Chapter 28

Chloroplast Photoprotection and the Trade-Off Between Abiotic and Biotic Defense

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Summary

This chapter places two key photoprotective processes in the chloroplast (thermal dissipation of excess excitation energy and removal of reactive oxygen species, ROS, by anti-oxidants) into the context of whole-leaf and whole-plant function in the environment. The emerging evidence for possible trade-offs between effects of altered thermal dissipation and/or anti-oxidation capacity on plant resistance to abiotic stresses (unfavorable physical conditions) versus biotic stresses (pests and pathogens) is summarized. We conclude that more research on this topic is urgently needed, especially for specific crop species and agriculturally relevant environments, including various combinations of multiple abiotic and biotic stresses. As an example of a key redox-signaling pathway impacted by thermal dissipation and/or anti-oxidation capacity, the formation of lipid-peroxidation-derived hormonal messengers of the oxylipin family, with critical roles in plant growth, development, and defenses, is discussed. The available evidence for specific effects of the capacities of thermal dissipation and/or anti-oxidation on sugar loading into foliar phloem conduits, sugar export from leaves, whole-plant growth rate, and plant biotic defenses is reviewed. Lastly, leaf responses to moderate versus massive ROS formation via multiple feedback loops are compared and contrasted.

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I Introduction

The present volume is dedicated to a key aspect of the suite of photoprotective mechanisms (see Logan et al., Chap. 7) employed by photosynthetic organisms to avoid potential damage encountered during the collection and processing of solar energy. This key photoprotective process is the thermal dissipation of excess excitation energy from the singlet-excited state of chlorophyll *a*, assessed as the non-photochemical quenching of chlorophyll fluorescence, operating in conjunction with cellular anti-oxidant networks (see Havaux and García-Plazaola, Chap. 26). Assessing the level of employment of these different photoprotective processes in different species under various environmental conditions provides information about how those species cope with stress and excess light, furthering our understanding of each species' role in the habitats where they thrive. Moreover, a better understanding of the mechanisms of thermal dissipation (and anti-oxidation) promises to provide the tools for identification, breeding, and molecular engineering of crop plants enhanced in various aspect of this component of photoprotection. It is often tacitly assumed that plants with a *greater* capacity for photoprotective thermal dissipation and/

or anti-oxidation would be better protected against losses in productivity caused by unfavorable environmental conditions, thus resulting in plants with *higher yields of food and fuels* under not-always-perfect growing conditions. Do we, however, know this to be the case? Will crops with augmented photoprotection be higher-yielding crops?

The currently available evidence indeed suggests that losses in plant productivity caused by *abiotic* stresses, such as drought or unfavorable (excessively low or high) temperatures or soil mineral content, may be counteracted by augmented photoprotection (for abiotic stress tolerance of, e.g., zeaxanthin-deficient and zeaxanthin-overexpressing mutant lines, see Davison et al. 2002; Du et al. 2010; Gao et al. 2010; Wang et al. 2010; Chen et al. 2011). However, net crop productivity is affected not only by plant biomass yield, but is crucially dependent on limiting the staggering losses (over 50 %) to pests and pathogens as *biotic stresses* (Fletcher et al. 2006). In this chapter, we summarize the emerging evidence that plant resistance to pests and pathogens is affected by factors serving in thermal dissipation and/or anti-oxidation. In fact, it appears from the evidence available to date (as detailed below) that plant biotic stress resistance can be *either lowered or increased* by augmented anti-oxidation, and can be *increased* by a *decrease* in thermal dissipation capacity. Future work in specific crop species and specific environments (pointed out by, e.g., Horton, Chap. 3; Brooks et al., Chap. 13). This future work should also consider differences between defenses against pests versus herbivores as well as defenses against specific biotic agents within each of the two categories.

The call for more research into the potential costs of anti-oxidant overexpression echoes a parallel development in the medical field. For example, Jackson (2008) stated "From the initial view that ROS [reactive oxygen species] were potentially damaging and that prevention of their actions would inevitably be beneficial, we are reaching an understanding that these substances play fundamental roles in metabolism", and Poljsak and Milisav

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Abbreviations: ^{1}Chl* – Singlet-excited chlorophyll; $^1O_2$* – Singlet-excited oxygen; ^{3}Chl* – Triplet-excited chlorophyll; Chl – Chlorophyll; LHC – Light-harvesting complex; NPQ – Non-photochemical quenching of chlorophyll fluorescence; *npq1* – Non-photochemical quenching mutant #1 of *Arabidopsis* deficient in violaxanthin de-epoxidase catalyzing conversion of violaxanthin to zeaxanthin; *npq4* – Non-photochemical quenching mutant #4 of *Arabidopsis* deficient in the photosystem II protein PsbS; $O_2^{\cdot-}$ – Oxygen radical anion (superoxide); OEC – Oxygen-evolving complex; PS II – Photosystem II; PsbS – Photosystem II protein S (member of the stress-associated sub-family of light-harvesting proteins); RC – Reaction center; ROS – Reactive oxygen species; *vte* – Vitamin E-deficient *Arabidopsis* mutant lacking alpha-, beta-, gamma-, and delta-tocopherol as a result of deficiency in the enzyme tocopherol cyclase (*vte1*) or homogentisate phytyltransferase (*vte2*)
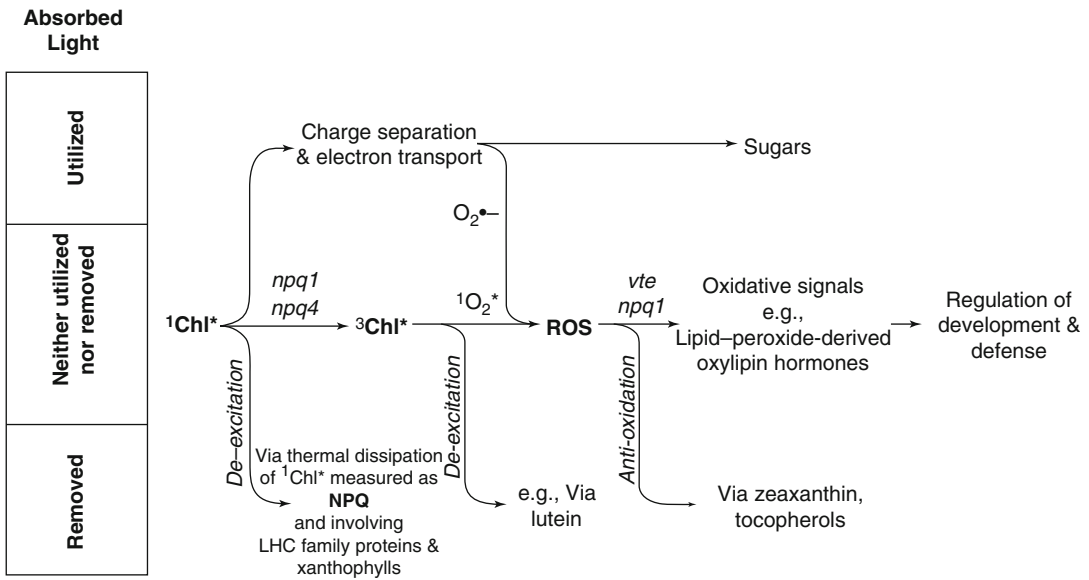
Fig. 28.1 Schematic depiction, placing excitation energy conversion during the primary light reactions of photosynthesis in the context of ROS formation and ROS-triggered signaling with an emphasis on lipid-peroxide-derived oxylipin hormones. $^1Chl^*$: singlet-excited chlorophyll, $^3Chl^*$: triplet-excited chlorophyll, $^1O_2^*$: singlet-excited oxygen, $O_2^{\cdot-}$: oxygen radical anion (superoxide), *LHC*: light-harvesting complex, *NPQ*: non-photochemical quenching of chlorophyll fluorescence from singlet-excited chlorophyll *a*, *npq1 Arabidopsis*: mutant deficient in violaxanthin de-epoxidase catalyzing conversion of violaxanthin to zeaxanthin, *npq4 Arabidopsis*: mutant deficient in the photosystem II protein PsbS, *ROS*: reactive oxygen species, *vte Arabidopsis*: mutants lacking alpha-, beta-, gamma-, and delta-tocopherol as a result of deficiency in either the enzyme tocopherol cyclase (*vte1*) or homogentisate phytyltransferase (*vte2*).

(2012) elaborated that "Oxidative stress is not necessarily an un-wanted situation, since its consequences are beneficial for many physiological reactions in cells. On the other hand, there are potentially harmful effects of 'anti-oxidative stress'". Furthermore, Villanueva and Kross (2012) stated that, "the normal balance between antioxidants and free radicals in the body is offset when either of these forces prevails… In summary, a hypothesis is presented that 'antioxidant-induced stress' results when antioxidants overwhelm the body's free radicals."

II Integration of Photoprotection into Whole-Plant Functioning

Strong evidence has accumulated that (i) foliar levels of reactive oxygen species (ROS), and multiple signaling networks responding to cellular redox state (as the balance between oxidants and anti-oxidants), contribute chiefly to the regulation of plant growth and development as well as the orchestration of abiotic and biotic plant defenses (for a review, see, e.g., Munné-Bosch et al. 2013), and that (ii) chloroplast ROS level and redox state is modulated by thermal energy dissipation and anti-oxidant level (Ledford and Niyogi 2005).

Figure 28.1 places thermal dissipation and anti-oxidation into the context of oxidative signaling networks regulating plant growth and development as well as plant defenses against pathogens and herbivores. When exposed to light levels that are limiting to photosynthesis, plants use as much of the light they absorb as possible in the photochemical pathway leading (via charge separation and photosynthetic electron transport) to sugar production. Plants grown in environments with fluctuating light levels harmlessly dissipate excess excitation energy (light absorbed in

excess of what can be utilized photochemically) via photoprotective thermal dissipation of excessive excitation energy, which represents a non-photochemical route leading to non-photochemical quenching (NPQ) of chlorophyll fluorescence (Fig. 28.1). Excess singlet-excited chlorophyll a (^{1}Chl*) is de-excited via thermal dissipation (involving, as summarized in the present volume, various xanthophyll pigments and proteins of the family of light-harvesting proteins; Fig. 28.1; see also, e.g., Büchel, Chap. 11; Brooks et al., Chap. 13; Morosinotto and Bassi, Chap. 14). Remaining ^{1}Chl* (not dissipated via the combination of photochemical and non-photochemical pathways) can convert to triplet-excited chlorophyll (^{3}Chl*) that readily passes its excitation energy to ground-state oxygen (Fig. 28.1). The NPQ-deficient *Arabidopsis* mutants *npq1* (lacking violaxanthin de-epoxidase that catalyzes the conversion of violaxanthin to zeaxanthin; see, e.g., Demmig-Adams et al., Chap. 24) and *npq4* (lacking the PsbS protein; see Brooks et al., Chap. 13), both deficient in NPQ (and thermal dissipation), are likely to form elevated levels of ^{3}Chl* under excess light. While light-harvesting proteins possess potent quenchers of ^{3}Chl* (see, e.g., Dall'Osto et al. 2006), any remaining ^{3}Chl* will transfer its energy on to oxygen, forming highly reactive singlet oxygen ^{1}O$_2$* (Fig. 28.1). Tocopherols (vitamin E) as well as zeaxanthin are potent anti-oxidant quenchers of ROS like ^{1}O$_2$* (see Havaux and García-Plazaola, Chap. 26; concerning the terminology of vitamin E versus tocopherols, vitamins are specifically defined as compounds not synthesized in the organism in which they have essential functions, which does not apply to photosynthetic organisms proficient in the synthesis of compounds serving as vitamins in non-photosynthetic organisms. We will, therefore, refer mainly to tocopherols with occasional reference to the fact that these compounds also serve as vitamin E).

Any remaining ROS (not removed by anti-oxidants) can give rise to oxidative signals – for example, lipid-peroxidation-derived oxylipin hormones like jasmonic acid (Fig. 28.2; see also section III below) that regulate plant growth and development as well as plant biotic defense (see also Brooks et al., Chap. 13). It should be noted that ROS-dependent oxylipin production is but one of a multitude of signaling pathways controlling plant function via redox-based signals (Munné-Bosch et al. 2013). For reviews of other redox signaling networks, see Baginsky and Link (2006), Dietz et al. (2006), Foyer et al. (2006), and Mullineaux et al. (2006).

III Lipid-Peroxidation-Derived Hormones as an Example for Redox Modulation of Plant Form and Function

Tocopherol-deficient plants exhibit defects in growth and development (for a review, see Falk and Munné-Bosch 2010), in particular impaired phloem loading, decreased carbohydrate export from leaves, enhanced foliar starch accumulation (Russin et al. 1996; Botha et al. 2000; Provencher et al. 2001; Sattler et al. 2003, 2004, 2006; Hofius et al. 2004; Maeda et al. 2006, 2008), and premature senescence (Abbasi et al. 2009). Falk and Munné-Bosch (2010) pointed out that these *impairments* in the growth of tocopherol-deficient plants are most obvious when the corresponding wild type plants exhibit a *high sink demand* (i.e., high carbohydrate consumption by the plant's growing or carbohydrate-storing sinks); they stated that "the sucrose export phenotype is more clearly observed in tocopherol-deficient plants in species with a higher sink demand, such as potato or maize, and also in other species when grown at low temperature, such as *Arabidosis thaliana*." Our own work has shown that *Arabidopsis* wild type increases the number of phloem cells in its leaf veins when grown under cool versus warm temperature, which likely contributes to a maintenance of carbon export flux and elevated levels of photosynthesis despite increased phloem sap viscosity at lower temperature (Cohu et al. 2013a,b).
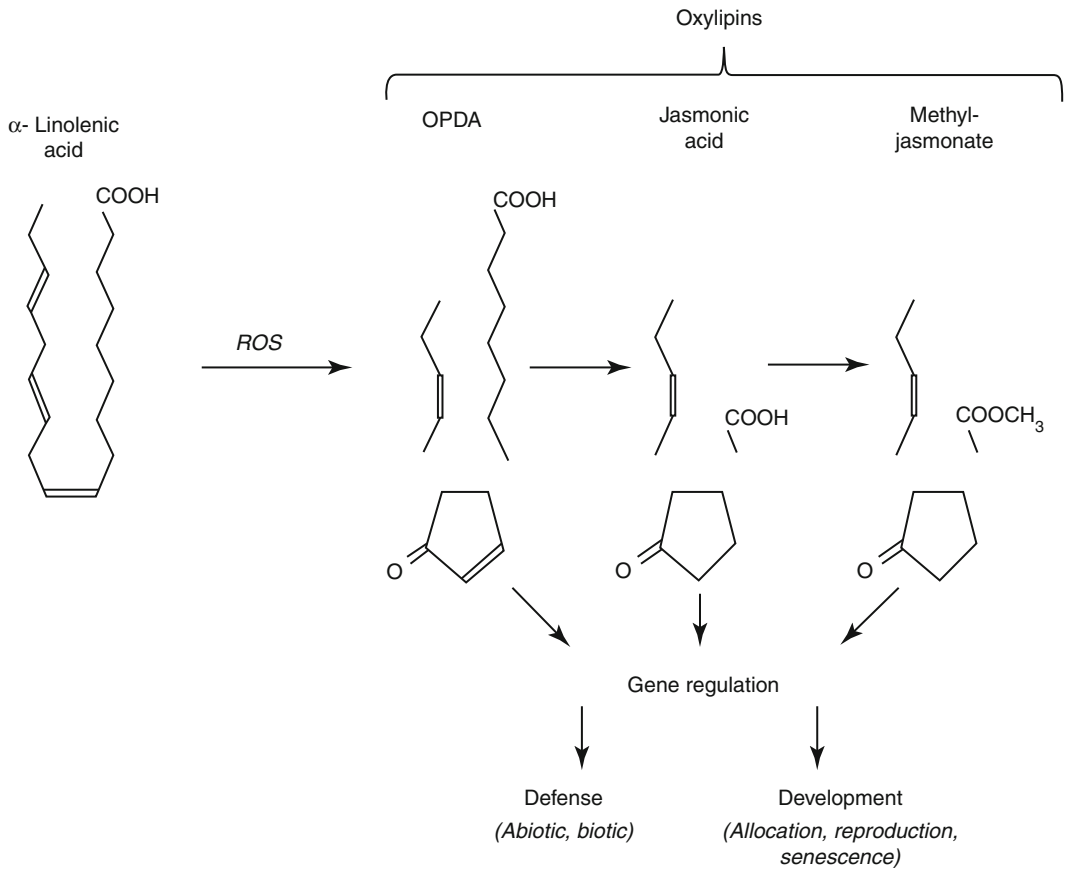
Fig. 28.2 Features of the biosynthesis of selected oxylipins with gene regulatory functions in *Arabidopsis.* *ROS* reactive oxygen species, *OPDA* 12-oxo-phytodienoic acid. Modified after Devoto and Turner (2005) and Demmig-Adams et al. (2013).

The mechanism of growth inhibition in tocopherol-deficient plants apparently involves increased levels of ROS, lipid-peroxidation-derived hormonal messengers (such as jasmonic acid and related oxylipins), and deposition of callose in cells involved in phloem loading (in the leaf's sugar-loading veins; Amiard et al. 2007). Callose formation has been shown to be associated with lipid peroxidation (Yamamoto et al. 2001), and a closing of phloem-loading routes via callose deposition occurs in plants infected by fungi (Koh et al. 2012), presumably to prevent the spread of the pathogen (Zavaliev et al. 2011). It has been shown that fungi and other pathogens utilize the plant's phloem conduits to spread themselves throughout the plant (Gilbertson and Lucas 1996; Waigman et al. 2004; Scholthof 2005). While the *benefit* of an ROS-triggered shutdown of phloem conduits may lie in a greater pathogen resistance, the *cost* of this protection would be the plant's diminished ability to export sugars from the photosynthesizing leaves for plant growth. Conversely, upregulation of foliar anti-oxidant levels, and a corresponding suppression of lipid-peroxidation-derived (and other oxidative) signals, has the potential *benefit* of preventing obstruction of phloem conduits (allowing unimpaired carbon export from source leaves to sinks), while potentially simultaneously rendering plants *more vulnerable* to succumbing to pathogen attack.

Characterized feature	Wild Type	Tocopherol ("Vitamin E")-deficient (*vte*)	PsbS-deficient (*npq4*)	Zeaxanthin-deficient (*npq1* or *npq1 lut2*)
ROS level	+ (1)	++ (2)	++ (1)	(unknown)
Oxylipin level (e.g., jasmonic acid)	+ (3)	++ (5)	++ (3)	++ (4)
Defense against herbivores	+ (3)	(unknown/ complex)	++ (3)	(unknown)
Callose deposition in phloem / putative pathogen defense	+ (4)	++ (6)	(unknown)	++ (4)

Fig. 28.3 Summary of the available evidence for a role of the anti-oxidant tocopherol, the photosystem II protein PsbS, and the xanthophyll zeaxanthin in modulating ROS and oxylipin levels as well as in plant biotic defenses. (*1*) Jänkänpää et al. 2013, (*2*) Havaux et al. 2005; Sattler et al. 2006; Semchuk et al. 2009, (*3*) Frenkel et al. 2009, (*4*) Amiard et al. 2007; Demmig-Adams et al. 2013, (*5*) Munné-Bosch et al. 2007, (*6*) Botha et al. 2000; Maeda et al. 2006. Both *npq1* and *npq1 lut2* exhibited a trend (albeit not significant for *npq1*) for increased cell wall ingrowths/reinforcements. The double mutant *npq1 lut2* exhibited even more pronounced cell wall ingrowths than *npq1* and yielded significant differences versus wild type. Both *npq1* and *npq1 lut2* were transferred from low to moderate (double mutant) or high light (*npq1*) to trigger cell wall ingrowths. The PsbS-deficient *npq4* mutant exhibited significant differences in oxylipin levels under herbivore attack in the field but not in the absence of herbivores. *npq1*, *Arabidopsis* mutant deficient in violaxanthin de-epoxidase catalyzing conversion of violaxanthin to zeaxanthin (this mutant was used to demonstrate elevated oxylipin levels in plants transferred from low growth light to high light; Demmig-Adams et al. 2013); *npq1 lut2*; *Arabidopsis* mutant deficient in both zeaxanthin (deficient in violaxanthin de-epoxidase catalyzing conversion of violaxanthin to zeaxanthin) and lutein (this mutant was used to demonstrate elevated cell wall ingrowths in phloem cells in plants transferred from low growth light to moderate light; Demmig-Adams et al. 2013); *npq4*, *Arabidopsis* mutant deficient in the photosystem II protein PsbS; *ROS* reactive oxygen species, *vte*, *Arabidopsis* mutant deficient in tocopherol synthesis and lacking alpha-, beta-, gamma-, and delta-tocopherol as a result of deficiency in either the tocopherol cyclase (*vte1*) or homogentisate phytyltransferase (*vte2*).

We note that a multitude of oxylipins is formed by either non-enzymatic or enzymatic lipid peroxidation, with both processes being modulated by ROS and anti-oxidants. Enzymatic lipid peroxidation occurs via lipoxygenases (LOX) with a catalytic iron center; ROS oxidize LOX from the inactive form LOX-Fe^{2+} to the LOX-Fe^{3+} form active in lipid peroxidation (Maccarrone et al. 1996), while anti-oxidants, such as tocopherols reduce LOX from the LOX-Fe^{3+} active form to the inactive form LOX-Fe^{2+} (Maccarrone et al. 1999). Products of both enzymatic and non-enzymatic lipid peroxidation serve as gene regulators in many organisms (see, e.g., Demmig-Adams and Adams 2010; Demmig-Adams et al. 2013).

Figure 28.3 summarizes the available evidence on altered redox state, altered oxidative messenger production, and altered biotic defenses in plants deficient in tocopherols (as key anti-oxidants: see, e.g., Havaux and García-Plazaola, Chap. 26), the PsbS protein (as a PS II protein and member of the light-stress-associated sub-family of light-harvesting proteins: see, e.g., Brooks et al., Chap. 13; Morosinottto and Bassi, Chap. 14), or the xanthophyll zeaxanthin (as a close correlate of thermal dissipation in
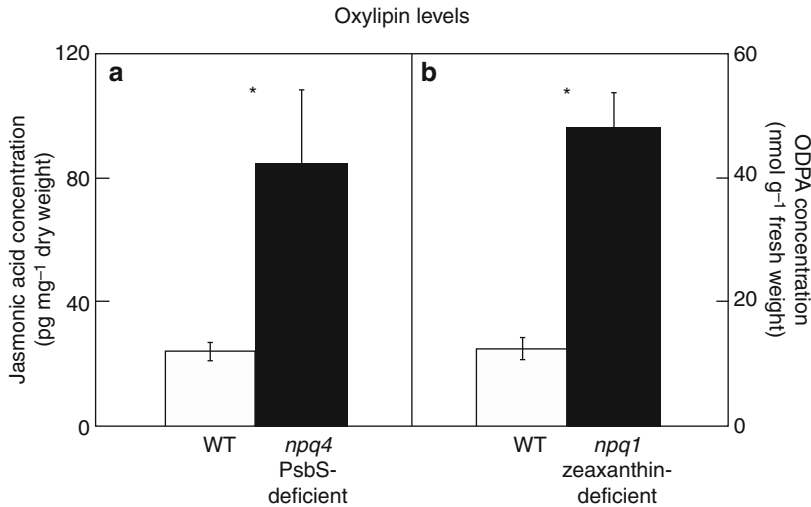
Oxylipin levels

Fig. 28.4 Augmented foliar oxylipin concentrations in *Arabidopsis* mutants deficient in either of two components of the photoprotective system. (**a**) Difference in jasmonic acid concentration between wildtype *Arabidopsis* (*WT*) and the PsbS-deficient *npq4* mutant under outdoors/field conditions in the presence of herbivores (Modified after Demmig-Adams et al. 2013; based on original data from Frenkel et al. 2009). (**b**) Difference in the concentration of the jasmonic acid precursor OPDA (12-oxo-phytodienoic acid) between WT *Arabidopsis* and the zeaxanthin-deficient *npq1* mutant upon transfer from low growth light to high light (modified after Demmig-Adams et al. 2013). *, significantly different at $p < 0.05$.

plants grown in nature; see, e.g., Demmig-Adams et al., Chap. 24). Here we summarize the evidence showing *increased ROS* levels, *increased oxylipin* levels, and *increased defenses* against certain pests or pathogens, respectively, in plants deficient in tocopherols (Fig. 28.3; Vitamin E-deficient, *vte*), PsbS (Figs. 28.3, 28.4a, and 28.5a; PsbS-deficient, *npq4*), or zeaxanthin (Figs. 28.3, 28.4b, and 28.5b; Zeaxanthin-deficient, *npq1* or *npq1 lut2*). Increased callose deposition in sugar-loading phloem complexes in tocopherol-deficient plants and in plants deficient in zeaxanthin and lutein has been observed (Fig. 28.5b). Furthermore, PsbS-deficient plants growing under highly variable light conditions in the field exhibit *decreased* sensitivity to a specific herbivore (Fig. 28.5a; see also Brooks et al., Chap. 13). To date, no information is available on the susceptibility of tocopherol-deficient plants to herbivore attack; future research is needed to address the possibility that the latter plants may have augmented herbivore defense, as seen in

PsbS-deficient plants. However, it is clear that the defenses against multiple pests and pathogens involve multiple defense pathways that are regulated by different signaling pathways, and can have *opposite* effects (Derksen et al. 2013). For example, resistance of a range of pepper varieties against the major pepper insect pest flower thrips was *positively* correlated with foliar tocopherol content, with insect development apparently being inhibited by foliar tocopherol (Maharijaya et al. 2012). Future research is thus urgently needed to address the role of components of the photoprotection network in plant defense to *multiple* different biotic challenges (and, e.g., pests versus pathogens) as well as to *combinations* of biotic and abiotic stresses under actual field conditions (see also, e.g., Horton, Chap. 3; Brooks et al., Chap. 13). The possibility that trade-offs may exist between defenses against herbivores versus pathogens, or against any two specific biotic agents, also needs to be considered.
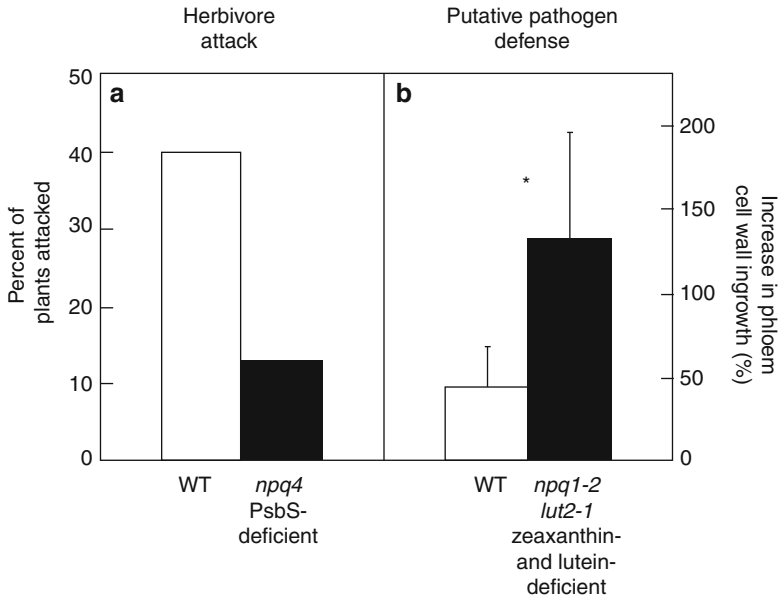
Fig. 28.5 (**a**) Augmented defense against herbivores in the PsbS-deficient *npq4* mutant and (**b**) augmented cell-wall deposition in phloem-loading complexes (as a putative defense against pathogens) in the zeaxanthin- and lutein-deficient double mutant *npq1-2 lut2*. The percent of plants attacked by herbivores was compared in wild type *Arabidopsis* (*WT*) and the PsbS-deficient *npq4* mutant under outdoors/field conditions (Modified after Demmig-Adams et al. 2013; based on original data from Frenkel et al. 2009); WT and the zeaxanthin- and lutein-deficient double mutant *npq1-2 lut2-1* were compared upon transfer from low growth light to high light (modified after Demmig-Adams et al. 2013). *, significantly different at p < 0.05.

IV Feedback Loops Between Photoprotection and Whole-Plant Function Under Moderately Versus Highly Excessive Light

Redox signaling networks include multiple feedback loops between primary photosynthetic reactions on the one hand, and plant growth, development and defenses on the other hand (Figs. 28.6 and 28.7). The overall outcome of the action of these multiple feedback loops apparently varies depending on how much excessive light is absorbed by a given leaf. Two contrasting scenarios are envisioned in Fig. 28.6 (*moderate* ROS formation) and Fig. 28.7 (*massive* ROS formation). Figure 28.6 depicts the putative situation in a leaf consuming/dissipating *most* of the absorbed light via the combination of photochemistry and thermal dissipation (NPQ), but also forming ROS levels sufficient to trigger production of redox signals

that, in turn, fully induce synthesis of factors (i) involved in thermal dissipation (NPQ) and anti-oxidation as well as (ii) biotic defenses. It has, for example, been reported that jasmonic acid triggers de-novo synthesis of the anti-oxidant tocopherols (Sandorf and Holländer-Czytko 2002) and ascorbic acid (Wolucka et al. 2005) as well as of carotenoids (Pérez et al. 1993).

Figure 28.7 depicts a putative situation for a leaf experiencing *massive* ROS formation. Continuing massive ROS formation and, e.g., leaf death (for an overview of senescence and death, see Biswal et al. 2013) can theoretically be prevented by a *shutdown* of the sources of ROS production. Two major forms of ROS produced in the primary light reactions are electronically excited singlet oxygen, formed in light-harvesting complexes (LHCs), and singly reduced superoxide, formed with electrons ultimately stemming from charge separation
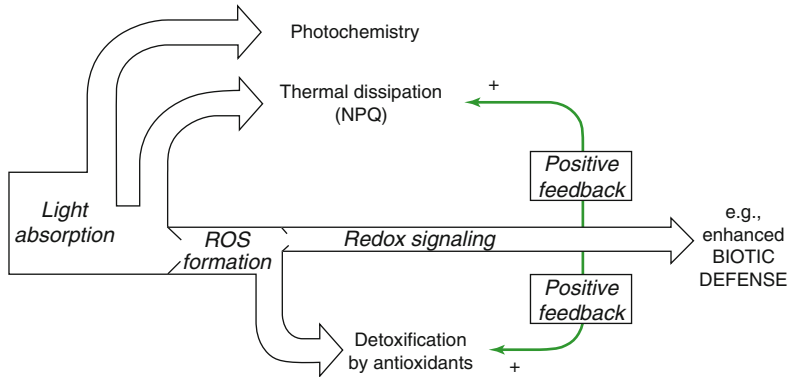
Fig. 28.6 Schematic depiction of the link between light absorption, formation of reactive oxygen species (*ROS*), redox signaling, and plant responses like biotic defense under moderate ROS formation as well as (positive) feedbacks (*green arrows*) between redox signals and (i) anti-oxidation or (ii) thermal energy dissipation in the chloroplast (leading to non-photochemical quenching of chlorophyll fluorescence, NPQ).
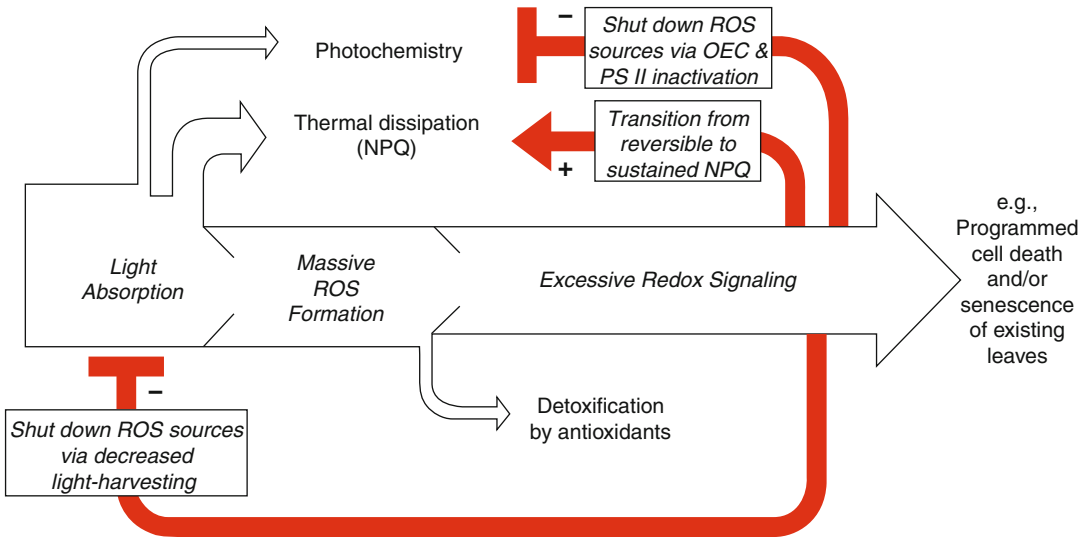

Fig. 28.7 Schematic depiction of the links among light absorption, formation of reactive oxygen species (*ROS*), redox signaling, and plant responses under massive ROS formation as well as feedbacks (*red arrow* and *red* feedback loops) between redox signals and shut-down of ROS sources via (i) inactivation of the oxygen-evolving complex and photosystem II (*PS II*) photochemistry and/or (ii) decreased light harvesting as well as (iii) transition from reversible to sustained non-photochemical quenching of chlorophyll fluorescence (*NPQ*).

in the oxygen-evolving complex (OEC) and photosystem II reaction center (PS II-RC). Consequently, degradation of LHCs and inactivation and/or degradation of OEC and PS II-RC would theoretically serve to *shut-down* ROS formation (Fig. 28.7). The inactivation and/or net degradation of PS II-RC under highly excessive light indeed involves an inhibition of de-novo synthesis of the D1 protein of the PS II-RC by ROS (e.g., Murata et al. 2007). Future research is needed to address the possibility that inactivation/ degradation of PS II-RC and/or OEC does serve to shut down massive ROS formation as proposed in Fig. 28.7 – while simultaneously shutting down photosynthesis (see

also, e.g., Murchie and Harbinson, Chap. 25; Morales et al., Chap. 27). Degradation of PS II-RCs and OEC, and photosynthetic shutdown ("photoinhibition"), in overwintering evergreens is associated with maintenance of high Chl (and LHC) levels, and a transition from rapidly reversible NPQ to a sustained form of NPQ locked-in for an entire season during which plant growth is arrested (Adams et al. 1995, 2002, 2004, 2006; Zarter et al. 2006a,b,c).

Adams et al. 2013 (see also Adams et al., Chap. 23) have reviewed the evidence indicating that photoinhibition (as a long-term inactivation of primary photochemistry and photosynthesis) in evergreens, as well as in deciduous perennials, is associated with foliar carbohydrate accumulation, and presumably with either sink limitation or limiting sugar export from leaves. Conversely, species other than evergreens, exposed to mineral stress, typically exhibit pronounced degradation of LHCs and elimination of excess light-harvesting capacity, which is expected to lower ROS formation without requiring sustained NPQ or OEC/PS II-RC shutdown/degradation (see Morales et al., Chap. 27).

Placement of PS II/OEC inactivation into a whole-organism context also begs for re-assessment of the assignment of such inactivation as "damage", similar to the re-evaluation efforts currently occurring in the medical field,

> Reactive oxygen species (ROS) and chronic changes in membrane lipids are not the result of accidental damage. Instead, these changes are the result of a highly evolved, stereotyped, and protein-catalyzed 'oxidative shielding' response that all eukaryotes adopt when placed in a chemically or microbially hostile environment… Research efforts need to be redirected. Oxidative shielding is protective and is a misguided target for therapy…An alternative title for this review might be, 'Oxidative stress or oxidative shielding: can 50 years of research be wrong?' (Naviaux 2012).

It is important to elucidate whether ROS-triggered responses represent actual damage or a form of prevention, favored by natural selection, of actual damage. Furthermore, accelerated death of pre-existing leaves may actually hasten redeployment of resources towards growth of new leaves better adjusted to altered environmental conditions. Lastly, what is favorable for plant fitness in natural settings may not necessarily be favorable in agricultural settings, and differences among crop species or varieties in the degree of photosynthetic shutdown in response to environmental stress might be exploited in such settings.

V Conclusions

As illustrated by many examples in plant ecology, *trade-offs* between the benefits and costs of plant adaptations make many specific strategies beneficial only in certain environments but not in others. This principle needs to be further applied to the case of plant photoprotection via thermal dissipation of excess excitation energy and/or anti-oxidation. While plant resistance against losses in productivity due to abiotic stresses may possibly be improved by augmented thermal dissipation/anti-oxidation in certain cases, plant resistance to pests and pathogens can apparently be either *lowered* or *increased* by augmented thermal dissipation/anti-oxidation. Future work is urgently needed to address these questions in specific crop species and specific agriculturally relevant environments, including various combinations of abiotic and biotic stresses as well as specific pests and herbivores.

While the benefit of a shutdown of (sugar-exporting) foliar phloem conduits triggered by reactive oxygen species may lie in a greater resistance to, e.g., specific pathogens, the cost to the plant may be a diminished ability to export sugars from photosynthesizing leaves to the plant's sinks for plant growth (and/or sugar and starch accumulation in fruits, roots, or tubers). We need to address the question of whether upregulation of foliar anti-oxidant levels and of the capacity for thermal dissipation, as well as putative corresponding suppression of oxidative

signals, has the potential benefit of preventing any obstruction of phloem conduits, while possibly simultaneously coming with the cost of rendering plants more vulnerable to pathogen attack.

We suggest that research be done to further elucidate specific feedback loops between redox signals and gene regulation of the biosynthesis of factors involved in thermal dissipation and/or anti-oxidation. The possibility also needs to be addressed that inactivation/degradation of PS II reaction centers and the oxygen-evolving complex serves to shut down massive ROS formation under highly excessive light conditions. It should, furthermore, be considered that naturally occurring differences among plant species and varieties/ecotypes in maximal NPQ capacity, antioxidant levels, and lipid peroxidation levels have evolved as a result of trade-offs between the needs for specific abiotic and biotic defenses in specific environments.

Acknowledgments

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