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*Review Article*

# Alterations in Photosynthetic Processes Due to the Effect of Oxidative Stress Induced by Cold and UV-B Radiation

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## Abstract

Plants of tropical and subtropical origin are damaged by temperatures below 15°C; these conditions produce cold stress in plants. The degradation of the ozone layer leads to a greater penetration of ultraviolet-B (UV-B) radiation to the planet's surface, especially in tropical and subtropical latitudes, which interferes with plant development. Cold and UV-B radiation can inhibit photosynthesis by altering gene expression and damaging photosynthetic components, either directly or through the production of reactive oxygen species (ROS). The photosynthetic components that are most affected are photosystems I (PSI) and II (PSII), light-harvesting complex II (LHCII) and the membranes. Through evolutionary processes, plants have developed different defense mechanisms against these types of stress, such as the synthesis of carotenoids and flavonoids, to protect themselves against the incidence of UV-B radiation and the ROS caused by these stressors. Therefore, this review is focused on analyzing the evidence that describes the effects of cold and UV-B radiation on the main components of the photosynthetic apparatus directly and through the production of ROS induced by photoinhibition as well as the mechanisms that plants use to eradicate the excessive production of ROS. 

**Keywords:** Photosystems; Defense system; Carotenoids; Flavonoids; ROS

## Introduction

In 1987, the Montreal Protocol was established to reduce the emissions of substances that deplete the ozone layer; despite this protocol, the degradation has not been reversed (IPCC, 2014; Abbasi and Abbasi, 2017), and deterioration continues to increase by 0.6% per year (Prado *et al*., 2012). In general, a 1% reduction of the ozone layer causes a 1.3 to 1.8% increase in ultraviolet-B (UV-B) radiation reaching the earth's surface (Hollósy, 2002). The levels of UV-B radiation at tropical latitudes are higher than in temperate regions due to the thinner ozone layer in the equatorial region, affecting the organisms that inhabit that region to a greater extent (Jaakola and Hohtola, 2010; Prado *et al*., 2012).

Exposure of plants to UV-B radiation induce a great number of variations in their metabolism, morphology, physiology and molecular levels (Mandi, 2016a; Jenkins, 2017). In fact, UV-B radiation can inhibit the process of photosynthesis directly, causing alteration in gene expression and damage to the photosynthetic machinery, mainly to photosystems I and II (PSI and PSII, respectively) as well as to light-harvesting complex II (LHCII). It can also affect photosynthesis indirectly through the generation of reactive oxygen species (ROS) (Smith *et al*., 2009; Mandi, 2016b). Tropical and subtropical plants are also damaged by temperatures below 15°C (Knight and Knight, 2012). At the cellular level the greatest damage caused by cold occurs in chloroplasts, since increasing the temperature again increases the activity of galactolipases (enzymes that release fatty acids) and lipoxygenases (LOX, catalysts of the hydroperoxidation of free fatty acids), which occurs mainly in the presence of light due to the production of ROS, all of which ultimately causes oxidation of the membranes (Kaniuga, 2008). In response to cold and UV-B radiation plants exhibit decreased leaf area, reduced stem elongation, increased leaf thickness, altered stomatal density, increased antioxidant enzyme activity and secondary metabolite synthesis, among other effects (Prado *et al*., 2012).

Synthesis of secondary metabolites of the phenylpropanoid pathway has been reported as a defense mechanism to counteract the deleterious effects of cold and UV-B radiation on plants; these compounds include phenolic acids, insoluble polyphenols and flavonoids (Theocharis *et al*., 2012; Zlatev *et al*., 2012). Flavonoids are found mainly in the epidermis of plant tissues, so they have been associated with the property of absorbing UV-B radiation as their main activity (Barnes *et al*., 2016; Mandi, 2016a). However, they

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also have the capacity to counteract the deleterious effects caused by ROS due to their antioxidant potential (Castañeda-Ovando *et al*., 2009). The objective of this review is to document the most recent advances in the literature regarding the effects of cold and UV-B radiation on the photosynthesis process, the mechanisms of ROS production during the photoinhibition process and the involvement of defense mechanisms to reverse the effects of stress.

**Morphological Changes in Plants Due to the Effects of Cold and UV-B Radiation**

Cold and UV-B radiation cause morphological changes in leaf thickness and area, increased axillary branching, cuticle wax production, reduced stem elongation and reduced dry matter content (Kumari *et al*., 2015; Jenkins, 2017). During cold stress, an increase in stem and leaf flaccidity in pepper plants has been demonstrated (Airaki *et al*., 2012). In addition, the growth of the leaves is due to an increase in parenchymal cells and an increase in intercellular spaces (Wang *et al*., 2014). Furthermore, if plants are exposed to temperatures outside optimal growth ranges, the negative effects of UV-B radiation increase (Singh *et al*., 2014b; Martel and Qaderi, 2016). Physiological changes depend on the plant species exposed to stress. For example, Kakani *et al*. (2003) reported that out of 40 studies involving 23 plant species exposed to UV-B radiation, only 54% had a biomass reduction, 35% were not affected and 5% exhibited an increase in biomass, suggesting that factors such as genotype, temperature and the intensity of the radiation are relevant in the response. Kataria *et al.* (2013) showed that dicotyledonous plants are more susceptible to UV-B radiation than monocotyledonous plants, which may be related to their morphological differences, since dicotyledons arrange their leaves more horizontally, are more exposed to radiation and are therefore more susceptible to damage to their photosynthetic components, particularly PSII.

**Effects of Cold and UV-B Radiation on the Activity of Photosystems I and II (PSI and PSII)**

**Photosystem II:** PSII is the first component of the luminous phase of photosynthesis. It consists of a complex of proteins and pigments that is responsible for taking the energy of the photons of solar radiation and transforming it into chemical energy (Nishiyama and Murata, 2014). The PSII antenna complex consists of the major chlorophyll complex, called LHCII; the minor complex consisting of the proteins CP29, CP26 and CP24; and the central protein complexes CP43 and CP47 (Pospíšil and Prasad, 2014). The central part of PSII consists of a pair of proteins called D1 and D2, which contain linked chlorophyll and carotenoid pigments as well as cofactors that are responsible for the flow of electrons from water molecules. This protein dimer consists of a manganese complex in which water oxidation occurs, the $P680^+$ reaction center, as well as the primary electron acceptor quinone $Q_A$ and the secondary acceptor quinone $Q_B$, which are bound to the D2 and D1 proteins, respectively (Rodrigues *et al*., 2006). All the above components are sensitive to UV-B radiation (Dobrikova *et al*., 2013; Kataria *et al*., 2014). Studies of the effects of UV-B radiation have mainly focused on PSII because the D1 and D2 proteins are very sensitive due to chemical transformations in amino acids with double bonds (Table 1) (Zlatev *et al*., 2012). Amino acids capable of absorbing UV-B radiation are those of the aromatic type, such as phenylalanine, tryptophan and tyrosine, as well as cysteine, cystine and histidine (Hollósy, 2002; Mandi, 2016b). The amino acid histidine is found in protein D1, which contains bound chlorophylls that, during conformational changes by the UV-B radiation effects are released to facilitate their photo-oxidation (Mahdavian *et al*., 2008).

Protein D1 is the component of PSII primarily affected by radiation, so plants have developed a complex system of continuous repair for the replacement of damaged D1 proteins, resulting in a process of intense regulation between degradation and D1, synthesis (Nishiyama and Murata, 2014). The repair process begins with the phosphorylation of the PSII containing the inactive D1, which promotes the transport of the PSII from the covered thylakoid membrane to the part of the membrane exposed to the stroma, where the complex is dephosphorylated. Then begins the action of two protease families called Deg and FtsH (Nath *et al*., 2013). Deg proteins are endopeptidases of which the DegP 2, isoform is found in the stromal part of the thylakoid membrane associated with D1 dissociation in two 23- and 10-kDa polypeptides (Haußühl *et al*., 2001). Deg 1 is bound to the part of the thylakoid membrane of the lumen that produces two polypeptides from D1 corresponding to 16 and 5.2 kDa (Kapri-Pardes *et al*., 2007). However, in mutants of *Arabidopsis thaliana* lacking DegP 2 the same phenotype and D1 change is obtained as in normal plants (Huesgen *et al*., 2006). So, it can be assumed that the activity of these proteases does not dependent on both to carry out the complete D1 degradation process. On the other hand, the participation of FtsH proteins, also called zinc metalloproteases, has been demonstrated in the degradation of the segments after the action of the endopeptidases (Lindahl *et al*., 2000). This process is essential for the replacement of D1 proteins and thereby maintenance of PSII (Bailey *et al*., 2002). In fact, the accumulation of FtsH family has been observed as a recovery system in plants exposed to UV-B radiation (Pascual *et al.*, 2017). Once the inactive D1 is degraded, the assembly of the new D1 pre-protein proceeds; which undergoes some post-translational modifications, and the restored PSII is subsequently returned to the grana (Nath *et al*., 2013). Finally, Mlinarić *et al.* (2017) reported that the completely PSII recovery is totally restored to the initial values during the night.

Cold and hydrogen peroxide ($H_2O_2$) produced during stress do not have a direct effect on PSII damage; however,

**Table 1:** Effects of UV-B radiation and cold stress in photosystems

| Photosystem | Effects of UV-B radiation | Effects of cold | Reference(s) |
|---|---|---|---|
| PSII | Damages proteins D1 and D2 through conformational changes in aromatic amino acids | Cold has no a direct effect on proteins D1 and D2 | Hollósy, 2002; Allakhverdiev and Murata, 2004; Zlatev *et al*., 2012; Mandi, 2016b |
| | Conformational changes produced in proteins D1 and D2 release chlorophylls increasing their photo-oxidation | Cellular activity decreases, so the solar radiation becomes excessive producing photo-oxidation of chlorophyll | Rodrigues *et al*., 2006; Mahdavian *et al*., 2008; Sonoike, 2011; Zlatev *et al*., 2012 |
| | Induces the accumulation of proteases which participate in D1 repair system | The protein synthesis is inhibited reducing the D1 repair system | Allakhverdiev and Murata, 2004; Nishiyama *et al*., 2011; Pascual *et al.*, 2017 |
| PSI | Increases ROS production which can form hydroperoxides from free fatty acids released during cold stress | Induces the activation of galactolipases increasing free fatty acids and therefore, the production of hydroperoxides which damage PSI because is more exposed to stroma than PSII | Zhang and Scheller, 2004; Kaniuga, 2008; Mandi, 2016b |
| | Reduces the RuBisCO activity which promote PSI photoinhibition by ROS produced | Reduces the RuBisCO activity which promote PSI photoinhibition by ROS produced | Lidon and Ramalho, 2011; Fu *et al*., 2016; Pascual *et al.*, 2017 |

these two factors lead to a reduction in the D1 repair system, thereby reducing PSII activity (Allakhverdiev and Murata, 2004). The main point of inhibition of the D1 repair system by ROS is an elongation factor during the translation process, thus also affecting the production of other proteins (Nishiyama *et al*., 2011). The mechanism consists of the formation of a disulfide bridge due to the oxidation of two cysteine residues, Cys 105 and Cys 242, caused by ROS. These residues are conserved in several plants as well as in prokaryotic organisms (Kojima *et al*., 2009). Therefore, the reduction in PSII activity due to cold and UV-B radiation is mainly attributed to damage caused by UV-B radiation directly in D1, whereas the excessive production of ROS as a consequence of exposure to both stress factors leads to a reduction in the PSII repair system, which leads to a prolonged recovery time for the plant.

**Photosystem I:** Most studies mention that PSI is less sensitive than PSII to damage by UV-B radiation (Kataria *et al*., 2014). However, cold, stress affects PSI more than PSII (Zhang *et al*., 2014). During exposure to cold cellular metabolic activity decreases, so that the solar radiation becomes excessive for the plant, and PSII is damaged (Table 1); however, as mentioned above, PSII has a repair system, and when the plant is again exposed to higher temperatures, PSII recovers. In contrast, PSI does not have such an efficient repair system. As a result, the electrons from PSII to PSI are not used properly, thus promoting the production of ROS and consequently damaging PSI (Sonoike, 2011). Another process that occurs during cold stress is the activation of galactolipases, which have the function of releasing fatty acids from the membranes. Most fatty acids in chloroplasts are polyunsaturated, making them more susceptible to oxidation by ROS and LOX. Once the temperature increases, LOX activity increases by producing a high amount of fatty acid hydroperoxides that are harmful to membranes and proteins (Kaniuga, 2008). This process coincides with the protein reduction that occurs in PSI during the time after increasing the temperature after cold stress (Zhang and Scheller, 2004). Therefore, the damage to PSI is related to the production of hydroperoxides from the damaged membranes because this photosystem is found mainly in stromal membranes and is therefore more exposed to this type of damage than PSII, which is found mostly in the inner chloroplast membrane.

Cold and UV-B radiation have a negative effect on the Calvin cycle, mainly affecting the activity of the ribulose-1,5-bisphosphate carboxylase (RuBisCO) enzyme, so the NADH produced in PSI is not used, maintaining the P700, reaction center in a reduced state (Lidon and Ramalho, 2011; Fu *et al*., 2016; Pascual *et al.*, 2017). This process causes the electrons coming from PSII to promote the production of ROS in PSI and, thus its photoinhibition. By blocking the activity of P700 the activity of the electron transport chain is reduced, which induces the reduction of the plastoquinones that causes photoinhibition of PSII. The inactivation of PSI decreases the production of ATP, which is necessary for the production of D1 in PSII repair. Therefore, inactivation of PSI negatively affects PSII. In contrast, inactivation of PSII does not negatively affect PSI activity (Sonoike, 2011). In fact, Zhang *et al*. (2016) suggest that the reduction of PSII repair or its damage produced by UV-B would be necessary when PSI is damaged by cold stress, because this reduces the production of ROS in PSI and consequently, allow PSI recovery. However, UV-B radiation is a higher inductor of ROS than cold and other stress factors (Wu *et al.*, 2016) therefore, is necessary to search better ways to improve the PSI recovery.

## Oxidative Stress and Photoinhibition of Photosynthesis

Because they need oxygen to survive, aerobic organisms are exposed to the negative effects of ROS. In the case of plants, the maximum production of these compounds occurs in chloroplasts (Hasanuzzaman *et al*., 2011). ROS can be classified into two groups: molecular or non-radical ROS, which are $H_2O_2$ and singlet oxygen ($^1O_2$) and free radicals, such as the superoxide radical ($O_2^{\bullet-}$), alkoxyl radicals (RO•) and peroxyls (ROO•), the perhydroxyl radical ($HO_2$•) and hydroxyl radical (OH•) (Gill and Tuteja, 2010). The last is the

radical with the greatest oxidation capacity in plants (Perl-Treves and Perl, 2002). Both cold and UV-B radiation promote a reduction in the activity of the Calvin cycle and thus a lower need for radiation in the photosynthesis process, resulting in an excess of incident radiation in the photosystems, promoting the formation of $^1O_2$, and $O_2^{\bullet-}$, which alter the redox state and, therefore, change the chloroplast metabolism (Nishiyama and Murata, 2014; Chan *et al*., 2016). PSII is the highest producer of $^1O_2$, and PSI the greatest producer of $O_2^{\bullet-}$ (Kataria *et al*., 2014).

During the exposure of chloroplasts to the energy of solar radiation, the chlorophyll molecules go from being in a basal state to a state of higher energy ($^1$Chl*). The energy is transferred between these molecules until reaching reaction center P680 (in the case of PSII) to continue with the electron transport chain. However, in the case of excess radiation, the chlorophyll remains in an excited state for a prolonged time, whereby the formation of chlorophyll triplets in the excited state ($^3$Chl*) occurs. When $^3$Chl* is formed, the energy of this molecule can be transmitted to an oxygen molecule and produces $^1O_2$ (Pospíšil and Prasad, 2014). During exposure to UV-B radiation, the D1 protein is damaged, so that the part that blocks the passage of the oxygen molecules produced during the oxidation of water allows access to the P680 reaction center (Nishiyama *et al*., 2011). During exposure to solar radiation, reaction center P680 also enters an excited state that subsequently also forms an excited triplet ($^3$P680*) and transmits its energy to an oxygen molecule, thereby forming $^1O_2$ (Gill and Tuteja, 2010; Pospíšil and Prasad, 2014).

On the other hand, in PSI the production of NADPH is accomplished by the reduction of $NADP^+$ and the use of the electrons coming from PSII. When the Calvin cycle undergoes alterations due to cold and/or UV-B radiation, the consumption of NADPH decreases, and $O_2^{\bullet-}$ is formed in the membrane. This radical is spontaneously transformed into $H_2O_2$ by the effect of a low pH or by the action of the superoxide dismutase (SOD) enzyme (Perl-Treves and Perl, 2002). Subsequently, $H_2O_2$ can be reduced to two molecules of water by the action of ascorbate peroxidase (APX) using ascorbic acid as a substrate (Asada, 2006). However, $O_2^{\bullet-}$ can also be protonated to form the radical $HO_2\bullet$, or, through the presence of metals such as copper and iron, the OH• radical can be formed by Haber-Weiss or Fenton reactions (Gill and Tuteja, 2010). It has also been demonstrated that UV-B radiation impinges on $H_2O_2$ molecules, causing OH• formation (Czégény *et al*., 2014). Finally, OH• participates in the oxidation of proteins and lipids in the membranes, the result of which is chain reactions that lead to the formation of RO• and ROO• radicals.

## Plant Defense Systems against Oxidative Stress Produced by Cold and UV-B Radiation

Due to the excessive production of ROS caused by cold and UV-B radiation, plants have a number of mechanisms, to prevent and combat oxidative stress (Zlatev *et al*., 2012). According to the location of the defense mechanisms with respect to the main production sites of ROS in chloroplasts, these organelles have several lines of defense against ROS: first, their content of carotenoids and tocopherols (Pospíšil and Prasad, 2014); second, antioxidant enzymes; and third, phenolic compounds, such as flavonoids.

### Carotenoids and Tocopherols

Carotenoids are part of the structure of membranes as well as of photosynthetic complexes, where they have the functions of radiation collectors as well as protectors against ROS (Domonkos *et al*., 2013). β-Carotene, the most abundant carotene in chloroplasts, is mainly bound to the reaction centers of PSI and PSII (Esteban *et al*., 2015) and the xanthophylls are mainly present in the antenna complexes (Ramel *et al*., 2012). In PSII, most of the β-carotene is in contact with the chlorophyll molecules, thus decreasing the probability of forming $^3$Chl*, whereas close to reaction center P680, β-carotene is responsible for containing the $^1O_2$ production (Cazzaniga *et al*., 2012). When β-carotene comes in contact with $^3$Chl* or with $^1O_2$, it acquires the energy of these molecules, releasing them to their basal state, and β-carotene then releases that energy in the form of heat (Fig. 1) (Pospíšil and Prasad, 2014). The products obtained from the exposure of β-carotene to ROS (β-apo-14'-carotenal, β-apo-10'-carotenal, etc) are more susceptible to oxidation (Ramel *et al*., 2012), which leads to the rapid oxidation of these compounds during stress due to UV-B radiation (Mahdavian *et al*., 2008).

It has been reported that the highest amount of $^1O_2$ is produced in PSII (Kataria *et al*., 2014); however, Cazzaniga *et al*. (2012) found that the PSI light-harvesting complex (LHCI) produces twice as much $^1O_2$ during stress at 8°C and high irradiance in Arabidopsis mutants lacking β-carotene production. This outcome explains the higher content of β-carotene in PSI of normal plants, which decreases the production of $^1O_2$ and therefore the difficulty detecting $^1O_2$ in this complex.

According to the location of β-carotene in the photosystems and their oxidation products (Ramel *et al*., 2012), this compound is mainly directed to the neutralization of $^1O_2$. In contrast, due to the increased presence of xanthophylls in light-harvesting complexes (LHCs), these compounds are intended to reduce the production of $^3$Chl* (Pospíšil and Prasad, 2014). This was verified by Dall'Osto *et al*. (2013) in Arabidopsis mutants unable to produce xanthophylls but not α- and β-carotene, in which they observed damage in the LHC. In the same work, a significant reduction in PSI activity was described because of a deficiency in the ratio of Psa A/B subunits. This deficiency is attributed to the fact that xanthophylls may play the role of mediators in the interactions between the PSI subunits or with the chaperones that direct these proteins. Alternatively, xanthophylls also have a role in

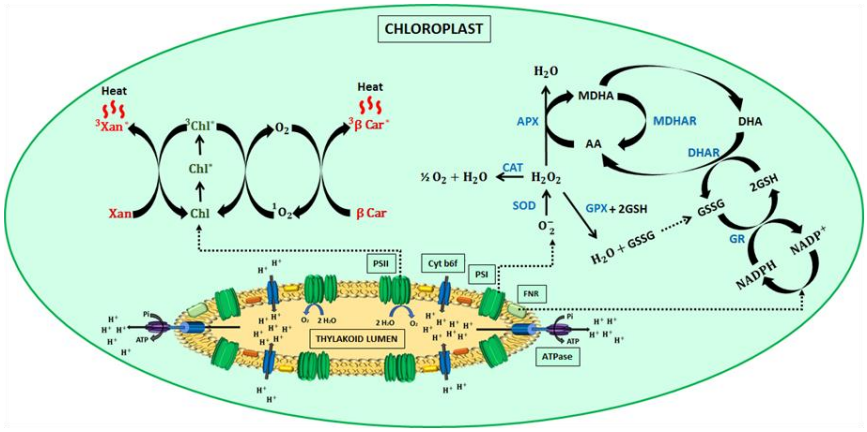


**Fig. 1:** Mechanism to quench triplet excited chlorophylls ($^3$Chl*) and ROS in the chloroplast. The exceding energy from chlorophyll prodduce $^3$Chl*, this energy is transferred to oxygen molecules to form the singlet oxygen $^1O_2$. The energy of this molecules, $^3$Chl* and $^1O_2$, is transferred to xanthophylls (Xan) and β-carotene (β-Car), respectively and is released as heat. The superoxide radical ($O_2^{\bullet-}$) produced in chloroplast is reduced throw the system of reactions of the antioxidant enzymes. PSII, photosystem II; PSI, photosystem I; Cyt b6f, cytochrome complex b6f; FNR, ferredoxin-NADP reductase; ATPase, ATP synthase; $^3$Xan*, triplet exited xanthophyll; $^3$β-Car*, triple excited β-carotene; SOD, superoxide dismutase; $H_2O_2$, hydrogen peroxide, CAT, catalase; APX, ascorbate peroxidase; AA, ascorbic acid; MDHA, monodehydroascorbate; MDHAR, MDHA reductase; DHA, dehydroascorbate; DHAR, DHA reductase; GPX, glutathione peroxidase; GSH, reduced glutathione; GSSG, oxidized glutathione; GR, glutathione reductase

the post-translational events of these proteins. However, it has also been shown that ROS induce changes in a elongation factor, which causes a decrease in protein production. In addition, the proteins of the photosystems are susceptible to attack from these compounds (Nishiyama *et al*., 2011) and because the lack of xanthophylls promotes a higher production of ROS, the reduction of PSI may be a consequence of the processes produced by these compounds.

When the carotenoid concentration is insufficient to decrease the production of $^1O_2$, this compound causes oxidation of proteins and thylakoid membranes. In the case of membranes, ROO• radicals are produced that can be neutralized by tocopherol molecules producing tocopherol radicals that can be reduced by another molecule, of tocopherol or react with another ROO• to form the molecule tocopherolquinol (Pospíšil and Prasad, 2014). During cold stress and high radiation levels α-tocopherol, has been shown to reduce membrane lipid damage and has the ability to reduce oxidation of chlorophyll molecules and damage to PSII, assuming that there are α-tocopherol molecules very close to PSII that could be protecting β-carotene and D1 through the neutralization of $^1O_2$ (Havaux *et al*., 2005). It has recently been proposed that α-tocopherol due to its ability to neutralize ROS, prevents these compounds from affecting the PSII repair system (Murata *et al*., 2012). In addition, it has been determined that α-tocopherol and xanthophyll molecules participate in similar actions together, since mutants with α-tocopherol deficiencies exhibit increases in xanthophyll content and vice versa. However, mutants with deficiencies of both compounds present severe damage to membranes, chlorophylls and PSII (Havaux *et al*., 2005).

**Enzymatic Antioxidants**

The main components of the antioxidant enzymatic machinery of plant cells are superoxide dismutase (SOD), catalase (CAT), ascorbate peroxidase (APX), monodehydroascorbate reductase (MDHAR), dehydroascorbate reductase (DHAR), glutathione peroxidase (GPX) and glutathione reductase (GR) (Fig. 1) (Gill and Tuteja, 2010). SOD is the first enzyme to participate during the production of ROS and is responsible for transforming the radical $O_2^{\bullet-}$ into $H_2O_2$. This process is accomplished by the metal ion contained by SOD (Cu, Mn or Fe). The Fe-SOD and Cu/Zn-SOD isozymes are in the chloroplasts (Asada, 2006). However, $H_2O_2$ is also a molecule that causes damage to the cell so it presents a further degree of detoxification that is performed by the remaining enzymes together. First, $H_2O_2$ is reduced to water molecules by the CAT, GPX and/or APX enzymes (Gill and Tuteja, 2010). In the case of CAT no reduced substrate is required to perform the action, which results in water and oxygen as products (Perl-Treves and Perl, 2002). In contrast, APX and GPX require ascorbic acid (AA) and reduced glutathione (GSH) to obtain water and monodehydroascorbate (MDHA) and oxidized glutathione (GSSG) molecules, respectively. These molecules are transformed to their reduced state by the enzymes MDHAR and GR, respectively. In the case of GR activity, NADPH produced during photosynthesis (Gill and Tuteja, 2010) is used. In chloroplasts, which are the sites of greatest ROS

production, APX is the most prevalent $H_2O_2$-neutralizing antioxidant enzyme. Different chloroplast APX molecules have been classified as tAPX for the molecules located in thylakoids and sAPX for the molecules found in the stroma (Foyer and Noctor, 2009; Murata *et al*., 2012).

In the majority of cases, both cold stress and UV-B radiation increase the activity of antioxidant enzymes, with some exceptions (Hasanuzzaman *et al*., 2012), which may depend on the type of plant and even on its nutritional status (Singh *et al*., 2014a). It has been observed that an increase in SOD activity is greater in the chloroplasts than in the cytoplasm, as is the increase in APX due to the effects of UV-B radiation, whereby the antioxidant activity focuses on metabolizing $H_2O_2$ and thus $O_2^{\bullet-}$ (Majer *et al*., 2014), which is mostly produced in PSI (Kataria *et al*., 2014). However, when cold stress and/or UV-B radiation is very severe, CAT activity (as well as that of APX in chloroplasts) is inhibited and/or insufficient for the amount of $H_2O_2$ produced (Hasanuzzaman *et al*., 2012; Liu *et al*., 2012). A reduction in tocopherols and carotenoids can also be observed (Mahdavian *et al*., 2008; Zlatev *et al*., 2012), which increases the concentration of $H_2O_2$ because it is not neutralized. This outcome is relevant because $H_2O_2$ is liposoluble and is able to pass through the membranes to migrate to other cellular compartments and cause greater damage (Mubarakshina *et al*., 2010).

**Flavonoids**

Based on the flavonoid structure they can be classified in: chalcones, flavones, flavonols, dihydroflavonols, flavandiols, anthocyanins, proanthocyanidins, isoflavonoids and aurones (Zhang *et al*., 2017). Flavonoids are widely distributed in the plant kingdom and in almost all plant organs, though they predominate in the epidermis of the leaves, stems and fruit skin. Due to the absorption capacity of UV-B radiation by compounds such as kaempferol and anthocyanins, the role of photoprotectors is attributed to flavonoids (Croteau *et al.,* 2000). However, overproduction of flavonoids under the effects of cold and UV-B radiation has also been linked to its antioxidant ability to reverse oxidative stress on the cell (Mazid *et al*., 2011; Cruceriu *et al*., 2017; Zhang *et al.,* 2017). Flavonoids containing two hydroxyl groups on their B ring are mostly produced under conditions of exposure to UV-B or high levels of PAR (photosynthetically active radiation) radiation (Guidi *et al.,* 2016) and contain a greater ability to inhibit ROS than those molecules with a single hydroxyl group, which have a greater capacity to absorb UV-B radiation. In addition, dihydroxylated flavonoids have the ability to form complexes with metals such as Al, Cu and Fe, thus also reducing the risk of producing OH• radicals through Fenton reactions with $H_2O_2$ molecules produced during exposure to cold and UV-B radiation (Castañeda-Ovando *et al*., 2009; Agati *et al*., 2013). It has been reported that the content of cinnamic acids is replaced by a higher content of flavonoids, which have a lower UV-B radiation absorption capacity, during exposure to this type of radiation (Agati *et al*., 2013), therefore, the plant's defenses are mainly focused on reducing the amount of ROS produced by UV-B radiation rather than absorbing the radiation (Hideg *et al*., 2013).

Quercetin has the ability to neutralize $^1O_2$ in chloroplast membranes (Dobrikova and Apostolova, 2015) and its presence in the nucleus suggests a role in preventing DNA oxidation (Agati *et al*., 2012). However, after being synthesized at the periphery of the endoplasmic reticulum, most flavonoids are stored in the vacuoles. Under extreme stress conditions, APX activity is inhibited, so that up to 75% of the $H_2O_2$ produced in the chloroplasts can migrate into the cytoplasm (Mubarakshina *et al*., 2010), and $H_2O_2$ can then be transferred into vacuoles transported by TIP aquaporins, where it is neutralized by flavonoids, such as anthocyanins (Agati *et al*., 2012). The increase in the content of anthocyanins has been tied to the redox changes of the cell caused by ROS such as $H_2O_2$ that can participate as either an oxidant or a reductant in many reactions (Perl-Treves and Perl, 2002). There are more than 100 members of the MYB transcription factors reported in plants (Wu *et al.,* 2016). Some of this MYB transcription factors participate in the regulation of flavonoid biosynthesis, affecting the capacity to inhibit or increase the production of flavonoids according to their conformation, which is regulated by changes in their structure according to the redox state of the molecules (Fig. 2). Therefore, $H_2O_2$ can activate these transcription factors and induce the synthesis of flavonoids (Heine *et al*., 2004; Dubos *et al*., 2008; Queval and Foyer, 2012). Then, $H_2O_2$ act as a mobile secondary messenger of the chloroplast. In fact, has been mentioned that $H_2O_2$ produced in chloroplast induces different signals compared to the $H_2O_2$ produced in other organelles (Chan *et al*., 2016). Guo and Wang (2010) observed, over time, consecutive increases and decreases in anthocyanin accumulation and expression of the *SlPAL5* gene (phenylalanine ammonia-lyase) in different tissues of tomato plants exposed to UV-A radiation. Therefore, during the exposure of plants to cold stress and/or UV-B radiation, these metabolites may have the ability to regulate their biosynthesis and other metabolic pathways through the neutralization of ROS (Foyer and Shigeoka, 2011).

## Conclusion

The damages caused by cold and UV-B radiation are the result of the overproduction of ROS in the chloroplasts due to induced damage in the electron transport chain. The functionality of the photosystems is the first component attacked by ROS, especially the protein synthesis system, which is fundamental for the recovery of damaged parts of photosystems, such as protein D1 of PSII. However, the damages in PSI are more critical for the functional recovery of the photosynthetic apparatus. Increase

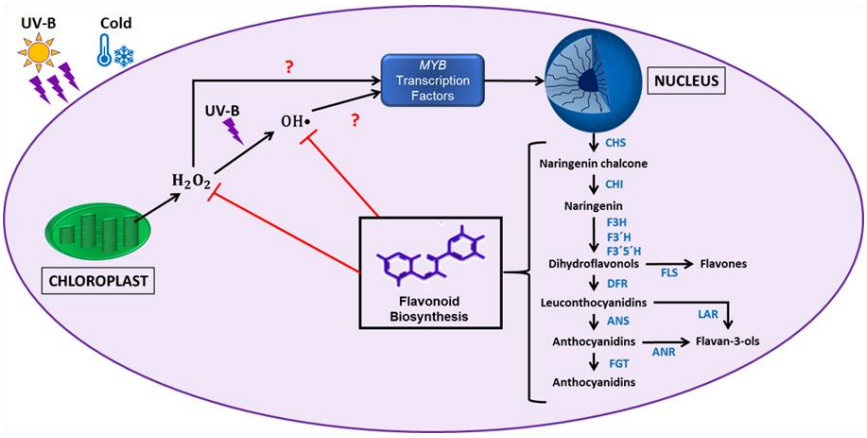


**Fig. 2:** Schematic model of the flavonoid biosynthesis regulation throw the ROS produced during the stress induced by cold and/or UV-B radiation. The excess of hydroxide peroxide ($H_2O_2$) produced during the stress is transformed to hydroxyl radical (OH•) by UV-B radiation, Fenton or Harber-Weiss reactions; this ROS can produce redox reactions in transcription factors MYB that active the gene transcription to produce enzymes of the flavonoid biosynthesis. Flavonoids have the capacity to neutralize the ROS produced during the stress and, therefore, regulate their own biosynthesis and other metabolic process affected by this ROS. CHS, chalcone synthase; CHI, chalcone isomerase; F3H, flavanone 3-hydroxylase; F3´H; flavonoid 3´-hydroxylase; F3´5´H, flavonoid 3´,5´-hydroxylase; FLS, flavonol synthase; DFR, dihidroflavonoid-4-reductase; LAR, leucocyanidin-4-reductase; ANS, anthocyanidin synthase; ANR, anthocyanidin reductase; FGT, flavonoid glycosyltransferase

in the production of certain antioxidant and flavonoid enzymes is induced by the increase in ROS, such as $H_2O_2$, due to the inability of the primary defense system to combat the increase caused by exposure to cold and UV-B radiation.

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