Higher plants and UV-B radiation: balancing damage, repair and acclimation

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Although UV-B is a minor component of sunlight, it has a disproportionately damaging effect on higher plants. Ultraviolet-sensitive targets include DNA, proteins and membranes, and these must be protected for normal growth and development. DNA repair and secondary metabolite accumulation during exposure to UV-B have been characterized in considerable detail, but little is known about the recovery of photosynthesis, induction of free-radical scavenging and morphogenic changes. A future challenge is to elucidate how UV-B-exposed plants balance damage, repair, acclimation and adaptation responses in a photobiologically dynamic environment.

Photosynthetic organisms need sunlight and are thus, inevitably, exposed to UV-B radiation. The UV-B wavelength band ranges from 280 nm to 320 nm, though only wavelengths greater than 290 nm can reach the Earth's surface. In sunlight, the ratio of UV-B to photosynthetically active radiation (PAR; 400–700 nm) fluctuates, primarily caused by changes in solar angle and thickness of the ozone layer. The thickness of the UV-screening ozone layer varies with season, meteorological conditions and latitude[1].

Depletion of the ozone layer results from emissions of halogenated chemicals, such as chlorofluorocarbons[2]. The resulting increase of solar UV-B in the biosphere is predicted to be minor in comparison with seasonal variations in UV-B flux. Nonetheless, a statistically significant trend of increasing UV-B photon flux has been measured[1,2]. Thinning of the ozone layer also results in a shift of the spectral UV-composition towards shorter wavelengths[1]. In general, biological damage is exacerbated as the wavelength becomes shorter. Thus, even modest increases in total UV-B are likely to cause significant biological damage.

Effects of solar UV-B on plants

UV-B radiation has many direct and indirect effects on plants, including damage to DNA, proteins and membranes (Box 1); alterations in transpiration and photosynthesis; and changes in growth, development and morphology[3]. UV-B exposure was found to lead to a reduction in biomass accumulation in some studies[3,4], but not in others[5,6]. Contradictory results might be caused by methodological differences, including levels of UV-B[5,7], PAR[8] and interactions with other environmental factors[4,8]. Indeed, a common problem in the field of UV-B physiology is to separate biologically relevant effects from those elicited by UV-fluences that would never actually be encountered in nature[5,6]. An additional source of contradictory results is the variation in UV-B sensitivity between species[9]. Plants distributed along lower latitudes or higher elevations, where UV-B fluences are greater, have more pronounced adaptive mechanisms than those from higher latitudes and/or lower elevations[9].

Tolerance to UV-B depends on the balance between a variety of damaging reactions, and both repair and acclimation responses. The analysis of the balancing act is difficult as distinctions between damage, repair and acclimation are not always clear. For example, the rapid UV-B-driven degradation of the D1 and D2 proteins of photosystem II (PSII) can be viewed either as damage or as part of a repair cycle designed to replace damaged components of PSII[10]. Similarly, UV-induced axillary branching can be viewed either as a consequence of the disruption to auxin metabolism[11] or as an acclimation response, possibly controlled through a UV-B photoreceptor[7]. Damage and acclimation may also be indistinguishable. Acclimation responses that reduce metabolic efficiencies or strain cellular resources may lower plant productivity and provide an apparent UV-sensitive phenotype. Thus, reductions in plant height assist a plant, at least in a canopy, in avoiding UV-B radiation, but as a consequence will also result in decreased interception of PAR[12].

The expression of acclimation responses is carefully regulated[13–17]. Continuous re-adjustment of UV-acclimation may be

Box 1. Molecular targets of UV-B

DNA
- Formation of cyclobutane-pyrimidine dimers (CPDs) and (6–4) photoproducts[20]

Photosynthetic machinery
- Inactivation of photosystem II (PSII)[30,31]
- Degradation of the D1 and D2 proteins of PSII[14–18]
- Decreased thylakoid membrane integrity[31]
- Reduced activity of Rubisco and other enzymes[3,31]
- Decreased levels of chlorophyll and carotenoids[31]
- Down-regulation of photosynthetic genes[31]
- Changes in chloroplast ultrastructure[31]

Membranes
- Peroxidation of lipids[19]

Phytohormones
- Photooxidation of IAA[15]

Secondary metabolism
- Activation of UV-B photoreceptor[16,43]
- Up-regulation of genes of the phenylpropanoid pathway[3,31,48,49]
- Accumulation of flavonoids and anthocyanins[39–41]
- Accumulation of alkaloids[59], waxes[60] and polyamines[19,56]

Free-radical scavenging
- Rise in levels of glutathione and ascorbate[63,64]
- Increased activity superoxide dismutase and glutathione reductase[18,62]
- Increased peroxidase activity[62]

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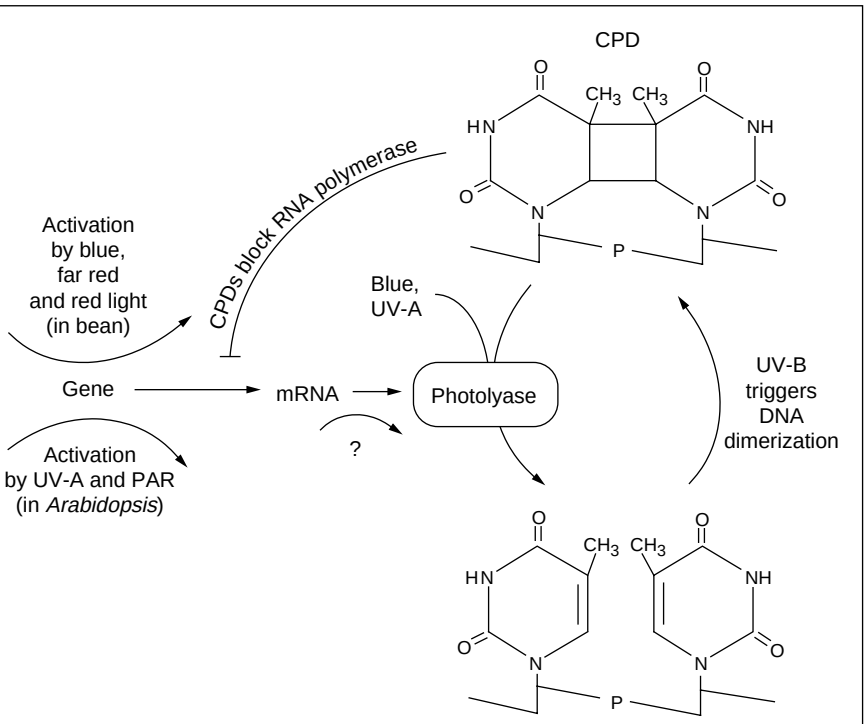

Fig. 1. Diagram showing the regulation of cyclobutane-pyrimidine dimer (CPD) photoreactivation. Transcription of genes encoding photolyases is minimal in the dark, but induced by blue, far-red and red wavelengths, possibly involving phytochrome[23], and/or by UV-A and PAR, possibly involving a UV-A/B photoreceptor[13]. Photolyase activity requires blue or UV-A radiation[19,22], but dimerization is mainly driven by UV-B[19]. If dimerization is not reversed, it will block transcriptional activity by impeding RNA-polymerases[19].

required in response to a photobiologically dynamic environment. For example, *Spirodela* plants exposed to supplemental UV-B acquire, within a day, enhanced radical-scavenging activity, the onset of which correlates with UV-B tolerance[18]. After a few days, scavenging activity declines, although UV-B tolerance remains high. Possibly enhanced radical scavenging is a rapid UV-B defence response, and this is subsequently supplemented or even replaced by other mechanisms such as the accumulation of UV-screening pigments, which, in *Spirodela*, requires at least two days.

Damage and repair

DNA as a target for UV-B

Absorbance of UV-B photons by DNA triggers the formation of cyclobutane-pyrimidine dimers (CPDs) and, to a lesser extent, pyrimidine (6–4)-pyrimidinone dimers [(6–4) photoproducts][19]. Apart from being mutagenic, DNA modifications disrupt cellular metabolism. Both RNA- and DNA-polymerase are unable to read through unrepaired dimers, leading to a blockage in gene transcription and DNA replication[19]. Repair of UV-B-damaged DNA is mainly via light-dependent photoreactivation[19]. *Arabidopsis* mutants deficient in light-dependent repair of CPDs[20] or (6–4) photoproducts[21] are UV-sensitive. Photolyases reverse dimerization, restoring the bases to their native form[19,22] (Fig. 1). An *Arabidopsis* photolyase has recently been cloned[13]. Its sequence shows considerable homology with type II photolyases from prokaryotes and animals. Photolyases carry two chromophores – either folate- or flavin-type – that have absorption maxima between 350 and 400 nm. Excitation energy is transferred to a flavin adenine dinucleotide-containing active site, which subsequently furnishes the electron that mediates the splitting of the dimer[22].

The low levels of UV-B required for DNA dimerization[16] are, in the biosphere, always accompanied by considerably higher levels of UV-A (10–20 fold) and PAR (60–600 fold). This makes the UV-A and PAR wavelengths admirably suited to drive photoreactivation and to control photoreactivation capacity. Photoreactivation itself is driven most effectively by blue and UV-A (Fig. 1)[19,22]. In beans, induction of photoreactivation capacity is under phytochrome control, possibly at the level of gene activation (Fig. 1)[23]. In *Arabidopsis*, accumulation of transcripts encoding photoreactivating enzymes is enhanced by PAR, blue or UV-A, but not by red or UV-B wavelengths[13]. An increased ability to repair DNA is an important aspect of adaptation to UV-B radiation. However, it remains unclear to what extent DNA damage and repair determine plant performance under environmentally relevant conditions.

The photosynthetic machinery

UV-B impinges on various aspects of photosynthesis[3,16,24], but effects on PSII have drawn considerable attention[3,7,10,25]. PSII is a highly structured protein–pigment complex (Fig. 2) that catalyses the transfer of electrons from water to plastoquinone. The structurally and functionally similar D1 and D2 proteins form the core of PSII. A very sensitive UV-B response is the rapid light-driven degradation of these two proteins[10]. Degradation is, *in vivo*, discernible at fluences of <1 μmol m^{-2} s^{-1} UV-B. In an environmentally relevant background of PAR, UV-B-driven (but not UV-A-driven) degradation of the D2 and D1 proteins is synergistically accelerated[10]. The degradation response is maximal at 300 nm, with shorter wavelengths having less effect[26]. Rapid PAR-driven turnover of D1 (D2 is stable under PAR) has been proposed to be part of a damage–repair cycle essential for maintaining PSII function under photoinhibitory conditions[27]. By analogy, it is possible that UV-B-driven D1-D2 turnover is also part of a repair cycle, preventing accumulation of UV-inactivated PSII.

D1-D2 degradation and PSII inactivation are distinct processes with different wavelength dependencies. Unlike the D1-D2 degradation reaction, PSII inactivation accelerates at shorter UV-C wavelengths[26]. Accumulation of inactive PSII is commonly measured as a decrease in oxygen evolution or variable chlorophyll fluorescence. Such measurements yield no information about the turnover rate of the damage–repair cycle of PSII. Moreover, although *in vivo* measurements of variable fluorescence are often intended as a measure of radiation-damage to PSII, it is not clear whether decreases in variable fluorescence always proportionally reflect UV-damage. Parallel UV-effects on the oxidizing and reducing sides of PSII might complicate analysis[16]. A further complexity is the possibility of multiple UV-B chromophores, including redox-active tyrosines (Z and D), plastosemiquinones (Q_A^- and Q_B^-), and the manganese cluster of the water-splitting complex[25] (Fig. 2). Identification of these chromophores is based on *in vitro* experiments; however, because of the non-realistic UV-B fluences commonly used in such experiments, the environmental relevance of the findings remains to be proven[25].

UV-B exposure may also result in decreased levels of photosynthetic pigments, altered thylakoid integrity, increased stomatal diffusion, and reduced Rubisco activity[16,24]. The decline in Rubisco activity correlates with a decrease in soluble protein[7,16]. These direct UV-B effects are often seen under high UV-B fluences and/or low accompanying PAR. At lower UV-B fluences, a down-regulation of the transcription of major photosynthetic genes is observable, and this might lead to long-term adaptations[16]. The up-regulation of enzymes of the phenyl propanoid pathway that occurs at the same time suggests that the regulation is controlled rather than being a simple consequence of DNA damage.

The photosynthetic machinery is a potential target for UV-B radiation. However, under field conditions, diminished biomass accumulation does not necessarily correlate with decreases in photosynthetic activity[5,6]. Thus, the relevance of UV-B-induced loss of photosynthetic activity, often observed under lab and/or glasshouse conditions, might not translate to environmentally relevant damage. Nonetheless, it has proven to be an important means of assessing the underlying biochemical mechanisms of UV-B damage, repair and acclimation[18].

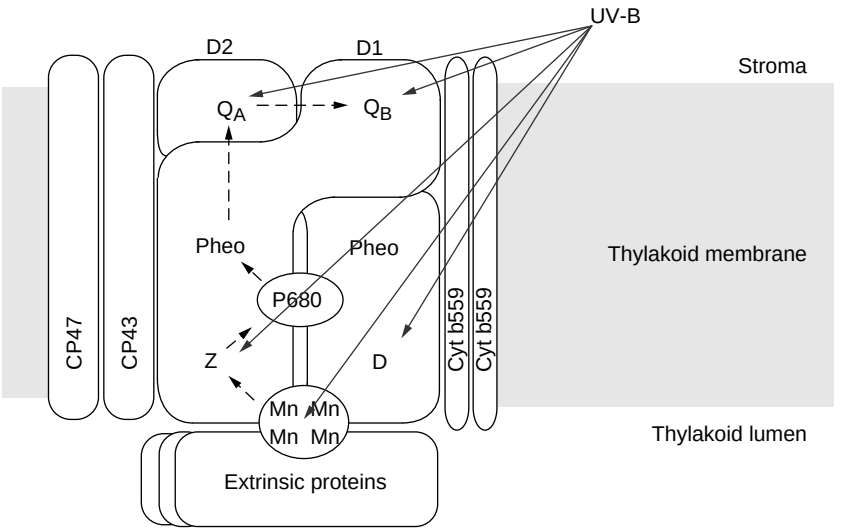

Fig. 2. Schematic presentation of photosystem II (PSII), indicating photosensitizers proposed to be involved in its UV-B-mediated inactivation. P680 is the primary electron donor of PSII. Z and D are redox active tyrosines located on the D1 and D2 proteins, respectively; Z normally serves as the electron donor to P680. Electrons originate from water, the splitting of which is catalysed by a cluster of four manganese atoms. Extrinsic proteins are involved in stabilizing this reaction. On the acceptor site, a pheophytin (Pheo) serves as the primary electron acceptor. The plastoquinones, Q_A and Q_B, are the secondary electron acceptors. Photosensitizers that absorb in the UV-B range and that have been proposed to play a role in PSII inactivation and/or D1-D2 degradation are marked by arrows.

Acclimation responses

Accumulation of secondary metabolites

UV-B induces accumulation of a range of secondary metabolites, which in turn affect numerous physiological functions. Low fluences of UV stimulate the general phenylpropanoid pathway, resulting in accumulation of flavonoids and sinapic esters[28,29]. *Arabidopsis* mutants that do not accumulate flavonoids and/or sinapic esters are highly UV sensitive[29]. Flavonoids and sinapic esters protect by specifically absorbing in the wavelength region from 280 to 340 nm (but not in the PAR waveband, which would diminish photosynthetic yields). Flavonoid accumulation is mainly in the upper epidermal cell layer[14,30]. Indeed, <10% of incident UV-B is generally transmitted through the epidermis[31]. Flavonoids also possess free-radical scavenging activity[32], which might offer additional protection to cells accumulating these compounds.

Regulation of the biosynthesis of UV-screening flavonoids is at the level of transcription and is under the control of a UV-B photoreceptor[7,14]. Analysis of UV-induced accumulation of anthocyanins revealed that the photoreceptor has its maximum activity at 290 nm, and works either alone or in association with phytochrome[15]. UV-B boosts transcript levels for phenylalanine ammonia lyase, 4-coumarate:CoA ligase, chalcone flavone isomerase and dihydroflavonol-4-reductase[7]. Chalcone synthase (CHS) catalyses the committal step in flavonoid biosynthesis and can be activated by UV-B and a variety of other environmental and developmental stimuli[7]. UV-B induced CHS expression is mainly in epidermal cells, where the flavonoids are localized[14]. Analysis of the CHS gene promoter revealed two UV-responsive units that provide binding sites for transcription factors. *Trans*-acting protein factors that bind to the promoter regions, and increase transcription, have been characterized[7]. Likewise, *trans*-acting factors have been identified that down-regulate CHS transcription. The resulting modulation of CHS activity allows the plant to re-direct the flow of intermediates of the phenylpropanoid pathway in response to environmental stimuli.

Polyamines, waxes and specific alkaloids have all been suggested to contribute to UV-tolerance. Polyamines accumulate in response to environmentally relevant doses of UV-B and PAR[33]. In soybean, a correlation was found between levels of polyamines and tolerance to UV-B[33]. Possibly, the radical-scavenging activity of polyamines (and polyamine conjugates) moderates UV-B radiation stress, as was demonstrated for other free-radical scavengers[18].

Selected alkaloids that absorb UV-B wavelengths, or possess free-radical scavenging activity, might also contribute to UV-protection. Levels of UV-absorbing tetrahydrocannabiol increase linearly with UV-B dose in *Cannabis*[34]. Similarly, cannabinoid content increases with altitude at which plants are grown. UV-induced accumulation responses could be of economic interest, as many alkaloids are used therapeutically.

The cuticle can attenuate UV-B penetration, either through reflection or through absorption by soluble flavonoids localized in the waxy layer, or by ferulic acid co-polymerized with cutin. In *Dudleya*, accumulation of glaucescence, a powdery wax, increases reflection of UV-B to a larger extent (about 25%) than that of PAR[35]. However, no differential UV-B sensitivity was discernible in pea lines that differ quantitatively in wax deposition and composition[36]. Possibly, UV-B reflection is only significant in heavily glaucous plants, such as the succulent *Dudleya*.

Free-radical scavengers

Active oxygen species play a role in mediating UV-damage[16,29,37]. Scavenging of active oxygen and other radical species, through either enzymatic or non-enzymatic systems, can alleviate UV stress[18]. In turn, low fluences of UV-B induce scavenging capacity. Levels of the key antioxidants glutathione and ascorbate are up-regulated in response to UV-B[38]. Similarly, UV-B boosts

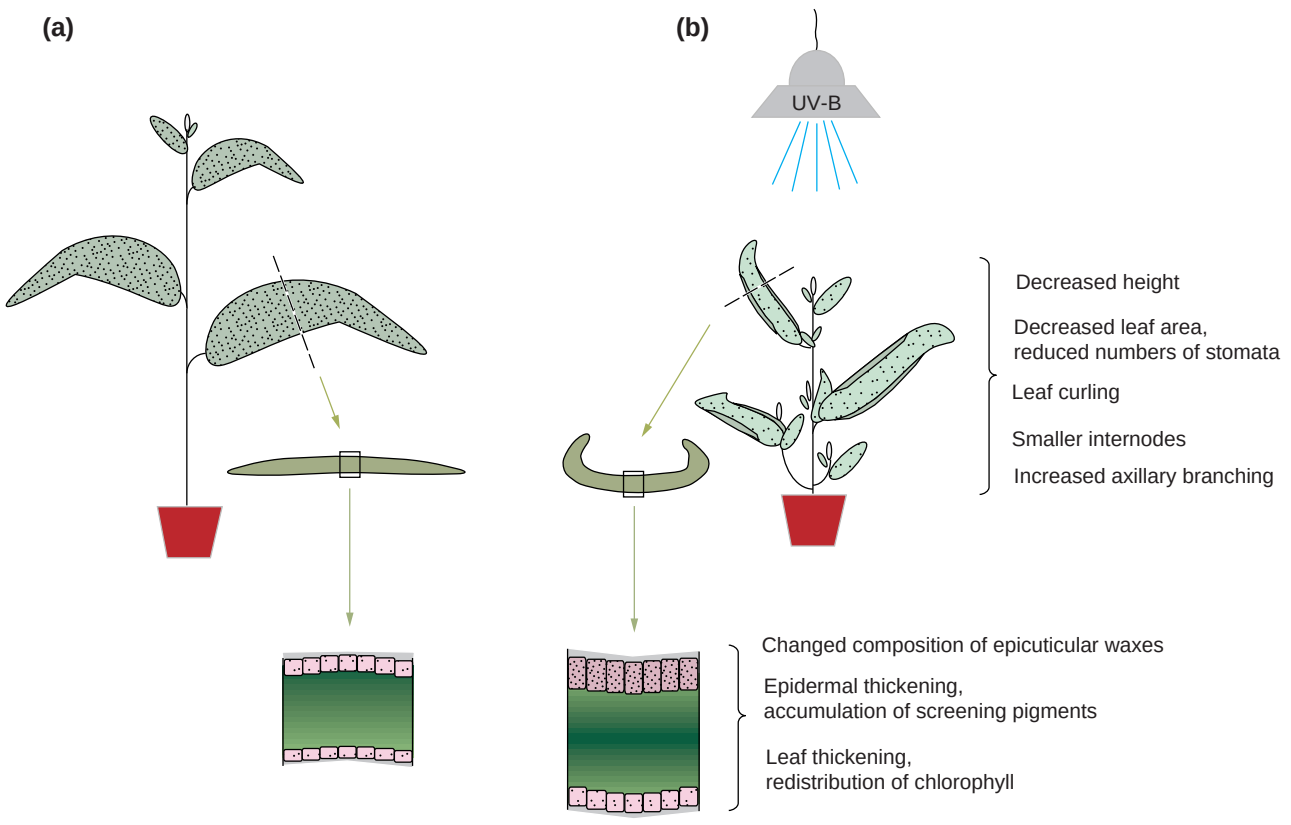

Fig. 3. Diagram showing UV-B-induced changes in leaf and plant morphology. Part (a) is the control; part (b) is a plant exposed to supplementary UV-B.

activities of superoxide dismutase[18,37] (SOD), gluthathione reductase[18,37] and ascorbate peroxidase[29,38]. Genes encoding scavenging enzymes are differentially expressed in response to UV-B: transcript levels of glutathione reductase and glutathione peroxidase rise, while those of SOD remain unaltered or even drop[16,17]. Similar differential responses have also been noted after exposure to O_3 or SO_2 (Ref. 17).

Peroxidase activity increases substantially in *Arabidopsis* following UV-exposure[37]. The increase is a well-regulated process involving the induction of specific isoforms of the enzyme[37]. Interestingly, UV-B also induces NADPH-oxidase activity, leading to peroxide production[37]. Peroxide scavenging by anionic peroxidases results in the formation of phenoxy radicals that can spontaneously polymerise, leading to lignification. In field studies, lignin accumulation reportedly increases in UV-B-exposed plants[6]. Possibly, lignification is beneficial for the UV-exposed plant, as conjugates of lignin with ferulic acid and other phenylpropanoids contribute to UV-screening. Additionally, lignification may be of ecological and economic importance, as it affects the digestibility of the plant and decomposition of plant litter[6].

Whole-plant responses

UV-B induces changes in leaf and plant morphology (Fig. 3)[6]. The mechanism underlying these alterations is not clear. Leaf curling is a photomorphogenic response, observable at low fluences of UV-B, that helps diminish the leaf area exposed to UV[7]. A protective function has also been hypothesized for leaf or epidermal thickening, as this would increase the length of the UV-B screening pathway (Fig. 3). Indeed, the thick epidermis of field-grown conifers screens UV-B exceptionally efficiently[31]. In pea, leaf thickening is accompanied by a redistribution of chlorophyll away from the adaxial surface (Fig. 3)[28]. UV-B also induces changes in leaf shape, possibly resulting from a non-homogeneous inhibition of growth[7].

Changes in plant morphology occur in the absence of decreases in biomass accumulation, and may be triggered by a UV-B photoreceptor[6,7]. UV-B also interferes with indoleacetic acid (IAA) metabolism, possibly via photooxidation of IAA, resulting in hormonal imbalances that would certainly induce morphogenic effects[11]. IAA photooxidation was studied under lab conditions that might not directly relate to environmentally relevant conditions. However, in field trials with relevant doses of UV-B, some of the observed morphological alterations (i.e. branching) resemble known IAA effects. Developmental alterations might also be controlled through jasmonic acid. This signalling molecule, derived from linolenic acid, is produced upon exposure of membranes to UV-B or other stresses[39]. The changes in plant morphology that result from UV-B exposure are thought to be of greater importance for the competitive balance between species than changes in photosynthesis[12]. Shifts in competitive balance might, ultimately, lead to an alteration in the composition of natural vegetation or in the productivity of agricultural systems.

Conclusions

The biological impact of UV-B radiation on plants is a combined function of damage, repair and acclimation. Multiple targets for UV-B radiation have been identified, although little is known about their environmental relevance. Acclimation and repair mechanisms that diminish the damaging effects of UV-B radiation have evolved[9,12]. This is especially true for plants from regions of naturally high levels of UV-B irradiation[9]. Since plants

are exposed to continuously varying levels of UV-B, they may well be continuously adjusting their UV defence. Thus, future challenges are not limited to the elucidation of adaptive pathways, but also need to address the process of adjustment and coordination of the multiple protective mechanisms in a photobiologically dynamic environment.

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