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Author: Lucas Vanhaelewyn Paolo Schumacher Dirk Poelman Christian Fankhauser Dominique Van Der Straeten Filip Vandenbussche

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***REPRESSOR OF ULTRAVIOLET-B PHOTOMORPHOGENESIS* function allows efficient phototropin mediated ultraviolet-B phototropism in etiolated seedlings**

Lucas Vanhaelewyn^a, Paolo Schumacher^b, Dirk Poelman^c, Christian Fankhauser^b, Dominique Van Der Straeten^a, Filip Vandenbussche^{a,1}

- a. Laboratory of Functional Plant Biology, Department of Physiology, Faculty of Sciences, Ghent University, KL Ledeganckstraat 35, B-9000 Gent, Belgium;
- b. Center for Integrative Genomics, Faculty of Biology and Medicine, Université de Lausanne, CH-1015 Lausanne, Switzerland.
- c. Lumilab, Department of Solid State Sciences, Faculty of Sciences, Ghent University, Krijgslaan 181, B-9000 Gent, Belgium.

¹To whom correspondence should be addressed. E-mail: Filip.Vandenbussche@ugent.be

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Running title: RUP function in phototropism

Highlights

- Phototropin independent phototropism occurs in UV-B and short wave UV-A light
- UVR8 does not enhance phototropin action
- The phototropin pathway is more sensitive to UV-B than the UVR8 pathway
- The mechanism of phototropin action in UV-B is similar to that in blue light
- RUP function is necessary for efficient phototropin mediated bending towards UV-B
- Defective UV-B phototropism in *rup1rup2* double mutants correlates with elongation

Abstract

Ultraviolet B (UV-B) light is a part of the solar radiation which has significant effects on plant morphology, even at low doses. In Arabidopsis, many of these morphological changes have been attributed to a specific UV-B receptor, UV resistance locus 8 (UVR8). Recent findings showed that next to phototropin regulated phototropism, UVR8 mediated signaling is able of inducing directional bending towards UV-B light in etiolated seedlings of Arabidopsis, in a phototropin independent manner. In this study, kinetic analysis of phototropic bending was used to evaluate the relative contribution of each of these pathways in UV-B mediated phototropism. Diminishing UV-B light intensity favors the importance of phototropins. Molecular and genetic analyses suggest that UV-B is capable of inducing phototropin signaling relying on phototropin kinase activity and regulation of NPH3. Moreover, enhanced UVR8 responses in the UV-B hypersensitive *rup1rup2* mutants interferes with the fast phototropin mediated phototropism. Together the data suggest that phototropins are the most important receptors for UV-B induced phototropism in etiolated seedlings, and a RUP mediated negative feedback pathway prevents UVR8 signaling to interfere with the phototropin dependent response.

1. Introduction

Although UV-B radiation is a minor fraction of the sunlight which is reaching Earth's surface, it causes significant biological effects in plants. These effects can be classified as damaging or regulatory, often influencing plant morphology [1]. UV-B induced morphological effects include curling, glazing and bronzing of the leaf, reducing the leaf area and increasing its thickness. Furthermore, elongation of the petiole and stems are inhibited, axillary branching is increased and shoot-root ratios differ compared with the control [2]. In the model dicot plant *Arabidopsis*, UV-B counteracts the shade avoidance response, causing inhibition of elongation and cell expansion, yielding compact plants [3, 4]. In addition, UV-B regulates differential growth in seedlings and rosettes [5, 6].

Phototropism is the most studied light controlled differential growth response. It is defined as the directional growth of plants towards light in order to optimize the position of photosynthetic tissues [7]. The main photoreceptors that regulate phototropism are the phototropins [8]. They allow perception of blue and UV-A light with a flavin mononucleotide (FMN) as chromophore [9]. *Arabidopsis* has two phototropins, phototropin1 (PHOT1) and phototropin2 (PHOT2); these are membrane-associated photoreceptors which trigger a downstream signal transduction cascade [10]. This signaling mechanism relies on the kinase activity of the phototropin receptor, and eventually results in the dephosphorylation of NONPHOTOTROPIC HYPOCOTYL3 (NPH3) [11, 12]. The dephosphorylated NPH3 interacts with PHOT1, making the latter a target for degradation by the 26S proteasome. This NPH3 mediated degradation is essential for phototropic bending towards blue light [13]. Recently, we have shown that UV-B light induced phototropism relies on two distinct photoreceptor systems. On the one hand, phototropins are involved, yet the exact mechanism remains elusive, while on the other hand the specific UV-B photoreceptor UVR8 can induce phototropic bending in the absence of phototropins [5].

UVR8 was originally discovered in a genetic screen for mutants which are hypersensitive to UV-B [14]. Later, UVR8 was identified as a specific UV-B receptor [15]. UVR8 is a 440 amino acid, seven-bladed β -propeller protein that forms homodimers, which instantly monomerize upon UV-B absorption. UV-B absorbance by Trp residues in the UVR8 protein leads to an excited state of UVR8, disrupting salt bridges at the dimer interface, thus initiating monomerization [16, 17]. Nuclear UVR8 accumulation alone is insufficient for downward signaling; supplementary UV-B is required to stimulate UVR8 monomerization and further signaling [18]. A suggested mode of action of UVR8 is through direct binding of chromatin [19], hence controlling expression of a range of downstream genes, including the transcription factor ELONGATED HYPOCOTYL5 (HY5), this mechanism is however currently under debate [20]. At the protein level, the mechanism of UVR8 signaling involves interaction with

CONSTITUTIVELY PHOTOMORPHOGENIC 1 (COP1). COP1 is part of an E3 ubiquitin ligase which enables selective protein ubiquitination and degradation [21]. COP1 regulates light proteins such as ELONGATED HYPOCOTYL5 (HY5) [22, 23]. The (inactive) homodimeric ground state of UVR8 is restored through redimerization of the UVR8 monomers, thus terminating UV-B-mediated signaling [24]. This process usually occurs within 1 hour following the UV-B exposure and is stirred by the negative feedback regulatory proteins REPRESSOR OF UV-B PHOTOMORPHOGENESIS 1 and 2 (RUP1, RUP2) [16, 24, 25]. The RUPs are transcriptionally activated by UV-B in an UVR8-, COP1- and HY5-dependent fashion, then they interact directly with UVR8, causing the repressor function that serves as “brake” in the UV-B-specific signalling [26]. RUP1 and RUP2 are considered largely redundant for their physiological effects on plants [24, 26]. Nevertheless, single mutants of *rup1* do not have any reported phenotype, while mutants of *rup2* appear slightly hypersensitive to UV-B radiation [26].

Recent findings showed that both phototropin and UVR8 mediated signaling each by themselves are capable of inducing directional growth towards UV-B light in etiolated seedlings of Arabidopsis [5]. However, up until now, the relative contribution of each of these pathways to UV-B mediated phototropism remain unclear. Here we study whether the two types of photoreceptor pathways always occur jointly. By diminishing light intensity or monitoring enhanced UVR8 response using the UV-B hypersensitive *rup1rup2* mutants, we clarify the contribution of the UVR8 pathway in phototropism in etiolated seedlings.

2. Methods

2.1. Plant materials

Col-0 was from NASC, Nottingham, UK. *Hy5-215* was a kind gift from Prof X.W. Deng (Yale University). *Rup1rup2*, *rup1rup2uvr8-6*, *rup1rup2hy5-215* and *uvr8-6* [26] were a kind gift from Prof. Roman Ulm (University of Geneva). *Nph3-6* was from Prof. E. Liscum (University of Missouri) [27]. *Phot1phot2amiRUVR8* was as described [5]. The kinase dead phototropin 1 lines *D806N-2* and *D806N-19* were a kind gift from Prof. K. Shimazaki (Kyushu University) [28].

2.2. Light sources

The 302nm light source was as described [5], consisting of a UVM57 lamp (UVP) and a NS297 band pass filter (International Light Technologies). Blue light was given using 470nm LEDs [29]. Monochromatic light was generated by a Xenon short arc lamp (Bausch and Lomb Optical Co, Toronto, Canada) combined with a Zeiss monochromator (Zeiss, Germany), with slit width 0.8 mm. The desired fluence rate was obtained by altering the distance between the plants and the light source.

2.3. Kinetics of bending

All seeds were sown on Jiffy peat pellets (Jiffy Stange, Norway) moistened with tap water, and kept for two days at 4°C in darkness. Subsequently, they were exposed to white light for 6h to stimulate germination. The seeds were left to germinate in darkness and seedlings were allowed to grow for 2 days at 21°C. After two days, the seedlings were transferred to a growth chamber with infrared (930nm) light and 21°C temperature. They were photographed every 5 minutes using a webcam based time lapse photography set-up [30, 31]. The focal plane of the pictures was parallel with the unilateral incoming light. Using the angle measurement tool of ImageJ (NIH, USA), the photographs were digitally analyzed. The angle of curvature is defined as the angle between the vertical and the upper end of the hypocotyl, and can be seen as deviation from the vertical, with 0° being vertical, 90° being horizontal and directed towards the light source and -90° being horizontal and directed away from the light source. Hypocotyl length measurements were done with ImageJ (NIH, USA).

2.4. Quantitative RT-PCR analysis

Seeds were sown on Jiffy peat pellets (Jiffy, Stange, Norway) and kept for 2 days at 4°C. Subsequently, they were exposed to 6h of white light at room temperature (RT) for germination. Seedlings were grown for 2 days in darkness and then exposed to UV-B light for varying duration. The shoot part of the seedlings was harvested for analysis. RNA was prepared using Qiagen RNeasy Plant Mini kits (Qiagen, Venlo, the Netherlands). Reverse transcription was performed with a cDNA Verso kit (Thermo Scientific, Erembodegem, Belgium). Quantitative PCR was done with KAPA SYBR FAST qPCR Kit (KAPA Biosystems, Boston, Massachusetts, USA) in the iCycler (Bio-Rad) as a thermal cycler and IQTM 5 (Bio-Rad) as optical module for multicolor real-time quantitative PCR detection. Primers for amplification of a RUP1 fragment were RUP1 qRTPCR-1 5'-CGGTCGGGTTATCGGGTCAG-3', RUP1 qRTPCR-2 5'-GAGCCATTGTAAAGCGTGTAGTCC-3' and for RUP2, RUP2 qRTPCR-1 5'-AGCAGCAACAAGAACAAGCACAAAC-3' and RUP2 qRTPCR-2 5'-CGGAGGAGGAGGAGGAAGATACG-3'. Control transcripts used for normalization were *EIF4a*, using primers EIF4a fwd 5' - CTCATCACCCTGACCTCTTAGC-3' and EIF4a rev 5' -AACCTTCCACTTCTCCGATAC-3' , and *EF1α* (AT5G60390) with primers TGAGCACGCTCTTCTTGCTTCA and GGTGGTGGCATCCATCTTGTACA. Data analysis was performed with QBase software [32].

2.5. Western blot

One hundred seedlings were grown in darkness for two days and then kept in darkness or unilaterally illuminated for 30 minutes with UV-B (0.002 $\mu\text{mol}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$) or blue light (10 $\mu\text{mol}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$) and immediately frozen in liquid nitrogen. Protein extraction was done in boiling Laemmli buffer. Antibodies are kind gifts from Emanuel Liscum (University of Missouri) for NPH3 and Karen

Schumacher (University of Heidelberg) for DET3. For NPH3 hybridization, the membranes were blocked for 1h at RT with 5% powder milk (Dietisa) in PBS-Tween. Membranes were then hybridized with NPH3 antibody in a 1:3000 solution of milk 5% PBS-Tween over night at 4°C. Afterwards they were quickly rinsed 3 times with PBS-Tween and washed 3 times for 15 minutes with PBS-Tween at RT. Subsequently, the secondary antibody was applied: anti-rabbit antibody (Promega) in a 1:10000 solution of 5% milk PBS-Tween for 1h at RT. Membranes were then quickly rinsed 3 times with PBS Tween and washed 3 times for 15 minutes with PBS Tween at RT. Tween was then washed away by rinsing 3-4 times with PBS. Detection was done by using Immobilon Western Chemiluminescent horse radish peroxidase substrate in an ImageQuant LAS 4000 mini detection machine.

3. Results

3.1. Kinetic analysis of UV-B and UVR8 controlled bending in etiolated seedlings

Recently it was shown that both phototropins and UVR8 can regulate bending towards UV-B [5], yet the dynamics of the response have not been studied in detail. To better understand the action of the photoreceptor systems involved in UV-B induced directional bending, a kinetic analysis of the bending angle was performed on etiolated seedlings exposed to unilateral UV-B light (302nm) at $0.12 \mu\text{mol}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$, a fluence rate previously shown sufficient to induce UVR8 dependent gene expression to UV-B, indicating that UVR8 signaling is activated in these conditions [33]. Wild type plants started orienting to the light within an hour after onset of exposure. As shown previously [5], the response of the *phot1phot2* double mutant (with normal UVR8 signaling) was delayed, it only started around 2h of exposure and reached a maximum after 8h (Fig. 1A). Up until now, it remains unknown at which wavelength range this response occurs. Using the kinetic analysis assay, the wavelength dependence of phototropin independent bending was determined for wavelengths in the UV-region and included in the solar radiation reaching the surface of the earth (295nm and longer) by means of a monochromator. The time point of 5h after onset of illumination was chosen to generate a response spectrum of bending in *phot1phot2* background. The response was clearly visible between 295 and 315nm, was reduced between 315 and 335nm and absent at wavelengths longer than 335nm (Fig. 1B). This shows that this phototropic response is particularly strong in UV-B light, yet also noticeable in short wave UV-A (below 340nm). Further detailed kinetic analysis of *UVR8* defective plants showed that severely reducing the levels of UVR8 protein by introducing an artificial micro-RNA construct [5] in *phot1phot2* background virtually abolished the response (Fig. 1A). By contrast, *uvr8-6* single knockout mutants [34] did not show any difference with the wild type in their kinetics of bending at a fluence rate of $0.12 \mu\text{mol}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$. Together the data suggest that the phototropin pathway masks the UVR8 effect by acting temporally before the UVR8 pathway, and that the UVR8 pathway is responsible for the phototropin independent bending.

3.2. Diminishing UV-B light favors the phototropin pathway

In the above mentioned conditions, it appears that phototropins are predominant in regulating the response, masking the UVR8 effect in wild type plants. However, since UVR8 is the only UV-B specific photoreceptor described to date, it may modulate the response depending on the strength of the signal, which in wild type plants is determined by the amount of UV-B light. To evaluate the relative contribution of either of the two photoreceptor systems in the phototropic response to UV-B, wild type, *phot1phot2* and *rup1rup2* (with enhanced UVR8 signaling) were exposed to UV-B light (302nm,) of lower fluence rates than used for the data in Fig. 1A. Kinetic analysis of the change in bending angle shows that at low fluence rates ($0.002 \mu\text{mol}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$), wild type plants bend towards the light with a similar velocity as in higher fluence rates, yet the *phot1phot2* mutant plants do not respond by unidirectional bending (compare Fig. 1A with Fig. 2A). The lack of phototropic response in *phot1phot2* indicates that an intact UVR8 pathway by itself is not capable of inducing unidirectional bending at these intensities. Furthermore, *rup1rup2* mutants which are known to have enhanced UVR8 signaling [26] responded as the wild type (Fig. 2A). This indicates that at these low light intensities, the phototropin pathway is the principal system to induce bending while the UVR8 pathway is not functional. Interestingly, the kinetics of high and low fluence rate phototropin dependent bending as seen in the wild type are very similar, indicating little effect of fluence rate on bending kinetics. Our data thus suggest that phototropins regulate bending towards UV-B independently of UVR8. We further tested which mechanisms could be involved in the UV-B regulation of phototropin action in these conditions. *phot1phot2* double mutants harboring a PHOT1 protein with a kinase-dead LOV2 domain did not show bending towards UV-B, indicating functional dependence on LOV2 kinase activity of the UV-B response (Fig. 2B). Phototropin action in blue light results in the dephosphorylation of NPH3, which can be detected by western blot [11, 12]. We analyzed NPH3 protein in wild type and *phot1phot2* double mutant seedlings. UV-B treatment caused a mobility shift of NPH3, displaying a faster migrating NPH3 protein in the wild type and *uvr8* mutant, yet not in a phototropin double mutant background (Fig. 2C). This situation is similar to that of blue light exposure, [11, 12] although seemingly less effective (Fig. 2C), and suggests that NPH3 is at least partially dephosphorylated in wild type plants upon UV-B exposure. NPH3 is indeed involved in the response, since *nph3* loss of function mutants do not react to low levels of UV-B (Fig. 2B). Together the data indicate that phototropins function in UV-B in a similar way as they do in blue light.

3.3. Enhanced UVR8 signaling in *rup1rup2* mutants interferes with the fast phototropic response

To obtain additional insights in the balance between the phototropin and the UVR8 pathway, we investigated the behavior of mutants with enhanced UVR8 signaling more profoundly. Again, *rup1rup2* double mutants that lack feedback inhibition of the UVR8 response were used. At fluence rates of $0.12 \mu\text{mol}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$, *rup1rup2* bending started at the same time as in wild type, but the velocity of bending was reduced from 2h after onset of exposure (Fig. 3A). This response was not observed in blue light (Fig. 3B). Coincidentally, this is the same time point at which the UVR8-dependent bending response starts in *phot1phot2* mutants (compare Fig. 3A

with Fig. 1A), suggesting that this is the moment of first visible output of the UVR8 pathway. Thus, an overresponsive UVR8 pathway in *rup1rup2* mutants causes interference with bending. Transcriptional induction of the RUP genes is regulated by the UVR8 pathway, as part of a negative feedback loop [26]. In order to know whether in our conditions the phenotype of *rup1rup2* mutants is the result of permanent absence of RUP proteins, or may relate to their UV-B inducibility, a time lapse analysis of transcript accumulation was performed by RT-qPCR on etiolated wild type shoots (Fig. 3C). In our conditions RUP1 and RUP2 transcript levels were elevated in parallel within the hour, which fits within the time frame for interfering with UVR8 signaling at the time when the *rup1rup2* phenotype is visible around 120 minutes after the start of exposure (Fig. 3A). At later time points both RUP transcript levels remained elevated, with RUP2 relatively more present. The parallel induction suggests redundancy of the RUP1 and RUP2 function. Triple mutants of *rup1*, *rup2* with *uvr8* or *hy5* could rescue the *rup1rup2* bending phenotype (Fig. 3D), indicating that the canonical UVR8 pathway is involved. This pathway is known to regulate hypocotyl elongation [34]. Therefore we assayed the length of the upper part of the hypocotyl, known for its capacity for elongation [12, 35, 36], that extends towards a light source upon bending. When keeping the plants exposed to $0.12 \mu\text{mol.m}^{-2}\text{s}^{-1}$ of UV-B for a longer time (24h), a marked difference in length of the hypocotyl portion oriented towards the light was detected (Fig. 3E). In *rup1rup2* mutants, elongation towards the light source was severely inhibited compared to wild type plants. Elongation towards the light was completely restored in the *rup1rup2uvr8* mutant yet only partially in *rup1rup2hy5*. This suggests that after bending has occurred, factors additional to HY5 regulate elongation.

4. Discussion

4.1. Phototropins in UV-B perception

In recent years, UVR8 has been the focal point with respect to UV-B signaling. UVR8 uses tryptophanes to specifically capture the UV-B light. However chromophores from other photoreceptors also absorb in the UV-B range and may thus also trigger responses. For instance, phototropins not only absorb blue and UV-A, but their FMN chromophores also absorb in the UV-B region [37]. We have shown that the kinase activity of phototropins (Fig. 2A, B) and dephosphorylation of NPH3 is associated with this response to UV-B. The existence of these hallmarks for phototropin signaling in UV-B light explains why the phototropin pathway is capable of generating a growth response towards unilateral UV-B light in etiolated seedlings. In addition, other photoreceptors with chromophores, such as phytochromes [38], and cryptochromes [39] can absorb beyond their characteristic response-associated waveband range, including in the UV-B part of the spectrum. Previous data on phytochrome studies have shown that although they are well known to perceive red and far red light, their mutants have phenotypes in blue light [40, 41]. However, to date it is not known what the

contribution is of their blue light absorption to the blue light phenotypes. These phenotypes may arise from interaction with typical blue light photoreceptors [42-44]. It is likely that phototropins themselves function as UV-B receptors without assistance from other photoreceptors, contrary to what is believed for red light enhancement of phototropism. Considering our genetic data showing that UVR8 does not enhance the phototropin response, that the phototropin and UVR8 response are separated in time and that these photoreceptors by themselves can perceive and signal UV-B, we hypothesize a complete separation of UVR8 and phototropin UV-B perception in phototropism of etiolated seedlings.

4.2. Timing of the UV-B response

Our kinetic analysis suggests that the earliest macroscopic UVR8 pathway output is only visible after two hours. By contrast the molecular events that precede this event, seem to occur very rapidly. Initial monomerization of UVR8 and interaction with COP1 is observable within 15 minutes of broadband UV-B irradiation [24]. Increased levels of HY5 in our system were observed between 30 and 90 minutes after the onset of exposure [45]. Considering that *RUP1* and *RUP2* are transcriptionally induced after 60 minutes (Fig. 3C), and that their induction is dependent on HY5 function [26], this timing is in support of them being direct candidate targets of HY5. The influence of RUP1 and RUP2 proteins on the bending phenotype starts at almost the exact same time (Fig. 3A) as the UVR8 visible output for differential elongation (Fig. 1A). This indicates that in wild type plants the RUP proteins accumulate/are functional before the effect on elongation starts, or that these are parallel processes. In the latter case, the molecular machinery for feedback (RUP proteins) and the machinery for inhibiting elongation is ready for function at the same time.

4.3. RUP1 and RUP2 tightly control UVR8 signaling

RUP1 and RUP2 redundantly restrain UVR8 signaling thus preventing excessive photomorphogenesis in UV-B light, including pigment accumulation, inhibition of hypocotyl elongation and dwarfism [26]. Similarly, during UV-B regulated phototropism, they inhibit the UVR8 signaling pathway that controls elongation (Fig. 3E). This pathway is very likely to be the same UVR8 and HY5 dependent pathway as the one that controls differential elongation [5, 45]. The enhanced UV-B signaling in *rup1rup2* mutants would cause strong inhibition of elongation both at the illuminated and shaded side of the hypocotyl. In the wild type, elongation at the illuminated side is inhibited strongly while this is less the case at the shaded side. This results in a marked effect, especially at the shaded side and therefore generates a different ratio of elongation between illuminated and shaded side compared to wild type. Whereas in the wild type, the shaded side elongates faster, this elongation is counteracted by an increased UVR8 signal in *rup1rup2* mutants. This phenotype is visible in etiolated seedlings, yet it remains to be seen whether light exposed and pigment containing seedlings, respond in the same way. Nevertheless, the timing of induction of the RUP proteins (Fig. 3C) and the phenotype of the *rup1rup2* mutant (Fig. 3A) is in agreement with the necessity of rapidly switching on the

negative feedback (within 2h). This is important to avoid interference with the phototropin pathway. Indeed, our data indicate that the control of elongation by the UVR8 pathway should not interfere with phototropism in dark grown seedlings, in order to optimally orient towards the light source, for instance after underground germination. The biological role for RUP1 and RUP2 is to keep UVR8 signaling under control, to allow fast completion of the phototropin response. This allows seedlings to optimally orient their cotyledons towards the light. Furthermore, the UVR8 pathway is not active at lower light intensities, which appear very suitable for phototropin signaling (Fig. 2). This observation also supports the notion that UVR8 signaling predominantly serves in generating photoprotection against potentially damaging UV-B of higher intensities.

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Figure 1. Kinetic analysis of the bending response to monochromatic UV-B light. (A) Kinetic analysis of the bending angle of wild type (Col-0), *phot1phot2*, *uvr8-6 (uvr8)* and *phot1phot2amiuvr8* in unilateral UV-B. Plants were grown for two days in darkness and exposed to monochromatic UV-B light (302nm, $0.12 \mu\text{mol.m}^{-2}\text{s}^{-1}$). Error bars represent SD (n=10). (B) Response spectrum 5h after the onset of unilateral UV-B light. Plants were grown for two days in darkness and exposed to monochromatic UV light ($0.12 \mu\text{mol.m}^{-2}\text{s}^{-1}$). Error bars represent SD (n=10).

Figure 2. Phototropins mediate UV-B response at low light intensity. (A) Kinetic analysis of the UV-B bending response. 2 day old etiolated wild type (Col-0), *uvr8*, *phot1phot2* and *rup1rup2* mutant seedlings were exposed to unilateral UV-B (302nm) ($0.002 \mu\text{mol.m}^{-2}\text{s}^{-1}$) and followed in time for orientation towards the light source. Error bars are SD (n=10). (B) As in (A), with wild type (Col-0) and kinase dead phototropin transgenic plants (*D806N-2* and *D806N-19*) and *nph3-6* (*nph3*) mutants. (C) Western blot analysis of proteins extracted from wild type (Col-0), *phot1phot2*, *uvr8-6* and *phot1phot2amiUVR8* seedlings kept in darkness or exposed to 30 minutes of UV-B or blue light. Hybridization was done with anti-NPH3 antibodies (αNPH3), and anti-DET3 (αDET3) antibodies as loading control. Arrows indicate the bands of the phosphorylated (phos) and dephosphorylated (dephos) form of NPH3.

Figure 3. Analysis of the phototropic response to unilateral light of $0.12 \mu\text{mol m}^{-2}\text{s}^{-1}$ in *rup1rup2* mutants. (A) Kinetic analysis of bending towards UV-B (302nm) light. Error bars are SD (n=10). (B) Kinetic analysis of bending towards blue (450nm) light. Error bars are SD (n=10). (C) QRT-PCR time lapse analysis of RUP1 and RUP2 in wild type Col-0 plants (302nm light). Values are relative to the control genes *EF1alpha* and *EIF1a*. Error bars are SEM (n=3). (D) Kinetic analysis of triple mutants *rup1rup2uvr8* and *rup1rup2hy5* (302nm light) (E) Length of the upper hypocotyl part that grows towards the light source. Error bars represent SD (n≥15). Asterisks indicate statistically significant differences ($p < 0.01$) from the wild type.

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Figure(s)





