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CELL BIOLOGY

Damaging and Defense Processes Induced in Plant Cells by UVB Radiation

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Abstract—UVB radiation (290–320 nm) activates various signaling mechanisms triggering the processes of programmed cell death in plants or their protection against the damaging action of this type of radiation. In the case of high dosages of UVB radiation, the mechanisms of cell death are associated with DNA damage and oxidative stress. In the first case, activation of DNA damage checkpoints and cell cycle arrest may occur; in the second case, cytochrome *c* is released from mitochondria with the subsequent activation of metacaspases. According to the existing data, both mechanisms induce DNA fragmentation and other changes typical for apoptotic cells, while low-intensity UVB radiation, which is perceived by a UVR8 photoreceptor, initiates protective processes promoting plant acclimatization to sunlight.

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INTRODUCTION

Sunlight is the source of energy required for the photosynthetic process in plants and is also an important adaptive stimulus. It allows plants to adapt to lighting conditions including changes in the intensity of ultraviolet B (UVB, 290–320 nm) radiation (Paul and Gwynn, 2003; Caldwell *et al.*, 2007; McKenzie *et al.*, 2007). Plants react to light signals through special photoreceptors, which initiate transduction of light signals to biochemical signaling cascades. As a result, these signaling pathways cause various photomorphogenetic responses and also initiate defense reactions and processes providing plant resistance to the UVB part of the sunlight (Frohnmeyer and Staiger, 2003; Ulm and Nagy, 2005). The known regulatory photoreceptors include phytochromes representing sensors for the red (R) and far red (FR) light (600–750 nm), cryptochromes and phototropins sensitive to the blue (400–500 nm) and UVA (320–400 nm) parts of the spectrum, and the UVR8 (UV resistance locus 8) protein functioning as a specific sensor of photons from the UVB region (Fraikin et al., 2013). This protein was identified as a UVB photoreceptor of plants and was characterized at the molecular level (Rizzini *et al.*, 2011).

The percentage of UVB radiation that is actively filtered by the ozone layer, in full-spectrum sunlight reaching the Earth's surface is 1.5%. However, UVB photons are high-energy particles, so UVB radiation provides the strongest damaging effect on the growth and development of plants (Caldwell *et al.*, 2007; Jenkins, 2009). The ozone layer represents a key component of the environment able to reduce this damaging effect, though some anthropogenic factors may cause its depletion. At present the global ozone level is lower than at the beginning of 1970s and its recovery is not expected in the next few decades (McKenzie *et al.*, 2007). Accordingly, a significant increase in the level of UVB radiation in the biosphere was observed (Ballare *et al.*, 2011). This tendency makes it possible to suppose that living organisms will continually undergo the action of high-intensive UVB radiation at increasing dosages.

Plants differentially respond to the intensity of UVB radiation (Ulm and Nagy, 2005). At low-intensity UVB radiation, photons regulate expression of various genes by a UVR8 photoreceptor (Morales *et al.*, 2013). Some of these genes are of special importance for plant adaptation to UVB radiation; they include genes encoding enzymes involved in the biosynthesis of flavonoids, which shield epidermal tissues from this type of radiation, and enzymes providing for photorepair of UVB-induced DNA damage (Jenkins, 2009).

Highly intensive UVB radiation causes various molecular damages in cell structures accompanied by the impairment of their functions (Brosche and Strid, 2003; Frohnmeyer and Staiger, 2003; Jenkins, 2009). Taking into account the biological consequences of the impact of UVB radiation on cells, DNA damage associated with the formation of some photoproducts, mainly pyrimidine dimers, is of special importance (Cadet *et al.*, 2015).

DNA damage triggers a complex signaling mechanism activating DNA damage checkpoints, which, in turn, arrest the cell cycle. On the one hand, such a response increases the lifetime of DNA repair enzymes; on the other hand, it may trigger programmed cell death (PCD) to destroy cells with nonrepaired DNA. This signaling pathway described by the phrase "response to DNA damage" represents both a fundamental process of the DNA protection in the cell and the mechanism of transfer of correct genetic information from one generation to another (Ciccia and Elledge, 2010).

The molecular mechanisms of DNA damage responses were studied mainly in yeasts and animals. Recently started detailed study of such mechanisms in plants, including those activated in response to UVB radiation, is facilitated by the sequencing of some plant genomes. Based on the results of such sequencing, researchers revealed many homologues of evolutionarily conservative components of the DNA damage response system (Mannuss *et al.*, 2012).

In addition to the responses to DNA damage, high intensity UVB radiation causes generation of reactive oxygen species (ROS) in plant cells and results in photo-oxidative stress accompanied by DNA fragmentation, changes in the nuclear morphology and, finally, PCD (Lam, Zhang, 2012). All the responses to high-intensity UVB radiation mentioned are not mediated by the UVR8 photoreceptor and are regulated by other signaling pathways.

The purpose of this study was to summarize the recent data on the mechanisms of UVB-induced PCD of plant cells and the UVR8-mediated regulatory pathway providing plant protection and acclimatization under the effect of UVB radiation.

THE UVR8-INDUCED SIGNALING PATHWAY IN PLANT CELLS

In recent years some authors have shown that photomorphogenetic responses in plants induced by low-intensity UVB radiation, such as the expression of genes involved in cell protection against UVB (Brown *et al.*, 2005, 2009; Favory *et al.*, 2009; Jenkins, 2009; Heijde and Ulm, 2012), are mediated by UVR8 (Rizzini *et al.*, 2011). In the absence of UVB light, this photoreceptor forms a functionally inactive homodimer. Binding of two identical subunits in the UVR8 dimer is provided by a complex network of salt bridges between charged residues of arginine, asparagine, and glutamine located on the contact surface of the subunits. Tryptophan (Trp) residues located between arginine residues form a unique pyramidal structure and function as UVB sensors. Unlike other known photoreceptors, UVR8 does not have a photoactive prosthetic chromophore.

Absorption of UVB photons by Trp285 and Trp233 initiates the breakage of salt bridges accompanied by dimer dissociation and the formation of functionally active UVR8 monomers (Christie *et al.*, 2012; Wu *et al.*, 2012).

Monomers interact with E3 ligase COP1. This protein is a photomorphogenetic represser able to ubiquitinize the transcription factor HY5 that results in its proteasomal degradation (Rizzini *et al.,* 2011; O'Hara and Jenkins, 2012). The interaction between the UVR8 monomer and COP1 occurs within several minutes after UVB light adsorption by a UVR8 dimer and represents a basic mechanism for triggering a signal providing stabilization of the transcription factor HY5. As a result, expression of the majority of genes involved in the UVR8-photoregulating pathway occurs (Brown and Jenkins, 2008), which provides cell protection against potential UVB-induced damage and, therefore, the survival of plants exposed to sunlight (Fig. 1; Favory *et al.*, 2009; Stracke *et al.*, 2010). A study of the effect of low-intensity UVB radiation on *uvr8* mutants showed that UVR8 regulates the induction of genes encoding the enzymes involved in the synthesis of flavonoids and other phenolic compounds and the enzymes providing repair of UVB-damaged DNA (Brown *et al.*, 2005; Favory *et al.*, 2009; Cloix *et al.*, 2012). Flavonoids, which function as an optical shield due to their ability to absorb and dissipate energy of UVB photons, are also characterized by some antioxidant properties and may quench free radicals providing additional protection of cells against the UVB-induced damage (Buer *et al.*, 2010; Xie *et al.*, 2012).

Along with activation of genes involved in the UVB protection of cells, UVR8 mediates activation of genes encoding RUP1 and RUP2 (repressors of photomorphogenesis 1 and 2) proteins, which act as negative regulators of the UVR8 pathway by direct protein–protein interaction (Gruber *et al.*, 2010; Cloix *et al.*, 2012). It has been shown recently that the interaction between RUPs and UVR8 causes a rapid UVR8 reversion from the active monomeric form to the homodimeric basic state (Fig. 1; Heijde and Ulm, 2013; Heilmann and Jenkins, 2013). A negative regulation of the UVB-triggered signaling pathway is also observed during the interaction between the COP1 and BBX24 proteins resulting in stimulation of the COP1 function in the process of suppression of HY5 transcriptional activity (Jiang *et al.*, 2012).

Note that the COP1 protein is involved in light-regulating responses, which are mediated not only by the UVR8 receptor, but also by crypto- and phytochrome representing photoreceptors activated by blue/UVA and red/far red light, respectively. All three photoreceptor types interact with the same WD40 domain of the COP1 protein (Heijde and Ulm, 2012). This probably explains the fact that the UVB-induced interaction between UVR8 and COP1 occurring under the joint influence of UVB and visible light results in COP1 elimination from the signaling pathways that

are triggered by phyto- and cryptochrome (Favory *et al.*, 2009).

Identification of UVR8 as a photoreceptor of the UVB light and the establishment of a connection between the processes of protection against UVB and the UVR8–COP1–HY5 pathway determined the progress in the understanding of principles of UVB photon perception by plants followed by light signal transduction. At the same time, experiments with the *uvr8-2* and *hy5* lines of etiolated plants did not reveal any significant phenotypic differences between these plants and wild types of plants, which resulted in a hypothesis about the presence of other UVB receptors in plants (Gardner *et al.*, 2009). According to the hypothesis, this photoreceptor absorbs UVB with shorter wavelengths and provides regulation of the stress response. The UVB-triggered stress pathway known as the "UV response" is conservative in plants, yeasts, and animals and involves activation of mitogen-activated protein kinases (MPKs; Ulm, 2003; Herrlich *et al.*, 2008). In *Arabidopsis*, MPK3 and MPK6 are activated in response to the UVB stress, while their wrong regulation in a *mkp1* mutant in relation to phosphatase 1 of MAP kinases (MPK1) results in manifestation of UVB-sensitive phenotypes (Gonzalez Besteiro *et al.*, 2011). MPK6 is a known positive regulator of PCD during plant development, for example, during ageing (Bartels *et al.*, 2009; Zhou *et al.*, 2009). Taking into account these data, it was supposed that MPK1 provides protection against UVB-triggered PCD by inhibition of the UVB-induced activity of MPK3 and MPK6 (Gonzalez Besteiro *et al.*, 2011). The UVB-triggered signaling pathways described, namely, the stress-induced MAPK pathway and UVR8–COP1–HY5-pathway, do not depend on each other, but may jointly influence plant tolerance to UVB radiation.

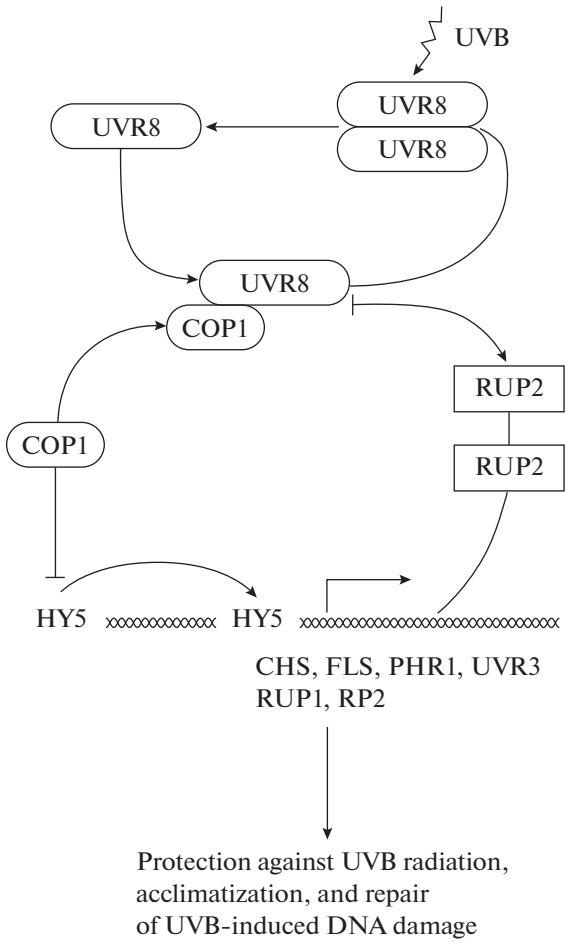

Fig. 1. Scheme of the UVR8-mediated signaling pathway regulating gene expression. *CHS, chalcone synthase; FLS, flavonol synthase*; *PHR1, photolyase 1*; *UVR3, UV repair defective 3.*

PROGRAMMED CELL DEATH IN PLANTS INDUCED BY HIGH-INTENSITY UVB RADIATION

High-intensity UVB radiation induces oxidative stress and severe DNA damage in plant cells that may activate a PCD process. In animals, the best studied PCD form is apoptosis, a genetically controlled mechanism intended for sequential destruction of barely damaged cells. Apoptosis is characterized by some specific manifestations, such as DNA fragmentation with DNA ladder formation, chromatin condensation, and cell shrinkage. In plants, the PCD mechanism is less studied; nevertheless, it includes many conservative components typical for the apoptosis of animal cells (Fomicheva *et al.*, 2012; Lam and Zhang, 2012; Yoshiyama *et al.*, 2013a). In addition, in the case of UVB-triggered PCD, plant cells demonstrate the above-described morphological traits (Reape and McCabe, 2008; Lytvyn *et al.*, 2010). Note that some of the plant genes involved in the UVB-triggered PCD processes have already been identified (Nawkar *et al.*, 2013).

Role of UVB-caused DNA damage in the cell cycle arrest and PCD induction processes. DNA is one of the main targets for the damaging effect of UVB radiation in plant cells. An increase in the level of sunlight UVB radiation caused by prolonged depletion of the stratospheric ozone layer reduces the genomic stability of plant populations (Ries *et al.*, 2000). Like "nonecological" UVC radiation (220–290 nm), UVB photons efficiently induce the formation of several types of DNA photoproducts including the most important cyclobutane pyrimidine dimers (CPDs) and (6–4) adducts (Sinha and Hader, 2002).

Three basic systems in plants intended for repair of UV-induced DNA damage include enzymatic photoreactivation (Teranishi *et al.*, 2008), nucleotide excision repair (Liu *et al.*, 2000), and homologous recombination (Dubest *et al.*, 2002). Photoreactivation involves two enzymes, CPD photolyase and (6–4) photolyase, which provide photorepair of CPD and

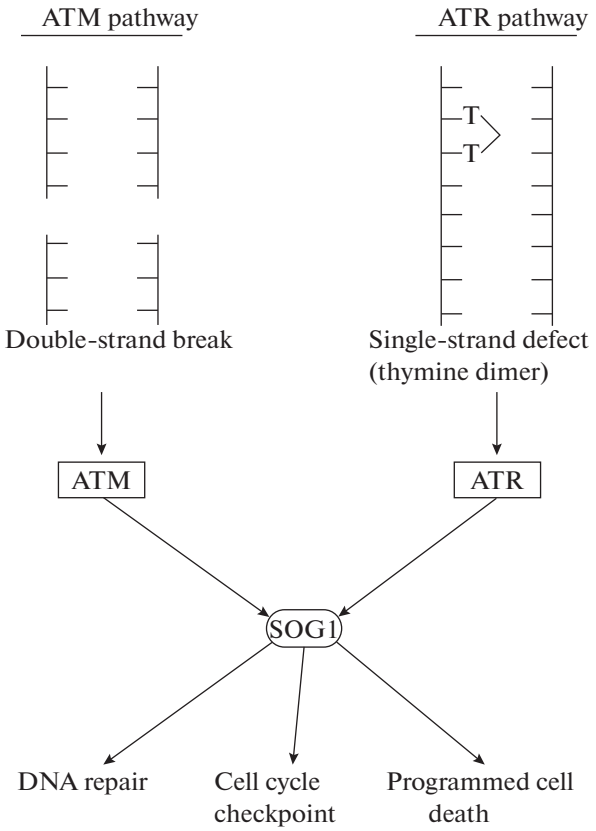

Fig. 2. Simplified scheme describing pathways of the plant cell response to UVB-induced DNA damage. DNA damage signal transmitted by signal ATM or ATR kinases and a central effector SOG1 may cause cell cycle arrest and either DNA repair or programmed cell death.

the (6–4) adducts, respectively (Sancar, 2008). Note that mutant plants deficient in the genes encoding these photolyases demonstrate an increased UVB sensitivity (Nakajima *et al.*, 1998; Teranishi *et al.*, 2008). A similar effect was observed for mutant plants with impaired excision repair ability (Liu *et al.*, 2000).

As we mentioned earlier, the efficiency of DNA repair systems can be improved by activation of DNA damage checkpoints able to arrest the cell cycle (Zhou and Elledge, 2000). In animals, the molecular mechanism of DNA damage checkpoints includes several protein components, such as ATM and ATR kinases, which detect DNA damage and initiate signal transduction cascades by the phosphorylation of signal checkpoint kinases (Cimprich and Cortez, 2008; Shiloh and Ziv, 2013). These signal kinases activate the p53 protein (tumor suppressor) and inactivate cyclin-dependent kinases to inhibit cell cycle progression from G1 to S (the G1/S checkpoint), DNA replication (the intra-S checkpoint), or G2 to mitosis (the G2/M checkpoint; Sancar *et al.*, 2004; Ciccia and Elledge, 2010). Activated (phosphorylated) p53 plays a key role in the choice of cell response to DNA damage: either cell cycle arrest, which provides some time for DNA damage repair, or the initiation of apoptosis in the case of multiple DNA disruptions that cannot be repaired (Helton and Chen, 2007).

Among the above-listed components, only the ATM and ATR kinases, which orchestrate responses to DNA damage, were found in plants (Fig. 2). ATM kinase reacts to double-strand DNA breaks, while ATR kinase detects breaks and CPD formed in a single DNA strand. In *Arabidopsis*, ATR kinase and its partner, ATRIP (ATR-Interacting Protein), participate in the increasing cell tolerance to UVB radiation (Culligan *et al.*, 2004; Sakamoto *et al.*, 2009). This fact is confirmed by the data on the increased UVB sensitivity in ATR-deficient mutants and the high death rate of mutant cells caused by the changed checkpoint at the G2 phase of the cell cycle (Culligan *et al.*, 2004). Experiments with synchronized cells of *Arabidopsis* roots showed that UVB-induced DNA damage causes modulation of the expression of genes regulating the cell cycle during the G1/S transition and determining its arrest at this stage (Jiang *et al.*, 2011). Blocking of the G1/S transition is considered to be a mechanism of protection against UVB radiation providing an increased time for repair of UVB-induced DNA damage prior to DNA replication.

The data on a direct connection between (1) cell cycle arrest at the G1/S transition and the high death rate of BY-2 cells of tobacco and (2) accumulation of CPD and single-strand breaks in DNA at high dosages of UVB radiation seem to be very interesting (Takahashi *et al.,* 2015). Moreover, at the S phase of a cell cycle, double-strand breaks may appear in *Arabidopsis* cells due to a collapse in the DNA replication fork; these breaks represent a major obstacle to the genome integrity and the main reason for PCD (Furukawa *et al.*, 2010; Curtis and Hays, 2011). Further increase in the UVB dosage induces specific nucleosomal DNA fragmentation (an integral PCD component) in BY-2 cells and the development of morphological traits typical for apoptosis, such as cell shrinkage and chromatin condensation (Lytvyn *et al.*, 2010).

An important stage in the study of the mechanisms of checkpoints and PCD in plants was connected with the revealing of the transcription factor SOG1 (SUPPRESSOR OF GAMMA RESPONSE 1) in *Arabidopsis* (Yoshiyama *et al.*, 2009). SOG1 orchestrates the cell response to DNA damage; its activation is required for the majority of such responses in plants including transcription, cell cycle arrest, and cell death (Fig. 2). SOG1 is phosphorylated by ATM kinase in response to DNA damage (Yoshiyama *et al.*, 2013b). A nonphosphorylated SOG1 mutant loses almost all SOG1 functions; therefore, phosphorylation is required for its proper functioning. The analysis of the SOG1 functions and mechanism of regulation showed that the role of SOG1 in a system of plant cell responses to DNA damage is comparable with that of p53 in animals. Thus, SOG1 is considered to be a key

regulator of the plant cell response to DNA damage (Yoshiyama *et al.*, 2013a).

There are also interesting data on the SOG1 functions in different responses to the UVB-induced DNA damage, such as PCD (Furukawa *et al.*, 2010) and hypocotyl growth inhibition in etiolated seedlings caused by cell cycle arrest (Biever *et al.*, 2014). The last example first demonstrated that the typical photomorphogenetic response may be induced by initiation of a mechanism related to the response to UVB-induced DNA damage rather than by a special regulating receptor of UVB light (for example, UVR8).

The role of UV-induced ROS formation and activation of metacaspases in the PCD process. High-intensity UVB radiation causes oxidative stress in plant cells by ROS generation in mitochondria and chloroplasts (Mackerness *et al.*, 2001). NADPH oxidase of the cytolemma absorbs UVB light and actively generates a superoxide anion radical $O_2^{\cdot -}$ (Kalbina and Strid, 2006). This radical is generated by a single-electron reduction of molecular oxygen and almost does not react with the majority of biological molecules. However, it can occur with a spontaneous or enzymatic dismutation with the formation of hydrogen peroxide (H_2O_2), which represents another low-reactive ROS able to migrate across the cell. In the presence of Fe^{2+}, hydrogen peroxide initiates a Fenton reaction generating a highly reactive hydroxyl radical $^{\cdot}OH$. This hydroxyl radical reacts with DNA and also membrane proteins and lipids through attachment to double bonds or a detachment of a hydrogen atom. Both reactions result in the formation of neutral radicals, which represent precursors of peroxy radicals generating in the presence of molecular oxygen.

Along with the OH radical, singlet oxygen ($^1O_2$) is also a strong oxidant of biological molecules. Singlet oxygen is generated by energy transfer from the triplet level of a photosensitizer to the oxygen molecule that results in its transition from the basic triplet state ($^3O_2$) to the singlet excited state due to spin reversal in one of the two uncoupled electrons of $^3O_2$. In chloroplasts of plant cells, $^1O_2$ can be generated by the $^3O_2$ reaction with photoexcited chlorophyll. A nonadequate dissipation of excess visible light energy during photosynthesis may result in the formation of triplet chlorophyll able to react with $^3O_2$ with the generation of $^1O_2$. The last one may also be generated by other molecules (porphyrins and flavins) located in the cytoplasm and organelles of plant cells and able to absorb UVA (320–400 nm) and visible light. However, to date there is no information about cell chromophores able to sensitize $^1O_2$ formation under UVB radiation. Therefore, in this paper we discuss only oxidative reactions, which are initiated in biologically important molecules by the OH radical produced after the initial UVB-induced generation of a superoxide anion radical.

Reactions occurring with the participation of the OH radical cause various oxidative damage to DNA, such as strand breaks and the formation of oxidized pyrimidine bases and modified purine bases, especially 8-oxoguanine. The mechanism of 8-oxoguanine generation may include the $^{\cdot}OH$ attachment to the C8 position of guanine with the formation of an 8-hydroxy-7,8-dihydroguanyl radical and its further single-electron oxidation to 8-oxoguanine. The most likely mechanism for the formation of DNA breaks is connected with the $^{\cdot}OH$-mediated detachment of hydrogen atoms from the C3, C4, and C5 positions of 2-deoxyribose. Note that DNA damage caused by oxidative processes makes up ~1% of the oxygen-independent formation of pyrimidine dimers, which represents the predominant type of biologically significant UVB-induced DNA defects (Cadet *et al.*, 2015).

In proteins, oxidation affects histidine, tryptophan, tyrosine, cysteine, methionine, arginine, and lysine residues. As a result, peroxides and carbonyl-containing compounds able to inhibit protein activity may be generated (Moller *et al.*, 2007). In the course of interaction between the OH radical and cysteine-containing proteins, a hydrogen atom is detached from its sulfhydryl group with the formation of a thiyl radical. Crosslinks of thiyl radicals between two cysteine molecules form cysteine dimers through disulfide bridges, as well as higher order aggregates. Crosslinks may also appear due to the oxidation of hystidine residues to carbonyl-containing compounds during their interaction with side chains of lysine, cysteine, and arginine residues. Covalent crosslinks important for the formation of high-molecular aggregates represent a common consequence of protein oxidation. Oxidative degradation of amino acid residues is accompanied by notable changes in the physicochemical properties of proteins, suppression of the activity of membrane enzymes, and failures in membrane transport functions.

ROS-induced oxidative degradation of membrane lipids is based on peroxidation of unsaturated fatty acids (UFAs). A hydroxyl radical initiates this process through the detachment of a hydrogen atom from the UFA lipid (LH) with the formation of an alkyl radical ($L^{\cdot}$). The interaction between $L^{\cdot}$ and oxygen results in the formation of peroxyl radicals ($LOO^{\cdot}$), which react with other LH and generate new $L^{\cdot}$ radicals and equivalent amounts of hydroperoxides (LOOH). Malonic dialdehyde (MDA) and other compounds containing carbonyl groups represent the accompanying products of free-radical lipid peroxidation (LP); they are generated during cyclization of peroxyl radicals and considered as the final LP products (Girotti, 2001). It is interesting that electrophylic MDA is able to migrate across the cell and react with several amino acid residues in proteins and also with guanine in DNA molecules. After MDA attachment to the guanine amino group and the further cyclization and

dehydratation reactions, a MDA-guanine adduct is formed. To date, there is no information on whether or not the generation of this adduct is caused by UVB radiation (Cadet *et al.*, 2015).

The excess ROS generation by high-intensity UVB radiation, accompanied by intensification of the LP and oxidative degradation processes in proteins, causes structural damages to the plasmalemma, as well as in chloroplast and mitochondrial membranes and also failures in the functions and permeability barriers of these membranes that may result in unavoidable cell death. However, it usually does not happen, which can be explained by the presence of a large group of enzymes and nonenzymatic compounds possessing antioxidant properties and neutralizing ROS without the formation of any other toxic compounds. ROS detoxication is also performed by high-molecular enzymes having antioxidant activity, which include superoxide dismutase (SOD), catalase, ascorbate peroxidases, and glutathione peroxidases. Low-molecular antioxidants include ascorbic acid, glutathione, α-tocopherol, carotinoids, and flavonoids (Mittler *et al.*, 2004).

SOD is the most efficient enzymatic antioxidant present in all subcellular compartments. SOD removes $O_2^{\bullet-}$ by catalyzing its dismutation and, therefore, reduces the risk of the $^{\bullet}OH$ formation. Hydrogen peroxide generating during the $O_2^{\bullet-}$ dismutation, is removed by catalase and ascorbate peroxidases including the enzymes of the glutathione–ascorbate cycle. Ascorbate peroxidases use ascorbic acid as the donor of electrons reducing H_2O_2 to H_2O. Glutathione peroxidases use glutathione for H_2O_2 reduction. In addition, glutathione is able to reduce lipid hydroperoxides to alcohols, so the detoxification of hydroperoxides by glutathione peroxidase may prevent a LOOH-dependent initiation of the peroxidation chain in chloroplasts and mitochondria, where glutathione peroxidases are localized.

ROS detoxification by antioxidant enzymes and low-molecular antioxidants provides protection of plant cells in the case of low-intensity UVB radiation. However, under high-intensity UVB radiation, the balance between the formation and neutralization of ROS is broken, which results in the increased ROS level within cells. Interestingly, ROS generated in chloroplasts under intensive UVB radiation and continuous action of visible light may damage neighboring mitochondria (Gao *et al.*, 2008). ROS can induce destructive oxidative reactions in molecular components of cell structures resulting in failures of their functions; they are also able to initiate PCD in plants. It was shown that ROS act as signaling molecules causing the opening of mitochondrial permeability transition pores. This process results in the generation of a large amount of ROS and forms a feedback loop, which enhances the initial PCD-triggering stress signal (Reape and McCabe, 2008).

The involvement of mitochondria in PCD has been studied in detail in animals. There are some data evidencing the role of plant cell mitochondria in PCD triggered by various stress stimuli including UV radiation (Yao *et al.*, 2004; Gao *et al.*, 2008). Such impacts cause a loss of the transmembrane potential by mitochondria, which is accompanied by cytochrome *c* release into the cytoplasm, activation of caspase-like proteases, and subsequent cell death (Yao *et al.*, 2004).

Caspases are cysteine proteases possessing aspartate-specific activity; these enzymes play a key role in PCD triggering in animals. Plants do not have direct homologues of apoptotic caspases, though they contain a small family of proteins known as metacaspases and are similar to caspase-like domains. The *Arabidopsis* genome includes nine identified genes encoding metacaspases, which are considered to be functional analogues of animal caspases (Watanabe and Lam, 2005).

According to the existing data, UVC radiation in the presence of visible light induces plant protease, which cleaves the substrate of caspase, Asp-Glu-Val-Asp (DEVD activity). The protease is induced within 30 min, and its activity reaches the maximum 1 h after the irradiation (Danon *et al.*, 2004). Overexpression of *AtDAD1* and *AtDAD2* (*A. thaliana homologs of Defenders against Apoptotic Death*) genes prevents DNA fragmentation caused by the activity of DEVD protease; as a result, the survival of cells under UV-induced stress conditions increases (Danon *et al.*, 2004). Use of the FRET (fluorescence resonance energy transfer) method provided the possibility for a real-time observation of the process of activation of a caspase-3-like protease during a UV-triggered PCD in plants (Zhang *et al.*, 2009). These data confirm the similar functioning of caspase-like plant proteases and apoptotic animal proteases.

Another study demonstrated induction of the *metacaspase8* (*AtMC8*) gene in *Arabidopsis* in response to oxidative stresses caused by UV radiation and H_2O_2 (He *et al.*, 2008). The *AtMC8* induction depends on the *AtRCD1* (*Radical-induced Cell Death1*) gene, so mutant lines deficient in *atmc8-1/2* or *rcd1-1* are tolerant to ROS, oxidative stress, and cell death triggered by UVB radiation (Jiang *et al.*, 2009). Interestingly, the induction of *AtMC8* transcripts in response to UVB radiation, like the induction of the above-mentioned protease with DEVD activity, is inhibited in the dark and occurs only under light. This feature is typical for UV-triggered PCD in plants; however, the specific role of visible light in this process still remains unclear.

CONCLUSIONS

The main recent achievement in the investigation of UVB light perception by plants is connected with the identification of a regulating receptor of UVB photons (URV8) and the elucidation of the mechanism of its involvement in cell protection against sunlight UVB radiation by the UVR8–COP–HY5 signaling pathway (Fig. 1). Significant progress was also observed for the study of signaling pathways resulting in PCD in plants. High intensity and high dosages of UVB radiation induce the formation of pyrimidine dimers and strand breaks in intracellular DNA. Accumulation of such damage triggers some cell responses including activation of DNA damage checkpoints, cell cycle arrest, and PCD characterized by typical DNA fragmentation and morphological traits of apoptotic cells. Recently it was found that the transcription factor SOG1 plays a key role in PCD regulation in plants (Fig. 2). In response to the oxidative stress caused by UVB-induced ROS, mitochondria lose their transmembrane potential, which is accompanied by a cytochrome *c* release into the cytoplasm and activation of metacaspases. All these events result in manifestation of typical apoptotic traits. Though it was shown that antiapoptotic *AtDAD1* and *AtDAD2* genes are involved in UV-triggered PCD, no direct evidence of the involvement of their transcripts into cell defense have been found. Note that the results of several of the above-discussed studies, which are of great importance for understanding the PCD mechanisms in plants, were obtained using UVC radiation, which does not penetrate into the biosphere. Though the mechanisms of cell damage induced by UVC and UVB radiation are very similar, we consider the revealing and investigation of the action of various components of signaling pathways resulting in PCD under UVB radiation of plants to be important in the ecophysiological aspect.

COMPLIANCE WITH ETHICAL STANDARDS

Conflict of interests. The authors declare that they have no conflict of interest.

Statement on the welfare of animals. This article does not contain any studies with animals performed by any of the authors.

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