Journal of Photochemistry & Photobiology, B: Biology 153 (2015) 334–343

Contents lists available at ScienceDirect

Journal of Photochemistry & Photobiology, B: Biology

journal homepage: www.elsevier.com/locate/jpb

Short Review

Changing scenario in plant UV-B research:UV-B from a generic stressor to a specific regulator

Parul Parihar [a], Samiksha Singh [a], Rachana Singh [a], Vijay Pratap Singh [b,*], Sheo Mohan Prasad [a,*]

[a] Ranjan Plant Physiology and Biochemistry Laboratory, Department of Botany, University of Allahabad, Allahabad-211002, India
[b] Govt. Ramanuj Pratap Singh dev Post Graduate College, Baikunthpur, Koriya-497335, Chhattisgarh, India

ARTICLE INFO

Article history:
Received 31 March 2015
Received in revised form 8 October 2015
Accepted 11 October 2015
Available online 22 October 2015

Keywords:
Generic stressor
Photomorphogenesis
Specific regulator
UVR8
UVR8 signalling
UV-B radiation

ABSTRACT

Although UV-B accounts for <0.5% of total sunlight energy reaching the earth's surface, however, it has multifaceted impact on plants as well as animals. High energy UV-B radiation is reported to have damaging impact on plant growth and productivity. After discovery of UV RESISTANCE LOCUS8 (UVR8), perceptions of biological impact of UV-B radiation on plants, have changed dramatically in last few years. This review focuses on the changing concept about the role of UV-B from a generic stressor to a specific regulator in plant science and has tried compiling the historical aspects of UVR8 starting with discovery, localisation and regulatory role played by UVR8 and also its interaction with other regulators.

Contents

1. Introduction

UVR has been divided into three different bands *viz.* UV-A (320-400 nm), UV-B (280-320 nm) and UV-C (100-280 nm). Most of the UV-B and UV-C are absorbed by the stratospheric ozone layer therefore,

* Corresponding authors. (Mobile): +91 9451373143.
E-mail addresses: vijaypratap.au@gmail.com (V.P. Singh), profsmprasad@gmail.com (S.M. Prasad).

http://dx.doi.org/10.1016/j.jphotobiol.2015.10.004
1011-1344/

only UV-A and a little UV-B reaches on the earth's surface. Although UV-B accounts for <0.5% of total sunlight energy reaching the earth's surface, still it has the highest energy of daylight spectrum, thus having high potential of damaging important biomolecules. These damages induced by UV-B are becoming more alarming due to depletion of ozone layer, which is a consequence of human activities. Stratospheric ozone is the shielding of earth surface for preventing from the harmful rays of sun, but due to release of chemicals like chlorofluorocarbons (CFCs) and also due to several free radicals like hydroxyl radical ($OH^{\bullet}$), nitric oxide radical ($NO^{\bullet}$), atomic chlorine ion ($Cl^{\bullet}$) and atomic bromine ion ($Br^{\bullet}$) ozone layer is getting depleted. Although all these ozone depleting substances (ODs) originate due to natural as well man-made activities but the contribution by human activities has increased their quantity in atmosphere. Though ozone layer is assuming its level gradually but due to the stability of these ODs for longer time in atmosphere and low reactivity they are continuously depleting the ozone layer. For instance, chlorine atom reacts with ozone molecule and dissociates ozone molecule and then reacts with one oxygen molecule and forms chlorine monoxide (ClO), which further reacts with another molecule of ozone and continues to destroy this layer and thus increased UV-B radiation reaches at earth's surface. The UV-B radiation has a deep tendency to cause multifaceted negative effects on morphology, physiology and biochemical processes of crops [1–3] either through direct damage or *via* various regulatory effects [4,5]. The negative effect of UV-B leads to changes in plant growth and productivity at the cellular level where general and specific as well as direct and indirect effects are found. Besides the direct effect of UV-B i.e. DNA injury, membrane changes and protein denaturation, UV-B also triggers oxidative stress by the formation of reactive oxygen species (ROS), thus resulting into increased oxygen toxicity in plants [6–8] which in turn, causes enhanced lipid and protein oxidations [9].

However, due to advancement in the field of UV-B studies and manipulation of UV-B doses in laboratory conditions, there was a renaissance of interest towards the environmental role of UV-B and emphasis shifted from damage and stress towards information and specific regulation. Wellmann [10,11] demonstrated that low doses of UV-B initiates photomorphogenic responses which includes, the suppression of hypocotyl extension and root growth, promotion of cotyledon opening [10–18] and biosynthesis of flavonoids in low UV-B fluence range [19–23]. Works of these authors clearly indicate that UV-B is perceived as a signal by the plants for some photomorphogenetic responses and also about the existence of UV-B photoreceptor but nature and functioning of this photoreceptor were not characterized.

The perception about the biological impact of UV-B radiation changed dramatically [24,25] with the finding of photomorphogenic responses that could not be explained by the action of known photoreceptors and were not a consequence of DNA damages. Kliebenstein et al. [26] isolated UV RESISTANCE LOCUS8 (UVR8) in *Arabidopsis thaliana* that was subsequently shown to act as a UV-B photoreceptor [27]. This review focuses on the changing concept about role of UV-B from a generic stressor to a specific regulator in plant science and also deals with several key players of UVR8 signalling.

2. UV-B as a Generic Stressor

Photosynthetic organisms need sunlight and are inevitably exposed to UV-B radiation which is an important component of the environment with the potential to alter plant growth and photosynthesis [28]. Even a small increase in incident UV-B radiation has significant biological effect because it is readily absorbed by number of important macromolecules such as nucleic acids, proteins and lipids [29,30]. Studies have demonstrated the extent of damage caused by ambient UV-B [31,32] and enhanced UV-B radiation [33–35] on morphological, physiological, biochemical and molecular components of crop plants (Table 1). The morphological effects caused by UV-B include many changes such as the reduction of plant height, leaf length and leaf area [35], increased auxiliary branching [36,37], leaf bronze, glazing, chlorosis, and necrotic spots [34]. Overall, enhanced UV-B radiation reduces main stem and rate of branch elongation resulting into more compact and shorter plants [34,35]. Decrease in the plant height is mainly due to shorter internodes rather than fewer nodes [38]. Similar to plant height, leaf area is also a sensitive growth parameter that responds to elevated UV-B radiation. Under enhanced UV-B radiation, decrease in leaf area has been reported [38,39] to serve as a protective mechanism. In addition, other morphogenic changes include delayed seedling emergence and flowering, and fruit ripening [40,41]. It has also been postulated that some UV-B-driven morphogenic responses are consequences of UV-B-induced changes in hormone metabolism and cell wall loosening [30,42]. Dicotyledonous species found to be more sensitive than monocotyledonous species to UV-B radiation [32,43]. Reduction in biomass accumulation due to UV-B exposure found in several plant species [34,44] ultimately results into reduction in crop yield [33].

Along with morphological changes, UV-B also affects patterns of root and rhizome development. Kumari et al. [45] found that root length was decreased under supplemental UV-B exposure in *Acorus calamus*. Several species and their varieties such as corn [46–48], rice [49], barley [50], wheat [51] and soybean [52–54] have been used to assess the detrimental effects of UV-B. The physiological effects caused by UV-B include reduction in photosynthetic activity mainly related to the degradation of photosystem II (PS II) proteins [55], destruction of pigments (chlorophylls and carotenoids) and reduced rubisco activity [56], and effects on stomatal functions [57]. The effects of UV-B include flavonoids accumulation in the epidermis which act as a shield to protect plant from UV-B radiation [58,59]. UV-B damages the biomolecules by generating ROS which can cause oxidations of lipids and proteins, and DNA damage [8,26,59]. With context to damage caused by UV-B, DNA is very sensitive as UV-B absorption causes phototransformation resulting in production of dimers *viz.* cyclobutane pyrimidine dimers (CPDs) and pyrimidine (6-4) pyrimidinone dimers (6-4 PPs) and due to inefficiency of polymerases to read through these photoproducts, affects the process of replication and transcription and ultimately the survival of organisms [60]. To lessen the impact of ROS generated, plants have evolved several strategies for avoiding as well as repairing the damages caused by UV-B exposure. The protection mechanisms at cellular level include accumulation of surface waxes, hairs and also production and accumulation of phenolic compounds that further reduce penetration of UV-B [19,24]. However, at biochemical level DNA damage is ameliorated by antioxidants such as ascorbic acid and alpha tocopherol [8,26] and by ROS scavenging enzymes such as superoxide dismutase, ascorbate peroxidase, glutathione reductase and guaiacol peroxidase [8,31,58]. While, protective responses originating at cytological level deploy a set of repairing complex that includes photoreactivation, excision and recombination repair. Photoreactivation involves monomerisation of dimers using UV-A/blue light, where the photolyase breaks the bonds between cyclobutane rings using the light energy, thus restoring the bases integrity. While excision repairing takes place by endonucleolytic cleavage, thereby releasing the nucleotides damaged by UV-B and then strand is resynthesized [61]. Moreover, repairments by homologous recombination are also initiated under UV-B irradiation [62]. These protective responses either at cellular or biochemical level were found to be stimulated under UV-B exposure only. With the advances in manipulation of UV-B radiation as low fluence rate [63–65], it was discovered that rather than damaging DNA they were evoking protective responses which were photomorphogenic and a loophole was there in understanding these responses as neither phytochrome nor cryptochrome could absorb UV-B and thus explaining these responses with known photoreceptors was not possible [10,11]. This signifies towards involvement of DNA molecule itself having UV-B receptor. Responses in plants were related with UV-B absorption by DNA as they were stimulated at wavelength ranging between 250 and 280 nm, while the action spectra for UV-B responses were found to be stimulated between 290 and 310 nm and wavelength below 290 nm inhibited these responses [66–68].