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REVIEW

DNA Repair Mechanisms in UV-B Tolerant Plants

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Abstract

Understanding the mechanisms responsible for UV-B irradiation-induced DNA damage repair in plants is important for agricultural technology in that it will potentially enable the development of plants with enhanced growth rates and crop yields. Unlike yeast and mammalian cells, intact plants use sunlight for photosynthesis and are thus chronically exposed to the UV band wavelengths present in solar radiation. UV induces DNA damage, which can be corrected by DNA repair mechanisms such as photoreactivation and excision repair. Recently, details of several DNA repair mechanisms have become clear in plants. We made transgenic rice overexpressing genes involved in excision repair or plant-specific DNA repair, and measured their tolerance to UV-B. We found that OsUV-DDB2 and OsSEND-1 transgenic lines had higher tolerance to UV-B than the wild type. In this review, recent advances in understanding repair of DNA damage from UV-B radiation in plants and the prospects for the development of UV resistant plants are discussed.

Discipline: Biotechnology
Additional key words: BER, NER, photoreactivation

Introduction

Environmental agents such as UV light, chemical mutagens, fungal and bacterial toxins, and ionizing radiation can inhibit growth and productivity in plants[1,25]. Recently, ozone depletion in the stratosphere has resulted in increased UV-B irradiation at the earth's surface. Because plants are dependent on solar radiation for photosynthesis, they are bombarded by UV for much longer periods than animals or yeast[29] and cannot avoid or escape from the effects of UV. With the future of the protection afforded by stratospheric ozone levels unclear, producing higher tolerance to UV-B irradiation in plants may become an important goal for agricultural technology.

Light energy from UV-B wavelengths of the electromagnetic spectrum causes two major types of DNA-damage: the formation of cyclobutane pyrimidine dimers (CPDs) and pyrimidine (6-4) pyrimidine dimers (6-4 photoproducts)[23]. Plants are thought to use two main strategies to protect against the adverse effects of UV: prophylactic shielding by flavonoids and phenolic compounds[27,30] and DNA repair mechanisms such as photoreactivation and the Nucleotide Excision Repair (NER) function[4,12,19,34]. In addition, UV-B irradiation also results in oxidative damage that can be corrected by Base Excision Repair (BER). Consequently, understanding and potentially using DNA repair mechanisms could become very important for producing UV-B tolerant plants.

This review presents an outline of the current understanding of DNA repair mechanisms and development of UV-B tolerant plants.

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Plant DNA repair pathways for UV damaged DNA

1. Photoreactivation

Photoreactivation, which is mediated by photolyase, is thought to be the DNA repair pathway for CPDs and (6-4) photoproducts in higher plants. Photolyases bind specifically to DNA lesions and remove them directly by absorbing light in the 300–600 nm range, the dimer is reduced to monomer pyrimidines and the enzyme is released (Fig. 1(a))[18,24,31]. Photolyases are known to be very specialized in terms of substrate specificity. The *uvr2* and *uvr3* mutants of *Arabidopsis* were each mutated in (6-4) photolyase and CPD photolyase[24]. In rice, the UV irradiation-sensitive rice cultivar Norin 1 is deficient in photorepair of CPDs, which is likely due to a mutation in CPD photolyase[13,33].

2. Nucleotide Excision Repair (NER)

The other DNA repair mechanism for UV-induced DNA lesions is NER, which sequentially recognizes DNA damage, makes an incision on the damaged strand, excises the damaged oligonucleotides, and fills the gap through DNA synthesis and ligation of the nick[2]. There are two subpathways of NER, designated global genomic repair (GGR) and transcription-coupled repair (TCR). While GGR repairs DNA damage over the entire genome, TCR is sensitive for the transcribed DNA strand in expressed genes (Fig. 1(b)). In lily, excision repair cross-complementation group 1 (ERCC1) could correct the mitomycin C (MMC) sensitivity in ERCC1-deficient Chinese hamster ovary cells that further suggested that the NER mechanism is conserved in animals, yeast and plants[36].

Recently, rice and *Arabidopsis* homologs of some of the components involved in mammalian NER pathways

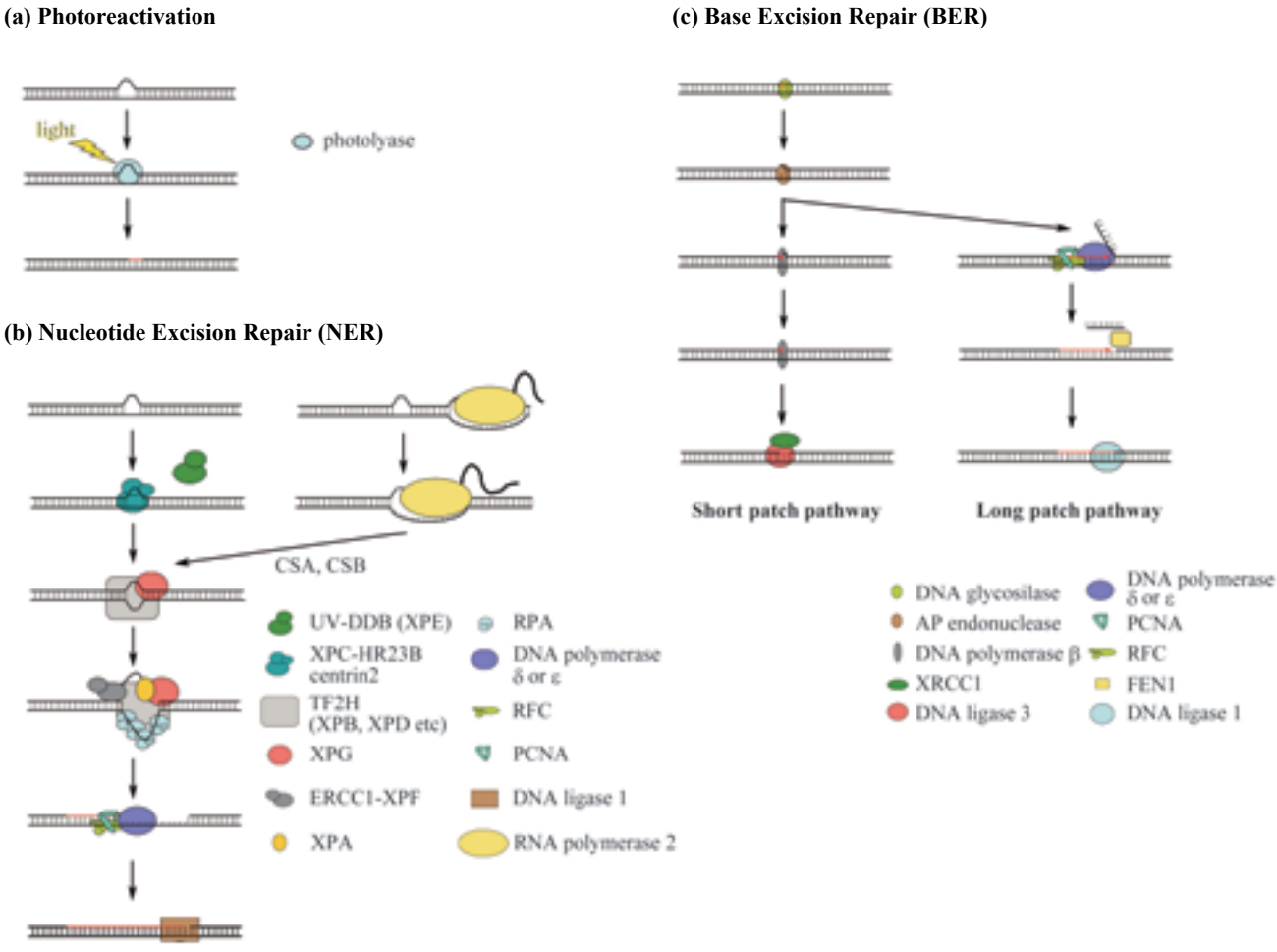

Fig. 1. DNA repair mechanisms for UV-B irradiation
(a): Photoreactivation, (b): Nucleotide Excision Repair (NER), (c): Base Excision Repair (BER).

have been reported[9,14,15,18] (Table 1). Several mutants in these pathways have also been reported which were sensitive to UV-B irradiation, including, for example, *Arabidopsis uvh3* (*atrad2/xpg*), *uvh6* (*atxpd*), *atrad1* (*atrad1/xpf*) and *uvr7* (*atercc1*)[6,7,21,22] (Table 1).

3. Base Excision Repair (BER)

UV irradiation also causes oxidative damage to DNA that is repaired by Base Excision Repair (BER)[28]. In BER, the damaged base is removed, the resulting single-stranded gap is filled in by DNA synthesis, and the

Table 1. DNA repair mechanisms in rice and *Arabidopsis*

Pathway	Gene	Function/Remarks	Accession no.		Reported mutants of UV-B sensitivity
			Oryza sativa	*Arabidopsis thaliana*	
Photoreactivation	CPD photolyase	Removal of CPD	AK111418	NM_179320	*atuvr2*
	Cry3	Removal of DNA damage, Targets to organelles	AK072287	AY102138	
	(6-4) photolyase	Removal of (6-4) photoproduct	–	NM_112432	*atuvr3*
Nucleotide excision repair	XPA	Binds to DNA damage	–	–	
(NER)	XPB	DNA helicase, Subunit of THIIH	AK060447	U29168	*atxpb*
	XPC	Binds to DNA damage	AK102608	NM_121669	
	XPD	DNA helicase, Subunit of THIIH	AK099724	AF188623	*atuvh6*
	XPF (Rad1)	5' incision	AK068556	AF191494	*atuvh1*
	ERCC1 (Rad10)	5' incision	AK070764	AF276082	*atuvr7*
	XPG (Rad2)	3' incision	AC123568	NM_113721	*atuvh3*
	UV-DDB1	UV damaged DNA binding protein-1	AK065508	NM_116781	
	UV-DDB2 (XPE)	UV damaged DNA binding protein-2	AB082381	BT010570	
	Rad23	Binds to DNA damage	AK064881	NM_123208	
	Rad23	Binds to DNA damage	AK103728	↑	
	Rad23	Binds to DNA damage	AK069065	NM_202452	
	Rad23	Binds to DNA damage	AK061556	NM_111121	
	CSA	Transcription-coupled NER	AK111811	NM_102549	
	CSB	Transcription-coupled NER	AK064456	NM_127432	
	CSB	Transcription-coupled NER	AK071717	NM_100254	
	CSB	Transcription-coupled NER	AK099822	NM_125791	
	XAB2	Transcription-coupled NER	AK066726	NM_122757	
	MMS19	Transcription-coupled NER	AK070264	NM_124186	
	TF2H1	TFIIH subunits p62	AK068124	NM_104451	
	TF2H2	TFIIH subunits p44	XM_473124	NM_148434	
	TF2H3	TFIIH subunits p34	XM_463960	NM_101692	
	TF2H4	TFIIH subunits p52	XM_474374	NM_202835	
	Cyclin H	Kinase subunit THIIH	AK101854	NM_122644	
	CDK7	Kinase subunit THIIH	AK068916	NM_104184	
	CDK7	Kinase subunit THIIH	AK064909	NM_104338	
	CDK7	Kinase subunit THIIH	AK101089	↑	
	CDK7	Kinase subunit THIIH	AK067238	NM_101725	
	CDK7	Kinase subunit THIIH	AK072696	NM_112366	
	CDK7	Kinase subunit THIIH	AK073808	NM_121065	
	MAT1	Kinase subunit THIIH	AK103771	NM_179145	
	MAT1	Kinase subunit THIIH	AK065754	↑	
	DNA ligase I	Ligates DNA ends	AK110056	NM_100689	
NER and BER	PCNA	Accessory protein of DNA polymerases	AK071591	NM_128510	
	PCNA	Accessory protein of DNA polymerases	AK063098	NM_100611	
	RFC1	Accessory protein of DNA polymerases	AK070564	NM_147883	
	RFC2	Accessory protein of DNA polymerases	AK103718	NM_179364	
	RFC3	Accessory protein of DNA polymerases	AK069984	NM_106396	
	RFC4	Accessory protein of DNA polymerases	AK069025	NM_104994	
	RFC5	Accessory protein of DNA polymerases	AK103751	NM_122656	
	RPA70a	ssDNA binding	AK101212	NM_201704	
	RPA70b	ssDNA binding	AK060582	NM_120884	
	RPA70c	ssDNA binding	AK073598	NM_123908	
	RPA32-1	ssDNA binding	AK103235	NM_128010	
	RPA32-2	ssDNA binding	AK073723	NM_111162	
	RPA32-3	ssDNA binding	AK102353	–	
	RPA14	ssDNA binding	AK058837	NM_117973	
	XPG	NER	AC123568	–	
	DNA polymerase δ	DNA replication, NER, BER	AB037899	NM_125792	
	DNA polymerase δ	DNA replication, NER, BER	AK110500		
	DNA polymerase δ2	DNA polymerase δ small subunit	AK067991	NM_201935	
	DNA polymerase ε	DNA replication, cell cycle regulation	AK107241		

(continued)

Table 1. DNA repair mechanisms in rice and *Arabidopsis (continued)*

Pathway	Gene	Function/Remarks	Accession no.		Reported mutants of UV-B sensitivity
			Oryza sativa	*Arabidopsis thaliana*	
Base excision repair (BER)	TagI	Glycosylase	AK063273	NM_106165	
	TagI	Glycosylase	AK110707	NM_112038	
	TagI	Glycosylase	AK065590	NM_202089	
	TagI	Glycosylase	AK069193	NM_125182	
	TagI	Glycosylase	AK109346	NM_112107	
	MutM	Glycosylase	AK063295	NM_104128	
	MutM	Glycosylase	AK065376	AB010690	
	AlkA	Glycosylase	AK073046	NM_114948	
	Ung	Glycosylase	XM_474316	NM_112749	
	Ogg	Glycosylase	XM_466174	NM_102020	
	MutY	Glycosylase	AC138002	NM_117343	
	AP endonuclease	DNA-(apurinic or apyrimidinic site) lyase	AK101426	NM_129709	
	AP endonuclease	DNA-(apurinic or apyrimidinic site) lyase	AK102132	-	
	AP endonuclease 2	DNA-(apurinic or apyrimidinic site) lyase	AK103074	NM_202962	
	PARP	Poly (ADP-ribose) polymerase	AK103479	NM_179834	
	PARP	Poly (ADP-ribose) polymerase	AK102681	NM_122152	
	DNA polymerase β	BER, meiosis	–	–	
	DNA polymerase λ	BER, Contains a BRCT domain	AB099525	NM_100926	
	FEN-1a	Removal of Okazaki fragment, BER	AK103819	NM_180546	
	FEN-1b	Class II member of RAD2 nuclease family	AK062149	–	
	DNA ligase III	Ligates DNA ends	–	–	
	XRCC1	Interacts with DNA ligase III	AK068998	NM_106691	
Other related genes	EXO1	Involved in DNA repair	AB179769	NM_179353	
	SEND-1	Class IV member of RAD2 nuclease family	AK102542	NM_114750	
	GEN-L	Class IV member of RAD2 nuclease family	AK063534	NM_100069	
	DNA ligase	Ligates DNA ends	AK064463	NM_105343	

new segment is ligated to the preexisting strand 3' to the damaged area (Fig. 1(c)). There are also two subpathways of BER, short patch BER and long patch BER. Short patch BER eliminates a damaged site with a glycosylase and AP endonuclease and the gap is ligated, but in long patch BER neighboring nucleotides of the eliminated site are displaced by DNA polymerase ε, δ and other components. BER, while known to be present in plants, has not been as well studied in plants as in animals[26]. BER homologous components in rice and *Arabidopsis* have been reported[5,11,17,18,20] (Table 1).

UV-B tolerant plants

Approximately 10% of UV-B radiation reaches the surface of the earth. A loss of 1% portions of the protective stratospheric ozone layer can result in an increase of 1.5% UV-B reaching the surface of the earth. In particular, ozone layer destruction in the polar region is an especially serious problem. UV-B irradiation causes damage to plants, decreasing crop yield, and potentially affecting the ability of the plant to manage the number of mutations passed on to progeny in the seeds. Therefore, making UV-B tolerant plants may become a very important tool to solve this problem. The *Arabidopsis* mutant UV-B insensitive 1 (*uvi1*), which exhibits an enhanced capacity for repair, is more resistant to UV-B than the wild type[32]. Another reported tolerance to UV-B irradiation is in the *uvt1* mutant. *Uvt1* is represented by the *atmyb4* mutant, and accumulates flavonoid and sinapate esters[3,16]. Ozone-sensitive *radical-induced cell death1-2* (*rcd1-2*) mutants also exhibit a higher tolerance to UV-B irradiation, likely due to an increased accumulation of UV absorbing compounds[8].

In rice, a wide variation in the level of UV-B resistance has been observed among different varieties. On the whole, Japanese lowland rice varieties (Japonica rice) are more resistant to UV-B radiation, while Indica varieties tend to be more sensitive. Sasanishiki, one of the most popular rice cultivars in Japan, has a higher UV-resistance than Norin 1 (Japonica rice), but has no difference in growth compared to Sasanishiki under normal growth conditions. Marich-bati, an Indica rice cultivar, also indicates the UV-resistance more than Surjamkhi (UV-sensitive Indica rice cultivar)[13,33]. Quantitative trait locus (QTL) analysis indicates that UV-B resistance in rice is associated with the CPD photolyase gene *qUVR-10*[35].

Recently, we produced several transformant rice plants with overexpression of genes related either to NER or plant-specific DNA repair. Previously, we indicated that expressions of *OsSEND-1* (plant specific RAD2 fam-

ily single-strand endonuclease 1) and *OsUV-DDB2* (UV-damaged DNA binding protein 2) were induced by UV-B irradiation[10,15]. Exposure of *OsSEND-1* and *OsUVDDB2* overexpression lines to UV-B irradiation indicated increased UV-B tolerance in the callus (Fig. 2(a)) and in seedlings (Fig. 2(c)). OsUV-DDB2 participates in the recognition of DNA damage in NER. Overexpressed OsUV-DDB2 in plants may aid in the recognition of DNA damage faster. *OsSEND-1* is a new plant specific gene of the RAD2 family. Overexpressed *OsSEND-1* has resistance to UV-B, therefore it is suggested that *OsSEND-1* is related to plant specific DNA repair. We also overexpressed several NER-related or plant-specific DNA repair genes and DNA replication genes. However, lines overexpressing other genes associated with both DNA repair and replication did not have any apparent increase in tolerance for UV-B irradiation in the callus (data not shown).

Concluding remarks

The importance of DNA repair mechanisms for UV-B tolerance in plants was emphasized in this review. Notably, we have concluded that rice transformant lines overexpressing *OsSEND-1* and *OsUV-DDB2* had an enhanced tolerance for UV-B irradiation. Little is known about the proteins or genes involved in DNA repair of UV-B irradiation damage in higher plants in comparison

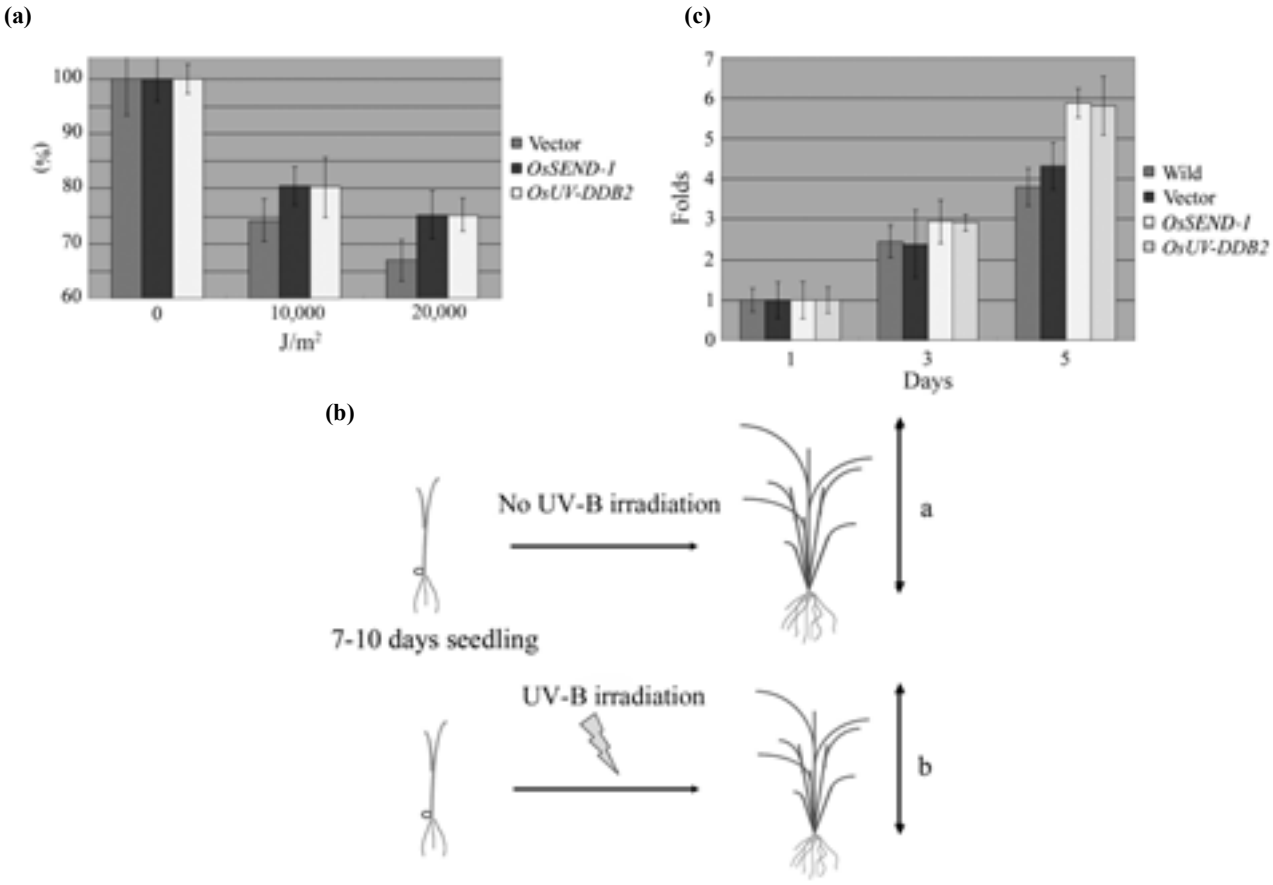

Fig. 2. Overexpressed OsSEND-1 or OsUV-DDB2 rice plants indicates UV-B tolerance in callus (a) and seedling (b)
The fragments were cloned to the PBE2113 vector. Cloned vectors were transformed into *Agrobacterium tumefaciens* EHA101. Rice plants used for transformation were *Oryza sativa* L. cv. Nipponbare. The agrobacterium transformation system was performed. Transformed (T1) seeds were collected and grown in callus induction medium. Northern blotting analysis and UV tolerance in callus were performed on T1 callus.
(a): T1 calli on callus induction medium were irradiated by UV-B using a UV-B lamp (TL 20 W/01RS UV-B Medical; Philips). The UV-B irradiation intensity was measured using a spectroradiometer (model DRC100X; Spectronics Corporation). The sizes of calli were measured in the dark for 3 and 5 days after UV-B irradiation.
(b): Outline of UV-B tolerance seedling assay.
(c): UV-B tolerance seedling assay. Rice seeds (T2) were germinated in water and then planted to soil. About 1.5 cm seedlings were irradiated continuously with 1.3 J/m^2 by only UV-B using UV-B lamp (Philips) at 28ºC. The a/b length was measured after 3 and 5 days of continuous UV-B irradiation.

to animals or yeast, or the amount of variation in repair systems throughout the plant taxa. Therefore, the identification and understanding of plant specific DNA repair, and further characterization of already known systems, will be necessary to exploit UV tolerance in a wide range of agronomically important crop plants.

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