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# Gene expression profile of *zeitlupe/lov kelch protein1* T-DNA insertion mutants in *Arabidopsis thaliana*: Downregulation of auxin-inducible genes in hypocotyls

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**Keywords:** *Arabidopsis thaliana*, auxin, high temperature, hypocotyl elongation, microarray, *SAUR*, *ZEITLUPE*

**Abbreviations:** *ACT2*, *ACTIN2*; *ARGOS*, *AUXIN-REGULATED GENE INVOLVED IN ORGAN SIZE*; *ARL*, *ARGOS-LIKE*; *AUX/IAA*, *AUXIN/INDOLE-3-ACETIC ACID*; *BEE1*, *BR ENHANCED EXPRESSION1*; *BRI1*, *BRASSINOSTEROID INSENSITIVE1*; *CCA1*, *CIRCADIAN CLOCK ASSOCIATED1*; *LHY*, *LATE ELONGATED HYPOCOTYL*; LKP1, LOV KELCH PROTEIN1; *PRR*, PSEUDO RESPONSE REGULATOR; qRT-PCR, quantitative reverse-transcription PCR; *SAUR*, *SMALL AUXIN UP RNA*; *ZTL*, zeitlupe.

Elongation of hypocotyl cells has been studied as a model for elucidating the contribution of cellular expansion to plant organ growth. ZEITLUPE (ZTL) or LOV KELCH PROTEIN1 (LKP1) is a positive regulator of warmth-induced hypocotyl elongation under white light in *Arabidopsis*, although the molecular mechanisms by which it promotes hypocotyl cell elongation remain unknown. Microarray analysis showed that 134 genes were upregulated and 204 genes including 15 auxin-inducible genes were downregulated in the seedlings of 2 *ztl* T-DNA insertion mutants grown under warm conditions with continuous white light. Application of a polar auxin transport inhibitor, an auxin antagonist or an auxin biosynthesis inhibitor inhibited hypocotyl elongation of control seedlings to the level observed with the *ztl* mutant. Our data suggest the involvement of auxin and auxin-inducible genes in ZTL-mediated hypocotyl elongation.

Plants use light not only as an energy source but also as an environmental cue for growth and development.[1,2] Numerous genes and proteins are involved in the perception and transduction of environmental light signals, including photoreceptors.[1,2] ZEITLUPE (ZTL)/LOV KELCH PROTEIN1 (LKP1) is a blue-light photoreceptor harbouring an F-box in *Arabidopsis*.[3-5] ZTL determines the period of circadian oscillation, regulates photoperiodic flowering and promotes hypocotyl elongation under light.[3-6] The molecular mechanisms of circadian oscillation and flowering-time control have been relatively well studied, whereas those for promotion of hypocotyl elongation remain largely unknown. Elongation of hypocotyl cells has been studied as a model for elucidating the contribution of cellular expansion to plant organ growth.[7] *ZTL*-overexpressing plants have elongated hypocotyls when grown at 22°C with continuous white light.[3,4] *ztl* mutants are indistinguishable from the wild type in the dark, but they have short hypocotyls when grown at 22°C with continuous white light.[8] The difference in hypocotyl length between the *ztl* mutant and wild-type seedlings was more significant when they were grown at 28°C with continuous white light.[8] Thus, ZTL functions as a positive regulator in warmth-induced hypocotyl elongation under white light. In this study, we report our microarray analysis, real-time quantitative reverse-transcription PCR (qRT-PCR) results and auxin inhibitor experiments using *ztl* mutant and wild-type seedlings to support the idea that auxin and auxin-inducible genes are involved in the hypocotyl elongation mediated by ZTL.

Seeds of 2 *ztl* T-DNA insertion mutants, *ztl-3* and *ztl-105*,[9] and the wild-type Columbia accession were sown on 1/2-basal-salt Murashige and Skoog agar (0.8% w/v) medium without sucrose and incubated at 4°C for 7 d in the dark. The seeds were then incubated at 22°C for 3 d and then 28°C for 5 d with continuous white light (80 $\mu mol \cdot m^{-2} \cdot s^{-1}$) as reported.[8] Total RNA was isolated from 8-day-old *ztl-3, ztl-105* and wild-type seedlings (2 biological replicates: Col1 and Col2) using an RNeasy Plant Mini Kit (Qiagen, Valencia, CA, USA). The microarray experiment was performed using *Arabidopsis* (V4) Gene Expression Microarrays 4 × 44K (G2519F-021169) (Agilent Technologies,

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Böblingen, Germany) as previously described.[10] A total of 134 genes were upregulated (average fold change > 2.0; $p < 0.01$) and 204 genes were downregulated (average fold change < −2.0; $p < 0.01$) in *ztl* mutants. These genes are listed in **Table S1 and S2**. Downregulation of *ZTL* in *ztl* mutants (AT5G57360; average fold change −63.0) validated this microarray analysis. The downregulation of the clock genes *PSEUDO RESPONSE REGULATOR9* (*PRR9*)[11,12] (AT2G46790; average fold change −14.0), *LATE ELONGATED HYPOCOTYL* (*LHY*)[12,13] (AT1G01060; average fold change −4.7), *CIRCADIAN CLOCK ASSOCIATED1* (*CCA1*)[12,14] (AT2G46830; 3 probes: average fold changes −2.9, −2.5 and −2.4) in *ztl* mutants was consistent with the involvement of ZTL in circadian clock regulation. Pathway analysis of upregulated and downregulated genes with the Gene Functional Classification Tool in DAVID Bioinformatics Resources 6.7 (http://david.abcc.ncifcrf.gov/home.jsp) classified them into several groups. As for the upregulated genes, no group was detected with a significant *P*-value (<0.05) (data not shown). Among the downregulated genes, 24 genes involved in the response to endogenous stimulus were detected as the group with the smallest *P*-value (3.3E-08; **Table S3**). Based on this grouping and the result from the Arabidopsis eFP Browser (http://bar.utoronto.ca/efp/cgi-bin/efpWeb.cgi), we constructed **Table 1**, which lists auxin-inducible genes downregulated in *ztl* seedlings. They included 7 *SMALL AUXIN UP RNA* (*SAUR*)[15,16] genes (*SAUR1, 9, 10, 22, 23, 46* and *68*), 4 *AUXIN/INDOLE-3-ACETIC ACID* (*AUX/IAA*)[15-17] genes (*IAA1, 5, 29* and *34*), a gene for indole-3-acetic acid-amido synthetase (*GH3.5*),[15,16,18] a gene for 1-aminocyclopropane-1-carboxylic acid synthase (*ASC4*),[19] *AUXIN-REGULATED GENE INVOLVED IN ORGAN SIZE* (*ARGOS*)[20] and *ARGOS-LIKE* (*ARL*).[21] Among these 15 genes, 13 were included in the group for response to endogenous stimulus and 12 were included in the group for response to auxin stimulus in **Table S3**.

We performed real-time quantitative reverse-transcription qRT-PCR to confirm the downregulation of 3 auxin-inducible genes, *SAUR22, SAUR23* and *IAA29*, in hypocotyls of 8-day-old *ztl* mutants in comparison with wild-type seedlings grown as for microarray analysis. RNA was isolated from hypocotyls with RNAiso Plus (Takara, Otsu, Japan). The reverse transcription reaction was conducted with an oligo dT primer and PrimeScript II (Takara). PCR was performed with a StepOnePlus Real-Time PCR System (Life Technologies, Carlsbad, CA). The PCR primers are listed in **Table S4**. The expression level of *ACTIN2* (*ACT2*) was used to normalize the expression levels of the target genes. The expression of *SAUR22, SAUR23* and *IAA29* was significantly lower in both *ztl* mutant hypocotyls than in wild-type hypocotyls (**Fig. 1**), confirming that these genes are downregulated in the hypocotyls of 2 *ztl* mutants.

To assess the role of auxin in hypocotyl elongation in *ztl* mutants and wild-type seedlings, we used 3 auxin inhibitors. The *ztl* mutant and wild-type seedlings were grown as described above in the absence or presence of the auxin transport inhibitor *N*-1-naphthylphthalamic acid (NPA),[22] the auxin antagonist α-(phenyl ethyl-2-one)-indole-3-acetic acid (PEO-IAA)[23] or the auxin biosynthesis inhibitor 5-(4-chlorophenyl)-4*H*-1,2,4-triazole-3-thiol (YUCASIN).[24] In the presence of 1 μM NPA, 5 μM PEO-IAA or 25 μM YUCASIN, the hypocotyl lengths of wild-type seedlings were reduced, although their hypocotyls were still significantly longer than those of *ztl* mutants (**Fig. 2**). Enhanced

**Table 1.** Auxin-inducible genes downregulated in *ztl* seedlings in comparison with wild-type seedlings

| | | | *ztl* vs Col | |
|---|---|---|---|---|
| **Array element** | **Gene Locus** | **Gene name** | **Fold change** | **P-value** |
| A_84_P17344 | AT2G22810 | *ACS4* | −6.569 | 8.83.E-15 |
| A_84_P16724 | AT1G15050 | *IAA34* | −5.317 | 9.89.E-13 |
| A_84_P12605 | AT2G18010 | *SAUR10* | −3.601 | 0.00.E+00 |
| A_84_P13527 | AT2G37030 | *SAUR46* | −3.333 | 6.83.E-07 |
| A_84_P18064 | AT1G15580 | *IAA5* | −2.899 | 4.58.E-28 |
| A_84_P843096 | AT1G29490 | *SAUR68* | −2.876 | 3.35.E-14 |
| A_84_P55550 | AT4G32280 | *IAA29* | −2.756 | 6.87.E-09 |
| A_84_P794374 | AT4G27260 | *GH3.5* | −2.585 | 6.11.E-25 |
| A_84_P524502 | AT3G59900 | *ARGOS* | −2.541 | 5.04.E-44 |
| A_84_P230289 | AT2G44080 | *ARL* | −2.268 | 3.87.E-16 |
| A_84_P10124 | AT4G14560 | *IAA1* | −2.145 | 1.47.E-15 |
| A_84_P21449 | AT4G34770 | *SAUR1* | −2.120 | 2.67.E-14 |
| A_84_P12944 | AT4G36110 | *SAUR9* | −2.058 | 4.26.E-03 |
| A_84_P94979 | AT5G18060 | *SAUR23* | −2.052 | 3.10.E-10 |
| A_84_P141269 | AT5G18050 | *SAUR22* | −2.029 | 1.03.E-15 |

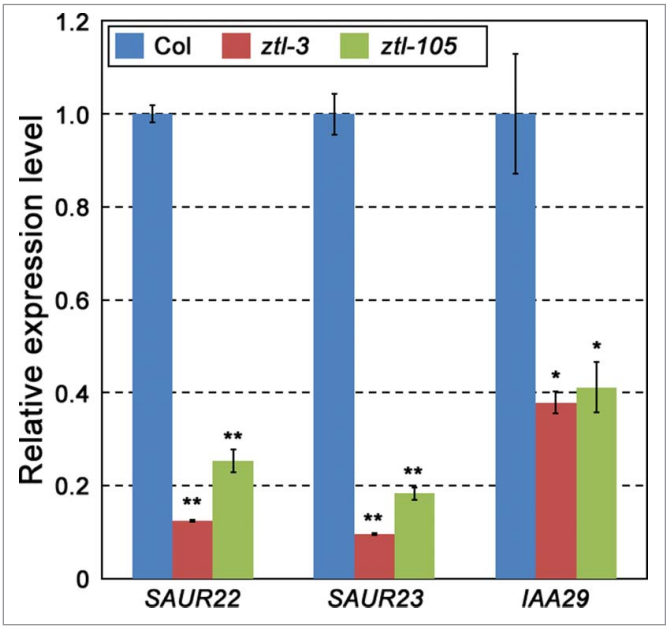


**Figure 1.** Expression of *SAUR22, SAUR23* and *IAA29* in hypocotyls of wild-type Columbia (Col) and 2 *ztl* mutant seedlings (*ztl-3* and *ztl-105*) quantified by real-time quantitative reverse-transcription PCR (qRT-PCR). Seedlings of wild-type Col plants and *ztl* mutants were grown on 1/2-basal-salt Murashige and Skoog agar (0.8% w/v) medium without sucrose for 3 d at 22°C and then for 5 d at 28°C with continuous white light (80 $\mu mol \cdot m^{-2} \cdot s^{-1}$). The expression levels were normalized using the actin transcript (*ACT2*) expression levels. The values for *ztl-3* and *ztl-105* are shown relative to wild-type Col. Error bars represent standard error of the mean ($n = 3$; technical replicates); *(***P**)< 0.05, **(***P*)< 0.01 (Student's *t* test) in comparison with wild-type Col.

hypocotyl elongation in wild-type seedlings was completely inhibited to *ztl* mutant levels by 5 μM NPA, 50 μM PEO-IAA or 100 μM YUCASIN. These results suggest the involvement of auxin in the hypocotyl elongation in seedlings mediated by ZTL.

Auxin regulates the expression of a large number of genes involved in growth, development and differentiation.[25] Among them, the early or primary auxin-inducible genes are grouped into at least 3 families: *AUX/IAA, GH3* and *SAUR* genes.[15,16] *AUX/IAA* genes encode transcriptional repressors and function in negative-feedback pathways to attenuate auxin responses.[17] *GH3* genes encode auxin-conjugating enzymes and function to attenuate auxin responses by downregulation of endogenous active auxin levels.[18] The expression of *SAUR* genes is usually associated with tissue elongation.[26,27] Among *SAUR* genes, members of 2 subfamilies, *SAUR19–24* and *SAUR61–68*, are postulated as positive effectors of cell expansion.[27-30] The sequences of *SAUR19–24* are also highly identical to one another and these 6 genes are located in a cluster on chromosome 5, suggesting their functional redundancy.[15,27] Plants overexpressing *SAUR19* have longer hypocotyls than wild-type plants under continuous light,[27] and plants overexpressing a *GFP-SAUR19, 21, 23* or *24* fusion gene also have elongated hypocotyls under long-day conditions.[28] SAUR19 stimulates plasma membrane $H^+$ -ATPase by inhibiting the activity of type 2C protein phosphatases, leading to cell expansion via an acid growth mechanism.[29] Thus, the downregulation of *SAUR22* and *23* detected by microarray analysis and confirmed by qRT-PCR may contribute to reducing hypocotyl elongation in *ztl* mutants. As for *SAUR61–68*, these genes are located in a cluster on chromosome 1 with high DNA sequence identity.[15,30] The positive function of SAUR63 in hypocotyl elongation was reported, although *SAUR68* may be a pseudogene, as it encodes a shorter open reading frame than other members in the gene subfamily.[30] Thus, the meaning of the downregulation of *SAUR68* in the hypocotyls of *ztl* mutants is unknown.

We observed downregulation of the expression of 2 genes for gibberellin biosynthesis emzymes, i.e. twenty-oxidase (*GA5*) (AT4G25420; average fold change −2.9) and ent-copalyl diphosphate synthase (GA1) (AT4G02780; average fold change −2.4), and 2 genes involved in brassinosteroid signaling, i.e BRASSINOSTEROID INSENSITIVE1 (BRI1) (AT4G39400; average fold change of −2.4) and BR ENHANCED EXPRESSION1 (BEE1) (AT1G18400; average fold change −2.3). These data suggest *ZTL*-dependent changes in gibberellin level and brassinosteroid signaling. Gibberellins stimulate cell elongation in *Arabidopsis* hypocotyls.[31] Brassinosteroids act on light-grown hypocotyl elongation independent of, but co-operatively with, gibberellins and auxin.[32] The downregulation of gene expression for gibberellin biosynthesis and brassinosteroid signaling in *ztl* seedlings suggests that these hormones also contribute to the effect of ZTL on hypocotyl elongation.

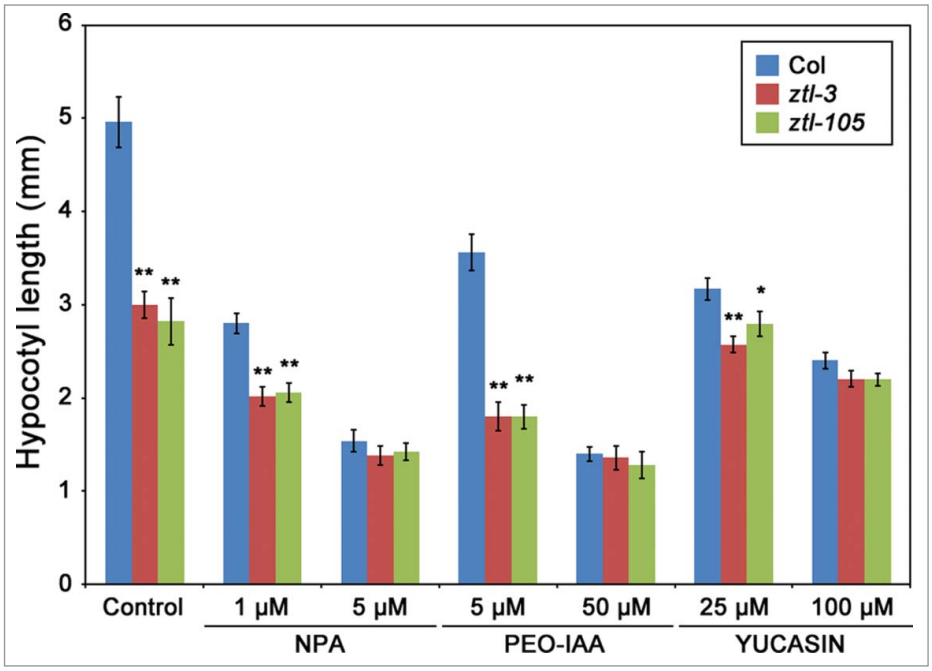


**Figure 2.** Inhibition of hypocotyl elongation by auxin inhibitors. Seedlings of wild-type Col plants and *ztl* mutants were grown for 3 d at 22°C and then for 5 d at 28°C with continuous white light (80 $\mu mol \cdot m^{-2} \cdot s^{-1}$) on 1/2-basal-salt Murashige and Skoog agar (0.8% w/v) medium without sucrose in the absence or presence of indicated concentrations of *N*-1-naphthylphthalamic acid (NPA), α-(phenyl ethyl-2-one)-indole-3-acetic acid (PEO-IAA) or 5-(4-chlorophenyl)-4H-1,2,4-triazole-3-thiol (YUCASIN). Error bars represent standard error of the mean ($n = 5–10$); *(***P***)< 0.05, **(***P***)< 0.01 (Student's *t* test) in comparison with wild-type Col.

### Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.


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### Supplemental Material

Supplemental data for this article can be accessed on the publishers website.

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