Priming and memory by photoperiod stress in *Arabidopsis thaliana*

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Summary

In the frame of this thesis, the effects of a suddenly occurring prolongation of the light period leading to photoperiod stress in *Arabidopsis thaliana* were investigated. In particular, the photoperiod stress response is characterised through three approaches. First, the response of plants to a prolonged light (PL) period of 0.5 h to 8 h is investigated in sensitivity experiments. Second, the effects of a photoperiod stress stimulus on plant responses to a subsequently occurring similar photoperiod stress are examined in *cis*-priming experiments. Third, the impact of photoperiod stress on plant responses to infections by pathogens are studied in *trans*-priming experiments.

Transcriptome analysis by RNA-seq showed that a prolongation of the light period by one hour is sufficient to alter the expression level of 22 genes at the end of the following night in four-week-old short day-grown plants. The expression of the photoperiod stress marker genes *ZINC FINGER OF ARABIDOPSIS THALIANA12* (*ZAT12*) and *BON ASSOCIATION PROTEIN1* (*BAP1*) was not significantly affected by PL periods up to 2.5 h. An extension of the PL period by 2.5 h, 4 h or 8 h is associated with an increase in the number of genes regulated at the end of the night that follows a PL period. Three genes regulated independent of the duration of the PL period were identified: *PHYTOCHROME INTERACTING FACTOR4*, *BES1-INTERACTING MYC-LIKE1*, and *COLD REGULATED 314 INNER MEMBRANE1*.

The *cis*-priming experiments revealed that photoperiod stress induced by a 4 h-prolonged light period (priming stimulus) causes in wild-type plants a different response to a second photoperiod stress (triggering stimulus). The first photoperiod stress induces the expression of *ZAT12* and *BAP1*, an accumulation of peroxides, and a decrease in catalase activity. These responses are suppressed in response to a second photoperiod stress. The suppression to a second photoperiod stress lasts for several days over a stress-free lag phase indicating the existence of a memory. Transcriptome analysis by RNA-seq revealed different kinds of memory genes for photoperiod stress showing a sustained, altered, or sensitized expression when exposed to a 4 h-PL period after a first similar PL period. A prolongation of the light period up to 2 h results only in a weak photoperiod stress response in wild-type plants and is not sufficient to prime (induce) the plants' resistance suggesting that the first stimulus needs to induce a substantial response to be memorized. The responsiveness of Arabidopsis wild-type plants to photoperiod stress and their ability to become primed by a photoperiod stress depends on their developmental phase. Analysis of plants of different age exposed to a 4 h-PL period showed that only three- to five-week-old plants responded to and were primed by the PL period indicated by induction of *ZAT12* and *BAP1* or by accumulation of peroxides and suppression of these responses, respectively. The memory of photoperiod stress-sensitive mutants *arabidopsis histidine kinase2* (*ahk2*) *ahk3* and *circadian clock associated1* (*cca1*) *long elongated hypocotyl* (*lhy*) is shorter than the memory of wild-type plants indicating that a functional cytokinin signalling and circadian clock are required for maintaining memory.

Trans-priming experiments revealed that photoperiod stress induced by an 8 h-PL period improves resistance of wild-type plants against infections with the bacterium *Pseudomonas syringae* pv. *tomato* DC3000 and the fungus *Botrytis cinerea*. Photoperiod stress-induced resistance against *P. syringae* and *B. cinerea* requires salicylic acid- and jasmonic acid-related responses as revealed by analysing mutants

defective in salicylic acid or jasmonic acid biosynthesis/signalling. *NONEXPRESSOR OF PATHOGENESIS RELATED GENES1* (*NPR1*) is crucial for the oxidative burst-like response of photoperiod stress, since *npr1* mutants do not accumulate peroxides during photoperiod stress and several genes were differently regulated in these mutants. Further investigation is necessary to fully understand the molecular mechanisms involved in photoperiod stress-induced resistance against pathogens.

Zusammenfassung

Im Rahmen dieser Arbeit wurden die Auswirkungen einer plötzlich auftretenden längeren Lichtperiode, die in *Arabidopsis thaliana* zu photoperiodischem Stress führt, untersucht. Die photoperiodische Stressantwort wurde mit Hilfe von drei experimentellen Ansätzen untersucht. Als Erstes wurde die Pflanzenantwort auf verlängerte Lichtperioden von einer halben Stunde bis acht Stunden in Sensitivitätsexperimenten analysiert. Als Zweites wurden die Effekte von photoperiodischem Stress auf Pflanzenantworten im Hinblick auf einen identischen photoperiodischen Stress in *cis*-Priming Experimenten evaluiert. Im dritten Teil der Arbeit wurden die Auswirkungen eines photoperiodischen Stresses auf Pflanzenantworten während einer Infektion mit Pathogenen in *trans*-Priming Experimenten studiert.

Transkriptomanalysen mittels RNA-Sequenzierung zeigten, dass bereits eine Verlängerung der Lichtperiode von lediglich einer Stunde ausreichend ist, um am Ende der folgenden Nacht die Expression von 22 Genen in Pflanzen, die unter Kurztag-Bedingungen angezogen wurden, zu beeinflussen. Die Expression von Markergenen photoperiodischen Stresses, *ZINC FINGER OF ARABIDOPSIS THALIANA12* (*ZAT12*) und *BON ASSOCIATION PROTEIN1* (*BAP1*), wurde nicht signifikant beeinflusst, wenn die Lichtperioden um weniger als vier Stunden verlängert wurden. Eine Verlängerung der Lichtperiode um zweieinhalb, vier oder acht Stunden war mit einem Anstieg der Expression von Genen, die am Ende der auf die verlängerte Lichtperiode folgenden Nacht reguliert sind, assoziiert. Ein Vergleich der Gene, die unter verschiedenen verlängerten Lichtperioden reguliert sind, ermöglichte die Identifikation von drei Genen, die unabhängig von der Länge der Lichtperiode reguliert wurden: *BES1-INTERACTING MYC-LIKE1*, *PHYTOCHROME INTERACTING FACTOR4* und *COLD REGULATED 314 INNER MEMBRANE1*.

Cis-priming Experimente zeigten, dass photoperiodischer Stress, der durch eine vierstündige Verlängerung der Lichtperiode (*priming stimulus*) ausgelöst wurde, wildtypische Pflanzen auf einen zweiten photoperiodischen Stress (*triggering stimulus*) vorbereitete. Der erste photoperiodische Stress induzierte die Expression von *ZAT12* und *BAP1*, die Akkumulierung von Peroxiden und reduzierte die Katalase-Aktivität. Diese Antworten wurden bei einem zweiten photoperiodischen Stress unterdrückt, auch nach einer stressfreien Lag-Phase, was auf ein „Gedächtnis“ hinweist. Transkriptomanalysen identifizierten verschiedene Arten von Gedächtnis-Genen für photoperiodischen Stress, die eine anhaltende, veränderte oder sensitivere Expression zeigten, wenn die Pflanzen einer zweiten vierstündigen Verlängerung der Lichtperiode ausgesetzt wurden. Eine Verlängerung der Lichtperiode von bis zu zwei Stunden resultierte nicht in einem signifikanten photoperiodischen Stress in Wildtyp-Pflanzen und war nicht ausreichend, um Resistenz zu induzieren (*priming*). Dies deutet darauf hin, dass der erste Stimulus eine substanzielle Antwort auslösen muss, um in „Erinnerung“ zu bleiben. Die Reaktionsfähigkeit von Wildtyp-Pflanzen auf photoperiodischen Stress und deren Fähigkeit, durch photoperiodischen Stress geprimed zu werden, hängt von ihrem Entwicklungsstatus ab: Ausschließlich drei bis fünf Wochen alte Pflanzen reagieren auf eine verlängerte Lichtperiode mit Priming, was aus der Induktion von *ZAT12* und *BAP1* oder der Akkumulierung von Peroxiden, sowie der Unterdrückung dieser Antworten hervorging. Das Gedächtnis der Mutanten *arabidopsis histidine kinase2* (*ahk2*) *ahk3* und *circadian clock associated1* (*cca1*) *long elongated hypocotyl* (*lhy*), die besonders sensitiv für

photoperiodischen Stress sind, ist kürzer als das Gedächtnis von Wildtyp-Pflanzen. Dies deutet darauf hin, dass ein funktioneller Cytokinin-Signalweg sowie eine intakte circadiane Uhr für die Aufrechterhaltung des pflanzlichen Gedächtnisses notwendig sind.

Trans-priming Experimente zeigten, dass photoperiodischer Stress, der durch eine achtstündige Verlängerung der Lichtperiode induziert wird, die Resistenz von Pflanzen gegenüber Infektionen mit *Pseudomonas syringae* pv. *tomato* DC3000 und *Botrytis cinerea* verbessert. Die durch photoperiodischen Stress induzierte Resistenz gegenüber *P. syringae* und *B. cinerea* erfordert Salicylsäure- und Jasmonsäure-abhängige Antworten, wie durch Analyse entsprechender Arabidopsis-Mutanten gezeigt werden konnte. *NONEXPRESSOR OF PATHOGENESIS RELATED GENES1* (*NPR1*) ist wichtig für die pflanzliche Antwort auf photoperiodischen Stress, der einem oxidativen Burst ähnelt. Dies zeigte sich daran, dass in der *npr1*-Mutante keine Peroxide nach photoperiodischem Stress akkumulierten und viele Gene in dieser Mutante anders als in Wildtyp-Pflanzen reguliert waren. Weitere Untersuchungen sind notwendig, um die molekularen Mechanismen, die der durch photoperiodischen Stress induzierten Resistenz zu Grunde liegen, besser zu verstehen.

List of publications

Significant parts of the work presented in this thesis is being prepared for publication:

Roeber-Terstegen, V. M., Illgen, S., Schmülling, T. and Cortleven, A., Priming and memory in response to photoperiod stress in *Arabidopsis thaliana*. *Manuscript in preparation. Journal to be determined.*

Roeber-Terstegen, V. M., Illgen, S., Lortzing, V., Schmülling, T. and Cortleven, A., Priming by photoperiod stress prepares *Arabidopsis thaliana* for future pathogen attacks. *Manuscript in preparation. Journal to be determined.*

In addition to the work presented in this thesis, the following publications resulted from the work during my dissertation time:

Roeber, V. M., Bajaj, I., Rohde, M., Schmülling, T. and Cortleven, A. (2021) Light acts as a stressor and influences abiotic and biotic stress responses in plants. *Plant Cell Environ*, 44, 645-664. https://doi.org/10.1111/pce.13948

Roeber, V. M., Schmülling, T. and Cortleven, A. (2022) The photoperiod: Handling and causing stress in plants. *Front. Plant Sci.*, 12, 781988. https://doi.org/10.3389/fpls.2021.781988

Cortleven. A., Roeber, V. M., Frank, M., Bertels, J., Lortzing, V., Beemster, G. T. S. and Schmülling, T. (2022) Photoperiod stress in *Arabidopsis thaliana* induces a transcriptional response resembling that of pathogen infection. *Front Plant Sci.*, 12, 13:838284. https://www.frontiersin.org/articles/10.3389/fpls.2022.838284/full

Hönig, M.*, Roeber, V. M.*, Schmülling, T. and Cortleven, A. (2023) Chemical priming of plant defense responses to pathogen attacks. *Front Plant Sci.*, 14:1146577. https://doi.org/10.3389/fpls.2023.1146577

*shared first authorship

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List of abbreviations

The following abbreviations were used in this work.

ABA	Abscisic acid
AGI	Arabidopsis Genome Identifier
AT	Annealing temperature
B	Blue light
BH	Benjamini Hochberg
Bonf	Bonferroni
CAPS	Cleaved amplified polymorphic sequence
CAT	Catalase
CCE	CRY C-terminal extension
CDL	Critical day length
Col-0	Columbia-0
DAG	Days after germination
cDNA	Complementary desoxyribonucleic acid
DNA	Desoxyribonucleic acid
dNTPs	Deoxyribose nucleoside triphosphates
EEE	Excess excitation energy
ER	Endoplasmic reticulum
ET	Ethylene
ETI	Effector triggered immunity
FC	Fold change
FP	Forward primer
FPKM	Fragments Per Kilobase Million
FW	Fresh weight
GP	Guaiacol peroxidase
FAD	Flavin adenine dinucleotide
FR	Far-red light
HR	Hypersensitive response
IR	Induced resistance
ISR	Induced systemic resistance
JA	Jasmonic acid
JA-Ile	Jasmonic acid-isoleucine
LED	Light-emitting diode
LD	Long day
LOV	Light oxygen voltage
LRR	Leucine rich repeat
MAMP	Microbe associated molecular pattern
MAPK	Mitogen activated protein kinase
MATE	Multidrug And Toxic Compound Extrusion
mRNA	messenger RNA
MTI	Microbe triggered immunity
NASC	European Arabidopsis Stock Centre
NB	Nucleotide binding
P	Priming
padj	adjusted *p*-value
PAMP	Pathogen associated molecular pattern
PCR	Polymerase chain reaction

PDA	Potato dextrose agar
PHR	Photolyase homologous region
PL	Prolonged light
PL0	Priming without a lag phase
PL1	Priming plus one day lag phase
PL0T	Priming without a lag phase plus triggering
PL1T	Priming plus one day lag phase plus triggering
PC	Principal component
PCA	Principal component analysis
PCR	Polymerase chain reaction
PE	Primer efficiency
PQ	Plastoquinone
PS	Product size
PTI	PAMP-triggered immunity
RP	Reverse Primer
R	Resistance gene
RL	Red light
RK	Receptor kinase
RLK	Receptor-like kinase
RLP	Receptor-like protein
QR	QuantPrime
qRT-PCR	Quantitative real time-polymerase chain reaction
QT	Quality Treshold
PRR	Pattern recognition receptors
RNA	Ribonucleic acid
ROS	Reactive oxygen species
rRNA	Ribosomal ribonucleic acid
SA	Salicylic acid
SAR	Systemic acquired resistance
SD	Short day
T	Triggering
tAPX	Thylakoid ascorbate peroxidase
TCA	Trichloroacetic acid
T-DNA	Transfer-DNA
TF	Transcription factor
Tm	Melting temperature
TTFL	transcriptional-translational feedback loops
UV-A	Ultraviolet-A
UV-B	Ultraviolet-B
WT	Wild type

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1 Introduction

Life on Earth is characterized by regularly occurring light and dark periods that alternate in a time of approximately 24 hours due to the Earth's rotation around its own axis. Eucaryotes including plants have adjusted several life processes to this regular rhythm of light and darkness (Nitschke *et al.*, 2017; Roeber *et al.*, 2021).

In the frame of this thesis, the effects of a suddenly occurring change in this regular rhythm of light and darkness were investigated in *Arabidopsis thaliana*. A special focus was placed on the question how a suddenly prolonged light period leading to photoperiod stress influences the response of Arabidopsis to other stimuli, such as an additional change in the daily light-dark rhythm (**section 3.2 - *cis*-priming**) or infections with plant pathogens (**section 3.3 - *trans*-priming**).

The topics that are introduced in this thesis are graphically summarized in **Figure 1**. The first section of this introduction considers plant responses under non-stress conditions (**section 1.1**), the following sections deal with plant responses under stressful environments (**section 1.2, 1.3, 1.4 and 1.5**).

In the first section of this introduction (**section 1.1**), the perception of light environments by plants is introduced. Light perception in plants relies on photoreceptors (**section 1.1.1**), and chloroplasts (**section 1.1.2**). Time measurement is enabled by the plant circadian clock (**section 1.1.3**). The second section (**section 1.2**) gives an overview on conditions that induce stress in plants, focusing especially on photoperiod (**section 1.2.1**) and biotic stresses (**section 1.2.2**). In the third section (**section 1.3**), the roles of phytohormones (**section 1.3.1**) and reactive oxygen species (**section 1.3.2**) in the regulation of plant stress responses is introduced. The fourth section introduces the effects of the light environment (light availability, light intensity, photoperiod, and light quality) on biotic stress responses of plants (**section 1.4**). The fifth section (**section 1.5**) deals with the responses of plants to stress combinations, focusing on induced resistance (**section 1.5.1**) and priming (**section 1.5.2**). In the sixth section of the introduction (**section 1.6**), the research aims underlying this work are summarized.

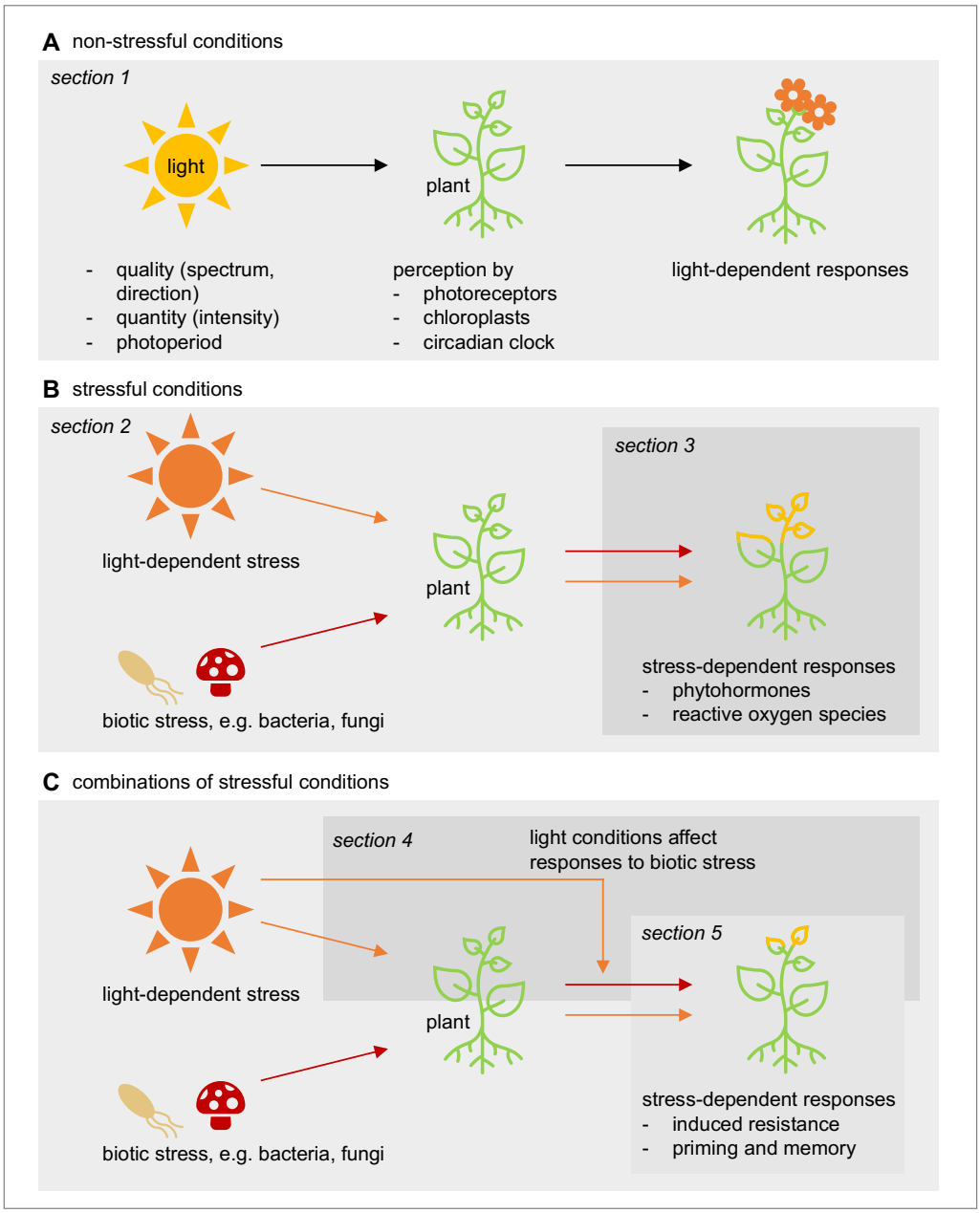

Figure 1. Graphical summary of Introduction.

Figure 1 summarizes the different topics of the Introduction in the order they are thematized in the text. **(A)** Perception of the light environment by plants under non-stress conditions. Section 1 introduces how plants determine light quality (spectral distribution, direction), quantity (intensity) and photoperiod (duration of the daily light length) using their photoreceptors, chloroplasts, and the circadian clock. **(B)** Perception of and responses to stress conditions by plants. Section 2 highlights the effects of photoperiod stress (orange arrows) and biotic stress (dark red arrows). Section 3 gives an overview on the roles of phytohormones and reactive oxygen species in the regulation on photoperiod and biotic stress. **(C)** Effects of combinations of environmental conditions on plants. Section 4 introduces the effects of the plant light environment (with a special focus on photoperiod stress) on responses to biotic stresses. Section 5 deals with stress-dependent resistance induction and priming by abiotic and biotic stresses.

1.1 Light environment perception by plants

Light providing plants with energy for photosynthesis represents an important environmental stimulus to which plants adjust their life processes (Franklin & Quail, 2010; Casal, 2013; Paik & Huq, 2019). Using photoreceptors (**section 1.1.1**), chloroplasts (**section 1.1.2**) and the circadian clock (**section 1.1.3**), plants can determine light quality (spectral distribution, direction), quantity (intensity) and photoperiod (duration of the daily light length) (Sanchez *et al.*, 2020; Roeber *et al.*, 2021; 2022).

Sensing of and responding to the photoperiod that depends on season and latitude (Jackson, 2009; Adole *et al.*, 2019) enables plants to precisely synchronize their developmental processes to certain times of the year thereby optimising growth and offspring production (Casal *et al.*, 2004). One of the responses influenced by the photoperiod in many plant species is flowering (Carré, 2001; Mouradov *et al.*, 2002; Roeber *et al.*, 2022). Depending on their ability to respond to different photoperiods, plants are classified as short day (SD), long day (LD) or day neutral plants (Jackson, 2009). While flowering is induced in SD plants when the photoperiod is shorter than a so-called critical day length (CDL), LD plants flower when the photoperiod exceeds the plants' critical day length. Plants that flower independently of their surrounding photoperiod, such as tomatoes, potatoes, or cucumbers, are considered as day neutral plants (Jackson, 2009). The model organism Arabidopsis is considered as a facultative LD plant meaning that Arabidopsis flowers earlier under LD but is also capable to flower under SD (Mouradov *et al.*, 2002). Interestingly, a single LD is sufficient to induce flowering in SD-grown Arabidopsis (Corbesier *et al.*, 1996) showing the responsiveness of Arabidopsis to changes of the photoperiod. However, only plants in a certain developmental phase respond to changes of the photoperiod (**Figure 2**) (Corbesier *et al.*, 1996; Matsoukas, 2014; Shibaeva *et al.*, 2022).

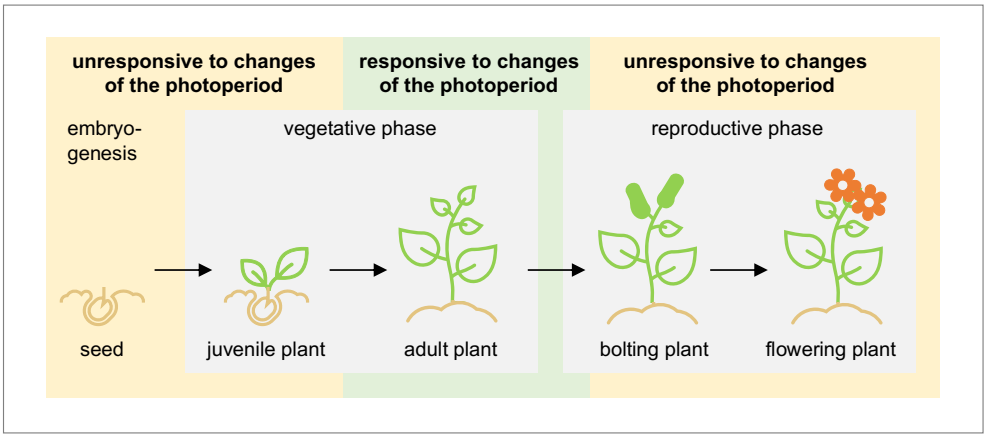

Figure 2. Responsiveness of plants to changes of the photoperiod.

Only Arabidopsis in certain developmental stages respond to changes of the photoperiod. Seeds and juvenile plants are responsive to changes of the photoperiod, while adult plants can sense photoperiods and respond accordingly. When plants entered their reproductive stage, they become again insensitive to photoperiods. Figure is inspired by Matsoukas (2014).

During their juvenile phase, Arabidopsis plants are insensitive to photoperiods, while they become sensitive in their adult phase (Matsoukas, 2014), thereby ensuring that only plants with a certain age initiate flowering. In addition to flowering, the photoperiod regulates several other developmental

processes in plants, such as growth cessation, bud setting and dormancy in perennial plants (Singh *et al.*, 2017) or senescence in annual plants (Serrano-Bueno *et al.*, 2021). Besides the influence of the photoperiod on developmental processes, the photoperiod can act as a signal to prepare plants for future stressful events. For instance, in autumn, plants sense the shortening of the day length, which prepares them for colder temperatures in winter (Lee & Thomashow, 2012). Moreover, the tolerance of warmer temperatures is also influenced by the light environment (Roeber *et al.*, 2022).

1.1.1 Light perception by photoreceptors

The ability of plants to respond to light including photoperiods relies on the perception of light by photoreceptors and chloroplasts (**section 1.1.2**) and measurement of the time through the plant circadian clock (**section 1.1.3**) (Jackson, 2009; Serrano-Bueno *et al.*, 2021).

In Arabidopsis, at least five photoreceptor families exist that can sense specific wavelengths of the light spectrum (**Figure 3**) (Sanchez *et al.*, 2020).

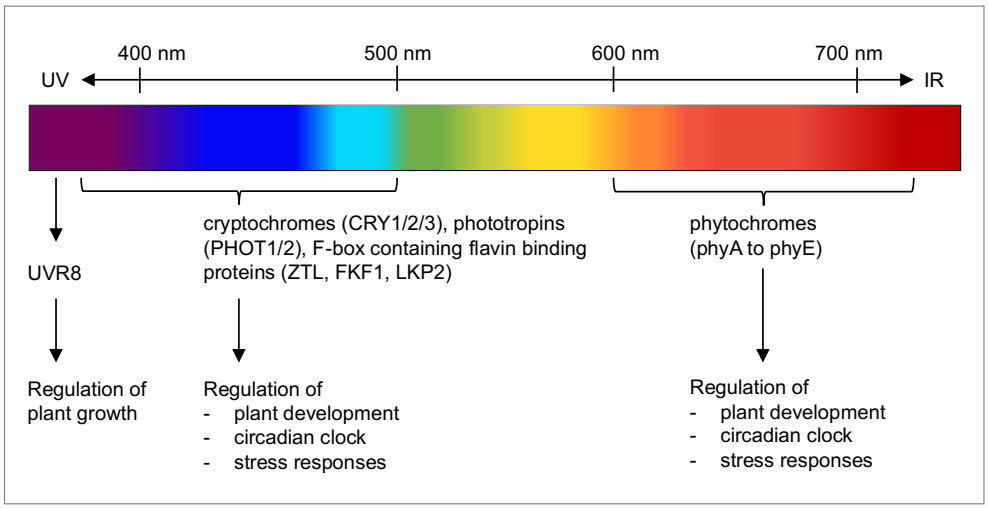

Figure 3. Perception of light environment by photoreceptors in Arabidopsis.

At least five photoreceptor families exist in Arabidopsis that sense specific wavelengths of the light environment. Light perception by certain photoreceptors regulates various aspects of plant performance. UV, ultraviolet light; IR, infrared radiation. Figure is inspired by Ghorbel *et al.* (2023) and Breen *et al.* (2023).

Wavelengths in the range of 600 to 750 nm (red (R) to far-red (FR) light) are absorbed by phytochromes in Arabidopsis and in the range of 320 to 500 nm (blue and ultraviolet (UV)-A light) are detected by cryptochromes, phototropins and F-box containing flavin-binding proteins (Paik & Huq, 2019). In addition, Arabidopsis perceive wavelengths ranging from 280 to 320 nm (UV-B light) employing UV RESISTANCE LOCUS8 (UVR8) photoreceptors. All these photoreceptors, except UVR8, interact with chromophores, which are light-absorbing ligands (Paik & Huq, 2019; Sanchez *et al.*, 2020).

Phytochromes are important regulators of plant development that are involved in germination, de-etiolation, photomorphogenic growth, gravitropism, shade avoidance responses, and stomatal and reproductive tissue development (Franklin & Quail, 2010; Casal, 2013; Pierik & De Wit, 2014; Yeom *et*

al., 2014). In addition, phytochromes have been shown to function as thermosensors (Jung *et al.*, 2016), affect the circadian clock, and regulate various stress responses (**section 1.2**) (Junior *et al.*, 2020).

In Arabidopsis, phytochromes phyA to phyE are homodimerizing photoreceptors consisting of an apoprotein attached to a single tetrapyrrole chromophore (phytochromobilin) that absorbs R to FR light, and to a lesser extent also blue light (Casal, 2013). Phytochromes are photoconvertible: in response to R light, the biologically inactive, cytoplasmic Pr-state of phytochromes (with an absorption maximum at 660 nm) convert into the biologically active Pfr-state (with an absorption maximum at 730 nm) that translocate into the nucleus (Sanchez *et al.*, 2020). Photo-activated phytochromes physically interact with and mediate the 26S proteasomal degradation of PHYTOCHROME INTERACTING FACTORS (PIFs) representing a family of basic helix-loop-helix (bHLH) TFs that repress photomorphogenesis (Duek & Fankhauser, 2005; Monte *et al.*, 2007; Franklin & Quail, 2010). Phytochromes also interact with the E3 ubiquitin ligase SUPPRESSOR OF PHYA1 (SPA1)/CONSTITUTIVELY PHOTOMORPHOGENIC1 (COP1), thereby preventing COP1-mediated degradation of ELONGATED HYPOCOTYL5 (HY5) (**Figure 4**) (Lian *et al.*, 2017). HY5 is a basic leucine zipper (bZIP) family TF that promotes several plant developmental processes including photomorphogenesis (Oyama *et al.*, 1997; Gangappa & Botto, 2016). Exposure to FR light transforms the Pfr-state of phytochromes back into the R light-absorbing state (Bae & Choi, 2008; Casal, 2013). Also, via thermal reversion, certain phytochromes, such as phyB, revert independent of light back to their inactive Pr state (**Figure 4**) (Klose *et al.*, 2020).

Another group of photoreceptors are cryptochromes that regulate several plant developmental processes (germination of seeds, de-etiolation of seedlings, root elongation, stomata development, reproductive tissue development) (Canamero *et al.*, 2006; Kang *et al.*, 2009). In addition, cryptochromes modulate the plant circadian clock and mediate several plant stress responses (Ponnu & Hoecker, 2022). In Arabidopsis, cryptochromes (CRY1 to CRY3) are photolyase-like photoreceptors that are composed of a conserved photolyase homologous region (PHR) harbouring a flavin adenine dinucleotide (FAD) as chromophore and a CRY C-terminal extension (CCE) domain (Casal, 2013; Ma *et al.*, 2016; Wang & Lin, 2020; Paik & Huq, 2019). Photo-activated cryptochromes interact with COP1, thereby inhibiting the activity of the E3 ubiquitin ligase SPA1/COP1, which promotes downstream signalling, such as photomorphogenesis. In addition, CRY1 and CRY2 interact with PIFs allowing blue light-dependent regulation of hypocotyl elongation (Pedmale *et al.*, 2016; Ponnu & Hoecker, 2022). Besides, photo-activated CRY2 interacts through its PHR domain with CRYPTOCHROME-INTERACTING bHLH (CIB) family TFs, thereby regulating flowering time (Liu *et al.*, 2011; Wang & Lin, 2020). The photoactivation of cryptochromes is inhibited by BLUE LIGHT INHIBITORS OF CRYPTOCHROMES1 (BIC1) and BIC2 (**Figure 4**) (Wang *et al.*, 2017; Ma *et al.*, 2016).

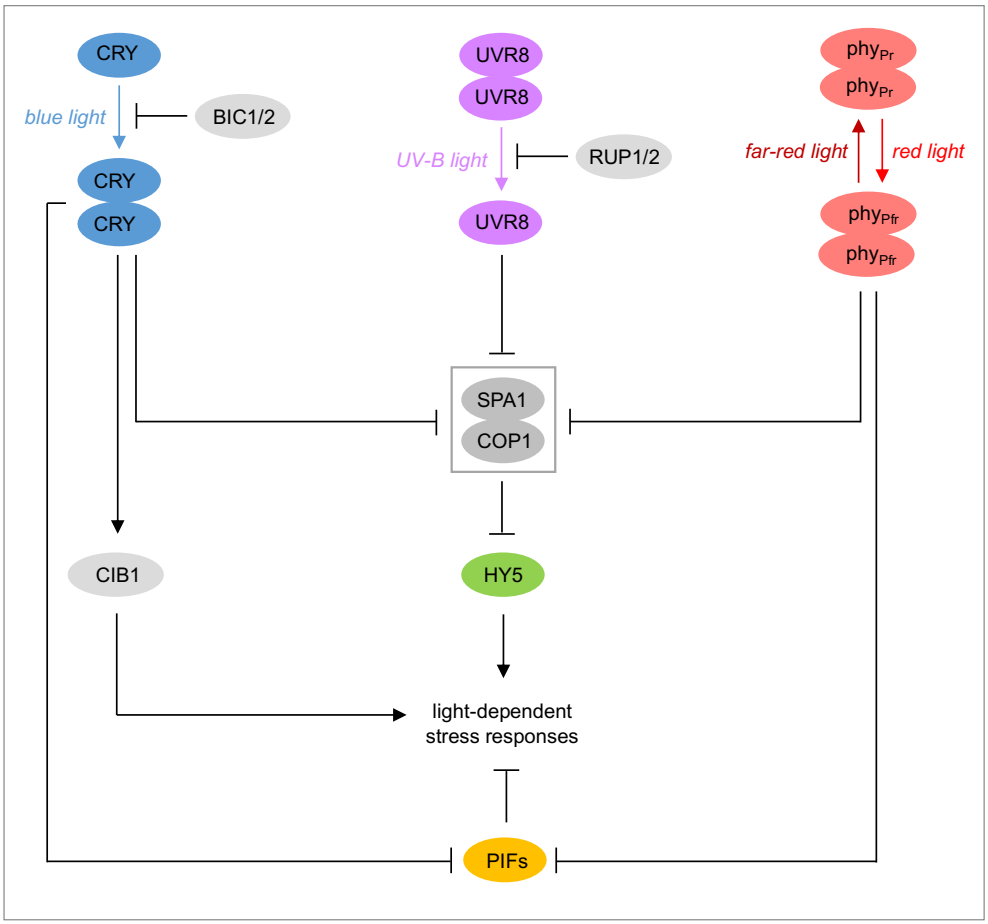

Figure 4. Simplified scheme of plant light signalling.

In Arabidopsis, the E3 ubiquitin ligase SPA1/COP1 is negatively regulated by phytochromes, cryptochromes, and UVR8 photoreceptors, thereby preventing COP1-mediated degradation of HY, a transcription regulator of various plant responses. Red light converts homodimerizing phytochromes in their biologically inactive Pr-state (phyPr) into their biologically active Pfr-state (phyPfr) that repress SPA1/COP1 and mediate the degradation of PIF transcription factors. Far-red light converts phytochromes from the Pfr-state (phyPfr) back to the Pr-state (phyPr). Blue light causes conformational changes of monomeric cryptochromes (CRY) resulting in the formation of biologically active oligomers that repress SPA1/COP1 and interact with CIB-family transcription factors. Photoactivation of cryptochromes is inhibited by BIC1/2. UV-B light stimulates the dissociation of homodimeric UVR8 photoreceptors into monomers that repress SPA1/COP1. RUP1/2 mediate the reformation of homodimeric UVR8. Arrows indicate positive regulations; cut lines indicate negative regulations. Figure is inspired by Roeber et al. (2021) and Xu and Zhu (2021). PIFs, PHYTOCHROME INTERACTING FACTORs; SPA1/COP1, SUPPRESSOR OF PHYA1/CONSTITUTIVELY PHOTOMORPHOGENIC1; HY5, ELONGATED HYPOCOTYL5; BIC1/2, INHIBITORS OF CRYPTOCHROMES1/2; CIB, CRYPTOCHROME-INTERACTING bHLH-family; RUP1/2, REPRESSOR OF PHOTOMORPHOGENESIS1/2.

Phototropins form another family of photoreceptors sensitive to blue light. In Arabidopsis, phototropins (PHOT1 and PHOT2) function redundantly in different processes, such as phototropism, stomata opening, chloroplast accumulation, leaf movement and cotyledon/leaf expansion (Christie *et al.*, 2015). In addition, phototropins have also distinct functions, for instance PHOT2 mediates the chloroplast avoidance movement, while PHOT1 possesses a higher sensitivity in some its regulated processes (Sullivan *et al.*, 2008; Aihara *et al.*, 2008; Christie *et al.*, 2015).

The LOV domain-containing F-box proteins, often referred to as ZEITLUPE (ZTL) family, form the third family of blue light-absorbing photoreceptors, to which the proteins ZTL, FLAVIN-BINDING KELCH

REPEAT F-BOX 1 (FKF1) and LOV KELCH PROTEIN2 (LKP2) belong (Ito *et al.*, 2012; Paik & Huq, 2019). ZTL, FKF1 and LKP2 contribute to the blue light-dependent proteasomal degradation of proteins (Sanchez *et al.*, 2020). Among the proteins whose stability is controlled by ZTL, FKF1 and LKP2 are related to the circadian clock and flowering (Christie *et al.*, 2015).

The UVR8 photoreceptor detects UV-B light (Yin & Ulm, 2017). UVR8 photoreceptors form cytoplasmic homodimers under natural conditions that dissociate into monomers when exposed to UV-B light (Findlay & Jenkins, 2016; Sanchez *et al.*, 2020). UVR8-dependent signalling promotes plant growth by regulating phytohormone signalling: UVR8 monomers interact with and inhibit the TFs BRI-EMS-SUPPRESSOR1 (BES1) and BES-INTERACTING MYC-LIKE1 (BIM1) representing two regulators of brassinosteroid signalling (Liang *et al.*, 2018; Yang *et al.*, 2019a). Monomeric UVR8 interacts with the E3 ubiquitin ligase SPA1/ COP1, thereby preventing COP1-mediated degradation of HY5 and initiating downstream signalling (Favory *et al.*, 2009; Yin & Ulm, 2017; Henry-Kirk *et al.*, 2018). The UVR8-COP1 interaction is disturbed by REPRESSOR OF PHOTOMORPHOGENESIS1 (RUP1) and RUP2 that mediate the reformation of UVR8 dimers (independent of COP1), thereby inactivating UVR8 photoreceptors (**Figure 4**) (Heijde & Ulm, 2013).

1.1.2 Light perception by chloroplasts

In addition to photoreceptors, also chloroplasts perceive light (Lepistö & Rintamäki, 2012; Roeber *et al.*, 2021; 2022). Chloroplasts represent specialized plant cell organelles that convert light energy into chemical energy during photosynthesis (Dobrogojski *et al.*, 2020).

Biogenesis of chloroplasts is regulated by signalling pathways that rely on light signals detected by photoreceptors (Jan *et al.*, 2022). Both photo-activated phytochromes and cryptochromes mediate the removal of SPA1/COP1 and PIFs from the plant nucleus, thereby preventing SPA1/COP1- or PIF-mediated suppression of their targets and thus activating the expression of photosynthesis-associated nuclear genes (PhANGs) (Liu *et al.*, 2007; Bae & Choi, 2008; Bu *et al.*, 2011; Liu *et al.*, 2011; Lepistö & Rintamäki, 2012; Pedmale *et al.*, 2016) which are important for chloroplast assembly (Hills *et al.*, 2015). The expression of PhANGs is also affected by retrograde signals (Lepistö & Rintamäki, 2012).

The composition of chloroplasts is adjusted depending on the light environment (Walters & Horton, 1995; Walters, 2005; Lepistö & Rintamäki, 2012). Chloroplasts possess more electron carries and higher quantities of photosystems, ATP synthase complexes and enzymes involved in the Calvin-Benson cycle, including ribulose-1,5-bisphosphate carboxylase/oxygenase (RuBisCo), under high light. Under low light, chloroplasts optimise their light harvesting by increasing the proportion of light-harvesting complexes (LHC), stacking of thylakoid membranes, and decreasing their relative ratios of chlorophyll *a* to *b* (Weston *et al.*, 2000; Walters, 2005). Acclimation of plants to different light intensities relies on redox signals (**section 1.3.2**) derived from chloroplasts (Pfannschmidt *et al.*, 2003). In addition, the ultrastructure of chloroplasts is also affected by the photoperiod. Chloroplasts of long day (LD)-grown Arabidopsis contain smaller grana stacks than short day (SD)-grown plants, and the relative ratio of chlorophyll *a* to *b* increases under LD conditions (Lepistö & Rintamäki, 2012; Roeber *et al.*, 2022). Another characteristic that is influenced by the photoperiod is the degradation rate of starch, which is

stored during the day in chloroplasts and allocated during the night (Lu *et al.*, 2005; Zeeman *et al.*, 2007; Roeber *et al.*, 2022). Shifting Arabidopsis from LD (16 h light/8 h dark) to SD (8 h light/16 h dark) decreased the starch degradation rate during the night following the photoperiod shift. Transferring SD-grown Arabidopsis to LD conditions had the opposite effect (Lu *et al.*, 2005). This points to the importance of the photoperiod in regulation of chloroplast-related processes.

1.1.3 Time measurement by the plant circadian clock

The circadian clock represents an endogenous time keeping mechanism enabling plants to anticipate the daily light-and-dark rhythm and to synchronize their biological processes with their environment (Covington *et al.*, 2008; Hsu & Harmer, 2014; Sanchez & Kay, 2016), thereby enhancing the plants' fitness (Seo & Mas, 2015). The plant circadian clock is entrained (reset) by environmental conditions, such as light and temperature (McClung, 2006; Creux & Harmer, 2019). In Arabidopsis, entrainment of the circadian clock by light involves all photoreceptor families except phototropins (**section 1.1.1**) (Sanchez *et al.*, 2020).

Plant circadian clocks are composed of several interlocked transcriptional-translational feedback loops (TTFL) (McClung, 2006; Hsu & Harmer, 2014). TTFLs consist of activating and repressing transcription factors whose levels alternate depending on the time of the day and night, thereby regulating the transcription of clock genes (Adams *et al.*, 2015).

In Arabidopsis, the circadian clock consists of three loops, the morning, central and evening loop, that regulate each other and together form the central oscillator (Venkat & Muneer, 2022). The main components of the circadian clock are shown in **Figure 5**. The morning loop contains the MYB-like TFs CIRCADIAN CLOCK ASSOCIATED1 (CCA1) and LATE ELONGATED HYPOCOTYL (LHY) (Haydon *et al.*, 2011). The expression of both *CCA1* and *LHY* peaks at dawn and are therefore considered as morning-phased genes (Nohales, 2021). CCA1 and LHY form homo- and heterodimers (Lu *et al.*, 2005; Yakir *et al.*, 2009). They both function as transcription regulators inhibiting their own expression but also the expression of several other clock genes, such as the *PSEUDO-RESPONSE REGULATOR5* (*PRR5*), the *PRR1*/*TIMING OF CAB EXPRESSION1* (*TOC1*), *GIGANTEA* (*GI*), *LUX ARRYTHMO* (*LUX*) or *EARLY FLOWERING4* (*ELF4*) (Alabadi *et al.*, 2001; Hazen *et al.*, 2005; Perales & Mas, 2007; Nakamichi *et al.*, 2010; Li *et al.*, 2011; Gendron *et al.*, 2012), and activating the expression of *PRR9* and *PRR7* (Locke *et al.*, 2006; Zeilinger *et al.*, 2006; Nohales, 2021). The expression of several of these genes is also stimulated by LIGHT-REGULATED WD1 (LWD1) and LWD2, REVEILLE4 (RVE4), RVE6, RVE8 and NIGHT LIGHT-INDUCIBLE AND CLOCK-REGULATED GENE1 (LNK1) and LNK2, acting as transcription activators (Sanchez *et al.*, 2020). The *PRR* genes are chronologically expressed peaking at specific times of the day, namely *PRR9* in the morning, *PRR7* at midday, *PRR5* in the afternoon and *PRR1*/*TOC1* in the evening (Nakamichi *et al.*, 2010; Gendron *et al.*, 2012). Each of the PRR proteins represses the transcription of its preceding *PRR* and additionally also *CCA1* and *LHY1* transcription (McClung, 2006; Nohales, 2021). The repression of *CCA1* and *LHY* by PRR9 and PRR7 is considered as the morning loop (Nakamichi *et al.*, 2010) and by PRR1/TOC1 as the evening loop (Haydon *et al.*, 2011).

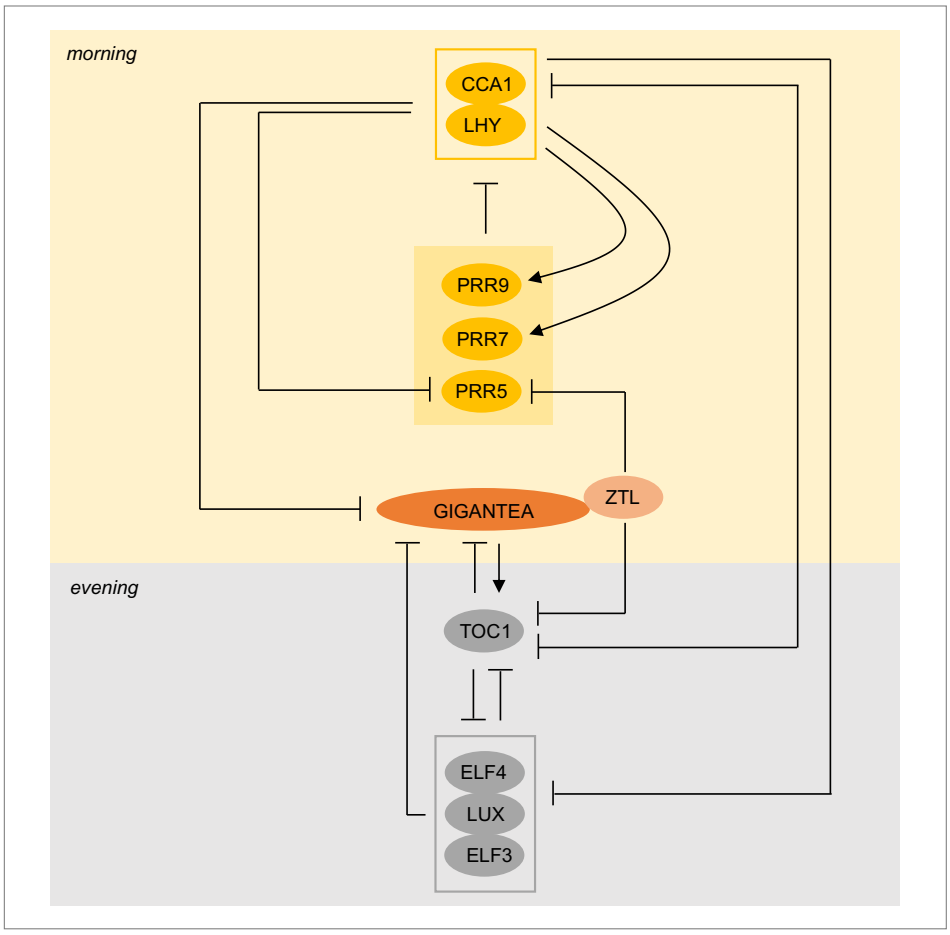

Figure 5. Simplified scheme of the circadian clock of Arabidopsis.

The circadian clock represents an endogenous time keeping mechanism allowing plants to anticipate the daily light-and-dark rhythm and to adjust their biological processes accordingly. The central regulatory components of the circadian clock are displayed: The expression of CCA1 and LHY peaks in the morning. CCA1 and LHY proteins function as transcription regulators that repress their own expression, and that of other genes including PRR5, TOC1, GI, LUX and ELF4. In addition, CCA1 and LHY activate the expression of PRR9 and PRR7. The PRR genes are chronologically expressed, starting with PRR9 in the morning, PRR7 at midday, PRR5 in the afternoon and TOC1 in the evening. PRRs repress the expression of their preceding PRR gene, CCA1 and LHY. In the evening, TOC1 represses expression of GI, LUX and ELF4. Degradation of TOC1 and PRR5 proteins is mediated by ZTL that is regulated by GI. Arrows indicate positive regulation; cut lines, indicate negative regulation. Figure is inspired by Roeber et al. (2022). CCA1, CIRCADIAN CLOCK ASSOCIATED1; LHY, LATE ELONGATED HYPOCOTYL; PRR5/7/9, PSEUDO-RESPONSE REGULATOR5/7/9; TOC1, TIMING OF CAB EXPRESSION1; ELF3/4, EARLY FLOWERING3/4; LUX, LUX ARRYTHMOK; GI, GIGANTEA; ZTL, ZEITLUPE.

During the central loop, which connects the morning and evening loops, decreasing levels of CCA1 and LHY result in increasing expression of *PRR1*/*TOC1* at dusk (Haydon *et al.*, 2011; Venkat & Muneer, 2022). In the evening, PRR1/TOC1, whose nuclear accumulation is enhanced by PRR5, inhibits the expression of several clock genes including *GI*, *LUX* and *ELF4* (Haydon *et al.*, 2011). Protein levels of PRR1/TOC1 and PRR5 are regulated by the blue light-sensitive F-box protein ZEITLUPE (ZTL), which is a component of an SCF^{ZTL} E3 ubiquitin ligase complex that targets both for proteasomal degradation in the darkness (Kim *et al.*, 2007). Degradation of PRR1/TOC1 is prevented by cytosolic GI, which forms heterodimers with ZTL and thereby modulates the accumulation of ZTL (Más *et al.*, 2003; Kim *et al.*, 2007; 2013; Sanchez *et al.*, 2020), and by PRR3, which competes with ZTL for interaction with PRR1/TOC1 (Haydon *et al.*, 2011). During night, *PRR1*/*TOC1* as well as *PRR9*, *PRR7*, *GI* and *LUX* are

repressed by the Evening Complex (EC), which is composed of LUX (or its close homolog NOX), ELF3 and ELF4. Just before dawn, the transcription of *CCA1* and *LHY* is activated again, which is likely mediated by EC and GI (Sanchez *et al.*, 2020; Nohales, 2021).

Although initially considered as a unidirectional pathway relying on input signals transduced via the circadian oscillator into output signals, increasing evidence proposes that the plant circadian system is a highly complex regulatory network, in which the circadian oscillator and plant signalling pathways regulate each other (Pruneda-Paz & Kay, 2010). Plant responses to several environmental stimuli, such as light, temperature, nutrients, or stresses (**section 1.2**), have been shown to influence and are in turn also regulated by the circadian oscillator. This multidirectional regulation involves the activity of phytohormones (**section 1.3.1**), regulation of the plant redox state (**section 1.3.2**) and calcium signals (Seo & Mas, 2015; Pruneda-Paz & Kay, 2010; Guadagno *et al.*, 2018; Karapetyan & Dong, 2018).

1.2 Stress perception and responses in plants

Plants are exposed to constantly changing environmental conditions. Suddenly occurring changes in environmental conditions that affect their growth, development, and productivity have the potential to induce stress in plants (Lichtenthaler, 1998; Mosa *et al.*, 2017; Gull *et al.*, 2019).

Plant stresses are classified as abiotic and biotic according to their trigger (Mosa *et al.*, 2017). While abiotic stress is elicited by either physical or chemical conditions, biotic stress is caused by living organisms (Gull *et al.*, 2019). Besides their classification as abiotic and biotic, plant stresses can be assigned as eustress or distress referring to stresses with positive (non-harmful) or negative (harmful) effects on plant performance, respectively (Lichtenthaler, 1998). Depending on the stress itself, its intensity and duration, the effects of stresses on plants can be temporary or lethal (Lichtenthaler, 1998; Gull *et al.*, 2019).

To successfully cope with stresses, sensing of unfavourable environments is requisite for plants. Perception of stressful environments allows plants to trigger downstream responses. These responses involve transcriptional, biochemical, physiological, and morphological mechanisms (Gull *et al.*, 2019; Lamers *et al.*, 2020). The complexity of these plant responses is further increased when different stresses occur simultaneously (**section 1.5**), which is common in nature. The responses triggered by stress combinations might interact positively or negatively or are unique to specific stress combinations. This means that responses of plants to certain stress combinations cannot be derived from their responses to single stresses (Suzuki *et al.*, 2014).

Already today, plant stresses are a major cause of economic losses in agriculture and forestry. This situation will be further aggravated due to climate change, which results in more severe and often simultaneously occurring stresses (Teshome *et al.*, 2020; Singh *et al.*, 2023) making it important to enhance our understanding of plant stress responses especially in response to stress combinations.

In the following section the responses of Arabidopsis to photoperiod stress (**section 1.2.1**), which is a form of abiotic stress, is introduced. The ensuing section introduces plant responses to biotic stresses (**section 1.2.2**). In addition, the effects of the light environment on plant response to biotic stresses are

highlighted (**section 1.5**). A special focus is placed on responses of plants to combinations of photoperiod stress and biotic stress (**section 1.4.3**).

1.2.1 Photoperiod stress

Photoperiod stress, originally called circadian stress, is a form of abiotic stress occurring in plants exposed to a sudden prolongation of the light period that exceeds the natural photoperiod, which depends on latitude and season (Nitschke *et al.*, 2016; Roeber *et al.*, 2021; 2022). Photoperiod stress has been first described in Arabidopsis grown under short day (SD) conditions (8 h light/16 h darkness) before exposure to a 24 h-prolonged light period (Nitschke *et al.*, 2016). Recent results indicate that a suddenly prolonged light period also induces stress in other plant species, such as tomato (Shibaeva *et al.*, 2022) (Anne Cortleven, personal communication). Interestingly, the developmental stage determines if Arabidopsis plants are capable of responding to photoperiod stress. The photoperiod stress response is only visible in mature plant leaves, while neither an induction of oxidative stress marker genes nor a programmed cell death-related necrosis is observed in younger Arabidopsis leaves. Besides, the strength of the photoperiod stress response depends on plant age. The duration of the dark period that follows the prolonged light treatment but also the length of the prolonged light period itself determine the strength of the photoperiod stress response (Nitschke, 2014). In SD-grown Arabidopsis plants, the response to photoperiod stress involves an induction of oxidative stress marker genes, such as *ZINC FINGER OF ARABIDOPSIS THALIANA12* (*ZAT12*) or *BON ASSOCIATED PROTEIN1* (*BAP1*), and an increase in peroxides that is accompanied by enhanced peroxidase and decreased catalase enzyme activities during the night that follows a 24 h-prolonged light period (Abuelsoud *et al.*, 2020). The next day, photoperiod stress-responsive plants suffer from a decreased photosynthetic efficiency and programmed cell death ensuing in their leaves (**Figure 6**) (Nitschke *et al.*, 2016).

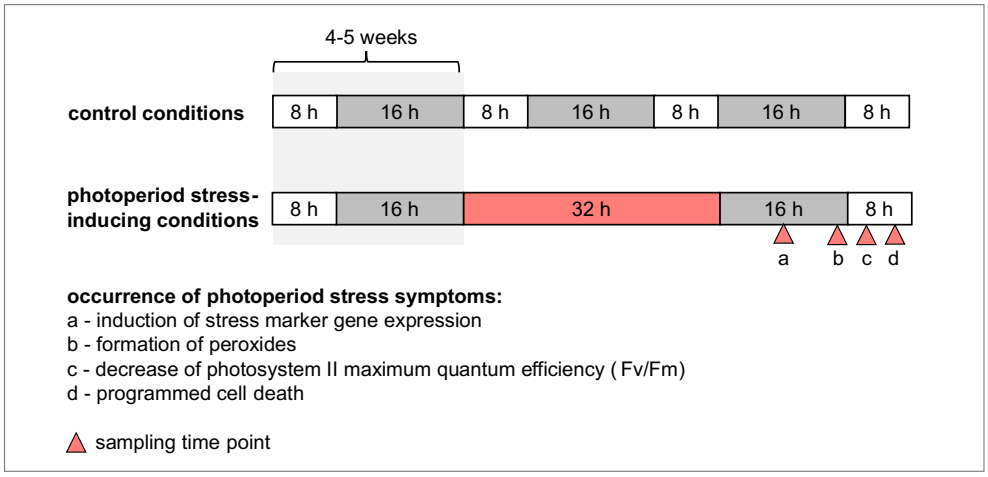

Figure 6. Schematic overview of photoperiod stress-inducing conditions in Arabidopsis.

Exposure of Arabidopsis to a prolonged light period of 32 hours induces photoperiod stress after growth for four to five weeks under short day conditions (8 h light/16 h darkness). During the night that follows the prolonged light treatment, the expression of redox-related stress marker genes (*ZAT12, BAP1*) is induced, which is accompanied by accumulation of peroxides in photoperiod stress-sensitive plants. During the next day, the photosynthetic efficiency decreases, and programmed cell death ensues in strongly stressed plants. Figure is inspired by Nitschke *et al.* (2016) and Frank (2019).

In addition to effects related to the plant redox state, also the phytohormone content is affected in response to photoperiod stress. The levels of jasmonic acid (JA), auxin as well as salicylic acid (SA) increase during the night that follows a suddenly 24 h-prolonged light treatment (Nitschke *et al.*, 2016; Cortleven *et al.*, 2022; Frank *et al.*, 2022). Cytokinin (CK), especially root-derived *trans*-zeatin derivatives, protect Arabidopsis against photoperiod stress (Frank *et al.*, 2020). The protective function of CK relies on the CK receptors ARABIDOPSIS HISTIDINE KINASE2 (AHK2) and AHK3 and involves the downstream-acting transcription regulators ARABIDOPSIS RESPONSE REGULATOR2 (ARR2), ARR10 and ARR12 (Nitschke *et al.*, 2016; Frank *et al.*, 2020). The protective function of CK against photoperiod stress is linked to auxin: Indole-3-acetic acid acts as an antagonist to CK, thereby promoting a strong photoperiod stress response (**Figure 7**) (Frank *et al.*, 2022).

To cope with photoperiod stress, a functional circadian clock is required. Mutants with a lowered expression or impaired function of CCA1 and its homolog LONG HYPOCOTYL (LHY) are particularly responsive to photoperiod stress. A similar responsiveness was observed in the CK-deficient mutants *ahk2 ahk3*, which have a lowered *CCA1* and *LHY* expression in common with the clock mutants (Nitschke *et al.*, 2016; Cortleven *et al.*, 2022).

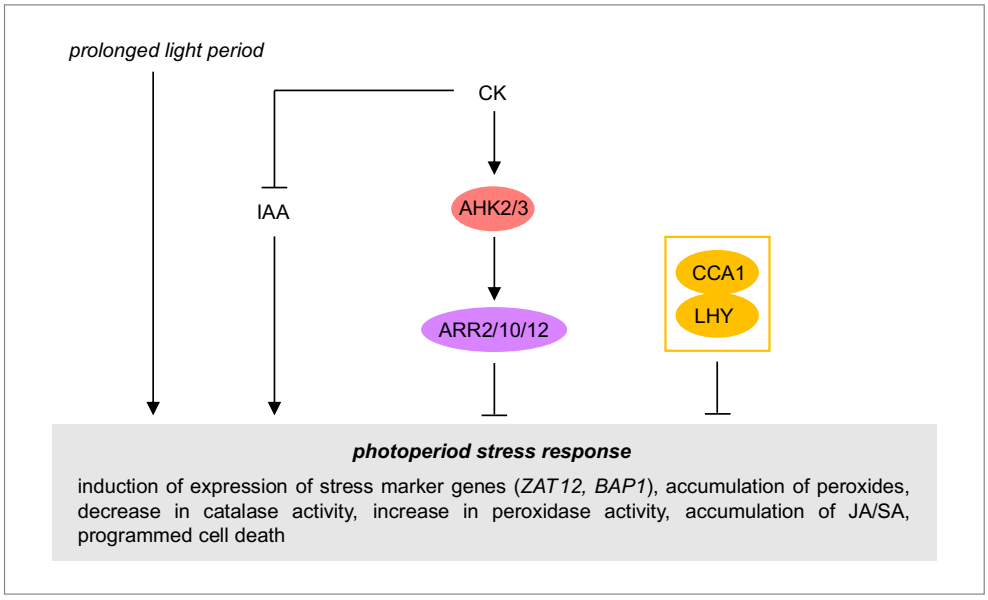

Figure 7. Model of the photoperiod stress response in Arabidopsis.

A prolongation of the light period results in short day-grown Arabidopsis in a photoperiod stress response that is characterized by induction of the expression of stress response marker genes, accumulation of peroxides, increases in phytohormone levels, such as JA, and SA, decreases of photosynthetic efficiency and programmed cell death. The phytohormone CK, especially *trans*-zeatin, negatively regulates the photoperiod stress response involving AHK2/AHK3 and ARR2/ARR10/ARR12. The protective function of CK is antagonized by IAA. Besides, the circadian clock components CCA1 and LHY negatively regulate the photoperiod stress response. Figure is inspired by Roeber *et al.* (2022). JA, jasmonic acid; SA, salicylic acid; CK, cytokinin; AHK2/3, ARABIDOPSIS HISTIDINE KINASE2/3; ARR2/10/12, ARABIDOPSIS RESPONSE REGULATOR2/10/12; IAA, indole-3-acetic acid. CCA1; CIRCADIAN CLOCK ASSOCIATED1; LHY, LATE ELONGATED HYPOCOTYL.

1.2.2 Biotic stress

Biotic stress can be caused by different kinds of living organisms, such as other plants, animals, insects, or pathogens (Gull *et al.*, 2019; Mosa *et al.*, 2017). Biotic stress induced by animals or insects typically involves physical damage of plants (Mosa *et al.*, 2017), while pathogens including bacteria, fungi, oomycetes, viruses, and nematodes cause diseases in plants (Iqbal *et al.*, 2021; Singh *et al.*, 2023). According to their lifestyles, plant pathogens are often classified as biotrophs, hemibiotrophs or necrotrophs (Glazebrook, 2005; Singh *et al.*, 2023). While biotrophs exploit nutrients from living plant cells, hemibiotrophic pathogens switch from a biotrophic to a necrotrophic lifestyle. Plant pathogens with a necrotrophic lifestyle kill invaded plant cells rapidly and use the resulting nutrients (Glazebrook, 2005). In addition to the different lifestyles that plant pathogens possess, also their infection strategies and the plant tissue they target vary dependent on the pathogen species (Singh *et al.*, 2023). While bacteria proliferate in the extracellular space of plant tissues, most fungi and oomycetes additionally break through plant cell walls using specialised structures (e.g. haustoria) (Dodds & Rathjen, 2010).

To recognize and cope with diverse pathogens, plants have evolved a multi-layered immune system (De Wit, 2007; Ngou *et al.*, 2022). Plant cell walls form the first physical barrier against invading pathogens and function as sensors of external conditions (Wan *et al.*, 2021). Exposure to biotic (or abiotic) stresses can damage plant cell walls, thereby impairing their functional integrity (Vaahtera *et al.*, 2019), which is transduced to the plant innate immunity (Wan *et al.*, 2021). The innate immunity of plants consists of two layers, (i) the microbe or pathogen associated molecular pattern (MAMP/PAMP)-triggered immunity (MTI/PTI) and (ii) the effector-triggered immunity (ETI) (**Figure 8**) (Chisholm *et al.*, 2006; Jones & Dangl, 2006; Wan *et al.*, 2021).

PTI is activated when plants recognize MAMPs or PAMPs (such as flagellin, chitin or lipopolysaccharides) in their extracellular space using plasma membrane-located pattern recognition receptors (PRRs) (Dodds & Rathjen, 2010; Trdá *et al.*, 2015). Depending on their intracellular structure, plant PRRs are classified as receptor-like kinases (RLKs) or receptor-like proteins (RLPs) containing or lacking an intracellular serine/threonine kinase domain, respectively (**Figure 8**) (Padmanabhan *et al.*, 2009; Trdá *et al.*, 2015). Plant PRRs possess a transmembrane domain and an extracellular domain (Hake & Romeis, 2019). The extracellular domain of plant PRRs that recognizes and binds specific MAMPs/PAMPs is often composed of leucine rich repeats (LRR) representing a common structural motif of several immune receptors (Padmanabhan *et al.*, 2009; Trdá *et al.*, 2015).

To circumvent PTI, pathogens often secrete effector proteins into plant cells (Dodds & Rathjen, 2010). Plants recognize several of the pathogen-derived effectors by intracellular receptor proteins containing a nucleotide-binding (NB) domain-LRR (NLR) (Jones & Dangl, 2006; Wan *et al.*, 2021; Ngou *et al.*, 2022). NLRs either directly interact and detect effectors or indirectly perceive effector-induced changes in infected plant cells (Jones & Dangl, 2006; Hake & Romeis, 2019). Recognition of effectors allows plants to activate ETI (Jones & Dangl, 2006), which involves strong and robust responses, and is often connected to the hypersensitive response (HR), which is a form of localized cell death that prevents pathogen spread (Hake & Romeis, 2019; Balint-Kurti, 2019).

Although PTI and ETI are differently activated, both trigger similar responses in plants (Yuan *et al.*, 2021) and synergistically boost each other (Nguyen *et al.*, 2021; Tena, 2021). Among the overlapping responses of PTI and ETI are an increase in cytoplasmic calcium levels, activation of mitogen-activated protein kinases (MAPK), receptor-like cytoplasmic kinases (RLCK) and calcium-dependent protein kinases (CDPK), production of reactive oxygen species (ROS), alterations in phytohormone and metabolite content and transcriptional reprogramming (Hake & Romeis, 2019; Yuan *et al.*, 2021). However, the duration and amplitude of responses during ETI are often stronger than those during PTI (Peng *et al.*, 2018; Hake & Romeis, 2019; Nguyen *et al.*, 2021). PTI for example leads to an early and transient accumulation of ROS, whereas ETI initiates a second, stronger ROS burst (Yuan *et al.*, 2021).

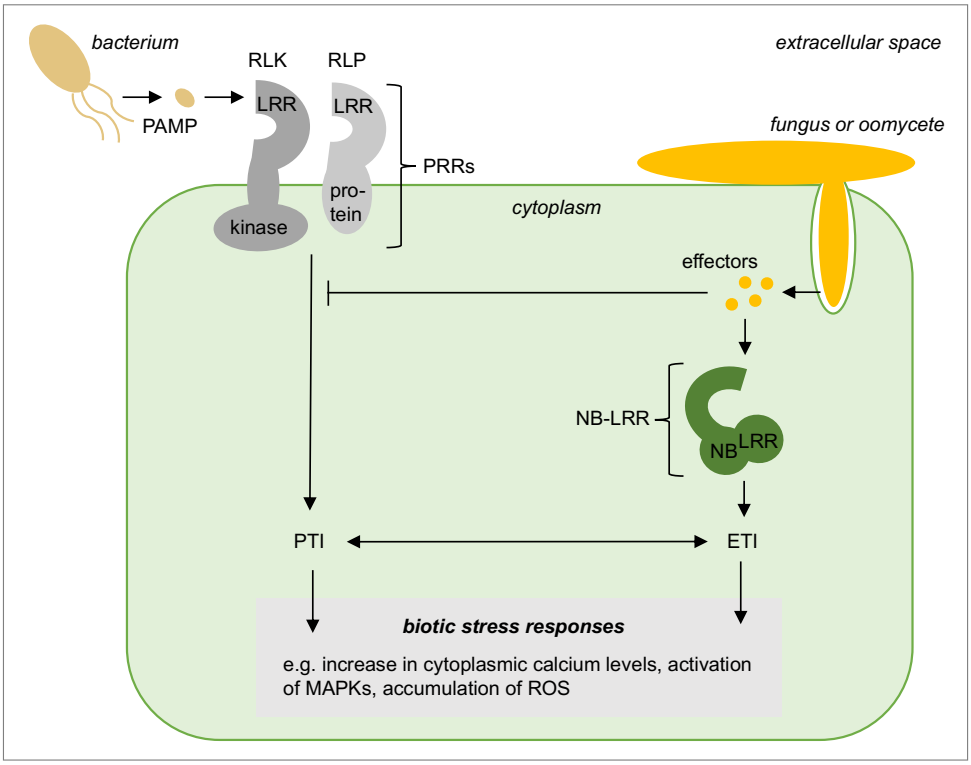

Figure 8. Schematic representation of innate immune responses in Arabidopsis.

The plant innate immunity is composed of two layers: PTI and ETI that synergistically boost each other. PTI is triggered when plants recognize MAMPs or PAMPs in their extracellular space using plasma membrane-located PRRs. Plant PRRs are classified as RLKs or RLPs. To circumvent PTI, plant pathogens frequently secrete effector proteins into plant cells. ETI is activated when plants recognize effector proteins by intracellular receptor proteins. Although PTI and ETI are differently activated, both trigger similar responses, for example increasing the cytoplasmic calcium levels, activating MAPKs, and accumulating ROS. Arrows, indicate positive regulations. Figure is inspired by Dodds and Rathjen (2010) and Nguyen *et al.* (2021). PTI, PAMP-triggered immunity; ETI, effector-triggered immunity. MAMP/PAMP, microbe or pathogen associated molecular pattern; PRR, pattern recognition receptor; RLK, receptor-like kinase; RLP, receptor-like protein; MAPK, mitogen-activated protein kinases; LRR, leucine rich repeat; NB, nucleotide-binding domain.

In addition to the induction of local resistance by PTI and ETI, plants can acquire a systemic resistance in distal tissues that have not been infected by pathogens (Hake & Romeis, 2019; De Kesel *et al.*, 2021). This relies on the formation of local signals that are transported through the whole plant (Gao *et al.*, 2015). Resistance induction in the whole plant is connected to systemic acquired resistance (SAR) (Conrath, 2006). In contrast to ETI that is linked to programmed cell death, SAR stimulates cell survival.

In Arabidopsis, salicylic acid (SA) and *N*-hydroxypipecolic acid (NHP) are the predominant signals mediating SAR (Fu & Dong, 2013; Hartmann & Zeier, 2019; Tian & Zhang, 2019). NHP is biosynthesized from pipecolic acid (Pip) by FLAVIN-DEPENDENT MONOOXYGENASE1 (FMO1) (Hartmann & Zeier, 2019). Pip represents a non-proteinogenic amino acid that is also connected to SAR. Formation of Pip from lysine is mediated by AGD2-LIKE DEFENSE RESPONSE PROTEIN1 (ALD1) and SAR-DEFICIENT4 (SARD4) (Wang *et al.*, 2018a; Yildiz *et al.*, 2021).

1.3 Regulation of plant responses to photoperiod and biotic stress

Phytohormones (**section 1.3.1**) and reactive oxygen species (**section 1.3.2**) are crucial for plants to regulate responses to various stresses (Verma *et al.*, 2016; Mittler *et al.*, 2022) including photoperiod and biotic stresses (Roeber *et al.*, 2022; Cortleven *et al.*, 2022). The involved signalling pathways are introduced in the following sections.

1.3.1 Phytohormones

Phytohormones represent plant-derived signalling compounds that mediate light-dependent processes, such as growth and development, or photosynthesis (Lau & Deng, 2010; Müller & Munne-Bosch, 2021), and responses to biotic stresses (Kumari & Singh, 2022).

The phytohormone content regulates the responsiveness of plants to incoming light signals (Lau & Deng, 2010) and accumulation of certain phytohormones, such as SA or JA, is, in turn, influenced by light signals in Arabidopsis (Nitschke *et al.*, 2016; Sharma *et al.*, 2022; Lajeunesse *et al.*, 2023). In addition, signalling by several other phytohormones, including abscisic acid (ABA), ethylene (ET), gibberellin (GA) and cytokinin (CK), is influenced by light. More specifically by central light signalling components, such as PIFs and HY5 (Lau & Deng, 2010; Liu *et al.*, 2017b).

For coping with biotic stresses, plants especially rely on signalling by SA, JA, and ET (Kumari & Singh, 2022). Respective signalling pathways can work antagonistically or synergistically (Van der Does *et al.*, 2013).

In the following sections, biosynthesis of and signalling by phytohormones that are important in plant responses to different light environments and biotic stresses will be considered in more detail (**section 1.3.1.1 and 1.3.1.2**).

1.3.1.1 Jasmonates

Jasmonates represent a group of lipid-derived phytohormones that comprises JA, and its derivatives (Wasternack & Song, 2017). Jasmonates are known to modulate a variety of processes and responses, including developmental processes, production of secondary metabolites and signalling in response to different abiotic and biotic stresses (**section 1.2**) (Wasternack & Song, 2017; El Sabagh *et al.*, 2022). Signalling pathways mediated by JA and light depend on each other (Robson *et al.*, 2010; Kazan & Manners, 2011; Balfagon *et al.*, 2019). For example, jasmonates regulate the expression of genes

important for photosynthesis, and stimulate the turnover of proteins required for light capture. Chlorophyll biosynthesis is repressed by CORONATINE INSENSITIVE1 (COI1) that is centrally involved in JA signalling (Robson *et al.*, 2010). Besides, JA biosynthesis/signalling mediate responses to certain light conditions, such as FR light, while phytochromes, such as phyA, modulate the expression of JA-responsive genes (Kazan & Manners, 2011).

In Arabidopsis, biosynthesis of JA involves chloroplasts, peroxisomes, and cytoplasm (**Figure 9**) (Wasternack & Song, 2017). First, α-linolenic acid (α-LeA) is enzymatically released from galactolipids representing a class of chloroplast thylakoid membranes (Hölzl *et al.*, 2006; Wasternack & Song, 2017; Wasternack & Hause, 2019).

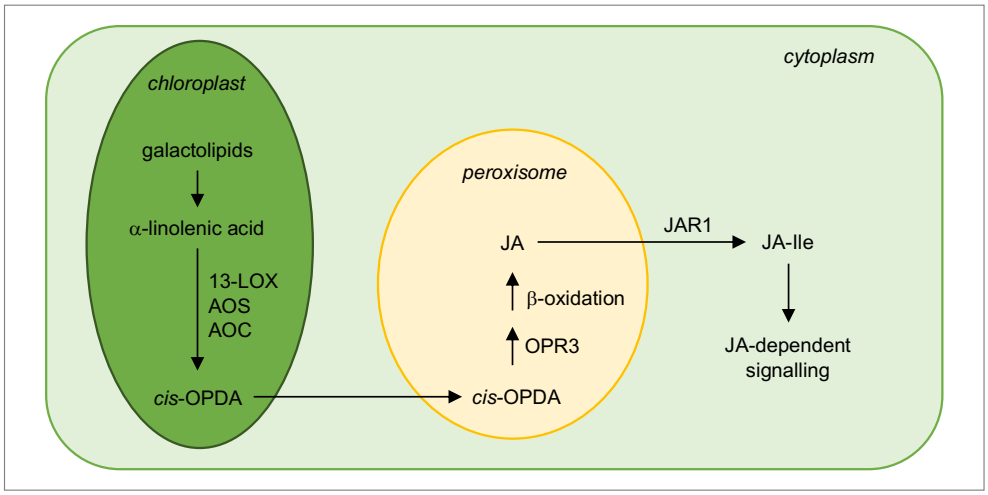

Figure 9. JA biosynthesis in Arabidopsis.

JA biosynthesis involves three cellular compartments: chloroplasts, peroxisomes, and cytoplasm. In chloroplasts, α-linolenic acid is released from galactolipids. Afterwards, α-linolenic acid is enzymatically processed by 13-LOX, AOS, and AOC resulting in the formation of *cis*-OPDA, which is transported to peroxisomes. In the peroxisomes, *cis*-OPDA is reduced by OPR3 and β-oxidized which results in the formation of JA. JA is released to the cytoplasm where it becomes metabolized, thereby forming a variety of JA derivatives. Conjugation of JA by JAR1 forms JA-Ile, the most biologically active JA derivative. Figure is inspired by Wasternack and Song (2017). 13-LOX, 13-LIPOXYGENASE; AOS, ALLENE OXIDE SYNTHASE; AOC, ALLENE OXIDE CYCLASE; *cis*-OPDA, *cis*-12-oxophytodienoic acid; OPR3, OPDA REDUCTASE3; JA, jasmonic acid; JAR1, JASMONOYL-ISOLEUCINE SYNTHASE1; JA-Ile, jasmonic acid isoleucine.

Afterwards, α-LeA is enzymatically processed by 13-LIPOXYGENASE (13-LOX), ALLENE OXIDE SYNTHASE (AOS), and ALLENE OXIDE CYCLASE (AOC), resulting in the formation of *cis*-12-oxophytodienoic acid (OPDA) that is exported from chloroplasts. Second, OPDA is imported into peroxisomes where it is reduced by OPDA REDUCTASE3 (OPR3) (Wasternack & Song, 2017), and subsequently shortened in its pentenyl side chains by β-oxidation, resulting in the formation of JA (also referred to as 7-iso-JA). Third, JA is released into the cytoplasm where it is metabolized resulting in the formation of a variety of JA derivatives. Conjugation of JA by JASMONOYL-ISOLEUCINE SYNTHASE1 (JAR1) results in the formation of JA-isoleucine (JA-Ile) representing the most biologically active JA derivative (**Figure 9**) (Wasternack & Hause, 2019; Liu & Timko, 2021).

Jasmonate signalling in the nucleus involves the SCF^{COI1}-JAZ coreceptor complex consisting of (i) the ubiquitin E3 ligase complex SCF^{COI1} and (ii) JASMONATE-ZIM DOMAIN (JAZ) proteins (**Figure 10**) (Pauwels & Goossens, 2011).

JAZ proteins interact and repress transcription regulators of JA-responsive genes, such as JASMONATE INSENSITIVE1 (JAI1/MYC2), a positive regulator of JA signalling, under conditions with low JA-Ile in the nucleus (Wasternack & Song, 2017). Under conditions with high JA-Ile levels, JA-Ile promotes interaction of the SCF^{COI1} E3 ubiquitin ligase complex and JAZ proteins resulting in the formation of the SCF^{COI1}-JAZ coreceptor complex (Thines *et al.*, 2007). This leads to the polyubiquitination of JAZ proteins, which marks them for degradation by the 26S proteasome, thereby removing the JAZ protein-mediated repression of JA-responsive genes (**Figure 10**) (Wasternack & Song, 2017; El Sabagh *et al.*, 2022).

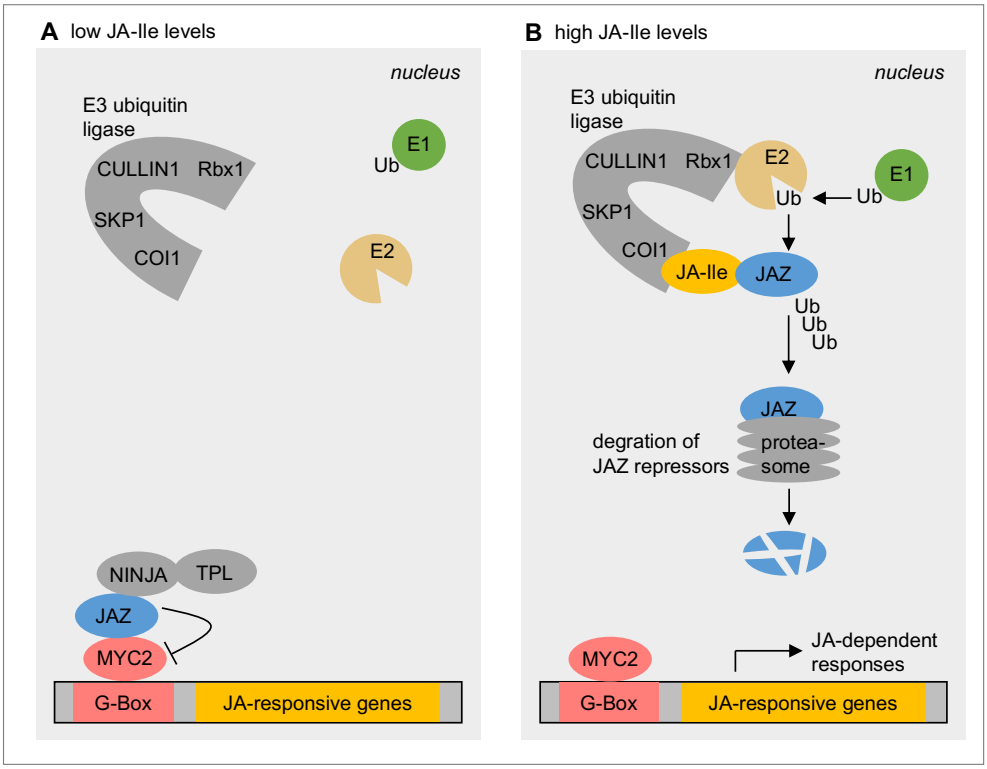

Figure 10. JA signalling in the nucleus of Arabidopsis.

(A) Under conditions with low levels of JA-Ile, JAZ proteins interact and repress transcription regulators of JA-responsive genes, such as MYC2. **(B)** Under conditions of high levels of JA-Ile, JA-Ile promotes the interaction of the SCF^{COI1} E3 ubiquitin ligase complex and JAZ proteins resulting in the formation of the SCF^{COI1}-JAZ coreceptor complex. JAZ proteins in this coreceptor complex are polyubiquitinated and subsequently degradated via the 26S proteasome. This removes JAZ-mediated repression of JA-responsive genes, thereby inducing JA-dependent responses in plants. Arrows, indicate positive regulations; cut lines, indicate negative regulations. Figure is inspired by Wasternack and Song (2017). JA, jasmonic acid; JA-Ile, jasmonic acid-isoleucine, Ub, ubiquitin; MYC2, JASMONATE INSENSITIVE1; JAZ, JASMONATE-ZIM DOMAIN.

1.3.1.2 Salicylic acid

The phytohormone salicylic acid (SA, 2-hydroxybenzoic acid) represents a phenolic compound (Backer *et al.*, 2019; Ullah *et al.*, 2022). SA (and its derivatives) regulates plant growth and development and

signalling in response to both abiotic and biotic stresses (**section 1.2**). In view of biotic stress responses, SA is known to be particularly important for both local and systemic induction of plant immune responses against (biotrophic) pathogen attacks (Maruri-Lopez *et al.*, 2019). SA regulates phytochromes, and phyA and phyB modulate in turn the expression of SA-responsive genes (Karpinski *et al.*, 1999; Genoud *et al.*, 2002; Lajeunesse *et al.*, 2023). SA contents alternate dependent on the plant circadian clock as plants show an increase in SA levels during the day and a decrease during the night (Zheng *et al.*, 2015; Lajeunesse *et al.*, 2023). The regulation of the SA content is important for the acclimation to different light conditions, as plant growth and photosynthesis are optimized by SA (Mateo *et al.*, 2004).

Biosynthesis of SA is realised through two independent pathways, (i) the PHENYLALANINE AMMONIA LYASE (PAL) pathway, and (ii) the ISOCHORISMATE SYNTHASE (ICS) pathway (**Figure 11**) (Backer *et al.*, 2019; El Sabagh *et al.*, 2022; Peng *et al.*, 2021). Both pathways rely on chorismic acid (chorismate) representing the final product of the shikimic acid pathway for SA biosynthesis (Seyfferth & Tsuda, 2014; Ullah *et al.*, 2022). In the (i) PAL pathway, chorismate-derived phenylalanine is converted to *trans*-cinnamic acid by PAL enzymes in the cytoplasm. Oxidation of *trans*-cinnamic acid results in the formation of benzoic acid (BA), which is hydroxylated, thereby generating SA (Peng *et al.*, 2021). In the (ii) ICS pathway, chloroplast-localized ICS1/2 enzymatically convert chorismate to isochorismate (Wildermuth *et al.*, 2001), which is exported by ENHANCED DISEASE SUSCEPTIBILITY5 (EDS5/SID1), representing a MULTIDRUG AND TOXIC COMPOUND EXTRUSION (MATE)-transporter into the cytoplasm. Cytoplasmic isochorismate is converted to isochorismate-9-glutamate by AVRPPHB SUSCEPTIBLE3 (PBS3) enzymes, which allocates to SA (spontaneously or by ENHANCED *PSEUDOMONAS* SUSCEPTIBILITY1, EPS1) (**Figure 11**) (Seyfferth & Tsuda, 2014; Lefevere *et al.*, 2020; Peng *et al.*, 2021).

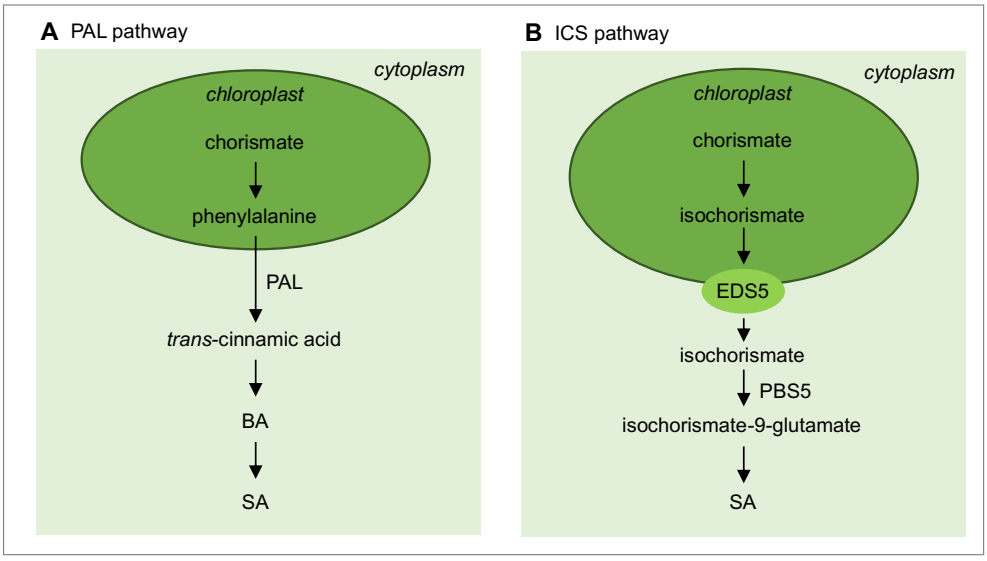

Figure 11. SA biosynthesis in Arabidopsis.

SA biosynthesis is realised through two independent pathways, PAL, and ICS pathways, that both require chorismate. **(A)** In the PAL pathway, chorismate-derived phenylalanine is converted to *trans*-cinnamic acid, thereby forming BA that becomes hydroxylated resulting in formation of SA. **(B)** In the ICS pathway, chorismate is enzymatically converted to isochorismate by chloroplast-localized ICS1/2 and afterwards exported to the cytoplasm by EDS5. In the cytoplasm, isochorismate is enzymatically converted to isochorismate-9-glutamate by PBS3, which afterwards allocates to SA. Figure is inspired by Peng *et al.* (2021). SA, salicylic acid; PAL, PHENYLALANINE

AMMONIA LYASE; ICS, ISOCHORISMATE SYNTHASE; BA, benzoic acid; EDS5/SID1, ENHANCED DISEASE SUSCEPTIBILITY5; PBS3, AVRPPHB SUSCEPTIBLE3.

Responses to SA are mediated by the transcriptional cofactor NONEXPRESSOR OF PATHOGENESIS-RELATED GENES1 (NPR1) and its paralogs NPR3/4 (Janda *et al.*, 2020; Peng *et al.*, 2021). As transcriptional cofactors, NPR1 and NPR3/4 indirectly regulate the expression of the SA-responsive *PATHOGENESIS-RELATED GENE1* (*PR1*), encoding an important plant defence protein, positively or negatively, respectively (Backer *et al.*, 2019).

Transcription coregulated by NPR1 depend on the plant cellular redox state (**section 1.3.2**). NPR1 forms cytoplasmic oligomers through intermolecular cysteine bonds (at Cysteine-82 and Cysteine-216 residues) under non-stressful conditions (**Figure 12**) (Mou *et al.*, 2003; Mittler *et al.*, 2022).

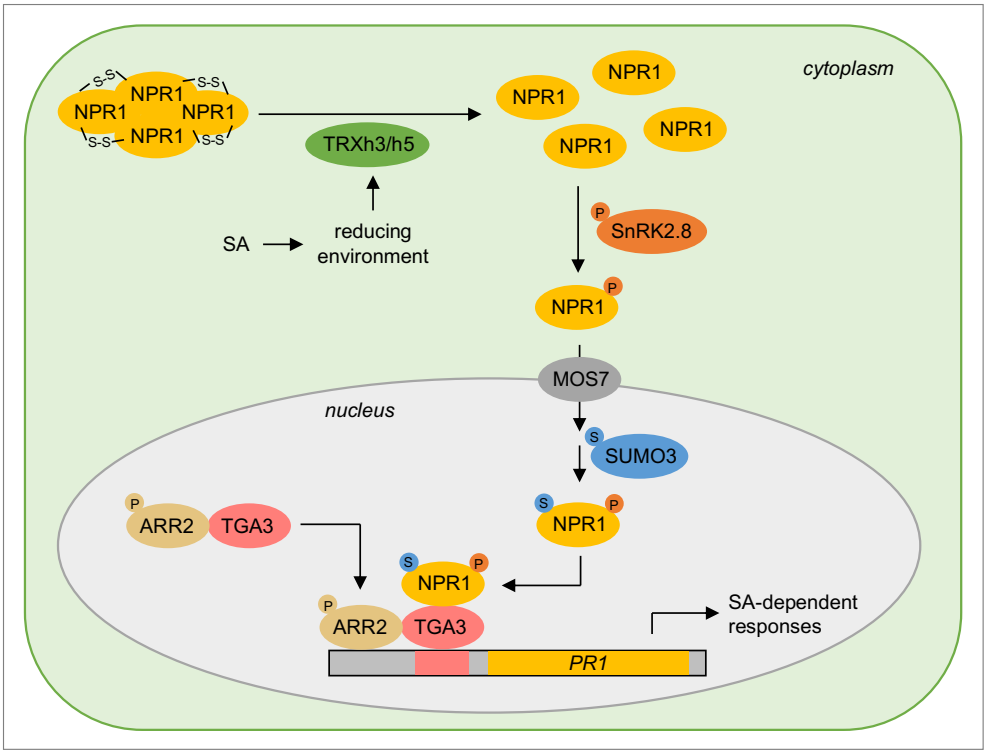

Figure 12. SA signalling involving NPR1.

Under non-stressful conditions, NPR1 forms cytoplasmic oligomers through intermolecular cysteine bonds (indicated by -S-S- in the Figure). Under stressful conditions, accumulation of SA and oxidative stress stimulate the reduction of cysteines of NPR1 oligomers involving TRXh3/h5. Monomeric NPR1 is phosphorylated by SnRK2.8 (P, indicates phosphorylated protein) and imported to the nucleus requiring the nucleoporin MOS7. In the nucleus, NPR1 is sumoylated (S, indicates sumoylated protein). Nuclear NPR1 coregulates the transcription of several transcription factors, such as TGA3 that interacts with ARR2, thereby regulating the expression of genes, e.g. *PR1*. Figure is inspired by Backer *et al.* (2019) and Choi *et al.* (2010). NPR1, NONEXPRESSOR OF PATHOGENESIS-RELATED GENES1; SA, salicylic acid; TRXh3/h5, THIOREDOXIN H-type 3/H-type 5; SnRK2.8, SNF1-RELATED PROTEIN KINASE 2.8; MOS7, MODIFIER OF SNC1,7; TGA3, TGACG-binding transcription factor; ARR2, ARABIDOPSIS RESPONSE REGULATOR2; *PR1*, *PATHOGENESIS-RELATED GENE1*; SUMO3, SMALL UBIQUITIN-LIKE MODIFIER3.

Accumulation of SA that is followed by oxidative stress, thereby reducing the plant cellular environment, results in a reduction of the Cysteine-156 of NPR1 oligomers by THIOREDOXIN H-type 3 (TRXh3) and

H-type 5 (TRXh5) that leads to the dissociation of oligomeric NPR1 into monomers (**Figure 12**) (Mou *et al.*, 2003; Tada *et al.*, 2008; Backer *et al.*, 2019). Monomeric NPR1 is phosphorylated (at Serine-589 and Threonine-373) by the serine/threonine kinase SNF1-RELATED PROTEIN KINASE 2.8 (SnRK2.8) and subsequently translocated into the nucleus through a bipartite nuclear localization sequence (NLS) located at the C-terminus of NPR1 (Kinkema *et al.*, 2000; Spoel *et al.*, 2009; Lee *et al.*, 2015; Backer *et al.*, 2019). Nuclear import of NPR1 to the nucleus requires the nucleoporin MODIFIER OF SNC1,7 (MOS7/Nup88) representing a plant nuclear pore complex component that is essential for plant immune responses (Wiermer *et al.*, 2010; Lee *et al.*, 2015; Cheng *et al.*, 2016). Nuclear NPR1 regulates transcription by interaction with several TFs, such as TGA TFs and the cytokinin-regulated ARABIDOPSIS RESPONSE REGULATOR2 (ARR2), thereby modulating plant responses to several stimuli (**Figure 12**) (Backer *et al.*, 2019).

1.3.2 Reactive oxygen species and antioxidants

Reactive oxygen species (ROS) are important regulators of light-dependent processes and biotic stress responses in plants (D'Alessandro *et al.*, 2020; Borbely *et al.*, 2022).

ROS form a group of molecules, including singlet oxygen ($^1O_2$), superoxide ($O_2^{\cdot-}$), hydrogen peroxide (H_2O_2), and the hydroxyl radical ($HO^{\cdot}$), that originate from molecular oxygen (O_2). In contrast to molecular oxygen, ROS can, due to their high chemical reactivity, oxidize cellular molecules. To prevent oxidative damage by excess ROS, ROS are eliminated (scavenged) by both non-enzymatic and enzymatic antioxidants (Mittler *et al.*, 2022; Qamer *et al.*, 2021). Reduced glutathione (GSH), phenolics, α-tocopherol, and flavonoids are examples of non-enzymatic antioxidants (Mohammadi *et al.*, 2021; Qamer *et al.*, 2021). Examples for enzymatic antioxidants are ASCORBATE PEROXIDASEs (APXs), CATALASEs (CATs), GLUTATHIONE PEROXIDASEs (GPXs), SUPEROXIDE DISMUTASEs (SODs), GLUTATHIONE *S*-TRANSFERASEs (GSTs), PEROXIDASE REDUCTASEs (PRXs), GLUTATHIONE REDUCTASEs (GRs), DEHYDROASCORBATE REDUCTASEs (DHAR) and MONO-DEHYDROASCORBATE REDUCTASEs (MDARs) (Qamer *et al.*, 2021). In addition to production and scavenging of ROS, transport of ROS to certain other plant cellular compartments or tissues adjusts the respective levels (Mittler, 2017; 2022).

In plants, basal cellular ROS levels are influenced by several parameters including their developmental stage, and physiological condition (e.g. phytohormone content (Müller & Munne-Bosch, 2021), the circadian clock, or external stimuli. Changes in cellular ROS levels affect the structures and functions of various plant proteins (Mittler *et al.*, 2022). In addition, ROS can function as signalling molecules in many different pathways and are crucial for the responses to both abiotic and biotic stresses (Mittler, 2017). Specificity in ROS signalling is achieved by different signatures (patterns) of ROS accumulation and scavenging which depend on the stimuli and are specific to the respective cellular compartment (Mittler *et al.*, 2022).

The light environment represents an external stimulus that affects both the cellular ROS levels as well as the activity of antioxidants that scavenge ROS (Shim & Imaizumi, 2015; Borbely *et al.*, 2022; Roeber *et al.*, 2022). In leaves of Arabidopsis adapted to low light intensities, already a short period (30 min) of

high light intensities is sufficient to induce the expression of *ASCORBATE PEROXIDASE2* (*APX2*) and to increase the levels of H_2O_2 (Fryer *et al.*, 2003). Exposure to high light intensities for a limited time positively influences photosynthesis of low light-adapted plants; however, too long exposure to high light intensities can result in excess excitation energy (EEE) referring to energy that is in excess absorbed by light-harvesting complexes (Karpinski *et al.*, 1999). Extended exposure to EEE-causing conditions results in the accumulation of ROS (Karpinski *et al.*, 1999; Vanderauwera *et al.*, 2005; Mühlenbock *et al.*, 2008).

In addition, changes in the light quality influence ROS levels (Borbely *et al.*, 2022). In Arabidopsis, red light treatment (6 h) sensed by phyB that interacts with the plant light signalling TF HY5 induces the production of H_2O_2 and EDS1-mediated accumulation of SA (Ahn *et al.*, 2022). Besides, red light affects the plant plastoquinone (PQ) pool, thereby regulating LESION SIMULATING DISEASE1 (LSD1) that functions as a negative regulator of programmed cell death (Chai *et al.*, 2015; Bernacki *et al.*, 2019). In addition to red light, blue light also influences the redox state. Light-dependent activation of cryptochromes regulates the activity of antioxidants, stimulates accumulation of ROS, and induces the expression of ROS-responsive genes (El-Esawi *et al.*, 2017; Borbely *et al.*, 2022). Notably, Borbely *et al.* (2022) discussed that blue light activates the plant redox system more efficiently than red light.

Another parameter of the light environment that influences the plant redox state is the photoperiod (Roeber *et al.*, 2022). In Arabidopsis, growth under certain photoperiods (SD or LD) modulates the responsiveness of the plants to conditions that stimulate ROS accumulation (Shim & Imaizumi, 2015). In particular, the enzymatic activity of CATs that are responsible for the dismutation of H_2O_2 molecules into water and molecular oxygen (Mhamdi *et al.*, 2010) is influenced by the photoperiod (Becker *et al.*, 2006; Shim & Imaizumi, 2015). Plants grown in long photoperiods possess a higher enzymatic activity of CATs than plants grown in short photoperiods (Shim & Imaizumi, 2015). In addition to CATs, also the activities of other enzymes, such as APXs and NAPD-MALATE DEHYDROGENASE, are enhanced in Arabidopsis plants that are grown in long photoperiods (Becker *et al.*, 2006; Roeber *et al.*, 2022). Interestingly, also the duration of the dark period can affect the cellular ROS levels (Borbely *et al.*, 2022). Moreover, also a suddenly occurring prolongation of the photoperiod (leading to photoperiod stress, **section 1.2.1**) results in accumulation of peroxides, affects the activities of enzymatic antioxidants, and induces the expression of apoplastic *PRX* genes (*PRX4/33/34/37*) in Arabidopsis previously grown in short photoperiods (during the night following the treatment) (Abuelsoud *et al.*, 2020).

1.4 Regulation of biotic stress responses by the light environment in plants

The light environment, including light quality, quantity and photoperiod, influences plant responses to abiotic and biotic stresses (Roeber *et al.*, 2021). How plants perform against attacks by insects and pathogens is strongly influenced by light (Bechtold *et al.*, 2005; Delprato *et al.*, 2015; Roeber *et al.*, 2021; Lajeunesse *et al.*, 2023).

1.4.1 Light availability

In Arabidopsis, complete resistance responses are only activated when light is available during the primary pathogen infection (Zeier *et al.*, 2004; Griebel & Zeier, 2008; Shimizu *et al.*, 2021). Absence of light (during the normal photoperiod leading to extended darkness) increases the apoplastic bacterial growth of the avirulent *Pseudomonas syringae* pv. *maculicola* (*Psm*) strain ES4326 containing the *avrRpm1* avirulence gene (Zeier *et al.*, 2004; Griebel & Zeier, 2008) or avirulent *P. syringae* pv. *tomato* (*Pst*) DC3000 carrying the avirulence gene *avrRpt2* in Arabidopsis (Genoud *et al.*, 2002). Similar observations were made by Griebel and Zeier (2008) who showed that absence of light after infection enhanced the susceptibility of *A. thaliana* ecotype Dijon-17 to Turnip Crinkle Virus (TCV). In Arabidopsis, several early and local resistance responses depend on light, including the transcription induction of defence genes like *PATHOGENESIS-RELATED1* (*PR1*) and *PHENYLALANINE AMMONIA LYASE* (*PAL*), the accumulation of free salicylic acid (SA) and glucoside-bound SA (SAG), and the development of hypersensitive response (HR) (Lozano & Sequeira, 1969; Guo *et al.*, 1993; Genoud *et al.*, 2002; Zeier *et al.*, 2004; Griebel & Zeier, 2008). The development of HR in Arabidopsis in response to infection with *Pst avrRpt2* requires functional chloroplasts (Genoud *et al.*, 2002). A possible explanation for the light dependency of HR in *Nicotiana tabacum* is suggested by Liu *et al.* (2007) who proposed that generation of ROS (**section 1.3.2**) by chloroplasts is inhibited in the absence of light; thereby delaying pathogen-induced HR. Chloroplast-derived ROS, for example singlet oxygen ($^1O_2$), induce the expression of nuclear-encoded defence genes via retrograde signals in response to recognition of PAMPs (Nomura *et al.*, 2012; Iqbal *et al.*, 2021; Littlejohn *et al.*, 2021; Foyer & Hanke, 2022). In Arabidopsis, this is most likely controlled by the calcium-sensing receptor CAS that is associated with chloroplast thylakoid membranes. Apart from generating calcium signals, CAS promotes biosynthesis of SA in response to flg22 (PAMPs). Experiments with *cas-1* mutants infected with virulent *Pst* DC3000, avirulent *Pst avrRpm1* or *Pst avrRpt2* showed that CAS is essential for both PTI and ETI, including HR, suggesting that chloroplasts are also important for both (Nomura *et al.*, 2012). To circumvent the positive effects of chloroplast-mediated signalling on plant resistance responses, *P. syringae* has evolved effector proteins, such as HopI1 or HopN1, that prevent SA accumulation and alter the structure of thylakoid membranes in chloroplasts or repress the generation of chloroplast-derived ROS and impact the activity photosystem II, respectively (Jelenska *et al.*, 2007; Kangasjärvi *et al.*, 2012; Rodriguez-Herva *et al.*, 2012). In *A. thaliana* ecotype Dijon-17, signalling mediated by *HRT*, a putative plant resistance (R) gene, that activates HR is dependent on light but independent of the photoreceptors phyA and phyB (**section 1.1.1**) (Chandra-Shekara *et al.*, 2006). Zeier *et al.* (2004) pointed out that transcription of *PAL*, encoding an enzyme that contributes in addition to ISOCHORISMATE SYNTHASE1 (ICS1) (Wildermuth *et al.*, 2001) to SA accumulation in Arabidopsis, stimulates *PR1* transcription and establishment of HR under light conditions. As SA and JA signalling pathways act often mutually antagonistic (Van der Does *et al.*, 2013), higher SA content in the presence of light might restrict the accumulation of JA, which is not the case in the absence of light. In addition to JA, camalexin accumulates in Arabidopsis infected by *Psm avrRpm1* only in the absence of light (Zeier *et al.*, 2004) indicating that light availability might restrict both. Besides, there are also local resistance responses that are activated independent of light upon infection by *Psm avrRpm1*, such as the transcription induction of *GLUTATHIONE S-TRANSFERASE1* (*GST1*) or oxidative burst-related hydrogen peroxide accumulation. In addition to these local resistance

responses, light availability during a primary infection by *Psm avrRpm1* is crucial for establishment of systemic resistance responses. This was concluded from experiments showing that light illumination during a primary infection by *Psm avrRpm1* decreases bacterial growth in systemic Arabidopsis leaves exposed to virulent *Psm* as secondary infection. Under normal light conditions, SA and glucoside-bound SA (SAG) is accumulated and *PR1* transcription is induced in systemic, uninfected tissues during a primary infection by *Psm avrRpm1*, which was not the case in the absence of light (Zeier *et al.*, 2004).

In addition to its influence on plant responses to pathogen attack, light also directly affects the virulence and mobility of plant pathogens (Oberpichler *et al.*, 2008; Roden & Ingle, 2009). Plant pathogens, for example *Pst* or *Botrytis cinerea*, possess photoreceptors allowing them to perceive light quality, quantity, and duration, and to respond accordingly (Schumacher, 2017; Losi & Gärtner, 2021). Light sensing and responding might enable plant pathogens to improve their host infection and/or their fitness (Losi & Gärtner, 2021); however, little is known about the concrete roles of pathogenic light signalling in their interactions with plants (Schumacher, 2017).

1.4.2 Light intensity

Light availability *per se* is not the only light environment parameter shaping plant resistance responses, light intensity is another (Roeber *et al.*, 2021): In Arabidopsis, exposure to highlight conditions during a primary infection by *Psm avrRpm1* limits the bacterial growth in systemic tissue when exposed to virulent *Psm* as secondary infection. In contrast to normal light conditions, the systemic resistance establishment under high light is less dependent on SA (Zeier *et al.*, 2004; Bechtold *et al.*, 2005). Similar observations were made by Mühlenbock *et al.* (2008) who showed that exposure to high light intensities decreases growth of virulent *Pseudomonas syringae* pv. *tomato* (*Pst*) DC3000 in Arabidopsis locally and systemically. In non-acclimated Arabidopsis, high light increases the excitation energy in excess to the energy that is normally needed for photosynthesis (Bechtold *et al.*, 2005; Roden & Ingle, 2009). The resulting excess excitation energy (EEE) is potentially destructive for the photosynthetic apparatus (Bechtold *et al.*, 2005). Responses to EEE share characteristics with plant responses providing resistance against pathogens: In both cases responses are modulated by redox changes of the plastoquinone (PQ) pool in chloroplasts and involve programmed cell death (Mühlenbock *et al.*, 2008). The redox modifications of the PQ pool activate the transcription of genes commonly regulated in response to EEE and resistance against pathogens; and stimulate the production of ROS (**section 1.3.2**) and ethylene (Bechtold *et al.*, 2005; Mateo *et al.*, 2004; Mühlenbock *et al.*, 2008; Roden & Ingle, 2009; Iqbal *et al.*, 2021). The programmed cell death stimulated in response to EEE relies on well-known regulators of plant resistance, namely ENHANCED DISEASE SUSCEPTIBILITY1 (EDS1) and PHYTOALEXIN DEFICIENT4 (PAD4), acting upstream of ROS and ethylene production. The effects of EDS1 and PAD4 on ROS production are regulated by LESION SIMULATING DISEASE1 (LSD1) that limits the spread of programmed cell death, thereby enabling acclimation to EEE or inducing resistance to pathogens (Mateo *et al.*, 2004; Mühlenbock *et al.*, 2008). The similarities between responses to both EEE and pathogens allow for light intensity-mediated modulation of plant resistance responses.

1.4.3 Photoperiod

Another light environment parameter regulating plant resistance responses is the photoperiod. The photoperiod influences plant resistance against bacterial, viral, and fungal pathogens in different species including Arabidopsis, rhododendron, tomato, maize, and brown mustard (Roeber *et al.*, 2022).

For *A. thaliana* ecotype Landsberg *erecta* (L*er*) it was observed that plants grown under LD conditions are less susceptible to infection by cauliflower mosaic virus (CaMV isolate Cabb B-JI) than plants grown under SD conditions. Interestingly, the positive effect of the longer photoperiod is detected although the virus accumulation is even higher under this condition (Cecchini *et al.*, 2002). Growth under longer photoperiods accelerates cell division and leaf expansion in WT ecotype Columbia-4 (Col-4) but decreases at the same time the total number of rosette leaves (**Figure 13**) (Baerenfaller *et al.*, 2015). Differences in protein levels observed in the Arabidopsis WT under SD and LD conditions are mainly associated with the photoperiod-dependent plant growth. However, certain proteins known to be involved in defence responses, such as PATHOGENESIS-RELATED GENE2 (PR2) and PR5, are specifically higher abundant in WT plants grown under longer photoperiods (**Figure 13**). In addition, transcripts of genes involved in plant biotic stress responses and flavonoid metabolism are enriched in the WT grown under longer photoperiods (**Figure 13**) (Baerenfaller *et al.*, 2015).

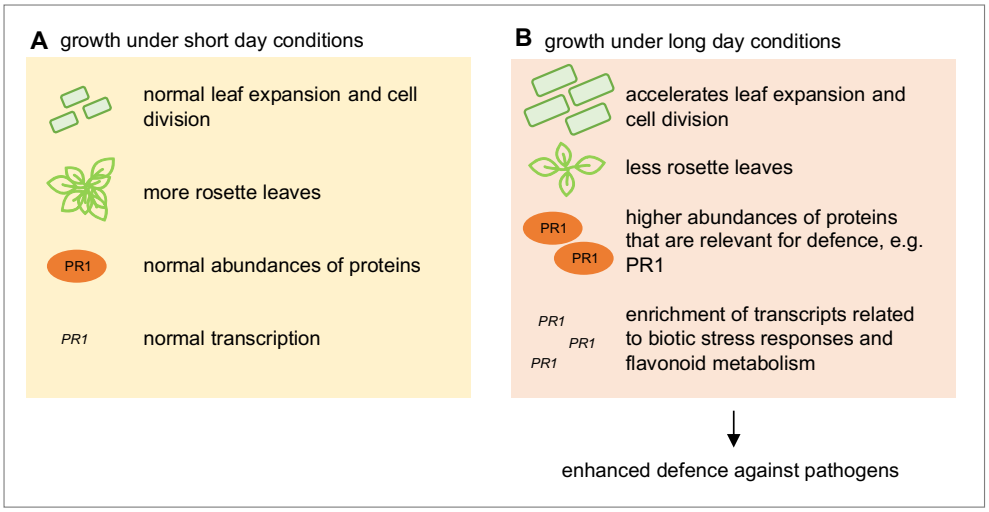

Figure 13. Comparison of growth under short and long day condition.

(A-B) In comparison to growth under short day conditions, growth under long day conditions accelerates leaf expansion and cell division of Arabidopsis, while the total number of rosette leaves decreases. Proteins and transcripts that are relevant for defence responses are enriched under long day conditions.

A positive effect of a longer photoperiod (in combination with high light) on the susceptibility of plants to pathogens is also shown for *Rhododendron* (cv. Elisabeth) infected with the fungus *Erysiphe* sp. Cut plant leaves are colonized by longer primary hyphae of *Erysiphe* sp. when kept under SD independent of the light intensity or LD at low light than under LD at high light conditions (Kenyon *et al.*, 2002). These results indicate that both photoperiod and light intensity are important parameters determining the outcome of plant-pathogen-interactions.

In addition, also a transfer from SD to LD conditions influences the susceptibility of plants to pathogen attacks. Arabidopsis plants shifted from SD to LD conditions when infected with *B. cinerea* are less susceptible to this necrotrophic fungus than plants kept under SD conditions. The transfer to longer photoperiods activates JA-dependent responses in the plants that are known to be particularly effective against *B. cinerea* infections. Although the JA content is not affected by the transfer to a longer photoperiod, the expression of JA signalling genes, such as the *JASMONATE INSENSITIVE1* (*JAI1/MYC2*), is induced (Cagnola *et al.*, 2018). The transcription activation of target genes by MYC2 is negatively regulated by JA ZIM-DOMAIN (JAZ) proteins that are in turn negatively regulated by GIBBERELLIN ACID INSENSITIVE (DELLA) proteins (Hou *et al.*, 2010; Song *et al.*, 2022). Cagnola *et al.* (2018) showed that a transfer from SD to LD conditions leads to an accumulation of the DELLA protein REPRESSOR OF *ga1-3* (RGA) (**Figure 14**). The latter depends on CONSTITUTIVE PHOTOMORPHOGENIC1 (COP1) whose nuclear abundance decreases in response to the photoperiodic shift.

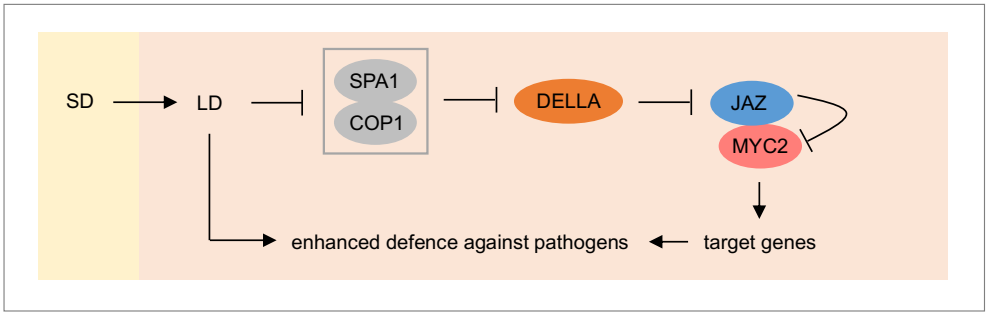

Figure 14. Transferring plants from short day to long day condition enhances resistance against pathogens.

Shifting Arabidopsis from SD to LD conditions, decreases the nuclear abundance of SPA1/COP1 functioning as a negative regulator of DELLA proteins. The DELLA protein RGA accumulates in response to a shift to LD and the expression of JA signalling genes, such as *MYC2*, increases. SD, short day; LD, long day; SPA1/COP1, SUPPRESSOR OF PHYA1/CONSTITUTIVE PHOTOMORPHOGENIC1; JAZ, JA ZIM-DOMAIN; MYC2, *JASMONATE INSENSITIVE1*.

Similarly, *Zea mays Hm1*^{A} (maize) seedlings that are transferred to a longer photoperiod after infection with *Cochliobolus carbonum* race 1 (CCR1) are less susceptible to this fungus than seedlings kept under the normal growing photoperiod. *Z. mays Hm1*^{A} plants possess a partial loss-of-function allele of *Hm1* that encodes a NADPH-dependent HC-toxin reductase (HCRT) required for the inactivation of the HC-toxin effector of CCR1, thereby increasing seedling susceptibility to CCR1. The authors discussed that the longer photoperiod is sufficient to overcome the enhanced susceptibility of *Z. mays Hm1*^{A} seedlings conferred by the lack of HCRT (Marla *et al.*, 2018).

For Arabidopsis it has been shown that also a treatment with continuous light decreases the susceptibility to various pathogens, such as *Pst* DC3000 bacteria (Cortleven *et al.*, 2022; Lajeunesse *et al.*, 2023), *Psm avrRpm1* (Griebel & Zeier, 2008), the oomycete *Hyaloperonospora parasitica* Noco2 (*Hpa*) (Evrard *et al.*, 2009) or the hemibiotrophic fungus *Pyricularia oryzae* (syn. *Magnaporthe oryzae*) (Shimizu *et al.*, 2021). A suddenly occurring treatment with continuous light that exceeds the normal photoperiod and is followed by a dark period leads to photoperiod stress (**section 1.2.1**). Analysis of

Arabidopsis showed that photoperiod stress regulates the transcription of many genes known to be responsive to pathogen attack, such as SA and JA biosynthesis/signalling genes. In addition, photoperiod stress increases the levels of several phytohormones coordinating the responses to pathogens, including SA, JA, JA-Ile and camalexin (Cortleven *et al.*, 2022). Especially the length of the light period directly before infection by *Psm avrRpm1* rather than the circadian clock determines the amount of SA that is accumulates in Arabidopsis (Griebel & Zeier, 2008). Under continuous light, potentiation of SA-dependent responses promotes opening of plant stomata, thereby preventing the establishment of aqueous microenvironments in leaves that are beneficial for *Pst* DC3000 bacteria, although ABA biosynthesis and signalling appears unaffected. More specifically, the effects of continuous light rely on SA biosynthesis by SALICYLIC ACID INDUCTION DEFICIENT2 (SID2) and SA signalling by NONEXPRESSOR OF PR1 (NPR1), as stomata are closed and microenvironments developed under continuous light in *sid2-2* and *npr1-1* mutants infected with *Pst* DC3000 (Lajeunesse *et al.*, 2023). The phytohormone abscisic acid (ABA) regulates stomatal closure during pathogen attacks, thereby acting antagonistically to SA. Using their effector proteins HopM1 and AvrE1, *Pst* bacteria stimulate ABA biosynthesis and signalling to induce stomatal closure in infected plant tissues. Effector-induced ABA biosynthesis and signalling is not influenced by continuous light, as respective marker genes (*NCED3* and *RD29A*) are similarly expressed in WT under continuous light, normal day-night-cycles, and extended darkness (Lajeunesse *et al.*, 2023). Similar observations were made by Yang *et al.* (2015) who showed that night-time light treatments, in particular red light, decrease the susceptibility of *Solanum lycopersicum* (tomato cv. Ailsa Craig) to *Pst* DC3000. Tomatoes infected with *Pst* DC3000 accumulated higher levels of free and conjugated SA and activated transcription of defence-related genes, when exposed to red light. Among the genes that are differently expressed in *Pst* DC3000-infected tomato exposed red light are (i) circadian clock genes, such as *CCA1*, (ii) phytohormone biosynthesis and signalling genes, especially SA-related genes, like *PAL* and *PR1*, (iii) genes regulating the plant redox state, such as *RBOH* and *GSTs*, (iv) genes mediating calcium signalling, and (v) transcription factor genes, for instance *WRKYs*. The authors highlighted that NPR1 is required for decreasing bacterial growth by red light treatments in tomato, as silencing of *NPR1* compromises the positive effects of the red light treatment (Yang *et al.*, 2015). Taking the observations for Arabidopsis and tomato plants into account, the current research indicates that SA signalling including NPR1 is relevant for decreasing the susceptibility against bacterial infections by continuous light in different plant species (**Figure 15**).

Besides its effects on the susceptibility of plants to *Pst* DC3000, continuous light also influences responses to fungal and oomycete attack (as mentioned above). More specifically, Evrard *et al.* (2009) characterized the hexameric promoter motif FORC^{A} of Arabidopsis that interacts with nuclear proteins and mediates responses to both light and fungal or oomycete pathogens, including *Hpa*. Interactions of FORC^{A} with its targets are inhibited by defence-related signals, such as SA or *Hpa* recognition; however, these are promoted by continuous light. Thereby, FORC^{A} contributes to finetuning of the transcriptome in response to both light and fungal or oomycete pathogens (Evrard *et al.*, 2009). Shimizu *et al.* (2021) observed that night-time light treatment (leading to continuous light) after inoculation of Arabidopsis with *P. oryzae* decreases the penetration and spore germination rates of the fungus. The authors related the effects of the photoperiod on the susceptibility of Arabidopsis to *P. oryzae* to photoperiod-dependent

changes in JA signalling, ROS, and tryptophan-derived metabolites (Shimizu *et al.*, 2021). In addition to the experiments performed with Arabidopsis, research by Macioszek *et al.* (2021) showed for *Brassica juncea* (brown mustard) that growth under continuous light decreases necrosis development when infected with the fungal pathogen *Alternaria brassicicola*. Altogether, these publications provide evidence that continuous light affects various types of plant-pathogen-interactions.

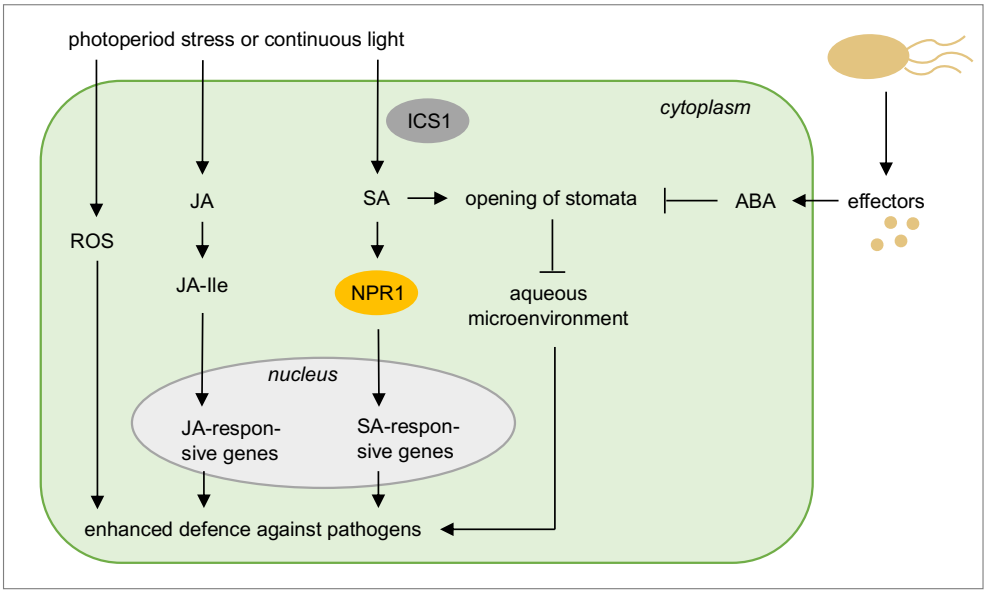

Figure 15. Continuous light enhances resistance against *Pst* bacteria.

Photoperiod stress induces the accumulation of peroxides, and increases the levels of JA, JA-Ile, SA and camalexin, thereby activating responses that resemble those that are effective against pathogen infections. SA accumulated in response to continuous light stimulates opening of plant stomata, thereby counteracting effector-induced ABA biosynthesis/signalling. Figure is inspired by Lajeunesse *et al.* (2023). ROS, reactive oxygen species; JA, jasmonic acid; JA-Ile, jasmonic acid-isoleucine; SA, salicylic acid; ICS1, ISOCHORISMATE SYNTHASE1; NPR1, NONEXPRESSOR OF PATHOGENESIS-RELATED GENES1; ABA, abscisic acid.

1.4.4 Light quality

Light quality, more specifically the spectral composition of light, represents another parameter affecting the susceptibility of plants to pathogens (Roeber *et al.*, 2021).

In particular, phytochrome-mediated signalling is important for plant responses against several pathogens (Genoud *et al.*, 2002; Griebel & Zeier, 2008; Zhao *et al.*, 2014; Yang *et al.*, 2015; Meng *et al.*, 2020; Gallé *et al.*, 2021). As part of the plant innate immunity, stomata restrict bacterial invasion (Underwood *et al.*, 2007). Under red light, stomatal opening is mediated by phyB. Under white light, phyB acts in addition to CRY1/2 and PHOT1/2 to regulate stomatal opening in Arabidopsis (Wang *et al.*, 2010). Downstream of phyB, the light signalling components PIF3/4 and COP1 repress stomatal opening (Mao *et al.*, 2005). PhyA functions partially redundant to phyB in mediating stomatal opening under white light (Wang *et al.*, 2010). Through the regulation of stomata opening by photoreceptors in Arabidopsis, light perception is linked to plant innate immunity.

The effects of phytochromes on plant resistance differ depending on the occurring plant-pathogen-interaction (Roeber *et al.*, 2021). While phytochrome-mediated signalling does not improve the

resistance of Arabidopsis against viral infections (Chandra-Shekara *et al.*, 2006), phytochrome-mediated signalling enhances the resistance against bacteria (Genoud *et al.*, 2002; Griebel & Zeier, 2008; Zhao *et al.*, 2014). More specifically, experiments with *A. thaliana* ecotype Dijon-17 mutated in either *phyA* or *phyB* that are infected by the virus TCV suggested that *HRT*-mediated signalling activating HR in the plants is independent of phytochromes (but dependent on light) (Chandra-Shekara *et al.*, 2006). However, both phyA and phyB are required for SA-mediated induction of *PR1* expression or development of hypersensitive response in Arabidopsis, when infected with *Pst avrRpt2* (Genoud *et al.*, 2002). Additional experiments with Arabidopsis plants that were infected by *Psm* showed that induction of systemic resistance responses (SAR), including accumulation of SA and expression of *PR1* in systemic, uninfected leaves, depends on phyA and phyB and that phytochromes are essential for the expression of *FLAVIN-DEPENDENT MONOOXYGENASE1* (*FMO1*) in systemic tissues (Griebel & Zeier, 2008). Research by Zhao *et al.* (2014) further suggested that red light signalling through phyB enhances the resistance of Arabidopsis against virulent *Pst* DC3000. The authors observed that phyB activates the expression of *LIPOXYGENASE2/3/4* (*LOX2/3/4*) by promoting the degradation of PIF3 under red light. In addition, LIPOXYGENASE enzyme activity is promoted by calcium-regulated MITOGEN-ACTIVATED PROTEIN KINASE3 (MPK3) and MPK6 under red light, when infected with *Pst* DC3000 (Zhao *et al.*, 2014).

Similar positive effects of red light on the susceptibility to pathogens have also been described for other plant species than Arabidopsis, such as tomato (Yang *et al.*, 2015) and strawberries (Meng *et al.*, 2020). As described above (**section 1.4.3**), night-time red light (which extends the normal photoperiod) enhances the resistance of tomato plants against *Pst* DC3000 bacteria, which is associated with an increase in the SA content and induction of defence gene transcription (Yang *et al.*, 2015). Meng *et al.* (2020) observed that growing *Fragaria ananases* (cv. Elsanta referring to strawberry) under red light conditions decreases their susceptibility to infections with *B. cinerea*, while growth under blue light conditions increased their susceptibility. The authors discussed that red light might activate JA-dependent signalling pathways through phyB, thereby counteracting *B. cinerea* infection (Meng *et al.*, 2020).

In contrast to red light that has been shown to decrease susceptibility to different pathogens, far-red (FR) light promotes disease development in different plant species, such as Arabidopsis (Kazan & Manners, 2011; Cerrudo *et al.*, 2012) or tomato (Courbier *et al.*, 2021). Plants typically experience an enrichment of FR light, when they are shaded, for example under high plant densities, where red light is depleted, and the proportion of FR light is increased resulting in low red to far-red light ratios (R:FR < 1) in comparison to natural sunlight (R:FR = 1.2) (Courbier *et al.*, 2021). During an enrichment of FR light, degradation of DELLA proteins promotes gibberellin (GA)-stimulated growth of Arabidopsis and inhibits JA-dependent defence responses (Leone *et al.*, 2014; Courbier *et al.*, 2021; Lazzarin *et al.*, 2021). Changes in the ratios of red to far-red light are perceived by phytochromes, predominantly by phyB that is converted into its inactive Pr form under low R:FR ratios (Courbier *et al.*, 2021). Inactivation of phyB (by low R:FR ratios or mutation) increases the susceptibility of Arabidopsis to the fungi *B. cinerea* (Cerrudo *et al.*, 2012; Fernandez-Milmanda *et al.*, 2020) or *Fusarium oxysporum* (Kazan & Manners, 2011), and to *Pst* DC3000 bacteria (De Wit *et al.*, 2013). The higher susceptibility of Arabidopsis to *B. cinerea* in response to FR-mediated inactivation of phyB relies on JA-dependent but

SA-independent signalling (Cerrudo *et al.*, 2012). More specifically, inactivation of phyB by FR light represses the expression of defence genes, such as the JA-responsive *PLANT DEFENSIN1.2* (*PDF1.2*) or the ethylene-/JA-regulated *ETHYLENE RESPONSE FACTOR1* (*ERF1*), in Arabidopsis infected with *B. cinerea*. The effects of FR light on Arabidopsis plant resistance against *B. cinerea* depend on JA perception by the SCF^{COI1}-JAZ complex (Cerrudo *et al.*, 2012). The stability of the JA signalling repressor protein JASMONATE ZIM DOMAIN PROTEIN10 (JAZ10) increases in response to inactivation of phyB (under low R:FR or through respective mutation), thereby JAZ10 attenuates JA-dependent signalling (Ballaré, 2014; Leone *et al.*, 2014). Recent research by Fernandez-Milmanda *et al.* (2020) showed that the sulfotransferase ST2a decreases the pool of bioactive JAs available in Arabidopsis in response to low R:FR ratios, thereby influencing JA-dependent plant defence responses. The effect of STa2 on bioactive JAs is caused by a phyB-PIF-mediated increase in *ST2a* transcripts under low R:FR ratios (Fernandez-Milmanda *et al.*, 2020). In addition to this direct effect on the pool of JAs, inactivation of phyB (by FR light or mutation) decreases the sensitivity to JA (Moreno *et al.*, 2009). In tomato plants, inactivation of phyB also enhances the plants' susceptibility to *B. cinerea*. The disease development in this plant-pathogen-interaction is associated to the soluble sugar content in tomato that is modulated by JA-dependent signalling. Inactivation of phyB increases the content of soluble sugars (glucose, fructose) in tomato plants, which is associated with a lowered JA signalling under low R:FR light (Courbier *et al.*, 2021).

Furthermore, defence responses mediated by SA, such as transcription of SA-dependent genes, are also inhibited by low R:FR ratios (De Wit *et al.*, 2013; Campos *et al.*, 2016; Iqbal *et al.*, 2021). The effects of low R:FR ratios on transcription of SA-dependent genes result from inhibition of SA-inducible kinases. Monomerization of the transcription coregulator NPR1 increases under low R:FR ratios, however, phosphorylation of NPR1 is not proportionally enhanced under this condition. Thereby, target gene transcription is prevented and susceptibility to pathogens, such as *Pst* DC3000, is enhanced (De Wit *et al.*, 2013; Roeber *et al.*, 2021).

In addition to phytochromes, also cryptochromes and phototropins influence plant resistance against pathogens (Wu & Yang, 2010; Jeong *et al.*, 2010); however, the distinct functions of blue light photoreceptors in plant resistance vary depending on the respective plant-pathogen-interaction. In Arabidopsis, CRY2 and PHOT2 stabilize HRT, thereby decreasing the susceptibility to TCV (Jeong *et al.*, 2010). Moreover, *CRY1* overexpression improves the local resistance of Arabidopsis against avirulent *Pst avrRpt2*, when the plants are exposed to continuous white or blue light after infection. The enhanced resistance in Arabidopsis overexpressing *CRY1* is associated with a stronger transcription of *PR1* and a slightly higher SA content in response to infection with *Pst avrRpt2*. However, overexpression of *CRY1* does not limit growth of *Pst* DC3000 under continuous white light. The experiments thus suggest that CRY1 is particularly important for resistance mediated by plant R proteins rather than basal resistance. In addition to the effects of CRY1 on local resistance, overexpression of *CRY1* decreases the susceptibility to virulent *Pst* DC3000 in distal plant leaves after primary infection with avirulent *Pst avrRpt2* (Wu & Yang, 2010). In contrast, induction of SAR by primary infection with virulent *Psm* is independent of blue light photoreceptors, including CRY1/2 and PHOT1/2, in Arabidopsis (Griebel & Zeier, 2008; Delprato *et al.*, 2015).

Interestingly, also ultraviolet (UV) irradiation influences the susceptibility of plants to pathogens (Demkura & Ballaré, 2012; Escobar-Bravo *et al.*, 2017). Notably, already relatively low doses of UV-B irradiation induce plant resistance responses that are effective against pathogens. In Arabidopsis, low doses of UV-B irradiation that cause no visible damage to leaves increase the resistance against *B. cinerea*. The positive effects of UV-B irradiation on resistance against this fungus does not rely on JA signalling. Instead, UV-B irradiation increases the production of sinapates (sinapoyl malate and sinapoyl glucose), which relies on rely on the UVR8 photoreceptor, thereby increasing the plants' resistance (Demkura & Ballaré, 2012). In addition to UV-B, also UV-C irradiation affects resistance against pathogens (Kunz *et al.*, 2008). In Arabidopsis, UV-C irradiation induces the accumulation of SA and expression of *EDS5* (Nawrath *et al.*, 2002). Kunz *et al.* (2008) further investigated the effects of UV-C radiation on plant resistance against the oomycete *Hyaloperonospora parasitica* and showed that UV-C irradiation induces DNA damage that promotes resistance without the necessity to recognize the pathogen.

1.5 Effects of stress combinations on plants

In nature, plants are typically exposed to combinations of various stimuli (Suzuki *et al.*, 2014). The experience of a previous condition can influence the plants' response to subsequent stimuli (**section 1.5.1, section 1.5.2**) (Hönig *et al.*, 2023).

1.5.1 Induced resistance

Certain stimuli, such as pathogens, herbivores, beneficial microbes, or chemicals, can further enhance basal resistance of plants that is established by PTI and ETI (De Kesel *et al.*, 2021; Hönig *et al.*, 2023). Such an induced resistance (IR) provides plants with a higher capacity to defend against a broad range of pathogens and in some cases to cope with abiotic stresses. Depending on the spatial effects in plants, IR phenotypes result from local and/or systemic resistance establishment (**Figure 16**). The term induced systemic resistance (ISR) refers to the latter form of IR phenotypes, which improve the resistance in the whole plant after occurrence of a local IR stimulus (Hake & Romeis, 2019; De Kesel *et al.*, 2021). Systemic acquired resistance (SAR) represents a well-described form of ISR. Depending on the underlying defence responses, IR phenotypes result from direct and/or primed (induced) plant responses (**section 1.5.2**). The final IR phenotypes are the outcome of combinations of local and/or systemic as well as direct and/or primed responses (De Kesel *et al.*, 2021; Hönig *et al.*, 2023).

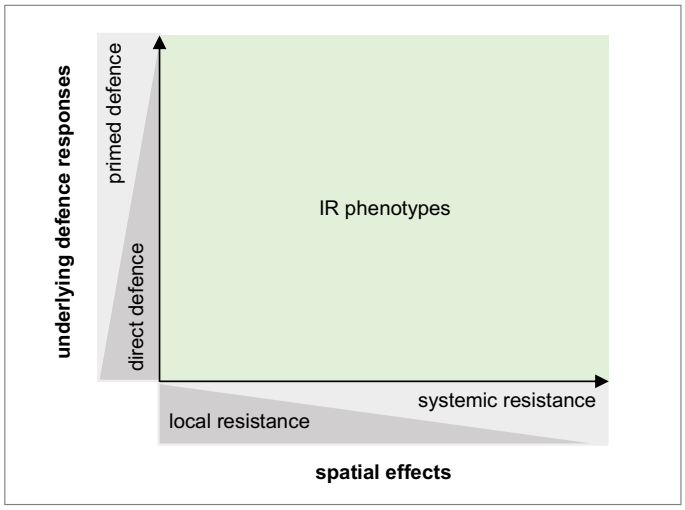

Figure 16. Induced resistance phenotypes.

IR phenotypes depend on spatial effects (local or systemic resistance induction) and the underlying defence responses (direct or induced/primed plant responses). The proportions of these parameters are affected by the IR stimulus, its timepoint of occurrence, and intensity, and the plant itself including its species, developmental stage, and respective tissue. Figure is inspired by De Kesel *et al.* (2021). IR, Induced resistance.

1.5.2 Priming of plant stress responses

Although plants lack a classical nervous system, they can adjust their responses to previous environmental conditions indicating a “memory”. The memory, which can be preserved over a stress-free period (lag phase, L), allows plants to prime (induce, P) their responses to future, potentially more severe environmental challenges (trigger, T) (Hilker *et al.*, 2016; Hilker & Schmülling, 2019). Primed plant responses are characterized by a potentiation leading to earlier, faster, more sensitive and/or stronger responses when the plants are exposed to a second stimulus (De Kesel *et al.*, 2021; Hönig *et al.*, 2023). Various levels of responses can be affected by an initial stimulus, thereby improving plant performance (Hilker & Schmülling, 2019). Among the plant responses that can be indicative for an establishment of a stress memory are transcriptional regulations (**Figure 17**), posttranscriptional and -translational modifications, epigenetic regulations and chromatin modifications, activities of enzymes as well as levels and locations of proteins, phytohormones or metabolites (Hilker & Schmülling, 2019; Liu *et al.*, 2022).

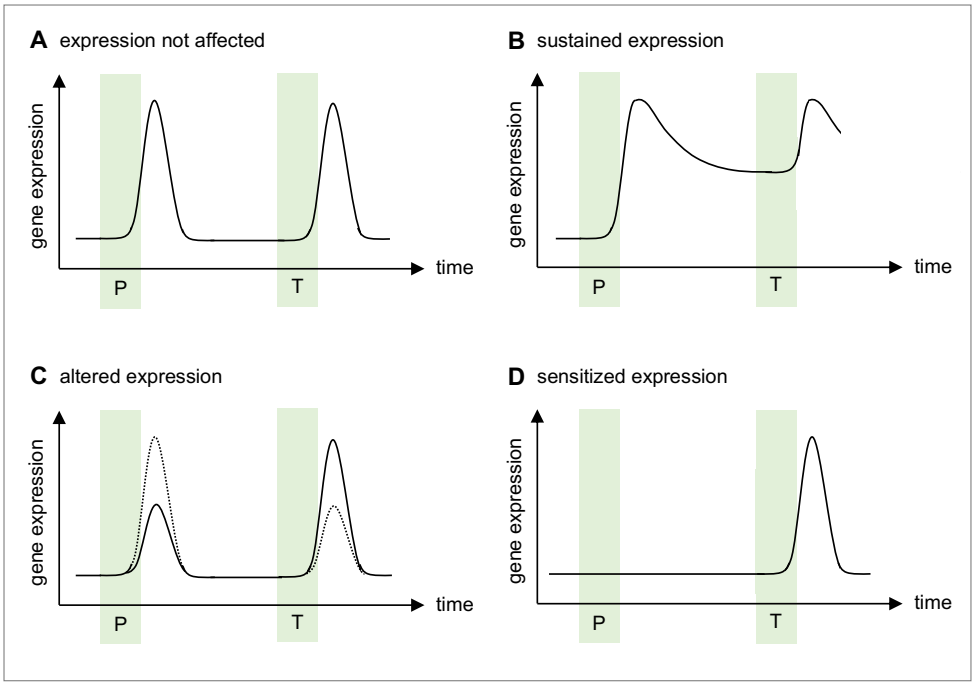

Figure 17. Expression of plant stress memory genes.

Exposure of plants to a first stimulus, priming, may affect the expression of genes in response to a second stimulus, triggering, indicating priming and memory. **(A)** Expression pattern of non-memory genes: Induction in response to priming and triggering stimuli is similar. **(B-C)** Expression patterns of memory genes: **(B)** Expression of genes is induced by a priming stimulus and remains at a higher level during the entire memory phase, thereby affecting the expression of certain genes when the plants are exposed to a second stimulus. **(C)** Expression of genes is affected by a priming stimulus, returns to non-induced expression during the memory phase and is stronger/weaker induced when plants are exposed to a second stimulus. **(D)** A few memory genes are not responsive to the priming but become sensitized to other stimuli, thus expression occurs only in response to a second stimulus. Figure is inspired by Friedrich *et al.* (2019) and Bäurle and Trindade (2020). P, Priming; T, Triggering.

The memory depends on the plant species or genotype, its developmental stage, or its nutritional condition, as well as the initial priming stimulus, its intensity or duration, and its timepoint of occurrence (Hilker *et al.*, 2016; Liu *et al.*, 2022). Several abiotic and biotic (temporally limited) environmental stimuli but also chemicals have been shown to prime plants, thereby preventing a delay in the induction of responses during acute stress and thus improving the plants' fitness with lower investments of their resources (Hilker *et al.*, 2016; Hilker & Schmülling, 2019; Hönig *et al.*, 2023). In the following thesis, the term *cis*-priming describes that a certain primary stimulus intensifies the responses of a plant against a similar kind of secondary stimulus. The term *trans*-priming indicates that a primary stimulus boosts plant responses against a different kind of secondary stimulus (Hilker & Schmülling, 2019).

In the two following sections (**section 1.5.2.1 and 1.5.2.2**), currently identified mechanisms involved in priming and memory of Arabidopsis and other plant species are presented.

1.5.2.1 Priming by abiotic stimuli

Among the abiotic stimuli that prime plants are alterations in temperature (Hilker *et al.*, 2016; Olas *et al.*, 2021), drought stress (Ding *et al.*, 2012; 2013), flooding stress (Liu *et al.*, 2022), mild salt stress (Sani

et al., 2013), and photoperiod stress (Cortleven *et al.*, 2022). The effects of photoperiod stress on plant responses to biotic stress are considered in **section 1.4.3**.

One well-described example for the long-lasting memory of cold periods is the vernalization response that enables Arabidopsis to adjust the timepoint of its flowering to its environment. In response to longer cold periods (but not to shorter cold periods), the floral repressor gene *FLOWERING LOCUS C* (*FLC*) is epigenetically silenced (Hepworth & Dean, 2015; Friedrich *et al.*, 2019; Hilker & Schmülling, 2019). The repression of *FLC* during longer cold periods is mediated by VERNALIZATION3 (VIN3) (Kim *et al.*, 2010; Hepworth & Dean, 2015; Friedrich *et al.*, 2019). Besides, a memory of low but non-freezing temperatures (< 10°C) allows Arabidopsis to improve its performance under subsequent freezing temperatures (below 0°C) without the necessity to constantly keep the plants in a cold-acclimated condition (Thomashow, 1999; Friedrich *et al.*, 2019; Leuendorf *et al.*, 2020). Zuther *et al.* (2019) observed that exposure to low temperature (4°C for 3 days) enhances the freezing tolerance of rosette leaves of 28-day-old soil-grown Arabidopsis to cold periods (4°C for 3 days) following a recovery phase at normal temperatures (lag phase, 22°C, 7 days). Cold priming affects the transcriptome as well as the lipid content and primary metabolite composition (Zuther *et al.*, 2019). Experiments by van Buer *et al.* (2016) indicated that cold priming (4°C for 1 day) is regulated by thylakoid-bound ascorbate peroxidases (tAPX) that modulates chloroplast-to-nucleus ROS signalling. Interestingly, Leuendorf *et al.* (2020) showed that even night-time exposure to low temperature (4°C for 16 hours) is sufficient to improve the freezing tolerance in 14- and 21-day-old Arabidopsis seedlings, however, not in 7-day-old seedlings, thereby indicating that this priming effect depends on their age. In addition, freezing experiments showed that the positive effects of a cold priming (4°C for 3 days) on the plants' resistance in response to a second cold stimulus are memorized (by 21-day-old Arabidopsis) for 3 days but are lost after a recovery phase (22°C) of five days, indicating that low temperatures primes Arabidopsis seedlings only transiently due to a shorter memory than soil-grown plants (Zuther *et al.*, 2019; Leuendorf *et al.*, 2020).

In addition to low temperature, also moderately high temperature has been shown to prime plants allowing their survival at a subsequent more severe temperatures (Bäurle & Trindade, 2020; Olas *et al.*, 2021; Balazadeh, 2022). In contrast to memory of cold conditions, memory of a previous heat stimulus (37°C for 1 hour) after a recovery phase (23°C for 1.5 hours) requires FORGETTER1 (FGT1) in 4-day-old Arabidopsis seedlings triggered by severe heat (44°C for 45 minutes). FGT1 interacts with protein complexes that remodel plant chromatin and contributes to the depletion of nucleosomes, thereby facilitating the transcription of memory genes after heat stress (Brzezinka *et al.*, 2016; Friedrich *et al.*, 2019). Among the genes that are more strongly induced in Arabidopsis seedlings memorizing a previous heat stimulus are *HEAT SHOCK PROTEINs* (for example *HSP22*) and *ASCORBATE PEROXIDASE2* (*APX2*) (Lämke & Bäurle, 2017). The enhanced induction of these genes in response to reoccurring heat stimuli is dependent on HEAT SHOCK FACTOR A2 (HSFA2) that associates with memory-related genes after heat conditions and promotes histone methylation (H3K4me2, H3K4me3) in response to heat (Charng *et al.*, 2007; Lämke & Bäurle, 2017; Charng *et al.*, 2023). HSFA2 is also required for transcription activation of memory genes in response to heat in the shoot apical meristem of Arabidopsis (Olas *et al.*, 2021).

In addition to abiotic stimuli related to temperature also water-related stimuli, such as flooding and drought, can prime plants for future conditions. Memory of both flooding and drought is associated to changes in the antioxidant system of plants (**section 1.3.2**) and their phytohormone signalling (**section 1.3.1**) (Hartman *et al.*, 2019; Keska *et al.*, 2021; Liu *et al.*, 2022).

Priming by dehydration (air-drying for 2 hours) prevents water loss when Arabidopsis seedlings are exposed to a second dehydration event after a recovery phase of watering (rehydration for 22 hours). Two types of genes, non-trainable and trainable genes, were identified based on their expression patterns in response to a single and multiple dehydration events. Non-trainable genes are similarly expressed in response to a single and multiple dehydration stimuli, while trainable genes are more strongly induced when exposed to multiple dehydration events compared to a single dehydration stimulus, although their expression returns to basal levels during the recovery phases which is comparable to those in unstressed plants. The expression pattern of trainable genes is characteristic for transcriptional memory (Ding *et al.*, 2012; 2013).

1.5.2.2 Priming by biotic stimuli

In addition to abiotic stimuli, also biotic stimuli prime plants for future challenges (Hake & Romeis, 2019). One well-described example for priming by biotic stimuli is the deposition of herbivorous insect eggs that improves the performance of Arabidopsis and other members of the *Brassicaceae* family against subsequent feeding by large white butterfly, *Pieris brassicae*, larvae (Lortzing *et al.*, 2019; Valsamakis *et al.*, 2020; 2022). Interestingly, the length of the deposition of *P. brassicae* eggs determines how Arabidopsis plants perform against larvae. More specifically, herbivorous insect eggs that remained longer on plant tissue (5 days) affected the larval feeding stronger than shorter egg deposition periods (1 to 4 days). The authors identified three patterns of plant responses induced by egg priming: (i) The first pattern includes responses that are directly induced one day after egg deposition and maintained at a high level until hatching of larvae, such as SA and JA levels. Larval feeding further improved expression of SA-related (but not JA-related) genes in egg-primed Arabidopsis. (ii) The second pattern refers to late induced responses that increase five to six days after egg deposition, such as JA-Ile and camalexin levels. Experiments with the *jar1-1* mutant that is impaired in the conjugation of JA to JA-Ile indicate that JA-Ile is essential for the effects of egg priming against larvae feeding, as the biomass of larvae feeding on *jar1-1* mutants is similar on egg-primed and non-primed mutants. (iii) The third pattern includes responses that are gradually increased over time after egg deposition, such as expression of plant defence genes, including *PAD3*, *CAX3*, *PDF1.4*, SA-dependent *PR1*, *PR5*. The effects of egg priming on larval feeding depend on SA biosynthesis and signalling, as the biomass of larvae feeding on *sid2* and *pad4* mutants is similar on egg-primed and non-primed mutants. However, priming by eggs improving the defence of Arabidopsis against *P. brassicae* larvae is independent of NPR1 or WRKY70. Experiments with the *pad3* mutant that is impaired in camalexin biosynthesis suggest that the increase in camalexin content in response to egg priming is not responsible for the negative effects on the larvae (Lortzing *et al.*, 2019; Valsamakis *et al.*, 2020; 2022).

Similar as cold priming in Arabidopsis (**section 1.5.2.1**), the effects of egg priming depend on the developmental stage but not the chronological age of the plants: During their vegetative phase, deposition of *P. brassicae* eggs primes Arabidopsis against larval feeding, however, not during their reproductive phase. Interestingly, the transcriptional status that Arabidopsis acquire in response to the deposition of *P. brassicae* eggs during the vegetative phase resembles the transcriptome that is detected in plants during their reproductive phase without any stimulus. This could be because the Arabidopsis transcriptome is reprogrammed during plant development from a state that is capable of being primed by insect eggs to a non-primable state that is characterized by a higher general resistance (Valsamakis *et al.*, 2022). In addition to members of the *Brassicaceae* family, insect egg deposition also improves the resistance of perennial plants, for example the elm tree *Ulmus minor*, against subsequently occurring larval feeding (Austel *et al.*, 2016; Schott *et al.*, 2021); Schott *et al.* (2023). A comparison of plant species with different lifestyles revealed conserved transcriptomic changes to insect eggs and feeding (Lortzing *et al.*, 2020). More specifically, the comparison of Arabidopsis exposed to *P. brassicae* eggs (Lortzing *et al.*, 2019), bittersweet nightshade (*Solanum dulcamara*) treated with *Spotoptera exigua* eggs, wild tobacco (*Nicotiana attenuate*) exposed to eggs of *Manduca sexta* or *Spotoptera exigua*, and elm tree *Ulmus minor* exposed to elm leaf beetle *X. luteola* eggs have at least one-fifth (22%) of the biological processes that are transcriptionally regulated in common (Lortzing *et al.*, 2020).

Another well-described example for priming by biotic stimuli represent beneficial microbes that can trigger induced systemic resistance (ISR, **section 1.5.1**) when interacting with plants, thereby improving the plants' resistance to subsequently occurring stimuli (Pieterse *et al.*, 2014; Hilker & Schmülling, 2019; Hönig *et al.*, 2023). Beneficial microbes, such as plant growth-promoting rhizobacteria (PGPR) or fungi (PGPF), present in the rhizosphere, can improve the plants' resistance against necrotrophic and (hemi) biotrophic pathogens or herbivores (Conrath *et al.*, 2002; Pieterse *et al.*, 2014). Priming of ISR by beneficial PGPR/F relies on plant phytohormone signalling, especially JA and ET defence signalling, but also JA and SA synergisms are involved (Pieterse *et al.*, 2014; Vlot *et al.*, 2021); however, the specific responses depend on the plant species, the respective beneficial microbes as well as the biotic stressors (Vlot *et al.*, 2021; Hönig *et al.*, 2023). In Arabidopsis, for example, colonization of the rhizosphere by PGPR triggers JA- and ET-dependent ISR, thereby improving the resistance against different pathogens (Pieterse *et al.*, 1996; 2014).

1.6 Research aims

Light represents an important environmental stimulus for plants determining numerous of their life processes (**section 1.1, 1.2.1, 1.3**) (Paik & Huq, 2019). Although research elucidating light perception and signalling in plants is steadily progressing (**section 1.1**), the knowledge about the effects of a suddenly occurring prolongation of the light period leading to photoperiod stress is currently limited (**section 1.2.1**). Therefore, one research aim of this thesis is to further characterize photoperiod stress in Arabidopsis (**section 3 and 4**). As part of this characterisation, different prolongations of the light period (1 h, 2.5 h, 4 h) are analysed concerning their effects on plant gene expression (using RNA-sequencing) to enhance our understanding of conditions that induce photoperiod stress (**section 3.1 and 4.1**). The main research aim of this work is to investigate photoperiod stress-mediated priming and

memory in Arabidopsis (**section 3.2, 3.3, 4.2 and 4.3**). To this end, conditions that cause photoperiod stress in Arabidopsis (like 4 h and 8 h prolonged light periods) are used to study priming and memory caused by this stress (**section 3.2, 3.3, 4.2 and 4.3**). The present work aims to increase our knowledge of the mechanisms underlying photoperiod stress-induced priming and memory. To this end, the roles of parameters known to be affected by a single photoperiod stress event, such as expression of photoperiod stress-responsive genes, ROS content, and phytohormone levels, are examined in memory experiments. In the frame of this thesis, two setups of priming and memory experiments are used: *cis*-priming and *trans*-priming experiments. In *cis*-priming experiments, the effects of a single photoperiod stress event on Arabidopsis plant responses that are treated with a second similar photoperiod stress are investigated (**section 3.2 and 4.2**). In *trans*-priming experiments, the influence of photoperiod stress on plant responses against pathogen attack (*P. syringae* pv. *tomato* DC3000; *B. cinerea*) is explored (**section 3.3 and 4.3**). The experiments provide evidence that photoperiod stress transfers Arabidopsis plants into an alarm condition that enhances plant resistance against several stresses (**section 5**).

2 Materials and methods

2.1 Plant material and growth conditions

Arabidopsis thaliana accession Columbia-0 (Col-0) was used as wild type (WT). The mutant and transgenic Arabidopsis plants used in this work are listed in **Table 1**. Seeds were obtained from the European Arabidopsis Stock Centre (NASC) or from colleagues as indicated.

Table 1. Mutant and transgenic Arabidopsis plants.

Name	Source	Additional information	Reference
acbp3	NASC	SALK_012290C	(Alonso *et al.*, 2003)
ahk2-5 ahk3-7	Obtained from Anne Cortleven (Berlin)	-	(Riefler *et al.*, 2006)
ald1	Obtained from Vivien Lortzing (Berlin)	SALK_007673	(Mishina & Zeier, 2006)
arr2-1	Obtained from Sören Werner (Berlin)	GK269G01	(Nitschke, 2014; Frank, 2019)
aos1	Obtained from Ivo Feussner (Göttingen)	SALK_017756	(Alonso *et al.*, 2003)
cca1 lhy	Obtained from Anne Cortleven (Berlin)	-	(Nitschke, 2014; Nitschke *et al.*, 2016)
dde2-2	Obtained from Anne Cortleven (Berlin)	-	(Von Malek *et al.*, 2002)
fitness-1	NASC	SALK_140249C	(Osella *et al.*, 2018)
fmo1-1	Obtained from Vivien Lortzing (Berlin)	SALK_026163	(Bartsch *et al.*, 2006)
jar1-1	Obtained from Anne Cortleven (Berlin)	EMS-mutant N8072	(Staswick *et al.*, 2002)
lox3	Obtained from Ivo Feussner (Göttingen)	SALK_062064	(Alonso *et al.*, 2003; Ding *et al.*, 2016)
lox4	Obtained from Ivo Feussner (Göttingen)	SALK_071732	(Alonso *et al.*, 2003)
myc2 myc3 myc4	Obtained from from Alain Goossens (Ghent)	*myc2* SALK-061267C, *myc3* GABI_445B11, *myc4* GABI_491E10	(Fernandez-Calvo *et al.*, 2011)
npr1-1	Obtained from Vivien Lortzing (Berlin)	EMS-mutant N3726	(Cao *et al.*, 1997)
npr1-2	Obtained from Xinnian Dong and Mindy Sponsel (USA)	EMS-mutant N3801	(Cao *et al.*, 1997)
ora59	Obtained from Anne Cortleven (Berlin), NASC	GABI_061A12 (N405772)	(Van der Does *et al.*, 2013)
pad3	Obtained from Erich Glawischnig (München), NASC	SALK_026585C	(Schuhegger *et al.*, 2006)
pad4	Obtained from Vivien Lortzing (Berlin)	SALK_089936	(Jirage *et al.*, 1999)
sid2/ics1	Obtained from Vivien Lortzing (Berlin)	SALK_088254	(Glazebrook *et al.*, 1996)
snrk2.8-1	Obtained from Christiane Gatz (Berlin)	SALK_073395	(Olate *et al.*, 2018)
tga1 tga3 tga7	Generated by Jan Erik Leuendorf (Berlin)	*tga1* SALK_028212, *tga3* SALK_086928, *tga7* GABI_434F04	(Kesarwani *et al.*, 2007; Choi *et al.*, 2010)
tga2 tga5 tga6	Obtained from Xinnian Dong (USA)	CS72346	(Kesarwani *et al.*, 2007)
tga3	Obtained from Jan Erik Leuendorf (Berlin)	SALK_086928, knock-down	(Choi *et al.*, 2010)
trxh3	NASC	SALK_111160C	(Alonso *et al.*, 2003)
trxh5	Obtained from Christiane Gatz (Berlin)	SALK_144259	(Olate *et al.*, 2018)

Seeds were sown on PT soil consisting of soil type T and type P (Einheitserde Classic Profisubstrat, Einheitserdewerke Werkverband e.V., Sinntal-Altengronau, Germany), and sand in a 2:2:1 ratio. After sowing, seeds were stratified for 2 days at 4°C. For photoperiod stress experiments, plants were grown on “sowing soil” for four weeks under short day (SD) condition (8 h light/16 h dark cycles) in Arabidopsis cultivation shelves (Photon Systems Instruments, Drasov, Czech Republic) with light-emitting diode (LED) lightning (cool white LEDs added with far-red LEDs, 100 to 150 µmol m^{-2} s^{-1}) at 22°C and 60% relative humidity. For genotyping and propagation or crossings, plants were grown under long day (LD) condition (16 h light/8 h dark cycles) in the greenhouse at 22°C during light and 18°C during dark periods. Greenhouse-grown plants were sown on PT soil and transferred to single pots for separation containing PT soil with Perligran G instead of sand.

2.2 Photoperiod stress treatments of Arabidopsis plants

Different photoperiod stress treatments were used in this thesis. For sensitivity experiments, plants were exposed to a prolonged light (PL) period of 1 h, 2.5 h, 4 h or 8 h, in addition to the normal 8 h SD light period (**Figure 18**). The PL treatment was followed by a dark period, which was shortened to 15 h, 13.5 h, 12 h or 8 h darkness, respectively, to maintain a 24 h day/night cycle. For priming experiments, plants were subjected to a PL period of 0.5 h, 1 h, 2 h, 4 h or 8 h, representing the first stress event (priming stimulus, P), which was followed by a second stress event (triggering stimulus, T). The triggering occurred either on the day directly following the priming stimulus (PL0T) or after a lag phase ranging from one to ten days (PL1T to PL10T), in which the plants received normal SD conditions. In *cis*-priming experiments, the plants were exposed to a second prolonged light treatment as the triggering stimulus. In *trans*-priming experiments, plants were infiltrated with *P. syringae* pv. *tomato* DC3000 or infected with fungal spores of *B. cinerea* (during the light period) as the triggering stimulus. Control plants remained under SD conditions during the whole experiments.

For sensitivity and *cis*-priming experiments, harvests were performed during the dark period in green light (except otherwise indicated). For plants exposed to PL periods at 14 days after germination (DAG), whole rosettes were sampled. For plants treated with PL periods at 21 DAG and 28 DAG, whole leaves were sampled, while for plants at 35 DAG or 42 DAG only the tips of the leaves were used.

2.3 Genotypic analysis of Arabidopsis plants

Arabidopsis plants used in this work were genotypically analysed. This included extraction of genomic DNA, which was followed by polymerase chain reaction and agarose gel electrophoresis. A combination of gene-specific and T-DNA insertion-specific primers were used in PCR reactions to identify T-DNA insertion mutants. The gene- and T-DNA insertion-specific primers used for genotyping are listed in **Table 2** and **Table 3**, respectively. Primer sequences in **Table 3** were derived from information provided by the Salk Institute Genomic Analysis Laboratory (SIGnAL, http://signal.salk.edu/tdnaprimers.2.html). Mutants with a point mutation were genotyped using cleaved amplified polymorphic sequences (CAPS) markers. To this end, genomic DNA of respective mutants was amplified by PCR and the PCR products were subsequently digested with specific restriction enzymes that cut at the position of the polymorphism. Primer sequences for this analysis are listed in **Table 4**. The *npr1-1* mutant contains a point mutation, which is likely to destabilize the entire protein and lower *PR1* expression (Cao *et al.*, 1997).

Table 2. Gene-specific primers used for genotyping.

PS, product size; AT, annealing temperature; FP, forward primer; RP, reverse primer. T-DNA insertion-specific primers are listed in **Table 3**.

Mutant	Forward primer sequence (FP)	Reverse primer sequence (RP)	PS for WT [bp]	Primer combination for mutant, [bp]	AT [°C]
acbp3	GTGGTTGCGTAGAAAACGAAG	GATTCACCTCTCCCCTGAAAC	1125	R + SALK, 552 - 852	60
ahk2-5	GCAAGAGGCTTTAGCTCCAA	TTGCCCGTAAGATGTTTTCA	672	F + SAIL, 650	59
ahk3-7	CCTTGTTGCCTCTCGAACTC	CGCAAGCTATGGAGAAGAGG	558	R + GABI, 450	59
ald1	TTACGATGCATTTGCTATGACC	TTTTAAATGGAACGCAAGGAG	1164	R + SALK, 553 - 853	60

Mutant	Forward primer sequence (FP)	Reverse primer sequence (RP)	PS for WT [bp]	Primer combination for mutant, [bp]	AT [°C]
arr2-1	ATGGTAAATCCGGGTCAC	ACATTCCACTCGTTACGC	1017	R + GABI, ~ 1110	58 - 60
aos1	TTTTCTCAATCGCTCCCATC	ATGCCGTCAACGGAACTAAC	688	R + SALK, ~ 600	59
cca1-1	TGTCCAGATAAGAAGTCACGCTCAGAAA	TTTATTCATGGAGGATGCAGCAGAGA	914	F + F_T-DNA, ~ 250	65
fitness-1	TGAAAGGATATGGAATCCGTG	GTTTTGGGAGAATAAGCCGAC	1013	R + SALK, 491 - 791	60
fmo1-1	CTTTTCGGTTGGACTTGGAAC	CTGCTTTGGACGTATCCTACG	1039	R + SALK, 485 - 785	60
lox3	AACGAAGTTGCCGAAGAAAA	TCACTCCACTTCCATCTCCTC	572	F + SALK, ~ 500	59
lox4	AAAGCGGCAGTTTTGAAAGA	TCATATCGGTGTCGGTTGAA	684	F + SALK, ~ 650	59
lhy-20	GAGAGCGATGGACTGAGGA	TTTTCGGGGTAGAGATGATAGAG	795	R + SALK, ~ 500	55
myc2	GACGGATACGGAATGGTTTT	GTTTGCTGGCTTTCTTCCTC	853	R + SALK, ~ 550	60
myc3	TGAAGCAGAGAGGCAGAGAAG	CCACTTATTTTCCTCAGCTTTTTG	953	F + GABI, 250; R + GABI, 800	60
myc4	CTCCTTGACAAATTTGATCCG	CGCTACACACACCATTGTTTG	995	R + GABI, 600	60
ora59	CAATCATTTGACCAATCCTTCC	TCTTGCGTCATAACAACACTCTG	551	F + GABI, ~ 350	59
pad3	TCGGTCAGTGAAGTCTACATGC	CCGGGAAAGAAATCAGAGAAC	1119	R + SALK, 435 - 735	60
pad4	TCATTCCGCGTCTTTTGTATC	TCGCCTCCCACACACTATAAC	1129	R+ SALK, 585 - 885	60
sid2/ics1	AGTGACTGTATTTGATCGCCG	TTTACGAATTTCTGCAATGGC	1086	R + SALK, 449 - 749	60
snrk2.8-1	ATTTTCCAAAGAGCTTTTCGC	GGTGATAGGTTTCCGAGCTTC	1244	R + SALK, 596 - 896	60
tga1-1	TCTTCGAAGAATTTGGCGAAGA	TTCCTGCTGTTCCATGGGAAGTAT	-	F + SALK_b1	58-60
tga2-1	ATCAAGCCCTTTACTTGTGCACCTTCAAG	CGGATGAACGAAATCCACCGA	-	F + JL-202 (*TGA5* is directly linked to *TGA2*)	58-60
tga3kd	CCACTCTTGTCCCACAAAATG	TCCATATCTCTAAAATTGCATTGC	1114	R + SALK, 487-787	60
tga6-1	GGACTGATGTCTCAACTGATGGTGACACAG	GACTATTCTCCAGCTGCTGAACA	-	R + JL-202	58-60
tga7-1	GTCCCAATACTGCTACTTCCTC	CTTGTAGCCAGTGTGAAT	-	No band in mutant because of deletion	58-60
trxh3	GCTGCGAGTAATCAAGTTTGC	ACCGACACAGAGACGAAGAAG	1161	R + SALK, 476 - 776	59
trxh5	CTCGAATCATCTCTCTGTGCC	TTCTCTTGTTATGTCCAGGGC	1137	R + SALK, 484 - 784	59 - 60

Table 3. T-DNA insertion-specific primers for genotyping.

Primer name	Primer sequence	Purpose of usage
SALK_LBb1.3_BP (SALK)	ATTTTGCCGATTTCGGAAC	Genotyping of SALK lines
SALK_LBb1 (SALK_b1)	GCGTGGACCGCTTGCTGCAACT	Genotyping of SALK lines
Feldmann-T-DNA-LB (F_T-DNA)	GATGCACTCGAAATCAGCCAATTTTAGAC	Genotyping of *cca1-1* (derived from the Feldmann T-DNA collection), (Krysan *et al.*, 1999)
JL-202	CATTTTATAATAACGCTGCGGACATCTAC	Genotyping of TGA lines, (Kesarwani *et al.*, 2007)
SAIL_IT1_F (SAIL)	GCCTTTTCAGAAATGGATAAATAGCCTTGCTTCC	Genotyping of SAIL lines
GABI_Kat (GABI)	CCCATTTGGACGTGTAGACAC	Genotyping of GABI-Kat lines

Table 4. Primers for genotyping with CAPS markers.

RE, restriction enzyme. PS, product sizes after digestion with restriction enzyme.

Primer name	Forward primer sequence (FP)	Reverse primer sequence (RP)	RE	Wild-type PS [bp]	Mutant PS [bp]	Mutation	Reference
dde2-2	CATACCGGGAAACTACGGTTTACC	GCTTGAAACTCAGGGAAGATCCGG	*Bst*UI	376	196, 210	Deletion results in frameshift leading to a stop codon	(Von Malek *et al.*, 2002)
jar1-1	CAATGGAAACGCTACTGACC	CGGGACTACAGGAAGGAGAC	*Hpy*188III	221, 411	632	Ser112Phe (TCT → TTT)	(Nitschke, 2014)
npr1-1	TGCGTGTGCTCTTCATTTC	ATCGTTTCCCGAGTTCCA	*Cvi*AII	115, 98, 200, 377	213, 200, 377	His334Tyr (CAT → TAT)	(Nitschke, 2014)
npr1-2	CCTGATGTATCTGCTCT	GCTTAATGCAGATGGTG	*Fsp*I	330, 134	464	Cys150Tyr (TGC → TAC)	(Nitschke, 2014)

2.3.1 Extraction of genomic DNA

Plant leaf material (approximately 0.5 x 0.5 cm^2) was harvested and transferred to 1.5-ml Eppendorf tubes containing two stainless-steel beads (2 mm diameter). Afterwards, 400 µl extraction buffer composed of 0.2 M Tris/HCl (pH 7.5), 0.25 M NaCl, 0.025 M EDTA and 0.5% (w/v) SDS was added. Samples were ground in a Retsch Mixer Mill MM2000 (Retsch, Haan, Germany) twice for two minutes at 30 Hz and then centrifuged at room temperature for 15 min at 13,000 rpm in a table-top centrifuge. The supernatants (300 µl per sample) were transferred into fresh 1.5-ml Eppendorf tubes, which was followed by the addition of 300 µl isopropanol. Samples were vortexed and incubated for two minutes at room temperature before they were centrifuged at room temperature for five minutes at 10,000 rpm. Afterwards, supernatants were discarded, and the pellets were washed with 300 µl 70% ethanol. This was followed by another centrifugation at room temperature for three minutes at 10,000 rpm. The supernatants were removed, pellets were dried at 60°C and then resolved in 40-100 µl distilled water.

2.3.2 Polymerase chain reaction

The extraction of genomic DNA was followed by polymerase chain reaction (PCR). PCR reactions contained DNA polymerase buffer including 2 mM $MgCl_2$, 1x *Taq* DNA polymerase, 200 µM dNTPs, 625 nM of a gene-specific forward and reverse primer and 1 µl of the undiluted gDNA extract in a 20 µl reaction. Primer pairs for PCRs were designed using the T-DNA Express software (http://signal.salk.edu/cgi-bin/tdnaexpress). Melting temperatures were checked using Tm Calculator software (https://tmcalculator.neb.com/). PCRs were performed with a thermocycler (Biometra, Analytik Jena GmbH, Jena, Germany) using the following cycling conditions: 3 min at 95°C for first strand dissociation, 25 to 32 cycles of 30 s at 94°C for denaturation, 55 s at 58-60°C depending on the primers for annealing, and 1 min per kb PCR product at 72°C for elongation, which was followed by a final extension for 3 min at 72°C. PCR products were analysed using agarose gel electrophoresis.

2.3.3 Agarose gel electrophoresis

Amplification of genomic DNA through PCR was followed by agarose gel electrophoresis. Agarose gels contained 1% to 1.5% (w/v) agarose dissolved in 1x TAE (40 mM TRIS, 40 mM acetic acid, 1 mM EDTA) and ethidium bromide solution (0.5 µl of 10 mg/ml stock solution was used for 50 ml 1x TAE). After addition of 6x loading buffer (to a final concentration of 1x, 30 % glycerol, 0.25 % bromophenol blue, 0.25 % xylene cyanol FF) to the PCR products, DNA fragments were separated according to their sizes in the agarose gel at 80 V using an Electrophoresis Power Supply (Bio-Rad Laboratories, California, USA). Separated DNA fragments were visualized using an ultraviolet (UV) transilluminator (Genoplex, VWR International GmbH, Darmstadt, Germany) and analysed using the software GenoCapture. For assessment of the DNA fragment sizes, 5 µl of the DNA ladder Hyperladder I (Thermo Scientific, Massachusetts, USA) were included in the agarose gel electrophoresis runs.

2.4 Analysis of transcript levels

2.4.1 RNA extraction and cDNA synthesis

For RNA extraction, plant leaf material (leaves eight to ten) was harvested, directly transferred to 2-ml Eppendorf tubes containing two stainless-steel beads (2 mm diameter) and immediately flash frozen in liquid nitrogen. Homogenization of the frozen plant material was performed in pre-chilled vessels with a Retsch Mixer Mill MM2000 (Retsch, Haan, Germany). Total RNA was extracted using the NucleoSpin® RNA plant kit (Machery and Nagel, Düren, Germany) as described in the user's manual. To prevent contamination with genomic DNA, DNase I (Fermentas, Life Technologies, Darmstadt, Germany) digestion was performed. RNA concentration was determined spectrophotometrically at a wavelength of 260 nm using a Nanodrop ND-1000 spectrophotometer (Nanodrop Technologies, Wilmington, USA). The quality of the extracted RNA was checked using the 260/280 nm and 260/230 nm ratios. DNase-digested RNA was used for RNA sequencing and quantitative real time-PCR experiments. For complementary DNA (cDNA) synthesis, 1 µg DNase-digested RNA was used in a 20 µl SuperScript® III Reverse Transcriptase reaction. First-strand cDNA synthesis was performed as follows: 1 µg DNase-digested RNA was mixed with 1 µl 50 µM oligo(dT)-primers, 1.8 µl 50 µM random hexamers (N9), 2 µl 5 mM dNTPs and water resulting in a final volume of 14.5 µl. This mix was incubated for 5 min at 65°C and then immediately placed on ice. Afterwards 4 µl 5x first-strand buffer, 1 µl 0.1 M DTT and 0.5 µl 200 U/µl SuperScript III Reverse Transcriptase (Invitrogen, Life Technologies, Thermo Fisher Scientific) were added. The resulting reaction mixture was incubated for 5 min at 25 °C, for 60 min at 50 °C, and 15 min at 70 °C in a thermocycler. The cDNA was diluted 1:10 for quantitative real time-PCR (**section 2.4.2**).

2.4.2 Quantitative real time-PCR

Quantitative real-time PCR (qRT-PCR) was used for expression analysis of genes of interest. qRT-PCR using FAST SYBR Green I technology was performed with a CFX96 Touch Real-Time Detection System (Biorad, Feldkirchen, Germany) using the following cycling conditions: 15 min 95°C, 40 cycles of 5 s at 95°C, 15 s at 55°C and 10 s at 72°C, which was followed by a dissociation analysis to check for specificity of the amplification. Reactions contained SYBR Green I, Immolase (Bioline, Memphis, USA), 300 nM (or 600 nM) of a gene-specific forward and reverse primer and 2 µl of a 1:10 diluted cDNA in a 20-µl reaction. Primer pairs for qRT-PCR were designed with Quantprime software (Arvidsson *et al.*, 2008) under the following conditions: Optimum melting temperature (Tm) at 60°C, GC content between 20% and 80%, 150 bp maximum length. Primer efficiency of the used primer pairs was checked using a standard curve. Sequences of the primers are listed in **Table 5**. Gene expression data were normalized against two or three reference genes (*UBC21, PP2A* and *MCP2A*) according to Vandesompele *et al.* (2002).

AGI codes of genes analysed in this study are: AT4G24230 (*ACBP3*), AT1G33960 (*AIG1*), AT2G37770 (*AKR4C9*), AT3G61190 (*BAP1*), AT4G39030 (*EDS5*), AT3G13610 (*F6'H1*), AT1G07050 (*FITNESS*), AT1G74710 (*ICS1*), AT1G19250 (*FMO1*), AT2G19190 (*FRK1*), AT1G79310 (*MCP2A*), AT1G79310

(*PP2A*), AT2G14610 (*PR1*), AT5G13830 (*TRM7C*), AT5G25760 (*UBC21*), and AT5G59820 (*ZAT12*). Additional AGI codes are provided in **Supplementary Table S1** to **Supplementary Table S41**.

Table 5. Primer sequences used for quantitative RT-PCR.

PE, primer efficiency. QP, Quantprime (https://quantprime.mpimp-golm.mpg.de/).

Primer name	Forward primer (FP) sequence	Reverse primer (RP) sequence	PE [%]	Reference
ACBP3	GACGAAAGCTGGTCATACTGTAG	TAAGCACTGGATTCACCTCTCC	105	Designed with QP
AIG1	CAAGGCAATGGCAGAGATGATG	GCGCACAGTGAATGATCAGAG	95	Designed with QP
AKR4C9	CCCGATACTGAATATGGTTGCG	CCAGTGACTAACCTAGCCTGTTC	97	Designed with QP
BAP1	CCAGAGATTACGGCGCGTGTT	TACAGACCCCAAACCGGAACTCC	99	Designed with QP
EDS5	GGTTCGTTCTCGTCGGATT	TTCTTGACATTGGTGCCTGA	91	Designed with QP
F6'H1	CCTGATATCTGCAGGAATGAAACG	GAGAGAAGAGACGTCTGAGTGG	91	Designed with QP
FITNESS	ACGTGGGATTTCTGGGAAGAAG	CCCTATAAACGGAATTGTCCAGAG	94	Designed with QP
ICS1	CGTCGTTCGGTTACAGGTT	CCGTTTCCGTTCTCGTTAG	94	(Choi *et al.*, 2010; Argueso *et al.*, 2012)
FMO1	CTCCATGATAGGCCTAACCAAAGC	AAACTACGGCACGCAGAAGAGAG	90	Designed with QP
FRK1	GAAGCGGTCAGATTTCAACA	TCAAGAAGAACAACCCCAAGA	-	(Choi *et al.*, 2010; Argueso *et al.*, 2012)
MCP2A	AACCCGCTATGCAGACACACG	CAGTTGGTTTCCCCGCTGGA	98	(Watanabe & Lam, 2011)
PP2A	CCATTAGATCTTGTCTCTCTGCT	GACAAAACCCGTACCGAG	94	Designed with QP
PR1	ATGCAGTGGGACGAGAGGGT	AACCCACATGTTCACGGCGG	86	(Ochsenbein *et al.*, 2006)
TRM7C	TCGGGAGTTATCACCTCAGC	AAGTACACCAGGACGACTTTCG	101	Designed with QP
UBC21	CTCTTAACTGCGACTCAGGGAATC	TGCCATTGAATTGAACCCTCTCAC	99	Designed with QP
ZAT12	CGCTTTGTCGTCTGGATTG	AGCAGCCCCACTCTCGTT	98	Designed with QP

2.4.3 RNA-sequencing

For RNA-sequencing (RNA-seq) analysis, total RNA of plant leaf material was extracted from three biological replicates using the NucleoSpin RNA plant kit (Machery and Nagel, Düren, Germany) as described in the user's manual. For the sensitivity (**section 3.1**) and *cis*-priming (**section 3.2**) experiments, the isolated RNA was sent to BGI (Hongkong, China) for sequencing. For the *trans*-priming experiments (**section 3.3**), the isolated RNA was sent to Novogene (Novogene Company Ltd, Cambridge, United Kingdom), where the RNA was processed and sequenced. Briefly, RNA amount and purity, RNA integrity (RIN) and contamination by rRNA were determined using a Nanodrop NA-1000, a Bioanalyzer Agilent 2100 (Agilent Technologies, Santa Clara, CA, United States) and agarose gel electrophoresis. The mRNA of DNase I-treated samples that passed the quality check was enriched and the mRNA was fragmented into shorter fractions. Using reverse transcription, cDNA was synthesized. After library preparation, the products were sequenced using the Illumina Novaseq or BGI Genomics platforms. BGI and Novogene obtained more than 44 million raw reads per sample. Adapter sequences of raw reads and low-quality sequences were removed from the raw reads. The resulting clean reads (approximately 40 million clean reads per sample) were stored as FASTQ files. Sequencing data were aligned to the Arabidopsis TAIR10 reference genome (https://www.arabidopsis.org/download/index-auto.jsp%3Fdir%3D%252Fdownload_files%252FGenes%252FTAIR10_genome_release) using the program Hierarchical Indexing for Spliced Alignment of Transcripts (HISAT2). Bowtie2 was used for aligning clean reads to the reference genes (Langmead & Salzberg, 2012). Gene expression levels were determined as Fragments Per Kilobase Million (FPKM) values.

Using R (version 4.3.1) and R studio, the DESeq2 method (Love *et al.*, 2014) was applied to identify differentially expressed genes (DEGs). Genotype and treatment were considered, and DEGs were filtered for Bonferroni-corrected *p*-value ≤ 0.05 and for $\log_2$ fold change values ≥ I1I.

Principal component analysis (PCA) was done using R (version 4.3.1) and R studio with the packages *PCAplot* and *ggplot2*. Non-normalized raw data (FPKM values) were used as input for PCA. Gene ontology (GO) term enrichment analysis was performed with PANTHER (Mi *et al.*, 2019) (https://www.arabidopsis.org/tools/go_term_enrichment.jsp) with the GO aspect biological process. Clustering analysis was performed with Multiple Experiment Viewer (MEV) (Howe *et al.*, 2011). The following parameters were used for quality threshold (QT) clustering: diameter = 0.5, minimum cluster size = 25 to 50, absolute distance = false, Pearson's correlation.

Venn diagrams were created with Venny2.1 (https://bioinfogp.cnb.csic.es/tools/venny/) or the Venn diagram tool of the University Ghent (https://bioinformatics.psb.ugent.be/webtools/Venn/).

2.5 Analysis of biochemical and physiological parameters under photoperiod stress

Exposure of SD-grown Arabidopsis plants to a PL period of 24 hours induced photoperiod stress. To evaluate the responses of Arabidopsis plants to prolongations of the light period shorter than 24 h, biochemical and physiological parameters were analysed.

2.5.1 Reactive oxygen species levels

Peroxides were determined according to Abuelsoud *et al.* (2020). Briefly, leaf material (leaves six to eleven of four-week-old plants) was harvested, weighted, and directly transferred to 2-ml Eppendorf tubes containing two stainless-steel beads (2 mm diameter). Subsequently, the plant material was flash-frozen in liquid nitrogen. Homogenization of the frozen plant material was performed in pre-chilled vessels with a Retsch Mixer Mill MM2000 (Retsch, Haan, Germany). Peroxides were extracted by addition of 300 µl 0.1% ice cold trichloroacetic acid (TCA) to 100 mg frozen plant material. After homogenization by vortexing, the thawed samples were incubated for 15 min on ice and then centrifuged at 4°C for 15 min at 11,000 rpm. The number of water-soluble peroxides including hydrogen peroxide was determined in the supernatant using a xylenol orange-based method (PierceTM Quantitative Peroxide Assay Kit (aqueous), ThermoFisher Scientific, Berlin, Germany) according to user's manual. Hydrogen peroxide was used as a standard. The absorbance was measured at 595 nm in a 96-well plate reader (Synergy HT, Biotek, Vermont, USA). Water-soluble peroxides were expressed as nmol ROS equivalents per g fresh weight.

2.5.2 Malondialdehyde levels

Malondialdehyde (MDA) levels were determined according to Hodges *et al.* (1999). Sampling for peroxide measurements was done as described. A volume of 500 µl 0.1% ice cold TCA was added to approximately 100 mg finely-ground leaf material. After homogenization by vortexing, samples were

centrifuged at 4°C for 15 min at 10,000 rpm. The supernatant was incubated at 95°C for 15 min with 0.5% thiobarbituric acid (TBA), which was dissolved in 20% TCA by 5 min-incubation in a water bath, resulting in the formation of thiobarbituric acid-malondialdehyde (TBA-MDA). Absorbance was measured at 532 and 600 nm in a 96-well plate reader (Synergy HT, Biotek, Vermont, USA).

2.5.3 Enzyme activities

Activities of guaiacol peroxidase (GP, EC 1.11.1) and catalase (CAT, EC 1.11.1.6) were determined according to Abuelsoud *et al.* (2020) and modified from Murshed *et al.* (2008). Sampling was done as described for peroxide measurements. The antioxidant enzyme activities were measured in approximately 100 mg finely ground, frozen plant leaf material and extracted by addition of 1 ml ice cold 50 mM MES-KOH buffer (pH 6.0) containing 40 mM KCl, 2 mM $CaCl_2$, 10% glycerol, 0.1% Triton X-114, 1 mM L-ascorbic acid, 1 mM phenylmethylsulfonyl fluoride (PMSF) and 1% PVPP. After homogenization by vortexing, samples were centrifuged at 4°C for 20 min at 16,000 rpm. Supernatants were used for enzyme activity measurements, which were performed in triplicates in a final volume of 0.2 ml in a 96-well plate reader (Synergy HT, Biotek, Vermont, USA). Guaiacol peroxidase activity was determined at 470 nm by monitoring the oxidation of guaiacol (ε_{470} = 26.6 mM^{-1} cm^{-1}) in 50 mM K-phosphate buffer (pH 7.0) containing 25 mM H_2O_2 and 25 mM guaiacol (Kumar & Khan, 1982). Catalase activity was evaluated at 240 nm by monitoring the decomposition of H_2O_2 (ε_{240} = 43.6 mM^{-1} cm^{-1}) in 50 mM K-phosphate buffer (pH 7.0) containing 25 mM H_2O_2 (Aebi, 1984). The protein content in the enzyme extracts was determined using Bradford assay (BioRad) at 595 nm (Bradford, 1976) with a 96-well plate reader (Synergy HT, Biotek, Vermont, USA).

2.5.4 Phytohormone and camalexin levels

Concentrations of camalexin, SA, JA, and JA-Ile were determined by UPLC-MS/MS (Q-ToF-ESI; Synapt G2-S HDMS; WatersR, Milford, Massachusetts, United States) according to Valsamakis *et al.* (2020). The analysis was done in collaboration with Dr. Vivien Lortzing.

2.6 Infections by pathogens

2.6.1 Infection by *Pseudomonas syringae*

Four-week-old Arabidopsis plants were inoculated with virulent *Pseudomonas syringae* pv. *tomato* DC3000 (*Pst*) bacteria as described in Griebel and Zeier (2008) with some modifications. Briefly, *Pst* was grown for one day at 28°C in LB medium (Carl Roth, Karlsruhe, Germany) containing rifampicin (final concentration of 50 µg/ml) under permanent shaking. For plant inoculation, bacteria were streaked out on LB-agar plates containing rifampicin (50 µg/ml). After two days of growth at 28°C, the bacteria were re-streaked onto new plates and grown for two further days at 28°C. Bacteria were collected with a Drigulski spatula from the plate by addition of 4 ml 10 mM $MgCl_2$. The resulting *Pst* suspension was transferred to a falcon tube and 10 mM $MgCl_2$ was added to a final volume of 25 ml. After centrifugation

for 10 min at 4000 rpm at 21°C, the resulting bacteria pellet was resuspended in 5 ml 10 mM $MgCl_2$ and the optical density was spectrophotometrically determined at 600 nm in a spectrophotometer (Windaus-Labortechnik, Clausthal-Zellerfeld, Germany). Using 10 mM $MgCl_2$, the bacterial solution was diluted to obtain bacterial titres of 0.002 or 0.005 for inoculation of Arabidopsis leaves (three leaves per plant, leaves 8 - 10) for bacterial growth experiments or RNA extraction, respectively. Inoculation of the abaxial leaf side was done using a 2-ml syringe without a needle. A mock inoculation was performed with 10 mM $MgCl_2$. Sampling was done one or three days post infection for RNA extraction or bacterial growth experiments, respectively. Bacteria were extracted from two leaves per plant by homogenisation in 0.1 ml 10 mM $MgCl_2$. Dilutions of the bacterial suspension were pipetted on LB-agar plates containing rifampicin and cycloheximide (50 µg/ml). Resulting colony numbers were counted after incubating the plates for two days at 28°C.

2.6.2 Infection by *Botrytis cinerea*

For infection of Arabidopsis plants with *B. cinerea*, the mycelium of the fungus was grown on Petri dishes containing 0.5x sterile potato dextrose agar for 14 days (10 h light/14 h dark cycles). For harvesting, 5 ml sterile water were added onto the plates, *Botrytis* spores were collected using a Drigalski spatula, filtered through Miracloth and collected in a 50-ml falcon tube. The number of spores/ml was determined using a light microscope (LEICA DM IL LED inverse light microscope, Wetzlar, Germany) and a Fuchs-Rosenthal counting chamber (0.200 mm, 0.0625 mm^{-1}). For infection, spore concentrations were adjusted to $1.5 * 10^4$ spores per ml. Droplets with a volume of 20 µl were pipetted on Arabidopsis leaves (one droplet per leaf, three leaves per plant, leaves 8 - 10). The plants were kept under SD conditions for three days in sealed, transparent plastic boxes ensuring high humidity. The degree of infection was captured in photographs using a Nikon camera D3000 (Nikon Corporation, Tokyo, Japan) and evaluated with ImageJ (https://imagej.nih.gov/ij/download.html).

2.7 Statistical analysis

Datasets were statistically evaluated with the software R (version 4.3.1) and RStudio (http://www.rstudio.com). Normal distribution of data and homoscedasticity were evaluated using Shapiro-Wilk normality and Levene's test, respectively. Data with normal distribution and homogenous variances were analysed using ANOVA followed by Tukey's post hoc tests. If data were not normally distributed, data were log-transformed to fulfil the criteria for parametric test procedures. When normality or homogeneity of variances were not met, the non-parametric Kruskal-Wallis test was performed followed by a Wilcoxon-Mann-Whitney test to perform pairwise comparisons. Data were visualized using Microsoft Excel and PowerPoint.

3 Results

3.1 Sensitivity of Arabidopsis plants to photoperiod stress

In the first part of this thesis, the transcriptomic response of SD-grown Arabidopsis plants to different prolonged light (PL) periods (1 h, 2.5 h and 4 h) was investigated by RNA-seq (**Figure 18**) to improve our understanding of the light conditions that induce photoperiod stress. In addition, samples were harvested at the end of a SD to exclude genes that are regulated by the time of the day (end of SD *vs.* control, **Figure 18**).

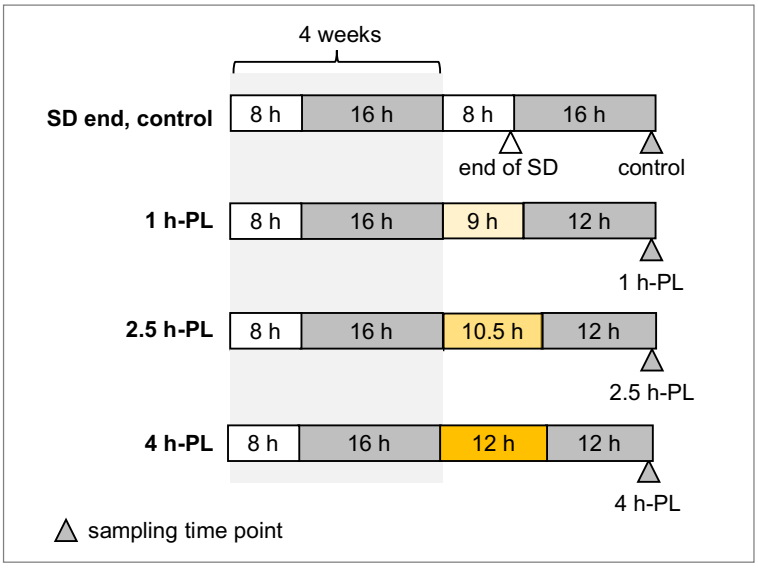

Figure 18. Analysis of sensitivity of Arabidopsis to a suddenly occurring prolonged light period.

Experimental setup of RNA-seq analysis for the sensitivity experiment. Four-week-old SD-grown (8 h light/16 h darkness) Arabidopsis plants were exposed to an 1 h-, 2.5 h- or 4 h-PL period or remained under SD conditions (control). Sampling (indicated by triangles) was performed at the end of the night following the light treatments. To exclude genes regulated by the time of the day from the analysis, additional plant material was harvested at the end of a normal SD (indicated by white triangle). White, light period; grey, dark period; orange, light period that is longer than a normal SD.

3.1.1 Exploratory data analysis of RNA-seq data

First, a PCA was performed. The PCA revealed that principal component 1 (PC1, x-axis) accounted for 55% and PC2 (y-axis) for additional 39% of the total variation in the RNA-seq dataset (**Figure 19A**). Thus, PC1 and PC2 together covered 94% of the variation of the expression in the dataset (Koch *et al.*, 2018). Biological replicates clustered together suggesting similarities among them and reproducibility of the expression data (**Figure 19A**). Notably, samples harvested at the end of an SD formed a cluster far away from the other samples that were harvested at the end of the night. This suggests that the time of the day strongly influenced the expression data. Samples of plants that were treated with a short PL period of 1 h or 2.5 h closely clustered together with control samples, while samples of plants treated with a longer PL of 4 h clustered further away from the control indicating that significant changes in transcript abundance are caused by this extended PL treatment (**Figure 19A**). The PCA suggested that the RNA-seq dataset was qualified for further data analysis.

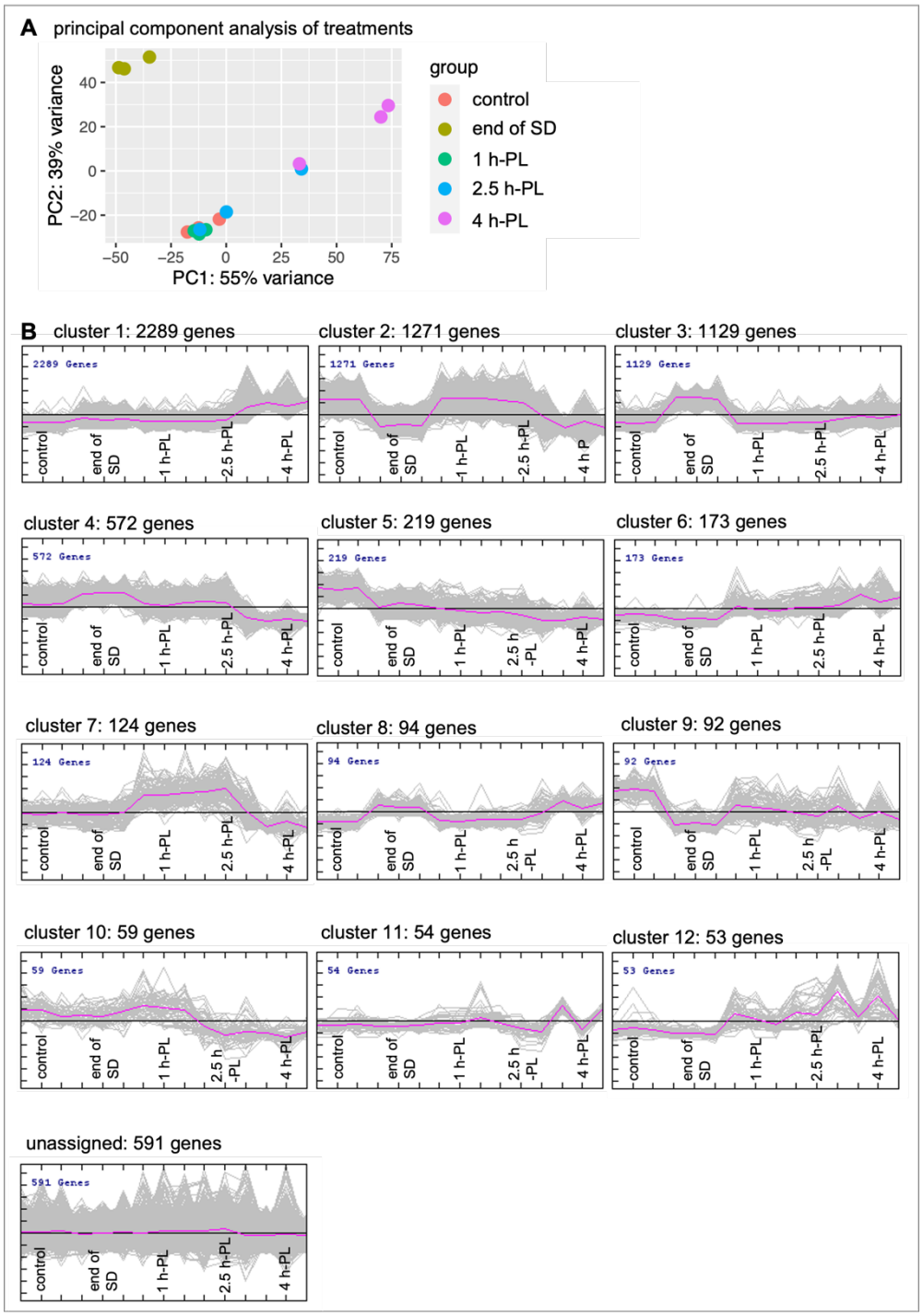

Figure 19. Analysis of fragments per kilobase million (FPKM) values in Arabidopsis plants under control conditions, at the end of a SD and exposed to a 1 h, 2.5 h or 4 h PL period.

(A) PCA of Arabidopsis plants under control conditions, at the end of a SD or exposed to a 1 h-, 2.5 h- or 4 h-PL period (three biological replicates per treatment). **(B)** QT clustering, performed with the following parameters: diameter = 0.5, minimum cluster size = 50 genes, absolute distance = false, Pearson's correlation, resulted in 12 clusters and one additional cluster with unassigned genes. Purple line, average levels of FPKM values (BH-corrected) of genes in the respective clusters.

To get first insights into the RNA-seq dataset, QT clustering was performed (**Figure 19B**) allowing to partition the expression data into clusters (Danalis *et al.*, 2012). The resulting clusters were further

analysed by GO term enrichment analysis (**Figure 20** and **Figure 21**). QT clustering revealed 12 clusters and one further cluster with unassigned genes (**Figure 19B**). Eighty-four percent of all regulated genes were detected in clusters 1 to 6.

Cluster 1 contained genes showing an upregulation in response to a 4 h-PL period corresponding to 34% of all regulated genes (2289 genes) (**Figure 19B**). According to GO term analysis, genes involved in toxin biosynthesis, (indole) phytoalexin biosynthesis and metabolism, responses to molecules of bacterial or fungal origin (chitin), responses to ER unfolded proteins, and responses to ozone were enriched in this cluster (**Figure 20A**). This suggests that most genes induced by a 4 h-PL period are also regulated during pathogen defence responses.

Cluster 2 comprised 19% of all regulated genes (1271 genes) displaying a downregulation at the end of a SD and in response to a 4 h-PL period (**Figure 19B**). According to GO term analysis, this cluster contained genes that were assigned to light-dependent biological processes, such as photosynthesis, light harvesting and reactions, chlorophyll biosynthesis and metabolism, responses to light intensities (**Figure 20B**). The similar regulation of genes at the end of a SD and in response to a 4 h-PL period suggests that several genes regulated by a 4 h-PL are also modulated by the time of the day.

Cluster 3 contained 17% of all regulated genes (1129 genes) showing an upregulation at the end of a SD (**Figure 19B**). This cluster comprised genes important for ribosomal RNA (rRNA) biogenesis, maturation, modification and processing, and ribosome biogenesis (**Figure 20C**).

Cluster 4 contained 9% of all regulated genes (572 genes) which were upregulated at the end of a SD and downregulated in response to a 4 h-PL period (**Figure 19B**). This cluster was enriched with genes assigned to plant (cellular) biosynthetic and metabolic processes, such as biosynthesis of small molecules or organic substances (**Figure 20D**).

Clusters 5 and 6 each comprised around 3% of all regulated genes (219 genes in cluster 5, 173 genes in cluster 6) which revealed a downregulation (cluster 5) or an upregulation (cluster 6) in response to PL periods (**Figure 19B**). Notably, a longer duration of the PL period is associated with a higher amplitude of the expression of genes in this cluster. The expression patterns of the genes in clusters 5 and 6 suggest that a longer PL period promotes a more pronounced transcriptomic response. According to GO term analysis, genes involved in responses to auxin, (red or far-red) light, developmental growth, cell wall organization and biosynthesis, and lipid biosynthesis are enriched in cluster 5 (**Figure 20E**). Cluster 6 is enriched with genes involved in responses to SA, regulation of (bacterial or fungal) plant defence responses, oxidative stress, and responses to wounding (**Figure 20F**). The latter suggests that PL periods induce responses that resemble those regulated during pathogen attack.

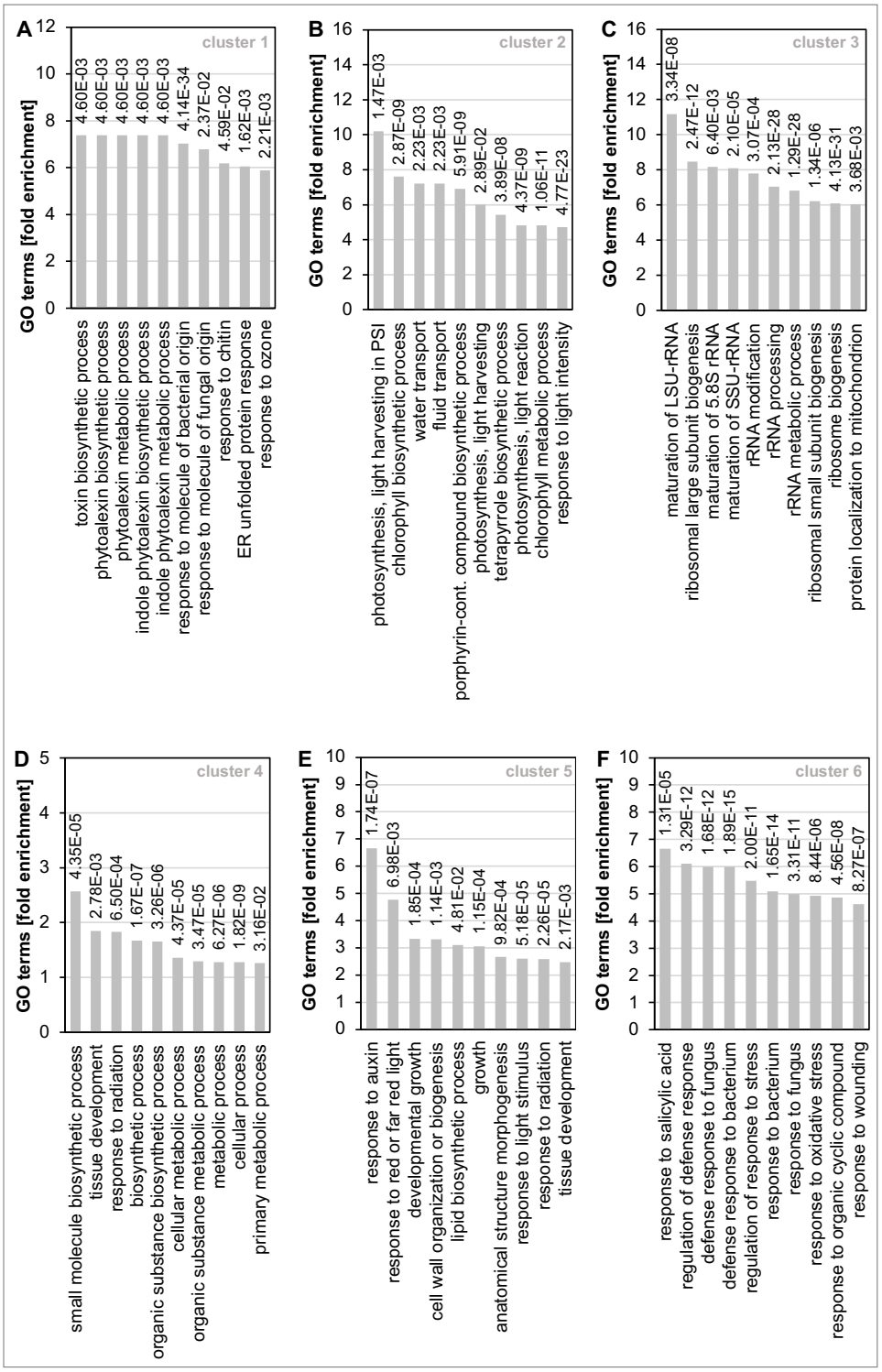

Figure 20. GO term analysis of QT clusters 1 to 6 for genes regulated in response to PL periods of 1 h, 2.5 h or 4 h or at the end of a SD.

GO term analysis of clusters derived from QT clustering displayed in **Figure 19**. **(A)** Cluster 1 (upregulation in response to a 4 h-PL period). **(B)** Cluster 2 (downregulation at the end of a SD and in response to a 4 h-PL period). **(C)** Cluster 3 (upregulation at the end of a SD). **(D)** Cluster 4 (upregulation at the end of a SD and downregulation in response to a 4 h-PL period). **(E)** Cluster 5 (downregulation in response to PL periods of 1 h to 4 h). **(F)** Cluster 6 (upregulation in response to PL periods of 1 h to 4 h). The top ten GO terms are shown. Numbers above the bars represent *p*-values.

Cluster 7 contained 2% of all regulated genes (124 genes) which were upregulated in response to 1 h- and 2.5 h-PL periods but downregulated in response to 4 h-PL (**Figure 19B**). Genes in this cluster were assigned to plant biosynthetic and metabolic processes (**Figure 21A**). The opposite regulation of genes in this cluster suggests that biological processes exist that are regulated in dependence of the length of the PL period.

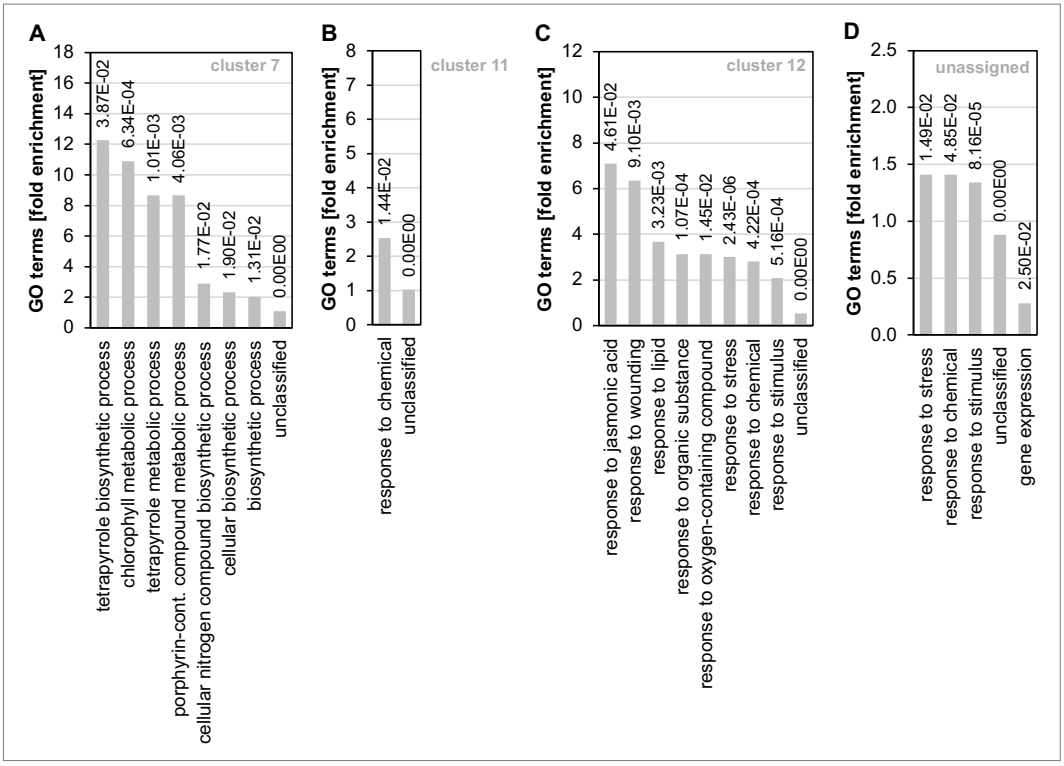

Figure 21. GO term analysis of QT clusters 7 to 12.

GO term analysis of clusters derived from QT clustering displayed in **Figure 19**. **(A)** Cluster 7 (upregulation in response to 1 h- and 2.5 h-PL periods but downregulation in response to 4 h-PL). **(B)** Cluster 11 (inconsistent regulation in response to a 4 h-PL period). **(C)** Cluster 12 (genes showing an upregulation in response to all PL periods having a stronger amplitude (especially in response to 4 h-PL periods) than genes in cluster 5). **(D)** Unassigned genes. GO term analysis of the remaining clusters 8, 9, and 10 resulted in unassigned biological processes. The top ten GO terms are shown. Numbers above the bars represent *p*-values.

Each of clusters 8 to 12 contained around 1% of all regulated genes (**Figure 19B**). Genes in clusters 8, 9 and 10 remained unclassified by GO term analysis. GO terms of clusters 11 and 12 are shown in **Figure 21B-C**. Due to the low number of genes in these clusters, their regulation and associated GO terms are not described in detail.

Notably, 9% of all regulated genes (591 genes) remained unassigned in the QT clustering (**Figure 19B**). It is important to mention that among the unassigned genes several are also involved in plant responses of plants to environmental stresses or treatment with chemicals (**Figure 21D**).

3.1.2 Comparison of transcriptomic changes in response to 1 h-, 2.5 h- and 4 h-PL periods in Arabidopsis plants

To investigate the transcriptomic changes in response to 1 h, 2.5 h and 4 h PL periods, differentially expressed genes (DEGs) were analysed in Arabidopsis plants using the RNA-seq data. In total 10223 genes were differentially expressed in a treatment-dependent manner (as calculated by Bonferroni correction, *p*-value ≤ 0.05).

Pairwise comparisons were performed to get more insights into the DEGs following a PL period. Comparisons between PL-treated and control samples revealed that 119 genes were differentially expressed in response to a 1 h-PL period, 421 genes were differentially expressed in response to a 2.5 h-PL period and 4299 genes were differentially expressed in response to a 4 h-PL period. This shows that the number of DEGs increases when the PL period is extended. Already a PL period of 1 h is sufficient to affect transcription in Arabidopsis plants. Comparisons between samples harvested at the end of a SD and the end of the night (control) resulted in 5366 DEGs.

The top 20 up- and down-regulated genes derived from the pairwise comparisons are summarized in **Supplementary Table S1** to **Supplementary Table S8**. Among the top 20 upregulated genes in response to a 1 h-PL period, genes involved in circadian regulation, such as *REVEILLE2* (*RVE2*), light signalling, such as *B-BOX DOMAIN PROTEIN7* (*BBX7*) and *BBX32*, and redox regulation, such as *GLUTATHIONE PEROXIDASE7* (*GPX7*), are included (**Supplementary Table S3**). In response to a 2.5 h-PL period, genes associated with light harvesting and signalling, such as *LIGHT HARVESTING COMPLEX PHOTOSYSTEM II* (*LHCB4.3*) or *CONSTANS* (*CO*), and genes connected to JA biosynthesis and signalling, such as *ALLENE OXIDE CYCLASE2* (*AOC2*) and *JASMONATE-ZIM-DOMAIN PROTEIN7* (*JAZ7*), were identified among the top 20 upregulated genes (**Supplementary Table S5**). Among the top 20 upregulated genes in response to a 4 h-PL, genes that are responsive to pathogens, such as *AVRRPT2-INDUCED GENE1* (*AIG1*) or *RESISTANCE METHYLATED GENE1* (*RMG1*), the camalexin biosynthesis gene *PAD3* and the hypoxia response gene *ZAT8* are included (**Supplementary Table S7**). Notably, genes connected to auxin biosynthesis and signalling, such as *INDOLE-3-ACETIC ACID INDUCIBLE29* (*IAA29*) and *SMALL AUXIN UP RNA22/23* (*SAUR22/23*), were identified among the top 20 downregulated genes in response to a 1 h-PL period (**Supplementary Table S4**), to a 2.5 h-PL (**Supplementary Table S6**) and to a 4 h-PL (**Supplementary Table S8**).

To identify biological processes associated to the most strongly regulated genes in response to PL periods of 1 h, 2.5 h and 4 h, DEGs derived from the pairwise comparisons were further filtered for genes that were either two-fold up- or downregulated (Bonferroni correction, *p*-value ≤ 0.05, $\log_2$fold change = I1I). The resulting gene lists were subjected to GO term enrichment analysis (**Figure 22A-C**). This analysis revealed that genes assigned to responses to auxin, regulation of and responses to hormones, responses to (endogenous) stimuli and chemicals are overrepresented after exposure to a 1 h-PL period in comparison to control (1 h-PL *vs.* control, **Figure 22A**). Genes associated with responses to the light environment, such as responses to far red or red light and light intensities, were overrepresented after treatment with a 2.5 h-PL in comparison to control. Similar as observed for 1 h-PL *vs*. control, treatment with a 2.5 h-PL period resulted in the enrichment of genes connected to the regulation of and responses to hormones, including auxin (2.5 h-PL *vs.* control, **Figure 22B**). Among

the top ten significantly enriched GO terms after a 4 h-PL period, genes associated with responses to molecules of bacterial origin, SAR, (cellular) responses to SA, hormone transport, such as auxin (polar) transport, and responses to hypoxia and endoplasmic reticulum (ER) stress were overrepresented (4 h-PL *vs*. control, **Figure 22C**). The consistent appearance of the regulation of genes related to responses to hormones including auxin-dependent responses suggests that this might be a common response to PL periods as the response to a 24 h-PL period also depends on auxin (Frank *et al.*, 2022).

To identify genes commonly regulated by PL periods, the gene lists (filtered for *p*-value (Bonferroni-corrected) ≤ 0.05, log_2fold change = I1I) were compared with each other. Genes regulated in the comparison between the end of a SD and the control were excluded. The results of this comparison were visualised in a Venn diagram (**Figure 22D**). Excluding genes that were regulated by the time of the day (end of SD *vs*. control), 22 genes were regulated in response to a 1 h-PL period *vs.* control, 74 genes were regulated in response to a 2.5 h-PL period *vs.* control, and 2332 genes were regulated in response to a 4 h-PL period *vs.* control (**Figure 22D**). Among those genes, in total 12 were regulated by all analysed PL periods (**Figure 22D**, orange box). They might represent potential candidates functionally involved in plant responses to different PL periods; however further analyses are needed to clarify this. Three genes, namely AT3G55646, AT5G59010 (*BRASSINOSTEROID-SIGNALING KINASE5*, *BSK5*), AT5G22460, were solely regulated in response to 1 h-PL *vs*. control. Five additional genes, namely AT5G56840, AT3G24460, AT4G31870 (*GLUTATHIONE PEROXIDASE7*, *GPX7*), AT5G10170 (*MYO-INOSITOL-1-PHOSPHATE SYNTHASE3*, *MIPS3*), AT5G66400, were regulated by both 1 h-PL *vs*. control and 2.5 h-PL *vs*. control. In total 14 genes were solely regulated by 2.5 h-PL *vs*. control (**Figure 22D**), including AT1G52590, AT1G70985, AT4G09970, AT1G21110 (*IGMT3*), AT5G37550, AT2G41540 (*GPDHC1*), AT5G08640 (*FLS1*), AT2G44130, AT1G66670 (*CLPP3*), AT3G26320 (*CYP71B36*), AT3G02310 (*SEP2*), AT5G40160 (*EMB506*), AT2G07718, AT2G40100 (*LHCB4.3*).

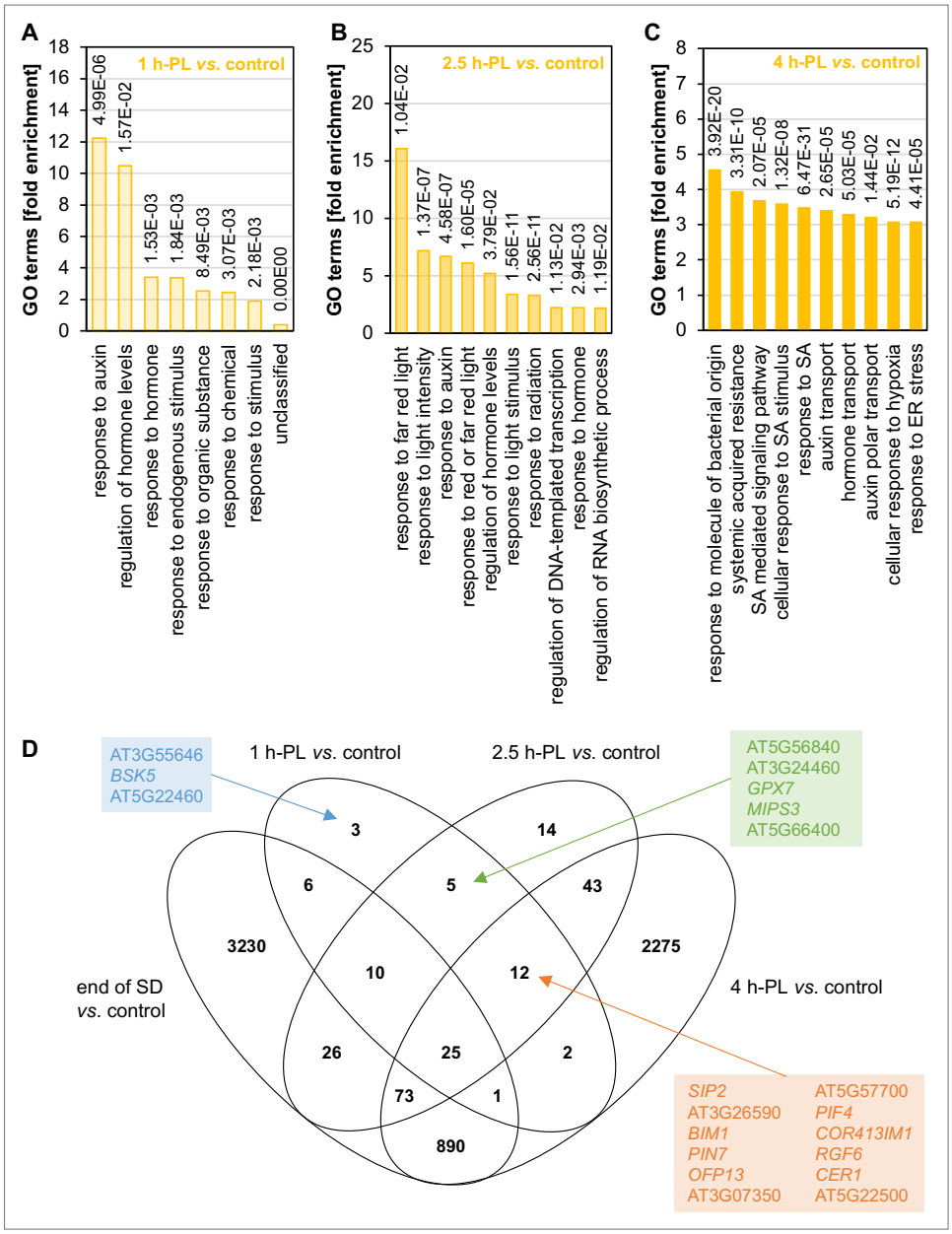

Figure 22. Analysis of genes significantly regulated in Arabidopsis plants treated with a PL period of 1 h, 2.5 h or 4 h in comparison to control conditions.

(A-C) GO term enrichment analysis of DEGs determined in the pairwise comparison (A) of 1 h-PL *vs.* control, (B) of 2.5 h-PL *vs.* control, and (C) of 4 h-PL *vs.* control. The top ten GO terms are shown. Numbers above the bars represent *p*-values. **(D)** Venn diagram comparing DEGs in Arabidopsis plants at the end of a SD or treated with a PL period of 1 h, 2.5 h or 4 h in comparison to control conditions (Bonferroni-corrected values, adjusted *p*-value ≤ 0.05; $\log_2$fold change ≥ |1|). Blue, genes only regulated by 1 h-PL *vs.* control; green, genes regulated by 1 h-PL *vs.* control and 2.5 h-PL *vs.* control; orange, genes regulated by 1 h-PL *vs.* control, 2.5 h-PL *vs.* control and 4 h-PL *vs.* control.

3.1.3 Comparison of transcriptomic changes in response to PL periods of different durations in Arabidopsis

To identify genes ubiquitously regulated in Arabidopsis plants in response to PL periods of different durations, the above-mentioned gene lists (**section 3.1.2**) were compared with lists of genes regulated in response to an 8 h-PL (this study, **section 3.3**) and a 24 h-PL period (Cortleven *et al.*, 2022). The comparisons were visualised in Venn diagram (**Figure 23A**).

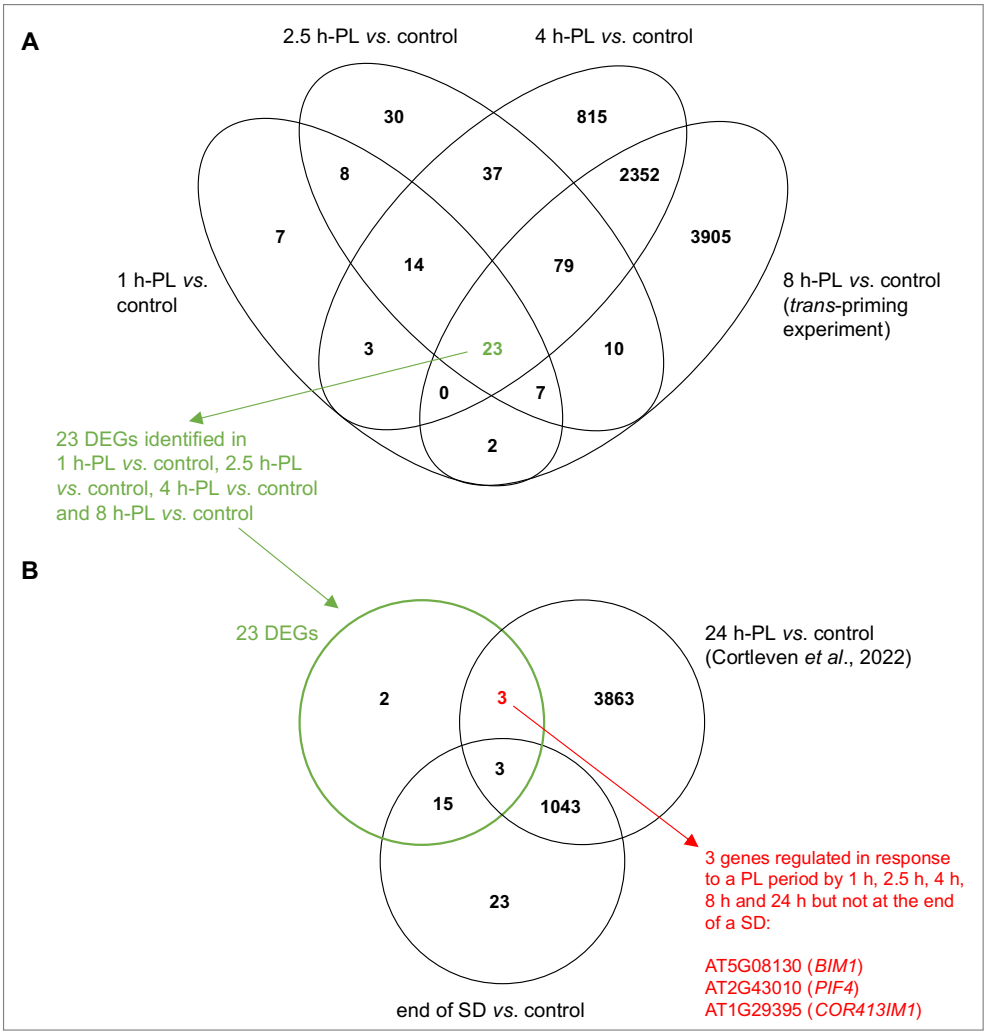

Figure 23. Comparison of the transcriptomic response in Arabidopsis plants to PL periods of different durations.

Comparison of the transcriptomic response in Arabidopsis plants to mild photoperiod stress (induced by a PL period by 1 h, 2.5 h, 4 h or 8 h) and strong photoperiod stress (induced by a 24 h-PL period; Cortleven *et al.* (2022)). **(A)** Venn diagram comparing Arabidopsis plants exposed to a PL period by 1 h, 2.5 h, 4 h and 8 h in comparison to control (Bonf-corrected values, adjusted *p*-value ≤ 0.05; $\log_2$fold change ≥ I1I). Genes (23 in total listed in **Supplementary Table S9**) that are significantly regulated in response to an 1 h, 2.5 h, 4 h and 8 h-prolongation of the light period are marked in green. **(B)** Venn diagram comparing Arabidopsis plants exposed to strong photoperiod stress (Cortleven *et al.*, 2022) with 23 DEGs that occurred in 1 h-PL *vs.* control, 2.5 h-PL *vs.* control, 4 h-PL *vs.* control and 8 h-PL *vs.* control (derived from (A)) and with genes regulated at the end of a SD in comparison to control conditions. Genes (3 in total) that are significantly regulated by all prolongations of the light period but at the same time not regulated in samples harvested at the end of a normal SD are marked in red.

In total 23 genes were commonly regulated in response to 1 h-PL, 2.5 h-PL, 4 h-PL, and 8 h-PL periods (**Supplementary Table S9**, **Figure 23B**). These 23 genes were further compared with DEGs detected in response to a 24 h-PL period in Arabidopsis plants (Cortleven *et al.*, 2022) and with genes regulated at the end of a SD in comparison to control, thereby excluding genes regulated by the time of day. In summary, three genes were detected to be regulated in response to all PL periods independent of their duration (but not regulated by the time of the day), namely AT5G08130 (*BES1-INTERACTING MYC-LIKE1*, *BIM1*), AT2G43010 (*PHYTOCHROME INTERACTING FACTOR4*, *PIF4*), and AT1G29395 (*COLD REGULATED 314 THYLAKOID MEMBRANE1*, *COR413IM1*) (**Supplementary Table S9**).

3.1.4 Regulation of the expression of genes encoding antioxidant enzymes in response to PL periods of different durations

Previous studies have shown that photoperiod stress is associated with an increase in peroxide levels and a regulation of genes related to oxidative stress (Cortleven *et al.*, 2022). Notably, a 2 h-PL period is sufficient to induce the accumulation of peroxides in Arabidopsis plants (Abuelsoud *et al.*, 2020). To determine if an increase in peroxides in response to PL shorter than 24 h is accompanied by transcriptomic changes related to the plant redox system, the regulation of genes encoding antioxidants was investigated. An available list of 221 genes (Cortleven *et al.*, 2022) was compared with the gene lists derived from pairwise comparisons (**section 3.1.2**). The RNA-seq analysis revealed that most genes encoding antioxidants are regulated, however not significantly, in response to PL periods of 1 h, 2.5 h and 4 h compared to control (**Supplementary Table S10**). Only one of the 221 analysed genes encoding antioxidants (*GPX7*) was significantly regulated in response to a PL period of 1 h. Three genes (*GPX7*, AT5G51100, AT4G33040) were significantly affected by a 2.5 h-PL period, and 46 genes by a 4 h-PL period (**Supplementary Table S10**). In a few cases, the expression of redox-related genes appeared to be more strongly affected by a 4 h-PL than by PL periods of 1 h and 2.5 h, for example for *AOX1A* (**Supplementary Table S10**). This indicates that the duration of the PL period influences the extent of regulation of genes related to the plant redox system.

3.1.5 Regulation of the expression of genes related to SA and JA biosynthesis/signalling in response to PL periods of different durations

Photoperiod stress strongly upregulates the expression of several SA- and JA-related genes (Cortleven *et al.*, 2022). To investigate the effects of 1 h-, 2.5 h- and 4 h-PL periods on the transcription of SA- and JA-related genes, respective gene lists (Cortleven *et al.*, 2022) were compared with the gene lists derived from pairwise comparisons (**section 3.1.2**). The RNA-seq data revealed that several genes related to SA and JA were regulated in response to PL periods of 1 h, 2.5 h and 4 h (**Supplementary Table S11** and **S12**). As observed for genes related to the redox system, the expression of several SA- and JA-related genes was more strongly regulated by a 4 h-PL than by PL periods of 1 h and 2.5 h (**Supplementary Table S11** and **S12**). Several genes related to SA biosynthesis, such as *EDS5/16* and *PBS3*, and signalling, such as *PAD4*, *EDS1*, *NIM1-INTERACTING1/2* (*NIMIN1/2*), *ALD1*, *SARD1/4* and *FMO1*, were strongly induced by a 4 h-PL. In addition, genes related to JA biosynthesis, such as *AOC2/3*

and *LOX2/4*, and signalling, such as *JAZ1/5/7*, were induced in response to a 4 h-PL. Numerous genes tended to be similarly regulated by shorter PL periods, however, often with smaller amplitudes (**Supplementary Table S11** and **S12**) suggesting that the duration of the PL period determines the extent of expression regulation of SA- and JA-related genes.

3.2 *Cis*-priming by photoperiod stress

In the second part of the thesis, the effects of a first photoperiod stress on plant responses to a similar second photoperiod stress were investigated. *Cis*-priming is described in the context of different stresses (**section 1.5.2**) but has not been investigated for photoperiod stress so far.

3.2.1 A PL period of 4 h primes Arabidopsis for future PL periods

Photoperiod stress induced by a 24 h-PL stimulates an oxidative burst in Arabidopsis. Not only PL periods of 24 h but also shorter prolongations in the range of a few hours induce oxidative stress (Abuelsoud *et al.*, 2020). To further confirm and characterize the oxidative stress-like response after a 4 h-PL, markers of oxidative stress were evaluated including stress marker gene expression, peroxide content, MDA levels, and enzyme activities. The experiments revealed that several markers of oxidative stress were affected by a 4 h-PL (**Figure 24**). More precisely, *ZAT12* and *BAP1* expression was induced, and peroxide content increased in response to 4 h-PL period, while the activity of the antioxidant enzyme catalase (CAT) decreased (**Figure 24B, C, E, G**) which suggests that a 4 h-PL period is sufficient to induce an oxidative stress-like response. The decreased CAT activity might be causative for the observed increase in peroxide levels and subsequent increase in the expression of redox-regulated genes such as *ZAT12* and *BAP1*. Other oxidative stress markers, such as MDA levels, functioning as an indicator of membrane damage, and the enzyme activity of guaiacol peroxidase (GP), were not affected by a 4 h-PL period (**Figure 24D, F**).

The effects of priming by photoperiod stress for future PL periods have not been investigated before. To study this, Arabidopsis plants were exposed to a 4 h-PL period as priming and triggering stimuli. Two setups were considered: The priming and triggering stimuli were applied (i) without a lag phase (PL0T), or (ii) with one day lag phase in-between (PL1T). As described above, *ZAT12* and *BAP1* were induced and the peroxide content increased, while CAT activity decreased in Arabidopsis plants in response to a single 4 h-PL period as priming (P) or triggering (T0, T1) (**Figure 24B, C, E, G**). One day after the priming stimulus (PL1), the expression of the analysed photoperiod stress-responsive genes, the peroxide content and CAT activity returned to control levels (**Figure 24B, C, E, G**). This suggests that a one-day lag phase is sufficient for Arabidopsis plants to compensate photoperiod stress-induced changes related to oxidative stress.

Exposure of Arabidopsis plants pre-treated with 4 h-PL revealed that the expression of *ZAT12* and *BAP1* and the accumulation of peroxides was suppressed when plants were exposed to a second 4 h-PL (PL0T, PL1T, **Figure 24B, C, E**). In addition, CAT activity did not decrease in plants exposed to a second 4 h-PL period after pre-treatment (PL0T, PL1T, **Figure 24G**). MDA levels and GP activity remained

comparable to those observed in response to a single 4 h-PL (**Figure 24D, F**). The lacking responsiveness to a PL period in plants that were pre-treated with a 4 h-PL suggests priming of respective plant responses by photoperiod stress. The observation that plants responded differently to a second 4 h-PL when applied after a lag phase of one day (PL1T) indicates a memory of photoperiod stress.

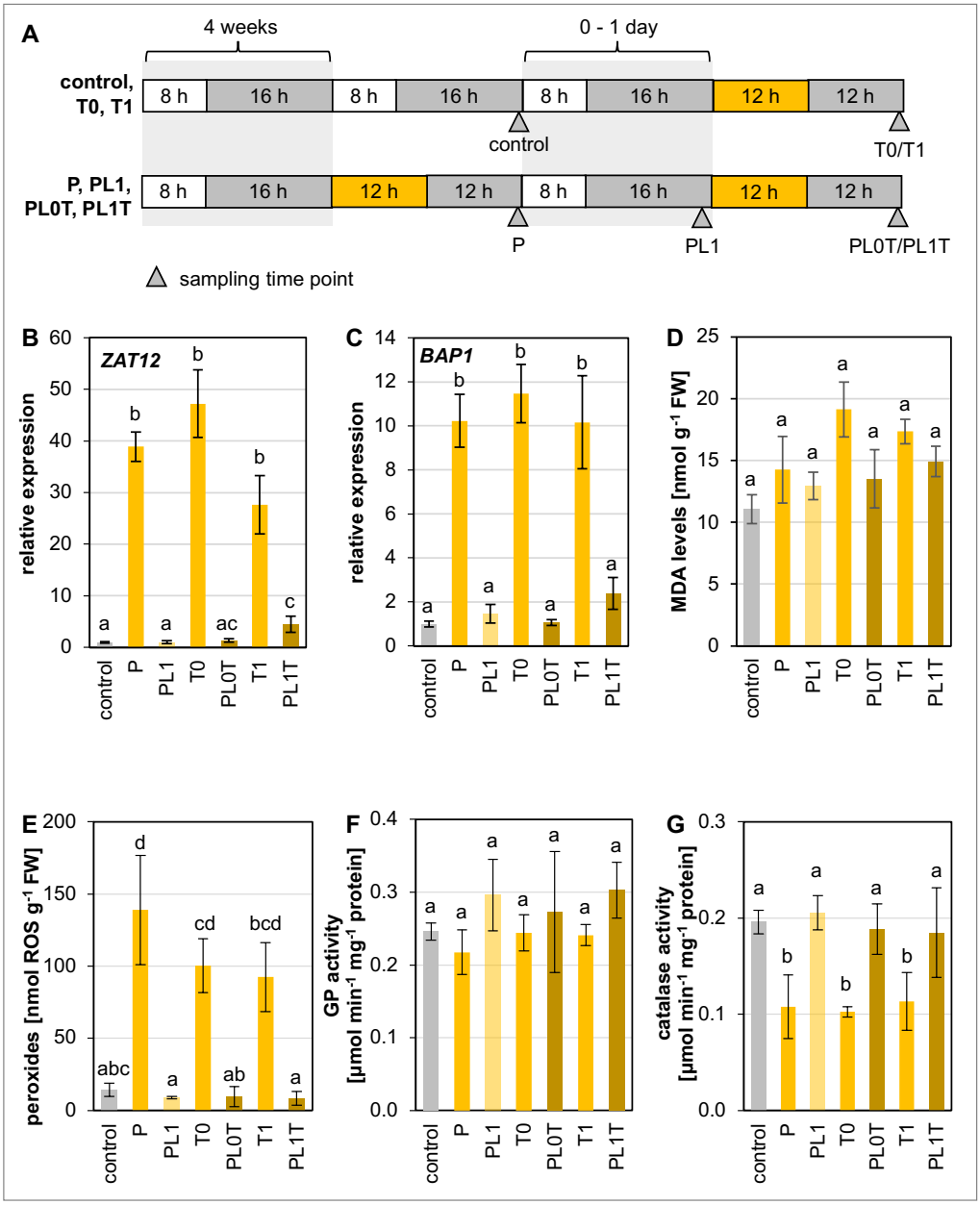

Figure 24. A 4 h-PL period primes plant responses to future PL periods.

(A) Schematic overview of experimental conditions used in (B-G). Arabidopsis plants were grown under SD condition for four weeks (control) before exposure to a 4 h-PL period as priming stimulus (P) or triggering stimulus directly after the P stimulus (T0) or one day later (T1). Directly after the prolonged light treatment (PL0) or after one day under SD condition (PL1), primed plants were exposed to a second prolonged light period of 4 hours (PL0T, PL1T). Leaf samples were collected at the end of the night following the respective stress treatments (arrowheads). White, light period; grey, dark period. **(B, C)** Relative expression of photoperiod stress marker genes (B) *ZAT12* and (C) *BAP1*. Expression levels of the controls were set to 1. **(D)** MDA levels. **(E)** Peroxide levels. **(F, G)** Enzyme activities of (F) guaiacol peroxidase (GP) and (G) catalase (CAT). Data are mean values (n = 4 ± SE). Experiments were run three times independently. Letters indicate significantly different groups (one-way ANOVA with post-hoc Tukey test or pairwise *t*-test; *p*-value ≤ 0.05). FW, fresh weight.

3.2.2 A PL period up 2 hours does not prime the response to a future PL period

To investigate if shorter PL treatments (less than 4 hours), which normally do not induce changes in the expression of photoperiod stress marker genes *ZAT12* and *BAP1* (Abuelsoud *et al.*, 2020), still prime plant responses for a 4 h-PL period, Arabidopsis plants were exposed to PL periods of 0.5 h, 1 h and 2 h representing the priming stimulus (**Figure 25**). Analysis of *ZAT12* and *BAP1* expression revealed that their induction was only suppressed when plants were exposed to a 4 h-PL period as priming stimulus but not when they were pre-treated with a PL period of 0.5 h, 1 h or 2 h (**Figure 25B-C**). This indicates that a PL period up to 2 h does not prime Arabidopsis. Thus, the photoperiod stress needs to induce a significant response to be memorized.

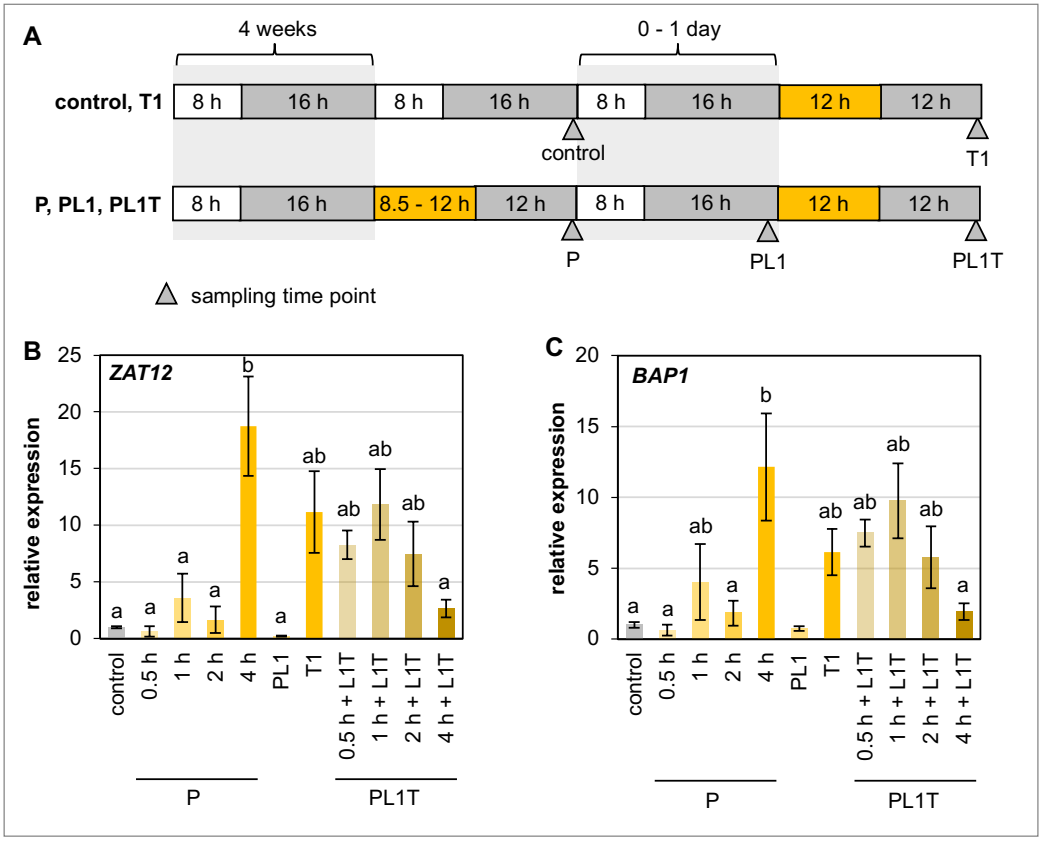

Figure 25. A PL period up to 2 h does not prime plants for future photoperiod stress.

(A) Schematic overview of experimental conditions used in **(B-C)**. Arabidopsis plants were grown under SD condition for four weeks (control) before exposure to a PL period of 0.5 h to 4 h (priming stimulus, P) or 4 h (triggering stimulus, T1). After one day under SD condition (PL1), primed plants were exposed to a second prolonged light period of 4 hours (P + L1T). Leaf samples were collected at the end of the night following the respective stress treatments (arrowheads). White, light period; grey, dark period. **(B, C)** Relative expression of photoperiod stress marker genes (B) *ZAT12* and (C) *BAP1*. Expression levels of the controls were set to 1. Data are mean values (n = 4 ± SE). Experiments were run three times independently. Letters indicate significantly different groups (one-way ANOVA with post-hoc Tukey test; *p*-value ≤ 0.05).

3.2.3 Responsiveness of Arabidopsis plants to a 4 h-PL period is dependent on the developmental phase

The ability to respond to photoperiods depends on the developmental phase of Arabidopsis (Matsoukas, 2014). To study if also the responsiveness to a PL period is influenced by the developmental phase,

Arabidopsis plants of different ages were exposed to a 4 h-PL. The experiment included plants that were grown for 14 days, 21 days, 28 days, 35 days, and 42 days after germination (DAG) under SD. Analysis of peroxide content revealed that a 4 h-PL period (P) increased peroxide levels at 21 DAG, 28 DAG and 35 DAG (**Figure 26A**). Interestingly, younger plants (14 DAG) or older plants (42 DAG) did not respond to a 4 h-PL period (**Figure 26A**). Similarly, the photoperiod stress-responsive genes *ZAT12* and *BAP1* were most strongly induced at 28 DAG (**Figure 26C-D**). The results suggest that the responsiveness of Arabidopsis plants to oxidative stress-like responses is influenced by their developmental phase. How plants sense a sudden PL period and if sensing itself depends on the developmental phase, remain to be investigated.

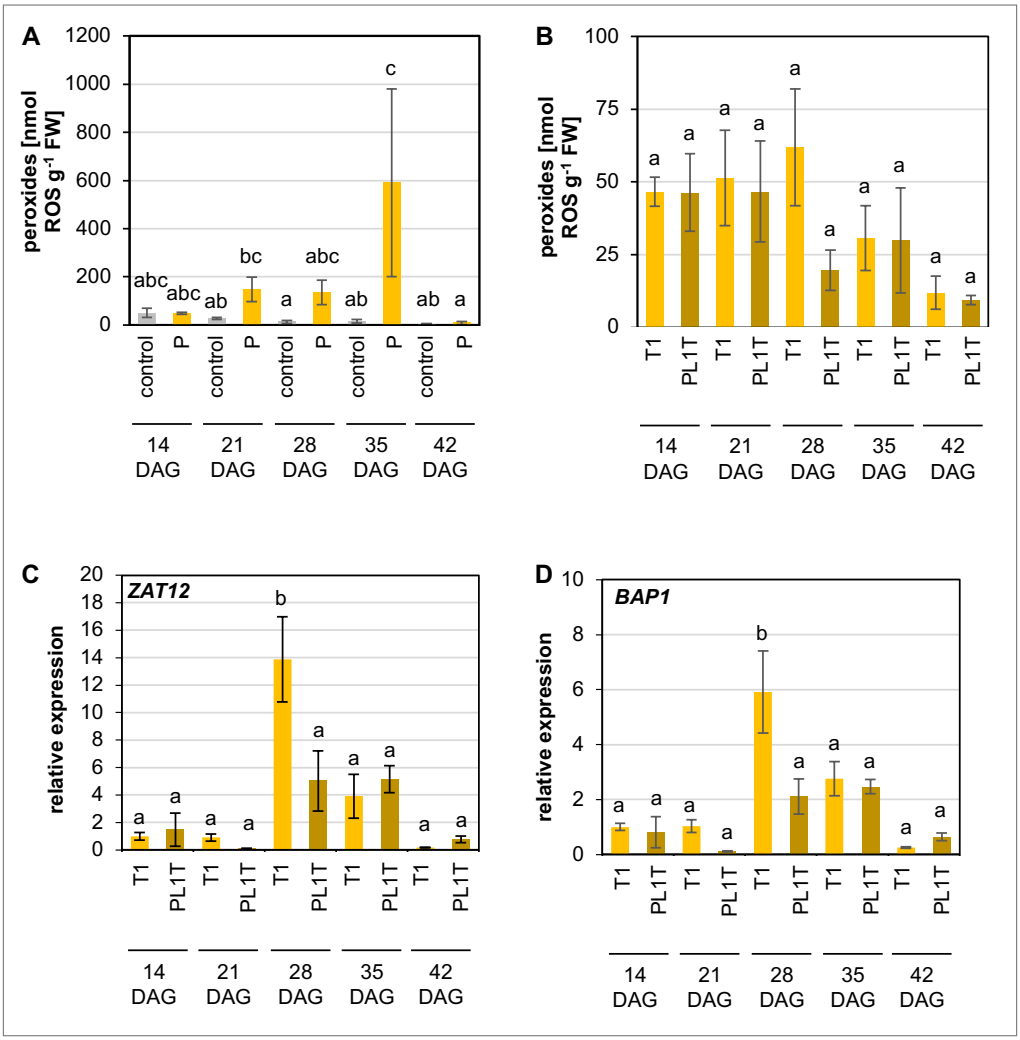

Figure 26. Responsiveness to 4 h-PL periods is dependent on the developmental phase.

Arabidopsis plants were grown under SD condition for 14 days after germination (DAG), 21 DAG, 28 DAG, 35 DAG or 42 DAG (control) before exposure to a 4 h-PL period as priming stimulus (P) or triggering stimulus (T1). After one day under SD condition, primed plants were exposed to a second PL period of 4 hours (PL1T). Samples were collected at the end of the night following the respective treatments. The same experimental setup as shown in **Figure 24** was used. **(A, B)** Relative expression of photoperiod stress marker genes (A) *ZAT12* and (B) *BAP1*. Expression levels in T1 plants 14 DAG were set to 1. Data are mean values (n = 4 ± SE). Experiments were run at least two times independently. **(C, D)** Peroxide levels. Data are mean values (n = 4 ± SE). Letters indicate significantly different groups (two-way ANOVA with post-hoc Tukey test; $p \leq 0.05$). FW, fresh weight.

To investigate if priming by photoperiod stress is influenced by the developmental phase, Arabidopsis plants of different ages were exposed to 4 h-PL periods as priming and triggering. Peroxide accumulation was suppressed only at 28 DAG in plants primed and triggered with a 4 h-PL period, while this was not observed for younger or older plants (**Figure 26B**). Analysis of the expression of the photoperiod stress-responsive *ZAT12* and *BAP1* resulted in similar observations: Both genes were suppressed in response to a 4 h-PL in plants at 21 DAG and 28 DAG when primed; this, however, was not observed not in younger or older plants (**Figure 26C-D**) indicating an absence of priming.

A possible explanation for our results might be that Arabidopsis plants at 14 DAG are still in their juvenile developmental phase and thus unresponsive to photoperiods as reported by Matsoukas (2014). Likewise, plants in the reproductive developmental phase are also responsive to photoperiods, while plants in adult phase within the vegetative phase are unresponsive to photoperiods (Matsoukas, 2014). Because of this, it was hypothesized that the loss of sensitivity to and primability by a 4 h-PL in Arabidopsis plants, is a result of their starting transition to the reproductive phase. Thus, expression of the *SENESCENCE ASSOCIATED GENE12* (*SAG12*), which is expressed in senescing tissues thereby displaying the progression of the reproductive phase, was analysed in Arabidopsis plants at 35 DAG, 37 DAG, and 39 DAG (**Figure 27A**).

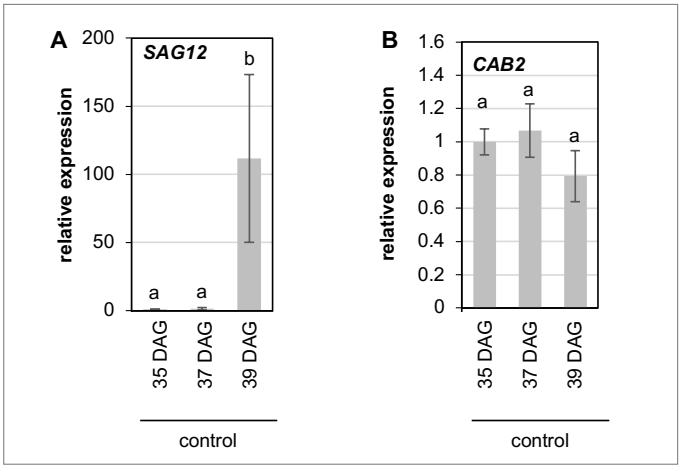

Figure 27. Loss of photoperiod stress responsiveness and priming coincides with progression in plant age.

Arabidopsis plants were grown under SD condition for 35 days after germination (DAG), 37 DAG, or 39 DAG. Leaf tips of plants were harvested. **(A-B)** Relative expression of (A) senescence marker gene *SAG12* and (B) photosynthesis gene *CAB2*. Expression levels at 35 DAG were set to 1. Data are mean values (n > 3 ± SE). Letters indicate significantly different groups (one-way ANOVA with post-hoc Tukey test; $p \leq 0.05$).

In addition, expression of the *CHLOROPHYLL A/B-BINDING PROTEIN 2* (*CAB2*), encoding a component of the light harvesting complex pf photosystem II, was investigated in the same setup (**Figure 27B**). The experiment revealed that *SAG12* expression strongly increased, while *CAB2* expression decreased when Arabidopsis plants progressed in their age. Thus, it was concluded that the reproductive phase progressed in these plants, which might be the reason for the loss of responsiveness to and primability by a 4 h-PL.

3.2.4 Priming by a 4 h-PL period is memorized by Arabidopsis for several days

Above (**section 3.2.1**), it was shown that Arabidopsis plants are able to memorize a 4 h-PL period for at least one day. To investigate if plants can memorize a previous PL period for longer durations, Arabidopsis plants were exposed to a 4 h-PL period as priming and triggering stimulus and grown under SD for up to ten days (PL0T - PL10T) between the two stimuli. The experimental setup is shown in **Figure 31**. Expression analysis of *ZAT12* and *BAP1* revealed that both genes were induced in response to a single 4 h-PL (P, T0 - T10) (**Figure 28A-F**). This induction was suppressed in Arabidopsis pre-treated with a 4 h-PL period and kept for up to seven days under SD before exposure to a second PL period (PL0T - PL7T). A longer lag phase, such as ten days between priming and triggering (PL10T), did not suppress the expression induction of *ZAT12* and *BAP1* anymore (**Figure 28A-F**). To avoid that the analysed plants entered the developmental phase in which they are not responsive to photoperiod stress (**section 3.2.3**), the experiment was in parts performed with younger plants primed at 21 or 24 DAG (instead of the standard experimental start at 28 DAGs). Taken together, the results indicate that a previous 4 h-PL period is memorized by transcription of *ZAT12* and *BAP1* for up to seven days in Arabidopsis plants.

To explore whether a 4 h-PL is memorized by changes in peroxide content for a similar duration, the peroxide content was determined. Peroxides accumulated in response to a single 4 h-PL period; however, accumulation of peroxides was suppressed in plants pre-treated with a 4 h-PL period and kept for up to five days under SD before exposure to a similar PL period (PL0T - PL5T) (**Figure 28G**). In response to longer lag phases (PL7T, PL10T), Arabidopsis plants accumulated similar levels of peroxides as plants treated only once with the 4 h-PL period (T7, T10) (**Figure 28H-I**). This suggests that Arabidopsis plants memorize a previous 4 h-PL for five days by changes in peroxide content demonstrating that the memory reflected by changes in peroxide content lasts shorter than the memory by transcription of photoperiod stress marker genes.

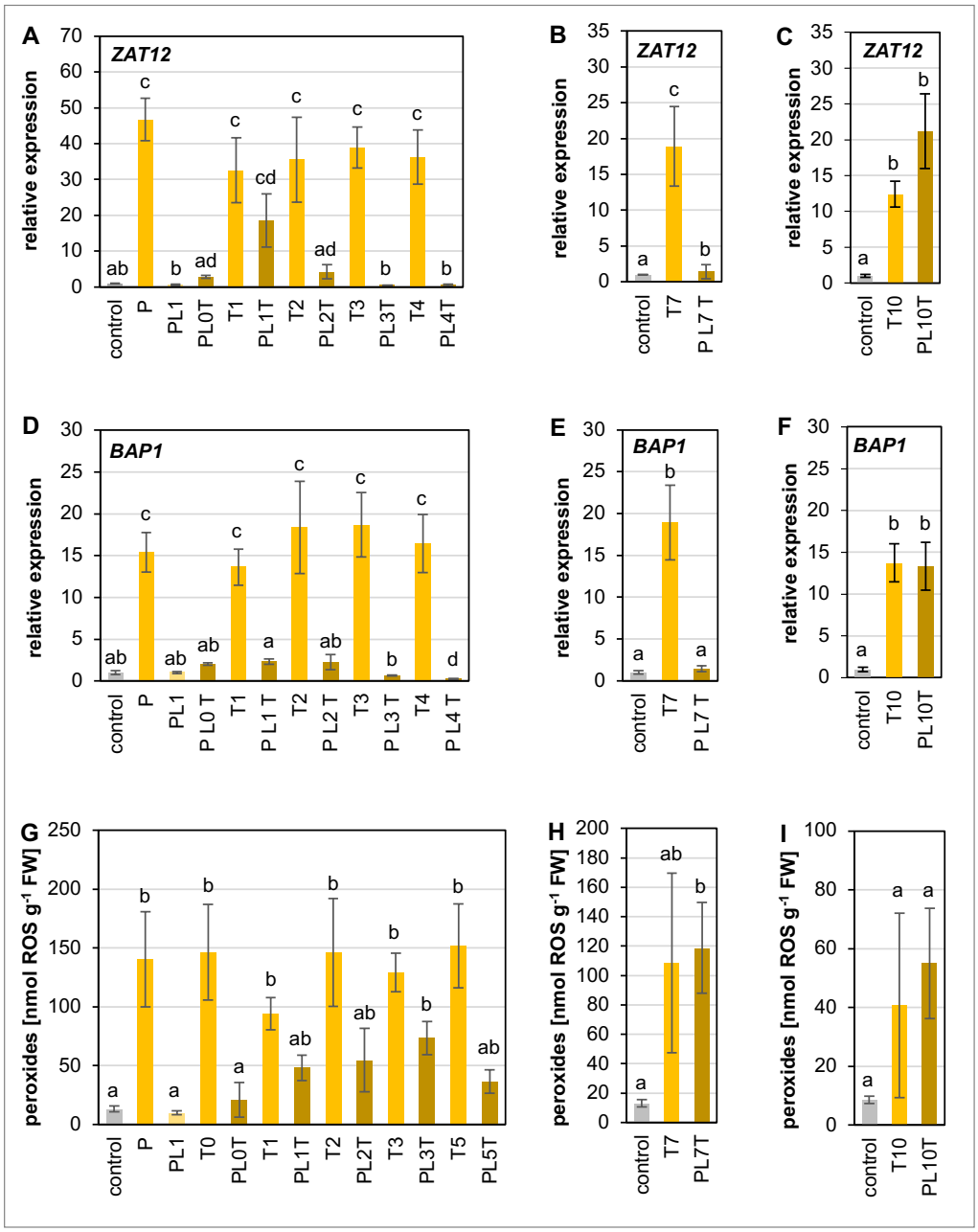

Figure 28. Arabidopsis memorizes priming by photoperiod stress.

Arabidopsis plants were grown under SD condition (control) before exposure to a 4 h-PL period as priming stimulus (P) or triggering stimulus (T0, T1, T2, T3, T4, T5, T7, T10). Directly after the prolonged light treatment (PL0) or after one day under SD condition (PL1), the primed plants were exposed to a second PL period of 4 hours (PL0T, PL1T, PL2T, PL3T, PL4T, PL5T, PL7T, PL10T). Leaf samples were collected at the end of the night following the respective stress treatments. **(A-C)** Peroxide levels. **(D-I)** Relative expression of photoperiod stress marker genes (D-F) *ZAT12* and (G-I) *BAP1*. Expression levels of the controls were set to 1. Data are mean values (n = 4 ± SE). Experiments were run three times independently. Letters indicate significantly different groups (one-way ANOVA with post-hoc Tukey test or pairwise *t*-test; *p*-value ≤ 0.05). FW, fresh weight.

3.2.5 Photoperiod stress-sensitive mutants *ahk2 ahk3* and *cca1 lhy* are primed by a 4 h-PL period, but have a shorter memory than WT Arabidopsis plants

The cytokinin receptor mutant *ahk2 ahk3* and the circadian clock mutant *cca1 lhy* have been described to be particularly sensitive to photoperiod stress (Nitschke *et al.*, 2016). Using these mutants, it was investigated if an enhanced sensitivity to photoperiod stress influences the priming including the length of the memory. Exposure of the mutants to a single 4 h-PL resulted in accumulation of peroxides in both double mutants (**Figure 29** and **Figure 30**). The mutants accumulated higher total levels of peroxides than WT Arabidopsis plants in response to a 4 h-PL, thereby supporting the stronger sensitivity of *ahk2 ahk3* and *cca1 lhy* mutants described by Nitschke *et al.* (2016). To investigate the memory of the mutants, a similar experimental setup as for WT plants (**section 3.2.4**) was used, meaning that the mutants were exposed to 4 h-PL periods as priming and triggering with an up to seven days lag phase in-between both treatments (PL0T - PL7T). Measurements indicated that the accumulation of peroxides in response to a 4 h-PL period was suppressed in *ahk2 ahk3* and *cca1 lhy* primed by a similar 4 h-PL period and with a lag phase of three to four days between priming and triggering (PL3T, PL4T, **Figure 29A** and **Figure 30A**). A longer lag phase did not suppress the accumulation of peroxides anymore. Although the data indicated a higher sensitivity of *ahk2 ahk3* and *cca1 lhy* mutants than WT plants, the results suggested a shorter memory in the mutants than in WT. This was further supported by analysis of *ZAT12* and *BAP1* expression; the induction of both genes in response to a 4 h-PL was suppressed in *ahk2 ahk3* and *cca1 lhy* mutants when a lag phase of three to four days between priming and triggering (PL4T, PL3T) was applied. In response to longer lag phases, induction of *ZAT12* and *BAP1* was not suppressed anymore in the mutants (**Figure 29B-E** and **Figure 30B-E**). Taken together, the experiments demonstrate that *ahk2 ahk3* and *cca1 lhy* mutants memorize a previous 4 h-PL by changes in peroxide content and transcription of photoperiod stress marker genes less long than WT plants. Moreover, the results indicate that a perception of cytokinin as well as a functional circadian clock are required for maintaining the photoperiod stress-induced priming and memory in Arabidopsis.

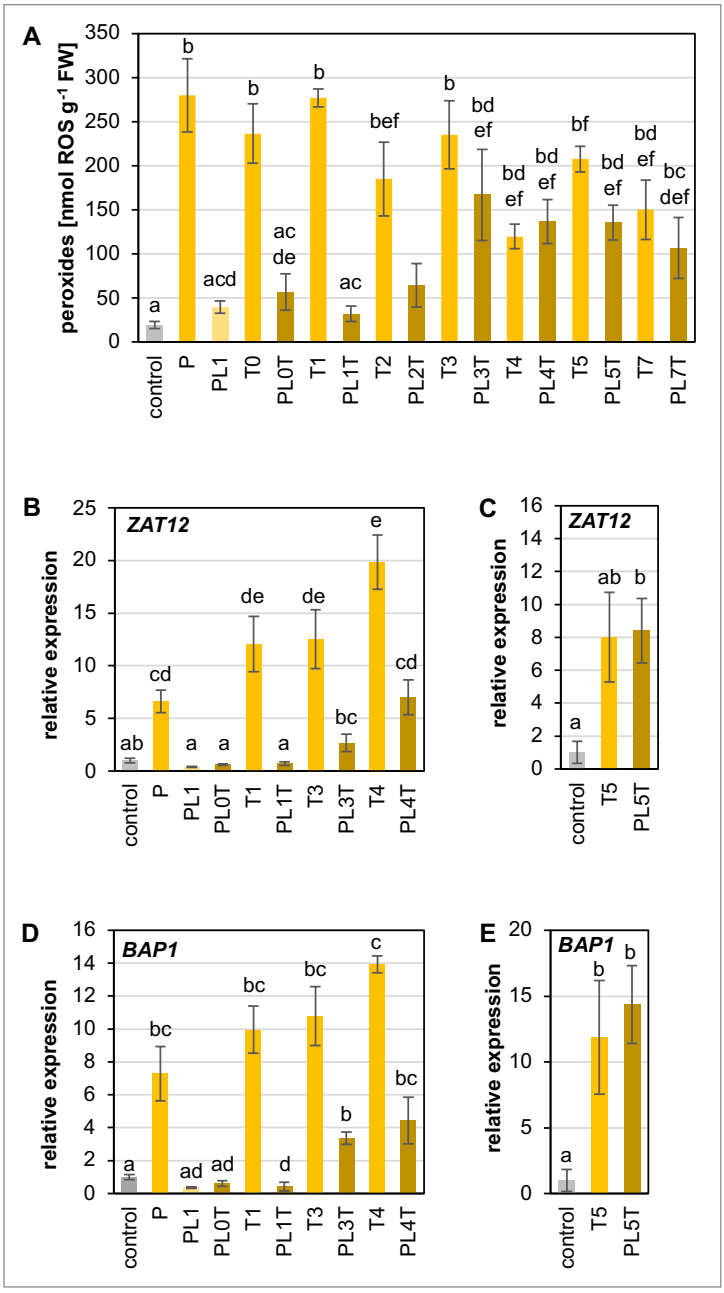

Figure 29. Photoperiod stress-sensitive mutant *ahk2 ahk3* has a shorter memory than WT plants.

Photoperiod stress-sensitive mutant *ahk2 ahk3* was grown under SD condition for four weeks (control) before exposure to a 4 h-PL period as priming stimulus (P) or triggering stimulus (T1, T3, T4, T5, T7). After one day at SD condition (PL1), the primed plants were exposed to a second PL period of 4 hours (PL1T, PL3T, PL4T, PL5T, PL7T). Leaf samples were collected at the end of the night following the respective stress treatments. **(A)** Peroxide levels. **(B-E)** Relative expression of photoperiod stress marker genes (B-C) *ZAT12* and (D-E) *BAP1*. Expression levels of the controls were set to 1. Data are mean values (n = 4 ± SE). Experiments were run three times independently. Letters indicate statistical significantly different groups (one-way ANOVA with post-hoc Tukey test; *p*-value ≤ 0.05). FW, fresh weight.

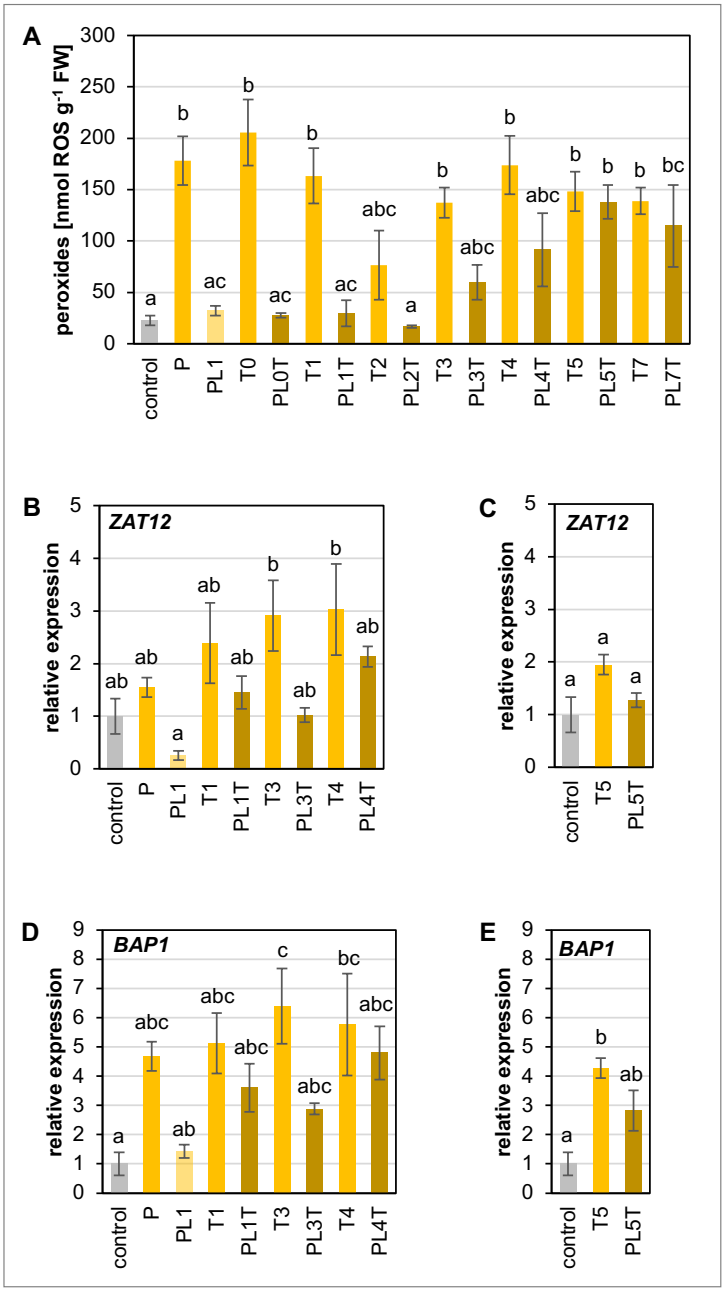

Figure 30. Photoperiod stress-sensitive mutant *cca1 lhy* has a shorter memory than WT plants.

Photoperiod stress-sensitive mutant *cca1 lhy* was grown under SD condition for four weeks (control) before exposure to a 4 h-PL period as priming stimulus (P) or triggering stimulus (T1, T3, T4, T5, T7). After one day at SD condition (PL1), the primed plants were exposed to a second PL period of 4 hours (PL1T, PL3T, PL4T, PL5T, PL7T). Leaf samples were collected at the end of the night following the respective stress treatments. **(A)** Peroxide levels. **(B-E)** Relative expression of photoperiod stress marker genes (B-C) *ZAT12* and (D-E) *BAP1*. Expression levels of the controls were set to 1. Data are mean values (n = 4 ± SE). Experiments were run three times independently. Letters indicate statistical significantly different groups (one-way ANOVA with post-hoc Tukey test; *p*-value ≤ 0.05). FW, fresh weight.

3.2.6 Transcriptomic changes during *cis*-priming by photoperiod stress

Changes of the transcript abundance in WT plants in response to a 4 h-PL periods as priming and triggering stimulus were investigated by RNA-seq. **Figure 31** outlines the experimental setup underlying this study aiming to investigate how many and which genes are regulated during *cis*-priming by photoperiod stress.

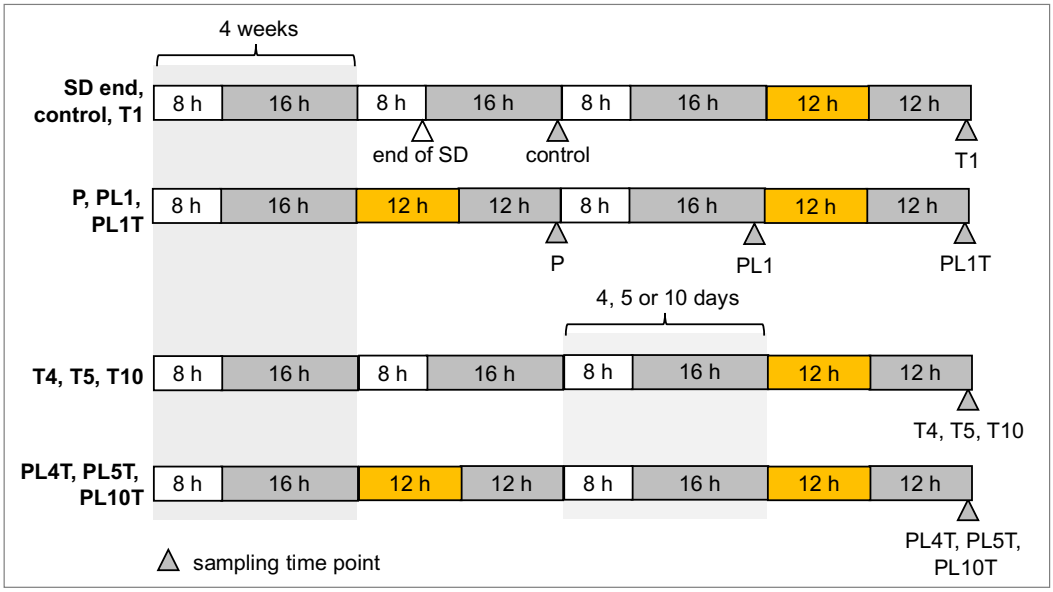

Figure 31. Setup for the RNA-seq experiment exploring *cis*-priming.

Schematic overview of the experimental conditions used in the *cis*-priming analysis. SD-grown WT Arabidopsis plants were exposed to a 4 h-PL period as priming (P) or triggering stimulus (T1, T4, T5, T10), or they remained under SD condition as a control. After one day at SD condition (PL1), primed plants were exposed to a second 4 h-PL period (PL1T). In addition, plants were treated with a 4 h-PL period and remained for 4, 5 or 10 days under standard SD condition before exposed to a second 4 h-PL period (PL4T, PL5T, PL10T). The only-triggered plants (T1, T4, T5 and T10) received the triggering stimulus at the same moment as the primed and triggered plants (PL4T, PL5T and PL10T). Sampling was performed at the end of the night following the light treatments. To filter out time-of-the-day-regulated genes from the analysis, additional plant material was harvested at the end of the standard SD period (indicated by a white triangle). White, light period; grey, dark period; orange, light period that is longer than a normal SD.

3.2.6.1 Exploratory data analysis of the *cis*-priming RNA-seq experiment

To evaluate the RNA-seq data, a PCA was performed. The PCA showed that PC1 (x-axis) accounted for 82%, and PC2 (y-axis) accounted for additional 6% of the total variation in the RNA-seq dataset (**Figure 32**). The biological replicates clustered together (**Figure 32**) suggesting similarities among the biological replicates and, therefore, reproducibility of the expression data. Samples harvested at the end of a SD clustered together (**Figure 32**, cluster 1). Control samples as well as samples treated with a 4 h-PL period but returned to SD for one day (PL1) clustered together (**Figure 32,** cluster 2). This indicates that after a one-day lag phase, regulated transcripts return to the control state. Samples treated with one 4 h-PL period (P, T1, T4, T5 and T10) or two 4 h-PL periods (PL1T, PL4T, PL5T and PL10T) clustered together (**Figure 32**, cluster 3) indicating that there are no major differences between primed and non-primed samples. The PCA indicates that the RNA-seq data were qualified for further analysis.

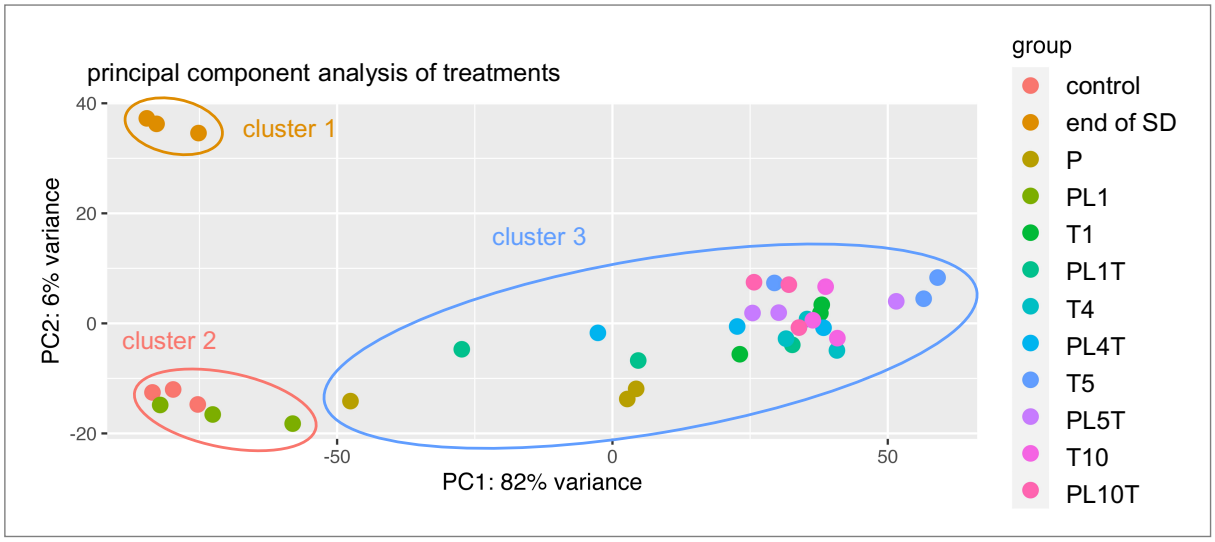

Figure 32. PCA for *cis*-priming experiment.

PCA of the RNA-seq dataset including WT Arabidopsis plants exposed to a 4 h-PL period as priming (P, PL1) and/or triggering (T1, T4, T5, T10) with one to ten days lag phase (PL1T, PL4T, PL5T, PL10T) (three biological replicates per treatment).

To get more insights into the RNA-seq dataset, QT clustering was performed. QT clustering revealed 17 clusters and one further cluster with unassigned genes (**Figure 33**). The clusters were analysed by GO term enrichment analysis (**Figure 34, Figure 35** and **Figure 36**).

Cluster 1 comprised 27% of all regulated genes (2233 genes, **Figure 33**) showing an upregulation under control, at the end of a SD, in P and PL1, in parts in PL1T and PL4T, and a downregulation under the remaining treatments. According to GO term analysis, genes involved in photosynthesis, light harvesting, responses to low light, phototropism and water and fluid transport are enriched in this cluster (**Figure 34A**).

Cluster 2 contained 25% of all regulated genes (2012 genes, **Figure 33**) showing a downregulation under control, at the end of a SD, in PL1 and PL1T, and an upregulation under the remaining treatments. Induction of genes by a 4 h-PL period (P) that is followed by a downregulation in response to a SD (PL1) suggests that these genes were only transiently induced by a PL period. The lower amplitude of genes exposed to two PL periods with one day lag phase in-between (PL1T) compared to amplitudes in response to a single 4 h-PL period (P, T1) suggests that pre-treatment with a PL period affects the expression of genes in this cluster when exposed to a second PL period. Due to their altered expression in response to PL1T, genes in this cluster may serve as marker genes for memory, such as the photoperiod stress marker genes *ZAT12* and *BAP1* (**section 3.2.4**). The lower amplitude of genes in this cluster in response to two PL periods is not visible anymore when the lag phase increased to five days (PL5T) or became even longer (PL10T, **Figure 33**), suggesting a loss of memory when the lag phase increases. Cluster 2 comprised genes that are responsive to molecules of bacterial origin, involved in toxin catabolism or metabolism, cellular responses to hypoxia or (decreased) oxygen levels, and the responses to SA (**Figure 34B**).

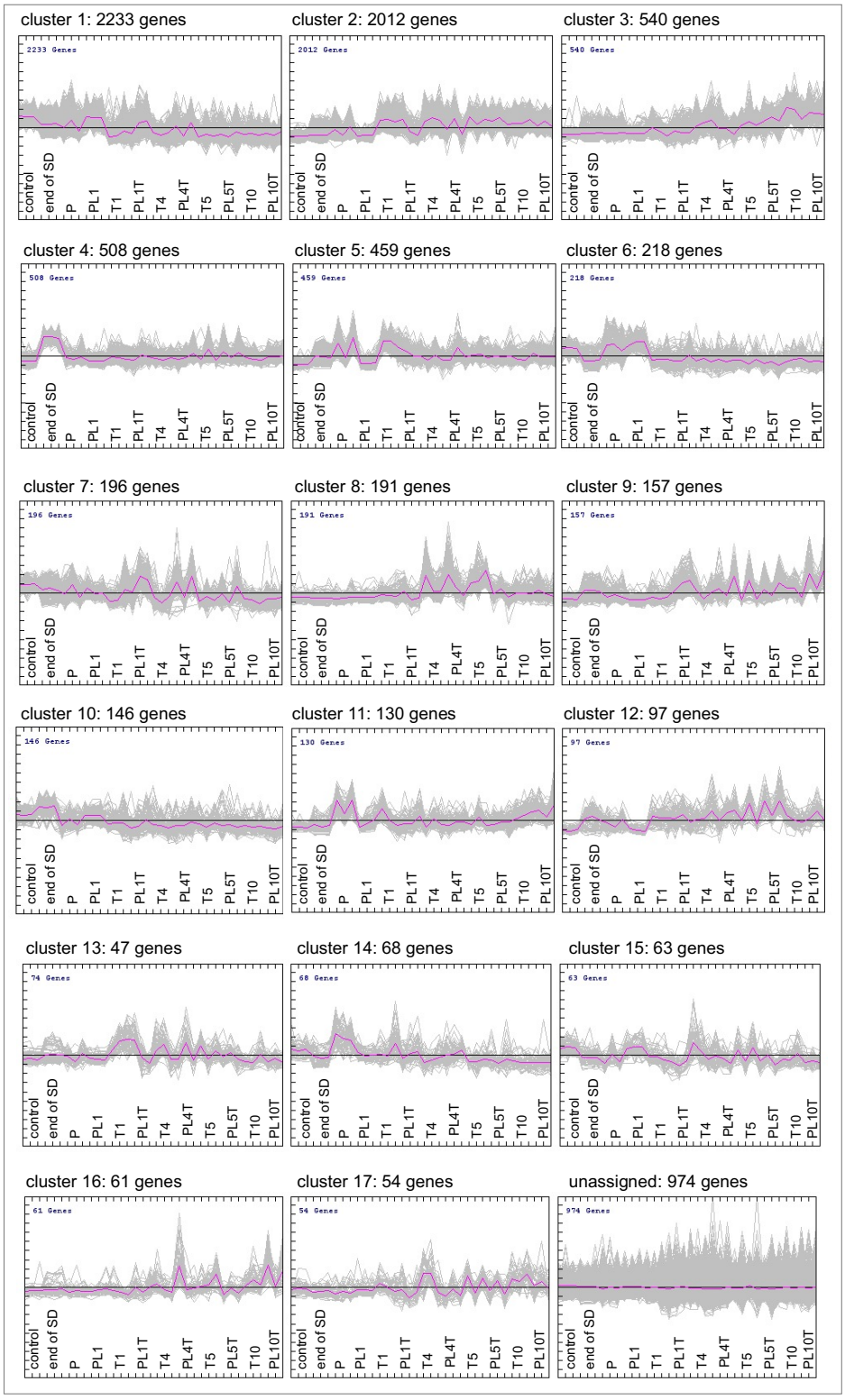

Figure 33. QT clustering for the *cis*-priming experiment.

QT clustering of the RNA-seq dataset from WT Arabidopsis plants under control conditions, at the end of an SD growth period, or WT Arabidopsis plants exposed to a 4 h-PL period as priming (P, PL1) and/or triggering (T1, T4, T5, T10) with a lag phase of one to ten days (PL1T, PL4T, PL5T, PL10T). Purple line, average levels of FPKM values of genes in the respective clusters.

Cluster 3 contained 7% of all regulated genes (540 genes, **Figure 33**) showing an upregulation in response to one or two 4 h-PL periods in older plants (T4, T5, T10, PL5T, PL10T) and a downregulation under the remaining treatments. This suggests that genes exist induced by PL periods in plants with a progressing developmental state. According to GO term analysis, genes in this cluster are associated to metabolic processes (indole glucosinolate metabolism, ketone metabolism), responses to molecules of bacterial origin, (cellular) responses to hypoxia or (decreased) oxygen levels (**Figure 34C**).

Cluster 4 comprised 6% of all regulated genes (508 genes, **Figure 33**) showing an upregulation at the end of a SD, and a downregulation under the remaining treatments. Genes in this cluster are regulated in response to the time of the day, but not, or only slightly, in response to PL periods. Genes in this cluster are assigned to DNA replication signalling and initiation, (mitochondrial) (m)RNA modification, and RNA metabolic processes (**Figure 34D**).

Cluster 5 contained 6% of all regulated genes (459 genes, **Figure 33**) showing an upregulation in response to a single 4 h-PL period (P, T1). A one-day lag phase is sufficient to downregulate their induced expression. The lower amplitude of the expression in response to PL1T compared to P or T1 indicates that the pre-treatment with a 4 h-PL period affects the expression of respective genes when exposed to a second similar PL, period after one SD. According to GO term analysis, genes in this cluster are related to protein refolding, (cellular) responses to unfolded proteins (including responses to ER unfolded proteins), chaperone-mediated protein refolding, and protein targeting/localization to the mitochondrion (**Figure 34E**).

Three percent of all regulated genes (218 genes) are included in cluster 6 (**Figure 33**) showing a slight upregulation under control, a stronger upregulation in response to P and PL1, and a downregulation under the remaining treatments. Genes in this cluster are assigned to responses to organic cyclic compounds, growth, responses to (abiotic) stimuli and chemicals (**Figure 34F**).

Clusters 7 to 11 each contained around 2% of all regulated genes. Genes in cluster 7 (196 genes) showing a strong upregulation in response to PL1T and PL4T that was not or only in parts noticeable in response to single 4 h-PL periods (P, T1, T4, **Figure 33**), thus indicating priming. According to GO term analysis, cluster 7 contained genes related to cell division (**Figure 35A**). Genes in cluster 8 (191 genes) showing an upregulation in T4, PL4T, and T5 (**Figure 33**) were assigned to responses to JA, fatty acids, wounding, cellular responses to extracellular stimuli, water deprivation and water, and ABA (**Figure 35B**). Genes in cluster 9 (157 genes) showing an upregulation in response to two PL periods (e.g. PL1T, **Figure 33**) were assigned to anthocyanin-containing compound biosynthetic or metabolic processes, and flavonoid/flavonol/flavone biosynthetic and metabolic processes (**Figure 35C**). Cluster 10 comprised genes upregulated under control conditions, at the end of a SD and in response to PL1, while the genes were downregulated in response to the remaining conditions (**Figure 33**). Genes in this cluster were not assigned to GO terms. The 130 genes in cluster 11 showing an upregulation in P, T1, T10 and PL10T (**Figure 33**) were responsive to molecules of bacterial origin or SA, connected to SAR, involved in regulation of defence responses, including responses to bacteria or fungi, regulate responses to stresses or to organic cyclic compounds (**Figure 35D**).

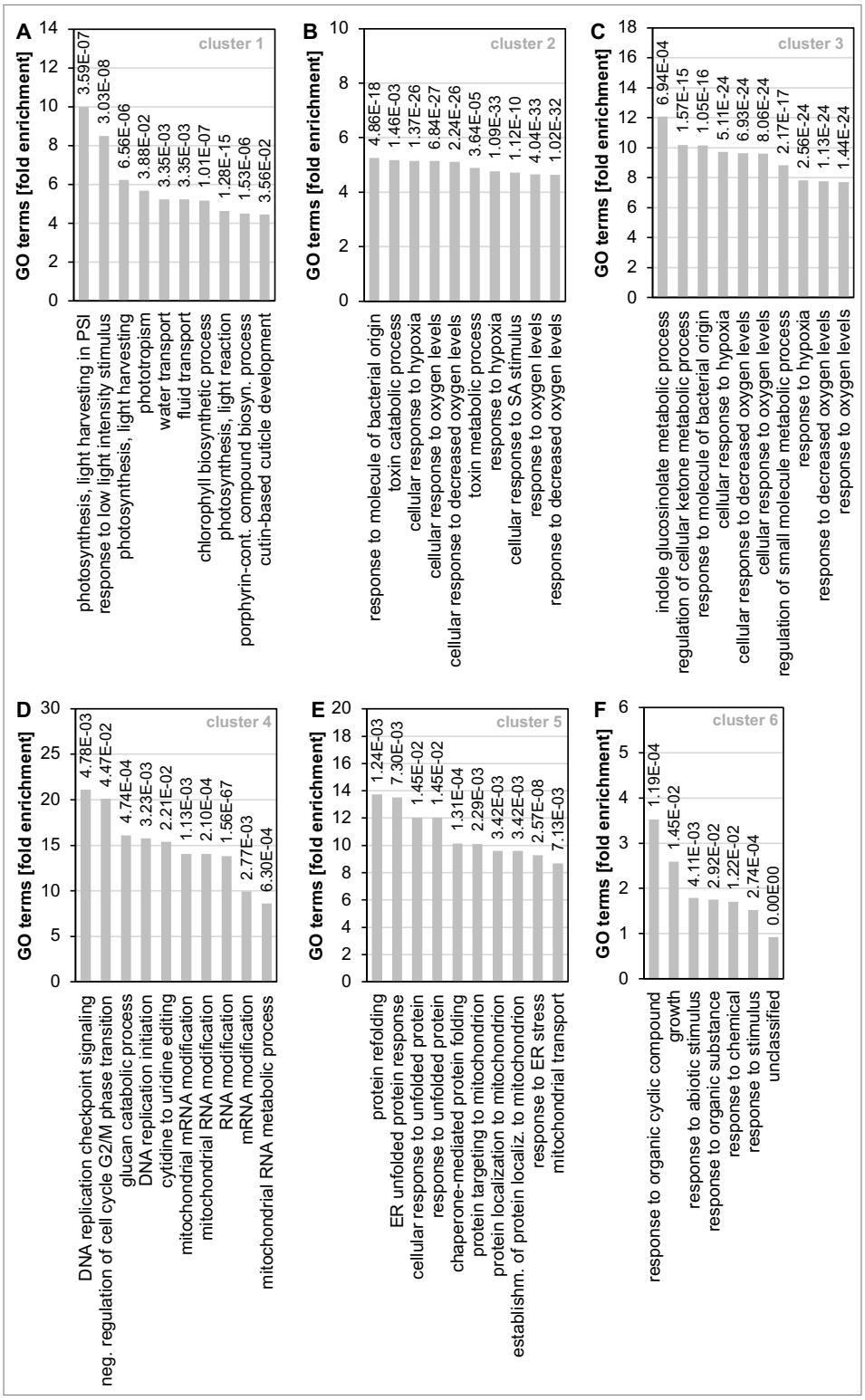

Figure 34. GO term analysis of QT clusters 1 to 6 of the *cis*-priming experiment.

GO term analysis of QT clusters 1 to 6 (panels A to F). The top ten GO terms are shown. Numbers above the bars represent respective *p*-values.

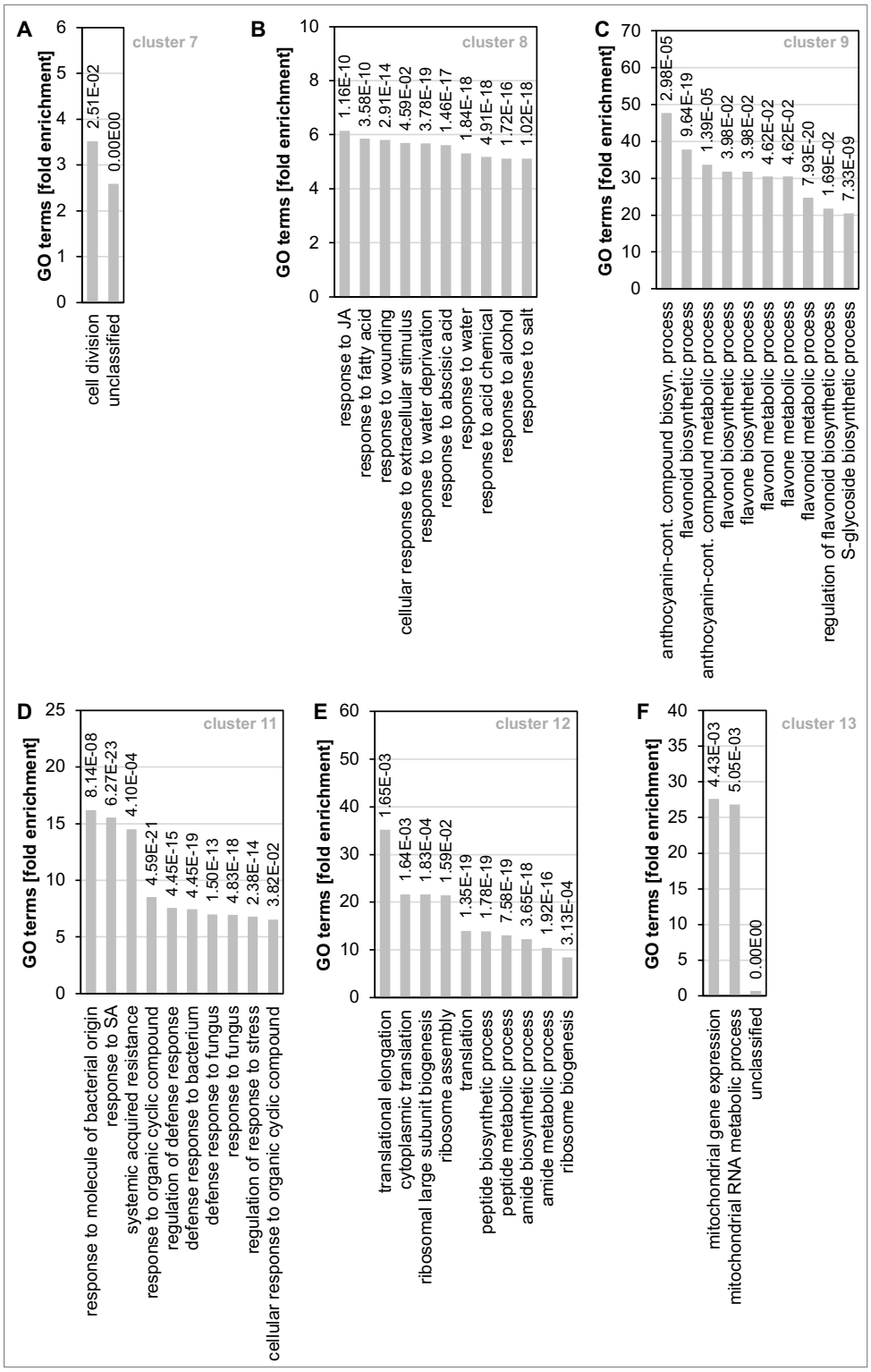

Figure 35. GO term analysis of QT clusters 7 to 13 of the *cis*-priming experiment.

GO term analysis of QT clusters 7 to 13 (panels A to F). The top ten GO terms are shown. Numbers above the bars represent respective *p*-values.

Clusters 12 to 17 each contained around 1% of all regulated genes (cluster 12: 97 genes; cluster 13: 74 genes; cluster 14: 68 genes; cluster 15: 63 genes; cluster 16: 61 genes; cluster 17: 54 genes; **Figure 33**). GO term analyses of these clusters are displayed in **Figure 35E-F** and **Figure 36**. Due to the low number of genes in these clusters, their regulation and associated GO terms are not described in detail.

In total, 12% of the genes (974 genes) were not assigned to clusters (**Figure 33**). Several of the unassigned genes were involved in *S*-glucoside or glucosinolate biosynthesis and metabolism (**Figure 36C**).

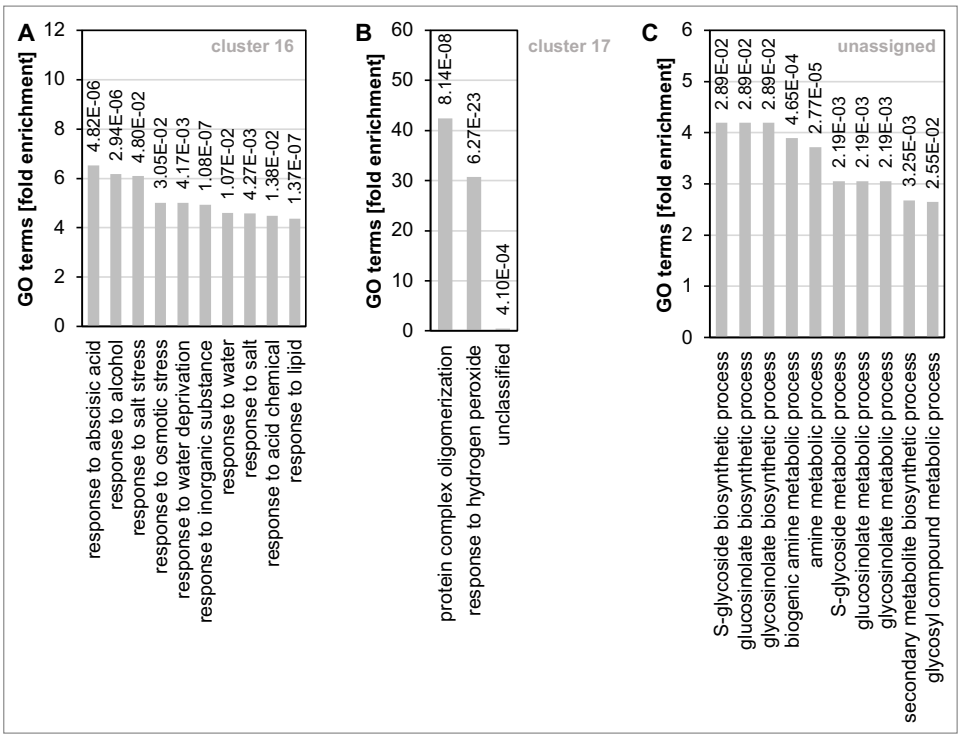

Figure 36. GO term analysis of QT clusters 16, 17 and unassigned genes of the *cis*-priming experiment.

GO term analysis of QT clusters 16, 17 (panels A, B) and unassigned genes (panel C). The top ten GO terms are shown. Numbers above the bars represent respective *p*-values.

3.2.6.2 Transcriptomic changes during *cis*-priming by photoperiod stress

In total, 13991 genes were differentially expressed (DEGs) in a treatment-dependent manner in the RNA-seq *cis*-priming dataset (as calculated by Bonferroni-correction, *p*-value ≤ 0.05). Pairwise comparisons were performed to get deeper insights into the DEGs regulated by one 4 h-PL period (P, T1), in combination with a lag phase (PL1), or by two PL periods (PL1T) in comparison to control. These comparisons revealed that 2931 genes were differentially expressed in response to priming by a 4 h-PL period (P *vs.* control) and 7035 genes were differentially expressed by triggering (T1 *vs.* control). In total 144 genes were differentially regulated in primed plants after one SD (PL1 *vs.* control). 2501 genes were differentially expressed in photoperiod stress primed-and-triggered plants (PL1T *vs.* control). This shows that the number of DEGs decreased when PL-treated plants were returned to SD; however, part of the DEGs were still differentially regulated. Notably, the number of DEGs in response to a photoperiod priming or triggering event resulted in a large difference.

The top 20 up- and downregulated genes are summarized in **Supplementary Table S13** to **Supplementary Table S20**. Among the top 20 upregulated genes in response to priming (P *vs.* control) and triggering (T1 *vs.* control) are genes involved in plant defence responses, such as *FMO1* and

RESPIRATORY BURST OXIDASE HOMOLOG C (*RBOHC*), genes responsive to hypoxia, such as *ZAT8* and *ETHYLENE RESPONSE FACTOR71* (*ERF71*), and genes encoding members of the *CYTOCHROME P450* gene family, such as *CYP76C5* and *CYP81G1* (**Supplementary Table S13** and **Supplementary Table S17**). Several genes connected to auxin biosynthesis and signalling, such as *SMALL AUXIN UP RNAs,* genes regulated by light, such as *DEVIL1* (*DVL1*), genes encoding membrane-localized proteins, like *ARABINOGALACTAN PROTEIN3* (*AGP3*), or ER membrane-localized proteins involved in fatty acid biosynthesis, like *DELTA 9 DESATURASE1* (*ADS1*), were identified among the top 20 downregulated genes in response to P and T (**Supplementary Table S14** and **Supplementary Table S18**). Notably, several of the genes detected among the top 20 up- and downregulated genes in response to priming (P) or triggering (T1) were also particularly strongly regulated after two PL periods (PL1T *vs.* control) such as *CYP76C5*, *CYP81G1*, *ERF71*, *RBOHC*, *ZAT8* (**Supplementary Table S19**). The auxin-related genes *IAA29* and *SAUR22*, and the light gene *DVL1* were among the top 20 downregulated genes in PL1T in addition to their strong regulation in response to priming or triggering (**Supplementary Table S20**).

Among the top 20 genes still higher expressed in primed plants after returning to SD (PL1 *vs.* control) are genes connected to plant defence responses, such as *PR2* and *PR5*, genes involved in calcium sensing or signalling, such as *CALMODULIN-LIKE41* (*CML41*) and *MULTIPLE C2 DOMAIN AND TRANSMEMBRANE REGION PROTEIN9* (*MCTP9*), and genes responsive to ethylene like *ERF55/72* (**Supplementary Table S15**). Genes related to the plant circadian rhythm, such as *B-BOX DOMAIN PROTEIN30* (*BBX30*), *PATHOGEN AND CIRCADIAN CONTROLLED1* (*PCC1*) and *CYCLING DOF FACTOR6* (*CDF6*), and genes involved in the regulation of the plant redox state, such as *GPX7*, were detected among the top 20 downregulated genes in PL1 (**Supplementary Table S17**). The lacking overlap of genes most strongly regulated in response to P/T1 and PL1 suggests that a single SD is sufficient to abrogate their strong regulation caused by priming or triggering.

DEGs derived from the pairwise comparisons were further filtered for genes that were either two-fold up- or downregulated to exclude genes that are regulated to a lower extent and resulting gene lists were analysed by GO term enrichment analysis (**Figure 37A-C**). Genes connected to SAR, responses to light/radiation, and responses to (external) biotic stimuli, such as to fungi, were detected by GO term analysis in primed plants after returning to SD (PL1 *vs.* control, **Figure 37A**). Genes associated with responses to molecules of bacterial origin, important for SAR, SA-mediated signalling, and responses to hypoxia or (decreased) oxygen levels were overrepresented in response to a 4 h-PL period (T1 *vs.* control) (**Figure 37B**). This is consistent with GO terms detected in a previous section in response to a 4 h-PL period (**section 3.1.2**). Several of the GO terms that were detected in response to triggering (T1 *vs.* control) were also identified in PL1T *vs.* control, such as SAR, responses to molecules of bacterial origin, and (cellular) responses to hypoxia or (decreased) oxygen levels (**Figure 37C**). In addition, other GO terms were overrepresented in PL1T *vs.* control, such as PRR signalling pathway, toxin catabolism and metabolism, and glutathione metabolism (**Figure 37C**).

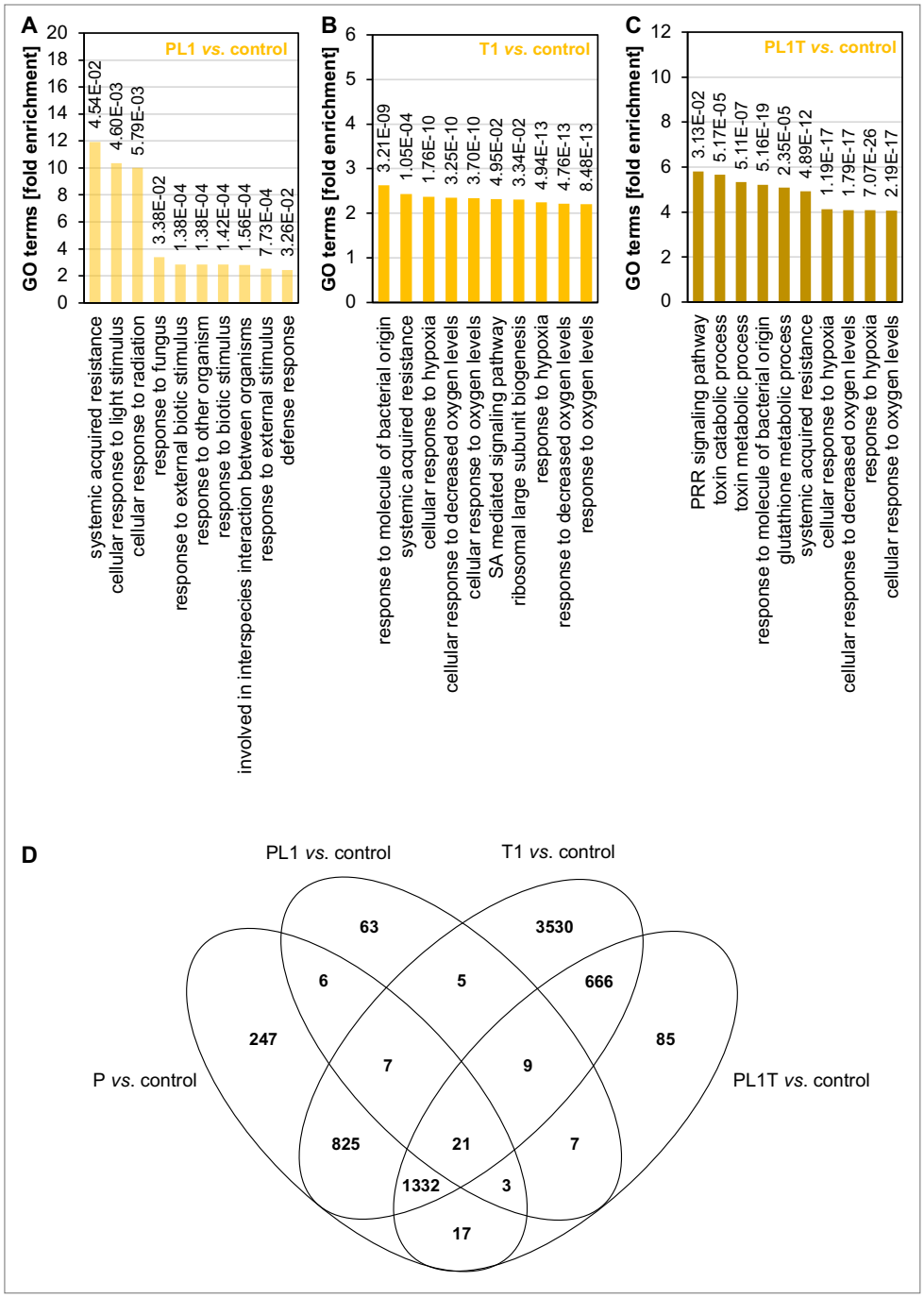

Figure 37. Analysis of transcriptomic changes in response to priming and triggering by photoperiod stress with one day lag phase.

(A-C) GO term enrichment analysis of DEGs determined in the pairwise comparison of (A) of PL1 *vs*. control, (B) T1 *vs*. control, and (C) PL1T *vs*. control. The top ten GO terms are shown. Numbers above the bars represent *p*-values. **(D)** Venn diagram comparing DEGs in Arabidopsis plants treated with one 4 h-PL treatment (P, T1), in combination with a lag phase (PL1), or with two PL treatments (PL1T) in comparison to control condition (Bonferroni-corrected values, adjusted *p*-value ≤ 0.05; log2fold change ≥ |1|).

To identify genes that are commonly regulated by priming and triggering, the gene lists (filtered for *p*-value (Bonferroni) ≤ 0.05, $\log_2$fold change = |1|) were compared with each other and the results were visualised in a Venn diagram (**Figure 37D**, gene lists are provided in an Excel file). In total 1332 genes

were regulated in response to priming, triggering and PL1T, however not in primed plants after returning to SD (**Figure 37D**). *PIF4* and *COR413IM1* were detected in this subset. In addition, also the photoperiod stress marker genes *ZAT12/RHL41* and *BAP1* were found in this subset. Genes in this subset represent interesting candidates for the analysis of memory, i.e. genes with an altered expression in response to priming that returns to a non-induced expression in PL1 (during the memory phase) and are differently induced when exposed to the triggering stimulus. In total 825 genes were regulated by priming and triggering, but not during the memory phase or in response to PL1T (**Figure 37D**). This subset contains genes that are regulated by priming but become insensitive to a second PL period. In a third subset, in total 21 genes were found that were regulated by priming, triggering, PL1T and were still regulated after the lag phase (PL1, **Figure 37D**). This subset represents promising candidates for the analysis of memory, i.e. genes that were induced or downregulated by priming and remained regulated during the memory phase, thereby affecting the expression when exposed to a second stimulus. *BIM1* was identified in this subset. In subset 4, in total 85 genes were identified to be regulated in response to PL1T, however not by priming or triggering or during the memory phase (PL1) (**Figure 37**). This subset contains genes that are not responsive to the priming but become sensitized by the first stimulus, thereby being stronger regulated only in response to the triggering.

3.2.6.3 Transcriptomic changes in response to 4 h-PL periods in Arabidopsis plants at different days after germination

To investigate the transcriptomic changes in response to a 4 h-PL period in Arabidopsis plants at different days after germination (DAG), DEGs in pairwise comparisons of T1 (30 DAG), T4 (33 DAG), T5 (34 DAG) and T10 (39 DAG) with control condition were analysed. Comparisons between PL-treated and control samples revealed 7035 DEGs in T1 *vs.* control, 4234 DEGs in T4 *vs.* control, 6708 DEGs in T5 *vs.* control and 4371 DEGs in T10 *vs.* control.

The top 20 up- and downregulated genes derived from the pairwise comparisons are summarized in **Supplementary Table S17** and **S18**, **Supplementary Table S21** and **S22**, **Supplementary Table S25** and **S26**, and **Supplementary Table S29** and **S30**. Several genes detected among the top 20 up- and downregulated genes in T4 *vs.* control, T5 *vs.* control, and T10 *vs.* control were also previously identified to be regulated in P *vs.* control and T1 *vs.* control. This includes *CYP81G1* and *CYP76C5*, *ERF71*, *ZAT8* and *RBOHC* which were upregulated in response to a single 4 h-PL period independent of DAG. Similarly, several genes, including *ADS1*, *AGP3* and *SAURs*, were identified to be consistently downregulated by a single 4 h-PL period independent of the DAG. This suggests that several genes that are particularly strongly responsive to a 4 h-PL period are regulated independent of the plant developmental state.

To further evaluate the transcriptomic changes in response to a 4 h-PL period in Arabidopsis at different DAGs, the DEGs derived from the pairwise comparisons were further filtered for genes that were either two-fold up- or downregulated and resulting gene lists were analysed by GO term enrichment analysis (**Figure 38**).

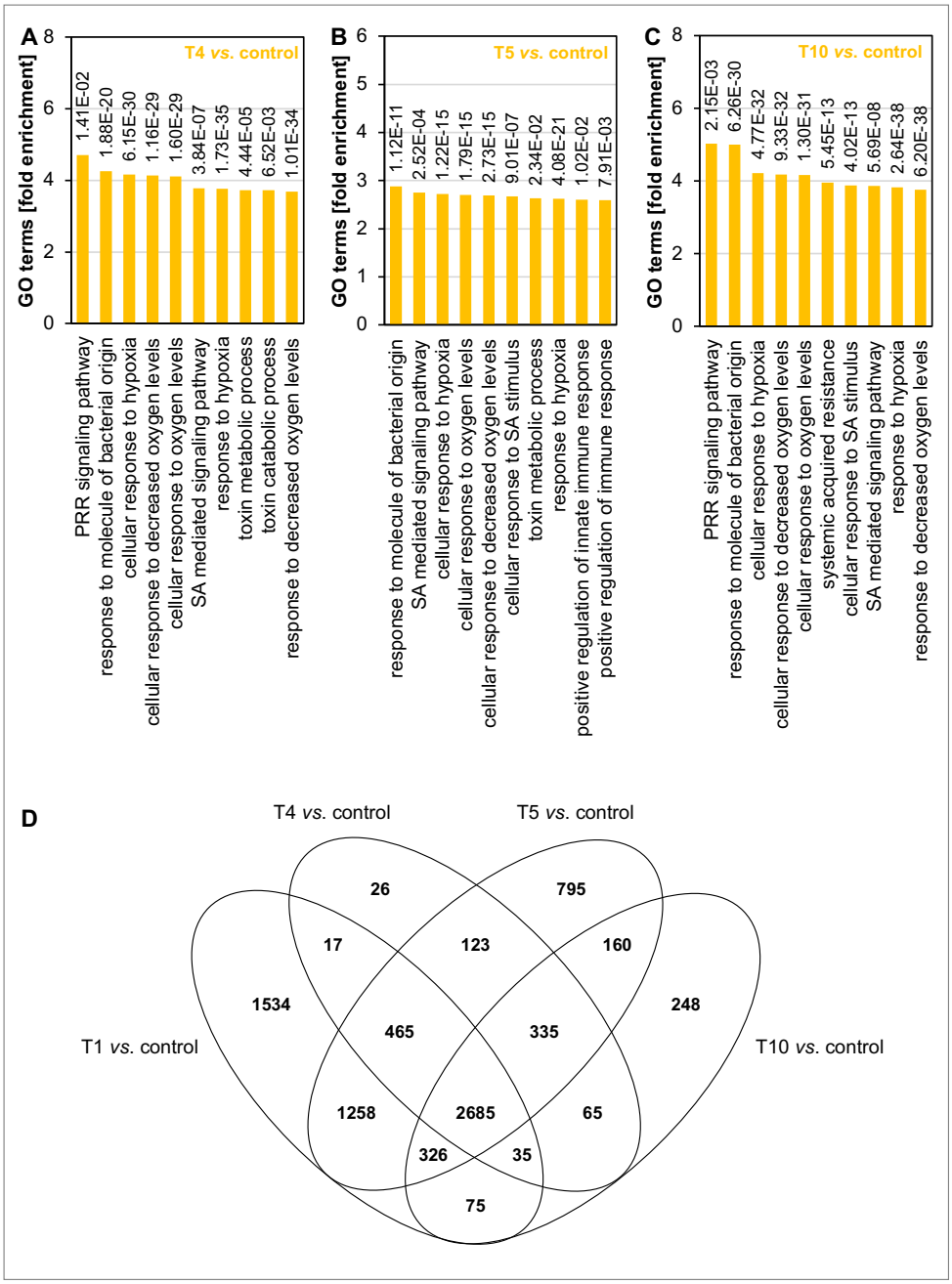

Figure 38. Analysis of transcriptomic changes in response to a 4 h-PL at different days after germination.

(A-C) GO term enrichment analysis of DEGs determined in the pairwise comparison of (A) T4 *vs.* control, (B) T5 *vs.* control, and (C) T10 *vs.* control. The top ten GO terms were shown. Numbers above the bars represent respective *p*-values. **(D)** Venn diagram comparing DEGs in Arabidopsis plants treated with one 4 h-PL period (T1, T4, T5, T10) in comparison to control conditions (Bonferroni-corrected values, adjusted *p*-value ≤ 0.05; $\log_2$fold change ≥ |1|).

Genes associated with responses to molecules of bacterial origin, mediate SA-dependent signalling, and (cellular) responses to hypoxia or (decreased) oxygen levels were identified in response to triggering (T1 *vs.* control, **Figure 37B** and T4 *vs.* control, T5 *vs.* control, T10 *vs.* control, **Figure 38A-C**) independent of the DAG. The majority of DEGs, in total 2685 genes, were regulated in response to every triggering (Venn diagram, **Figure 38D**). The strong overlap in regulation of transcript abundances in

response to a 4 h-PL at different DAG suggests that several DEGs are regulated independently of the DAG regulated (at least within the analysed time frame). However, genes exist that are differentially regulated only at a certain DAGs (1535 genes in T1 *vs.* control, 26 genes in T4 *vs.* control, 795 genes in T5 *vs.* control, 248 genes in T10 *vs.* control, **Figure 38D**) suggesting that the response to a 4 h-PL period is in part influenced by the DAG.

3.2.6.4 Transcriptomic changes of genes that might function as markers for memory of photoperiod stress in Arabidopsis

To investigate the transcriptomic changes of genes that may function as markers for memory of a 4 h-PL period in Arabidopsis, DEGs in PL1T, PL4T, PL5T and PL10T were analysed and pairwise comparisons performed; this allowed getting a deeper insight into the DEGs regulated by two 4 h-PL periods at different durations of the lag phases, in comparison to control condition. Comparisons between primed-and-triggered and control samples revealed that 2501 DEGs in PL1T *vs.* control, 3300 DEGs in PL4T *vs.* control, 4919 DEGs in PL5T *vs.* control and 3829 DEGs in PL10T *vs.* control. The top 20 up- and downregulated genes derived from the pairwise comparisons are summarized in **Supplementary Table S19** and **S20**, **Supplementary Table S23** and **S24**, **Supplementary Table S27** and **S28**, **Supplementary Table S31** and **S32**. Notably, several genes up- and downregulated in PL4T *vs.* control, PL5T *vs.* control and PL10T *vs.* control were also regulated by PL1T *vs.* control. The genes *CYP76C5*, *ERF71*, and *RBOHC* were among the upregulated genes in response to priming and triggering independent of the duration of the lag phase. *ADS1*, *AGP3* and *IAA29* were among the top 20 downregulated genes in response to priming and triggering in PL1T *vs.* control, PL4T *vs.* control, PL5T *vs.* control and PL10T *vs.* control. Their consistent strong regulation independent of the duration of the lag phase suggests that these genes rather represent genes that are responsive to PL periods instead of functioning as markers for priming and memory.

To further study the transcriptomic changes of genes that may function as markers for photoperiod stress memory in Arabidopsis, DEGs derived from the pairwise comparisons were filtered for those two-fold up- or downregulated. The resultant genes were then analysed by GO term enrichment analysis (**Figure 39A-C**). Genes associated with responses to molecules of bacterial origin, toxin metabolism and responses to (decreased) oxygen levels are overrepresented in PL1T *vs.* control (**Figure 37C**), PL4T *vs.* control, PL5T *vs.* control and PL10T *vs.* control (**Figure 39A-C**).

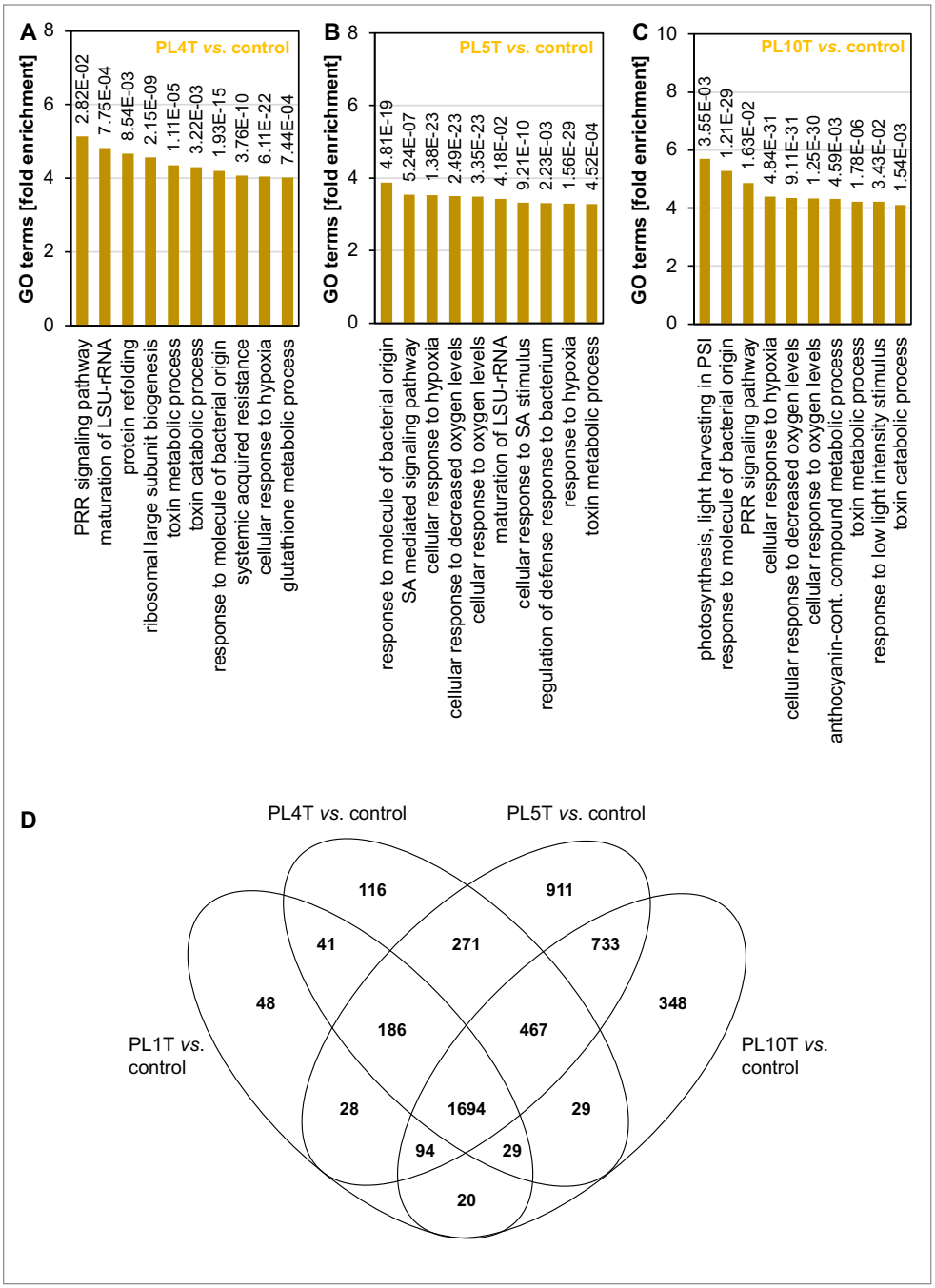

Figure 39. Analysis of transcriptomic memory of photoperiod stress.

(A-C) GO term enrichment analysis of DEGs determined in the pairwise comparison of (A) PL4T *vs.* control, (B) PL5T *vs.* control, and (C) PL10T *vs.* control. The top ten GO terms are shown. Numbers above the bars represent *p*-values. **(D)** Venn diagram comparing DEGs in Arabidopsis plants treated with two 4 h-PL period (PL1T, PL4T, PL5T, PL10T) in comparison to control conditions (Bonferroni-corrected values, adjusted *p*-value ≤ 0.05; $\log_2$fold change ≥ |1|).

The largest number of DEGs, 1694 genes, were regulated in response to every priming-and-triggering scenario, independent of the duration of the lag phase (**Figure 39D**, gene lists are provided in an Excel file); they likely represent genes responsive to PL periods rather than being marker genes for photoperiod stress memory. However, this subset also contained genes that were regulated by all

primed-and-triggered combinations, but to different extents, such as *RHL41*/*ZAT12*, *BAP1*, *PIF4* and *IAA29*. Different abundances of transcripts in PL10T *vs.* control compared to PL1T *vs*. control or PL4T *vs.* control suggest that such genes might function as markers for a photoperiod stress memory. In a second subset, 48 genes were differentially regulated in response to PL1T *vs.* control, but not in response to priming-and-triggering when a longer lag phase was included (**Figure 39**); these genes might function as markers for short-term memory of photoperiod stress.

3.3 *Trans*-priming by photoperiod stress

In the third part of the thesis, the effects of mild photoperiod stress on plant resistance against pathogens were investigated. Strong photoperiod stress caused by a 24 h-PL period in SD-grown Arabidopsis induces alterations of transcripts resembling those during pathogen infection. The transcriptional reprogramming was associated with an induction of SAR in the absence of a pathogen. Especially SA biosynthesis and signalling genes have been identified as photoperiod stress-responsive (Cortleven *et al.*, 2022).

3.3.1 Mild photoperiod stress regulates transcription of defence response genes

To assess the effect of mild photoperiod stress on the transcript abundance of pathogen-responsive genes, four-week-old WT plants were exposed to a PL period of 4 or 8 h (**Figure 40A**). The experiments revealed that a PL of 4 h induces the expression of defence response genes; this upregulation was even stronger after an 8 h PL period. Among the defence response genes showing an increased transcript level in response to a PL period of 4 h or 8 h were the early defence signalling gene *FRK1* as well as SA-dependent biosynthesis/signalling genes such as *ICS1*, *EDS5* and *PR1*, and the SA-independent basal resistance gene *FMO1* (**Figure 40B-F**), thereby confirming the RNA-seq data (**Supplementary Table S11** and **Supplementary Table S39**).

After one SD (lag phase, PL1), expression of the induced genes returned to levels similar to those in control conditions (**Figure 41**). Notably, although *PR1* expression levels decreased after one day of SD condition, it was not as low as in control plants (**Figure 41D**). Together, the results indicate that mild photoperiod stress associated with a PL period of 4 or 8 hours, induces strong but transient alterations in the expression of defence response genes.

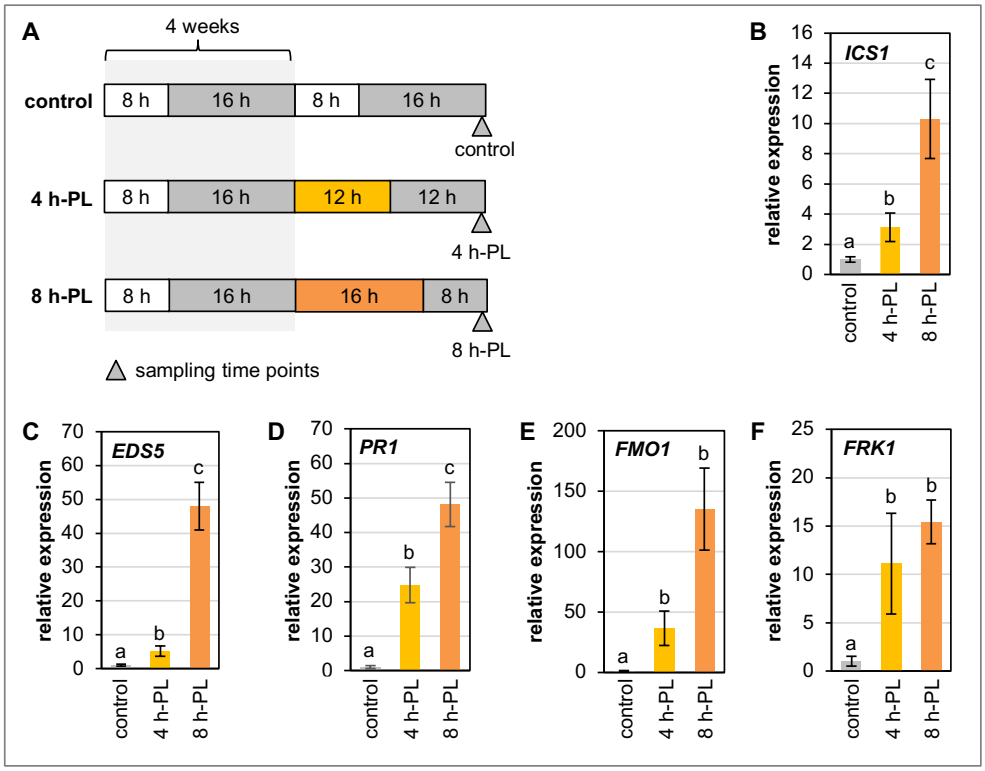

Figure 40. A PL period by 4 h and 8 h regulates the expression of defence response genes.

(A) Schematic overview of the experimental setup. SD-grown Arabidopsis plants were exposed to a 4 h- or 8 h-PL period or remained under SD condition. White, light period; grey, dark period; yellow or orange, light period that is 4 h or 8 h longer than a SD, respectively. **(B-F)** Relative expression of (B) *ICS1*, (C) *EDS5*, (D) *PR1*, (F) *FMO1*, and (F) *FRK1* in Arabidopsis plants under control condition or at the end of the night that followed a 4 h- or 8 h-PL period. Values are expressed relative to control samples, which were set to 1. Error bars represent SE (n ≥ 3). Letters indicate different statistical groups ($p \leq 0.05$) as determined by ANOVA (of logarithmically transformed data when normality or homogeneity of variances were not met) followed by Tukey's post-hoc test.

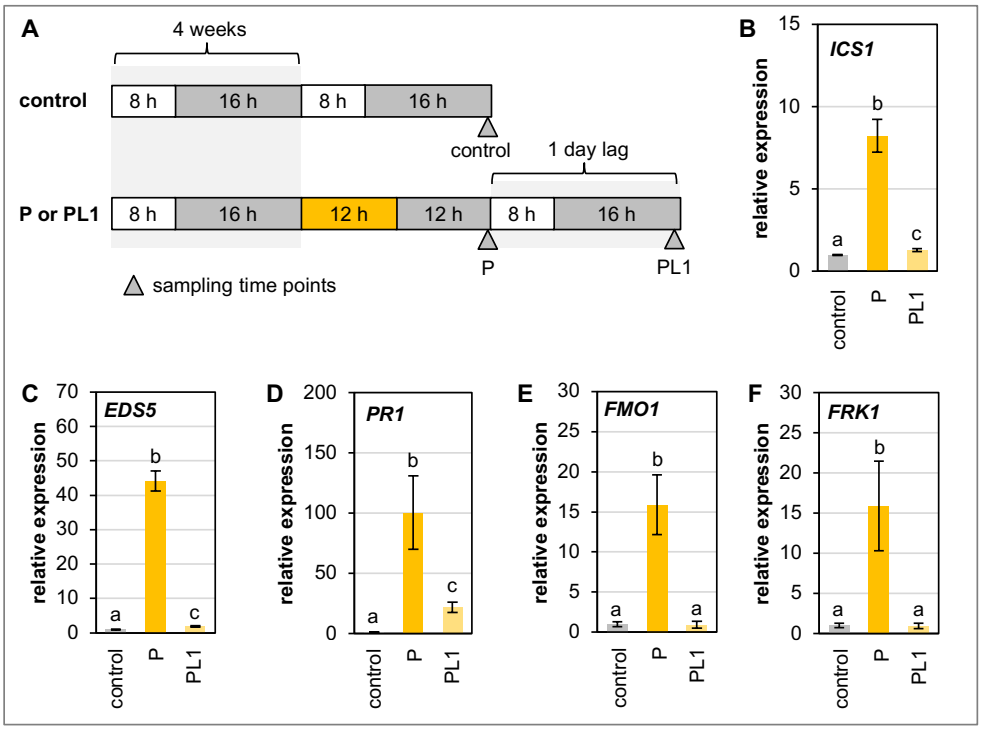

Figure 41. Expression of photoperiod stress-induced genes after a one-day lag phase.

(A) Schematic overview of the experimental setup. SD-grown Arabidopsis plants were exposed to a 4 h-PL period or remained under SD condition. In addition, plants that were treated with a 4 h-PL period and returned to SD conditions for 1 day afterwards (PL1) were analysed. White, light period; grey, dark period; yellow, light period that 4 h is longer than a SD. **(B-F)** Relative expression of (B) *ICS1*, (C) *EDS5*, (D) *PR1*, (F) *FMO1*, and (F) *FRK1* in Arabidopsis plants under control conditions or at the end of the night that followed a 4 h prolonged light period. In addition, plants that were treated with a 4 h-PL period and returned to SD conditions for 1 day (PL1) were analysed. Values are expressed relative to control samples, which were set to 1. Error bars represent SE (n ≥ 3). Letters indicate different statistical groups ($p \leq 0.05$) as determined by ANOVA (of logarithmically transformed data when normality or homogeneity of variances were not met) followed by Tukey's post-hoc test.

3.3.2 Mild photoperiod stress increases salicylic acid and camalexin levels

Several phytohormones, including SA, JA and JA-Ile, are involved in both light signalling pathways and defence against biotic stresses (Roeber *et al.*, 2021). In the context of photoperiod stress, Nitschke *et al.* (2016) highlighted that JA and JA-Ile levels strongly increase in response to a 24 h-PL period. Cortleven *et al.* (2022) recently pointed out that SA levels also increase in response to strong photoperiod stress. Additionally, the concentration of the phytoalexin camalexin is strongly elevated (Cortleven *et al.*, 2022).

To determine, if shorter PL periods (less than 24 hours) also influence phytohormone levels, Arabidopsis plants were treated with a 4 h-PL period (**Figure 42A**). Sampling was performed at the end of the prolonged light period. Additionally, samples were harvested from plants kept for one, two or three days under SD condition after the PL treatment (PL1, PL2 or PL3, respectively) to investigate the late effects of a PL period on phytohormone and camalexin levels.

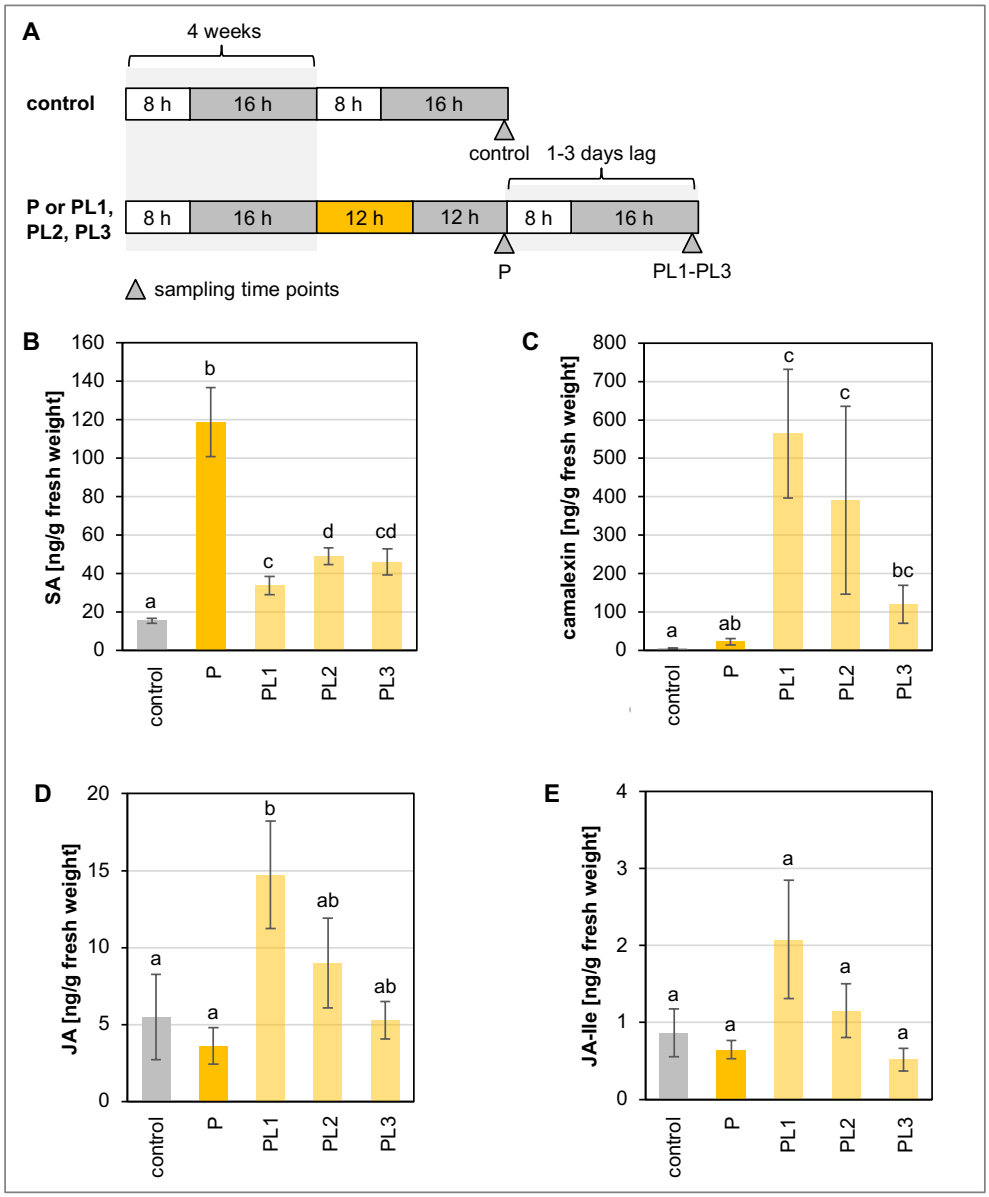

Figure 42. Mild photoperiod stress affects the levels of phytohormones and camalexin involved in plant defence responses.

(A) Schematic overview of the experimental setup. SD-grown Arabidopsis plants were exposed to a 4 h-PL period or remained under SD condition. In addition, plants treated with a 4 h-PL period and returned to SD conditions for 1 to 3 days (PL1-PL3) were analysed. White, light period; grey, dark period; yellow, light period that 4 h is longer than a standard SD. **(B-E)** Levels of (B) salicylic acid (SA), (C) camalexin, (D) JA, and (E) JA-Ile. Values represent means ± SE (n ≥ 8). Letters indicate different statistical groups ($p \leq 0.05$) as determined by ANOVA (of logarithmically transformed data when normality or homogeneity of variances were not met) followed by Tukey's post-hoc test.

Interestingly, JA and JA-Ile levels were only modestly affected by a 4 h-PL period, whereas SA content strongly increased at the end of the night following a 4 h-PL period (**Figure 42B, D, E**). The SA content decreased (but tended to be slightly higher than in control plants) in plants that were returned to SD. In addition to SA, camalexin levels were strongly elevated up to 500-fold by a 4 h-PL period (**Figure 42C**). The highest camalexin levels were reached after one SD (PL1, **Figure 42C**). As camalexin biosynthesis during plant defence responses involves SA signalling (Glawischnig, 2007), it seemed reasonable that

camalexin levels peaked later than SA concentrations. Taken together, the results suggest that moderate photoperiod stress induced by 4 h-PL period particularly affects SA biosynthesis/signalling pathways, whereas a 24 h-PL period, representing a stronger photoperiod stress, additionally influences JA and JA-Ile biosynthesis/signalling (Nitschke *et al.*, 2016; Cortleven *et al.*, 2022). As both SA and camalexin are important for pathogen defence, the results indicate that moderate photoperiod stress activates signalling cascades normally induced after exposure to biotic stresses in plants.

3.3.3 Mild and strong photoperiod stresses induce similar changes of gene expression

The results have shown that mild and more severe photoperiod stress show significant similarities with respect to gene regulation, increases in SA levels, and peroxide content (Cortleven *et al.*, 2022). To analyse the consequences of mild and strong stress treatments on gene expression in more detail, an RNA-seq dataset of Cortleven *et al.* (2022) for a PL treatment of 24 h and those generated in this study for WT (**section 3.3.10**) were compared. About one-third of all DEGs (34%) were commonly regulated in response to mild and strong photoperiod stress (**Figure 43A**). However, most DEGs (41.7%) were only regulated in response to mild photoperiod stress, while one-fourth (24.3%) was only regulated in response to strong photoperiod stress (**Figure 43A**).

GO term enrichment analysis revealed that processes related to phytoalexin camalexin (and indole) biosynthesis and metabolism, as well as cellular responses to hypoxia are enriched among the commonly regulated DEGs (**Figure 43C**). DEGs only detected in plants treated with mild photoperiod stress were enriched for genes involved in responses to (low) light intensity, red/blue light, biotic stimuli, and auxin (**Figure 43B**). DEGs only identified in plants exposed to strong photoperiod stress have functions in processes related to the assembly of NAD(P)H dehydrogenase complexes or photosystem II, plastid transcription and translation, and chloroplast (r)RNA processing (**Figure 43D**).

Taken together, the analysis suggests that the length of the prolonged light period (in combination with the following dark period) has a significant impact on the set of responsive genes. Interestingly, especially genes related to phytoalexin (including camalexin and indole) biosynthesis and metabolism are regulated independently of the length of the PL period.

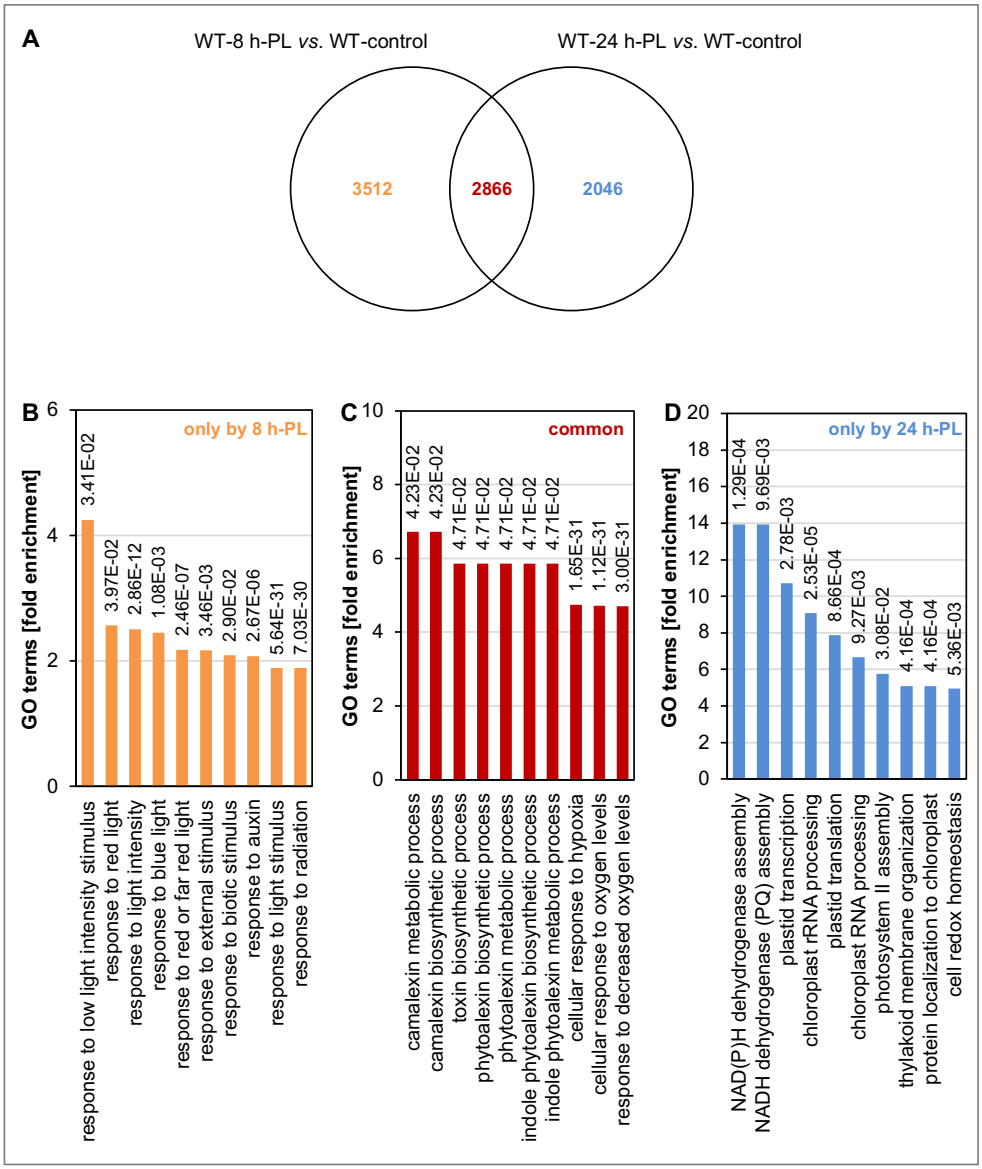

Figure 43. Comparison of changes in transcript abundances of Arabidopsis in response to mild and strong photoperiod stress.

(A) Venn diagram comparing DEGs in Arabidopsis plants treated with a PL period of 8 h or 24 h in comparison to control condition (Bonferroni-corrected values, adjusted *p*-value ≤ 0.05; $\log_2$fold change ≥ I1I). The RNA-seq dataset for WT plants treated with an 8 h-PL period is further characterized in **section 3.3.10**. **(B-D)** Top 10 GO terms of DEGs that are regulated in WT Arabidopsis plants in response to (B) 8 h-PL *vs.* control, (C) both a PL period of 8 h and 24 h, and (D) 24 h-PL *vs.* control. Numbers above the bars represent *p*-values.

3.3.4 Mild photoperiod stress primes the defence of Arabidopsis plants against a subsequent *Pseudomonas* infection

A previous study showed that a PL period of 24 h strongly decreases the infection by of *P. syringae* pv. *tomato* (*Pst*) DC3000 in Arabidopsis (Cortleven *et al.*, 2022). As mild photoperiod stress activates reactions similar to those triggered by pathogen infection, it was investigated whether PL periods of 4 or 8 h improve the resistance of plants against a subsequent pathogen infection and whether plants memorize mild photoperiod stress over a stress-free period.

To investigate this, Arabidopsis plants were inoculated with *Pst* DC3000 bacteria (triggering stimulus, T) after being exposed to a PL period as priming (P, **Figure 44**). As mock treatment, plants were infiltrated with 10 mM magnesium chloride. Control plants remained untreated under SD condition. Plants pre-treated with PL periods of 8 hours and inoculated with *Pst* directly the next day (no lag phase, L0) or after one day SD condition (with lag phase, L1) showed an enhanced resistance against *Pst* infection (**Figure 44B**). This suggests that mild photoperiod stress is memorized by plants and primes resistance against *Pst* infection. Longer lag phases resulted again in a higher susceptibility to *Pst* (**Figure 44B**) indicating that the memory for photoperiod stress against pathogen attack lasts for about one day.

In addition, a PL period of 4 hours decreased the bacterial growth in Arabidopsis plants directly in the next day (no lag phase, PL0T, **Figure 45B**) demonstrating that also shorter PL periods improve the resistance against *Pst*. However, plants pre-treated with PL periods of 4 hours and *Pst*-inoculated after lag phases (of one to three days) showed a similar bacterial growth as plants only infected with *Pst* (**Figure 45B**), indicating that the PL period influences the response and the length of the photoperiod stress memory.

To assess changes in expression levels, expression of genes typically induced by mild photoperiod stress or in response to *Pst* were investigated in plants previously treated with photoperiod stress and triggered by *Pst* infection without a lag phase (PL0T) and after one day (PL1T) (**Figure 44C-D**). In experiments without a lag phase (PL0T), expression of *PR1* and *FMO1* was more strongly induced in plants exposed to 4 h or 8 h-PL period before infection than in only *Pst*-infected plants (**Figure 44C-D**, **Figure 45C-D**). This stronger induction may indicate priming by mild photoperiod stress. However, in experiments with a one-day lag phase (PL1T), no increased induction of *PR1* and *FMO1* was observed (**Figure 44C-D**, **Figure 45C-D**) suggesting that photoperiod stress primes the expression of these two analysed genes. Altogether, the experiments revealed that PL periods by 4 h or 8 h improve the resistance of Arabidopsis plants against *Pst* infection. The changes in transcript levels of *PR1* and *FMO1* seem to function as reliable indicators of photoperiod stress-induced priming and memory.

To further investigate the effects of mild photoperiod stress on the resistance of Arabidopsis against pathogen attack, the following experimental setup was used as a standard condition: Plants were treated with a PL period by 8 hours as the priming stimulus and infected with *Pst* as the triggering stimulus directly at the next day (without a lag phase, PL0T). This treatment was chosen as it resulted in a strong transcript regulation in Arabidopsis plants.

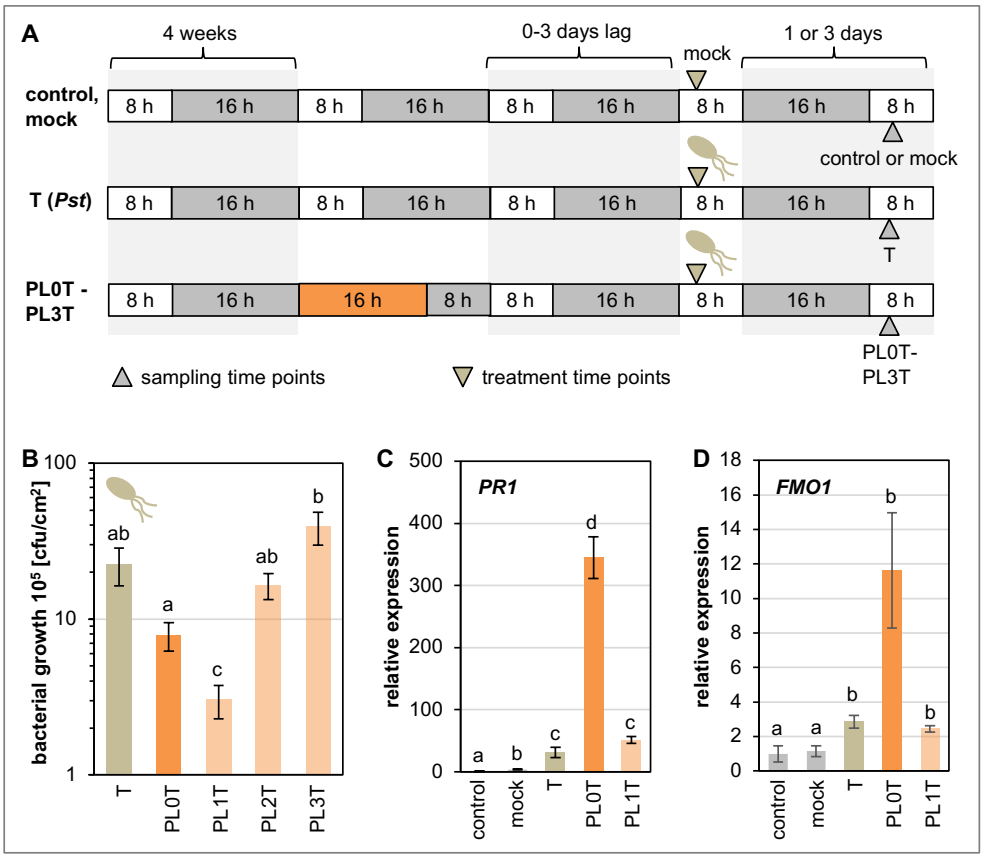

Figure 44. A PL period of 8 hours improves the resistance of Arabidopsis plants against *Pst* DC3000 infection.

(A) Schematic overview of the experimental setup. SD-grown plants kept under SD condition (control) were treated with 10 mM magnesium chloride (mock) or infected with *Pst* DC3000 (T) and pre-treated with a prolonged light period of 8 hours in advance (PL0T-PL3T). **(B)** Bacterial growth in plants infected with *Pst* DC3000 (T) and pre-treated with a prolonged light period of 8 hours directly before infection (PL0T) or one, two or three days under SD condition between both treatments (PL0T-PL3T). **(C-D)** Relative expression of (C) *PR1*, and (D) *FMO1* in plants under control condition, treated with 10 mM magnesium chloride (mock) or infected with *Pst* DC3000 (T) and pre-treated with a prolonged light period of 8 hours in advance (PL0T, PL1T). Values represent means (n ≥ 8 for bacterial growth analysis, n ≥ 4 for expression analysis) ± SE. Expression values are expressed relative to control samples, which were set to 1. Letters indicate statistically different groups (*p*-value ≤ 0.05) as determined by ANOVA followed by Tukey's post-hoc test.

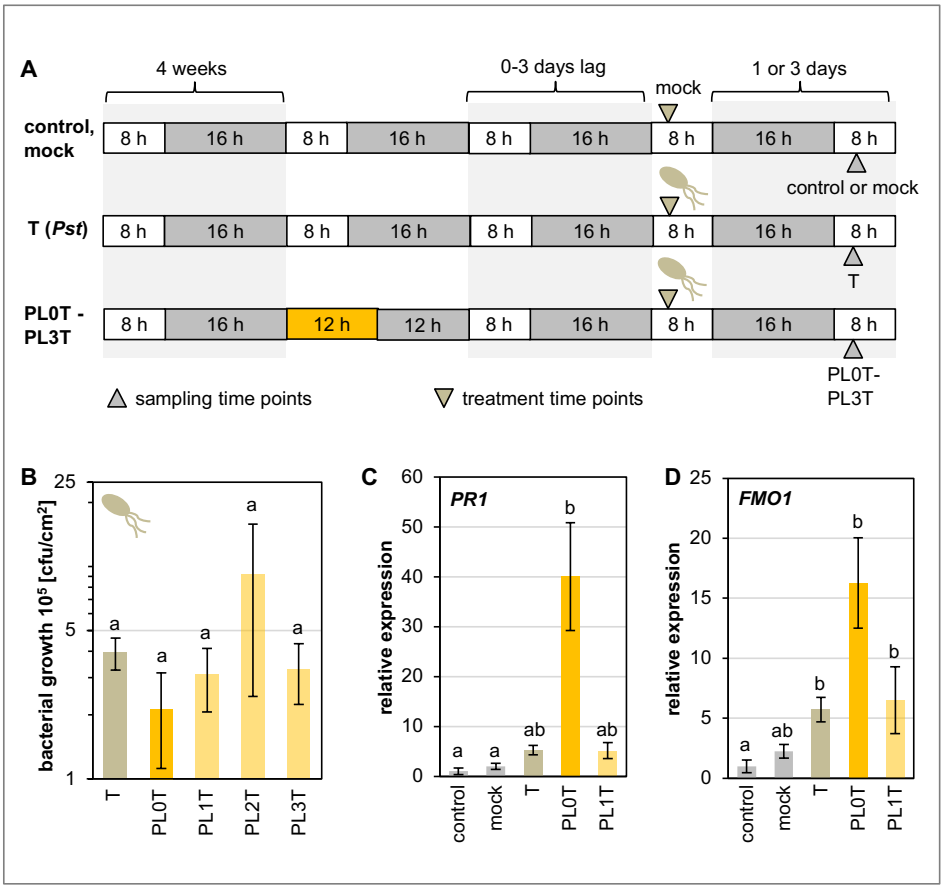

Figure 45. A PL period of 4 hours slightly improves the resistance of Arabidopsis plants against *Pst* DC3000 infection.

(A) Schematic overview of the experimental setup. Plants kept under SD condition (control), were treated with 10 mM magnesium chloride (mock) or infected with *Pst* DC3000 (T) and pre-treated with a PL period of 4 h in advance (PL0T-PL3T). **(B)** Bacterial growth in plants infected with *Pst* DC3000 (T) and pre-treated with a PL period of 4 hours directly before infection (PL0T) or one, two or three days under SD condition between both treatments (PL0T-PL3T). **(C-D)** Relative expression of (C) *PR1*, and (D) *FMO1* in plants under control condition, treated with 10 mM magnesium chloride (mock) or infected with *Pst* DC3000 (T) and pre-treated with a PL period of 4 h in advance (PL0T, PL1T). Values represent means (n ≥ 8 for bacterial growth analysis, n ≥ 4 for expression analysis) ± SE. Expression values are expressed relative to control samples, which were set to 1. Letters indicate statistically different groups (*p*-value ≤ 0.05) as determined by ANOVA followed by Tukey's post-hoc test.

3.3.5 Photoperiod stress-induced resistance against *Pseudomonas* attacks requires SA biosynthesis/signalling and components of SAR

SA-dependent defence is particularly effective against biotrophic pathogens, including *Pst* (Beckers & Spoel, 2006). Therefore, we investigated if SA biosynthesis/signalling is required for improvement of plant resistance against *Pst* that was observed after a 4 h- or 8 h-PL period. To this end, mutants with impaired SA biosynthesis (*ics1*) or signalling (*pad4*, *npr1*) were used. In addition, the involvement of pipecolic acid (Pip) and N-hydroxy pipecolic acid (NHP), which are important for mediating SAR (Lim, 2023), was examined using mutants with impaired Pip accumulation (*ald1*) or impaired conversion of Pip to NHP (*fmo1*).

First, the responsiveness of the mutants to an 8 h-PL period was analysed by measuring peroxide content at the end of the night that followed the PL treatment. The experiments revealed increased peroxide levels in WT plants and all SA- and SAR-related mutants except *npr1* (**Figure 46A**) indicating that NPR1 is required for the redox-related photoperiod stress response in Arabidopsis.

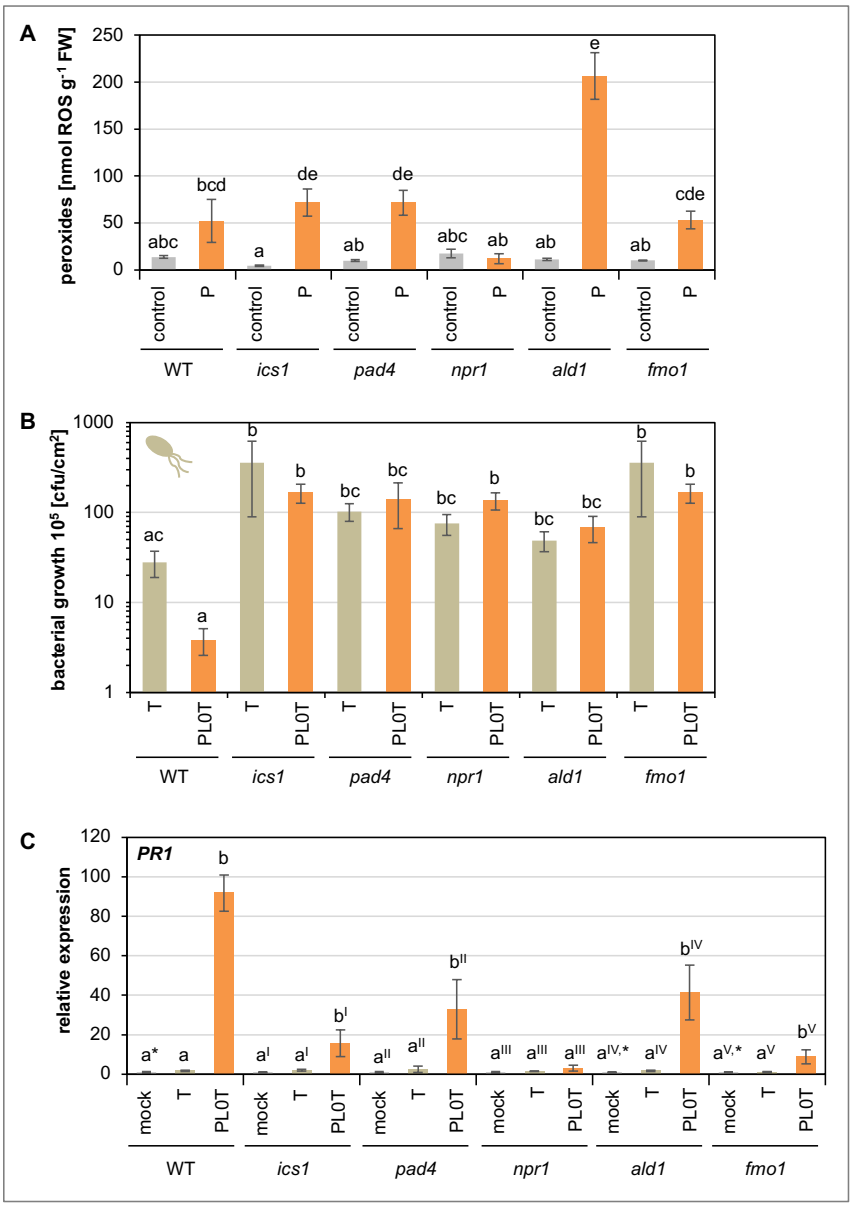

Figure 46. Activation of resistance against *Pst* DC3000 requires components of SA biosynthesis/signalling and SAR.

(A) Peroxide content in leaves of WT plants and *ics1*, *pad4*, *npr1*, *ald1* and *fmo1* mutants at the end of the night that followed an 8 h-PL period (P). **(B)** Bacterial growth in WT plants and mutants related to SA biosynthesis/signalling (*ics1*, *pad4*, *npr1*) or SAR (*ald1*, *fmo1*) infected with *Pst* DC3000 (T) and pre-treated with a PL period of 8 h directly before infection (PL0T). Bacteria were extracted three days after infection. **(C)** Relative expression of *PR1* in WT plants and mutants related to SA biosynthesis/signalling (*ics1*, *pad4*, *npr1*) or SAR (*ald1*, *fmo1*) treated with 10 mM magnesium chloride (mock) or infected with *Pst* DC3000 (T) and pre-treated in advance. Values represent means (n ≥ 8 for bacterial growth experiment, n ≥ 4 for expression analysis and peroxide measurements) ± SE. Expression values are expressed relative to the mock samples of the respective genotype, which were set to 1. Letters indicate statistically different groups (p-value ≤ 0.05) as determined by ANOVA followed by Tukey's post-hoc test. Statistical analysis was performed for every genotype separately as indicated by Roman numbers. Mock treatments of different genotypes were additionally compared by statistical analysis (all mock treatments marked with an asterisk belong to the same statistical group). FW, fresh weight.

Second, the mutants were exposed to an 8 h-PL period and infected with virulent *Pst* bacteria (without a lag phase, **Figure 46B**). All mutants exposed to a PL period in advance to inoculation with *Pst* (PL0T) were as strongly infected as mutants that had not received a PL treatment (T, **Figure 46B**). These results suggest that a functional plant immune system including SA biosynthesis (*ICS1*) or signalling (*PAD4*, *NPR1*) and Pip/NHP biosynthesis (*ALD1*, *FMO1*) is required for improving resistance against *Pst* infection by mild photoperiod stress.

To further unravel photoperiod stress-induced priming, *PR1* expression after infection with *Pst* with (PL0T) or without (T) a prior PL treatment was analysed. The experiments revealed a stronger *PR1* induction in all PL0T-treated mutants except *npr1* (**Figure 46C**). Although bacterial growth was similar in all primed-and-triggered and triggered SA- and SAR-related mutants and did therefore not indicate priming against *Pst*, the *PR1* expression analysis suggested that *ics1*, *pad4*, *ald1* and *fmo1* were primed at least to some extent (**Figure 46C**). Nevertheless, the observed priming and the responsiveness to the PL treatment (as measured by the increased peroxide content) were not sufficient to decrease bacterial growth in those mutants pre-treated by a PL period. This suggests that a functional plant immune system including SA biosynthesis and signalling but also Pip/NHP biosynthesis is required for the improvement of the resistance against *Pst* induced by mild photoperiod stress.

As *PR1* expression is (due to its direct regulation by NPR1) not induced in the *npr1* mutant (Cao *et al.*, 1994; 1997; Chen *et al.*, 2019), the analysis of *PR1* transcript levels could not be used to evaluate the priming response. Therefore, it was searched for a gene that is not regulated by NPR1 but responsive to both photoperiod stress and the combined treatment by photoperiod stress and pathogen infection. Thus, expression of the NHP biosynthesis gene *FMO1* was tested in WT plants and the *npr1* mutant; notably, expression of *FMO1* was increased in PL0T WT plants compared to only *Pst*-infected plants (**Figure 47**). For *npr1* mutants, similar, however not significant, tendencies were observed (**Figure 47**). This indicates that mutation of *NPR1* partly abrogates the response otherwise observed in WT plants in response to a combination of mild photoperiod stress and *Pst* infection.

Taken together, the results showed that SA biosynthesis/signalling and components of SAR are required for improving resistance against *Pst* by a PL period. Although an 8 h-PL period seems to prime expression of *PR1* in SA- and SAR-related mutants (*ics1*, *pad4*, *ald1*, *fmo1*) similar to WT, this was not sufficient to enhance resistance against *Pst* in the mutants. Interestingly, the responsiveness to photoperiod stress is not necessarily connected to the primability of a plant against *Pst* (as observed for *npr1*). SA-regulated *NPR1* is required for the photoperiod stress-induced increase in peroxide content.

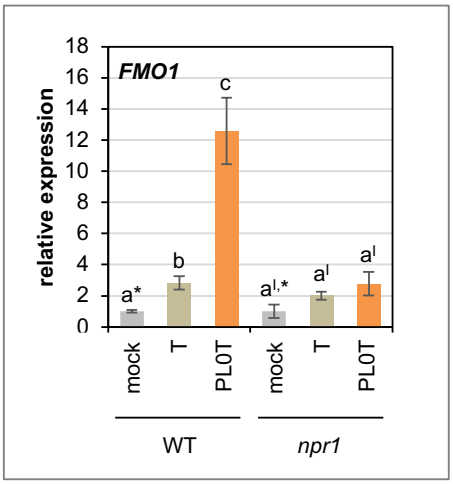

Figure 47. Mutation of *NPR1* partly suppresses the transcription induction of *FMO1* in response to a combination of mild photoperiod stress and an infection with *Pst* DC3000.

Relative expression of *FMO1* in WT plants and *npr1* mutants treated with 10 mM magnesium chloride (mock) or infected with *Pst* DC3000 (T) and pre-treated with a PL period of 8 hours (PL0T). Values represent means (n ≥ 4) ± SE. Expression values are expressed relative to the mock samples of the respective genotype, which were set to 1. Letters indicate statistically different groups (*p*-value ≤ 0.05) as determined by ANOVA followed by Tukey's post-hoc test. Statistical analysis was performed for every genotype separately as indicated by Roman numbers. Mock treatments of different genotypes were additionally compared by statistical analysis (mock treatments marked with an asterisk belong to the same statistical group).

3.3.6 Photoperiod stress-induced resistance against *Pseudomonas* attack requires ARR2 and TGA transcription factors

Plant resistance against *Pst* infection is enhanced by interaction of CK-regulated transcription factor ARR2 with the SA-responsive factor TGA3, which additionally interacts with NPR1, thereby regulating transcription of defence-related genes, such as *PR1* (Choi *et al.*, 2010). In the context of strong photoperiod stress, ARR2, which functions together with ARR10 and ARR12 as regulators of photoperiod stress, improves the tolerance of Arabidopsis to a suddenly PL period (Nitschke *et al.*, 2016; Frank, 2019).

In view of the importance of ARR2 in plant resistance against *Pst* infection and photoperiod stress, the involvement of ARR2 in photoperiod stress-induced resistance against *Pst* infection was investigated. Measurements of the peroxide content in *arr2* mutants exposed to an 8 h-PL period showed a responsiveness to mild photoperiod stress (**Figure 48A**). Nevertheless, *arr2* mutants were not protected against a subsequent *Pst* infection by an 8 h-PL period; the bacterial growth was similar in only *Pst*-infected *arr2* (T) and primed-and-triggered *arr2* mutants (PL0T, **Figure 48B**). This suggests that ARR2 is required to improve the resistance against *Pst* infection in response to a prolongation of the light period.

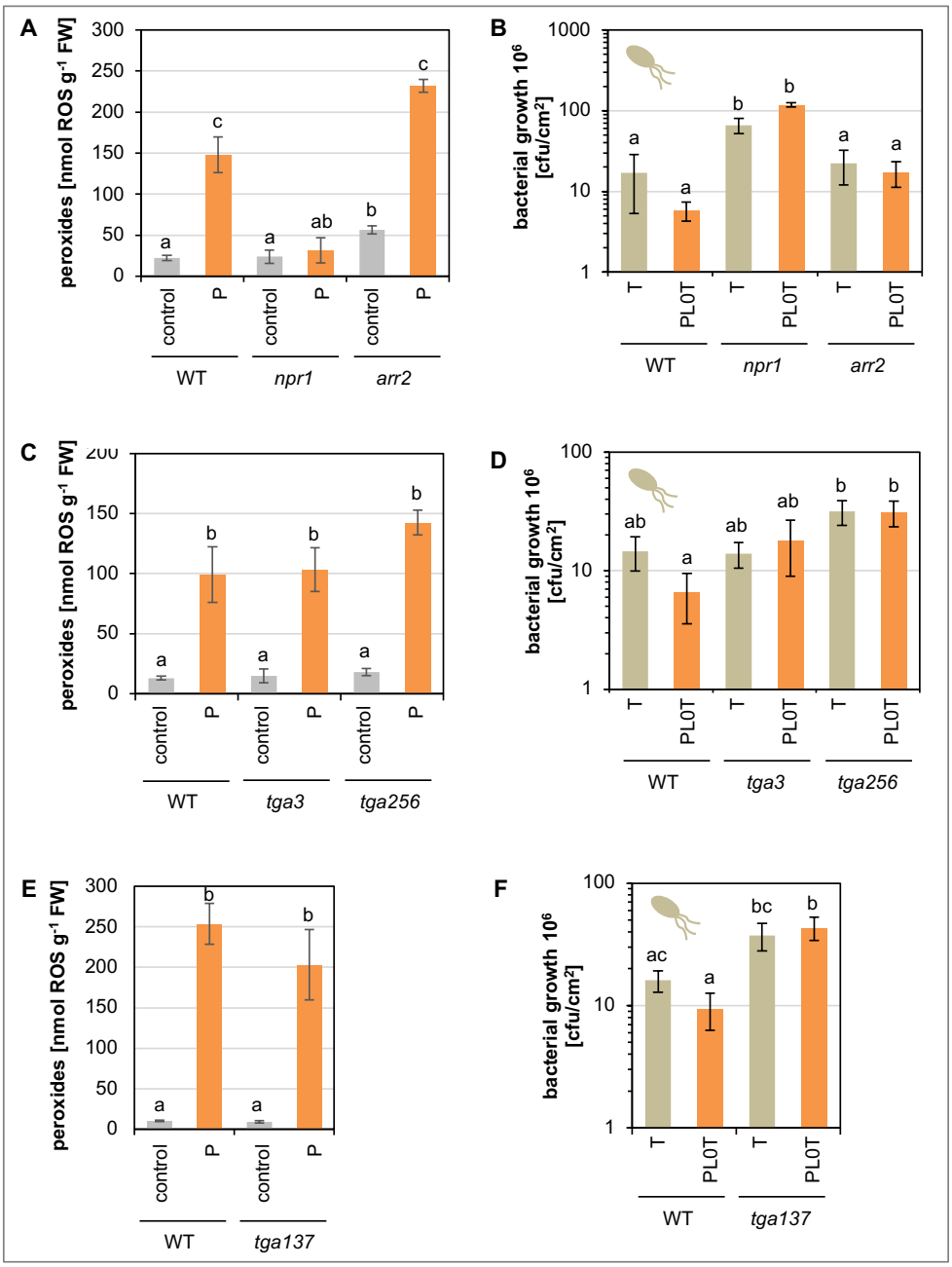

Figure 48. Activation of resistance against *Pst* DC3000 requires ARR2 and TGA transcription factors.

(A, C, E) Peroxide content in leaves of WT plants, and (A) *npr1*, and *arr2*, (C) *tga3*, and *tga256*, and (E) *tga137* mutants at the end of the night that followed an 8 h PL period (P). **(B, D, F)** Bacterial growth in WT plants, and (B) *npr1*, and *arr2*, (D) *tga3*, and *tga256*, and (F) *tga137* infected with *Pst* DC3000 (T) and pre-treated with a PL period of 8 hours directly before infection (PL0T). Data represent means (n ≥ 8 for bacterial growth experiment, n ≥ 4 for peroxide measurements) ± SE. Letters indicate statistically different groups (*p*-value ≤ 0.05) as determined by ANOVA followed by Tukey's post-hoc test. FW, fresh weight.

ARR2 interacts with the SA responsive factor TGA3 which additionally interacts with NPR1 (Choi *et al.*, 2010). As NPR1-interacting TGAs have been shown to be important regulators of biotic and abiotic stress responses in Arabidopsis (Tomaz *et al.*, 2022), their involvement in photoperiod stress-induced resistance against *Pst* infection was investigated. As both NPR1 and ARR2 interact with TGA3, *tga3*

mutants were first analysed. Their exposure to an 8 h-PL period revealed that they are photoperiod stress-responsive indicated by the fact that peroxide content increased in response to a prolonged light treatment (**Figure 48C**). However, the resistance of *tga3* mutants against a subsequent *Pst* infection was not improved by an 8 h-PL period, similar to *arr2* mutants (**Figure 48D**), indicating that the photoperiod stress-induced resistance against *Pst* infection requires TGA3 in addition to NPR1 and ARR2.

Furthermore, it was investigated if also other NPR1-interacting TGAs convey photoperiod stress-induced resistance against *Pst* infection. Exposure of *tga2 tga5 tga6* (*tga256*) and *tga1 tga3 tga7* (*tga137*) mutants to an 8 h-PL period revealed that both triple mutants are photoperiod stress-responsive; their peroxide content increased in response to the PL treatment (**Figure 48C, E**). However, also these mutants were not protected against a subsequent *Pst* infection by a prolongation of the light period (**Figure 48D, F**) indicating that interactors of NPR1 are required for photoperiod stress-induced resistance against *Pst* infection, thereby supporting the importance of NPR1 in this process.

3.3.7 Photoperiod stress-induced resistance against a future *Pseudomonas* infection requires thioredoxin-mediated monomerization and phosphorylation of nuclear NPR1

In the absence of oxidative stress, NPR1 proteins form oligomers in the plant cytoplasm (Backer *et al.*, 2019). Increasing SA levels leading to oxidative stress result in the dissociation of NPR1 oligomers into monomers, which translocate into the nucleus. Dissociation of oligomeric NPR1 is realized by thioredoxin H-type 3 (TRX-h3) and H-type 5 (TRX-h5), which reduce NPR1 cysteines thereby releasing NPR1 monomers (Mou *et al.*, 2003; Tada *et al.*, 2008). The NPR1 monomers are subsequently imported into the nucleus involving the SNF-1 RELATED PROTEIN KINASE 2-8 (SnRK2-8), which interacts with NPR1 monomers and phosphorylates them (Lee *et al.*, 2015; Backer *et al.*, 2019).

Because of the importance of NPR1 monomerization, phosphorylation and nuclear import for its function as transcription co-activator we investigated the involvement of TRX-h3, TRX-h5 and SNRK2-8 in photoperiod stress-induced resistance against *Pst* infection. Upon exposure of *trxh3*, *trxh5* and *snrk2-8* mutants to an 8 h-PL treatment the peroxide content increased, showing that the mutants were responsive to photoperiod stress (**Figure 49A**). However, *trxh3*, *trxh5* and *snrk2-8* responded similar to *Pst* with or without an 8 h-PL treatment in contrast to WT (**Figure 49B**). This suggests TRX-h3- and TRX-h5-mediated NPR1 monomerization and SNRK2-8-dependent phosphorylation and nuclear import are required for photoperiod stress-induced resistance against *Pst* infection. This further supports an important role for NPR1 in this process.

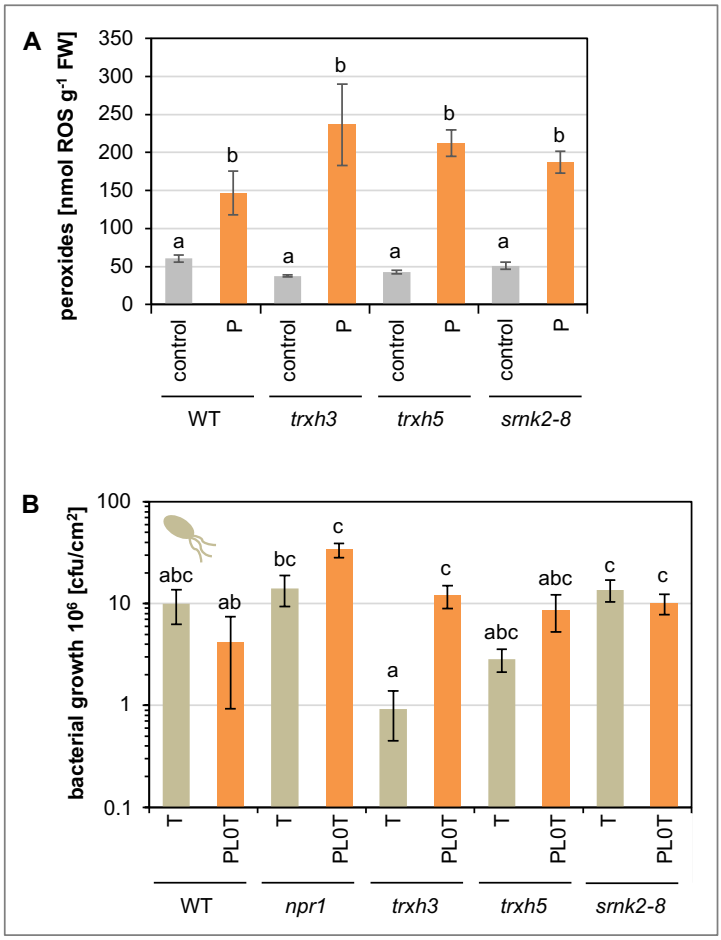

Figure 49. Activation of resistance against *Pst* DC3000 requires thioredoxin-mediated monomerization and phosphorylation of nuclear NPR1.

(A) Peroxide content in leaves of WT plants and *npr1*, *trxh3*, *trxh5*, and *srnk2.8* mutants at the end of the night that followed an 8 h-PL period (P). **(B)** Bacterial growth in WT plants and mutants required for thioredoxin-mediated monomerization of NPR1 and phosphorylation of nuclear NPR1 (*npr1*, *trxh3*, *trxh5*, and *srnk2.8*) infected with *Pst* DC3000 (T) and pre-treated with a PL period of 8 hours directly before infection (PL0T). Values represent means (n ≥ 7 for bacterial growth experiment, n ≥ 4 for peroxide measurements) ± SE. Letters indicate statistically different groups (*p*-value ≤ 0.05) as determined by ANOVA followed by Tukey's post-hoc test. FW, fresh weight.

3.3.8 Photoperiod stress-induced resistance against *Pseudomonas* attack requires JA biosynthesis and signalling

In addition to SA signalling, also JA signalling regulates the defence against *P. syringae* infection (Cui *et al.*, 2005). Depending on the conditions, SA and JA signalling either improve the plants' resistance together (van Wees *et al.*, 2000) or negatively regulate respective plant defence responses (Thaler *et al.*, 2001; Traw & Bergelson, 2003; Cui *et al.*, 2005). In response to an 8 h-PL period, JA and JA-Ile levels increase in WT pointing to the involvement of JA signalling in mild photoperiod stress (Cortleven *et al.*, 2022). Due to the importance of JA signalling for both, defence against *Pst* infection and photoperiod stress responses, the involvement of JA biosynthesis/signalling in photoperiod stress-induced resistance against *Pst* infections was investigated using mutants with an impaired biosynthesis of the JA precursor OPDA (*lox3*, *lox4*, *aos*), JA biosynthesis (*dde2*), JA-Ile biosynthesis (*jar1*) or disturbed JA signalling (*myc234*, *ora59*, **Figure 50**).

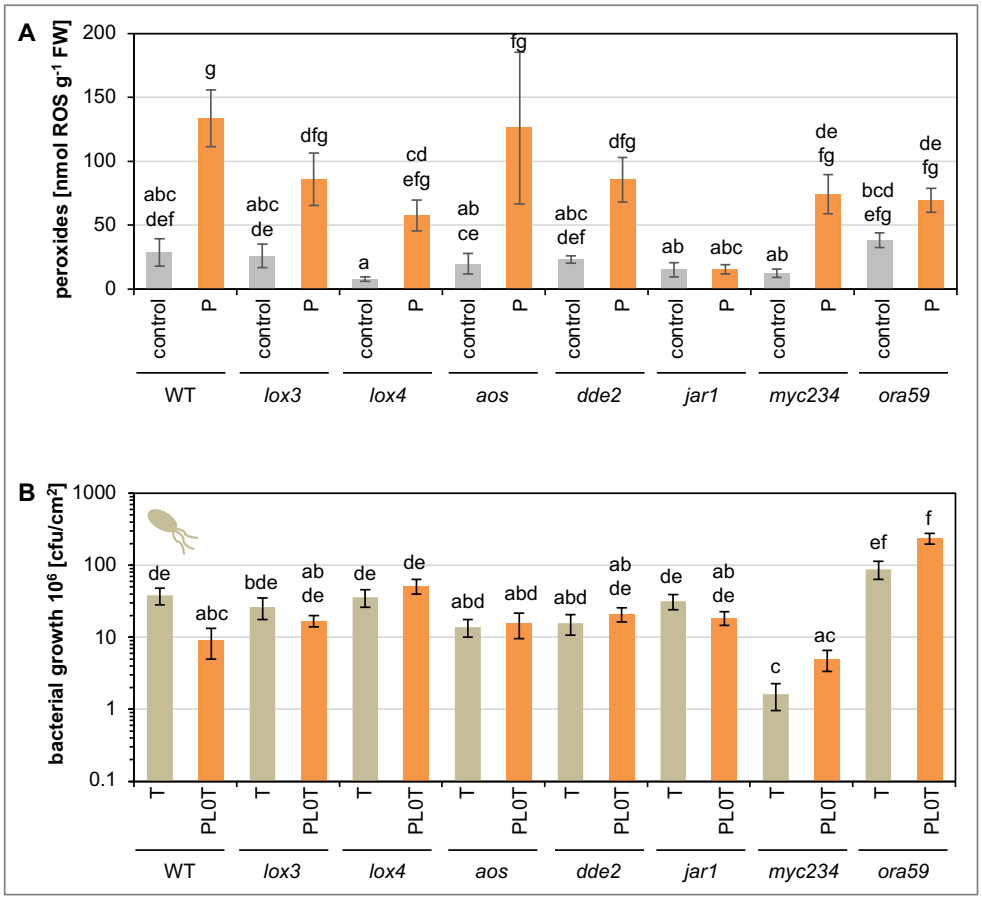

Figure 50. Activation of resistance against *Pst* DC3000 requires components of JA biosynthesis/signalling.

(A) Peroxide content in leaves of WT plants, *lox3*, *lox4*, *aos*, *dde*, *jar1*, *myc234* and *ora59* mutants at the end of the night that followed an 8 h PL period (P). **(B)** Bacterial growth in WT plants and mutants related to JA biosynthesis/signalling (*lox3*, *lox4*, *aos*, *dde*, *jar1*, *myc234* and *ora59*) infected with *Pst* DC3000 (T) and pre-treated with a prolonged light period of 8 hours directly before infection (PL0T). Values represent means (n ≥ 8 for bacterial growth experiment, n ≥ 3 for peroxide measurements) ± SE. Letters indicate statistically different groups (*p*-value ≤ 0.05) as determined by ANOVA followed by Tukey's post-hoc test. FW, fresh weight.

First, the responsiveness of the JA and/or JA-Ile biosynthesis/signalling mutants to an 8 h-PL period was analysed by measuring the peroxide content at the end of the night that followed the PL treatment (**Figure 50A**). Interestingly, peroxide levels increased in all mutants related to JA biosynthesis or signalling except *jar1* in response to an 8 h-PL period (**Figure 50A**). A similar attenuated phenotype of *jar1* mutants has already been described by Nitschke *et al.* (2016) in response to a 24 h-PL period. The lacking responsiveness to photoperiod stress of *jar1* mutants might indicate that JA-Ile biosynthesis is required for the photoperiod stress-induced increase in peroxide content in Arabidopsis. However, currently ongoing research rather indicates that a second site mutation, more specifically an insertion of GAA repeats, in the *jar1-1* background rather than impaired JA-Ile biosynthesis is responsible for the lacking responsiveness to prolongations of the light period (Anne Cortleven, personal communication).

Second, the mutants were exposed to an 8 h-PL period and infected with virulent *Pst* (without a lag phase, **Figure 50B**). In bacterial growth experiments, all mutants except *jar1* and WT exposed to a PL

period before inoculation with *Pst* (PL0T) were as strongly infected as only *Pst*-infected mutants (T, **Figure 50B**). This suggests that JA biosynthesis/signalling is required to improve the resistance against *Pst* infection by photoperiod stress, in addition to SA biosynthesis and signalling and components of SAR. The only slight induction of photoperiod stress-induced resistance against *Pst* that was observed in *jar1* indicates that JA-Ile biosynthesis is not required in this process.

3.3.9 Mild photoperiod stress induces resistance against a subsequent *Botrytis* infection in Arabidopsis plants

To explore whether photoperiod stress enhances the resistance of Arabidopsis also to other plant pathogens, WT plants were treated with an 8 h-PL period and afterwards infected with fungal spores of *B. cinerea* (without a lag phase, **Figure 51**). Evaluation of disease symptoms of infected leaves revealed that WT plants pre-treated with an 8 h-PL period (PL0T) suffered less than non-PL-treated plants (T, **Figure 51A**). Expression analysis of *PR1* showed increased transcript levels in plants infected with *Botrytis* and pre-treated with an 8 h-PL period (PL0T) compared to only *Botrytis*-infected plants (T, **Figure 51B**). Together, this indicates that mild photoperiod stress primes Arabidopsis against a subsequent *Botrytis* infection.

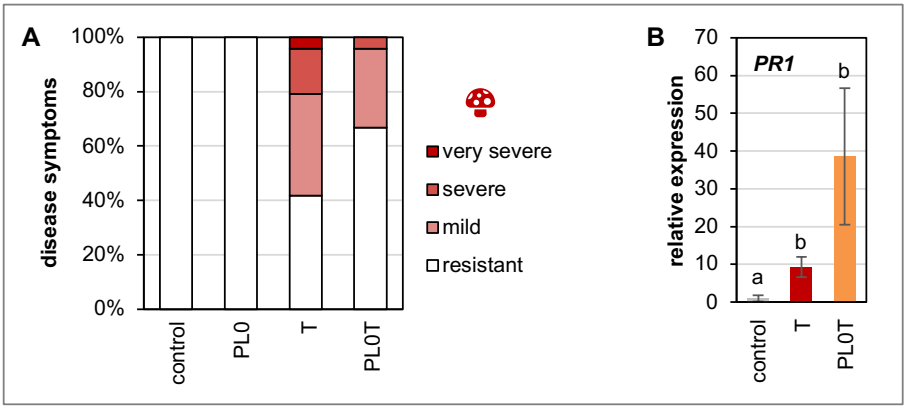

Figure 51. Mild photoperiod stress improves resistance against *B. cinerea* infection.

(A) Evaluation of disease symptoms in WT plants under control condition, treated with an 8 h PL period (PL0) or infected with *B. cinerea* (T) and pre-treated with a PL period of 8 hours (PL0T). Disease symptoms of infected leaves were evaluated 3 to 5 days post infection and categorized according to visible necrosis into 4 groups: very severe disease symptoms, whole leaf suffered from infection and leaf turned (at least in parts) yellow; severe, necrotic leaf area beneath the position of *B. cinerea* application; mild, small separated necrotic leaf areas beneath the position of *B. cinerea* application; resistant, no necrosis visible. **(B)** Relative expression of *PR1* in WT plants under control condition or infected with *B. cinerea* (T) and pre-treated with a PL period of 8 hours (PL0T). Values represent means (n ≥ 4 for expression analysis) ± SE. Expression values are expressed relative to the control (SD grown plants treated with buffer), which was set to 1. Letters indicate statistically different groups (*p*-value ≤ 0.05) as determined by ANOVA followed by Tukey's post-hoc test.

Depending on the pathogen that attacks, plants activate specific immune responses upon recognition of the pathogen. SA signalling pathways are particularly effective to defend biotrophic pathogens, such as *Pseudomonas* (Beckers & Spoel, 2006), while JA-dependent mechanisms are in general more important to defend necrotrophic pathogens (El Oirdi *et al.*, 2011), such as *B. cinerea*. Both SA- and JA-

biosynthesis/-signalling are important for the resistance induction by mild photoperiod stress against *Pst* infections (**sections 3.3.5, 3.3.8**). To assess whether photoperiod stress-induced resistance against *Botrytis* infection also requires those signalling pathways, the JA-biosynthesis mutant *dde2* and the SA-signalling mutant *npr1* were analysed (**Figure 52A**). Evaluation of disease symptoms of infected leaves showed that both *dde2* and *npr1* pre-treated with an 8 h-PL period (PL0T) developed only slightly less necrosis than directly *Botrytis*-infected mutants (T, **Figure 52B-C**). Thus, in contrast to WT plants, there is almost no improvement of resistance after priming in *dde2* and *npr1* mutants pointing to an involvement of JA biosynthesis and SA signalling in photoperiod stress-induced resistance. However, *PR1* expression increased in primed-and-triggered *dde2* mutants similar as in WT compared to directly *Botrytis*-infected *dde2* (**Figure 52D**) suggesting that JA biosynthesis is not necessary for priming of *PR1* expression. As expected, in *npr1* mutants *PR1* expression was not affected by the treatments (**Figure 52D**). Taken together, these results suggest that photoperiod stress-induced priming against *Botrytis* infection requires DDE2-mediated JA biosynthesis and NPR1-dependent SA signalling.

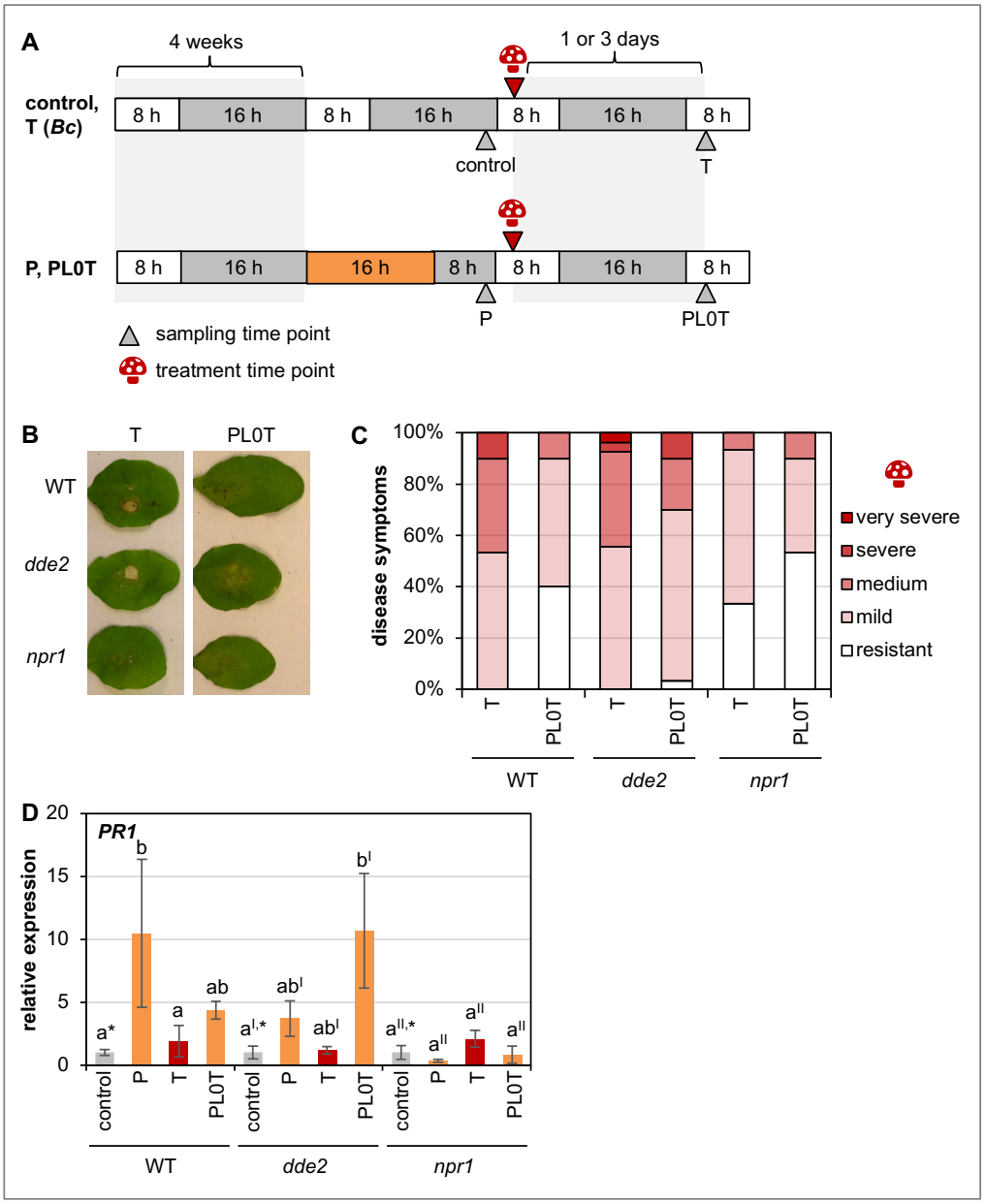

Figure 52. Activation of resistance by mild photoperiod stress against a *Botrytis* infection requires JA biosynthesis and SA signalling.

(A) Schematic overview of the experimental setup. WT plants and mutants remained under SD (control), treated with a PL period of 8 hours (P) or infected with *B. cinerea* (T) after pre-treatment with a PL period of 8 hours (PL0T). **(B)** Representative leaves of WT, *dde2* and *npr1* infected with *B. cinerea* (T) after exposure to mild photoperiod stress (induced by an 8 h-PL period, PL0T). **(C)** Evaluation of disease symptoms in WT, *dde2*, and *npr1*, infected with *B. cinerea* (T) and pre-treated with a PL period of 8 hours (PL0T). Disease symptoms of infected leaves were evaluated 3 to 5 days post infection and categorized according to visible necrosis into 5 groups: very severe disease symptoms, whole leaf suffered from infection and leaf turned yellow; severe, half leave suffered and turned in parts yellow; medium, grey necrotic leaf area beneath the position of *B. cinerea* application; mild, small separated grey necrotic leaf areas beneath the position of *B. cinerea* application; resistant, no necrosis visible. **(D)** Relative expression of *PR1* in WT, *dde2* and *npr1* treated with an 8 h-PL period (P) or infected with *B. cinerea* (T) after pre-treatment with 8 h-PL period (PL0T). Values represent means (n ≥ 3 for expression analysis) ± SE. Expression values are expressed relative to the control samples of the respective genotype, which were set to 1. Letters indicate statistically different groups ($p \leq 0.05$) as determined by ANOVA followed by Tukey's post-hoc test. Statistical analysis was performed for every genotype separately as indicated by Roman numbers. The control treatments of different genotypes were additionally compared by statistical analysis (all control treatments marked with an asterisk belong to the same statistical group).

3.3.10 NPR1 controls partly the altered gene expression caused by photoperiod stress

During abiotic and biotic stresses and developmental processes, NPR1 functions as a transcription co-activator that is sensitive to the plant redox status (Seo *et al.*, 2020). In view of the requirement of NPR1 in the photoperiod stress-induced increase of peroxides and its role in the subsequently increased pathogen resistance, further investigation of the role of NPR1 in the photoperiod stress response was of interest. To understand the genome-wide transcriptomic changes and especially the role of NPR1 under mild photoperiod stress conditions, an RNA-seq analysis, in which we compared changes of transcript abundance of WT and *npr1* treated with an 8 h-PL period, was performed (sampling according to control and 8 h-PL in **Figure 40A**).

PCA revealed that control samples of WT and *npr1* clustered together (**Figure 53A**, grey circle) indicating only small differences of transcript abundance between the two genotypes in the absence of photoperiod stress. In contrast, upon stress treatment distinct clusters were identified for WT and *npr1* indicating strong differences in their stress responses (**Figure 53A**). To get further insights, the number of genes regulated were analysed: 5727 genes (Benjamini Hochberg, BH) were regulated considering the interaction effects of genotype and treatment, 13033 genes (BH) in a genotype-dependent manner, and 3413 genes (BH) in a treatment-dependent manner.

To assess differences in the response of WT and *npr1* to an 8 h-PL period, FPKM values of the genes regulated in a genotype-dependent manner were analysed by clustering and GO term analysis. QT clustering revealed 7 clusters and one additional cluster with 102 unassigned genes (**Figure 53B**). Eighty-nine percent of all genotype-dependent regulated genes were found in clusters 1 and 2, which showed an upregulation (cluster 1) or a downregulation (cluster 2) in both genotypes at the end of the night that followed an 8 h-PL period (**Figure 53B**) indicating that the majority of genes was similarly regulated in WT and *npr1*. The analysis showed that responses to bacterial molecules, camalexin and indole phytoalexin biosynthetic and metabolic processes, cellular responses to hypoxia and decreasing oxygen levels, as well as biological processes linked to SAR and responses to salicylic acid are enriched in cluster 1 (**Figure 54A**). Similar GO terms were detected in WT in response to a PL period of 24 h (**Figure 43**). GO term analysis of cluster 2 revealed that responses to (low) light intensity or blue light as well as biological processes connected to photosynthesis and light harvesting, water or fluid transport are enriched in this cluster. Nine percent of all genotype-dependent regulated genes (clusters 3-7) were differently regulated in WT and *npr1* in response to an 8 h-PL period (**Figure 54B**) and therefore represent genes dependent on NPR1.

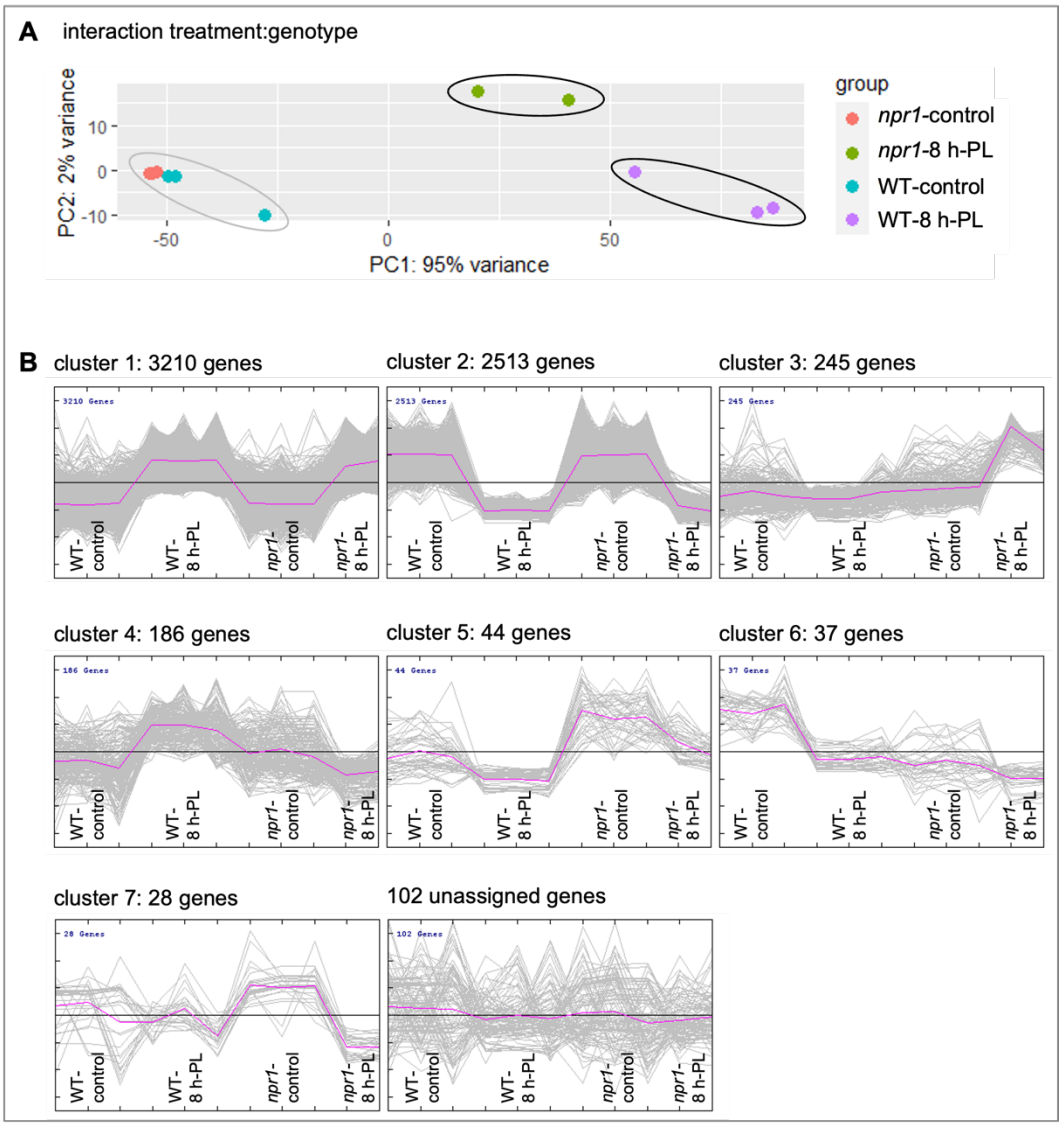

Figure 53. Analysis of FPKM values in WT and *npr1* plants exposed to an 8 h-PL period.

(A) PCA of WT and *npr1* plants under control condition and in response to an 8 h-PL period. Grey circle, control samples; black circles, samples treated with an 8 h-prolongation of the light period. **(B)** QT clustering (performed with the following parameters: diameter = 0.5, minimum cluster size = 10 genes, absolute distance = false, Pearson's correlation) resulted in seven clusters and one additional cluster with unassigned genes. Purple line, average levels of FPKM values of genes.

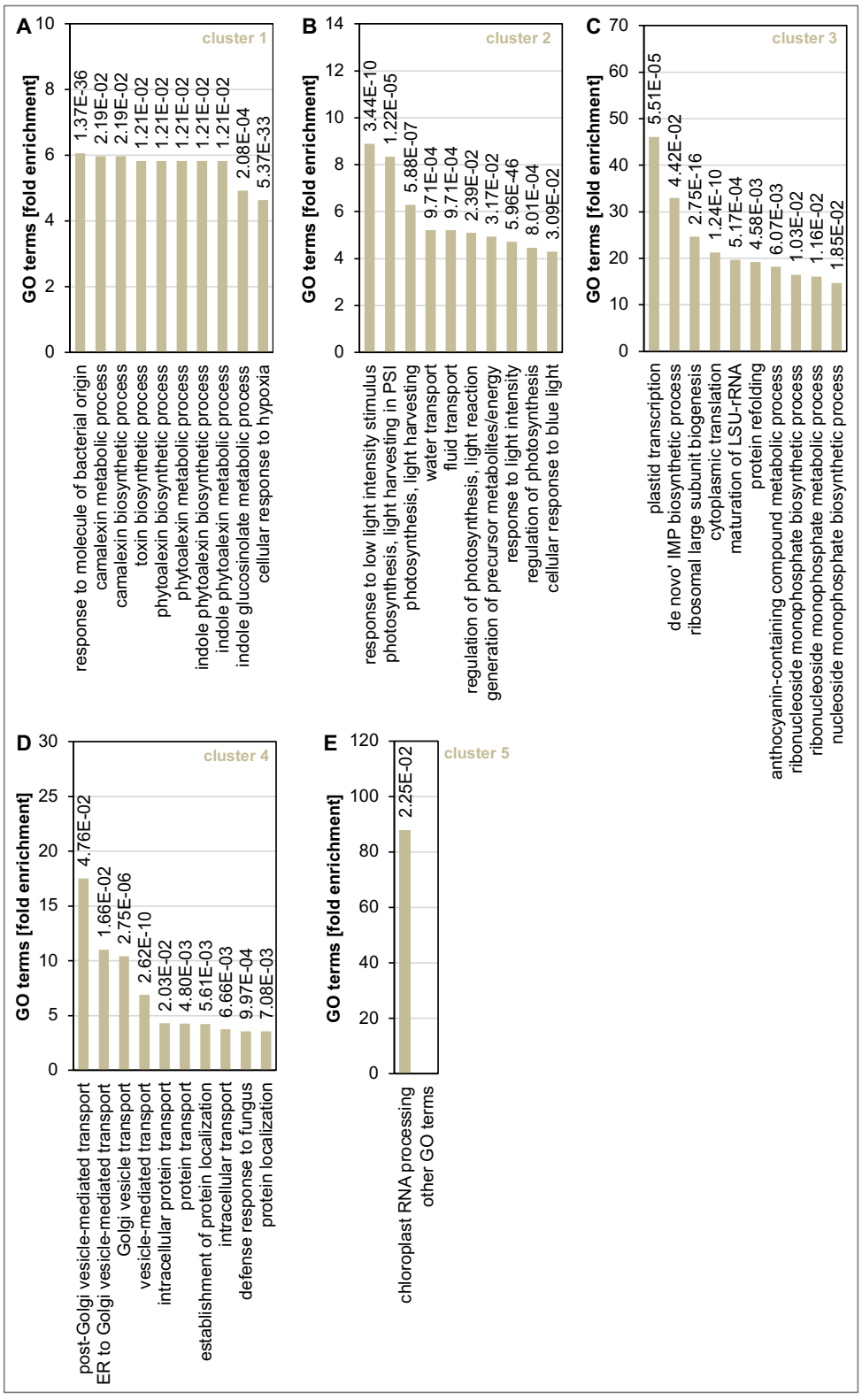

Figure 54. GO term analysis of QT clusters 1 to 5.

(A-E) Top 10 GO terms in WT plants and *npr1* mutants corresponding to genes regulated in (A) cluster 1, (B) cluster 2, (C) cluster 3, (D) cluster 4, and (E) cluster 5. Numbers above the bars represent *p*-values.

Next, pairwise comparisons were performed between photoperiod stress-treated (8 h-PL period) and control samples for each genotype (**Supplementary Table S33** and **Supplementary Table S34**). DEGs (Bonferroni-corrected values, adjusted *p*-value ≤ 0.05; log_2fold change ≥ I1I) significantly regulated only in WT-8 h-PL *vs.* WT-control or *npr1*-8 h-PL *vs. npr1*-control, as well as commonly regulated DEGs were identified. Interestingly, the number of DEGs that were significantly regulated only in WT-8 h-PL *vs.* WT-control (4703 DEGs) was considerably higher than the number of DEGs that were commonly regulated in WT-8 h-PL *vs.* WT-control and *npr1*-8 h-PL *vs. npr1*-control (2521 DEGs) or significantly regulated only in *npr1*-8 h-PL *vs. npr1*-control (759 DEGs; **Figure 55**). This is in accordance with the generally stronger responsiveness of WT plants to an 8 h-PL period.

GO enrichment analysis revealed that cellular responses to hypoxia and oxygen levels, protein refolding processes, and responses to pathogen-derived molecules (bacterial and fungal), SA responses and SAR are enriched in both genotypes (**Figure 55**). This indicates that an 8 h-PL period induces responses resembling those activated by pathogen infection in both, WT and *npr1*. The analysis also revealed that protein localization to the endoplasmic reticulum, Golgi vesicle-mediated transport, as well as aerobic respiration and photosynthesis are enriched in WT plants, while processes related to translation (involving ribosomes) are enriched in the *npr1* mutant (**Figure 55**).

Additional analysis of the pairwise comparisons (**Figure 55**) resulted in the top 20 up- and downregulated DEGs commonly regulated in WT and *npr1* (**Supplementary Table S33** and **Supplementary Table S34**), or significantly regulated in only one of the genotypes (**Supplementary Table S35** to **Supplementary Table S38**). The results indicate that genes relevant for mediating oxidative stress responses, NPR1-independent biotic stress responses, auxin-dependent responses, or the integration of light signals are commonly regulated in WT and *npr1* by an 8 h-PL treatment. The results also suggested that genes relevant for ABA biosynthesis and signalling are more strongly induced in WT by an 8 h-PL period, while genes that are temperature-responsive or encode detoxifying proteins or redox regulators, such as *FITNESS* or *F6'H1*, are predominantly affected in *npr1*. This suggests that NPR1 is important for a full activation of ABA-dependent responses induced by an 8 h-PL period in WT plants. Taken together, the genome-wide transcriptomic analysis suggests that NPR1 is important for parts of the response to an 8 h-PL period.

Taken together, the genome-wide transcript analysis suggests that NPR1 contributes to the altered gene expression in response to an 8 h-PL period. However, it seems that destabilization of NPR1 in *npr1* mutants (Cao *et al.*, 1997) does not result in complete unresponsiveness to mild photoperiod stress, as most genes are similarly regulated in WT and *npr1* mutants.

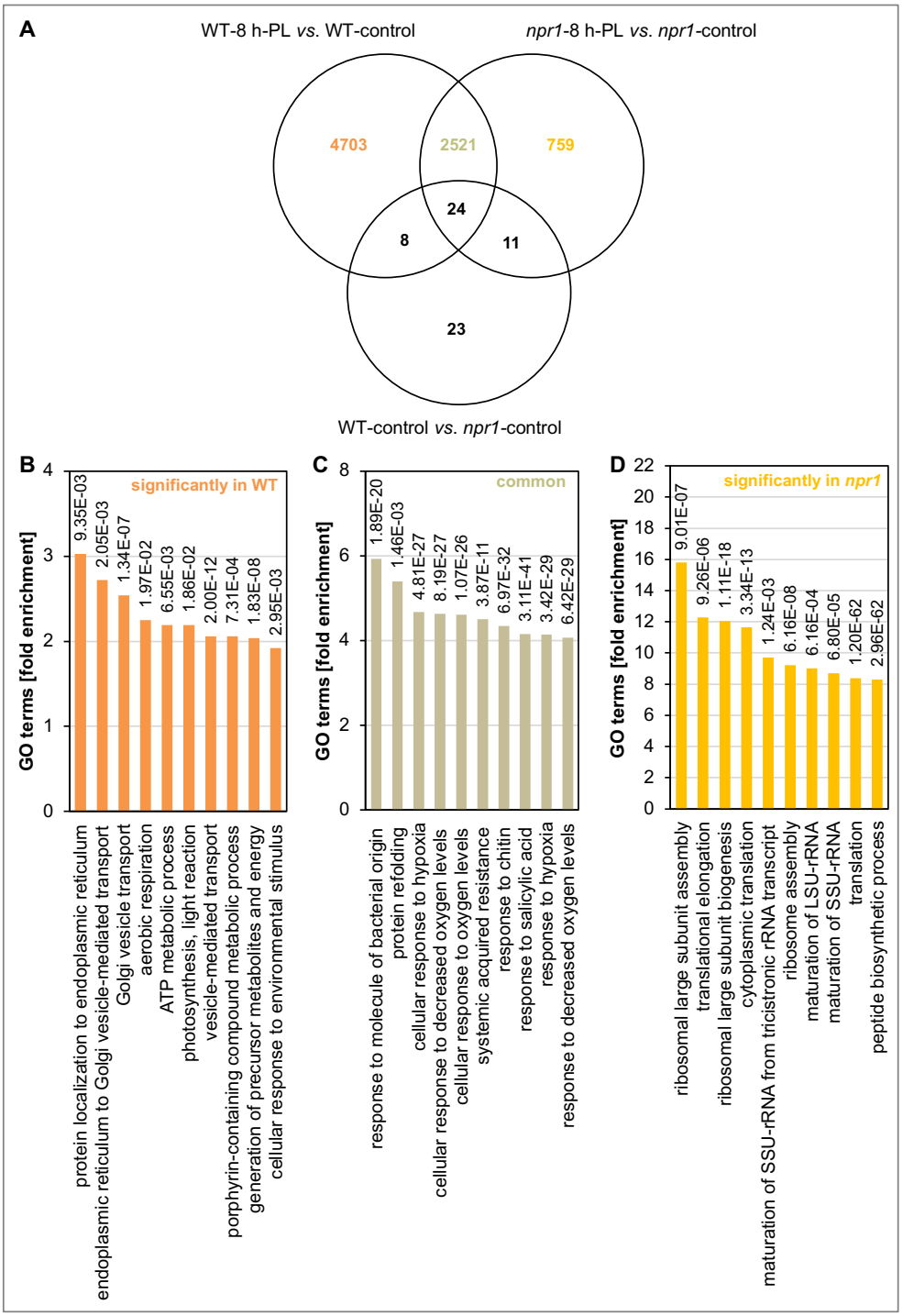

Figure 55. Analysis of genes significantly regulated in WT plants and *npr1* mutants treated with a PL period of 8 h.

(A) Venn diagram comparing DEGs in WT plants and *npr1* treated with a prolonged light of 8 h in comparison to control conditions (Bonferroni-corrected values, adjusted *p*-value ≤ 0.05; $\log_2$fold change ≥ l1l). **(B-D)** Top 10 GO terms of DEGs that are regulated in (B) WT-P *vs.* WT-control, (C) both WT-P *vs.* WT-control and *npr1*-P *vs. npr1*-control in response to a PL period of 8 h, and (D) *npr1*-P *vs. npr1*-control. Numbers above the bars represent *p*-values.

3.3.11 *FITNESS* and *ACBP3* are not required for the response to mild photoperiod stress

Under mild photoperiod stress, NPR1 is important as a transcription regulator. To get a better insight, how transcription in response to mild photoperiod stress is altered in *npr1* mutants, the top 20 regulated DEGs in *npr1* were considered in more detail (**Supplementary Table S37**). The aim was to identify genes strongly regulated specifically in *npr1* but not WT plants in response to an 8 h-PL period. Besides others, *FITNESS, TRM7C* and *AKR4C9* were identified as significantly responsive genes in *npr1* mutants in the RNA-seq analysis (**Supplementary Table S37**). Further analysis by qRT-PCR revealed that these are indeed particularly strongly induced by an 8 h-PL period in *npr1* mutants (**Figure 56A-E**), thereby confirming the RNA-seq data.

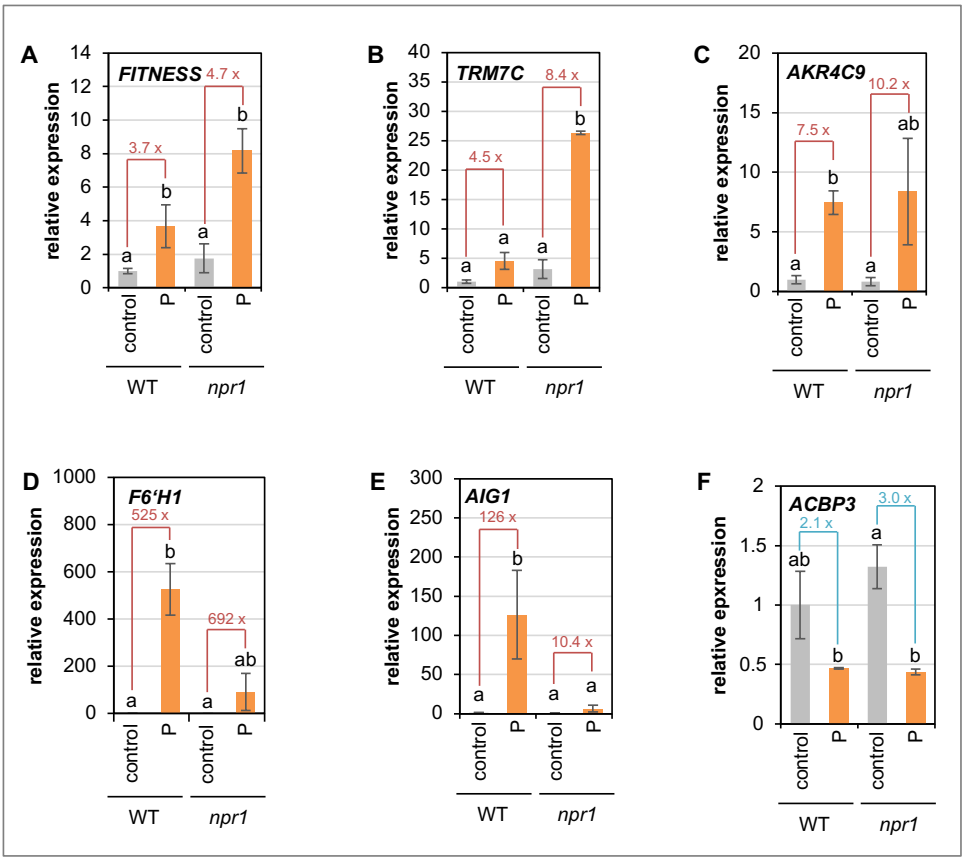

Figure 56. Evaluation of RNA-seq analysis by qRT-PCR.

(A-F) Relative expression of (A) *FITNESS*, (B) *TRM7C*, (C) *AKR4C9*, (D) *F6'H1*, (E) *AIG1*, and (F) *ACBP3* in WT plants and *npr1* mutants in response to an 8 h-PL period (analysed by qRT-PCR). Red and blue values indicate increase and decrease in fold change, respectively. All values are expressed relative to WT control samples, which were set to 1. Error bars represent SE (n ≥ 3). Letters indicate statistically different groups ($p \leq 0.05$) as determined by ANOVA followed by Tukey's post-hoc test. Related RNA-seq data are shown in **Supplementary Table S37**.

For most genes identified among the top 20 most strongly upregulated DEGs in *npr1* in response to an 8 h-PL period there is only limited information available so far. Further analysis was performed for genes for which literature was available: *FITNESS* encodes a protein with a single CONSTANS, CONSTANS-like and TOC1 (CCT)-domain that maintains cellular redox homeostasis, negatively regulates seed yield as well as NPR1-dependent resistance against *Pst* infection (Osella *et al.*, 2018; Mengarelli *et al.*, 2021).

To investigate if *FITNESS* might also be important for the increase of the peroxide content under mild photoperiod stress, the *fitness* mutant was exposed to an 8 h-PL period (**Figure 57**). The peroxide levels were similar in WT plants and *fitness* mutants after exposure to an 8 h-PL period (**Figure 57**) indicating that *FITNESS* is not required for the photoperiod stress-induced increase in peroxide content.

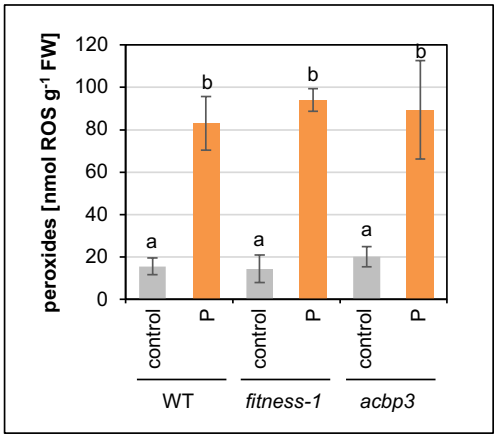

Figure 57. Mutation of *FITNESS* or *ACBP3* does not affect the responsiveness to mild photoperiod stress.

Peroxide content in leaves of WT plants, *fitness*, and *acbp3* mutants at the end of the night following an 8 h-PL period (P). Values represent means (n ≥ 4 for expression analysis) ± SE. Letters indicate statistically different groups (*p*-value ≤ 0.05) as determined by ANOVA followed by Tukey's post-hoc test. FW, fresh weight.

Two other genes among the top 20 significantly upregulated DEGs in *npr1* were tested, namely *F6'H1* and *AIG1* (**Supplementary Table S37**). Although there is currently limited information available about their relevance in biological processes available, expression of both genes is enhanced in *ACBP3*-overexpressing Arabidopsis plants (Xiao & Chye, 2011). *ACBP3* belongs to a group of six *ACBP* genes in Arabidopsis that encode acyl-coenzyme A (CoA)-binding proteins. Interestingly, overexpression of *ACBP3* improves the NPR1-dependent resistance against *Pst* infection (Xiao & Chye, 2011). As there was an induction of both *F6'H1* and *AIG1* in response to an 8 h-PL period (**Figure 56**), it was analysed if this increase was linked to higher expression of *ACBP3* under mild photoperiod stress (**Figure 56**). However, expression analysis revealed the opposite: An 8 h-PL period lowered the expression of *ACBP3* in WT and *npr1* mutants (**Figure 56**). These results suggest that enhanced expression of *F6'H1* and *AIG1* in response to an 8 h-PL period in WT and *npr1* is not due to an enhanced *ACBP3* expression. The increase in peroxide content in *acbp3* mutants after an exposure to an 8 h-PL period was similar to that in WT plants (**Figure 57**), indicating that *ACPB3* is, like *FITNESS*, not required for the photoperiod stress-induced increase in peroxide content.

Another approach for further investigation of genes relevant for the lacking response to PL periods in *npr1* is the analysis of DEGs that are specifically regulated in WT but not *npr1* (**Supplementary Table S35** and **Supplementary Table S36**). Since such a regulation is applicable to more than 4000 DEGs (**Figure 55A**), they were not considered in detail in this work. A first analysis of the genes was performed in **chapter 3.3.10**.

3.3.12 Mild photoperiod stress affects transcript abundance of salicylic and jasmonic acid biosynthesis/signalling genes in WT and *npr1*

Strong photoperiod stress alters the expression of several SA and JA biosynthesis/signalling genes (Cortleven *et al.*, 2022). Here, it was analysed whether this also occurs in response to mild photoperiod stress. To this end, the RNA-seq dataset of WT plants exposed to an 8 h-PL period (WT-P *vs.* WT-control) was employed (**Supplementary Table S39** and **S40**).

Expression of approximately one-third of SA-related genes was upregulated in WT plants by an 8 h-PL period (**Supplementary Table S39**). Among them were SA biosynthesis genes (including *PAD4*, *ICS1*), *WRKY* transcription factor genes (including *WRKY8/28/70*), *NPR* genes (*NPR1/3/4*), and genes encoding interactors of NPR1 including several TGAs (*TGA1/4/5/6*) and NIMIN (*NIMIN1/2*) proteins (**Supplementary Table S39**). Furthermore, one-fifth of the SA-related genes were downregulated by the stress treatment (**Supplementary Table S39**). Among these were found SA biosynthesis genes (including *ENHANCED DISEASE SUSCEPTIBILITY1* (*EPS1*)), cinnamic acid biosynthesis genes (including *PHENYLALANINE AMMONIA LYASE3/4* (*PAL3/4*)) and genes encoding NPR1 interactors (including *TGA2*, *NIMIN3*) (**Supplementary Table S39**)).

Analysis of JA-related genes revealed that half of them were upregulated after stress treatment in WT (**Supplementary Table S40**). Among them were several JA biosynthesis genes (including *LOX3*, *OPR3*), the JA-Ile biosynthesis gene *JAR1* as well as genes encoding JA signalling proteins (including *JAZ1/2/5/6/7/8/10/12*, *MYC3*) (**Supplementary Table S40**). In addition, several other JA genes were downregulated, such as *JAZ9/11*, *FAD3/4* or *MYC4* (**Supplementary Table S40**). These results show that both SA and JA biosynthesis/signalling are regulated by mild photoperiod stress in Arabidopsis WT plants.

As responses to SA and JA/ET are both mediated by NPR1 (Backer *et al.*, 2019), the regulation of the SA- and JA-related genes by photoperiod stress in *npr1* was compared to WT. The analysis revealed that most SA-related genes are similarly regulated in *npr1* and WT plants (**Supplementary Table S40**). However, a set of nine genes (including e.g. *TGA4/5*) were detected to be differently regulated in WT and *npr1*, showing an upregulation in WT and a downregulation in *npr1* (**Supplementary Table S40**). Also, most of the JA-related genes were similarly regulated in *npr1* and WT (**Supplementary Table S40**). However, two sets of genes showed an opposite response in *npr1* and WT (**Supplementary Table S40**). One of them included genes upregulated in WT, but downregulated in *npr1*, such as *JAR1* and *NINJA*. The second set included genes downregulated in WT, but upregulated in *npr1*, such as *JAZ9* and *FAD8* (**Supplementary Table S40**).

Taken together, the analysis of SA- and JA-related genes supports the notion that the regulation of SA and JA biosynthesis/signalling genes in response to mild photoperiod stress is in part dependent on NPR1.

3.3.13 Mild photoperiod stress regulates genes related ROS signalling in WT and *npr1*

To investigate if mild photoperiod stress affects the expression of genes related to ROS signalling, the RNAseq dataset of WT plants exposed to an 8 h-PL period was used for analysis of genes related to ROS (**Supplementary Table S41**). One-third of the ROS-related genes are significantly regulated by photoperiod stress (**Supplementary Table S41**). Among them are several thioredoxin family genes, *NADPH OXIDASE* genes (including *RBOHF*, *RBOHD*), some *PEROXIDASE* genes (*PER4/28/33/34/46/49/70*), *ASCORBATE PEROXIDASE* genes (*APX1/3*), *GLUTATHIONE PEROXIDASE2* (*GPX2*), *CAT1*, and *AOX1A* (**Supplementary Table S41**). The most strongly regulated genes were *PER33*, AT1G28480, *MDAR2*, *RBOHC* and *PER4*, which were at the end of the night following an 8 h-PL treatment at least 7-fold (log_2fold change) induced in WT-P *vs.* WT-control. Among the downregulated ROS-related genes, several ferric-chelate reductase genes, numerous *PEROXIDASE* genes (*PER11/12/16/18/20/26/36/42/43/45/63*), *APX2/6*, and the catalase genes *CAT2/3* were found (**Supplementary Table S41**). Most strongly downregulated were the genes *PER39*, AT5G49730, *PER1/16/32* showing at least a 4-fold (log_2fold change) downregulation in WT-P *vs.* WT-control. In summary, the analysis suggests that an extensive regulation of genes related to ROS signalling occurs in response to mild photoperiod stress.

Many of the ROS-related genes were similarly regulated in WT and *npr1* plants (**Supplementary Table S41**). However, one gene, AT1G32350 encoding the *ALTERNATIVE OXIDASE 1D* (*AOX1D*), was particularly strongly induced in *npr1*, namely 25-fold (log_2fold change) in *npr1*-P *vs. npr1*-control compared to 7-fold in WT-P *vs.* WT-control (**Supplementary Table S41**). ROS-related genes, such as *PER4* or *PER33*, previously shown to be strongly induced at the end of the night following a 24 h-PL period (Abuelsoud *et al.*, 2020), were stronger upregulated in WT-P *vs.* WT-control after an 8 h-PL period compared to *npr1*-P *vs. npr1*-control (**Supplementary Table S41**). The altered regulation of ROS-related genes in *npr1* might be connected to the lacking accumulation of peroxides in the mutants (**section 3.3.5**).

Taken together, the results indicate that expression of ROS-related genes during the response to mild photoperiod stress is dependent on NPR1. Clearly, the analysis of the transcriptomic response showed that NPR1 did not function as the “one-and-only” signalling component mediating responses to mild photoperiod stress.

4 Discussion

4.1 Sensitivity of Arabidopsis to PL periods

As a continuation of previous research studying photoperiod stress in Arabidopsis induced by a 24 h-PL period (Nitschke, 2014; Nitschke *et al.*, 2016; Frank, 2019; Abuelsoud *et al.*, 2020; Frank *et al.*, 2020; Cortleven *et al.*, 2022), this work investigated the effects of PL periods shorter than 24 h on the strength of the photoperiod stress response in SD-grown Arabidopsis plants.

4.1.1 Effects of PL periods of different durations on the Arabidopsis transcriptome

In the first part of the thesis, the sensitivity of the transcriptome of WT Arabidopsis plants to PL periods of different durations was investigated (**section 3.1**). The research demonstrated that already a PL period of 1 h was sufficient to affect the transcript level of numerous genes at the end of the night that followed the PL period (**Figure 22**) indicating a high sensitivity of Arabidopsis to changes in the light-dark rhythm. This is consistent with previous reports highlighting that plants adjust their life processes to daily fluctuations of the photoperiod (Shim & Imaizumi, 2015; Nitschke *et al.*, 2017; Adole *et al.*, 2019). Since the work presented here, considers only responses and numbers of regulated genes at a single time point, transiently regulated genes were most likely not discovered. The most strongly regulated genes in response to a 1 h-PL period might be interesting candidates for the analysis of PL periods even shorter than 1 h aiming to elucidate the effects of PL periods in the range of minutes, as they occur as daily changes in the light-dark rhythm. Especially genes important for circadian regulation, light signalling and redox regulation were induced by a 1 h-PL period in WT plants (**Supplementary Table S3**), while genes connected to auxin biosynthesis and signalling were downregulated (**Supplementary Table S4**). This indicates that already a 1 h-PL period influences genes connected to similar processes as those found for the response to a 24 h-PL period, such as the plant circadian clock, redox or auxin status (Cortleven *et al.*, 2022) although their number is much lower. It is also consistent with findings of Nitschke *et al.* (2016), Abuelsoud *et al.* (2020) and Frank *et al.* (2020) who used a 24 h-PL period indicating the relevance of the clock, redox status and auxin for the response to photoperiod stress. It is known that the plant circadian clock regulates the transcription of auxin-related genes (Covington & Harmer, 2007) and genes connected to the plant redox state (Nitschke, 2014; Guadagno *et al.*, 2018). As photoperiod stress causes a dysregulation of the circadian clock, it might be that this then causes the altered transcript levels of auxin-related and ROS-related genes.

The strength of the responses of plants to PL periods is influenced by several parameters, including the light-dark rhythm before exposure to a PL period, the duration of the PL period itself and the subsequently dark period (Nitschke *et al.*, 2017). In this work it was shown that the total number of genes differentially expressed in response to PL periods increased when the PL period was extended (**Figure 22**). Similarly, the peroxide content and the expression of photoperiod stress marker genes *ZAT12* and *BAP1* increased when Arabidopsis plants were exposed to longer PL periods (Abuelsoud *et al.*, 2020). Already a 4 h-PL period differentially regulated more than 3000 genes in WT Arabidopsis (**Figure 22**) suggesting a strong regulation. Most strongly upregulated were amongst others in WT *CYP76C5*, *CYP81G1*, *FMO1*, *ERF71*, and *ALD1*, while *IAA29*, *SAUR22* were strongly downregulated

(**Supplementary Table S7** and **Supplementary Table S8**). Strong effects of a PL period shorter than 24 h were also reported by previous research showing that a single LD is sufficient to induce flowering in SD-grown Arabidopsis (Corbesier *et al.*, 1996).

Comparisons of transcriptomic changes in response to PL periods with different durations revealed three genes, *PIF4*, *BIM1* and *COR413IM1*, that were differentially regulated independent of the length of the PL period (**Figure 23**). In response to PL periods of 1 h to 8 h, the expression of all three genes was downregulated (**Supplementary Table S9**). An 1 h-PL period was sufficient to downregulate *PIF4*, *BIM1* and *COR413IM1* at least one-fold (log_2fold change, **Supplementary Table S9**) in comparison to control. Longer PL treatments of 2.5 h to 8 h further downregulated the transcript abundance of *PIF4* and *COR413IM1* but not *BIM1*.

The consistent regulation of these genes in response to PL periods of different durations raised the question of the functional relevance underlying their regulation.

PIF4 encodes a basic helix loop helix (bHLH) TF interacting with phytochromes and cryptochromes (Huq & Quail, 2002). Overexpression of *PIF1*, belonging to the same bHLH superfamily in Arabidopsis (Zhu *et al.*, 2016), suppresses the response to a 24 h-PL period (personal communication, Ishita Bajaj). The expression of *PIF4* is negatively regulated by the evening complex that is a component of the circadian clock in Arabidopsis (Nusinow *et al.*, 2011; Xu & Zhu, 2021). Since changes in the light-dark rhythm affect the circadian clock (Nitschke *et al.*, 2016), the effects of PL treatments on the circadian clock including the EC might cause a downregulation of *PIF4* expression. The PIF4 protein, whose activity and stability are repressed under light, represents an important regulator of plant growth integrating both light and hormonal signals (Choi & Oh, 2016). In the view of hormonal signalling, PIF4 positively regulates the transcription of auxin biosynthesis and signalling genes, such as *IAA29* (Choi & Oh, 2016). Negative regulation of PIF4 by PL periods might result in the downregulation of numerous auxin biosynthesis and signalling genes as was observed in response to PL periods of different durations (**Supplementary Table S4**, **Supplementary Table S6**, **Supplementary Table S8**). During photoperiod stress responses of plants, auxin stimulates the formation of ROS, which is counteracted by CK, especially root-derived *trans*-zeatin, acting through AHK3, ARABIDOPSIS HISTIDINE PHOSPHATASE2 (AHP2) AHP3/5 and ARR2/10/12 (**Figure 7**) (Frank *et al.*, 2020; Frank *et al.*, 2022). The negative regulation of auxin biosynthesis by CK during photoperiod stress might be regulated by PIF4, since it has been reported by Di *et al.* (2016) that PIF4 regulates auxin biosynthesis in a CK-dependent manner in Arabidopsis roots. The effects of CK on PIF4 might be mediated through AHK3 and ARR12, since in roots *PIF4* transcription is positively regulated by treatment with *trans*-zeatin involving AHK3 and ARR1/12 (Di *et al.*, 2016). However, if this is indeed true for plant responses to photoperiod stress remains to be investigated, for example with an analysis of respective mutants.

BIM1 encodes a bHLH TF interacting with UVR8 photoreceptors (Liang *et al.*, 2018) and cryptochromes (Wang *et al.*, 2018b). CRY2 is required for a response to photoperiod stress (personal communication, Ishita Bajaj). The BIM1 protein positively mediates BR signalling genes by interaction with BRI1-EMS SUPPRESSOR1 (BES1) (Wang *et al.*, 2018b) belonging to the same protein family as BZR1 (Shi *et al.*, 2022) which is also modulated by PIF4 (Xu & Zhu, 2021). In addition to its regulation of BR signalling, BIM1 has been hypothesized to be negatively regulated by phytochromes and to stimulate shade

avoidance responses, similar as PIF4, by positively regulating auxin biosynthesis (Roig-Villanova & Martinez-Garcia, 2016). The downregulation of *BIM1* in response to PL periods of different durations might contribute (together with the downregulation of *PIF4*) to a repression of auxin biosynthesis and signalling genes observed in this study. However, this needs to be further investigated in the context of photoperiod stress.

COR413IM1 encodes an integral inner envelope membrane protein of chloroplasts whose expression is induced by abscisic acid or cold treatments (Okawa *et al.*, 2008). It is so far not clear how a regulation of *COR413IM1* might be of functional relevance for plant responses to PL periods.

4.1.2 A PL period of 4 h induces an oxidative stress-like response in Arabidopsis

As an extension of previous research performed by Abuelsoud *et al.* (2020), this work investigated the oxidative stress-like response of Arabidopsis WT plants to a PL period of 4 h (**Figure 24**). The experiments demonstrated that a 4 h-PL period is sufficient to regulate ROS-related genes (**Supplementary Table S10**). In addition, peroxides accumulated, and the enzyme activity of catalase decreased in WT plants (**Figure 24**) indicating that a 4 h-PL induces an oxidative burst-like response in WT. This is consistent with observations made in response to a PL period of 24 h showing that strong photoperiod stress induces an oxidative burst-like response that is connected to an induction of ROS-related genes, such as peroxidase genes, and decreased catalase activities (Abuelsoud *et al.*, 2020; Cortleven *et al.*, 2022). However, the oxidative burst-like response to a 4 h-PL period was in parts different to the response to a 24 h-PL period: MDA levels and the enzyme activity of guaiacol peroxidase were not affected by a 4 h-PL (**Figure 24**), although a 24 h-PL increased both (Abuelsoud *et al.*, 2020). This suggests that the magnitude of the oxidative burst-like response induced by PL periods might be determined by the duration of the PL period.

4.2 *Cis*-priming and memory by photoperiod stress

In the second part of the thesis, the effects of PL periods on plant responses to future PL treatments, i.e. priming, were investigated (**section 3.2**). From previous research it is known that the light environment regulates plant responses to several abiotic and biotic stresses (Roeber *et al.*, 2021; Cortleven, 2022) and in addition light signals can prime the response of plants to stress (Han *et al.*, 2019; Holness *et al.*, 2023).

4.2.1 *Cis*-priming and memory by photoperiod stress involves oxidative stress-like responses in Arabidopsis

To investigate priming and memory by photoperiod stress, a PL period of 4 h was used, since priming can in many cases be induced by low doses of a stress. The research demonstrated that induction of photoperiod stress marker genes *ZAT12* and *BAP1* and accumulation of peroxides in response to a 4 h-PL period did not occur after a second PL period (**Figure 24**). In accordance with this, a decrease in

catalase activity was not detected anymore after a second PL treatment (**Figure 24**). Such altered responses are characteristic for primed plants (Hilker & Schmülling, 2019; Liu *et al.*, 2022) indicating that a 4 h-PL period primes Arabidopsis for future PL treatments. Priming by light involving the plant redox system has also been previously described in Arabidopsis by Han *et al.* (2019). Red light signals perceived by phyB induce the transcription of *APX2* encoding an antioxidant enzyme that detoxifies ROS, thereby improving the tolerance of Arabidopsis to heat (Han *et al.*, 2019). The lack of a response in primed plants to a second 4 h-PL period as it is the case in this work contrasts observations made for other stress combinations where priming is often associated with a stronger response to a second stress (Hilker & Schmülling, 2019).

The inability to respond to a second PL period after a lag phase (PL1T) suggests that a previous 4 h-PL is memorized over a stress-free time. Memory in plants requires some kind of storage of information that can be for example achieved by transcriptional priming, epigenetic regulation, conformational or quantitative changes of proteins, or effects on hormones or metabolites (Hilker & Schmülling, 2019). Since different plant responses, including photoperiod stress marker gene expression, the peroxide content and catalase enzyme activity, remained unaffected in response to a 4 h-PL period after pre-treatment with a similar PL period (**Figure 24**), it was concluded that WT plants memorize a previous PL period by different parameters. The experiments suggested that the memory of a 4 h-PL period that is connected to photoperiod stress marker gene expression persisted over a lag phase of seven days in WT plants, while the memory associated to the peroxide content lasted a few days less (**Figure 28**). A similar discrepancy in the length of memory in dependence of the analysed parameter has also been shown for example in response to heat stress (Liu *et al.*, 2018).

A PL period up to two hours was not memorized in WT plants (**Figure 25**) suggesting that the first PL period needs to induce noticeable response to affect plant responses to a subsequent PL period in order to be memorized. In case of priming and memory by photoperiod, Arabidopsis plants become insensitive through the priming when exposed to a second stress. Thereby photoperiod stress differs from other abiotic environmental stimuli that prepare plants by a mild exposure to subsequent severe stress conditions leading to stronger and faster responses after priming when exposed to the second stimulus (Hilker & Schmülling, 2019). For example, non-freezing temperatures as well as moderate temperatures improve performance of Arabidopsis to subsequent more severe cold or heat stresses, respectively (Zuther *et al.*, 2019; Olas *et al.*, 2021). Similar observations were also made in the context of drought and osmotic stresses (Suzuki *et al.*, 2014; Hilker & Schmülling, 2019).

Experiments with the photoperiod stress-sensitive mutants *ahk2 ahk3* and *cca1 lhy* showed that both mutants memorized a 4 h-PL period shorter than WT plants (**Figure 29**, **Figure 30**). This suggested that CK perception through AHK2/3 and/or a functional circadian clock including *CCA1* and *LHY* are required for maintaining the memory of a previous PL treatment over longer time periods. The shorter memory length that was observed in both mutants might be linked to a lowered expression of *CCA1* and *LHY*, which the mutants have in common (Nitschke *et al.*, 2016; Cortleven *et al.*, 2022). However, this is not the only link between the plant circadian clock and cytokinin: Several genes that are regulated by the circadian clock are also under control of cytokinin (Singh & Mas, 2018) making it possible that the regulation of commonly targeted genes is required for maintaining the memory state.

4.2.2 Importance of the plant developmental phase for priming by photoperiod stress

The developmental phase influences the ability of Arabidopsis plants to respond to photoperiods (**Figure 2**) (Matsoukas, 2014). Previous research by Nitschke (2014) and Frank (2019) showed that the sensitivity and responsiveness of Arabidopsis to photoperiod stress depends on the plants age. As an extension to their research, I have studied the transcriptomic response of WT plants with different ages ranging from 24 DAG to 35 DAG to a 4 h-PL period (**Figure 38**). Few DEGs existed that were only regulated at certain DAG by a 4 h-PL period; however, most DEGs were regulated in response to a 4 h-PL period at different days after germination (**Figure 38**) suggesting that the majority of the transcriptome regulated by 4 h-PL period in Arabidopsis does not depend on the plants' age.

Analysis of the oxidative stress response in younger and older plants demonstrated that the responsiveness to a 4 h-PL period was influenced by the developmental phase, since only plants at 21 DAG to 35 DAG, corresponding to an age of three to five weeks, responded to the 4 h-PL treatment (**Figure 26**). Further experiments showed that the response to a second 4 h-PL period was only suppressed in plants of a certain age, namely those that were three to four-week-old when primed (**Figure 26**), suggesting that the developmental state influences *cis*-priming by PL periods. This is in accordance with previous implications (**section 4.2.1**) that the first PL period needs to induce a noticeable response to prime Arabidopsis plants, i.e. plants of a certain age that were not responding to the PL treatment were also not primed. A possible explanation might be that WT plants at 14 DAG are still in their juvenile developmental phase and thus not responsive to photoperiods, as reported by Matsoukas (2014). Likewise, plants in the reproductive developmental phase are also unresponsive to photoperiods, while plants in the adult developmental phase are responsive to photoperiods (Matsoukas, 2014). The strong induction of *SAG12*, which is known to be expressed in senescing tissues (James *et al.*, 2018), in analysed WT plants at 39 DAG (**Figure 27**) suggests the progression to the reproductive phase. A similar influence of plant age on priming has been previously reported in the context of other abiotic and biotic stresses (**section 1.5.2**): Leuendorf *et al.* (2020) showed that night-time cold treatments improved the freezing tolerance of Arabidopsis seedlings at 14 DAG and 21 DAG, but not 7 DAG. Valsamakis *et al.* (2022) published that the effects of egg priming depend on the developmental stage but not on the chronological age of the plants, since deposition of *P. brassicae* eggs primes Arabidopsis against larval feeding during their vegetative phase but not during their reproductive phase. The transcriptional status that Arabidopsis acquires in response to the deposition of *P. brassicae* eggs during their vegetative phase resembles the transcriptome of plants during their reproductive phase without any stimulus (Valsamakis *et al.*, 2022).

4.2.3 Transcriptomic memory of photoperiod stress in WT plants

An altered response of changes in gene expression to stress can be indicative for the establishment of a memory (**Figure 17**) (Hilker & Schmülling, 2019). Comparisons of the transcriptomic profiles in response to 4 h-PL periods as priming and triggering stimuli were performed to investigate the transcriptomic memory of photoperiod stress in Arabidopsis (**section 3.2.6**).

Different patterns of gene expression were considered to identify potential genes indicative for a memory of photoperiod stress:

(i) In total 1332 genes were regulated in response to priming, triggering and PL1T, but not in primed plants after returning to SD (**Figure 37**). Genes in this subset that return to non-induced expression in PL1 (during the memory phase) and are differently induced when exposed to the triggering stimulus after priming represent candidates for further investigation of the transcriptomic memory of photoperiod stress. The photoperiod stress marker genes *ZAT12* and *BAP1* used in this study to evaluate the transcriptomic memory of photoperiod were detected in this subset. In addition, *PIF4*, *COR413IM1* and several auxin-related genes, such as *IAA29* and *SAUR22/23*, were found in this subset. Expression of *PIF4* and auxin biosynthesis genes is connected to transcriptomic memory of thermomorphogenesis (Xie *et al.*, 2021). Epigenetic modifications of *PIF4* during thermomorphogenesis are linked to stress memory (Xie *et al.*, 2021). Since *PIF4* and *COR413IM1* are responsive to PL periods of different durations, analysis of their expression might be interesting to further investigate the effects of priming by PL periods shorter than 4 h. Besides, also the gene *HEAT SHOCK TRANSCRIPTION FACTOR A2* (*HSFA2*) encoding a transcription factor known to positively regulate memory of heat stress (Friedrich *et al.*, 2021; Xie *et al.*, 2021; Sedaghatmehr *et al.*, 2022) was detected in this subset. HSFA2 prolongs the acquired thermotolerance (Charng *et al.*, 2007).

(ii) A second subset of 825 genes was identified that were regulated by priming and triggering stimuli, but not during the memory phase or in response to PL1T (**Figure 37**). Further investigation of genes in this subset might be interesting to identify genes with a sustained expression, i.e. genes that are regulated by priming, remain at high levels during the memory phase and become insensitive to a second PL period. The pathogen-responsive gene *PR1* is included in this subset showing an upregulation in response to priming or triggering, and a high regulation during the memory phase (PL1) that was not further affected by a second 4 h-PL. Expression of *PR1* is often potentiated in response to pathogen infections in plants previously primed by chemicals (Hönig *et al.*, 2023). A stronger induction of *PR1* after pathogen infection in plants primed by photoperiod stress was also observed in this work. Besides, the gene *CLAVATA1* (*CLV1*) known to be associated with transcriptomic memory of heat stress in the shoot apical meristem of Arabidopsis (Olas *et al.*, 2021; Zuo *et al.*, 2023) was detected in this subset.

(iii) A third subset contained 21 genes that were regulated by priming, triggering, PL1T and PL1 (**Figure 37**). To identify memory genes, i.e. genes up- or downregulated by priming, regulated during the memory phase, and affected by an exposure to a second stimulus, an analysis of genes in this subject was of interest; *BIM1* and *PR5* were among them. *PR5* was previously used as a priming marker gene by Valsamakis *et al.* (2022).

(iv) A fourth subset of 85 genes contained genes regulated in response to PL1T, but not by priming or triggering, or during the memory phase (PL1) (**Figure 37**). Their further investigation might allow identifying genes with sensitized expression, i.e. genes not responsive to priming but becoming responsive after challenging by the first stimulus, thereby being more strongly regulated only in response to triggering.

To investigate the memory of a 4 h-PL period after longer lag phases of four to ten days, DEGs in response to PL4T, PL5T and PL10T were compared to DEGs in PL1T (**Figure 39**).

(i) The analysis demonstrated that most DEGs (1694 genes) were regulated independently of the duration of the lag phase in response to priming and triggering (**Figure 39**). Genes with a consistent strong regulation unaffected by the lag phase likely represent genes responsive to PL periods rather than markers of memory. Besides, this subset contained genes that were regulated by all primed-and-triggered combinations although not to the same extent, such as *RHL41*/*ZAT12*, *BAP1* and *HSFA2*, which showed an induction after one to four days lag phase (PL1T, PL4T), while being more strongly induced after a ten-day lag phase (PL10T). Conversely, genes like *PIF4* and *IAA29* that were downregulated in PL1T and PL4T, but more strongly downregulated after longer lag phases, were also included in this subset. Thus, also genes that were regulated by all primed-and-triggered combinations might still represent candidates for a further analysis of the memory of photoperiod stress when showing different extents of regulation.

(ii) A second subset contained 48 genes that were significantly regulated in response to PL1T, but not in response to priming-and-triggering when a longer lag phase was included (**Figure 39**). These genes might be informative for short-term memory of photoperiod stress.

4.3 *Trans*-priming and memory by photoperiod stress

In the third part of this thesis, the effects of mild photoperiod stress in Arabidopsis were investigated focusing on plant responses related to pathogen infections. Mild photoperiod stress not only induced SA-dependent plant responses (**Figure 40**, **Figure 41**, **Figure 42**) but also enhanced/primed the resistance of plants against both *Pst* DC3000 and *B. cinerea* infections (**Figure 44**, **Figure 45**, **Figure 51**). The SA signalling protein NPR1 is required for the responses to photoperiod stress.

4.3.1 The effects of mild photoperiod stress on SA-related responses

Genes involved in SA biosynthesis/signalling were induced (**Figure 40**) and SA accumulated (**Figure 42**) at the end of the night following a 4 h-PL in WT plants. Similar observations were made after strong photoperiod stress (Cortleven *et al.*, 2022). SA levels accumulated by mild photoperiod stress were around one-fourth of levels measured in response to strong photoperiod stress (Cortleven *et al.*, 2022) indicating that the duration of the PL period influences the SA accumulation. Nevertheless, the induction of SA-dependent responses characterises both strong and mild photoperiod stress.

The induction of SA-dependent responses by mild photoperiod stress is only transient as expression of most SA biosynthesis/signalling genes and SA levels decreased again under SD conditions (**Figure 41**, **Figure 42**). However, the expression of certain SA-dependent genes (such as *PR1*) and the levels of free SA stayed at higher levels than in non-induced plants (**Figure 41**, **Figure 42**) indicating the preservation of a partly induced state under SDs following mild photoperiod stress treatments. Since SA biosynthesis/signalling is triggered by ROS (Herrera-Vásquez *et al.*, 2015), the induction of SA-dependent responses might result from the oxidative burst-like response observed during photoperiod

stress (Abuelsoud *et al.*, 2020). On the other hand, SA also stimulates ROS production (Herrera-Vásquez *et al.*, 2015).

An increase in SA levels activates many pathways, including the biosynthesis of camalexin representing a phytoalexin that is produced during biotic stresses (Glawischnig, 2007). Cortleven *et al.* (2022) observed that camalexin levels increased in response to photoperiod stress. The current study suggests that mild photoperiod stress does not directly affect camalexin levels. Instead, mild photoperiod stress first induces an accumulation of SA at the end of the night following a 4 h-PL, which is then followed by an increase in camalexin levels (**Figure 42**). The order of these events resembles those observed during SAR which can prime plants against pathogen attacks (Bernsdorff *et al.*, 2016).

4.3.2 The effects of mild photoperiod stress on JA-related responses

Mild photoperiod stress caused an only very moderate increase in JA levels (**Figure 42**) in contrast to the strong accumulation of JA in response to strong photoperiod stress (Cortleven *et al.*, 2022). Nevertheless, the expression of genes involved in JA biosynthesis/signalling is still regulated by mild photoperiod stress (**Supplementary Table S12**) indicating that mild photoperiod stress activates at least part of JA-related responses. This agrees with observations made by Cagnola *et al.* (2018) who showed that a shift from SD to LD activates JA-dependent responses in Arabidopsis including an induction of JA signalling genes, while the JA content is not affected by the transfer to a longer photoperiod.

Several studies highlight the antagonistic regulation of SA- and JA-mediated plant responses (Koornneef *et al.*, 2008; Thaler *et al.*, 2012), while neutral or synergistic regulations of SA- and JA-mediated plant responses occur less often (Rostás *et al.*, 2013; Lortzing *et al.*, 2019). Synergistic effects on the expression of genes important for SA- or JA-dependent responses emerge when Arabidopsis plants are treated with both SA and JA transiently and at low concentrations (Mur *et al.*, 2006; Lortzing *et al.*, 2019). SA and JA synergisms depend on NPR1 and COI1 (Mur *et al.*, 2006). Notably, SA and JA have antagonistic effects when Arabidopsis is treated with both phytohormones for a longer time and at high concentrations. This suggests that the outcomes of SA- and JA-mediated plant responses depend on their relative concentrations and the duration of their availability (Mur *et al.*, 2006; Lortzing *et al.*, 2019). SA and JA antagonism occurs downstream of COI1, probably by SA-mediated regulation of JA-dependent transcription factors (Caarls *et al.*, 2015). Mild photoperiod stress particularly influences SA-dependent responses, while strong photoperiod stress additionally impacts JA-dependent pathways. In view of the concentration-dependent regulation of SA- and JA-mediated responses that has been reported by others (Mur *et al.*, 2006; Caarls *et al.*, 2015; Lortzing *et al.*, 2019), the results suggest that mild and strong photoperiod stress activate qualitatively different downstream responses, likely causing different degrees of defence against pathogens. Since the interactions of SA- and JA-dependent signalling are very complex (Yang *et al.*, 2019b), further investigations are necessary to evaluate interactions of respective pathways in response to photoperiod stress.

4.3.3 The effects of mild photoperiod stress on the induction of plant resistance

Mild photoperiod stress transiently induces the expression of defence genes (**Figure 40)** and an accumulation of phytohormones and camalexin important for plant resistance (**Figure 42**). These responses document that mild photoperiod stress activates responses that are also regulated in response to pathogen infection without an actual pathogen attack as noted before for strong photoperiod stress (Cortleven *et al.*, 2022). Having this in mind, it was investigated if mild photoperiod stress improves resistance against *Pst*. The experiments showed that mild photoperiod stress induced by a 4 h- or 8 h-PL period decreased bacterial growth in Arabidopsis (**Figure 44**, **Figure 45**); however, with less prominent effects with by a shorter PL period. Importantly, the response of the defence genes *PR1* and *FMO1* to bacterial infection was much stronger after pre-treatment with mild photoperiod stress indicating priming. However, priming of the expression of these two defence genes was already lost after one subsequent SD, while priming of bacterial defence lasted one day longer.

For their virulence, pathogenic bacteria including *Pst* DC3000 require an aqueous microenvironment in the plant apoplast that is characterised by visible water-soaking lesions (Xin *et al.*, 2016; Lajeunesse *et al.*, 2023). Such an aqueous microenvironment is maintained under conditions with high humidity, which occurs when stomata are closed. To stimulate stomatal closure and induce water-soaking lesions, *Pst* DC3000 secretes effector proteins (like HopM1 and AvrE1) that promote abscisic acid biosynthesis/signalling in infected plant tissue, thereby regulating stomatal aperture and closure (Xin *et al.*, 2016). Notably, treatment with continuous light increases the SA content in Arabidopsis, which prevents stomatal closure (Lajeunesse *et al.*, 2023). Elevated SA levels in response to both mild and strong photoperiod stress might also prevent stomatal closure, thereby hampering the establishment of aqueous microenvironments. However, the concrete effects of mild photoperiod stress on plant stomata width and water-soaking lesion formation remain to be investigated. Water-soaked lesions are part of the stress syndrome in mature Arabidopsis leaves after strong photoperiod stress (Nitschke *et al.*, 2016; Frank *et al.*, 2020), most likely contributing to pathogen infection. To exclude an interference of water-soaking lesions on pathogen infection, the effects of strong photoperiod stress on *Pst* resistance were investigated in not fully developed leaves (Cortleven *et al.*, 2022) evolving less lesions (Frank, 2019). Also, after mild photoperiod stress similar less pronounced lesions are visible, thus most likely avoiding an interference of the lesions on pathogen responses. The similar phenotypic alterations in response to photoperiod stress and pathogen infection support the finding that a PL period induces similar responses as pathogen attack.

Mild photoperiod stress also enhances resistance against the fungus *B. cinerea*, which is reflected by less necrosis development and induced expression of *PR1* in WT plants pre-treated with an 8 h-PL period (PL0T) (**Figure 51**, **Figure 52**). This suggests that mild photoperiod stress transfers Arabidopsis into a general alarm status without an actual pathogen attack instead of activating resistance responses that are specific to pathogens.

4.3.4 Roles of SA- and JA-related signalling and components of SAR in mild photoperiod stress-induced resistance

While the induction of plant resistance against (hemi-) biotrophic pathogens, such as *Pst*, depends on SA-mediated signalling, resistance induction against necrotrophic pathogens, such as *B. cinerea*, requires JA-dependent responses (Van der Does *et al.*, 2013). Having this in mind, the roles of SA- and JA-mediated responses in mild photoperiod stress-induced resistance against *Pst* and *B. cinerea* were explored.

Since SA accumulated in plants treated with mild photoperiod stress (**Figure 42**), its role in mild photoperiod stress-induced resistance against *Pst* infection was investigated. The number of bacteria did not decrease in mutants impaired in SA biosynthesis (*ics1*) or signalling (*pad4*, *npr1*) when a mild photoperiod stress was applied (**Figure 46B**). Similar observations were made for mutants impaired in Pip accumulation (*ald1*) or the conversion of Pip to NHP (*fmo1*) (**Figure 46B**). The results may suggest that components of SA biosynthesis/signalling and SAR play a role in resistance induction against *Pst* by mild photoperiod stress. However, it needs to be considered that priming of defence against *Pst* may not be recognizable as the mutant genes are required for resistance establishment. Pre-treatment with mild photoperiod stress still increased the expression of *PR1* in *ics1*, *pad4*, *ald1* and *fmo1* mutants compared to plants that were directly infected with *Pst* (**Figure 46C**) indicating priming of *PR1* expression. However, the induction of *PR1* expression by the infection with *Pst* was considerably lower than in the WT plants. This suggests that priming is partially independent of components of SA biosynthesis/signalling and SAR. Nevertheless, these are required for full priming of *PR1* expression and for priming of pathogen defence. Furthermore, the analysed mutants except *npr1* responded to PL period pre-treatments by accumulation of peroxides to similar levels as WT plants (**Figure 46A**). It is thus possible that priming occurred in these mutants and resistance was induced, however not realised as increased resistance when infected by *Pst* due to the impaired defence pathway in the mutants. This interpretation also applies for JA-related mutants accumulating peroxides except *jar1* in response to PL periods without showing an improved resistance against *Pst* afterwards (**Figure 50**). Also in this case, it needs to be it needs to be considered that priming of defence against *Pst* may not be recognizable as the mutant genes are required for pathogen defence. However, since resistance against (hemi-) biotrophic pathogens, such as *Pst*, depends especially on SA-mediated signalling (Van der Does *et al.*, 2013), this result suggests that also JA biosynthesis/signalling is important for resistance induction against *Pst* by mild photoperiod stress. The involvement of both SA- and JA-related signalling in priming by mild photoperiod stress against *Pst* infections would indicate that both phytohormones jointly contribute to mediating the response to mild photoperiod stress.

To investigate the roles of SA- and JA-mediated responses in mild photoperiod stress-induced resistance against *B. cinerea* infection, mutants impaired in SA signalling (*npr1*) and JA biosynthesis (*dde2*) were analysed. Previous research showed that *dde2* mutants are highly susceptible to *B. cinerea* (Liu *et al.*, 2017a), while *npr1* mutants are similarly susceptible as WT plants. SA application improves the resistance of plants against *B. cinerea* (Thomma *et al.*, 1999; Ferrari *et al.*, 2003). In contrast to observations made for WT plants, pre-treatment by mild photoperiod stress did only slightly improve the resistance against the fungus in the analysed mutants (**Figure 52**) indicating that SA- and JA-related

responses are required for mild photoperiod stress-induced resistance against *B. cinerea*. Further investigation is necessary to explore the complete mechanisms enabling mild photoperiod stress-induced resistance, which seems to be dependent of SA and JA-related responses.

4.3.5 The role of NPR1 in the response to photoperiod stress

Mild photoperiod stress was associated with an oxidative burst-like response (**Figure 24**) and accumulation of SA (**Figure 42**), thereby resembling responses against infecting pathogens. During plant defence responses, an increase in SA levels stimulates the generation of ROS through repression of antioxidant enzymes, such as catalases and ascorbate peroxidases (Chen *et al.*, 1993). SA can also promote scavenging of ROS for example during ozone stress (Yoshida *et al.*, 2009). *Vice versa*, apoplastic ROS stimulates SA biosynthesis and signalling (Herrera-Vásquez *et al.*, 2015; Cortleven *et al.*, 2022). ROS function as signalling molecules modulating downstream responses, such as SAR (Bolwell *et al.*, 2002). The transcription coregulator NPR1, whose activity is determined by the plant redox state (Mou *et al.*, 2003; Mittler *et al.*, 2022), is essential for the induction of SAR by NHP (Backer *et al.*, 2019; Yildiz *et al.*, 2021). NPR1 directly induces the expression of several *WRKY* TF genes acting as positive or negative regulators of SAR (Wang *et al.*, 2006). Furthermore, NPR1 reinforces the circadian clock by controlling morning- and evening-phased genes (Zhou *et al.*, 2015).

Under non-stressed conditions, NPR1 forms cytoplasmic oligomers through intermolecular cysteine bonds (Mou *et al.*, 2003; Mittler *et al.*, 2022). During pathogen infections, SA accumulates in plants and the plant redox state is affected resulting in reduction of NPR1 by TRXh3 and TRXh5, which leads to the dissociation of oligomeric NPR1 into monomers (Mou *et al.*, 2003; Tada *et al.*, 2008; Backer *et al.*, 2019). Monomeric NPR1 is phosphorylated by SnRK2.8 and translocates into the nucleus (Kinkema *et al.*, 2000; Spoel *et al.*, 2009; Lee *et al.*, 2015; Backer *et al.*, 2019). Nuclear NPR1 coregulates transcription of *PR1* (Kinkema *et al.*, 2000), together with TGA TFs (Kesarwani *et al.*, 2007; Cortleven, 2022).

During plant defence responses, NPR1 regulates the crosstalk between SA- and JA/ET-dependent signalling (Spoel *et al.*, 2003; Cortleven, 2022). In addition, NPR1 mediates crosstalk between SA and auxin (Wang *et al.*, 2007) as well as SA and gibberellin (Yu *et al.*, 2022; Zavaliev & Dong, 2023). Furthermore, SA-dependent defence signalling is enhanced by CK requiring interaction of ARR2, TGA3 and NPR1 (Choi *et al.*, 2010).

The effects of light on plant defence responses often involve NPR1. In tomato, NPR1 is required for resistance induction by night-time red light treatments (Yang *et al.*, 2015). In Arabidopsis, continuous light induces SA biosynthesis by SID2 and signalling by NPR1, thereby increasing SA-dependent responses, which promote stomatal opening, thereby preventing the formation of aqueous microenvironments that are beneficial for *Pst* (Lajeunesse *et al.*, 2023). In response to low R:FR ratios, monomerization of NPR1 increases (independent of SA) without inducing defence genes, while phosphorylation of NPR1 is not enhanced under respective low R:FR ratios (De Wit *et al.*, 2013; Roeber *et al.*, 2021). The decreased phosphorylation of NPR1 has been proposed to result from inhibition of SA-dependent kinases by low R:FR ratios and due to effects of phyB on SA signalling and pathogen

defence (De Wit *et al.*, 2013; Moreno & Ballare, 2014). Excess white light or red light perceived by phyB stimulates accumulation of ROS, which keeps plant stomata open (Devireddy *et al.*, 2020).

Besides the importance of NPR1 during defence responses, NPR1 stimulates plant cell survival under conditions of oxidative damage, UV irradiation, and heat stress (Zavaliev *et al.*, 2020; 2023). In addition, NPR1 mediates cold acclimation by interaction with different TFs, such as HSFA1, thereby inducing the transcription of genes regulating cold acclimation (Olate *et al.*, 2018). During ER stress, NPR1 interacts with the basic leucine zipper (bZIP) domain TFs bZIP28 and bZIP60, in this way repressing the induction of unfolded protein response genes (Lai *et al.*, 2018). In response to salt stress, NPR1 transiently accumulates in the stroma of chloroplasts where NPR1 functions as a chaperone and antioxidant, thereby being involved in protein and redox homeostasis (Seo *et al.*, 2020).

This study demonstrated that NPR1 is essential for the photoperiod stress response of Arabidopsis, since *npr1* mutants did not accumulate peroxides in response to photoperiod stress (**Figure 46**). Additional analysis showed that the nuclear pore complex component MODIFIER OF SNC1,7 (MOS7) mediating nuclear import of NPR1 (Cheng *et al.*, 2009) is required for the oxidative burst-like photoperiod stress response, since *mos7-1* mutants did not accumulate peroxides in response to PL periods (data not shown, personal communication, Marcel Wiermer). However, the oxidative burst-like photoperiod stress response neither depended on SA biosynthesis by *ICS1* or signalling by *PAD4*, nor required it *ALD1* or *FMO1*, as respective mutants accumulated peroxides like WT plants (**Figure 46**).

The *npr1* mutant used in this work contains a single point mutation (caused by EMS mutagenesis) that changes a conserved histidine to a tyrosine in the encoded protein, thereby impacting the third ankyrin-repeat of NPR1, which is likely to destabilize the entire NPR1 protein and ends in very little *PR1* expression (Cao *et al.*, 1997). To further verify the role of NPR1 in the photoperiod stress response, *npr1-2* mutants were analysed carrying a point mutation in the BTB/POZ domain of the NPR1 protein causing a cysteine-to-tyrosine exchange (Kinkema *et al.*, 2000). Since the effects of the mutation in *npr1-2* are in part weaker as for example still residual induction of *PR1* expression takes place (Cao *et al.*, 1997), it was expected that *npr1-2* mutants are still responsive to photoperiod stress, which was confirmed by accumulation of peroxides after a PL period of 8 h (data not shown, personal communication, Anne Cortleven). The different results obtained for *npr1-1* and *npr1-2* mutants point to an involvement of functional NPR1 proteins in the accumulation of peroxides during photoperiod stress.

To gain further insights into the role of NPR1 in response to PL periods, Western blot experiments can be performed to investigate whether total NPR1 protein levels are affected by PL treatments. Another possibility is that the protein state changes in response to PL periods from oligomeric to monomeric NPR1, which occurs during SAR (Mou *et al.*, 2003). In line with this, PL treatments are likely to affect the subcellular localization of NPR1 from cytoplasm to nucleus or chloroplasts, which might be connected to the function of NPR1, as observed in response to salt stress (Seo *et al.*, 2020). To investigate the subcellular localization of NPR1 in response to PL periods, confocal microscopy experiments using *35S:NPR1-GFP* plants in the *npr1-1* mutant background can be performed.

The analysis of transcript abundances performed with *npr1* mutants indicated that NPR1 is required for around one-tenth of the transcriptomic response to photoperiod stress (**Figure 53**, **Figure 54**). Since

NPR1-dependent transcriptional reprogramming and priming of immune responses is induced by NHP (Yildiz *et al.*, 2021), it would be interesting to investigate NHP levels during photoperiod stress.

In Arabidopsis, SA-induced priming of pathogen defence requires NPR1 (Kohler *et al.*, 2002; Backer *et al.*, 2019). The results of the thesis suggest that priming by photoperiod stress also involves NPR1-dependent signalling, thereby inducing resistance against *Pst* (**Figure 46**, **Figure 47**, **Figure 48**, **Figure 49**, **Figure 50**) and *B. cinerea* (**Figure 52**). The importance of NPR1 in photoperiod stress-induced resistance against *Pst* was investigated by analysis of *trxh3*, *trxh5* and *snrk2-8* mutants impaired in monomerization and phosphorylation of NPR1 proteins, respectively (**Figure 49**). In addition, *arr2*, *tga137*, *tga256* were investigated that are mutated in TFs interacting with NPR1 (**Figure 48**). Photoperiod stress did not induce resistance against *Pst* in these mutants, although they were responsive to photoperiod stress. This implies that activation of resistance against *Pst* requires monomerization and phosphorylation of NPR1 as well as transcription factors known to interact with NPR1.

The responses to photoperiod stress and genetic analyses made in this thesis have shown similarities with the known SA signalling pathway involving NPR1 (**Figure 12**). Based on this and previous publications describing photoperiod stress responses, a summary of changes caused by photoperiod stress and priming of pathogen resistance was developed (**Figure 58**). A suddenly occurring PL period induces the accumulation of phytohormones, camalexin and peroxides. The resulting reducing environment leads to the TRXh3/5-dependent dissociation of cytoplasmic NPR1 oligomers into monomers and SnRK2.8-mediated phosphorylation of NPR1. Subsequently nuclear-imported NPR1 coregulates different transcription factors, such as TGA3 and ARR2, thereby regulating the expression of defence response genes, such as *PR1*. Mutation of *NPR1* prevented an accumulation of peroxides and affected the transcript abundance of around one-tenth of the genes regulated in response to an 8 h-PL period in WT (**Figure 53**). NPR1 is thus required for the response to photoperiod stress in addition to its function in pathogen defence. In this way, activation of NPR1-mediated responses by PL periods contribute to defence priming against *Pst* and *B. cinerea*. *PR1* expression is primed by pre-treatment with PL periods and infection with both *Pst* and *B. cinerea* in WT plants. In plants carrying mutations in defence genes (like *ics1*, *pad4*, *ald1*, *fmo1*) pre-treated with PL periods, priming of *PR1* expression after *Pst* infection still occurs, although less strong than in WT plants, suggesting that this expression priming is likely partially independent of components of SA biosynthesis/signalling and SAR. Nevertheless, these are required for resistance establishment after pre-treatment by photoperiod stress, in addition to NPR1. Further investigation is necessary to fully understand the molecular mechanisms involved in photoperiod stress-induced resistance and the priming process (especially in the context of *B. cinerea* infections).

Mild photoperiod stress might be useful to improve plant resistance in controlled light environments (for example in greenhouses) in a sustainable manner and without damaging plants as observed under strong photoperiod stress, which is associated with leaf necrosis. The fact that also other plant species, such as tomato, respond to conditions that induce photoperiod stress and show subsequently increased pathogen resistance (Yang *et al.*, 2015) (personal communication, Anne Cortleven) demonstrates that it might be a promising tool to enhance pathogen resistance in different plant species.

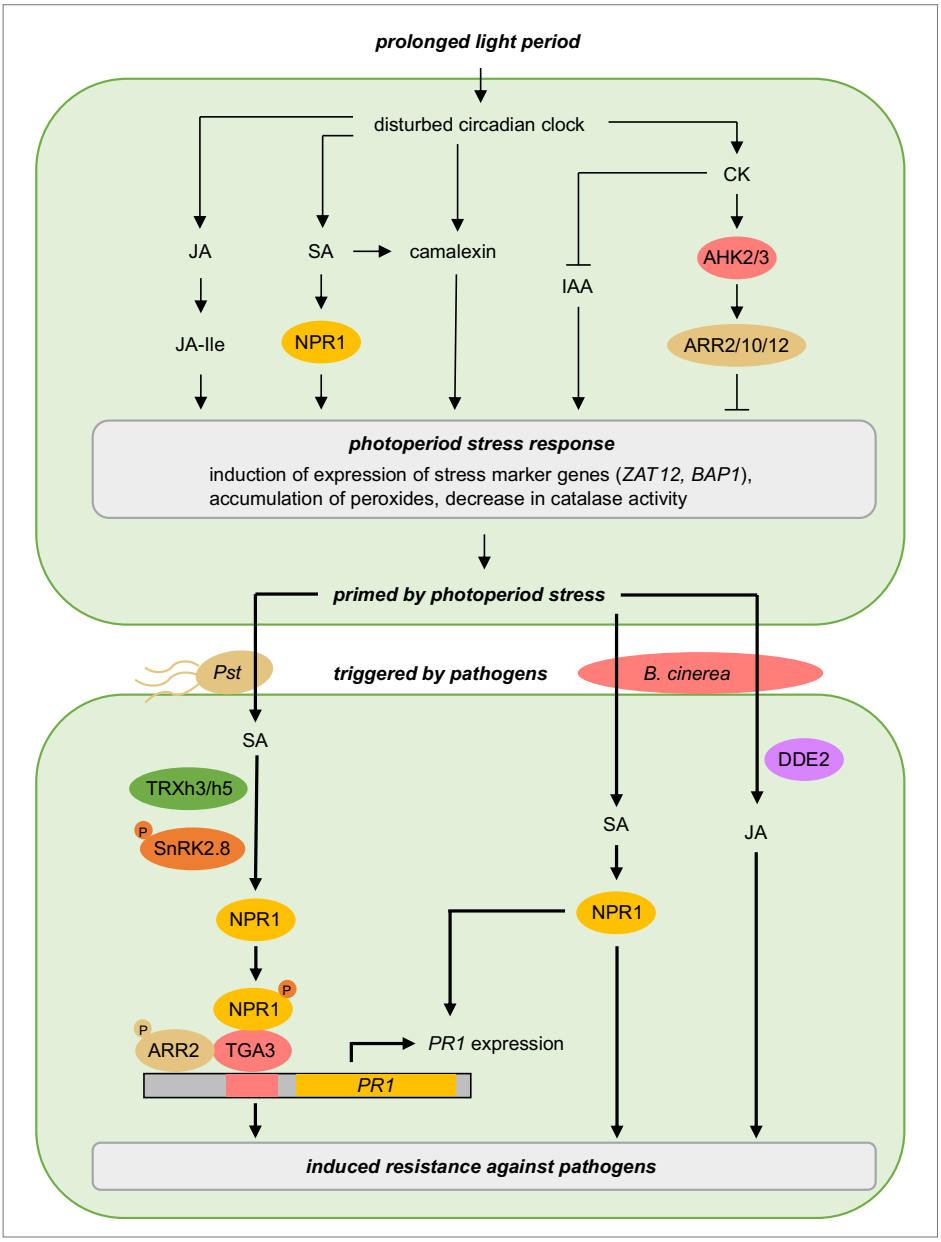

Figure 58. Summary of changes caused by photoperiod stress and priming of pathogen resistance.

A suddenly occurring PL period disturbs the plant circadian clock. This stimulates the accumulation phytohormones and camalexin, which activates downstream signalling pathways and leads to the photoperiod stress response characterised by induction of expression of photoperiod stress marker genes, accumulation of peroxides and a decrease in catalase activity. Activation of CK signalling by PL periods negatively regulates the photoperiod stress response involving AHK2/AHK3 and ARR2/ARR10/ARR12. The protective function of CK is antagonized by IAA. Priming by photoperiod stress enhances the resistance against *Pst* and *B. cinerea*. In case of *Pst* infection, NPR1-mediated SA signalling contributes to pathogen resistance. SA and peroxides promote the TRXh3/5-mediated dissociation of NPR1 oligomers in the cytoplasm, which is followed by SnRK2.8-dependent phosphorylation of NPR1. Monomeric NPR1 is imported into the nucleus, where it interacts with TFs, such as TGA3 and ARR2, in this way coregulating the expression of *PR1*. In case of *B. cinerea*, NPR1-mediated SA signalling and DDE2-mediated JA biosynthesis improve the resistance. JA, jasmonic acid; JA-Ile, jasmonic acid-isoleucine; NPR1, NONEXPRESSOR OF PATHOGENESIS-RELATED GENES1; SA, salicylic acid; TRXh3/h5, THIOREDOXIN H-type 3/H-type 5; SnRK2.8, SNF1-RELATED PROTEIN KINASE 2.8; TGA3, TGACG-binding transcription factor; ARR2, ARABIDOPSIS RESPONSE REGULATOR2; DDE2, DELAYED DEHISCENCE2/ALLENE OXIDE SYNTHASE; CK, cytokinin; AHK2/3, ARABIDOPSIS HISTIDINE KINASE2/3;; ARR2/10/12, ARABIDOPSIS RESPONSE REGULATOR2/10/12; TGA3; IAA, indole-3-acetic acid; *PR1*, *PATHOGENESIS-RELATED GENE1*; P, indicates phosphorylation of proteins. Thick arrows indicate stronger regulation after pre-treatment with a PL period.

5 Conclusions

At this point of research, the data suggests that the transcriptome of Arabidopsis plants is influenced by PL periods in the range of a few hours. An extension of the duration of the PL period increases the number of differentially regulated genes. Three genes, *PIF4*, *COR413IM1* and *BIM1*, are regulated independently of the duration of the PL period, or the time of the day.

Besides affecting the plant transcriptome, a 4 h-PL period is sufficient to induce oxidative stress-like responses connected to photoperiod stress. Mild photoperiod stress transiently transfers Arabidopsis into a kind of alarm state that improves its performance against a similar photoperiod stress and enhances its resistance against pathogens. The memory of photoperiod stress depends on the plants' genotype, its developmental stage, and the initial priming stimulus including its intensity and duration.

In most studies, in which plants were treated with different photoperiods (including this work), light intensity remained unchanged throughout experiments (Shibaeva *et al.*, 2022). This raises the question, if photoperiod stress results from the longer photoperiod itself or from the increased daily light integral. Preliminary experiments revealed that photoperiod stress also occurs under lower light intensities than applied in this study (personal communication, Anne Cortleven, Ishita Bajaj) indicating that photoperiod stress priming and memory results from the longer photoperiod (in combination with a shortened night) and are not induced by an increased light intensity. This is further supported by Shibaeva *et al.* (2022) who showed that in tomato and cucumber plants a higher daily light integral is not causal for stress symptoms induced by continuous light.

From an ecological perspective, it remains open why priming and memory of photoperiod stress occurs in plants. Obviously, plants in nature do not experience PL periods exceeding the normal changes in day length, which are in the range of minutes depending on season and latitude (Jackson, 2009). However, increasing light availability and pollution, particularly in urban environments, make it important to investigate the effects of a sudden prolongation of the light period on plants. Previous publications indicate that not only Arabidopsis but also other plant species, such as tomato, are sensitive to a sudden PL period, thereby affecting the responses of plants to other environmental cues, such as biotic stresses (Yang *et al.*, 2015; Cagnola *et al.*, 2018). The increasing use of greenhouses in agriculture in combination with the observation that photoperiod stress prepares plants for a subsequent biotic stress (Cortleven *et al.*, 2022) offers new possibilities to use photoperiod stress as a sustainable strategy to improve plant performance.

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Annex

Supplementary Table S1. Top 20 upregulated genes in WT plants at the end of a SD in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change; SD, short day.

AGI	End of SD *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT5G23240	12.83514174	1.74E-50	*DNA J PROTEIN C76* (*DJC76*), *DNA J PROTEIN C76* (*DJC76*), DNAJ heat shock N-terminal domain-containing protein.
AT4G33980	12.62892667	3.97E-16	*COLD-REGULATED GENE 28* (*COR28*), *RESPONSE UNKNOWN PROTEIN 41* (*HUP41*), acts with COR27 as a key regulator in the COP1-HY5 regulatory hub by regulating HY5 activity to ensure proper skotomorphogenic growth in the dark and photomorphogenic development in the light.
AT1G76790, *IGMT5*	12.49308238	7.61E-20	*INDOLE GLUCOSINOLATE O-METHYLTRANSFERASE 5* (*IGMT5*), encodes a protein with similarity to N-acetylserotonin O-methyltransferase (ASMT) but it does not have ASMT activity *in vitro*.
AT1G71000	11.45140908	9.01E-15	Chaperone DnaJ-domain superfamily protein.
AT4G30650	10.84850706	2.2E-145	Low temperature and salt responsive protein family.
AT1G07050	10.6961554	1.87E-32	*FITNESS,* encodes a protein with a single CCT domain and belongs to the CCT motif family genes (CMF). FITNESS acts upstream JUB1 thereby controlling H2O2 levels. FITNESS has a role in cellular redox homeostasis controlling H2O2 levels, due to changes in enzymes, metabolites and transcripts related to ROS detoxification
AT5G60100, *PRR3*	10.53375037	7.16E-20	*PSEUDO-RESPONSE REGULATOR 3* (*PRR3*), encodes pseudo-response regulator 3 (APRR3/PRR3). *PRR3* transcript levels vary in a circadian pattern with peak expression at dusk under long and SD conditions. PRR3 affects the period of the circadian clock and seedlings with reduced levels of PRR3 have shorter periods, based on transcriptional assays of clock-regulated genes. *PRR3* is expressed in the vasculature of cotyledons and leaves where it may help stabilize the TOC1 protein by preventing interactions between TOC1 and the F-box protein ZTL.
AT1G22770, *GI*	10.24413692	4.14E-98	*GIGANTEA* (*GI*), together with CONSTANTS (CO) and FLOWERING LOCUS T (FT), GIGANTEA promotes flowering under long days in a circadian clock-controlled flowering pathway. GI acts earlier than CO and FT in the pathway by increasing CO and FT mRNA abundance. Located in the nucleus. Regulates several developmental processes, including photoperiod-mediated flowering, phytochrome B signaling, circadian clock, carbohydrate metabolism, and cold stress response. The gene's transcription is controlled by the circadian clock, and it is post-transcriptionally regulated by light and dark. Forms a complex with FKF1 on the CO promoter to regulate *CO* expression. The mRNA is cell-to-cell mobile.
AT5G24470, *PRR5*	9.229962221	3.37E-30	*PSEUDO-RESPONSE REGULATOR 5* (*PRR5*), encodes a pseudo-response regulator whose mutation affects various circadian-associated biological events such as flowering time in the long-day photoperiod conditions, red light sensitivity of seedlings during early photomorphogenesis, and the period of free-running rhythms of certain clock-controlled genes including CCA1 and APRR1/TOC1 in constant white light. Acts as transcriptional repressor of CCA1 and LHY. Acts additively with EC, PRR7 and PRR9 to regulate hypocotyl growth under photoperiodic conditions.
AT4G16740, *TPS03*	9.136456831	1.08E-09	*TERPENE SYNTHASE 03* (*TPS03*), encodes an (E,E)-alpha-farnesene synthase in the Col ecotype of Arabidopsis. This enzyme can also catalyze the formation of (E)-beta-ocimene as well as trace amounts of myrcene and other related compounds in vitro. The cytosolic localization of the protein may make it favor (E,E)-alpha-farnesene biosynthesis because the precursor of this product, FPP, is primarily cytosolic. Transcript levels for this gene increase in response to treatment with the jasmonic acid mimic coronalon or in response to the insect *Plutella xylostella*.
AT2G40080, *ELF4*	9.118939484	8.1E-66	*EARLY FLOWERING 4* (*ELF4*), encodes a novel nuclear 111 amino-acid phytochrome-regulated component of a negative feedback loop involving the circadian clock central oscillator components CCA1 and LHY. ELF4 is necessary for light-induced expression of both *CCA1* and *LHY*, and conversely, CCA1 and LHY act negatively on light-induced *ELF4* expression. ELF4 promotes clock accuracy and is required for sustained rhythms in the absence of daily light/dark cycles. It is involved in the phyB-mediated constant red light induced seedling de-etiolation process and may function to coregulate the expression of a subset of phyB-regulated genes.
AT1G17665	9.11812244	3.43E-13	CA-responsive protein.
AT5G20630, *GER3*	9.097763603	7.03E-13	*GERMIN-LIKE PROTEIN 3* (*GLP3*), encodes a germin-like protein. Its transcripts are more abundant in RNA from leaves collected in the evening, suggesting some kind of circadian regulation.
AT5G47240	8.708536907	2.65E-65	*NUDIX HYDROLASE HOMOLOG 8* (*NUDT8*)
AT2G39920	8.698853335	3.54E-20	HAD superfamily, subfamily IIIB acid phosphatase
AT3G61920	8.666668379	5.85E-07	PADRE protein
AT3G10185	8.656106464	6.34E-07	Encodes a Gibberellin-regulated GASA/GAST/Snakin family protein (GASA13)
AT2G21660, *GRP7*	8.46564889	7.1E-264	Encodes a small glycine-rich RNA binding protein that is part of a negative-feedback loop through which AtGRP7 regulates the circadian oscillations of its own transcript. Gene expression is induced by cold. GRP7 appears to promote stomatal opening and reduce tolerance under salt and dehydration stress conditions but promotes stomatal closing and thereby increases stress tolerance under conditions of cold tolerance. Loss of function mutations have increased susceptibility to pathogens suggesting a role in mediating innate immune response.
AT2G42530, *COR15B*	8.39352711	1.22E-88	*COLD REGULATED 15B* (*COR15B*), encodes COR15B, a protein that protects chloroplast membranes during freezing.
AT5G52310, *LTI78*	8.355958394	7.55E-54	*LOW-TEMPERATURE-INDUCED 78* (*LTI78*), *RESPONSIVE TO DESICCATION 29A* (*RD29A*), cold regulated gene, the 5' region of cor78 has cis-acting regulatory elements that can impart cold-regulated gene expression The mRNA is cell-to-cell mobile.

Supplementary Table S2. Top 20 downregulated genes in WT plants at the end of a SD in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change; SD, short day.

AGI	End of SD *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT1G01060, *LHY*	-10.38934609	4.4071E-80	*LATE ELONGATED HYPOCOTYL* (*LHY*), LHY encodes a myb-related putative transcription factor involved in circadian rhythm along with another myb transcription factor CCA1.
AT2G46830, *CCA1*	-9.519099844	2.13E-215	CIRCADIAN CLOCK ASSOCIATED 1 (CCA1), encodes a transcriptional repressor that performs overlapping functions with LHY in a regulatory feedback loop that is closely associated with the circadian oscillator of Arabidopsis. Binds to the evening element in the promoter of TOC1 and represses TOC1 transcription. CCA1 and LHY colocalize in the nucleus and form heterodimers in vivo. CCA1 and LHY function synergistically in regulating circadian rhythms of Arabidopsis. CCA1 binds the GI promoter.
AT1G35140, *PHI-1*	-9.35815323	6.0589E-65	*PHOSPHATE-INDUCED 1 (PHI-1), HYPOXIA RESPONSE UNKNOWN PROTEIN 46 (HUP46), EXORDIUM LIKE 7 (EXL7), PHOSPHATE-INDUCED 1 (PHI-1*), EXL1 is involved in the C-starvation response. Phenotypic changes of an exl1 loss of function mutant became evident only under corresponding experimental conditions. For example, the mutant showed diminished biomass production in a short-day/low light growth regime, impaired survival during extended night, and impaired survival of anoxia stress.
AT1G26790	-9.273684638	1.3623E-13	*CYCLING DOF FACTOR 6* (*CDF6*), Dof-type zinc finger DNA-binding family protein
AT3G09450	-9.233025128	5.6207E-09	Fusaric acid resistance family protein.
AT5G16023, *RTFL18*	-9.205355581	1.3274E-09	ROTUNDIFOLIA LIKE 18 (RTFL18), encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.
AT3G09600, *RVE8*	-9.154930985	3.772E-142	*REVEILLE 8 (RVE8), LHY-CCA1-LIKE5 (LCL5*), encodes a MYB-like transcription factor similar to CIRCADIAN CLOCK-ASSOCIATED1 (CCA1) and ELONGATED HYPOCOTYL (LHY). Involved in the regulation of circadian clock by modulating the pattern of histone 3 (H3) acetylation. Functions as a transcriptional activator of evening element containing clock genes. Involved in heat shock response.
AT3G02380, *COL2*	-9.029443281	3.0206E-66	*CONSTANS-LIKE 2 (COL2), CONSTANS-LIKE 2 (ATCOL2), B-BOX DOMAIN PROTEIN 3 (BBX3*), homologous to the flowering-time gene CONSTANS (CO) encoding zinc-finger proteins.
AT1G10550, *XTH33*	-8.832796937	9.8779E-28	*XYLOGLUCAN:XYLOGLUCOSYL TRANSFERASE 33* (*XTH33*), encodes a membrane-localized protein that is predicted to function during cell wall modification. Overexpression of XTH33 results in abnormal cell morphology. Its expression is under epigenetic control by ATX1.
AT3G15310	-8.792378714	2.8033E-16	Transposable element gene.
AT4G15430	-8.711123769	5.4559E-81	*REDUCED HYPEROSMOLALITY-INDUCED CA2+ INCREASE 1.6 (OSCA1.6*), ERD (early-responsive to dehydration stress) family protein.
AT5G17300, *RVE1*	-8.696419941	4.042E-108	*REVEILLE1* (*RVE1*), Myb-like transcription factor that regulates hypocotyl growth by regulating free auxin levels in a time-of-day specific manner.
AT4G08950, *EXO*	-8.642946314	6.241E-143	*EXORDIUM* (*EXO*), cell wall localized protein of unknown function. Expressed in areas with rapidly dividing cells. Overexpression induces elements of brassinosteroid signaling pathways.
AT3G27170, *CLC-B*	-8.495420608	6.1071E-37	*CHLORIDE CHANNEL B (CLC-B*), member of Anion channel protein family The mRNA is cell-to-cell mobile.
AT5G48490	-8.293571641	2.5999E-17	*DIR1-LIKE (DIR1-LIKE*), encodes a protein with similarity to a lipid transfer protein that may contribute to systemic acquired resistance (SAR).
AT1G32900, *GBSS1*	-7.864352166	2.8661E-63	*GRANULE BOUND STARCH SYNTHASE 1* (*GBSS1*), Glucosyltransferase specifically responsible for elongating amylose polymers.
BETA-OHASE 2	-7.772361577	1.592E-108	*BETA-OHASE 2*
AT3G47340, *ASN1*	-7.67422298	3.1854E-13	*GLUTAMINE-DEPENDENT ASPARAGINE SYNTHASE 1 (ASN1), DARK INDUCIBLE 6 (DIN6*), encodes a glutamine-dependent asparagine synthetase, the predicted ASN1 peptide contains a purF-type glutamine-binding domain, and is expressed predominantly in shoot tissues, where light has a negative effect on its mRNA accumulation. Expression is induced within 3 hours of dark treatment, in senescing leaves and treatment with exogenous photosynthesis inhibitor. Induction of gene expression was suppressed in excised leaves supplied with sugar.
AT2G41250	-7.452383386	2.463E-264	Haloacid dehalogenase-like hydrolase (HAD) superfamily protein
AT4G40090	-7.346411501	0.00016929	*ARABINOGALACTAN PROTEIN 3 (AGP3)*

Supplementary Table S3. Top 20 upregulated genes in WT plants in response to an 1 h-PL period in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	1 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT5G56840	3.936412829	0.00507648	Myb-like transcription factor family protein.
AT3G24460	2.883143941	1.7551E-07	Serinc-domain containing serine and sphingolipid biosynthesis protein.
AT1G55740	2.470465029	3.004E-13	*SEED IMBIBITION 1 (SIP1), RAFFINOSE SYNTHASE 1 (RS1*), seed imbibition 1.
AT1G24580	2.010539352	0.00643204	RING/U-box superfamily protein.
AT5G42760	1.698538543	0.02190185	Leucine carboxyl methyltransferase.
AT3G55646	1.670959635	0.01497347	TPRXL
AT5G23660, *SWEET12*	1.645926831	1.2602E-06	*HOMOLOG OF MEDICAGO TRUNCATULA MTN3 (MTN3), (AtSWEET12*), encodes a member of the SWEET sucrose efflux transporter family proteins.

AGI	1 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT4G31870, *GPX7*	1.608377408	0.04636598	*GLUTATHIONE PEROXIDASE 7 (GPX7*), encodes glutathione peroxidase. Role in the degradation of H2O2 to water using glutathione as electron donor.
AT5G37260, *RVE2*	1.576367258	1.1268E-21	*REVEILLE2 (RVE2*), encodes a MYB family transcription factor Circadian 1 (CIR1). Involved in circadian regulation in Arabidopsis.
AT1G57770	1.441149725	0.00014123	Oxidoreductase located in the chloroplast.
AT3G07650, *COL9*	1.425506898	0.00347261	*CONSTANS-LIKE 9 (COL9), B-BOX DOMAIN PROTEIN 7 (BBX7*), this gene belongs to the CO (CONSTANS) gene family. This gene family is divided in three subgroups: groups III, to which COL9 belongs, is characterized by one B-box (supposed to regulate protein-protein interactions) and a second diverged zinc finger. COL9 downregulates expression of CO (CONSTANS) as well as FT and SOC1 which are known regulatory targets of CO. The mRNA is cell-to-cell mobile.
AT1G65486	1.407084284	0.00143759	Secreted peptide which functions in plant growth and pathogen defense. (STMP4)
AT3G21150, *BBX32*	1.371630482	0.00044774	*B-BOX DOMAIN PROTEIN 32 (BBX32), EMF1-INTERACTING PROTEIN 6 (EIP6*), encodes a protein with a B-box domain predicted to act as a transcription factor. Expression of the BBX32 gene is affected by monochromatic red light. Genetic analysis shows BBX32 is under circadian control; it is a morning gene under clock regulation.
AT3G30775, *ERD5*	1.345957549	0.01813755	EARLY RESPONSIVE TO DEHYDRATION 5 (ERD5), encodes a proline oxidase that is predicted to localize to the inner mitochondrial membrane, its mRNA expression induced by high levels of Al and by osmotic stress. The promoter contains an L-proline-inducible element.
AT5G10170, *MIPS3*	1.334919451	3.8709E-05	MYO-INOSITOL-1-PHOSPHATE SYNTHASE 3 (MIPS3), myo-inositol-1-phosphate synthase isoform 3. Expressed in leaf, root and silique. Immunolocalization experiments with an antibody recognizing MIPS1, MIPS2, and MIPS3 showed endosperm localization.
AT3G26590	1.331607713	1.6171E-08	MATE efflux family protein.
AT3G60160	1.32380688	0.00999669	*ATP-BINDING CASSETTE C9 (ABCC9*), member of MRP subfamily.
AT5G02440	1.289680478	0.00195534	60S ribosomal protein L36
AT1G23090, *AST91*	1.143575743	5.1158E-11	*SULFATE TRANSPORTER 91 (AST91), (SULTR3;3*), encodes AST91 mRNA for sulfate transporter.
AT4G37320, *CYP81D5*	1.079505258	0.025781	*CYTOCHROME P450, FAMILY 81, SUBFAMILY D, POLYPEPTIDE 5 (CYP81D5*), member of CYP81D.

Supplementary Table S4. Top 20 downregulated genes in WT plants in response to an 1 h-PL period in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	1 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT5G22500	-4.118540341	4.0556E-06	*FATTY ACID REDUCTASE 1 (FAR1*), encodes a member of the eight-member gene family encoding alcohol-forming fatty acyl-CoA reductases (FARs) identified in Arabidopsis thaliana. Three of the FARs, FAR1 (At5g22500), FAR4 (At3g44540) and FAR5 (At3g44550), are shown to generate the fatty alcohols found in root, seed coat, and wound-induced leaf tissue.
AT1G02205, *CER1*	-3.333431189	0.01384509	*ECERIFERUM 1 (CER1*), expression of the CER1 gene associated with production of stem epicuticular wax and pollen fertility. Biochemical studies showed that cer1 mutants are blocked in the conversion of stem wax C30 aldehydes to C29 alkanes, and they also lack the secondary alcohols and ketones. These suggested the CER1 protein is an aldehyde decarbonylase, but the exact molecular function of this protein remains to be determined.
AT1G03010	-3.272743028	0.00923559	Phototropic-responsive NPH3 family protein.
AT3G21330	-3.181477455	0.0470511	Basic helix-loop-helix (bHLH) DNA-binding superfamily protein.
AT2G14900	-2.549324493	0.00198339	Gibberellin-regulated family protein, (GASA7).
AT5G24580	-2.340964235	0.00055223	Heavy metal transport/detoxification superfamily protein (HIPP09), *HEAVY METAL ASSOCIATED PROTEIN 47 (ATHMP47*).
AT5G22460	-2.270894092	0.00071485	Alpha/beta-Hydrolases superfamily protein.
AT5G02540	-2.109575457	1.7821E-07	NAD(P)-binding Rossmann-fold superfamily protein
AT4G25260	-2.083027963	4.6097E-06	*PECTIN METHYLESTERASE INIHIBITOR 7 (PMEI7*), pectin methylesterase inhibitor. Forms pH dependent complex with PME3.
AT5G18050	-2.075078506	3.7381E-05	*SMALL AUXIN UP RNA 22 (SAUR22*), SAUR-like auxin-responsive protein family.
AT4G16515, *RGF6*	-1.976870621	0.00980944	Encodes a root meristem growth factor (RGF). Belongs to a family of functionally redundant homologous peptides that are secreted, tyrosine-sulfated, and expressed mainly in the stem cell area and the innermost layer of central columella cells. RGFs are required for maintenance of the root stem cell niche and transit amplifying cell proliferation.
AT1G75780, *TUB1*	-1.968910519	0.01303106	*TUBULIN BETA-1 CHAIN (TUB1*), beta tubulin gene downregulated by phytochrome A (phyA)-mediated far-red light high-irradiance and the phytochrome B (phyB)-mediated red light high-irradiance responses.
AT5G16023, *RTFL18*	-1.887053248	0.02381395	*ROTUNDIFOLIA LIKE 18 (RTFL18), DEVIL 1 (DVL1*), encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.
AT4G32280, *IAA29*	-1.789378735	0.00017354	*INDOLE-3-ACETIC ACID INDUCIBLE 29 (IAA29*), indole-3-acetic acid inducible 29 protein involved in IAA signaling. Downstream target of PIF4.
AT5G54510, *DFL1*	-1.780503436	8.3214E-05	*DWARF IN LIGHT 1 (DFL1), GRETCHEN HAGEN3.6 (GH3.6),* encodes an IAA-amido synthase that conjugates Ala, Asp, Phe, and Trp to auxin. Lines overexpressing this gene accumulate IAA-ASP and are hypersensitive to several auxins. Identified as a dominant mutation that displays shorter hypocotyls in light grown plants when compared to wild type siblings. Protein is similar to auxin inducible gene from pea (GH3).
AT5G18010, *SAUR19*	-1.759833097	0.03011447	*SMALL AUXIN UP RNA 19 (SAUR19*), encodes SAUR19 (small auxin up RNA 19). Note that TAIR nomenclature is based on Plant Mol Biol. 2002, 49:373-85 (PMID:12036261).
AT5G66400	-1.759764566	0.00034538	*RESPONSIVE TO ABA 18 (RAB18), ARABIDOPSIS THALIANA DROUGHT-INDUCED 8 (ATDI8*), belongs to the dehydrin protein family, which contains highly conserved stretches

AGI	1 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
			of 7-17 residues that are repetitively scattered in their sequences, the K-, S-, Y- and lysine rich segments. ABA- and drought-induced glycine-rice dehydrin protein. The ABA-induced expression of RAB18 was reduced following ACC application, indicating that ethylene inhibits the ABA signaling pathway.
AT1G29395, *COR413IM1*	-1.703002322	0.00789064	*COLD REGULATED 314 INNER MEMBRANE 1 (COR413IM1*), Integral membrane protein in the inner envelope of chloroplasts. Provide freezing tolerance. Expression is induced by short-term cold-treatment, water deprivation, and abscisic acid treatment. Involved in response to salt tolerance.
AT5G18060	-1.698092579	0.00388429	*SMALL AUXIN UP RNA 23 (SAUR23*), SAUR-like auxin-responsive protein family.
AT1G69830, *AMY3*	-1.638001665	8.3572E-30	*ALPHA-AMYLASE-LIKE 3 (AMY3*), encodes a plastid-localized & -amylase. Expression is reduced in the SEX4 mutant. Loss of function mutations show normal diurnal pattern of starch accumulation/degradation. Expression follows circadian rhythms.

Supplementary Table S5. Top 20 upregulated genes in WT plants in response to a 2.5 h-PL period in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	2.5 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT5G23240	5.341731863	2.2612E-06	*DNA J PROTEIN C76 (DJC76*), DNAJ heat shock N-terminal domain-containing protein.
AT5G56840	4.314986808	0.0002437	Myb-like transcription factor family protein.
AT5G15840, *CO*	4.247869297	5.6709E-07	*CONSTANS (CO), B-BOX DOMAIN PROTEIN 1 (BBX1*), encodes a protein showing similarities to zinc finger transcription factors, involved in regulation of flowering under long days. Acts upstream of FT and SOC1.
AT2G34600, *JAZ7*	3.619968344	1.1111E-06	*JASMONATE-ZIM-DOMAIN PROTEIN 7 (JAZ7*), key regulator in alternative splicing in the jasmonate signaling pathway, alone and in collaboration with other regulators.
AT3G49320	3.600835172	0.00294291	Metal-dependent protein hydrolase.
AT2G27690, *CYP94C1*	3.119170591	0.00109018	*CYTOCHROME P450, FAMILY 94, SUBFAMILY C, POLYPEPTIDE 1 (CYP94C1*), encodes a CYP94C1. Has highest omega-hydroxylase activity with 9,10-epoxystearic acid, while also metabolized lauric acid (C12:0) and C18 unsaturated fatty acids. Gene expression is induced in response to wounding and jasmonic acid treatment.
AT5G24110	3.041362161	0.01144109	*WRKY DNA-BINDING PROTEIN 30 (WRKY30*), member of WRKY TF; Group III.
AT3G24460	3.017437925	1.6215E-08	Serinc-domain containing serine and sphingolipid biosynthesis protein.
AT3G25770, *AOC2*	2.788701947	0.01653156	*ALLENE OXIDE CYCLASE 2 (AOC2*), encodes allene oxide cyclase. One of four genes in Arabidopsis that encode this enzyme, which catalyzes an essential step in jasmonic acid biosynthesis. Gene expression is induced during senescence, a process that involves jasmonic acid signalling pathway.
AT3G16330	2.611457378	0.02565608	Avr9/Cf-9 rapidly elicited protein.
AT5G08640, *FLS1*	2.548076835	0.03089795	*FLAVONOL SYNTHASE 1 (FLS1*), encodes a flavonol synthase that catalyzes formation of flavonols from dihydroflavonols. Co-expressed with CHI and CHS (qRT-PCR).
AT1G21110, *IGMT3*	2.532957841	0.02604753	*INDOLE GLUCOSINOLATE O-METHYLTRANSFERASE 3 (IGMT3*), O-methyltransferase family protein.
AT3G57520, *SIP2*	2.497312162	1.2701E-13	*SEED IMBIBITION 2 (SIP2), RAFFINOSE SYNTHASE 2 (RS2*), encodes a raffinose-specific alpha-galactosidase that catalyzes the breakdown of raffinose into alpha-galatose and sucrose.
AT1G70985	2.437655522	0.0153154	Hydroxyproline-rich glycoprotein family protein.
AT1G06000	2.370157638	3.5577E-05	UGT89C1, encodes a flavonol-7-O-rhamnosyltransferase involved in the formation of rhamnosylated flavonols.
AT5G37550	2.325338307	0.00051506	Hypothetical protein.
AT1G78000, *SULTR1;2*	2.307126564	0.00729298	*SULFATE TRANSPORTER 1;2 (SULTR1;2), SELENATE RESISTANT 1 (SEL1*), encodes a sulfate transporter that can restore sulfate uptake capacity of a yeast mutant lacking sulfate transporter genes.
AT2G40100, *LHCB4.3*	2.206439882	0.00082775	*LIGHT HARVESTING COMPLEX PHOTOSYSTEM II (LHCB4.3*), Lhcb4:3 protein (Lhcb4.3), light harvesting complex of photosystem II The mRNA is cell-to-cell mobile.
AT4G31800, *WRKY18*	2.064221802	0.00217719	*WRKY DNA-BINDING PROTEIN 18 (WRKY18*), pathogen-induced transcription factor. Binds W-box sequences *in vitro*. Forms protein complexes with itself and with WRKY40 and WRKY60. Constitutive expression of WRKY18 enhanced resistance to *P. syringae*, but its coexpression with WRKY40 or WRKY60 made plants more susceptible to both *P. syringae* and *B. cinerea*. WRKY18, WRKY40, and WRKY60 have partially redundant roles in response to the hemibiotrophic bacterial pathogen *P. syringae* and the necrotrophic fungal pathogen *B. cinerea*, with WRKY18 playing a more important role than the other two.
AT5G48470	2.053515296	8.3821E-07	*PEP-RELATED DEVELOPMENT ARRESTED 1 (PRDA1*), hypothetical protein.

Supplementary Table S6. Top 20 downregulated genes in WT plants in response to a 2.5 h-PL period in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	2.5 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT1G06080, *ADS1*	-6.6837918	3.7115E-12	*DELTA 9 DESATURASE 1 (ADS1*), encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.

AGI	2.5 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT3G47340, *ASN1*	-4.6568966	0.00013221	*GLUTAMINE-DEPENDENT ASPARAGINE SYNTHASE 1 (ASN1)*, encodes a glutamine-dependent asparagine synthetase, the predicted ASN1 peptide contains a purF-type glutamine-binding domain, and is expressed predominantly in shoot tissues, where light has a negative effect on its mRNA accumulation.
AT5G16023, *RTFL18*	-3.764207	1.3488E-11	*ROTUNDIFOLIA LIKE 18 (RTFL18), DEVIL 1 (DVL1)*, encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.
AT4G32280, *IAA29*	-3.7590274	9.2963E-26	*INDOLE-3-ACETIC ACID INDUCIBLE 29 (IAA29)*, indole-3-acetic acid inducible 29 protein involved in IAA signaling. Downstream target of PIF4.
AT1G02205, *CER1*	-3.6182446	0.00142717	*ECERIFERUM 1 (CER1)*, expression of the CER1 gene associated with production of stem epicuticular wax and pollen fertility. Biochemical studies showed that cer1 mutants are blocked in the conversion of stem wax C30 aldehydes to C29 alkanes, and they also lack the secondary alcohols and ketones. These suggested the CER1 protein is an aldehyde decarbonylase, but the exact molecular function of this protein remains to be determined.
AT5G22500	-3.5841727	0.00035996	*FATTY ACID REDUCTASE 1 (FAR1)*, encodes a member of the eight-member gene family encoding alcohol-forming fatty acyl-CoA reductases (FARs) identified in Arabidopsis thaliana. Three of the FARs, FAR1 (At5g22500), FAR4 (At3g44540) and FAR5 (At3g44550), are shown to generate the fatty alcohols found in root, seed coat, and wound-induced leaf tissue.
AT5G18050	-3.3752083	2.9364E-15	*SMALL AUXIN UP RNA 22 (SAUR22)*, SAUR-like auxin-responsive protein family.
AT3G21330	-3.155704	0.04702301	Basic helix-loop-helix (bHLH) DNA-binding superfamily protein.
AT5G02540	-3.1440179	4.9115E-19	NAD(P)-binding Rossmann-fold superfamily protein.
AT2G46790, *PRR9*	-3.0624751	6.6054E-05	*PSEUDO-RESPONSE REGULATOR 9 (PRR9), TOC1-LIKE PROTEIN 1 (TL1)*, Pseudo-response regulator PRR9. Involved in clock function. PRR7 and PRR9 are partially redundant essential components of a temperature-sensitive circadian system. CCA1 and LHY had a positive effect on PRR9. Interact with TOC1 in a yeast two-hybrid assay. Acts as transcriptional repressor of CCA1 and LHY. Acts additively with EC, PRR5 and PRR7 to regulate hypocotyl growth under photoperiodic conditions.
AT1G29395, *COR413IM1*	-2.8814886	3.2502E-13	*COLD REGULATED 314 INNER MEMBRANE 1 (COR413IM1)*, Integral membrane protein in the inner envelope of chloroplasts. Provide freezing tolerance. Expression is induced by short-term cold-treatment, water deprivation, and abscisic acid treatment. Involved in response to salt tolerance.
AT5G18060	-2.8212826	1.1708E-12	*SMALL AUXIN UP RNA 23 (SAUR23)*, SAUR-like auxin-responsive protein family.
AT5G62280	-2.815052	4.4427E-05	DUF1442 family protein (DUF1442).
AT5G24580	-2.7466162	3.2611E-06	*HEAVY METAL ASSOCIATED PROTEIN 47 (ATHMP47); (HIPP09)*, Heavy metal transport/detoxification superfamily protein.
AT4G01335	-2.5868022	0.00023597	TATA box-binding protein associated factor RNA polymerase I subunit B-like protein.
AT5G18030	-2.5563428	7.805E-22	*SMALL AUXIN UP RNA 21 (SAUR21)*, SAUR-like auxin-responsive protein family.
AT2G14900	-2.5516208	0.00136914	Gibberellin-regulated family protein, (GASA7)
AT5G66400	-2.4448328	3.0401E-10	*ARABIDOPSIS THALIANA DROUGHT-INDUCED 8 (ATDI8), RESPONSIVE TO ABA 18 (RAB18)*, belongs to the dehydrin protein family, which contains highly conserved stretches of 7-17 residues that are repetitively scattered in their sequences, the K-, S-, Y- and lysine rich segments. ABA- and drought-induced glycine-rice dehydrin protein. The ABA-induced expression of RAB18 was reduced following ACC application, indicating that ethylene inhibits the ABA signaling pathway. RAB18 is also expressed in response to the formation of the phospholipid diacylglycerol pyrophosphate. COR47 and RAB18 double overexpressor plants are cold tolerant. Expressed in guard cells.
AT5G44260	-2.4131152	2.9174E-15	*TANDEM CCCH ZINC FINGER PROTEIN 5 (TZF5); (ATC3H61*), encodes a Tandem CCCH Zinc Finger protein. Interacts and co-localizes with MARD1 and RD21A in processing bodies (PBs) and stress granules (SGs).
AT5G39860	-2.3843337	1.1791E-06	*PACLOBUTRAZOL RESISTANCE1 (PRE1);BASIC HELIX-LOOP-HELIX PROTEIN 136 (BHLH136);BANQUO 1 (BNQ1)*, encodes PRE1 (PACLOBUTRAZOL RESISTANCE1). PRE1 and IBH1 form a pair of antagonistic HLH/bHLH transcription factors that function downstream of BZR1 to mediate brassinosteroid regulation of cell elongation. BNQ1 is directly and negatively regulated by AP3 and PI in petals.Required for appropriate regulation of flowering time.

Supplementary Table S7. Top 20 upregulated genes in WT plants in response to an 4 h-PL period in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	4 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT1G33730, *CYP76C5*	9.258702457	0.00147155	*CYTOCHROME P450, FAMILY 76, SUBFAMILY C, POLYPEPTIDE 5 (CYP76C5)*, cytochrome P450, family 76, subfamily C, polypeptide 5.
AT1G44130	9.058076478	0.00156357	Eukaryotic aspartyl protease family protein.
AT3G46080	8.601579292	0.00120016	C2H2-type zinc finger family protein, (*ZAT8*).
AT4G14630, *GLP9*	8.274448699	0.03586999	*GERMIN-LIKE PROTEIN 9 (GLP9)*, germin-like protein with N-terminal signal sequence that may target it to the vacuole, plasma membrane and/or outside the cell. The mRNA is cell-to-cell mobile.
AT5G45090	8.188970778	0.00704969	*PHLOEM PROTEIN 2-A7 (PP2-A7)*, phloem protein 2-A7.
AT5G59490	7.891434093	0.00090959	Haloacid dehalogenase-like hydrolase (HAD) superfamily protein.
AT2G04515	7.842720661	0.00036207	Transmembrane protein.
AT5G38900	7.712317184	0.03230884	*PROTEIN DISULFIDE ISOMERASE (PDI)*, Thioredoxin superfamily protein.
AT3G13950	7.622910282	0.04333726	Ankyrin.
AT2G35980, *YLS9*	7.57629186	0.00034865	*YELLOW-LEAF-SPECIFIC GENE 9 (YLS9), NDR1/HIN1-LIKE (NHL10); ARABIDOPSIS NDR1/HIN1-LIKE 10 (ATNHL10)*, encodes a protein whose sequence is similar to tobacco hairpin-induced gene (HIN1) and Arabidopsis non-race specific disease resistance gene (NDR1). Expression of this gene is induced by cucumber mosaic virus, spermine and during

AGI	4 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
			senescence. The gene product is localized to the chloroplast. The mRNA is cell-to-cell mobile.
AT3G22600	7.543939078	0.00611864	*GLYCOSYLPHOSPHATIDYLINOSITOL-ANCHORED LIPID PROTEIN TRANSFER 5 (LTPG5*), glycosylphosphatidylinositol (GPI)-anchored LTPg protein, downregulated in syncytia induced by the beet cyst nematode Heterodera schachtii and root knot nematode Meloidogyne incognita. Infection with bacteria (Pseudomonas syringae) and fungi (Botrytis cinerea) leads to the induction of the gene in leaves.
AT2G38340, *DREB19*	7.493462043	0.01799577	*DEHYDRATION RESPONSE ELEMENT-BINDING PROTEIN 19 (DREB19*), encodes a member of the DREB subfamily A-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are eight members in this subfamily including DREB2A AND DREB2B that are involved in response to drought.
AT5G09470, *DIC3*	7.404412348	6.5129E-09	Encodes one of the mitochondrial dicarboxylate carriers (DIC): *DIC1* (AT2G22500), *DIC2* (AT4G24570), *DIC3* (AT5G09470).
AT1G33960, *AIG1*	7.320070658	0.00201285	*IMMUNE ASSOCIATED NUCLEOTIDE BINDING 8 (IAN8);AVRRPT2-INDUCED GENE 1 (AIG1*), identified as a gene that is induced by avirulence gene avrRpt2 and RPS2 after infection with Pseudomonas syringae pv maculicola strain ES4326 carrying avrRpt2.
AT3G26830, *PAD3*	7.266038642	0.00741143	*PHYTOALEXIN DEFICIENT 3 (PAD3), (CYP71B15),* mutations in pad3 are defective in biosynthesis of the indole derived phytoalexin camalexin. Encodes a cytochrome P450 enzyme that catalyzes the conversion of dihydrocamalexic acid to camalexin. The mRNA is cell-to-cell mobile.
AT5G11920, *cwINV6*	7.264314868	8.213E-07	*6-&1-FRUCTAN EXOHYDROLASE (AtcwINV6); 6-&1-FRUCTAN EXOHYDROLASE (cwINV6*), encodes a protein with fructan exohydrolase (FEH) activity acting on both inulin and levan-type fructans (1- and 6-FEH). The enzyme does not have invertase activity.
AT4G21840	7.263455823	7.3068E-06	*METHIONINE SULFOXIDE REDUCTASE B8 (MSRB8*), methionine sulfoxide reductase B8.
AT1G32960, *SBT3.3*	7.216163893	8.8953E-05	Expression induced by pectin-related DAMPs (Damage Associated Molecular Patterns). Together with SBT3.5 contributes to the activation of defence-related PMEs and Botrytis resistance. Influences the expression of specific defense genes and the structure of pectin against Botrytis.
AT4G11170	7.1566336	0.0496088	*RESISTANCE METHYLATED GENE 1 (RMG1*), a NB-LRR disease resistance protein with a Toll/interleukin-1 receptor (TIR) domain at its N terminus. RMG1 is expressed at high levels in response to flg22 and in naive met1/nrpd2 relative to wild-type plants. Expression of this gene is controlled by DNA methylation in its promoter region.
AT1G14880	7.123408208	1.0902E-05	*PLANT CADMIUM RESISTANCE 1 (PCR1*)

Supplementary Table S8.Top 20 downregulated genes in WT plants in response to an 4 h-PL period in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	4 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT1G06080, *ADS1*	-7.7345137	1.6833E-14	*DELTA 9 DESATURASE 1 (ADS1*), encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.
AT4G40090	-7.4334493	0.00010769	*ARABINOGALACTAN PROTEIN 3 (AGP3*), arabinogalactan protein 3.
AT4G32280, *IAA29*	-6.863475	3.7618E-45	*INDOLE-3-ACETIC ACID INDUCIBLE 29 (IAA29*), indole-3-acetic acid inducible 29 protein involved in IAA signaling. Downstream target of PIF4.
AT3G47340, *ASN1*	-6.1240959	2.3831E-09	*GLUTAMINE-DEPENDENT ASPARAGINE SYNTHASE 1 (ASN1);DARK INDUCIBLE 6 (DIN6);ARABIDOPSIS THALIANA GLUTAMINE-DEPENDENT ASPARAGINE SYNTHASE 1 (AT-ASN1*), encodes a glutamine-dependent asparagine synthetase, the predicted ASN1 peptide contains a purF-type glutamine-binding domain, and is expressed predominantly in shoot tissues, where light has a negative effect on its mRNA accumulation. Expression is induced within 3 hours of dark treatment, in senescing leaves and treatment with exogenous photosynthesis inhibitor. Induction of gene expression was suppressed in excised leaves supplied with sugar.
AT5G16023, *RTFL18*	-6.1099289	1.4228E-12	*ROTUNDIFOLIA LIKE 18 (RTFL18); DEVIL 1 (DVL1*), encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.
AT5G50335	-5.9145194	8.6243E-08	Hypothetical protein.
AT2G45610	-5.3122766	0.03759078	Alpha/beta-Hydrolases superfamily protein.
AT3G55240	-5.087083	8.9692E-38	Overexpression leads to PEL (Pseudo-Etiolation in Light) phenotype. (RPGE3)
AT5G18050	-5.047864	5.4785E-25	*SMALL AUXIN UP RNA 22 (SAUR22*), SAUR-like auxin-responsive protein family.
AT2G14900	-4.692513	1.6744E-11	Gibberellin-regulated family protein, (GASA7).
AT4G01460	-4.6161599	1.1657E-18	Basic helix-loop-helix (bHLH) DNA-binding superfamily protein.
AT5G62280	-4.6010648	4.4382E-16	DUF1442 family protein (DUF1442).
AT3G16240, *DELTA-TIP*	-4.5797882	4.6027E-07	*DELTA TONOPLAST INTEGRAL PROTEIN (DELTA-TIP*), Delta tonoplast intrinsic protein, functions as a water channel and ammonium (NH3) transporter. Highly expressed in flower, shoot, and stem. Expression shows diurnal regulation and is induced by ammonium (NH3). Protein localized to vacuolar membrane. The mRNA is cell-to-cell mobile.
AT5G02760	-4.5293098	3.4465E-46	*SENESCENCE-SUPPRESSED 51 PROTEIN PHOSPHATASE (SSPP);ARABIDOPSIS PP2C CLADE D 7 (APD7*), encodes a phosphatase that functions in sustaining proper leaf longevity and preventing early senescence by suppressing or perturbing SARK-mediated senescence signal transduction.
AT1G17700	-4.5114243	0.00017107	*A PRENYLATED RAB ACCEPTOR 1.F1 (PRA1.F1*), prenylated RAB acceptor 1.
AT2G29170	-4.4184652	0.02054088	NAD(P)-binding Rossmann-fold superfamily protein.

AGI	4 h-PL *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT4G33790, *CER4*	-4.3965529	4.8986E-11	*ECERIFERUM 4 (CER4), FATTY ACID REDUCTASE 3 (FAR3*), encodes an alcohol-forming fatty acyl-CoA reductase, involved in cuticular wax biosynthesis. Lines carrying recessive mutations are deficient in primary alcohol and have glossy stem surfaces.
AT4G01335	-4.3613885	2.211E-11	TATA box-binding protein associated factor RNA polymerase I subunit B-like protein.
AT5G18060	-4.3536836	4.757E-28	*SMALL AUXIN UP RNA 23 (SAUR23*), SAUR-like auxin-responsive protein family.
AT4G10150	-4.3510829	1.9332E-07	*ARABIDOPSIS TOXICOS EN LEVADURA 7 (ATL07*), RING/U-box superfamily protein.

Supplementary Table S9. DEGs regulated in response to different PL periods in comparison to WT-control.

Genes in WT plants that were regulated (according to Bonferroni correction, filtered for log_2fold change (FC) ≥ I1I and adjusted *p*-values (padj) ≤ 0.05) in response to a 1 h,- 2.5 h-, 4 h-, 8 h- and 24 h-PL period in comparison to control conditions. Cells remained empty when genes were not detected after the filtering.

gene id	1 h-PL *vs.* control		2.5 h-PL *vs.* control		4 h-PL *vs.* control		8 h-PL *vs.* control		24 h-PL *vs.* control	
	log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
1 h-PL *vs.* control										
AT3G55646	1.6710	0.014973467								
BSK5	-1.1098	8.85011E-05								
AT5G22460	-2.2709	0.000714851								
1 h-PL *vs.* control and 2.5 h-PL *vs.* control										
AT5G56840	3.9364	0.005076483	4.3150	0.0002437						
AT3G24460	2.8831	1.75513E-07	3.0174	1.6215E-08						
GPX7	1.6084	0.046365975	1.7074	0.00966856						
MIPS3	1.3349	3.8709E-05	1.5330	9.106E-08						
AT5G66400	-1.7598	0.000345377	-2.4448	3.0401E-10						
1 h-PL *vs.* control, 2.5 h-PL *vs.* control and 4 h-PL *vs.* control										
AT3G57520, *SIP2*	2.4705	3.00396E-13	2.4973	1.2701E-13	2.2918	4.9363E-11				
AT3G26590	1.3316	1.61711E-08	1.4668	5.0831E-11	1.4689	4.2315E-11				
AT1G23080, *PIN7*	-1.1635	0.002960238	-1.5121	1.7481E-07	-2.8609	3.254E-33				
AT5G04820, *OFP13*	-1.1749	0.020703516	-1.4926	2.1075E-05	-1.4839	1.0385E-05				
AT3G07350	-1.3007	0.04971647	-2.0765	4.1522E-09	-2.4528	1.4838E-13				
AT5G57700	-1.3198	1.56416E-07	-1.2570	7.5752E-07	-1.2400	7.8191E-07				
AT4G16515, *RGF6*	-1.9769	0.009809442	-2.2317	0.00030061	-3.7088	5.2704E-14				
1 h-PL *vs.* control, 2.5 h-PL *vs.* control, 4 h-PL *vs.* control and 8 h-PL *vs.* control										
AT5G23660, *SWEET12*	1.6460	1.26021E-06	1.8907	1.0593E-09	1.5795	6.5609E-06	1.1684	0.0146846		
AT1G64660, *MGL*	-1.0883	0.012432576	-1.2965	6.0247E-05	-1.2723	9.2968E-05	-1.6424	0.0000986		
AT5G12050	-1.1260	5.67093E-08	-1.9446	2.0405E-28	-2.9888	4.5975E-67	-5.3480	8.44E-27		
AT2G20670	-1.3230	0.044013279	-2.0235	1.0116E-08	-2.5942	5.0555E-16	-2.4198	0.0001316		
AT5G18080, *SAUR24*	-1.4142	8.79034E-06	-2.1867	1.8713E-15	-2.4554	6.073E-20	-5.0117	8.71E-21		
AT5G18030	-1.4623	1.8938E-06	-2.5563	7.805E-22	-3.5974	1.1494E-37	-5.8576	7.04E-26		
AT5G02760	-1.4810	0.008429367	-2.1818	2.1864E-09	-4.5293	3.4465E-46	-6.2525	8.83E-27		
AT3G15540, *IAA19*	-1.5541	1.58509E-06	-1.6949	2.5324E-08	-2.1233	2.1973E-14	-1.7824	0.00281508		
AT3G55240	-1.5663	0.0020233	-2.0635	7.7203E-08	-5.0871	8.9692E-38	-11.7755	2.17E-17		
AT5G18060, *SAUR23*	-1.6981	0.003884295	-2.8213	1.1708E-12	-4.3537	4.757E-28	-5.6019	1.22E-13		
AT5G18010, *SAUR19*	-1.7598	0.030114465	-2.1583	0.00017617	-3.3879	1.1017E-10	-7.5776	4.06E-18		
AT4G32280, *IAA29*	-1.7894	0.000173535	-3.7590	9.2963E-26	-6.86347	3.7618E-45	-8.5035	1.15E-15		
AT5G16023, RTFL18	-1.8871	0.023813947	-3.7642	1.3488E-11	-6.1099	1.4228E-12	-7.9285	1.45E-05		
AT5G18050, *SAUR22*	-2.0751	3.73812E-05	-3.3752	2.9364E-15	-5.0479	5.4785E-25	-9.4733	3.89E-10		
AT4G25260	-2.0830	4.60973E-06	-2.0744	4.7244E-06	-3.3642	2.151E-19	-2.3073	8.18E-05		
AT5G02540	-2.1096	1.78212E-07	-3.1440	4.9115E-19	-4.2974	9.4908E-35	-2.4374	1.98E-07		
AT5G24580	-2.3410	0.000552234	-2.7466	3.2611E-06	-3.0842	2.4745E-08	-2.6970	0.01901833		
AT2G14900	-2.5493	0.001983385	-2.5516	0.00136914	-4.6925	1.6744E-11	-3.7099	4.76E-09		
AT1G02205, *CER1*	-3.3334	0.013845094	-3.6182	0.00142717	-4.1807	1.1717E-05	-5.1368	8.28E-35		
AT5G22500	-4.1185	4.05557E-06	-3.5841	0.00035996	-3.8284	3.8008E-05	-4.8781	0.01991909		
all compared meaning 1 h-PL *vs.* control, 2.5 h-PL *vs.* control, 4 h-PL *vs.* control, 8 h-PL *vs.* control and 24 h-PL *vs.* control										
AT2G43010, *PIF4*	-1.5694	2.08429E-07	-2.3820	1.9118E-20	-3.2669	4.3875E-40	-6.9255	1.82E-36	3.2404	1.8E-09
AT1G29395, *COR413IM1*	-1.7030	0.007890639	-2.8814	3.2502E-13	-3.4557	5.2347E-20	-3.5774	1.19E-21	1.6498	0.034064559
AT5G08130, *BIM1*	-1.0676	3.06315E-13	-1.5421	5.8887E-30	-1.8248	8.5026E-43	-1.3010	4.81E-07	1.4159	2.2E-16

Supplementary Table S10. Regulation of the transcript abundance of genes related to the redox system in response to PL periods of 1 h, 2.5 h and 4 h in comparison to WT-control.

Genes in WT plants that were regulated (according to Bonferroni correction) in response to a 1 h-, 2.5 h- and 4 h-PL periods in comparison to control conditions. Cells remained empty when expression levels of respective genes were not detected. Blue or yellow backgrounds of cells indicate downregulation or upregulation, respectively. FC, fold change; padj, adjusted *p*-value according to Bonferroni correction.

AGI	gene id	end of SD *vs.* control		1 h-PL *vs.* control		2.5 h-PL *vs.* control		4 h-PL *vs.* control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT4G25100	*FSD1*	-1.18226529	1	-0.242630687	1	-0.372219692	1	-1.347650164	0.115511957
AT5G51100	*FSD2*	2.530238179	8.23E-28	0.817107431	1	1.73981797	1.95E-11	1.65033967	4.62E-10
AT5G23310	*FSD3*	2.898875258	4.88E-30	0.358779237	1	0.74774216	1	-0.001061489	1
AT1G08830	*CSD1*	-0.045017377	1	-0.404338951	1	1.010868042	1	1.364422987	1
AT2G28190	*CSD2*	-0.333426959	1	-0.324247615	1	0.529799193	1	-0.486329819	1
AT5G18100	*CSD3*	-0.327402713	1	-0.233246372	1	-0.497219357	1	-1.313784864	0.218887014
AT3G10920	*MSD1*	0.880860066	4.90E-21	0.138680376	1	0.298557721	1	0.462688948	0.000960857
AT3G56350	*MnSOD-like*		NA		NA		NA		NA
AT1G07890	*APX1*	1.465437491	0.00024375	-0.136127019	1	0.648422503	1	1.768160104	1.20E-07
AT3G09640	*APX2*		NA		NA		NA		NA
AT4G35000	*APX3*	0.394965107	1	0.044794265	1	-0.056889707	1	-0.404015962	1
AT4G09010	*TL29*	0.330460521	1	0.177501865	1	0.166355587	1	-1.223377874	1
AT4G35970	*APX5*	-0.549892392	1	-0.703827715	1	-0.67499921	1	-0.743391	1
AT4G32320	*APX6*	-0.153162916	1	0.205751859	1	0.090272772	1	-0.888329204	1
AT1G33660	*APX7*								
AT4G08390	*SAPX*	1.272381356	1	0.251568306	1	1.182308539	1	1.62869832	0.014596382
AT1G77490	*TAPX*	-0.376454539	1	0.085780204	1	-0.149075218	1	-1.150159232	0.912653337
AT1G63940	*MDAR6*	0.807997201	0.00502938	-0.069972978	1	0.619026856	1	1.017474592	1.57E-06
AT3G09940	*MDAR3*								
AT3G27820	*MDAR4*	1.12316927	2.49E-11	0.446779087	1	0.496042896	1	0.032236595	1
AT3G52880	*MDAR1*	0.583246898	0.041411787	0.091120737	1	0.416063208	1	0.85898524	5.19E-08
AT5G03630	*ATMDAR2*	2.299610673	0.014030243	0.436075932	1	1.227837317	1	3.43150081	2.58E-09
AT5G16710	*DHAR3*	0.068471645	1	0.230628877	1	0.208400498	1	-0.291100346	1
AT5G36270	*DHAR5*								
AT1G75270	*DHAR2*	1.242499691	1	0.093308498	1	0.408832241	1	2.89477526	3.95E-07
AT1G19550	*DHAR4*	-1.263204345	NA	-0.970328749	NA	0.505131431	NA	-1.350241296	NA
AT1G19570	*DHAR1*	2.885309947	1.21E-18	0.017917718	1	-0.214914888	1	-0.39350108	1
AT3G24170	*GR1*	-2.35914548	3.39E-21	0.172881804	1	0.760388257	1	1.526246792	1.85E-07
AT3G54660	*GR2*								
AT1G20630	*CaT1*	1.411595881	0.017581898	-0.584890117	1	0.196432728	1	0.70681892	1
AT4G35090	*CaT2*	-1.437687546	5.28E-14	0.464634813	1	-0.348088894	1	-1.019776892	1.02E-05
AT1G20620	*CaT3*	2.276637106	4.22E-19	-0.4427921	1	-0.818585544	1	-1.305175103	0.00021098
AT2G25080	*GPX1*	-0.982441689	1	0.021932942	1	-0.410860368	1	-1.909141508	1.15E-08
AT2G31570	*GPX2*	-1.581518215	1	-0.014744564	1	0.326797845	1	2.109047411	0.001948951
AT2G43350	*GPX3*	-0.475293356	1	-0.06877938	1	0.146466165	1	0.31391802	1
AT2G48150	*GPX4*	1.168112549	NA	1.469749363	NA	1.804532287	NA	3.458713396	NA
AT3G63080	*GPX5*	-0.48053004	1	-0.098548997	1	0.161580497	1	1.107727395	0.019264618
AT4G31870	*GPX7*	-1.050119303	1	1.608377408	0.04636598	1.70735297	0.009668564	0.895047371	1
AT1G63460	*GPX8*	0.131766505	1	-0.131965712	1	0.548717412	1	1.404220064	0.037447371
AT4G11600	*GPX6*	3.247765344	4.52E-22	0.13129491	1	0.651663063	1	1.986296472	1.61E-06
AT5G01600	*FER1*	-1.022991289	1	-1.302363339	1	-1.034122884	1	-1.418019707	1
AT3G56090	*FER2*	-0.156990923	1	-0.742348514	1	-0.20716657	NA	0.933800759	NA
AT2G40300	*FER3*	2.84338283	6.59E-13	0.038107178	1	0.750977658	1	-0.250825713	1
AT3G11050	*FER4*	-0.546858558	1	-0.334666438	1	0.058484386	1	-0.764922295	1
AT5G20230									
AT1G72230		-0.702797886	1	-1.585713439	1	-1.67243833	1	-1.582632347	1
AT3G27200		-0.682510486	1	-0.33131426	1	0.091399461	1	-0.342532654	1
AT3G60280									
AT4G12880	*ENODL19*	1.677224022	1	-0.564406235	1	-0.781661782	1	-1.852517122	1
AT5G26330		0.233493629	1	0.494893414	1	0.520060662	1	-0.597781606	1
AT2G33740	*CUTA*	-0.578076863	0.306682644	0.183274374	1	-0.138630854	1	-0.515760048	1
AT4G28365	*ENODL3*	0.660317535	NA	0	NA	0	NA	0	NA
AT2G31050									
AT5G07390	*RbohA*								
AT1G09090	*RBOHB*		NA		NA		NA		NA
AT5G51060	*RbohC*								
AT5G47910	*RBOHD*	0.428513835	1	0.46122861	1	0.474998444	1	1.279579342	8.90E-10
AT1G19230	*RbohE*	-1.763113734	1	0.995338075	1	1.192924005	NA	0.656585988	1
AT1G64060	*RbohF*								
AT4G25090	*RbohG*	0	NA	0	NA	0.797505141	NA	0.573269241	NA
AT5G60010	*RbohH*		NA		NA		NA		NA
AT4G11230	*RbohI*	2.161690257	1	0.996535333	NA	0.845740643	NA	3.401492895	NA
AT3G45810	*RbohJ*								
AT5G23980	*FRO4*	0.927145649	1	-0.312841946	1	-0.625988454	1	-2.113624265	0.015687903
AT1G01590	*FRO1*	0.4866653	1	1.117293607	1	1.456798505	NA	-1.023493035	1
AT1G01580	*FRO2*	-3.007781177	2.33E-06	-0.200761267	1	-0.788266674	1	-2.237692078	0.003271398
AT5G23990	*FRO5*		NA		NA		NA		NA
AT5G49730	*FRO6*	-3.331710842	2.66E-11	0.34373349	1	-0.41990973	1	-2.132717875	0.00573059
AT5G49740	*FRO7*	-2.041538389	1.32E-07	0.537963696	1	-0.162276247	1	-1.824618327	1.57E-05
AT5G50160	*FRO8*	1.189953521	0.00025743	0.04330123	1	-0.158999425	1	-0.397116929	1
AT5G67590									
AT1G23020	*FRO3*	-0.462899024	1	0.567687684	1	0.221424807	1	0.098723518	1

Annex

AGI	gene id	end of SD *vs.* control		1 h-PL *vs.* control		2.5 h-PL *vs.* control		4 h-PL *vs.* control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT1G32350	*AOX1D*	-7.018317612	1	-6.725439983	1	0.558579732	1	7.347773058	1
AT3G22370	*AOX1A*	1.628931263	1	0.023558911	1	1.088347076	1	4.855771679	1.44E-11
AT3G22360	*AOX1B*	0	1	0	NA	0	NA	6.098734296	NA
AT3G27620	*AOX1C*	1.781967282	1	0.308810355	NA	1.210860214	NA	0.776132856	NA
AT5G64210	*AOX2*	-2.145597526	NA	-1.85272193	NA	-1.046613226	NA	-2.232634477	NA
AT4G22260	*IM*	0.047742489	1	0.305469372	1	0.381636472	1	0.93085213	0.000348346
AT1G48130	*1-Cys PrxR*								
AT3G11630	*2-cys PrxR A*	0.315730968	1	0.40253215	1	0.587666374	1	-0.143885928	1
AT5G06290	*2-Cys Prx B*	1.159765454	0.959399908	0.417624941	1	0.501818139	1	-0.346314657	1
AT3G06050	*PRXIIF*	0.437541131	1	0.094320852	1	0.595858756	1	1.398185128	2.57E-12
AT3G26060	*PRXQ*	0.887829607	1	0.499743623	1	0.895693953	1	0.112838364	1
AT1G65990	*Type 2 PrxR A*								
AT1G65980	*Type 2 PrxR B*								
AT1G65970	*Type 2 PrxR C*								
AT1G60740	*Type 2 PrxR D*	-1.263253262	1	-0.970375067	1	1.146151789	NA	5.548321398	NA
AT3G52960	*Type 2 PrxR E*	1.970510926	3.07E-07	0.283677303	1	0.93753869	1	0.244406532	1
AT3G03405									
AT2G04700		0.968082195	0.024835843	0.401833077	1	0.368430792	1	-0.099480351	1
AT1G62180									
AT1G43560	*ty2*	0.875738398	1	0.539292636	1	0.769047348	1	-0.22086495	1
AT1G31020	*TO2*	-0.091429656	1	0.138327357	1	0.199992225	1	0.734412113	0.577070034
AT1G52990	*Thioredoxin family*	0.122030228	1	1.58484809	1	1.493198769	NA	0.172225341	1
AT1G53300	*TTL1*	0.291122442	1	-0.04473421	1	-0.077350871	1	-0.880094999	0.125231327
AT1G76760	*TY1*	-0.925671459	1	0.582151589	1	0.700814442	1	2.290671863	0.053019199
AT2G33270	*ACHT3*	0	NA	0	NA	0	NA	5.18282402	NA
AT2G42580	*TTL3*	-2.748628067	4.92E-41	-0.403554649	1	-0.319516509	1	-0.454639507	1
AT3G06730	*TRX z*	1.960611997	4.77E-15	0.253651808	1	0.672873961	1	0.137180329	1
AT3G08710	*TH9*	0.422212397	1	0.072753374	1	0.290831634	1	1.227386398	2.21E-06
AT3G20560	*PDIL5-3*	0.869715469	1.55E-05	0.282487909	1	0.400971776	1	0.234623767	1
AT3G56420	*Thioredoxin family*		NA		NA		NA		NA
AT4G04950	*GRXS17*	0.282081204	1	-0.165307768	1	-0.035346961	1	0.182505275	1
AT3G56420	*Thioredoxin family*		NA		NA		NA		NA
AT4G29670	*ACHT2*	1.57889864	0.000145475	0.459970367	1	0.663394172	1	1.547845489	0.000270682
AT4G32580	*Thioredoxin family*	0	NA	0	NA	0.797516484	NA	0	NA
AT4G37200	*HCF164*	-0.242595088	1	0.3563693	1	0.395825283	1	-0.429849768	1
AT2G40790	*CXXS2*	0.660319415	NA	2.43783081	NA	2.577451242	NA	2.118567813	NA
AT3G51030	*TRX1*	-0.629973321	1	-0.429928238	1	-0.336246916	1	-0.647890921	1
AT5G39950	*TRX2*	0.536743484	1	-0.025420122	1	0.477493884	1	1.589306137	0.000172469
AT5G42980	*TRX3*	0.046989639	1	0.339711999	1	0.246610237	1	0.188852096	1
AT1G19730	*ATTRX4*	-1.222380291	4.90E-05	-0.06708445	1	-0.022865161	1	-0.042106625	1
AT1G45145	*TRX5*	2.039975791	1	0.526100804	1	2.362640824	1	4.695654746	2.19E-06
AT1G03680	*THM1*	1.054708355	0.577310504	0.405241968	1	0.365507453	1	-0.214728238	1
AT4G03520	*ATHM2*	0.766083852	1	0.244749291	1	0.373632065	1	0.044741402	1
AT2G15570	*ATHM3*	-1.203801356	9.84E-09	-0.050058719	1	-0.128870315	1	0.25141476	1
AT3G15360	*TRX-M4*	0.655130114	1	0.100630582	1	0.045660624	1	-0.31912521	1
AT4G35460	*NTRB*	-0.654824378	0.275733711	-0.197565891	1	-0.170388243	1	-0.171994885	1
AT2G17420	*NTRA*	-0.242480188	1	0.214927977	1	0.399698873	1	1.3487242	0.000215629
AT2G41680	*NTRC*	0.35078552	1	0.295120708	1	0.47826558	1	-0.311281276	1
AT1G50320	*THX*	-0.14235	1	0.311163	1	0.315515	1	-0.03444	1
AT1G03020		-0.648293777	1	-0.513834372	1	-0.601894277	1	-0.524677989	1
AT1G03850	*GRXS13*	0.490436065	NA	-0.380219896	NA	1.583859616	NA	4.399440462	NA
AT1G06830		-0.68810343	1	0.866466706	1	0.78343335	1	-0.545925053	1
AT1G28480	*GRX480*	4.363292594	1	0.876371405	1	4.028298479	1	6.605345585	6.11E-05
AT2G20270		0.811012794	9.99E-06	0.095595167	1	0.473352049	1	0.587067672	0.131714615
AT2G30540		-1.596556761	0.004671406	0.50786765	1	-0.355115186	1	-0.934585851	1
AT2G47870		7.129655864	0.000252079	2.47283739	1	-0.164262125	1	2.501129116	1
AT2G47880		-0.754513068	1	0.497278195	1	-0.38284369	1	-1.013631993	0.067847031
AT3G02000									
AT3G62930		-0.410348958	1	0.011336634	1	-0.017035009	1	-0.280882372	1
AT3G62950		-1.070811453	1	-0.081180559	1	-0.77830988	1	-1.721571483	0.139700791
AT3G62960		-1.304034287	1	-0.155362558	1	0.177559176	NA	0.458793907	NA
AT4G15660		0.654706129	1	0.433955616	1	0.034898116	1	-0.871552473	1
AT4G15660		0.654706129	1	0.433955616	1	0.034898116	1	-0.871552473	1
AT4G15680		1.213261984	1	0.630018339	1	0.223340505	1	-0.722505443	1
AT4G15690		0.760769659	1	-0.053749923	1	-0.72435194	1	-1.904931648	0.088368096
AT4G15700		0.351368628	1	0.239101393	1	0.025585445	1	-0.731445401	1
AT4G28730	*GrxC5*	0.010990202	1	0.05786455	1	-0.099725883	1	-0.419578862	1
AT4G33040		1.347528193	0.148110677	0.787950195	1	1.658997867	0.000553835	1.872148771	4.88E-06
AT5G11930		-0.205636856	1	-0.056155396	1	0.190380196	1	1.374194244	1
AT5G14070	*ROXY2*	-2.38326084	1	0.299607924	1	0.02979052	NA	-0.113403468	1
AT5G18600		-1.235102009	0.734641244	-0.288124813	1	-0.564979001	1	-1.082028856	1
AT1G77370		-0.313808318	1	0.129376029	1	0.210971737	1	1.16079379	0.002646985
AT5G20500		-0.383720753	1	0.034144659	1	0.037181865	1	0.267812376	1
AT5G40370	*GRXC2*	0.929858918	0.000261138	0.19943027	1	0.480895897	1	1.302842237	2.91E-11
AT5G63030	*GRXC1*	-1.358846984	7.89E-10	-0.008361354	1	0.030433319	1	0.602164546	1
AT3G11920		-1.182756499	NA	-0.889879258	NA	-0.192593069	NA	-2.23158469	NA
AT1G05240	*PER1*								

AGI	gene id	end of SD *vs.* control		1 h-PL *vs.* control		2.5 h-PL *vs.* control		4 h-PL *vs.* control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT1G05250	*PER2*	0	NA	0	NA	0	NA	3.735246811	NA
AT1G05260	*RCI3*	-0.338482748	NA	-2.47866343	NA	-2.634340363	NA	-1.111565628	NA
AT1G14540	*PER4*	-3.179933783	1	0.952191263	1	1.933964618	1	2.769482158	1
AT1G14550	*PER5*		NA		NA		NA		NA
AT1G24110	*PER6*		NA		NA		NA		NA
AT1G30870	*PER7*	0	NA	0	NA	0	NA	0.573280584	NA
AT1G34510	*PER8*								
AT1G44970	*PER9*		NA		NA		NA		NA
AT1G49570	*PER10*								
AT1G68850	*PER11*		NA		NA		NA		NA
AT1G71695	*PER12*	1.797427645	8.60E-05	-0.103112001	1	-0.250785393	1	-0.958928662	1
AT1G77100	*PER13*								
AT2G18140	*PER14*								
AT2G18150	*PER15*		NA		NA		NA		NA
AT2G18980	*PER16*	-2.464901833	1	0.139721492	NA	0.558937295	NA	-0.722278255	NA
AT2G22420	*PER17*	-1.512903062	1	-0.08078231	1	-0.173315219	1	-0.802705979	1
AT2G24800	*PER18*								
AT2G34060	*PER19*	-0.707363265	1	0.549904588	1	0.508694154	1	-2.270710957	0.061676238
AT2G35380	*PER20*	0	NA	0.953160607	NA	0.79748396	NA	1.407482289	NA
AT2G37130	*PER21*	-0.017198954	1	1.215170617	1	1.381281941	1	0.882933086	1
AT2G38380	*PER22*		NA		NA		NA		NA
AT2G38390	*PER23*	0	NA	0	NA	0.797516484	NA	0	NA
AT2G39040	*PER24*		NA		NA		NA		NA
AT2G41480	*PER25*	-2.783588391	0.000314877	-0.827195022	1	-0.945615006	1	-1.732443251	1
AT2G43480	*PER26*								
AT3G01190	*PER27*	-0.301434202	NA	-0.008558456	NA	-0.164235183	NA	-1.350257038	NA
AT3G03670	*PER28*	0	NA	0	NA	0	NA	5.080539238	NA
AT3G17070	*PER29*	-0.463915585	1	-0.557927565	1	-0.710382712	1	-0.534066326	1
AT3G21770	*PER30*	-1.711287912	1	0.381742988	1	0.642916658	NA	0.319076773	1
AT3G28200	*PER31*	-2.708379073	1.08E-09	-0.165292415	1	-0.228927606	1	-0.344331256	1
AT3G32980	*PER32*	0.136012542	1	0.402488461	1	-0.540324422	1	-2.82455847	0.749971988
AT3G49110	*PER33*								
AT3G49120	*PER34*								
AT3G49960	*PER35*	-1.055274084	NA	-3.190120579	NA	-3.345798628	NA	-1.707732747	NA
AT3G50990	*PER36*	0	NA	0.953181788	NA	0	NA	0.573269241	NA
AT4G08770	*PER37*								
AT4G08780	*PER38*	0	NA	0	NA	0	NA	0.573280584	NA
AT4G11290	*PER39*	0	NA	0	NA	1.645299282	NA	0	NA
AT4G16270	*PER40*								
AT4G17690	*PER41*		NA		NA		NA		NA
AT4G21960	*PER42*								
AT4G25980	*PER43*	1.4374466	1	-0.004746328	NA	0.045041186	NA	0.612570344	NA
AT4G26010	*PER44*	0	NA	0.953160915	NA	1.64530136	NA	0.573248339	NA
AT4G30170	*PER45*		NA		NA		NA		NA
AT4G31760	*PER46*	0	NA	0.953171362	NA	0	NA	1.457326715	NA
AT4G33420	*PER47*	-6.937818208	2.95E-34	-0.608580392	1	-0.522166174	1	-0.021509459	1
AT4G33870	*PER48*	3.368340728	NA	0	NA	0	NA	0.573260042	NA
AT4G36430	*PER49*	0	1	0.95319537	NA	4.309507013	NA	5.832351335	NA
AT4G37520	*PER50*	-4.437478449	3.81E-24	-0.27082338	1	-0.052803369	1	0.062194333	1
AT4G37530	*PER51*	-3.713933475	0.00059938	-0.074409303	1	1.825031554	1	3.124370033	0.02326576
AT5G05340	*PER52*								
AT5G06720	*PER53*								
AT5G06730	*PER54*	0.660288034	NA	0	NA	0.797487543	NA	4.082199066	NA
AT5G14130	*PER55*	0	NA	0.953181788	NA	0.797505141	NA	0	NA
AT5G15180	*PER56*	-0.246083929	1	-0.914180399	1	-0.86335462	NA	-1.38443806	1
AT5G17820	*PER57*								
AT5G19880	*PER58*	-1.263285434	1	-0.970408219	1	1.823645005	1	7.596552769	1
AT5G19890	*PER59*		NA		NA		NA		NA
AT5G22410	*PER60*								
AT5G24070	*PER61*		NA		NA		NA		NA
AT5G39580	*PER62*	-3.960123637	1	2.174136751	1	2.368815251	1	2.158603787	1
AT5G40150	*PER63*	0.847260245	1	-0.209228994	1	-0.121654464	1	-1.52103341	1
AT5G42180	*PER64*	-0.3014111	NA	-0.970320855	NA	-1.125997502	NA	-1.350233402	NA
AT5G47000	*PER65*	-1.263185108	NA	-0.970309512	NA	-1.125986159	NA	-1.350222059	NA
AT5G51890	*PER66*	-1.384067914	0.055535221	-0.592324215	1	-0.505283776	1	-0.659298105	1
AT5G58390	*PER67*	0.451912	1	-1.322667918	1	-0.822092359	1	-2.835525875	1
AT5G58400	*PER68*								
AT5G64100	*PER69*	-2.742806307	NA	-2.44992814	NA	0.439389242	NA	0.056312492	NA
AT5G64110	*PER70*	-2.742982167	NA	-0.000762842	NA	-0.873507107	NA	0.331413784	NA
AT5G64120	*PER71*	-2.588156038	1	-0.981733459	1	1.75054964	1	2.496134658	1
AT5G66390	*PER72*	-0.756180542	1	-0.879988548	1	-0.219935021	NA	0.699700568	NA
AT5G67400	*PER73*	0	NA	0	NA	0	NA	0.573281	NA

Supplementary Table S11. Regulation of the transcript abundance of SA-related genes in response to PL periods of 1 h, 2.5 h and 4 h in comparison to WT-control.

Genes in WT plants that were regulated (according to Bonferroni correction) in response to a 1 h-, 2.5 h- and 4 h-PL periods in comparison to control conditions. Cells remained empty when expression levels of respective genes

were not detected. Blue or yellow backgrounds of cells indicate downregulation or upregulation, respectively. FC, fold change; padj, adjusted *p*-value according to Bonferroni correction.

AGI	gene id	end of SD *vs.* control		1 h-PL *vs.* control		2.5 h-PL *vs.* control		4 h-PL *vs.* control	
		log$_2$FC	padj	log$_2$FC	padj	log$_2$FC	padj	log$_2$FC	padj
AT1G74710	*EDS16*	-0.31412	1	0.312868	1	1.050912	1	3.817777	1.21E-11
AT1G18870	*ICS2*	2.760975	2.81E-08	-0.98605	1	-0.88328	1	-0.59236	1
AT5G13320	*PBS3*	0.026465	1	0.138812	1	1.604583	1	5.575909	1.63E-11
AT5G67160	*EPS1*	-1.12472	1	0.29495	1	0.933528	1	0.788802	1
AT4G39030	*EDS5*	1.374601	1	0.810494	1	1.958386	1	5.477246	1.36E-17
AT3G29200	*CM1*	0.088082	1	0.391423	1	0.71765	0.000756	-0.26413	1
AT5G10870	*CM2*	0.958786	1	-0.17703	1	0.497287	1	1.517848	0.096422601
AT1G69370	*CM3*	0.707237	1	-0.08231	1	-0.16792	1	-0.71933	1
AT2G37040	*PAL1*	-2.09002	3.28E-25	-0.31901	1	0.109401	1	-0.05811	1
AT3G53260	*PAL2*	-3.99946	3.67E-38	0.278299	1	0.001468	1	-1.41173	0.020076914
AT5G04230	*PAL3*	-0.12929	1	0.47761	1	0.317307	1	-0.66687	1
AT3G10340	*PAL4*	-2.42255	7.53E-20	-1.02524	0.032395	-1.34338	1.08E-05	-0.96593	0.064734887
AT4G29010	*AIM1*	0.913446	0.01261	-0.00737	1	0.304364	1	0.617574	1
AT3G06860	*MFP2*	0.686226	1	-0.09496	1	0.591656	1	1.566516	0.002547308
AT1G73805	*SARD1*	0.340607	1	0.055687	1	1.004496	1	3.387872	4.24E-10
AT5G26920	*CBP60g*	-1.17814	1	0.518827	1	0.334538	1	1.831001	0.108142621
AT1G58100	*TCP8*	-1.22439	2.90E-26	-0.02024	1	0.095439	1	0.447694	0.099520318
AT2G45680	*TCP9*	-1.23642	1.10E-05	0.505884	1	1.21188	2.05E-06	1.880739	1.23E-19
AT4G35580	*NTL9*	0.25647	1	-0.08052	1	0.025746	1	0.426853	1
AT5G08330	*CHE*								
AT4G18170	*WRKY28*	-1.51372	1	0.392758	1	-0.55092	1	0.812813	1
AT2G46400	*WRKY46*	-0.54011	1	0.549926	1	1.295231	1	4.191055	6.23E-06
AT5G46350	*WRKY8*	-1.22074	1	-0.18969	1	0.236923	1	2.435509	0.03094282
AT5G49520	*WRKY48*	-3.85548	2.79E-06	0.379952	1	0.906608	1	2.082008	0.012295246
AT3G20770	*EIN3*	-0.94415	3.04E-06	-0.21594	1	-0.48405	1	-0.32575	1
AT2G27050	*EIL1*	-1.40182	3.32E-10	-0.43793	1	-0.78756	0.283243	-1.78353	3.69E-18
AT1G52890	*NAC019*	2.732563	1	-0.58635	1	1.584822	1	3.353535	1
AT3G15500	*ANAC055*								
AT4G27410	*ANAC072*								
AT5G65210	*TGA1*	-0.63865	1	0.030722	1	0.600564	1	1.203791	0.000826054
AT5G10030	*TGA4*	-0.99752	1.13E-05	0.013516	1	0.016847	1	0.004012	1
AT1G33240	*GTL1*	-2.82028	3.83E-17	0.13656	1	-0.10649	1	-0.88358	1
AT5G09410	*EICBP.B*	-0.66975	1.90E-09	-0.19424	1	-0.30789	1	-0.54228	2.36E-05
AT5G64220	*CAMTA2*	-0.25296	1	-0.10452	1	-0.14208	1	0.102356	1
AT2G22300	*CAMTA3/SR1*	-0.38304	1	0.1458	1	0.06037	1	0.5576	1
AT3G56400	*WRKY70*	1.129754	1	-0.6487	1	-0.04246	1	2.025233	0.008484591
AT3G09830	*PCRK1*	0.674395	1	0.231991	1	0.728534	1	2.083011	4.04E-07
AT5G03320	*PCRK2*	-0.04368	1	-0.05011	1	0.144565	1	1.193631	3.50E-05
AT3G48090	*EDS1*	0.45055	1	-0.01337	1	0.55314	1	2.996634	5.46E-16
AT3G52430	*PAD4*	-0.05361	1	0.486625	1	0.878952	1	3.219285	3.39E-09
AT1G33560	*ADR1*	0.560805	1	0.950213	1	1.01798	1	1.725135	4.92E-05
AT4G33300	*ADR1-L1*	0.117549	1	0.199062	1	0.303259	1	1.519125	2.60E-05
AT4G04720	*ADR1-L2*	0.058189	1	0.398834	1	0.331435	1	1.33687	0.000103294
AT5G40770	*PHB3*	2.671847	1.12E-07	-0.19864	1	0.728997	1	2.449531	5.11E-06
AT1G64280	*NPR1*	0.438184	1	-0.225	1	0.02555	1	1.073952	0.02703603
AT5G06950	*TGA2/AHBP-1B*	-0.12479	1	-0.19866	1	-0.19451	1	-0.11663	1
AT5G06960	*TGA5/OBF5*	-0.51599	1	0.035572	1	0.369071	1	1.373914	0.004013247
AT3G12250	*TGA6*	-0.17805	1	-0.08967	1	-0.23227	1	0.400699	1
AT1G02450	*NIMIN1*	5.961399	0.020493	-0.59261	1	2.884747	1	6.560325	0.001400249
AT3G25882	*NIMIN2*	2.089763	1	-1.92589	1	1.503771	1	5.178812	5.49E-05
AT1G09415	*NIMIN3*	0.245982	1	0.214602	1	0.100934	1	-0.54335	1
AT5G45110	*NPR3*	-0.29861	1	-0.03664	1	0.071879	1	1.602619	2.26E-05
AT4G19660	*NPR4*	1.186261	0.014778	-0.26467	1	-0.07251	1	1.093754	0.095080625
AT2G13810	*ALD1*	-1.42571	1	-3.58311	1	5.402885	1	8.181609	0.184688049
AT5G52810	*SARD4*	1.259694	1	-0.10064	1	2.096422	1	4.323882	5.49E-10
AT1G19250	*FMO1*	-0.56328	1	-2.57841	1	5.753965	1	8.824529	1

Supplementary Table S12. Regulation of the transcript abundance of JA-related genes in response to PL periods of 1 h, 2.5 h and 4 h in comparison to WT-control.

Genes in WT plants that were regulated (according to Bonferroni correction) in response to a 1 h-, 2.5 h- and 4 h-PL periods in comparison to control conditions. Cells remained empty when expression levels of respective genes were not detected. Blue or yellow backgrounds of cells indicate downregulation or upregulation, respectively. FC, fold change; padj, adjusted *p*-value according to Bonferroni correction.

AGI	gene id	end of SD *vs.* control		1 h-PL *vs.* control		2.5 h-PL *vs.* control		4 h-PL *vs.* control	
		log$_2$FC	padj	log$_2$FC	padj	log$_2$FC	padj	log$_2$FC	padj
AT1G05800	*DGL*	0	NA	0	NA	0	NA	0.573281	NA
AT2G44810	*DAD1*		NA		NA		NA		NA
AT2G29980	*FAD3*	-1.37329	1	-0.09275	1	-0.25762	1	-1.51149	0.65231
AT3G11170	*FAD7*	-0.21826	1	0.482945	1	0.168228	1	-0.98469	1
AT5G05580	*FAD8*	2.157542	0.000292	0.746252	1	1.178711	1	0.01161	1
AT3G45140	*LOX2*	3.703768	2.25E-11	0.380407	1	1.288041	1	2.026757	0.217237
AT1G17420	*LOX3*	2.448876	3.33E-21	-0.09869	1	0.101368	1	0.390058	1
AT172520	*LOX4*	0.055534	1	0.433361	1	0.679213	1	2.38521	0.001637

AGI	gene id	end of SD *vs.* control		1 h-PL *vs.* control		2.5 h-PL *vs.* control		4 h-PL *vs.* control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT5G42650	*AOS*	2.409547	3.94E-23	0.184294	1	0.208473	1	-0.18583	1
AT3G25770	*AOC2*	6.76717	1.66E-28	0.747373	1	2.788702	0.016532	3.88093	1.50E-07
AT3G42650	*AOC3*	0.819587	1	-0.28631	1	1.422225	1	4.050506	0.005509
AT2G06050	*OPR3*	3.283197	7.05E-22	0.33637	1	1.165883	1	2.236261	1.15E-08
AT2G46370	*JAR1*	1.338909	6.78E-23	0.038296	1	0.227407	1	0.864156	7.04E-08
AT1G19180	*JAZ1*	2.357668	0.154533	0.745915	1	0.941939	1	2.731279	0.004181
AT1G74950	*TIFY10B*	1.877779	4.06E-28	0.178711	1	0.218035	1	0.688244	0.357278
AT3G17860	*JAZ3*	-0.7152	0.005899	0.210245	1	-0.20491	1	-0.78533	0.000343
AT1G48500	*JAZ4*	-0.02356	1	0.305278	1	-0.75488	1	-1.28565	1
AT1G17380	*JAZ5*	1.765014	1	0.747318	1	1.460683	1	2.885785	0.00291
AT1G72450	*JAZ6*	-0.89694	0.389247	0.294217	1	-0.00863	1	0.114506	1
AT2G34600	*JAZ7*	4.743113	6.69E-14	-0.00941	1	3.619968	1.11E-06	2.996802	0.001437
AT1G30135	*JAZ8*	2.489614	1	-0.90679	NA	2.379459	NA	1.194338	NA
AT1G70700	*TIFY7*	1.898541	1.37E-11	0.741477	1	0.626439	1	-0.30437	1
AT5G13220	*JAZ10*	0.536769	1	-0.07071	1	0.251972	1	0.944522	1
AT3G43440	*JAZ11*	-0.32712	1	-0.10681	1	-0.02399	1	0.390279	1
AT5G20900	*JAZ12*	0.578525	1	-0.04383	1	-0.08025	1	0.323072	1
AT4G28910	*NINJA*	-0.09589	1	-0.17491	1	0.043994	1	0.284271	1
AT2G39940	*COI1*	-0.37775	1	0.042054	1	-0.05479	1	0.053247	1
AT1G06160	*ORA59 (ERF 59)*	3.156472	1	1.279636	1	1.429871	1	0.589586	1
AT3G23240	*ERF1*	-0.05907	1	-2.2335	1	1.027437	NA	2.726655	NA
AT1G32640	*MYC2, JIN1*	3.537548	6.69E-41	0.178866	1	-0.1123	1	0.234528	1
AT5G46760	*MYC3*	0.635797	1	0.187479	1	0.263609	1	0.743258	0.160715
AT4G17880	*MYC4*	-0.99699	4.04E-05	0.512308	1	0.481988	1	-0.56143	1
AT5G44420	*PDF1.2a*	2.712012	1	0.811094	1	3.018458	1	1.867101	1

Supplementary Table S13. Top 20 upregulated genes in WT plants treated with a 4 h-PL period (P) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	P *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT1G76470	9.553283071	0.000000317	NAD(P)-binding Rossmann-fold superfamily protein.
AT1G33730	9.250923117	0.0000325	*CYTOCHROME P450, FAMILY 76, SUBFAMILY C, POLYPEPTIDE 5 (CYP76C5)*, Cytochrome P450, family 76, subfamily C, polypeptide 5.
AT3G44830	9.083074513	0.0000217	Lecithin:cholesterol acyltransferase family protein.
AT1G44130	9.046953276	5.64E-09	Eukaryotic aspartyl protease family protein.
AT5G67310	8.825445012	0.0000507	*CYTOCHROME P450, FAMILY 81, SUBFAMILY G, POLYPEPTIDE 1 (CYP81G1)*, member of CYP81G.
AT1G19250	8.811492279	4.24E-12	*FLAVIN-DEPENDENT MONOOXYGENASE 1 (FMO1)*, FMO1 is required for full expression of TIR-NB-LRR conditioned resistance to avirulent pathogens and for basal resistance to invasive virulent pathogens. Functions in an EDS1-regulated but SA-independent mechanism that promotes resistance and cell death at pathogen infection sites. FMO1 functions as a pipecolate N-hydroxylase and catalyzes the biochemical conversion of pipecolic acid to N-hydroxypipecolic acid (NHP). NHP systemically accumulates in the plant foliage and induces systemic acquired resistance to pathogen infection.
AT4G19970	8.662356502	0.000373535	Nucleotide-diphospho-sugar transferase family protein.
AT2G47520	8.606156916	0.000707281	*ARABIDOPSIS THALIANA ETHYLENE RESPONSE FACTOR 71 (AtERF71)*, encodes a member of the ERF (ethylene response factor) subfamily B-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 5 members in this subfamily including RAP2.2 AND RAP2.12. It plays a role in hypoxia-induced root slanting.
AT3G46080	8.594094365	1.11E-12	*ZAT8*, C2H2-type zinc finger family protein.
AT1G61080	8.42628077	0.0000707	Hydroxyproline-rich glycoprotein family protein.
AT5G17390	8.404195456	0.00000386	Adenine nucleotide alpha hydrolases-like superfamily protein.
AT1G47890	8.397308197	0.00000588	*RECEPTOR LIKE PROTEIN 7 (AtRLP7)*, receptor like protein 7.
AT2G26400	8.387128901	0.005953374	Encodes a protein predicted to belong to the acireductone dioxygenase (ARD).
AT4G14630	8.266784684	0.000658977	*GERMIN-LIKE PROTEIN 9 (GLP9)*, germin-like protein with N-terminal signal sequence that may target it to the vacuole, plasma membrane and/or outside the cell. The mRNA is cell-to-cell mobile.
AT3G28510	8.247323645	0.00012624	P-loop containing nucleoside triphosphate hydrolases superfamily protein.
AT4G15270	8.188896684	0.000148515	Glucosyltransferase-like protein.
AT5G45090	8.181719472	0.0000915	PHLOEM PROTEIN 2-A7 (AtPP2-A7), phloem protein 2-A7.
AT2G13810	8.171169464	4.22E-13	*AGD2-LIKE DEFENSE RESPONSE PROTEIN 1 (ALD1);EDS TWO SUPPRESSOR 5 (EDTS5); (ATALD1)*, ALD1 is a L-lysine alpha-aminotransferase. It is part of the pipecolic acid biosynthetic pathway, where it catalyzes the biochemical conversion of lysine to epsilon-amino-alpha-ketocaproic acid (KAC) which is subject to subsequent transamination, cyclization and isomerization to form 2,3-dehydropipecolic acid.
AT4G37010	8.130535069	0.00000579	*CENTRIN 2 (CEN2);CALMODULIN-LIKE 19 (CML19)*, encodes a member of the Centrin family. Mutants are hypersensitive to UV and prone to UV induced DNA damage. Based on sequence similarity and mutant phenotype CEN2 is thought to be involved in nucelotide excision repair/DNA repair.
AT1G65610	8.091185956	5.58E-10	*ARABIDOPSIS THALIANA GLYCOSYL HYDROLASE 9A2 (ATGH9A2); KORRIGAN 2 (KOR2); (ATKOR2)*, Six-hairpin glycosidases superfamily protein.

Supplementary Table S14. Top 20 downregulated genes in WT plants treated with a 4 h-PL period (P) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	P *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT1G06080	-7.750434718	9.94E-09	*DELTA 9 DESATURASE 1 (ADS1*), encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.
AT4G40090	-7.44098496	0.0000229	*ARABINOGALACTAN PROTEIN 3 (AGP3)*
AT4G32280	-6.884500238	8.35E-17	*INDOLE-3-ACETIC ACID INDUCIBLE 29 (IAA29*), Indole-3-acetic acid inducible 29 protein involved in IAA signaling. Downstream target of PIF4.
AT5G16023	-6.117229992	1.52E-12	*ROTUNDIFOLIA LIKE 18 (RTFL18);DEVIL 1 (DVL1*), encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.
AT5G50335	-5.910515058	0.0000096	Hypothetical protein.
AT2G45610	-5.324009531	0.018495162	α/β-Hydrolases superfamily protein
AT3G55240	-5.061800644	0.0000327	Overexpression leads to PEL (Pseudo-Etiolation in Light) phenotype (*RPGE3*)
AT5G18050	-5.048098305	0.000895879	*SMALL AUXIN UP RNA 22 (SAUR22*), SAUR-like auxin-responsive protein family
AT2G14900	-4.705755506	4.28E-14	Gibberellin-regulated family protein (*GASA7*)
AT5G62280	-4.600557451	0.029720805	*DUF1442 family protein (DUF1442)*
AT4G01460	-4.596527947	0.000635021	basic helix-loop-helix (bHLH) DNA-binding superfamily protein.
AT3G16240	-4.586683864	0.020746599	(*TIP2;1); (DELTA-TIP1); (ATTIP2;1); (AQP1);DELTA TONOPLAST INTEGRAL PROTEIN (DELTA-TIP*), delta tonoplast intrinsic protein, functions as a water channel and ammonium (NH3) transporter. Highly expressed in flower, shoot, and stem. Expression shows diurnal regulation and is induced by ammonium (NH3). Protein localized to vacuolar membrane. The mRNA is cell-to-cell mobile.
AT5G02760	-4.535292123	0.004033404	*ARABIDOPSIS PP2C CLADE D 7 (APD7), SENESCENCE-SUPPRESSED 51 PROTEIN PHOSPHATASE (SSPP);* encodes a phosphatase that functions in sustaining proper leaf longevity and preventing early senescence by suppressing or perturbing SARK-mediated senescence signal transduction.
AT2G29170	-4.423681733	0.036961937	NAD(P)-binding Rossmann-fold superfamily protein.
AT4G01335	-4.367781981	5.84E-11	AT4G01335 AT4G01335.1 TATA box-binding protein associated factor RNA polymerase I subunit B-like protein;(source:Araport11) protein_coding
AT4G10150	-4.358844876	4.89E-08	*ARABIDOPSIS TOXICOS EN LEVADURA 7 (ATL07)*, RING/U-box superfamily protein.
AT5G18060	-4.358277694	0.0000154	*SMALL AUXIN UP RNA 23 (SAUR23),* SAUR-like auxin-responsive protein family.
AT5G66110	-4.314461639	0.001598674	*HEAVY METAL ASSOCIATED ISOPRENYLATED PLANT PROTEIN 27 (HIPP27);HEAVY METAL ASSOCIATED PROTEIN 55 (ATHMP55),* Heavy metal transport/detoxification superfamily protein.
AT5G02540	-4.299582035	4.71E-12	NAD(P)-binding Rossmann-fold superfamily protein.
AT4G08109	-4.1455294	0.0001046	Transposable_element_gene.

Supplementary Table S15. Top 20 upregulated genes in WT plants treated with a 4 h-PL period after one day lag phase (PL1) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	PL1 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT3G50770	6.790556571	4.40844E-06	*CALMODULIN-LIKE 41 (CML41)*
AT2G19800	6.508315564	8.60923E-07	*MYO-INOSITOL OXYGENASE 2 (MIOX2),* encodes a myo-inositol oxygenase family gene.
AT1G62420	4.952025408	0.004446639	*RXR3*, phosphorus (P) stress-inducible DUF506 gene family member; overexpression represses root hair growth; interacts with calmodulins; influences on root hair [Ca2+]cyt oscillation.
AT1G75040	4.839083473	9.59214E-07	*PATHOGENESIS-RELATED GENE 5 (PR5*), thaumatin-like protein involved in response to pathogens. mRNA level of the PR-5 gene (At1g75040)is significantly changed after cutting the inflorescence stem indicating the existence of a network of signal transducing pathways as other stress-regulated genes (At5g01410, At3g17800, At1g29930)do not response to the treatment. The mRNA is cell-to-cell mobile.
AT3G51860	4.616997337	0.002564787	Vacuolar Ca(2+)/H(+) transporter that forms a complex with CAX1 that plays a role in diverse processes including phosphate homeostasis and heavy metal tolerance.
AT1G77380	4.351956826	0.043177042	AMINO ACID PERMEASE 3 (AAP3), amino acid permease which transports basic amino acids.
AT3G57260	4.314582365	0.000369176	PATHOGENESIS-RELATED PROTEIN 2 (PR2) BETA-1,3-GLUCANASE 2 (BGL2), beta 1,3-glucanase.
AT5G10380	4.086927536	1.06743E-08	*RING1*, encodes a RING finger domain protein with E3 ligase activity that is localized to the lipid rafts of the plasma membrane. Expression is increased in response to fungal pathogen. May be involved in regulation of programmed cell death by facilitating degredation of regulation of PDC activators. The mRNA is cell-to-cell mobile.
AT2G43570	4.018160694	0.019845467	CHITINASE, PUTATIVE (CHI), putative basic chitinase.
AT4G00700	3.883315041	0.003535466	*MULTIPLE C2 DOMAIN AND TRANSMEMBRANE REGION PROTEIN 9 (*MCTP9), C2 calcium/lipid-binding plant phosphoribosyltransferase family protein.
AT1G21310	3.790986304	0.03694701	*EXTENSIN 3 (EXT3*), encodes extensin 3.
AT1G72060	3.600428753	0.000289555	Serine-type endopeptidase inhibitor.
AT5G64000	3.562087355	0.008681488	*SAL2*, 3'(2'),5'-bisphosphate nucleotidase.
AT1G36060	3.520568506	0.019258992	*TRANSLUCENT GREEN (TG); (WIND3); (ERF55*), encodes a member of the DREB subfamily A-6 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 8 members in this subfamily including RAP2.4.Overexpression results in increased drought tolerance and vitrified leaves. Binds to DRE/GCC promoter elements and activates

AGI	PL1 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
			expression of aquaporin genes AtTIP1;1, AtTIP2;3, and AtPIP2;2.Involved in light induced suppression of germination via regulation of PIF1 and SOM expression. Its DNA binding activity is regulated by phyA and phyB.
AT5G07200	3.449732402	0.001639946	*ARABIDOPSIS THALIANA GIBBERELLIN 20-OXIDASE 3 (ATGA20OX3); (YAP169*), encodes a gibberellin 20-oxidase.
AT1G34180	3.375169108	0.033777457	*NAC DOMAIN CONTAINING PROTEIN 16 (NAC016*), NAC domain containing protein 16.
AT5G18470	3.273867831	0.002548797	Curculin-like (mannose-binding) lectin family protein.
AT1G12211	3.199768616	0.044722781	Hypothetical protein.
AT2G37130	3.133698013	0.04922	Peroxidase superfamily protein.
AT3G16770	3.102670014	2.81112E-07	*RELATED TO AP2 3 (RAP2.3), ETHYLENE RESPONSE FACTOR 72 (ERF72); ETHYLENE-RESPONSIVE ELEMENT BINDING PROTEIN (EBP*), encodes a member of the ERF (ethylene response factor) subfamily B-2 of the plant specific ERF/AP2 transcription factor family (RAP2.3). The protein contains one AP2 domain. There are 5 members in this subfamily including RAP2.2 AND RAP2.12.It is localized to the nucleus and acts as a transcriptional activator through the GCC-box. It has been identified as a suppressor of Bax-induced cell death by functional screening in yeast and can also suppress Bax-induced cell death in tobacco plants. Overexpression of this gene in tobacco BY-2 cells confers resistance to H2O2 and heat stresses. Overexpression in Arabidopsis causes upregulation of PDF1.2 and GST6. It is part of the ethylene signaling pathway and is predicted to act downstream of EIN2 and CTR1, but not under EIN3. The mRNA is cell-to-cell mobile.

Supplementary Table S16. Top 20 downregulated genes in WT plants treated with a 4 h-PL period after one day lag phase (PL1) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	PL1 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT4G15248	-5.914653845	0.047207402	B-*BOX DOMAIN PROTEIN 30 (BBX30) B-BOX DOMAIN PROTEIN 30 (BBX30);MICROPROTEIN 1B (MIP1B);MICROPROTEIN 1A (MIP1A*), encodes a microprotein that delays floral transition by forming a complex with CONSTANS (CO) and the co-repressor protein TOPLESS.
AT5G24150	-5.221773181	1.4013E-13	(*SQP1);SQUALENE MONOOXYGENASE 5 (SQE5*), squalene monooxygenase gene homolog.
AT5G66740	-4.436433729	0.007129702	*BOUNDARY OF ROP DOMAIN8 (BDR8*), spindle assembly abnormal protein (DUF620)
AT3G22231	-3.920234462	8.83859E-06	*PATHOGEN AND CIRCADIAN CONTROLLED 1 (PCC1*), encodes a member of a novel 6 member Arabidopsis gene family. Expression of PCC1 is regulated by the circadian clock and is upregulated in response to both virulent and avirulent strains of *Pseudomonas syringae* pv. *tomato*.
AT2G15020	-3.633408435	1.51516E-08	Hypothetical protein.
AT5G42760	-3.554580576	0.003241464	Leucine carboxyl methyltransferase.
AT3G44450	-3.49902416	1.49189E-05	*BLUE-LIGHT INHIBITOR OF CRYPTOCHROMES 2 (BIC2*), plant specific protein. BIC1 and BIC2 inhibit cryptochrome function by blocking blue light-dependent cryptochrome dimerization. Light activated transcription of BICs is mediated by cryptochromes.
AT1G24580	-3.136447705	0.006405865	RING/U-box superfamily protein.
AT5G04950	-2.93323753	3.52098E-08	*NICOTIANAMINE SYNTHASE 1 (NAS1*), encodes a nicotianamide synthase.
AT1G73600	-2.846173398	0.000744012	*PHOSPHOETHANOLAMINE METHYLTRANSFERASE3 (ATPMT3); (DEG26*), encodes a S-adenosyl-L-methionine-dependent phosphoethanolamine N-methyltransferase whose expression is responsive to both phosphate (Pi) and phosphite (Phi) in roots. It catalyzes the three sequential P-base methylation of phosphoethanolamine to phosphocholine. Homologous biochemical function to NMT1 (At3g18000). Double mutants of NMT1 and NMT3 are defective in leaf, root, flower, seed, and pollen development.
AT1G26790	-2.722909296	0.039130611	*CYCLING DOF FACTOR 6 (CDF6*), Dof-type zinc finger DNA-binding family protein.
AT1G70830	-2.717868504	0.025055722	*MLP-LIKE PROTEIN 28 (MLP28*), MLP-like protein 28.
AT3G22235	-2.429889417	4.20917E-05	*CYSTEINE-RICH TRANSMEMBRANE MODULE 8 (ATHCYSTM8*), cysteine-rich TM module stress tolerance protein.
AT1G07610	-2.268539612	7.29436E-06	*METALLOTHIONEIN 1C (MT1C*). one of the five metallothioneins (MTs) genes identified in Arabidopsis. MTs are cysteine-rich proteins required for heavy metal tolerance. The mRNA is cell-to-cell mobile.
AT4G15620	-2.239836695	0.001281685	*CASP-LIKE PROTEIN 1E2 (CASPL1E2*), uncharacterized protein family (UPF0497).
AT1G19640	-2.137784999	1.92537E-06	*JASMONIC ACID CARBOXYL METHYLTRANSFERASE (JMT*), encodes a S-adenosyl-L-methionine:jasmonic acid carboxyl methyltransferase that catalyzes the formation of methyljasmonate from jasmonic acid. Its expression is induced in response to wounding or methyljasmonate treatment.
AT1G48260	-2.131298855	0.000317532	*CBL-INTERACTING PROTEIN KINASE 17 (CIPK17);SNF1-RELATED PROTEIN KINASE 3.21 (SnRK3.21*), encodes a member of the SNF1-related kinase (SnRK) gene family (SnRK3.21), which has also been reported as a member of the CBL-interacting protein kinases (CIPK17).
AT4G31870	-2.100857804	0.028342206	*GLUTATHIONE PEROXIDASE 7 (GPX7); (GPXL7*), encodes glutathione peroxidase. Role in the degradation of H2O2 to water using glutathione as electron donor.
AT5G14760	-2.0936539	1.21269E-16	*FLAGELLIN-INSENSITIVE 4 (FIN4);L-ASPARTATE OXIDASE (AO*), At5g14760 encodes for L-aspartate oxidase involved in the early steps of NAD biosynthesis. In contrary to the EC 1.4.3.16 (l-aspartate oxidase - deaminating) the enzyme catalyzes the reaction L-aspartate + O2 = iminoaspartate (alpha-iminosuccinate) + H2O2. Flavoenzyme-encoding gene.
AT4G15920	-2.033509551	0.043617501	*SWEET17*, encodes a vacuolar fructose transporter expressed in parenchyma and xylem that controls leaf fructose content. When its expression is reduced, fructose accumulates in leaves.

Supplementary Table S17. Top 20 upregulated genes in WT plants treated with a 4 h-PL period (T1) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, *Arabidopsis* Genome Initiative locus identifier; FC, fold change.

AGI	T1 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT3G44830	11.6770121	8.75E-11	Lecithin:cholesterol acyltransferase family protein.
AT2G47520	11.6248269	1.81E-09	*ARABIDOPSIS THALIANA ETHYLENE RESPONSE FACTOR 71 (AtERF71*), encodes a member of the ERF (ethylene response factor) subfamily B-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 5 members in this subfamily including RAP2.2 AND RAP2.12. It plays a role in hypoxia-induced root slanting.
AT1G33730	11.6179084	6.95E-10	*CYTOCHROME P450, FAMILY 76, SUBFAMILY C, POLYPEPTIDE 5 (CYP76C5*), cytochrome P450, family 76, subfamily C, polypeptide 5.
AT1G76470	11.3084337	2.87E-11	NAD(P)-binding Rossmann-fold superfamily protein.
AT4G19970	11.2514568	5.26E-09	Nucleotide-diphospho-sugar transferase family protein.
AT3G46080	11.1004754	5.65E-23	C2H2-type zinc finger family protein, (*ZAT8*)
AT5G67310	10.934105	3.03E-09	*CYTOCHROME P450, FAMILY 81, SUBFAMILY G, POLYPEPTIDE 1 (CYP81G1*), member of CYP81G.
AT2G44460	10.9211467	4.11E-10	*BETA GLUCOSIDASE 28 (BGLU28*), beta-glucosidase, major myrosinase which initiates sulfur reallocation by hydrolyzing particular GL species, conferring sulfur deficiency tolerance, especially during early development.
AT5G51060	10.5901377	1.69E-18	*A. THALIANA RESPIRATORY BURST OXIDASE HOMOLOG C (ATRBOHC*), RHD2 (along with RHD3 and RHD4) is required for normal root hair elongation. Has NADPH oxidase activity. Gene is expressed in the elongation and differention zone in trichoblasts and elongating root hairs. RDH2 is localized to the growing tips of root hair cells. It is required for the production of reactive oxygen species in response to extracellular ATP stimulus. The increase in ROS production stimulates Ca2+ influx.
AT1G44130	10.4838896	5.18E-13	Eukaryotic aspartyl protease family protein.
AT1G66570	10.3211197	2.72E-08	*SUCROSE-PROTON SYMPORTER 7 (SUC7*), sucrose-proton symporter 7.
AT5G17390	10.2905791	1.16E-10	Adenine nucleotide alpha hydrolases-like superfamily protein.
AT5G59490	10.2742297	2.38E-10	Haloacid dehalogenase-like hydrolase (HAD) superfamily protein.
AT1G13480	10.2252119	2.28E-11	Hypothetical protein (DUF1262)
AT5G48410	10.2161832	1.85E-08	*GLUTAMATE RECEPTOR 1.3 (GLR1.3)*, member of Putative ligand-gated ion channel subunit family protein.
AT4G22070	10.1811672	3.88E-08	*WRKY DNA-BINDING PROTEIN 31 (WRKY31)*, member of WRKY Transcription Factor; Group II-b.
AT1G15520	10.0954449	1.26E-12	*ATP-BINDING CASSETTE G40 (ABCG40);PLEIOTROPIC DRUG RESISTANCE 12 (ATPDR12*), ABC transporter family involved in ABA transport and resistance to lead. Localizes to plasma membrane. Upregulated by lead. Expressed in leaves, flowers, stomata and roots.
AT2G42480	10.074386	5.32E-09	MATH domain/coiled-coil protein.
AT1G19250	10.0605876	1.32E-16	*FLAVIN-DEPENDENT MONOOXYGENASE 1 (FMO1*), FMO1 is required for full expression of TIR-NB-LRR conditioned resistance to avirulent pathogens and for basal resistance to invasive virulent pathogens. Functions in an EDS1-regulated but SA-independent mechanism that promotes resistance and cell death at pathogen infection sites. FMO1 functions as a pipecolate N-hydroxylase and catalyzes the biochemical conversion of pipecolic acid to N-hydroxypipecolic acid (NHP). NHP systemically accumulates in the plant foliage and induces systemic acquired resistance to pathogen infection.
AT4G15270	10.0114414	2.91E-08	Glucosyltransferase-like protein.

Supplementary Table S18. Top 20 downregulated genes in WT plants treated with a 4 h-PL period (T1) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	T1 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT4G34530	-10.924516	0.012652	*CRYPTOCHROME-INTERACTING BASIC-HELIX-LOOP-HELIX 1 (CIB1*), encodes a transcription factor CIB1 (cryptochrome-interacting basic-helix-loop-helix). CIB1 interacts with CRY2 (cryptochrome 2) in a blue light-specific manner in yeast and Arabidopsis cells, and it acts together with additional CIB1-related proteins to promote CRY2-dependent floral initiation. CIB1 positively regulates FT expression.
AT1G06080	-10.665791	3.96E-08	*DELTA 9 DESATURASE 1 (ADS1)*, encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.
AT3G47340	-10.116271	6.00E-05	*GLUTAMINE-DEPENDENT ASPARAGINE SYNTHASE 1 (ASN1); DARK INDUCIBLE 6 (DIN6);ARABIDOPSIS THALIANA GLUTAMINE-DEPENDENT ASPARAGINE SYNTHASE 1 (AT-ASN1)*, encodes a glutamine-dependent asparagine synthetase, the predicted ASN1 peptide contains a purF-type glutamine-binding domain, and is expressed predominantly in shoot tissues, where light has a negative effect on its mRNA accumulation. Expression is induced within 3 hours of dark treatment, in senescing leaves and treatment with exogenous photosynthesis inhibitor. Induction of gene expression was suppressed in excised leaves supplied with sugar. The authors suggest that the gene's expression pattern is responding to the level of sugar in the cell.
AT4G33790	-9.2318315	9.39E-06	*ECERIFERUM 4 (CER4); (G7); FATTY ACID REDUCTASE 3 (FAR3)*, encodes an alcohol-forming fatty acyl-CoA reductase, involved in cuticular wax biosynthesis. Lines carrying recessive mutations are deficient in primary alcohol and have glossy stem surfaces.
AT5G16023	-9.1955996	1.47E-09	*ROTUNDIFOLIA LIKE 18 (RTFL18); DEVIL 1 (DVL1*), encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.

AGI	T1 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT3G55240	-9.1006396	4.74E-08	*RPGE3,* overexpression leads to PEL (Pseudo-Etiolation in Light) phenotype.
AT4G40090	-8.298577	2.27E-07	*ARABINOGALACTAN PROTEIN 3 (AGP3)*, arabinogalactan protein 3.
AT1G10550	-8.2398389	9.48E-11	*TRANSFERASE 33 (XTH33); XYLOGLUCAN:XYLOGLUCOSYL TRANSFERASE 33 (XET)*, encodes a membrane-localized protein that is predicted to function during cell wall modification. Overexpression of XTH33 results in abnormal cell morphology. It's expression is under epigenetic control by ATX1.
AT2G42870	-8.2260678	4.70E-09	*PHY RAPIDLY REGULATED 1 (PAR1); HELIX-LOOP-HELIX 1 (HLH1)*, encodes PHYTOCHROME RAPIDLY REGULATED1 (PAR1), an atypical basic helix-loop-helix (bHLP) protein. Closely related to PAR2 (At3g58850). Up regulated after simulated shade perception. Acts in the nucleus to control plant development and as a negative regulator of shade avoidance response. Functions as transcriptional repressor of auxin-responsive genes SAUR15 (AT4G38850) and SAUR68 (AT1G29510).
AT4G14130	-7.8426246	0.002961	*XYLOGLUCAN ENDOTRANSGLYCOSYLASE7 (XTR7); XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE15 (XTH15),* xyloglucan endotransglycosylase-related protein.
AT5G65390	-7.7792143	1.34E-13	*ARABINOGALACTAN PROTEIN 7 (AGP7)*, arabinogalactan protein 7.
AT5G50335	-7.7273681	0.000135	Hypothetical protein.
AT2G19660	-7.6542228	0.000407	Cysteine/Histidine-rich C1 domain family protein.
AT3G48260	-7.5768941	0.002325	*WITH NO LYSINE (K) KINASE 3 (WNK3)*, encodes a member of the WNK family (9 members in all) of protein kinases, the structural design of which is clearly distinct from those of other known protein kinases, such as receptor-like kinases and mitogen-activated protein kinases.
AT3G46490	-7.5706835	0.001691	2-oxoglutarate (2OG) and Fe(II)-dependent oxygenase superfamily protein.
AT4G34790	-7.5633119	4.84E-06	*SMALL AUXIN UPREGULATED RNA 3 (SAUR3) SMALL AUXIN UPREGULATED RNA 3 (SAUR3); (DEG4*), putative OXS2-binding DEGs were constitutively activated by OXS2.
AT5G44260	-7.5449325	5.28E-17	*TANDEM CCCH ZINC FINGER PROTEIN 5 (TZF5)*, encodes a Tandem CCCH Zinc Finger protein. Interacts and co-localizes with MARD1 and RD21A in processing bodies (PBs) and stress granules (SGs).
AT4G17460	-7.3248373	9.71E-21	(*HAT1); JAIBA (JAB*), encodes a class II HD-ZIP protein that regulates meristematic activity in different tissues, and that it is necessary for the correct formation of the gynoecium.
AT4G01335	-7.1941569	1.23E-06	TATA box-binding protein associated factor RNA polymerase I subunit B-like protein.
AT5G45820	-7.184961	1.60E-08	*CBL-INTERACTING PROTEIN KINASE 20 (CIPK20);PROTEIN KINASE 18 (PKS18); SNF1-RELATED PROTEIN KINASE 3.6 (SnRK3.6),* encodes a CBL-interacting serine/threonine protein kinase comprised of an N-terminal kinase catalytic domain similar to SNF1/AMPK and a unique C-terminal regulatory domain.

Supplementary Table S19. Top 20 upregulated genes in WT plants treated with a 4 h-PL period as priming and triggering with one day lag phase in-between (PL1T) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	PL1T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT3G44830	10.7413574	1.08E-08	Lecithin:cholesterol acyltransferase family protein.
AT1G33730	10.5195321	1.36E-07	*CYTOCHROME P450, FAMILY 76, SUBFAMILY C, POLYPEPTIDE 5 (CYP76C5)*, cytochrome P450, family 76, subfamily C, polypeptide 5.
AT1G76470	10.4766573	2.85E-09	NAD(P)-binding Rossmann-fold superfamily protein.
AT2G47520	10.3944245	5.33E-07	*ARABIDOPSIS THALIANA ETHYLENE RESPONSE FACTOR 71 (AtERF71)*, encodes a member of the ERF (ethylene response factor) subfamily B-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 5 members in this subfamily including RAP2.2 AND RAP2.12. It plays a role in hypoxia-induced root slanting.
AT5G51060	9.99485917	3.51E-16	*A. THALIANA RESPIRATORY BURST OXIDASE HOMOLOG C (ATRBOHC)*, RHD2 (along with RHD3 and RHD4) is required for normal root hair elongation. Has NADPH oxidase activity. Gene is expressed in the elongation and differention zone in trichoblasts and elongating root hairs. RDH2 is localized to the growing tips of root hair cells. It is required for the production of reactive oxygen species in response to extracellular ATP stimulus. The increase in ROS production stimulates Ca2+ influx.
AT2G44460	9.85617884	1.05E-07	*BETA GLUCOSIDASE 28 (BGLU28)*, Beta-glucosidase, major myrosinase which initiates sulfur reallocation by hydrolyzing particular GL species, conferring sulfur deficiency tolerance, especially during early development.
AT4G14630	9.82688779	9.99E-07	*GERMIN-LIKE PROTEIN 9 (GLP9),* germin-like protein with N-terminal signal sequence that may target it to the vacuole, plasma membrane and/or outside the cell. The mRNA is cell-to-cell mobile.
AT5G67310	9.72328836	0.00000103	*CYTOCHROME P450, FAMILY 81, SUBFAMILY G, POLYPEPTIDE 1 (CYP81G1),* member of CYP81G.
AT5G17220	9.69508767	0.0000124	*GLUTATHIONE S-TRANSFERASE PHI 12 (GSTF12);TRANSPARENT TESTA 19 (TT19);GLUTATHIONE S-TRANSFERASE 26 (GST26)*, encodes glutathione transferase belonging to the phi class of GSTs. Naming convention according to Wagner et al. (2002). Mutants display no pigments on leaves and stems. Likely to function as a carrier to transport anthocyanin from the cytosol to tonoplasts.
AT3G46080	9.64137783	1.14E-16	C2H2-type zinc finger family protein, *ZAT8.*
AT2G39030	9.40342779	4.86E-08	*N-ACETYLTRANSFERASE ACTIVITY 1 (NATA1)*, encodes a protein that acts as an ornithine N-delta-acetyltransferase, leading to the formation of N-delta-actetylornithine. This compound is likely used in plant defense and levels of it are increased in Arabidopsis plants in response to MeJA and ABA. The mRNA is cell-to-cell mobile.
AT5G17390	9.3709687	2.38E-08	Adenine nucleotide alpha hydrolases-like superfamily protein.
AT1G66570	9.32447793	0.00000305	*SUCROSE-PROTON SYMPORTER 7 (SUC7)*, sucrose-proton symporter 7.
AT4G08770	9.2927677	0.00000896	*PEROXIDASE 37 (Prx37)*, encodes a putative apoplastic peroxidase Prx37. Primarily expressed in the vascular bundles. Overexpression renders a dwarf phenotype with smaller

AGI	PL1T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
			plants and delayed development. Plants overexpressing Prx37 also shows an increase in the amount of esterified phenolic material associated with their walls.
AT3G19615	9.23829885	0.00000326	Beta-1,4-xylosidase.
AT1G44130	9.19127341	2.35E-09	Eukaryotic aspartyl protease family protein.
AT5G48410	9.14344549	0.00000333	*GLUTAMATE RECEPTOR 1.3 (GLR1.3)*, member of Putative ligand-gated ion channel subunit family.
AT4G19970	9.00286074	0.00010057	Nucleotide-diphospho-sugar transferase family protein.
AT1G69930	8.98879386	0.0000237	*GLUTATHIONE S-TRANSFERASE TAU 11 (ATGSTU11),* encodes glutathione transferase belonging to the tau class of GSTs. Naming convention according to Wagner et al. (2002).
AT4G23700	8.96519928	0.00000487	*CATION/H+ EXCHANGER 17 (CHX17*), member of Putative Na+/H+ antiporter family.

Supplementary Table S20. Top 20 downregulated genes in WT plants treated with a 4 h-PL period as priming and triggering with one day lag phase in-between (PL1T) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	PL1T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT1G06080	-10.71067	3.21E-08	*DELTA 9 DESATURASE 1 (ADS1)*, encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.
AT2G42870	-9.2762813	7.46E-08	*PHY RAPIDLY REGULATED 1 (PAR1);HELIX-LOOP-HELIX 1 (HLH1)*, encodes PHYTOCHROME RAPIDLY REGULATED1 (PAR1), an atypical basic helix-loop-helix (bHLP) protein. Closely related to PAR2 (At3g58850). Up regulated after simulated shade perception. Acts in the nucleus to control plant development and as a negative regulator of shade avoidance response. Functions as transcriptional repressor of auxin-responsive genes SAUR15 (AT4G38850) and SAUR68 (AT1G29510).
AT5G16023	-8.2786836	3.25E-07	*ROTUNDIFOLIA LIKE 18 (RTFL18);DEVIL 1 (DVL1)*, encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.
AT5G44260	-7.3549486	1.4E-16	*TANDEM CCCH ZINC FINGER PROTEIN 5 (TZF5); (ATC3H61); (ATTZF5)*, encodes a Tandem CCCH Zinc Finger protein. Interacts and co-localizes with MARD1 and RD21A in processing bodies (PBs) and stress granules (SGs).
AT2G14900	-6.7787265	1.61E-09	*GASA7,* Gibberellin-regulated family protein.
AT4G14130	-6.3995536	0.00462239	*XYLOGLUCAN ENDOTRANSGLYCOSYLASE7 (XTR7); XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE15 (XTH15),* xyloglucan endotransglycosylase-related protein.
AT1G52830	-6.1280133	0.00000836	*INDOLE-3-ACETIC ACID 6 (IAA6);SHORT HYPOCOTYL 1 (SHY1)*, an extragenic dominant suppressor of the hy2 mutant phenotype. Also exhibits aspects of constitutive photomorphogenetic phenotype in the absence of hy2. Mutants have dominant leaf curling phenotype shortened hypocotyls and reduced apical hook. Induced by indole-3-acetic acid.
AT5G08150	-5.820022	0.01074387	*SUPPRESSOR OF PHYTOCHROME B 5 (SOB5)*, encodes SOB5. Activation tagging lines accumulated higher level of cytokinin.
AT5G62280	-5.7785679	0.000081	DUF1442 family protein (*DUF1442*).
AT1G52750	-5.7045577	0.00013919	Alpha/beta-Hydrolases superfamily protein.
AT4G32280	-5.6497836	2.12E-12	*INDOLE-3-ACETIC ACID INDUCIBLE 29 (IAA29)*, Indole-3-acetic acid inducible 29 protein involved in IAA signaling. Downstream target of PIF4.
AT4G40090	-5.4883416	8.8E-08	*ARABINOGALACTAN PROTEIN 3 (AGP3)*, arabinogalactan protein 3.
AT4G16563	-5.4674647	7.7E-35	Eukaryotic aspartyl protease family protein.
AT3G45970	-5.3046977	5.16E-13	*EXPANSIN L1 (EXPL1);* member of EXPANSIN-LIKE. Naming convention from the Expansin Working Group (Kende et al, 2004. Plant Mol Bio) The mRNA is cell-to-cell mobile.
AT4G08950	-5.2573599	6.38E-40	*EXORDIUM (EXO)*, cell wall localized protein of unknown function. Expressed in areas with rapidly dividing cells. Overexpression induces elements of brassinosteroid signaling pathways.
AT1G10550	-5.0746071	0.00392241	*TRANSFERASE 33 (XTH33);XYLOGLUCAN:XYLOGLUCOSYL TRANSFERASE 33 (XET),* encodes a membrane-localized protein that is predicted to function during cell wall modification.Overexpression of XTH33 results in abnormal cell morphology. It's expression is under epigenetic control by ATX1.
AT1G50040	-5.0672076	2.09E-20	Formin-like protein, putative (*DUF1005*).
AT5G65390	-4.9971002	3.48E-12	*ARABINOGALACTAN PROTEIN 7 (AGP7)*, arabinogalactan protein 7.
AT3G21330	-4.7912074	0.00086147	Basic helix-loop-helix (bHLH) DNA-binding superfamily protein.
AT5G18050	-4.7170981	0.00482749	*SMALL AUXIN UP RNA 22 (SAUR22),* SAUR-like auxin-responsive protein family.

Supplementary Table S21. Top 20 upregulated genes in WT plants treated with a 4 h-PL period (T4) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	T4 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT2G39030	12.4991922	2.43E-16	*N-ACETYLTRANSFERASE ACTIVITY 1 (NATA1)*, encodes a protein that acts as an ornithine N-delta-acetyltransferase, leading to the formation of N-delta-actetylornithine. This compound is likely used in plant defense and levels of it are increased in Arabidopsis plants in response to MeJA and ABA. The mRNA is cell-to-cell mobile.

AGI	T4 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT2G47520	12.0722407	1.97E-10	*ARABIDOPSIS THALIANA ETHYLENE RESPONSE FACTOR 71 (AtERF71)*, encodes a member of the ERF (ethylene response factor) subfamily B-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 5 members in this subfamily including RAP2.2 AND RAP2.12. It plays a role in hypoxia-induced root slanting.
AT5G51060	11.4072335	6.83E-22	*A. THALIANA RESPIRATORY BURST OXIDASE HOMOLOG C (ATRBOHC)*, RHD2 (along with RHD3 and RHD4) is required for normal root hair elongation. Has NADPH oxidase activity. Gene is expressed in the elongation and differention zone in trichoblasts and elongating root hairs. RDH2 is localized to the growing tips of root hair cells. It is required for the production of reactive oxygen species in response to extracellular ATP stimulus. The increase in ROS production stimulates Ca2+ influx.
AT3G44830	11.4050529	3.7E-10	Lecithin:cholesterol acyltransferase family protein.
AT1G76470	11.393673	1.76E-11	NAD(P)-binding Rossmann-fold superfamily protein.
AT1G69930	11.3678769	2.83E-10	*GLUTATHIONE S-TRANSFERASE TAU 11 (ATGSTU11),* encodes glutathione transferase belonging to the tau class of GSTs. Naming convention according to Wagner et al. (2002).
AT5G67310	11.3340845	3.83E-10	*SUBFAMILY G, POLYPEPTIDE 1 (CYP81G1)*, member of CYP81G.
AT1G33730	11.3133016	3.16E-09	*CYTOCHROME P450, FAMILY 76, SUBFAMILY C, POLYPEPTIDE 5 (CYP76C5)*, cytochrome P450, family 76, subfamily C, polypeptide 5.
AT4G08770	11.0906672	1.94E-09	*PEROXIDASE 37 (Prx37)*, encodes a putative apoplastic peroxidase Prx37. Primarily expressed in the vascular bundles. Overexpression renders a dwarf phenotype with smaller plants and delayed development. Plants overexpressing Prx37 also shows an increase in the amount of esterified phenolic material associated with their walls.
AT3G46080	11.0660365	8.14E-23	C2H2-type zinc finger family protein.
AT2G38240	11.0223091	5.13E-25	*JASMONIC ACID OXIDASE 4 (JAO4)*, one of 4 paralogs encoding a 2-oxoglutarate/Fe(II)-dependent oxygenases that hydroxylates JA to 12-OH-JA.
AT3G49110	10.8521459	1.83E-09	*PEROXIDASE 33 (PRX33);PEROXIDASE CA (ATPCA); (ATPRX33),* Class III peroxidase Perx33. Expressed in roots. Located in the cell wall. Involved in cell elongation. Expression activated by light. May play a role in generating H2O2 during defense response. The mRNA is cell-to-cell mobile.
AT2G44460	10.7841467	8.64E-10	*BETA GLUCOSIDASE 28 (BGLU28)*, Beta-glucosidase, major myrosinase which initiates sulfur reallocation by hydrolyzing particular GL species, conferring sulfur deficiency tolerance, especially during early development.
AT2G35730	10.7152019	3.48E-08	*HEAVY METAL ASSOCIATED PROTEIN 19 (ATHMP19)*, heavy metal transport/detoxification superfamily protein.
AT5G48410	10.5515831	3.27E-09	*GLUTAMATE RECEPTOR 1.3 (GLR1.3)*, member of Putative ligand-gated ion channel subunit family.
AT4G22070	10.5506236	5.98E-09	*WRKY DNA-BINDING PROTEIN 31 (WRKY31),* member of WRKY Transcription Factor; Group II-b.
AT4G14630	10.447413	5.63E-08	*GERMIN-LIKE PROTEIN 9 (GLP9)*, germin-like protein with N-terminal signal sequence that may target it to the vacuole, plasma membrane and/or outside the cell. The mRNA is cell-to-cell mobile.
AT4G16260	10.4312634	2.74E-07	Encodes a putative beta-1,3-endoglucanase that interacts with the 30C02 cyst nematode effector. May play a role in host defense.
AT1G67980	10.3847289	2.16E-08	*CAFFEOYL-COA 3-O-METHYLTRANSFERASE (CCOAMT),* encodes S-adenosyl-L-methionine: transcaffeoyl Coenzyme A 3-O-methyltransferase. Methyltransferase in the lignin biosynthetic pathway.
AT2G30750	10.3824763	8.47E-21	*CYTOCHROME P450, FAMILY 71, SUBFAMILY A, POLYPEPTIDE 12 (CYP71A12)*, putative cytochrome P450; together with CYP71A13 produces dihydrocamalexic acid (DHCA), the precursor to the defense-related compound camalexin, which accumulates in the intercellular space and contributes to the resistance of mature Arabidopsis to P. syringae without directly inhibiting bacterial growth.

Supplementary Table S 22. Top 20 downregulated genes in WT plants treated with a 4 h-PL period (T4) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	T4 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT4G29020	-10.00447	0.0000117	Glycine-rich protein.
AT1G06080	-8.3949648	6.92E-09	*DELTA 9 DESATURASE 1 (ADS1)*, encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.
AT1G11850	-7.6370476	0.00096733	Transmembrane protein.
AT2G42870	-6.9910577	2.67E-08	*PHY RAPIDLY REGULATED 1 (PAR1);HELIX-LOOP-HELIX 1 (HLH1)*, encodes PHYTOCHROME RAPIDLY REGULATED1 (PAR1), an atypical basic helix-loop-helix (bHLP) protein. Closely related to PAR2 (At3g58850). Up regulated after simulated shade perception. Acts in the nucleus to control plant development and as a negative regulator of shade avoidance response. Functions as transcriptional repressor of auxin-responsive genes SAUR15 (AT4G38850) and SAUR68 (AT1G29510).
AT1G52750	-6.9123215	0.0000209	alpha/beta-Hydrolases superfamily protein.
AT4G14130	-6.876164	0.01045614	*XYLOGLUCAN ENDOTRANSGLYCOSYLASE 7 (XTR7); XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE 15 (XTH15),* xyloglucan endotransglycosylase-related protein (XTR7).
AT5G48490	-6.7822979	0.00164518	*(DEG15);DIR1-LIKE (DIR1-LIKE),* encodes a protein with similarity to a lipid transfer protein that may contribute to systemic acquired resistance (SAR).
AT4G38825	-6.2605521	0.00305738	*SMALL AUXIN UPREGULATED RNA 13 (SAUR13)*, SAUR-like auxin-responsive protein family.

AGI	T4 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT3G23730	-6.1413331	5.44E-10	*XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE 16 (XTH16)*, xyloglucan endotransglucosylase/hydrolase 16.
AT5G65390	-6.0167489	9.41E-15	*ARABINOGALACTAN PROTEIN 7 (AGP7)*, arabinogalactan protein 7.
AT4G40090	-5.7899317	0.00000199	*ARABINOGALACTAN PROTEIN 3 (AGP3)*, arabinogalactan protein 3.
AT5G22940	-5.6597303	2.34E-14	Homolog of FRA8 (AT2G28110), a member of a member of glycosyltransferase family 47; exhibits high sequence similarity to tobacco (Nicotiana plumbaginifolia) pectin glucuronyltransferase.
AT2G34430	-5.6535834	0.00079206	*LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX II SUBUNIT B1 (LHCB1.4); (DEG11);LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX II SUBUNIT B1 (LHB1B1),* Photosystem II type I chlorophyll a/b-binding protein The mRNA is cell-to-cell mobile.
AT1G04800	-5.5576606	0.0000301	Glycine-rich protein.
AT1G11740	-5.5060085	0.00025491	Ankyrin repeat family protein.
AT5G18430	-5.4547207	0.03361077	GDSL-motif esterase/acyltransferase/lipase. Enzyme group with broad substrate specificity that may catalyze acyltransfer or hydrolase reactions with lipid and non-lipid substrates.
AT1G10550	-5.4539669	0.00051128	*XYLOGLUCAN:XYLOGLUCOSYL TRANSFERASE 33 (XET)*, encodes a membrane-localized protein that is predicted to function during cell wall modification.Overexpression of XTH33 results in abnormal cell morphology.
AT1G52830	-5.4379779	0.0000192	*INDOLE-3-ACETIC ACID 6 (IAA6) INDOLE-3-ACETIC ACID 6 (IAA6);SHORT HYPOCOTYL 1 (SHY1),* an extragenic dominant suppressor of the hy2 mutant phenotype. Also exhibits aspects of constitutive photomorphogenetic phenotype in the absence of hy2. Mutants have dominant leaf curling phenotype shortened hypocotyls and reduced apical hook. Induced by indole-3-acetic acid.
AT3G60290	-5.33561	0.00018547	2-oxoglutarate (2OG) and Fe(II)-dependent oxygenase superfamily protein.
AT1G75590	-5.3098412	0.04174198	*SMALL AUXIN UPREGULATED RNA 52 (SAUR52)*, SAUR-like auxin-responsive protein family.

Supplementary Table S23. Top 20 upregulated genes in WT plants treated with a 4 h-PL period as priming and triggering with four days lag phase in-between (PL4T) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	PL4T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT2G39030	12.2349259	1.49E-15	*N-ACETYLTRANSFERASE ACTIVITY 1 (NATA1)*, encodes a protein that acts as an ornithine N-delta-acetyltransferase, leading to the formation of N-delta-acetylornithine. This compound is likely used in plant defense and levels of it are increased in Arabidopsis plants in response to MeJA and ABA.
AT1G76470	11.4415171	1.3E-11	NAD(P)-binding Rossmann-fold superfamily protein.
AT4G08770	11.2364534	8.98E-10	*PEROXIDASE 37 (Prx37)*, encodes a putative apoplastic peroxidase Prx37. Primarily expressed in the vascular bundles. Overexpression renders a dwarf phenotype with smaller plants and delayed development. Plants overexpressing Prx37 also shows an increase in the amount of esterified phenolic material associated with their walls.
AT1G61120	11.196888	2.05E-09	*(GES);TERPENE SYNTHASE 04 (TPS04);TERPENE SYNTHASE 4 (TPS4)*, encodes a geranyllinalool synthase that produces a precursor to TMTT, a volatile plant defense C16-homoterpene. GES transcript levels rise in response to alamethicin, a fungal peptide mixture that damages membranes. This transcriptional response is blocked in JA biosynthetic and JA signaling mutants, but GES transcript levels still rise in response to alamethicin in mutants with salicylic acid and ethylene biosynthetic and/or signaling defects.
AT3G44830	11.1267793	1.52E-09	Lecithin:cholesterol acyltransferase family protein.
AT2G44460	11.0658526	1.81E-10	*BETA GLUCOSIDASE 28 (BGLU28)*, Beta-glucosidase, major myrosinase which initiates sulfur reallocation by hydrolyzing particular GL species, conferring sulfur deficiency tolerance, especially during early development.
AT2G47520	10.7994137	8.57E-08	*ARABIDOPSIS THALIANA ETHYLENE RESPONSE FACTOR 71 (AtERF71)*, encodes a member of the ERF (ethylene response factor) subfamily B-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 5 members in this subfamily including RAP2.2 AND RAP2.12. It plays a role in hypoxia-induced root slanting.
AT5G51060	10.742221	4.02E-19	*A. THALIANA RESPIRATORY BURST OXIDASE HOMOLOG C (ATRBOHC)*, RHD2 (along with RHD3 and RHD4) is required for normal root hair elongation. Has NADPH oxidase activity. Gene is expressed in the elongation and differention zone in trichoblasts and elongating root hairs. RDH2 is localized to the growing tips of root hair cells. It is required for the production of reactive oxygen species in response to extracellular ATP stimulus. The increase in ROS production stimulates Ca2+ influx.
AT4G16260	10.722883	7.11E-08	Encodes a putative beta-1,3-endoglucanase that interacts with the 30C02 cyst nematode effector. May play a role in host defense.
AT1G69930	10.6890269	9.07E-09	*GLUTATHIONE S-TRANSFERASE TAU 11 (ATGSTU11)*, encodes glutathione transferase belonging to the tau class of GSTs. Naming convention according to Wagner et al. (2002).
AT1G33730	10.6292612	8.03E-08	*CYTOCHROME P450, FAMILY 76, SUBFAMILY C, POLYPEPTIDE 5 (CYP76C5),* cytochrome P450, family 76, subfamily C, polypeptide 5.
AT5G67310	10.6258612	1.38E-08	*CYTOCHROME P450, FAMILY 81, SUBFAMILY G, POLYPEPTIDE 1 (CYP81G1)*, member of CYP81G.
AT1G67980	10.1974589	5.3E-08	*CAFFEOYL-COA 3-O-METHYLTRANSFERASE (CCOAMT)*, encodes S-adenosyl-L-methionine: transcaffeoyl Coenzyme A 3-O-methyltransferase. Methyltransferase in the lignin biosynthetic pathway.
AT2G38240	10.1825513	5.71E-21	*JASMONIC ACID OXIDASE 4 (JAO4),* one of 4 paralogs encoding a 2-oxoglutarate/Fe(II)-dependent oxygenases that hydroxylates JA to 12-OH-JA.

AGI	PL4T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT4G15210	10.0728207	0.00000827	*REDUCED BETA AMYLASE 1 (RAM1); (BMY1);BETA-AMYLASE 5 (BAM5),* cytosolic beta-amylase expressed in rosette leaves and inducible by sugar. RAM1 mutants have reduced beta amylase in leaves and stems.
AT1G66570	10.0291164	1.11E-07	*SUCROSE-PROTON SYMPORTER 7 (SUC7)*, sucrose-proton symporter 7.
AT3G19615	10.0099258	8.24E-08	Beta-1,4-xylosidase.
AT1G43160	9.99706298	0.0000065	*RELATED TO AP2 6 (RAP2.6);ETHYLENE RESPONSIVE FACTOR113 (ERF113),* encodes a member of the ERF (ethylene response factor) subfamily B-4 of ERF/AP2 transcription factor family (RAP2.6). The protein contains one AP2 domain. There are 7 members in this subfamily.
AT5G17220	9.93765672	0.00000445	*GLUTATHIONE S-TRANSFERASE PHI 12 (GSTF12);TRANSPARENT TESTA 19 (TT19);GLUTATHIONE S-TRANSFERASE 26 (GST26)*, encodes glutathione transferase belonging to the phi class of GSTs. Naming convention according to Wagner et al. (2002). Mutants display no pigments on leaves and stems. Likely to function as a carrier to transport anthocyanin from the cytosol to tonoplasts.
AT5G48410	9.92851919	7.68E-08	*GLUTAMATE RECEPTOR 1.3 (GLR1.3)*, member of Putative ligand-gated ion channel subunit family.

Supplementary Table S24. Top 20 downregulated genes in WT plants treated with a 4 h-PL period as priming and triggering with four days lag phase in-between (PL4T) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	PL4T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT4G40090	-8.3257823	0.00000019	*ARABINOGALACTAN PROTEIN 3 (AGP3)*, arabinogalactan protein 3.
AT1G06080	-7.4773696	3.11E-08	*DELTA 9 DESATURASE 1 (ADS1)*, encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.
AT1G11740	-6.343446	0.00017388	Ankyrin repeat family protein.
AT4G16563	-6.1082217	1.57E-39	Eukaryotic aspartyl protease family protein.
AT4G14130	-5.988341	0.00586603	*XYLOGLUCAN ENDOTRANSGLYCOSYLASE 7 (XTR7); XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE 15 (XTH15)*, xyloglucan endotransglycosylase-related protein (XTR7).
AT2G42870	-5.9195777	0.00000228	*PHY RAPIDLY REGULATED 1 (PAR1); HELIX-LOOP-HELIX 1 (HLH1)*, encodes PHYTOCHROME RAPIDLY REGULATED1 (PAR1), an atypical basic helix-loop-helix (bHLP) protein. Closely related to PAR2 (At3g58850). Up regulated after simulated shade perception. Acts in the nucleus to control plant development and as a negative regulator of shade avoidance response. Functions as transcriptional repressor of auxin-responsive genes SAUR15 (AT4G38850) and SAUR68 (AT1G29510).
AT1G10550	-5.836131	0.0000552	*XYLOGLUCAN:XYLOGLUCOSYL TRANSFERASE 33 (XET)*, encodes a membrane-localized protein that is predicted to function during cell wall modification.Overexpression of XTH33 results in abnormal cell morphology. It's expression is under epigenetic control by ATX1.
AT5G16023	-5.780331	7E-13	*ROTUNDIFOLIA LIKE 18 (RTFL18);DEVIL1 (DVL1)*, encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.
AT1G72620	-5.2807075	0.00247583	Alpha/beta-Hydrolases superfamily protein.
AT5G65390	-5.2800002	3.36E-13	*ARABINOGALACTAN PROTEIN 7 (AGP7)*, arabinogalactan protein 7.
AT5G02760	-5.2515	0.0000391	*SENESCENCE-SUPPRESSED 51 PROTEIN PHOSPHATASE (SSPP);ARABIDOPSIS PP2C CLADE D 7 (APD7)*, encodes a phosphatase that functions in sustaining proper leaf longevity and preventing early senescence by suppressing or perturbing SARK-mediated senescence signal transduction.
AT5G44260	-5.2400211	1.59E-08	*TANDEM CCCH ZINC FINGER PROTEIN 5 (TZF5); (ATC3H61); (ATTZF5)*, encodes a Tandem CCCH Zinc Finger protein. Interacts and co-localizes with MARD1 and RD21A in processing bodies (PBs) and stress granules (SGs).
AT5G62280	-5.2310917	0.0012844	DUF1442 family protein (DUF1442).
AT4G01335	-5.2236795	3.26E-11	TATA box-binding protein associated factor RNA polymerase I subunit B-like protein.
AT5G57760	-4.8309456	0.00000253	Hypothetical protein.
AT3G61090	-4.8254635	0.005598	Putative endonuclease or glycosyl hydrolase.
AT1G52750	-4.7402578	0.00718772	Alpha/beta-Hydrolases superfamily protein.
AT5G57550	-4.7203507	0.03158985	*XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE 25 (XTH25); XYLOGLUCAN ENDOTRANSGLYCOSYLASE 3 (XTR3)*, xyloglucan endotransglycosylase-related protein.
AT4G32280	-4.6543298	3.58E-08	*INDOLE-3-ACETIC ACID INDUCIBLE 29 (IAA29)*, Indole-3-acetic acid inducible 29 protein involved in IAA signaling. Downstream target of PIF4.
AT1G50040	-4.6250738	5.34E-17	Formin-like protein, putative (DUF1005).

Supplementary Table S25. Top 20 upregulated genes in WT plants treated with a 4 h-PL period (T5) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	T5 *vs.* control		Short description derived by TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT2G39030	13.6649381	4.67E-20	*N-ACETYLTRANSFERASE ACTIVITY 1 (NATA1)*, encodes a protein that acts as an ornithine N-delta-acetyltransferase, leading to the formation of N-delta-actetylornithine. This compound is likely used in plant defense and levels of it are increased in Arabidopsis plants in response to MeJA and ABA. The mRNA is cell-to-cell mobile.
AT1G76470	12.8109148	3.02E-15	NAD(P)-binding Rossmann-fold superfamily protein.
AT2G47520	12.7236554	6.76E-12	*ETHYLENE RESPONSE FACTOR 71 (ERF71);ARABIDOPSIS THALIANA ETHYLENE RESPONSE FACTOR 71 (AtERF71)*, encodes a member of the ERF (ethylene response factor) subfamily B-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 5 members in this subfamily including RAP2.2 AND RAP2.12. It plays a role in hypoxia-induced root slanting.
AT1G61120	12.5530398	1.55E-12	*TERPENE SYNTHASE 4 (TPS4)*, encodes a geranyllinalool synthase that produces a precursor to TMTT, a volatile plant defense C16-homoterpene. GES transcript levels rise in response to alamethicin, a fungal peptide mixture that damages membranes. This transcriptional response is blocked in JA biosynthetic and JA signaling mutants, but GES transcript levels still rise in response to alamethicin in mutants with salicylic acid and ethylene biosynthetic and/or signaling defects. GES transcripts also accumulate in response to a larval infestation. This enzyme does not localize to the plastids, and it may be present in the cytosol or endoplasmic reticulum.
AT1G52690	12.4922058	0.00271	*LATE EMBRYOGENESIS ABUNDANT 7 (LEA7)*, Intrinsically disordered protein that stabilizes lactate dehydrogenase (LDH) during drying and freezing.
AT1G69930	12.4808765	5.90E-13	*GLUTATHIONE S-TRANSFERASE TAU 11 (GSTU11);GLUTATHIONE S-TRANSFERASE TAU 11 (ATGSTU11)*, encodes glutathione transferase belonging to the tau class of GSTs. Naming convention according to Wagner et al. (2002).
AT1G33730	12.4046071	1.17E-11	*CYTOCHROME P450, FAMILY 76, SUBFAMILY C, POLYPEPTIDE 5 (CYP76C5)*, cytochrome P450, family 76, subfamily C, polypeptide 5.
AT1G43160	12.3903659	1.37E-10	*RELATED TO AP2 6 (RAP2.6);ETHYLENE RESPONSIVE FACTOR113 (ERF113)*, encodes a member of the ERF (ethylene response factor) subfamily B-4 of ERF/AP2 transcription factor family (RAP2.6). The protein contains one AP2 domain. There are 7 members in this subfamily.
AT5G51060	12.3404413	4.53E-26	*A. THALIANA RESPIRATORY BURST OXIDASE HOMOLOG C (ATRBOHC)*, RHD2 (along with RHD3 and RHD4) is required for normal root hair elongation. Has NADPH oxidase activity. Gene is expressed in the elongation and differention zone in trichoblasts and elongating root hairs. RDH2 is localized to the growing tips of root hair cells. It is required for the production of reactive oxygen species in response to extracellular ATP stimulus. The increase in ROS production stimulates Ca2+ influx.
AT3G44830	12.1369506	7.12E-12	Lecithin:cholesterol acyltransferase family protein.
AT4G08770	11.9744143	1.82E-11	*PEROXIDASE 37 (Prx37)*, encodes a putative apoplastic peroxidase Prx37. Primarily expressed in the vascular bundles. Overexpression renders a dwarf phenotype with smaller plants and delayed development. Plants overexpressing Prx37 also shows an increase in the amount of esterified phenolic material associated with their walls.
AT5G67310	11.9460463	1.41E-11	*CYTOCHROME P450, FAMILY 81, SUBFAMILY G, POLYPEPTIDE 1 (CYP81G1)*, member of CYP81G.
AT1G67980	11.78656	1.27E-11	*CAFFEOYL-COA 3-O-METHYLTRANSFERASE (CCOAMT),* encodes S-adenosyl-L-methionine: transcaffeoyl Coenzyme A 3-O-methyltransferase. Methyltransferase in the lignin biosynthetic pathway.
AT4G14630	11.7794222	6.66E-11	*GERMIN-LIKE PROTEIN 9 (GLP9)*, germin-like protein with N-terminal signal sequence that may target it to the vacuole, plasma membrane and/or outside the cell. The mRNA is cell-to-cell mobile.
AT4G16260	11.711888	6.32E-10	Encodes a putative beta-1,3-endoglucanase that interacts with the 30C02 cyst nematode effector. May play a role in host defense.
AT3G46080	11.4775921	9.59E-25	*ZAT8,* C2H2-type zinc finger family protein.
AT3G19615	11.3677474	6.99E-11	β-1,4-xylosidase.
AT1G66570	11.3237504	1.49E-10	*SUCROSE-PROTON SYMPORTER 7 (SUC7)*, sucrose-proton symporter 7.
AT5G48410	11.2908529	5.91E-11	*GLUTAMATE RECEPTOR 1.3 (GLR1.3)*, member of Putative ligand-gated ion channel subunit family.
AT3G49110	11.2238203	2.56E-10	*PEROXIDASE CA (PRXCA);PEROXIDASE 33 (PRX33);PEROXIDASE CA (ATPCA); (ATPRX33)*, Class III peroxidase Perx33. Expressed in roots. Located in the cell wall. Involved in cell elongation. Expression activated by light. May play a role in generating H2O2 during defense response. The mRNA is cell-to-cell mobile.

Supplementary Table S26. Top 20 downregulated genes in WT plants treated with a 4 h-PL period (T5) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	T5 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT1G06080	-11.38833	1.16E-09	*DELTA 9 DESATURASE 1 (ADS1)*, encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.
AT2G42870	-10.846269	2.08E-09	*PHY RAPIDLY REGULATED 1 (PAR1);HELIX-LOOP-HELIX 1 (HLH1)*, encodes PHYTOCHROME RAPIDLY REGULATED1 (PAR1), an atypical basic helix-loop-helix (bHLP) protein. Closely related to PAR2 (At3g58850). Up regulated after simulated shade perception. Acts in the nucleus to control plant development and as a negative regulator of shade avoidance response. Functions as transcriptional repressor of auxin-responsive genes SAUR15 (AT4G38850) and SAUR68 (AT1G29510).
AT5G48490	-10.747937	4.69E-06	*(DEG15);DIR1-LIKE (DIR1-LIKE)*, encodes a protein with similarity to a lipid transfer protein that may contribute to systemic acquired resistance (SAR).
AT5G18050	-9.9636424	1.93E-07	*SMALL AUXIN UP RNA 22 (SAUR22)*, SAUR-like auxin-responsive protein family.
AT2G34430	-9.7271319	8.77E-17	*LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX II SUBUNIT B1 (LHB1B1)*, Photosystem II type I chlorophyll a/b-binding protein The mRNA is cell-to-cell mobile.

AGI	T5 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT4G32280	-9.6285845	3.23E-11	*INDOLE-3-ACETIC ACID INDUCIBLE 29 (IAA29)*, Indole-3-acetic acid inducible 29 protein involved in IAA signaling. Downstream target of PIF4.
AT5G45820	-9.1792082	4.56E-11	*CBL-INTERACTING PROTEIN KINASE 20 (CIPK20);PROTEIN KINASE 18 (PKS18);SNF1-RELATED PROTEIN KINASE 3.6 (SnRK3.6)*, encodes a CBL-interacting serine/threonine protein kinase comprised of an N-terminal kinase catalytic domain similar to SNF1/AMPK and a unique C-terminal regulatory domain.
AT5G16023	-8.956344	6.33E-09	*ROTUNDIFOLIA LIKE 18 (RTFL18);DEVIL 1 (DVL1)*, encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.
AT1G52750	-8.6293647	7.76E-05	Alpha/beta-Hydrolases superfamily protein.
AT4G14130	-8.5651641	0.000191	*XYLOGLUCAN ENDOTRANSGLYCOSYLASE 7 (XTR7);XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE 15 (XTH15),* xyloglucan endotransglycosylase-related protein (XTR7)
AT3G21330	-8.5452866	4.14E-05	Basic helix-loop-helix (bHLH) DNA-binding superfamily protein.
AT1G10550	-8.1009017	3.85E-10	*TRANSFERASE 33 (XTH33);XYLOGLUCAN:XYLOGLUCOSYL TRANSFERASE 33 (XET)*, encodes a membrane-localized protein that is predicted to function during cell wall modification.Overexpression of XTH33 results in abnormal cell morphology. It's expression is under epigenetic control by ATX1.
AT5G62280	-8.0825355	1.98E-07	DUF1442 family protein (DUF1442).
AT3G27690	-8.0765916	2.80E-11	*DEG13*, encodes Lhcb2.4. Belongs to the Lhc super-gene family encodes the light-harvesting chlorophyll a/b-binding (LHC) proteins that constitute the antenna system of the photosynthetic apparatus.The mRNA is cell-to-cell mobile. Activated by OXS2 under the treatment of salt.
AT4G40090	-8.0593214	8.63E-07	*ARABINOGALACTAN PROTEIN 3 (AGP3)*, arabinogalactan protein 3.
AT5G57550	-8.0483299	1.26E-10	*XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE 25 (XTH25);XYLOGLUCAN ENDOTRANSGLYCOSYLASE 3 (XTR3),* xyloglucan endotransglycosylase-related protein (XTR3).
AT5G45670	-7.9407417	0.027008	GDSL-motif esterase/acyltransferase/lipase. Enzyme group with broad substrate specificity that may catalyze acyltransfer or hydrolase reactions with lipid and non-lipid substrates.
AT4G24275	-7.8282867	2.93E-05	Identified as a screen for stress-responsive genes.
AT5G22940	-7.7656448	2.90E-16	Homolog of FRA8 (AT2G28110), a member of a member of glycosyltransferase family 47; exhibits high sequence similarity to tobacco (Nicotiana plumbaginifolia) pectin glucuronyltransferase.
AT3G58120	-7.7223196	1.47E-08	*(ATBZIP61); (BZIP61),* encodes a member of the BZIP family of transcription factors. Forms heterodimers with the related protein AtbZIP34. Binds to G-boxes in vitro and is localized to the nucleus in onion epidermal cells.

Supplementary Table S27. Top 20 upregulated genes in WT plants treated with a 4 h-PL period as priming and triggering with five days lag phase in-between (PL5T) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	PL5T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT2G47520	12.1485439	1.34E-10	*HYPOXIA RESPONSIVE ERF (ETHYLENE RESPONSE FACTOR) 2 (HRE2); ETHYLENE RESPONSE FACTOR 71 (ERF71);ARABIDOPSIS THALIANA ETHYLENE RESPONSE FACTOR 71 (AtERF71)*, encodes a member of the ERF (ethylene response factor) subfamily B-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 5 members in this subfamily including RAP2.2 AND RAP2.12. It plays a role in hypoxia-induced root slanting.
AT5G51060	11.8551527	7.39E-24	*A. THALIANA RESPIRATORY BURST OXIDASE HOMOLOG C (ATRBOHC)*, RHD2 (along with RHD3 and RHD4) is required for normal root hair elongation. Has NADPH oxidase activity. Gene is expressed in the elongation and differention zone in trichoblasts and elongating root hairs. RDH2 is localized to the growing tips of root hair cells. It is required for the production of reactive oxygen species in response to extracellular ATP stimulus. The increase in ROS production stimulates Ca2+ influx.
AT3G44830	11.5905549	1.39E-10	Lecithin:cholesterol acyltransferase family protein.
AT2G39030	11.3502708	5.39E-13	*N-ACETYLTRANSFERASE ACTIVITY 1 (NATA1)*, encodes a protein that acts as an ornithine N-delta-acetyltransferase, leading to the formation of N-delta-actetylornithine. This compound is likely used in plant defense and levels of it are increased in Arabidopsis plants in response to MeJA and ABA. The mRNA is cell-to-cell mobile.
AT2G44460	11.2666544	6.06E-11	*BETA GLUCOSIDASE 28 (BGLU28)*, Beta-glucosidase, major myrosinase which initiates sulfur reallocation by hydrolyzing particular GL species, conferring sulfur deficiency tolerance, especially during early development.
AT1G76470	11.0929219	9.78E-11	NAD(P)-binding Rossmann-fold superfamily protein.
AT3G46080	10.9708246	2.22E-22	*ZAT8,* C2H2-type zinc finger family protein.
AT1G69930	10.9542026	2.44E-09	*GLUTATHIONE S-TRANSFERASE TAU 11 (GSTU11); GLUTATHIONE S-TRANSFERASE TAU 11 (ATGSTU11)*, encodes glutathione transferase belonging to the tau class of GSTs. Naming convention according to Wagner et al. (2002).
AT2G02010	10.8142936	8.74E-36	Glutamate decarboxylase.
AT1G33730	10.7789006	4.10E-08	*CYTOCHROME P450, FAMILY 76, SUBFAMILY C, POLYPEPTIDE 5 (CYP76C5),* cytochrome P450, family 76, subfamily C, polypeptide 5.
AT5G67310	10.7668222	7.04E-09	*CYTOCHROME P450, FAMILY 81, SUBFAMILY G, POLYPEPTIDE 1 (CYP81G1)*, member of CYP81G.
AT3G49580	10.6823828	4.77E-31	*RESPONSE TO LOW SULFUR 1 (LSU1)*, RESPONSE TO LOW SULFUR gene family member; expressed during sulfur deficiency.
AT3G49110	10.6259321	5.90E-09	*PEROXIDASE CA (PRXCA); PEROXIDASE 33 (PRX33); PEROXIDASE CA (ATPCA); (ATPRX33)*, Class III peroxidase Perx33. Expressed in roots. Located in the cell wall. Involved in cell elongation. Expression activated by light. May play a role in generating H2O2 during defense response. The mRNA is cell-to-cell mobile.

AGI	PL5T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT4G08770	10.5507909	2.84E-08	*PEROXIDASE 37 (Prx37)*, encodes a putative apoplastic peroxidase Prx37. Primarily expressed in the vascular bundles. Overexpression renders a dwarf phenotype with smaller plants and delayed development. Plants overexpressing Prx37 also shows an increase in the amount of esterified phenolic material associated with their walls.
AT2G30750	10.5310071	1.78E-21	*CYTOCHROME P450, FAMILY 71, SUBFAMILY A, POLYPEPTIDE 12 (CYP71A12)*, Putative cytochrome P450; together with CYP71A13 produces dihydrocamalexic acid (DHCA), the precursor to the defense-related compound camalexin, which accumulates in the intercellular space and contributes to the resistance of mature Arabidopsis to P. syringae without directly inhibiting bacterial growth.
AT1G61120	10.4196626	9.18E-08	*GERANYLLINALOOL SYNTHASE (GES); TERPENE SYNTHASE 04 (TPS04);TERPENE SYNTHASE 4 (TPS4)*, encodes a geranyllinalool synthase that produces a precursor to TMTT, a volatile plant defense C16-homoterpene. GES transcript levels rise in response to alamethicin, a fungal peptide mixture that damages membranes. This transcriptional response is blocked in JA biosynthetic and JA signaling mutants, but GES transcript levels still rise in response to alamethicin in mutants with salicylic acid and ethylene biosynthetic and/or signaling defects. GES transcripts also accumulate in response to a larval infestation. This enzyme does not localize to the plastids, and it may be present in the cytosol or endoplasmic reticulum. The mRNA is cell-to-cell mobile.
AT4G14630	10.3755108	7.93E-08	*GERMIN-LIKE PROTEIN 9 (GLP9)*, germin-like protein with N-terminal signal sequence that may target it to the vacuole, plasma membrane and/or outside the cell. The mRNA is cell-to-cell mobile.
AT1G05680	10.3559641	7.53E-13	*URIDINE DIPHOSPHATE GLYCOSYLTRANSFERASE 74E2 (UGT74E2),* encodes a UDP-glucosyltransferase, UGT74E2, that acts on IBA (indole-3-butyric acid) and affects auxin homeostasis. The transcript and protein levels of this enzyme are strongly induced by H2O2 and may allow integration of ROS (reactive oxygen species) and auxin signaling. This enzyme can also transfer glycosyl groups to several compounds related to the explosive TNT when this synthetic compound is taken up from the environment.
AT5G48410	10.3318381	1.03E-08	*ARABIDOPSIS THALIANA GLUTAMATE RECEPTOR 1.3 (ATGLR1.3); GLUTAMATE RECEPTOR 1.3 (GLR1.3),* member of Putative ligand-gated ion channel subunit family.
AT3G25250	10.2312035	2.11E-20	*AGC2 KINASE 1 (AGC2-1); (AGC2); OXIDATIVE SIGNAL-INDUCIBLE1 (OXI1); (AtOXI1),* Arabidopsis protein kinase The mRNA is cell-to-cell mobile.

Supplementary Table S28. Top 20 downregulated genes in WT plants treated with a 4 h-PL as priming and triggering with five days lag phase in-between (PL5T) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	PL5T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT5G48490	-10.071654	6.07E-06	*DIR1-LIKE (DIR1-LIKE*), encodes a protein with similarity to a lipid transfer protein that may contribute to systemic acquired resistance (SAR).
AT2G42870	-10.064534	1.04E-07	*PHY RAPIDLY REGULATED 1 (PAR1);HELIX-LOOP-HELIX 1 (HLH1)*, encodes PHYTOCHROME RAPIDLY REGULATED1 (PAR1), an atypical basic helix-loop-helix (bHLP) protein. Closely related to PAR2 (At3g58850). Up regulated after simulated shade perception. Acts in the nucleus to control plant development and as a negative regulator of shade avoidance response. Functions as transcriptional repressor of auxin-responsive genes SAUR15 (AT4G38850) and SAUR68 (AT1G29510).
AT1G06080	-9.1757311	1.30E-08	*(ATADS1);DELTA 9 DESATURASE 1 (ADS1)*, encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.
AT3G47340	-9.0402044	9.63E-05	*GLUTAMINE-DEPENDENT ASPARAGINE SYNTHASE 1 (ASN1);DARK INDUCIBLE 6 (DIN6);ARABIDOPSIS THALIANA GLUTAMINE-DEPENDENT ASPARAGINE SYNTHASE 1 (AT-ASN1),* encodes a glutamine-dependent asparagine synthetase, the predicted ASN1 peptide contains a purF-type glutamine-binding domain, and is expressed predominantly in shoot tissues, where light has a negative effect on its mRNA accumulation. Expression is induced within 3 hours of dark treatment, in senescing leaves and treatment with exogenous photosynthesis inhibitor. Induction of gene expression was suppressed in excised leaves supplied with sugar. The authors suggest that the gene's expression pattern is responding to the level of sugar in the cell.
AT2G34430	-8.7705887	3.51E-13	*LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX II SUBUNIT B1 (LHCB1.4); (DEG11);LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX II SUBUNIT B1 (LHB1B1),* Photosystem II type I chlorophyll a/b-binding protein The mRNA is cell-to-cell mobile.
AT4G40090	-8.2393818	3.17E-07	*ARABINOGALACTAN PROTEIN 3 (AGP3)*, arabinogalactan protein 3.
AT5G16023	-8.1746091	5.80E-07	*ROTUNDIFOLIA LIKE 18 (RTFL18);DEVIL 1 (DVL1)*, encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.
AT4G32280	-7.9960482	4.65E-17	*INDOLE-3-ACETIC ACID INDUCIBLE 29 (IAA29),* Indole-3-acetic acid inducible 29 protein involved in IAA signaling. Downstream target of PIF4.
AT4G14130	-7.7834294	0.003669	*XYLOGLUCAN ENDOTRANSGLYCOSYLASE 7 (XTR7); XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE 15 (XTH15),* xyloglucan endotransglycosylase-related protein (XTR7).
AT1G52750	-7.3736839	2.54E-05	Alpha/beta-Hydrolases superfamily protein.
AT5G18050	-7.3128185	2.13E-06	*SMALL AUXIN UP RNA 22 (SAUR22)*, SAUR-like auxin-responsive protein family.
AT1G04180	-7.0869786	0.003093	*YUCCA 9 (YUC9*).
AT5G45820	-7.0724626	3.35E-08	*CBL-INTERACTING PROTEIN KINASE 20 (CIPK20);PROTEIN KINASE 18 (PKS18);SNF1-RELATED PROTEIN KINASE 3.6 (SnRK3.6)*, encodes a CBL-interacting serine/threonine protein kinase comprised of an N-terminal kinase catalytic domain similar to SNF1/AMPK and a unique C-terminal regulatory domain.
AT1G29490	-7.0664139	0.005195	*SMALL AUXIN UPREGULATED 68 (SAUR68)*, SAUR-like auxin-responsive protein family.
AT1G62510	-6.7253443	5.35E-05	Expressed in the root cortex.

AGI	PL5T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT4G08109	-6.6839863	0.002368	Transposable element gene.
AT1G02620	-6.5445838	0.044719	Ras-related small GTP-binding family protein.
AT5G26280	-6.5150221	0.024944	TRAF-like family protein.
AT5G57550	-6.4691812	1.64E-06	*ENDOTRANSGLUCOSYLASE/HYDROLASE 25 (XTH25); XYLOGLUCAN ENDOTRANSGLYCOSYLASE 3 (XTR3),* xyloglucan endotransglycosylase-related protein (XTR3).
AT2G43010	-6.4005806	5.25E-27	Isolated as a semidominant mutation defective in red-light responses. Encodes a nuclear localized bHLH protein that interacts with active PhyB protein. Negatively regulates phyB mediated red light responses. Involved in shade avoidance response. Protein abundance is negatively regulated by PhyB.Involved in the regulation of response to nutrient levels. Controls the resistance to B. cinerea in a COI1- and EIN2-dependent manner.

Supplementary Table S29. Top 20 upregulated genes in WT plants treated with a 4 h-PL period (T10) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	T10 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT2G47520	12.1731322	1.21E-10	*HYPOXIA RESPONSIVE ERF (ETHYLENE RESPONSE FACTOR) 2 (HRE2);ETHYLENE RESPONSE FACTOR 71 (ERF71);ARABIDOPSIS THALIANA ETHYLENE RESPONSE FACTOR 71 (AtERF71)*, encodes a member of the ERF (ethylene response factor) subfamily B-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 5 members in this subfamily including RAP2.2 AND RAP2.12. It plays a role in hypoxia-induced root slanting.
AT4G14630	11.2874083	9.00E-10	*GERMIN-LIKE PROTEIN 9 (GLP9),* germin-like protein with N-terminal signal sequence that may target it to the vacuole, plasma membrane and/or outside the cell. The mRNA is cell-to-cell mobile.
AT3G46080	11.2858109	7.93E-24	*ZAT8,* C2H2-type zinc finger family protein.
AT5G51060	11.1721378	7.02E-21	*A. THALIANA RESPIRATORY BURST OXIDASE HOMOLOG C (ATRBOHC)*, RHD2 (along with RHD3 and RHD4) is required for normal root hair elongation. Has NADPH oxidase activity. Gene is expressed in the elongation and differention zone in trichoblasts and elongating root hairs. RDH2 is localized to the growing tips of root hair cells. It is required for the production of reactive oxygen species in response to extracellular ATP stimulus. The increase in ROS production stimulates Ca2+ influx.
AT1G69930	11.0374083	1.63E-09	*GLUTATHIONE S-TRANSFERASE TAU 11 (GSTU11);GLUTATHIONE S-TRANSFERASE TAU 11 (ATGSTU11)*, encodes glutathione transferase belonging to the tau class of GSTs. Naming convention according to Wagner et al. (2002).
AT1G33730	10.9686404	1.71E-08	*CYTOCHROME P450, FAMILY 76, SUBFAMILY C, POLYPEPTIDE 5 (CYP76C5),* cytochrome P450, family 76, subfamily C, polypeptide 5.
AT2G02010	10.9576822	8.47E-37	Glutamate decarboxylase.
AT2G44460	10.9053099	4.59E-10	*BETA GLUCOSIDASE 28 (BGLU28)*, Beta-glucosidase, major myrosinase which initiates sulfur reallocation by hydrolyzing particular GL species, conferring sulfur deficiency tolerance, especially during early development.
AT5G37490	10.8407326	1.52E-09	*PUB21,* Plant U-box type E3 ubiquitin ligase (PUB).
AT3G44830	10.7855864	8.93E-09	Lecithin:cholesterol acyltransferase family protein.
AT1G61120	10.7586702	1.87E-08	*GERANYLLINALOOL SYNTHASE (GES);TERPENE SYNTHASE 04 (TPS04);TERPENE SYNTHASE 4 (TPS4)*, encodes a geranyllinalool synthase that produces a precursor to TMTT, a volatile plant defense C16-homoterpene. GES transcript levels rise in response to alamethicin, a fungal peptide mixture that damages membranes. This transcriptional response is blocked in JA biosynthetic and JA signaling mutants, but GES transcript levels still rise in response to alamethicin in mutants with salicylic acid and ethylene biosynthetic and/or signaling defects. GES transcripts also accumulate in response to a larval infestation. This enzyme does not localize to the plastids, and it may be present in the cytosol or endoplasmic reticulum. The mRNA is cell-to-cell mobile.
AT3G49580	10.6643571	6.35E-31	*RESPONSE TO LOW SULFUR 1 (LSU1)*, RESPONSE TO LOW SULFUR gene family member; expressed during sulfur deficiency.
AT2G30750	10.6574043	4.78E-22	*CYTOCHROME P450, FAMILY 71, SUBFAMILY A, POLYPEPTIDE 12 (CYP71A12),* Putative cytochrome P450; together with CYP71A13 produces dihydrocamalexic acid (DHCA), the precursor to the defense-related compound camalexin, which accumulates in the intercellular space and contributes to the resistance of mature Arabidopsis to P. syringae without directly inhibiting bacterial growth.
AT1G21850	10.6246195	1.60E-06	*SKU5 SIMILAR 8 (sks8)*, SKU5 similar 8.
AT2G39030	10.541212	8.13E-11	*N-ACETYLTRANSFERASE ACTIVITY 1 (NATA1)*, encodes a protein that acts as an ornithine N-delta-acetyltransferase, leading to the formation of N-delta-actetylornithine. This compound is likely used in plant defense and levels of it are increased in Arabidopsis plants in response to MeJA and ABA. The mRNA is cell-to-cell mobile.
AT5G48410	10.5301762	3.75E-09	*ARABIDOPSIS THALIANA GLUTAMATE RECEPTOR 1.3 (ATGLR1.3);GLUTAMATE RECEPTOR 1.3 (GLR1.3)*, member of Putative ligand-gated ion channel subunit family.
AT4G16260	10.456995	2.50E-07	Encodes a putative beta-1,3-endoglucanase that interacts with the 30C02 cyst nematode effector. May play a role in host defense.
AT3G25250	10.377591	4.66E-21	*AGC2 KINASE 1 (AGC2-1); (AGC2);OXIDATIVE SIGNAL-INDUCIBLE1 (OXI1); (AtOXI1)*, Arabidopsis protein kinase The mRNA is cell-to-cell mobile.
AT5G67310	10.3604584	5.32E-08	*CYTOCHROME P450, FAMILY 81, SUBFAMILY G, POLYPEPTIDE 1 (CYP81G1)*, member of CYP81G.
AT4G31950	10.3021252	4.43E-07	*CYTOCHROME P450, FAMILY 82, SUBFAMILY C, POLYPEPTIDE 3 (CYP82C3),* member of CYP82C.

Supplementary Table S30. Top 20 downregulated genes in WT plants treated with a 4 h-PL period (T10) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	T10 *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT1G06080	-11.567936	4.79E-10	*DELTA 9 DESATURASE 1 (ADS1),* encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature.
AT4G29020	-10.011615	1.17E-05	Glycine-rich protein.
AT5G48490	-8.7141474	5.75E-06	*(DEG15);DIR1-LIKE (DIR1-LIKE),* encodes a protein with similarity to a lipid transfer protein that may contribute to systemic acquired resistance (SAR).
AT4G40090	-8.2389269	3.25E-07	*ARABINOGALACTAN PROTEIN 3 (AGP3)*, arabinogalactan protein 3.
AT1G11850	-8.2033833	0.001209	Transmembrane protein.
AT2G42870	-8.1754922	7.21E-09	*PHY RAPIDLY REGULATED 1 (PAR1);HELIX-LOOP-HELIX 1 (HLH1)*, encodes PHYTOCHROME RAPIDLY REGULATED1 (PAR1), an atypical basic helix-loop-helix (bHLP) protein. Closely related to PAR2 (At3g58850). Up regulated after simulated shade perception. Acts in the nucleus to control plant development and as a negative regulator of shade avoidance response. Functions as transcriptional repressor of auxin-responsive genes SAUR15 (AT4G38850) and SAUR68 (AT1G29510).
AT2G14900	-8.156458	7.33E-07	*GASA7,* Gibberellin-regulated family protein.
AT1G52750	-7.9149115	7.60E-05	Alpha/beta-Hydrolases superfamily protein.
AT1G59030	-7.8176943	0.016899	GDSL-motif esterase/acyltransferase/lipase. Enzyme group with broad substrate specificity that may catalyze acyltransfer or hydrolase reactions with lipid and non-lipid substrates.
AT2G34430	-7.7692347	9.44E-10	*LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX II SUBUNIT B1 (LHCB1.4); (DEG11);LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX II SUBUNIT B1 (LHB1B1),* Photosystem II type I chlorophyll, a/b-binding protein The mRNA is cell-to-cell mobile.
AT5G03130	-7.7519095	0.007745	Hypothetical protein.
AT1G11740	-7.730377	0.000443	Ankyrin repeat family protein.
AT5G65390	-7.7197371	2.43E-13	*ARABINOGALACTAN PROTEIN 7 (AGP7),* arabinogalactan protein 7.
AT5G50335	-7.667718	0.000181	Hypothetical protein.
AT2G19660	-7.5945727	0.000536	Cysteine/Histidine-rich C1 domain family protein.
AT5G02760	-7.3227886	7.89E-12	*SENESCENCE-SUPPRESSED 51 PROTEIN PHOSPHATASE (SSPP);ARABIDOPSIS PP2C CLADE D 7 (APD7)*, encodes a phosphatase that functions in sustaining proper leaf longevity and preventing early senescence by suppressing or perturbing SARK-mediated senescence signal transduction.
AT2G20750	-7.1329918	6.31E-08	*EXPANSIN B1 (ATEXPB1);EXPANSIN B1 (EXPB1); (ATHEXP BETA 1.5)*, member of BETA-EXPANSINS.
AT1G72620	-7.0978227	0.004234	Alpha/beta-Hydrolases superfamily protein.
AT1G11070	-7.0889919	0.013318	Hydroxyproline-rich glycoprotein family protein.
AT1G78260	-6.9369857	0.005422	RNA-binding (RRM/RBD/RNP motifs) family protein.

Supplementary Table S31. Top 20 upregulated genes in WT plants treated with a 4 h-PL period as priming and triggering with ten days lag phase in-between (PL10T) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	PL10T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT5G17220	11.8086959	9.81E-10	*ARABIDOPSIS THALIANA GLUTATHIONE S-TRANSFERASE PHI 12 (ATGSTF12);GLUTATHIONE S-TRANSFERASE PHI 12 (GSTF12);TRANSPARENT TESTA 19 (TT19);GLUTATHIONE S-TRANSFERASE 26 (GST26)*, encodes glutathione transferase belonging to the phi class of GSTs. Naming convention according to Wagner et al. (2002). Mutants display no pigments on leaves and stems. Likely to function as a carrier to transport anthocyanin from the cytosol to tonoplasts.
AT1G03940	11.2352931	6.86E-10	HXXXD-type acyl-transferase family protein.
AT2G47520	11.2315601	1.22E-08	*HYPOXIA RESPONSIVE ERF (ETHYLENE RESPONSE FACTOR) 2 (HRE2);ETHYLENE RESPONSE FACTOR 71 (ERF71);ARABIDOPSIS THALIANA ETHYLENE RESPONSE FACTOR 71 (AtERF71)*, encodes a member of the ERF (ethylene response factor) subfamily B-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 5 members in this subfamily including RAP2.2 AND RAP2.12. It plays a role in hypoxia-induced root slanting.
AT4G14630	10.911301	6.02E-09	*GERMIN-LIKE PROTEIN 9 (GLP9)*, germin-like protein with N-terminal signal sequence that may target it to the vacuole, plasma membrane and/or outside the cell. The mRNA is cell-to-cell mobile.
AT2G44460	10.8509519	6.16E-10	*BETA GLUCOSIDASE 28 (BGLU28)*, Beta-glucosidase, major myrosinase which initiates sulfur reallocation by hydrolyzing particular GL species, conferring sulfur deficiency tolerance, especially during early development.
AT3G46080	10.6518002	6.21E-21	*ZAT8,* C2H2-type zinc finger family protein.
AT5G51060	10.5571919	2.34E-18	*A. THALIANA RESPIRATORY BURST OXIDASE HOMOLOG C (ATRBOHC)*, RHD2 (along with RHD3 and RHD4) is required for normal root hair elongation. Has NADPH oxidase activity. Gene is expressed in the elongation and differention zone in trichoblasts and elongating root hairs. RDH2 is localized to the growing tips of root hair cells. It is required for the production of reactive oxygen species in response to extracellular ATP stimulus. The increase in ROS production stimulates Ca2+ influx.

AGI	PL10T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT3G49580	10.5371272	3.96E-30	*RESPONSE TO LOW SULFUR 1 (LSU1)*, RESPONSE TO LOW SULFUR gene family member; expressed during sulfur deficiency.
AT1G33730	10.5321314	1.32E-07	*CYTOCHROME P450, FAMILY 76, SUBFAMILY C, POLYPEPTIDE 5 (CYP76C5),* cytochrome P450, family 76, subfamily C, polypeptide 5.
AT4G15210	10.4718717	1.72E-06	*ARABIDOPSIS THALIANA BETA-AMYLASE (ATBETA-AMY); (AT-BETA-AMY);REDUCED BETA AMYLASE 1 (RAM1); (BMY1);BETA-AMYLASE 5 (BAM5),* cytosolic beta-amylase expressed in rosette leaves and inducible by sugar. RAM1 mutants have reduced beta amylase in leaves and stems.
AT2G39030	10.4694481	1.24E-10	*N-ACETYLTRANSFERASE ACTIVITY 1 (NATA1)*, encodes a protein that acts as an ornithine N-delta-acetyltransferase, leading to the formation of N-delta-actetylornithine. This compound is likely used in plant defense and levels of it are increased in Arabidopsis plants in response to MeJA and ABA. The mRNA is cell-to-cell mobile.
AT3G08860	10.3976288	8.85E-18	*PYRIMIDINE 4 (PYD4)*, encodes a protein that is predicted to have beta-alanine aminotransferase activity.
AT3G44830	10.2817025	1.03E-07	Lecithin:cholesterol acyltransferase family protein.
AT2G02010	10.2195538	1.14E-31	AT2G02010 AT2G02010.2 Glutamate decarboxylase protein_coding
AT4G16590	10.1925809	0.000463	*CELLULOSE SYNTHASE-LIKE A01 (ATCSLA01); (CSLA01);CELLULOSE SYNTHASE-LIKE A1 (ATCSLA1);CELLULOSE SYNTHASE-LIKE A01 (CSLA01)*, encodes a gene similar to cellulose synthase.
AT1G69930	10.1734757	1.18E-07	*GLUTATHIONE S-TRANSFERASE TAU 11 (GSTU11);GLUTATHIONE S-TRANSFERASE TAU 11 (ATGSTU11)*, encodes glutathione transferase belonging to the tau class of GSTs. Naming convention according to Wagner et al. (2002).
AT5G37490	10.1454453	5.26E-08	*PUB21, CMPG5,* Plant U-box type E3 ubiquitin ligase (PUB).
AT1G61120	10.1002126	4.11E-07	*GERANYLLINALOOL SYNTHASE (GES);TERPENE SYNTHASE 04 (TPS04);TERPENE SYNTHASE 4 (TPS4)*, encodes a geranyllinalool synthase that produces a precursor to TMTT, a volatile plant defense C16-homoterpene. GES transcript levels rise in response to alamethicin, a fungal peptide mixture that damages membranes. This transcriptional response is blocked in JA biosynthetic and JA signaling mutants, but GES transcript levels still rise in response to alamethicin in mutants with salicylic acid and ethylene biosynthetic and/or signaling defects. GES transcripts also accumulate in response to a larval infestation. This enzyme does not localize to the plastids, and it may be present in the cytosol or endoplasmic reticulum. The mRNA is cell-to-cell mobile.
AT2G30750	10.0369734	2.98E-19	*CYTOCHROME P450, FAMILY 71, SUBFAMILY A, POLYPEPTIDE 12 (CYP71A12),* Putative cytochrome P450; together with CYP71A13 produces dihydrocamalexic acid (DHCA), the precursor to the defense-related compound camalexin, which accumulates in the intercellular space and contributes to the resistance of mature Arabidopsis to P. syringae without directly inhibiting bacterial growth.
AT1G56650	9.9561696	4.43E-06	*PRODUCTION OF ANTHOCYANIN PIGMENT 1 (PAP1);PHOSPHATIDIC ACID PHOSPHATASE 1 (PAP1);MYELOBLASTOSIS PROTEIN 75 (MYB75);SUC-INDUCED ANTHOCYANIN ACCUMULATION 1 (SIAA1);MYB DOMAIN PROTEIN 75 (ATMYB75);ARABIDOPSIS THALIANA PRODUCTION OF ANTHOCYANIN PIGMENT 1 (ATPAP1); (PAP1-D)*, encodes a putative MYB domain containing transcription factor involved in anthocyanin metabolism and radical scavenging. Essential for the sucrose-mediated expression of the dihydroflavonol reductase gene. Auxin and ethylene responsiveness of PAP1 transcription is lost in myb12 mutants. Interacts with JAZ proteins to regulate anthocyanin accumulation.

Supplementary Table S32. Top 20 downregulated genes in WT plants treated with a 4 h-PL period as priming and triggering with ten days lag phase in-between (PL10T) in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	PL10T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
AT1G06080	-11.661467	2.97E-10	*(ATADS1);DELTA 9 DESATURASE 1 (ADS1)*, encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.
AT2G42870	-10.15761	6.78E-08	*PHY RAPIDLY REGULATED 1 (PAR1);HELIX-LOOP-HELIX 1 (HLH1)*, encodes PHYTOCHROME RAPIDLY REGULATED1 (PAR1), an atypical basic helix-loop-helix (bHLP) protein. Closely related to PAR2 (At3g58850). Up regulated after simulated shade perception. Acts in the nucleus to control plant development and as a negative regulator of shade avoidance response. Functions as transcriptional repressor of auxin-responsive genes SAUR15 (AT4G38850) and SAUR68 (AT1G29510).
AT5G48490	-9.5434347	2.21E-06	*(DEG15);DIR1-LIKE (DIR1-LIKE)*, encodes a protein with similarity to a lipid transfer protein that may contribute to systemic acquired resistance (SAR).
AT2G34430	-8.9305419	9.35E-14	*LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX II SUBUNIT B1 (LHCB1.4); (DEG11);LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX II SUBUNIT B1 (LHB1B1),* Photosystem II type I chlorophyll a/b-binding protein The mRNA is cell-to-cell mobile.
AT4G40090	-8.3324579	1.91E-07	*ARABINOGALACTAN PROTEIN 3 (AGP3)*, arabinogalactan protein 3.
AT2G14900	-8.2499891	4.38E-07	*GASA7,* Gibberellin-regulated family protein.
AT5G65390	-8.2252046	2.46E-12	*ARABINOGALACTAN PROTEIN 7 (AGP7)*, arabinogalactan protein 7.
AT1G72620	-8.1531488	5.03E-05	Alpha/beta-Hydrolases superfamily protein.
AT4G01335	-8.1232247	1.52E-06	TATA box-binding protein associated factor RNA polymerase I subunit B-like protein.
AT5G02760	-8.0413463	3.21E-14	*SENESCENCE-SUPPRESSED 51 PROTEIN PHOSPHATASE (SSPP);ARABIDOPSIS PP2C CLADE D 7 (APD7),* encodes a phosphatase that functions in sustaining proper leaf longevity

AGI	PL10T *vs.* control		Short description derived from TAIR
	log_2 FC	*p*-value adj (Bonf)	
			and preventing early senescence by suppressing or perturbing SARK-mediated senescence signal transduction.
AT1G52750	-8.0044788	4.91E-05	Alpha/beta-Hydrolases superfamily protein.
AT1G11850	-7.7251668	0.000661	Transmembrane protein.
AT4G29020	-7.2425864	0.000821	Glycine-rich protein.
AT5G62280	-6.9847659	3.92E-07	DUF1442 family protein (DUF1442).
AT4G14130	-6.9755636	0.007272	*XYLOGLUCAN ENDOTRANSGLYCOSYLASE 7 (XTR7);XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE 15 (XTH15),* xyloglucan endotransglycosylase-related protein (XTR7).
AT1G11740	-6.9246886	0.000589	Ankyrin repeat family protein.
AT4G28900	-6.8827833	8.80E-09	Transposable element gene.
AT5G45820	-6.8026463	1.44E-07	*CBL-INTERACTING PROTEIN KINASE 20 (CIPK20);PROTEIN KINASE 18 (PKS18);SNF1-RELATED PROTEIN KINASE 3.6 (SnRK3.6),* encodes a CBL-interacting serine/threonine protein kinase comprised of an N-terminal kinase catalytic domain similar to SNF1/AMPK and a unique C-terminal regulatory domain.
AT4G32280	-6.7990057	2.31E-16	*INDOLE-3-ACETIC ACID INDUCIBLE 29 (IAA29),* Indole-3-acetic acid inducible 29 protein involved in IAA signaling. Downstream target of PIF4.
AT2G19660	-6.7263085	0.01686	Cysteine/Histidine-rich C1 domain family protein.

Supplementary Table S33. Top 20 commonly upregulated genes in WT and *npr1* after mild photoperiod stress in comparison to control-WT or control-*npr1*, respectively.

Genes were sorted according to their regulation in *npr1*. Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs. npr1*-control		Short description derived from TAIR
	log_2 FC	*p*-value (Bonf)	log_2 FC	*p*-value (Bonf)	
AT5G02780	7.9909177	0.00030805	11.7149323	4.66E-05	*GLUTATHIONE TRANSFERASE LAMBDA 1* (*GSTL1*), encodes a member of the lambda family of glutathione transferases. It has thiol transferase activity and self S-glutathionylation activity *in vitro*.
AT2G23910	9.814718	0.00015072	11.5930499	2.48E-06	NAD(P)-binding Rossmann-fold superfamily protein
AT1G13480	10.1104543	3.47E-25	11.2688222	1.69E-12	*DUF1262*. hypothetical protein
AT1G19250	6.91201593	0.00246566	11.2194456	2.87E-05	*FLAVIN-DEPENDENT MONOOXYGENASE 1* (*FMO1*), FMO1 is required for full expression of TIR-NB-LRR conditioned resistance to avirulent pathogens and for basal resistance to invasive virulent pathogens. Functions in an EDS1-regulated but SA-independent mechanism that promotes resistance and cell death at pathogen infection sites. FMO1 functions as a pipecolate N-hydroxylase and catalyzes the biochemical conversion of pipecolic acid to N-hydroxypipecolic acid (NHP). NHP systemically accumulates in the plant foliage and induces systemic acquired resistance to pathogen infection.
AT4G22880	8.78845175	4.69E-13	11.1704594	2.99E-17	*LEUCOANTHOCYANIDIN DIOXYGENASE* (*LDOX*), encodes leucoanthocyanidin dioxygenase. which is involved in proanthocyanin biosynthesis. Mutant analysis suggests that this gene is also involved in vacuole formation.
AT1G08860	7.56442378	2.02E-05	11.1033614	1.07E-05	*BONZAI 3* (*BON3*), encodes a copine-like protein. which is a member of a newly identified class of calcium-dependent. phospholipid binding proteins that are present in a wide range of organisms. Overexpression of this gene suppresses bon1-1 phenotypes. Double mutant analyses with bon1-1 suggest that BON1 and BON3 have overlapping functions in maintaining cellular homeostasis and inhibiting cell death.
AT5G62480	8.56181591	8.54E-12	10.9619416	6.20E-07	*GLUTATHIONE S-TRANSFERASE TAU 9* (*GSTU9*), encodes glutathione transferase belonging to the tau class of GSTs.
AT5G17220	9.3402575	1.77E-23	10.6309986	3.42E-30	*GLUTATHIONE S-TRANSFERASE PHI 12* (*GSTF12*), encodes glutathione transferase belonging to the phi class of GSTs. Naming convention according to Wagner et al. (2002). Mutants display no pigments on leaves and stems. Likely to function as a carrier to transport anthocyanin from the cytosol to tonoplasts.
AT4G09820	8.16537152	0.00021341	10.4301313	3.26E-08	*TRANSPARENT TESTA 8* (*TT8*), TT8 is a regulation factor that acts in a concerted action with TT1. PAP1 and TTG1 on the regulation of flavonoid pathways. namely proanthocyanidin and anthocyanin biosynthesis. Affects dihydroflavonol 4-reductase gene expression. It is thought that a ternary complex composed of TT2. TT8 and TTG1 is necessary for correct expression of BAN in seed endothelium. Also important for important for marginal trichome development. It binds the promoter of both AT3G26790 and AT1G28300.TT8 interacts with JAZ proteins to regulate anthocyanin accumulation. TT8 acts maternally to affect seed FA biosynthesis and inhibits seed FA accumulation by down-regulating a group of genes either critical to embryonic development or important in the FA biosynthesis pathway. TT8 represses the activities of LEAFY COTYLEDON1. LEAFY COTYLEDON2. and FUSCA3. the critical transcriptional factors important for seed development.

AGI	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs. npr1*-control		Short description derived from TAIR
	log_2 FC	*p*-value (Bonf)	log_2 FC	*p*-value (Bonf)	
AT1G28480	7.99802447	1.34E-26	10.3826494	3.52E-10	*GRX480*, encodes a member of the glutaredoxin family that regulates protein redox state. GRX480 interacts with TGA factors and suppresses JA-responsive *PDF1.2* transcription. *GRX480* transcription is SA-inducible and requires NPR1. Maybe involved in SA/JA cross-talk. It has also been shown to interact with the transcription factor TGA2 and suppress ORA59 promoter activity.
AT5G11210	6.75170565	0.02454033	10.2567471	0.001655779	Member of Putative ligand-gated ion channel subunit family
AT5G59490	9.76724255	2.34E-19	10.1914271	7.03E-11	Haloacid dehalogenase-like hydrolase (HAD) superfamily protein
AT1G05880	6.84194051	0.00036881	9.97742849	3.97E-05	*ARIADNE 12* (*ARI12*), ARI12 belongs to a family of `RING between RING fingers' (RBR) domain proteins with E3 ligase activity. Expression of *ARI12* is induced by UV-B exposure.
AT2G02010	9.11854555	2.13E-06	9.87586282	4.99E-05	Glutamate decarboxylase
AT3G02840	7.81887396	7.44E-40	9.82248437	2.36E-22	ARM repeat superfamily protein
AT4G39670	7.32699037	2.15E-08	9.77538988	1.86E-11	*PHOSPHOLIPASE-LIKE PROTEIN* (*GLTP*), member of the glycolipid transfer protein (GLTP) superfamily. shuttles ceramide-1-phosphate (C1P) between membranes.
AT1G44130	6.76699364	0.00032395	9.715426	0.000665304	Eukaryotic aspartyl protease family protein
AT3G28210	7.14648393	1.52E-06	9.64311872	5.89E-09	*STRESS-ASSOCIATED PROTEIN 12* (*SAP12*), encodes a putative zinc finger protein (PMZ).
AT3G25250	9.056848	3.19E-07	9.61507908	4.15E-05	*OXIDATIVE SIGNAL-INDUCIBLE1* (*OXI1*), *Arabidopsis* protein kinase. The mRNA is cell-to-cell mobile.
AT1G26380	6.16263598	0.01271564	9.46076609	2.50E-06	*FAD-LINKED OXIDOREDUCTASE 1* (*FOX1*), functions in the biosynthesis of 4-hydroxy indole-3-carbonyl nitrile (4-OH-ICN). a cyanogenic phytoalexin in *Arabidopsis*. FOX1 acts as a dehydrogenase on indole cyanohydrin to form indole carbonyl nitrile.

Supplementary Table S34. Top 20 commonly downregulated genes in WT and *npr1* after mild photoperiod stress in comparison to WT-control or control-*npr1*, respectively.

Genes were sorted according to their regulation in *npr1*. Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs. npr1*-control		Short description derived from TAIR
	log_2 FC	*p*-value (Bonf)	log_2 FC	*p*-value (Bonf)	
AT5G64170	-5.70414368	1.71E-46	-6.83501848	6.97E-44	*NIGHT LIGHT-INDUCIBLE AND CLOCK-REGULATED GENE 1* (*LNK1*). LNK1 is a member of a small family (4 proteins) in *Arabidopsis* that have some overlap in function. LNK1 functions in the integration of light signaling and circadian clock. It is regulated by the clock TOC1 complex. Functions as a transcriptional coactivator.
AT3G54510	-9.35658448	2.88E-10	-7.17291759	0.019431087	*ERD4*, early-responsive to dehydration stress protein
AT5G35510	-7.33155003	8.80E-05	-7.19907481	0.039885856	TIR-NBS-LRR class disease resistance protein
AT5G54585	-11.6476734	5.64E-18	-7.20472996	2.62E-55	Hypothetical protein
AT3G08405	-7.56098396	5.63E-05	-7.37424189	0.032720523	Natural antisense transcript overlaps with AT3G54500, antisense long-noncoding RNA.
AT2G43010	-6.92550055	1.81E-36	-7.38722846	3.10E-27	Isolated as a semidominant mutation defective in red - light responses. Encodes a nuclear localized bHLH protein that interacts with active PhyB protein. Negatively regulates phyB mediated red light responses. Involved in shade avoidance response. Protein abundance is negatively regulated by PhyB. Involved in the regulation of response to nutriant levels. Controls the resistance to *B. cinerea* in a COI1- and EIN2-dependent manner.
AT1G29490	-6.90494838	0.002788258	-7.44753836	0.044398242	*SMALL AUXIN UPREGULATED 68* (*SAUR68*), SAUR-like auxin-responsive protein family.
AT2G34430	-7.43112002	9.54E-31	-7.48245536	1.56E-24	*LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX II SUBUNIT B1* (*LHCB1.4*), photosystem II type I chlorophyll a/b-binding protein. The mRNA is cell-to-cell mobile.
AT5G49360	-7.18250689	3.15E-48	-7.53483048	3.56E-40	*BETA-XYLOSIDASE 1* (*BXL1*), encodes a bifunctional β-D-xylosidase/α-L-arabinofuranosidase required for pectic arabinan modification. Located in the extracellular matrix. Gene is expressed specifically in tissues undergoing secondary wall thickening. This is a member of glycosyl hydrolase family 3 and has six other closely related members.
AT4G21745	-6.18533214	0.024642987	-7.6961795	0.00952392	PAK-box/P21-Rho-binding family protein
AT3G47340	-7.56623947	0.000121182	-7.78038311	0.01350058	*GLUTAMINE-DEPENDENT ASPARAGINE SYNTHASE 1* (*ASN1*), encodes a glutamine-dependent asparagine synthetase. the predicted ASN1 peptide contains a purF-type glutamine-binding

AGI	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs. npr1*-control		Short description derived from TAIR
	log_2 FC	*p*-value (Bonf)	log_2 FC	*p*-value (Bonf)	
					domain. and is expressed predominantly in shoot tissues. where light has a negative effect on its mRNA accumulation. Expression is induced within 3 hours of dark treatment. in senescing leaves and treatment with exogenous photosynthesis inhibitor. Induction of gene expression was suppressed in excised leaves supplied with sugar. The authors suggest that the gene's expression pattern is responding to the level of sugar in the cell.
AT5G36910	-7.43918264	0.000267642	-7.79914395	0.014432908	*THIONIN 2.2* (*THI2.2*), encodes a thionin that is expressed at a low basal level in seedlings and shows circadian variation. Predicted to encode a PR (pathogenesis-related) protein. Belongs to the plant thionin (PR-13) family with the following members: At1g66100, At5g36910, At1g72260, At2g15010, At1g12663, At1g12660.
AT5G16023	-7.92848014	1.44848E-05	-7.85469048	0.008431762	*ROTUNDIFOLIA LIKE 18* (*RTFL18*), encodes a plant peptide that could be involved in the coordination of socket cell development in wild-type plants.
AT5G62280	-9.55247277	2.41506E-10	-7.99577279	2.83848E-13	DUF1442 family protein
AT5G44260	-9.65947417	1.24902E-10	-8.12117163	0.000953346	*TANDEM CCCH ZINC FINGER PROTEIN 5* (*TZF5*), encodes a Tandem CCCH Zinc Finger protein. Interacts and co-localizes with MARD1 and RD21A in processing bodies (PBs) and stress granules (SGs).
AT4G32280	-8.50351585	1.15093E-15	-8.12814784	1.98929E-21	*INDOLE-3-ACETIC ACID INDUCIBLE 29* (*IAA29*)
AT5G06980	-8.43932369	8.88E-50	-9.19070698	4.70E-41	*NIGHT LIGHT-INDUCIBLE AND CLOCK-REGULATED GENE 4* (*LNK4*), member of a small gene family. Appears to be clock regulated. Somewhat redundant with LNK1/2 though more like LNK3 in having effects on biomass accumulation and phototrophism.
AT1G06080	-8.87297947	5.58E-06	-9.50157402	0.000285044	*DELTA 9 DESATURASE 1* (*ADS1*), encodes a protein homologous to delta 9 acyl-lipid desaturases of cyanobacteria and acyl-CoA desaturases of yeast and mammals. expression down-regulated by cold temperature. It is involved in the desaturation of VLCFAs to make monounsaturated VLCFAs.
AT3G55240	-11.7755326	2.16E-17	-11.4104532	4.72E-10	Overexpression leads to PEL (Pseudo-Etiolation in Light) phenotype.
AT5G45820	-9.90613038	1.08E-26	-12.2941597	6.20E-13	*CBL-INTERACTING PROTEIN KINASE 20* (*CIPK20*), encodes a CBL-interacting serine/threonine protein kinase comprised of an N-terminal kinase catalytic domain similar to SNF1/AMPK and a unique C-terminal regulatory domain.

Supplementary Table S35. Top 20 upregulated genes in WT plants after mild photoperiod stress in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	WT-8 h-PL *vs.* WT-control		Short description derived from TAIR
	log_2 FC	*p*-value (Bonf)	
AT1G64940	10.70232	1.61E-12	*CYTOCHROME P450, FAMILY 87, SUBFAMILY A, POLYPEPTIDE 6* (*CYP89A6*)
AT1G06135	10.1951	0.021876824	*SMALL PHYTOCYTOKINES REGULATING DEFENSE AND WATER LOSS 1* (*SCREW1*), transmembrane protein
AT4G18430	10.19448	1.62E-09	*RAB GTPASE HOMOLOG A1E* (*RABA1e*)
AT2G21900	9.79704	4.16E-10	*WRKY DNA-BINDING PROTEIN 59* (*WRKY59*), member of WRKY transcription factor (group II-c)
AT2G40340	9.299325	4.81E-06	Encodes a member of the DREB subfamily A-2 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are eight members in this subfamily including DREB2A AND DREB2B that are involved in response to drought.
AT4G34410	9.216652	3.95E-05	*REDOX RESPONSIVE TRANSCRIPTION FACTOR 1* (*RRTF1*), encodes a member of the ERF (ethylene response factor) subfamily B-3 of ERF/AP2 transcription factor family. The protein contains one AP2 domain. There are 18 members in this subfamily including ATERF-1, ATERF-2 AND ATERF-5. Regulates programmed cell death (PCD) inhibitor genes.
AT3G48450	9.131756	5.01E-06	RPM1-interacting protein 4 (RIN4) family protein
AT5G36907	9.006842	0.000590859	Transmembrane protein
AT1G68250	8.951148	0.000104663	Hypothetical protein
AT4G33070	8.933902	5.35E-11	*PYRUVATE DECARBOXYLASE 1* (*PDC1*), encodes hypoxia and drought induced pyruvate decarboxylase. which catalyzes the first step in ethanolic fermentation.
AT2G07798	8.88788	1.55E-05	Hypothetical protein
AT3G54520	8.758447	6.20E-06	Hypothetical protein
AT5G40010	8.655084	0.005173941	*AAA-ATPASE 1* (*AATP1*), encodes a mitochondrial ATPase involved in seed and silique development
AT5G47740	8.649704	1.61E-06	Adenine nucleotide alpha hydrolases-like superfamily protein
AT1G17615	8.626973	7.01E-05	*IR-NBS2* (*TN2*), TN2 is an atypical TIR-NBS protein that lacks the LRR domain common in typical NLR receptors. It interacts with EXO70B1. a subunit of the exocyst complex.
AT3G28510	8.579268	6.56E-09	P-loop containing nucleoside triphosphate hydrolases superfamily protein
AT1G78390	8.575326	0.000970615	*NINE-CIS-EPOXYCAROTENOID DIOXYGENASE 9* (*NCED9*), encodes 9-cis-epoxycarotenoid dioxygenase. a key enzyme in the biosynthesis of abscisic acid.

AGI	WT-8 h-PL *vs.* WT-control		Short description derived from TAIR
	log_2 FC	*p*-value (Bonf)	
AT2G02250	8.571363	0.000314964	*PHLOEM PROTEIN 2-B2* (*PP2-B2*)
AT2G07835	8.445763	0.028468547	Hypothetical protein
AT5G40000	8.378344	1.76E-05	P-loop containing nucleoside triphosphate hydrolases superfamily protein.

Supplementary Table S36. Top 20 downregulated genes in WT plants after mild photoperiod stress in comparison to WT-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	WT-8 h-PL *vs.* WT-control		Short description derived from TAIR
	log_2 FC	*p*-value (Bonf)	
AT4G36105	-5.72592	0.000686768	Polyamine-modulated factor 1-binding protein
AT5G18020	-5.76839	9.73E-12	*SMALL AUXIN UP RNA 20* (*SAUR20*), SAUR-like auxin-responsive protein family.
AT3G24518	-5.79636	0.045134867	Natural antisense transcript overlaps with AT3G24520, antisense long non-coding RNA
AT5G50335	-5.80982	5.28E-10	Hypothetical protein
AT5G66590	-5.9313	2.63E-12	CAP (Cysteine-rich secretory proteins, Antigen 5, and Pathogenesis-related 1 protein) superfamily protein.
AT5G02515	-5.94592	1.22E-15	Natural antisense transcript overlaps with AT5G16030, antisense long non-coding RNA.
AT1G52750	-5.97246	0.002381528	Alpha/beta-hydrolases superfamily protein
AT1G29910	-6.06877	6.73E-06	*CHLOROPHYLL A/B BINDING PROTEIN 3* (*CAB3*), member of Chlorophyll a/b-binding protein family.
AT5G65730	-6.37783	3.27E-16	*XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE 6* (*XTH6*), xyloglucan endotransglucosylase/hydrolase 6.
AT4G11780	-6.41111	0.03881389	*TON1 RECRUITING MOTIF 10* (*TRM10*), GAR2-like protein
AT2G46790	-6.45007	0.045257671	*PSEUDO-RESPONSE REGULATOR 9* (*PRR9*), PRR7 and PRR9 are partially redundant essential components of a temperature-sensitive circadian system. CCA1 and LHY had a positive effect on PRR9. Interact with TOC1 in a yeast two-hybrid assay. Acts as transcriptional repressor of CCA1 and LHY. Acts additively with EC. PRR5 and PRR7 to regulate hypocotyl growth under photoperiodic conditions.
AT1G19540	-6.45046	0.0485963	NmrA-like negative transcriptional regulator family protein
AT1G15830	-6.74024	0.000179692	Hypothetical protein
AT1G04223	-6.7465	0.006246362	Small nucleolar RNA
AT5G40790	-6.87638	0.0027507	*AITR3,* induced transcription repressor that acts as feedback regulator in ABA signalling.
AT5G02155	-6.88751	0.006718319	Natural antisense transcript overlaps with AT5G14120.
AT5G45830	-6.91835	0.004462274	*DELAY OF GERMINATION 1* (*DOG1*), a quantitative trait locus involved in the control of seed dormancy. DOG1 expression is seed-specific.
AT1G62510	-7.18141	0.000568351	Expressed in the root cortex.
AT4G03725	-7.25461	0.000548092	Natural antisense transcript overlaps with AT4G00750, antisense long non-coding RNA.
AT4G40090	-7.42928	0.001190543	*ARABINOGALACTAN PROTEIN 3* (*AGP3*), arabinogalactan protein 3

Supplementary Table S37. Top 20 upregulated genes in *npr1* mutants after mild photoperiod stress in comparison to *npr1*-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	*npr1*-8 h-PL *vs. npr1*-control		Short description derived from TAIR
	log_2 FC	*p*-value (Bonf)	
AT3G46080	11.05068239	0.029750123	C2H2-type zinc finger family protein
AT3G13610	10.83475207	0.014278981	*F6'H1,* encodes a Fe(II)- and 2-oxoglutarate-dependent dioxygenase family gene F6'H1. Mutations in this gene compromise iron uptake and the production of fluorescent phenolics involved in Fe uptake.
AT1G33960	6.547326425	0.011047792	*AVRRPT2-INDUCED GENE 1* (*AIG1*), identified as a gene that is induced by avirulence gene *avrRpt2* and *RPS2* after infection with *Pseudomonas syringae* pv. *maculicola* strain ES4326 carrying *avrRpt2.*
AT1G07050	6.46331575	0.000609596	*FITNESS* (*FITNESS*). *FITNESS* encodes a protein with a single CCT domain and belongs to the CCT motif family genes (CMF). FITNESS acts upstream JUB1 thereby controlling H2O2 levels. FITNESS has a role in cellular redox homeostasis controlling H2O2 levels. due to changes in enzymes. metabolites and transcripts related to ROS detoxification.
AT5G13830	6.171918357	0.004531719	*TRNA METHYLTRANSFERASE 7C* (*TRM7C*). FtsJ-like methyltransferase family protein.
AT3G04300	5.455618566	0.001704553	RmlC-like cupins superfamily protein
AT1G78930	4.990587404	1.11E-09	Mitochondrial transcription termination factor family protein (*MTERF16*)
AT5G62210	4.853139697	0.000135141	Embryo-specific protein 3 (*ATS3*)
AT3G15357	4.687416478	0.010405657	Phosphopantothenoylcysteine decarboxylase subunit
AT3G23550	4.640262276	0.008127351	*DETOXIFICATION 18* (*DTX18*). MATE efflux family protein
AT2G40010	4.40034703	6.65E-12	Ribosomal protein L10 family protein
AT3G20440	4.34253517	2.70E-34	*EMBRYO DEFECTIVE 2729* (*EMB2729*). encodes BE1. a putative glycoside hydrolase. Involved in organogenesis and somatic embryogenesis by regulating carbohydrate metabolism. Mutation in *BE1* has pleotrophic effect on the whole plant development.
AT1G03940	4.305465006	1.65E-07	HXXXD-type acyl-transferase family protein
AT2G37260	4.295091934	4.91E-05	*TRANSPARENT TESTA GLABRA 2* (*TTG2*). encodes a protein similar to WRKY transcription factors that is expressed in the seed integument and endosperm.
AT1G48570	4.243007074	5.38E-34	Zinc finger (Ran-binding) family protein
AT2G37770	4.161961764	0.041983689	*ALDO-KETO REDUCTASE FAMILY 4 MEMBER C9* (*AKR4C9*). encodes an NADPH-dependent aldo-keto reductase that can act on a wide variety of substrates *in vitro* including

AGI	*npr1*-8 h-PL *vs.* *npr1*-control		Short description derived from TAIR
	log_2 FC	*p*-value (Bonf)	
			saturated and unsaturated aldehydes. steroids. and sugars. GFP-tagged AKR4C9 localizes to the chloroplast where it may play a role in detoxifying reactive carbonyl compounds that threaten to impair the photosynthetic process. Transcript levels for this gene are upregulated in response to cold. salt. and drought stress.
AT5G14580	4.051956481	5.60E-11	Polyribonucleotide nucleotidyltransferase
AT1G51380	3.906557359	2.98E-08	DEA(D/H)-box RNA helicase family protein
AT1G10522	3.752174463	2.28E-30	*PLASTID REDOX INSENSITIVE 2* (*PRIN2*). *PRIN2* mutants are impaired in PEP (plastid-encoded RNA polymerase) activity and high light-dependent plastid redox signalling to the nucleus.
AT1G52770	3.74294807	0.00312723	Phototropic-responsive NPH3 family protein

Supplementary Table S38. Top 20 downregulated genes in *npr1* mutants after mild photoperiod stress in comparison to *npr1*-control.

Filtered for padj ≤ 0.05. AGI, Arabidopsis Genome Initiative locus identifier; FC, fold change.

AGI	*npr1*-8 h-PL *vs.* *npr1*-control		Short description derived from TAIR
	log_2 FC	*p*-value (Bonf)	
AT2G17880	-1.925030067	0.000103146	*DNA J PROTEIN C24* (*DJC24*), chaperone DnaJ-domain superfamily protein
AT4G15780	-1.954154621	1.19E-13	*VESICLE-ASSOCIATED MEMBRANE PROTEIN 724* (*VAMP724*), member of VAMP72 Gene Family; TGN/PM localized
AT4G17730	-1.966946408	1.71E-10	*SYNTAXIN OF PLANTS 23* (*SYP23*), member of SYP2 Gene Family. Together with SYP23 interacts with Tobacco mosaic virus 126 kDa protein; required for normal local virus accumulation and spread.
AT5G65207	-1.995720268	0005111486	Hypothetical protein
AT1G11175	-1.999916569	0.006340715	Other RNA
AT2G14878	-2.028004776	1.29E-15	Other RNA
AT4G20070	-2.080892583	2.29E-06	*ALLANTOATE AMIDOHYDROLASE* (*AAH*), the gene encoding Arabidopsis thaliana Allantoate Amidohydrolase (AtAAH). which catalyzes the allantoate deiminase reaction (EC 3.5.3.9) is expressed in all parts of the plant being consistent with a function in purine turnover in Arabidopsis.
AT3G58490	-2.11778376	1.93E-05	*SPHINGOID PHOSPHATE PHOSPHATASE 1* (*SPP1*), encodes a long-chain base 1-phosphate (LCBP) phosphatase that is expressed in the endoplasmic reticulum.
AT4G31290	-2.261343021	0.000869874	*GAMMA-GLUTAMYL CYCLOTRANSFERASE 2;2* (*GGCT2;2*). ChaC-like family protein
AT5G64180	-2.320376211	5.07E-14	Tropomyosin
AT3G13520	-2.366507894	0.007907345	*ARABINOGALACTAN PROTEIN 12* (*AGP12*), encodes a GPI-anchored arabinogalactan (AG) peptide with a short 'classical' backbone of 10 amino acids. seven of which are conserved among the 4 other *Arabidopsis* AG peptides. These peptides may be involved in cell signaling.
AT1G49500	-2.398273697	0.004656558	Transcription initiation factor TFIID subunit 1b-like protein
AT3G47420	-2.69738153	5.24E-11	*GLYCEROL-3-PHOSPHATE PERMEASE 1* (*G3Pp1*), encodes a Pi starvation-responsive protein AtPS3
AT5G49700	-2.72567169	0.003404156	*AT-HOOK MOTIF NUCLEAR LOCALIZED PROTEIN 17* (*AHL17*), putative AT-hook DNA-binding family protein
AT1G80440	-2.727658812	4.76E-06	*KISS ME DEADLY 1* (*KMD1*). *KELCH REPEAT F-BOX 20* (*KFB20*). encodes a member of a family of F-box proteins. called the KISS ME DEADLY (KMD) family. that targets type-B ARR proteins for degradation and is involved in the negative regulation of the cytokinin response. Also named as KFB20. a member of a group of Kelch repeat F-box proteins that negatively regulate phenylpropanoid biosynthesis by targeting the phenypropanoid biosynthesis enzyme phenylalanine ammonia-lyase.
AT1G73480	-3.099764547	7.32E-17	Alpha/beta-hydrolases superfamily protein (*MAGL4*)
AT4G36850	-3.411810907	0.015399202	PQ-loop repeat family protein / transmembrane family protein
AT5G42900	-3.590706341	0.011913459	*COLD REGULATED GENE 27* (*COR27*), acts with COR28 as a key regulator in the COP1-HY5 regulatory hub by regulating HY5 activity to ensure proper skotomorphogenic growth in the dark and photomorphogenic development in the light.
AT2G38530	-3.644205367	0.013639853	*LIPID TRANSFER PROTEIN 2* (*LTP2*), involved in lipid transfer between membranes and plays a role in maintaining the integrity of the cuticle-cell wall interface. Belongs to a family of Lipid transfer proteins. Sequence similarity to other plant/*Arabidopsis* LPT genes but highest similarity to LPT1. Stress and pathogen-inducible motifs found in the upstream region. Expressed in flower. leaves and siliques but absent in roots.
AT3G05890	-4.494469748	0.002406812	*RARE-COLD-INDUCIBLE 2B* (*RCI2B*), low temperature and salt responsive protein family.

Supplementary Table S39. Regulation of transcript abundance of SA-related genes in response to PL periods of 8 h in WT and *npr1.*

Genes in WT plants and *npr1* mutants that were regulated (according to Bonferroni correction) in response to an 8 h-PL periods in comparison to control. Cells remained empty when expression levels of respective genes were not detected. Blue or yellow backgrounds of cells indicate downregulation or upregulation, respectively. FC, fold change; padj, adjusted *p*-value according to Bonferroni correction.

AGI	gene id	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs.* *npr1*-control		*npr1*-8 h-PL *vs.* WT-8 h-PL		*npr1*-control *vs.* WT-control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT1G74710	*ICS1*	5.58635158	3.0095E-40	5.15126558	1.3346E-26	-0.6418102	1	-0.2067242	1
AT1G18870	*ICS3*	-1.085391	1	-1.1606426	1	0.60808803	1	0.68333957	1

AGI	gene id	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs.* *npr1*-control		*npr1*-8 h-PL *vs.* WT-8 h-PL		*npr1*-control *vs.* WT-control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT5G13320	*PBS*	6.42936631	3.9556E-34	6.2131399	8.9865E-25	-0.7361141	1	-0.5198877	1
AT5G67160	*EPS1*	-1.7874371	7.8975E-05	-0.3720904	1	1.39694146	0.51827424	-0.0184052	1
AT4G39030	*EDS5*	6.92966969	1.1379E-41	6.41546694	3.7543E-27	-1.660854	1	-1.1466513	1
AT3G29200	*AtCM1*	-0.9044162	0.74659998	0.24805166	1	1.43111951	6.5743E-05	0.27865167	1
AT5G10870	*AtCM2*	1.09322486	0.13759642	0.35106404	1	-0.5096734	1	0.23248743	1
AT1G69370	*AtCM3*	-0.9236687	1	-0.1959811	1	0.81408517	1	0.08639757	1
AT2G37040	*PAL1*	2.09818073	2.1813E-17	2.18908617	7.2234E-15	0.28466543	1	0.19375999	1
AT3G53260	*PAL2*	0.08315166	1	0.23591145	1	-0.059421	1	-0.2121808	1
AT5G04230	*PAL3*	-2.2717504	4.7172E-06	-1.566512	1	0.7595422	1	0.05430384	1
AT3G10340	*PAL4*	-2.5450889	0.02929402	-1.6770642	1	1.27262631	1	0.40460162	1
AT4G29010	*AIM1*	-0.2557203	1	-0.2222627	1	-0.0106304	1	-0.044088	1
AT3G06860	*MFP2*	1.07340309	1.6788E-07	0.63651335	1	-0.4476105	1	-0.0107208	1
AT1G73805	*SARD1*	3.7957806	7.9839E-28	3.50723747	3.3561E-18	-0.5411428	1	-0.2525996	1
AT5G26920	*CBP60g*	3.97467603	4.5837E-37	4.14482235	1.1326E-31	-0.4988578	1	-0.6690041	1
AT1G58100	*TCP8*	0.19849372	1	-0.1069713	1	-0.2645796	1	0.04088539	1
AT2G45680	*TCP9*								
AT4G35580	*NTL9*	0.39196923	1	-0.078127	1	-0.5723623	1	-0.1022661	1
AT5G08330	*CHE*	-3.7016284	1.2037E-18	-1.7329762	0.49281972	1.93769283	0.06550945	-0.0309593	1
AT4G18170	*WRKY28*	3.94136828	2.4896E-05	1.99313165	1	-3.1411063	0.20994597	-1.1928696	1
AT2G46400	*WRKY46*	6.44654311	1.2905E-54	5.6156186	1.1305E-31	-1.4506637	1	-0.6197392	1
AT5G46350	*WRKY8*	4.09538572	2.0785E-08	1.60015937	1	-2.6240338	0.57792206	-0.1288075	1
AT5G49520	*WRKY48*	5.79820382	1.1494E-23	5.138757	2.2064E-13	-1.5799972	1	-0.9205504	1
AT3G20770	*EIN3*	0.33025177	1	-0.50727	1	-1.0705678	0.02941374	-0.2330461	1
AT2G27050	*EIL1*	-2.4827459	2.3005E-13	-1.2970138	1	1.14416165	1	-0.0415705	1
AT1G52890	*ANAC0198*	5.81470752	0.00093936	5.10755258	0.66709988	-3.2494091	1	-2.5422541	1
AT3G15500	*ANAC055*	1.08515162	1	2.21136498	1	-3.3808155	1	-4.5070289	1
AT4G27410	*ANAC072*	1.96524361	0.00048521	0.96829858	1	-1.6401135	0.52339113	-0.6431685	1
AT5G65210	*TGA1*	0.6599633	1	0.00216478	1	-1.1025587	0.08922907	-0.4447602	1
AT5G10030	*TGA4*	0.86414516	1	-0.1307073	1	-1.1754062	0.10963026	-0.1805538	1
AT1G33240	*GTL1*	-3.8857472	8.3756E-12	-1.779898	1	2.06809315	1	-0.037756	1
AT5G09410	*CAMTA1*	0.36643847	1	-0.5963275	1	-1.0960957	0.01656675	-0.1333298	1
AT5G64220	*CAMTA2*	0.95307442	0.67835751	-0.1279639	1	-1.1660987	0.09949594	-0.0850604	1
AT2G22300	*CAMTA3*	2.22965446	1.5009E-08	0.97426188	1	-1.4735971	0.37741415	-0.2182045	1
AT3G56400	*WRKY70*	3.76373552	5.3844E-32	1.12300765	1	-2.7263104	5.2229E-12	-0.0855825	1
AT3G09830	*PCRK1*	3.00765831	4.6633E-29	2.74874798	2.4702E-18	-0.9279169	1	-0.6690066	1
AT5G03320	*PCRK2*	1.77268212	5.0154E-12	0.78642367	1	-1.0825705	0.11099665	-0.0963121	1
AT3G48090	*EDS1*	3.07046986	2.4431E-16	2.99463732	8.3019E-12	-0.316694	1	-0.2408615	1
AT3G52430	*PAD4*	3.51193535	1.0077E-18	2.63527656	6.4767E-07	-1.2896187	1	-0.41296	1
AT1G33560	*ADR1*	1.20964819	0.00738702	1.18664486	0.15799546	-0.324425	1	-0.3014216	1
AT4G33300	*ADR1-L1*	2.19703331	8.3373E-15	1.57095324	0.00021995	-1.0413149	1	-0.4152349	1
AT4G04720	*ADR1-L2*	0.00126257	1	-0.0684092	1	-0.1176765	1	-0.0480047	1
AT5G40770	*PHB3*	2.37598521	9.3728E-24	2.46754387	2.9299E-20	-0.0678471	1	-0.1594058	1
AT1G64280	*NPR1*	1.75416173	1.7975E-11	0.88673886	1	-1.2534816	0.00428138	-0.3860587	1
AT5G06950	*TGA2*	-1.3794314	0.01950836	-0.6402206	1	0.6830475	1	-0.0561633	1
AT5G06960	*TGA5*	0.5683175	1	-0.0961934	1	-0.7281916	0.2366474	-0.0636807	1
AT3G12250	*TGA6*	0.55442249	1	0.10017771	1	-0.5127821	1	-0.0585373	1
AT1G02450	*MININ1*	7.75976748	8.3691E-15	3.82432462	1	-4.2128648	0.00017846	-0.277422	1
AT3G25882	*MININ2*	6.70416263	7.9517E-29	3.3525251	0.00221075	-3.7620066	2.6779E-06	-0.4103691	1
AT1G09415	*MININ3*	-1.3135591	1	-0.4616034	1	0.86453508	1	0.0125794	1
AT5G45110	*NPR3*	3.50438123	6.2373E-33	1.59508398	0.00407156	-1.9723954	1.8662E-06	-0.0630981	1
AT4G19660	*NPR4*	1.89097387	8.095E-17	0.77613432	1	-1.2113021	0.00093352	-0.0964625	1
AT2G13810	*ALD1*	7.09565325	1.6085E-06	8.50717532	4.7004E-06	-1.7326978	1	-3.1442199	1
AT5G52810	*SARD4*	3.95519743	5.6435E-23	3.3423652	6.2546E-12	-1.1526454	1	-0.5398132	1
AT1G19250	*FMO1*	6.91201593	0.00246566	11.2194456	2.8729E-05	-1.6292232	1	-5.9366529	1

Supplementary Table S40. Regulation of transcript abundance of JA-related genes in response to PL periods of 8 h in WT and *npr1*.

Genes in WT plants and *npr1* mutants that were regulated (according to Bonferroni correction) in response to an 8 h-PL periods in comparison to control. Cells remained empty when expression levels of respective genes were not detected. Blue or yellow backgrounds of cells indicate downregulation or upregulation, respectively. FC, fold change; padj, adjusted *p*-value according to Bonferroni correction.

AGI	gene id	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs.* *npr1*-control		*npr1*-8 h-PL *vs.* WT-8 h-PL		*npr1*-control *vs.* WT-control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT1G05800	*DGL*								
AT2G44810	*DAD*	4.56958743		0		-4.7954725		0	
AT2G29980	*FAD3*	-1.5538289	6.6848E-65	-2.066624189	1.3009E-92	-0.3812618	1	0.13153348	1
AT3G11170	*FAD7*	-0.2534347	1	-0.301241021	1	-0.2728746	1	-0.2250683	1
AT5G05580	*FAD8*	0.02209386	1	0.926075276	0.05053001	1.09000563	0.00059589	0.18602421	1
AT3G45140	*LOX2*	0.67524177	1	1.941127703	0.6278613	0.52681233	1	-0.7390736	1
AT1G17420	*LOX3*	4.75311649	4.5919E-13	3.081846617	0.01737147	-2.0723143	1	-0.4010444	1
	LOX4								
AT5G42650	*AOS*	1.54032728	4.4274E-07	1.192856285	0.06940822	-0.897572	1	-0.5501011	1
AT3G25770	*AOC2*	4.08839494	2.5671E-25	4.503049117	4.479E-24	-1.2038917	1	-1.6185459	0.19831635
	AOC3								

AGI	gene id	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs.* *npr1*-control		*npr1*-8 h-PL *vs.* WT-8 h-PL		*npr1*-control *vs.* WT-control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT2G06050	*OPR3*	3.9546702	8.1799E-23	2.479652182	3.0291E-05	-1.9301641	0.03737632	-0.4551461	1
AT2G46370	*JAR1*	0.21240653	1	-0.206275798	1	-0.3223489	1	0.09633348	1
AT1G19180	*JAZ1*	6.25399273	1.3706E-19	5.466550108	1.0007E-10	-2.0716446	1	-1.284202	1
AT1G74950	*JAZ2*	1.49701783	0.00586066	0.409399136	1	-1.3726632	0.43335493	-0.2850445	1
AT3G17860	*JAZ3*	-0.7811866	1	-1.546767222	0.07498803	-1.429241	0.35571694	-0.6636604	1
AT1G48500	*JAZ4*	-1.3467561	1	-3.153539489	1	-1.7356224		0.07116098	
AT1G17380	*JAZ5*	6.28246793	1.7271E-22	4.726158975	3.0448E-08	-2.4359067	1	-0.8795977	1
AT1G72450	*JAZ6*	1.15913537	1	-0.273141104	1	-1.6932801	0.80434223	-0.2610036	1
AT2G34600	*JAZ7*	3.96037577	1	1.308166159		-1.0308738		1.62133581	
AT1G30135	*JAZ8*	4.72227335	1	1.503895797		-1.3268774		1.89150016	
AT1G70700	*JAZ9*	-0.2207329	1	0.047915145	1	-0.0722015	1	-0.3408495	1
AT5G13220	*JAZ10*	4.27060963	1.9538E-06	2.774436741	1	-1.903141	1	-0.4069681	1
AT3G43440	*JAZ11*	-0.55697	1	-0.25148424	1	0.28642574	1	-0.0190601	1
AT5G20900	*JAZ12*	0.70834312	1	-0.286969354	1	-1.2093061	0.00019148	-0.2139936	1
AT4G28910	*NINJA*	0.29259559	1	-0.246054602	1	-0.4362869	1	0.10236329	1
AT2G39940	*COI1*	-0.1332732	1	-0.741637788	3.1803E-05	-0.6470903	0.00292676	-0.0387258	1
AT1G06160	*ORA59 (ERF 59)*	1.74156708	1	3.441821374	1	-0.1105909		-1.8108452	1
AT3G23240	*ERF1*	3.65377754	0.00941421	7.281220511	0.00153774	0.03770848	1	-3.5897345	1
AT1G32640	*MYC2, JIN1*								
AT5G46760	*MYC3*	1.44723205	5.7728E-05	0.452601909	1	-1.2192214	0.12937179	-0.2245913	1
AT4G17880	*MYC4*	-2.4385447	5.1747E-05	-0.985736487	1	1.5246206	1	0.0718124	1
AT5G44420	*PDF1.2 a*	0.83052418	1	0.382502209	1	0.8486675	1	1.29668946	1

Supplementary Table S41. Regulation of transcript abundance of ROS-related genes in response to PL periods of 8 h in WT and *npr1*.

Genes in WT plants and *npr1* mutants that were regulated (according to Bonferroni correction) in response to an 8 h-PL periods in comparison to control. Cells remained empty when expression levels of respective genes were not detected. Blue or yellow backgrounds of cells indicate downregulation or upregulation, respectively. FC, fold change; padj, adjusted *p*-value according to Bonferroni correction.

AGI	gene id	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs.* *npr1*-control		*npr1*-8 h-PL *vs.* WT-8 h-PL		*npr1*-control *vs.* WT-control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT4G25100	*FSD1*	-1.345819	9.4678E-07	-0.5890463	1	0.88384764	1	0.127074938	1
AT5G51100	*FSD2*	1.6918028	4.2358E-27	1.92196142	4.4704E-30	0.40801591	1	0.177857293	1
AT5G23310	*FSD3*	0.53439933	1	2.06687655	6.1059E-05	2.04823707	8.0006E-05	0.515759862	1
AT1G08830	*Cu/ZnSOD (CSD1*	1.46288479	0.49110646	0.81305697	1	-0.7275534	1	-0.077725608	1
AT2G28190	*CSD2)*	0.24243842	1	-0.0487538	1	-0.1929845	1	0.098207763	1
AT5G18100	*CSD3*	-0.6983419	0.10267971	-0.4790073	1	0.45823077	1	0.2388961	1
AT3G10920	*MSD1*	0.78838278	1.8746E-06	0.19702187	1	-0.3745709	1	0.216789965	1
AT3G56350	*MnSOD-like*								
AT1G07890	*APX1*	2.61208321	2.2245E-10	0.80325941	1	-1.448195	1	0.36062879	1
AT3G09640	*APX2*	0		0.23414291		0.99907616		0.963400865	
AT4G35000	*APX3*	0.00113474	1	-0.082183	1	-0.0862693	1	-0.002951539	1
AT4G09010	*APX4*	-1.5745998	5.1328E-13	-0.9333829	0.12818898	0.84271823	0.87487187	0.201501355	1
AT4G35970	*APX5*	-0.027976	1	-0.5927557	1	-0.3324328	1	0.232346909	1
AT4G32320	*APX6*	-1.202312	0.08449863	-0.4807802	1	0.63686669	1	-0.084665182	1
	APX7								
AT4G08390	*sTromal-APX*	1.16959489	0.21160112	1.41094639	0.03668515	0.50464281	1	0.263291319	1
AT1G77490	*Thylakoid-APX*	-1.2694049	6.387E-43	-1.0822505	2.4785E-24	0.22287051	1	0.035716052	1
AT1G63940	*MDAR1*	1.75826821	4.811E-101	1.54740129	1.6436E-62	0.08950492	1	0.30037185	1
AT3G09940	*MDAR2*	7.77526301	2.454E-26	5.12448112	2.1704E-06	-3.6672445	0.00236041	-1.016462629	1
AT3G27820	*MDAR3*	-0.9522877	1	0.01632229	1	1.0665254	0.88646667	0.097915449	1
AT3G52880	*MDAR4*	0.54921061	2.6353E-09	0.22838984	1	-0.2411646	1	0.079656147	1
AT5G03630	*MDAR5*	4.27514161	5.4604E-15	1.97078471	1	-2.96966	0.00040438	-0.665303057	1
AT5G16710	*DHAR1*	-0.1759445	1	-0.0777184	1	0.13909306	1	0.040866895	1
	DHAR2								
AT1G75270	*DHAR3*	5.24436627	6.963E-36	2.73032378	1.2738E-05	-2.7669763	4.8538E-06	-0.252933771	1
AT1G19550	*DHAR4*								
AT1G19570	*DHAR5*	0.90527744	1	0.86647353	1	-0.0466731	1	-0.007869196	1
AT3G24170	*GR1*	0.59277082	0.15054708	-0.2502469	1	-0.8546494	0.00013346	-0.011631771	1
AT3G54660	*GR2*	-0.8556408	2.7787E-15	-0.5249233	0.01217538	0.19473819	1	-0.135979308	1
AT1G20630	*CaT1*	1.40728252	0.00299502	0.38749698	1	-0.7785487	1	0.241236808	1
AT4G35090	*CaT2*	-2.3192189	4.5563E-08	-0.8066742	1	1.24433945	1	-0.268205285	1
AT1G20620	*CaT3*	-2.1128064	8.1733E-08	-1.0706257	1	0.67526752	1	-0.366913227	1
AT2G25080	*GPX1*	-2.4042346	2.168E-122	-1.7985696	5.9639E-54	0.179056	1	-0.426609045	0.297806674
AT2G31570	*GPX2*	2.16169294	3.6874E-18	-0.3408346	1	-2.6755009	7.4685E-23	-0.172973282	1
AT2G43350	*GPX3*	-0.2616359	1	-0.2881788	1	0.19553635	1	0.222079292	1
AT2G48150	*GPX4*	2.53523381	1	2.35373269	1	-1.6351739		-1.453672825	
AT3G63080	*GPX5*	1.0510738	8.4682E-05	-0.0804494	1	-1.2561184	6.0345E-06	-0.124595264	1
AT4G31870	*GPX7*	3.47060409	7.6639E-28	3.52487111	4.0472E-23	-0.1526117	1	-0.206878674	1

AGI	gene id	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs. npr1*-control		*npr1*-8 h-PL *vs.* WT-8 h-PL		*npr1*-control *vs.* WT-control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT1G63460	*GPX8*	1.10015164	0.01549074	-0.154098	1	-1.1387286	0.07968505	0.115521089	1
AT4G11600	*GPX6*	2.77018794	8.4658E-18	1.25605159	1	-1.4883862	0.05341672	0.025750143	1
AT5G01600	*FerriTin 1*	0.53458309	1	-0.191874	1	-0.4059498	1	0.320507293	1
AT3G56090	*FerriTin 2*	1.01596868	1	1.85679352	0.00016673	1.15729965	1	0.316474811	1
AT2G40300	*FerriTin 3*	-1.0001606	1	-0.2732162	1	1.21788517	1	0.490940812	1
AT3G11050	*FerriTin 4*	1.49022412	1	1.01862933	1	0.3187262		0.790320988	1
AT5G20230		6.49379355	3.5627E-16	6.99923049	1.2588E-14	-2.4662141	1	-2.971651034	0.491166342
AT1G72230		-1.2761504	1	-1.5712965	0.06296946	0.59298175	1	0.888127835	1
AT3G27200		-0.5339169	1	0.12765936	1	0.81395402	1	0.152377808	1
AT3G60280									
AT4G12880		-1.0769523	1	-0.286348	1	1.12573362	1	0.335129295	1
AT5G26330		-1.4667214	0.01749248	-0.4441858	1	1.27247538	1	0.249939796	1
AT2G33740		-0.6911121	0.0029442	-0.7653299	0.00357218	-0.2371448	1	-0.162926986	1
AT4G28365		2.32726744		1.19587643		-1.1297243		0	
AT2G31050									
AT5G07390	*RbohA*	0		-1.189268		0		0.963388027	
AT1G09090	*RbohB*	2.92205838		0		-3.1479427		0	
AT5G51060	*RbohC*	7.56053095	1.0636E-28	5.44854504	3.0629E-10	-2.895177	0.58935707	-0.783191076	1
AT5G47910	*RbohD*	3.63459249	5.755E-17	2.08429114	0.0228314	-1.5878905	1	-0.037589184	1
AT1G19230	*RbohE*	3.95687895	0.847917	2.92249481	1	-1.3114276		-0.277043442	1
AT1G64060	*RbohF*	1.00680546	1	0.33029285	1	-0.7090278	1	-0.032515159	1
AT4G25090	*RbohG*	2.66390407		2.780552		0.118332		0	
AT5G60010	*RbohH*	5.42568786		0		-5.6515721		0	
AT4G11230	*RbohI*	3.3949841	0.21919417	2.91095971	1	-0.7015809		-0.217556521	1
AT3G45810	*RbohJ*								
AT5G23980		-2.0178175	0.31039328	-0.4295111	1	1.47626774		-0.112038692	1
AT1G01590	*FRO1*	-1.1291093	1	0.11964473	1	0.68300159		-0.565752419	1
AT1G01580	*FRO2*	-2.7680434	1.0135E-05	-1.2664686	1	1.72817394	1	0.226599126	1
AT5G23990	*FRO5*								
AT5G49730	*FRO6*	-4.8523159	1.4979E-09	-2.5774761	1	1.74131516	1	-0.533524682	1
AT5G49740	*FRO7*	-3.1407859	0.00238079	-0.952055	1	1.69249574	1	-0.496235111	1
AT5G50160	*FRO8*	-1.6697015	1	-0.1173608	1	1.8469985	1	0.294657781	1
AT5G67590		0.13272802	1	-0.002165	1	0.06949559	1	0.204388626	1
AT1G23020		-0.838549	1	-0.9987136	1	-0.966431	1	-0.80626633	1
AT1G32350		7.05753551	1	25.0457445	9.8009E-16	-4.0028491	1	-21.99105816	2.80686E-14
AT3G22370	*AOX1A*	6.46511684	3.3559E-26	3.85029961	1.8701E-05	-3.0372321	0.02308914	-0.422414827	1
AT3G22360	*AOX1B*	6.31434318	1	2.16538419		-4.147307		0	
AT3G27620	*AOX1C*	1.40492662	1	1.63819756		2.33906112		2.105790184	
AT5G64210	*AOX2*	4.17605713		2.27568741		-1.8986766		0	
AT4G22260	*ImmuTans*	1.41048511	0.00011877	0.46428019	1	-1.1190682	0.61439218	-0.172863297	1
AT1G48130	*1-Cys PrxR*								
AT3G11630	*2-cysPrxRA*	0.14681817	1	0.04626302	1	0.16207082	1	0.262625978	1
AT5G06290	*2-cysPrxRB*	-0.089187	1	0.05077757	1	0.42371253	1	0.283747968	1
AT3G06050	*2-cysPrxRF*	1.13628933	1.0497E-22	0.46337587	1	-0.4056744	1	0.2672391	1
AT3G26060	*PrxR Q*	0.66073939	4.577E-08	0.33227637	1	0.24519715	1	0.573660171	2.05956E-05
AT1G65990									
AT1G65980		1.36733952	1.6643E-36	0.36939942	1	-0.9295598	4.5663E-12	0.068380308	1
AT1G65970		4.37912419	1	8.28265824	1	-4.4178927	1	-8.321426772	1
AT1G60740		4.08100142	1	3.77022775	1	-3.5491637		-3.238390002	
AT3G52960		0.79804929	0.04465147	0.70840403	1	0.36469147	1	0.454336738	1
AT3G03405									
AT2G04700		0.06326578	1	-0.255382	1	-0.2207795	1	0.097868332	1
AT1G62180		-0.6901265	0.00105646	-0.786346	0.0005138	0.29896348	1	0.395182935	1
AT1G43560		-0.1024276	1	-0.0070832	1	0.23486182	1	0.139517466	1
AT1G31020		0.68077197	1	0.2558946	1	-0.4652657	1	-0.040388343	1
AT1G52990		-1.7058018	1	-0.7621749	1	0.95993916		0.016312226	
AT1G53300		-1.2509545	3.5045E-06	-0.3810987	1	0.98528241	0.11155956	0.115426691	1
AT1G76760		1.27592117	1	0.56423711	1	-0.8875335	1	-0.175849432	1
AT2G33270		3.67749544	1	2.21524557	1	-5.0666691		-3.604419212	
AT2G42580		-2.3252637	1.6857E-13	-2.3048458	3.9884E-10	0.09611288	1	0.075694984	1
AT3G06730		0.41167546	1	1.50648273	4.7951E-08	0.94794582	0.15765961	-0.146861454	1
AT3G08710		2.41204898	1.5619E-09	1.19652445	1	-1.2819032	1	-0.066378685	1
AT3G20560		0.99103093	0.05371245	0.31411275	1	-0.4174558	1	0.259462392	1
AT3G56420		1.20578061		-1.8886066		-2.3934454		0.700941814	
AT4G04950		-0.7351238	1	0.05644122	1	0.677558	1	-0.114006994	1
AT3G56420		1.20578061		-1.8886066		-2.3934454		0.700941814	
AT4G29670		3.35126587	4.341E-14	0.99147864	1	-2.1954058	0.00496627	0.164381425	1
AT4G32580		-0.4318706		2.27570025		1.11712494		-1.590445938	
AT4G37200		-1.3038009	1	-0.4280128	1	0.75359985	1	-0.122188293	1
AT2G40790		-1.6448375		-0.7657184		2.0192986		1.140179544	
AT3G51030	*TRX-H-1*	-0.119046	1	0.32494927	1	0.89777389	1	0.453778576	1
AT5G39950		1.17164713	1.1696E-15	0.45482874	1	-0.8980747	4.638E-06	-0.181256343	1
AT5G42980		0.69350047	1	0.19654832	1	-0.3959707	1	0.100981484	1
AT1G19730	*TRX-H-4*	-0.3837241	1	-1.0552095	1.9403E-06	-0.7254359	0.24395773	-0.05395051	1
AT1G45145	*TRX-H-5*	4.85741714	2.4888E-23	2.33387652	0.05767718	-3.0502765	1.3589E-05	-0.526735901	1
AT1G03680	*TRX-M1*	0.2771026	1	-0.0314098	1	-0.2586777	1	0.049834706	1
AT4G03520		0.20841005	1	-0.1046733	1	-0.2314119	1	0.081671472	1
AT2G15570		0.87689808	1	-0.5975783	1	-1.6040494	0.00025025	-0.12957299	1
AT3G15360		-0.3298062	1	-0.3962654	1	0.00471116	1	0.071170379	1
AT4G35460		0.27142157	1	-0.4480052	1	-0.6140345	1	0.105392273	1

AGI	gene id	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs.* *npr1*-control		*npr1*-8 h-PL *vs.* WT-8 h-PL		*npr1*-control *vs.* WT-control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT2G17420		1.76608581	8.8895E-13	0.3069477	1	-1.4426736	1.3689E-05	0.016464537	1
AT2G41680		-0.1177873	1	0.15648736	1	0.27580229	1	0.001527659	1
AT1G50320		0.19705834	1	-0.3799546	1	-0.7076669	0.13545277	-0.130654018	1
AT1G03020		1.53888578	1	0.04486869	1	-1.7491499	1	-0.25513279	1
AT1G03850		6.02487822	6.2817E-18	1.55282796	1	-6.3266612	2.2158E-15	-1.854610932	1
AT1G06830		0.12001535	1	0.36765837	1	-0.2262796	1	-0.473922624	1
AT1G28480		7.99802447	1.3404E-26	10.3826494	3.5242E-10	-2.5711906	1	-4.955815561	1
AT2G20270		0.85154224	1.3122E-05	0.09268264	1	0.02663332	1	0.785492926	0.000209469
AT2G30540		-2.0208542	3.3045E-09	-2.0989815	3.3998E-07	-0.5379937	1	-0.459866451	1
AT2G47870		4.45010903	1	4.64534347	1	-2.1925597		-2.387794114	
AT2G47880		-0.4824999	1	-1.865646	1	-2.5430579	1	-1.159911863	1
AT3G02000									
AT3G62930		0.27560356	1	-0.0732008	1	-0.449851	1	-0.101046677	1
AT3G62950		-2.244982	2.2252E-26	-2.5729925	1.2547E-24	-1.0736687	0.10995387	-0.745658158	1
AT3G62960		-0.4920061	1	0.55212963		0.05836703		-0.985768678	
AT4G15660		0.75360789	1	0.56278843	1	-0.1331165	1	0.057702955	1
AT4G15660		0.75360789	1	0.56278843	1	-0.1331165	1	0.057702955	1
AT4G15680		1.74948327	1	1.21113633	1	-1.5593564	1	-1.021009469	1
AT4G15690		0.94310283	1	0.55663024	1	-1.1149902	1	-0.728517577	1
AT4G15700		1.65667229	0.59639162	0.99369664	1	-1.0651801	1	-0.402204455	1
AT4G28730		0.03971327	1	-0.0596903	1	0.22857299	1	0.327976526	1
AT4G33040		1.02100347	1	-0.1006655	1	-0.2956499	1	0.826019057	1
AT5G11930		1.42881179	1	0.75993138	1	0.70015334		1.369033748	1
AT5G14070		-3.6644311	1	-5.162887		-2.152647		-0.654191159	
AT5G18600		-1.25801	1.5056E-06	-1.3867892	2.5037E-06	-0.1074805	1	0.021298672	1
AT1G77370		2.10203596	4.0511E-07	0.39742325	1	-2.0975767	3.7232E-05	-0.392963957	1
AT5G20500		-0.055789	1	-0.2888555	1	-0.2252957	1	0.007770801	1
AT5G40370		1.02270013	8.6061E-08	0.57338201	1	-0.5298035	1	-0.080485429	1
AT5G63030		0.19916021	1	-0.6751652	0.09815271	-0.9333937	4.9434E-06	-0.059068268	1
AT3G11920		-3.2346929		-3.2946829		0		-0.364348193	
AT1G05240	*PER1*	-4.226972	1	-0.0355591		3.86435549		-0.327057382	
AT1G05250	*PER2*								
AT1G05260	*PER3*	1.16025563		-0.3971803		0.10528608		1.662722042	
AT1G14540	*PER4*	7.15416744	1.5668E-16	5.63390219	6.0606E-06	-2.2889413	1	-0.768676014	1
AT1G14550	*PER5*								
AT1G24110	*PER6*								
AT1G30870	*PER7*								
AT1G34510	*PER8*								
AT1G44970	*PER9*								
AT1G49570	*PER10*								
AT1G68850	*PER11*	-2.3179407		0		0		-2.51475088	
AT1G71695	*PER12*	-1.9289861	0.02083536	-0.2613143	1	1.8455355	0.4893154	0.177863674	1
AT1G77100	*PER13*								
AT2G18140	*PER14*								
AT2G18150	*PER15*	3.17315682		2.15489706		-1.0165767		0	
AT2G18980	*PER16*	-4.073938		0.10159635		4.1837664		0.008232076	
AT2G22420	*PER17*	-2.0307432	1	-1.4767771	1	1.15907823	1	0.605112151	1
AT2G24800	*PER18*	0		1.26386978		1.06705033		0	
AT2G34060	*PER19*	-3.8854145	2.4061E-15	-0.8776376	1	2.81466102	1.975E-05	-0.193115881	1
AT2G35380	*PER20*	-0.7633204		3.52565361		3.32886324		-0.96011079	
AT2G37130	*PER21*	0.59583142	1	0.8113252	1	0.34918538	1	0.133691599	1
AT2G38380	*PER22*	-2.31794		-1.1892907		0		-1.552996897	
AT2G38390	*PER23*								
AT2G39040	*PER24*								
AT2G41480	*PER25*	-1.8796018	1	-1.6492325	1	-0.5484613		-0.778830533	1
AT2G43480	*PER26*	-2.3179407		0		0		-2.51475088	
AT3G01190	*PER27*								
AT3G03670	*PER28*	6.09046488	1	2.75378434		-3.3350395		0	
AT3G17070	*PER29*	-1.9054106	0.00115396	-1.4898973	1	0.81380947	1	0.398296166	1
AT3G21770	*PER30*	0.52756169	1	-0.5643939	1	-1.1087715		-0.016815922	1
AT3G28200	*PER31*	-2.9519804	5.9715E-17	-1.2274153	1	1.78584018	0.00500987	0.061275062	1
AT3G32980	*PER32*	-4.4042774	0.0003062	-3.4938399	0.33564211	0.35636339	1	-0.554074159	1
AT3G49110	*PER33*	8.34440079	0.00181828	5.11631892	1	-3.8092721	1	-0.581190283	1
AT3G49120	*PER34*	5.19744826	1.7061E-22	3.54646517	1.2211E-06	-2.4736812	0.08448252	-0.822698152	1
AT3G49960	*PER35*	0.52921128		-0.2278755		0.56321192		1.320298719	
AT3G50990	*PER36*	0		3.278249		3.08144772		0	
AT4G08770	*PER37*	6.28239484	1	7.83329607	1	-4.2543622	1	-5.805263378	1
AT4G08780	*PER38*	3.08770282	1	3.16003446	1	-3.6611412		-3.73347285	
AT4G11290	*PER39*	-5.9378091	1	-4.4388931		0		-1.923267256	
AT4G16270	*PER40*	0.48587762	1	-0.9145273		0.77979231		2.180197249	
AT4G17690	*PER41*								
AT4G21960	*PER42*	-1.5485843	1	-0.5590417	1	1.15014169	1	0.160599051	1
AT4G25980	*PER43*	-0.6042282		0.17925949		0.65993997		-0.123547724	
AT4G26010	*PER44*	3.16421414		0.94225312		0.33929614		2.561257154	
AT4G30170	*PER45*	-2.7341797		0		0		-2.930986164	
AT4G31760	*PER46*	2.79825708		0		-3.9859294		-0.960112519	
AT4G33420	*PER47*	1.27745513	1	-1.4498374	1	-2.7413365	0.05266269	-0.014043959	1
AT4G33870	*PER48*								
AT4G36430	*PER49*	6.1399123	0.9040342	1.25301501	1	-4.8852685		0.001628817	
AT4G37520	*PER50*	-0.6205635	1	-0.9953546	1	-0.8123907	1	-0.437599539	1

AGI	gene id	WT-8 h-PL *vs.* WT-control		*npr1*-8 h-PL *vs.* *npr1*-control		*npr1*-8 h-PL *vs.* WT-8 h-PL		*npr1*-control *vs.* WT-control	
		log_2FC	padj	log_2FC	padj	log_2FC	padj	log_2FC	padj
AT4G37530	*PER51*	1.68768915	0.00180191	0.37510045	1	-1.6202599	0.07383936	-0.307671223	1
AT5G05340	*PER52*	3.56373165	0.95736386	1.30802449	1	-4.1910617		-1.935354554	1
AT5G06720	*PER53*	-0.1658244	1	-0.6983442	1	-0.2977804		0.234739325	
AT5G06730	*PER54*	5.46400393	1	2.04496325	1	-2.6464137		0.772626964	
AT5G14130	*PER55*								
AT5G15180	*PER56*	0.30402267	1	-1.0157901	1	0.38029372		1.700106476	1
AT5G17820	*PER57*								
AT5G19880	*PER58*	7.13321122	3.2397E-09	6.3426024	0.10894031	-3.6246296	0.02795865	-2.834020822	1
AT5G19890	*PER59*								
AT5G22410	*PER60*								
AT5G24070	*PER61*								
AT5G39580	*PER62*	1.48478672	1	4.59552119	1	-1.8126819		-4.923416364	1
AT5G40150	*PER63*	-1.7703015	0.10291723	0.04206012	1	1.86117099	0.17335794	0.048809396	1
AT5G42180	*PER64*	2.2113688		0		-3.3990411		-0.960117812	
AT5G47000	*PER65*								
AT5G51890	*PER66*	-0.2663006	1	-0.3601212	1	0.53859031	1	0.632410964	1
AT5G58390	*PER67*	-2.1262422	0.00574553	-1.188178	1	0.37042968	1	-0.567634464	1
AT5G58400	*PER68*								
AT5G64100	*PER69*	4.54662379		3.73914337		-1.7676051		-0.960124659	
AT5G64110	*PER70*	5.02317906		4.20206596		-0.819472		0	
AT5G64120	*PER71*	3.89889341	1.728E-08	4.12289544	3.6059E-07	-0.9740338	1	-1.19803584	1
AT5G66390	*PER72*	1.80314749	1	0.67480896	1	-0.0547789		1.07355966	1
AT5G67400	*PER73*	1.74832449		0		-1.9742088		0	