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The *Arabidopsis* bZIP transcription factor family — an update

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The basic (region) leucine zippers (bZIPs) are evolutionarily conserved transcription factors in eukaryotic organisms. Here, we have updated the classification of the *Arabidopsis thaliana* bZIP-family, comprising 78 members, which have been assorted into 13 groups. *Arabidopsis* bZIPs are involved in a plethora of functions related to plant development, environmental signalling and stress response. Based on the classification, we have highlighted functional and regulatory aspects of selected well-studied bZIPs, which may serve as prototypic examples for the particular groups.

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Organisation of the *Arabidopsis* bZIP-family

Sequencing of various eukaryotic genomes highlighted the importance of transcriptional control as approximately 8% of the protein-encoding capacity is attributed to transcription factors (TFs) [1••]. In general, TFs are classified in families according to their conserved DNA-binding domains. Currently, around 100 TF families have been defined for the model plant *Arabidopsis* [1••]. Here, we will focus on basic (region) leucine zipper (bZIP) TFs, which are characterized by a basic DNA-binding region and an adjacent so-called leucine zipper, enabling bZIP dimerisation (for details see Figure 1) [2,3]. As bZIPs generally perform as dimers, heterodimerisation results in an enormous regulatory flexibility, for example, with respect to target-side selection or protein interactions [4••].

With respect to numbers, the bZIP-family size differs considerably with 53 in humans [4••], 78 in *Arabidopsis*, 92 in rice [5], 125 in maize and 247 in rapeseed [6]. Whole genome, chromosome and tandem duplications appear to be important mechanisms in shaping bZIP families, particularly in plants [6]. Phylogenetic studies could trace back bZIPs to early ancestors in green algae [5]. In line with this finding, plant bZIPs have been associated with a plethora of functions, supporting a recruitment early in plant evolution [7,5].

Jakoby *et al.* [7] provided the first near-complete genome-wide update on the *Arabidopsis* bZIP family, which was further revised and discussed in an evolutionary context [5]. A grouping based on homology of the basic region and additional conserved motifs has been proposed, which is somewhat subjective, but has found wide acceptance by the community. Nevertheless, after 15 years it is reasonable to summarise new insights and update this classification as outlined in the phylogenetic tree in Figure 2. Overall, data mining retrieved 78 bZIP members, adding bZIP76-bZIP79, which were in part previously described [5]. Moreover, we excluded one pseudogene (bZIP73). Following the previous nomenclature [7], these bZIPs were classified into 13 groups (designated A-M).

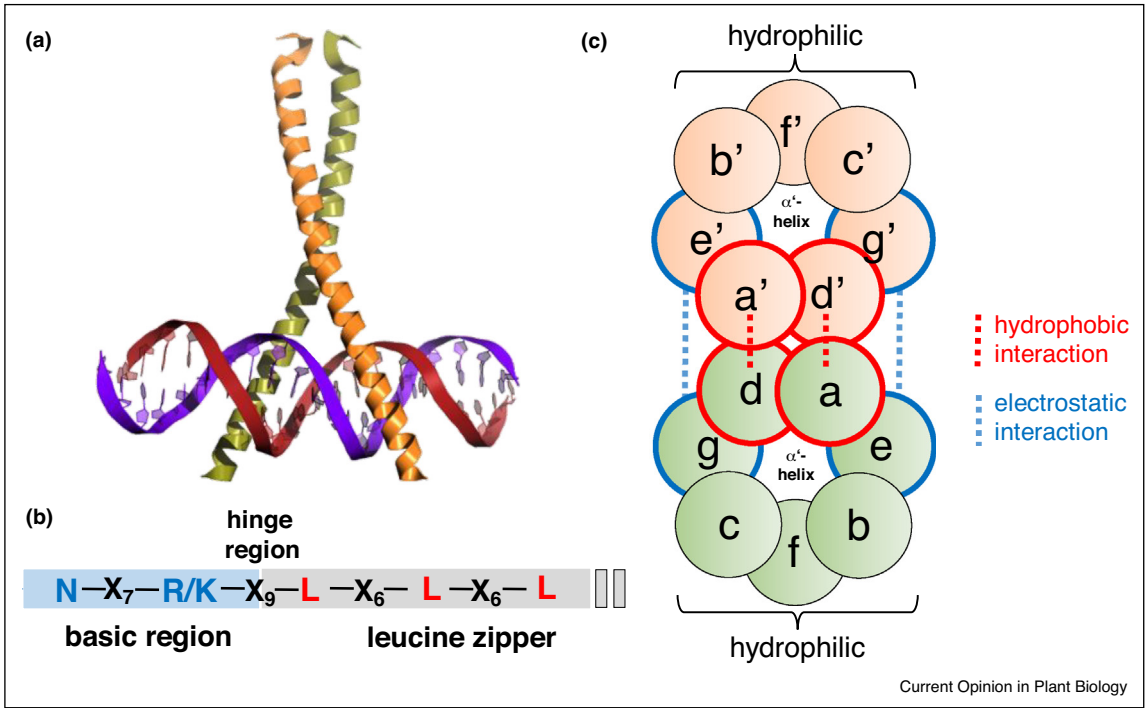
As an enormous amount of functional data has been collected on *Arabidopsis* bZIPs, an exhaustive overview is not feasible. Thus, this review will focus on selected, well-studied bZIPs, which may stand as prototypic examples for functional and regulatory aspects or display group-specific properties.

Function of *Arabidopsis* bZIPs

Group D

Group D comprises the so-called TGA factors (TGAs), named according to their cognate TGACG DNA-binding motif. From the historical perspective, tobacco TGA1a was the first TF isolated from plants, binding to the viral *activation sequence-1* (*as-1*) motif. TGAs are characterized by a short zipper domain consisting of three repeats, a rather conserved C-terminus including two Q-rich domains, and a more variable N-terminus. The ten *Arabidopsis* TGAs are classified into five clades as recently reviewed [8].

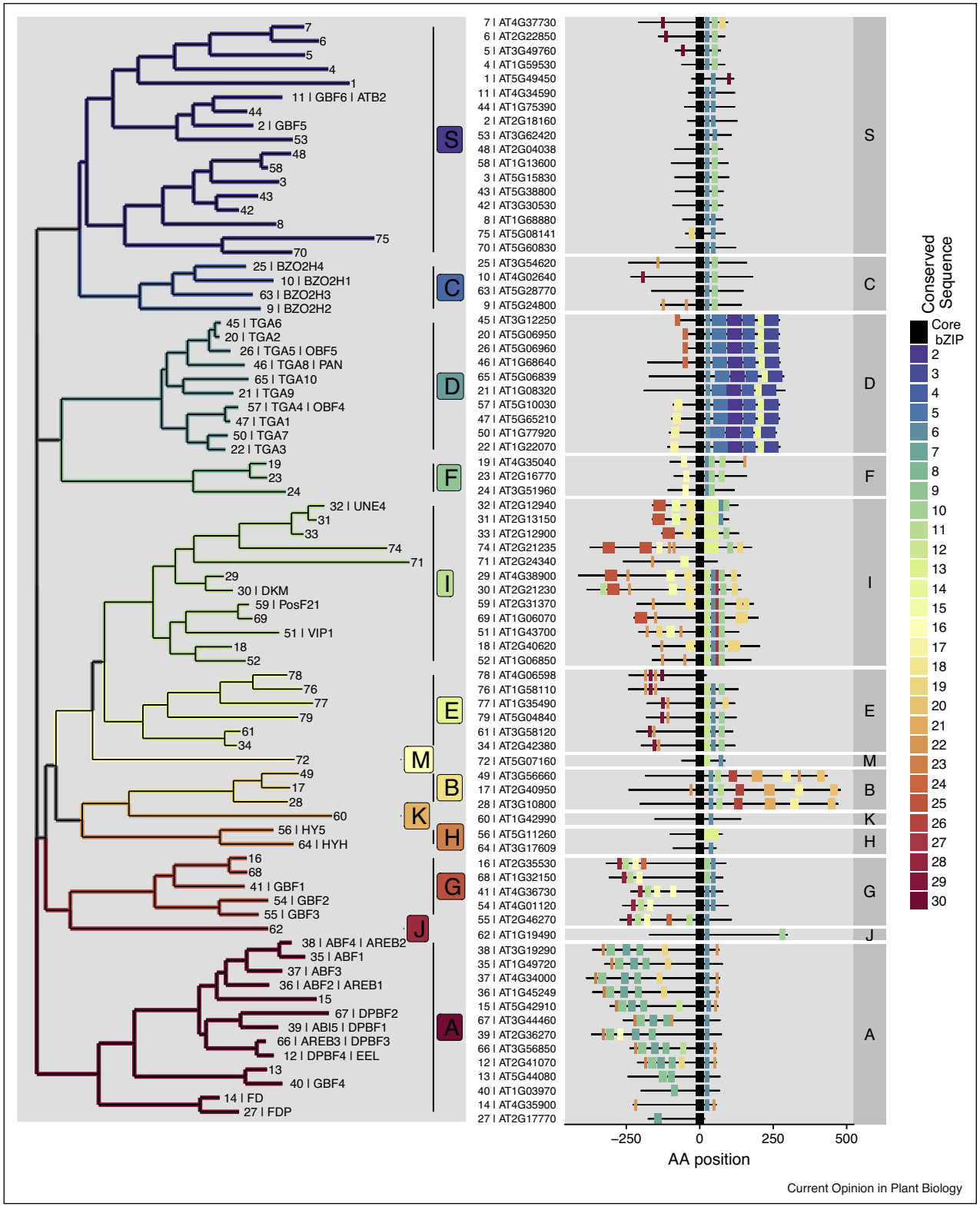
Figure 1



Structural aspects of bZIP transcription factors [2,5,7,129]. **(a)** Structural model of a dimer of the bZIP domains, exemplified for the prototypic human TF, CREB (https://commons.wikimedia.org). According to the 'scissors-grip-model' [3], each bZIP domain forms a continuous α-helical structure and dimerises via the so-called leucine zipper (ZIP). Like a pair of scissors, the bZIP proteins contact the major groove of double-stranded DNA. **(b)** Schematic representation of the bZIP domain, consisting of a basic DNA-binding region (blue) and the adjacent ZIP domain (grey). The ZIP domain consists of heptad repeats of leucine (L) or related hydrophobic aa. The number of repeats differs considerably from three (group D), to more than eight (groups C and S). Structural data demonstrate that only five aa of each basic domain facilitate the contact to DNA. Here, an invariant N-X_7-R/K motif with asparagine (N) and basic (R/K) residues with exact spacing is found. As an exception, group E bZIP76-bZIP79 harbour a deletion and hence lack the N residue. In the basic region of the group J member bZIP62, R/K is exchanged by I. Yet, a possible functional relevance of these alterations has not been analysed. The bZIP target sequences often consist of an ACGT-core with adjacent nucleotides, forming palindromic or pseudo-palindromic hexamers. With respect to the 3′ flanking nucleotide, these boxes are designated palindromic G-boxes (CACGTG), C-boxes (GACGTC) etc. Nevertheless, some bZIPs perform binding to non-ACGT-motifs [79,80,81••,102•]. Heterodimerisation may account for a more complex target-site selection. **(c)** Schematic view on the bZIP dimerisation interphase. The amphipathic C-terminal zipper domain dimerises to form coiled-coils. A heptameric repeat is formed by seven aa, arranged in two turns and labelled a–g. As depicted in the scheme, the positions a–d and a′–d′ are hydrophobic (typically L) and expose their site-chains to form a hydrophobic face facilitating intermolecular interactions. Charged aa in positions g and e often form crosswise electrostatic interactions between helices (g–e′ and g′–e). Deduced from these interactions, structure-based rules have been used for predictions of heterodimerisation, proposing that preferentially homodimers or dimers within related group-members (quasi-homodimers) are formed by *Arabidopsis* bZIPs [129]. Nevertheless, non-predicted heterodimerisation patterns between groups have been demonstrated, for example, between E and I, C and S_1 or H and G [30,59,80].

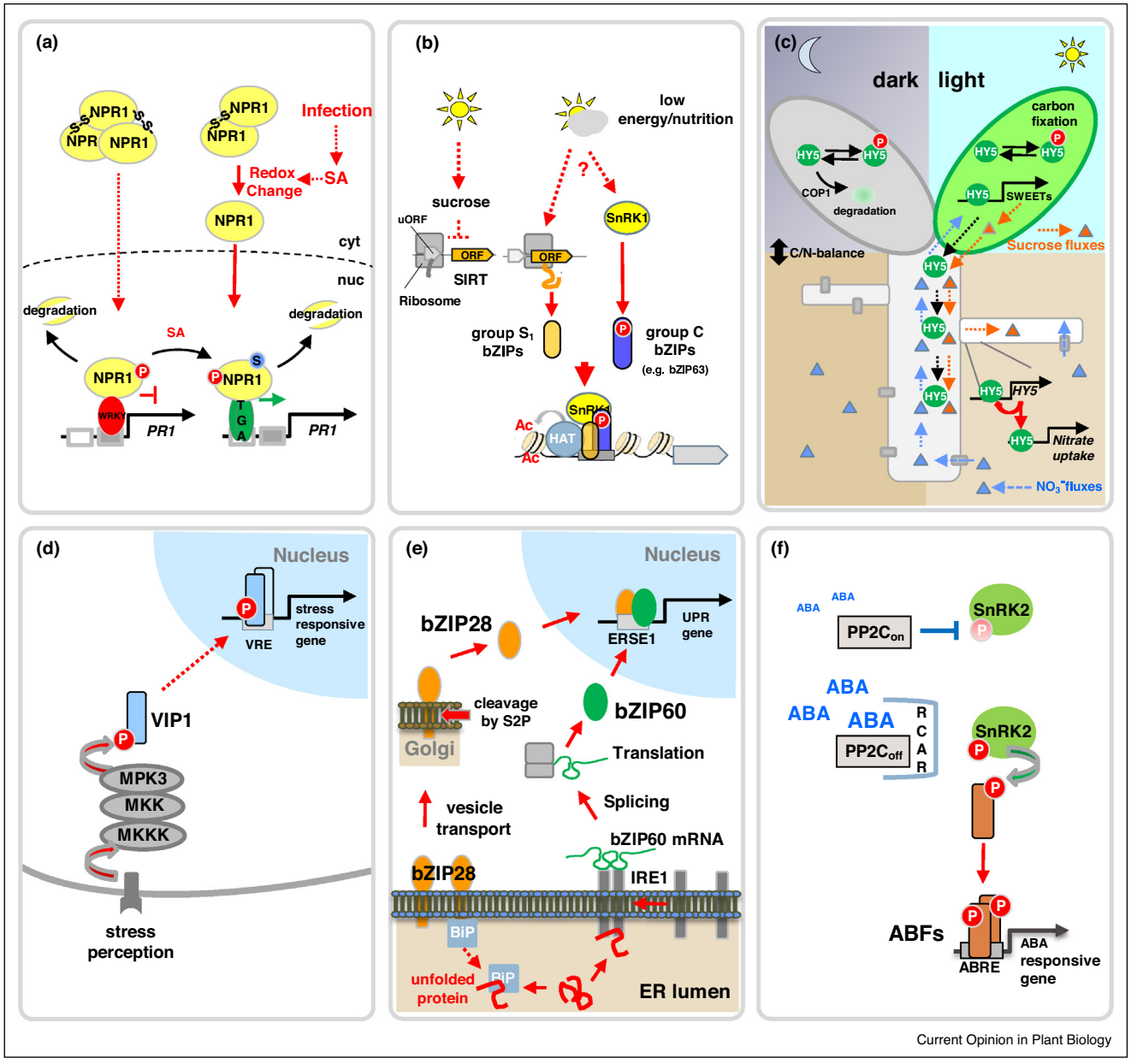
The clade II factors TGA2, TGA5, TGA6 redundantly act as crucial transcriptional regulators in systemic acquired resistance (SAR). SAR is part of the plants' immune system and implements plant-wide, broad-spectrum pathogen immunity that is triggered by a prior local infection [9]. Importantly, clade II and several other TGAs physically interact with nonexpressor of pr genes1 (NPR1), which represents a major regulator of plant pathogen responses [9]. In the non-induced state, NPR1 resides as an inactive multimeric complex in the cytosol. However, upon infection, local redox changes mediated by accumulation of the important defence-related hormone salicylic acid (SA) initiate the disaggregation of NPR1 complexes, enabling translocation of monomers to the nucleus [10] (Figure 3a). Expression of the classical SA-induced marker gene *pathogenesis related1* (*PR1*) depends on class II TGAs and is impaired in the *npr1* mutant [8,9]. Although it was generally accepted, that NPR1 acts as a transcriptional co-activator of TGAs, a mechanistic gene regulatory model was presented only very recently for the clade III factor TGA3. This model proposes the formation of dynamic promoter TF-NPR1 complexes, controlled by multiple post-translational NPR1 modifications [11••,10]. Both *as-1* and W-boxes are well-described *PR1* promoter *cis*-elements for TGAs and WRKYs, respectively [12]. During the

Figure 2



Organisation of the *Arabidopsis thaliana* bZIP-family. All sequences encoding a bZIP domain were retrieved from genome resources as outlined below. A phylogenetic tree (left) was calculated and grouping of bZIPs was performed based on similarities in the bZIP and other conserved motifs (right). We identified 78 bZIP proteins (bZIP1 to bZIP79), following the generic naming according to [7]. As the previously annotated bZIP73 (group I) turned out to be a pseudogene, it was excluded from the family. The classification into 10 groups (A-I and S) [7] was further extended by

Figure 3



Schematic representation of selected signalling pathways employing bZIP TFs modified according to [11••,37••,44,61••,89]. For details see text. P: phosphorylation; S: sumoylation; Ac: acetylation; HAT: histone acetyl-transferase.

(Figure 2 Legend Continued) introducing the single-member groups M, K and J, as these bZIPs differ considerably in their motif structure. Besides the generic names, At identifiers and trivial names are given, if generally accepted in the community. Bioinformatic procedures: bZIP protein sequences of the longest splice variant of each gene were obtained from EnsemblPlants Biomart (plants.ensembl.org/biomart/martview/). HMM profiles were obtained from pfam (http://pfam.xfam.org/family/PF00170#tabview=tab6 and http://pfam.xfam.org/family/PF07716#tabview=tab6). These HMM profiles were used to reassure the bZIP motif was present in the obtained genes. Genes were aligned applying the Einsi function of MAFFT, then alignments were trimmed using TRimAl, at 30% conserved AA. (trimal -in Bzip_mafft_einsi_input.txt -out Bzip_trimal03_output .txt -gt 0.3). Iqtree was used for model selection, after which the best model LG+R7 was selected to calculate the phylogenetic tree (settings: iqtree-omp -s Bzip_input -m LG+R7 -alrt 1000 -nt AUTO). The tree figure was made using R and package 'ggtree2'. Conserved sequences were determined using MEME (http://meme-suite.org/tools/meme), generating the 30 most significantly conserved sequences (in MEME and MAST output) in the full length bZIP sequences. Conserved sequences found by MAST were visualized using R and 'ggplot2' (for details see Supplementary Figure S1).

non-induced state, phosphorylated NPR1 interacts with WRKY70, forming a repressive complex at a W-box. Upon SA-induction, this complex dissociates due to a change in the NPR1 phosphorylation pattern and subsequent sumoylation. Post-translationally modified NPR1 now switches partners, and forms a transient TGA3-NPR1 complex on the *as-1* element to activate *PR1* transcription. Whether this highly dynamic mechanism generally accounts for SA-induced genes and also for clade II TGAs, needs to be established.

In a simplified view, plants rely on SA-dependent defences when they encounter biotrophic pathogens, whereas the hormones jasmonic acid (JA) and ethylene (ET) coordinate defences against necrotrophs. Interestingly, clade II TGAs are also required for the activation of the later pathway [13•] by supporting ethylene-insensitive3 (EIN3)-mediated activation of *ORA59*, which encodes a master regulator of the JA/ET pathway [14]. Since SA is well-known to repress JA/ET signalling in a manner that requires clade II TGAs [15], these TFs might serve as a point of pathway integration. TGAs interact with land plant-specific glutaredoxins (called ROXYs in *Arabidopsis*) [16]. Most of the ROXYs interact with the transcriptional co-repressor TOPLESS [17]. Specific members of the ROXY family are induced by SA and since they negatively regulate the JA/ET pathway when ectopically expressed, they are candidates to mediate the negative effect of SA on the JA/ET pathway [16].

Besides their involvement in defence responses, clade II TGAs have also been implicated in controlling detoxification processes [18]. In this regard, the TGAs regulate a distinct set of detoxification genes by recruiting the GRAS co-activator scarecrow-like14 (SCL14) [19]. This NPR1-independent response mediates plant tolerance towards various toxic endogenous and exogenous compounds and is also repressed by specific ROXYs [20].

Among the clade III members, only the previously mentioned TGA3 has been analysed in detail. TGA3 is involved in basal pathogen resistance and in mediating hormonal cross-talk between SA and cytokinin [21]. Cytokinin signalling leads to phosphorylation of the response regulator ARR2, which together with TGA3 binds and activates SA-dependent promoters. Moreover, recent work positioned TGA3 in the context of heavy metal detoxification [22].

TGA1 and TGA4 (clade I) are not involved in establishing SAR, but control basal resistance [8]. Although TGA1 physically interacts with NPR1, clade I-mediated gene regulation is largely NPR1-independent [23]. TGA1 and TGA4 induce apoplastic defences and ER stress responses [24] and directly control transcription of *SARD1* and *CBP60g*, both important TFs involved in biosynthesis of SA and further signalling molecules in pathogen defence [25].

A previously unexpected function in root nitrate uptake and nitrate responses has recently been established for clade I TGAs [26]. Interestingly, *TGA1/4* transcripts accumulate in a nitrate dependent manner and most of the genes differentially expressed in wild-type compared to the *tga1tga4* double mutant are nitrate responsive. Moreover, direct binding to promoters of nitrate uptake transporter genes, as well as clade I dependent alterations in root architecture has been observed [26,27].

Whereas limited data on developmental functions of clade IV (TGA9, TGA10) are available [8], the single clade V member TGA8/PAN (PERIANTHIA) controls the formation of floral organ primordia [28]. In the mutant, the outer floral whorls are transformed from a tetramerous to pentamerous organisation. From the evolutionary perspective it is interesting to note that PAN interacts not only with ROXYs, but also with the NPR1-like proteins BOP1/2, indicating mechanistic conservation in TGA regulatory patterns [8].

Taken together, most of the group D bZIPs increase the plant's ability to survive pathogen and xenobiotic stress. However, some members adopted developmental functions and (partially) exploit conserved interaction partners to control their activities.

Groups C and S

Group S comprises the largest bZIP-cluster of 17, generally intron-less genes, which encode small TF proteins of approximately 20 kD in size. Yet, only the group S_1 members (bZIP1, bZIP2, bZIP11, bZIP44, bZIP53) have been characterized, in detail. Based on the finding that all four group C-bZIPs (bZIP9, bZIP10, bZIP25, bZIP63) preferentially heterodimerise with group S_1 members and on results demonstrating that these heterodimers [29,30] are functionally interlinked in plant starvation signalling, they are referred to as the C/S_1-bZIP network, as recently reviewed [31].

The proposed function of this network in plant energy management is based on its activation by several energy/nutrient-dependent signalling inputs: first, due to conserved upstream open reading frames (uORFs), all S_1-bZIPs share a unique translational regulation. Encoded by the uORF, a nascent peptide is produced leading to ribosome stalling and inhibition of main ORF-translation in a sucrose-dependent manner [32,34] (Figure 3b). Remarkably, sucrose-induced repression of translation (SIRT) provides a mechanism by which cellular nutrient/energy availability can be relayed into gene expression. Second, with respect to group C, bZIP63 has been demonstrated to be phosphorylated by SNF1-related kinase1 (SnRK1), an evolutionarily conserved major

metabolic kinase responding to energy and nutritional starvation [35••]. SnRK1 phosphorylates bZIP63 *in vivo* and thereby, enhances its ability to heterodimerise with S_1-bZIPs [36••]. Moreover, a transiently formed, ternary C/S_1-bZIP/SnRK1 complex was found to be recruited to target promoters, facilitating histone acetylation and gene activation [36••,37••,38]. In a genome-wide view, a distinct subset of the SnRK1 and starvation-induced genes depends on S_1-bZIP downstream activity. Particularly striking, a co-ordinated activation of a pathway leading to catabolism of branched-chain amino acids has been observed, providing ATP to sustain plant survival during stress [37••]. Along this line, overexpression of bZIP11/ATB2 mimics responses observed in carbon-starved plants [39]. Third, transcription of *bZIP1* was found to be repressed by the presence of the metabolic signal glucose, depending on the glucose-sensor hexokinase1 (HXK1) [40]. Finally, bZIP1 has been implicated in the integration of darkness-induced starvation and NO_3^- signalling [41••]. Taken together, based on their heterodimerisation properties the C/S_1-bZIP network has been proposed to function as signalling hub to facilitate metabolic reprogramming upon energy/nutritional starvation leading to metabolic adaptation and survival upon stress.

Energy homeostasis is critical to all phases of plant life, which might explain why C/S_1-bZIPs have been described to control a plethora of energy-demanding processes both, in stress and development. For example S_1-bZIPs related to bZIP11 control auxin-dependent primary root growth [38,42•] and similar to OPAQUE2 in maize, the orthologous group C members bZIP10 and bZIP25 dimerise with bZIP53 to activate seed maturation genes [43]. Moreover, bZIP1 and its partners control metabolic reprogramming upon salt stress in roots [44] and bZIP10 is involved in pathogen defence responses, regulated by a cytosolic anchor protein [45••].

Functional analyses of the C/S_1-network is complicated by partial redundancy [37••,46], and complex transcriptional, translational and post-translational regulation [31]. Synergistic gene activation by C/S_1-heterodimers has been demonstrated [29,43] however, nuclear abundance of particular bZIPs and their specific heterodimerisation properties [30] need to be studied to unravel transcriptional control. Gene activation by direct recruitment of the histone acetylation machinery has been demonstrated for several, but not all C-bZIPs and S_1-bZIPs [39]. A striking regulatory property of bZIP1 has recently been described as a 'hit-and-run' mechanism in protoplasts. Transient binding of the TF to a promoter ('hit') and its subsequent mobilization to a second promoter ('run') leads to sustainable alterations in gene expression at the primary binding site [41••]. Studies unravelling the mechanism in planta are however required.

Taken together, a significant advance has been made to link C/S_1-signalling to the plant's low energy management system. The importance of these TFs is highlighted by their evolutionary conservation, as C/S-bZIPs can be traced back to an algal ancestor and a functional SIRT-regulation was demonstrated in gymnosperms [33]. Nevertheless, redundant and/or specific functions of particular C-bZIPs and S_1-bZIPs or their impact on the integration of non-energy related stimuli remain to be elucidated.

Group H

Group H consists of only two members, elongated hypocotyl5 (HY5) and HY5 homolog (HYH) and has recently been reviewed [47]. Initially, *hy5* was identified in a mutant screen, showing elongated hypocotyls under various light conditions. Meanwhile, the functional relevance of HY5 in numerous developmental processes is well-established, such as promoting photomorphogenesis, chloroplast development and pigment accumulation downstream of phytochromes, cryptochromes and UV-B photoreceptors [47,48]. HY5 is also implicated in multiple hormone signalling pathways, related to abscisic acid (ABA), auxin [49•,50], cytokinin, gibberellin, brassinosteroids [51], ET [52] or strigolactones [47]. Moreover, HY5 participates in the shade avoidance response [53] and it translates red:far-red light quality changes that are sensed in leaves into auxin-controlled lateral root development [49•]. In short, HY5 performs as an evolutionarily conserved master regulator in plants, which co-ordinates light, environmental and developmental signalling [47].

HY5 activity is particularly controlled by post-translational mechanisms. The central negative regulator of photomorphogenesis, the E3 ubiquitin-protein ligase constitutive photomorphogenic1 (COP1) was found to interact with HY5, resulting in its ubiquitination and degradation in darkness (Figure 3c) [54]. HY5 protein appears in a phosphorylated and a non-phosphorylated pool, but only the latter is physiologically active and binds DNA. In contrast, phosphorylated HY5 was demonstrated to be insensitive to COP1-dependent degradation and hence provides a pool of TF proteins, which can be rapidly activated by de-phosphorylation [47].

Thousands of genes were found to be directly controlled by HY5 [55,56] via binding to ACGT-core and non-ACGT-motifs. In particular, genes implicated in the circadian clock, chlorophyll biosynthesis, light and hormone signalling as well as distinct miRNAs are under control of HY5 [47,56]. Typical mechanisms which have been described for HY5-mediated gene regulation are competition for *cis*-elements and interaction with other TFs. For example, integration of light, temperature and reactive oxygen species (ROS) signalling has been studied by antagonistic binding of HY5 and phytochrome interacting factor (PIF) bHLH factors (basic-helix-loop-helix) to G-boxes (CACGTG) [57]. Physical TF interactions have been described for MYBs, calmodulin7 (CAM7) or group-G bZIPs [47].

The HY5 zipper structure has been disclosed and exhibits significant differences to other bZIP proteins [58]. Whereas homodimers and heterodimers were described between HY5 and HYH, dimerisation properties reach out to groups G, C and S_1 [59]. HY5 and HYH display partly overlapping functions, for example, with respect to hypocotyl and lateral root growth or pigment accumulation [50,47]. Nevertheless, the *hyh* mutant does not show the pronounced *hy5* phenotype. Importantly, HY5 and HYH activate *HY5* transcription, suggesting an auto-regulatory circuit and supporting the view that protein availability and interplay is critical for HY5/HYH function [50,60,47].

Recent work supported a pivotal function of HY5 in controlling C/N-homeostasis and root architecture [61••]. Indeed, shoot photosynthetic C-fixation and acquisition of N via the roots have to be tightly coordinated to sustain optimal plant growth (Figure 3c). In photosynthetic leaves, HY5 protein is stabilized and supports sucrose export to roots by activating transcription of *SWEET*-facilitator genes. As supported by grafting experiments and tracking of mobile HY5-GFP fusions, HY5 was proposed to be translocated as a phloem-mobile TF from the shoot to the root system. In roots, HY5 auto-activates its own transcription, and directly binds and activates the *NRT2.1* gene, encoding a high-affinity nitrate uptake transporter. Subsequently, nitrate is transported from the root to the shoot to meet demands of the growing tissues.

Groups G and J

Group G comprises five members from which only G-box-binding factor1 (GBF1) has been studied in detail. Although related to other group G members, the so far uncharacterized bZIP62 was placed in group J, as it misses several conserved group G motifs.

GBF1 is a long-known, negative regulator of blue-light dependent hypocotyl expansion and thus, performs largely antagonistically to HY5 and HYH [62]. In fact, whereas HY5 and its homolog HYH are degraded in darkness in a COP1-dependent manner, GBF1 is addressed by a different degradation mechanism. In contrast to HY5, both GBF1 and HYH were found to act synergistically in chlorophyll and anthocyanin accumulation [62]. A mechanistic insight arises from studies on GBF1 dimerisation properties, indicating that gene regulation largely depends on the interaction partner [59]. Whereas HY5 homodimers activate G-box controlled genes, GBF1-HY5 heterodimers decrease their transcription and GBF1-HYH heterodimers do not bind or regulate transcription at all [63]. Moreover, genome-wide binding studies of overexpressed GBF1 in a *hy5* or *hyh* background demonstrate that HY5 is almost indispensable for GBF1 binding, whereas HYH serves more specific functions [64]. Moreover, GBF1 interacts with the bHLH master regulator MYC2 and the CAM7-TF [62,65].

Additional important functions of GBF1 have been established in promoting lateral root development [65] and natural senescence [66•]. At bolting time and the onset of senescence, GBF1 binds to the *catalase2* (*cat2*) promoter to repress transcription. By reducing the H_2O_2-scavenging activity of cat2, GBF1 triggers ROS accumulation, which is proposed to function as a signal orchestrating the senescence program. Similarly, GBF1 effects the SA-dependent pathogen defence, presumably by enhancing ROS-dependent hypersensitive cell-death and the activation of the well-known defence regulator *phytoalexin deficient 4* (*PAD4*) [67].

As GBF1 is usually not regulated on transcriptional level, post-transcriptional regulation of GBF1 is assumed. Indeed, redox control of GBF1 [68,69] and *in vitro* phosphorylation to alter DNA binding properties [70] have been demonstrated. However, *in vivo* studies are required to substantiate these mechanisms. Moreover, the individual functions of other group G members, their heterodimerisation properties and the nuclear translocation of the partially cytosolic bZIP68 need to be addressed [59].

Group I

Among the 12 group I members, VirE2-interacting protein1 (VIP1; bZIP51) has attracted attention as a putative host factor in Agrobacterium-mediated T-DNA transfer [71]. Agrobacterium VirE2 is well-known as a single-stranded DNA-binding protein, which protects the T-DNA strand while it is mobilized to the plant nucleus. Via protein interaction, VIP1 was shown to bridge between VirE2 and the nuclear importin α [71,72]. This observation led to the 'Trojan horse hypothesis', proposing that Agrobacterium is hitchhiking VIP1 to transport the T-DNA strand to the plant nucleus [73••]. However, a recent study questions the impact of this mechanism both with respect to subcellular localisation of VirE2 and Agrobacterium-mediated transformation [74].

Work by Djamei *et al.* [73••] analysed the mechanistic properties of the nuclear import of VIP1, establishing a cascade in which pathogen-activated mitogen-activated protein kinase3 (MPK3), results in VIP1 phosphorylation, which in turn triggers its translocation to the nucleus (Figure 3d). In here, VIP1 controls stress-related genes by binding to VIP1 response elements (VRE; ACNGCT) [75,76]. However, ensuing studies support a more pleiotropic function of VIP1, such as in low sulphur tolerance [77] or osmosensory signalling [78,79]. Indeed, nuclear translocation was also found to be driven by hypo-osmotic stress whereas the impact of particular phosphorylation sites are still a matter of debate [80,78]. In line with the proposed function, key genes involved in ABA inactivation have been found to be activated by VIP1, targeted to an AGCTGT/G-motif [79,80]. Genome-wide *in vitro* binding studies propose a CAGCT-element for group I

factors, which resembles data from several studies and clearly harbours no ACGT-core [81••].

As single *vip1* mutants are not impaired in the dehydration/rehydration response [79], functional redundancy between related group I members was proposed. Accordingly, stimulated nuclear translocation and heterodimerisation were demonstrated for VIP1, bZIP59, bZIP69, bZIP29, bZIP30 and bZIP52 [80]. Work on the VIP1 homolog bZIP29 further broadens the view, as members of the sub-group of VIP1-related TFs have also been identified as VIP1 protein interaction partners [82]. To address the issue of redundancy, a dominant negative bZIP29:SRDX repressor was expressed under the native promoter, resulting in developmental alterations concerning leaf cell number and root meristem function. Accordingly, bZIP29 was found to be expressed in proliferative tissues and ChIPseq and transcriptome experiments support a function in cell cycle control and cell wall organisation [82]. Moreover, bZIP59 (PosF21) has been defined to function in auxin-induced callus formation and plant regeneration [83] and bZIP30 (DKM, DRINK ME) and bZIP18 have been implicated in female and pollen reproductive organ development, respectively [84,85].

Classical studies on group I orthologues in *Arabidopsis* and other plant species have proposed a function in vascular development [86]. Although only a subset of group I bZIPs has been analysed yet, a more complex picture emerges, linking group I bZIPs to stress response, cell-cycle regulation and various developmental aspects.

Groups E and M

In addition to the previously classified group E-members bZIP34 and bZIP61 [7], we extended this group by bZIP76-79. Whereas these bZIPs share group E-specific motifs, this is not the case for bZIP72, which thus was placed in the single-member group M. It needs to be noted that the highly conserved Asn (Figure 1b) is missing in the basic region of the novel group E members. Moreover, bZIP78 stands out, as it harbours a truncated zipper domain. The functional implications of these alterations with respect to DNA binding and dimerisation need to be analysed.

Yet, only bZIP34 has been partially characterized by linking it to pollen germination and pollen tube growth [87]. Strikingly, bZIP34 and bZIP61 are unable to form homodimers, due to the presence of a proline residue, which breaks the α-helical structure of the zipper [88]. In fact, a mutant approach exchanging the proline to alanine, results in homodimers binding a G-box. Moreover, heterodimerisation with VIP1 related bZIPs (group I) or bZIP43 (group S) has been demonstrated for bZIP34 and bZIP61 [82,84,88], proposing a functional link between these groups.

Groups B and K

The three members of group B (bZIP17, bZIP28, bZIP49) and the one group K member (bZIP60) perform as important regulators of the evolutionarily conserved endoplasmic reticulum (ER) stress response [89]. Proper folding of proteins in the ER lumen is crucial for survival and is facilitated by chaperones such as binding proteins (BiPs). Upon adverse environmental conditions, the load of misfolded proteins exceeds the cellular protein folding capacity leading to their degradation, referred to as unfolded protein response (UPR). In contrast to three ER-stress pathways identified in mammals, only two were found to be conserved in plants, executed by B-bZIP and K-bZIP, respectively. Although involved in a related functional aspect, their domain structure, the evolutionary relationship and distinct mechanistic differences support the separation in two groups.

The mechanisms of bZIP activation in UPR are particularly striking (Figure 3e). Group B bZIP28 is tethered to the ER membrane, facing via its N-terminus to the cytosol. In the ER-lumen, the C-terminus is covered by BiPs, which are allocated to unfolded proteins during ER-stress. Subsequently, membrane bound bZIP28 is transported via vesicles to the Golgi. Here, the N-terminal part of bZIP28 is cleaved off, releasing the truncated active TF, which relocates to the nucleus [90,89]. A sequential activity of S1P and S2P proteases has been proposed however, recent studies do not support participation of S1P [90]. In the nucleus bZIP28 binds promoters harbouring the ER stress–response element 1 (ERSE1), composed of two sub-elements (CCAAT-N10-CACG)[91]. The CACG consensus is recognized by a bZIP dimer and the adjacent CCAAT-box is bound by heterotrimeric nuclear factor Y (NF-Y-TFs). Indeed, a larger TF-complex has been proposed to activate ER stress genes [91]. Although most studies focus on bZIP28, structural similarities support an analogous mechanism of activation for the other group B members [89,92,93]. To evaluate the biotechnological potential, transgenic plants overexpressing bZIP17 under a stress-inducible promoter were constructed, which display enhanced tolerance to salt treatment. However, constitutive activation of the ER stress pathway leads to significant growth retardation [94].

The second, evolutionary more ancient pathway of ER stress response [89] involves the dual-functioning kinase/ribonuclease inositol-requiring enzyme 1 (IRE1) and group K bZIP60, which when inactive was also found to be tethered to the ER via a trans-membrane domain [89] (Figure 3e). Similarly to group B proteins, a truncated bZIP60 was found to be the active form, which is mobilized to the nucleus to regulate ER stress-genes [95,96]. However, maturation of bZIP60 does not rely on proteolysis. In fact, the ER membrane localised IRE1 dimerises in response to ER stress, enabling its ribonuclease activity to excise a 23 bp fragment of the bZIP60

mRNA. This splicing event leads to a frame-shift, which eliminates the trans-membrane domain and fuses a nuclear-localisation sequence to the mature bZIP60 [97•,98,99].

Interestingly, both ER-stress pathways may converge on distinct target gene sets, as bZIP60 and bZIP28/bZIP17 were found to heterodimerise [91,93]. Moreover, recent studies focus on cell survival or cell death [100] and cross-talk between UPR and light signalling facilitated by HY5 and bZIP28 [101].

Group F

Group F consists of three members (bZIP19, bZIP23 and bZIP24) from which bZIP19 and bZIP-23 redundantly control genes that encode for Zn transporters. By these means, they are proposed to facilitate plant adaptation to Zn-limiting conditions [102•,103].

Moreover, group F-bZIPs bind palindromic zinc deficiency response elements (ZDREs; RTGTCGACAY) *in vitro*, which do not contain an ACGT-core motif [102•]. Phylogenetic studies reveal a conservation of these TFs and their binding motifs in transporter gene families within land plants [104]. The third member (bZIP24) was not found to regulate genes in Zn homeostasis, but was proposed to act as a negative regulator in the salt-stress response [105,106].

Group A

Group A consists of 13 members, thereby representing the second biggest cluster, which can be further classified in four distinct subgroups.

The prominent subgroup of abscisic acid responsive element binding factors (ABFs) contains the highly related ABF1–4 that have been found to act at the core of ABA signalling in a partially redundant manner [107]. Being transcriptionally induced by abiotic stresses that impair water availability, such as cold, salinity or drought and post-transcriptionally activated by ABA signalling these factors implement adaptive responses to counteract water deficit in vegetative tissues [107]. For example, in response to drought, they participate in stomatal closure [108] and reprogram metabolism to ensure accumulation of protective osmolytes or directly induce expression of late embryogenesis abundant (LEA) genes to protect cells from dehydration [109,107]. Applying reverse genetic and molecular approaches the upstream ABA signalling components of ABFs were identified [110••,111] (Figure 3f). In short, members of the clade A serine/threonine protein phosphatases 2Cs (PP2Cs) were found to negatively control ABA signalling by inactivating the positively acting SnRK2.2, SnRK2.3 and SnRK2.6 (SNF1-related kinases 2) via dephosphorylation. Upon ABA perception by pyrabactin resistance proteins/PYR-like proteins/regulatory component of ABA receptor (PYR/PYL/RCAR) co-receptors, PP2Cs are sequestered, thereby preventing their interaction with SnRK2 kinases [112••,111,113]. Predominantly activated by auto-phosphorylation, SnRK2s directly phosphorylate ABFs to strongly enhance their transactivation properties [114,109]. Via direct binding to ABA responsive element (ABRE; ACGTGG/TC) *cis*-elements, ABFs control expression of their stress-responsive target genes [109,115]. An interplay of ABFs and C/S_1 bZIPs in controlling gene regulation in salt-treated roots has been described, but requires further analyses [44].

Besides ABFs also individual members of the closely related DPBF subgroup (DPBF1-4; DC3 promoter binding factors), have been found to be central for ABA signalling, especially in the late phase of seed development [116]. In particular, ABA insensitive 5/DPBF1 (ABI5) has been extensively characterized with respect to its function in ABA-dependent seed maturation and seed germination [117] and might be prototypic for other DPBFs, such as enhanced EM levels/DPBF4 (EEL), which also shows a seed specific expression profile [118]. Developmentally induced ABI5 activity is controlled by the ABA signalling cascade [119] and, moreover was found to be modified through several post-translational modifications such as ubiquitination, sumoylation and *S*-nitrosylation [117]. Apart from its impact on seed development, ABI5 has been found to directly control genes involved in abiotic stress responses [120], chlorophyll catabolism [121], lateral root development and dark-induced leaf senescence (DIS) [117]. With respect to DIS both ABI5 and ABFs seem to fulfil in part redundant functions [122].

Moreover, it was demonstrated that ABI5 also participates in plant floral transition control [123] by directly affecting transcription of flowering locus C (FLC), which represses the florigen gene flowering locus T (FT) [124]. Interestingly, the highly related flowering locus D (FD) and FD paralog (FDP) TFs, which form another subgroup of A-bZIPs are required for FT function [125••]. Both regulators are expressed in the shoot apex and are able to interact with FT in the nucleus to promote transcription of pivotal floral meristem identity genes such as APETALA1 (AP1) [124]. By these means, the bZIP factors FD and FDP promote floral transition and flower development. Importantly, recent findings in rice demonstrate that the homologs of FT and FD also interact with 14-3-3 proteins to form a ternary complex to control AP1 mediated flowering [126]. As the rice anti-florigen terminal flower 1 (TFL1) competes for binding to the 14-3-3 protein partner these results provide a molecular basis how flowering is controlled by the balance of the florigen FT and the anti-florigen TFL1. Concerning the last subgroup (bZIP13 and bZIP40) no functional data are available, yet.

Concluding remarks

Since publication of the first classification of *Arabidopsis* bZIPs around 15 years ago, a tremendous amount of

functional data has accumulated, placing bZIPs at central hubs in environmental and developmental signalling. Indeed, novel genome-wide techniques have supported TF research in general, for example, by using near-complete yeast-one-hybrid TF-collections [1••], genome-wide TF-binding studies [81••] or routine ChIP-seq methods to unravel TF-hierarchies [127]. However, future studies have to specify cellular TF-actions by focusing analyses on defined tissues under selected developmental and environmental conditions in preference to whole seedling approaches.

A long-standing enigma in TF research is how specificity is gained from limited *cis*-element sequence information particular for bZIP and bHLH factors, which both preferentially bind G-boxes. Flanking DNA sequences, TF-heterodimerisation and complex formation as well as accessibility to DNA due to chromatin modifications provide explanations for *in vivo* G-box specificity. Although recent bioinformatic attempts are straightforward [128], they did not yet gain a sufficient level of insight. More striking, the complex regulation of promoters with multiple binding sites is not unravelled for any plant promoter, yet.

The bZIP family provides many examples how TF activity is controlled on multiple levels, namely transcriptionally, translationally (e.g. SIRT) or post-translationally (e.g. by phosphorylation, nuclear translocation, protein stability). It is self-evident, that insight in these regulatory mechanisms is crucial for understanding bZIP function.

The impact of (partial) redundancies (e.g. group I) and the functional importance of heterodimerisation remain important research topics, particularly for bZIPs. Yet, only a limited number of dimer interactions have been tested experimentally, by using two-hybrid, BIFC or pull-down approaches [30,59,82]. Although predictions of bZIP dimerisation patterns have been conducted, they do not sufficiently match experimental data [129]. Analysing group-specific interactions, quasi-homodimers within groups have frequently been found (e.g. within groups G and H), which may partially explain functional redundancies. Nevertheless, the importance of inter-group heterodimerisation (e.g. C and S_1, E and I) has been demonstrated and a family-wide interaction matrix would be straightforward. In human bZIP research large-scale heterodimerisation and its consequence on DNA-binding has been assessed [4••]. In this respect, detailed knowledge on bZIP expression and protein distribution is essential to predict *in vivo* heterodimerisation. Moreover, tools to study dynamic (hetero)-dimerisation in a single-cell or a single-promoter context (e.g. by using fluorescent-labelled TFs) yet need to be established.

Finally, approaches to define crucial TF protein interaction partners and their structural properties are highly important. In this respect, elucidating the mechanism controlling co-activator and co-repressor function need to be addresses, which appear to be highly regulated by post-translational modifications, as demonstrated for NPR1. Finally, the interplay between chromatin dynamics and TF-action needs to be further elucidated. As described for animal systems, 'pioneer factor' which target and remodel silent chromatin to enable accessibility for other TFs to facilitate gene control may be important research topics [130].

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at https://doi.org/10.1016/j.pbi.2018.05.001.

References and recommended reading

Papers of particular interest, published within the period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

1. •• Pruneda-Paz JL, Breton G, Nagel DH, Kang SE, Bonaldi K, Doherty CJ, Ravelo S, Galli M, Ecker JR, Kay SA: **A genome-scale resource for the functional characterization of Arabidopsis transcription factors**. *Cell Rep* 2014, **8**:622-632.
Resource of almost 2000 Arabidopsis TFs in GATEWAY-compatible vectors to perform Yeast-1-Hybrid studies.

2. Kouzarides T, Ziff E: **Leucine zippers of fos, jun and GCN4 dictate dimerization specificity and thereby control DNA binding**. *Nature* 1989, **340**:568-571.

3. Vinson CR, Sigler PB, McKnight SL: **Scissors-grip model for DNA recognition by a family of leucine zipper proteins**. *Science* 1989, **246**:911-916.

4. •• Rodríguez-Martínez JA, Reinke AW, Bhimsaria D, Keating AE, Ansari AZ: **Combinatorial bZIP dimers display complex DNA-binding specificity landscapes**. *Elife* 2017, **6**:1-29.
Development and application of an EMSA-FRET technique to study large-scale heterodimerisation of human bZIPs.

5. Corrêa LGG, Riaño-Pachón DM, Schrago CG, Vicentini dos Santos R, Mueller-Roeber B, Vincentz M: **The role of bZIP transcription factors in green plant evolution: adaptive features emerging from four founder genes**. *PLoS ONE* 2008, **3**: e2944.

6. Zhou Y, Xu D, Jia L, Huang X, Ma G, Wang S, Zhu M, Zhang A, Guan M, Lu K *et al.*: **Genome-wide identification and structural analysis of bZIP transcription factor genes in *Brassica napus***. *Genes* 2017, **8**:288.

7. Jakoby M, Weisshaar B, Dröge-Laser W, Vicente-Carbajosa J, Tiedemann J, Kroj T, Parcy F: **bZIP transcription factors in Arabidopsis**. *Trends Plant Sci* 2002, **7**:106-111.

8. Gatz C: **From pioneers to team players: TGA transcription factors provide a molecular link**. *Mol Plant Microbe Interact* 2013, **26**:151-159.

9. Fu ZQ, Dong X: **Systemic acquired resistance: turning local infection into global defense**. *Annu Rev Plant Biol* 2013, **64**:839-863.

10. Withers J, Dong X: **Posttranslational modifications of NPR1: a single protein playing multiple roles in plant immunity and physiology**. *PLOS Pathog* 2016, **12**:e1005707.

11. Saleh A, Withers J, Mohan R, Marqués J, Gu Y, Yan S, Zavaliev R, Nomoto M, Tada Y, Dong X: **Posttranslational modifications of the master transcriptional regulator NPR1 enable dynamic but tight control of plant immune responses**. *Cell Host Microbe* 2015, **18**:169-182.

•• The authors present a conclusive model on NPR1-dependent transcriptional control, based on several post-translational modifications. By changing TF partners, NPR1 can form repressing and activating TF-DNA-complexes.

12. Pape S, Thurow C, Gatz C: **The Arabidopsis PR-1 promoter contains multiple integration sites for the coactivator NPR1 and the repressor SNI1**. *Plant Physiol* 2010, **154**:1805-1818.

13. Zander M, La Camera S, Lamotte O, Metraux JP, Gatz C: ***Arabidopsis thaliana* class-II TGA transcription factors are essential activators of jasmonic acid/ethylene-induced defense responses**. *Plant J* 2010, **61**:200-210.

• Besides the well-known impact of TGAs on SA-dependent gene activation, the authors demonstrate the impact of TGA factors on the JA/ET-pathway acting against necrotrophs.

14. Zander M, Thurow C, Gatz C: **TGA transcription factors activate the salicylic acid-suppressible branch of the ethylene-induced defense program by regulating ORA59 expression**. *Plant Physiol* 2014, **165**:1671-1683.

15. Caarls L, Pieterse CMJ, Van Wees SCM: **How salicylic acid takes transcriptional control over jasmonic acid signaling**. *Front Plant Sci* 2015, **6**:1-11.

16. Ndamukong I, Abdallat A, Thurow C, Fode B, Zander M, Weigel R, Gatz C: **SA-inducible Arabidopsis glutaredoxin interacts with TGA factors and suppresses JA-responsive PDF1.2 transcription**. *Plant J* 2007, **50**:128-139.

17. Uhrig JF, Huang L, Barghahn S, Willmer M, Thurow C, Gatz C: **CC-type glutaredoxins recruit the transcriptional co-repressor TOPLESS to TGA-dependent target promoters in *Arabidopsis thaliana***. *Biochim Biophys Acta Gene Regul Mech* 2017, **1860**:218-226.

18. Müller S, Hilbert B, Dueckershoff K, Roitsch T, Krischke M, Müller MJ, Berger S: **General detoxification and stress responses are mediated by oxidized lipids through TGA transcription factors in Arabidopsis**. *Plant Cell* 2008, **20**:768-785.

19. Fode B, Siemsen T, Thurow C, Weigel R, Gatz C: **The Arabidopsis GRAS protein SCL14 interacts with class II TGA transcription factors and is essential for the activation of stress-inducible promoters**. *Plant Cell* 2008, **20**:3122-3135.

20. Huang L, Li N, Thurow C, Wirtz M, Hell R, Gatz C: **Ectopically expressed glutaredoxin ROXY19 negatively regulates the detoxification pathway in *Arabidopsis thaliana***. *BMC Plant Biol* 2016, **16**:200.

21. Choi J, Huh SU, Kojima M, Sakakibara H, Paek KH, Hwang I: **The cytokinin-activated transcription factor ARR2 promotes plant immunity via TGA3/NPR1-dependent salicylic acid signaling in arabidopsis**. *Dev Cell* 2010, **19**:284-295.

22. Fang H, Liu Z, Long Y, Liang Y, Jin Z, Zhang L, Liu D, Li H, Zhai J, Pei Y: **The Ca^{2+}/calmodulin2-binding transcription factor TGA3 elevates LCD expression and H_2S production to bolster Cr^{6+} tolerance in Arabidopsis**. *Plant J* 2017, **91**:1038-1050.

23. Shearer HL, Cheng YT, Wang L, Liu J, Boyle P, Després C, Zhang Y, Li X, Fobert PR: **Arabidopsis clade I TGA transcription factors regulate plant defenses in an NPR1-independent fashion**. *Mol Plant Microbe Interact* 2012, **25**:1459-1468.

24. Wang L, Fobert PR: **Arabidopsis clade I TGA factors regulate apoplastic defences against the bacterial pathogen *Pseudomonas syringae* through endoplasmic reticulum-based processes**. *PLOS ONE* 2013, **8**:e77378.

25. Sun T, Busta L, Zhang Q, Ding P, Jetter R, Zhang Y: T: **GACG-binding factor 1 (TGA1) and TGA4 regulate salicylic acid and pipecolic acid biosynthesis by modulating the expression of systemic acquired resistance deficient 1 (SARD1) and calmodulin-binding protein 60 g (CBP60 g)**. *New Phytol* 2018, **217**:344-354.

26. Alvarez JM, Riveras E, Vidal EA, Gras DE, Contreras-López O, Tamayo KP, Aceituno F, Gómez I, Ruffel S, Lejay L *et al.*: **Systems approach identifies TGA1 and TGA4 transcription factors as important regulatory components of the nitrate response of *Arabidopsis thaliana* roots**. *Plant J* 2014, **80**:1-13.

27. Canales J, Contreras-López O, Álvarez JM, Gutiérrez RA: **Nitrate induction of root hair density is mediated by TGA1/TGA4 and CPC transcription factors in *Arabidopsis thaliana***. *Plant J* 2017, **92**:305-316.

28. Maier AT, Stehling-sun S, Offenburger S, Lohmann JU: **The bZIP transcription factor PERIANTHIA: a multifunctional hub for meristem control**. *Front Plant Sci* 2011, **2**:1-17.

29. Weltmeier F, Ehlert A, Mayer CS, Dietrich K, Wang X, Schutze K, Alonso R, Harter K, Vicente-Carbajosa J, Dröge-Laser W: **Combinatorial control of Arabidopsis proline dehydrogenase transcription by specific heterodimerisation of bZIP transcription factors**. *EMBO J* 2006, **25**:3133-3143.

30. Ehlert A, Weltmeier F, Wang X, Mayer CS, Smeekens S, Vicente-Carbajosa J, Dröge-Laser W: **Two-hybrid protein–protein interaction analysis in Arabidopsis protoplasts: establishment of a heterodimerization map of group C and group S bZIP transcription factors**. *Plant J* 2006, **46**:890-900.

31. Dröge-Laser W, Weiste C: **The C/S_1-bZIP-network: a regulatory hub orchestrating plant energy homeostasis**. *Trends Plant Sci* 2018, **23**:422-433.

32. Yamashita Y, Takamatsu S, Glasbrenner M, Becker T, Naito S, Beckmann R: **Sucrose sensing through nascent peptide-meditated ribosome stalling at the stop codon of Arabidopsis bZIP11**. *FEBS Lett* 2017, **591**:1266-1277.

33. Peviani A, Lastdrager J, Hanson J, Snel B: **The phylogeny of C/S1 bZIP transcription factors reveals a shared algal ancestry and the pre-angiosperm translational regulation of S1 transcripts**. *Sci Rep* 2016, **6**:30444.

34. Rahmani F, Hummel M, Schuurmans J, Wiese-Klinkenberg A, Smeekens S, Hanson J: **Sucrose control of translation mediated by an upstream open reading frame-encoded peptide**. *Plant Physiol* 2009, **150**:1356-1367.

35. Baena-González E, Rolland F, Thevelein JM, Sheen J: **A central integrator of transcription networks in plant stress and energy signalling**. *Nature* 2007, **448**:938-942.

•• Milestone paper on the impact of SnRK1 on plant metabolism, first proposing C/S_1-bZIP TFs as downstream mediators of SnRK1.

36. Mair A, Pedrotti L, Wurzinger B, Anrather D, Simeunovic A, Weiste C, Valerio C, Dietrich K, Kirchler T, Nägele T *et al.*: **SnRK1-triggered switch of bZIP63 dimerization mediates the low-energy response in plants**. *Elife* 2015, **4**:e05828.

•• Using an unbiased biochemical approach, the authors define bZIP63 as direct target of the SnRK1 kinase, identify the *in vivo* phosphorylation sites in bZIP63 and demonstrate the impact of phosphorylation in planta. Moreover, a phosphorylation dependent bZIP heterodimerisation is proposed.

37. Pedrotti L, Weiste C, Nägele T, Wolf E, Lorenzin F, Dietrich K, Mair A, Weckwerth W, Teige M, Baena-González EWD-L: **Snf1-related kinase1-controlled C/S1-bZIP signaling activates alternative mitochondrial metabolic pathways to ensure plant survival under low energy stress provoked by extended darkness**. *Plant Cell* 2018, **30**:495-509.

•• Using transcriptome analyses the authors define the overlap of SnRK1 and group S_1-regulated genes and support bZIP function in activating alternative metabolic pathways to promote survival under energy-limiting conditions. Moreover, a ternary complex of C/S_1-bZIPs and SnRK1 is proposed to directly bind promoters and to regulate transcription via histone acetylation.

38. Weiste C, Dröge-Laser W: **The Arabidopsis transcription factor bZIP11 activates auxin-mediated transcription by recruiting the histone acetylation machinery**. *Nat Commun* 2014, **5**:3883.

39. Ma J, Hanssen M, Lundgren K, Hernández L, Delatte T, Ehlert A, Liu C-M, Schluepmann H, Dröge-Laser W, Moritz T *et al.*: **The sucrose-regulated Arabidopsis transcription factor bZIP11 reprograms metabolism and regulates trehalose metabolism**. *New Phytol* 2011, **191**:733-745.

40. Kang SG, Price J, Lin P-CC, Hong JC, Jang J-CC: **The Arabidopsis bZIP1 transcription factor is involved in sugar signaling, protein networking, and DNA binding**. *Mol Plant* 2010, **3**:361-373.

41. •• Para A, Li Y, Marshall-Colón A, Varala K, Francoeur NJ, Moran TM, Edwards MB, Hackley C, Bargmann BOR, Birnbaum KD *et al.*: **Hit-and-run transcriptional control by bZIP1 mediates rapid nutrient signaling in Arabidopsis**. *Proc Natl Acad Sci U S A* 2014, **111**:10371-10376.
Using omics approaches on transiently transformed protoplasts, bZIP1 is proposed to function as a pioneer TF ('hit and run' model), which alters transcriptional properties of a promoter by transient binding. However, the mechanism needs to be established and verified in planta.

42. • Weiste C, Pedrotti L, Selvanayagam J, Muralidhara P, Fröschel C, Novák O, Ljung K, Hanson J, Dröge-Laser W: **The Arabidopsis bZIP11 transcription factor links low-energy signalling to auxin-mediated control of primary root growth**. *PLOS Genet* 2017, **13**:e1006607.
Energy availability and primary root growth are linked via bZIP11-related bZIPs, which control the important negative regulator of root meristem function IAA3/SHY2.

43. Alonso R, Onate-Sanchez L, Weltmeier F, Ehlert A, Diaz I, Dietrich K, Vicente-Carbajosa J, Dröge-Laser W: **A pivotal role of the basic leucine zipper transcription factor bZIP53 in the regulation of Arabidopsis seed maturation gene expression based on heterodimerization and protein complex formation**. *Plant Cell* 2009, **21**:1747-1761.

44. Hartmann L, Pedrotti L, Weiste C, Fekete A, Schierstaedt J, Göttler J, Kempa S, Krischke M, Dietrich K, Mueller MJ *et al.*: **Crosstalk between two bZIP signaling pathways orchestrates salt-induced metabolic reprogramming in Arabidopsis roots**. *Plant Cell* 2015, **27**:2244-2260.

45. •• Kaminaka H, Nake C, Epple P, Dittgen J, Schutze K, Chaban C, Holt BF III, Merkle T, Schafer E, Harter K *et al.*: **bZIP10-LSD1 antagonism modulates basal defense and cell death in Arabidopsis following infection**. *EMBO J* 2006, **25**:4400-4411.
The authors describe an interesting mechanism, how the well-known cell-death regulator LSD1 functions as anchor protein to control nuclear import of bZIP10. The mechanism, how the TF is released needs to be established.

46. Dietrich K, Weltmeier F, Ehlert A, Weiste C, Stahl M, Harter K, Dröge-Laser W: **Heterodimers of the Arabidopsis transcription factors bZIP1 and bZIP53 reprogram amino acid metabolism during low energy stress**. *Plant Cell* 2011, **23**:381-395.

47. Gangappa SN, Botto JF: **The multifaceted roles of HY5 in plant growth and development**. *Mol Plant* 2016, **9**:1353-1365.

48. Yang Y, Liang T, Zhang L, Shao K, Gu X, Shang R, Shi N, Li X, Zhang P, Liu H: **UVR8 interacts with WRKY36 to regulate HY5 transcription and hypocotyl elongation in Arabidopsis**. *Nat Plants* 2018, **4**:98-107.

49. • van Gelderen K, Kang C, Paalman R, Keuskamp D, Hayes S, Pierik R: **Far-red light detection in the shoot regulates lateral root development through the HY5 transcription factor**. *Plant Cell* 2018, **30**:101-116.
The authors link light perception (red:far-red) in the shoot to auxin-mediated lateral root emergence via the HY5 transcription factor.

50. Sibout R, Sukumar P, Hettiarachchi C, Holm M, Muday GK, Hardtke CS: **Opposite root growth phenotypes of hy5 versus hy5 hyh mutants correlate with increased constitutive auxin signaling**. *PLoS Genet* 2006, **2**:1898-1911.

51. Li QF, He JX: **BZR1 interacts with HY5 to mediate brassinosteroid- and light-regulated cotyledon opening in arabidopsis in darkness**. *Mol Plant* 2016, **9**:113-125.

52. Yu Y, Wang J, Zhang Z, Quan R, Zhang H, Deng XW, Ma L, Huang R: **Ethylene promotes hypocotyl growth and HY5 degradation by enhancing the movement of COP1 to the nucleus in the light**. *PLoS Genet* 2013, **9**:e1004025.

53. Ciolfi A, Sessa G, Sassi M, Possenti M, Salvucci S, Carabelli M, Morelli G, Ruberti I: **Dynamics of the shade-avoidance response in Arabidopsis**. *Plant Physiol* 2013, **163**:331-353.

54. Hoecker U: **The activities of the E3 ubiquitin ligase COP1/SPA, a key repressor in light signaling**. *Curr Opin Plant Biol* 2017, **37**:63-69.

55. Lee J, He K, Stolc V, Lee H, Figueroa P, Gao Y, Tongprasit W, Zhao H, Lee I, Deng XW: **Analysis of transcription factor HY5 genomic binding sites revealed its hierarchical role in light regulation of development**. *Plant Cell* 2007, **19**:731-749.

56. Zhang H, He H, Wang X, Wang X, Yang X, Li L, Deng XW: **Genome-wide mapping of the HY5-mediated gene networks in Arabidopsis that involve both transcriptional and post-transcriptional regulation**. *Plant J* 2011, **65**:346-358.

57. Toledo-Ortiz G, Johansson H, Lee KP, Bou-Torrent J, Stewart K, Steel G, Rodríguez-Concepción M, Halliday KJ: **The HY5-PIF regulatory module coordinates light and temperature control of photosynthetic gene transcription**. *PLoS Genet* 2014, **10**:e1004416.

58. Yoon MK, Kim HM, Choi G, Lee JO, Choi BS: **Structural basis for the conformational integrity of the *Arabidopsis thaliana* HY5 leucine zipper homodimer**. *J Biol Chem* 2007, **282**:12989-13002.

59. Llorca CM, Berendzen KW, Malik WA, Mahn S, Piepho H-P, Zentgraf U: **The elucidation of the interactome of 16 Arabidopsis bZIP factors reveals three independent functional networks**. *PLOS ONE* 2015, **10**:e0139884.

60. Binkert M, Kozma-Bognar L, Terecskei K, De Veylder L, Nagy F, Ulm R: **UV-B-responsive association of the arabidopsis bZIP transcription factor ELONGATED HYPOCOTYL5 with target genes, including its own promoter**. *Plant Cell* 2014, **26**:4200-4213.

61. •• Chen X, Yao Q, Gao X, Jiang C, Harberd NP, Fu X: **Shoot-to-root mobile transcription factor HY5 coordinates plant carbon and nitrogen acquisition**. *Curr Biol* 2016, **26**:640-646.
HY5 is demonstrated to act as a presumably phloem-mobile TF, balancing root and shoot growth by activating export of photosynthetically-produced sugars in the leaf and NO_3^- uptake in the root.

62. Gangappa SN, Srivastava AK, Maurya JP, Ram H, Chattopadhyay S: **Z-box binding transcription factors (ZBFs): a new class of transcription factors in Arabidopsis seedling development**. *Mol Plant* 2013, **6**:1758-1768.

63. Singh A, Ram H, Abbas N, Chattopadhyay S: **Molecular interactions of GBF1 with HY5 and HYH proteins during light-mediated seedling development in *Arabidopsis thaliana***. *J Biol Chem* 2012, **287**:25995-26009.

64. Ram H, Priya P, Jain M, Chattopadhyay S: **Genome-wide DNA binding of GBF1 is modulated by its heterodimerizing protein partners, HY5 and HYH**. *Mol Plant* 2014, **7**:448-451.

65. Maurya JP, Sethi V, Gangappa SN, Gupta N, Chattopadhyay S: **Interaction of MYC2 and GBF1 results in functional antagonism in blue light-mediated Arabidopsis seedling development**. *Plant J* 2015, **83**:439-450.

66. • Smykowski A, Zimmermann P, Zentgraf U: **G-box binding factor1 reduces CATALASE2 expression and regulates the onset of leaf senescence in arabidopsis**. *Plant Physiol* 2010, **153**:1321-1331.
The authors mechanistically link GBF1 function to senescence, by demonstrating that GBF1 binds and represses CATALASE2 promoters to increase ROS as an important signal in senescence.

67. Giri MK, Singh N, Banday ZZ, Singh V, Ram H, Singh D, Chattopadhyay S, Nandi AK: **GBF1 differentially regulates CAT2 and PAD4 transcription to promote pathogen defense in *Arabidopsis thaliana***. *Plant J* 2017, **91**:802-815.

68. Shaikhali J, Norén L, de Dios Barajas-López J, Srivastava V, König J, Sauer UH, Wingsle G, Dietz K-J, Strand Å: **Redox-mediated mechanisms regulate DNA binding activity of the G-group of basic region leucine zipper (bZIP) transcription factors in Arabidopsis**. *J Biol Chem* 2012, **287**:27510-27525.

69. Shaikhali J: **GIP1 protein is a novel cofactor that regulates DNA-binding affinity of redox-regulated members of bZIP transcription factors involved in the early stages of Arabidopsis development**. *Protoplasma* 2015, **252**:867-883.

70. Smykowski A, Fischer S, Zentgraf U: **Phosphorylation Affects DNA-binding of the senescence-regulating bZIP transcription factor GBF1**. *Plants* 2015, **4**:691-709.

71. Tzfira T, Vaidya M, Citovsky V: **VIP1, an Arabidopsis protein that interacts with Agrobacterium VirE2, is involved in VirE2 nuclear import and Agrobacterium infectivity**. *EMBO J* 2001, **20**:3596-3607.

72. Citovsky V, Kapelnikov A, Olie S, Zakai N, Rojas MR, Gilbertson RL, Tzfira T, Loyter A: **Protein interactions involved in nuclear import of the Agrobacterium VirE2 protein in vivo and in vitro**. *J Biol Chem* 2004, **279**:29528-29533.

73. •• Djamei A, Pitzschke A, Nakagami H, Rajh I, Hirt H: **Trojan horse strategy in Agrobacterium transformation: abusing MAPK defense signaling**. *Science* 2007, **318**:453-456.
The authors link nuclear import of the group I bZIP VIP1 to its phosphorylation by a MAPK pathway.

74. Shi Y, Lee L-Y, Gelvin SB: **Is VIP1 important for Agrobacterium-mediated transformation**. *Plant J* 2014.

75. Pitzschke A, Djamei A, Teige M, Hirt H: **VIP1 response elements mediate mitogen-activated protein kinase 3-induced stress gene expression**. *Proc Natl Acad Sci U S A* 2009, **106**:18414-18419.

76. Lacroix B, Citovsky V: **Characterization of VIP1 activity as a transcriptional regulator *in vitro* and in planta**. *Sci Rep* 2013, **3**:2440.

77. Wu Y, Zhao Q, Gao L, Yu XM, Fang P, Oliver DJ, Xiang CB: **Isolation and characterization of low-sulphur-tolerant mutants of Arabidopsis**. *J Exp Bot* 2010, **61**:3407-3422.

78. Tsugama D, Liu S, Takano T: **VIP1 is very important/interesting protein 1 regulating touch responses of Arabidopsis**. *Plant Signal Behav* 2016, **11**:1-3.

79. Tsugama D, Liu S, Takano T: **A bZIP protein, VIP1, is a regulator of osmosensory signaling in Arabidopsis**. *Plant Physiol* 2012, **159**:144-155.

80. Tsugama D, Liu S, Takano T: **Analysis of functions of VIP1 and its close homologs in osmosensory responses of *Arabidopsis thaliana***. *PLoS ONE* 2014, **9**:e103930.

81. •• O'Malley RC, Huang SC, Song L, Lewsey MG, Bartlett A, Nery JR, Galli M, Gallavotti A, Ecker JR: **Cistrome and epicistrome features shape the regulatory DNA landscape**. *Cell* 2016, **165**:1280-1292.
Resource presenting a genome-wide data-set on *in vitro* DNA-binding properties of roughly 500 TFs.

82. Van Leene J, Blomme J, Kulkarni SR, Cannoot B, De Winne N, Eeckhout D, Persiau G, Van De Slijke E, Vercruysse L, Vanden Bossche R *et al.*: **Functional characterization of the Arabidopsis transcription factor bZIP29 reveals its role in leaf and root development**. *J Exp Bot* 2016, **67**:5825-5840.

83. Xu C, Cao H, Zhang Q, Wang H, Xin W, Xu E, Zhang S, Yu R, Yu D, Hu Y: **Control of auxin-induced callus formation by bZIP59–LBD complex in Arabidopsis regeneration**. *Nat Plants* 2018, **4**:108-115.

84. Gibalová A, Steinbachová L, Hafidh S, Bláhová V, Gadiou Z, Michailidis C, Müller K, Pleskot R, Dupľáková N, Honys D: **Characterization of pollen-expressed bZIP protein interactions and the role of ATbZIP18 in the male gametophyte**. *Plant Reprod* 2017, **30**:1-17.

85. Lozano-Sotomayor P, Chávez Montes RA, Silvestre-Vañó M, Herrera-Ubaldo H, Greco R, Pablo-Villa J, Galliani BM, Diaz-Ramirez D, Weemen M, Boutilier K *et al.*: **Altered expression of the bZIP transcription factor DRINK ME affects growth and reproductive development in *Arabidopsis thaliana***. *Plant J* 2016, **88**:437-451.

86. Pyo H, Demura T, Fukuda H: **Vascular cell expression patterns of Arabidopsis bZIP group I genes**. *Plant Biotechnol* 2006, **23**:497-501.

87. Gibalová A, Reňák D, Matczuk K, Dupl'Áková N, Cháb D, Twell D, Honys D: **AtbZIP34 is required for Arabidopsis pollen wall patterning and the control of several metabolic pathways in developing pollen**. *Plant Mol Biol* 2009, **70**:581-601.

88. Shen H, Cao K, Wang X: **A conserved proline residue in the leucine zipper region of AtbZIP34 and AtbZIP61 in *Arabidopsis thaliana* interferes with the formation of homodimer**. *Biochem Biophys Res Commun* 2007, **362**:425-430.

89. Howell SH: **Endoplasmic reticulum stress responses in plants**. *Annu Rev Plant Biol* 2013, **64**:477-499.

90. Liu J-X, Srivastava R, Che P, Howell SH: **An endoplasmic reticulum stress response in arabidopsis is mediated by proteolytic processing and nuclear relocation of a membrane-associated transcription factor, bZIP28**. *Plant Cell* 2007, **19**:4111-4119.

91. Liu JX, Howell SH: **bZIP28 and NF-Y transcription factors are activated by ER stress and assemble into a transcriptional complex to regulate stress response genes in Arabidopsis**. *Plant Cell* 2010, **22**:782-796.

92. Zhou SF, Sun L, Valdés AE, Engström P, Song ZT, Lu SJ, Liu JX: **Membrane-associated transcription factor peptidase, site-2 protease, antagonizes ABA signaling in Arabidopsis**. *New Phytol* 2015, **208**:188-197.

93. Kim J-S, Yamaguchi-Shinozaki K, Shinozaki K: **ER-anchored transcription factors bZIP17 and bZIP28 regulate root elongation**. *Plant Physiol* 2018, **176**:2221-2230.

94. LIU J-X, SRIVASTAVA R, HOWELL SH: **Stress-induced expression of an activated form of AtbZIP17 provides protection from salt stress in Arabidopsis**. *Plant Cell Environ* 2008, **31**:1735-1743.

95. Iwata Y, Fedoroff NV, Koizumi N: **Arabidopsis bZIP60 is a proteolysis-activated transcription factor involved in the endoplasmic reticulum stress response**. *Plant Cell* 2008, **20**:3107-3121.

96. Iwata Y, Koizumi N: **An Arabidopsis transcription factor, AtbZIP60, regulates the endoplasmic reticulum stress response in a manner unique to plants**. *Proc Natl Acad Sci U S A* 2005, **102**:5280-5285.

97. • Deng Y, Humbert S, Liu J-X, Srivastava R, Rothstein SJ, Howell SH: **Heat induces the splicing by IRE1 of a mRNA encoding a transcription factor involved in the unfolded protein response in Arabidopsis**. *Proc Natl Acad Sci U S A* 2011, **108**:7247-7252.
The authors define stress-induced splicing as a novel mechanism to generate nuclear-localized TFs.

98. Rubio C, Pincus D, Korennykh A, Schuck S, El-Samad H, Walter P: **Homeostatic adaptation to endoplasmic reticulum stress depends on Ire1 kinase activity**. *J Cell Biol* 2011, **193**:171-184.

99. Nagashima Y, Mishiba K, Suzuki E, Shimada Y, Iwata Y, Koizumi N: **Arabidopsis IRE1 catalyses unconventional splicing of bZIP60 mRNA to produce the active transcription factor**. *Sci Rep* 2011, **1**:29.

100. Ruberti C, Lai Y, Brandizzi F: **Recovery from temporary endoplasmic reticulum stress in plants relies on the tissue-specific and largely independent roles of bZIP28 and bZIP60, as well as an antagonizing function of BAX-Inhibitor 1 upon the pro-adaptive signaling mediated by bZIP28**. *Plant J* 2018, **93**:155-165.

101. Nawkar GM, Kang CH, Maibam P, Park JH, Jung YJ, Chae HB, Chi YH, Jung IJ, Kim WY, Yun D-J *et al.*: **HY5, a positive regulator of light signaling, negatively controls the unfolded protein response in *Arabidopsis***. *Proc Natl Acad Sci U S A* 2017, **114**:2084-2089.

102. • Assunção AGL, Herrero E, Lin Y-F, Huettel B, Talukdar S, Smaczniak C, Immink RGH, van Eldik M, Fiers M, Schat H *et al.*: ***Arabidopsis thaliana* transcription factors bZIP19 and bZIP23 regulate the adaptation to zinc deficiency**. *Proc Natl Acad Sci U S A* 2010, **107**:10296-10301.
The authors define a novel function of the group F members bZIP19 and bZIP23 in regulating Zn homeostasis.

103. Inaba S, Kurata R, Kobayashi M, Yamagishi Y, Mori I, Ogata Y, Fukao Y: **Identification of putative target genes of bZIP19, a**

transcription factor essential for Arabidopsis adaptation to Zn deficiency in roots. *Plant J* 2015, **84**:323-334.

104. Castro PH, Lilay GH, Muñoz-Mérida A, Schjoerring JK, Azevedo H, Assunção AGL: **Phylogenetic analysis of F-bZIP transcription factors indicates conservation of the zinc deficiency response across land plants**. *Sci Rep* 2017, **7**:3806.

105. Popova OV, Yang O, Dietz K-J, Golldack D: **Differential transcript regulation in *Arabidopsis thaliana* and the halotolerant *Lobularia maritima* indicates genes with potential function in plant salt adaptation**. *Gene* 2008, **423**:142-148.

106. Yang O, Popova OV, Süthoff U, Lüking I, Dietz K-J, Golldack D: **The Arabidopsis basic leucine zipper transcription factor AtbZIP24 regulates complex transcriptional networks involved in abiotic stress resistance**. *Gene* 2009, **436**:45-55.

107. Banerjee A, Roychoudhury A: **Abscisic-acid-dependent basic leucine zipper (bZIP) transcription factors in plant abiotic stress**. *Protoplasma* 2017, **254**:3-16.

108. Sirichandra C, Davanture M, Turk BE, Zivy M, Valot B, Leung J, Merlot S: **The arabidopsis ABA-activated kinase OST1 phosphorylates the bZIP transcription factor ABF3 and creates a 14-3-3 binding site involved in its turnover**. *PLoS ONE* 2010:5.

109. Yoshida T, Mogami J, Yamaguchi-Shinozaki K: **Omics approaches toward defining the comprehensive abscisic acid signaling network in plants**. *Plant Cell Physiol* 2015, **56**:1043-1052.

110. •• Soon FF, Ng LM, Zhou XE, West GM, Kovach A, Tan MHE, Suino-Powell KM, He Y, Xu Y, Chalmers MJ *et al.*: **Molecular mimicry regulates ABA signaling by SnRK2 kinases and PP2C phosphatases**. *Science* 2012, **335**:85-88.
Structural analysis of the PP2C-SnRK2 complex unravels a simple mechanism how ABA binding to PP2C can release SnRK2 from PP2C repression and initiate ABA signalling.

111. Umezawa T, Nakashima K, Miyakawa T, Kuromori T, Tanokura M, Shinozaki K, Yamaguchi-Shinozaki K: **Molecular basis of the core regulatory network in ABA responses: sensing, signaling and transport**. *Plant Cell Physiol* 2010, **51**:1821-1839.

112. •• Fujii H, Chinnusamy V, Rodrigues A, Rubio S, Antoni R, Park S-Y, Cutler SR, Sheen J, Rodriguez PL, Zhu J-K: **In vitro reconstitution of an abscisic acid signalling pathway**. *Nature* 2009, **462**:660-664.
Employing a protoplast transfection system the authors provide first insights into the mode of action of the core ABA signalling components.

113. Cutler SR, Rodriguez PL, Finkelstein RR, Abrams SR: **Abscisic acid: emergence of a core signaling network**. *Annu Rev Plant Biol* 2010, **61**:651-679.

114. Furihata T, Maruyama K, Fujita Y, Umezawa T, Yoshida R, Shinozaki K, Yamaguchi-Shinozaki K: **Abscisic acid-dependent multisite phosphorylation regulates the activity of a transcription activator AREB1**. *Proc Natl Acad Sci U S A* 2006, **103**:1988-1993.

115. Nakashima K, Yamaguchi-Shinozaki K: **ABA signaling in stress-response and seed development**. *Plant Cell Rep* 2013, **32**:959-970.

116. Kim SY: **Arabidopsis ABI5 subfamily members have distinct DNA-binding and transcriptional activities**. *Plant Physiol* 2002, **130**:688-697.

117. Skubacz A, Daszkowska-Golec A, Szarejko I: **The role and regulation of ABI5 (ABA-insensitive 5) in plant development, abiotic stress responses and phytohormone crosstalk**. *Front Plant Sci* 2016:7.

118. Bensmihen S, Giraudat J, Parcy F: **Characterization of three homologous basic leucine zipper transcription factors (bZIP) of the ABI5 family during *Arabidopsis thaliana* embryo maturation**. *J Exp Bot* 2005, **56**:597-603.

119. Nakashima K, Fujita Y, Kanamori N, Katagiri T, Umezawa T, Kidokoro S, Maruyama K, Yoshida T, Ishiyama K, Kobayashi M *et al.*: **Three arabidopsis SnRK2 protein kinases, SRK2D/SnRK2.2, SRK2E/SnRK2.6/OST1 and SRK2I/SnRK2.3, involved in ABA signaling are essential for the control of seed development and dormancy**. *Plant Cell Physiol* 2009, **50**:1345-1363.

120. Finkelstein RR, Lynch TJ: **The Arabidopsis abscisic acid response gene ABI5 encodes a basic leucine zipper transcription factor**. *Plant Cell* 2000, **12**:599-609.

121. Sakuraba Y, Kim D, Kim YS, Hörtensteiner S, Paek NC: **Arabidopsis STAYGREEN-LIKE (SGRL) promotes abiotic stress-induced leaf yellowing during vegetative growth**. *FEBS Lett* 2014, **588**:3830-3837.

122. Gao S, Gao J, Zhu X, Song Y, Li Z, Ren G, Zhou X, Kuai B: **ABF2, ABF3 and ABF4 promote ABA-mediated chlorophyll degradation and leaf senescence by transcriptional activation of chlorophyll catabolic genes and senescence-associated genes in Arabidopsis**. *Mol Plant* 2016, **9**:1272-1285.

123. Shu K, Chen Q, Wu Y, Liu R, Zhang H, Wang S, Tang S, Yang W, Xie Q: **Abscisic acid-insensitive 4 negatively regulates flowering through directly promoting Arabidopsis flowering locus C transcription**. *J Exp Bot* 2016, **67**:195-205.

124. Shu K, Luo X, Meng Y, Yang W: **Toward a molecular understanding of abscisic acid actions in floral transition**. *Plant Cell Physiol* 2018, **59**:215-221.

125. •• Abe M, Kobayashi Y, Yamamoto S, Ichinoki H, Notaguchi M, Goto K: **FD, a bZIP protein mediating signals from the floral pathway integrator FT at the shoot apex**. *Science* 2005, **309**:1052-1057.
The authors present mechanistic insights how photoperiodic induction of floral transition is controlled by mobile FT and the shoot apex expressed group A bZIP FD.

126. Kaneko-Suzuki M, Kurihara-Ishikawa R, Okushita-Terakawa C, Kojima C, Nagano-Fujiwara M, Ohki I, Tsuji H, Shimamoto K, Taoka KI: **TFL1-like proteins in rice antagonize rice FT-like protein in inflorescence development by competition for complex formation with 14-3-3 and FD**. *Plant Cell Physiol* 2018, **59**:458-468.

127. Song L, Huang SC, Wise A, Castanon R, Nery JR, Chen H, Watanabe M, Thomas J, Bar-Joseph Z, Ecker JR: **A transcription factor hierarchy defines an environmental stress response network**. *Science* 2016, **354**:558-566.

128. Ezer D, Shepherd SJ, Brestovitsky A, Dickinson P, Cortijo S, Charoensawan V, Box MS, Biswas S, Jaeger K, Wigge PA: **The G-box transcriptional regulatory code in Arabidopsis**. *Plant Physiol* 2017, **175**:628-640.

129. Deppmann CD, Acharya A, Rishi V, Wobbes B, Smeekens S, Taparowsky EJ, Vinson C: **Dimerization specificity of all 67 B-ZIP motifs in *Arabidopsis thaliana*: a comparison to *Homo sapiens* B-ZIP motifs**. *Nucleic Acids Res* 2004, **32**:3435-3445.

130. Zaret KS, Lerner J, Iwafuchi-Doi M: **Chromatin scanning by dynamic binding of pioneer factors**. *Mol Cell* 2016, **62**:665-667.