita et al., 2005), and by solving the crystal structure of BRL1 in complex with BL (She et al., 2013). Thus, it was proposed that the substitution of Ile642 (in BRL1) or Met657 (in BRI1) to Glu614 (in BRL2) might interfere with the BL-binding by changing the hydrophobicity of this region (She et al., 2013). Several *bri1* mutants with point mutations in the island domain-LRR interface have been identified (Li and Chory, 1997; Noguchi et al., 1999; Sun et al., 2017). It remains to be demonstrated if BRI1 mutants carrying these molecular lesions are deficient in BR binding, which would further confirm the importance of this region. The BL binding pocket in BRI1 is highly hydrophobic and relatively small. Accordingly, the introduction of polar or bulky groups into the BL molecule attenuates its bioactivity (Wang et al., 2001; Back and Pharis, 2003). This further emphasizes the significance of hydrophobic interactions between BL and the BRI1 island domain. Although most of the residues contributing to the formation of the BL binding pocket are conserved, BRL2 does not bind to BL, and BRL3 showed decreased BL binding compared to BRI1 (Cano-Delgado et al., 2004; Kinoshita et al., 2005). Further studies are needed to identify the detailed molecular basis for the differences in BL binding among BRI1, BRL2, and BRL3.

BRs function as a molecular glue to bring BRI1 and its co-receptors together

Upon BL binding, the island domain in the BRI1 ectodomain becomes ordered and its position with respect to the LRR core becomes fixed (Hothorn et al., 2011; She et al., 2011), which creates a docking platform for the binding of a co-receptor protein required for BRI1 activation. One such co-receptor is SOMATIC EMBRYOGENESIS RECEPTOR KINASE3 (SERK3)/BRI1-ASSOCIATED KINASE1 (BAK1). This protein was previously characterized as a BRI1-interacting protein (Li and Nam, 2002; Nam and Li, 2002; Russinova et al., 2004; Wang et al., 2005a; Wang et al., 2008), a genetic component of BR signaling (Li et al., 2002; Nam and Li, 2002), and a BRI1 phosphorylation target (Li et al., 2002; Nam and Li, 2002). SERK3/BAK1 belongs to a subfamily of five smaller LRR RKs (SERK1-5) that regulate plant growth, development, and immunity and play a critical, redundant role in BR signaling (Chinchilla et al., 2007; Heese et al., 2007; Gou et al., 2012; Meng et al., 2015; Hohmann et al., 2018b). The interaction between BRI1 and SERK3/BAK1 is ligand-dependent (Wang et al., 2005b; Wang et al., 2008; Hothorn et al., 2011; Jaillais et al., 2011a; She et al., 2011; Santiago et al., 2013), although a portion of BRI1 and BAK1 heterodimers may exist in the absence of BRs (Bucherl et al., 2013). The crystal structures of the BRI1-BL-SERK1 and BRI1-BL-SERK3/BAK1 ectodomain complexes suggest that BL acts as a molecular glue, promoting the association between BRI1 and BAK1 (Santiago et al., 2013; Sun et al., 2013). These two structures are comparable because BL- and BRI1-interacting amino acids are highly conserved among the SERK proteins (Santiago et al., 2013; Sun et al., 2013). Structural data reveal that the ectodomain of SERK1 makes contacts with the BRI1-bound BL, the island domain, and LRR25 of BRI1 (Santiago et al., 2013). Consistent with this finding, a substitution of Thr750 with a bulkier Ile in BRI1 may perturb the direct BRI1-SERK3/BAK1 interactions, causing the compromised BR signaling observed in *bri1-102* (Friedrichsen et al., 2000). In addition, a substitution of Asp122 with a less hydrophilic Asn in SERK3/BAK1 may cause additional interactions between SERK3/BAK1 and BRI1, causing a BR-hypersensitive phenotype (Jaillais et al., 2011a). The hydrogen bonds established between SERK1 and the 2a, 3a-diol moiety of BL are important for BR signaling activation, as BR derivatives in which the two hydroxyls in BL were replaced by methyl ethers (Back et al., 2002) or acetonide (Muto and Todoroki, 2013) exhibited weakened activity.

Negative regulators of the BRI1-SERK3/BAK1 association

In the absence of BRs, BRI1 is kept in an inactive state by auto-inhibition through its C-terminal domain (Wang et al., 2005a), auto-phosphorylation in the kinase domain (Wang et al., 2005b; Oh et al., 2009; Oh et al., 2012b; Bojar et al., 2014), and interaction with the inhibitory protein BRI1 KINASE INHIBITOR1 (BKI1) (Wang and Chory, 2006; Jaillais et al., 2011b; Jiang et al., 2015b). BKI1 associates with the PM (Jaillais et al., 2011b) and interacts with BRI1 by binding to the C-lobe of its kinase domain (Wang et al., 2014a). As this part of BRI1 is required for the binding of the SERK3/BAK1 kinase domain, BKI1 interferes with BRI1-SERK3/BAK1 interactions (Jaillais et al., 2011b; Bojar et al., 2014). BR-induced heterodimerization of BRI1 and the SERK3/BAK1 ectodomains brings their cytoplasmic kinase domains to the correct orientation to remove BKI1-induced inhibition and to trigger trans-phosphorylation of these two kinase domains. Activated BRI1 rapidly phosphorylates BKI1 (Wang et al., 2014a), thereby affecting the positive charge of the BKI1 membrane association domain (Simon et al., 2016), leading to its release from the PM and the full activation of the BRI1-SERK3/BAK1 complex. Negative regulators of the co-receptor SERK3/BAK1 have also been reported. For example, BAK1-INTERACTING RECEPTOR-LIKE KINASES3 (BIR3), which was identified as an *in vivo* SERK3/BAK1 complex partner (Halter et al., 2014), inhibits the formation of BRI1-SERK heterodimers by interacting with the ectodomains of SERKs (Hohmann et al., 2018a), thus negatively regulating BR signaling. After BR exposure, BIR3 is released from SERK3/BAK1 and BRI1 (Imkampe et al., 2017). Along with the removal of negative regulators, reciprocal phosphorylation between the BAK1 and BRI1 kinase domains occurs. This process begins with

BRI1 phosphorylating SERK3/BAK1 (Wang et al., 2008). The phosphorylated SERK3/BAK1 locks itself into the active conformation (Yan et al., 2012) and further phosphorylates BRI1 (Wang et al., 2008) to fully activate the receptor complex.

BR SIGNALING

Following BR perception at the PM by BRI1 and SERK3/BAK1, a well-established cascade relays BR signals to BRI1-EMS-SUPPRESSOR1 (BES1) and BRASSINAZOLE-RESISTANT1 (BZR1) family TFs, which control BR-regulated gene expression (He et al., 2002; Wang et al., 2002; Yin et al., 2002; Zhao et al., 2002; Yin et al., 2005; Yu et al., 2011). In this section, we provide an update on core BR signaling mechanisms and focus on emerging research that is revealing how BR signaling is modulated and connected with other signaling pathways.

Inhibition of BR signaling

When BR levels are low, BR signaling is attenuated through multiple mechanisms. The glycogen synthase kinase3 (GSK3)-like kinase BRASSINOSTEORID INSENSITIVE2 (BIN2) functions as a negative regulator of BR signaling. BIN2 phosphorylates numerous substrates including BES1 and BZR1 (Li et al., 2001; He et al., 2002; Li and Nam, 2002; Youn and Kim, 2015). BIN2-induced phosphorylation inactivates BES1 and BZR1 by promoting their cytoplasmic retention via 14-3-3 proteins (Gampala et al., 2007; Ryu et al., 2007), inhibiting their DNA-binding activity (Vert and Chory, 2006), and stimulating their degradation (He et al., 2002; Yin et al., 2002; Nolan et al., 2017b; Kim et al., 2019). Moreover, BES1 and BZR1 are kept in an inactivate state through interactions with the BROAD COMPLEX/TRAMTRACK/BRIC-A-BRAC (BTB) family protein BRZ-SENSITIVE-SHORT HYPOCOTYL1 (BSS1) under BR-deficient conditions (Shimada et al., 2015).

Activation of BR signaling

When BRs are present, the BRI1 and BAK1 receptor complex activates downstream cytoplasmic kinases BR SIGNALING KINASES (BSKs) and CONSTITUTIVE DIFFERENTIAL GROWTH1 (CDG1) (Tang et al., 2008; Kim et al., 2011; Sreeramulu et al., 2013), which in turn activate the phosphatase BRI1-SUPPRESSOR1 (BSU1) (Kim et al., 2009; Kim et al., 2011). A recent genetic screen identified several semi-dominant mutants of *bsk3*. Analysis of these mutants revealed that anchoring of BSK3 to the PM via N-myristoylation is essential for BR signaling. BSK3 interacts with BRI1 and additional BSK family proteins BSU1 and BIN2, suggesting that BSK3 functions as a BR signaling scaffold (Ren et al., 2019). Moreover, BIN2 phosphorylates BSK3, promoting BSK-BSK, BSK-BRI1, and BSK-BSU1 interactions. In turn, BSK3 upregulates *BSU1* transcript and protein levels to enhance BR signaling (Ren et al., 2019). Thus, although BIN2 functions primarily as a negative regulator of BR signaling, it also engages in a feedback loop that promotes BR signaling through phosphorylation of the positive regulator BSK3.

In line with the notion that scaffolding plays an important role in BR signaling, TETRATRICOPEPTIDE THIOREDOXIN-LIKE (TTL) proteins TTL1, TTL3, and TTL4 have recently been implicated in bringing BR signaling components together at the PM (Amorim-Silva et al., 2019). TTL3 forms a complex with several players in BR signaling, including BRI1, BSU1, and BZR1. TTL3 is localized to the cytoplasm, but BR treatment leads to its association with the PM where it serves to bring BR signaling components together. This allows BSU1 to dephosphorylate Tyr200 in BIN2, thereby inactivating this protein (Kim et al., 2009). Several other mechanisms that control the activity of BIN2 have been described (Figure 2). For example, BIN2 is degraded by the proteasome in the presence of BRs (Peng et al., 2008). The F-box E3 ubiquitin ligase KINK SUPPRESSED IN *BZR1-1D* (KIB1) mediates the ubiquitination and subsequent degradation of BIN2 in the presence of BRs while also blocking BIN2-substrate interactions (Zhu et al., 2017). BIN2 is also regulated in a cell type-specific manner through scaffolding. For example, OCTOPUS sequesters BIN2 to the PM in the phloem (Truernit et al., 2012; Anne et al., 2015), and POLAR sequesters this protein to the PM in the stomatal cell lineage (Houbaert et al., 2018). The BIN2-OCTOPUS interaction inhibits BIN2-induced phosphorylation of BES1 and BZR1 by confining BIN2 to the PM, thereby promoting phloem differentiation (Truernit et al., 2012; Anne et al., 2015). Similarly, POLAR regulates the activity of BIN2 and several related GSK3-like kinases by controlling their localization, thus allowing different BIN2/GSK3-like kinase substrates to be phosphorylated (Houbaert et al., 2018). Several post-translational modifications also affect the activity of BIN2. HDA6 deacetylates BIN2, thereby repressing its kinase activity (Hao et al., 2016), whereas BIN2 is activated via oxidation at specific cysteine residues (Song et al., 2018b). Dephosphorylation by ABI1 and ABI2 (which negatively regulate abscisic acid [ABA] signaling) activates BIN2 in the presence of ABA

(Wang et al., 2018a; Jiang et al., 2019). These diverse regulatory mechanisms highlight the multifaceted control of BIN2 kinase activity under different conditions and in different tissues. The complex regulatory mechanisms of BIN2, coupled with the multitude of substrates that are phosphorylated by this protein (Youn and Kim, 2015), underline the importance of this versatile kinase.

BES1 and BZR1 control BR-regulated gene expression

The inactivation of BIN2 by BRs along with the dephosphorylation of BES1 and BZR1 by PROTEIN PHOSPHATASE2A (PP2A) (Tang et al., 2011) allow BES1 and BZR1 to become active in the nucleus to control BR-responsive gene expression (He et al., 2002; Wang et al., 2002; Yin et al., 2002; Zhao et al., 2002; Yin et al., 2005; Yu et al., 2011). BES1, BZR1, and the homologs BEH1-BEH4 are atypical basic helix-loop-helix (bHLH) TFs that function redundantly as master regulators of BR-responsive gene expression (Wang et al., 2002; Yin et al., 2002; Yin et al., 2005; Chen et al., 2019a). BRs modulate the expression levels of ~5,000-8,000 genes, approximately half of which are induced and the other half repressed by BRs (Guo et al., 2013; Wang et al., 2014b; Nolan et al., 2017b). The identification of BES1 and BZR1 target genes genome-wide played a central role in characterizing the BR-regulated gene network (Sun et al., 2010; Yu et al., 2011; Oh et al., 2012a). These studies revealed hundreds of BES1/BZR1-targeted TFs (BTFs) that are regulated by BRs (Sun et al., 2010; Yu et al., 2011; Guo et al., 2013). Signal amplification through these TFs may at least partially explain how BRs are able to regulate a large number of genes. A theme that has emerged is that BTFs often physically interact with BES1 and/or BZR1 to cooperatively or antagonistically regulate gene expression. Thus, these BTFs interface with the BR signaling pathway at multiple levels: they are both targets of BR regulation, as BES1 or BZR1 binds to their promoters, and mediators of BR responses through interacting with BES1 or BZR1 to regulate the expression of other BR-responsive genes.

The observation that BES1 and BZR1 can either induce or repress gene expression leads to the question of what dictates their activation versus repressive activity. Comparisons of BES1 and BZR1 targets using BR-responsive transcriptome data showed that BR-induced genes are enriched in E-Box (CANNTG) binding sites, whereas BES1 and BZR1 repress gene expression by binding to BRRE elements (CGTG(T/C)G) in the promoters of their target genes (Sun et al., 2010; Yu et al., 2011). BES1 and BZR1 cooperate with other TFs, histone-modifying enzymes, and transcriptional regulators to activate BR-induced gene expression. For example, BES1-INTERACTING MYC-LIKE PROTEIN1 (BIM1) interacts with BES1; these proteins synergistically bind to E-Box elements in their BR-induced target genes to activate their expression (Yin et al., 2005). Similarly, BES1 cooperates with MYB30 to promote BR-induced gene expression (Li et al., 2009). Further insight into how BES1 and BZR1 repress gene expression comes from studies of MYBL2 and HAT1, which positively regulate the BR pathway by assisting BES1 in BR-repressed gene expression (Ye et al., 2012; Zhang et al., 2014b). BES1 and BZR1 also contain an ETHYLENE RESPONSE FACTOR (ERF)-associated amphiphilic repression (EAR) domain, which mediates interactions with TOPLESS (TPL) proteins (Oh et al., 2014a; Ryu et al., 2014; Espinosa-Ruiz et al., 2017), allowing the recruitment of HISTONE DEACETYLASE19 (HDA19) to mediate histone deacetylation and thus repress gene expression. BES1- and BZR1-repressed genes include genes encoding rate-limiting enzymes involved in BR biosynthesis, forming a negative feedback loop to inhibit the BR pathway (He et al., 2005). Analysis of BES1 binding sites by protein binding microarray analysis suggested that BES1 likely binds to the BRRE sites of BR-repressed genes as a homodimer. By contrast, the regulation of BR-induced gene expression involves the formation of heterodimers between BES1 or BZR1 and other TFs such as PHYTOCHROME INTERACTING FACTOR4 (PIF4) (Martinez et al., 2018).

Recent structural studies indicated that BZR1 family TFs contain a bHLH-like DNA binding domain that recognizes both CACGTG (G-box, a specific E-box) and CGTG (core of BRRE site) elements (Nosaki et al., 2018). Amino acid residues in this domain that determine binding specificities, binding affinities, and dimer formation were identified (Nosaki et al., 2018). Further structural studies with full-length BES1 or BZR1 and possible heterodimers between BES1, BZR1, and other classical bHLH protein, such as BIM1 and PIF4, should help reveal how BES1 and BZR1 can either activate or repress a large number of genes to generate various biological responses.

BR-induced gene expression involves cooperative TFs and interplay with light signaling, auxin, and gibberellins

The first BR-related Arabidopsis mutants were isolated in a forward genetic screen for seedlings with de-etiolated morphology in the dark. These mutants were characterized by short, thick hypocotyls and open, expanded cotyledons (Chory et al., 1991). It was therefore clear from very early genetic studies that this group of hormones is involved in regulating light responses in plants.

Extensive research in recent years has led to the characterization of several points of interaction between BR and light signaling components. First, a direct link came from identifying protein-protein interactions between BZR1 and PIF4, explaining the binding of these proteins to numerous overlapping genomic targets (Oh et al., 2012a). BES1, PIF4, and the BES1-PIF4 complex recognize different DNA elements. Interaction with PIF4 alters the binding site of BES1, switching specificity from a BRRE site associated with BR-repressed genes to a CATGTG element that is enriched in BR- and PIF-upregulated genes (Martinez et al., 2018). Consequently, the formation of BES1-PIF4 dimers leads to de-repression of BR biosynthetic genes at dawn and an increase in BR levels (Martinez et al., 2018). Crosstalk between these two signaling pathways also occurs at the level of BIN2 kinase, which phosphorylates PIF4 and PIF5 and targets them for proteasomal degradation. In this way, BR signaling, which leads to the inactivation of BIN2, promotes the stabilization of PIF4 and contributes to the timing of hypocotyl elongation to late at night, before the activation of light signaling (Bernardo-Garcia et al., 2014). Moreover, the active form of BZR1 interacts with LONG HYPOCOTYL5 (HY5), another major TF involved in light signaling. BZR1-HY5 interactions regulate cotyledon development and opening during photomorphogenesis (Li and He, 2016). Finally, several photoreceptors impinge on BES1 to inhibit the BR pathway. UV light receptor UVR8 inhibits the DNA binding activity of BES1 and BIM1, providing a molecular mechanism by which UV light inhibits plant growth (Liang et al., 2018). Similarly, cryptochromes CRY1 and CRY2 interact with BIM1 and dephosphorylate BES1 in response to blue light to inhibit BES1-DNA binding (Wang et al., 2018b). CRY1 interacts with BZR1 to inhibit DNA binding, and BZR1 phosphorylation is also promoted by the CRY1-BIN2-BZR1 regulatory module, providing another mechanism by which BR signaling is inhibited in blue light (He et al., 2019). In response to red light, photoactivated phyB interacts with dephosphorylated BES1 to inhibit its transcriptional activity (Wu et al., 2018). Taken together, these observations implicate BES1 and BZR1 as major targets for light-mediated inhibition of hypocotyl elongation by BRs and suggest that BR-activated gene expression is attenuated by light signals. In addition to the regulation of plant development in darkness, BRs play a role in blue-light-mediated shade avoidance. Under reduced blue light conditions, BRs modify the expression levels of genes encoding XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASES (XTHs; enzymes that regulate cell wall extensibility), thereby governing hypocotyl elongation synergistically with auxin (Keuskamp et al., 2011).

BES1 and BZR1 interact with other growth-promoting TFs such as the auxin-regulated ARFs. ARF6 interacts with both PIFs and BZR1 to cooperatively regulate gene expression (Oh et al., 2014b). ARF6 co-occupies nearly half of the target genes of BZR1 and PIF4, and the BZR1-PIF4-ARF6 trio controls cell elongation through downstream targets such as PACLOBUTRAZOL-RESISTANT (PRE), ILI1 BINDING bHLH PROTEIN1 (IBH1), and HOMOLOG OF BEE2 INTERACTING WITH IBH1 (HBI1), which form a tri-antagonistic loop (Wang et al., 2009; Zhang et al., 2009a; Bai et al., 2012b; Ikeda et al., 2012). BRs also undergo crosstalk with the GA pathway. Under low GA conditions, BES1, BZR1, PIF4, and ARF6 are inhibited by DELLAs (Bai et al., 2012a; Gallego-Bartolome et al., 2012; Li et al., 2012). Hence, when GA is present, the degradation of DELLAs allows for the activation of the BZR1-PIF4-ARF6 module to promote growth responses. BRs also regulate GA levels (Tong et al., 2014; Unterholzner et al., 2015), suggesting that BR-GA crosstalk is manifested through both GA biosynthesis and signaling. Taken together, BRs integrate multiple hormonal and environmental inputs, which eventually leads to the activation or repression of BR-regulated genes, but some components of the signaling cascade, such as BZR1/BES1 family members, are shared with other, BR-independent pathways.

A recently isolated hextuple BZR1/BES1 family mutant (*bzr-h*) exhibited defects in anther development, which are not present in the triple BR signaling mutant *bri1 brl1 brl3* (Chen et al., 2019a). Follow-up studies clearly demonstrated that besides their role in BR signaling, members of the BZR1/BES1 TF family function in tapetum development, which is mediated by another LRR RK, EXCESS MICROSPOROCYTES1 (EMS1), via a BR-independent signaling cascade (Chen et al., 2019b; Zheng et al., 2019). The extracellular domain of EMS1 perceives the signaling peptide TAPETUM DETERMINANT1 (TPD1) to regulate tapetum development, but EMS1 shares an interchangeable intracellular kinase domain with BRI1, suggesting that these two receptors have a common ancestor (Zheng et al., 2019). It would be interesting to determine the mechanisms by which TPD1-EMS1 signaling modulates BES1 activity.

BR-regulated gene expression involves histone modifications and chromatin remodeling

DNA within the nucleus is packaged in chromatin, which involves interactions between DNA and histone proteins. Histone tails can be modified (e.g. by methylation, ubiquitination, or acetylation) to affect chromatin compaction and activate or repress gene expression. BR-regulated gene expression involves epigenetic mechanisms including histone modifications. One such mark associated with the repression of gene expression is histone 3 lysine 27 di- and tri-methylation (H3K27me2/H3K27me3). BES1 interacts with EARLY FLOWER6 (ELF6) and RELATIVE OF EARLY FLOWER6 (REF6), which function as positive regulators in the BR pathway by removing repressive H3K27me2/H3K27me3 marks (Yu et al., 2008; Lu et al., 2011), thus allowing BES1 to activate gene expression. BR-induced gene expression at these loci likely involves chromatin remodeling, since REF6 interacts with the SWI/SNF-type chromatin remodeler BRAHMA (Li et al., 2016). Likewise, BZR1 and PIF3 interact with the chromatin remodeler PICKLE to repress H3K27me3 on target gene promoters and allow for BR-induced gene expression (Zhang et al., 2014a). These observations underscore the importance of histone-modifying enzymes and chromatin-remodeling factors in the de-repression of BR-regulated genes.

Other histone modifications promote gene activation. This is the case for histone 3 lysine 36 di- and tri-methylation (H3K36me2/3), which positively regulates BR responses and is required for the activation of a large portion of BR-induced genes (Sui et al., 2012; Wang et al., 2014b). BES1 interacts with the transcription elongation factor INTERACTING-WITH-SPT6-1 (IWS1) to promote BR-regulated gene expression (Li et al., 2010). In turn, BES1 and IWS1 recruit the H3K36 methyltransferase SDG8 to the promoters of BR-regulated genes to increase H3K36me2/3 levels and allow for the activation of BR-induced gene expression (Wang et al., 2014b). Together, the removal of repressive H3K27me2/3 and the addition of H3K36me2/3 marks allow for the activation of BR-regulated genes. Further studies should explore the relationship between BR signaling and other histone modifications such as H3K9 or H3K4 methylation, which generally repress or activate transcription, respectively. Given the connection of BRs with chromatin remodeling factors, it would also be interesting to study how BRs influence chromatin accessibility, as such studies have yielded important insights into how other hormones regulate gene expression (Potter et al., 2018).

BES1 and BZR1 regulation: more than just phosphorylation

Non-phosphorylated forms of BES1 and BZR1 can be observed within minutes of BR treatment (Yin et al., 2002; Yin et al., 2005). Given the availability of high-quality BES1 (Yu et al., 2011) and BZR1 antibodies (Jeong et al., 2015; Zhang et al., 2016), the phosphorylation status of BES1 and BZR1 has been one of the most reliable, widely used markers of BR pathway activity. While BIN2-mediated phosphorylation inhibits the activity of BES1 and BZR1, MITOGEN-ACTIVATED PROTEIN KINASE6 (MPK6) can phosphorylate BES1 to promote its activity (Kang et al., 2015). Hence, the perception of pathogen-associated molecular patterns (PAMPs) leads to the MPK6-mediated phosphorylation of BES1 (Kang et al., 2015). The residues of BES1 phosphorylated by MPK6 affect its role in immunity but not BR-regulated growth, indicating that site-specific phosphorylation differentially modulates BES1 activity. Phosphorylation is not the only way to regulate BES1 and BZR1 activity. Recent work is revealing that BES1 and BZR1 are controlled through numerous additional mechanisms including oxidation, alternative splicing, ubiquitination, and degradation (Figure 3).

One such modification involves the oxidation of BES1 and BZR1 by reactive oxygen species (ROS). Although they were once thought to merely be toxic reaction byproducts, there is a growing appreciation for the role of ROS (including H_2O_2) as important signaling molecules (Mittler et al., 2011). BRs induce H_2O_2 production to modulate several processes including stomatal movement and stress responses (Xia et al., 2014; Shi et al., 2015; Tian et al., 2018). H_2O_2 is required for BR-regulated growth and BR-responsive gene expression (Tian et al., 2018). BRs trigger an NADPH oxidase-dependent burst of H_2O_2 through an unknown mechanism, which leads to the oxidation of BZR1 and BES1 on residues Cys63 and Cys84, respectively. Oxidation of BZR1 promotes BZR1-ARF6 and BZR1-PIF4 interactions (Tian et al., 2018). Thus, in addition to inhibiting BIN2-induced phosphorylation of BES1/BZR1, BRs activate BES1 and BZR1 through H_2O_2-mediated oxidation. Furthermore, the thioredoxin TRXh5 interacts with BZR1 to promote its reduction, leading to its inactivation. BRs inhibit the expression of *TRXh5*, thus ensuring the redox-mediated activation of BZR1 and BES1 (Tian et al., 2018).

Another layer of BES1 regulation comes from alternative splicing of the *BES1* transcript. *BES1* exists in two forms: the canonical and widely studied transcript encoding a 335 amino acid protein (*BES1-S*) and an alternative splicing variant that adds an additional 22 amino acids to the N-terminus of BES1 (*BES1-L*). The additional sequence in *BES1-L* adds another nuclear localization signal (NLS), rendering BES1-L constitutively nuclear localized. Overexpression of *BES1-L* results in stronger BR-gain-of-function phenotypes compared to *BES1-S*, suggesting that *BES1-L* is more active *in planta.* These phenotypes may be explained by the nuclear accumulation of BES1-L along with its ability to interact with BES1-S and BZR1 to promote their nuclear localization. Despite the strong activity of BES1-L, it appears that *BES1-S* is more highly expressed. *BES1-S* is induced by BR treatment or in *bes1-D* mutants, whereas *BES1-L* is repressed (Jiang et al., 2015a). These findings point towards complex regulation of *BES1* and illustrate that different splice variants can result in altered biological activities.

Lastly, BES1 and BZR1 are regulated by multiple E3 ubiquitin ligases that control their degradation through the 26S proteasome or selective autophagy. *bes1-D* and *bzr1-D* mutants were originally identified as harboring gain-of-function mutations leading to a Pro to Leu substitution in the PEST domain of BES1 or BZR1 (Wang et al., 2002; Yin et al., 2002). This mutation is associated with dramatic accumulation of BES1 protein, as monitored by immunoblotting, suggesting that BES1 is subject to post-translational control (Yin et al., 2002). Indeed, BES1 and BZR1 interact with E3 ubiquitin ligases such as the F-Box protein MORE AXILLARY GROWTH LOCUS2 (MAX2). BES1 is ubiquitinated by MAX2 in response to strigolactone (SL) to suppress shoot branching (Wang et al., 2013). Interestingly, the mutation found in *bes1-D* impairs the interaction between BES1 and MAX2, indicating that the stabilization of BES1 in *bes1-D* mutants may be at least partially explained by the disruption of BES1-E3 ubiquitin ligase interactions. Similarly, BZR1 interacts with PLANT U-BOX40 (PUB40), which specifically degrades BZR1 in roots but not in shoots. PUB40 is phosphorylated and stabilized by BIN2 kinase, leading to the degradation of BZR1 under low BR concentrations (Kim et al., 2019). Like the BES1-MAX2 interaction, the gain-of-function *bzr1-D* mutation diminishes the interaction between PUB40 and BZR1. These studies point toward the regulation of BES1 and BZR1 in specific tissue/organ, developmental, or environmental contexts. In line with this idea, several E3 ubiquitin ligases degrade BES1 or BZR1 under changing light conditions. CONSTITUTIVE PHOTOMORPHOGENIC1 (COP1) targets the inactive, phosphorylated form of BZR1 in the dark, whereas SINAT (SINA of *Arabidopsis thaliana*) E3 ubiquitin ligases mediate the degradation of the active dephosphorylated form of BES1/BZR1 in the light (Kim et al., 2014; Yang et al., 2017; Yang and Wang, 2017). SINAT levels decrease in the dark but increase in the light, explaining why these regulators target BES1 in the light (Yang et al., 2017).

Diurnal rhythms add to the complexity of BES1/BZR1 protein regulation. Under short-day conditions, BES1 protein levels fluctuate dramatically, peaking ~2 hours after dawn. BES1-GFP is stabilized by light under these conditions, but BZR1 and BES1-L do not display light-induced stabilization (Martinez et al., 2018). Additional studies are needed to separate the effects of light, the circadian clock, and BR levels on BES1 and BZR1 protein regulation, which could further define the roles of the different E3 ubiquitin ligases involved in BES1 and BZR1 degradation. One important observation is that no E3 ubiquitin ligase mutant has been identified with phenotypes or BES1/BZR1 protein accumulation comparable to *bes1-D* or *bzr1-D*. Therefore, the construction of higher-order mutants for different E3 ubiquitin ligases involved in BES1 and BZR1 degradation and/or the identification of additional players in this process will be an important direction for future research. BSS1, also known as BLADE ON PETIOLE1 (BOP1), acts as part of a CUL3$^{BOP1/BOP2}$ E3 ubiquitin ligase complex that facilitates PIF4 degradation during photomorphogenesis (Zhang et al., 2017). Thus, given that BSS1/BOP1 also interacts with BES1 and BZR1 and affects their protein levels (Shimada et al., 2015), it would be interesting to determine if BSS1 plays a role in BES1 and BZR1 ubiquitination.

Upon ubiquitination, protein degradation typically occurs through the 26S proteasome or the autophagy pathway (Floyd et al., 2012). Specific cargos can be recruited for autophagy-mediated degradation with the help of autophagy receptor proteins that bind to the autophagy protein ATG8, which decorates autophagic membranes (Marshall and Vierstra, 2018). DOMINANT SUPPRESSOR OF KAR2 (DSK2) is an autophagy receptor for BES1 degradation (Nolan et al., 2017b). DSK2 contains a ubiquitin-associated domain that recognizes poly-ubiquitin chains (Farmer et al., 2010; Lin et al., 2011), along with an ATG8-interacting motif (Nolan et al., 2017b) that binds to ATG8. DSK2 interacts with ubiquitinated BES1, and BES1-DSK2-ATG8 interactions mediate BES1 degradation through the autophagy pathway during drought or fixed-carbon starvation stress. Furthermore, BIN2 phosphorylates DSK2 proximal to its ATG8-interacting motif, thereby promoting DSK2-ATG8 interactions and BES1 degradation through autophagy. SINAT2, an E3 ubiquitin ligase for BES1, also interacts with DSK2, suggesting that the ubiquitination and subsequent degradation of BES1 may be coordinated (Nolan et al., 2017b). Although a link between DSK2 and BZR1 remains to be explored, BZR1 was shown to be stabilized by TOR kinase, a negative regulator of autophagy. Decreased TOR levels in *tor RNAi* plants led to reduced BZR1 levels, which were restored by treatment with the autophagy inhibitor 3-methyladenine (Zhang et al., 2016). Therefore, both BES1 and BZR1 appear to be degraded by the autophagy pathway, although much remains to be learned about this process, including what controls whether these proteins are degraded through the proteasome versus the autophagy pathway.

THE ROLE OF BRs IN DEVELOPMENT

Characterization of BR biosynthetic and signaling mutants, together with studies of the effects of exogenous BR application, have unambiguously shown that BRs are important for plant development. Numerous developmental processes are affected when BR signaling is perturbed (Figure 4), such as seed development (Jiang et al., 2013a), flowering time (Domagalska et al., 2010), and pollen development (Ye et al., 2010). BRs coordinate the tropic responses of plant organs by regulating polar auxin transport (Li et al., 2005). The modulation of auxin transport by BRs is also reflected by the promotion of lateral root primordial initiation during lateral root development (Bao et al., 2004), while root hair cell development is an example of BR signaling taking part in cell fate determination through the regulation of the WEREWOLF-GLABRA3/ENHANCER OF GLABRA3-TRANSPARENT TESTA GLABRA1 transcriptional complex (Cheng et al., 2014). Differentiation of cambial cells into xylem vessels is dependent on GSK3-like kinases; these master regulators of the BR signaling pathway act as downstream components of the TRACHEARY ELEMENT DIFFERENTIATION INHIBITORY FACTOR (TDIF) signaling pathway (Kondo et al., 2014).

BR signaling is tissue specific

Most phenotypic defects of BR biosynthetic mutants can be rescued by exogenous BR treatment. As with other hormones, plant responses to exogenous BR application are dose-dependent, with growth-promoting effects observed for lower concentrations and growth retardation when higher doses are used (Chaiwanon and Wang, 2015; Belda-Palazon et al., 2018). Initial dissection of BR signaling at the tissue level has provided clues that the epidermal layer is the site of the most intensive BR signaling events, as the dwarf phenotypes of BR receptor or BR biosynthetic enzyme mutants could be rescued by epidermis-specific expression of the mutated genes (Savaldi-Goldstein et al., 2007). These findings led to more detailed studies of the Arabidopsis root meristem, which is a favorite model organ for developmental biologists due to its simplicity (Jaillais and Vert, 2016). However, these studies have led to some conflicting conclusions. Several papers emphasize the importance of BR signaling in the root tip epidermis. Two studies (Hacham et al., 2011; Vragovic et al., 2015a) demonstrated that BR signaling in the root epidermis and not in the inner tissues is sufficient to restore meristem size in the *bri1* mutant, just as others demonstrated that BZR1 mainly acts in the root epidermis to promote meristem growth (Chaiwanon and Wang, 2015). This view was recently challenged by a study showing that expressing fluorescently tagged BRI1-CITRINE specifically in the phloem complemented the meristem size and architecture of BR receptor triple mutants (*bri1 brl1 brl3)* (Kang et al., 2017). These opposing observations are quite puzzling, since it was previously shown that BRI1 activity in inner root meristem cell files primarily plays a role in differentiation, while its activity in the epidermis leads to cell proliferation (Vragovic et al., 2015b). However, the promoter used in this study to express BRI1 in the stele is not active in phloem poles (Kang et al., 2017), which could explain the lack of phenotype recovery. It remains to be seen what makes BR signaling in phloem poles so important that it can lead to the full recovery of mutant root meristem phenotypes.

What is the mode of action of BRs and how do they orchestrate plant growth?

Cell divisions (production) and cell expansion (elongation) are the main factors controlling plant organ growth. Cell expansion determines how much a certain organ will grow, but the number of cells that elongate in a tissue is determined by cell division rates. These two processes are tightly connected, because cell expansion can also regulate cell division rates and cells must reach the proper size before they divide (Beemster and Baskin, 1998; Jones et al., 2017). Whether the growth defects observed in BR mutants are caused by aberrant cell elongation and differentiation or perturbations in cell cycle progression remains an open question. Roots treated with BRs initially show increased growth, which later ceases and leads to reduced meristem size due to the promotion of cell elongation and exit from the meristem (Chaiwanon and Wang, 2015). The current consensus is that BR signaling orchestrates both cell division and elongation. Balanced BR signaling is required to maintain normal meristem size in roots (Gonzalez-Garcia et al., 2011), possibly through the control of cell cycle components. Recent findings challenge this view, suggesting that BRs play a dominant role in regulating cell elongation, as the small root meristem size of *bri1 brl1 brl3* mutants can be entirely explained by reduced cell elongation based on the correlation of plots of cumulative cell length along the cortex files of mutant and wild-type plants (Kang et al., 2017). Several lines of evidence support this explanation and suggest that BRs only control cell expansion and that the cell cycle perturbations in these mutants are an indirect consequence of their primary role. First, pollen grains are one of the richest sources of BRs (Fujioka and Sakurai, 1997), and BRs promote the *in vitro* germination of pollen and pollen tube growth rates (Vogler et al., 2014). Pollen tubes are single cells that do not divide but undergo extreme elongation, which means that at least in some developmental contexts, BRs promote only cell elongation. A second example is embryo development, which is tightly coupled with cell cycle progression (Jenik et al., 2005), but the phenotypic defects of BR mutants start to be visible only during later stages of embryo development after the onset of cell elongation (Jiang et al., 2013a). This lack of phenotypic deviation during early embryo development in BR mutants suggests that BRs primarily control cell elongation. Finally, the elongation of light-grown Arabidopsis hypocotyls in response to BR application occurs only via cell elongation and not cell division (Tanaka et al., 2003).

Based on the early studies, which showed that BRs positively regulate the expression of *CYCD3* (Hu et al., 2000), it was obvious that this group of hormones exerts profound effects on cell division and cycle progression in plant cells. Subsequently, it was demonstrated that BRs control root meristem size by promoting cell cycle progression (Gonzalez-Garcia et al., 2011), and several molecular players have since been identified. Maintenance of the root stem cell niche is achieved through the repression of cell divisions in the quiescent center via the TF BRAVO, which is under direct negative regulation by BES1 (Vilarrasa-Blasi et al., 2015). In addition, BRs positively regulate the expression of *ERF115*, encoding a TF triggering cell divisions and stem cell niche replenishment when surrounding cells are damaged (Heyman et al., 2013).

Another example of BRs regulating cell division is the involvement of BR signaling components in stomatal development via the regulation of asymmetric cell divisions. During this process, a protodermal cell commits to the stomatal lineage and becomes a meristemoid mother cell, which undergoes asymmetric cell division to produce meristemoid and stomatal lineage ground cells. Meristemoid cells can then undergo additional amplifying divisions or differentiate into a guard mother cell, which gives rise to a pair of guard cells (Lau and Bergmann, 2012). This process is regulated by signals perceived by several LRR RKs (Shpak et al., 2005) at the cell surface and downstream signaling events coordinated by a mitogen-activated protein kinase (MAPK) pathway (Lampard et al., 2008). MAPK module activity leads to the phosphorylation and downregulation of SPEECHLESS (SPCH), which drives asymmetric cell divisions of meristemoid mother cells. BIN2 regulates the stomatal development signaling cascade in seemingly two opposite ways: by phosphorylating and inhibiting YDA (a MAPKKK) and possibly MKK4 and MKK5 (Khan et al., 2013), leading to an increase in stomata number; and by phosphorylating and degrading SPCH, thereby decreasing the stomata number (Gudesblat et al., 2012; Kim et al., 2012). The dual role of BIN2 has been explained recently: BIN2 associates and forms complexes with BASL and POLAR proteins, which can relocate BIN2 from the nucleus, where it acts on SPCH, to the cortical BASL polarity site in the PM, where it can attenuate the MAPK signaling module (Houbaert et al., 2018).

Clearly, BRs have effects on both cell elongation and cell division. However, even after decades of research, it is difficult to determine whether the role of BRs in cell cycle modulation is just a consequence of perturbed cell elongation. It is highly likely that BRs can affect both processes and that the cellular and tissue context is crucial for determining which effect will be the prevailing one. In the future, it will be crucial to uncouple these two processes to determine the direct mode of action of BRs.

THE ROLES OF BRs IN PLANT RESPONSES TO TEMPERATURE AND DROUGHT STRESS

The structure and composition of vegetation throughout the Earth has changed dramatically since the last ice age, and a similar magnitude of change is expected in the coming century if emissions continue at a high rate (Nolan et al., 2018). Therefore, understanding how plants can better withstand changing environments represents an important challenge. Beyond their roles in growth and development, BRs also control responses to stresses such as heat, cold, and drought stress (Nolan et al., 2017a). The relationship between BRs and stress responses is complex. Although the application of BRs promotes tolerance to several stresses (Kagale et al., 2007; Bajguz and Hayat, 2009; Yuan et al., 2010; Anjum et al., 2011; Divi et al., 2016), analysis of BR deficient and insensitive mutants revealed that impairment of the BR pathway is often associated with increased survival in the face of stresses such as drought stress (Feng et al., 2015; Northey et al., 2016; Nolan et al., 2017b). In this section, we focus on responses to drought and temperature stress, which has been the topic of a number of recent studies related to the BR signaling pathway. This research affirms that multiple aspects of the BR signaling pathway interface with stress responses but suggests that the outcome depends on the particular components and tissues that are affected.

BR and ABA pathways antagonize one another

Early studies showed that BR-deficient mutants are hypersensitive to ABA (Clouse et al., 1996; Li et al., 2001). Since ABA production is induced during drought stress to promote stress tolerance (Cutler et al., 2010), these observations point towards a close relationship between BR and ABA pathways (Zhang et al., 2009b). Indeed, the molecular basis for BR-ABA antagonism has been extensively defined, ranging from interactions between downstream TFs such as BES1 and ABI3 or ABI5 (Ryu et al., 2014; Yang et al., 2016) to BIN2 kinase, a negative regulator of the BR signaling pathway that becomes activated in the presence of ABA (Wang et al., 2018a). BIN2 phosphorylates and promotes the activity of positive regulators in the ABA pathway including SnRK2 kinases (Cai et al., 2014) and TFs such as ABI5 (Hu and Yu, 2014). Given the antagonism between BR and ABA pathways and the role of ABA in promoting drought stress responses, it might be expected that BRs inhibit drought stress responses. This appears to be the case for the BR signaling pathway operating through BRI1 to downstream BES1 and BZR1 TFs (Chen et al., 2017; Nolan et al., 2017b; Ye et al., 2017). However, overexpression of the vascular BR receptor BRL3 promotes drought responses without penalizing growth (Fabregas et al., 2018).

Several mechanisms converge on BES1 to balance BR-regulated growth and stress responses

Depending on the duration and severity of drought stress, plants must carefully coordinate growth and stress responses. When water limitation is mild, inhibited growth would cause a yield and fitness penalty. On the other hand, when drought is severe, plants cease growth to ensure survival (Claeys and Inze, 2013), although it is not clear if growth limitation represents an energy trade-off or is simply co-regulated with stress responses. Hence, understanding the molecular mechanisms that coordinate growth and stress responses is crucial for engineering crops with optimized stress responses. BES1 is emerging at the nexus coordinating BR-mediated growth and stress responses. Several mechanisms that inhibit BES1 activity during stress have been described. First, as discussed above, BES1 is degraded through DSK2-mediated selective autophagy during drought stress to inhibit BR-regulated growth. Mutants with impaired BES1 degradation such as *dsk2* loss-of-function mutants have decreased survival during drought, which can be restored by inhibiting *BES1* expression using *BES1 RNAi* (Nolan et al., 2017b). These findings indicate that BES1 degradation is an important mechanism to limit BR-regulated growth during drought stress to promote plant survival.

In addition to modulating the protein abundance of BES1, drought also affects its activity through interactions with RESPONSIVE TO DESICCATION26 (RD26), a drought-inducible TF. BES1 is activated by BRs to promote growth and inhibit the expression of *RD26.* By contrast, RD26 is both transcriptionally induced during drought and activated by an ABA-ABI1-BIN2 signaling module, in which BIN2 activity is promoted by ABA during stress to phosphorylate and stabilize RD26 protein (Jiang et al., 2019). Subsequently, RD26 inhibits BES1 and promotes drought responses. At a mechanistic level, this occurs through protein-protein interactions between BES1 and RD26. These two TFs interact, likely forming heterodimers that synergistically bind to G-Box (CACGTG) promoter elements, but BES1 and RD26 have opposite activities. For example, BES1 inhibits many drought-induced genes whereas RD26 activates these genes (Ye et al., 2017). Therefore, the inhibition of BES1 activity through interaction with RD26 on a common promoter element is another means by which BES1 is inhibited when stress is encountered.

TINY, an APETALA2/ETHYLENE RESPONSIVE FACTOR TF, has also been implicated in inhibiting growth and promoting drought responses. *TINY* is induced during drought stress and antagonizes BES1 to inhibit BR-regulated growth and promote drought-responsive gene expression; however, TINY does so by binding to a different promoter element, the Drought responsive element (DRE) (Xie et al., 2019). TINY is also a substrate of BIN2 kinase; the phosphorylation of TINY by BIN2 promotes its stability. Since BRs inhibit BIN2 activity under optimal growth conditions, this provides a mechanism to restrain stress responses mediated by TFs such as TINY and RD26 while still allowing them to become rapidly activated by stress via BIN2-mediated phosphorylation and stabilization.

Finally, the degradation of BES1-interacting TFs such as WRKY46, WRKY54, and WRKY70 (WRKY46/54/70) during drought represents another mechanism that inhibits BR-regulated growth. *WRKY46/54/70* are direct targets of BES1 and function as positive regulators of BR biosynthesis and signaling (Chen et al., 2017). The *wrky54 wrky46 wrky70* triple mutant *(wrky54t)* exhibits dwarf phenotypes, which is consistent with the notion that these factors are required for BR-regulated growth. *wrky54t* plants are more tolerant to drought stress than wild-type plants, as they exhibit the constitutive activation of thousands of drought-regulated genes. Therefore, WRKY46/54/70 cooperate with BES1 to inhibit drought-responsive gene expression. Similar to BES1, WRKY46/54/70 are phosphorylated and destabilized by BIN2 kinase, and WRKY54 protein levels decrease during drought stress (Chen et al., 2017). These findings indicate that the degradation of growth-promoting TFs during drought stress extends beyond BES1. However, the role of BIN2 in WRKY54 degradation and the E3 ubiquitin ligase(s) and downstream pathways involved in this process remain to be further characterized.

In summary, the modulation of BES1 represents an important point of crosstalk between BR and drought-stress responses. BES1 is inhibited through targeted protein degradation and transcriptional inactivation during drought stress, whereas BES1 inhibits drought-stress responses under growth-promoting conditions. Although these studies provide mechanistic insight into the coordination of plant growth and stress responses, they did not examine specific tissues or developmental contexts, which present potential opportunities for designing ways to overcome growth-stress tradeoffs.

Tissue-specific modulation of the BR pathway allows for increased growth and drought tolerance

Many efforts to increase plant drought survival also negatively affect growth. A number of approaches have been taken to attempt to circumvent this phenomenon. For example, stress-inducible expression of factors that promote drought tolerance has been explored (Reguera et al., 2013), but this method requires extensive knowledge of stress-inducible promoters, which can be challenging for generalization to other species. Alternatively, gene-stacking approaches have proven fruitful in overcoming growth inhibition using positive regulators of drought responses such as DEHYDRATION-RESPONSIVE ELEMENT-BINDING PROTEIN1A (DREB1A). Overexpression of *DREB1A* led to increased drought tolerance and growth inhibition, but the growth limitation could be overcome by simultaneous overexpression of *DREB1A* together with the rice homolog of *PIF4* (*OsPIL1*) in Arabidopsis (Kudo et al., 2016). Along these lines, an elegant study demonstrated that overexpressing the BR receptor gene *BRL3* increased plant survival during drought without the growth penalty observed in BR mutants such as the loss-of-function mutant *bri1* (Fabregas et al., 2018). In this case, *BRL3* expression was driven by a constitutive promoter, but BRL3 protein primarily accumulates in the vascular tissue of roots, where it promotes the accumulation of osmoprotective metabolites and stress-responsive gene expression (Fabregas et al., 2018). These findings demonstrate that understanding the spatiotemporal complexity of BR signaling holds great promise for engineering stress-resistant crops. Although BES1 appears to inhibit drought responses in Arabidopsis, BES1 and BRs promote the expression of a subset of drought-responsive genes (Ye et al., 2017). Therefore, further dissection of factors involved in the BR transcriptional network might also allow TFs that positively influence both growth and stress responses to be identified.

TaBZR2, a BES1 and BZR1 homolog in wheat (*Triticum aestivum*), promotes drought responses by inducing the expression of wheat *glutathione S-transferase 1* (*TaGST1*), which is involved in superoxide scavenging (Cui et al., 2019). Likewise, studies in tomato (*Solanum lycopersicum*) revealed that BRs promote drought tolerance, whereas overexpression of the BR receptor gene *SlBRI1* had an opposite effect (Nie et al., 2019). Therefore, the regulation of drought stress by BRs operates at different levels, including stress-responsive gene expression, modulation of ABA levels, H_2O_2 production, and the production of antioxidants and osmoprotectant compounds (Planas-Riverola et al., 2019). The cumulative effects of these changes on plant survival and growth appear to depend on the species, method of manipulation, and spatiotemporal context.

BRs regulate heat and cold stress responses

BRs regulate growth and stress responses under both increased and decreased temperatures. Under increased temperatures, BES1 and BZR1 accumulate and function along with PIF4 to promote thermogenic growth (Ibañez et al., 2018; Martinez et al., 2018). Increased BES1 and BZR1 levels promote the expression of *PIF4,* and increased PIF4 levels allow for the derepression of BR biosynthesis by switching BES1 from a repressive homodimer to a PIF4-BES1 heterodimer that activates transcription (Martinez et al., 2018). In a seemingly opposite manner, increased temperatures decrease BRI1 levels, which tempers BR signaling and increases root growth (Martins et al., 2017). BRI1 undergoes ubiquitination, endocytosis, and degradation (Martins et al., 2015; Zhou et al., 2018), which are required for the heat-induced decrease in BRI1 accumulation (Martins et al., 2017). While PUB12 and PUB13 ubiquitinate BRI1 following BR perception (Zhou et al., 2018), the E3 ubiquitin ligase responsible for this ubiquitination during heat stress remains to be identified.

BR signaling also regulates plant tolerance to cold stress. One aspect of this regulation involves the BR-mediated promotion of cold tolerance through the accumulation of the active unphosphorylated forms of BZR1 and BES1, promoting the expression of *C-REPEAT/DEHYDRATION-RESPONSIVE ELEMENT BINDING FACTOR1* (*CBF1*) and *CBF2*, which positively regulate cold-stress responses (Li et al., 2017). CESTA, a positive regulator of BR signaling (Poppenberger et al., 2011), also promotes cold-stress responses. CESTA is dephosphorylated and SUMOylated in response to BRs, which leads to the CESTA-mediated activation of *COLD-RESPONSIVE (COR)* genes through both *CBF*-dependent and independent pathways. This in turn promotes basal and acquired freezing tolerance (Eremina et al., 2016). BIN2 also plays a role in BR-mediated regulation of cold-stress responses by phosphorylating INDUCER OF CBF EXPRESSION1 (ICE1) during prolonged exposure to cold, promoting its degradation to attenuate *CBF* induction (Ye et al., 2019). In summary, BRs can either promote or inhibit several stress responses. Future efforts should focus on untangling the roles of the specific BR signaling components in stress responses and understanding their spatiotemporal regulation.

ROLES OF BR HORMONES IN CROPS

BRs play pivotal roles in plant growth, development, and responses to adverse conditions, making them major targets for manipulation to improve agronomic traits. In this section, we provide an update on unique aspects of BR signaling outside the model plant Arabidopsis and report on promising aspects and challenges when manipulating BRs to improve crops.

Unique factors for BR signaling in crops

It is apparent that BR biosynthetic and signaling pathways are conserved among species. For example, *DWARF4* (*DWF4*), which encodes the rate-limiting 22α hydroxylase in the BR biosynthetic pathway in Arabidopsis, has homologs with similar functions in rice (Sakamoto et al., 2006) and maize (*Zea mays*) (Liu et al., 2007; Makarevitch et al., 2012). Likewise, BRI1 homologs have been identified in rice (Yamamuro et al., 2000), maize (Kir et al., 2015), and tomato (Holton et al., 2007) that likely serve as BR receptors based on mutant phenotypes and BL binding activity (Holton et al., 2007).

At the same time, unique factors that contribute to BR signaling have been described in cereals. Among these are factors regulating GSK protein levels, such as *qGL3*, encoding a putative protein phosphatase with a Kelch-like repeat domain in rice (OsPPKL1) that is an ortholog of Arabidopsis BSU1 (Zhang et al., 2012). Unlike the dephosphorylation activity of BSU1 on BIN2, which leads to BIN2 degradation, OsPPKL1 dephosphorylates OsGSK3 and stabilizes it (Gao et al., 2019). Other factors include *QTL for GRAIN WIDTH AND WEIGHT ON CHROMOSOME5* (*GW5*), encoding a calmodulin binding protein that inhibits auto-phosphorylation of GSK2 and trans-phosphorylation of OsBZR1 and DWARF AND LOW-TILLERING (DLT) by GSK2 to enhance BR signaling in rice (Liu et al., 2017). In addition to BZR1 homologs in rice, other TFs involved in BR signaling in rice are directly regulated by GSK2, such as GROWTH-REGULATING FACTOR4 (OsGRF4) (Che et al., 2015) and DLT (Tong et al., 2012). Another example is rice KNOTTED1-LIKE HOMEOBOX (KNOX) TRANSCRIPTION FACTOR HOMEOBOX1 (OSH1), which represses BR biosynthesis by activating BR catabolism genes (Tsuda et al., 2014). DLT, OSH1, and OVATE family protein 19 (OsOFP19) interact with each other, and OsOFP19 promotes the activity of OSH1 while suppressing the function of DLT, indicating that OsOFP19 is a negative regulator of BR signaling (Yang et al., 2018a).

Epigenetic modifications are also implicated in the regulation of BR signaling in crops. *Epi-d1*, an epigenetic allele of *DWARF1 (encoding* G protein alpha subunit in rice RGA1) contains DNA methylation in the promoter of this gene, causing a dwarf phenotype (Ashikari et al., 1999) (Miura et al., 2009). SDG725-mediated H3K36 methylation is a positive regulator of BR signaling, as it is required for the expression of BR-related genes in rice (Sui et al., 2012). In addition, the loss of DNA methylation in the promoter of *RELATED TO ABSCISIC ACID INSENSITIVE3* (*ABI3*)/*VIVIPAROUS1* (*VP1*)*6* (*RAV6*) in the *Epi-rav6* rice mutant leads to the ectopic expression of *OsBRI1* and the BR biosynthetic genes *DWARF2* (*D2*) (Hong et al., 2003), *DWARF11*, and *BR-DEFICIENT DWARF1* (Zhang et al., 2015).

Manipulation of BRs in crops

Leaf angle, plant height, and inflorescence architecture are three key determinants of yield that are potentially regulated by BRs (Yamamuro et al., 2000; Hong et al., 2003; Sakamoto et al., 2006; Yang et al., 2018b). BRs play a unique role in controlling leaf erectness by inhibiting the division of abaxial sclerenchyma cells; these cells provide mechanical support for the lamina joints in *Oryza sativa* when clustered (Sun et al., 2015). Leaf angle could be adjusted by altering the expression of BR biosynthesis genes, but in most cases, this is accompanied by a severe dwarf phenotype, as observed for rice plants harboring a deletion of the *D2* gene (Li et al., 2013). The *osdwaf4* knockout mutant (Sakamoto et al., 2006) and *OsBU1 RNAi* transgenic plants (Tanaka et al., 2009) showed a more erect leaf angle phenotype without affecting plant height or fertility and produced higher grain yields under dense planting compared to the wild type (Sakamoto et al., 2006; Tanaka et al., 2009), suggesting that it is possible to manipulate the BR pathway in an agriculturally relevant manner. BRs also regulate inflorescence architecture. In green foxtail (*Setaria viridis*), a mutation in *CYP724B1*, encoding an inflorescence-expressed CYP enzyme involved in BR biosynthesis, resulted in homeotic conversion of bristles to spikelets and the emergence of two florets per spikelet (Yang et al., 2018b).

Secondary cell walls in the form of wood and fibers are the most abundant, renewable plant products, and there is a high demand for improving wood and fiber production. Overexpression of *Populus trichocarpa CYP85A3* (*PtCYP85A3*), encoding a P450 monooxygenase that catalyzes the conversion of castasterone to BL, enhanced xylem formation and wood production in poplar while the composition of cellulose and lignin and cell wall thickness were not affected, making *PtCYP85A3* a good target for engineering fast-growing woods (Jin et al., 2017). BR signaling also regulates cotton (*Gossypium hirsutum*) fiber development by modifying the expression of *DET2* and *PAGPDA1* (*PAG1*). *PAG1* encodes a homolog of the Arabidopsis *PHYB ACTIVATION-TAGGED SUPPRESSOR1* (*BAS1*), which inactivates BRs via C-26 hydroxylation (Luo et al., 2007; Yang et al., 2014).

BRs are involved in sex determination in maize, as *nana plant1* (*na1*), which carries a loss-of-function mutation in a *DET2* homolog, contains feminized male flowers with a tassel-seed phenotype (Hartwig et al., 2011). However, RNAi knockdown of maize *BRI1* and its homologs led to plants with a strong dwarf phenotype but lacking a sex-determination phenotype (Kir et al., 2015). As such, it remains to be determined if this function of BRs is conferred by tissue- or developmental stage-specific signaling components. BR-promoted pollen and seed development in rice is achieved by stimulating the expression of *CARBON STARVED ANTHER* (*CSA*), encoding a R2R3-type MYB TF, which further triggers the expression of sugar partitioning and metabolic genes through OsBZR1 (Zhu et al., 2015).

BRs are also important for nutrient accumulation. In grapevine (*Vitis vinifera*), sugar allocation is regulated by BRs to increase soluble sugar contents in berries, which is achieved by promoting the activities of both invertases and sucrose synthase and by up-regulating the expression of genes encoding invertase and mono- and di-saccharide transporters (Xu et al., 2015).

Crops are grown under ever-changing environmental conditions in the field, and inevitably, they face adverse conditions. At least in some cases, BR application helps alleviate stress in plants. For example, BR treatment increases tolerance to rice blast and bacterial blight diseases in rice (Nakashita et al., 2003), to cadmium in tomato (Hayat et al., 2010; Hasan et al., 2011), and to cold-induced damage in cucumber (*Cucumis sativus*) (Jiang et al., 2013b). Therefore, understanding the mechanisms by which BRs regulate these stress responses in crops represents an important direction for the future.

Tissue-specific modification of gene expression, a way to overcome undesirable effects of BRs

Manipulating the expression of BR-regulated genes by mutation or overexpression often causes pleiotropic phenotypes, some of which might be undesirable for crop breeding and planting, such as changes of leaf erectness (Sakamoto et al., 2006; Tanaka et al., 2009; Makarevitch et al., 2012; Li et al., 2013; Mantilla Perez et al., 2014), plant height (Yamamuro et al., 2000; Li et al., 2013; Hirano et al., 2017), inflorescence architecture (Liu et al., 2007; Makarevitch et al., 2012;

Li et al., 2013; Yang et al., 2018b), or biomass (Morinaka et al., 2006; Sakamoto et al., 2006; Tanaka et al., 2009). Tissue-specific promoters, such as vascular tissue-specific *S-ADENOSYLMETHIONINE SYNTHASE* (Wu et al., 2008) and seed-specific glutelin *Gt1* promoters (Li et al., 2018), have been employed to drive the overexpression of BR biosynthetic genes, such as genes encoding sterol C-22 hydroxylases (which control the conversion of campestanol to 6-deoxocathasterone) (Wu et al., 2008) and *OsDWF4* in rice to increase seed yield without impairing other traits. A CRISPR-based tissue-specific knockout system can be designed and used to generate mutations in particular cell types and tissues (Decaestecker et al., 2019). Applying both of these tissue-specific gene manipulation systems to fine-tune BR signaling as needed would facilitate the generation of improved crops.

Due to the importance of BRs for plant development, nutrient accumulation, and resistance to stress conditions, BR-related genes may be identified whose expression could be manipulated to simultaneously increase plant productivity and performance under adverse conditions. These genes might be manipulated by overexpression or knock-down in crops of interest. Alternatively, exogenous application of BRs holds promise for helping crops overcome certain stresses, although this approach is hindered by the high cost of BR synthesis. Systems and synthetic biology approaches such as introducing the BR biosynthetic pathway into microbes might help address this issue and allow for more economical production of BRs. Altogether, manipulation of the BR pathway for crop improvement holds great promise but requires further knowledge of how BR signaling operates in different crops, environments, and developmental contexts.

CONCLUSIONS AND PERSPECTIVES

Research in the last several decades has made the BR pathway arguably one of the best-studied signaling pathways in plants. Genetic approaches such as mutant screens demonstrated the importance of BRs in plant growth and uncovered many major players in BR biosynthesis and signaling (Clouse, 2015). Subsequently, molecular, biochemical, structural, and genomic approaches have increased our understanding of the BR signaling cascade, from PM-localized receptor kinases to the transcriptional networks controlled by BES1 and BZR1 (Kim and Wang, 2010; Clouse, 2011; Guo et al., 2013; Dejonghe et al., 2014; Nolan et al., 2017a). It is becoming increasingly evident that the BR pathway does not represent a linear signaling pathway that operates in isolation, but rather that BRs undergo crosstalk with multiple other hormones and stress responses (Nolan et al., 2017a). Moreover, BR signaling varies among different cells and tissues, which can be manipulated to improve plant growth and stress responses (Fabregas et al., 2018).

Despite the progress, many questions in the BR field remain to be addressed. We still do not fully understand how BRs control a large number of genes, when this regulation occurs, and in which cells these genes are activated or repressed to control BR-regulated growth, development, and responses to environmental cues. Technological advances such as single cell genomics (Shahan, 2019) and improved proteomic technologies (Song et al., 2018a) coupled with computational modeling will be instrumental in addressing these questions.

Furthermore, much remains to be learned about BR biosynthesis and potential modes of transport (Vukašinović and Russinova, 2018). Future studies should aim to obtain evidence that BRs are transported out of cells and to identify BR transporters. Finally, we need to better understand how BRs contribute mechanistically to the overall growth program of plants in particular environments such as shade, high temperature, and drought. As a long-term overarching objective, researchers should work to manipulate the BR pathway in crops and other plants so that plants can accommodate the stress created by greater fluctuations in these environmental parameters.

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FIGURE LEGENDS

Figure 1: Overview of the BR signaling pathway.

When BRs are absent (left), plasma membrane-localized receptors BRI1 and BAK1 are inhibited by several factors, including BKI1 and BIR3. Additionally, BIN2 kinase functions as a negative regulator and phosphorylates BES1 and BZR1 TFs to inhibit their activity through multiple mechanisms. BSS1 forms a complex with BES1 and BZR1 in the cytoplasm, and THXh5 reduces BZR1 in the nucleus, further inactivating these TFs. This leads to relatively low expression of BR-induced genes and higher expression of BR-repressed genes. When BRs such as brassinolide (BL) are present, they bind to the receptor BRI1 and co-receptor BAK1 to initiate BR signaling (right). BKI1 and BIR3 dissociate from the receptor complex, allowing BRI1 and

BAK1 to become phosphorylated and activated. BSKs/CDGs are phosphorylated and activate BSU1 phosphatase to inhibit BIN2. Dephosphorylation by PP2A allows BES1 and BZR1 to function with other TFs and co-factors to promote BR-induced gene expression and inhibit BR-repressed gene expression. Figure created with BioRender.com. BAK1, BRI1-ASSOCIATED KINASE1; BES1, BRI1-EMS-SUPPRESSOR 1; BIN2, BRASSINOSTEROID-INSENSITIVE 2; BIR3, BAK1-INTERACTING RECEPTOR-LIKE KINASES3; BKI1, BRI1 KINASE INHIBITOR1; BL, Brassinolide; BR, Brassinosteroid; BRI1, BRASSINOSTEROID INSENSITIVE 1; BRRE, BR Response Element; BSK, BR SIGNALING KINASES; BSS1, BRZ-SENSITIVE-SHORT HYPOCOTYL1; BSU, BRI1 SUPPRESSOR 1; BZR1, BRASSINAZOLE-RESISTANT 1; CDG, CONSTITUTIVE DIFFERENTIAL GROWTH; H_2O_2, Hydrogen Peroxide; HDA19, HISTONE DEACETYLASE19; IWS1, INTERACTING-WITH-SPT6-1; KIB1, KINK SUPPRESSED IN *BZR1-1D*; P, indicates phosphorylation; PP2A, PROTEIN PHOSPHATASE2A; PUB12/13, PLANT U-BOX12/13; REF6, RELATIVE OF EARLY FLOWER6; SDG8, SET DOMAIN GROUP 8; SH, indicates reduced Cys residue; SOH, indicates oxidized Cys residue; TF, transcription factor; TPL, TOPLESS; TRXh5, THIOREDOXIN H-TYPE 5; Ub, indicates ubiquitination.

Figure 2: Mechanisms regulating BIN2 activity

In addition to canonical dephosphorylation and inactivation of BIN2 by BSU1 in the presence of BRs, several other mechanisms also regulate BIN2 activity. BIN2 is ubiquitinated by the E3 ubiquitin ligase KIB1 and degraded by the proteasome in the presence of BRs. Deacetylation by HDA6 inhibits BIN2 activity, whereas oxidation by reactive oxygen species (ROS) promotes BIN2 activity. ABA also activates BIN2 through the inhibition of ABI1/2 phosphatases that dephosphorylate BIN2. Finally, BIN2 is sequestered in a cell-type specific manner by OPS in the phloem or POLAR and BASL in the stomatal cell lineage. Figure created with BioRender.com. ABA, Abscisic acid; ABI1/2, ABA INSENSITIVE1/2; Ac, indicates acetylation; BASL, BREAKING OF ASYMMETRY IN THE STOMATAL LINEAGE; BIN2, BRASSINOSTEROID-INSENSITIVE2; BR, Brassinosteroid; BSU, BRI1 SUPPRESSOR1; HDA6, HISTONE DEACETYLASE6; KIB1, KINK SUPPRESSED IN *BZR1-1D*; OPS, OCTOPUS; P, indicates phosphorylation; PM, Plasma membrane; POLAR, POLAR

LOCALIZATION DURING ASYMMETRIC DIVISION AND REDISTRIBUTION; ROS, Reactive oxygen species; SOH, indicates oxidized Cys residue; Ub, indicates ubiquitination.

Figure 3: Diverse regulatory mechanisms controlling BES1 and BZR1 activity

BES1 and BZR1 activity is modulated by multiple modes of regulation. *BES1* transcripts are subject to alternative splicing, with a longer BES1-L isoform displaying increased nuclear localization. Phosphorylation by BIN2 inactivates BES1 and BZR1, whereas MPK6 phosphorylation of BES1 in response to bacterial pathogens or Pathogen-associated molecular pattern (PAMPs) leads to its activation. PP2A dephosphorylates and activates BES1 and BZR1 in the presence of BRs. The production of hydrogen peroxide (H_2O_2) is promoted by BRs and activates BES1 and BZR1 via oxidation, whereas TRXh5 reduces BZR1. BES1 and BZR1 can be inactivated by cytoplasmic sequestration, photoreceptors that respond to UV, red and blue light, or ubiquitination. Several families of E3 ubiquitin ligases target BZR1 or BES1 in different tissues or in response to environmental cues, leading to their degradation by the proteasome or autophagy. DSK2 mediates selective autophagy for BES1 degradation during stress. Figure created with BioRender. ATG8, AUTOPHAGY-RELATED PROTEIN8; BES1, BRI1-EMS-SUPPRESSOR1; BIN2, BRASSINOSTEROID-INSENSITIVE 2; BR, Brassinosteroid; BSS1, BRZ-SENSITIVE-SHORT HYPOCOTYL1; BZR1, BRASSINAZOLE-RESISTANT1; COP1, CONSTITUTIVE PHOTOMORPHOGENIC1; CRY1, CRYPTOCHROME1; DSK2, DOMINANT SUPPRESSOR OF KAR2; H_2O_2, Hydrogen Peroxide; MAX2, MORE AXILLARY GROWTH LOCUS2; MPK6, MITOGEN-ACTIVATED PROTEIN KINASE6; P, indicates phosphorylation; PHYB, PHYB, PHYTOCHROME B; PP2A, PROTEIN PHOSPHATASE2A; PUB40, PLANT U-BOX40; SH, indicates reduced Cys residue; SINATs, SINA of *Arabidopsis thaliana*; SOH, indicates oxidized Cys residue; TRXh5, THIOREDOXIN H-TYPE 5; Ub, indicates ubiquitination; UVR8, UVB-RESISTANCE8.

Figure 4: Summary of BR regulated developmental processes in Arabidopsis.

Temperature and light modulate PHYB activity, regulate the concentration of PIF4, and indirectly determine the levels of PIF4-BES1 heterodimerization. The interaction of these TFs determines their gene targets and leads to different cellular responses. Xylem differentiation is governed by the TDIF signaling pathway. GSK3s are crucial components in this pathway, which act as negative regulators of xylem differentiation and enable cross-talk with the BR signaling pathway. Stomatal development is fine-tuned by the dual role of BIN2 and is dependent on its subcellular localization. When located in the nucleus, BIN2 mainly acts as a negative regulator of SPCH activity, whereas in complex with BASL and POLAR, it re-localizes to the PM polarized region of MMC and acts as a negative regulator of YDA and MKKs, leading to SPCH activation. BRs inhibit flowering by promoting the expression of flowering inhibitor FLC. Additionally, the expression BR biosynthetic genes exhibits diurnal changes. During the root epidermal cell fate determination process, BIN2 phosphorylates EGL3, leading to its trafficking from the nucleus to cytosol in trichoblast cells, which facilitates its movement from trichoblast to atrichoblast cells. BIN2 can also phosphorylate TTG1 to inhibit the activity of the WER-GL3/EGL3-TTG1 transcriptional complex. In the root apical meristem, BRs control the size of the stem cell niche by balancing the expression of BRAVO, which negatively regulates cell divisions in the quiescent center. BR signaling levels increase along the longitudinal axis, with higher levels present in cells closer to the differentiation/elongation zone. Arrows indicate activation and blunt-ended lines indicate inhibition. BES1, BRI1-EMS-SUPPRESSOR 1; BIN2, BRASSINOSTEROID-INSENSITIVE2; BR, Brassinosteroid; BRAVO, BRASSINOSTEROIDS AT VASCULAR AND ORGANIZING CENTER; BRI1, BRASSINOSTEROID INSENSITIVE1; BSU, BRI1 SUPPRESSOR1; BZR1, BRASSINAZOLE-RESISTANT1; EGL3, ENHANCER OF GLABRA3; EPF1/2, EPIDERMAL PATTERNING FACTOR 1/2; ERF115, ETHYLENE RESPONSE FACTOR 115; FLC, FLOWERING LOCUS C; GL2, GLABRA2; GSK3, GLYCOGEN SYNTHASE KINASE3; MKK4/5/7/9, MITOGEN-ACTIVATED PROTEIN KINASE KINASE 4/5/7/9; MMC, Meristemoid mother cell; MPK3/6, MITOGEN-ACTIVATED PROTEIN KINASE3; PHYB, PHYTOCHROME B; PIF4, PHYTOCHROME INTERACTING FACTOR4; QC, Quiescent center; SPCH, SPEECHLESS; TDIF, TRACHEARY ELEMENT DIFFERENTIATION INHIBITORY FACTOR; TDR, TDIF RECEPTOR; TTG1, TRANSPARENT TESTA GLABRA1; WER, WEREWOLF; WOX4, WUSCHEL RELATED HOMEOBOX 4; YDA, YODA.

Table 1: Important genes in BR signaling

Functional classification	Gene name	AGI	Function	References
BR perception	BRI1	At4g39400	BR receptors	He et al., 2000; Cano-Delgado et al., 2004
	BRL1	At1g55610		
	BRL3	At3g13380		
	SERK3/BAK1	At4g33430	Serves as a co-receptor of BRI1 along with homologs SERK1, SERK2, and SERK4	Nam and Li, 2002; Gou et al., 2012
	BKI1	At5g42750	BRI1 kinase inhibitor, inhibits BRI1/BAK1 interaction	Wang and Chory, 2006
	BIR3	At1g27190	Inhibits BRI1/BAK1 interaction	Hohmann et al., 2018a
	PUB12	At2g28830	Ubiquitinates BRI1 following BR perception	Zhou et al., 2018
	PUB13	At3g46510		
Phosphorylation and dephosphorylation cascade	BSK1	At4g35230	Together with their homologous proteins, phosphorylate and activate BSU1; BSK3 acts as a scaffolding protein to regulate BR signaling	Tang et al., 2008; Kim et al., 2011; Ren et al., 2019
	BSK3	At4g00710		
	CDG1	At3g26940		
	BSU1	At1g03445	Dephosphorylates and inactivates BIN2	Kim et al., 2009
	BIN2	At4g18710	Together with other GSK family members, phosphorylates and inactivates BES1 and BZR1	Li and Nam, 2002; Kim et al., 2009
	PP2A	At1g69960	Dephosphorylates and activates BES1 and BZR1	Tang et al., 2011
	BES1	At1g19350	Control BR-regulated gene expression along with homologs BEH1-4	Wang et al., 2002; Yin et al., 2002; Yin et al., 2005; He et al., 2005
	BZR1	At1g75080		
BIN2 interactors that modulate BIN2 activity	KIB1	At4g12810	Mediates BIN2 ubiquitination and subsequent degradation	Zhu et al., 2017
	OCTOPUS	At3g09070	Confines BIN2 to the PM, blocking its interaction with BES1/BZR1	Anne et al., 2015
	POLAR	At4g31805	Regulate the nuclear versus cytosolic and PM localization of BIN2	Houbaert et al., 2018
	BASL	At5g60880		
	HDA6	At5g63110	Deacetylates BIN2 and represses BIN2 kinase activity	Hao et al., 2016
	TTL1	At1g53300	Together with its homologs TTL3/4, act to scaffold BR signaling components at the PM	Amorim-Silva et al., 2019
Modulators of BES1/BZR1 degradation and activation	MAX2	At2g42620	Mediate BES1/BZR1 ubiquitination and degradation	Wang et al., 2013; Kim et al., 2014; Yang et al., 2017; Kim et al., 2019
	PUB40	At5g40140		
	COP1	At2g32950		
	SINAT2	At3g58040		
	DSK2	At2g17200	Autophagy receptor for BES1 degradation	Nolan et al., 2017b
	14-3-3λ	At5g10450	Together with other 14-3-3 proteins, retains phosphorylated BES1 and BZR1 in the cytoplasm	Gampala et al., 2007; Ryu et al., 2007
	TRXh5	At1g45145	Interacts with BZR1 to promote its reduction and inactivation	Tian et al., 2018
	RGA1	At2g01570	Together with other DELLA proteins, inhibits BES1, BZR1, PIF4, and ARF6 under low GA conditions	Bai et al., 2012a; Gallego-Bartolome et al., 2012
	BSS1/BOP1	At3g57130	Sequesters BES1 and BZR1 in the cytoplasm in the absence of BRs	Shimada et al., 2015
	BOP2	At2g41370		
	UVR8	At5g63860	UV light receptor, inhibits DNA binding activity of BES1	Liang et al., 2018
	CRY1	At4g08920	Interact with BES1, BZR1, and BIM1 in response to blue light to inhibit their activity	Wang et al., 2018b; He et al., 2019
	CRY2	At1g04400		
	PHYB	At2g18790	Inhibits the transcriptional activity of BES1 in response to red light	Wu et al., 2018
Transcriptional regulators involved in BR-mediated gene expression	IWS1	At1g32130	Interacts with BES1 to promote BR-regulated gene expression	Li et al., 2010
	BIM1	At5g08130	Together with its homologs BIM2 and BIM3, interacts with BES1 to activate the expression of BR-induced genes	Yin et al., 2005
	MYB30	At3g28910	Cooperates with BES1 to promote BR-induced gene expression	Li et al., 2009

	PIF4	**At2g43010**	**Interacts with BES1 and BZR1 to regulates BR-induced gene expression**	**Oh et al 2012a; Martinez et al., 2018**
	ARF6 **ARF8**	**At1g30330** **At5g37020**	**Interacts with both PIFs and BZR1 to regulate gene expression**	**Oh et al., 2014b**
	MYBL2 **HAT1**	**At1g71030** **At3g54610**	**BES1/BZR1 target transcription factors, assist BES1 in BR-repressed gene expression**	**Ye et al., 2012; Zhang et al., 2014b**
	HDA19	**At4g38130**	**Mediates histone deacetylation for BES1 and BZR1-repressed genes**	**Oh et al., 2014a; Ryu et al., 2014**
	TPL	**At1g15750**	**Interacts with BES1/BZR1 and recruits HDA19**	**Oh et al., 2014a; Ryu et al., 2014**
	ELF6 **REF6**	**At5g04240** **At3g48430**	**Remove repressive H3K27me2/H3K27me3 marks, allowing BES1 to activate gene expression**	**Yu et al., 2008; Lu et al., 2011**
	PICKLE	**At2g25170**	**Represses H3K27me3 marks for BR-induced genes**	**Zhang et al., 2014a**
	SDG8	**At1g77300**	**Increases H3K36me2/3 levels for BR-induced gene expression**	**Wang et al., 2014b**
	WRKY46 **WRKY54** **WRKY70**	**At2g46400** **At2g40750** **At3g56400**	**Cooperate with BES1 to inhibit drought-responsive gene expression**	**Chen et al., 2017**
	RD26	**At4g27410**	**Inhibits BES1 and promotes drought responses**	**Jiang et al., 2019**
	TINY	**At5g25810**	**Together with TINY2/3, regulates drought responses through an antagonistic interaction with BES1**	**Xie et al., 2019**

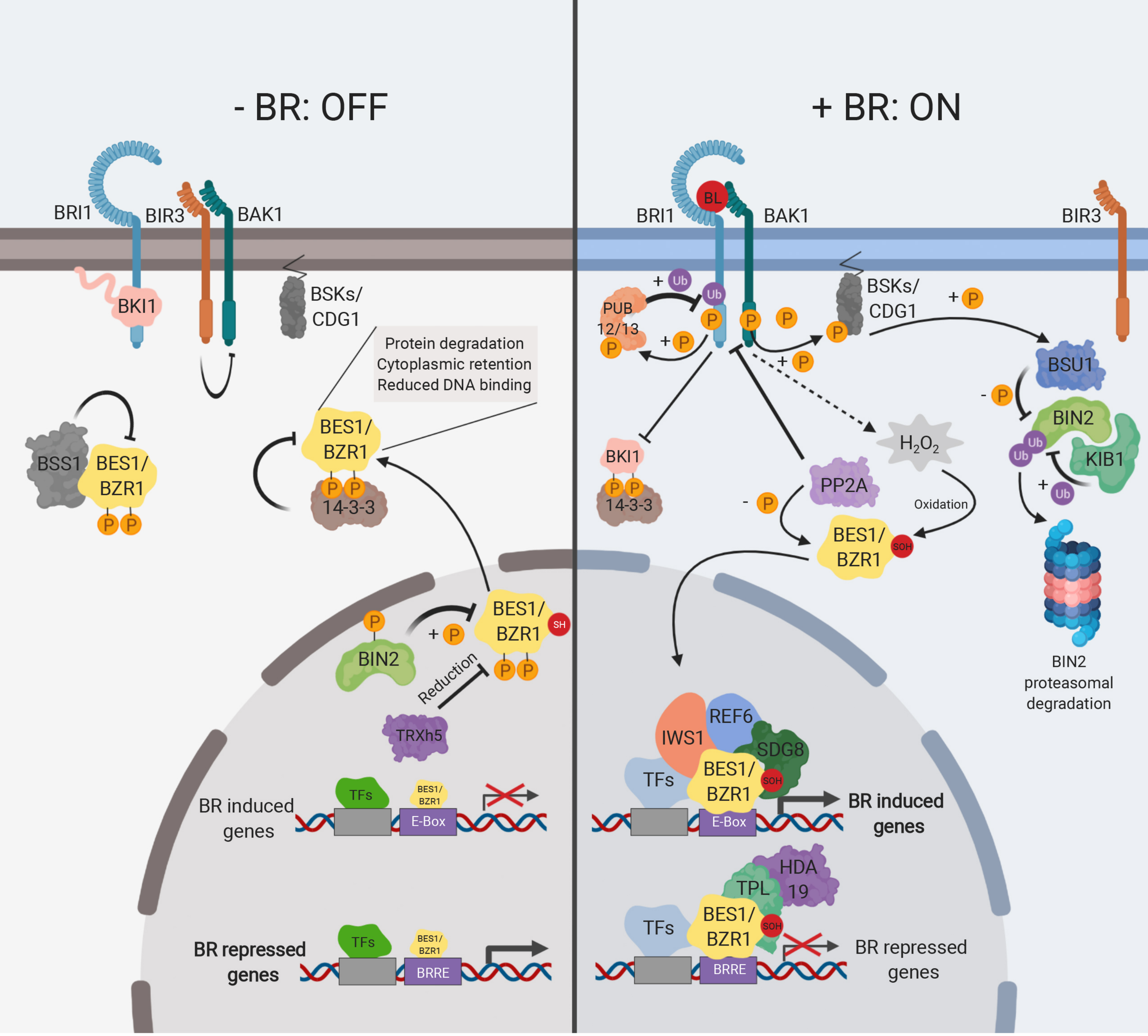

Figure 1: Overview of the BR signaling pathway.
When BRs are absent (left), plasma membrane-localized receptors BRI1 and BAK1 are inhibited by several factors, including BKI1 and BIR3. Additionally, BIN2 kinase functions as a negative regulator and phosphorylates BES1 and BZR1 TFs to inhibit their activity through multiple mechanisms. BSS1 forms a complex with BES1 and BZR1 in the cytoplasm, and THXh5 reduces BZR1 in the nucleus, further inactivating these TFs. This leads to relatively low expression of BR-induced genes and higher expression of BR-repressed genes. When BRs such as brassinolide (BL) are present, they bind to the receptor BRI1 and co-receptor BAK1 to initiate BR signaling (right). BKI1 and BIR3 dissociate from the receptor complex, allowing BRI1 and BAK1 to become phosphorylated and activated. BSKs/CDGs are phosphorylated and activate BSU1 phosphatase to inhibit BIN2. Dephosphorylation by PP2A allows BES1 and BZR1 to function with other TFs and co-factors to promote BR-induced gene expression and inhibit BR-repressed gene expression. Figure created with BioRender.com. BAK1, BRI1-ASSOCIATED KINASE1; BES1, BRI1-EMS-SUPPRESSOR 1; BIN2, BRASSINOSTEROID-INSENSITIVE 2; BIR3, BAK1-INTERACTING RECEPTOR-LIKE KINASES3; BKI1, BRI1 KINASE INHIBITOR1; BL, Brassinolide; BR, Brassinosteroid; BRI1, BRASSINOSTEROID INSENSITIVE 1; BRRE, BR Response Element; BSK, BR SIGNALING KINASES; BSS1, BRZ-SENSITIVE-SHORT HYPOCOTYL1; BSU, BRI1 SUPPRESSOR 1; BZR1, BRASSINAZOLE-RESISTANT 1; CDG, CONSTITUTIVE DIFFERENTIAL GROWTH; H2O2, Hydrogen Peroxide; HDA19, HISTONE DEACETYLASE19; IWS1, INTERACTING-WITH-SPT6-1; KIB1, KINK SUPPRESSED IN BZR1-1D; P, indicates phosphorylation; PP2A, PROTEIN PHOSPHATASE2A; PUB12/13, PLANT U-BOX12/13; REF6, RELATIVE OF EARLY FLOWER6; SDG8, SET DOMAIN GROUP 8; SH, indicates reduced Cys residue; SOH, indicates oxidized Cys residue; TF, transcription factor; TPL, TOPLESS; TRXh5, THIOREDOXIN H-TYPE 5; Ub, indicates ubiquitination.

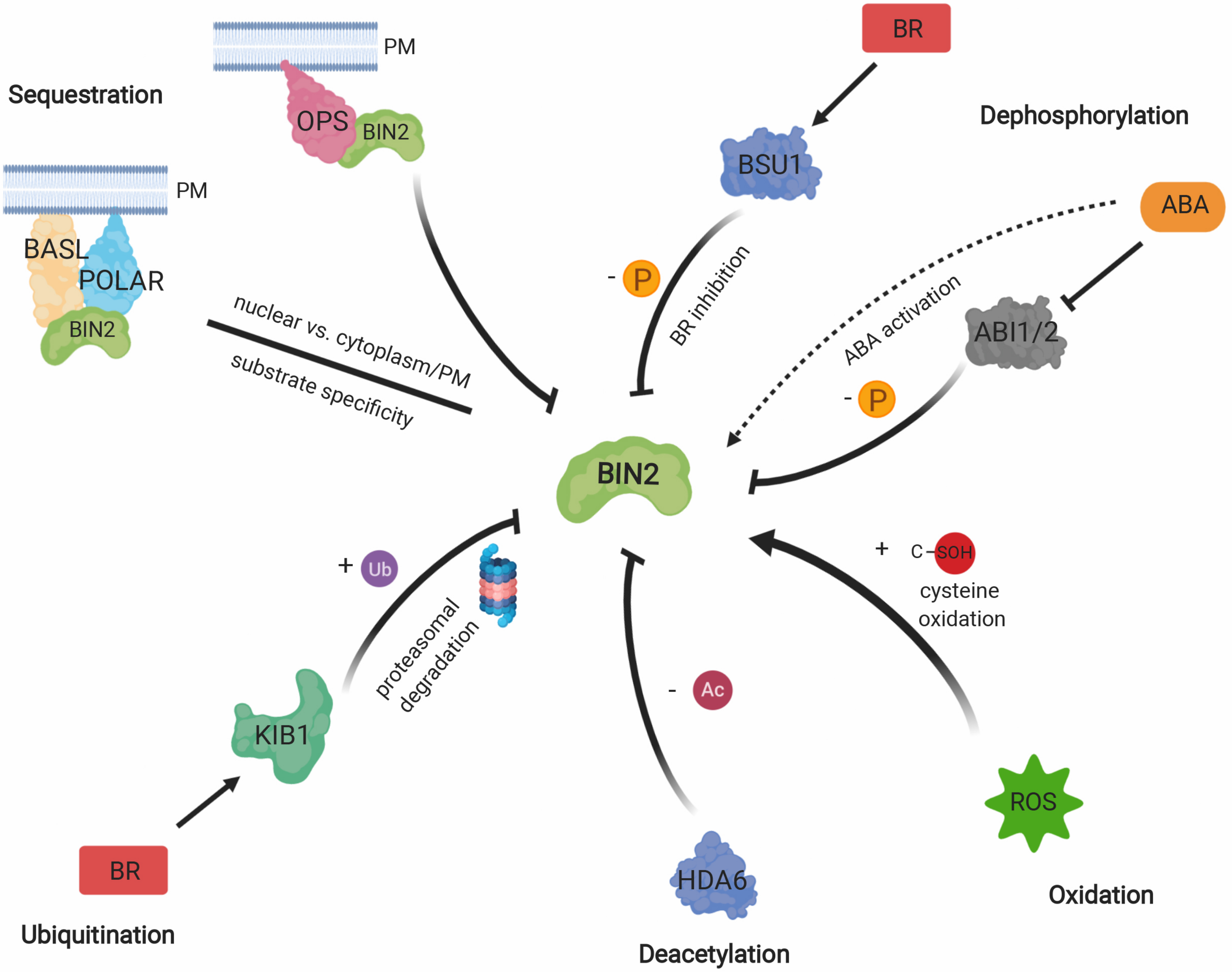

Figure 2: Mechanisms regulating BIN2 activity
In addition to canonical dephosphorylation and inactivation of BIN2 by BSU1 in the presence of BRs, several other mechanisms also regulate BIN2 activity. BIN2 is ubiquitinated by the E3 ubiquitin ligase KIB1 and degraded by the proteasome in the presence of BRs. Deacetylation by HDA6 inhibits BIN2 activity, whereas oxidation by reactive oxygen species (ROS) promotes BIN2 activity. ABA also activates BIN2 through the inhibition of ABI1/2 phosphatases that dephosphorylate BIN2. Finally, BIN2 is sequestered in a cell-type specific manner by OPS in the phloem or POLAR and BASL in the stomatal cell lineage. Figure created with BioRender.com. ABA, Abscisic acid; ABI1/2, ABA INSENSITIVE1/2; Ac, indicates acetylation; BASL, BREAKING OF ASYMMETRY IN THE STOMATAL LINEAGE; BIN2, BRASSINOSTEROID-INSENSITIVE2; BR, Brassinosteroid; BSU, BRI1 SUPPRESSOR1; HDA6, HISTONE DEACETYLASE6; KIB1, KINK SUPPRESSED IN BZR1-1D; OPS, OCTOPUS; P, indicates phosphorylation; PM, Plasma membrane; POLAR, POLAR LOCALIZATION DURING ASYMMETRIC DIVISION AND REDISTRIBUTION; ROS, Reactive oxygen species; SOH, indicates oxidized Cys residue; Ub, indicates ubiquitination.

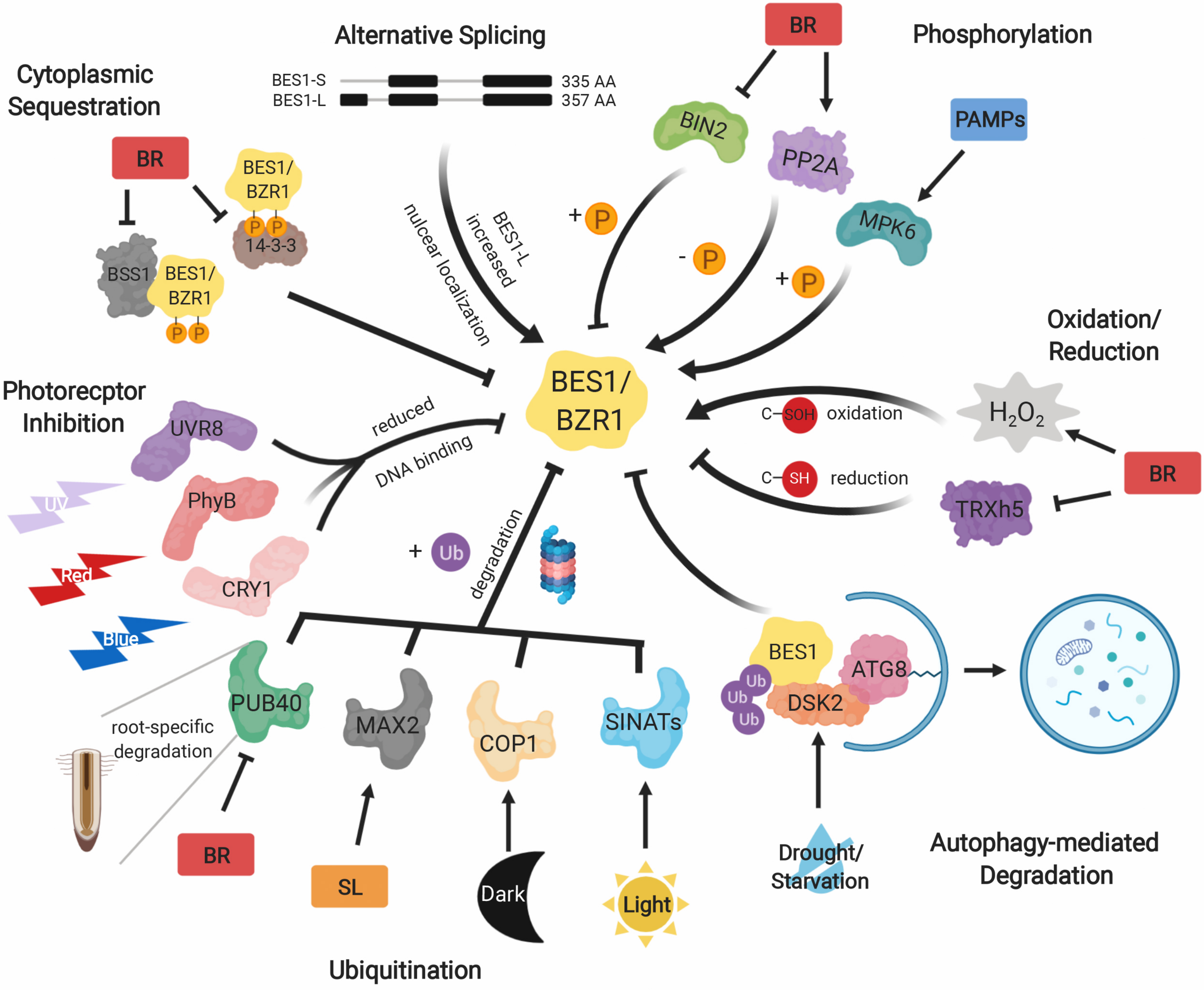

Figure 3: Diverse regulatory mechanisms controlling BES1 and BZR1 activity
BES1 and BZR1 activity is modulated by multiple modes of regulation. BES1 transcripts are subject to alternative splicing, with a longer BES1-L isoform displaying increased nuclear localization. Phosphorylation by BIN2 inactivates BES1 and BZR1, whereas MPK6 phosphorylation of BES1 in response to bacterial pathogens or Pathogen-associated molecular pattern (PAMPs) leads to its activation. PP2A dephosphorylates and activates BES1 and BZR1 in the presence of BRs. The production of hydrogen peroxide (H2O2) is promoted by BRs and activates BES1 and BZR1 via oxidation, whereas TRXh5 reduces BZR1. BES1 and BZR1 can be inactivated by cytoplasmic sequestration, photoreceptors that respond to UV, red and blue light, or ubiquitination. Several families of E3 ubiquitin ligases target BZR1 or BES1 in different tissues or in response to environmental cues, leading to their degradation by the proteasome or autophagy. DSK2 mediates selective autophagy for BES1 degradation during stress. Figure created with BioRender. ATG8, AUTOPHAGY-RELATED PROTEIN8; BES1, BRI1-EMS-SUPPRESSOR1; BIN2, BRASSINOSTEROID-INSENSITIVE 2; BR, Brassinosteroid; BSS1, BRZ-SENSITIVE-SHORT HYPOCOTYL1; BZR1, BRASSINAZOLE-RESISTANT1; COP1, CONSTITUTIVE PHOTOMORPHOGENIC1; CRY1, CRYPTOCHROME1; DSK2, DOMINANT SUPPRESSOR OF KAR2; H2O2, Hydrogen Peroxide; MAX2, MORE AXILLARY GROWTH LOCUS2; MPK6, MITOGEN-ACTIVATED PROTEIN KINASE6; P, indicates phosphorylation; PHYB, PHYB, PHYTOCHROME B; PP2A, PROTEIN PHOSPHATASE2A; PUB40, PLANT U-BOX40; SH, indicates reduced Cys residue; SINATs, SINA of Arabidopsis thaliana; SOH, indicates oxidized Cys residue; TRXh5, THIOREDOXIN H-TYPE 5; Ub, indicates ubiquitination; UVR8, UVB-RESISTANCE8.

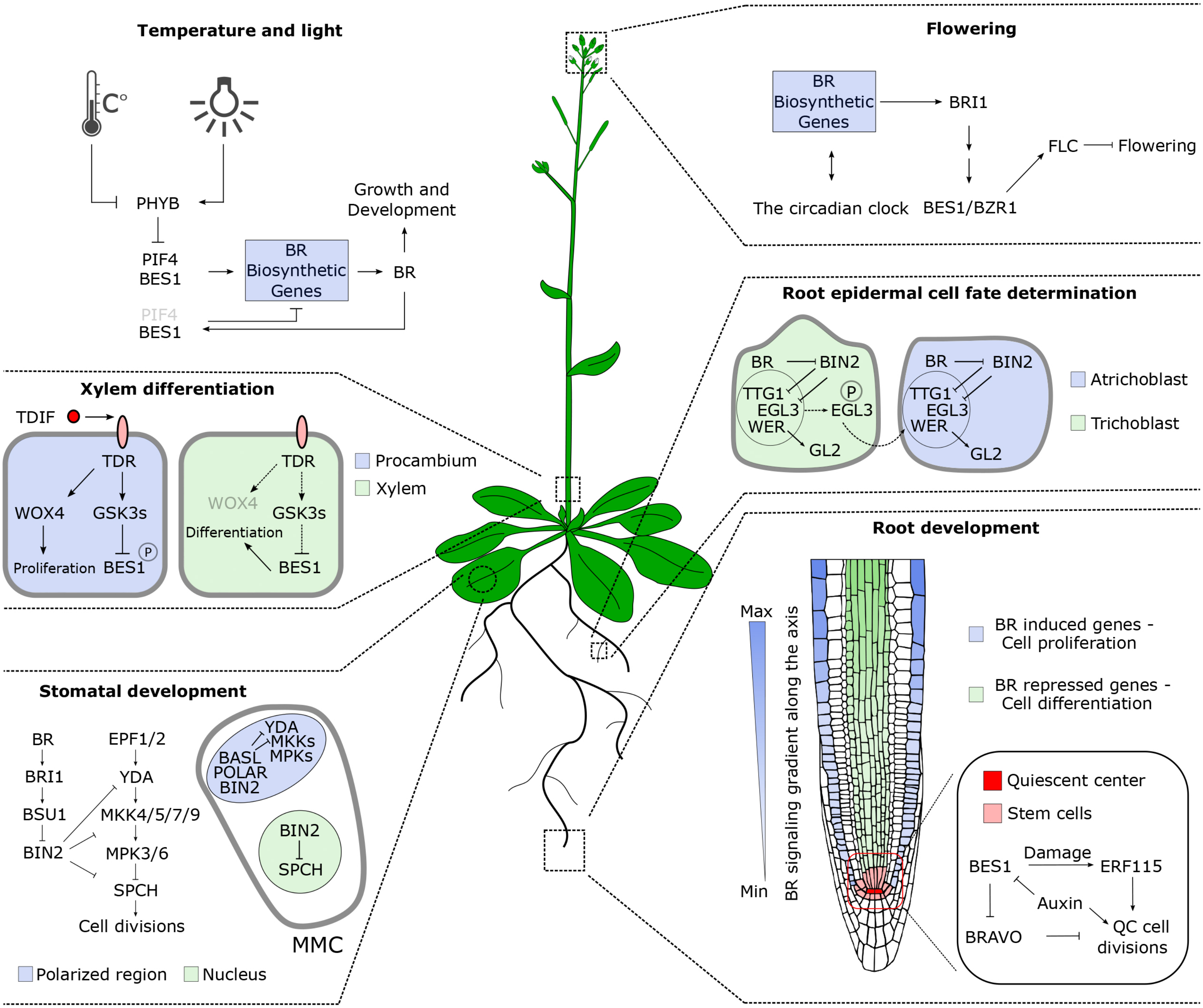

Figure 4: Summary of BR regulated developmental processes in Arabidopsis.

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