Thiol-Based Peroxidases and Ascorbate Peroxidases: Why Plants Rely on Multiple Peroxidase Systems in the Photosynthesizing Chloroplast?

Karl-Josef Dietz*

Photosynthesis is a highly robust process allowing for rapid adjustment to changing environmental conditions. The efficient acclimation depends on balanced redox metabolism and control of reactive oxygen species release which triggers signaling cascades and potentially detrimental oxidation reactions. Thiol peroxidases of the peroxiredoxin and glutathione peroxidase type, and ascorbate peroxidases are the main peroxide detoxifying enzymes of the chloroplast. They use different electron donors and are linked to distinct redox networks. In addition, the peroxiredoxins serve functions in redox regulation and retrograde signaling. The complexity of plastid peroxidases is discussed in context of suborganellar localization, substrate preference, metabolic coupling, protein abundance, activity regulation, interactions, signaling functions, and the conditional requirement for high antioxidant capacity. Thus the review provides an opinion on the advantage of linking detoxification of peroxides to different enzymatic systems and implementing mechanisms for their inactivation to enforce signal propagation within and from the chloroplast.

INTRODUCTION

Photosynthesis depends on dynamic input parameters. In particular photon flux density may change in fractions of seconds. Many other parameters like CO2-availability at the site of carbon fixation, temperature, local effects of pathogens and nutrient supply can fluctuate on the time scale of minutes to days and strongly affect photosynthesis. Photosynthesis is most efficiently regulated to avoid damage despite these variations (Allahverdiyeva et al., 2015). The robustness is realized by regulatory networks which exploit information from systems performance and adjust activities of critical processes. Reactive oxygen species and redox regulation by thiol switches play a major role in the control of photosynthesis (Dietz and Hell, 2015). In this context, the question arises as to why chloroplasts rely on two types of peroxidases, the thiol-based and ascorbate-dependent peroxidases.

SUPEROXIDE AND HYDROGEN PEROXIDE GENERATION IN PHOTOSYNTHESIS

The photosynthetic electron transport chain has two main mechanisms to produce superoxide anions. (i) Reduced plastocyanin donates electrons to O2 via the plastid terminal oxidase (PTOX) (Heyno et al., 2009). Thus the regulatory circuitry controlling the redox state of the intersystem electron transport carrier plastocyanine tends to adjust an intermediately reduced level of reduced to oxidized plastocyanine to minimize ROS generation. (ii) (Over-)reduced photosystem I (PSI) as consequence of an imbalance between light driven electron delivery and electron acceptor availability such as NADP+, nitrite or sulfite transfers electrons from ferredoxin or the Fe-S-center Fx to O2. This O2-reduction reaction is called the Mehler reaction. The magnitude of the Mehler reaction is discussed in a controversial manner ranging from rather low rates (Laisk et al., 2006) to high rates reaching 30% of total electron flow (Badger et al., 2000). A comparison of 101 species for their capacity of O2-dependent electron drainage from the photosynthetic electron transport revealed species-specific differences, e.g. between gymnosperms and angiosperms (Shirao et al., 2013). While O2-dependent electron flow ranged around 3% in diverse gymnosperms, the rates dropped to about 1% or less in angiosperms such as tobacco (Shirao et al., 2013). These authors also elaborated on the question whether PSI- and PTOX-dependent O2-reduction can experimentally be distinguished in wild type plants. Based on the similar affinity to O2 they argued that only the combined O2-reduction can be measured. The rate of the Mehler reaction changes in dependence on environmental conditions such as light, temperature, nutritional status, and restrictions in CO2-supply, e.g. following stress-induced stomatal closure. Surprisingly, Ruuska et al. (2000) observed no change in light-dependent O2 uptake in transgenic A. thaliana
with only 10% residual ribulose-1,5-bisphosphate carboxylase/oxygenase levels suggesting efficient and tight control even under conditions of metabolic imbalance. Chloroplasts contain Fe-dependent and Cu/Zn-dependent superoxide dismutases (FeSOD1-3, CuZnSOD2 in A. thaliana, Mittler et al., 2004) which catalyze the conversion of superoxide to hydrogen peroxide by the dismutation reaction \( 2 \text{O}_2^- + 2 \text{H}^+ \rightarrow \text{H}_2\text{O}_2 + \text{O}_2 \). Thus the Mehler reaction and PTOX-dependent generation of superoxide and dismutation are considered as major sources of \( \text{H}_2\text{O}_2 \) in chloroplasts. Lipid peroxidation occurs if unsaturated fatty acids react with singlet oxygen or superoxide, or by enzymatic catalysis (Farmer and Mueller, 2013). Lipid peroxides initiate radical chain reactions and are degraded to reactive electrophilic species (RES). RES react with macromolecules, alter gene expression and may cause cell damage and thus need to be tightly controlled.

**ASCORBATE PEROXIDASES**

Chloroplasts of A. thaliana contain two ascorbate peroxidases (APX), a thylakoid-bound tAPX and a soluble sAPX. APXs belong to the group of heme peroxidases that reduce \( \text{H}_2\text{O}_2 \) with ascorbate as electron donor (Fig. 1). In the absence of ascorbate nanomolar concentrations of \( \text{H}_2\text{O}_2 \) inactivate tAPX. This occurs with a rate constant of \( 7 \times 10^9 \text{ M}^{-1} \text{s}^{-1} \) in the presence of 2 mM \( \text{H}_2\text{O}_2 \) (Miyake and Asada, 1996). The inactivation occurs if the cosubstrate ascorbate does not convert the reactive intermediate state \( \text{Fe}^{IV} \cdot \) to the safe \( \text{Fe}^{III} \)-state of the heme (Fig. 1). Regular stromal ascorbate concentrations are in the range of 10 mM, thus the inactivation of tAPX and sAPX can only occur under severe stress conditions such as heavy metal toxicity or severe drought (Liu et al., 2008; Kitajima, 2008). Two reasons are discussed to explain the susceptibility of chloroplast APX to \( \text{H}_2\text{O}_2 \)-dependent inactivation, the increased turnover rate of plastid APX by adopting more reactive intermediates or the need of inactivation for signaling purposes (Kitajima, 2008). Since electrons are extracted from \( \text{H}_2\text{O} \) in the water splitting apparatus of photosystem II and finally transferred to \( \text{O}_2 \) in the Mehler reaction, the whole pathway has been termed water-water cycle (Asada, 1999). A conclusive simulation model has been assembled to describe the activity and functional consequences of the ascorbate-dependent water-water cycle in plants (Polle, 2001). This paper provides a survey of reported concentrations of enzymes, activities and substrates of the water water cycle. APX was given with a total concentration of 70 µM, a \( K_m \) (Asc) of 300 µM and the \( K_m \) (H2O2) of 30 µM (Nakano and Asada, 1987). The estimated local concentration of tAPX at the thylakoid membrane is 1 mM (Miyake and Asada, 1996). The mathematical model supports the view that the ascorbate and glutathione pools of the chloroplasts can be uncoupled under in vivo conditions (Polle, 2001).
**THIOL PEROXIDASES**

Thiol peroxidases are heme-free enzymes that use a cysteiny1 thiolate to attack the peroxide substrates. Any protein that reacts with peroxides via a cysteiny1 thiol tentatively might be called thiol peroxidase, e.g. annexins (Dalal et al., 2014) or glutathione-S-transferases (Dixon et al., 2009). However two groups of enzymes, the peroxiredoxins (Prx) and glutathione peroxidases (Gpx), should be considered as thiol peroxidases in sensu stricto due to their high affinity to peroxides. Prx-s were first described as protector proteins and thiol antioxidants in mixed function oxidation assays where DNA and glutamine synthase are degraded in the presence of Fe, thiols and O2 (Kim et al., 1988). Prx-s suppress the degradation process. Higher plants express a set of usually 9 to 10 Prx-s, three to four of which are targeted to plastids (Dietz et al., 2006). The plastid peroxiredoxins belong to the groups of classical 2-cysteine peroxiredoxins (2-CysPrx), peroxiredoxin Q (PrxQ) and the type II peroxiredoxin (PrxIIIE). Following reaction with peroxide substrate, oxidized 2-CysPrx receives its electrons from NADPH via NADPH-dependent thioredoxin reductase C (NTRC) or from thioredoxins like Trx-x which likely is linked to ferredoxin (Fd) via Fd-dependent thioredoxin reductase (FTR) (Collin et al., 2003; Pulido et al., 2010) (Fig. 1). Work with A.thaliana mutants lacking either NTRC or Trx-x suggests that the NTRC-pathway is predominant for regeneration of reduced 2-CysPrx (Pulido et al., 2010). If coupled to Trx-dependent regeneration 2-CysPrx equally accepts H2O2 and small peroxides such as tBOOH, but poorly reacts with lipid peroxides (König et al., 2003). The latter was confirmed by Award et al. (2015) using dithiothreitol as artificial regenerant. However in this artificial system, tBOOH reduction only reached about 30% of the rate of H2O2-reduction (Award et al., 2015). 2-CysPrx adopts an additional function as chaperone (Jang et al., 2004). Site-directed mutation of specific amino acids has revealed that chaperone activity of 2-CysPrx does not exclusively depend on the hyperoxidized conformation (König et al., 2013; Lee et al., 2015). PrxIIIE prefers H2O2 >4-fold over H2O2 (Treffon and Dietz, 2015). PrxIIIE prefers H2O2 >4-fold over tBOOH and has negligible rates with cumene hydroperoxide (Treffon and Dietz, unpublished). Oxidized PrxIIIE accepts electrons from glutathione/glutaredoxin-C5-system (Couturier et al., 2011). The preferred substrate of PrxQ is H2O2. Oxidized PrxQ is regener-ated by Trx-y (Lamkemeier et al., 2006). A small set of glutathione peroxidases (Gpx) is targeted to chloroplasts and functions as Trx-dependent peroxide reductases which hardly react with GSH (Navrot et al., 2006; Zhai et al., 2013). Gpx isoforms prefer lipid peroxides as substrates with usually >10-fold higher catalytic efficiency towards complex lipid peroxides such as phosphatidylcholine hydroperoxide as compared to H2O2 (Esh- dat et al., 1997; Matamoros et al., 2015).

**CONSEQUENCES OF APX, PRX AND GPX DEFICIENCY**

Rice and Arabidopsis plants devoid of chloroplast APX display no altered phenotype under normal growth conditions with constant light, but their development is strongly inhibited in the presence of methylviologen and at low temperature, thus under photoinhibitory conditions (Caverzan et al., 2014; Kangasjärvi et al., 2008). Compensatory upregulation of 2-CysPrx and cytosolic APX may allow for long term high light acclimation of sapa/tapx-plants (Kangasjärvi et al., 2008). Deficiency of 2-cysteine peroxiredoxin in antisense A. thaliana delays early seedling development with impairment of photosynthesis (Baier and Dietz, 1999; Baier et al., 2000). Growth is inhibited in antisense and T-DNA insertion lines and levels of H2O2 and carbonylated proteins increase significantly (Awad et al., 2015; Pulido et al., 2010). Photosynthesis of plants lacking 2-CysPrxA and B (2cpa 2cpb), and in particular in 2cpa 2cpb tapx-plants which in addition lack thylakoid APX showed decreased quantum yield of photosystem II, and lower CO2 fixation rates. Most important-ly, H2O2-responsive nuclear genes were unchanged in 2cpa 2cpb and only massively accumulated in 2cpa 2cpb tapx-plants (Award et al., 2015). The lack of NTRC, the predominant reductant of 2-CysPrx, caused a smaller increase in protein carbonylation of about 36% of that in 2-cysprx and 64% increase in H2O2 (Pulido et al., 2010). Depletion of Gpx1 and Gpx7 from Arabidopsis slightly altered leaf phenotype, compromised acclimation to excess light by increased photoinhibition and H2O2.

![Fig. 2. Overview of peroxidases in chloroplasts](http://molcells.org)
development, but interestingly has no effect on malondialdehyde (MDA) accumulation (Chang et al., 2009). MDA is a degradation product of lipid peroxidation. Thus other mechanisms appear to counteract the lack of Gpx-dependent lipid peroxide detoxification capacity and possibly are linked to up-regulated levels of ascorbate, glutathione and salicylic acid (Chang et al., 2009).

THE APX, PRX AND GPX NETWORK

Starting from this biochemical and physiological knowledge one can address the question as to why chloroplasts express so many peroxidases? The answer to this question apart from redundancy must consider eight characteristics and processes, namely (i) suborganellar localization, (ii) substrate preference, (iii) metabolic coupling, (iv) abundance, (v) regulation, (vi) interactions, (vii) signaling functions and (viii) the temporal and spatial requirement for high antioxidant capacity (Fig. 2).

Suborganellar localization

tAPX and PrxQ are associated with the thylakoids. It still needs to be resolved whether PrxQ associates with the grana stacks from the stromal side, the thylakoid lumen or both (Lamkemeyer et al., 2006; Petersson et al., 2006). 2-Cys Prx adopts different conformations in dependence on redox state and experimental evidence suggests that certain 2-CysPrx conformation states associate with thylakoids and photosystem II (König et al., 2003; Muthuramalingam et al., 2009), however these interactions may be short-lived since Cerveau et al. (2016) could not detect major portions of 2-CysPrx at intensively washed thylakoids and also found no correlation with overoxidation state. PrxlIE, sAPX, and Gpx are reported as stromal proteins. It is concluded that part of the peroxidase diversity is due to distinct suborganellar association.

Substrate preference

Only a limited number of peroxide substrates have been tested with the Prx, Apx and Gpx isoforms. The exclusive specificity of APX for H2O2, the strong preference of Gpx for lipid peroxides and the mixed but variable substrate specificity of the Prx-s indicate distinct peroxide detoxification or sensing function.

Metabolic coupling

The electron donor for APX and the reductants of oxidized Prx and Gpx link peroxide detoxification to different metabolic pathways. Coupling of GSH/ascorbate to APX, NADPH/NTRC and FTR/Tnx-y/z/CDSP to 2-CysPrx, PrxQ and also presumably Gpx, and GSH/Grx (as well as presumably FTR/Tnx) to PrxlIE diversifies electron drainage and generates robustness in detoxification. Importantly NTRC, but also likely Tnx-x, Tnx-y and other involved redox transmitters, serve multiple functions in redox regulation. An example is the role of NTRC in regulating photochemical activities of the photosynthetic electron transport chain (Naranjo et al., 2016). Thus diversion of electrons into ROS detoxification via thiol peroxidases may cause spatial oxidation of other target proteins which then alter their function (König et al., 2013).

Protein abundance

In vivo electron fluxes in the network depend on affinities, rate constants and concentrations of involved players. The abundance of the here discussed participants can be obtained from the At_Chloro database (Ferro et al., 2010) and has recently been assembled (König et al., 2012). The relative scores were described as follows: 2-Cys PRX A (100 relative units, roughly corresponding to concentration in µM): PrxlIE (24): PrxQ (8.6): Gpx1 (3.4): sAPX (4.4): tAPX (2.6). Among the redox interactors is Tnx-m4 (11 relative units) highly abundant while Tnx-x is present with only 0.24 relative units (König et al., 2012). The response of the network to any specific environmental challenge must take into account these distinct abundances. But in addition conditional changes in the relative abundances by quantitative proteomics need to be explored in future work.

Regulation of activity

It was discussed above that Apx is prone to inactivation in the absence of reduced ascorbate. Likewise, 2-CysPrx is sensitive to hyperoxidation. The peroxidatic Cys which reacts with the peroxide substrate and, thereby is converted to the sulfenic acid derivative within the catalytic cycle, occasionally converts to sulfenic acid. About 250 peroxide reduction reactions have been determined as average catalytic cycles prior to inactivation by hyperoxidation (Liebthal et al., 2016). Thus the rate of peroxide turnover and rereduction of the hyperoxidized form by sulfiredoxin (Liu et al., 2006) determine the active fraction of 2-CysPrx. Interestingly, the fraction of hyperoxidized 2-CysPrx did not strongly change under environmental stress (Cerveau et al., 2016). PrxlIE is regulated by S-nitrosylation and glutathionylation (Romero-Puertas et al., 2007; Treffon and Dietz, unpublished). Similar data are missing for chloroplast Gpx and PrxQ. But reports from other systems and non-plastidic isoforms suggest that posttranslational modifications also affect the other chloroplast thiol peroxidases (Cha et al., 2012; Seo et al., 2009). These mechanisms likely allow for conditional, spatial and species-specific control of ROS also in chloroplasts.

Interactions with other proteins

Bacterial, yeast and human Prx and Gpx interact with specific protein partners like receptors and signaling components (Bertoldi, 2016; Flohé, 2015). Plant 2-CysPrx interacts with multiple redox transmitters, but also other proteins such as fructose-1,6-bisphosphatase and cyclophilin 20-3 (Caporaletti et al., 2007;
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Liebthal et al., 2016; Muthuramalingam et al., 2009). There is a need for the comprehensive search for interaction partners of the various peroxidases, in particular the thiol peroxidases, in order to understand their role in cell signalling. The promiscuity of 2-CysPrx in interacting with thiol proteins like the AACTH-proteins (Dangoor et al., 2009) and chloroplastic drought-induced stress protein of 32 kD (CDSP32) (Broin et al., 2002) should be considered in both directions; within the catalytic cycle the reduction of oxidized 2-CysPrx enables repeated detoxification of peroxides. In the redox sensory pathway, the oxidation of the thiol protein in dependence on the peroxide concentration may feed information into e.g. the AACTH pathway and affect cell signalling.

Signaling functions
Transcript analyses revealed specific alterations of gene expression upon deletion of 2-CysPrx, PrxQ or sAPx1/sAPx2 (Baier and Dietz, 1999; Kangasjärvi et al., 2008; Lamkemeyer et al., 2006). This suggests that each peroxidase is involved in a specific and at least partly unique metabolic and signalling context. Transcriptome profiling of all mutants in a single experiment is needed to ultimately address the distinct and overlapping signalling role. Only a single transcript was significantly up-regulated in the tapx sapx-plants under control conditions (Kangasjärvi et al., 2008). Apparently, the mutant is able to acclimate under constant growth conditions and to compensate for the deletion. Thus transient and fluctuating environmental conditions, respectively, are needed to challenge the mutants and to pinpoint to the molecular and physiological consequences of the specific defects.

Conditional requirement for specific antioxidant capacity
Both tapx sapx- and 2cysprx-plants revealed chlorotic phenotype and growth inhibition during early seedling development (Baier et al., 1999; Kangasjärvi et al., 2008). Chloroplast APX activity dropped to very low levels at 2 to 3 d after radicle emergence (Pena-Ahumada et al., 2006). The authors concluded that the ascorbate-dependent water-water cycle only develops following a metabolic switch at 3 d after radicle emergence whereas Prx-s might be important during very early development (Pena-Ahumada et al., 2006). Similar versus dissimilar regulation can be deduced from transcriptome data sets by applying coexpression analysis tools (e.g. at ATTED-II, Obayashi et al., 2009). Figure 3 depicts the Mutual Rank (MR) among the pairwise comparison for coexpression following sorting from the strongest to the weakest co-expression regulation. Low numbers indicate high degree of coexpression. It can be seen that there is significant coregulation between 2-CysPrxA, 2-CysPrxB and PrxQ. Some coexpression is observed between Prx and Apx, but most other pairs of peroxidases reveal negligible coexpression in transcript regulation. The result shows that the peroxidases are specifically regulated in dependence on environmental and developmental cues. In summary the available evidence supports the eight hypotheses explaining why plants rely on multiple peroxidase systems in the photosynthesizing chloroplast.

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