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1 **Tanned or Sunburned: How Excessive Light Triggers Plant Cell Death**

2

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11 **Running Title:** Plant cell death induced by excessive light

12

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16

17 **Short Summary**

18 Capturing light intensities exceeding the capacity of photosynthesis can lead to cell death in plant
19 leaves. In this review, we summarize recent findings on the mechanisms mediating light-induced
20 cell death, highlighting the role of reactive carbonyl species, of the endoplasmic reticulum and of
21 phytohormones in the adoption of phototolerance versus cell death pathways.

22

23 **Keywords:** Arabidopsis, photosynthesis, excessive light, photooxidative stress, carotenoid, plant cell
24 death, stress response.

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33 **Abstract**

34 Plants often encounter light intensities exceeding the capacity of photosynthesis (excessive light)
35 mainly due to biotic and abiotic factors, which lower CO₂ fixation and reduce light energy sinks.
36 Under excessive light, the photosynthetic electron transport chain generates damaging molecules
37 hence leading to photooxidative stress and eventually to cell death. In this review, we summarize
38 the mechanisms linking excessive absorption of light energy in the chloroplasts to programmed cell
39 death in plant leaves. We highlight the importance of reactive carbonyl species generated by lipid
40 photooxidation, their detoxification, and the integrating role of the endoplasmic reticulum in the
41 adoption of phototolerance or cell death pathways. Finally, we invite the scientific community to
42 standardize the conditions of excessive light treatments.

43

44

45 **Abbreviations**

46 PFD, Photon Flux Density; ROS, Reactive Oxygen Species; RWC, Relative Water Content; OEC,
47 Oxygen Evolving Complex; PCD, Programmed Cell Death; RCS, Reactive Carbonyl Species; HNE, 4-
48 Hydroxy-2-nonenal; NPQ, Non-Photochemical Quenching; JA, Jasmonic acid; JA-Ile, Jasmonoyl-
49 Isoleucine; SA, Salicylic acid; OXI1, OXIDATIVE STRESS INDUCIBLE 1; EX1, EXECUTER 1; ER,
50 Endoplasmic Reticulum; UPR, Unfolded Protein Response; ABA, abscisic acid; ¹O₂, singlet oxygen.

51

52 **Introduction**

53 Plants often encounter light intensities that exceed the needs of photosynthesis (Ort, 2001). Light
54 energy that is not used to assimilate CO₂ or dissipated can be considered to be in excess and leads
55 to a depression of photosynthetic efficiency, especially at PSII (Kato et al., 2003; Murata et al., 2007).
56 Excessive light occurs mainly in response to biotic and abiotic factors, such as drought, heat or pests,
57 and lowers the capacity of light energy sinks, first and foremost CO₂ fixation. The excess electrons
58 and excitation energy can be transferred to molecular oxygen, generating biologically damaging
59 molecules such as reactive oxygen species (ROS), peroxides and radicals, hence leading to
60 photooxidative stress that can ultimately lead to cell death (Apel and Hirt, 2004; Li et al., 2009).

61

62 **High light under different conditions induces different responses**

63 The real condition of excessive light, which is dependent on photosynthetic activity and can also
64 occur at relatively low light intensities, is often confused with 'high light' treatment. Drought stress,
65 nutrient starvation or pathogens can lead to the same state of overexcitation of the photosystems,
66 without the imposition of high light (Garai and Tripathy, 2018; Wang et al., 2018; Pérez-Bueno et
67 al., 2019). Likewise, by playing with the CO₂ and O₂ composition of the gas phase, Montané et al.
68 (1998) generated the same excitation pressure on the photosystems at different photon flux
69 densities (PFDs) (Montané et al., 1998). In addition, the intensity of sunlight that leaves receive
70 under natural conditions often fluctuates, because of clouds and intermittent shading by other
71 leaves or plants. This "fluctuating light" can be very damaging to the photosynthetic machinery,
72 particularly PSI, even at relatively low PFDs (Kono et al., 2014; Yamori et al., 2016).

73

74 PFDs used in the literature to induce excessive light range from 500 to 2000 μmol photons m⁻² s⁻¹.
75 These treatments can also increase leaf temperature and, unless air temperature is controlled, will

76 impose a double condition of excessive light and heat stress. Lack of leaf temperature control in
77 experimental protocols increases interlaboratory data variability and makes comparison between
78 different studies more difficult.

79
80 Even when leaf temperature is controlled by lowering air temperature, different setups have been
81 adopted, such as 600 $\mu\text{mol photons m}^{-2} \text{s}^{-1}$ PFD at 23°C air temperature (Balfagón et al., 2019), 1500
82 $\mu\text{mol photons m}^{-2} \text{s}^{-1}$ at 7°C (Ramel et al., 2012; D'Alessandro et al., 2018), 1200 $\mu\text{mol photons m}^{-2}$
83 s^{-1} at 18°C (Lv et al., 2015), 1200 $\mu\text{mol photons m}^{-2} \text{s}^{-1}$ at 11.5°C (Huang et al., 2019) or 2000 μmol
84 $\text{photons m}^{-2} \text{s}^{-1}$ at 4°C (Mattila et al., 2019). These studies show that leaf temperature remains at
85 standard levels (20-22°C) when exposed to 1500 $\mu\text{mol photons m}^{-2} \text{s}^{-1}$ at 7°C or 1200 $\mu\text{mol photons}$
86 $\text{m}^{-2} \text{s}^{-1}$ at 11.5°C. However, leaf temperature increases by 4°C (from 23°C to 27°C) when exposed to
87 600 $\mu\text{mol photons m}^{-2} \text{s}^{-1}$ or 13°C (from 22°C to 35°C) at 1200 $\mu\text{mol photons m}^{-2} \text{s}^{-1}$ if air temperature
88 is not lowered.

89
90 Several studies have indicated that there can be significant overlap between high light and heat
91 stress responses when air temperature is maintained at normal growth temperature. Balfagón et
92 al. (2019) compared a 7-h high light treatment (HL) with a heat shock treatment (HS, 42°C) and the
93 combination of both (Balfagón et al., 2019). The HL treatment caused a 4°C increase in leaf
94 temperature, from 23°C to 27°C, the HS treatment increased leaf temperature by around 10°C, from
95 23°C to 33°C, and the combined stresses increased leaf temperature by around 10-12°C. Analysis
96 of transcriptomes showed an astonishing resemblance between HL (at 23°C air temperature) and
97 HS, with a large fraction of the modified genes being common between the two stresses,
98 corresponding to 58% (4439) of HL alone. This result suggests that performing HL experiments at
99 23°C air temperature causes a response partially overlapping with HS, likely due to the increase in
100 leaf temperature. While the combination of HS and HL was equally similar to HS and to HL (64%
101 overlap) on a transcriptional level, other experiments indicated a dominance of HS during HL stress.
102 In fact, while HL performed at 23°C caused stomatal closure, HS and HL+HS induced stomatal
103 opening. Relative water content (RWC) was unperturbed by HL but reduced from 75% to 60% by HS
104 and HS+HL. The number of starch granules diminished in HL but increased under HS and HS+HL.
105 H_2O_2 production increased in HS and HS+HL-treated plants but did not in HL-treated plants, in
106 agreement with previous analyses of pure excess light (D'Alessandro et al., 2018). Similar results

107 were observed in another study when analyzing the systemic response to HL, HS and the
108 combination of both stresses (Zandalinas et al., 2020).

109

110 **The lipid problem in excessive light**

111

112 ROS production under stress conditions initially elicits a signaling cascade and, if sufficient quantities
113 are produced to overwhelm defense mechanisms, also leads to cellular damage. Singlet oxygen ($^1\text{O}_2$)
114 is a very harmful and likely the major ROS produced under excessive light (Triantaphylidès et al.,
115 2008; Ramel et al., 2012; D'Alessandro and Havaux, 2019; Khorobrykh et al., 2020). In fact, a $^1\text{O}_2$
116 mediated transcriptional response can be detected after a 24-h high light treatment at controlled
117 air temperature, while marker genes for H_2O_2 are not induced (Shumbe et al., 2017). This result is
118 in line with the increased biosynthesis of H_2O_2 and the induction of *APX2* only when high light
119 conditions are combined with heat stress (Smirnoff and Arnaud, 2019; Balfagón et al., 2019; Huang
120 et al., 2019). Notably, the O_2 -evolving-complex (OEC) proteins are particularly susceptible to heat
121 stress (Berry and Bjorkman, 1980; Weis and Berry, 1988; Havaux, 1993), and OEC-depleted PSII
122 predominantly generates H_2O_2 (Khorobrykh et al., 2020). Only a prolonged high light stress is
123 therefore likely to cause a combined accumulation of $^1\text{O}_2$ and H_2O_2 (Laloi and Havaux, 2015).

124

125 ROS, and especially $^1\text{O}_2$, are highly reactive towards carbon-carbon double bonds. ROS are primarily
126 produced at the level of the photosystems in a lipid rich environment, and so oxidized lipids are one
127 of the major products of ROS generation in response to excessive light. Fatty acids are highly
128 unsaturated in plant leaves (Douce and Joyard, 1980) and are therefore very sensitive to oxidation
129 (Bour et al., 2019). The major fatty acids in leaves are the polyunsaturated fatty acids linolenic acid
130 (C18:3) and linoleic acid (C18:2) containing 3 and 2 double bonds, respectively. At the level of
131 thylakoids, a primary site of ROS production, the ratio between unsaturated to saturated lipids is
132 9.4 to 1, and linolenic acid corresponds to 70% of total fatty acids (Chapman and Barber, 1986;
133 Yalcinkaya et al., 2019). Considering *Arabidopsis* whole leaves, linolenic acid can represent up to 50
134 % of the total fatty acid content (Li-Beisson et al., 2013). Consequently, lipid peroxidation is usually
135 a primary event associated with ROS production and oxidative stress in plants (Birtic et al., 2011).
136 The attack of lipids by ROS generates specific peroxides, notably lipid endoperoxides specific to $^1\text{O}_2$,
137 while lipid radicals and lipid peroxy radicals are specific to reduced oxygen forms (Mano et al.,

138 2019b; Khorobrykh et al., 2020). In a second stage, all these molecules are transformed into lipid
139 hydroperoxides.

140

141 High light treatment with controlled air temperature was found to be systematically associated with
142 a lipid peroxidation signature typical of $^1\text{O}_2$ (Triantaphylidès et al., 2008), indicating a major role for
143 this ROS in photooxidative degradation of plant lipids. The abundance of polyunsaturated fatty
144 acids has been proposed to act as a sink for ROS, especially $^1\text{O}_2$, in chloroplasts, which could
145 constitute a protection mechanism against propagation of oxidative stress (Mène-Saffrané et al.,
146 2009). However, lipid peroxides are not only the signature of photooxidation but also directly
147 contribute to signaling and toxicity.

148

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

158

159 Lipid hydroperoxides spontaneously fragment into a variety of secondary compounds that can
160 exhibit high reactivity towards biomolecules. Reactive carbonyl species (RCS) are the α,β -
161 unsaturated aldehydes and ketones derived from lipid peroxides, which can further hinder the
162 physiological functioning of macromolecules and induce cell death (Farmer and Mueller, 2013;
163 Mano et al., 2019b). Some of these molecules, such as (E)-2-hexenal, have been studied as Green
164 Leaf Volatiles and previously reviewed (Ameye et al., 2018). RCS, such as hydroxynonenal (HNE),
165 acrolein or malondialdehyde (Fig. 1), are electrophiles and hence can react with nucleophilic groups
166 in biomolecules to make covalent bonds (Mano, 2012; Mano et al., 2019b). In this way RCS may
167 change protein conformation or alter protein function under stress conditions, consequently
168 affecting metabolic or signaling processes (Biswas and Mano, 2016; Yalcinkaya et al., 2019).

169

170 **Promiscuous, broad-spectrum detoxification of lipid peroxidation by-products**

171 Reactive molecules, both of endogenous or exogenous origin, may constitute a hazard for the cell
172 and lead to cell death. Plants have evolved a detoxification mechanism that can neutralize these
173 dangers. As this mechanism was discovered when studying the response to herbicides, it was called
174 the 'xenobiotic response' (Sandermann, 1992; Kreuz et al., 1996; Riechers et al., 2010). This process
175 is organized in three phases, modification, conjugation and compartmentalization, and enzyme
176 families participating in each phase of the xenobiotic response are reported in Fig. 2.

177

178 Enzymes of the modification phase are active towards RCS and catalyze their conversion into less
179 dangerous molecules (Mano et al., 2019b). RCS detoxification can occur through different enzymatic
180 reactions: (i) reduction of the α,β -unsaturated bond in an RCS molecule using NAD(P)H as the
181 electron donor, by alkenal reductase (AER) and alkenal/one oxidoreductase (AOR); (ii) reduction of
182 a carbonyl moiety to an alcohol using NAD(P)H, catalyzed with aldo-keto reductase (AKR); (iii)
183 oxidation of an aldehyde to a carboxylic acid using NAD⁺ as the electron acceptor, catalyzed by
184 aldehyde dehydrogenase (ALDH); (iv) oxidation of an aldehyde using O₂ as the electron acceptor
185 catalyzed by aldehyde oxidase (AO) (Mano et al., 2002; Stiti et al., 2011; Yamauchi et al., 2011;
186 Yamauchi et al., 2012; SAITO et al., 2013; Srivastava et al., 2017). In addition, formation of a
187 glutathione or glucoside adduct of RCS can be catalyzed by enzymes of the conjugation phase (i.e.
188 glutathione transferase (GST)) (Mano et al., 2017; Mano et al., 2019a).

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

197

198 Enzymes of the modification phase are usually associated with biosynthetic pathways for secondary
199 metabolites (Cytochrome P450 oxidases, Short Chain Dehydrogenases/Reductases, ...). At the same
200 time, part of these large families of enzymes was identified in the response to xenobiotic
201 compounds (Fode et al., 2008). It is likely that plants can adopt the less specific steps of the reactions

202 that they perform in secondary metabolism to cope with hazardous molecules, in what could be
203 considered a broad-spectrum (promiscuous) detoxification pathway. Ligand promiscuity and
204 promiscuous detoxification are well-known processes in animal systems where the two
205 promiscuous receptors CAR and PXR can bind to different molecules and controls the expression of
206 several CYP450 genes (Tolson and Wang, 2010). These processes, catalyzed by the same families of
207 enzymes as the xenobiotic response of plants, are active against endogenous toxicants and
208 constitute the base of drug metabolism and pharmacokinetics. We therefore propose to adopt the
209 animal systems nomenclature of promiscuous detoxification for the xenobiotic response in plants.

210

211 As promiscuous detoxification is active towards hazardous metabolites generated during
212 photooxidation, such as RCS, this pathway represents a new level of photoprotection. Light-induced
213 ROS generation is inevitably coupled with photosynthesis. ROS quenching and scavenging are
214 continuous, but under stress conditions they could become insufficient compared to ROS
215 production, leading to ROS accumulation. At this point, a first level of photoprotection, including
216 nonphotochemical energy quenching (NPQ), cyclic electron transport, plastid terminal oxidase
217 (PTOX) and state transition, reduces ROS production (Alric and Johnson, 2017; Roach and Krieger-
218 Liskay, 2019). A second level of photoprotection, including ROS scavenging (by SOD and APX, for
219 the superoxide anion radical and H₂O₂, respectively) and ROS quenching (by carotenoids,
220 tocopherols and plastoquinones for ¹O₂), avoids oxidation (Apel and Hirt, 2004; Krieger-Liskay et
221 al., 2008; Noctor et al., 2018; Filiz et al., 2019). The D1 protein of PSII, which is very susceptible to
222 oxidation, can also be considered a component of photoprotection together with the well described
223 D1 repair mechanism (Nath et al., 2013; Järvi et al., 2015; Yoshioka-Nishimura, 2016; Kato et al.,
224 2018). When these photoprotective processes are insufficient, or inhibited by stress, oxidation of
225 lipids generates RCS. Promiscuous detoxification can metabolize the hazardous reactive molecules
226 to protect the cell (Fig. 3) which, under certain conditions, can trigger programmed cell death (PCD)
227 (Biswas and Mano, 2016).

228

229 **From RCS to PCD**

230 ¹O₂-derived peroxidation of lipids, proteins and nucleic acids has been observed in treatments with
231 photosensitizers (methylene blue or rose Bengal) and in Arabidopsis mutant lines overproducing ¹O₂
232 such as *flu* and *ch1* (Triantaphylidès et al., 2008; Shao et al., 2013; Laloi and Havaux, 2015). *ch1*
233 showed increased lipid peroxidation and photooxidative damage under excess light conditions,

234 whereas $^1\text{O}_2$ is generated under non-photoinhibitory conditions in *flu* (Wagner et al., 2004; Ramel
235 et al., 2013a). In both *ch1* and *flu*, $^1\text{O}_2$ -induced cell death was dependent on signaling proteins,
236 OXIDATIVE STRESS INDUCIBLE 1 (OXI1) (in *ch1* and in the wild type), EXECUTER1 (EX1) or SAFE1 (in
237 *flu*), indicating the involvement of a signaling process leading to cell death (Shumbe et al., 2016;
238 Dogra et al., 2019; Beaugelin et al., 2019; Wang et al., 2020). The EX1 protein is located in the grana
239 margins of the thylakoids (Wang et al., 2016; Dogra et al., 2018; Dogra and Kim, 2020), relatively
240 distant from the main $^1\text{O}_2$ source under excessive light, the PSII reaction centers in the grana core
241 (D'Alessandro and Havaux, 2019). EX1 can sense $^1\text{O}_2$ produced from free chlorophylls associated
242 with the PSII repair process or released during non-photoinhibitory stress, through oxidative post-
243 translational modification and proteolysis by the protease FtsH2 (Wang et al., 2016; Dogra et al.,
244 2019). Following those modifications, EX1 can induce PCD. However, PCD in WT and *ch1* leaves
245 under high PFDs at controlled temperature is independent of EX1 (Ramel et al., 2013a; Shumbe et
246 al., 2016), indicating the existence of at least two $^1\text{O}_2$ signaling pathways leading to cell death.

247 Prolonged exposure to extreme light conditions would result in an increased accumulation of ROS,
248 principally a combination of $^1\text{O}_2$ and reduced/radical O_2 species (Laloi and Havaux, 2015;
249 Khorobrykh et al., 2020). While $^1\text{O}_2$ generates lipid endoperoxides, O_2 radicals oxidize lipids to lipid
250 radicals and peroxy radicals. Peroxy radicals can react with other lipids generating a new lipid
251 radical, and again a peroxy radical in a chain reaction (Mano et al., 2019b; Khorobrykh et al., 2020).
252 Another vicious circle has been highlighted between RCS and H_2O_2 (Biswas et al., 2020). RCS can
253 alter protein function by covalent modification (Colzani et al., 2013), and H_2O_2 scavenging enzymes
254 (catalase and ascorbate peroxidase) were observed in a study as targets of RCS (Mano et al., 2014).
255 In addition, an animal catalase has been shown to form adducts with HNE, leading to inactivation of
256 the enzyme and a compromised H_2O_2 scavenging (Bauer and Zarkovic, 2015; Biswas et al., 2020).
257 H_2O_2 itself can also modify the activity of enzymes of the detoxification pathway. Indeed, it was
258 recently demonstrated that, at relatively low concentrations (0.2 mM), H_2O_2 hinders almost 50% of
259 the HNE-reducing activity present in BY-2 cell cultures, and this can occur within minutes (Biswas et
260 al., 2020). Thus, RCS production can result in the accumulation of H_2O_2 that could inhibit RCS
261 detoxification, hence generating a feed-forward amplification mechanism.

262

263 H_2O_2 is a long-lived ROS that can travel to the nucleus and act as a signaling agent (Exposito-
264 Rodriguez et al., 2017). Nonetheless, it has been recently shown that RCS mediates H_2O_2 -induced
265 PCD by inducing caspase-3-like activity in BY-2 cell cultures (Biswas and Mano, 2016; Biswas et al.,

266 2020). Therefore, the overaccumulation of RCS could be a major mechanism for the induction of cell
267 death, and a mechanism for linking photosynthetic inhibition to PCD. Consistent with this, we have
268 observed that a 4-h pretreatment of Arabidopsis plants with HNE increased lipid peroxidation under
269 excessive light (D'Alessandro et al., 2018).

270

271 **Hormonal control of excessive light-induced cell death**

272 A key regulator of PCD under excessive light is the AGC kinase OXI1. OXI1 mediates cell death in
273 response to both biotic and abiotic stimuli, and it resides at the intersection of the H₂O₂ and ¹O₂
274 signaling pathways (Shumbe et al., 2016). OXI1 is also linked to the signaling pathways of several
275 phytohormones (Howden et al., 2011). OXI1 is required for the full activation of MAPK3 and MAPK6
276 to induce PCD under H₂O₂ stress (Rentel et al., 2004) and induces jasmonic acid (JA) and salicylic acid
277 (SA) biosynthesis in high light-exposed leaves (Shumbe et al., 2016; Beaugelin et al., 2019).

278

279 There are many examples in the literature indicating a link between plant/leaf photodamage and
280 induction of the JA pathway. For instance, the transfer of Arabidopsis plants from 80 to 800 μmol
281 m⁻² s⁻¹ PFD increased the concentration of JA and JA-Ile (Alsharafa et al., 2014). In apple, sun damage
282 was also accompanied by increased concentrations of those compounds (Torres et al., 2017).
283 Moreover, treatments of leaves with methyl-JA under excessive light enhances PSII photoinhibition
284 and photodamage (Wierstra and Kloppstech, 2000; Ramel et al., 2013a; Savchenko et al., 2017).
285 Photooxidative damage in the ¹O₂-overproducing Arabidopsis mutant *ch1* is also associated with a
286 marked upregulation of JA biosynthesis genes and by a strong accumulation of OPDA (12-oxo-
287 phytodienoic acid) and JAs (Ramel et al., 2013a). Very recently, an excessive light transcriptome of
288 Arabidopsis plants further confirmed that plants respond to excessive light by modifying the
289 metabolism of JA, and additionally revealed a potential role for SA and abscisic acid (ABA) (Huang
290 et al., 2019). Together these results support the idea that JA plays an important role in response to
291 exposure to excess light.

292

293 The role of JAs in PCD associated with plant defense against feeding insects, nematodes, fungal and
294 bacterial pathogens is already well documented (Rentel et al., 2004; Reinbothe et al., 2009) and
295 suggests that it may also mediate PCD under abiotic stress conditions. Indeed, JA accumulation
296 under excess light is associated with the induction of senescence-associated factors, such as SAG12,
297 SAG13, SEN1 and SEN4, as well as PCD inducers including MC8 and gVPE (Beaugelin et al., 2019).

298 Furthermore, the allene oxide synthase mutant of *Arabidopsis thaliana* (*dde2*), deficient in JA
299 biosynthesis, is much more tolerant to high light-induced cell death (at controlled temperature)
300 than the wild-type (Ramel et al., 2013b). Similarly, the acclimation of the *Arabidopsis ch1* mutant to
301 excessive light and ¹O₂ stress requires the down-regulation of the JA pathway and JA accumulation
302 (Ramel et al., 2013a).

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

308
309 Similarly to JA, both SA and ABA also accumulate in plants exposed to excess light energy (Galvez-
310 Valdivieso et al., 2009; Lv et al., 2015; Beaugelin et al., 2019). ABA deficiency in *Arabidopsis* was
311 reported to enhance plant sensitivity to high light (Huang et al., 2019), suggesting a positive role for
312 ABA in stress tolerance. Conversely, the SA-deficient *sid2* mutant of *Arabidopsis* shows enhanced
313 tolerance to high light stress as shown by a marked reduction of lipid peroxidation compared with
314 the wild type (Beaugelin et al., 2019). These results support the finding that SA overaccumulation
315 leads to a ¹O₂ burst in the chloroplast (Lv et al., 2019), resulting in a possible self-amplifying loop.
316 Furthermore, SA biosynthesis is downstream of JA accumulation (Beaugelin et al., 2019), suggesting
317 that the cell death mechanism under abiotic stress is different from the well-known SA-JA
318 antagonism in biotic stress. Clearly, the role of these two stress hormones in plant
319 tolerance/sensitivity to excess light energy still needs more investigation (Liu et al., 2016).

320
321 Emerging evidence has also implicated cytokinin signaling in abiotic stresses related to
322 photosynthetic dysfunction and ROS production (Zwack and Rashotte, 2015; Cortleven et al., 2019).
323 Loss of the cytokinin receptors AHK2 and AHK3 results in greater photooxidative stress tolerance
324 (induced by a combination of water deficit and moderately elevated PFD) as indicated by decreased
325 lipid peroxidation and by chlorophyll loss, and this was interpreted as an indication that cytokinins
326 negatively regulate photooxidative stress tolerance (Danilova et al., 2014). However, in another
327 study (Cortleven et al., 2014), *ahk2,3* and *arr1,12* double mutants were hypersensitive to high light
328 stress, indicating that cytokinin signaling may play a positive role in this response. Those

329 contradictory results illustrate even better the need for standardized conditions in imposing high
330 light stress (*i.e.* temperature) .

331

332 **The endoplasmic reticulum in the response to excess light energy**

333 Although excessive light is a stress that primarily affects the chloroplast, it induces an extended
334 response that can lead to cell death and senescence of a full organ. Counterintuitively, it was
335 recently shown in *Nicotiana tabacum* seedlings that chloroplastic gene expression and translation
336 showed only limited changes after an increase in PFD from 350 $\mu\text{mol photons m}^{-2} \text{s}^{-1}$ to 1000 μmol
337 $\text{m}^{-2} \text{s}^{-1}$ at 24°C (Schuster et al., 2020). These results are in agreement with the genetic response to
338 excessive light of the Arabidopsis $^1\text{O}_2$ -overproducer mutant *ch1*, which showed that only 9% of the
339 total genetic response involved chloroplastic protein-encoding genes (Beaugelin et al., 2020). The
340 vast majority of the response was nuclear, followed by genes encoding mitochondrial proteins and
341 endoplasmic reticulum (ER)-located proteins. A possible explanation for the ER response lies in the
342 extensive modification of proteins caused by RCS.

343

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

351

352 Both in plant and animal systems, the ER integrates cellular signals and resides at the intersection
353 between tolerance and cell death responses (Su et al., 2013; Li et al., 2015; Kørner et al., 2015).
354 Evidence that ROS production in chloroplasts and mitochondria triggers the UPR in plants are
355 accumulating, and Ozgur et al. (2015) depicted transcriptional modifications of UPR gene expression
356 upon application of H_2O_2 , rotenone, methyl viologen or 3-(3,4-dichlorophenyl)-1,1-dimethylurea
357 (DCMU), mimicking organellar ROS production (Ozgur et al., 2015). In agreement with these results,
358 the UPR is triggered in the $^1\text{O}_2$ -overproducing *ch1* mutant both under physiological growth
359 conditions (chronic induction) and during excessive light stress (Beaugelin et al., 2020). However,
360 $^1\text{O}_2$ lifetime has been estimated in the sub- μs range in the thylakoid membranes allowing an

361 estimated diffusion distance of around only 5.5 nm (Khorobrykh et al., 2020). Therefore, $^1\text{O}_2$ must
362 react with molecules in its proximity that then constitute the actual signal mediators (e.g. β -
363 carotene oxidation products, lipid peroxides, RCS). Walley and co-workers demonstrated that
364 MEcPP, an intermediate of isoprenoid biosynthesis in the chloroplast, was able to activate UPR
365 genes *in vivo* (Walley et al., 2015). In fact, the accumulation of MEcPP in the *Arabidopsis ceh1*
366 mutant causes the constitutive accumulation of SA, which triggers the UPR response in the ER and
367 regulates JA-responsive genes (Xiao et al., 2012; Lemos et al., 2016; Bjornson et al., 2017) .

368

369 Under high light conditions, mutants of the UPR signaling pathway have helped to reveal the key
370 role of the ER in triggering cell death mechanisms (Beaugelin et al., 2020). The double mutant *bzip60*
371 *bzip28* showed an increased sensitivity towards excessive light stress, while pretreatment with
372 tauroursodeoxycholic acid (TUDCA, a chemical chaperone alleviating UPR) leads to increased
373 tolerance (Beaugelin et al., 2020). Moreover, pre-treatment of plants with the ER stress inducer,
374 tunicamycin, was shown to enhance photosensitivity, with the induction of PCD marker genes and
375 the development of leaf cell death and lipid peroxidation under excessive light (Beaugelin et al.,
376 2020). The strict interconnection between chloroplasts and ER stress under excessive light is further
377 demonstrated by AtDAD1 and AtDAD2, two ER-located proteins, which play a key role as inhibitors
378 of excessive light-induced PCD (Beaugelin et al., 2019). DAD proteins appear to act as molecular
379 brakes on the $^1\text{O}_2$ -induced, OXI1/JA/SA-mediated cell death by inhibiting OXI1.

380

381 To date ER-mediated PCD is known to involve several mechanisms such as the regulation of pro- or
382 anti-death members of the Bcl-2 family, the induction of plant specific N-rich proteins (NRPs) cell
383 death signaling and the induction of proteins exhibiting caspase-1-like or caspase-3-like activities
384 (Williams et al., 2010; Reis and Fontes, 2012). AtBAG7 is a member of Bcl-2-associated athanogene
385 (BAG) located in the ER. AtBAG7 directly binds to AtBIP2 during UPR and facilitates heat and cold
386 stress tolerance. Carvalho et al. (2014) showed that overexpression of BIP in soybean induces
387 delayed senescence under developmental and abiotic stress conditions, and leads to the
388 attenuation of NRP-mediated cell death (Carvalho et al., 2014). Accordingly, a knockout mutant of
389 *BIP3* exhibited an increased sensitivity to high light stress in *Arabidopsis* (Beaugelin et al., 2020). In
390 NRP-overexpressing soybean lines, the transcription factor GmNAC81 (homologue of ANAC089 in
391 *Arabidopsis thaliana*) is strongly induced and promote activation of caspase 1- and caspase 3-like
392 activity (Reis and Fontes, 2012). GmNAC81 interacts with GmNAC30 which together bind to the

393 promoter of a vacuolar processing enzyme (VPE) involved in PCD (Mendes et al., 2013). Also in
394 Arabidopsis, ANAC089 controls a number of PCD regulators, including *MC5*, *BAG6* and *NAC094*,
395 under stress conditions (Yang et al., 2014). In agreement, the ¹O₂-overproducing *ch1* mutant exhibits
396 an increased expression of *ANAC089* under excessive light stress (Beaugelin et al., 2020). Finally,
397 caspase-like activity seems to be a pan-maker of plant PCD mediating excessive light-induced PCD,
398 downstream of RCS and prolonged ER stress signaling .

399

400 **Why do plants kill leaf cells under excessive light?**

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

417

418 **Main conclusions and some perspectives**

419 In this review, we provide a broad summary of the literature linking pure excessive light exposure
420 to programmed cell death. We highlight the importance of controlling the conditions of excessive
421 light treatments, particularly leaf temperature, to limit variability between laboratories. In fact, by
422 selecting results based on pure excessive light exposure, and not from the superposition of heat and
423 high light stress, we are able to propose a model explaining how this stress can trigger PCD (Fig. 5).
424 After an initial phase of stress response, in which the role of ¹O₂ is predominant and phototolerance

425 mechanism are activated, damage to PSII and the decay of lipid peroxides to RCS leads to the
426 accumulation of reduced forms of oxygen. H₂O₂ further enhances the increase in RCS by limiting
427 cellular detoxification, leading to ER stress. If the stress does not cease, ER stress may then lead to
428 the activation of PCD induced by caspase-like activity. At the same time, overproduction of ¹O₂ may
429 activate EX1 at the grana margins, together with the OXI1/JA/SA controlled PCD.

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

440

441

442 **Author contributions**

443 S.D. and M.H. planned, structured, and wrote the article; I.B. contributed to the article.

444

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449

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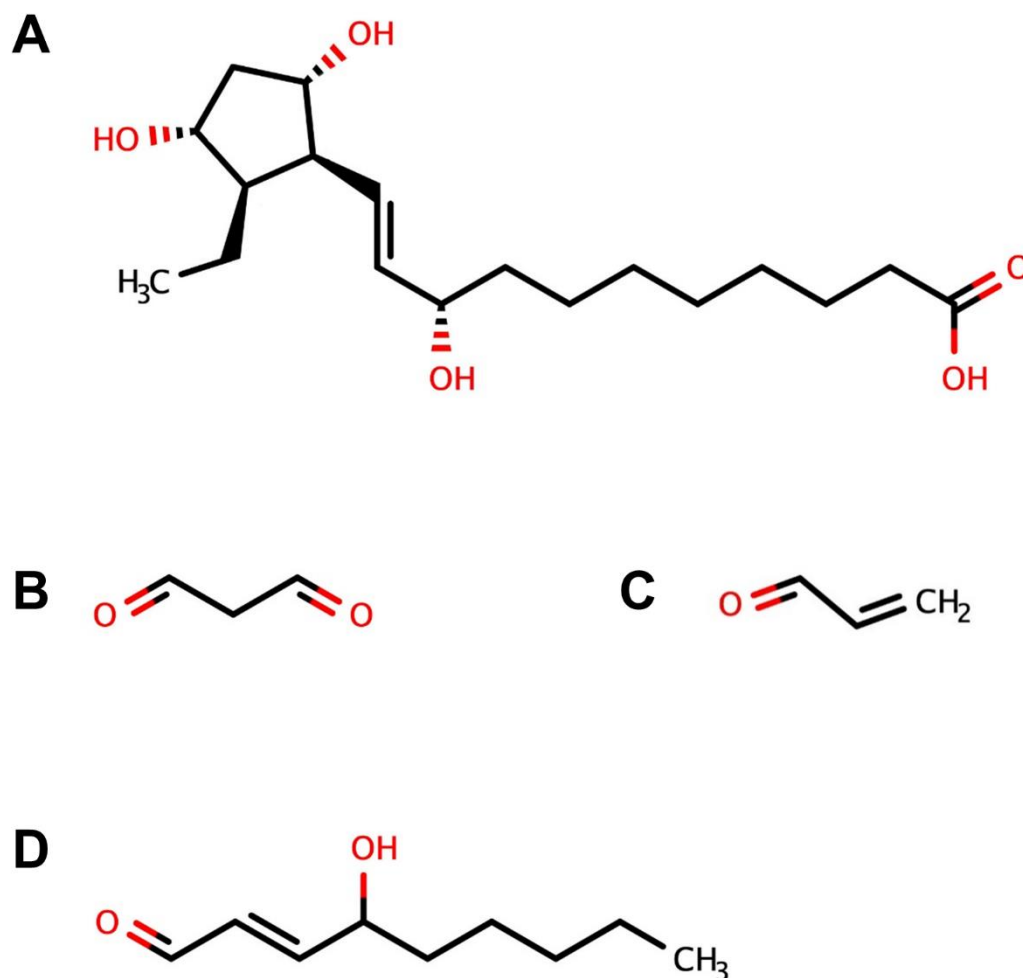
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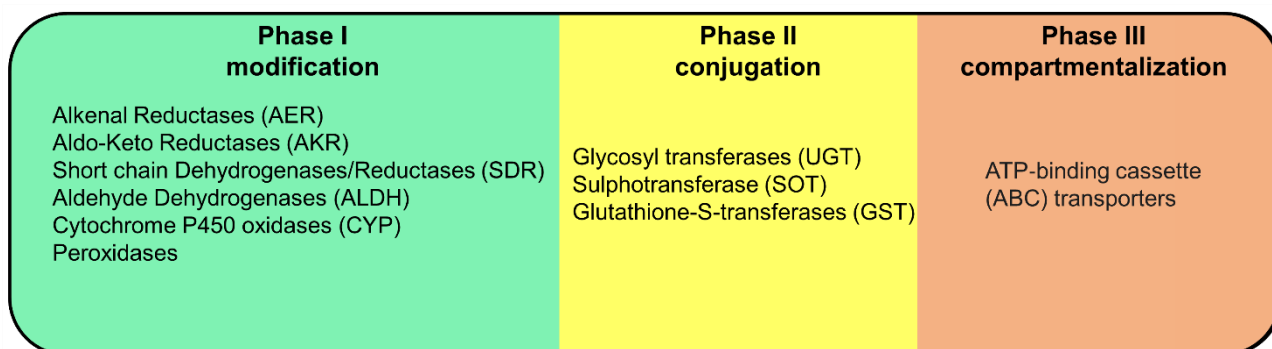
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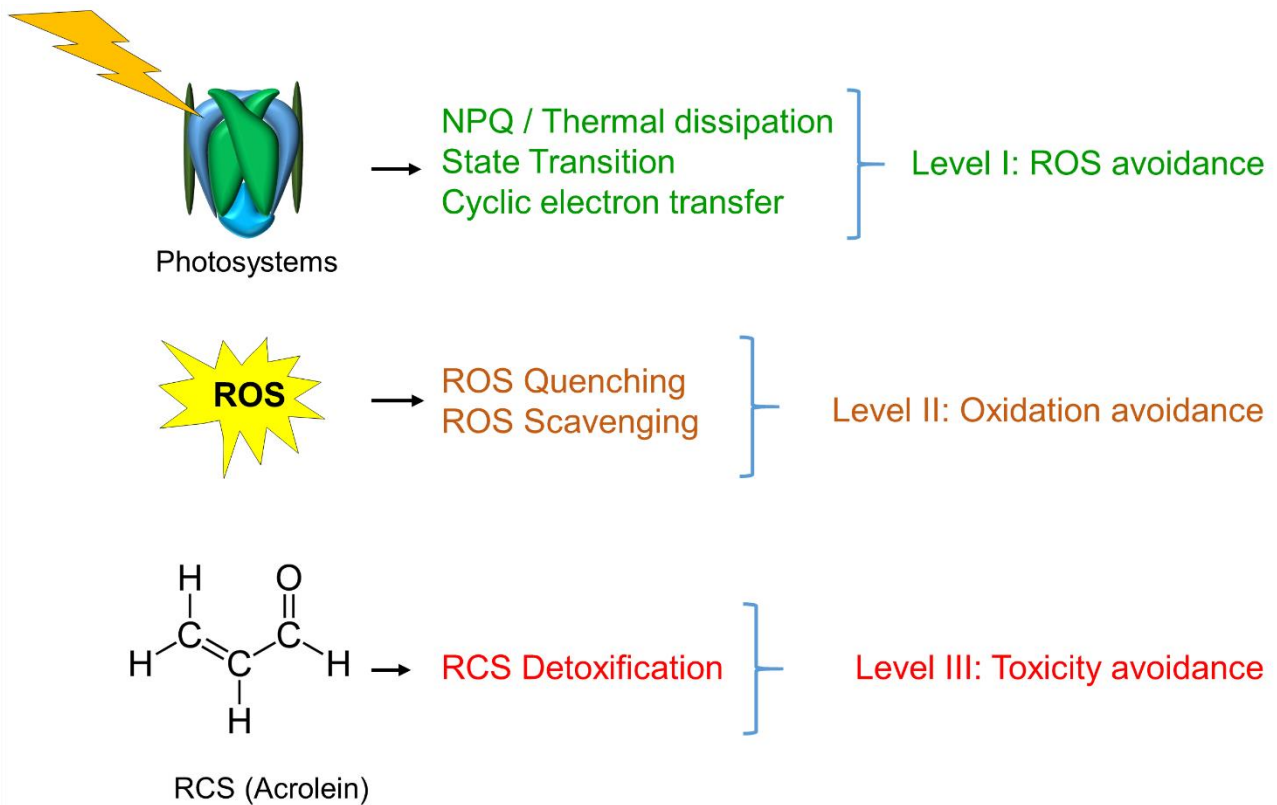
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784 Figure 1. Examples of secondary products generated by lipid peroxidation in plant leaves: A) 9-F1-
785 Phytostane, B) malondialdehyde (MDA), C) acrolein, D) 4-hydroxynonenal (HNE).



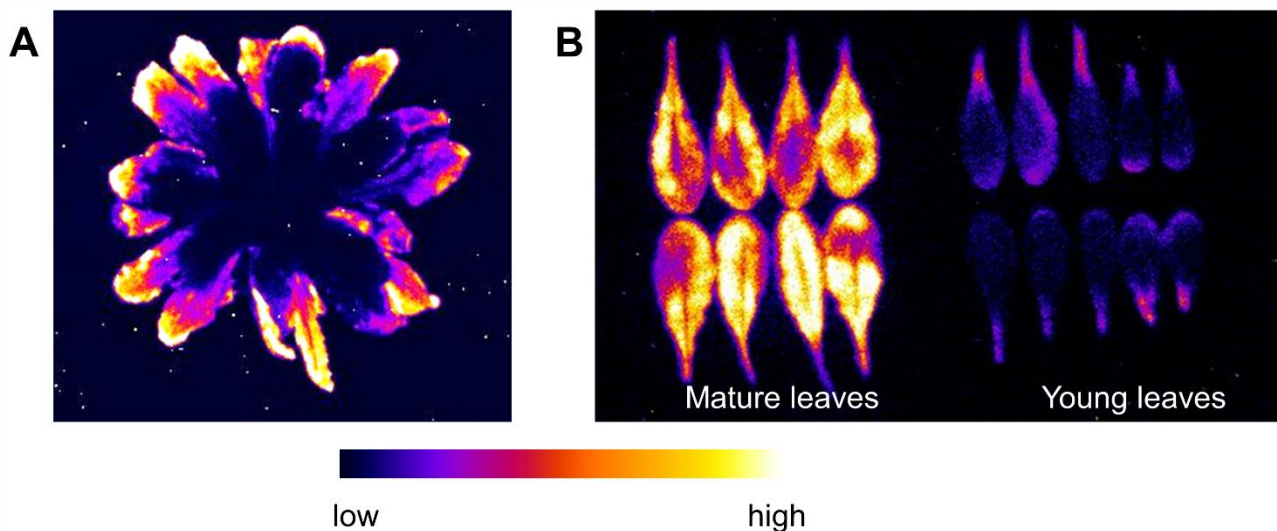
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787 Figure 2. The xenobiotic detoxification response is composed of three phases involving families of
788 detoxifying enzymes.



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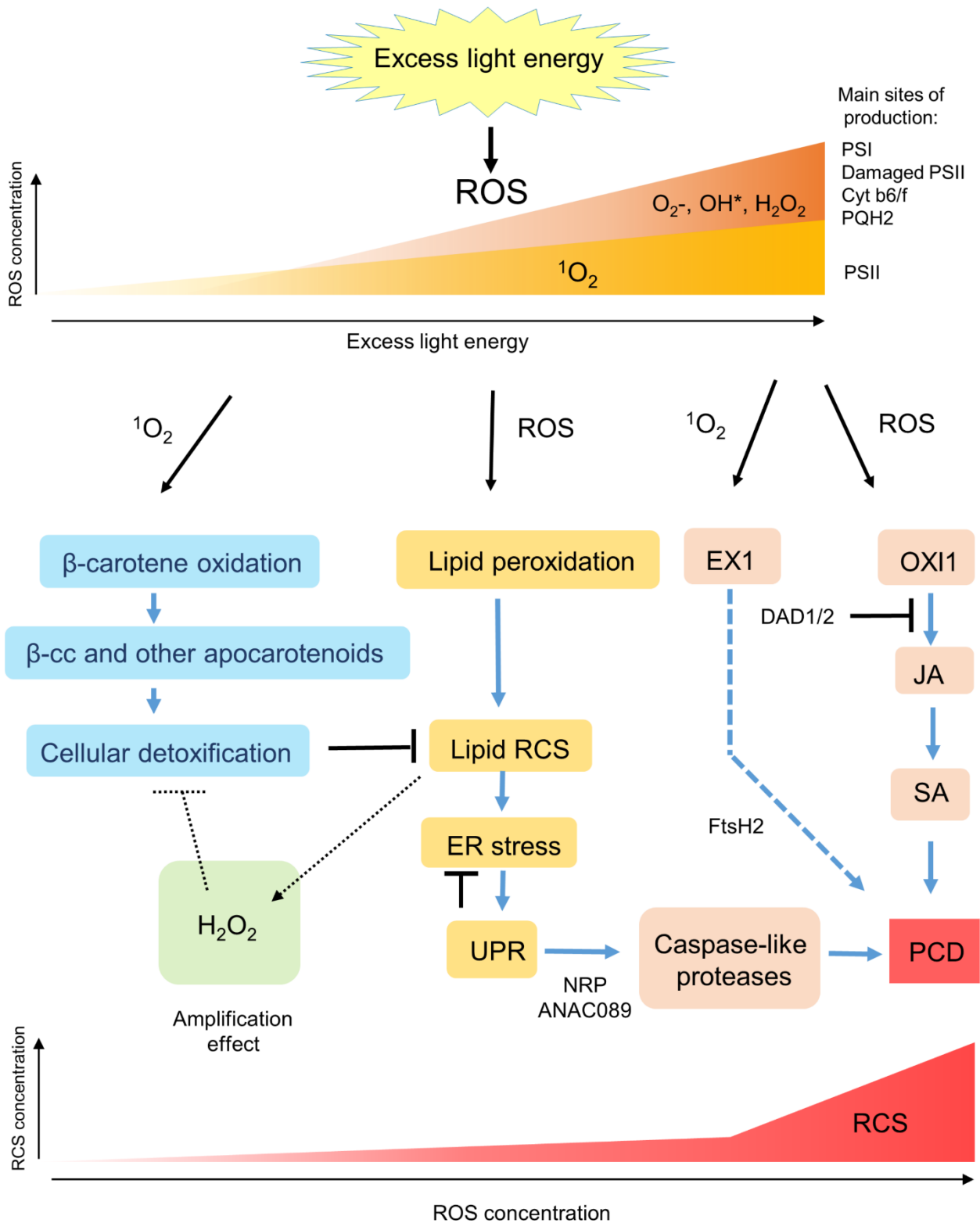
790 Figure 3. Different levels of photoprotective mechanisms are present in plants to sequentially (1)
 791 limit ROS photoproduction, (2) ROS-induced oxidation and (3) toxicity of photooxidation by-
 792 products.



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794 Figure 4. The extent of high light-induced lipid peroxidation is age dependent. A) Autoluminescence
 795 image of lipid peroxidation in an Arabidopsis plant exposed to high light ($1500 \mu\text{mol photons m}^{-2} \text{s}^{-1}$) and low air temperature (7°C), showing that lipid peroxides accumulate in old/mature leaves. B)
 796 Autoluminescence images of detached Arabidopsis leaves exposed to a PFD of $1100 \mu\text{mol photons}$
 797 $\text{m}^{-2} \text{s}^{-1}$ for 16 h in a cold chamber (6°C), confirming that young leaves are more tolerant to
 798

799 photooxidative damage than old leaves. Adapted from D'Alessandro et al. (2018). The color palette
 800 indicates signal intensity from low (blue) to high values (white).



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 802 Figure 5. Scheme of the pathways of excessive light induced PCD in plants. Excessive absorption of
 803 light energy in the chloroplasts brings about ROS production that triggers several signaling pathways
 804 leading to PCD. These pathways involve reactive products derived from lipid peroxidation,

805 phytohormones and PCD-regulatory proteins. The ER plays an important integrative function in the
806 orientation of the stress response towards PCD. Apocarotenoids, such as β -cyclocitral, are upstream
807 signals that can induce cellular detoxification mechanisms modulating the pro-death action of toxic
808 lipid oxidation products.

809