

SAUR Proteins as Effectors of Hormonal and Environmental Signals in Plant Growth

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ABSTRACT

The plant hormone auxin regulates numerous aspects of plant growth and development. Early auxin response genes mediate its genomic effects on plant growth and development. Discovered in 1987, *SMALL AUXIN UP RNAs (SAURs)* are the largest family of early auxin response genes. SAUR functions have remained elusive, however, presumably due to extensive genetic redundancy. However, recent molecular, genetic, biochemical, and genomic studies have implicated SAURs in the regulation of a wide range of cellular, physiological, and developmental processes. Recently, crucial mechanistic insight into SAUR function was provided by the demonstration that SAURs inhibit PP2C.D phosphatases to activate plasma membrane (PM) H⁺-ATPases and promote cell expansion. In addition to auxin, several other hormones and environmental factors also regulate SAUR gene expression. We propose that SAURs are key effector outputs of hormonal and environmental signals that regulate plant growth and development.

Key words: auxin, SAURs, cell expansion, acid growth, PP2C.D phosphatases, PM H⁺-ATPases

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INTRODUCTION

Auxin, derived from the Greek word *αυξειν* (auxein, meaning to grow or increase), was the first plant hormone identified. Since its discovery in the 1930s, auxin has fascinated plant biologists, who have sought to understand its biosynthesis, metabolism, transport, perception, signaling, and responses. Auxin influences nearly all aspects of plant growth and development through regulating cell division, expansion, differentiation, and patterning. In the nucleus, a co-receptor complex that is composed of TRANSPORT INHIBITOR RESPONSE1/AUXIN SIGNALING F-BOX PROTEINS (TIR1/AFBs) and AUXIN/INDOLE ACETIC ACID (AUX/IAA) transcriptional repressors perceives auxin to regulate gene transcription (reviewed in [Wang and Estelle, 2014](#)). These auxin-regulated genes then control downstream auxin responses.

Early or primary auxin response genes are transcriptionally induced by auxin within minutes and this induction does not require *de novo* protein synthesis ([Abel et al., 1994](#)). Most early auxin response genes are classified into three families: AUX/IAAs, *GRETCHEN HAGEN3s (GH3s)*, and SAURs (reviewed in [Hagen and Guilfoyle, 2002](#)). SAURs were originally identified in a differential hybridization screen for genes that were rapidly induced by auxin in elongating soybean hypocotyl sections ([McClure and Guilfoyle, 1987](#)). Since then, SAURs have been identified in diverse plant species, including mung bean

([Yamamoto et al., 1992](#)), pea ([Guilfoyle et al., 1993](#)), tomato ([Zurek et al., 1994](#)), *Arabidopsis* ([Gil et al., 1994](#)), apple ([Watillon et al., 1998](#)), radish ([Anai et al., 1998](#)), maize ([Yang and Poovaiah, 2000](#)), rice ([Jain et al., 2006](#)), moss ([Rensing et al., 2008](#)), sorghum ([Wang et al., 2010](#)), potato ([Wu et al., 2012](#)), cotton ([Yang et al., 2012](#)), litchi ([Kuang et al., 2012](#)), tobacco ([Wu et al., 2012](#)), pepper ([Wu et al., 2012](#)), petunia ([Wu et al., 2012](#)), peach ([Tatsuki et al., 2013](#)), and poplar ([Wang et al., 2014a](#)). While substantial progress has been made toward understanding the functions of both Aux/IAA and GH3 proteins in auxin responses ([Tiwari et al., 2001](#); [Staswick et al., 2002](#); [Tiwari et al., 2004](#); [Calderón Villalobos et al., 2012](#)), functional studies on SAURs have lagged behind. Nearly 30 years after their discovery, we have only just begun to unlock the secrets of the SAURs. In this review, we describe recent advances that implicate SAURs in regulating a wide range of cellular, physiological, and developmental processes.

SAURs, the Largest Family of Early Auxin Response Genes

Among early auxin response genes, the SAUR gene family is the most numerous. Genomic bioinformatic analyses have revealed that there are 81 SAURs (including two pseudogenes) in

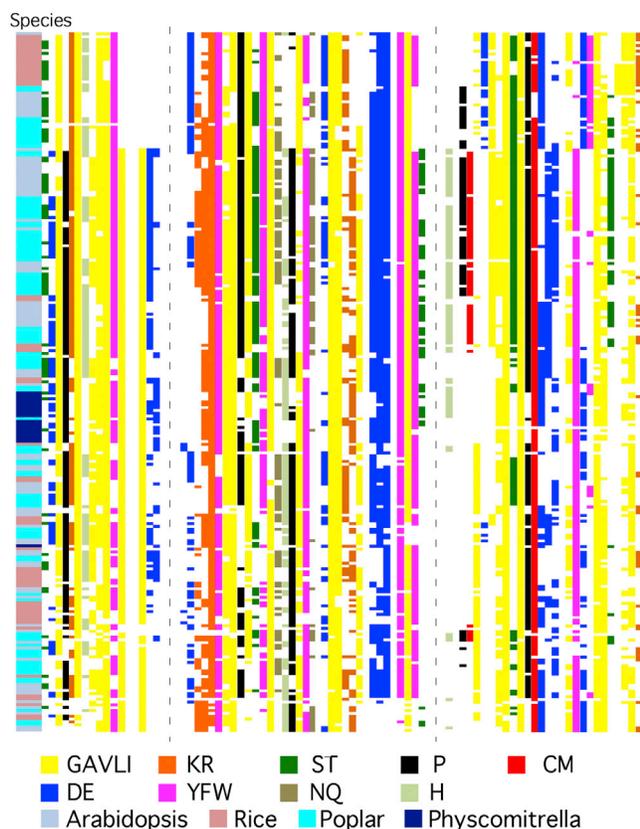


Figure 1. The SAUR Domain.

Multiple sequence alignment of the SAUR domain of 247 SAUR proteins from *Arabidopsis thaliana*, *Oryza sativa*, *Populus trichocarpa*, and *Physcomitrella patens* was performed using Clustal Omega. The consensus residue(s) at each position are color coded. Dotted lines indicate the positions of short, nonconserved insertions present in a few SAUR family members that were removed from the alignment.

Arabidopsis (Hagen and Guilfoyle, 2002), 58 SAURs (including two pseudogenes) in rice (Jain et al., 2006), 18 SAURs in moss (Rensing et al., 2008), 71 SAURs in sorghum (Wang et al., 2010), 134 SAURs in potato (Wu et al., 2012), 99 SAURs in tomato (Wu et al., 2012), and 79 SAURs in maize (Chen et al., 2014). Typically, SAUR genes are not randomly distributed in the genome, as many of them are found in tandem arrays of extremely highly related genes in soybean (McClure et al., 1989), *Arabidopsis* (Hagen and Guilfoyle, 2002), rice (Jain et al., 2006), tomato (Wu et al., 2012), and maize (Chen et al., 2014). Tandem and segmental duplication events likely contributed to the expansion of the SAUR gene family (Wu et al., 2012; Chen et al., 2014). The genomic structures of SAUR genes show similar features. The vast majority of SAUR genes lack introns. Many also contain one or more auxin response elements (AuxREs) within their promoter region and possess a downstream destabilizing (DST) element in the 3' untranslated region (UTR) (Hagen and Guilfoyle, 2002; Jain et al., 2006; Wu et al., 2012; Chen et al., 2014). The DST consists of three conserved elements separated by nonconserved bases of variable length (GGA(N)_xATAGAT(N)_xGTA) (McClure et al., 1989; Newman et al., 1993; Sullivan and Green, 1996). This sequence is found in 30 of the 79 *Arabidopsis* SAUR genes (Supplemental Table 1). For at least some SAUR transcripts, the DST element

confers instability (Sullivan and Green, 1996). However, the functional significance of these DST elements is uncertain, as *Arabidopsis* mutants defective in DST-mediated mRNA degradation exhibit no apparent phenotype (Johnson et al., 2000).

SAUR genes encode small proteins that are unique to plants and contain no obvious characterized motifs indicative of a biochemical function. The predicted molecular weights of *Arabidopsis* SAUR proteins range from 9.3 to 21.4 kDa. SAUR proteins have been predicted to reside in the nucleus, cytosol, mitochondrion, chloroplast, and on the plasma membrane (Wu et al., 2012; Chen et al., 2014). Studies employing SAUR fusion proteins have provided evidence for SAUR localization to the nucleus (ZmSAUR2, Knauss et al., 2003; SAUR32, Park et al., 2007; SAUR36, Narsai et al., 2011), cytosol (OsSAUR39, Kant et al., 2009; SAUR55, Narsai et al., 2011; SAUR41, Kong et al., 2013; SAUR40 and SAUR71, Qiu et al., 2013), and plasma membrane (SAUR63, Chae et al., 2012; SAUR19, Spartz et al., 2012). While such findings should be interpreted with caution, as most of these studies were conducted with SAUR overexpression constructs and sometimes in heterologous systems, the findings suggest that different SAURs may localize to distinct cellular compartments.

Protein multiple sequence alignment revealed that SAURs from different plant species contain a central domain specific to SAUR proteins (CDD superfamily c103633, Marchler-Bauer et al., 2013). This ~60 amino acid domain, referred to here as the SAUR domain, is highly conserved (Figure 1), suggesting that this domain is essential for SAUR protein function. The SAUR domain is dominated by hydrophobic amino acids and contains short, highly conserved, charged patches and a nearly invariant cysteine residue. Several proline and aromatic amino acids are also exceptionally highly conserved. Outside of the SAUR domain, SAUR proteins have variable length N- and C-terminal extensions, which exhibit much lower degrees of conservation (Supplemental Figure 1).

SAUR expression is regulated at multiple levels. Early studies revealed that several SAUR genes are transcriptionally induced by auxin in mung bean, pea, *Arabidopsis*, radish, and maize (reviewed in Hagen and Guilfoyle, 2002). More recent genomic methods of assessing gene expression have greatly expanded the number of known auxin-induced SAUR genes in *Arabidopsis* (Tian et al., 2002; Zhao et al., 2003; Goda et al., 2004; Nemhauser et al., 2004; Redman et al., 2004; Okushima et al., 2005; Chapman et al., 2012). However, this list likely remains incomplete as many SAUR genes are not represented on the *Arabidopsis* ATH1 gene chip and several of the SAUR probe sets that are on this chip are not specific to individual SAUR genes. With these caveats, it is estimated that about one-half of the *Arabidopsis* SAUR genes are upregulated to some extent by auxin treatment (Supplemental Figure 2). Interestingly, a small number of SAUR genes appear to be repressed by auxin (Supplemental Figure 2). In general, auxin-induced SAURs tend to be most highly expressed in shoots, while many auxin-repressed and nonresponsive SAURs are preferentially expressed in roots (Paponov et al., 2008).

A second layer of regulation of SAUR expression involves mRNA stability. At least some SAUR transcripts are highly unstable

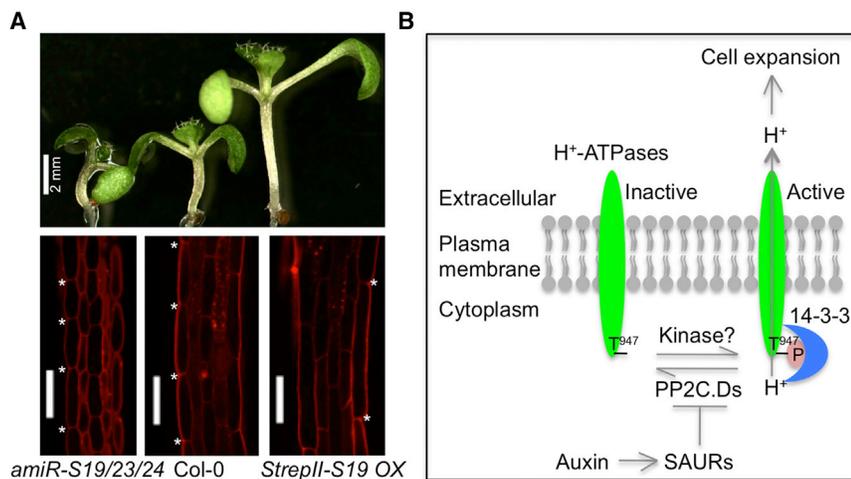


Figure 2. SAURs Activate PM H^+ -ATPases Via Inhibiting PP2C.D Phosphatases to Promote Cell Expansion.

(A) Seven-day-old light-grown *Arabidopsis* seedlings. Seedlings were stained in 10 $\mu\text{g/ml}$ propidium iodide, and hypocotyl epidermal cells were observed under a Nikon A1 spectral confocal microscope. Asterisks (*) indicate the intercellular space of two adjacent epidermal cells. Scale bar represents 100 μm . **(B)** A model for SAUR-regulated cell expansion. Adapted from Spartz et al. (2014).

or stabilized SAUR fusion proteins have been utilized. Such studies have implicated SAURs in a wide range of cellular, physiological, and developmental processes involving hormonal and environmental control

of plant growth and development. While the findings of overexpression studies should always be interpreted with caution, many of these findings have been supported by RNA silencing strategies that target multiple SAUR subfamily members, as well as additional biochemical and genetic findings. Below we review the evidence for SAUR-mediated regulation of several aspects of plant growth and development.

Cell Expansion

Cell expansion is a fundamental process essential for plant growth and development (reviewed in Braidwood et al., 2013). Hypocotyl growth is mainly caused by cell expansion, making it an excellent study system (Gendreau et al., 1997). Auxin plays a major role in regulating hypocotyl growth (Gray et al., 1998; Zhao et al., 2001). Many SAURs are highly expressed in elongating hypocotyls (McClure and Guilfoyle, 1987; Chae et al., 2012; Spartz et al., 2012; Stamm and Kumar, 2013), and it has long been hypothesized that SAURs may be involved in auxin-regulated cell expansion. Experimental results supporting a positive role for SAURs in cell expansion were obtained from several recent studies in *Arabidopsis*. Overexpression of SAUR36 (Stamm and Kumar, 2013), SAUR41 (Kong et al., 2013), or stabilized fusion proteins of SAUR19 (Spartz et al., 2012) or SAUR63 (Chae et al., 2012) promotes hypocotyl elongation as a result of increased cell expansion. By contrast, seedlings expressing artificial microRNAs (amiRNAs) targeting multiple members of the SAUR19 (Spartz et al., 2012) or SAUR63 (Chae et al., 2012) subfamilies have slightly shorter hypocotyls with smaller epidermal cells (Figure 2A). These findings suggest that these SAURs positively regulate cell expansion to promote hypocotyl growth. In addition to hypocotyl phenotypes, SAUR63 overexpression and amiRNA knockdown plants also exhibit longer and shorter stamen filaments, respectively. Likewise, SAUR19 and additional SAURs have also been implicated in leaf cell expansion, which we review below.

What is the mechanism underlying SAUR-mediated cell expansion? Spartz et al. (2014) recently proposed that SAURs promote cell expansion via an acid growth mechanism. The acid growth theory was proposed in the 1970s to explain auxin-mediated cell expansion (Rayle and Cleland, 1970, 1980, 1992;

(McClure and Guilfoyle, 1989; Franco et al., 1990). The DST element in the 3'-UTR of SAUR genes functions as an mRNA instability determinant, responsible for the rapid turnover of SAUR mRNAs (Newman et al., 1993). In the cases examined, auxin does not appear to regulate SAUR transcript stability (Gil and Green, 1996). Interestingly, some SAUR transcripts have also been shown to be specifically polyadenylated by PAPS1, one of the four canonical nuclear poly(A) polymerases of *Arabidopsis* (Vi et al., 2013). This appears to be functionally significant, as overexpression of a GFP-SAUR19 transgene containing a heterologous 3'-UTR could suppress a subset of *paps1* mutant phenotypes.

In addition to regulation at the transcriptional and posttranscriptional levels, SAUR expression is also subject to posttranslational control. Several SAUR proteins have been found to be highly unstable. The first such report was with the maize SAUR2 (ZmSAUR2) protein (Knauss et al., 2003). [^{35}S]Methionine pulse-chase labeling experiments revealed that ZmSAUR2 is a short-lived protein with a half-life of about 7 min. More recently, the *Arabidopsis* SAUR19 and SAUR63 proteins were found to be highly unstable. Treatment with the 26S proteasome inhibitor MG132 causes accumulation of SAUR19 and SAUR63, suggesting the involvement of the ubiquitin-26S proteasome pathway in regulating SAUR degradation (Chae et al., 2012; Spartz et al., 2012). However, neither the mechanism nor regulation of SAUR ubiquitylation has been elucidated, although in the case of SAUR63, the degradation rate was reported to be faster under dim light than bright light (Chae et al., 2012). Interestingly, the SAUR19 and SAUR63 proteins are dramatically stabilized when expressed as fusions with GFP, GUS, or even some small epitope tags (StrepII) (Chae et al., 2012; Spartz et al., 2012).

Roles of SAURs in Plant Growth and Development

Since their discovery, SAURs have puzzled plant biologists. All plant genomes appear to contain large SAUR gene families. Because of likely genetic redundancy and the difficulties in generating higher order *saur* mutants due to the tight linkage of tandemly arrayed SAUR genes, it is challenging to employ loss-of-function genetic approaches to elucidate SAUR functions in plant growth and development. To circumvent this problem, gain-of-function genetic approaches that overexpress SAURs

Hager, 2003). According to this theory, auxin stimulates susceptible cells to excrete protons, lowering apoplastic pH and elevating membrane potential. Plasma membrane (PM) H⁺-ATPases are responsible for pumping protons into the apoplast. The resulting reduction in apoplastic pH activates cell wall-loosening enzymes such as expansins and hyperpolarizes the plasma membrane, thus increasing cell wall extensibility and promoting solute and water uptake to generate the turgor increase required for cell expansion. Until recently, however, the acid growth theory has lacked strong genetic support and the underlying molecular mechanism(s) by which auxin activates PM H⁺-ATPase activity were uncertain.

The *Arabidopsis* genome encodes 11 PM H⁺-ATPases called AHAs (ARABIDOPSIS H⁺-ATPASES), and AHA1 and AHA2 are the most highly expressed isoforms (Baxter et al., 2003; Haruta et al., 2010). Takahashi et al. (2012) provided key insight into auxin-mediated PM H⁺-ATPase activation with the demonstration that auxin rapidly induces phosphorylation of the penultimate threonine residue of the C-terminal autoinhibitory domain of AHAs (Thr⁹⁴⁷ in AHA2). Phosphorylation of this residue coincides with 14-3-3 protein binding and PM H⁺-ATPase activation (Fuglsang et al., 1999; Kinoshita and Shimazaki, 1999). SAUR19 activates PM H⁺-ATPases by promoting AHA Thr⁹⁴⁷ phosphorylation (Spartz et al., 2014). This phosphorylation increase results in elevated PM H⁺-ATPase activity. Consistent with these findings, plants overexpressing stabilized SAUR19 fusion proteins exhibit remarkable phenotypic similarity to *open stomata2* (*ost2*) mutants. *ost2* is a gain-of-function allele of AHA1 that results in a constitutively active enzyme (Merlot et al., 2007). Like *ost2* mutants, seedlings expressing SAUR19 fusion proteins exhibit increased elongation growth, reduced apoplastic pH, drought hypersensitivity due to improper guard cell regulation, and constitutive expression of pathogen defense genes (Spartz et al., 2014). These phenotypes are also elicited by fusicoccin, a fungal wilting toxin that specifically activates PM H⁺-ATPases (Marre, 1979; Singh and Roberts, 2004).

SAUR19, as well as several additional SAURs, interact with several members of the 9-member PP2C.D family of protein phosphatases (Schweighofer et al., 2004; Spartz et al., 2014). Enzymatic assays revealed that SAUR binding inhibits phosphatase activity. Spartz et al. (2014) employed gain- and loss-of-function genetic studies to demonstrate that SAUR19 and PP2C.D phosphatases function antagonistically to regulate cell expansion by controlling AHA Thr⁹⁴⁷ phosphorylation and activity. For example, whereas seedlings overexpressing GFP-SAUR19 exhibit increased cell expansion, Thr⁹⁴⁷ phosphorylation, and PM H⁺-ATPase activity, PP2C.D1 overexpression confers reductions in all three processes. Importantly, both SAUR19-mediated growth promotion and PP2C.D1-mediated growth repression occur in a PM H⁺-ATPase-dependent manner.

Consistent with these gain-of-function findings, seedlings expressing a *PP2C.D2/5/7/8/9* amiRNA knockdown construct partially phenocopy *GFP-SAUR19* overexpression seedlings. Furthermore, PP2C.D1 interacts with AHA2 on the plasma membrane and can catalyze AHA Thr⁹⁴⁷ dephosphorylation both *in vitro* and when heterologously expressed in yeast. Based on these results, the authors proposed that auxin induction of

SAUR expression results in repression of PP2C.D-mediated dephosphorylation of PM H⁺-ATPase Thr⁹⁴⁷. Consequently, PM H⁺-ATPase equilibrium is shifted to the phosphorylated, active state, resulting in increased apoplastic acidification and cell expansion (Figure 2B).

Curiously, in contrast to the above findings, some SAURs have been suggested to negatively regulate cell expansion. Transgenic seedlings overexpressing SAUR32 (also known as AAM1, ABOLISHED APICAL HOOK MAINTENANCE1) exhibit reduced hypocotyl elongation when grown either in the dark or under red light (Park et al., 2007). Likewise, *saur36* T-DNA loss-of-function plants have larger leaves containing larger epidermal cells, suggesting that SAUR36 may negatively regulate cell expansion to inhibit leaf growth (Hou et al., 2013). However, Stamm and Kumar (2013) reported that SAUR36 overexpressing seedlings display a long hypocotyl phenotype. At this point, it is unclear whether these disparate findings with SAUR36 in leaves and hypocotyls are due to organ-specific regulation or some other factor. Future studies are required to elucidate the potential mechanisms of SAUR32 and SAUR36 inhibition of cell expansion. Nonetheless, it is tempting to speculate that different SAUR family members might act in an antagonistic fashion, a phenomenon frequently observed in other gene families, such as AUXIN RESPONSE FACTORS (ARFs) and ARABIDOPSIS RESPONSE REGULATORS (ARRs) (reviewed in Guilfoyle and Hagen, 2007; Argueso et al., 2010).

Shade Avoidance Responses

Under shade (a low red/far-red ratio) conditions, many plants exhibit increased elongation growth of stems and petioles, hypostomatic leaves, reduced branching, increased leaf senescence, and early flowering. These growth responses are known as shade avoidance responses. In *Arabidopsis* seedlings, the most dramatic shade avoidance response is hypocotyl elongation (reviewed in Casal, 2013; de Wit et al., 2014). Mutant seedlings defective in auxin biosynthesis, transport, signaling, and response exhibit reduced hypocotyl growth in response to shade, indicating that auxin is an important regulator of shade avoidance responses. In response to shade, the PHYTOCHROME INTERACTING FACTOR (PIF4, PIF5, and PIF7) transcription factors activate expression of auxin biosynthetic genes to upregulate indole-3-acetic acid (IAA) production.

A link between SAURs and shade avoidance response was suggested by Roig-Villanova et al. (2007). In *Arabidopsis*, PHYTOCHROME RAPIDLY REGULATED1 (PAR1) and PAR2 are atypical basic helix-loop-helix (bHLH) proteins, which lack the basic domain required for DNA-binding. Expression of *PAR1* and *PAR2* is upregulated by shade in seedlings, inhibiting shade avoidance responses. *PAR1* and *PAR2* repress the expression of SAUR15 (also known as SAUR-AC1), SAUR67 (misannotated as SAUR68), and several additional SAURs. *PAR1* and *PAR2* lack a DNA-binding domain. Instead, *PAR1* interacts with PIF4 to inhibit PIF4 DNA-binding activity (Hao et al., 2012). Many SAURs, including SAUR15, are potential direct targets of PIF4 (Oh et al., 2012). Therefore, *PAR1*-mediated transcriptional repression of SAUR15 likely involves *PAR1* inhibition of PIF4-mediated transcriptional activation. Like *PAR1* and

PAR2, *SAUR15* and *SAUR67* are also induced by shade. However, this induction is transient, suggesting that *PAR1* and *PAR2* repression of *SAUR* expression may serve as a feedback mechanism for attenuating shade-induced *SAUR* expression.

Additional evidence supporting a role of SAURs in shade-stimulated elongation growth was provided by [Hornitschek et al. \(2012\)](#). PIF4 and PIF5 function redundantly to promote shade-induced hypocotyl growth. The authors used chromatin immunoprecipitation sequencing (ChIP-seq) to identify the genomic binding sites for PIF5 in plants exposed to simulated shade for 2 h. The potential direct PIF5 targets include the *SAUR19* subfamily genes (*SAUR19*, 20, 21, 22, 23, and 24) as well as additional *SAUR* genes. In response to shade, expression of *SAUR19*, *SAUR21*, *SAUR23*, and *SAUR24* is rapidly induced in elongating hypocotyls ([Spartz et al., 2012](#)). Upregulation of *SAUR* gene expression by shade could be achieved both directly by PIF4 and PIF5-mediated gene activation and indirectly by increased auxin levels controlled by PIF4, PIF5, and PIF7. Presumably, these increases in *SAUR* expression promote the cell expansion underlying the increased hypocotyl and petiole growth characteristic of shade avoidance growth. However, genetic studies are clearly needed to support this hypothesis.

High Temperature-Induced Growth

Similar to shade, high temperature also induces rapid elongation growth. *Arabidopsis* seedlings exhibit elongated hypocotyls and petioles in response to high temperature. High temperature promotes auxin biosynthesis in *Arabidopsis* seedlings ([Gray et al., 1998](#)). This upregulation of auxin biosynthesis is controlled by PIF4, the primary PIF family member that controls high temperature-mediated growth ([Koini et al., 2009](#)). PIF4 directly regulates the expression of auxin biosynthesis genes *TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS1* (*TAA1*) and *CYP79B2* ([Franklin et al., 2011](#)) and *YUCCA8* (*YUC8*) ([Sun et al., 2012](#)) to increase auxin production in response to high temperature.

Recent findings indicate that SAURs contribute to PIF4-mediated elongation growth at high temperature. *SAURs* in the *SAUR19* subfamily positively regulate cell expansion to promote hypocotyl growth ([Spartz et al., 2012, 2014](#)). High temperature rapidly induces the expression of these genes in hypocotyls ([Franklin et al., 2011](#)). However, *pif4* null mutants exhibit dramatically reduced expression of these genes at high temperature, indicating that high temperature-induced *SAUR* gene expression is PIF4-dependent. Furthermore, *SAUR19* overexpression rescues the hypocotyl elongation defects of *pif4* mutants at high temperature. These results suggest that the *SAUR19* subfamily genes function downstream of PIF4 to regulate hypocotyl growth in response to high temperature ([Franklin et al., 2011](#)). PIF4-dependent *SAUR* gene expression could be explained by two distinct mechanisms. As described above, PIF4 upregulates auxin biosynthesis in response to elevated temperatures. Since the *SAUR19* subfamily genes are auxin inducible ([Spartz et al., 2012](#)), PIF4-mediated increases in auxin levels would induce the expression of these genes. In addition, PIF4 may directly regulate the expression of the *SAUR19* subfamily genes. [Oh et al. \(2012\)](#) provided experimental evidence to support this hypothesis, as *SAUR19*, 20, 21, 22, 23, and 24 were all

identified as high-confidence PIF4 target genes by ChIP-seq analysis. Although targeted follow-up experiments are needed to confirm that PIF4 directly binds to the promoters of these genes, it seems likely that both mechanisms contribute to PIF4-dependent *SAUR* gene expression to promote elongation growth at high temperature.

Tropic Growth

SAUR-mediated changes in cell expansion have also been implicated in tropic growth responses; directional growth in response to environmental stimuli such as light and gravity. Light and gravity signals cause an asymmetric auxin distribution, resulting in differential growth (bending) of shoots and roots during phototropic and gravitropic responses. Shoots exhibit positive phototropism, growing toward the light source. Auxin accumulates on the shaded side of plant stems, promoting the differential cell expansion that results in stem bending toward the light. In response to gravity, shoots exhibit negative gravitropism (grow upward), while roots exhibit positive gravitropism (grow downward). A higher concentration of auxin on the lower side of shoots causes increased growth, while a higher concentration of auxin on the lower side of roots inhibits growth (reviewed in [Goyal et al., 2013](#); [Vandenbrink et al., 2014](#)).

Several of the first studies on *SAUR* genes reported increases in *SAUR* expression within the elongating cells of tropically stimulated stems. Using a tissue printing technique, [McClure and Guilfoyle \(1989\)](#) examined the distribution of *SAUR* transcripts in the elongating region of excised soybean hypocotyls during gravitropism. Upon gravity stimulation, *SAUR* transcripts changed from a symmetric distribution to an asymmetric distribution, accumulating on the lower side of hypocotyls. Likewise, increased GUS activity was observed on the lower side of gravistimulated tobacco stems expressing a *pSAUR10A::GUS* reporter ([Li et al., 1991](#)), and RNA *in situ* hybridization detected more abundant soybean *SAUR* transcripts in the epidermis and cortex on the lower side of hypocotyls during gravitropic response ([Gee et al., 1991](#)). In addition to gravitropism, asymmetric *pSAUR10A::GUS* activity was also observed in tobacco stems during phototropic growth, with the shaded side exhibiting increased GUS staining ([Li et al., 1991](#)). More recent transcriptomic studies have revealed that many *SAUR* genes are preferentially expressed on the lower, elongating sides of gravistimulated rice shoots and *Arabidopsis* stems ([Hu et al., 2013](#); [Taniguchi et al., 2014](#)). In fact, of the 30 genes exhibiting differential expression between the upper and lower flanks of *Arabidopsis* stems following a 30-min gravistimulation, 14 were *SAUR* genes. Curiously, all 14 of these genes belong to the *SAUR19* and *SAUR63* clades of the phylogeny ([Supplemental Figure 2](#)).

Overexpression studies have provided some functional support for the involvement of SAURs in tropic growth responses. *Arabidopsis* seedlings expressing a *GFP-SAUR19* transgene driven by the cauliflower mosaic virus 35S promoter exhibit a dramatic delay in phototropic bending and a wavy root growth habit ([Spartz et al., 2012](#)). *SAUR41* is specifically expressed in the endodermis of the root elongation zone on the upper side of gravistimulated roots, and ectopic expression of *SAUR41* resulted in altered gravitropic root growth ([Kong et al., 2013](#)).

Together with the above gene expression studies correlating *SAUR* expression with differential growth of the two sides of tropically stimulated roots and shoots, it seems likely that asymmetric *SAUR* expression may promote the cell expansion underlying tropic growth. Disrupting the normal expression pattern by expressing *SAUR* genes from heterologous promoters results in the loss of the ability to control differential cell expansion. Interestingly, a recent modeling study (Hohm et al., 2014) found that apoplastic acidification is an important component of phototropic bending, suggesting that SAUR-mediated activation of PM H⁺-ATPases may drive the differential cell expansion associated with tropic growth.

Apical Hook Development

Like tropic growth, the development, maintenance, and opening of apical hooks involves the regulated formation of auxin gradients to control differential cell expansion (reviewed in Abbas et al., 2013). Dicotyledonous seedlings grown in the dark develop an apical hook structure caused by differential cell expansion. The apical hook protects the shoot apical meristem from damage when seedlings grow through soil. Upon reaching the soil surface and perceiving light, hook opening is triggered.

Gene expression and gain-of-function studies in *Arabidopsis* have suggested that SAURs are involved in apical hook development. The auxin-regulated reporter *DR5::GUS* is preferentially expressed on the inner side of the apical hook (Abbas et al., 2013). Although *SAUR32* expression does not appear to be auxin inducible, *SAUR32* expression in apical hooks closely resembles that of *DR5::GUS* (Park et al., 2007). While the apical hooks of a *saur32* T-DNA null mutant appear wild-type, *SAUR32* overexpression from the 35S promoter confers a hookless phenotype due to a defect in apical hook maintenance. Interestingly, *SAUR32* overexpression seedlings also have short hypocotyls, suggesting that *SAUR32* negatively regulates cell expansion. These findings suggest that during apical hook development, *SAUR32* might inhibit cell expansion of the inner side of the apical hook, which is important for hook maintenance. Paradoxically, however, overexpression of SAURs that promote hypocotyl cell expansion such as *SAUR19* and *SAUR36* also confers a hookless phenotype (Spartz et al., 2012; Stamm and Kumar, 2013). Although additional studies are clearly required, the above findings suggest that proper expression of both growth-promoting and growth-inhibiting *SAUR* genes is required to achieve the differential cell expansion necessary to achieve normal apical hook development. The challenge is to understand the molecular mechanisms behind SAUR-regulated differential growth in the apical hook. That said, initial genetic studies of the recently proposed SAUR-PP2C.D-mediated regulation of PM H⁺-ATPase activity support a role for this regulatory module in apical hook development. When grown in the dark, *pp2c.d1* and *pp2c.d2* mutants, as well as the constitutively active *ost2* PM H⁺-ATPase mutant, exhibit reduced apical hook angles and open cotyledon phenotypes (Spartz et al., 2014).

Leaf Growth and Senescence

Auxin plays a critical role in leaf growth and development, regulating leaf initiation, lamina formation, vein development, and leaf shape and size (reviewed in Scarpella et al., 2010; Byrne, 2012). Several lines of evidence have suggested that SAURs

regulate leaf growth through controlling cell expansion or division, contributing to auxin-regulated leaf growth and development.

Spartz et al. (2012) found that the *SAUR19* subfamily genes positively regulate leaf growth. Plants expressing an artificial miRNA targeting *SAUR19*, 23, and 24 display reduced leaf area, while plants expressing stabilized *SAUR19* fusion proteins exhibit larger leaves than wild-type. These changes in leaf size appear to be entirely due to altered cell size, suggesting that the *SAUR19* subfamily genes positively regulate cell expansion to promote leaf growth. Strikingly, *SAUR19* appears quite unique among leaf growth-promoting genes. For example, a recent study examining 13 genes that confer increased leaf size when either overexpressed or mutated revealed that *SAUR19* and *EXPANSIN10* were the only ones that specifically affected cell expansion rather than cell division (Vanhaeren et al., 2014). Furthermore, when tested in pairwise combinations with the other growth-promoting mutations/transgenes, *SAUR19* overexpression resulted in synergistic increases in leaf size. These findings suggest that the *SAUR19* subfamily genes are attractive targets for future genetic engineering efforts aimed at enhancing plant biomass.

Unlike the *SAUR19* subfamily genes, a few SAURs have been implicated as negative regulators of leaf growth. *SAUR76* is highly expressed in roots, but only weakly expressed in leaves. Interestingly, while auxin treatment resulted in a dramatic upregulation in roots, no increase in *SAUR76* expression was observed in leaves (Markakis et al., 2013). When overexpressed from the 35S promoter, *SAUR76* conferred a reduction in leaf size. This effect appears to be the result of a reduction in cell number rather than cell size, suggesting that *SAUR76* might negatively regulate cell division to inhibit leaf growth (Markakis et al., 2013). Similarly, *SAUR36* also appears to negatively regulate leaf growth, as *saur36* null mutants exhibit enlarged leaves (Hou et al., 2013). However, unlike *SAUR76* overexpression, *saur36* mutants exhibit an increase in leaf epidermal cell size, suggesting that *SAUR36* negatively regulates leaf cell expansion rather than cell division.

In addition to regulating leaf growth and development, auxin has also been implicated in leaf senescence, a process eventually leading to leaf death. However, the precise functions of auxin in leaf senescence are unclear, as contradictory results have been reported as to whether auxin negatively or positively regulates leaf senescence (reviewed in Jibrán et al., 2013; Khan et al., 2014). Recent studies of auxin-induced *SAUR* genes in *Arabidopsis* and rice suggest that auxin may promote leaf senescence through the expression of SAURs. *Arabidopsis SAUR36*, also known as *SAG201* (*SENESCENCE-ASSOCIATED GENE201*), is highly upregulated during leaf senescence (Hou et al., 2013) and leaf senescence of two independent *saur36* T-DNA mutants was significantly delayed. Furthermore, inducible overexpression of *SAUR36* promoted premature leaf senescence. Interestingly, only *SAUR36* expression constructs that lacked the DST element within the 3'-UTR conferred an early senescence phenotype, suggesting that posttranscriptional regulation of *SAUR36* transcript levels may play an important functional role. These findings convincingly demonstrate that *SAUR36* positively regulates leaf

senescence. Similar findings were obtained with *OsSAUR39* in rice. Although loss-of-function support is lacking, *OsSAUR39* expression is elevated in older leaves and overexpression of *OsSAUR39* promoted early senescence (Kant et al., 2009). Further studies are needed to provide mechanistic insight and to determine whether the upregulation of *SAUR36* and *OsSAUR39* expression during senescence is auxin dependent.

Root Growth and Development

Auxin is essential for root growth and development, regulating stem cell specification and division, meristem size, cell elongation, and differentiation (reviewed in Sozzani and Iyer-Pascuzzi, 2014; Takatsuka and Umeda, 2014). In addition to the primary roots, auxin also regulates the initiation and growth and development of lateral roots. While the majority of *SAUR* studies discussed above have focused on expression patterns and functional studies in shoots, several of the aforementioned *SAUR* overexpression lines also exhibit root phenotypes.

SAUR76 is expressed in the pericycle and endodermal layers of the root elongation zone and at lateral root initiation sites (Markakis et al., 2013). Auxin treatment strongly upregulates *SAUR76* expression in roots. While *saur76* null mutants exhibited no apparent phenotype, overexpression of *SAUR76* conferred increases in root meristem size and primary root growth, suggesting that *SAUR76* may positively regulate root growth. *SAUR41*, on the other hand, is specifically expressed in the quiescent center and cortical/endodermal initials of the stem cell niche of primary and lateral roots. Its expression pattern is expanded to the root endodermis in seedlings treated with auxin for 1 h. Plants overexpressing *SAUR41* exhibited increased primary root growth and lateral root numbers (Kong et al., 2013). Interestingly, expression of *SAUR41* under the control of tissue/cell type-specific promoters (*PIN1*, *WOX5*, *PLT2*, and others) caused defects in root meristem patterning and auxin distribution within root tips (as assayed using the *DR5_{rev}::GFP* reporter). As in shoots, there is some evidence for positive- and negative-acting SAURs in root growth and development. In contrast to the above findings with *Arabidopsis SAUR41* and *SAUR76*, overexpression of *OsSAUR39* in rice resulted in reductions in root elongation and lateral root development (Kant et al., 2009). To date, no molecular or biochemical mechanism has been elucidated to explain how SAUR proteins might regulate root growth and development.

Calcium Signaling

In response to cytosolic free Ca^{2+} increases, calcium-binding proteins, such as calmodulins (CaM) and CaM-like proteins, sense Ca^{2+} signals and initiate signaling cascades to regulate many cellular, physiological, and developmental processes (reviewed in Dodd et al., 2010; Kudla et al., 2010). Numerous studies have suggested that Ca^{2+} is involved in auxin signaling or responses (reviewed in Vanneste and Friml, 2013). Auxin induces a rapid and transient increase of cytosolic Ca^{2+} concentration in various plant species. However, the functions of Ca^{2+} in auxin signaling or responses are poorly understood. SAUR proteins may provide a functional link between Ca^{2+} and auxin responses.

Using recombinant ^{35}S -labeled CaM to screen a maize root cDNA expression library, Yang and Poovaiah (2000) isolated ZmSAUR1

as a CaM-interacting protein that binds to CaM in a calcium-dependent manner. Deletion analyses revealed that the CaM-binding motif is located in the N-terminal region of ZmSAUR1. This study and subsequent studies have identified additional SAUR proteins (maize ZmSAUR2, *Arabidopsis SAUR15* and *SAUR70*, and soybean SAUR10A5) capable of binding CaM (Knauss et al., 2003; Popescu et al., 2007). Our search for potential CaM-binding sites within the 79 *Arabidopsis SAUR* proteins using CALMODULIN TARGET DATABASE (<http://calcium.uhnres.utoronto.ca/ctdb/ctdb/sequence.html>) identified potential CaM-binding motifs in all family members except SAUR4, 6, 10, 18, 30, 33, 40, 41, 52, 57, 58, 67, and 74. To date, however, only SAUR70 has been shown to bind CaM or CaM-related proteins *in planta* (Popescu et al., 2007) and no functional significance has been ascribed to this potential activity of SAUR proteins.

Auxin Transport

Several studies have indicated that SAURs can modulate polar auxin transport. In *Arabidopsis*, *SAUR19* overexpression and *SAUR19/23/24* amiRNA knockdown seedlings conferred increased and decreased basipetal IAA transport, respectively, in hypocotyls (Spartz et al., 2012). Similar findings have been obtained with *SAUR41* and *SAUR63* (Chae et al., 2012; Kong et al., 2013). In contrast, overexpression of *OsSAUR39* in rice resulted in reduced basipetal IAA transport (Kant et al., 2009), again raising the possibility that different SAUR proteins may function antagonistically. The simplest explanation for increased auxin transport is the elevated PM H^+ -ATPase activity resulting from SAUR overexpression. As per the chemiosmotic model for IAA transport (Blakeslee et al., 2005), the increase in apoplastic acidification and the predicted increase in plasma membrane potential associated with SAUR overexpression would be expected to promote IAA transport. However, other mechanisms are also possible, including the aforementioned CaM-binding activity of SAURs, as polar auxin transport is highly dependent on calcium (Vanneste and Friml, 2013). Regardless of the mechanism, the fact that SAUR proteins are capable of modulating IAA transport provides a potential explanation for the diversity of effects, including altered cell expansion, division, and patterning that result from ectopic SAUR expression.

Hormonal Regulation of SAUR Expression - More than Just Auxin

In addition to auxin, brassinosteroids (BR), gibberellins (GA), jasmonate, and abscisic acid (ABA) have been reported to regulate the expression of some SAUR genes, indicating that SAURs likely contribute to other hormone-regulated aspects of plant growth and development. Similar to auxin, BR is a growth-promoting hormone that regulates many physiological and developmental processes (reviewed in Fàbregas and Caño-Delgado, 2014; Wang et al., 2014b). A large body of evidence suggests that the BR and auxin pathways exhibit extensive crosstalk and converge at the transcriptional level to regulate many shared target genes (reviewed in Vert and Chory, 2011; Wang et al., 2012), including many SAUR genes (Goda et al., 2004; Nemhauser et al., 2004). In the BR signaling pathway, the BRI1-EMS-SUPPRESSOR1 (BES1)/BRASSINAZOLE RESISTANT1 (BZR1) family transcription factors regulate gene expression

(He et al., 2005; Yin et al., 2005). Chromatin immunoprecipitation–DNA microarray (ChIP–chip) studies identified many *SAUR* genes as potential direct targets of BZR1 and BES1 (Supplemental Table 2) (Sun et al., 2010; Yu et al., 2011). Indeed, ChIP assays have confirmed that BZR1 and BES1 bind to the *SAUR15* promoter (Yin et al., 2005; Sun et al., 2010; Walcher and Nemhauser, 2012), and BES1 also binds to *SAUR36* and *SAUR59* promoters (Yu et al., 2011). While the precise roles of SAUR proteins in BR action remain unclear, given the well-established role of BR in promoting cell expansion, it seems likely that SAURs are downstream effectors that mediate at least some aspects of BR-mediated expansion growth.

GA is also a growth-promoting hormone that regulates diverse physiological and developmental processes, such as seed germination and stem elongation. The DELLA repressors function as central regulators in the GA signaling pathway, and GA promotes DELLA protein degradation by the 26S proteasome to stimulate growth (reviewed in Claeys et al., 2014; Xu et al., 2014). Consistent with a possible role for SAURs in GA-dependent growth, Bai et al. (2012) recently found that 27 *SAUR* genes were upregulated following a 12-h GA treatment (Supplemental Table 3). This increase in *SAUR* expression is likely the result of enhanced ARF, PIF, and BZR1/BES1 transcription factor binding to *SAUR* promoters following DELLA proteolysis, as DELLAs interact with and inhibit the binding of each of these transcription factors (Feng et al., 2008; de Lucas et al., 2008; Bai et al., 2012; Oh et al., 2014).

A second connection between SAUR proteins and GA-regulated growth is in seed germination. GA promotes germination and RGA-LIKE2 (RGL2) is a major DELLA protein that represses seed germination in *Arabidopsis*. Stamm et al. (2012) reported that RGL2 upregulates *SAUR36*, which is highly expressed in dry and imbibed seeds, and ChIP–qPCR analysis detected RGL2 binding to the *SAUR36* promoter. Consistent with the upregulation of *SAUR36* by RGL2, GA represses *SAUR36* expression (Stamm and Kumar, 2013). Genetic studies support a role for *SAUR36* in regulating seed germination, although curiously, germination of both *saur36* null mutants and *SAUR36* overexpression lines was hypersensitive to paclobutrazol (a GA biosynthesis inhibitor) and ABA treatments. Further research is required to understand the precise role of *SAUR36* in seed germination.

In contrast to the general trend of increased *SAUR* expression elicited by the above growth-promoting hormones, many *SAUR* genes are repressed by the stress-related hormones jasmonate and ABA (Supplemental Table 4) (Nemhauser et al., 2006). ABA is a major regulator of the adaptive responses of plants to stresses, such as drought and high salt, which cause plant growth inhibition (reviewed in Yoshida et al., 2014). Many *SAUR* genes exhibit reduced expression in response to ABA treatment, as well as drought and osmotic stresses (Kodaira et al., 2011). To investigate the mechanisms underlying abiotic stress-regulated growth inhibition, Kodaira et al. (2011) studied two transcriptional repressors AZF1 (ARABIDOPSIS ZINC-FINGER PROTEIN1) and AZF2. Expression of *AZF1* and *AZF2* is induced by ABA, drought, and high salt, and *AZF* overexpression confers growth repression. Transcriptome analyses of DEX-inducible *AZF1* and *AZF2* overexpression plants identified 27

SAUR genes, whose expression is downregulated. These genes included nearly all members of the *SAUR19* and *SAUR63* subfamilies, and gel shift assays demonstrated that recombinant AZF1 and AZF2 proteins could bind to the promoters of *SAUR20* and *SAUR63*, suggesting that at least these *SAUR*s are direct targets of AZF-mediated repression. Consistent with these findings, expression of *SAUR16* and *SAUR63* is increased in the *azf1 azf2* double mutant in response to high salt stress. As several studies have found that the *SAUR19* and *SAUR63* subfamily genes positively regulate cell expansion to promote plant growth (Chae et al., 2012; Spartz et al., 2012, 2014), these findings suggest that AZF-mediated downregulation of *SAUR* expression may contribute to the growth repression characteristic of ABA and abiotic stress treatments. Potentially, the molecular basis for this regulation could be the aforementioned SAUR regulation of PP2C.D phosphatase activity to modulate PM H⁺-ATPase activity. While both auxin treatment and *SAUR19* overexpression promote increases in PM H⁺-ATPase C-terminal Thr⁹⁴⁷ phosphorylation and enzymatic activity, ABA treatment results in reduced Thr⁹⁴⁷ phosphorylation and ATPase activity (Hayashi et al., 2014).

CONCLUSIONS AND FUTURE PERSPECTIVES

Following a protracted lag phase, the functions of the large, highly conserved, and plant-specific *SAUR* gene family are beginning to be elucidated. Data obtained from molecular, genetic, biochemical, and genomic studies have suggested that *SAUR*s are involved in regulating a wide range of cellular, physiological, and developmental processes in response to hormonal and environmental signals. We suggest that *SAUR*s play key roles in integrating hormonal and environmental signals into distinct growth and developmental responses (Figure 3). Based on our current knowledge, this regulation is accomplished largely through the induction or repression of *SAUR* gene transcription. In particular, the *SAUR19* and *SAUR63* subfamilies, both of which have been intimately linked with cell expansion, appear to be especially responsive to hormonal and environmental signals that regulate plant growth. That said, there is strong evidence that *SAUR* activities are also controlled post-transcriptionally, via the regulation of mRNA and protein stability. Determining the extent and mechanisms by which hormonal and environmental signals contribute to these modes of regulation is a crucial area for future research.

Although considerable progress has been made in recent years, we still have only a rudimentary understanding of the functions and molecular mechanisms of *SAUR*-regulated plant growth and development. Much of our current knowledge is based on expression patterns and gain-of-function studies, which, at best, can only suggest potential functions. While *SAUR* loss-of-function studies are complicated by likely extensive genetic redundancy and tight linkage arrangements, modern genome editing techniques such as TALEN and CRISPR/Cas9 nucleases offer potential solutions.

Molecular and biochemical studies are also desperately needed to provide additional mechanistic insight into *SAUR* protein function. The recent demonstration that *SAUR19* inhibits PP2C.D

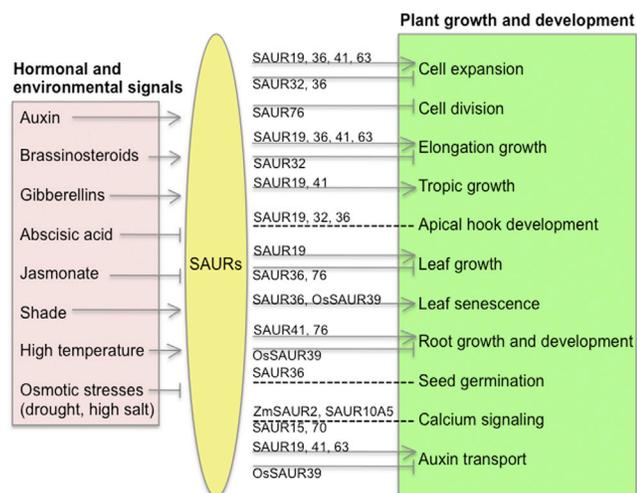


Figure 3. SAURs Integrate Hormonal and Environmental Signals to Regulate Plant Growth and Development.

Arrows and blunted lines indicate positive and negative regulation, respectively. Dashed lines indicate either positive or negative regulation. Hormones and environmental factors confer changes in SAUR expression. Particular SAURs then positively or negatively regulate specific cellular, physiological, and developmental processes. The indicated effects of the various hormones and environmental factors on SAUR expression reflect the overall trend.

phosphatase activity to control PM H⁺-ATPase activity is an exciting first step in this direction (Spartz et al., 2014). And indeed, the crucial role that PM H⁺-ATPases play in establishing electrochemical gradients across the plasma membrane could potentially control, either directly or indirectly, many of the growth responses that appear to be influenced by SAURs. However, the observations that different SAUR proteins exhibit distinct subcellular localizations strongly suggest that PM H⁺-ATPase regulation is only one of several SAUR functions. That said, the possibility that PP2C.D regulation is the primary function of SAUR proteins remains an intriguing prospect. There are 9 PP2C.D family members in *Arabidopsis* and initial work suggests that these proteins are differentially localized (Tovar-Mendez et al., 2014). Thus, a SAUR-PP2C.D code may exist, whereby distinct SAUR-PP2C.D combinations in different tissues and subcellular compartments could modulate the phosphorylation status and activities of many downstream effectors to control diverse aspects of growth and development. Detailed PP2C.D genetic studies, characterization of PP2C.D expression patterns, and the identification of additional PP2C.D substrates will likely be informative in testing this possibility and potentially forging new links with SAUR proteins. In addition, the CaM-binding activity exhibited by several SAUR proteins also warrants additional studies that may lead to the identification of distinct biochemical functions for SAUR proteins.

It now seems apparent that SAUR proteins are crucial regulators of diverse aspects of plant growth and development. Unraveling the molecular networks that link specific hormonal and environmental signals to particular SAUR proteins and cellular/developmental outputs will undoubtedly improve our understanding of SAUR function, and may lead to novel strategies for manipulating

plant growth. We expect many new and exciting findings on this long-neglected family of plant proteins in the years to come.

SUPPLEMENTAL INFORMATION

Supplemental Information is available at *Molecular Plant Online*.

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