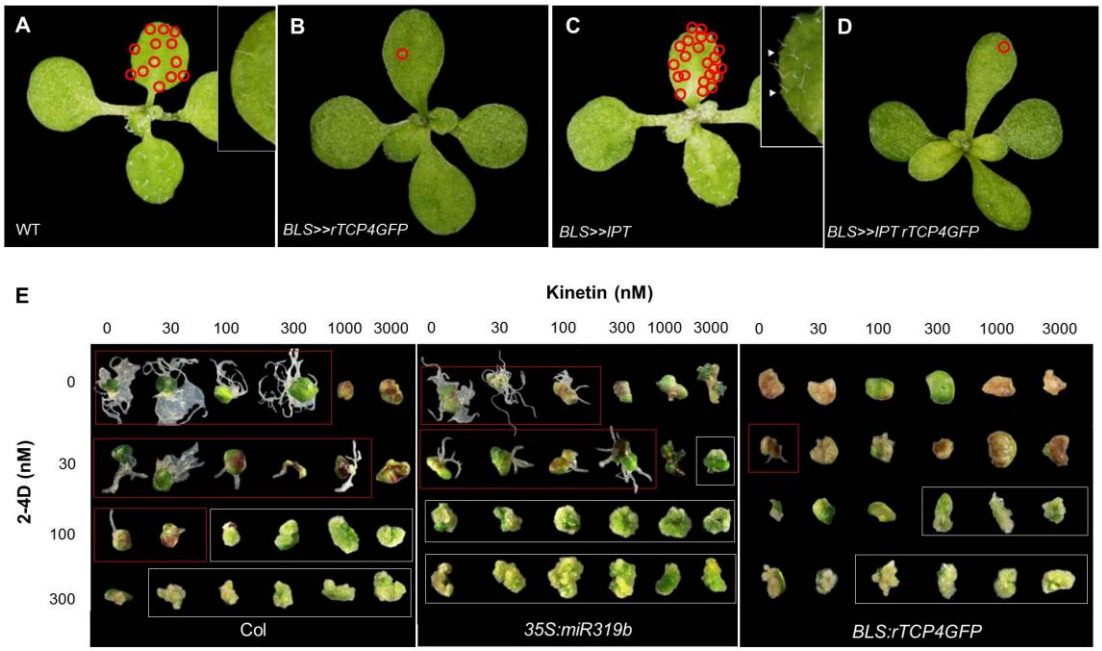

Figure S1, related to Figure 1. Modification of classical CK responses in leaves with altered CIN-TCP levels. A-D) 14-day-old wild type (A), *BLS>>IPT* (B), *BLS>>rTCP4GFP* (C) and *BLS>>rTCP4GFP IPT* (D) plants. Trichomes are marked in red circles on leaf 1. Insets show a close-up of the margin of leaf 1. White arrowheads mark serrations. **E)** Altered sensitivity to CK as monitored in a callus induction assay. Leaf 3 from ca. five 14-day-old seedlings was excised, incubated on plates containing varying concentrations of the CK kinetin and the auxin analog 2-4D and kept in constant light for 4 weeks. In low auxin concentrations, cuttings produce roots, and this is inhibited by the presence of kinetin. In the WT, inhibition of root formation in the absence of auxin occurred at 1 µM kinetin, while for *35S:miR319b* plants 300 nM kinetin already inhibited root formation. Similarly, while at 100 nM 2-4D, WT required 100 nM kinetin for callus formation, *35S:miR319b* required no kinetin, while *BLS:rTCP4GFP* required 1 µM kinetin to produce callus growth. Shown are representative tissue fragments arranged as a composite image. Red rectangles mark leaves that produced roots. White rectangles mark leaves producing callus.

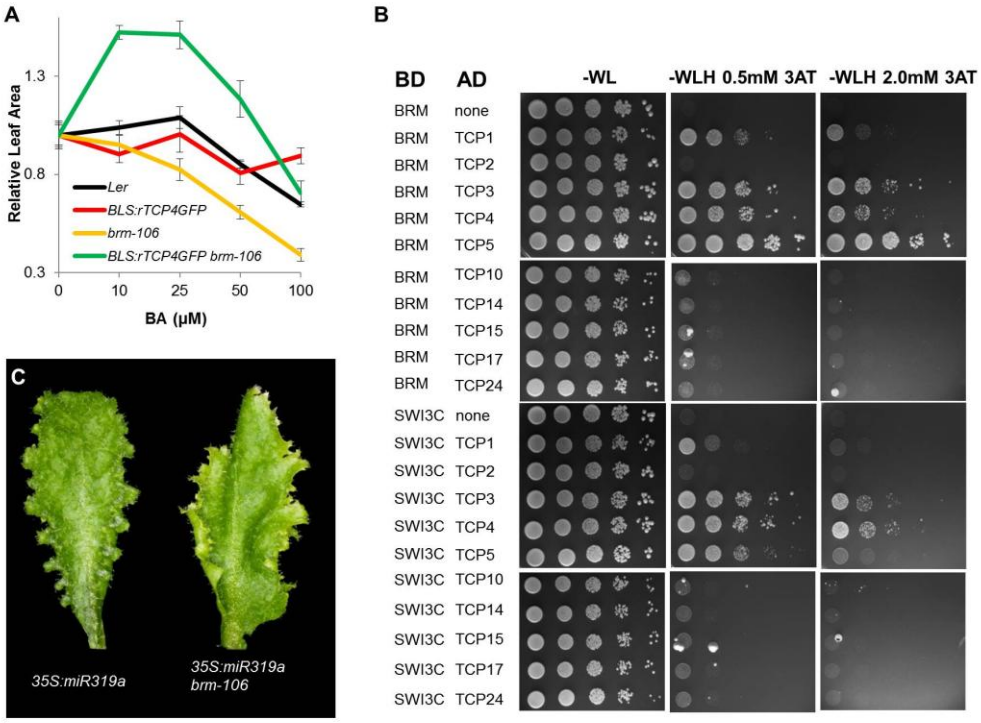

Figure S2, related to Figure 3. Genetic and physical interaction of CIN-TCPs with BRM and the BRM complex component SWI3C. A) The effect of bi-weekly exogenous BA application on the size of leaf 4 (mean leaf size at 0 μM BA is 101.7, 26.3, 94.3, and 31.37 mm^2 for Ler, *BLS:rTCP4GFP*, *brm-106*, and *BLS:rTCP4GFP brm-106*, respectively). Error bars are SE, n=7 to 10. **B)** Serial dilution plating of yeast expressing the N-terminal domain of BRM or full-length SWI3C fused to the GAL4 DNA binding domain (BD) and TCPs fused to the GAL4 activation domain (AD). An empty AD vector was used as a negative control (none). Interactions were probed by assaying growth on media lacking histidine supplemented with 0.5 or 2 mM 3-Amino-1,2,4-triazole (3AT). **C)** Reduction of BRM activity (*brm-106)* caused minor changes to *35S:miR319a* leaf morphology.

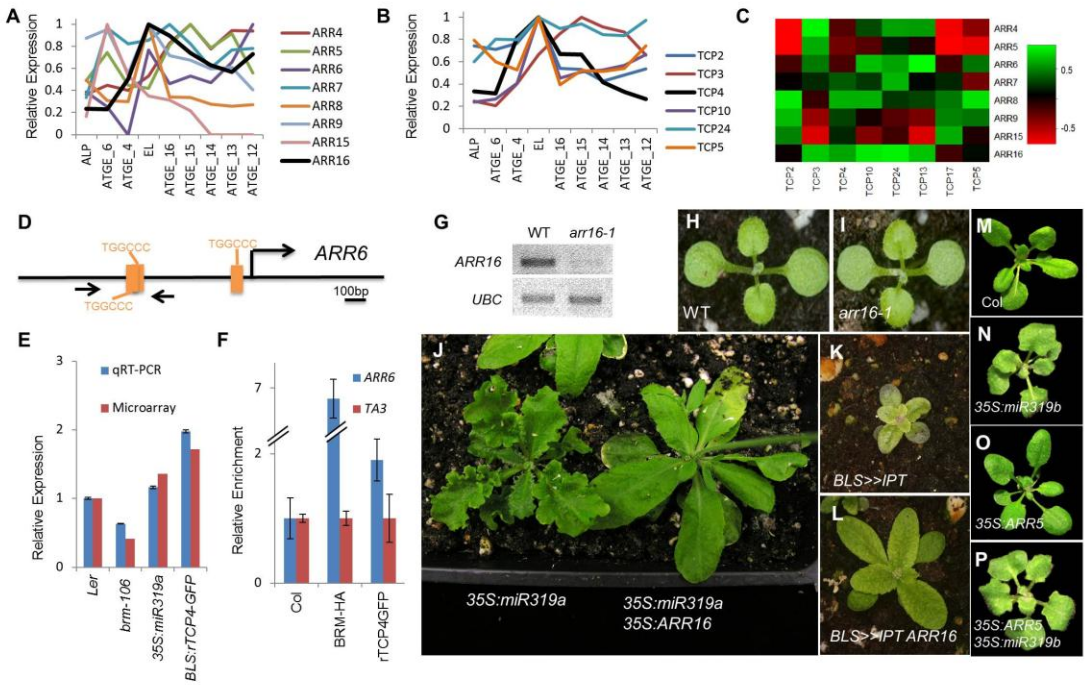

Figure S3, related to Figure 4. The relationship between *CIN-TCPs*, *ARR16* and CK responses in young leaves. A-B) Dynamic expression in gradually maturing leaves (Efroni et al., 2008). The first three samples are comprised of mixed leaf/meristem tissue. *ARR15* and *ARR7* peak early, in agreement with their roles in the shoot meristem (Leibfried et al., 2005). Expression of *ARR16*, as well as *ARR6*, *ARR8* and *ARR9* peaks in rapidly expanding leaves (EL), as does that of several *CIN-TCPs (TCP4*, *TCP5*, and *TCP10)*. **C)** Expression correlation matrix for the A-class ARRs and CIN-TCPs. R values are color-coded. **D)** Promoter of *ARR6* with TCP4 binding motifs. Arrows mark the primers used for the ChIP in (F). **E)** Expression level of *ARR6*, measured using qPCR of 21 DAS apices, and published microarrays (Efroni et al., 2006; Bezhani et al., 2007). Error bars are SE, n=3. **F)** ChIP of ARR6 promoter using *brm-1 pBRM:BRM-HA* and *BLS:rTCP4GFP* with anti-HA and anti-GFP antibodies, respectively. ChIP was repeated twice, and a representative result is shown. Error bars are SE for 3 technical replicates. **G)** Levels of *ARR16* are greatly reduced, but not eliminated in the homozygous T-DNA insertion line SAIL_398_C12. **H-I)** The morphology of 14DAS short-day-grown WT (H) and *arr16-1* (I) plants is very similar. **J)** Five-week-old *35S:miR319* and *35S:ARR16 35S:mir319* plants. **K-L**) Four-week-old *BLS>>IPT* (K) and *BLS>>IPT ARR16* rosettes (L). Note the loss of anthocyanin accumulation and growth retardation triggered by the *BLS>>IPT* in (K). **M-P)** Three-week-old Col (M) *35S:miR319b* (N) *35S:ARR5* (O) and *35S:miR319b*

35S:ARR5 (P) plants. The same result was obtained with two different published *35S:ARR5* lines (Salome et al., 2006; Ren et al., 2009).

Table S1, related to Figure 3. Transcription factors identified in yeast-2-hybrid interaction screen with BRM, SYD and core BRM or SYD complex components.

Table S2, related to Figure 3. Genes coordinately regulated in *brm-101*, and mutants with altered *CIN-TCPs* levels. Fold changes are listed.

Supplemental Experimental Procedures

Mutagenesis and Map based cloning

We tagged TCP4 with GFP to allow identification of response suppressors over modifiers of TCP4 levels. We employed a direct fusion transgene (*BLS:rTCP4GFP*) for the screen, which was identical in phenotype to *rTCP4* expressed from the *BLS* promoter (Efroni et al., 2008).

The phenotype of the three *BLS:rTCP4GFP* suppressors was similar to that of *ffo3*, a mutation previously mapped to chromosome 2 (Levin et al., 1998). Complementation tests revealed the three mutants were allelic with *ffo3*. Fine-mapping placed the *ffo3* mutation in a 200Kb interval flanked by the CD297A and GBF3 markers, a region with 67 annotated genes. By sequencing candidate genes, a missense mutation was identified in a proline in the ATPase domain of the *BRAHMA* (*BRM*) gene. Sequencing of the suppressors also revealed missense mutations in *BRM*, in one case the mutation was identical to that in *ffo3*.

Plasmid Construction

BiFC plasmids

The N-terminal domain of BRM plus the nuclear localization signal (NLS) from VirD was cloned into to pCL113 to obtain 35S:cYFP-BRMN; the BRM NLS is in the C terminal domain of this protein and hence missing from BRMN. Full-length TCP4 and SWI3C were cloned into pCL112 to obtain 35S:nYFP-TCP4 or 35S:nYFP SWI3C. 35S:2xmCherry was cloned into pEarley102 (Earley et al., 2006) as previously described.

Yeast two Hybrid Screen

All baits were in the Invitrogen pDEST32 vector except for the SYD N-terminal domain which was in pDBleu. The yeast strain used was PJ69-4A. The six baits used

were: BRM N-terminal domain (At2g46020, aa 1-976), SYD N-terminal domain (At2g28290, aa 1-657), SWI3C C-terminal domain (At1g21700, aa 383-807), full length SWI3A (At2g47620, aa 1-512), full-length SWI3B (At2g33610, aa 1-469), and as well as full length BSH (At3g17590, aa 1-242). Each bait host was transformed individually with one of the 1,400 transcription factors in the prey vector. The resulting 6 x1,400 strains plus relevant positive and negative controls were spotted on SD media lacking Leu (L) and Trp (W) to assess growth in the absence of selection. In addition, cells were spotted on selection media (-Trp -Leu –His SD media containing 0.1mM, 0.5mM, 1mM, 2mM) of 3-Amino-1,2,4-triazole (3-AT; Sigma, A8056). The growth of all interactors on the different media was scored. The interaction screen was performed at least 2 times for each bait/prey combination.

To confirm the interactions between TCPs and BRM/SWI3C, pDEST32-BRM-N or pDEST32-SWI3C-C bait and pDEST22-TCP prey were co-transformed into the PJ69-4A yeast strain. Transformed cells were plated on -Trp -Leu SD media. The resulting colonies were grown in -Trp -Leu SD liquid media overnight, adjusted for equal cell density, serially diluted ($1\sim 10^{-4}$) and spotted on selection media (-Trp -Leu –His /SD media with 0.1mM, 0.5mM, 1mM, 2mM 3-AT).

Primers used after reverse transcription (RT) or chromatin immunoprecipitaion (ChIP) for semi-quantitative (semiQ) or quantitative (Q) PCR.

RT-UBC-Q-F TAGCATTGATGGCTCATCCTGA
RT-UBC-Q-R TTGTGCCATTGAATTGAACCC
RT-ARR16-Q-F TGCAAAGTGACAACAGCAGA
RT-ARR16-Q-R CCAGGCATACAGTAATCGGA
RT-EIF4A1-Q-F AAACTCAATGAAGTACTTGAGGGACA
RT-EIF4A1-Q-R TCTCAAAACCATAAGCATAAATACCC
RT-ARR6-Q-F TGAAGCCGGTAAAACTCTCG
RT-ARR6-Q-R GTTTGAGCTGCGAGTGAACA
ChIP-TA3-Q-F CTGCGTGGAAGTCTGTCAAA
ChIP-TA3-Q-R CTATGCCACAGGGCAGTTTT
ChIP-ARR16 -Q-F CCTCCTTTTGTTTTGATTCGAC
ChIP-ARR16-Q-R TCACAAATGGACCACCAATC
ChIP-ARR6-Q-F TCCCTCCGGTTCACTGAGTA
ChIP-ARR6-Q-R CCACCTTTTCATTCACCAAA
ChIP-NC2-Q-F ACTCGCATCTTCGGTTTGTT
ChIP-NC2-Q-R GGATTGAAGGAAAGCCAGAA

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