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Tanned or Sunburned: How Excessive Light Triggers Plant Cell Death

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ABSTRACT

Plants often encounter light intensities exceeding the capacity of photosynthesis (excessive light) mainly due to biotic and abiotic factors, which lower CO_2 fixation and reduce light energy sinks. Under excessive light, the photosynthetic electron transport chain generates damaging molecules, hence leading to photooxidative stress and eventually to cell death. In this review, we summarize the mechanisms linking the excessive absorption of light energy in chloroplasts to programmed cell death in plant leaves. We highlight the importance of reactive carbonyl species generated by lipid photooxidation, their detoxification, and the integrating role of the endoplasmic reticulum in the adoption of phototolerance or cell-death pathways. Finally, we invite the scientific community to standardize the conditions of excessive light treatments.

Key words: *Arabidopsis*, photosynthesis, excessive light, photooxidative stress, carotenoid, plant cell death, stress response

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INTRODUCTION

Plants often encounter light intensities that exceed the needs of photosynthesis (Ort, 2001). Light energy that is not used to assimilate CO_2 or dissipated can be considered to be in excess and leads to a depression of photosynthetic efficiency, especially at Photosystem II (PSII) (Kato et al., 2003; Murata et al., 2007). Excessive light occurs mainly in response to biotic and abiotic factors, such as drought, heat, or pests, and lowers the capacity of light energy sinks, first and foremost CO_2 fixation. The excess electrons and excitation energy can be transferred to molecular oxygen, generating biologically damaging molecules, such as reactive oxygen species (ROS), peroxides, and radicals, leading to photooxidative stress that can ultimately lead to cell death (Apel and Hirt, 2004; Li et al., 2009).

HIGH LIGHT UNDER DIFFERENT CONDITIONS INDUCES DIFFERENT RESPONSES

The real condition of excessive light, which is dependent on photosynthetic activity and can also occur at relatively low light intensities, is often confused with "high light" treatment. Drought stress, nutrient starvation, or pathogens can lead to the same state of overexcitation of the photosystems, without the imposition of high light (Garai and Tripathy, 2018; Wang et al., 2018; Pérez-Bueno et al., 2019). Likewise, playing with CO_2 and O_2 compositions of the gas phase Montané et al. (1998) generates the same excitation pressure on the photosystems at different photon flux densities (PFDs) (Montané et al., 1998). In addition, the intensity of sunlight that leaves receive under natural conditions often fluctuates because of clouds and intermittent shading by other leaves or plants. This "fluctuating light" can be very damaging to the photosynthetic machinery, particularly Photosystem I (PSI), even at relatively low PFDs (Kono et al., 2014; Yamori et al., 2016).

PFDs used in the literature to induce excessive light range from 500 to 2000 μmol photons m^{-2} s^{-1}. These treatments can also increase leaf temperature and, unless the air temperature is controlled, will impose a double condition of excessive light and heat stress. The lack of leaf-temperature control in experimental protocols increases interlaboratory data variability and makes comparisons between different studies more difficult.

Even when the leaf temperature is controlled by lowering the air temperature, different setups have been adopted, such as

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600 μmol photons m^{-2} s^{-1} PFD at 23°C air temperature (Balfagón et al., 2019), 1500 μmol photons m^{-2} s^{-1} at 7°C (Ramel et al., 2012; D'Alessandro et al., 2018), 1200 μmol photons m^{-2} s^{-1} at 18°C (Lv et al., 2015), 1200 μmol photons m^{-2} s^{-1} at 11.5°C (Huang et al., 2019), or 2000 μmol photons m^{-2} s^{-1} at 4°C (Mattila et al., 2019). These studies show that the leaf temperature remains at standard levels (20°C–22°C) when exposed to 1500 μmol photons m^{-2} s^{-1} at 7°C or 1200 μmol photons m^{-2} s^{-1} at 11.5°C. However, the leaf temperature increases by 4°C (from 23°C to 27°C) at 600 μmol photons m^{-2} s^{-1} or 13°C (from 22°C to 35°C) at 1200 μmol photons m^{-2} s^{-1} if the air temperature is not lowered.

Several studies have indicated that there can be significant overlap between high light and heat stress responses when the air temperature is maintained at normal growth temperature. Balfagón et al. (2019) compared a 7-h high light (HL) treatment with a heat-shock (HS) treatment (42°C) and the combination of both (Balfagón et al., 2019). The HL treatment caused a 4°C increase in leaf temperature (23°C to 27°C), the HS treatment increased leaf temperature by around 10°C (23°C to 33°C), and the combined stresses increased leaf temperature by around 10°C–12°C. The analysis of transcriptomes showed an astonishing resemblance between HL (at 23°C air temperature) and HS, with a large fraction of the modified genes being common between the two stresses, corresponding to 58% (4439) of HL alone. This result suggests that performing HL experiments at 23°C air temperature causes a response that partially overlaps with HS, likely due to the increase in the leaf temperature. While the combination of HS and HL was equally similar to HS and HL (64% overlap) on a transcriptional level, other experiments indicated a dominance of HS during HL stress. In fact, while HL performed at 23°C caused stomatal closure, HS and HL + HS induced stomatal opening. The relative water content was unperturbed by HL but reduced from 75% to 60% by HS and HS + HL. The number of starch granules diminished in HL but increased under HS and HS + HL. H_2O_2 production increased in HS and HS + HL-treated plants but not in HL-treated plants, in agreement with previous analyses of pure excess light (D'Alessandro et al., 2018). Similar results were observed in another study when the systemic response to HL, HS, and the combination of both stresses was analyzed (Zandalinas et al., 2020).

THE LIPID PROBLEM IN EXCESSIVE LIGHT

ROS production under stress conditions initially elicits a signaling cascade and, if sufficient quantities are produced to overwhelm defense mechanisms, also leads to cellular damage. Singlet oxygen ($^1O_2$) is very harmful and likely the major ROS produced under excessive light (Triantaphylidès et al., 2008; Ramel et al., 2012; D'Alessandro and Havaux, 2019; Khorobrykh et al., 2020). In fact, a $^1O_2$ mediated transcriptional response can be detected after a 24-h HL treatment at controlled air temperature, while marker genes for H_2O_2 are not induced (Shumbe et al., 2017). This result agrees with the increased biosynthesis of H_2O_2 and the induction of *APX2* only when high light conditions are combined with heat stress (Balfagón et al., 2019; Huang et al., 2019; Smirnoff and Arnaud, 2019). Notably, the O_2-evolving-complex (OEC) proteins are particularly susceptible to heat stress (Berry and Bjorkman, 1980; Weis and Berry, 1988; Havaux, 1993), and OEC-depleted PSII predominantly generates H_2O_2 (Khorobrykh et al., 2020). Only a prolonged high light stress is therefore likely to cause a combined accumulation of $^1O_2$ and H_2O_2 (Laloi and Havaux, 2015).

ROS, and especially $^1O_2$, are highly reactive toward carbon–carbon double bonds. Because ROS are primarily produced at the level of the photosystems in a lipid-rich environment, oxidized lipids are one of the major products of ROS generation in response to excessive light. Fatty acids are highly unsaturated in plant leaves (Douce and Joyard, 1980) and are therefore very sensitive to oxidation (Bour et al., 2019). The major fatty acids in leaves are the polyunsaturated fatty acids linolenic acid (C18:3) and linoleic acid (C18:2), which contain three and two double bonds, respectively. At the level of thylakoids, a primary site of ROS production, the ratio between unsaturated to saturated lipids is 9.4 to 1, and linolenic acid corresponds to 70% of total fatty acids (Chapman and Barber, 1986; Yalcinkaya et al., 2019). In *Arabidopsis* whole leaves, linolenic acid can represent up to 50% of the total fatty acid content (Li-Beisson et al., 2013). Consequently, lipid peroxidation is usually a primary event associated with ROS production and oxidative stress in plants (Birtic et al., 2011). The attack of lipids by ROS generates specific peroxides, notably lipid endoperoxides specific to $^1O_2$, while lipid radicals and lipid peroxyl radicals are specific to reduced oxygen forms (Mano et al., 2019b; Khorobrykh et al., 2020). In a second stage, all these molecules are transformed into lipid hydroperoxides.

High light treatment with controlled air temperature was found to be systematically associated with a lipid peroxidation signature typical of $^1O_2$ (Triantaphylidès et al., 2008), indicating a major role for this ROS in the photooxidative degradation of plant lipids. The abundance of polyunsaturated fatty acids has been proposed to act as a sink for ROS, especially $^1O_2$, in chloroplasts, which could constitute a protection mechanism against the propagation of oxidative stress (Mène-Saffrané et al., 2009). However, lipid peroxides are not only the signature of photooxidation but also directly contribute to signaling and toxicity.

Phytoprostanes and phytofurans are compounds derived from non-enzymatic oxidation of α-linolenic acid in plants (Mueller, 2004; Durand et al., 2011; Collado-González et al., 2016; Galano et al., 2017) (Figure 1). Because of their structural analogies with isoprostanes and prostanoids, these lipid derivatives may constitute a new class of bioactive plant compounds in response to oxidative stress (Mueller et al., 2008). Some phytoprostanes contain an electrophilic site, which can react and form adducts with proteins (Liebler, 2008). Typical reactions are Schiff base formation and Michael addition. The accumulation of phytoprostanes/phytofurans has been reported in various photosynthetic organisms under oxidative stress (Loeffler et al., 2005; Yonny et al., 2016; Lupette et al., 2018), potentially playing a role in plant responses to photooxidative stress.

Lipid hydroperoxides spontaneously fragment into a variety of secondary compounds that can exhibit high reactivity

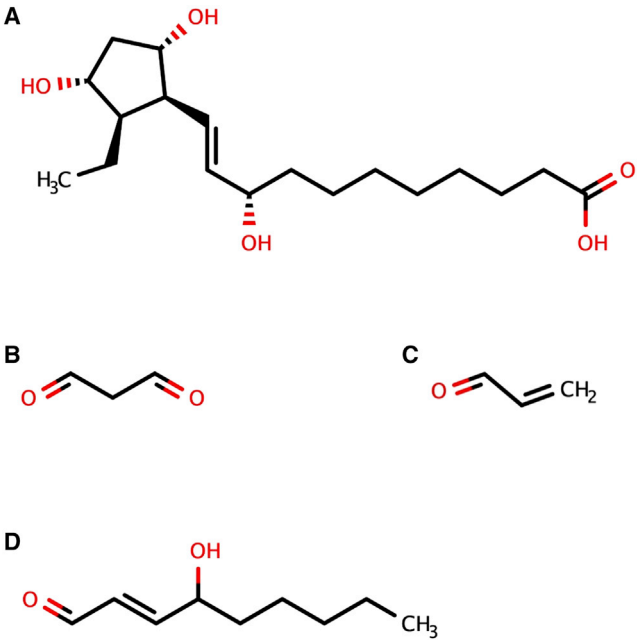

Figure 1. Examples of Secondary Products Generated by Lipid Peroxidation in Plant Leaves.
(A) 9-F1-Phytoprostane, **(B)** malondialdehyde (MDA), **(C)** acrolein, **(D)** 4-hydroxynonenal (HNE).

toward biomolecules. Reactive carbonyl species (RCS) are the α,β-unsaturated aldehydes and ketones derived from lipid peroxides, which can further hinder the physiological functioning of macromolecules and induce cell death (Farmer and Mueller, 2013; Mano et al., 2019b). Some of these molecules, such as (*E*)-2-hexenal, have been studied as green leaf volatiles and previously reviewed (Ameye et al., 2018). RCS, such as hydroxynonenal (HNE), acrolein, and malondialdehyde (Figure 1), are electrophiles, hence reacting with nucleophilic groups in biomolecules to establish covalent bonds (Mano, 2012; Mano et al., 2019b). In this way, RCS may alter the conformations or functions of proteins under stress conditions, consequently affecting metabolic or signaling processes (Biswas and Mano, 2016; Yalcinkaya et al., 2019).

PROMISCUOUS, BROAD-SPECTRUM DETOXIFICATION OF LIPID PEROXIDATION BY-PRODUCTS

Reactive molecules, both of endogenous or exogenous origin, may constitute a hazard for the cell and lead to cell death. Plants have evolved a detoxification mechanism that can neutralize these dangers. As this mechanism was discovered in studies that examined the response to herbicides, it was called the "xenobiotic response" (Sandermann, 1992; Kreuz et al., 1996; Riechers et al., 2010). This process is divided into three phases, namely, modification, conjugation, and compartmentalization, and enzyme families participating in each phase of the xenobiotic response are reported in Figure 2.

Enzymes of the modification phase are active toward RCS and catalyze their conversion into less dangerous molecules (Mano et al., 2019b). RCS detoxification can occur through different enzymatic reactions: (1) reduction of the α,β-unsaturated bond in an RCS molecule using NAD(P)H as the electron donor, which is catalyzed by alkenal reductase and alkenal/one oxidoreductase; (2) reduction of a carbonyl moiety to an alcohol using NAD(P)H, which is catalyzed by aldo-keto reductase; (3) oxidation of an aldehyde to a carboxylic acid using NAD^+ as the electron acceptor, which is catalyzed by aldehyde dehydrogenase; and (4) oxidation of an aldehyde using O_2 as the electron acceptor, which is catalyzed by aldehyde oxidase (Mano et al., 2002; Stiti et al., 2011; Yamauchi et al., 2011, 2012; Saito et al., 2013; Srivastava et al., 2017). In addition, the formation of a glutathione or glucoside adduct of RCS can be catalyzed by enzymes of the conjugation phase (i.e., glutathione *S*-transferase) (Mano et al., 2017, 2019a).

Several of these enzymes are induced under stress conditions, in agreement with the overproduction of RCS, and plants overexpressing alkenal reductases, aldo-keto reductases, and aldehyde dehydrogenases are usually more tolerant to stress conditions, as reviewed in Mano et al. (2019a, 2019b). In addition, we have recently reported a pathway controlling acclimation to excessive light mediated by the induction of RCS detoxification by the apocarotenoid β-cyclocitral (D'Alessandro et al., 2018; D'Alessandro and Havaux, 2019). Phytoprostanes and phytofurans may also require the induction of a detoxification response to limit their effects *in vivo*, and the rapid disappearance of phytoprostanoids in tobacco cells is consistent with this idea (Loeffler et al., 2005).

Enzymes of the modification phase are usually associated with biosynthetic pathways for secondary metabolites (e.g., cytochrome P450 oxidases, short-chain dehydrogenases/reductases). At the same time, some of these large families of enzymes were identified in the response to xenobiotic compounds (Fode et al., 2008). It is likely that plants can adopt the less-specific steps of the reactions that they perform in secondary metabolism to cope with hazardous molecules, in what could be considered a broad-spectrum (promiscuous) detoxification pathway. Ligand promiscuity and promiscuous detoxification are well-known processes in animal systems where the two promiscuous receptors CAR and PXR can bind to different molecules and control the expression of several CYP450 genes (Tolson and Wang, 2010). These processes, catalyzed by the same families of enzymes as those in the xenobiotic response of plants, are active against endogenous toxicants and constitute the base of drug metabolism and pharmacokinetics. We therefore propose to adopt the animal systems nomenclature of promiscuous detoxification for the xenobiotic response in plants.

As promiscuous detoxification is active toward hazardous metabolites generated during photooxidation, such as RCS, this pathway represents a new level of photoprotection. Light-induced ROS generation is inevitably coupled with photosynthesis. ROS quenching and scavenging are continuous, but under stress conditions they could become insufficient compared with ROS production, leading to ROS accumulation. At this point, the first level of photoprotection, including nonphotochemical energy quenching (NPQ), cyclic electron transport, plastid terminal oxidase, and state transition, reduces ROS

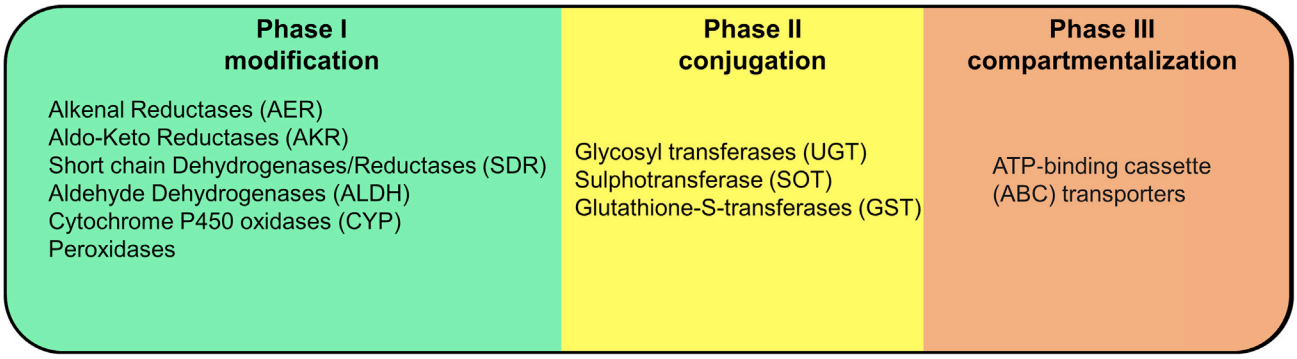

Figure 2. The Xenobiotic Detoxification Response Is Composed of Three Phases Involving Families of Detoxifying Enzymes.

production (Alric and Johnson, 2017; Roach and Krieger-Liszkay, 2019). The second level of photoprotection, including ROS scavenging (by superoxide dismutase and ascorbate peroxidase for the superoxide anion radical and H_2O_2, respectively) and ROS quenching (by carotenoids, tocopherols, and plastoquinones for $^1O_2$), avoids oxidation (Apel and Hirt, 2004; Krieger-Liszkay et al., 2008; Noctor et al., 2018; Filiz et al., 2019). The D1 protein of PSII, which is very susceptible to oxidation, can also be considered a component of photoprotection together with the well-described D1 repair mechanism (Nath et al., 2013; Järvi et al., 2015; Yoshioka-Nishimura, 2016; Kato et al., 2018). When these photoprotective processes are insufficient or inhibited by stress, the oxidation of lipids generates RCS. Promiscuous detoxification can metabolize the hazardous reactive molecules to protect the cell (Figure 3) which can trigger programmed cell death (PCD) under certain conditions (Biswas and Mano, 2016).

FROM RCS TO PCD

$^1O_2$-derived peroxidation of lipids, proteins, and nucleic acids has been observed in treatments with photosensitizers (methylene blue or rose Bengal) and in *Arabidopsis* mutant lines overproducing $^1O_2$ such as *flu* and *ch1* (Triantaphylidès et al., 2008; Shao et al., 2013; Laloi and Havaux, 2015). *ch1* shows increased lipid peroxidation and photooxidative damage under excessive light conditions, whereas $^1O_2$ is generated under non-photoinhibitory conditions in *flu* (Wagner et al., 2004; Ramel et al., 2013a). In both *ch1* and *flu*, $^1O_2$-induced cell death was dependent on signaling proteins, OXIDATIVE STRESS INDUCIBLE 1 (OXI1) (in *ch1* and in the wild type), EXECUTER 1 (EX1), or SAFE1 (in *flu*), indicating the involvement of a signaling process that leads to cell death (Shumbe et al., 2016; Beaugelin et al., 2019; Dogra et al., 2019; Wang et al., 2020). The EX1 protein is located in the grana margins of the thylakoids (Wang et al., 2016; Dogra et al., 2018; Dogra and Kim, 2020), relatively distant from the main $^1O_2$ source under excessive light, the PSII reaction centers in the grana core (D'Alessandro and Havaux, 2019). EX1 can sense $^1O_2$ produced from free chlorophylls associated with the PSII repair process or released during non-photoinhibitory stress through oxidative post-translational modification and proteolysis by the protease FtsH2 (Wang et al., 2016; Dogra et al., 2019). Following these modifications, EX1 can induce PCD. However, PCD in wild-type and *ch1* leaves under high PFDs at a controlled temperature is independent of EX1 (Ramel et al., 2013a; Shumbe et al., 2016), indicating the existence of at least two $^1O_2$ signaling pathways that lead to cell death.

Prolonged exposure to extreme light conditions results in an increased accumulation of ROS, especially a combination of $^1O_2$ and reduced/radical O_2 species (Laloi and Havaux, 2015; Khorobrykh et al., 2020). While $^1O_2$ generates lipid endoperoxides, O_2 radicals oxidize lipids to lipid radicals and peroxyl radicals. Peroxyl radicals can react with other lipids, generating a new lipid radical, as well as a peroxyl radical in a chain reaction (Mano et al., 2019b; Khorobrykh et al., 2020). Another vicious circle has been highlighted between RCS and H_2O_2 (Biswas et al., 2020). RCS can alter protein function by covalent modifications (Colzani et al., 2013), and H_2O_2 scavenging enzymes (catalase and ascorbate peroxidase) were observed in a study as targets of RCS (Mano et al., 2014). In addition, an animal catalase has been shown to form adducts with HNE, leading to enzyme inactivation and compromised H_2O_2 scavenging (Bauer and Zarkovic, 2015; Biswas et al., 2020). H_2O_2 itself can also modify the activity of enzymes in the detoxification pathway. Indeed, it was recently demonstrated that, at relatively low concentrations (0.2 mM), H_2O_2 hinders almost 50% of the HNE-reducing activity present in BY-2 cell cultures, and this can occur within minutes (Biswas et al., 2020). Thus, RCS production can result in the accumulation of H_2O_2 that could inhibit RCS detoxification, hence generating a feed-forward amplification mechanism.

H_2O_2 is a long-lived ROS that can travel to the nucleus and act as a signaling agent (Exposito-Rodriguez et al., 2017). Nonetheless, it has been recently shown that RCS mediates H_2O_2-induced PCD by inducing caspase-3-like activity in BY-2 cell cultures (Biswas and Mano, 2016; Biswas et al., 2020). Therefore, the overaccumulation of RCS can be a major mechanism for the induction of cell death and a mechanism for linking photosynthetic inhibition to PCD. Consistent with this, we have observed that a 4-h pretreatment of *Arabidopsis* plants with HNE increased lipid peroxidation under excessive light (D'Alessandro et al., 2018).

HORMONAL CONTROL OF EXCESSIVE LIGHT-INDUCED CELL DEATH

A key regulator of PCD under excessive light is the AGC kinase OXI1. OXI1 mediates cell death in response to both biotic and

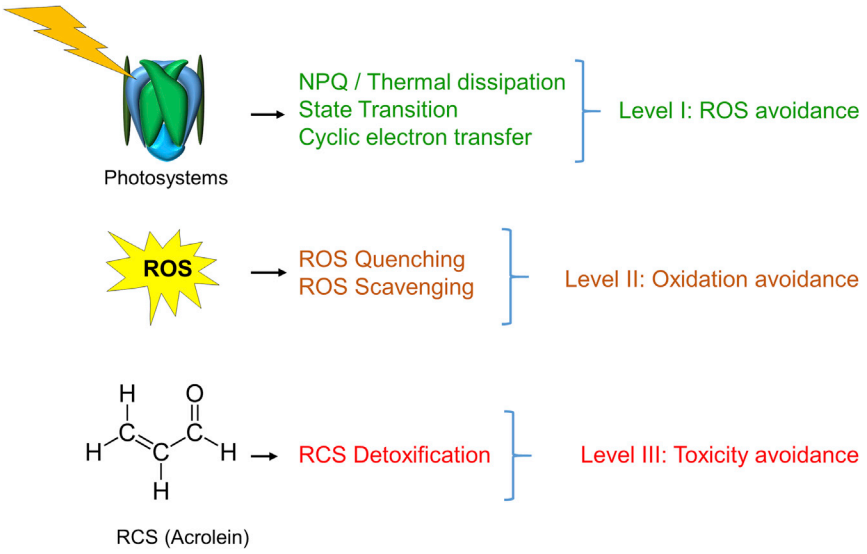

Figure 3. Levels of Photoprotective Mechanisms.
Different levels of photoprotective mechanisms are present in plants to sequentially limit (1) ROS photoproduction, (2) ROS-induced oxidation, and (3) toxicity of photooxidation by-products.

abiotic stimuli, and it resides at the intersection of H_2O_2 and $^1O_2$ signaling pathways (Shumbe et al., 2016). OXI1 is also linked to the signaling pathways of several phytohormones (Howden et al., 2011). OXI1 is required for the full activation of mitogen-activated protein kinase 3 (MAPK3) and MAPK6 to induce PCD under H_2O_2 stress (Rentel et al., 2004) and induces jasmonic acid (JA) and salicylic acid (SA) biosynthesis in leaves exposed to high light (Shumbe et al., 2016; Beaugelin et al., 2019).

There are many examples in the literature reporting a link between plant/leaf photodamage and induction of the JA pathway. For instance, the transfer of *Arabidopsis* plants from 80 to 800 μmol m^{-2} s^{-1} PFD increased the concentration of JA and JA-Ile (Alsharafa et al., 2014). In apple, sun damage was also accompanied by increased concentrations of these compounds (Torres et al., 2017). Moreover, treatments of leaves with methyl-JA under excessive light enhances PSII photoinhibition and photodamage (Wierstra and Kloppstech, 2000; Ramel et al., 2013a; Savchenko et al., 2017). Photooxidative damage in the $^1O_2$-overproducing *Arabidopsis* mutant *ch1* is also associated with a marked upregulation of JA biosynthesis genes and by a strong accumulation of OPDA (12-oxo-phytodienoic acid) and JAs (Ramel et al., 2013a). Recently, a study of the excessive light transcriptome of *Arabidopsis* further confirmed that plants respond to excessive light by modifying the metabolism of JA, and additionally revealed potential roles for SA and abscisic acid (ABA) (Huang et al., 2019). Taken collectively, these results support the idea that JA plays an important role in response to exposure to excess light.

The role of JAs in PCD associated with plant defense against feeding insects, nematodes, and fungal and bacterial pathogens is already well documented (Rentel et al., 2004; Reinbothe et al., 2009) and suggests that it may also mediate PCD under abiotic stress conditions. Indeed, JA accumulation under excessive light is associated with the induction of senescence-associated factors, such as SAG12, SAG13, SEN1, and SEN4, as well as PCD inducers, including MC8 and γ-vacuolar processing enzyme (γ-VPE) (Beaugelin et al., 2019). Furthermore, the allene oxide synthase mutant of *Arabidopsis thaliana* (*dde2*), which is deficient in JA biosynthesis, is much more tolerant to high light-induced cell death (at a controlled temperature) than the wild-type (Ramel et al., 2013b). Similarly, the acclimation of the *Arabidopsis ch1* mutant to excessive light and $^1O_2$ stress requires the downregulation of the JA pathway and JA accumulation (Ramel et al., 2013a).

The role of JA in response to excessive light appears to be dependent on temperature. When high light is combined with high temperature, the *dde2* JA mutant is less tolerant than the wild type (Balfagón et al., 2019). In line with these results, JA is known to act together with SA to confer basal thermotolerance (Clarke et al., 2009). This emphasizes further the fact that different light stress conditions induce different, and sometimes opposite, physiological responses.

Similarly to JA, both SA and ABA also accumulate in plants exposed to excess light energy (Galvez-Valdivieso et al., 2009; Lv et al., 2015; Beaugelin et al., 2019). ABA deficiency in *Arabidopsis* was reported to enhance plant sensitivity to high light (Huang et al., 2019), suggesting a positive role for ABA in stress tolerance. Conversely, the SA-deficient *sid2* mutant of *Arabidopsis* shows enhanced tolerance to high light stress as shown by a marked reduction of lipid peroxidation compared with the wild type (Beaugelin et al., 2019). These results support the finding that SA overaccumulation leads to $^1O_2$ burst in chloroplasts (Lv et al., 2019), resulting in a possible self-amplifying loop. Furthermore, SA biosynthesis is downstream of JA accumulation (Beaugelin et al., 2019), suggesting that the cell-death mechanism under abiotic stress is different from the well-known SA–JA antagonism in biotic stress. Clearly, the role of these two stress hormones in plant tolerance/sensitivity to excess light energy still needs more investigation (Liu et al., 2016).

Emerging evidence has also implicated cytokinin signaling in abiotic stresses related to photosynthetic dysfunction and ROS production (Zwack and Rashotte, 2015; Cortleven et al., 2019). The loss of the cytokinin receptors AHK2 and AHK3 results in greater photooxidative stress tolerance (induced by a combination of water deficit and moderately elevated PFD), as indicated by decreased lipid peroxidation and by chlorophyll loss, and this was interpreted as an indication that cytokinins negatively regulate photooxidative stress tolerance (Danilova et al., 2014). However, in another study (Cortleven et al., 2014), ahk2,3 and arr1,12 double mutants were hypersensitive to high light stress, indicating that cytokinin signaling may play a positive role in this response. These contradictory results illustrate even better the need for standardized conditions in imposing high light stress (i.e., temperature).

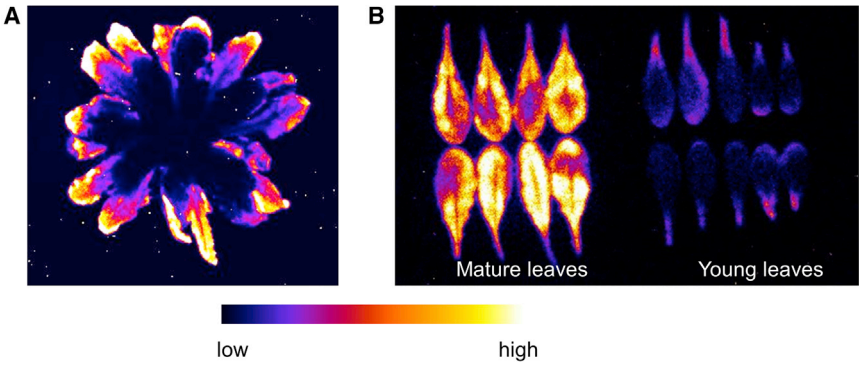

Figure 4. The Extent of High Light-Induced Lipid Peroxidation Is Age-Dependent.
(A) Autoluminescence image of lipid peroxidation in an *Arabidopsis* plant exposed to high light (1500 μmol photons m^{-2} s^{-1}) and low air temperature (7°C), showing that lipid peroxides accumulate in old/mature leaves.
(B) Autoluminescence images of detached *Arabidopsis* leaves exposed to a PFD of 1100 μmol photons m^{-2} s^{-1} for 16 h in a cold chamber (6°C), confirming that young leaves are more tolerant to photooxidative damage than old leaves. The color palette indicates the signal intensity from low (blue) to high values (white). Adapted from D'Alessandro et al. (2018).

ENDOPLASMIC RETICULUM IN THE RESPONSE TO EXCESS LIGHT ENERGY

Although excessive light is a stress that primarily affects the chloroplast, it induces an extended response that can lead to cell death and senescence of a full organ. Counterintuitively, it was recently shown in *Nicotiana tabacum* seedlings that chloroplastic gene expression and translation showed only limited changes after an increase in PFD from 350 μmol photons m^{-2} s^{-1} to 1000 μmol m^{-2} s^{-1} at 24°C (Schuster et al., 2020). These results are in agreement with the genetic response to excessive light of the *Arabidopsis* $^1O_2$-overproducer mutant *ch1*, which showed that only 9% of the total genetic response involved chloroplastic protein-encoding genes (Beaugelin et al., 2020). The vast majority of the response was nuclear, followed by genes encoding mitochondrial proteins and endoplasmic reticulum (ER)-located proteins. A possible explanation for the ER response lies in the extensive modification of proteins caused by RCS.

ER stress is a well-described phenomenon occurring in eukaryotic cells when unfolded or misfolded proteins accumulate in the ER lumen and overwhelm the folding capacity of the ER. Plant signaling in the ER was previously reviewed by Park and Park (2019): ER stress triggers a response called the unfold protein response (UPR) by activating two signaling branches via bZIP28 and IRE1/bZIP60, which can sense and transduce the information (Park and Park, 2019). Both branches are activated when binding immunoglobulin protein (BIP) chaperones are released from bZIP28 and IRE1 inside the ER and bind to unfolded proteins (Howell, 2013; Nawkar et al., 2018).

In both plant and animal systems, the ER integrates cellular signals and resides at the intersection between tolerance and cell-death responses (Su et al., 2013; Kørner et al., 2015; Li et al., 2015). Evidence that ROS production in chloroplasts and mitochondria triggers the UPR in plants is accumulating, and Ozgur et al. (2015) depicted transcriptional modifications of UPR gene expression upon application of H_2O_2, rotenone, methyl viologen, or 3-(3,4-dichlorophenyl)-1,1-dimethylurea, hence mimicking organellar ROS production (Ozgur et al., 2015). In agreement with these results, the UPR is triggered in the $^1O_2$-overproducing *ch1* mutant both under physiological growth conditions (chronic induction) and during excessive light stress (Beaugelin et al., 2020). However, the $^1O_2$ lifetime has been estimated in the sub-microsecond range in thylakoid membranes, allowing an estimated diffusion distance of around only 5.5 nm (Khorobrykh et al., 2020). Therefore, $^1O_2$ must react with molecules in its proximity that constitute the actual signal mediators (e.g., β-carotene oxidation products, lipid peroxides, RCS). Walley et al. (2015) demonstrated that MEcPP, an intermediate of isoprenoid biosynthesis in the chloroplast, was able to activate UPR genes *in vivo*. In fact, the accumulation of MEcPP in the *Arabidopsis ceh1* mutant causes the constitutive accumulation of SA, which triggers the UPR response in the ER and regulates JA-responsive genes (Xiao et al., 2012; Lemos et al., 2016; Bjornson et al., 2017).

Under high light conditions, mutants of the UPR signaling pathway have helped to reveal the key role of the ER in triggering cell-death mechanisms (Beaugelin et al., 2020). The double mutant *bzip60 bzip28* showed an increased sensitivity toward excessive light stress, while pretreatment with tauroursodeoxycholic acid (a chemical chaperone alleviating UPR) leads to increased tolerance (Beaugelin et al., 2020). Moreover, pretreatment of plants with the ER stress inducer, tunicamycin, was shown to enhance photosensitivity, with the induction of PCD marker genes and the development of leaf cell death and lipid peroxidation under excessive light (Beaugelin et al., 2020). The strict relationship between chloroplasts and ER stress under excessive light is further demonstrated by AtDAD1 and AtDAD2, two ER-located proteins, which play a key role as inhibitors of excessive light-induced PCD (Beaugelin et al., 2019). DAD proteins appear to act as molecular brakes on $^1O_2$-induced and OXI1/JA/SA-mediated cell death by inhibiting OXI1.

To date, ER-mediated PCD is known to involve several mechanisms such as the regulation of pro- or anti-death members of the Bcl-2 family, the induction of plant-specific N-rich protein (NRP) cell-death signaling, and the induction of proteins exhibiting caspase-1-like or caspase-3-like activities (Williams et al., 2010; Reis and Fontes, 2012). AtBAG7 is a member of the Bcl-2-associated athanogene (BAG) family located in the ER. AtBAG7 directly binds to AtBIP2 during UPR and facilitates heat and cold stress tolerance. Carvalho et al. (2014) showed that overexpression of BIP in soybean induces delayed senescence under developmental and abiotic stress conditions and leads to the attenuation of NRP-mediated cell death (Carvalho et al., 2014). Accordingly, a knockout mutant of *BiP3* exhibited increased sensitivity to high light stress in *Arabidopsis* (Beaugelin et al., 2020). In NRP-overexpressing

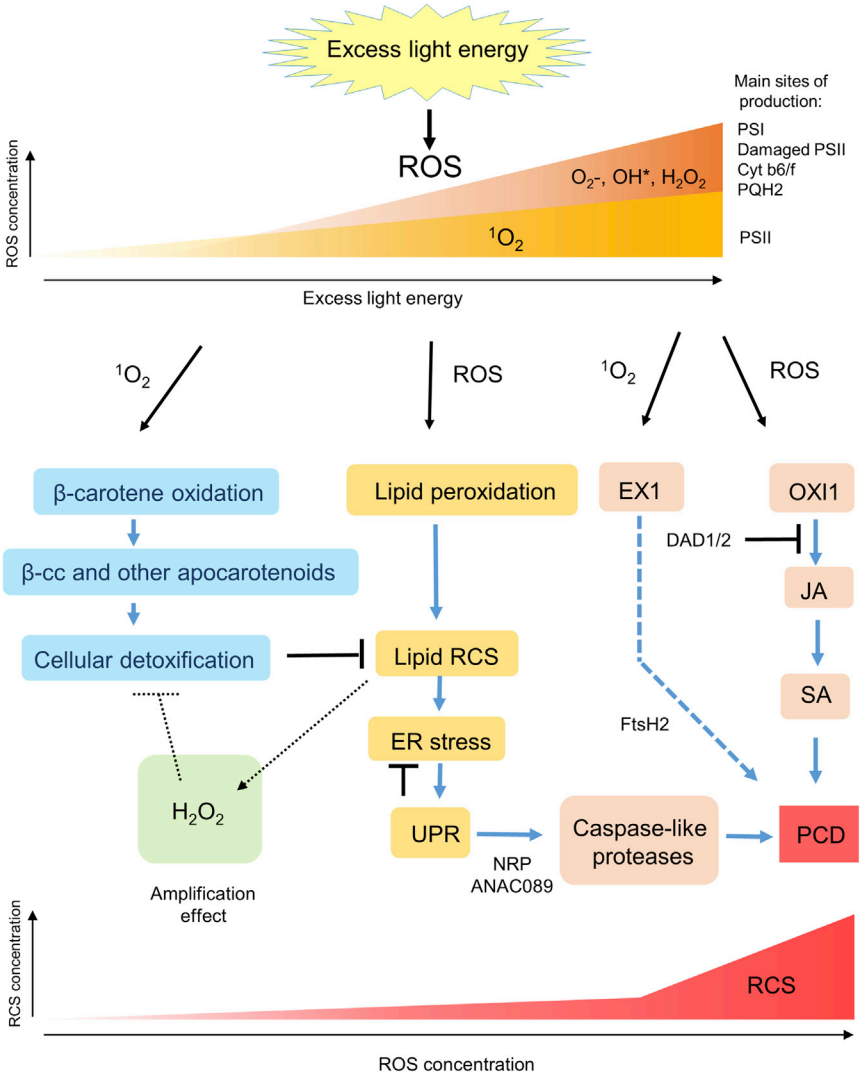

Figure 5. Scheme of the Pathways of Excessive Light-Induced PCD in Plants.
Excessive absorption of light energy in the chloroplasts brings about ROS production that triggers several signaling pathways, thereby leading to PCD. These pathways involve reactive products derived from lipid peroxidation, phytohormones, and PCD-regulatory proteins. The ER plays an important integrative function in the orientation of the stress response toward PCD. Apocarotenoids, such as β-cyclocitral, are upstream signals that can induce cellular detoxification mechanisms, hence modulating the pro-death action of toxic lipid oxidation products.

soybean lines, the transcription factor GmNAC81 (a homolog of ANAC089 in *A. thaliana*) is strongly induced, thereby promoting caspase-1-like and caspase-3-like activities (Reis and Fontes, 2012). GmNAC81 interacts with GmNAC30, which together bind to the promoter of a VPE involved in PCD (Mendes et al., 2013). In *Arabidopsis*, ANAC089 controls a number of PCD regulators, including MC5, BAG6, and NAC094, under stress conditions (Yang et al., 2014). In agreement with these findings, the $^1O_2$-overproducing *ch1* mutant exhibits increased expression of ANAC089 under excessive light stress (Beaugelin et al., 2020). Finally, caspase-like activity seems to be a pan-maker of plant PCD, hence mediating excessive light-induced PCD downstream of RCS and prolonging ER stress signaling.

WHY DO PLANTS KILL LEAF CELLS UNDER EXCESSIVE LIGHT?

The biological role of PCD is easy to understand in a phytopathological context: it limits further pathogen spreading and triggers defense mechanisms in noninfected neighbor tissues (Morel and Dangl, 1997; Heath, 2000). The physiological relevance of excessive light-induced cell death is less clear. A relevant aspect in this context is the fact that the photoprotective capacity strongly depends on leaf age (Bielczynski et al., 2017; D'Alessandro et al., 2018). In fact, when plants are exposed to excessive light, lipid peroxidation is usually much more pronounced in mature leaves (D'Alessandro et al., 2018) (Figure 4). Non-functional photosynthetic tissues become an unnecessary carbon sink, and damaged tissues are more exposed to pathogen attack (Savatin et al., 2014). Therefore, plants appear to choose to sacrifice old/mature leaves when they are photodamaged and recover important nutrients rather than invest in repair. In addition, cell membranes damaged under photooxidative conditions could lead to water loss (Carvalho et al., 2017; Li et al., 2017). Stomatal and leaf hydraulic systems are known to be very responsive to stress conditions, and they can decline even before cell integrity is lost (Trueba et al., 2019). Therefore, PCD may be a means to avoid dramatic water losses from photodamaged leaves to preserve water relations and promote plant survival. Therefore, the preservation of healthy organs by light-induced PCD of photooxidized organs is a defense mechanism that can come into play under many environmental constraints.

CONCLUDING REMARKS AND PERSPECTIVES

In this review, we provide a broad summary of the literature linking pure excessive light exposure to PCD. We highlight the importance of controlling the conditions of excessive light treatments, particularly leaf temperature, to limit variability between laboratories. In fact, by selecting results based on pure excessive light exposure, and not from the superposition of heat and high light stress, we are able to propose a model explaining how this stress can trigger PCD (Figure 5). After an initial phase of stress response, in which the role of $^1O_2$ is predominant and phototolerance mechanisms are activated, damage to PSII and decay of lipid peroxides to RCS leads to the accumulation of reduced forms of oxygen. H_2O_2 further enhances the increase in RCS by limiting cellular detoxification, leading to ER stress. If

the stress does not cease, ER stress may then lead to the activation of PCD induced by caspase-like activity. At the same time, overproduction of $^1O_2$ may activate EX1 at the grana margins, together with the OXI1/JA/SA controlled PCD.

Although we were able to identify several steps in the response to excessive light, many questions remain to be answered. Firstly, we know that our knowledge of phototolerance mechanisms is not exhaustive and that many molecular players in apocarotenoid sensing remain elusive. Secondly, it is not clear whether the signaling of these metabolites interacts with other known retrograde signaling pathways. Finally, PCD may be mediated by at least three pathways in our model, and the connections and interrelations between caspase-like inducers of PCD and EX1- and OXI1-induced PCD remain unknown. Therefore, in our opinion, it is pivotal to find similar working conditions between laboratories to maximize the compatibility between the results. At the same time, the substantial difference between excessive light alone or in combination with heat shock highlights the importance of studying stress combinations.

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