New Auxin Binding Protein1 (ABP1) mutants show impairment of auxin-related functions and defect in red and far red light responses

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abp1 mutants have an auxin-insensitive phenotype

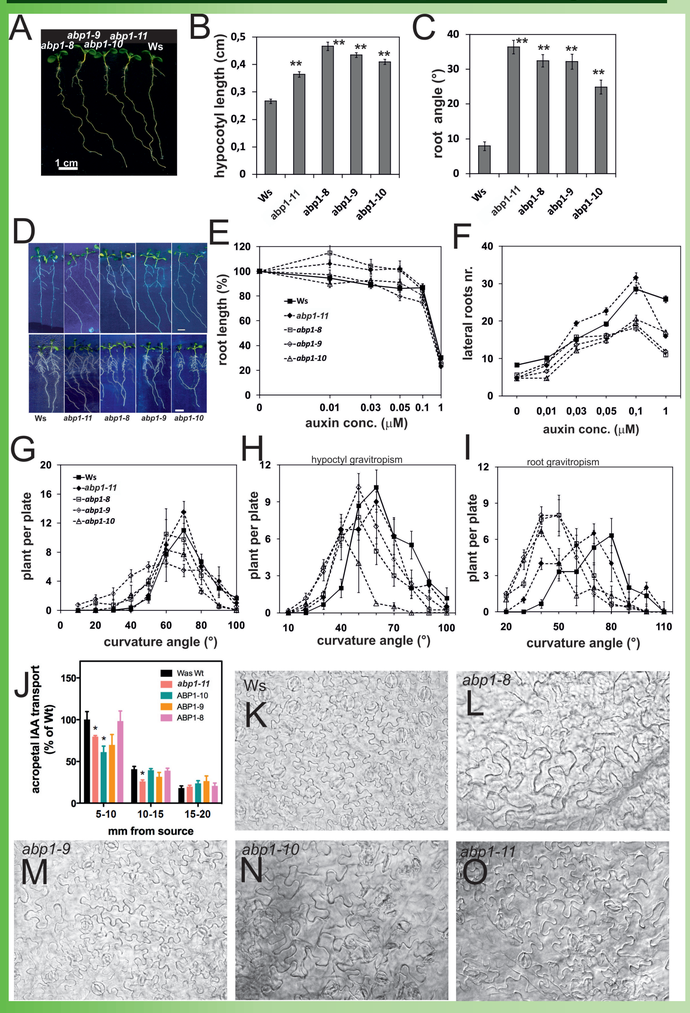

Fig. 1. Seedlings have an auxin-insensitive phenotype. (A,B) 7d old seedlings: tall hypocotyls in mutants. (C) Root slanting (D) Lateral root formation is auxin-insensitive (0µM & 0.1 µM IAA) (E, F) inhibition of main root and lateral root development. (G) Delayed phototropism (H, I) Delayed gravitropism. (J) Slower acropetal auxin transport in root. (K-O) Bigger cells and fewer lobes in epidermal cells

abp1 mutants delay up-regulation of expression of auxin-induced genes

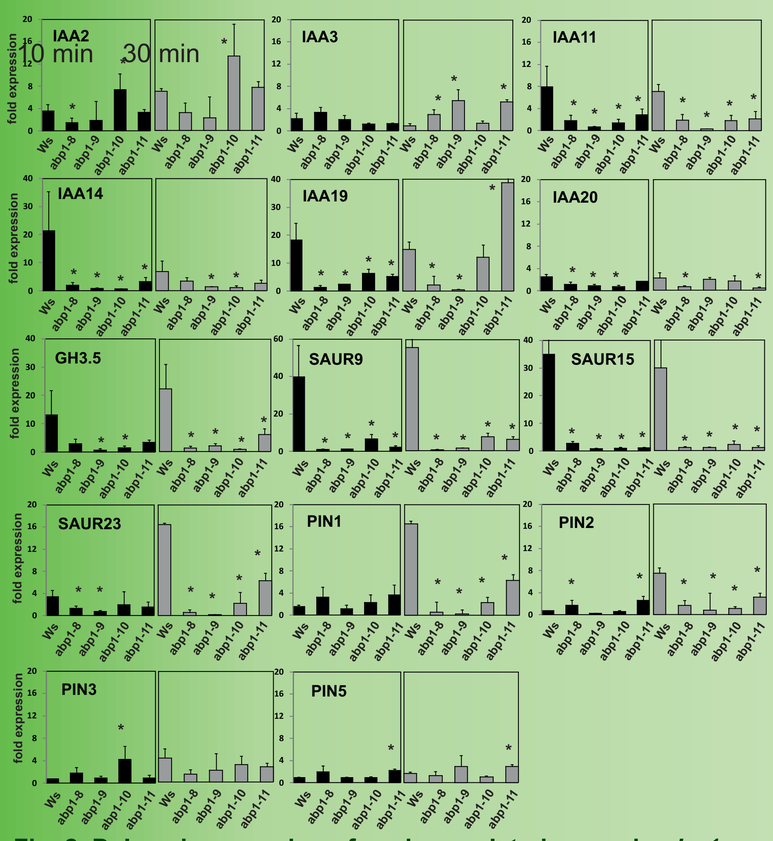

Fig. 2. Delayed expression of auxin-regulated genes in *abp1* mutants. Black bars: 10 min; gray bars: 30 min after auxin application. Expression of most genes is not properly up-regulated: The „10 min rule“ is kept.

Auxin signal transduction and the two receptor concept

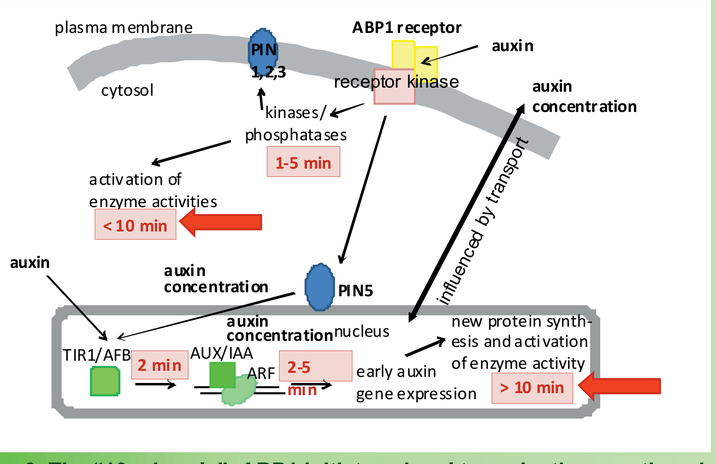

Fig. 3. The “10 min rule”: ABP1 initiates signal transduction reactions in the cytosol in less than 10 min. This needs a transmembrane protein which probably is a receptor kinase. One “end point” of regulation is the regulation of auxin transport and thus of cytosolic auxin concentration. This means that ABP1 is directing TIR1 activity so that changes of expression of early auxin genes can be measured within 10 min in *abp1* mutants (Fig. 2) and e.g. phospholipase A mutants (Labusch *et al.*, 2013). The *tir1* mutant mis-regulates only one of our test genes, *abp1* mutants about 80%.

ABSTRACT

Auxin Binding Protein1 (ABP1) has been proposed as membrane-bound auxin receptor in plants based on early studies. Recent progress in ABP1 research suggested that ABP1 could be auxin receptor for rapid auxin related processes. We designed and characterized four in-vitro abp1 mutants containing point mutation in the presumed auxin binding site of ABP1. The abp1 mutants showed defects in auxin-related functions such as in gravitropic and phototropic (root and shoot) responses, early flowering, insensitivity to auxin and reduced transcript levels of early auxin responsive genes (AUX/IAAs, GH3, SAURs) and auxin efflux transporter genes (PINs). Additionally, the abp1 mutants exhibited insensitivity of hypocotyl elongation inhibition to red and far-red light and showed hypersensitive hypocotyl elongation to shade light. qPCR data of shade-induced genes in response to FR-and R-enriched white light was altered in all abp1 mutants in compared to WT. This provides initial evidence of a regulatory link between auxin and phyB-mediated light responses via ABP1 action. Taken together, the new abp1 mutants showed mutant properties not only as auxin mutants but also as light mutants

abp1 mutants have a vegetative and early flowering phenotype

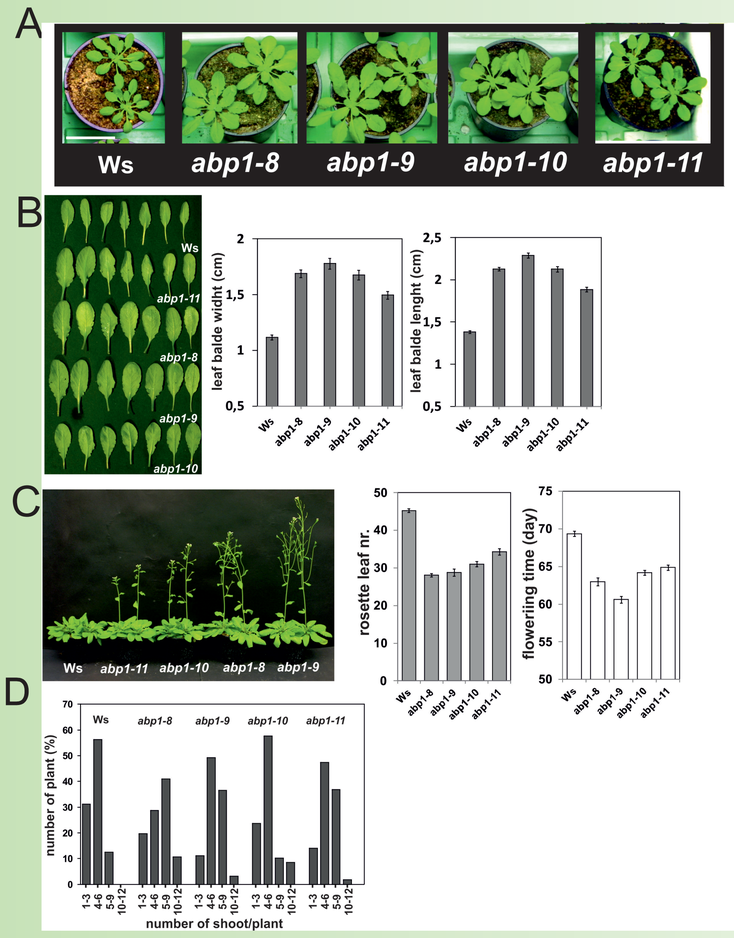

Fig. 4. (A, B) Rosette and leaf phenotype: similar to phyA plants. (C) *abp1* mutants flower early. (D) apical dominance is decreased.

Working model of the functional interaction of ABP1 and phytochrome B

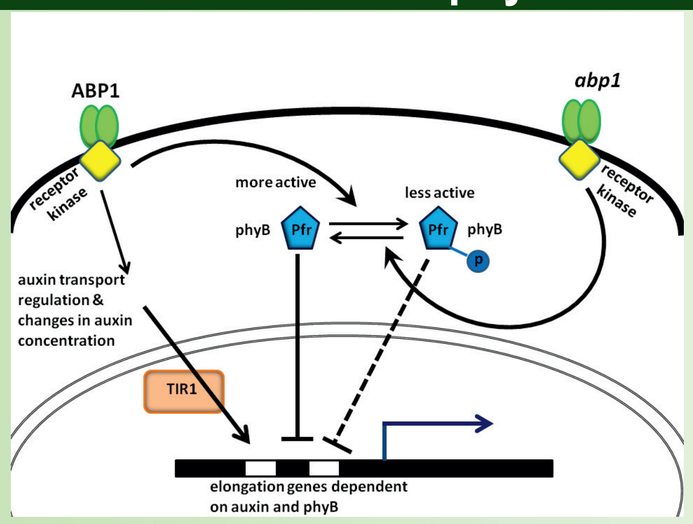

Fig.7. Model of functional interaction of ABP1 and phytochrome B. Phosphorylation of Ser-86 negatively regulates all physiological phyB responses including the response to shade. Faster dark reversion attenuates red light-induced nuclear import and interaction with the negative regulator PIF3 compared with the wild-type version phyBSer86-GFP (Medzihradsky *et al.*, 2013). It is suggested that ABP1 can influence this phosphorylation–dephosphorylation equilibrium towards the more active form. This more active form can still be inactivated by FR so that wt ABP1 plants show a small elongation to shade whereas the *abp1* mutants show a hyper-response.

abp1 mutants are hypersensitive to shade light and insensitive to R or FR

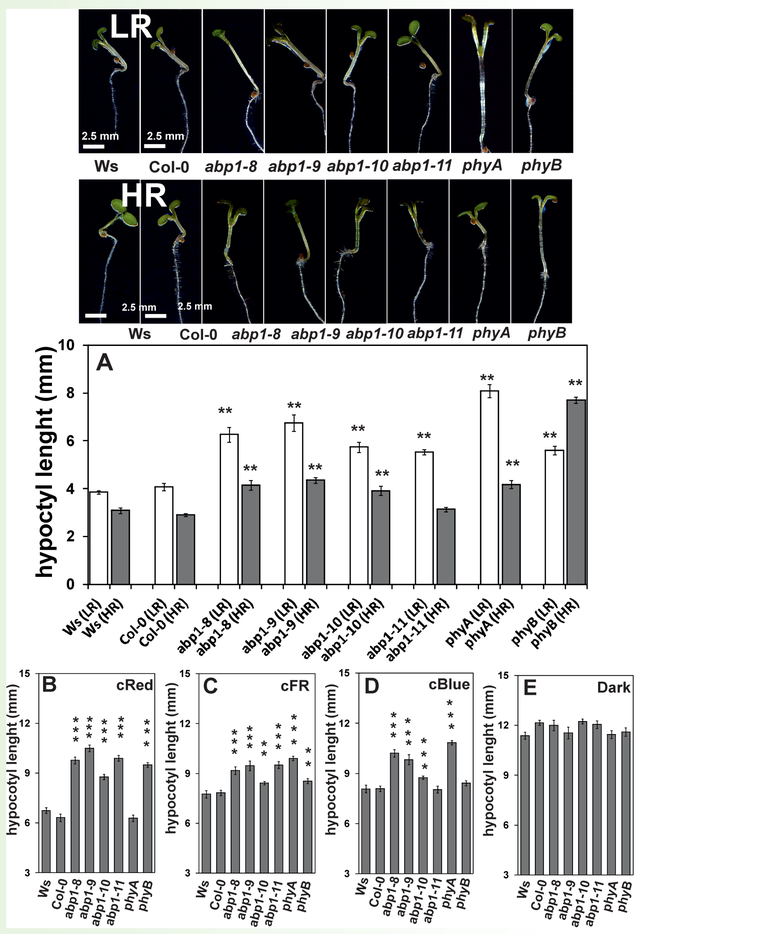

Fig. 5. (A) LR (gray bars): low ratio R/FR = far red rich white light; HR (white bars): high ratio R/FR red rich white light. (B-E) Seedlings were grown in continuous light (0.1 µmol m-2 s-1) for 4 days. *abp1* seedlings are blue, red- and far red-insensitive.

in *abp1* mutants regulation of shade-induced genes is aberrant

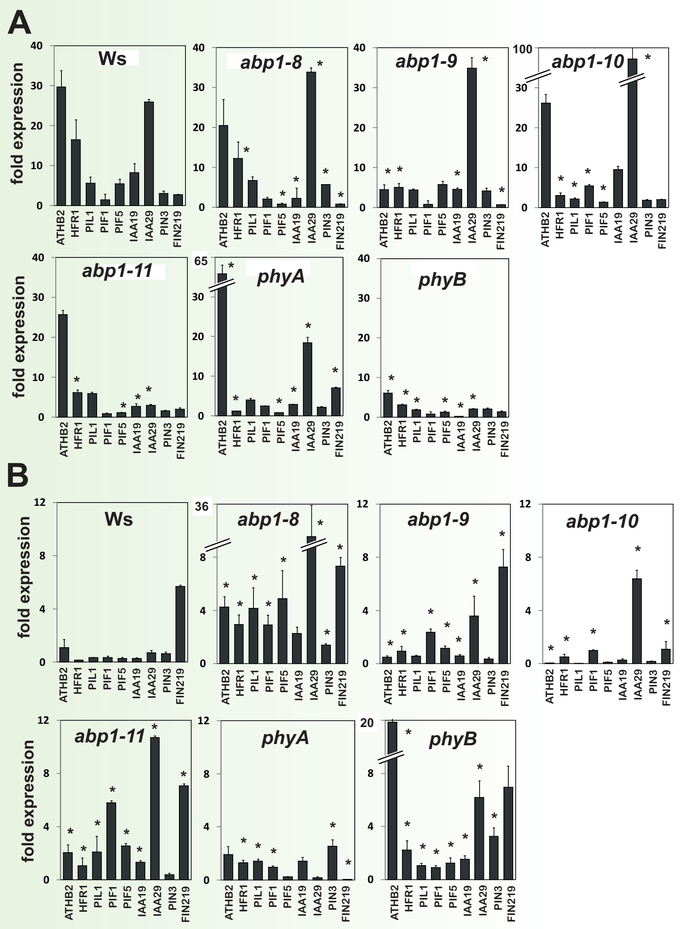

Fig.6. Gene induction after (A) 1h low ratio R/FR (far-red rich (B) 1h high ratio R/FR (red-rich) on a background of white light. Shade marker genes were selected from the literature. Shade induction of genes was low in phyB and strong in wt and ±lower in abp1 mutants. In high Red, shade genes were low and high in phyB and intermediate in abp1 mutants.

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