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REVIEW ARTICLE

Impact of UV-B radiation on photosynthesis – an overview

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Abstract

Ultraviolet-B (UV-B) radiation constitutes a minor part of the solar spectrum, being most of this solar radiation absorbed by the UV-screening stratospheric ozone layer. Yet, a global depletion of the ozone layer, largely due to the release of chlorofluorocarbons caused by human activities, has resulted in an increase of solar UV-B radiation at the earth's surface. Accordingly, in the temperate latitudes, such ozone decrease reached ca. 3% and 6% in the North and South hemispheres, respectively, between 2002 and 2005 (as compared to the 1970s). Despite the uncertainty of long-term predictions, it is also estimated an UV-B increase of 5-10% over temperate latitudes within the next 15 years In this context, this work aim at to present an overview of plants sensitivity/tolerance to UV-B irradiation mostly considering the key photosynthetic metabolism.

Key words: Ozone depletion, Photosynthesis, Reactive oxygen species, Ultraviolet-B radiation

Introduction

Ozone depletion and UV-B radiation on Earth's surface

Most of UV radiation does not reach Earth's surface due to its interaction to the atmospheric components. In fact, UV-C radiation might be completely absorbed by the atmospheric gases, UV-B radiation is absorbed by the stratospheric ozone layer, whereas UV-A radiation is hardly absorbed by this layer. The ozone layer can be depleted by free radical catalysts, including nitric oxide, nitrous oxide and hydroxyl, as well as atomic chlorine and bromine. Although there are natural sources for all of these species, the concentrations of chlorine and bromine have increased markedly in recent years due to the release of large quantities of man-made organohalogen compounds, especially chlorofluorocarbons, with a half-life ranging from 50 to 150 years (Madronich et al., 1998). These highly stable compounds are capable of surviving the rise to the stratosphere, where Cl and Br radicals are produced by the action of UV radiation. Each radical is then able to initiate and catalyze a chain reaction capable of breaking down over 100,000 ozone molecules. Such breakdown of ozone molecules in the stratosphere can result in a decrease of the effectiveness of UV radiation absorption and therefore more radiation reaches the Earth, with each 1% reduction in ozone causing an increase of 1.3-1.8% in UV-B radiation reaching the biosphere (Caldwell and Flint, 1994; McKenzie et al., 2003).

Nowadays ozone levels over the northern hemisphere have been dropping by 4% per decade. Much higher seasonal declines have been measured in approximately 5% of the Earth's surface, around the north and south poles, constituting the so called ozone-holes. Current stratospheric ozone levels are at the lowest point since measurements began in 1970s and global terrestrial UV-B radiation levels range between 0 and 12 kJm^{-2} on a given day, with near Equator and mid-latitudes receiving higher doses (McKenzie et al., 2011). The changes in ozone and UV-B are not uniform over the Earth's surface (Figure 1). The ozone concentrations in the high latitudes (comprising Antarctic and Arctic regions) are 40–50% lower than the pre-1980 values; in the mid-latitudes (35–60ºN and 35–60ºS) are 3–6% lower than the pre-1980 values; and, at the Equator, show minimum changes (Forster et al., 2011).

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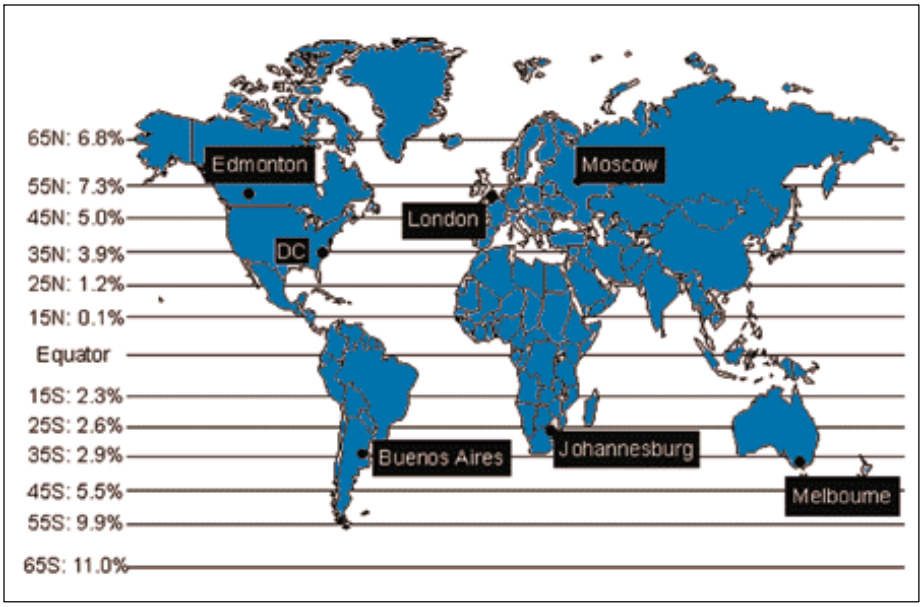

Figure 1. A 1996 study using satellite-based analyses of UV-B trends demonstrated that this radiation levels had increased at ground level. This figure shows the percent increases in average annual UV-B reaching the surface over the past 10 years. UV-B incidence is strongly dependent on latitude.
(http://www.epa.gov/airtrends/aqtrnd95/stratoz.html)

Current levels of UV-B during the cropping season are somewhere between 2-12 kJm^{-2} per day on the Earth's surface, which includes an increase of 6-14% of UV-B radiation (Forster et al., 2011) over the pre-1980 levels. A 30% increase in UV-B results in a maximum amount of 2.44 kJm^{-2} per day in UK (Forster et al., 2011), but such low levels of UV-B radiation are very uncommon during the cropping season in several parts of the world. For example, in the Cotton Belt of USA, current UV-B radiation levels are 4–11 kJm^{-2} per day during the summer season (Frederick et al., 2000), and the predicted UV-B levels based on Taalas et al. (2000) would be 4.56–12.54 kJm^{-2} per day. In China, ambient UV-B levels during soybean cropping period are in average of 8.85 kJm^{-2} per day (Li et al., 2002). A 30% increase in UV-B levels is expected to seriously affect crop production in these and other parts of the world.

Target sites of UV-B radiation in the photosynthetic pathway

UV-B radiation (280–320 nm) is a minor part of the solar spectrum, although the component that reaches Earth's surface is a promoter of a large number of responses in higher plants at the molecular, cellular and whole-organism level (Caldwell et al., 2007; Jenkins, 2009). In fact, UV-B radiation is readily absorbed by a large number of biomolecules (*e.g.*, nucleic acids, proteins, lipids), therefore leading to their photoexcitation what might promote changes on multiple biological processes, both with damaging or regulatory importance (Jenkins, 2009; Tian and Yu, 2009). Nevertheless, cconsiderable intra- and interspecific variability in sensitivity of crop plants to UV-B radiation have been reported (Teramura, 1983; Bornman and Teramura, 1993; Correia et al., 1998; Mazza et al., 2000), including at the photosynthetic level. In general, leaf photosynthesis might decrease more by enhanced UV-B radiation under growth chamber or glasshouse conditions than under field conditions due to low PAR or a low ratio of PAR to UV-B in the chambers. Despite the diversity of UV targets in plants, it seems that the photosynthetic apparatus is amongst the main action targets of UV-B, and its damage contributes significantly to the overall UV-B damage (Lidon and Ramalho, 2011; Lidon et al., 2012; Lidon, 2012).The photosynthetic pathway responses to UV-B radiation depend on experimental growth conditions and plant growth stage, UV-B dosage, flow rate and the ratio of PAR to UV-B radiation, as well as on the interaction with other environmental stresses (*e.g.*, cold, drought, mineral availability) (Bornman and Teramura, 1993; Tevini, 2004; Jenkins, 2009). The photosynthetic structures are widely impaired by UV-B radiation with impact

observed at several levels, namely through the induction of tissue chlorosis and necrosis, changes in leaf ultrastructure and anatomy (*e.g.*, in the thickness of epidermal and palisade mesophyll cell layers), degradation of photosynthetic pigments and thylakoid electron transport carriers (Bornman and Teramura, 1993; Tevini, 2004; Jenkins, 2009; Lidon and Ramalho, 2011).

Stomatal regulation and CO_2 fixation

Stomatal regulation is an important process limiting leaf photosynthesis. Although earlier studies have proposed that UV-B radiation does not significantly affect stomatal conductance (Teramura et al., 1984; Murali and Teramura, 1985, 1987; Agrawal et al., 1991; Keiller et al., 2003), several other studies demonstrated reduced stomatal conductance in response to UV-B radiation (Dai et al., 1992; Middleton and Teramura, 1993; Pal et al., 1998, 1999; Lidon and Ramalho, 2011). Also, Jansen and van-den-Noort (2000) found that high UV-B might stimulate either stomatal opening or closing in *Vicia faba*, depending on the metabolic state. However, although differences exist in genotypes tolerance to UV-B radiation (Correia et al., 1999), the induced reduction of stomatal conductance can barely be responsible for CO_2 limitation in several crops (Agrawal et al., 1991; Teramura et al., 1991; Ziska and Teramura, 1992; Zhao et al., 2003; Lidon and Ramalho, 2011). In fact, the stomatal conductance reduction is usually in much smaller extent than that of the net photosynthetic rate. Furthermore, the calculated intercellular CO_2 concentration of plants exposed to UV-B radiation presented values close or even higher than that of untreated control plants (Agrawal et al., 1991; Zhao et al., 2003; Lidon and Ramalho, 2011). All together, these results point to mesophyll imbalances that may arise from damages and/or regulatory mechanisms, both at biochemical and biophysical level (Lidon and Ramalho, 2011).

In fact, ribulose-1,5-bisphosphate carboxylase/oxygenase (rubisco) content and activity are also strongly affected by UV-B radiation in many field crops (Vu et al., 1982, 1984; Strid et al., 1990; Nedunchezhian and Kulandaivelu, 1991; Jordan et al., 1992; He et al., 1993, 1994; Huang et al., 1993; Kulandaivelu and Nedunchezhian, 1993; Mackerness et al., 1997; Correia et al., 1999; Savitch et al., 2001). That will affect V_{cmax} and, therefore, contribute to photosynthesis depression (Iwanzik et al., 1983; Teramura et al., 1991; Keiller et al., 2003; Tevini, 2004; Lidon and Ramalho, 2011; Lidon et al., 2012). Additionally, when the effect of UV-B with or without UV-A radiation on the mechanisms of UV-B reduced photosynthesis are considered, Savitch et al. (2001) found that in *Brassica napus* submitted to 200 μmol m^{-2} s^{-1} PAR the decrease in the CO_2 assimilation capacity for PAR + UV-B treated plants was associated with a decreasing capacity for sucrose biosynthesis and limited triose-P utilization. In addition, the RuBP regeneration (Allen et al., 1997; Savitch et al., 2001) and the amount of sedoheptulose 1,7-bisphosphatase (Allen et al., 1998) also decreases upon UV-B radiation exposure. On the other hand, high UV-B irradiance in combination with low PAR levels produces significant reduction in the concentration of carboxylating enzymes, whereas high PAR (higher than 1,000 μmol^{-2} s^{-1}) together with low UV-B levels do not affect Rubisco activity (Barbato et al., 1995). UV-B-induced inactivation of Rubisco could be due to modification of the peptide chain, degradation of the protein, and/or diminished transcription of the gene (Jordan et al., 1992; Caldwell, 1993). Furthermore, it is apparent that the UV-B-induced reduction in Rubisco is greater in UV-sensitive than in UV-resistant strains. These findings pose two questions: why did supplementary UV-B radiation causes a marked reduction in Rubisco; and why was this effect greater in UV-sensitive, than in UV-resistant strains? A possible explanation can be linked to the modification of proteins by photooxidation, or by reactive oxygen species (ROS) and free radicals produced during photosensitization (Caldwell, 1993; Foyer et al., 1994). These modifications would include cross-linking, aggregation, denaturation and degradation (Andley and Clark, 1989; Wilson and Greenberg, 1993; Borkman and Mclaughlin, 1995; Greenberg et al., 1996).

Photochemical reactions

As stated above, a major impact site of UV-B radiation is the chloroplast, leading to the impairment of the photosynthetic function (Bornman, 1989; Allen et al., 1998; Lidon et al., 2012). Furthermore, the integrity of the thylakoid membrane and structure seems to be even more sensitive than the activities of the photosynthetic apparatus bound within (Lidon and Ramalho, 2011; Lidon et al., 2012). Negative effects on several photosynthetic components are known, including through suppression of chlorophyll synthesis (Kulandaivelu et al., 1991), the inactivation oxygen evolution, LHCII, PSII reaction centres functionality and the thylakoid electron flux. Due to

the key role of LHCII in light absorption and energy transfer to the reaction center, as well as on thylakoid stacking, any damage to these structures can result in multiple effects on the photosynthetic functioning. Furthermore, it must be considered that UV-B radiation inhibition of LHCII (Vu et al., 1982, 1984; Strid et al., 1990; Lidon et al., 2012) is also eventually linked to a decrease in the transcription of the cab gene responsible for the synthesis of the chlorophyll a/b-binding proteins of LHCII, which may lead to the functional disconnection of LHCII from PSII (Jordan et al., 1994).

Numerous studies have also showed that in photophosphorylation processes, PSII is the most sensitive component of the thylakoid membrane on exposure to UV-B radiation (Brandle et al., 1977; Noorudeen and Kulandaivelu, 1982; Kulandaivelu et al., 1991; Melis et al., 1992; Allen et al., 1998; Chaturvedi et al., 1998; Correia et al., 1999; Bolink et al., 2001; Savitch et al., 2001; Lidon et al., 2012), what would be related to rapid degradation of the D_1 and D_2 proteins of PSII (Lidon et al., 2012). Moreover, relatively to PSII, UV-B radiation has a smaller effect on PSI (Kulandaivelu et al., 1991) and cytochrome b_6/f complex (Cen and Bornman, 1990; Lidon et al., 2012), although strong UV-B mediated effects on PSI linear electron transport (Lidon and Ramalho, 2011) and on cyclic phosphorylation (Pang and Hays, 1991) were repoted.

Within PSII, UV-B radiation acts on either the reaction center itself, producing dissipative sinks for excitation energy, which quenches the variable fluorescence and/or in the reducing site of PSII (Iwanzik et al., 1983; Lidon and Ramalho, 2011). Nevertheless other works suggest that the quinone electron acceptors (Melis et al., 1992), the redox active tyrosines (Tyr-Z, Tyr-D), or the primary step of PSII electron transport (Iwanzik et al., 1983; Kulandaivelu et al., 1991) are the primary targets of UV-B action in PSII electron transport. In this context, the water-oxidizing complex (OEC) seems to be UV-B sensitive (Lidon et al., 2012). Since the Mn cluster of water oxidation is the most fragile component of the electron transport chain, UV-B absorption by the protein matrix or by other redox components may lead to conformational changes and inactivation of the Mn cluster. Q_A and Q_B acceptors have also been frequently suggested as sensitizers of D_1 and D_2 protein damage due to the similarity of the action spectrum of D_1 protein degradation and the absorption spectrum of plastosemiquinones (Jansen et al., 1996). Thus, the importance of the donor side components of PSII primarily that of the water-oxidizing system, in sensitizing D_1 protein cleavage via hydroxyl radical formation must also be considered (Lidon et al., 2012). Indeed, a potential primary catabolism involved in UV-B induced physiological and biochemical injury has been related to the production of ROS (Caldwell, 1993; Foyer et al., 1994; Hideg et al., 1997). Through this pathway, triplet molecular oxygen continuously produced during light-driven photosynthetic electron transport, in the water splitting complex coupled to PSII, can be converted in the sequential reduction to superoxide, hydrogen peroxide and hydroxyl radical (Lidon and Henriques, 1993; Apel and Hirt, 2004).

Protection against UV-B radiation

Recovery from UV-induced dysfunction of enzymes is expected to involve protein and DNA synthesis and/or repair,, both in the chloroplast and nuclear DNA. Among proteins, the functions of D_1 and D_2 from PSII can be partially restored after UV-B damages. D_1 protein is rapidly turned over *in vivo* in a short time as 30 min (Wilson and Greenberg, 1993). On the other hand, D_2 degradation is activated by distinct photosensitizers in the UV-B and visible region of the spectrum and it has been suggested that its degradation is coupled with that of D_1, being influenced by events occurring at the quinone niche on the D_1 protein.

As defensive strategy, plants may trigger machanisms for the dissipation of excess excitation energy. Excess energy imposed to the photosynthetic apparatus may be thermally deactivated through photochemical and non-photochemical quenchers (Lidon and Ramalho, 2011). One of the main routes of heat dissipation is the xanthophylls cycle, in which violaxanthin is converted to zeaxanthin when the level of captured energy is higher than that used through photochemical events, what could happen even under moderate irradiance and depends of the species, ecological history of the plant and if environmental stresses that reduce the photochemical energy use are involved (Adams and Demmig-Adams, 1992; Ramalho et al., 2000; 2003; Batista et al., 2011). The key enzyme of this cycle is violaxanthin deeposidase, which is sensitive to UV-B radiation. Nevertheless, this process further links the catabolic action of ROS, which must also be controlled due to its damaging action over a wide number of biological molecules and structures (Lidon et al., 2012), among them prteins, lipids and nucleic acids (Öquist, 1982; Logan, 2005; Fortunato et al., 2010; Partelli et al., 2011).

The production of ROS is an inevitable consequence of photosynthetic activity (Ensminger et al., 2006), as under normal metabolic conditions, 10-30% of the thylakoid electron transport might lead to O_2 photoreduction (Bartoli et al., 1999; Logan, 2005), what further increases under stressful conditions that decrease the photochemical energy use. Oxygen itself is a strong oxidant since it possesses two unpaired electrons in its outermost π orbital. The reduction of oxygen by nonradical species needs the transfer of two electrons having parallel spins to oxygen in order to fit with parallel spins of two unpaired electrons. Oxygen, therefore, got converted to ROS by univalent reduction or by energy transfer. The more common ROS produced in plant include superoxide, hydrogen peroxide and hydroxyl radical.

The superoxide radical ($O_2^{\bullet-}$) in aqueous solution has a pKa of 4.9 and might occur in physical, chemical and biological processes (Lidon and Henriques, 1993). Moreover, hydrogen peroxide is a powerful oxidizing agent (E^O = + 1.36 V, at pH 7 for the system H_2O_2/H_2O), being the transition metals involved in its synthesis throughout the protonation of $O_2^{\bullet-}$(Lidon and Henriques, 1993), whereas hydroxyl radical has a short lifetime and a strongly positive redox potential (+2 V), being produced throughout the catalyzed Haber-Weiss cycle and therefore dependent on both H_2O_2 and $O_2^{\bullet-}$ (Lidon and Henriques, 1993).

ROS production arises in plant cells via a number of routes. Most of these highly reactive molecules of oxygen are formed in plant cells via dismutation of superoxide as a result of single electron transfer to molecular oxygen in electron transfer chains mainly during the Mehler reactions in chloroplast which is the higher source of ROS (Logan, 2005; Asada, 2006). The lack of $NADP^+$ in PSI, due to redox imbalance, causes spilling of electron on molecular oxygen, triggering the generation of superoxide. The majority of superoxide *in vivo* is thought to be produced via electron spilling from reduced ferridoxin to oxygen. Superoxide formed then undergoes dismutation, either spontaneously or facilitated by superoxide dismutases. Superoxide radicals generated by one electron reduction of molecular oxygen by Mehler reaction in PSI are rapidly converted into hydrogen peroxide by chloroplast Cu-Zn-superoxide dismutase, which is then removed by the action of ascorbate peroxidases and catalases. Yet, if such removal is compromised or insufficient much more reactive hydroxyl radicals can be formed from superoxide and hydrogen peroxide through Fe catalyzed Haber-Weiss reaction (Foyer and Harbinson, 1994; Logan, 2005; Bhattacharjee, 2010; Fortunato et al., 2010). That would further promote damages, namely at the level of the lipid matrix of chloroplast membranes (Harwood, 1998; Campos et al., 2003; Partelli et al., 2011) where the double links of polyunsaturated fatty acids (FAs) are preferential targets of ROS and free radicals (Öquist, 1982; Girotti, 1990). In addition, peroxy and alkoxy radicals formed as intermediates in membrane lipid peroxidation are also very toxic at high concentration and poses threat to several biomolecules (Lidon and Henriques, 1993). Therefore, since the production of ROS is an metabolic inevitable consequence (overexpressed under excess photochemical energy), plants have evolved efficient strategies by devising and integrating antioxidative defense mechanism that avoids the production (as the thermal dissipation mechanisms referred above) of promotes the scavenging of ROS, through a integrated network of enzyme (*e.g.*, Cu,Zn-superoxide dismutase, ascorbate peroxidase) and non-enzyme (ascorbate, glutathione) molecules acting through the ascorbate-glutathione (Haliwell-Asada) cycle to overcame the imposed oxidative stress conditions.

Although in living tissues, accumulation of ROS imposes ultimately oxidative stress and cellular damages, it seems that at low concentrations these highly reactive species may also act as signaling molecules or second messengers, therefore implicated in the modulation of normal plant development, including senescence (ageing) and many other physiological processes (Dröge, 2002; Bhattacharjee, 2012). In this context, increased levels of UV-B radiation can stimulate the generation of elevated amounts of ROS (Bhattacharjee, 2012; Lidon et al., 2012; Xie et al., 2012), thus enhancing the expression of several genes involved in natural senescence phenomena, where ROS are implicated (Hollósy, 2002).

Apart from their role in accelerated ageing and other developmental processes, ROS, including nitric oxide (Kliebenstein et al., 2002), can be seen as triggering agents of defense mechanisms against a range of stress factors, including UV-B radiation (Lidon and Henriques, 1993; Bhattacharjee, 2012; Lidon et al., 2012). In this context, Wang et al. (2009) supported that nitric oxide appeared to act in the same direction or synergistically with other ROS to induce ethyl synthesis in a defensive response under UV-B radiation in maize leaves. Also, it was reported that UV-B induced an

increase of nitric oxide that may act as a second messenger and perform antioxidant response to UV-B radiation (Zhang et al., 2003). Tossi et al. (2009) reported that when maize seedlings are UV-B-irradiated, cellular damage occurs and ROS becomes widely distributed in chloroplasts and mesophyll cells. Pretreatment with apocynin and coinciding nitric oxide accumulation prevented this damage. They also suggested that UV-B perception triggers an increase in abscisic acid concentration, which activates NADPH oxidase and H_2O_2 generation. Moreover, sensitivity to UV-B radiation also follows the rate of sequential reduction of triplet molecular oxygen produced during the photosynthetic light reactions (Lidon et al., 2012). After photoreduction of this chemical entity to superoxide and dismutation to hydrogen peroxide hydroxyl radicals are produced. Yet the rate of zeaxanthin production is stimulated in the xanthophylls cycle, limiting photodegradation of isoprenoids (Lidon et al., 2012). Moreover, UV-B radiation might become lethal if an unbalanced ascorbate peroxidation develops, as this process limits the functioning of the enzymatic antioxidant systems (i.e., the Asada Halliwell cycle), mostly because of substrate limitations for the kinetics of ascorbate peroxidase (Lidon et al., 2012). In this context, ROS can trigger an increasing lipid peroxidation (particularly of monogalactosyldiacylglycerol class) and photosystems proteolysis, as well as degradation of thylakoids structure and functioning (Lidon et al., 2012).

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