Peroxisomes as redox-signaling nodes in intracellular communication and stress responses

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Abstract

Redox compartmentalization in organelles is an effective evolutionary strategy (Box 1; Jones and Go, 2010). From an evolutionary perspective, peroxisomes, originating from the endoplasmic reticulum (ER), were selected to house a range of metabolic pathways involving the production of certain reactive oxygen species (ROS) such as H2O2 to avoid toxicity to other organelles such as mitochondria (Gabalón, 2018). Peroxisomes play a diverse range of roles in cell functionality and in the perception of and responses to changes in their environment (Sandalio and Romero-Puertas, 2015; Lismont et al., 2019). The range of functions associated with plant peroxisomes has increased considerably over the last two decades (Table 1). As most of these pathways produce ROS and nitric oxide (NO), disturbances in these metabolic processes trigger transitory changes in ROS/reactive nitrogen species (RNS) production. These changes regulate peroxisomal metabolism, leading to peroxisome-dependent signaling and organelle crosstalk, which triggers specific cell responses (Sandalio and Romero-Puertas, 2015). The biosynthesis of phytohormones jasmonic acid (JA), auxin IAA, and salicylic acid (SA) associated with the β-oxidation pathway contributes to the complex role of peroxisomes in development and stress responses (Kao et al., 2018; Figure 2A). Peroxisomes dynamically regulate their number, shape, and size.

ADVANCES

- Peroxosomal H2O2 regulates pathogen associated processes, DNA repair systems, cell cycles and phytohormone-dependent signalling.
- Peroxisomes regulate cellular processes in the cytosol and other cell compartments through moonlighting proteins such as CAT3, which is able to transnitrosylate and degrade GSNOR via autophagy.
- Peroxisomes are highly dynamic organelles that are capable of changing their number, size, morphology and speed in response to environmental redox changes.
- Peroxules are ROS- and NO-induced dynamic structures that are regulated by PEX11a, which connects peroxisomes to chloroplasts, mitochondria, ER and lipid bodies.
- Under basal and stress conditions, peroxisomal populations and quality are regulated by selective autophagy (pexophagy) which is controlled by ROS and the peroxosomal protease LON2.
protein content in response to changing environmental conditions and remain in close contact with other subcellular compartments such as mitochondria and chloroplasts (Sandalio and Romero-Puertas, 2015; Shai et al., 2016; Sandalio et al., 2020). Peroxisomes play a key role in the evolution of the metabolic networks of photosynthetic organisms by connecting oxidative and biosynthetic pathways operating in different compartments. This review updates our knowledge of peroxisomal redox homeostasis and the role of ROS and NO in the functionality, biogenesis and abundance of these organelles, as well as their role as redox hubs in metabolic regulation, signaling, and organelle crosstalk.

Peroxisomes are ROS and NO producers

**Peroxisomes produce and scavenge ROS**

ROS include an array of molecular oxygen derivatives that occur as a normal attribute of aerobic life (Figure 1). Peroxisomes are one of the main sources of cellular ROS production and one of the most oxidized cellular organelles (Smirnoff and Arnaud, 2019). However, peroxisomes have a complex antioxidant system to balance ROS levels, enabling them to strictly regulate organelle functionality, metabolism, and signaling networks. The first step in $O_2$ univale $O_2^-$ (Figure 1), is produced in ureide and nucleic acid catabolism by xanthine oxidoreductase (XOR) and urate oxidase or uricase (UO) reaction (Werner and Witte, 2011; Sandalio et al., 2013; Figure 2A); in the sulfite oxidation by sulfite oxidase (SO; Byrne et al., 2009); and in the NADH/NADPH-dependent electron transport chain in the peroxisomal membrane. Superoxide accumulation is regulated by different superoxide dismutases (SOD; reviewed in Sandalio and Romero-Puertas, 2015; Figure 2B).

In photosynthetic tissue, peroxisomes accumulate the highest concentrations of organelle $H_2O_2$ (the second step in $O_2$ reduction) with a flux of $\sim 10,000 \text{ nmol}^{-2} \text{ m}^{-2} \text{ s}^{-1}$ (Foyer and Noctor, 2003). The use of $H_2O_2$ ratiometric reporter HyperAs targeting peroxisomes has facilitated the imaging of changes in peroxisomal $H_2O_2$ accumulation in response to Cd treatment (Calero-Muñoz et al., 2019) and the increase in intraperoxisomal Ca$^{2+}$ levels (Costa et al., 2010). The main source of $H_2O_2$ in peroxisomes in green tissue is GOX in the photorepiration cycle (Figure 2A), which contributes up to 70% of total $H_2O_2$ production in plant cells (Reumann and Weber, 2006; Foyer et al., 2009). Photorespiration requires coordination of the chloroplast, peroxisome, mitochondrion, and cytosol; and photorespiration-dependent $H_2O_2$ production increases considerably under environmental stress conditions such as heat and drought (Talbi et al., 2015; Walker et al., 2016), heavy metal (Gupta et al., 2017), high light (Cui et al., 2016a), and biotic stress (Rojas et al., 2012; Hodges et al., 2016; Yang et al., 2018). Fatty acid β-oxidation, another source of $H_2O_2$ in peroxisomes by the Acyl-CoA oxidase (ACX), provides energy during the initial stage of seedling growth by oxidizing fats stored as triacylglycerol (TAG) in oil bodies (Rinaldi et al., 2016; Figure 2A). Other β-oxidation pathways are active in green tissues, including the synthesis of ubiquinone, hormones such as indole acetic acid (IAA) and JA, and secondary metabolites such as benzoic acid (BA) and phenylpropanoids (reviewed in Pan et al., 2020; Figure 2A). Polyamine catabolism and sarcosine oxidase are additional peroxisomal sources of $H_2O_2$ (Figure 2A; Goyer et al., 2004; Wang et al., 2019). Peroxosomal $H_2O_2$ levels are regulated by balancing $H_2O_2$ generation and scavenging rates (Figure 2B) by catalase (CAT), which account for 10%–25% of total peroxisomal proteins (Reumann et al., 2004; Figure 2B). Arabidopsis (Arabidopsis thaliana) plants contain three CAT genes, CAT1, CAT2, and CAT3, with CAT2 being the most important defense against

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**Table 1 Plant peroxisome functions**

- ROS and RNS metabolism
- $H_2O_2$ and NO signaling
- Photosynthesis
- Phytohormones biosynthesis (JA, IAA, SA)
- Fatty acid β-oxidation
- Glycolate cycle
- Polyamine catabolism
- Amino acids metabolism
- Indole glucosinolates metabolism
- Ureide metabolism
- Purine catabolism
- Biotin biosynthesis
- Ubiquinone biosynthesis
- Phyloquinone biosynthesis
- Isoprenoids biosynthesis
- BA derive biosynthesis
- Sulfite metabolism

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**Figure 1** Sequential reduction of $O_2$ and ROS production: superoxide ($O_2^-\cdot\cdot$), hydrogen peroxide ($H_2O_2$), and hydroxyl (OH) radicals.
Figure 2  Oxygen and nitrogen reactive species metabolism in peroxisomes. A, Principal peroxisomal metabolic pathways associated with peroxisomal ROS and NO production. ROS are produced in metabolic pathways such as \( \beta \)-oxidation, photorespiration, ureides metabolism, and polyamine oxidation, and in a small electron transport chain associated with the membrane (peroxisomal membrane proteins, PMP18 and PMP29; Figure 2B). NO is produced in peroxisomes by NOS-like (NOS-I) activity, although other sources, such as XOR, polyamine oxidation, and IBA metabolism, could also be involved. ROS, NO, and other RNS may leak out of the peroxisome (dashed arrows) and act as signal molecules that regulate cell metabolism and gene expression. B, Scheme of peroxisomal antioxidant defenses, RNS scavengers, and NAD(P)H supply. O\(_2^–\) is regulated by SODs, while H\(_2\)O\(_2\) is controlled by CAT, the ASC-GSH cycle, and Gpx. Peroxynitrite (ONOO\(^–\)) and GSNO are produced in peroxisomes by reaction of NO with O\(_2^–\) and glutathione (GSH), respectively. GSNO can negatively regulate MDHAR and CAT through S-nitrosylation and nitration, and SOD may be regulated by nitration. SOD may indirectly control ONOO\(^–\) by regulating O\(_2^–\) levels. Uric acid acts as an ONOO\(^–\) scavenger. NAD(P)H is supplied by the oxidative pentose phosphate pathway (G6PD; 6PGD), ICDH, MDH, and NUDIX19. 6PGD, 6 phosphogluconate dehydrogenase; AAT, amino acid translocator; AOC, allene oxide cyclase; AOS, allene oxide synthase; APX, ascorbate peroxidase; BADH, betaine aldehyde dehydrogenase; CAT, catalase; CuAO, copper amine oxidase1; DHAR, dehydroascorbate peroxidase; GOX1,2, glycolate oxidase1,2; G6PD, glucose-6-phosphate dehydrogenase; GGT, glutamate–glyoxylate aminotransferase; GlyT, glycerate–glycolate translocator; GR, glutathione reductase; Gpx, glutathione peroxidase; H-Acyl-CoA, 3-hydroxyacyl-CoA; HPR, hydroxypyruvate reductase; IAA, indole-3-acetic acid; IBA, indole-3-butyric acid; IB3, acyl-CoA dehydrogenase/oxidase-like IB3; ICDH, isocitrate dehydrogenase; KAT, L-3-ketoacyl-CoA-thiolase; LOX, lipoxygenase; MDH2, malate dehydrogenase; MDHAR, monodehydroascorbate peroxidase; MFP, multifunctional protein; OPCL1, OPCL-80 CoA ligase1; NOS-1, NO synthase-like; NUDIX19, nudix hydrolase homolog 19; OP3, OPDA reductase3; PAO3, polyamine oxidase3; PAO3/4, polyamine oxidase 3/4; PNC, peroxisomal ATP carrier; PXA1, peroxisomal ABC-transporter1; PXN, peroxisomal NAD carrier; SGT, serin–glyoxylate aminotransferase; UOX, urate oxidase.
photorespiratory $\text{H}_2\text{O}_2$ accounting for 80% of activity (reviewed in Mhamdi et al., 2012). In fact, physical GOX-CAT interactions regulated by SA occur in rice leaves (Zhang et al., 2016). A protective association between CAT and isocitrate lyase has also been observed in castor bean glyoxysomes (Yanik and Donaldson, 2005) and CAT2 also interacts with ACX2/ACX3 regulating their activity and therefore the SA-mediated regulation of JA biosynthesis, under biotrophic infection (Yuan et al., 2017). Although the extraordinarily low affinity of CAT for $\text{H}_2\text{O}_2$, with a Km of around 43 mM, reduces its efficiency in controlling $\text{H}_2\text{O}_2$ the abundance of CAT compensates for this low affinity (Foyer and Noctor, 2016). The peroxisomal ascorbate–glutathione cycle, which, in Arabidopsis, composed of ascorbate peroxidase (APX3 and APX5), monodehydroascorbate reductase (MDHAR1), dehydroascorbate reductase (DHAR1), and glutathione reductase (GR); reviewed in Mhamdi et al., 2012; Sandalio and Romero-Puertas 2015; Pan et al., 2020; Figure 2B) also contribute to $\text{H}_2\text{O}_2$ homeostasis. MDHAR and APX are associated with the peroxisomal membrane and the higher affinity for $\text{H}_2\text{O}_2$ of APX (100 μM) as compared to CAT, could regulate $\text{H}_2\text{O}_2$ leakage from peroxisomes to the cytosol (Del Río et al., 2003; Kaur et al., 2009; Eastmond, 2007; Figure 2B). Therefore, CAT and APX are positioned to enable $\text{H}_2\text{O}_2$ to act as a second messenger. Glutathione $S$-transferases support peroxide regulation in these organelles (Pan and Hu, 2018). The ascorbate–glutathione cycle also facilitates regeneration and maintenance of the peroxisomal redox buffers ASC/DHA and GSH/GSSG. The use of ratiometric glutathione redox potential reporters, such as roGFP2, targeting peroxisomes has facilitated the imaging of peroxisome oxidation under extended dark stress and the application of elicitors (Bratt et al., 2016).

**Peroxisomal NO/RNS production and scavenging**

Although NO is a well-known signaling molecule in plants, its metabolism has not been fully elucidated (León and Broseta, 2020). Peroxisomal NO production has been associated with a NO synthase-like activity (NOS-I; Figure 2A; Barroso et al., 1999), the conversion of IBA to IAA by β-oxidation (Figure 2A; Schlicht et al., 2013), polyamine catabolism (Figure 2A; Wimalasekera et al., 2011; Agural et al., 2018), and the XOR reaction (Figure 2A; Antonenkov et al., 2010; Wang et al., 2010). Other nitrogen-derived species, such as peroxynitrite (ONOO$^-\text{}$), resulting from the $\text{O}_2^-/\text{NO}$ reaction, and nitrosoglutathione (GSNO), resulting from the combination of NO and GSH and considered a cellular NO reservoir, have been detected in peroxisomes (Figure 2A; Ortega-Galisteo et al., 2012; Corpas and Barroso, 2014). Peroxisomal SOD could regulate ONOO$^-\text{}$ accumulation by controlling $\text{O}_2^-\text{'}$ availability and CAT could degrade it, as reported in animal cells (Gebicka and Didik, 2009), and thus play a key modulatory role at the cross-point between $\text{H}_2\text{O}_2$ and NO/ONOO$^-\text{'}$-mediated signaling pathways (Figure 2B). Urate, a well-known peroxynitrite scavenger (Hooper et al., 2000; Alamillo and García-Olmedo, 2001), may contribute also to regulate ONOO$^-\text{'}$ in peroxisomes (Figure 2B).

S-nitrosoglutathione reductase (GSNOR), which balances NO and S-nitrosothiol levels, has been proteomically identified in plant peroxisomes (reviewed in Sandalio et al., 2019), although this requires validation.

**NADH/NADPH regeneration in peroxisomes**

The concept of redox stress (oxidative and reductive) reflected by changes in NAD(H)/NADP(H) has gained increasing attention. The NAD(P)H cofactor is required to β-oxidation and antioxidative defenses MDHAR and GR. NAD(P)H regeneration in peroxisome take place by the oxidative pentose phosphate pathway (OPPP; Corpas et al., 1999; Reumann et al., 2007; Lansing and Doering, 2020; Figure 2B), NADP-dependent isocitrate dehydrogenase (ICDH; Corpas et al., 1999; Reumann et al., 2007; Figure 2B), NADH phosphorylation by NADH kinase 3 (NADK3; Waller et al., 2010) and possibly betaine aldehyde dehydrogenase (ALDH19; Hou and Bartels, 2015). The peroxisomal NADH pool is supported by malate dehydrogenase MDH2 (Cousins et al., 2008; Figure 2B). Peroxisomes also contain pyrophosphatase Nudix Hydrolase Homolog 19 (NUDT19), which hydrolyzes NADPH to NMNH, as well as 2′,5′-ADP and NADH to NMNH and AMP (Lingner et al., 2011).

**ROS- and NO-dependent PTMs in peroxisomal metabolism regulation**

Analysis of peroxisomal proteomes shows that a large number of peroxisomal proteins (35%) are targeted by multiple PTMs (Sandalio et al., 2019). Peroxisomal-dependent ROS/RNS can fine-tune post-translational redox changes in proteins, regulating stability, activity, location, and protein–protein interactions (Duan and Walther, 2015; Hashiguchi and Komatsu, 2016; Sandalio et al., 2019; Foyer et al., 2020) supporting peroxisomes capacity to regulate their metabolism and dynamics in response to environmental changes. Hydrogen peroxide leads to rapid and reversible oxidative protein modifications such as sulfenylation, sulfinylation, and intra- and intermolecular disulfide bond formation, which contribute to coordinated regulation of cellular processes, while overoxidation by sulfonlylation appears to be an irreversible process (reviewed in Noctor et al., 2018; Young et al., 2019; Sandalio et al., 2019; Sies and Jones, 2020). Given their transient nature, these sulfur modifications are regarded as redox switches (Huang et al., 2018). Peroxisomal antioxidant defenses, fatty acid β-oxidation, and photorepair are prone to $\text{H}_2\text{O}_2$-dependent redox regulation (reviewed in Sandalio et al., 2019). The glyoxalase 1 (GLX1) homolog is a putative sulfenylation protein involved in protection against carbonyls (Schmitz et al., 2018).

NO, in turn, modifies proteins through covalent PTMs including S-nitrosylation (Martín-Ruiz et al., 2011; Sánchez-Vicente et al., 2019). Putative peroxisomal S-nitrosylated proteins also include antioxidants and enzymes from the photorespiration cycle (Romero-Puertas and Sandalio, 2016; Sandalio et al., 2019) suggesting that S-nitrosylation plays an important role in regulating peroxisomal $\text{H}_2\text{O}_2$.
concentrations under physiological and stress conditions (Ortega-Galisteo et al., 2012). Recently, the noncanonical catalase CAT3, identified as a “repressor of” GSNO1 (ROG1), was reported to transnitrosylate GSNO1 to promote its degradation by autophagy, while CAT1 and CAT2 do not do it, thereby CAT3 positively regulates NO signaling and according to Arabidopsis rog1 mutants are more susceptible to NO than WT (Chen et al., 2020). CAT3 is localized in peroxisomes, the cytoplasm, and the plasma membrane (Li et al., 2015; Zou et al., 2015) and is recruited into the nucleus by the cucumber mosaic virus (CMV) 2b protein (Inaba et al., 2011; Murota et al., 2017). Zhan et al. (2018) have reported that S-nitrosylation induces selective autophagy of Arabidopsis GSNO1 during hypoxia responses. CAT3 also interacts with other proteins in the cytosol and plasma membrane, thus increasing the likelihood that these proteins are also substrates of CAT3 transnitrosylase activity (Chen et al., 2020). These findings suggest NO self-regulation and ROS/NO crosstalk. Zhang et al. (2020) have reported that glutathione denitrosylation is required to maintain the upregulation of GSNO activity; thus coordinating GSNO activity with protein S-nitrosylation levels to ensure appropriate signaling involving the SA pathway in response to H2O2.

Some fatty acid β-oxidation enzymes, including ACX2, 3, may be S-nitrosylation targets (Sandialo et al., 2019). OPC-8:0 CoA Ligase1 (OPCL1), involved in activating JA biosynthetic precursors in leaf peroxisomes (Koo et al., 2006), is also a putative target of S-nitrosylation, pointing to NO/JA-crosstalk. Proteomic analyses suggest that BRI1 suppressor 1 (BSU1)-like 3 is targeted by S-nitrosylation (Sandialo et al., 2019) suggesting NO-dependent brassinosteroids signaling. NO-dependent nitration also inhibits peroxisomal antioxidants such CAT (Lozano-Juste et al., 2011; Chaki et al., 2015) and SOD (Holzmeister et al., 2015). Therefore, NO and ROS, apart from self-regulation (Romero-Puertas and Sandialo, 2016), may also regulate specific proteins and/or metabolic pathways and metabolite channeling, depending on the redox environment both inside and outside the peroxisome.

Peroxisome-dependent redox regulation of transcriptional responses

ROS act as secondary messengers that are sensed by specific redox-sensitive proteins, which activate signal transduction pathways and alter gene expression (Suzuki et al., 2012; Mittler, 2017). Different ROS trigger different protein modifications, as shown by different gene expression patterns (Møller and Sweetlove, 2010; Mor et al., 2014). The subcellular site, where the ROS/oxidation state is modified, acts as a specific cellular redox network signal (Foyer and Noctor, 2003; König et al., 2012; Foyer et al., 2017) and leaves a specific imprint on the transcriptome response (Rosenwasser et al., 2011). The selective reactivity, stability, and diffusibility of H2O2 make it an ideal signaling molecule (Sewelam et al., 2014; Sies and Jones, 2020). Mutants lacking peroxisomal CAT2 (cat2) have been extensively studied in Arabidopsis and tobacco (Nicotiana tabacum) plants under control and stress conditions, showing that altering peroxisomal H2O2 induces changes in gene expression profiles (Vandenabeele et al., 2003, 2004; Chaouch et al., 2010; Queval et al., 2012). This profile showed specificity with transcriptional responses that differ from those induced by chloroplast-derived H2O2 (Sewelam et al., 2014). Analyses of WT plants grown at specific atmospheric CO2 levels to boost photorespiration and production of H2O2 (Chaouch et al., 2010; Queval et al., 2012) and of WT plants treated with aminotriazole, a catalase inhibitor (Gechev et al., 2005), have also shown that peroxisomal H2O2 plays a role in signaling, as a transcriptional footprint have been linked to peroxisomes (Rosenwasser et al., 2013). However, little is known about how peroxisome-derived H2O2 coordinates or relays signaling events. Although peroxisome-dependent gene regulation involves several metabolic categories (reviewed in Sandialo and Romero-Puertas, 2015; Su et al., 2019), those related to protein repair responses under stress conditions are regulated in cat2 mutants (Queval et al., 2007; Sewelam et al., 2014); suggesting that peroxisomes are involved in acclimation and survival processes under changing environmental conditions. The triple mutant cat1 cat2 cat3 shows serious redox disturbance and growth defects under physiological conditions, with differentially expressed genes belonging to plant growth regulation, as well as abiotic and biotic stress response categories. Some of these genes belong to transcription factor and receptor-like protein kinase categories (Su et al., 2019). The ROS signals derived from different cell compartments are proposed to connect in the cytoplasm with MAPK pathway to regulate the expression of nuclear genes (Noctor and Foyer, 2016). Several genes related to MAPK cascade pathways, such as MPK11, MPK13, and serine/threonine kinase oxidative signal inducible 1 (OXI1), are severely altered in the triple cat mutant (Su et al., 2019). Thus, peroxisomal H2O2 appears to participate in retrograde signaling, although little is known about the underlying molecular mechanisms and crosstalk with ROS from other compartments (Sandialo and Romero-Puertas, 2015; Su et al., 2019).

Redox-dependent regulation of peroxisomal plasticity

Peroxisome biogenesis and proliferation

Their high plasticity enables peroxisomes to adapt their number, morphology, movement, and metabolic pathways to changes in their environment (Figure 3). However, why certain signals and molecules trigger these changes, when these changes occur, and how dynamic peroxisomal changes function in relation to tolerance are not well understood. Some evidence shows that peroxisomal proliferation through the division of preexisting peroxisomes is regulated by ROS (López-Huertas et al., 2000; Figure 3; Box 2). Peroxisome proliferation occurs in response to abiotic stresses associated with ROS production: ozone (Oksanen et al., 2004), clofibrate (Nila et al., 2006; Castillo et al., 2008), salinity
Proteins involved in peroxisome biogenesis and maintenance are called peroxins (PEXs; Baker et al., 2016; Reumann and Bartel, 2016; Kao et al., 2018; Reumann and Chowdhary, 2018). Peroxisome proliferation under abiotic stress appears to be regulated by specific PEX11 genes which contain five members (PEX11a-PEX11e; Lingard and Trelease, 2006; Orth et al., 2007) and under abiotic stress-specific PEX11 appears to regulate peroxisome proliferation. Thus, salinity upregulated PEX11a and PEX11c in A. thaliana (Ebeed et al., 2017), while PEX11a and PEX11e were upregulated in response to Cd exposure in Arabidopsis plants (Rodríguez-Serrano et al., 2016; Terrón-Camero et al., 2020), and PEX11b, PEX11c, and PEX11d were upregulated by hypoxia (Li and Hu, 2015). Gene coexpression analysis in Arabidopsis plants under drought conditions shows clustering of photosynthetic genes and peroxisomal abundance, suggesting that H$_2$O$_2$ plays a role in peroxisomal abundance regulation (Li and Hu, 2015). This is supported by the absence of peroxisome proliferation in gox2 Arabidopsis mutants exposed to Cd (Calero-Muñoz et al., 2019). However, genome analyses of Physcomitrium, A. thaliana, and Triticum aestivum show upregulation of $\beta$-oxidation in response to drought, dehydration, and ABA (Ebeed et al., 2018). Interestingly, PEX11 gene family expression differs between drought-sensitive and resistant wheat genotypes, although the significance or not of these differences for drought tolerance has not been established (Ebeed et al., 2018). These findings suggest that peroxisomal H$_2$O$_2$ could be involved in environmental change perception and acclimation through differential PEX11 regulation and peroxisome proliferation. Plant peroxisome proliferation could be a protective response to ROS overflow in cell compartments due to highly efficient peroxisomal antioxidant defenses, as reported during protoplast transition from G0 to G1 (Tiew et al., 2015) and in response to salt stress in Arabidopsis and Oryza sativa by reducing

**Box 1 SUBCELLULAR REDOX COMPARTMENTALIZATION**

As oxygen-dependent redox reactions came to control life after O$_2$ appeared in the atmosphere, cells developed complex mechanisms to detect and regulate these changes to maintain metabolic functionality. Redox compartmentalization in organelles is an effective evolutionary strategy, which regulates physiological and stress conditions through site-specific footprinting (Jones and Go, 2010). This redox circuit flexibility facilitates rapid responses to changes in intracellular redox equilibrium, which, in turn, favors beneficial signaling and detrimental oxidative stress. Photosynthetic organisms have developed efficient redox control systems using redox signals as the most fundamental forms of information (Foyer and Noctor, 2016). The thiol/disulphide couples GSH/GSSG and Cys/CySS, the ASC/DHA couple and a broad range of redox dependent proteins, which are counterparts of ROS such as H$_2$O$_2$, and other oxidants, form the core of the redox state and regulate the cell signaling, structure and activity of proteins and transcription factors. Apart from ROS, RNS are also redox signaling molecules, which include NO and peroxynitrite (ONOO$^-$). Both ROS and RNS regulate covalent, often reversible, modifications, mainly targeting Cys, which regulates metabolic shifts and triggers signaling cell responses (Noctor et al., 2018; Sánchez-Vicente et al., 2019). Although irreversible oxidation products, such as sulfonic acid, carbonylation, and nitration, adversely affect proteins and lipids, they may be also involved in oxidative signaling (Foyer et al., 2017). Analyses of redox potential in plant tissue identified peroxisomes as some of the most oxidized cellular organelles, with a redox potential of approximately $-360$ mV (Bratt et al., 2016; Smirnoff and Arnaud, 2019)
Box 2 PEROXISOME BIOGENESIS AND PROLIFERATION

Plant peroxisome abundance is governed by (1) biogenesis, associated with physiological processes and division (fission) of a preexisting peroxisome, (2) proliferation, which is related to stress responses, and (3) pexophagy, a selective peroxisome degradation mechanism (Kao et al., 2018; Olmedilla and Sandalio, 2019). Proteins involved in peroxisome biogenesis and maintenance are called peroxins (PEXs; Kao et al., 2018). The import of peroxisomal membrane proteins (PMPs) in Arabidopsis involves peroxins PEX19, acting as the chaperone for PMPs, PEX3 acting as the membrane anchor for PEX19, and PEX16, which recruits PEX3 to the ER before the formation of pre-peroxisomes. Arabidopsis PEX16 also recruits PMPs to the ER in a PEX3/PEX19-independent manner (Pan et al., 2020). The import of peroxisomal matrix proteins containing the C-and N-terminal targeting signals PTS1 and PTS2, respectively, take place by the soluble receptors PEX5 (for PTS1) and PEX7 (for PTS2) in the cytosol (Baker et al., 2016; Reumann and Chowdhary, 2018). PEX5 is recycled from the peroxisomal matrix back to the cytosol by the ubiquitin conjugating enzyme PEX4 and its membrane anchor PEX22, three RING-type ubiquitin ligases, PEX2, PEX10 and PEX12, and two AAA ATPases, PEX1 and PEX6 (reviewed in Reumann and Bartel, 2016; Kao et al., 2018).

Peroxisomes proliferate through the division of preexistent peroxisomes, which involves peroxisome elongation regulated by PEX11, organelle constriction and fission, governed by dynamin-related proteins (DRP3A and DRP3B) and fission proteins (FIS1A and FIS1B; Pan et al., 2020; Su et al., 2019). The PEX11 gene family in Arabidopsis contain five members: PEX11a, PEX11b, PEX11c, PEX11d and PEX11e, (Lingard and Trelease, 2006; Orth et al., 2007). FIS1A and FIS1B are shared by peroxisomes and mitochondria; DRP3A and DRP3B regulate peroxisomal and mitochondrial fission; and DRP5B is involved in peroxisome and chloroplast fission (Kao et al., 2018), indicating a highly coordinated regulation of organelles populations.

ROS/RNS-dependent formation of peroxules

In vivo observation of plant tissues, with fluorescent proteins targeting peroxisomes, reveals the rapid formation of tubular peroxisome extensions called peroxules, induced in response to changes in ROS levels (Figure 3; Sinclair et al., 2009; Barton et al., 2013; Rodríguez-Serrano et al., 2016). Short periods of Cd exposure (15–30 min) induce peroxule formation, which is considerably reduced by H2O2 scavengers and in rboh mutants, suggesting regulation by external ROS (Rodríguez-Serrano et al., 2016). Confocal images show peroxule contacts with chloroplasts and mitochondria under Cd treatment and high light (Sinclair et al., 2009; Sandalio et al., 2013; Jaipargas et al., 2016; Rodríguez-Serrano et al., 2016). Peroxule formation in response to Cd and As is dependent upon PEX11a, while pex11a Arabidopsis mutants show altered ROS-dependent signaling networks (Rodríguez-Serrano et al., 2016). Peroxule production and peroxisome-dependent signaling are compromised in nia1 nia2 Arabidopsis mutants, which have lower NO levels than wild-type plants in response to Cd treatment, demonstrating the important role of NO in peroxule formation (Terrón-Camero et al., 2020). This could be due to oxidative changes and S-nitrosylation patterns in the antioxidant system (Terrón-Camero et al., 2020), which affect cellular redox balance. PEX11a and peroxule formation therefore play a key role in regulating stress perception and rapid cell responses to environmental cues (Rodríguez-Serrano et al., 2016; Terrón-Camero et al., 2020; Figure 3). Given rapid peroxule induction and no significant changes in PEX11a expression in nox1 mutants (Terrón-Camero et al., 2020), PEX11a can reasonably be assumed to be regulated by specific ROS- and NO-dependent PTMs. Activation of yeast peroxin Pex11p depends on redox changes in its cysteines (Knoblauch and Rachubinski, 2010; Schrader et al., 2012).

Although there is no direct evidence, peroxules could participate in the transfer of H2O2 and other metabolites to mitochondria and chloroplast (Figure 4). Stromules, which are dynamic structures similar to peroxules in chloroplasts, transfer H2O2 from chloroplasts to nuclei as part of a retrograde signaling process (Caplan et al., 2015; Kumar et al., 2018); however, to our knowledge, no connection between peroxules and nuclei has been established so far. Peroxules could also be involved in protein transport such as the transfer of the sugar-dependent 1 (SDP1) lipase from the peroxisomal membrane to the lipid body (Thazar-Poulot et al., 2015; Figure 4).

Peroxisomal speed is regulated by ROS

Time course analyses of peroxisomes in response to Cd in Arabidopsis seedlings have highlighted a considerable increase in peroxisomal speed after 24 h of treatment which is regulated by ROS produced by RBOHs and Ca2+ ions (Figure 3; Rodríguez-Serrano et al., 2009, 2016). Increased
peroxisomal movement could improve antioxidant defenses where Cd and other factors promote ROS accumulation and/or could aid signaling transduction and metabolite exchanges in different parts of the cell (Rodríguez-Serrano et al., 2009). Information on the role of peroxisomal motility under stress conditions is scarce; however, in myosin loss-of-function Arabidopsis mutants and in Arabidopsis treated with the herbicide 2,4-D, inhibition of organelle movement negatively affects plant growth (Rodríguez-Serrano et al., 2014; Ryan and Nebenführ, 2018). Oikawa et al. (2015) found that light-adapted Arabidopsis peroxisomes are much more mobile than dark-adapted peroxisomes (Oikawa et al., 2015) and, by using photosynthetic mutants shmt1 (defective in serine hydroxymethyltransferase) and ped2 (defective in PEX14), they concluded that both, peroxisome mobility and peroxisome–chloroplast interactions observed under light, are regulated by photosynthesis rather than by photo-receptors or photorespiration (Oikawa et al., 2015).

**Figure 4** Redox-dependent interorganellar crosstalk. Peroxisomes (P) collaborate and communicate with other cellular organelles, mitochondria (M), and chloroplasts (CH) by exchanging molecules such as H$_2$O$_2$, and redox metabolites, as well as Ca$^{2+}$ and proteins. These exchanges could take place through porins or MCSs. Peroxosome formation contributes to ROS/RNS, metabolite, and protein exchanges such as the transfer of TAG lipase sugar-dependent 1 (SDP1) to lipid bodies (LB). ROS/RNS-dependent posttranslational modifications regulate peroxule formation, MCSs, interorganellar crosstalk, and signaling transduction. Peroxosomal ROS/RNS interferes with cytosolic redox state and signaling processes and vice versa; the cytosolic redox state regulates peroxosomal protein import by affecting the redox state of peroxin 5 (PEX5). The peroxisomal redox state can also regulate redox changes in the nucleus (N), chloroplasts, and mitochondria.

**Pexophagy and peroxisomal homeostasis are regulated by oxidative processes.**

Excessive numbers of peroxisomes and those containing obsolete or dysfunctional proteins need to be eliminated to control cellular redox homeostasis. Some evidence shows that ROS and oxidative damage to peroxisomes regulate the degradation of oxidized whole peroxisomes by selective autophagy termed pexophagy (Figure 3; Shibata et al., 2013; Yoshimoto et al., 2014; Olmedilla and Sandalio, 2019).

Autophagy-related genes (ATGs) regulate autophagy in all eukaryotic cells including those in plants (Avin-Wittenberg et al., 2018). During photomorphogenesis, several authors have reported pexophagy using Arabidopsis atg mutants (Farmer et al., 2013; Kim et al., 2013; Shibata et al. 2013); however, unlike in mammals and yeast, the mechanism in plants is not fully understood. Shibata et al. (2013) have observed high levels of oxidized CAT and clusters of peroxisomes in atg mutants. Peroxosomal clusters were also observed in H$_2$O$_2$-treated Arabidopsis plants (Yoshimoto et al., 2014) and in atg5 and atg7 Arabidopsis mutants exposed to Cd treatment where phagophore and peroxosome colocalization was observed (Calero-Muñoz et al., 2019). Some evidence shows the important role of oxidative processes in pexophagy induction: (1) ubiquitinated CAT is accumulated in Arabidopsis mutants defective in NBR1 (nbr-1), a pexophagy adaptor (Zhou et al., 2013); (2) ATG8/CAT–CAT/NBR1 interactions have been observed in Arabidopsis plants exposed to Cd (Calero-Muñoz et al., 2019); (3) CAT activity is involved in starvation-induced pexophagy (Tyutereva et al., 2018); and (4) clustered peroxisomes in Arabidopsis atg mutants mainly accumulate in the aerial parts of plants, where oxidative metabolism is higher than in roots (Zhou et al., 2013; Yoshimoto et al., 2014). Glucose-mediated regulation of root meristem activity requires pexophagy to maintain ROS and auxin cellular homeostasis in Arabidopsis plants (Huang et al., 2019). The chaperone activity of peroxosomal protease LON2 negatively regulates pexophagy (Figure 3; Farmer et al., 2013; Young and Bartel, 2016). In plants, specific peroxisomal receptors have not been clearly identified, and the role of adaptors, such as NBR1-like proteins, which specifically interact with ubiquitinilated proteins, is under debate (Olmedilla and Sandalio, 2019; Young et al., 2019). The possibility of both NBR1-dependent and independent pexophagy cannot be ruled out. An alternative process independent of autophagy, induced under high CO$_2$ and increased H$_2$O$_2$ conditions, involves chloroplast vesiculation (CV) proteins which interact with PEX11-1 in rice (Figure 3; Umnajkitikorn et al., 2020).

**Peroxisome crosstalk with other organelles**

To optimize their multiple functions, peroxisomes collaborate and communicate with other cell organelles by exchanging substrates. Photorespiration is the best example of metabolic cellular interorganelle communication. However, peroxisomes are also cellular redox communication hubs, as well as guardians and modulators of H$_2$O$_2$ levels (Fransen and Lismont, 2019) given the following findings: (1) peroxisomes contain enzymes involved in producing and scavenging H$_2$O$_2$ and NO; (2) they contain proteins regulated by ROS- and NO-dependent PTMs and therefore act as ROS/NO sensors; (3) they regulate NAD(P) + /NAD(P)H, ascorbic (Asc)/dehydroascorbic acid (DHA) and GSH/GSSG pools;...

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and (4) \( \text{H}_2\text{O}_2 \) and NO act as second messengers in a wide range of developmental, physiological, and stress processes (Fransen and Lismont, 2019; Sandalio et al., 2020).

There is an intimate relationship between the peroxisomal redox state and changes in the redox state of other organelles. In mammalian systems, \( \text{H}_2\text{O}_2 \) released from peroxiomes into the cytosol diffuses into mitochondria, oxidizing directly or indirectly cysteine residues of mitochondrial proteins (Lismont et al., 2019). Chlamydomonas mutants deficient in peroxisomal NAD\(^+\)-dependent MDH2 show that MDH2 plays a key role in the reverse coupling of redox/\( \text{H}_2\text{O}_2 \) signals from peroxisomes to chloroplasts (Kong et al., 2018). Peroxisomal NAD(P)\(^+\)/NAD(P)H pools in Arabidopsis regulate photosynthesis performance to meet the demand for reducing equivalents under fluctuating light (Li et al., 2019). Peroxisomal basal \( \text{H}_2\text{O}_2 \) levels greatly affect antioxidative defense regulation in cytosol and chloroplasts, as observed in peroxisomal apx4 knockdown rice plants (Sousa et al., 2018). The inhibition of CAT activity in apx4 Arabidopsis mutants significantly affected networks involved in photosynthetic performance under adverse conditions promoting oxidative stress and favoring antioxidant enzyme accumulation in cytosol and chloroplasts (Sousa et al., 2018).

Despite the central role of \( \text{H}_2\text{O}_2 \) in peroxisome metabolism and cell functionality, no peroxiporin-like proteins have been identified in the peroxisomal membrane. Although porins are present in plant peroxisomes (Reumann et al., 1997; Corpas et al., 2000; Figure 4), their role in \( \text{H}_2\text{O}_2 \) permeability remains unclear. In yeast, Pex11A, Pex11B, and Pex11G have been reported to facilitate the permeation of molecules up to 400 Da (Mindthoff et al., 2016) and could be candidates to diffuse \( \text{H}_2\text{O}_2 \) through the peroxisomal membrane. However, recently, Lismont et al. (2019) provided evidence that neither the porin PXMP2 nor PEX11B is essential for \( \text{H}_2\text{O}_2 \) permeation across the peroxisomal membrane. Throughout membrane contact sites (MCSs), ROS accumulation could directly facilitate interorganelle signal transmission using as-yet-unknown ROS transporters (Figure 4; Yoboue et al., 2018). Electron microscopy images of leaf cells show physical contact between peroxisome and chloroplasts and, interestingly, \( \text{H}_2\text{O}_2 \) accumulation inside peroxisomes in the contact site with chloroplasts and vacuoles, suggesting a relationship between ROS accumulation and organelle communication (Romero-Puertas et al., 2004). Using femtosecond laser and optic tweezer techniques, tethering between the chloroplast and peroxisomes has been demonstrated (Oikawa et al., 2015; Gao et al., 2016). The area of peroxisomes interacting with chloroplasts increases under light conditions, whereas, in the dark, peroxisomes lost their connection with chloroplasts (Oikawa et al., 2015). The PEX10 Zn RING finger interacts with the chloroplast envelope’s outer membrane, which is necessary for full photorespiration functionality and could be a candidate for MCSs in plant peroxisomes (Schumann et al., 2007; Figure 4). Although the role of PTMs in regulating protein–protein interactions at interorganelle contact sites remains unexplored, it is reasonable to assume that tethering is regulated by specific PTMs.

The translocation of peroxisomal proteins to other cell compartments is part of interorganellar communication and signaling, although the mechanism(s) by which this occurs is still unknown. Thus, CAT interacts with nonperoxisomal proteins including cytosolic calcium-dependent kinase CDPK8 (Zou et al., 2015), plasma membrane-associated calcium-dependent kinase OsCPK10 (Bundó and Coca, 2017), cytosolic salt overly sensitive 2 (SOS2; Verslues et al., 2007), lesion simulating disease1 (LSD1; Li et al., 2013), receptor-like cytoplasmic kinase STRK1 (Zhou et al., 2018), chloroplast/cytosolic nucleoside diphosphate kinase 2 (NDPK2), no catalase activity 1 (NCA1; Hackenberg et al., 2013; Li et al., 2015), and nucleoredoxin 1 (NRX1; Kneeshaw et al., 2017) in addition to GSNO (mentioned above), which are all integral stress-signaling proteins. It is unclear whether CAT is translocated from peroxisomes by the ER-associated degradation (ERAD)-like system involved in the export of PEX5 from the peroxisomal membrane and export of matrix peroxisomal proteins to be degraded (Lingard et al., 2009) or is retained in the cytosol under oxidative conditions as in the case of mammalian cells (Walton et al., 2017). Walton et al. (2017) have reported that Cys-11 of human PEX5 acts as a redox switch that modulates the import of peroxisomal matrix proteins such as CAT. Under oxidative stress conditions, CAT is retained in the cytosol where it can protect against \( \text{H}_2\text{O}_2 \)-mediated redox changes and reinforce cellular defenses to prevent oxidative damage out of peroxisomes (Walton et al., 2017). Oxidative and nitrosative stress could enable swift control of CAT localization in compartments, thus helping to regulate redox signaling pathways.

In the case of dual-targeted OPPP enzymes, targeting decisions appear to depend on the cytosolic redox state. This has been suggested in relation to Arabidopsis G6PD1/G6PD4 (Meyer et al., 2011), PGL3 (Hölscher et al., 2014), and PGD2 upon interaction with nonperoxisomal isoforms PGD1 or PGD3, which retain heteromeric enzymes in the cytosol (Lutterbey and von Schaewen, 2016). NADPH-oxidase and peroxisomal AtPAO3 cross-talk to balance intracellular \( \text{O}_2^/-/\text{H}_2\text{O}_2 \), which in turn, affect the cyt-c/AOX pathways in mitochondria and regulates pollen tube elongation (Wu et al., 2010). As catalase (cat2)-deficient Arabidopsis mutants show upregulation of ASC-GSH components in the cytosol (Mhamdi et al., 2010), peroxisomes can interfere with cytosolic redox state. Cytosolic redox changes, in turn, impact the dual targeting of 6-phosphogluconolactonase 3 (PGL3) of chloroplasts and peroxisomes in Arabidopsis leaves, a process requiring thioredoxin m2 (Trxm2) in the cytosol (Hölscher et al., 2014).

**Future perspectives**

Many challenges in peroxisome redox biology remain to be addressed (Outstanding questions). One of them is to
OUTSTANDING QUESTIONS

- Do carboxylated and nitrated peroxisomal proteins act as signaling messengers?
- Does CAT3 transnitrosylate other proteins in addition to GSNOR? Are other transnitrosylases present in peroxisomes?
- Is S-nitrosylation involved in retrotranslocation of peroxisomal proteins to the cytosol?
- Which pexophagy receptors and adaptors are involved and what role do ROS and NO play in identifying peroxisomes for degradation?
- How do cells balance peroxule production, peroxisome proliferation and changes in peroxisomal speed in response to changes in their environment?
- Which ROS/redox sensor(s) is (are) present in the peroxisomal membrane?

determine the nature of proteins involved in peroxisomal NO production. We need to amplify our limited knowledge of the mechanisms underlying NO regulation of peroxisome dynamics, metabolism, and signaling, together with NO and ROS crosstalk with hormones, such as JA. Additional analyses of the interplay and hierarchy of ROS-, NO-dependent, and other peroxisomal PTMs such as phosphorylation and persulfidation are required. Peroxisome-dependent regulatory components also need to be characterized by analyzing gene network structures and by identifying downstream responses induced by peroxisomal ROS and NO. The components of contact sites and the factors involved in peroxule production, as well as the regulatory role of ROS and NO in both these areas, also need to be determined. Analysis of tethering techniques, specific fluorescence proteins, and ROS mutants, combined with meta-analyses of organelle proteome datasets, should provide a better understanding of peroxisomal dynamics and interorganelle interactions. Finally, identification of pexophagy receptors and adaptors and their regulation by ROS, NO, and S$_2$H should enable us to integrate biochemical processes and organelle dynamics into our understanding of cellular regulatory systems.

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