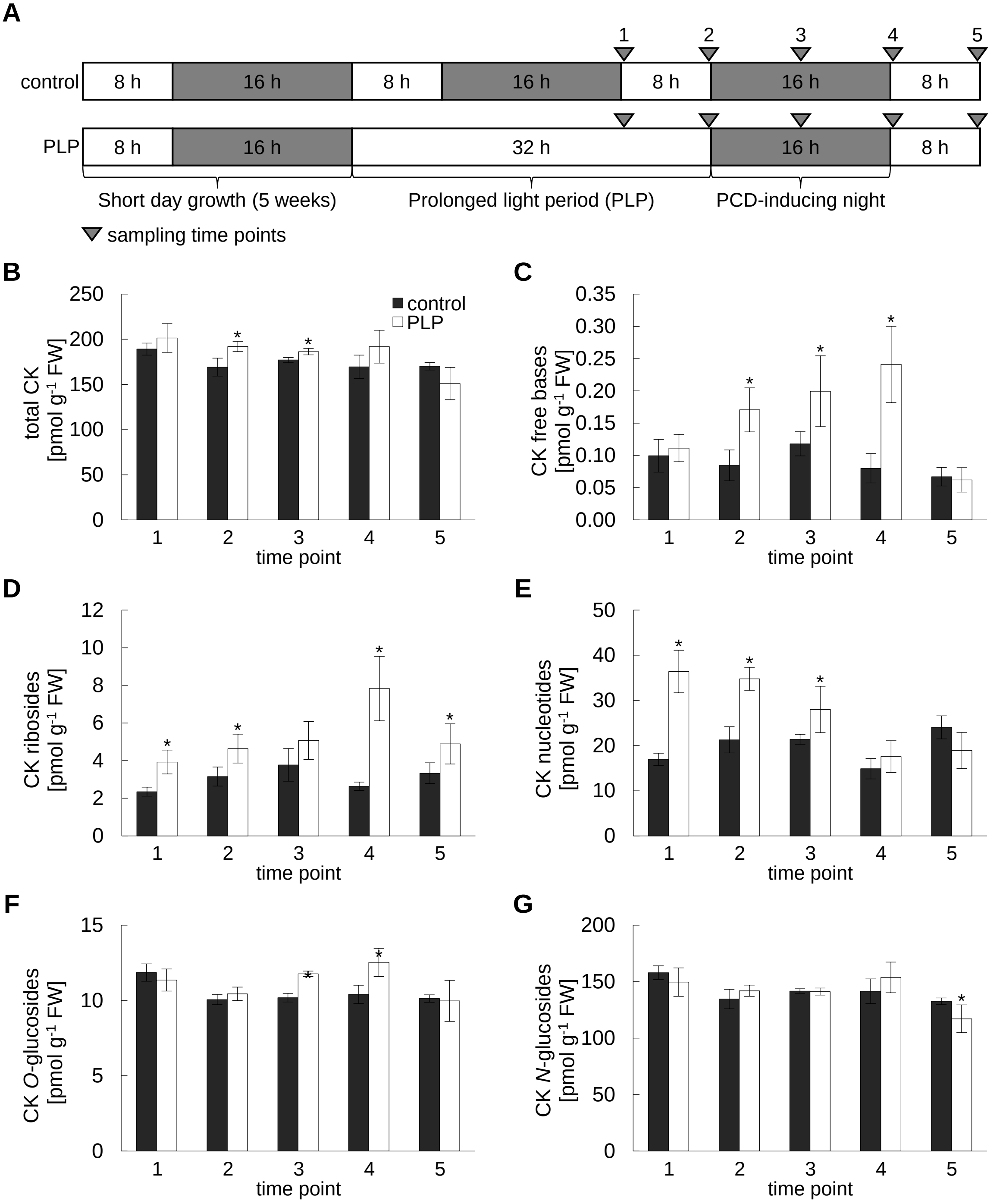

Figure 1. Photoperiod stress increases the CK concentration in wild-type plants. (A) Schematic overview of sampling time points for CK measurements. 5-weeks-old wild-type plants were cultivated under SD conditions and were further cultivated under these conditions (control) or were exposed to a prolonged light period (PLP) of 32 h. (B - G) Concentration of total CK (B), CK free bases (C), CK ribosides (D), CK nucleotides (E), CK *O*-glucosides (F) and CK *N*-glucosides (G) in control and PLP samples at the time points depicted in A. Stars indicate a statistically significant difference between PLP and the respective control samples at the given time point (1 to 5) in a paired Student's t-test ($p \leq 0.05$). Values are given as pmol g-1 FW ± SD (n = 5). The complete data set is shown in Table S1.

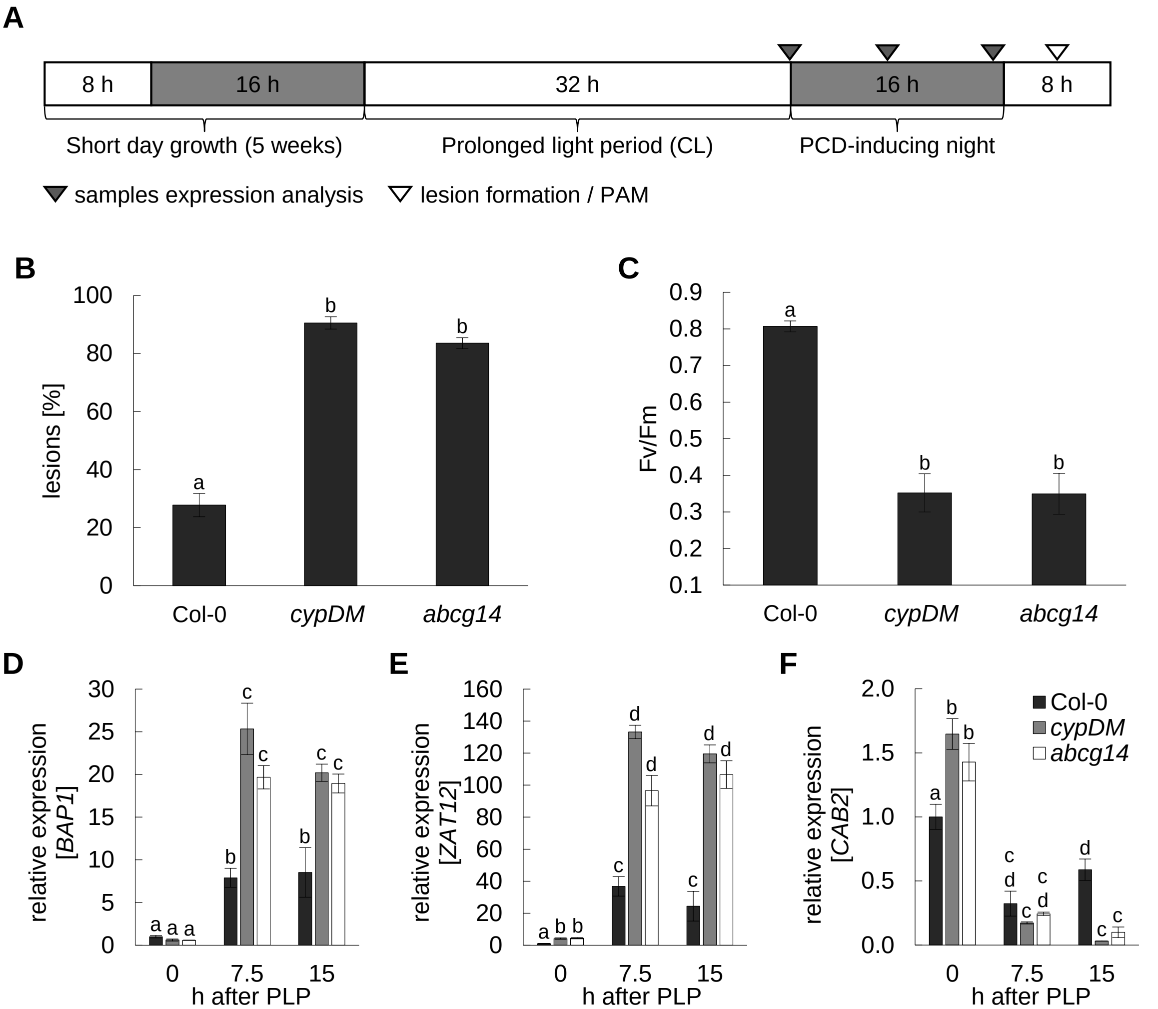

Figure 2. Plants deficient in *t*Z-type CKs are strongly affected by photoperiod stress. (A) Schematic overview of photoperiod stress treatment. Arrow points indicate sampling time points for the different analysis. (B) Lesion formation of leaves in 5-weeks-old Col-0, *cypDM* and *abcg14* plants the day after the PCD-inducing night (one-way ANOVA; $p \leq 0.05$; n = 15). (C) Photosystem II maximum quantum efficiency (Fv/Fm) of leaves the day after the PCD-inducing night (Paired Wilcoxon test; $p \leq 0.05$; n = 15). (D - F) Expression of marker genes (*BAP1*, *ZAT12*, *CAB2*) 0 h, 7.5 h and 15 h after PLP treatment. Letters indicate statistical groups (two-way ANOVA; $p \leq 0.05$; $p \leq 0.05$; $n \geq 3$). The expression level of wild type at timepoint 0 h was set to 1. Error bars indicate SE. Pictures of representative plants exposed to a 24-h prolongation of the light period are shown in Fig. S1A.

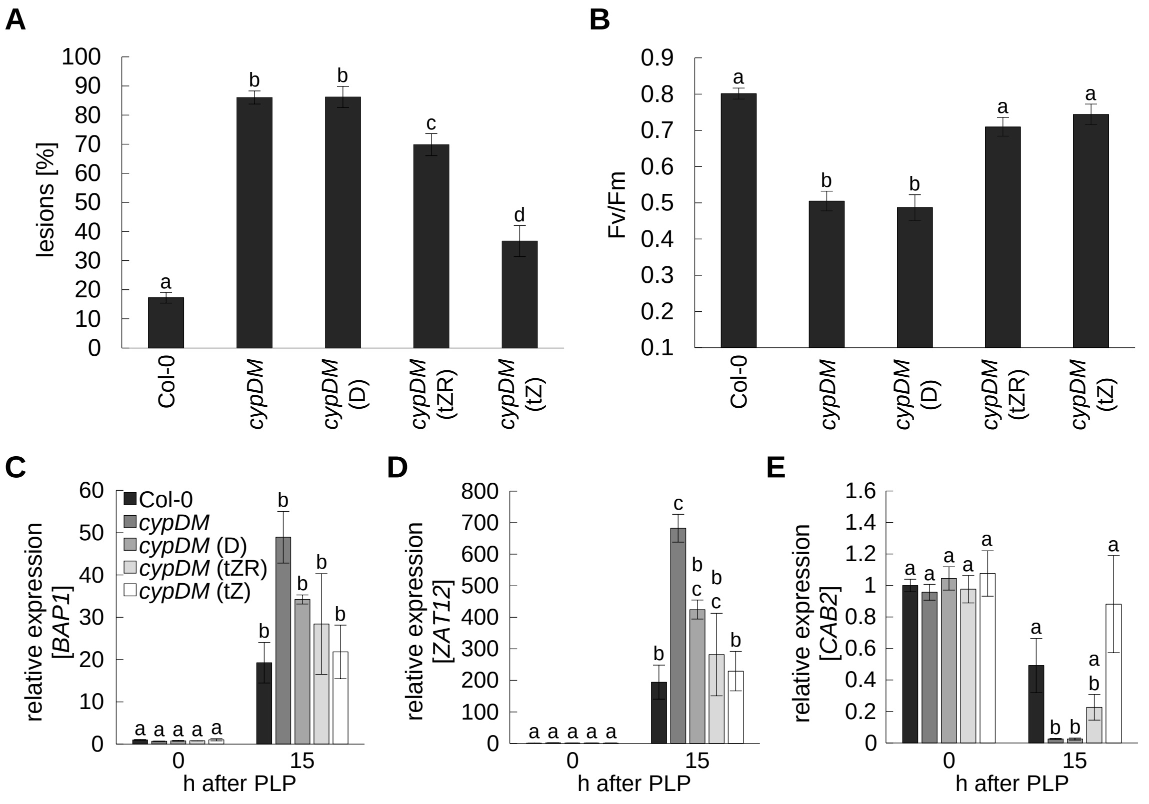

Figure 3. Pretreatment of CK-deficient plants with *t*Z-type CKs reduces the damage caused by photoperiod stress. *cypDM* mutant plants were watered-daily for five weeks with 10 µM *t*Z, 10 µM *t*ZR or DMSO solvent control (D). Thereafter, the consequences of PLP treatment on these plants were compared to untreated *cypDM* and wild-type plants. (A) Percentage of lesion formation in 5-weeks-old short day-grown plants the day after PLP treatment (one-way ANOVA; $p \leq 0.05$; n = 12). (B) Photosystem II maximum quantum efficiency (Fv/Fm) of leaves evaluated in A (one-way ANOVA; $p \leq 0.05$; n = 15). (C - E) Expression of marker genes (*BAP1*, *ZAT12*, *CAB2*) 0 h and 15 h after PLP treatment (one/two-way ANOVA; $p \leq 0.05$; n ≥ 3). The expression level of wild type at the end of the PLP treatment (0 h) was set to 1. Abbreviations: D, DMSO; *t*Z, *trans*-zeatin; *t*ZR, *trans*-zeatin-riboside. Letters indicate statistical groups ($p \leq 0.05$). Error bars indicate SE. Pictures of representative plants tested in A and B after PLP treatment are shown in Fig. S1B.

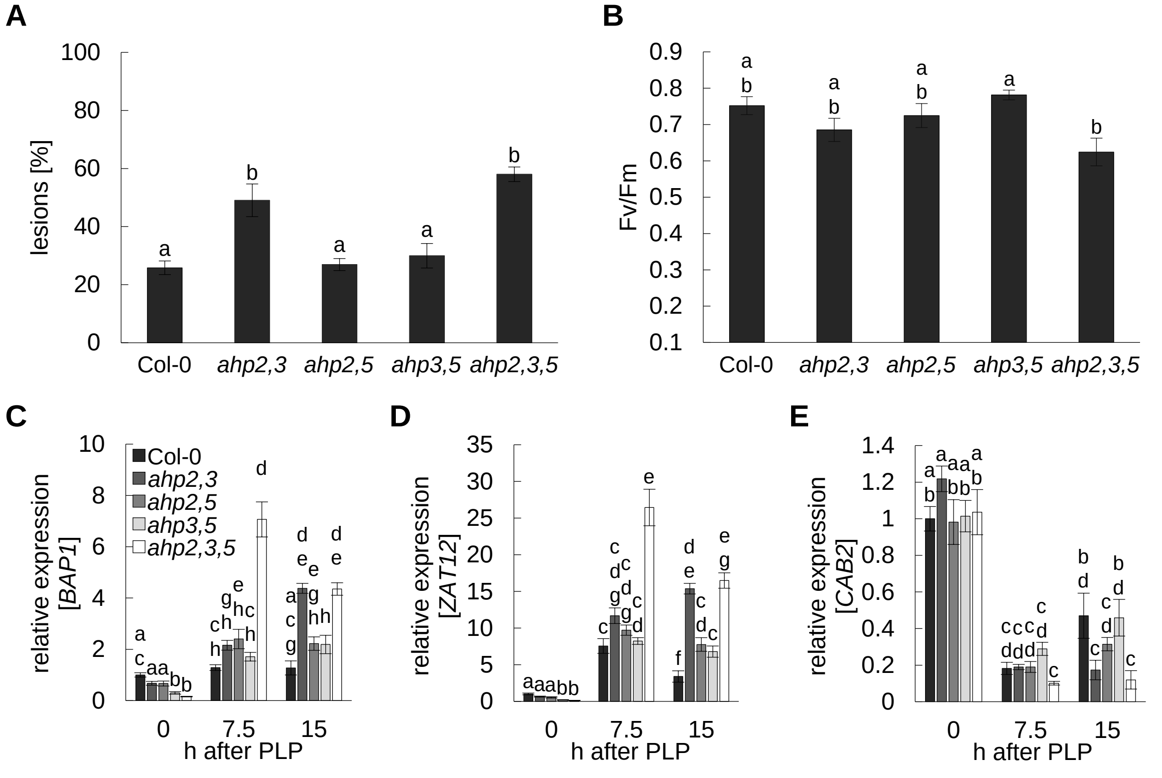

Figure 4. AHP2, AHP3 and AHP5 act redundantly during photoperiod stress. (A) Lesion formation in 5-weeks-old Col-0 and *ahp* mutant plants the day after PLP treatment (one-way ANOVA; $p \leq 0.05$; n = 15). (B) Photosystem II maximum quantum efficiency (Fv/Fm) of leaves the day after PLP treatment (one-way ANOVA; $p \leq 0.05$; n = 15). (C - E) Relative expression of marker genes (*BAP1*, *ZAT12*, *CAB2*) 0 h, 7.5 h and 15 h after PLP treatment. The expression level of wild type at time point 0 h was set to 1. Letters indicate statistical groups (two-way ANOVA; $p \leq 0.05$; $n \geq 3$). Error bars indicate SE. Pictures of representative plants tested in A and B after PLP treatment are shown in Fig. S1C.

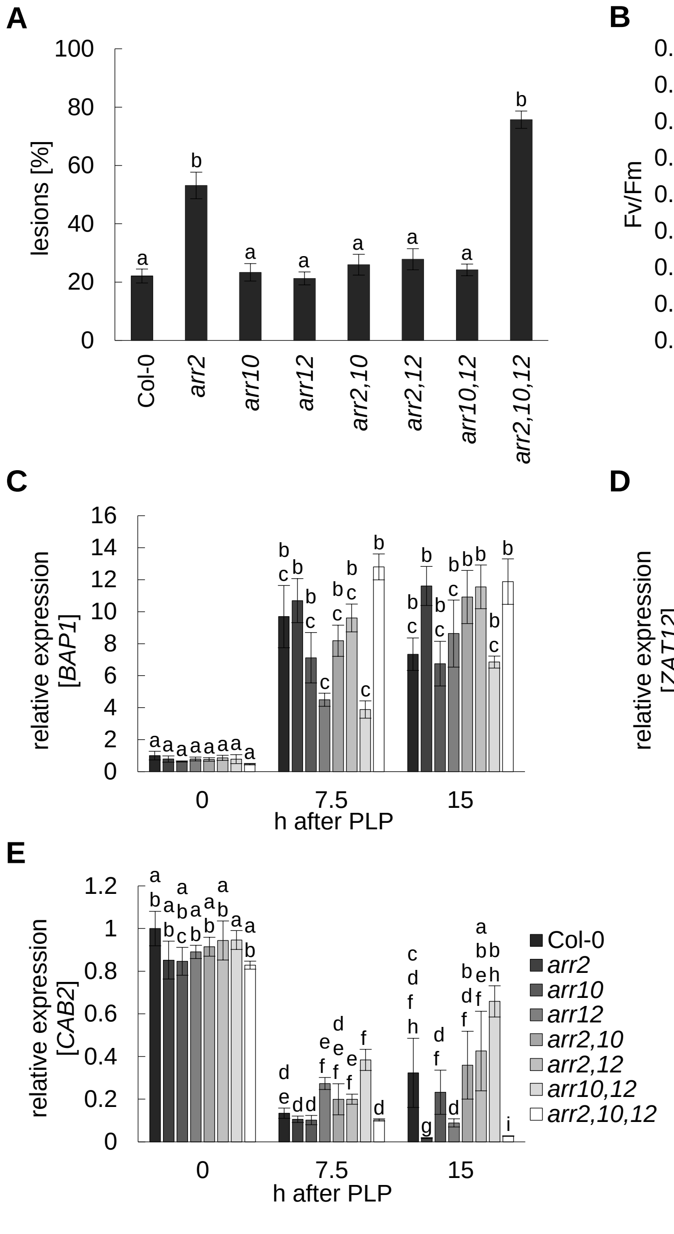

Figure 5. ARR2, ARR10 and ARR12 interact to respond to photoperiod stress. (A) Quantification of lesion forming leaves in 5-weeks-old Col-0 and type-B ARR mutants the day after the PLP treatment (one-way ANOVA; p ≤ 0.05; n = 15). (B) Photosystem II maximum quantum efficiency (Fv/Fm) of leaves the day after PLP treatment (one-way ANOVA; p ≤ 0.05; n = 15). (C - E) Relative expression of marker genes (*BAP1*, *ZAT12*, *CAB2*) 0 h, 7.5 h and 15 h after PLP treatment. The expression level of wild type at the end of the PLP treatment (0 h) was set to 1. Letters indicate statistical groups (two-way ANOVA/Paired Wilcoxon test; p ≤ 0.05; n ≥ 3). Error bars indicate SE. Pictures of representative plants tested in A and B after PLP treatment are depicted in Fig. S1D.

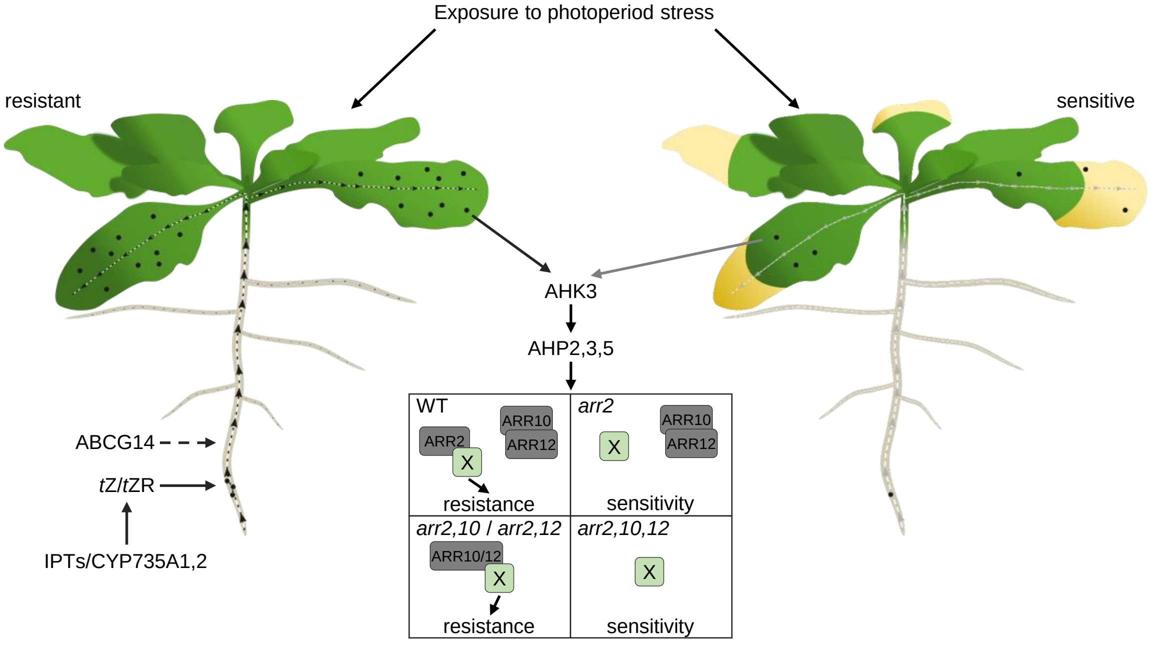

Figure 6. Model showing the role of CK in regulating the response to photoperiod stress. During exposure to photoperiod stress, wild-type plants (left) increase their CK levels. IPT and CYP735A proteins increase synthesis of *t*Z-type CK (black balls) in roots which are transported via ABCG14 to the shoot (black dashed line) where they activate CK signaling mainly through AHK3. AHP2, AHP3 and AHP5, and ARR2, ARR10 and ARR12. Impairment of either *t*Z-type CK synthesis or transport (less molecules and grey dashed lines) induce weaker CK signaling causing higher sensitivity to photoperiod stress (right plant). The central four rectangles show a model for type-B ARR-dependent regulation of the response. It is proposed that ARR2, ARR10 and ARR12 interact in the wild type (WT) with a yet unknown interaction partner (X) essential for photoperiod stress resistance (rectangle top left). The affinity of ARR2 to X is higher than the affinities of ARR10 and ARR12 to X. Additionally, ARR10 and ARR12 directly or indirectly interact with each other. In *arr2* plants (rectangle top right), X does not have an interaction partner and thus would be unable to function while ARR10 and ARR12 still interact with each other leading to the formation of the photoperiod stress syndrome. Resistance of *arr2,10* and *arr2,12* plants (rectangle bottom left) is caused by the loss of ARR10-ARR12 association and the resulting interaction of X with ARR10 or ARR12. Ultimately, the enhanced photoperiod stress sensitivity of *arr2,10,12* plants (rectangle bottom right) would be caused by the complete loss of interaction partners for X.

Table S1. Changes in CK concentration by PLP treatment. The indicated time points (control/PLP 1 to 5) correspond to those shown in Figure 1A. Bold numbers indicate statistically significant difference in PLP samples compared to the respective controls at the same time point in a paired Student's t-test ($p \leq 0.05$). Values are given as pmol g-1 FW ± SD (n = 5). Concentrations below detection limit are referred to as <LOD. RMP, riboside monophosphates; *O*G, *O*-glucosides; R*O*G, riboside-*O*-glucoside; 7G, 7-glucoside; 9G, 9-glucoside.

	1		**2**		**3**		**4**		**5**	
	control	**PLP**	**control**	**PLP**	**control**	**PLP**	**control**	**PLP**	**control**	**PLP**
iP	0.090±0.027	0.103±0.021	0.078±0.024	**0.161±0.034**	0.115±0.018	**0.193±0.056**	0.074±0.022	**0.234±0.059**	0.063±0.014	0.059±0.019
iPR	0.24 ± 0.06	**0.75 ± 0.11**	0.40 ± 0.10	**0.87 ± 0.10**	0.51 ± 0.05	0.61 ± 0.13	0.27 ± 0.05	**1.11 ± 0.25**	0.56 ± 0.16	0.54 ± 0.17
iPRMP	5.13 ± 0.74	**21.61± 1.81**	10.86± 1.77	**20.66± 1.46**	10.98± 1.02	14.69± 3.15	5.27 ± 1.22	6.56 ± 1.97	14.44± 1.19	**10.32± 2.84**
iP7G	22.04± 1.25	23.56± 2.89	20.03± 0.74	21.10± 1.31	23.19± 1.66	23.91± 0.85	20.64± 0.58	**24.65± 1.92**	21.19± 0.98	20.59± 2.74
iP9G	1.90 ± 0.11	2.10 ± 0.25	1.70 ± 0.04	1.91 ± 0.19	2.01 ± 0.22	2.04 ± 0.07	1.78 ± 0.11	1.96 ± 0.18	1.76 ± 0.10	1.58 ± 0.23
***t*Z**	0.009±0.003	0.009±0.002	0.007±0.001	**0.010±0.002**	0.003±0.001	**0.006±0.001**	0.006±0.001	0.007±0.001	0.004±0.001	0.003±0.001
***t*ZR**	1.67 ± 0.19	**3.03 ± 0.53**	2.43 ± 0.43	**3.51 ± 0.77**	2.90 ± 0.90	4.04 ± 0.89	1.90 ± 0.24	**3.36 ± 0.78**	2.59 ± 0.47	2.73 ± 0.46
***t*ZRMP**	8.11 ± 1.20	**13.25± 3.13**	7.56 ± 1.65	**11.89± 2.17**	6.01 ± 0.31	**8.88 ± 1.75**	6.04 ± 1.13	4.83 ± 1.25	7.85 ± 1.61	**3.98 ± 0.91**
***t*Z*O*G**	6.77 ± 0.43	6.47 ± 0.49	5.64 ± 0.31	6.02 ± 0.24	5.37 ± 0.17	5.88 ± 0.26	5.52 ± 0.46	**6.42 ± 0.61**	5.52 ± 0.16	**4.67 ± 0.57**
***t*ZR*O*G**	1.10 ± 0.05	1.08 ± 0.11	0.98 ± 0.08	1.06 ± 0.05	0.90 ± 0.04	**1.24 ± 0.09**	1.05 ± 0.08	**1.46 ± 0.17**	0.93 ± 0.04	0.90 ± 0.15
***t*Z7G**	87.53± 3.94	81.79± 6.96	74.79± 5.97	79.45± 2.35	77.34± 2.45	76.07± 2.10	79.79± 6.84	85.25± 8.16	73.74± 1.31	**63.65± 6.76**
***t*Z9G**	26.97± 0.60	**23.71± 2.26**	22.16± 1.99	22.78± 0.76	20.82± 1.05	20.23± 0.73	22.18± 2.32	24.80± 2.21	19.62± 1.60	16.82± 2.49
DHZ	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
DHZR	0.026±0.007	**0.053±0.015**	0.027±0.008	**0.049±0.010**	0.031±0.009	**0.075±0.021**	0.023±0.002	**0.138±0.037**	0.023±0.005	**0.067±0.020**
DHZRMP	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
DHZ*O*G	0.059±0.005	0.057±0.003	0.054±0.002	0.055±0.006	0.048±0.006	**0.062±0.005**	0.042±0.006	**0.064±0.006**	0.041±0.003	0.051±0.008
DHZR*O*G	0.059±0.005	0.062±0.013	0.067±0.006	0.072±0.015	0.057±0.009	**0.096±0.021**	0.056±0.007	**0.122±0.036**	0.056±0.010	0.068±0.017
DHZ7G	5.73 ± 0.17	5.56 ± 0.43	5.13 ± 0.20	5.58 ± 0.39	5.11 ± 0.20	**6.11 ± 0.35**	4.70 ± 0.28	**6.02 ± 0.86**	4.46 ± 0.24	4.38 ± 0.54
DHZ9G	0.17 ± 0.03	0.14 ± 0.01	0.14 ± 0.01	0.12 ± 0.01	0.11 ± 0.02	0.10 ± 0.01	0.11 ± 0.01	**0.16 ± 0.04**	0.11 ± 0.01	0.10 ± 0.02
***c*Z**	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
***c*ZR**	0.41 ± 0.04	**0.10 ± 0.02**	0.30 ± 0.10	0.21 ± 0.06	0.33 ± 0.02	0.34 ± 0.03	0.44 ± 0.01	**3.22 ± 0.92**	0.16 ± 0.03	**1.55 ± 0.46**
***c*ZRMP**	3.72 ± 0.58	**1.54 ± 0.15**	2.84 ± 0.42	2.22 ± 0.46	4.39 ± 0.23	4.43 ± 0.57	3.56 ± 0.19	**6.18 ± 0.81**	1.74 ± 0.34	**4.62 ± 0.88**
***c*Z*O*G**	1.14 ± 0.08	1.20 ± 0.12	1.02 ± 0.07	**1.18 ± 0.09**	1.16 ± 0.11	1.29 ± 0.06	0.99 ± 0.04	**1.29 ± 0.04**	1.04 ± 0.08	**1.72 ± 0.42**
***c*ZR*O*G**	2.73 ± 0.16	2.48 ± 0.21	2.30 ± 0.13	2.07 ± 0.17	2.64 ± 0.12	**3.20 ± 0.30**	2.73 ± 0.11	3.17 ± 0.38	2.54 ± 0.25	2.56 ± 0.31
***c*Z7G**	13.32± 0.99	12.53± 1.36	10.46± 0.46	10.82± 1.04	12.88± 0.79	12.59± 0.93	12.13± 1.30	10.67± 1.22	11.46± 1.05	**9.83 ± 0.73**
***c*Z9G**	0.28 ± 0.01	**0.23 ± 0.03**	0.21 ± 0.02	**0.17 ± 0.02**	0.24 ± 0.03	**0.17 ± 0.01**	0.22 ± 0.02	0.20 ± 0.02	0.25 ± 0.02	**0.18 ± 0.02**

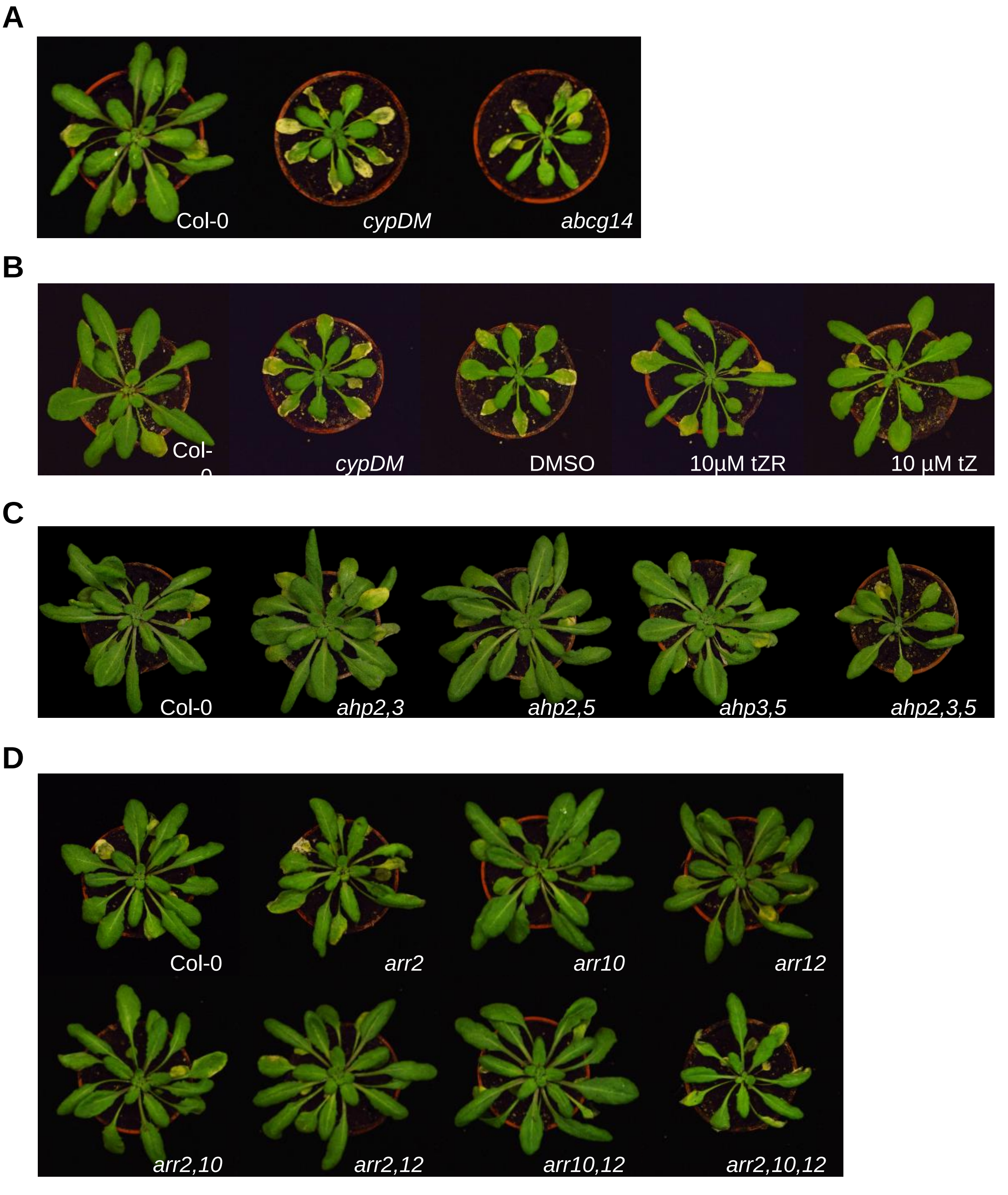

Figure S1. Representative plants after PLP treatment. The pictures illustrate the phenotype of plants used for experiments shown in Fig. 2 to Fig. 5. Pictures were taken two days after PLP treatment and belong to Fig. 2 (A), Fig. 3 (B), Fig. 4 (C), and Fig. 5 (D). Details of the experiments can be found in the legends of the respective figures.

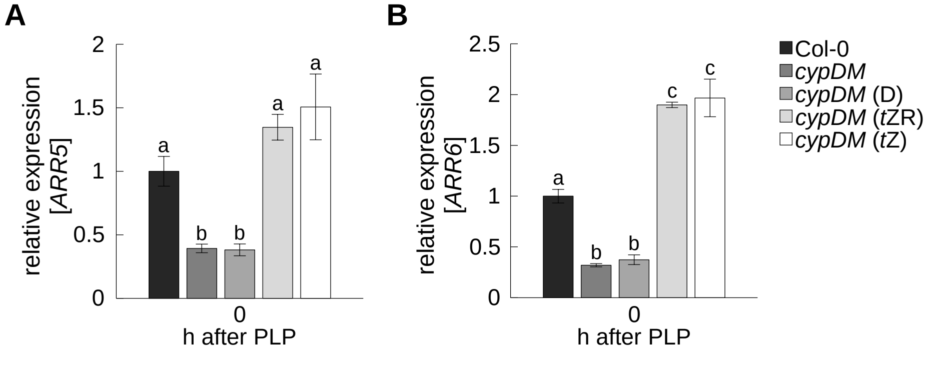

Figure S2. Pretreatment of CK-deficient plants with *t*Z-type CKs rescues differential expression of CK response genes. Expression of ARR5 (A) and ARR6 (B) 0 h after PLP treatment relative to wild type . Letters indicate statistical groups (one-way ANOVA; $p \leq 0.05$; $p \leq 0.05$; $n \geq 3$).