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BRIEF COMMUNICATION

Auxin binding proteins ABP1 and ABP4 are involved in the light- and auxin-induced down-regulation of phytochrome gene *PHYB* in maize (*Zea mays* L.) mesocotyl

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Abstract Previous research in maize suggested a possible involvement of auxin-binding proteins (ABPs) in light signaling during maize seedling development. To obtain more information about the interaction of auxin and light signaling, we investigated the gene expression of phytochrome B (*PHYB*) and phytochrome A (*PHYA*) in loss-of-function mutants in *ABP1* and *ABP4* genes in maize (*Zea mays* L.). We studied how expression of the *PHYB* and *PHYA* genes in mesocotyl is regulated by white light (WL), and whether exogenous auxin NAA influences the expression of the phytochrome genes. We found that knockout of *ABP1* or *ABP4* results in essentially reduced expression of *PHYB* gene in dark-grown mesocotyl. WL reduced *PHYB* expression in WT but not in the *ABPs* knockout seedlings. The data indicate that ABP1 and ABP4 are positively involved in *PHYB* expression in etiolated mesocotyl. Our results also indicate that in etiolated mesocotyl, ABP1 and ABP4 mediate the inhibitory effect of exogenous auxin on level of *PHYB* transcript. In contrast, in our experimental conditions, WL does not reduce expression of *PHYA*. Our results further suggest that ABP1 and ABP4 are not likely involved in the expression of *PHYA* gene and neither in auxin-induced suppression of *PHYA* transcript accumulation. Our results support the existence of cross-talk between auxin and light signaling and indicate for the first time that *ABP1*, *ABP4* and *PHYB* genes could share common signaling pathway(s).

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Abbreviations

ABP	Auxin binding protein
NAA	1-Naphthalene acetic acid
WL	White light
WT	Wild-type

Introduction

The primary step of auxin signaling is the binding of auxin to an auxin receptor. In addition to described function of auxin receptors from TRANSPORT INHIBITOR RESPONSE 1/AUXIN-BINDING F-BOX PROTEIN (TIR1/AFB) family, putative auxin receptor, the ABP1 (AUXIN-BINDING PROTEIN 1) has been identified (reviewed in Tromas and Perrot-Rechenmann 2010, and in Shishova and Lindberg 2010). ABP1 is a protein with high-affinity to auxin identified in maize coleoptiles over 30 years ago by its capacity to bind radiolabelled auxin (Hertel et al. 1972; Löbler and Klämbt 1985). Several studies demonstrated that ABP1 acts at the plasma membrane (Barbier-Brygoo et al. 1989; Leblanc et al. 1999). On the other hand, the predominant localization of ABP1 was found to be in the endoplasmic reticulum lumen (Jones and Herman 1993). It was demonstrated that ABP1 plays a role in cell expansion (Jones et al. 1998; Chen et al. 2001) and in cell division (Fellner et al. 1996; David et al. 2007). Effendi et al. (2011) provided evidence that ABP1 is involved in regulation of polar auxin transport thus affecting local auxin concentration and early auxin gene

regulation (Braun et al. 2008). Recently, the role of ABP1 was extensively reviewed (e.g. Tromas et al. 2010; Sauer and Kleine-Vehn 2011; Scherer 2011). Differently from *Arabidopsis*, where only one homolog of ABP1 is present, at least five ABPs, including ABP1, have been identified in maize so far (Schwob et al. 1993), but their roles in growth and development have yet to be elucidated.

Light as an external factor regulates plant growth in the complex interaction with internal factors including auxins. The mechanisms how auxins can be involved in light-induced growth inhibition is not yet fully understood. Various studies have shown a correlation between light responses and auxin levels or polar auxin transport (reviewed in Tian and Reed 2001; Liu et al. 2011), and a number of reports demonstrated the existence of signalling elements shared by light and auxin (reviewed in Halliday et al. 2009; Li et al. 2012) One of the hypotheses how light via phytochromes mediates the decrease in growth involves ABP1 (Walton and Ray 1981; Jones et al. 1989). This idea was supported by the observation that red light (RL) reduces the abundance of ABP1 (Jones and Venis 1989; Jones et al. 1991). Interestingly, the expression of another member of the ABP family, *ABP4* in maize mesocotyls was up-regulated in RL- and FR-grown seedlings (Fellner et al. 2006).

In *Arabidopsis*, the phytochrome family consists of five genes (*PHYA*, *PHYB*, *PHYC*, *PHYD* and *PHYE*) (Sharrock and Quail 1989; for review see Franklin and Quail 2000). In maize, the gene family consists of six phytochromes: *PHYA1*, *PHYA2*, *PHYB1*, *PHYB2*, *PHYC1* and *PHYC2* (Gaut and Doebley 1997; Sheehan et al. 2004; Sawers et al. 2005). Sheehan et al. (2004), the authors showed that all six phytochrome genes are transcribed in several seedling tissues, while the expression of *PHYA1*, *PHYB1*, and *PHYC1* predominate in all seedling tissues examined. The authors also showed that etiolated seedlings express higher levels of *PHYA* and *PHYB* than plants developing in light, whereas the expression of *PHYC* was not affected by light.

We previously reported that seedlings of modern maize hybrid 3394 with defect in *ABP4* gene expression show changes in its growth responses to auxin and light (Fellner et al. 2006). To obtain more information about the interaction of auxin and light signalling, we investigated the expression of phytochrome genes *PHYB* and *PHYA* in loss-of-function mutants in *ABP1* and *ABP4* genes in maize (*Zea mays* L.). We studied how the expression of the *PHYB* and *PHYA* in mesocotyls is regulated by white light (WL), and whether the artificial auxin NAA influences the expression of the phytochrome genes in darkness. Our results here support the existence of cross-talk between auxin and phytochrome signalling and indicate for the first time the involvement of ABP1 and ABP4 in phytochrome signalling pathways.

Materials and methods

The loss-of-function mutants in *ABP1* and *ABP4* genes in maize (*Zea mays* L.) were used for all experiments (Im et al. 2000). The *abp* mutants contained the Robertson's *Mutator* transposable elements (Bennetzen 1996) in *ABP1* and/or *ABP4* genes. Seeds of single mutants *abp1* (B2 allele) and *abp4* (B2/K1 allele), double mutants *abp1abp4* (B2/K1 allele) and a near isogenic line (here called WT) were a gift from Alan M. Jones (The University of North Carolina, Chapel Hill, NC). All *abp* mutants were tested for the lack of *ABP1* and/or *ABP4* gene expression, and they showed stable phenotypes. The fytochromobilin-deficient mutant *elm1* (*elongated mesocotyl 1*) was initially identified in the W22 background (Sawers et al. 2002) and was also introgressed into the B73 background (inbred maize line) by backcrossing five times (Dubois et al. 2010). Kernels of *elm1* and B73 were a gift from Thomas P. Brutnell from Boyce Thompson Institute for Plant Research, Ithaca, NY.

For experiments in vitro, seeds were surface sterilized (70 % ethanol for 3 min, soaked in 5 % sodium hypochlorite, and rinsed with sterile distilled water). The seeds germinated on a 0.7 % (w/v) agar medium in Magenta GA7 boxes (77 × 77 × 196 mm; Sigma-Aldrich, Prague, Czech Republic) (9 seeds per box). The basal medium (BM) contained Murashige and Skoog salts (MS medium; Sigma-Aldrich, Prague, Czech Republic) (Murashige and Skoog 1962), 1 % (w/v) sucrose and 1 mM MES (2-(N-morpholino)-ethanesulfonic acid; pH adjusted to 6.1 before autoclaving). In experiments with auxin, the BM was supplemented with 1-Naphthaleneacetic acid (NAA) in various concentrations. Seeds in the Magenta boxes were placed in a growth chamber (Percival PGC-10, IA, USA) and incubated in the dark or white light (WL; Philips PL-L-40 W/840/4P, USA; total photon fluence rate 150 μmol m^{-2} s^{-1}). For the development of etiolated seedlings, the boxes were wrapped in aluminium foil. In all light conditions, the seeds were incubated for 7 days at a temperature of 23 °C. The fluence rate was measured with a portable spectroradiometer (model LI-1800; Li-Cor; Lincoln, NE) calibrated by the Department of Biophysics at Palacky University in Olomouc. The mesocotyl length (from the scutellar to the coleoptilar node) was measured with a ruler to the nearest millimeter. Mesocotyl in 6–9 seedlings per treatment in each independent experiment was measured. Changes in mesocotyl growth (i.e. inhibition or stimulation) caused by auxin were expressed in percents based on the following formula: $X = 100 \times (A - B)/A$, where "X" is the change in growth (in %), "A" and "B" stand for growth (in mm) in the absence and presence, respectively, of auxin.

For gene expression experiments, total RNA was extracted from the mesocotyl of 7-day-old plants using an

RNeasy Plant Mini RNA kit (Qiagen Inc., USA, Valencia, CA) according to the manufacturer's instructions. After RNA extraction was performed, traces of genomic DNA from a batch of RNA was removed by a DNaseI treatment using RQ1 RNA-free DNase (Promega, USA) for 40 min at 37 °C in a water-bath. RNA was then mixed with phenol:chloroform:isoamylalcohol (25:24:1) vortexed and spun to allow the two phases to form. Afterwards, supernatant was transferred into a new tube and the volume completed with 20 μL of 1 M acetic acid and 550 μL of 96 % ethanol. The mixture was incubated overnight and then washed with ethanol. RNA quantity and quality was then measured by a spectrophotometer Smart Spec Plus 2000 (BioRad, Czech Republic).

A reverse transcription reaction was performed with 1 μg of total RNA by adding 4 μL of 5 × FS (First Strain) buffer, 1 μL of 10 mM dNTP, 1 μL of 0.1 M DTT, 1 μL of 50 μM oligo(dT)20 primer, 0.5 μL of RNaseOUT (InVitrogen, Carlsbad, CA, USA) and 1 μL of SuperScript III Reverse Transcriptase (InVitrogen Co., Carlsbad, CA, USA). The volume was filled up to 20 μL with RNase-free water. The reaction mixture was incubated in a thermocycler (MJ Mini Gradient Thermal Cycler, BioRad, Czech Republic) programmed for 50 °C for 60 min followed by 85 °C for 5 min and ended by cooling for 15 min. The cDNA product was directly used in PCR (Polymerase Chain Reaction). PCR amplification was performed in a mixture containing: 4 μL of 5X GoTaq polymerase buffer, 2 μL of 1 mM dNTP, 1 μL of 10 mM of each gene-specific primer, 1 μL of GoTaq polymerase (Promega, USA) and 1 μL of diluted cDNA. The *PHYA* (accession no. AT 1G09570) and *PHYB* (accession no. AT2G18790) gene specific primers (Table 1) were used and PCR reactions of 24 cycles were performed as described in Sheehan et al. (2004) and adjusted in initial experiments. Number of cycles for expression of *18S rRNA* was also carefully adjusted to detect possible differences in expression between genotypes and conditions. The template cDNAs were denatured at 95 °C for 15 min followed by cycles of 94 °C for 30 s, 55 °C for 30 s, and 72 °C for 1 min. Final elongation was performed at 72 °C for 10 min followed by cooling at 4 °C. The *18S rRNA* gene (accession no. AF168884) of maize was used as reference gene and amplified using the specific primers (Table 1). The template cDNAs were denatured at 94 °C for 3 min followed by cycles of 94 °C for 30 s, 55 °C for 45 s, and a 30 s extension at 72 °C for 22 cycles. The final extension was performed at 72 °C for 5 min followed by cooling at 15 °C. PCR products were size fractionated by electrophoresis in a 1 % (w/v) agarose gel stained with ethidium bromide. Detected bands were evaluated using the software package ImageJ to obtain semi-quantitative data on relative gene expression. The numbers on axis "y" in the expression graphs are number of pixels of band signals. When necessary, the statistical significance of the treatment differences was assessed using Student's *t* test.

Table 1 List of gene-specific primers used for semi-quantitative RT-PCR

Primer	Sequence
PHYA—F	5′-GAG AGA TCC ATG AAG CAA AAG GTT TAC-3′
PHYA—R	5′-GAA GGT TGA CAT GCC CAG CTT CCC TGA G-3′
PHYB—F	5′-GTT TTG GCT GAC TTC GCT AAG CAT G-3′
PHYB—R	5′-GGA CGA TGA GGA AGA AAC TCC GCT CTG-3′
18S rRNA—F	5′-ACG AAC AAC TGC GAA AGC-3′
18S rRNA—R	5′-CGG CAT CGT TTA TGG TTG-3′

Results and discussion

We previously reported that in the regulation of growth and development of young maize seedlings auxin interacts with light, and we hypothesized that *ABP4* plays an important role in this cross-talk (Fellner et al. 2006). To get insight into the interaction, we investigated whether the knockout of *ABP1* and/or *ABP4* affects expression of genes coding for phytochrome B (*PHYB*) and phytochrome A (*PHYA*) in maize mesocotyls developed in darkness or in white light (WL).

In maize seedlings with knockout *ABP1* or *ABP4* gene grown in darkness, the expression of *PHYB* was significantly lower than in mesocotyls of WT plants. Interestingly, when both genes were off in the double mutant *abp1/abp4*, expression of *PHYB* was similar to that observed in WT plants (Fig. 1). The results indicate that in darkness, ABP1 and ABP4 positively influence *PHYB* expression, and that ABP1 and ABP4 functionally depend on each other. Alternatively, it is possible that the knockout of *ABP1* and *ABP4* trigger alternative pathway(s) leading to the normal expression of *PHYB*. In our experimental conditions, WL strongly reduced level of *PHYB* transcript in WT plants, whereas it had not any essential effect on the level of *PHYB* transcript in single and double mutants (Fig. 1). Namely, the knockout of *ABP1* and/or *ABP4* gene led to the similar expression of *PHYB* in dark- and WL-developed mesocotyls. However, WL reduced the expression of *PHYB* in WT mesocotyl to the level similar to that in *abp* mutants. It is therefore possible that in the dark, ABPs do not stimulate *PHYB* expression directly but rather through positive regulation of some transcription factor,

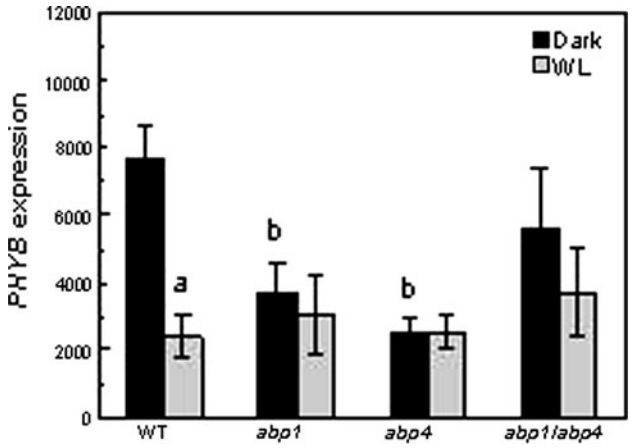

Fig. 1 Expression of *PHYB* gene in mesocotyls of etiolated or white light-grown WT and *abp* mutants. WT and mutant plants grew in conditions in vitro on the BM. *PHYB* expression analysis was performed by semi-quantitative RT-PCR, and *18S rRNA* was used as a reference gene. In each genotype and each condition, the data represent average from 10 identical independent experiments ±SE. In each experiment, in each condition and in each genotype, a mix of 20 mesocotyls was always used. **a** significantly different ($P < 0.05$) from the dark in each genotype; **b** significantly different ($P < 0.05$) from WT in the dark

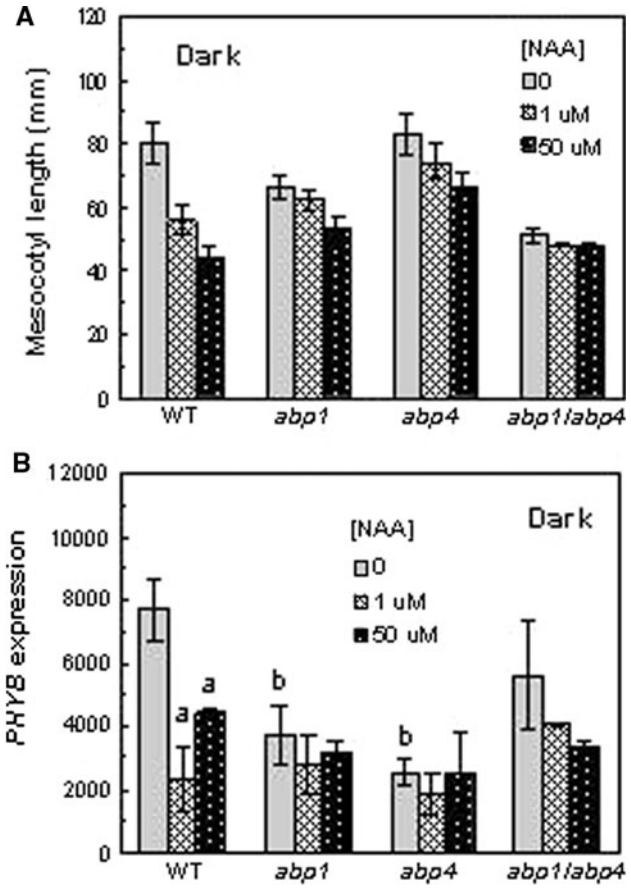

Fig. 2 Responsiveness of mesocotyls in etiolated WT and *abp* mutants to exogenous auxin in regards to elongation and *PHYB* gene expression. **a** Comparison of mesocotyl growth in etiolated single mutants *abp1*, *abp4*, double mutant *abp1abp4*, and corresponding WT, in response to NAA. Mesocotyl elongation was measured with a ruler to the nearest millimeter in 7-day-old seedlings grown in Magenta boxes in darkness, on the BM supplemented with 10^{-6} or 5×10^{-5} mol L^{-1} NAA. The results are the mean length ±SE obtained from 5 to 12 independent experiments. In each experiments, each genotype and in each condition, six to nine seedlings were measured. **b** Expression of *PHYB* in mesocotyls of WT and *abp* mutants developed in the dark as a function of exogenous auxin. WT and *abp* mutant plants grew in conditions in vitro on the BM supplemented with NAA at concentrations of 10^{-6} or 5×10^{-5} mol L^{-1}. *PHYB* expression analysis was performed by semi-quantitative RT-PCR, and *18S* rRNA as a reference gene was used. In each genotype and each condition, the data represent average from 10 identical independent experiments ±SE. In each experiment, in each condition and in each genotype, a mix of 20 mesocotyls was always used. **a** significantly different ($P < 0.05$) from corresponding control (absence of NAA); **b** significantly different ($P < 0.05$) from WT in the absence of NAA

which could be however blocked (destabilized) in WL conditions. The results on *PHYB* expression in WT developed under WL are not consistent with the results reported by Sheehan et al. (2004). The authors showed that dark- and WL-grown mesocotyls accumulates *PHYB* at similar levels. The discrepancy could reflect the differences of WL sources used by us and Sheehan et al. (2004), and/or that differently from the authors we used very young plants developed in conditions in vitro. On the other hand, the authors showed tissue-specific accumulation of *PHYB* is light dependent. Neither in the dark nor in WL, levels of *PHYB* transcript in WT and *abp* mutants correlate with mesocotyl length. However, this conclusion is obvious also from results of Sheehan et al. (2004).

It was reported that ABP1 in maize binds not only native auxin IAA but also artificial auxin NAA (Ray and Dohrmann 1977; Dahlke et al. 2009). Here we found that intact etiolated maize seedlings with knockout *ABP1* and/or *ABP4* are much less sensitive to the inhibitory effect of NAA than plants with the functional ABPs (Fig. 2a). We further revealed that the level of *PHYB* transcript was significantly reduced by NAA in etiolated WT mesocotyls but not in mesocotyls of the knockout plants (Fig. 2b). The data suggest that in etiolated mesocotyls, functional ABP1 and ABP4 are required for NAA-induced inhibition of mesocotyl elongation and NAA-induced inhibition of *PHYB* expression. As evident, the lack of NAA-induced inhibition of *PHYB* expression in the *abp* mutants correlates with the lack of the mutant sensitivity to exogenous auxin in elongation response. To support the existence of the cross-talk between *PHYB*- and *ABP*-mediated signalling pathway, we studied sensitivity to exogenous auxin of *elm1* (*elongated mesocotyl 1*) mutant deficient in fytochromobilin (Dubois et al. 2010). In agreement with our hypothesis, etiolated *elm1* mesocotyls showed distinctly less sensitivity to NAA than corresponding WT (Fig. 3).

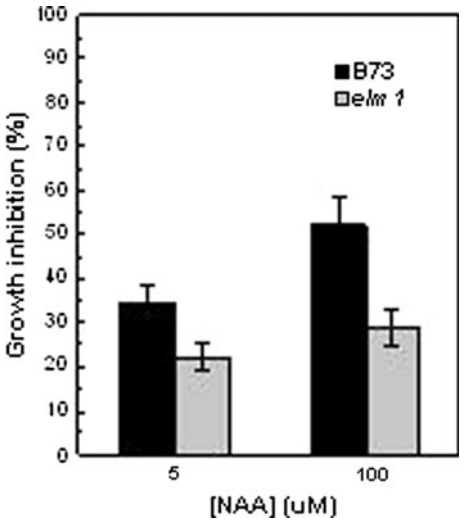

Fig. 3 Responsiveness of mesocotyls in intact etiolated *elm1* mutant to exogenous auxin. Growth responses of maize *elm1* mutant and corresponding WT (B73) cultured in the dark on the BM supplemented with NAA at concentrations of 5×10^{-6} or 10^{-4} mol L^{-1}. The values are the mean length ±SE from six independent experiments, with 6–9 plants measured in each experiment. Auxin-induced inhibition of mesocotyl growth (in %) was calculated as described in the "Materials and Methods"

In WL conditions, auxin had slight and very variable effects on *PHYB* expression in all genotypes tested. Although for each condition and each genotype, ten independent experiments were conducted, the obtained results were inconclusive (data not shown).

In comparison with experiments on *PHYB*, expression of *PHYA* was much variable. In contrast to *PHYB*, the level of *PHYA* transcript in etiolated maize mesocotyls was not affected by the loss of function in *ABP1* nor *ABP4* genes, suggesting that functional ABP1 or ABP4 do not play a role in regulation of *PHYA* expression. *PHYA* expression was not essentially influenced by WL in either genotype tested (Fig. 4a). It is unusual as PhyA belongs to light labile phytochromes (Sharrock and Quail 1989). Light-dependent decrease in *PHYA* transcript has been observed in several grass species (Cobert et al. 1989) and the same trend was observed in maize (Sheehan et al. 2004, and references therein). Here we found no reduction of the *PHYA* level by WL in maize mesocotyls. It could be explained by the fact that differently from Sheehan et al. (2004) our source of WL was not supplemented by incandescent lamps and has total photon irradiance 150 µmol m^{-2} s^{-1}. Interestingly, Franklin et al. (2007) reported that under high photon irradiances of RL (>100 µmol m^{-2} s^{-1}) degradation of a pool of nuclear-localized phyA was retarded. The authors demonstrated photoprotection of phyA at high photon irradiances of RL and provided evidence of significant functional activity for photoprotected phyA.

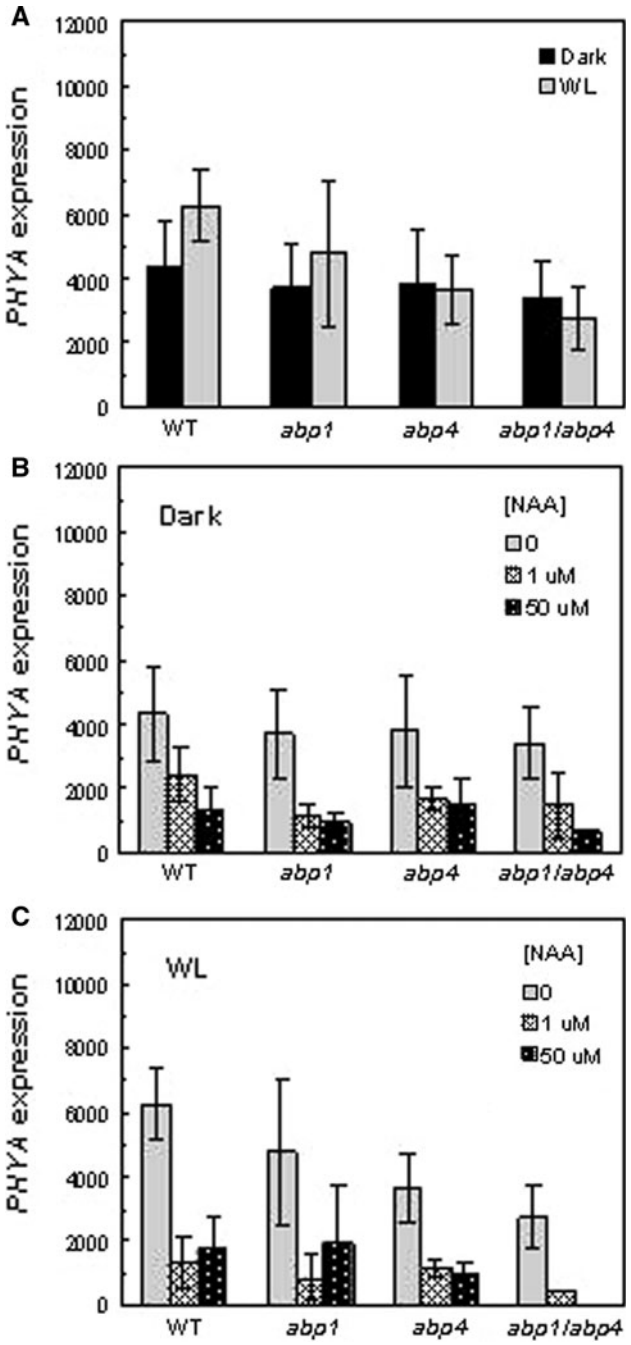

Fig. 4 Expression of *PHYA* gene in mesocotyls of etiolated or white light-grown WT and *abp* mutants as a function of exogenous auxin **a** Expression of *PHYA* in mesocotyls of WT and *abp* mutants developed in the dark or white light (WL). WT and mutant plants were grown in conditions in vitro on the BM. **b** Expression of *PHYA* in mesocotyls of WT and *abp* mutants developed in darkness or **c** in WL as a function of exogenous auxin. WT and mutant plants were grown in conditions in vitro on the BM supplemented with NAA at concentrations of 10^{-6} or 5×10^{-5} mol L^{-1}. *PHYA* expression analysis was performed by semi-quantitative RT-PCR, and *18S rRNA* was used as a reference gene. In each genotype and each condition, the data represent average from 10 identical independent experiments ±SE. In each experiment, in each condition and in each genotype, a mix of 20 mesocotyls was always used

Albeit the variability in *PHYA* expression, distinct trend of auxin-induced reduction of *PHYA* transcript was observed in etiolated as well as in WL-developed mesocotyls of all genotypes tested (Fig. 4b, c). Differently from *PHYB*, the inhibition of *PHYA* gene expression was however of similar trend in WT and the *abp* mutants. It therefore seems that ABP1 and ABP4 do not play a role in the auxin-mediated inhibition of *PHYA* expression. It supports the conclusion above that ABP1 and ABP4 are not involved in the expression of *PHYA*.

The possibility of the involvement of ABP in cross-talk between light and auxin signalling was brought out by Jones et al. (1991). The authors reported that RL reduces the abundance of the ABP1, which controls cell expansion in maize. In contrast, the expression of *ABP4* in maize mesocotyls was up-regulated in light grown seedlings (Fellner et al. 2006). Our results presented here show for the first time that knockout of *ABP1* and/or *ABP4* genes affects expression of *PHYB* in etiolated maize mesocotyls. We further report that etiolated mesocotyls of the *abp* mutants are insensitive to exogenous auxin NAA as regards to NAA-induced inhibitin of mesocotyl elongation and NAA-induced inhibition of *PHYB* expression. Similarly, reduction of sensitivity to exogenous auxin was observed in fytochromobilin-deficient mutant *elm1* (Fig. 3). Our results therefore indicate that in the dark, reduction of *PHYB* expression by exogenous auxin interferes with the signaling pathway involved in mesocotyl elongation. However, the molecular mechanism has to be elucidated.

Based on our results we hypothesize that in dark conditions, ABP1 and ABP4 activate a transcription factor, which trigger expression of *PHYB*, whereas in WL conditions, the TF could be blocked (destabilized) (Fig. 5). We further propose that after binding NAA, the ABPs lose their ability to activate the TF, which could come to the essentially reduced expression of *PHYB* gene. The fact that in the absence of exogenous auxin, ABP1 and/or ABP4 seems to play a positive role in *PHYB* expression (Fig. 1) may suggest a divergence of signaling pathways triggered by low (endogenous auxin) and high (endogenous plus exogenous auxin). Our results support the existence of cross-talk between auxin and light signaling and indicate for the first time that *ABP1*, *ABP4* and *PHYB* genes could share common signaling pathway(s).

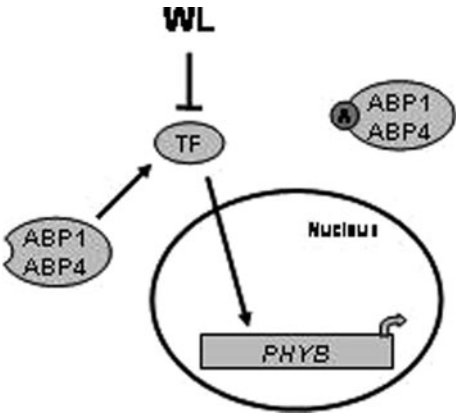

Fig. 5 Working model showing cross-talk between ABP and *PHYB* signaling pathways in etiolated or WL-grown maize mesocotyls in the absence or presence of exogenous auxin. The model shows that in dark conditions, ABP1 and ABP4 activate a transcription factor (*TF*), which trigger expression of *PHYB*, whereas in WL conditions, the TF could be blocked (destabilized). We further propose that after binding NAA (A), the ABPs lose their ability to activate the TF, which could come to the essentially reduced expression of *PHYB* gene. *Arrows* and *T-bars* represent positive and negative effects, respectively

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