Plant Science 264 (2017) 96–101

Contents lists available at ScienceDirect

Plant Science

journal homepage: www.elsevier.com/locate/plantsci

Review article

Developmental reprogramming by UV-B radiation in plants

Marcela Dotto[a], Paula Casati[b,*]

[a] Facultad de Ciencias Agrarias, Universidad Nacional del Litoral, 3080, Esperanza, Santa Fe, Argentina
[b] Centro de Estudios Fotosintéticos y Bioquímicos, Universidad Nacional de Rosario, 2000, Rosario, Santa Fe, Argentina

ARTICLE INFO

Keywords:
UV-B
Plant development
Plant growth
Molecular mechanisms

ABSTRACT

Plants are extremely plastic organisms with the ability to adapt and respond to the changing environmental conditions surrounding them. Sunlight is one of the main resources for plants, both as a primary energy source for photosynthesis and as a stimulus that regulates different aspects of their growth and development. UV-B comprises wavelengths that correspond to a high energy region of the solar spectrum capable of reaching the biosphere, influencing plant growth. It is currently believed that plants are able to acclimate when growing under the influence of this radiation and perceive it as a signal, without stress signs. Nonetheless, many UV-B induced changes are elicited after DNA damage occurs as a consequence of exposure. In this review we focus on the influence of UV-B on leaf, flower and root development and emphasize the limited understanding of the molecular mechanisms for most of this developmental processes affected by UV-B documented over the years of research in this area.

1. Introduction

Plants are normally exposed to a constantly changing environment, which influences the developmental programs executed throughout their lifetime. Light is undoubtedly the most important environmental factor affecting processes such as seed germination, leaf, root and seed development, and flowering time. Perception of light is mediated by different photoreceptors leading to the modulation of signaling networks and genetic pathways to adjust plant growth and development accordingly. Even though ultraviolet-B (UV-B) radiation (280–315 nm) represents only a small fraction of the solar radiation reaching the Earth's surface, it is a high-energy component to which plants need to respond. Some of these responses involve changes in plant morphology, physiology and production of secondary metabolites, which are commonly described and several aspects of the underlying mechanisms have been elucidated [1–3].

Diverse UV-B intensities have been used in different experiments described in the literature, which are usually in the range of UV-B intensities that can reach the earth surface, since this is not uniform in different points of the planet. The influence of UV-B radiation on plant growth include both general deleterious effects and specific regulatory responses [1,2,4]. The deleterious effects include DNA damage, membrane changes, protein crosslinking and the formation of reactive oxygen species (ROS) which results in oxidative stress, these effects are usually produced by high UV-B intensity, typically above 1 μmol m^{-2} s^{-2} UV-B [5–7]. The more specific photomorphogenic responses are usually triggered by lower intensities of UV-B radiation, usually below 1 μmol m^{-2} s^{-2} UV-B, some photomorphogenic responses can be activated even at 0.1 mol m^{-2} s^{-2} UV-B [8]. It is currently believed that plants are able to perceive this radiation as a signal, without stress signs [9]. However, higher UV-B doses can also affect plant growth.

So far, UV RESISTANT LOCUS (UVR8) is the only UV-B specific photoreceptor identified [10,11], which has been shown to enhance Arabidopsis plant survival under low UV-B levels [10,12]. This receptor mediates some photomorphogenic responses through changes in the expression of genes involved in DNA repair, antioxidative defense and production of phenolic compounds that can act as UV screening molecules [10]. The UVR8 photoreceptor interacts with CONSTITUTIVELY PHOTOMORPHOGENIC 1 (COP1, an E3 ubiquitin ligase) after UV-B exposure to activate transcription of UV-B signaling and response genes, which initiate UV-B acclimation pathways [10,13–15].

In addition, high intensities of UV-B induce damage responses in plants; which involve signaling pathways that are partially conserved among different organisms [16–18], and are independent of UVR8. For example, to deal with UV-B damaged DNA, the protein kinase ATM AND RAD3-RELATED (ATR) functions as a key regulator of the DNA damage response rendering *atr* mutants hypersensitive to UV-B in Arabidopsis [16]. Besides this, the mitogen activated protein kinases (MAPK) MPK3 and MPK6 are triggered after exposure to DNA-damaging agents in Arabidopsis; making the MPK3/MPK6 cascade an additional major pathway contributing to UV-B-induced DNA damage

* Corresponding author at: Suipacha 531, Rosario, 2000, Argentina.
E-mail addresses: mdotto@fca.unl.edu.ar (M. Dotto), casati@cefobi-conicet.gov.ar (P. Casati).

http://dx.doi.org/10.1016/j.plantsci.2017.09.006
Received 31 May 2017; Received in revised form 27 July 2017; Accepted 1 September 2017
Available online 13 September 2017
0168-9452/

Table 1
UV-B influence on developmental processes.

Leaf area reduction	[22,41–44]
Downward curling	[41]
Leaf number reduction	[58]
Higher trichome density	[43,66–68]
Reduced hypocotyl extension	[12,34–38]
UV-B phototropism	[39,40]
Reduced root growth	[77–79]
Delay in flowering time	[43,71–73]

[17,19].

A summary of photomorphological changes induced by UV-B radiation in plants is shown in Table 1. Even though the effect of UV-B on developmental aspects of plant growth has been several times described, the mechanistic details behind these changes are largely missing. Recent revision articles have focused on different aspects of UV-B responses in plants, including UV-B responses controlled by hormones [3] and UVR8-mediated photomorphogenic responses [20,21], therefore in this review examine the current knowledge of UV-B mediated developmental reprogramming in plants, including UVR8-independent responses and identify areas of limited knowledge.

2. The influence of UV-B radiation on plant growth and development

UV-B radiation can induce several plant photomorphogenic responses which appear to be adaptive, including hypocotyl growth inhibition, cotyledon curling, and rapid induction of the expression of a variety of genes, including those involved in the synthesis of phenylpropanoid and flavonoid UV sunscreens [12,22–26]. After the identification of a UV-B specific photoreceptor, important advances in the elucidation of the initial steps of signal transduction have been made. UVR8 acts as the primary chromophore for UV-B, in an analogous manner to other well-characterized light receptors [27]. However, UVR8 is unique because it lacks a prosthetic chromophore to perform photoreception; instead this photoreceptor senses UV-B light through a triad of tryptophan residues present in this protein [11]. UVR8-mediated signal transduction involves the action of the photomorphogenesis response regulators COP1 [10,14] ELONGATED HYPOCOTYL 5 (HY5) and HY5 HOMOLOG (HYH), two bZIP transcription factors [13,28]. In the dark, HY5 is ubiquitinated by COP1 which acts as an E3 ubiquitin ligase and therefore marks HY5 for degradation by the proteasome. HY5 plays an important role during de-etiolation in the initiation of photomorphogenesis as well as in UV-B signaling through UVR8. This receptor localizes both in the nucleus and cytoplasm, when plants are grown under white light, it exists as a dimer mainly in the cytoplasm, whereas nuclear localization prevails after UV-B irradiation. This nuclear localization has been recently proposed to be mediated by association with COP1 [29,30]. Several genes are regulated by this pathway shortly after UV-B exposure, including HY5 and HYH, which act downstream of UVR8-COP1 and are required for most of the UVR8-dependent the transcriptional regulation [12,13,15].

The molecular mechanisms behind the UVR8-mediated transcriptional changes are still not clear. Even though some evidence suggests UVR8 associates to chromatin at certain loci [31], there is currently some controversy on this subject [32]. ChIP experiments performed by different authors failed to confirm direct UVR8-chromatin association, therefore it was proposed that UVR8-COP1 could modulate gene expression through interaction with HY5 and HYH, without direct chromatin binding [32].

Analysis of UVR8 signaling through expression from tissue-specific promoters indicated that early steps occur tissue-autonomously and require expression of UVR8 in epidermal cells, such as induction of HY5 expression, whereas expression of UVR8 from mesophyll cell is necessary for the processed leading to protect adult plants from the damaging effects of UV-B, and the acclimation of adult plants require signaling from mesophyll and epidermal cells simultaneously [33]. Amongst others, the UVR8-mediated UV-B signaling pathway affects hypocotyl growth, leaf morphogenesis, and phototropism, which are next described in more detail. Even though UVR8-mediated responses are described in numerous reports, we also include information about UV-B signaling through alternative pathways.

2.1. UV-B radiation and hypocotyl elongation

Low levels of UV-B radiation inhibit hypocotyl growth in one of the best characterized responses in Arabidopsis. This inhibition seems to be a major developmental effect regulated by the UVR8 pathway. Involved in this UV-B response are COP1, UVR8, and HY5, and the negative regulators of this pathway REPRESSOR OF UV-B PHOTOMORPHOGENESIS 1 (RUP1) and RUP2, which are two highly related WD40-repeat proteins and interact with UVR8 as strong repressors of UV-B signaling [34]. Wild type plants show a significant inhibition of hypocotyl growth after UV-B exposure, while *uvr8* mutants do not [12]; similarly, overexpression of RUP2 leads to a reduction in UV-B–induced hypocotyl growth inhibition [34]. In addition to these, the transcription factor FAR-RED ELONGATED HYPOCOTYL3 (FHY3), together with HY5, has also an important role in the regulation of COP1 expression in the low UV-B photomorphogenic UV-B signaling. For example, in the absence of FHY3, UV-B–induced inhibition of hypocotyl growth is impaired [35].

UVR8 not only mediates the inhibition of hypocotyl growth as a consequence of UV-B exposure but also by small changes in ambient temperature, called as thermomorphogenesis [36]. Thermomorphogenesis induces stem elongation and leaf elevation, which are predicted to improve leaf cooling. UV-B perceived by UVR8 decreases thermomorphogenesis via different mechanisms inhibiting the activity of a bHLH transcription factor PHYTOCHROME INTERACTING FACTOR 4 (PIF4), which up-regulates the biosynthesis of auxins that is required to regulate plant growth under high temperature. This result suggests that there is a crosstalk between UV-B and high-temperature signaling, and that UV-B-mediated inhibition of hypocotyl elongation is affected with growth temperature [36].

Nonetheless, UV-B not only induces photomorphogenic hypocotyl growth inhibition via de UVR8 photoreceptor but also via DNA damage responses at higher doses. For example, Arabidopsis mutants of the endonucleases of the Nucleotide Excision Repair (NER) DNA repair pathway (*xpf-3* and *uvr1-1*) showed a higher inhibition of hypocotyl growth by UV-B [37,38]. Therefore, increased DNA damage by UV-B also results in hypocotyl growth inhibition after UV-B treatment in Arabidopsis plants. Interestingly, at the high UV-B doses used in these experiments, etiolated *uvr8* mutants showed a similar inhibition of hypocotyl growth as wild type seedlings, suggesting that under the experimental conditions used, this photomorphogenic response is directed by signals originating from UV-B absorption by DNA independently of the UVR8 pathway [38].

2.2. UV-B radiation and leaf responses

2.2.1. Phototropism and epinasty

Leaves are crucial organs for light capture and efficient photosynthesis; in consequence most plants are able to adapt their morphology in order to achieve this function in the process known as phototropism. Phototropins mediate this process in response to blue and UV-A light, but orientation towards UV-B light requires UVR8 and is independent from the phototropins PHOT1 and PHOT2 and the phototropin-mediated signaling component NPH3 [39]. Interestingly, UV-B through the UVR8 pathway seems to repress the expression of auxin-responsive genes affecting directional bending. However, a recent report indicates that at very low UV-B intensities, phototropins mediate the response to UV-B in etiolated wild type seedlings, acting as

the most important receptors for UV-B induced phototropism and signaling by the UVR8 pathway becomes important when the phototropin-dependent response is complete [40].

An additional leaf response induced by UV-B radiation is the downward curling (or epinasty). In *Arabidopsis thaliana*, UV-B provokes leaf epinasty of the edges of the blade in wild type plants, while this is not observed in *uvr8* mutants, suggesting that this photomorphogenic effect is mediated by the UVR8 pathway after UV-B irradiation, with a mechanism similar to that activated by phytocrome PHYB and its downstream components PHYTOCHROME INTERACTING FACTORS (PIFs) under red light [41]. Interestingly, under low levels of supplementary UV-B, PHYB signaling is necessary for epinasty of the blade edges. Moreover, downward leaf curling apparently rely on altered auxin distribution, as auxin levels accumulate at the leaf blade edges in the presence of UV-B [41]. Therefore, epinasty after UV-B exposure seems to require the co-action of PHYB and UVR8 signaling pathways, both affecting auxin levels [41].

2.2.2. Leaf growth

One of the most consistent responses to UV-B exposure is the reduction in leaf area [22,41–43]. This reduction has been attributed to both inhibition of cell division and decrease in cell expansion, which seems to be a species-specific response [44]. Different data about UV-B effects on cell proliferation and cell expansion seem to be contradictory, but these inconsistencies probably reflect differences in experimental conditions. For example, stress-inducing UV-B conditions that produce DNA damage and necrosis generally inhibit cell proliferation, while lower doses and/or chronic UV-B treatments can result in both inhibition of cell proliferation and expansion [9,45–53]. For example, fewer epidermal cells were observed after UV-B exposure in wheat and pea [52,54]; but UV-B radiation can also reduce epidermal cell expansion in leaves of *Arabidopsis thaliana* [44]. Moreover, several reports have shown that UV-B inhibits cell expansion in different plant species [22,45,54–56].

Molecular details regarding the mechanism behind the reduction in leaf area were provided for Arabidopsis plants. This process was demonstrated to be a result of inhibition of cell proliferation mediated by the action of miR396, a microRNA responsible for the downregulation of *GROWTH REGULATING FACTORS* (*GRF*) transcription factors [42]. miRNA396 levels are increased by UV-B in proliferating leaves, and this induction parallels the decrease in *GRF1*, *GRF2* and *GRF3* transcripts. Induction of miR396 results in inhibition of cell proliferation. Interestingly, this response is independent of UVR8 and ATR, but dependent on MPK3, which is activated by UV-B-induced DNA damage [42]. Hence, at intensities that can cause DNA damage in Arabidopsis, UV-B radiation restricts leaf growth by inhibiting cell division in proliferating tissues. Also, UV-B levels present in solar radiation inhibits maize leaf growth as a consequence of a decrease in cell production in the meristem of the developing leaf [57]. This decrease in the meristem size in maize leaves by UV-B correlates with a decrease in the concentration of gibberellins regulated by GRF1 and possibly other transcription factors of this family [57]. Moreover, a reduction in plant total leaf number was observed in some crop species, such as maize [58].

As described above, the reduction in leaf area by UV-B can be a consequence of either a decrease in cell division or in cell expansion, or both. Decreases in stem elongation by UV-B have also been reported for different species [59–62]. Recently, Roro et al. demonstrated that the reduction in shoot elongation and leaf area in pea in response to a daily UV-B exposure was associated with decreased levels of gibberellins in apical stem tissue and young leaves. However, it is not clear whether the inhibition of shoot elongation in UV-B exposed plants is a direct result of decreased cell expansion, or if cell division is also affected.

Characterization of the Arabidopsis mutant *uvi4* (*UV-B-insensitive 4*) indicated that the leaf cells of this mutant undergo one extra round of endoreduplication [63]. While the total leaf blade area *of uvi4* mutants was similar to that of wild type plants throughout leaf development; the cell area in the adaxial epidermis was larger than that of wild type leaves, demonstrating that the *uvi4* leaves have fewer but larger epidermal cells. Interestingly, tetraploid *uvi4* Arabidopsis leaves were more resistant to UV-B than diploid leaves, suggesting that endoreduplication (and as a consequence leaves with larger cells) can increase UV-B tolerance [63]. However, the link of endoreduplication with cell and organ growth and its regulation by UV-B radiation is not completely understood. The atypical E2F transcription factor E2Fe/DEL1 is an important regulator of the endocycle. Interestingly, E2Fe/DEL1 is also a transcriptional repressor of *PHR1*, a cyclobutane pyrimidine dimer photolyase in Arabidopsis [64]. After UV-B exposure, *e2fe* mutants show higher DNA repair than wild type plants and these mutants resume endoreduplication faster than control plants. These results lead to the conclusion that DNA damage after UV-B exposure regulates ploidy-dependent cell growth in Arabidopsis, probably independently of the UVR8 photoreceptor pathway. Despite this, UVR8 has also been previously implicated in the regulation of endopolyploidy in response to UV-B [45]; thus, it is possible that several UV-B independent pathways may converge to a common endoreduplication regulator under UV-B conditions. In fact, UV-B radiation has been identified as a significant positive climatic predictor for high endopolyploidy; and *Arabidopsis* accessions with increasing endopolyploidy also show higher tolerance to UV-B radiation, suggesting that endoreduplication may be a UV-B tolerance mechanism that exists in natural populations [65].

2.2.3. Trichome development

One of the strategies that plants have developed to avoid UV-B radiation damage is the ability to produce UV-B absorbing compounds, such as flavonoids and other phenolics, which are mainly located in superficial tissues, including the trichomes [66,67]. In fact, trichome density of leaves exposed to UV-B was reported to be higher than those of shaded leaves, and leaf hairs in the exposed region showed a considerably increased UV-B absorbing capacity [68]. The regulation of the density of leaf trichomes by UV-B is GLABRA3 (GL3)-dependent; a higher trichome density correlates with lower sensitivity to UV-B, suggesting a shielding role for trichomes in exposed leaves [43]. Similarly, an increase in the production of epicuticular wax was observed for maize plants exposed to UV-B, which is also believed to have a protective function [58]. The mechanistic details for these responses are still unknown.

2.3. UV-B radiation and inflorescence development

The correct timing of flowering is of outstanding importance, since it must ultimately result in the successful dispersal of seeds. In order to achieve the tight regulation of flowering, multiple pathways have evolved to integrate several environmental and endogenous cues [69,70]. Changes in flowering time after UV-B exposure have been described in several species including Arabidopsis, maize, *Phaseolus vulgaris* and *Vigna radiate* [43,71–73]. A delay in flowering time was observed in all cases, but no molecular details on the mechanism behind it are currently known. UV-B also induces a decrease in inflorescence height and an increase in the number of flowering stems in Arabidopsis [9]. There is also very limited knowledge regarding UV-B influence on flower development, and a few reports studying the effect of this radiation on fruit development describe mainly changes in secondary metabolites composition [74,75]. These changes are often interpreted as a consequence of the plant reallocating resources when growing under the influence of UV-B radiation. In many species, resource allocation toward reproductive parts occurs only after the plant reaches a certain mass, size or age [76].

2.4. UV-B radiation and root development

Roots are very important for the success of land plants, and several

reports have shown that Arabidopsis plants have a UV-B-sensing mechanism in roots that regulate morphogenesis and early seedling development [77]. In Arabidopsis wild type seedlings, UV-B exposure causes an inhibition of primary root elongation. However, the *root UVB sensitive 1* (*rus1*) mutant has a primary root that is hypersensitive to UV-B [77]. Interestingly, the roots but not other organs perceive the UV-B signal responsible for root growth inhibition, and this response seems to be independent of all other known plant photoreceptors. Arabidopsis has a second gene that modulates root growth inhibition by UV-B, *RUS2*, and *rus2* mutants seedlings show similar phenotypes as *rus1* seedlings under UV-B exposure, suggesting that the two proteins work in the same pathway [78]. It has been proposed that RUS1 and RUS2 physically interact in a root UV-B-sensing pathway regulating early seedling morphogenesis and development in Arabidopsis.

On the other hand, high levels of UV-B radiation also induce the expression of cell cycle regulatory and DNA damage genes in Arabidopsis roots, along with inhibition of primary root elongation [79]. These results suggest that in roots, UV-B-induced DNA damage may result in the delay of the G1-to-S transition of the cell cycle, which might be a protective mechanism to prevents cells with damaged DNA from dividing [79].

3. Conclusions

Several photomorphogenic responses are elicited after UV-B exposure in plants, which are probably an adaptation to allow them to survive under a hostile ambient. As described in this review, some of these adaptive growth changes are mediated by the UV-B specific UVR8 photoreceptor pathway; however, many morphological effects are activated by DNA damage responses independently of the UVR8 photoreceptor (Fig. 1). Despite this, it is clear that some, if not most, photomorphological changes that occur after UV-B exposure in plants, can be provoked by different UV-B response pathways depending on the intensity and/or duration of the exposure. Data discussed here show that, for example, hypocotyl growth inhibition can be a consequence of the activation of either the UVR8 pathway (see for example references [12,34]) or a direct response after DNA damage caused by UV-B exposure [37,38], being the activation of either pathways clearly dependent on the UV-B intensities/doses used in the different experiments. This is also true for the regulation of leaf growth [41,42] and the regulation of the entrance to the endoreduplication pathway [45,63] by UV-B. It seems that UV-B responses through the UVR8 pathway (activated by low UV-B levels) and after DNA damage, through the activation of the ATR, the MPK3 and 6 and RUS pathways in the roots (triggered after exposure of high levels of UV-B) converge to general regulators of plant growth to provoke reprogramming of the necessary developmental processes that would allow plants to complete their life cycle under adverse or changing environmental conditions.

Even though some reports indicate that there is a cross-talk between UVR8-mediated responses and the circadian clock, the knowledge is still very limited [25,80,81]. It has been proposed that UV-B light entrains the clock through transcriptional activation of clock genes mediated by UVR8 and COP1 [25] and that the clock could modulate the plant sensitivity to UV-B stress at different times of the day [80]. Since the circadian clock regulates multiple factors that affect plant growth and development, this is an interesting field of research that could shed light into the influence of this cross-talk in the developmental programs affected by UV-B radiation.

Interestingly, some of the photomorfological changes induced by UV-B exposure seem to be also regulated and mediated by hormones, such as the auxin requirement in UV-B induced phototropism [40] and downward leaf curling [41]; or the role of gibberellins in inhibition of leaf growth [57]. Although a crosstalk between UV-B light and hormone responses in plants exist and it has been recently reviewed by

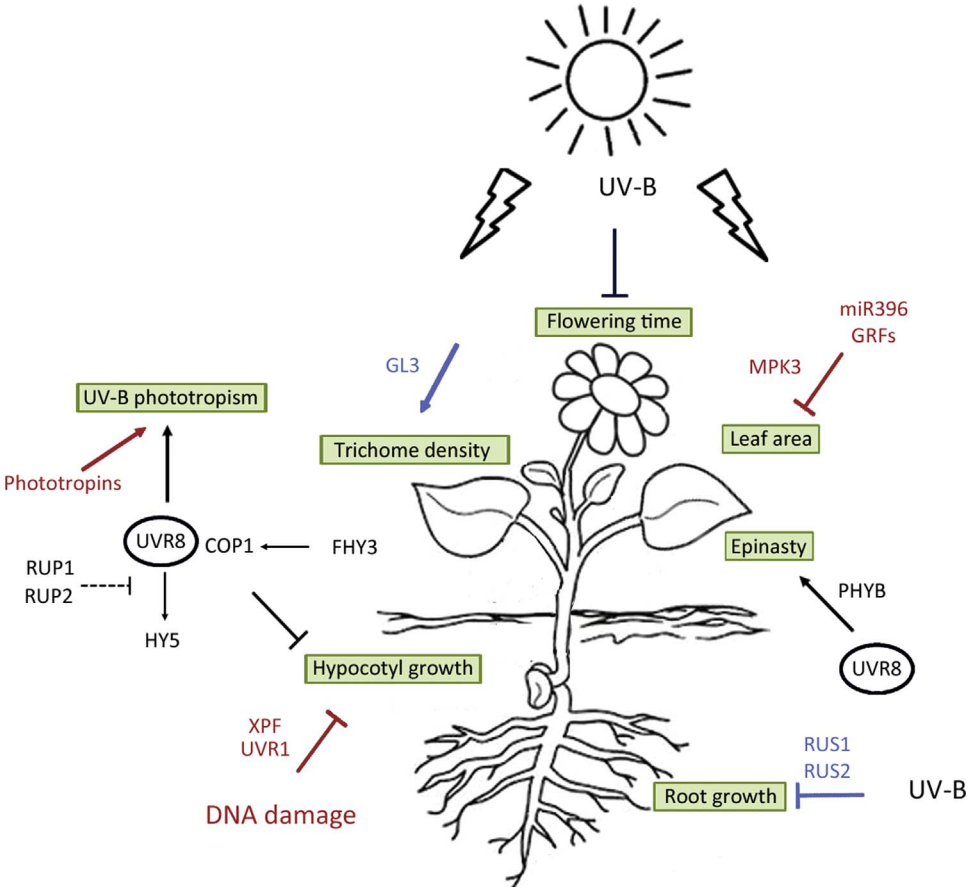

Fig. 1. UV-B influence on developmental processes in plants. The developmental processes discussed in this review are represented inside green rectangles. UVR8-dependent processes are indicated in black, UVR8-independent processes are represented in red and those with unknown dependency on this receptor are represented in blue color. Arrows indicate induction and blocked arrows represent inhibition of the developmental process. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Vanhaelewyn et al. [3], in the future it would be interesting to further investigate the connection between the hormone regulated pathways, the low UV-B, UVR8 photomorphogenic activated pathway, and the high UV-B, DNA damage activated morphogenic pathways, and see if there is a common UV-B signal transductor that converge to the same or different elements in hormone pathways to regulate photomorphogenic changes by this radiation.

Acknowledgements

This work was supported by FONCyT grant 2015-157 to P.C. P.C. and M.D. are members of the Researcher Career of the Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET) and P.C. is an Associate Professor of UNR.

References

[1] S.A. Mackerness, Plant responses to ultraviolet-B (UV-B: 280–320 nm) stress: what are the key regulators? Plant Growth Regul. 32 (2000) 27–39.
[2] H. Frohnmeyer, D. Staiger, Ultraviolet-B radiation-mediated responses in plants. Balancing damage and protection, Plant Physiol. 133 (2003) 1420–1428.
[3] L. Vanhaelewyn, E. Prinsen, D. Van Der Straeten, F. Vandenbussche, Hormone-controlled UV-B responses in plants, J. Exp. Bot. 67 (2016) 4469–4482.
[4] C.L. Ballare, a. L. Scopel, a. E. Stapleton, M.J. Yanovsky, Solar ultraviolet-B radiation affects seedling emergence, DNA, integrity, plant morphology, growth rate, and attractiveness to herbivore insects in datura ferox, Plant Physiol. 112 (1996) 161–170.
[5] S.S. Gill, N.A. Anjum, R. Gill, M. Jha, N. Tuteja, DNA damage and repair in plants under ultraviolet and ionizing radiations, Sci. World J. 2015 (2015) 5–7.
[6] V. Manova, D. Gruszka, DNA damage and repair in plants − from models to crops, Front. Plant Sci. 6 (2015) 1–26.
[7] P. Parihar, S. Singh, R. Singh, V.P. Singh, S.M. Prasad, Changing scenario in plant UV-B research:UV-B from a generic stressor to a specific regulator, J. Photochem. Photobiol. B. 153 (2015) 334–343.
[8] B.A. Brown, G.I. Jenkins, UV-B signaling pathways with different fluence-rate response profiles are distinguished in mature Arabidopsis leaf tissue by requirement for UVR8, HY5, and HYH, Plant Physiol. 146 (2008) 576–588.
[9] K. Hectors, E. Prinsen, W. De Coen, M.A.K. Jansen, Y. Guisez, Arabidopsis thaliana plants acclimated to low dose rates of ultraviolet B radiation show specific changes in morphology and gene expression in the absence of stress symptoms, New Phytol. 175 (2007) 255–270.
[10] L. Rizzini, J.-J. Favory, C. Cloix, D. Faggionato, A. O'Hara, E. Kaiserli, R. Baumeister, E. Schafer, F. Nagy, G.I. Jenkins, R. Ulm, Perception of UV-B by the Arabidopsis UVR8 protein, Science 332 (2011) 103–106.
[11] G.I. Jenkins, Structure and function of the UV-B photoreceptor UVR8, Curr. Opin. Struct. Biol. 29 (2014) 52–57.
[12] J.-J. Favory, A. Stec, H. Gruber, L. Rizzini, A. Oravecz, M. Funk, A. Albert, C. Cloix, G.I. Jenkins, E.J. Oakeley, H.K. Seidlitz, F. Nagy, R. Ulm, Interaction of COP1 and UVR8 regulates UV-B-induced photomorphogenesis and stress acclimation in Arabidopsis, EMBO J. 28 (2009) 591–601.
[13] R. Ulm, A. Baumann, A. Oravecz, Z. Máté, E. Adám, E.J. Oakeley, E. Schäfer, F. Nagy, Genome-wide analysis of gene expression reveals function of the bZIP transcription factor HY5 in the UV-B response of Arabidopsis, Proc. Natl. Acad. Sci. U. S. A. 101 (2004) 1397–1402.
[14] A. Oravecz, CONSTITUTIVELY PHOTOMORPHOGENIC1 is required for the UV-B response in arabidopsis, Plant Cell 18 (2006) 1975–1990.
[15] B.A. Brown, C. Cloix, G.H. Jiang, E. Kaiserli, P. Herzyk, D.J. Kliebenstein, G.I. Jenkins, A UV-B-specific signaling component orchestrates plant UV protection, Proc. Natl. Acad. Sci. 102 (2005) 18225–18230.
[16] K. Culligan, A. Tissier, A. Britt, ATR regulates a G2-phase cell-cycle checkpoint in arabidopsis thaliana, Plant Cell 16 (2004) 1091–1104.
[17] M.A. González Besteiro, R. Ulm, ATR and MKP1 play distinct roles in response to UV-B stress in Arabidopsis, Plant J. 73 (2013) 1034–1043.
[18] T. Cools, L. De Veylder, DNA stress checkpoint control and plant development, Curr. Opin. Plant Biol. 12 (2009) 23–28.
[19] M.A.G. Besteiro, S. Bartels, A. Albert, R. Ulm, Arabidopsis MAP kinase phosphatase 1 and its target MAP kinases 3 and 6 antagonistically determine UV-B stress tolerance, independent of the UVR8 photoreceptor pathway, Plant J. 68 (2011) 727–737.
[20] R. Yin, R. Ulm, How plants cope with UV-B: from perception to response, Curr. Opin. Plant Biol. 37 (2017) 42–48.
[21] G.I. Jenkins, Photomorphogenic responses to ultraviolet-B light, Plant Cell Environ. (2017).
[22] J.J. Wargent, V.C. Gegas, G.I. Jenkins, J.H. Doonan, N.D. Paul, UVR8 in Arabidopsis thaliana regulates multiple aspects of cellular differentiation during leaf development in response to ultraviolet B radiation, New Phytol. 183 (2009) 315–326.
[23] D.J. Kliebenstein, Arabidopsis UVR8 regulates ultraviolet-B signal transduction and tolerance and contains sequence similarity to human regulator of chromatin condensation 1, Plant Physiol. 130 (2002) 234–243.
[24] P.V. Demkura, C.L. Ballaré, UVR8 mediates UV-B-induced arabidopsis defense responses against Botrytis cinerea by controlling sinapate accumulation, Mol. Plant. 5 (2012) 642–652.
[25] B. Fehér, L. Kozma-Bognár, E. Kevei, A. Hajdu, M. Binkert, S.J. Davis, E. Schäfer, R. Ulm, F. Nagy, Functional interaction of the circadian clock and UV RESISTANCE LOCUS 8-controlled UV-B signaling pathways in Arabidopsis thaliana, Plant J. 67 (2011) 37–48.
[26] K. Tilbrook, A.B. Arongaus, M. Binkert, M. Heijde, R. Yin, R. Ulm, The UVR8 UV-B photoreceptor: perception, signaling and response, Arabidopsis Book 11 (2013) e0164.
[27] V.C. Galvão, C. Fankhauser, Sensing the light environment in plants: photoreceptors and early signaling steps, Curr. Opin. Neurobiol. 34 (2015) 46–53.
[28] B.A. Brown, L.R. Headland, G.I. Jenkins, UV-B action spectrum for UVR8-mediated HY5 transcript accumulation in arabidopsis, Photochem. Photobiol. 85 (2009) 1147–1155.
[29] R. Yin, M. Skvortsova, S. Loubery, R. Ulm, COP1 is required for UV-B-induced nuclear accumulation of the UVR8 photoreceptor, Proc. Natl. Acad. Sci. 113 (2016) E4415–E4422.
[30] C. Qian, W. Mao, Y. Liu, H. Ren, O.S. Lau, X. Ouyang, X. Huang, Dual-source nuclear monomers of UV-B light receptor direct photomorphogenesis in arabidopsis, Mol. Plant. 9 (2016) 1671–1674.
[31] C. Cloix, G.I. Jenkins, Interaction of the arabidopsis UV-B-specific signaling component UVR8 with chromatin, Mol. Plant. 1 (2008) 118–128.
[32] M. Binkert, C.D. Crocco, B. Ekundayo, K. Lau, S. Raffelberg, K. Tilbrook, R. Yin, R. Chappuis, T. Schalch, R. Ulm, Revisiting chromatin binding of the Arabidopsis UV-B photoreceptor UVR8, BMC Plant Biol. (2016) 1–11.
[33] P. Bernula, C.D. Crocco, A.B. Arongaus, R. Ulm, F. Nagy, A. Viczian, Expression of the UVR8 photoreceptor in different tissues reveals tissue-autonomous features of UV-B signalling, Plant Cell Environ. 40 (2017) 1104–1114.
[34] H. Gruber, M. Heijde, W. Heller, A. Albert, H.K. Seidlitz, R. Ulm, Negative feedback regulation of UV-B-induced photomorphogenesis and stress acclimation in Arabidopsis, Proc. Natl. Acad. Sci. 107 (2010) 20132–20137.
[35] X. Huang, X. Ouyang, P. Yang, O.S. Lau, G. Li, J. Li, H. Chen, X.W. Deng, Arabidopsis FHY3 and HY5 positively mediate induction of COP1 transcription in response to photomorphogenic UV-B light, Plant Cell 24 (2012) 4590–4606.
[36] S. Hayes, A. Sharma, D.P. Fraser, M. Trevisan, C.K. Cragg-Barber, E. Tavridou, C. Fankhauser, G.I. Jenkins, K.A. Franklin, UV-B perceived by the UVR8 photoreceptor inhibits plant thermomorphogenesis, Curr. Biol. 27 (2017) 120–127.
[37] G. Gardner, C. Lin, E.M. Tobin, H. Loehrer, D. Brinkman, Photobiological properties of the inhibition of etiolated Arabidopsis seedling growth by ultraviolet-B irradiation, Plant Cell Environ. 32 (2009) 1573–1583.
[38] J.J. Biever, D. Brinkman, G. Gardner, UV-B inhibition of hypocotyl growth in etiolated Arabidopsis thaliana seedlings is a consequence of cell cycle arrest initiated by photodimer accumulation, J. Exp. Bot. 65 (2014) 2949–2961.
[39] F. Vandenbussche, K. Tilbrook, A.C. Fierro, K. Marchal, D. Poelman, D. Van Der Straeten, R. Ulm, Photoreceptor-mediated bending towards UV-B in arabidopsis, Mol. Plant. 7 (2014) 1041–1052.
[40] L. Vanhaelewyn, P. Schumacher, D. Poelman, C. Fankhauser, D. Van Der Straeten, F. Vandenbussche, REPRESSOR OF ULTRAVIOLET-B PHOTOMORPHOGENESIS function allows efficient phototropin mediated ultraviolet-B phototropism in etiolated seedlings, Plant Sci. 252 (2016) 215–221.
[41] A.C. Fierro, O. Leroux, B. De Coninck, B.P.A. Cammue, K. Marchal, E. Prinsen, D. Van Der Straeten, F. Vandenbussche, Ultraviolet-B radiation stimulates downward leaf curling in Arabidopsis thaliana, Plant Physiol. Biochem. 93 (2015) 9–17.
[42] R. Casadevall, R.E. Rodriguez, J.M. Debernardi, J.F. Palatnik, P. Casati, Repression of growth regulating factors by the microRNA396 inhibits cell proliferation by UV-B radiation in Arabidopsis leaves, Plant Cell 25 (2013) 3570–3583.
[43] A. Yan, J. Pan, L. An, Y. Gan, H. Feng, The responses of trichome mutants to enhanced ultraviolet-B radiation in Arabidopsis thaliana, J. Photochem. Photobiol. B Biol. 113 (2012) 29–35.
[44] K. Hectors, E. Jacques, E. Prinsen, Y. Guisez, J.P. Verbelen, M.A.K. Jansen, K. Vissenberg, UV radiation reduces epidermal cell expansion in leaves of Arabidopsis thaliana, J. Exp. Bot. 61 (2010) 4339–4349.
[45] J.J. Wargent, J.P. Moore, A. Roland Ennos, N.D. Paul, Ultraviolet radiation as a limiting factor in leaf expansion and development, Photochem. Photobiol. 85 (2009) 279–286.
[46] S. Nogues, D.J. Allen, J.I. Morison, N.R. Baker, Ultraviolet-B radiation effects on water relations, leaf development, and photosynthesis in droughted pea plants, Plant Physiol. 117 (1998) 173–181.
[47] K.R. Reddy, S.K. Singh, S. Koti, V.G. Kakani, D. Zhao, W. Gao, V.R. Reddy, Quantifying the effects of corn growth and physiological responses to ultraviolet-B radiation for modeling, Agron. J. 105 (2013) 1367–1377.
[48] T.M. Robson, P.J. Aphalo, Species-specific effect of UV-B radiation on the temporal pattern of leaf growth, Physiol. Plant. 144 (2012) 146–160.
[49] I. Staxen, J.F. Bornman, A morphological and cytological study of Petunia hybrida exposed to UV-B radiation, Physiol. Plant. 91 (1994) 735–740.
[50] K. Laakso, J.H. Sullivan, S. Huttunen, The effects of UV-B radiation on epidermal anatomy in loblolly pine (Pinus taeda L.) and Scots pine (Pinus sylvestris L.), Plant, Cell Environ. 23 (2000) 461–472.
[51] R.W. Hofmann, B.D. Campbell, S.J. Bloor, E.E. Swinny, K.R. Markham, K.G. Ryan, D.W. Fountain, Responses to UV-B radiation in Trifolium repens L. – physiological links to plant productivity and water availability, Plant Cell Environ. (2003) 603–612.
[52] L. Hopkins, M.A. Bond, A.K. Tobin, S. Harold, M. Building, S. Andrews, Ultraviolet-B radiation reduces the rates of cell division and elongation in the primary leaf of wheat (Triticum aestivum L. cv Maris Huntsman)t, Plant Cell Environ. 65 (2002) 617–624.

[53] M.C. Rousseaux, S.D. Flint, P.S. Searles, M.M. Caldwell, Plant responses to current solar ultraviolet-B radiation and to supplemented solar ultraviolet-B radiation simulating ozone depletion: an experimental comparison, Photochem. Photobiol. 80 (2004) 224.
[54] R. Gonzalez, R. Mepsted, A.R. Wellburn, N.D. Paul, Non-photosynthetic mechanisms of growth reduction in pea (Pisum sativum L.) exposed to UV-B radiation, Plant Cell Environ. 21 (1998) 23–32.
[55] C.T. Ruhland, T.A. Day, Effects of ultraviolet-B radiation on leaf elongation, production and phenylpropanoid concentrations of Deschampsia antarctica and Colobanthus quitensis in Antarctica, Physiol. Plant. 109 (2000) 244–251.
[56] C.T. Ruhland, F.S. Xiong, W.D. Clark, T.A. Day, The Influence of Ultraviolet-B Radiation on growth, hydroxycinnamic acids and flavonoids of Deschampsia antarcfica during springtime ozone depletion in Antarctica, Photochem. Photobiol. 81 (2005) 1086–1093.
[57] J. Fina, R. Casadevall, H. AbdElgawad, E. Prinsen, M. Markakis, G. Beemster, P. Casati, UV-B inhibits leaf growth through changes in Growth Regulating Factors and gibberellin levels, Plant Physiol. 365 (2017).
[58] C. Wijewardana, W.B. Henry, W. Gao, K.R. Reddy, Interactive effects on CO2 drought, and ultraviolet-B radiation on maize growth and development, J. Photochem. Photobiol. B. 160 (2016) 198–209.
[59] N. Furness, P. Jolliffe, M. Upadhyaya, Competitive interactions in mixtures of broccoli and Chenopodium album grown at two UV-B radiation levels under glasshouse conditions, Weed Res. 45 (2005) 449–459.
[60] R.W. Hofmann, B.D. Campbell, Response of Trifolium repens to UV-B radiation: morphological links to plant productivity and water availability, Plant Biol. 13 (2011) 896–901.
[61] M. Germ, B. Breznik, N. Dolinar, I. Kreft, A. Gaberscik, The combined effect of water limitation and UV-B radiation on common and tartary buckwheat, Cereal Res. Commun. 41 (2013) 97–105.
[62] A. Roro, S. Dukker, T. Melby, K. Solhaug, S. Torre, J. Olsen, UV-B-induced inhibition of stem elongation and leaf expansion in pea depends on modulation of gibberellin metabolism and intact gibberellin signalling, J. Plant Growth Regul. (2017) 1–11.
[63] Y. Hase, K.H. Trung, T. Matsunaga, A. Tanaka, A mutation in the uvi4 gene promotes progression of endo-reduplication and confers increased tolerance towards ultraviolet B light, Plant J. 46 (2006) 317–326.
[64] A. Radziejwoski, K. Vlieghe, T. Lammens, B. Berckmans, S. Maes, M.A.K. Jansen, C. Knappe, A. Albert, H.K. Seidlitz, G. Bahnweg, D. Inze, L. De Veylder, Atypical E2F activity coordinates PHR1 photolyase gene transcription with endoreduplication onset, EMBO J. 30 (2011) 355–363.
[65] V.C. Gegas, J.J. Wargent, E. Pesquet, E. Granqvist, N.D. Paul, J.H. Doonan, Endopolyploidy as a potential alternative adaptive strategy for Arabidopsis leaf size variation in response to UV-B, J. Exp. Bot. 65 (2014) 2757–2766.
[66] G. Karabourniotis, K. Papadopoulos, M. Papamarkou, Y. Manetas, Ultraviolet-B radiation absorbing capacity of leaf hairs, Physiol. Plant. 86 (1992) 414–418.
[67] G. Karabourniotis, G. Kofidis, C. Fasseas, V. Liakoura, I. Drossopoulos, Polyphenol deposition in leaf hairs of olea europaea (Oleaceae) and quercus ilex (Fagaceae), Am. J. Bot. 85 (1998) 1007–1012.
[68] V. Liakoura, M. Stefanou, Y. Manetas, C. Cholevas, G. Karabourniotis, Trichome density and its UV-B protective potential are affected by shading and leaf position on the canopy, Environ. Exp. Bot. 38 (1997) 223–229.
[69] N.H. Battey, Aspects of seasonality, J. Exp. Bot. 51 (2000) 1769–1780.
[70] L.-H. Cho, J. Yoon, G. An, The control of flowering time by environmental factors, Plant J. 13461 (2016) 1–12.
[71] U. Mark, M. Saile-Mark, M. Tevini, Effects of solar UVB radiation on growth, flowering and yield of central and southern European maize cultivars (Zea mays L.), Photochem. Photobiol. 64 (1996) 457–463.
[72] M. Saile-Mark, M. Tevini, Effects of solar UV-B radiation on growth, flowering and yield of central and southern European bush bean cultivars (Phaseolus vulgaris L.), in: J. Rozema, W.W.C. Gieskes, S.C. Van De Geijn, C. Nolan, H. De Boois (Eds.), UV-B Biosph. Springer, Netherlands, Dordrecht, 1997, pp. 114–125.
[73] K. Rajendiran, M.P. Ramanujam, Improvement of biomass partitioning, flowering and yield by triadimefon in UV-B stressed Vigna radiata (L.) Wilczek, Biol. Plant. 48 (2004) 145–148.
[74] R. Alonso, F.J. Berli, A. Fontana, P. Piccoli, Malbec grape (Vitis vinifera L.) responses to the environment: berry phenolics as influenced by solar UV-B, water deficit and sprayed abscisic acid, Plant Physiol. Biochem. 109 (2016) 84–90.
[75] P. Carbonell-Bejerano, M. Diago, J. Martínez-Abaigar, J.M. Martínez-Zapater, J. Tardáguila, E. Núñez-Olivera, Solar ultraviolet radiation is necessary to enhance grapevine fruit ripening transcriptional and phenolic responses, BMC Plant Biol. 14 (2014) 1–16.
[76] P.A. Wigge, Integration of spatial and temporal information during floral induction in arabidopsis, Science 309 (2005) 1056–1059.
[77] H. Tong, C.D. Leasure, X. Hou, G. Yuen, W. Briggs, Z.-H. He, Role of root UV-B sensing in Arabidopsis early seedling development, Proc. Natl. Acad. Sci. U. S. A. 105 (2008) 21039–21044.
[78] C.D. Leasure, H. Tong, G. Yuen, X. Hou, X. Sun, Z.-H. He, ROOT UV-B SENSITIVE2 acts with ROOT UV-B SENSITIVE1 in a root ultraviolet B-Sensing pathway, Plant Physiol. 150 (2009) 1902–1915.
[79] L. Jiang, Y. Wang, L.O. Bjorn, S. Li, UV-B-induced DNA damage mediates expression changes of cell cycle regulatory genes in Arabidopsis root tips, Planta 233 (2011) 831–841.
[80] T. Takeuchi, L. Newton, A. Burkhardt, S. Mason, E.M. Farré, Light and the circadian clock mediate time-specific changes in sensitivity to UV-B stress under light/dark cycles, J. Exp. Bot. 65 (2014) 6003–6012.
[81] E. Horak, E.M. Farré, The regulation of UV-B responses by the circadian clock, Plant Signal. Behav. 10 (2015) e1000164.