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UV-B radiation induced alterations in plant photosynthetic processes of the thylakoid membranes in higher plants

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Abstract

In this article an attempt has been made to analyze the effect of UV-B radiation in primary events of photosynthesis under in vivo and in vitro conditions. It particularly alters photosystem II by causing inhibition in electron transport activity. In contrast it induces less inhibition in the PS I activity by altering plastocyanin. It also induces lipid peroxidation in thylakoid membranes during UV -B stress.

Introduction:

UV radiation is one of the serious issues since past few decades due to industrialization. Increase in the industrialization results in the increase in anthropogenically important atmospheric pollutants such as chlorofluorocarbons (CFCs), halocarbons, chloroform (MCF) and dioxins (NOx). Considerable amounts of natural production of reactive nitrogen species (RNS) such as nitric oxide (NO⁻), peroxynitrate (ONOO⁻) and nitrous oxide (N₂O) from unpolluted aquatic and terrestrial ecosystems also contribute to the depletion of ozone layer (Kramlich and Linak, 1994). These pollutants are being responsible for the depletion ozone layer in the stratosphere that helps in screening of UVR (Singh et al., 2010b). Due to increase of the pollutants depletion of ozone layer is occurring not only in the Antarctic region but also all over the earth surface resulting in the subsequent increase of UV radiation (UVR; 280-400 nm) entering to the earth surface (Crutzen, 1992). The depletion of ozone layer in Antarctic region has been reported to be more than 70% (Smith et al., 1992).

These harmful UV rays reaching the earth surface effects the production of algae and photosynthetic macrophytes as they can easily absorb UV light by biomolecules such as nucleic acids and proteins (Hader *et al.,* 2007; Fernanda Pessoa, 2012). All this is because UV-B radiation can penetrate into the water upto a depth of 20-30 m (Smith *et al.,* 1992). Aquatic organisms like algae and photosynthetic macrophytes which grow in marine ecosystem are the support for entire life, because these aquatic organisms mainly produce food, for aquatic organisms like fishes sponges shelter, as O₂ supplement and as p^H regulators. This negative impact of UV-radiation is not only for the primary producers but also to various aquatic and terrestrial ecosystems ranging from prokaryotes to eukaryotes i.e. from lower to higher plants, animals and also human (Norval *et al.,* 2007).

UV-B radiation and its impact on photosynthesis:

Blue-green algae (early aquatic organisms) began to use solar energy to convert H_2O and CO_2 molecule into organic compounds and molecular oxygen (O_2) through a process now called photosynthesis since ages (Dismukes *et al.*, 2001). Some of the photosynthetically created oxygen is combined with organic carbon to produce CO_2 molecules. The remaining O_2 accumulated in the atmosphere and touched off a massive ecological disaster with respect to early existing anaerobic organisms (Blankenship, 1992; Dismukes *et al.*, 2001).

UV-B radiation targets at molecular level:

Ultraviolet-B radiation have been studied during the last thirty years in small growth cabinets, growth chambers and green houses or in the field supplementing white light or ambient solar UV radiation with artificial UV-B or attenuating (or even excluding) the UV-B from the solar light. Supplementation studies were useful elucidating UV stress responses and accompanying mechanisms; however, they are less reliable in providing estimates for natural habitats, where many environmental parameters such as water and mineral stress and/or high sunlight irradiances are often interfering. Attenuation of solar UV-B by appropriate filters, such as ozone or plastic films, avoids the use of artificial UV (Tevini and Teramura, 1989; Mark *et al.*, 1996). However by this method only relative UV-B enhancement compared to the reduced solar UV-B can be evaluated. Present studies reveals that UV-B acts on macromolecules and molecules of biological importance, perturbing the processes in which they are involved as described below (McKenzie *et al.*, 2003). DNA is one of the most notable targets of UV. Irradiation in both the UV-B and UV-C regions results in a multitude of DNA photo products (Mac Kerness *et al.*, 1998), which may cause mutations during replication (Jiang and Taylor, 1993; Buma *et al.*, 2003). DNA

in detail in human beings, mammals, fungi and cyanobacteria (Stapleton, 1992; Malloy *et al.*, 1997; Buma *et al.*, 2003; Hader and Sinha, 2005; Wulff *et al.*, 2008).

exposure (Smith, 1989; Cannon et al., 1995; Kumar et al., 2004). UV induced damage to DNA has been studied

UV-B impact on photosystem II:

PS II is the membrane protein complex found complex found in oxygenic photosynthetic organisms (higher plants, green algae and cyanobacteria), which harnesses light energy to split H_2O into O_2 , protons and electrons (Anderson and Styring, 1991; Scmidth et al., 2010; Yunsheng et al., 2011). There is general consensus that UV-B radiation influences primarily PS II, there are many different reports on possible targets (Tevini, 2004; Imre Vass., 2011; Takahashi, et al., 2011). Different techniques were used to reveal the possible target sites of UV-B radiation such as fluorescence induction, flash-induced absorption changes, and measurement of O2 evolution. However it seems to be well established that the redox components of PS II are affected by UV-B to some degree. From previous experiments it has been assumed that UV-B acts on either reaction centre itself, producing dissipative sinks for excitation energy, which quenches the variable fluorescence and/ or the reducing site of PS II (Iwanzik et al., 1983; George et al., 2011). Recent comparative studies indicated the water oxidizing complex as the most UV-sensitive part of PS II (Bornman and Sundby-Emanuelsson, 1995). Since the Mn cluster of water oxidation seems to be the most fragile component of the electron transport chain, UV-B absorption by the protein matrix or other redox components may lead to conformational change and inactivation of the Mn cluster. Most observations support the notion that UV-B preferentially inactivates the water oxidizing complex with additional effects on the QA and QB acceptors, as well as on the Tyr-Z and Tyr-D donors (Rengeret al., 1989; Vass et al., 1996; Giacometti et al., 1996; Tystjarvi, 2008).

The acceptor or reducing side of the D_1 and D_2 proteins can be modified by UV-B radiation with a subsequent change in the number and activity of quinone binding sites. Specifically, it has been suggested that UV-B radiation primarily modifies the binding sites on the PS II acceptor side with a simultaneous blocking of pheophytin, the primary electron acceptor (Renger *et al.*, 1986b). UV-B radiation also decreases chlorophyll fluorescence with the fast components accelerated and the slow components retarded, suggesting the formation of additional quenchers of exciton energy in reaction centers (Renger *et al.*, 1991). It has been indicated that plastoquinone with its three redox states (quinine, semiquinone anion and the quinol) may act as a primary UV-B photosensitive molecule since all these forms absorb to the same extent in the UV-B region (Melis *et al.*, 1992).

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Recently, it was shown that UV-B induces both structural and excitonic uncoupling of chlorophyll with in the light harvesting complexes. Transient absorption measurements and low frequency infra red and Raman spectroscopy show that the predominant sites of UV-B damage in PS II are at the OEC itself as well as specific locations near the OEC-binding sites (Lukins et al., 2005). A combination of high intensity PAR and UV-B Radiation results in enhanced rates of photodamage and degradation of the D_1 protein (Greenberg *et al.*, 1989) although UV-B driven protein cleavage occurs at different sites as compared to that induced by PAR and is thought to be independent of the presence of oxygen (Melis et al., 1992; Barbato et al., 1995). Under supplemental UV-B, both D₁ and D₂ proteins are subjected to photodamage (Melis et al., 1992; Jesen et al., 1993; Friso et al., 1994; Vass et al., 1996). The interplay between PAR and supplemental UV-B radiation and the role of the later in photodamage and turnover of the D_1 and D_2 proteins are questions of current interest (Masi and Melis, 1997).

Impact on LHC II of PS II:

PS II is surrounded by its light harvesting antenna which is comprised of the inner minor antenna complex (built by CP24, CP26 and CP29, encoded by the genes LHC b 4, 5 and 6) and the outer major antenna complex LHC II (Susann et al., 2011; Kolyo et al., 2011). Its function is a topical area of research, not only because of its prevalence and light harvesting role, but also because it is a key target of several signal transduction pathways that control light energy used. UV-B radiation decreases the transcription of the *cab* genes responsible for the synthesis of Chl *a/b* binding proteins LHC II and may lead to the functional disconnection of LHC II from PS II (Jordan et al., 1994). In addition, it was showed that the increase in pr1 transcript and decrease in LHCb transcript in response to UV-B exposure in Arabidopsis thaliana is mediated through pathways involving hydrogen peroxide derived from superoxide anion (Mackerness et al., 2001).

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