Environmental and Experimental Botany 124 (2016) 89–99

Contents lists available at ScienceDirect

Environmental and Experimental Botany

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

Review

The relationship between multiple UV-B perception mechanisms and DNA repair pathways in plants

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ARTICLE INFO

Article history:
Received 9 October 2015
Received in revised form 14 December 2015
Accepted 26 December 2015
Available online 30 December 2015

Keywords:
Arabidopsis
Cell-cycle arrest
DNA repair
Photodimers
Photomorphogenesis
UV-B

ABSTRACT

UV-B radiation (280–320 nm) is a component of sunlight and a natural environmental stimulus for plants. The characterization of UVR8 (UV Resistance Locus 8) demonstrated that plants contain at least one UV-B-specific photoreceptor and signaling pathway. In plants, DNA damage caused by UV-B and the subsequent responses, historically, have often been considered general stress or non-photomorphogenic. Other UV-B-specific signaling pathways that function independently of the UVR8 photoreceptor suggest that multiple perception mechanisms exist in plants. Recently, however, plant perception of UV-B radiation and the initiation of photomorphogenic responses outside of the UVR8 pathway have been largely overlooked. Plant responses to UV-B are highly varied. Therefore, the existence of multiple perception pathways seems logical. The objective of this review is to highlight that the absorption of UV-B occurs through a variety of ways, for example through DNA, and induces photomorphogenic responses specific to that absorption that are distinct from the UVR8 signaling pathway.

Contents

1. Introduction

Plants are dependent on a wide array of environmental signals to modulate growth and morphology and have evolved sophisticated systems for perceiving and responding to such stimuli. Among these is the perception of light signals through photoreceptors that absorb light at specific wavelengths. UV-B radiation (280–320 nm) is an especially important component of sunlight. It

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http://dx.doi.org/10.1016/j.envexpbot.2015.12.010
0098-8472/

has the highest energy of the solar spectrum that reaches the earth's surface, making it a unique light stimulus. It can cause damage to biomolecules such as DNA (Britt, 2004; Taylor, 2006), but it also induces classic photomorphogenic responses like hypocotyl growth inhibition (Ballaré et al., 1991; Kim et al., 1998; Shinkle et al., 2004; Gardner et al., 2009), cotyledon expansion (Boccalandro et al., 2001), and leaf development (Brown and Jenkins, 2008; Wargent et al., 2009), among others (reviewed in Frohnmeyer and Staiger, 2003; Ulm, 2006). Tremendous progress has been made in defining UV-B-specific signaling pathways in plants as well as possible perception mechanisms, and this progress has been extensively reviewed elsewhere (Brosché and Strid, 2003; Ulm, 2006; Jenkins, 2009; Heijde and Ulm, 2012; Tilbrook et al., 2013; Ulm and Jenkins, 2015).

DNA damage caused by UV-B and the subsequent responses are well known. Historically, these responses have been considered non-photomorphogenic or as general responses in plants because they are also activated by other stimuli (reviewed in Brosché and Strid, 2003; Frohnmeyer and Staiger, 2003). Recently, the identity of a UV-B photoreceptor in plants was revealed to be UVR8 (Rizzini et al., 2011), a component that was known to function in UV-B-specific signaling (Kliebenstein et al., 2002; Brown and Jenkins, 2008; Favory et al., 2009). This has paved the way for a wealth of subsequent research concerned with elucidating the properties of UVR8 and its mechanism as a photoreceptor, as well as more thoroughly defining a UV-B photoreceptor pathway. However, mechanisms by which plants can perceive UV-B radiation and initiate photomorphogenic responses outside of the UVR8 pathway have been largely overlooked. The existence of several UV-B-specific signaling pathways in plants that are independent of the UVR8 photoreceptor suggests that other perception mechanisms exist (Brown and Jenkins, 2008; Wargent et al., 2009; González Besteiro et al., 2011; Biever et al., 2014). They are recognized to some extent in the literature, but are often ill-defined by the categorical restrictions used to separate the responses. The distinction between "photomorphogenic" and "damage" responses may be helpful for describing the varied effects of UV-B irradiation in plants, but they are perhaps not entirely accurate. Photomorphogenesis is development mediated by light (Briggs and Olney, 2001). Therefore, if signals originating from DNA after absorption of UV-B ultimately converge to regulate processes such as gene expression or the cell cycle, then development or growth is affected and photomorphogenesis has occurred. With that in mind, this review focuses on the initial perception of UV-B radiation in plants that induces downstream processes that ultimately affect growth. In particular, UV-B-induced DNA damage and responses to that damage will be discussed within the context of being a possible pathway for regulating early photomorphogenesis in plants in response to UV-B light (Fig. 1).

2. Historical UV-B research in plants

Although the impacts of solar UV on plant growth have interested scientists for over a century (reviewed in Caldwell, 1971), a research focus on increased UV-B fluxes and their effects on plants was prompted by concerns over decreasing stratospheric ozone, initially discovered in the 1980s (Farman et al., 1985). This was a concern because stratospheric ozone is the main barrier to the earth's surface of solar UV radiation. It is most efficient at absorbing higher energy wavelengths (<290 nm), where UV-C is essentially excluded along with a small portion of UV-B. UV-A and

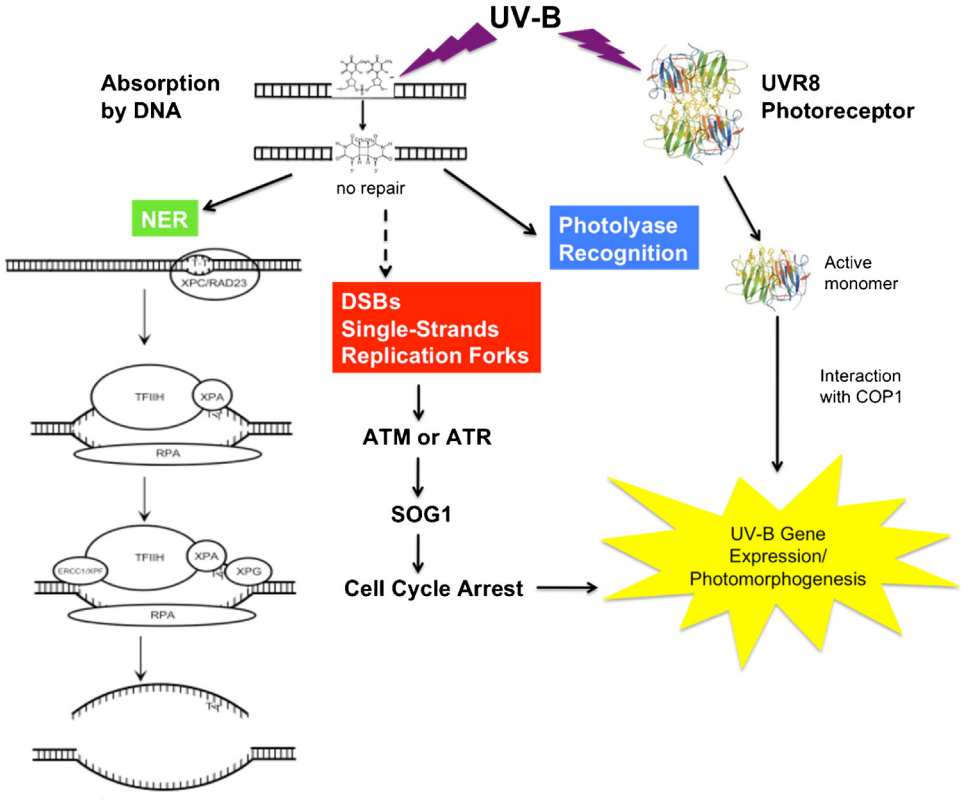

Fig. 1. Proposed UV-B perception pathways in etiolated *Arabidopsis* seedlings. UV-B is directly absorbed by the UVR8 photoreceptor. UVR8 monomerizes and interacts with COP1 to induce expression of genes under the control of HY5/HYH. Concurrently, DNA directly absorbs UV-B light to form photodimers. Repair processes like nucleotide excision repair (NER) and photoreactivation can efficiently repair photoproducts to a degree. Cell-cycle arrest is induced by unrepaired photodimers that are either recognized directly or through double-strand breaks (DSBs) or stalled replication sites by the ATM/ATR-SOG1 signaling pathways. Both mechanisms ultimately affect photomorphogensis. (Image of NER pathway appears in Britt, 2004; structures of UVR8 dimer and monomer appear in Heijde and Ulm, 2012).

the remaining UV-B photons are transmitted through the ozone layer; however, the UV-B wavelengths between 290 and 320 nm are greatly reduced (Ulm, 2006). Therefore, as stratospheric ozone levels decrease, the results are higher fluxes of those wavelengths that already pass through, and the transmission of shorter wavelength UV-B as well (Caldwell and Flint, 1994). It was known from human based research and associated model systems that DNA damage from UV-B is a primary source of skin cancer (Setlow, 1974). By extension, DNA damage caused by UV-B is a potential issue for plants because it could inflict cellular damage and decrease overall plant growth and productivity.

Banning the use of chlorofluorocarbons (CFCs) has helped alleviate the large loss of stratospheric ozone over Antarctica each year (Crutzen and Oppenheimer, 2008), but global levels of stratospheric ozone are in an overall decline (NASA, 1999; Forster et al., 2011). Interactions with greenhouse gases and other chemicals make it difficult to predict future levels and changes in stratospheric ozone (Weatherhead and Andersen, 2006). Therefore, increased UV-B radiation at the earth's surface is still a concern, and understanding how plants perceive UV-B is additionally important, regardless of possible increased fluxes, because it is an inherent component of sunlight and an environmental stimulus for plants.

3. UV-B perception in plants and its effects

The effects of UV-B radiation in plants are varied. Direct absorption of UV-B light by several cellular components leads to downstream effects either directly through that absorption or through indirect consequences. These early effects can manifest in a variety of morphological responses where decreased plant height and biomass accumulation are commonly observed (Jansen et al., 1998; Kakani et al., 2003; Ballaré et al., 2011). The inhibition of hypocotyl elongation is a classic photomorphogenic response (Beggs et al., 1980) and is often used to gauge sensitivity to UV-B light (Kim et al., 1998; Shinkle et al., 2004, 2005; Gardner et al., 2009). UV-B light induces the expansion of cotyledons and can cause curling in the cotyledon (Boccalandro et al., 2001). It also alters leaf expansion and growth (Hopkins et al., 2002; Wargent et al., 2009).

Several genes that encode enzymes in the phenylpropanoid pathway are strongly induced after UV-B irradiation, and the accumulation of flavonoids and anthocyanins helps plants shield UV-B before reaching other cellular components (Robberecht and Caldwell, 1978; Li et al., 1993; Stapleton and Walbot, 1994; Mazza et al., 2000). For example, *uvr8* mutants exhibited lower photosynthetic efficiency due to increased photoinhibition from UV-B irradiation, presumably because they lack flavonoids to screen UV-B light and protect the photosynthetic apparatus. The same *uvr8* plants were severely dwarfed and necrotic compared to wt (Davey et al., 2012). Therefore, increased levels of UV-B may have a significant impact on plant growth, especially if they lack sufficient screening compounds. Plant productivity was an initial concern regarding potential increases in UV-B radiation because irradiating plants with UV-B light mainly resulted in photosynthetic damage, reactive oxygen species (ROS) production, and both direct and indirect DNA damage (reviewed in Jansen et al., 1998).

Most of the UV-B irradiation effects in plants observed under laboratory conditions are unlikely to occur in nature (*e.g.*, UV-C irradiation, artificially high UV-B fluences beyond projected increases, *etc.*), and this has sparked debate as to what effects are relevant to plants under natural environmental conditions (reviewed in Hideg et al., 2013). For example, photosynthetic rates in plants grown under natural conditions have not shown significant differences under changes in UV-B radiation and do not explain the observed plant growth decreases (Ballaré et al., 2011). Studies using pea suggested that reductions in leaf area and biomass after UV-B exposure were the result of a decrease in cell divisions and smaller cell area (González et al., 1998; Nogués et al., 1998), providing evidence that growth inhibition can occur through alterations in cell cycle regulation.

Early hypotheses regarding the perception of UV-B light in plants recognized the possibility of multiple pathways that were likely linked to certain wavelengths due to the dependency of biological responses to particular ranges of UV. When action spectra were normalized to the most effective wavelengths, DNA was the main potential chromophore for a majority of the responses (Caldwell, 1971), and more recent work has provided evidence that DNA could be a sensor for photomorphogenic UV-B responses at shorter wavelengths (Shinkle et al., 2004; Shinkle et al., 2005). However, shorter wavelengths of UV-B (~280–300 nm) are typically regarded as "damaging" because of the higher energy associated with them (Ulm, 2006), so the idea that DNA could act as a specific sensor for UV-B light is not often considered. This is because formation of ROS, DNA damage, or lipid peroxidation by ROS are generally attributed to short wavelength UV-B, and these effects can ultimately trigger pathways responsive to other environmental stresses like wounding or pathogen attack (reviewed in Frohnmeyer and Staiger, 2003; Brosché and Strid, 2003).

Specific UV-B effects that lead to photomorphogenic responses, such as hypocotyl growth inhibition, cotyledon expansion, leaf elongation, or flavonoid biosynthesis, are typically considered as those induced by longer wavelengths (≥300 nm). Because of this distinction, most studies involving UV-B photomorphogenesis now routinely filter out wavelengths lower than 300 nm, which may provide a limited view of how plants actually respond to the full, natural UV-B spectrum. In addition to wavelength dependence, several studies have shown that certain responses are fluence-dependent (Kim et al., 1998; Boccalandro et al., 2001; Shinkle et al., 2004; Kalbina and Strid, 2006; Brown and Jenkins, 2008; Gardner et al., 2009), where responses to lower fluences are photomorphogenic and responses to higher fluences are stress-like. Regardless of specific categorizations of UV-B responses in plants, it is clear that plants perceive UV-B signals *via* multiple mechanisms either directly or indirectly, and the initial signal is the absorption of UV-B radiation.

3.1. Direct UV-B absorption in plants

A number of components in the cell, including proteins and nucleic acids, directly absorb UV-B radiation (Britt, 2004). It is important to emphasize that the direct absorption referred to in this review is the absorption of wavelength-specific UV-B photons that causes the excitation of electrons resulting in rearrangements of molecules (Clayton, 1970). This includes conformational changes in proteins that can be reversed and is distinct from UV-C and ionizing radiation, like gamma or X-rays, which have enough energy to release electrons from molecules, usually resulting in permanent changes.

The direct absorption of UV-B light by DNA is especially critical due to the formation of photodimers (discussed in more detail below) that create distortions in the DNA strand that block transcription and replication. Unrepaired photodimers can lead to mutations that threaten genome integrity as well as overall plant growth (Ries et al., 2000b). Consequences of damage products produced in RNA or through the direct absorption of UV-B light by cellular proteins are largely unknown and are not an extensively studied area. This is distinct from the identification of the UVR8 protein as a UV-B photoreceptor in plants (Rizzini et al., 2011) that absorbs UV-B directly and controls the transcriptional induction of genes involved in the production of flavonoids and

other genes regulated by the transcription factor HY5 (Brown et al., 2005). Flavonoids produced in the epidermis of leaves in response to UV radiation presumably absorb UV-B light directly, as well, to screen the radiation before it can damage cellular components in deeper layers (Robberecht and Caldwell, 1978; Li et al., 1993; Stapleton and Walbot, 1994). Flavonoid absorption *per se* is not thought to be informational, in that the energy from the absorbed UV-B photon is captured within the molecule (Edreva, 2005) and is not known to affect downstream processes or growth.

UV radiation can also activate cell membrane receptors involved in apoptosis in human cells (Kulms and Schwarz, 2002). Evidence suggesting a similar activation in plants has demonstrated the initiation of mitogen-activated protein kinase (MAPK) signals by UV-B (Stratmann, 2003; Holley et al., 2003; Ulm et al., 2004). More recently, the MKP1-regulated MAPK pathway was shown to operate independently of UVR8 (González Besteiro et al., 2011). This further demonstrates the involvement of mechanisms for UV-B perception in plants directly activated through UV-B absorption that are not limited to absorption by UVR8.

3.2. Indirect effects of UV-B absorption in plants

Perception of UV-B light also occurs indirectly. In addition to direct absorption of UV-B by photosynthetic components, disruption of photosynthetic processes is a common indirect effect of UV-B light exposure (Bornman, 1989; Day and Vogelmann, 1995; A.-H.-Mackerness et al., 1997). Photosynthetic electron transport is mainly inhibited through degradation of the D1 and D2 proteins of photosystem II (PSII) after UV-B irradiation (Jansen et al., 1996; Vass et al., 1996). However, photoinhibition can also occur through damage to PSI (Powles, 1984) and has been implicated as a potential source of ROS (Takahashi and Murata, 2008). ROS production is a common observation after UV-B irradiation in light-grown plants (Dai et al., 1997; A.-H.-Mackerness et al., 1998). ROS mainly affects membranes through lipid peroxidation, but ROS can also oxidize proteins, RNA, and DNA, and critical levels of the oxidation products will eventually lead to cell death (Mittler, 2002). ROS can function as systemic signals for several environmental stimuli, but this signal has not been documented in response to UV-B irradiation directly (Miller et al., 2009). Due to several links between ROS and gene expression changes (Krizek et al., 1993; Rao et al., 1996; Surplus et al., 1998; Kalbina and Strid, 2006), it is likely that a UV-B-induced systemic signaling pathway for ROS does exist in plants (A.-H.-Mackerness, 2000).

Chalcone synthase (CHS) catalyzes the first reaction devoted to flavonoid biosynthesis, and its gene expression is strongly up-regulated by UV-B irradiation. Accumulation of flavonoids and anthocyanins is a common response to UV-B exposure in plants. A suite of phenylpropanoid compounds accumulates in response to several environmental stresses such as herbivory, pathogen attack, or low temperatures (Dixon and Paiva, 1995). Although there is UV-B-specific flavonoid and anthocyanin production, synthesis of these molecules occurs after visible light exposure as well, as evidenced by *CHS* induction by blue and red light (Frohnmeyer et al., 1992; Christie and Jenkins, 1996). *UVR8* is required for the synthesis of flavonoids specifically after UV-B irradiation through the transcriptional induction of *CHS* and other biosynthetic genes involved in the phenylpropanoid pathway (Brown et al., 2005).

DNA repair mechanisms are ultimately activated to eliminate photodimers created by the direct absorption of UV-B light and oxidation products due to interactions with ROS formed as the result of UV-B irradiation. Photodimers can be directly reversed through photoreactivation with exposure to blue/UV-A light (Sancar, 1994), which is a process unique to this type of DNA damage. There are also general mechanisms like nucleotide excision repair (NER) or homologous recombination (HR) that repair all types of DNA damage. An accumulation of any unrepaired lesions will trigger DNA damage signaling pathways mediated by ATM and/or ATR that recognize double-strand breaks or blocked replication and transcription sites (discussed in more detail below). The consequence of DNA damage accumulation after UV-B exposure is mostly blocked replication (Culligan et al., 2004). The induction of DNA damage repair transcripts after UV-B reflects those mostly related to homologous recombination and, to a lesser extent, double-strand breaks (Missirian et al., 2014). NER components are involved in other processes and found in most plant tissues at low levels without much induction after UV-B irradiation (Mannuss et al., 2012).

4. UV-B induced DNA damage

When DNA absorbs UV-B light directly, energy from the photons causes covalent linkages between adjacent pyrimidine bases creating two main photoproducts, cyclobutane pyrimidine dimers (CPDs) and pyrimidine-6,4-pyrimidinone dimers (6,4PPs). Further exposure to UV irradiation causes photoisomeration of 6,4PPs into the Dewar photoproduct (Mitchell 1988; Takeuchi et al., 1998). In humans, DNA is the primary molecule that absorbs UV-B radiation, and DNA damage is the source of several downstream effects such as sunburn and skin cancer (Kulms and Schwarz, 2002). Plants do not develop cancer (Doonan and Sablowski, 2010), but disruption of the cell cycle can occur in response to UV-B-specific DNA damage (Jiang et al., 2011; Biever et al., 2014), and programmed cell death can be activated if DNA damage accumulates to a critical level in certain plant tissues (Fulcher and Sablowski, 2009; Furukawa et al., 2010).

CPDs are by far the most abundant dimers and are produced ~10× more efficiently than 6,4PPs (Taylor, 2006). UV-C radiation can reverse CPDs, but CPDs do not absorb UV-B, which make them fairly stable in natural light conditions (Taylor, 2006) and may be the reason why they are preferentially repaired in the light (Britt et al., 1993). On the other hand, 6,4PPs absorb maximally at 325 nm and are much less stable in sunlight (Taylor, 2006). Conversion of the 6,4PP to the Dewar photoisomer efficiently occurs by UV light at 325 nm, and both photodimers are rapidly removed by photoreactivation or NER (Mitchell, 1988; Takeuchi et al., 1998). 6,4PP repair was shown to be more rapid in the dark (Britt et al., 1993), possibly because of its more labile presence in the light.

4.1. Repair of photodimers

Plants are well equipped to cope with DNA damage and have evolved efficient repair mechanisms because they cannot simply move to avoid harmful radiation from the sun. They have two main repair mechanisms for photodimers: (a) photoreactivation and (b) nucleotide excision repair (NER). Photoreactivation occurs only for UV-B photodimers. CPD- or 6,4PP-specific photolyases reverse photodimer formation and restore the original bases using energy from UV-A or blue light (Sancar, 1994). This direct binding and reversal of photodimers is largely why plants are so efficient at repairing photodimers, making photoreactivation the more favorable for photodimer repair because an error that may result in a mutation is less likely to occur. Plants contain two different photolyases that specifically bind either CPDs or 6,4PPs but not both. At this time, an enzyme specific for Dewar photoproducts has not been identified. Expression of the CPD photolyase (PHR1) is induced by white light or UV-B, but the 6,4PP photolyase (UVR3) is constitutively expressed (Chen et al., 1994; Waterworth et al., 2002). The CPD photolyase appears to be regulated by HY5, under the control of the UVR8 photoreceptor signaling pathway (Brown et al., 2005; Brown and Jenkins, 2008; Li et al., 2015). Recent work

has shown that both photolyase genes are under transcriptional control by HY5/HYH and induced upon light exposure (Castells et al., 2010), but the requirement for UVR8 was not tested. There is little repair of CPD photodimers in the dark (Britt et al., 1993), and light-dependent repair seems to be the dominant pathway for their removal (Chen et al., 1994). In contrast, 6,4PPs are more efficiently removed in the dark *via* NER, rather than through photoreactivation (Britt et al., 1993). However, this may not be the case for all plant species (Hada et al., 1996). Why CPD and 6,4PP repair may be favored by one repair mechanism over another is unclear. 6,4PPs cause more of a disruption to the DNA strand (Taylor, 2006), which may be more of a problem for transcription and replication processes and could explain why NER is so efficient at removing this photodimer in plants (Britt et al., 1993; Mitchell et al., 1985). CPD formation is more efficient during light exposure, so photoreactivation, a light-dependent process, may be more necessary than the light-independent NER mechanism for CPD repair.

Nucleotide excision repair (NER) is a more universal mechanism that repairs other DNA damage products in addition to UV-B photodimers. It functions without the need for light energy, and several enzymes are involved (Table 1), resulting in the excision of a small strand of bases flanking, and including, the photodimer. The remaining gap is filled through the normal replication process. This method of repair is considered to be more "error-prone" because it must refill a gap of about 30 nucleotides and disrupts more of the original DNA strand. It can occur throughout the genome as global genomic repair (GGR) or as a more directed process coupled with transcription (TCR; Britt, 2002). Most of the information regarding the mechanism of NER has been worked out in human cell cultures, *Escherichia coli*, or yeast (Sancar and Smith, 1989; Sugasawa et al., 2001; Volker et al., 2001; Wang et al., 1993; You et al., 2003), and the knowledge regarding the specific biochemistry of the NER pathway in plants remains limited (Li et al., 2002).

Both photoreactivation and NER contribute to a plant's tolerance to UV-B radiation. *Arabidopsis* mutants of the photolyases and NER enzymes are hypersensitive when irradiated with UV-B or UV-C by displaying necrosis and decreased growth (Britt et al., 1993; Harlow et al., 1994; Jiang et al., 1997; Landry et al., 1997; Liu et al., 2000, 2001). Mutations in the 5′- and 3′-endonucleases involved in NER, in particular, seem to have the most dramatic effect on *Arabidopsis* growth under UV-B (Britt et al., 1993; Harlow et al., 1994; Gardner et al., 2009; Biever et al., 2014). Because NER components ultimately recognize single-stranded DNA at stalled replication or transcription sites or the other proteins involved in those processes, they usually have roles in other types of damage repair (Kunz et al., 2005). This means that mutations of NER components may lead to general growth consequences, so when plants are exposed to UV-B, it is not surprising that those mutants are especially sensitive.

Homologous recombination (HR) seems to, in part, be responsible for the removal of CPDs (Ries et al., 2000a,b), but not 6,4PPs. UV-stimulated homologous recombination (HR) activity was proportional to the amount of CPDs formed and dependent on photosynthetically active radiation but independent of the CPD photolyase (Ries et al., 2000b). CPD formation occurs at a much higher frequency than 6,4PPs, and this may be the reason that CPDs are the main photodimer targeted for HR (Ries et al., 2000b). However, a lack of data linking HR events to 6,4PPs cannot exclude HR as a possible repair mechanism for this photodimer as well. HR is likely a more secondary process for removal of photodimers. A study using a mutant lacking the CENTRIN2 protein, which stabilizes the photodimer recognition complex involved in NER, showed increased HR (Molinier et al., 2004), indicating that HR is more prominent only when other repair processes are inhibited.

5. DNA damage response signaling pathways

The detection of DNA damage is an important process for resistance and tolerance to environmental factors causing damage, in particular UV-B radiation (Culligan et al., 2004). An elaborate network of proteins is employed to recognize the damage and initiate a signaling cascade that inhibits progression of the cell cycle to limit the proliferation of potential mutations. This network is a conserved response among several organisms (Melo and Toczyski, 2002) and activated through the recognition of double-strand breaks or single-stranded DNA at replication forks by the protein kinases ATAXIA-TELANGIECTASIA MUTATED (ATM) and ATM AND RAD3-RELATED (ATR), respectively (Garcia et al., 2003; Culligan et al., 2004). As previously mentioned, the accumulation of unrepaired UV-B-photodimers results in stalled replication sites and, to a lesser extent, double strand breaks (Molinier et al., 2004), both of which activate DNA damage responses. SUPPRESSOR OF GAMMA 1 (SOG1) is a plant-specific transcription factor in this pathway and could be analogous to p53 in mammalian systems (Yoshiyama et al., 2009). SOG1 is necessary for downstream signaling from ATM and ATR and is required for transcriptional

Table 1
Arabidopsis genes involved in nucleotide excision repair (NER) and photoreactivation: a non-comprehensive list of the major components involved in damage recognition and early steps of NER.

Gene name and designation	Description/function	Reference
UVH3/UVR1 (At3g28030)	XPG/RAD2 homolog; 3′ DNA-specific endonuclease involved in NER	Liu et al. (2001)
UVH1/XPF (At5g41150)	XPF/RAD1 homolog; 5′ DNA-specific endonuclease involved in NER, functions with ERCC1/RAD10	Liu et al. (2000)
UVR7/ERCC1 (At3g15620)	ERCC1/RAD10 homolog; 5′ DNA-specific endonuclease involved in NER, functions with XPF/RAD1	Hefner et al. (2003)
UVH6 (At1g03190)	XPD/RAD3 homolog; DNA helicase involved in NER	Lui et al. (2003)
UVR2/PHR1 (At1g12370)	PHR1, CPD photolyase	Ahmad et al. (1997)
UVR3 (At3g15620)	6,4PP photolyase	Nakajima et al. (1998)
CENTRIN2 (At4g37010; At3g50360)	Modulates NER and homologous recombination (HR) pathways; interacts directly with RAD4	Molinier et al. (2004)
XPC (At5g16630)	RAD4 homolog; interacts with CEN2 and RAD23 in DNA damage recognition	Liang et al. (2006)
RAD23 (At1g79650; At1g16190; At3g02540; At5g38740; At5g16090)	HR23A,B homolog; stabilizes DNA damage recognition complex (XPC) in NER	Farmer et al. (2010)
RPA (At4g19130; At5g45400; At2g06510; At5g61000; At5g08020; At2g24490; At3g02920)	Replication protein A; binds and stabilizes single-stranded DNA	Kunz et al. (2005)

responses after gamma irradiation (Preuss and Britt, 2003; Yoshiyama et al., 2009). It may also function independently of ATM and ATR pathways in UV-B-specific DNA damage signaling (Biever et al., 2014).

Recently, Biever et al. (2014) showed that hypocotyl growth inhibition induced by UV-B light in etiolated *Arabidopsis* seedlings is influenced by signals originating from UV-B absorption by DNA that eventually trigger cell-cycle arrest. The authors used *Arabidopsis* mutants of the NER endonucleases *xpf-3* and *uvr1-1* that showed hypersensitivity to UV-B in terms of hypocotyl growth inhibition. What was striking about the hypersensitivity in these mutants was that it occurred at relatively lower fluences (3000–10, 000 μmol m^{-2} s^{-1}) compared to the wild type (Biever et al., 2014), which indicated that UV-B induced photodimer formation could be responsible for a photomorphogenic response (*e.g.*, hypocotyl growth inhibition) in etiolated seedlings. This idea was further tested using the *suppressor of gamma 1* (*sog1-1*) mutant (Preuss and Britt, 2003) that lacks a transcription factor responsible for gene induction and cell-cycle arrest after gamma irradiation in *xpf* mutants. UV-B-induced hypocotyl growth inhibition in the *sog1-1* mutant was similar to wild type, but the *xpf sog1-1* double mutant did not exhibit the hypersensitivity of *xpf*, showing that DNA damage response signaling governed by SOG1 was likely activated by UV-B-specific DNA damage accumulation (*i.e.*, photodimers). The ultimate effect of DNA damage responses is cell-cycle arrest. This was measured directly using a Col wt line containing a *CYCB1;1-GUS* reporter construct (Colon-Carmona et al., 1999). The accumulation of CYCB1;1-GUS after UV-B irradiation was apparent and consistent with the timeline for hypocotyl growth inhibition (Biever et al., 2014). In addition, the process initiated by DNA damage occurred independently of *UVR8* and its signaling pathway responsible for *CHS* induction. The *xpf-3* mutant showed CHS induction that was similar to wt. Hypocotyl growth inhibition by UV-B light in etiolated *uvr8* mutants was not different from wt, but a lack of *CHS* induction in these mutants was maintained. This work adds to the limited literature that provides evidence for a photomorphogenic pathway that is triggered by UV-B-induced photodimer formation and is independent of a known UV-B photoreceptor. It further shows that DNA damage can induce specific UV-B responses that are not simply those initiated by general plant stress.

Most DNA damage response (DDR) pathways in plants have been determined by studies using gamma irradiation to inflict damage, and the ultimate effect of DNA damage signaling is growth arrest through alteration of the cell cycle. UV-B induced the same signaling pathways that lead to programmed cell death in the root apical meristem after gamma irradiation (Furukawa et al., 2010). Gamma irradiation also initiated these pathways in the shoot primordia (Fulcher and Sablowski, 2009), but UV-B-induced DNA damage, specifically, was not studied. However, the existence of these signaling pathways shows that UV-B-induced DNA damage could affect plant growth in this way. Instead of cell-cycle arrest, DDR can cause cells to enter endoreduplication cycles (Adachi et al., 2011). Endoreduplication may be important for UV-B tolerance in certain plant tissues, but the involvement of the full suite of DDR components is unknown. UV-B irradiation stimulated endoreduplication rather than cell-cycle arrest in *Arabidopsis* leaves and was dependent on UVR8 (Wargent et al., 2009). The *uvi4* mutant isolated in *Arabidopsis* was less sensitive to UV-B irradiation than the wt because of additional endoreduplication rounds in the hypocotyl (Hase et al., 2006).

6. Perception of UV-B by UVR8

The UV-B specific signaling pathway regulated by UV RESISTANCE LOCUS 8 (UVR8) is probably the most characterized mechanism regarding photomorphogenic responses to UV-B in plants. The *uvr8-1* mutant was originally isolated as being more sensitive to UV-B than the wild type when grown in the light (Kliebenstein et al., 2002). *uvr8* mutants are deficient in UV-B specific *CHS* induction and also show increased levels of PR1 and PR5 (Kliebenstein et al., 2002; Brown and Jenkins, 2008), proteins involved in responses such as defense against pathogens. In addition, UVR8 regulates expression of the transcription factors ELONGATED HYPOCOTYL5 (HY5) and its homolog HYH (Brown et al., 2005; Brown and Jenkins, 2008) by directly interacting with CONSTITUTIVELY PHOTOMORPHOGENIC1 (COP1) during UV-B exposure. This interaction inhibits a repressor of UVR8 that is associated with *HY5/HYH* chromatin and allows activation of these transcription factors and subsequent genes under their control (Favory et al., 2009). The HY5 interaction with promoters of its target genes is enhanced by UV-B and requires UVR8 (Binkert et al., 2014).

Accumulation of UVR8 in the nucleus occurs shortly after UV-B irradiation (Kaiserli and Jenkins, 2007). The mechanism for UVR8 translocation into the nucleus has yet to be determined, but UVR8 also, constitutively and independently of UV-B radiation, binds to chromatin (Cloix and Jenkins, 2008). UVR8 is mainly located in the cytoplasm, but there is at least a small pool of UVR8 that already exists in the nucleus (Kaiserli and Jenkins, 2007). However, expression of genes regulated by UVR8 requires UV-B exposure (O'Hara and Jenkins, 2012). *UVR8* itself is not induced by UV-B and protein levels remain constant in dark grown compared to light grown plants (Kaiserli and Jenkins, 2007; Rizzini et al., 2011; O'Hara and Jenkins, 2012).

6.1. Mechanism for UV-B perception by UVR8

UVR8 was recently demonstrated to act as a UV-B photoreceptor *in vitro* (Rizzini et al., 2011). Early characterization of *UVR8* showed it was homologous to the human gene *REGULATOR OF CHROMATIN CONDENSATION* (*RCC1*), which is a guanine nucleotide exchange factor for the G-protein Ran (Kliebenstein et al., 2002), but this activity has not been observed in plants. UVR8 interacts with itself to form a dimer that monomerizes upon UV-B irradiation *in vitro* (Rizzini et al., 2011). Biochemical analyses demonstrated that specific tryptophan residues were required for dimer formation and formed the chromophore for UV-B absorption (Christie et al., 2012; Wu et al., 2012). Specifically, a "tryptophan pyramid" forms between UVR8 monomers and is surrounded by charged and other aromatic residues that create salt bridges at the dimer interface. Monomerization occurs when the cross-dimer salt bridges are disrupted through UV-B light absorption by the tryptophan pyramid (Christie et al., 2012; Miyamori et al., 2015). The monomer is the active form and binds to COP1 to regulate downstream gene expression (Fig. 1) (Favory et al., 2009; Rizzini et al., 2011). UVR8 contains a β-propeller domain that is necessary for UV-B dependent interaction with COP1, but UV-B-specific signaling and regulation requires a separate domain found in the C-terminus of UVR8 (Cloix et al., 2012; Yin et al., 2015).

The unique cluster of tryptophans at the center of the protein was originally hypothesized to be required for dimerization and interaction with COP1 because two of the tryptophans that were mutated to alanine lost the ability to form dimers but retained their interaction with COP1 (Rizzini et al., 2011). One particular mutation, $UVR8^{W285A}$, constitutively interacted with COP1, but did not form dimers. $UVR8^{W285F}$ did form dimers but was unresponsive to UV-B and showed no interaction with COP1 (Rizzini et al., 2011). It would seem that the $UVR8^{W285A}$ would show constitutive responses to UV-B that are regulated by UVR8 such as expression of *HY5* or *CHS*, but interestingly, *in vivo* experiments showed that

these mutants were phenotypically similar to *uvr8* mutants by lacking *HY5* and *CHS* expression and hypocotyl growth inhibition after UV-B irradiation (O'Hara and Jenkins, 2012). Biochemical analysis demonstrated that the $UVR8^{W285A}$ mutant was structurally very similar to the wt UVR8 dimer (Christie et al., 2012), which would explain the lack of downstream responses initiated by $UVR8^{W285A}$ after UV-B exposure previously reported (O'Hara and Jenkins, 2012). More recently, however, $UVR8^{W285A}$ showed constitutive photomorphogenic responses to UV-B (Heijde et al., 2013). Responses, such as constitutive expression of *HY5* and *CHS* would be expected to some degree, as well, based on the results mentioned above regarding UVR8 binding to chromatin independent of UV-B. Whether it was the dimer or monomer that was constitutively bound to chromatin, however, was not specified (Cloix and Jenkins, 2008). Jenkins (2014) has provided a thorough review of UVR8 structure and function.

6.2. UVR8-independent responses specific to UV-B

There are documented UV-B-specific responses that occur independently of UVR8, demonstrating that UV-B perception in plants must occur *via* multiple mechanisms. Brown and Jenkins (2008) described a high-fluence rate response in *Arabidopsis* leaves that induced gene expression specifically in response to UV-B irradiation but did not require *UVR8*. The three genes identified in this category were *WRKY30* (At5g24110), *UDPgtfp* (At1g05680), and *FAD oxred* (At1g26380). Both *UDPgtfp* and *FAD oxred* are known to be up-regulated by H_2O_2 (Inzé et al., 2011). Not much is known about *WRKY30* specifically, but *WRKY* transcription factors, in general, regulate a wide range of plant processes, and they function most notably in plant immunity, defense, and leaf senescence (Pandey and Somssich, 2009; Besseau et al., 2012). Because of the implicated functions of these genes and the fact that their expression was observed after irradiation with the highest UV-B fluences tested, it was concluded that this response likely overlaps with oxidative stress or wound signaling pathways (Brown and Jenkins, 2008). The overlap of UV-B-specific signaling with such pathways has been the subject of many studies (reviewed in Brosché and Strid, 2003; Frohnmeyer and Staiger, 2003).

Signal transduction from several different stress responses converge by activating mitogen-activated protein kinase (MAPK) networks (Holley et al., 2003). The signaling network involving MAP kinase phosphatase 1 (MKP1), in particular, is activated by UV-B irradiation and is independent of UVR8 (Holley et al., 2003; Kalbina and Strid, 2006; González Besteiro et al., 2011). The *mkp1* mutant was originally identified by its hypersensitivity in terms of root growth to genotoxic stress caused by UV-C irradiation (Ulm et al., 2001). Whether MAPK pathways are activated by UV-induced DNA damage directly, by ROS, or by other signals is unknown.

7. Regulation of UV-B light perception and responses

Plant responses to signals from the environment are ultimately regulated by downstream components that control gene expression or other aspects of growth. The E3 ubiquitin ligase, COP1, is a main regulator of photomorphogenesis, specifically (Deng et al., 1991), along with DE-ETIOLATED 1 (DET1; Chory et al., 1989) that targets other proteins for degradation. COP1/DET1 are negative regulators of light-mediated development because both mutants display light-grown phenotypes when grown in the dark. COP1's regulation of UV-B photomorphogenesis is different from other types of light as it typically degrades the transcription factor HY5 in the dark and, upon light exposure, is inhibited allowing HY5 to induce transcription of genes under its control (Oravecz et al., 2006; Favory et al., 2009). The photoreceptor UVR8 interacts directly with COP1 to promote UV-B photomorphogenesis in plants through transcriptional induction of *HY5* and, subsequently, the induction of genes that require HY5 (Favory et al., 2009).

Negative regulation of the UVR8-mediated UV-B signaling pathway is controlled by *RUP1* and *RUP2* (Gruber et al., 2010). The REPRESSOR OF UV-B PHOTOMORPHOGENESIS (RUP) proteins are highly homologous to one another and contain WD40-repeats similar to COP1. Transcription is induced for each one by UV-B light and dependent on UVR8-COP1 interaction and HY5. However, other types of light induce *RUP1* and *RUP2*, so they may have a more general role in light responses (Gruber et al., 2010). Induction of *CHS* after UV-B irradiation is much higher in the *rup2* mutant and is basically abolished in overexpression lines (Gruber et al., 2010). *rup1rup2* hypocotyl growth inhibition after UV-B light exposure is much more severe than wild type, but these plants seem to be more readily acclimated to UV-B (Gruber et al., 2010). RUP1 and RUP2 could physically facilitate UVR8 redimerization after UV-B-induced monomerization, which "turns off" UVR8-controlled photomorphogenesis (Heijde and Ulm, 2013). The RUP1 and RUP2 proteins bind the C27 domain of UVR8, and this happens in the absence of UV-B light (Cloix et al., 2012). Although the UVR8-signaling pathway is activated by UV-B irradiation, it appears that its regulation by RUP1/RUP2 is not.

DNA repair proteins are also under regulatory control by DET1 and COP1. Both DET1 and COP1 regulate the expression of the photolyase genes *PHR1* and *UVR3* by degrading HY5/HYH in the dark. *det1* mutants were more tolerant to UV-C irradiation due to a combined effect of increased expression of the photolyase genes and genes involved in the phenylpropanoid pathway (Castells et al., 2010). DET1 is also required for proper nucleotide excision repair through associations with the photodimer recognition factors DDB2 and CSA that detect conformational changes in the DNA strand or stalled RNA polymerases, respectively (Castells et al., 2011). Both proteins interact with CUL4-DDB1 complexes, which associate with DET1 during normal *Arabidopsis* development and are necessary for UV tolerance (Al Khateeb and Schroeder, 2007, 2009; Biedermann and Hellmann, 2010). The CUL4-DDB1-mediated degradation of DDB2 required ATR, indicating that DDB2 regulation is also linked to checkpoint responses (Molinier et al., 2008). The results of these studies are important because they provide evidence that DNA repair processes and DNA damage signaling are necessary for proper plant development and are under control of DET1 and COP1, major components that regulate photomorphogenesis.

8. Conclusions and future directions

Research leading to knowledge regarding how plants perceive and respond to UV-B radiation has made substantial progress in the last few years, especially with the characterization of UVR8 as a UV-B photoreceptor and further definition of its signaling pathway (Jenkins, 2014). While UVR8 no doubt plays a major role in UV-B photoperception (Christie et al., 2012), it cannot explain nor account for all UV-B responses observed in plants (Gardner et al., 2009; Wargent et al., 2009; González Besteiro et al., 2011; Biever et al., 2014). Plant responses to UV-B radiation are highly varied, and the existence of multiple perception pathways seems logical. While this idea is accepted to some degree, previous categorization of plant UV-B responses limits room for interpretation regarding "damage-like" or "photomorphogenic" effects. It seems naive to assume that plants would contain a single photoreceptor system for UV-B light, when plants have redundant or homologous photoreceptors for other light qualities. As highlighted throughout this review, the absorption of UV-B occurs through a variety of processes and induces responses specific to that absorption, including induction of photomorphogenic responses through perception mechanisms other than the UVR8 signaling pathway.

As discussed in Section 5, there is evidence that photomorphogenic responses, such as the inhibition of hypocotyl growth in etiolated *Arabidopsis* seedlings, are influenced by UV-B-specific DNA damage and do not require UVR8. This evidence reinforces the idea that multiple UV-B perception mechanisms exist in plants that could be more analogous to UV-B perception in human cells. The parallels to UV-B perception in humans were how initial UV-B perception hypotheses were formed for plants (Caldwell, 1971). More importantly, results from Shinkle et al. (2005) and Biever et al. (2014) suggest the possibility that a UV-B perception pathway initiated by UV-B-specific DNA damage can influence photomorphogenic growth in plants, rather than being a general stress response that is not necessarily specific to UV-B or part of UV-B-specific signaling.

In initial plant development, a germinating seedling extending out of the soil will have minimal synthesis of flavonoids due to the lack of prior light exposure and little protection from the first sunlight exposure making it more vulnerable to UV-B light. The UV-B light present in solar radiation is likely absorbed more readily by DNA at this stage leading to photodimer formation. If the recognition of photodimers occurs by DNA repair enzymes involved in either NER or photoreactivation, then downstream processes that require ATM, ATR, or the transcription factor SOG1, which eventually lead to growth inhibition through cell-cycle arrest, might be activated. UVR8 is required for UV-B-dependent production of flavonoids and, as the plant continues to grow, is important for protection from UV-B light. However, the UV-B perception pathway initiated through direct absorption by DNA is still relevant because some UV-B light would continue to pass through the leaf and reach the inner cellular components. These two pathways are distinct UV-B perception mechanisms, operating in tandem, to influence plant growth.

To fully determine how UV-B-induced photodimer formation influences plant growth, more sophisticated techniques for detecting and quantifying photodimers are needed, such as previously developed LC-MS methods (Douki et al., 2000). Despite the growing body of literature describing DNA repair processes in plants, there remains a lot left to decipher in terms of biochemistry and sequence of events. The endonucleases involved in NER also have documented functions in other DNA repair processes (Bardwell et al., 1994; Gallego et al., 2000). It could be that these enzymes are important for recognition and initiating DNA damage signaling downstream. Even though these enzymes may function in more general growth responses, they seem to have specific responses to UV-B light. The core proteins that are required for initial recognition of DNA damage based on studies in yeast and humans are XPC, Rad23B, XPA, RPA, TFIIH, and CENTRIN2 (reviewed in Kunz et al., 2005). Analysis of their functions in plants will help provide a comprehensive view of the exact steps from direct photodimer detection to cell-cycle arrest or other downstream effects. Plants contain genetic homologs of all of the listed proteins except XPA. There has been limited research on their biochemical functions in plants to determine whether they play a similar role to that in other systems. More work is necessary to help fully understand the involvement of ATM and ATR in UV-B-specific DNA damage signaling. Because the persistence of photodimers mostly leads to replication blocks, ATR is likely the major component. Focus on its role should help determine the specific link between UV-B-specific DNA damage recognition and ultimate downstream consequences. The ultimate regulation or influence on the cell cycle is a particularly interesting outcome of DNA damage signaling. Inhibition of auxin transport could also be a contributing factor to hypocotyl growth inhibition after UV-B irradiation. Since auxin also influences the cell cycle, measuring auxin transport could provide more insight into the regulation of the UV-B-induced hypocotyl growth response through possible interference from flavonoids (Stenlid, 1976; Jacobs and Rubery, 1988; Gardner and Sanborn, 1989; Brown et al., 2001; Hectors et al., 2012) or direct effects on the cell cycle. Exploration into how more precisely UV-B or other environmental stimuli control the cell cycle and the other components involved is an area for future research.

Characterizing plant perception of UV-B and subsequent responses is an important part in understanding how plants respond to their light environment in general. The understanding gained from this work may help researchers better predict how changes in the light environment, such as potential increased fluxes of UV-B, will affect plant growth to better determine how plants will respond overall and adapt to a changing environment.

Acknowledgements

The authors would like to thank the Minnesota Agricultural Experiment Station, the University of Minnesota Graduate School, and the Plant Biological Sciences Graduate Program for financial support.

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