Free Radical Biology and Medicine 197 (2023) 58–70

Contents lists available at ScienceDirect

Free Radical Biology and Medicine

journal homepage: www.elsevier.com/locate/freeradbiomed

Invited Review Article

Deciphering peroxisomal reactive species interactome and redox signalling networks☆

Luisa M. Sandalio *, Aurelio M. Collado-Arenal, María C. Romero-Puertas

Department of Biochemistry, Cell and Molecular Biology of Plants, Estación Experimental del Zaidín (EEZ), Consejo Superior de Investigaciones Científicas (CSIC), C/ Profesor Albareda 1, 18008, Granada, Spain

ARTICLE INFO

Keywords:
Interactome
Peroxisome
Reactive nitrogen species
Reactive oxygen species
Reactive sufur species
Reactive carbonyl species
Redox signaling
Retrograde signaling

ABSTRACT

Plant peroxisomes are highly dynamic organelles with regard to metabolic pathways, number and morphology and participate in different metabolic processes and cell responses to their environment. Peroxisomes from animal and plant cells house a complex system of reactive oxygen species (ROS) production associated to different metabolic pathways which are under control of an important set of enzymatic and non enzymatic antioxidative defenses. Nitric oxide (NO) and its derivate reactive nitrogen species (RNS) are also produced in these organelles. Peroxisomes can regulate ROS and NO/RNS levels to allow their role as signalling molecules. The metabolism of other reactive species such as carbonyl reactive species (CRS) and sulfur reactive species (SRS) in peroxisomes and their relationship with ROS and NO have not been explored in depth. In this review, we define a peroxisomal reactive species interactome (PRSI), including all reactive species ROS, RNS, CRS and SRS, their interaction and effect on target molecules contributing to the dynamic redox/ROS homeostasis and plasticity of peroxisomes, enabling fine-tuned regulation of signalling networks associated with peroxisome-dependent H_2O_2. Particular attention will be paid to update the information available on H_2O_2-dependent peroxisomal retrograde signalling and to discuss a specific peroxisomal footprint.

1. Introduction

Peroxisomes, which are ubiquitous organelles in different organisms, have common metabolic pathways, such as reactive oxygen species (ROS) metabolism and fatty acid β-oxidation, throughout their evolution (Table 1). Interestingly, from an evolutionary perspective, peroxisomes, originating from the endoplasmic reticulum (ER), were selected to house a range of pathways involving the production of certain ROS such as H_2O_2 [1]. The driving force behind the origin of peroxisomes from the ER has been proposed to be the separation of H_2O_2-producing enzymes involved in fatty acid metabolism that could be toxic to other processes, such as protein folding and modification, occurring in the ER [1]. Subsequently, other oxidative enzymes and pathways, such as certain β-oxidation enzymes originally localized to the mitochondrion, were relocated to peroxisomes [2]. This suggests that the relocation of the β-oxidation of very long fatty acids in peroxisomes could constitute a

Abbreviations: ACX, Acyl CoA oxidase; AO, Aldehyde oxidase; APX, Ascorbate peroxidase; ASC, Ascorbate AT: 3-aminotriazole; ATG, Autophagy-related genes; CAS, β-cyanoalanine synthase; CAT, Catalase; CDES, Cysteine desulfhydrases; CDPK8, calcium-dependent kinase 8; CMV, Cucumber mosaic virus; CRS, Carbonyl reactive species; CuAO, Copper amine oxidase; DHAR, Dehydroascorbate reductase; 2,4-D, 2,4-dichlorophenoxiacetic acid; DRP, Dynamin-related protein; ER, Endoplasmic reticulum; Fis1, Fission protein 1; GLX1, Glyoxalase 1; GOX, Glycolate oxidase; GPX, Glutathione peroxidase; GR, Glutathione reductase; GSH, Reduced glutathione; GSNO, S-nitrosoglutathione; GSNOR, S-nitroso-glutathione reductase; GSNOR-CysNO, GSNOR S-nitrosylated; GSSG, Oxidized glutathione; GST, Glutathione-S-Transferase; GUN1, Genome uncoupled 1; HPR, Hydroxypyruvate reductase; HSP, Heat shock protein; IAA, Indolacetic acid; IBA, Indole-3-butyric acid; ILV, Intraluminal vesicles; JA, Jasmonic acid; LAP2, Leucine aminopeptidase 2; MAPK, Mitogen-activated protein kinases; MDA, Malondialdehyde; MDAR, Monodehydroascorbate reductase; MGO, Methylglyoxal; NCA1, No catalase activity 1; NOS-like, Nitric oxide oxidase like activity; OASTL, O-acetylserine (thiol) lyase; PAP, 3'-Phosphoadenosine 5'-Phosphate; PEXs, Peroxines; PMP, Peroxisomal membrane protein; PP2A-B'γ, B'γ 2A protein phosphatase subunit; PRDX5, peroxiredoxin 5; PRSI, Peroxisomal reactive species interatome; PRX, Peroxiredoxin; PTM, Post-translational modifications; RNS, Reactive nitrogen species; RSI, Reactive species interactome; RSS, reactive sulfide species; SA, Salicylic acid; SAOX, sarcosine oxidase; SOD, Superoxide dismutases; SOS2, Salt overly sensitive 2; SRS, Sulfur reactive species; Ub, ubiquitin; UO, Urate oxidase; XOR, Xanthine oxidoreductase.

☆ This article is a contribution to the special issue entitled "Oxidative stress responses in plants" Guest Edited by Frank van Breusegem.

* Corresponding author.
E-mail address: luisamaria.sandalio@eez.csic.es (L.M. Sandalio).

https://doi.org/10.1016/j.freeradbiomed.2023.01.014
Received 14 October 2022; Received in revised form 19 December 2022; Accepted 12 January 2023
Available online 13 January 2023

Table 1
Main metabolic pathways associated to peroxisomes in plant and animal cells.

Main metabolic pathways	Plants	Animals	References
Reactive Oxygen and Nitrogen Species Metabolism	✓	✓	Rev in [3,4]
Sulfite metabolism	✓		Rev in [3,5]
Amino acids metabolism	✓	✓	Rev in [5]
Indole glucosinolates metabolism	✓		Rev in [6]
Ureide metabolism	✓	✓	Rev in [3,7]
Hormones biosynthesis	✓	✓	[8,9]
Polyunsaturated fatty acids biosynthesis		✓	[10]
Eter phospholipids biosynthesis		✓	[11]
Pyrimidines biosynthesis		✓	[10]
Biotin biosynthesis	✓		Rev in [5]
Ubiquinone biosynthesis	✓	✓	Rev in [5,12]
Phylloquinone biosynthesis	✓		Rev in [5]
Secondary metabolites biosynthesis	✓		Rev. in [5]
Isoprenoid and cholesterol biosynthesis	✓		Rev. in [5,10]
Bile acids biosynthesis		✓	Rev in [10]
Prostaglandin degradation		✓	Rev in [10]
Polyamine degradation	✓	✓	Rev in [10, 13]
Purines degradation	✓	✓	Rev in [10,7]
Phytol degradation	✓		[14]
Pseudouridine degradation	✓		[15]
Photorespiration	✓		Rev in [8]
Oxidation of fatty acids	✓	✓	Rev [8,10]
Glyoxylate cycle	✓		Rev in [8]

selective advantage in order to avoid an excess of FADH2/NADH produced during this process, leading to an unbalanced ratio of ubiquinone and its reduced form, ubiquinol [2]. This, in turn, interferes with the mitochondrial respiratory chain, which eventually leads to an increase in ROS production [2].

Plant peroxisomes are highly dynamic organelles with regard to metabolism, morphology and abundance. They participate in different metabolic processes involved in development, morphogenesis, reproduction, and cell responses to different stress conditions and also house nitric oxide (NO) and reactive nitrogen species (RNS) metabolism (reviewed in Refs. [3,8] Table 1). The contribution of peroxisomes to the management of other reactive species, such as carbonyl reactive species (CRS) and sulfur reactive species (SRS), is also under debate. The biosynthesis of phytohormones jasmonic acid (JA), auxin and salicylic acid (SA) (Table 1) which are associated to β-oxidation pathway and regulate multiple pathways in the cell, also contributes to the complex role of peroxisomes in development and stress responses [5]. Therefore, peroxisomes could be considered as a platform of signal molecules in the cell.

Peroxisomes are small organelles surrounded by a simple membrane containing a granular matrix. However, recently, using monomeric fluorescent reporter proteins to target sequences from two peroxisome membrane peroxins, Wright and Bartel (2020) [16] have demonstrated that peroxisomes contain internal membranes that accumulate over time, whose formation requires endosomal sorting complexes for transport (ESCRT) machinery, and appear to be derived from the peroxisomal membrane giving rise to the formation of intraluminal vesicles (ILV). These vesicles were mainly visualized in young seedlings when β-oxidation is more active and could be involved in a mechanism of lipid mobilization in the peroxisomal lumen [16].

Nowadays, peroxisomes are considered to be multipurpose organelles that are necessary for both animal and plant cell functionality, for the perception of changes in their environment and for the regulation of cell responses to environmental conditions, as well as to disease [3,4] (Table 1). The range of functions associated with peroxisomes, particularly in plants, is continuously increasing, with, in addition to ROS detoxification and fatty acid β-oxidation, peroxisomes housing ureide metabolism, polyamine and amino acid catabolism, photorespiration, the glyoxylate cycle, sulfur and indole glucosinolate metabolism, biotin biosynthesis, ubiquinone and phylloquinone biosynthesis, isoprenoid biosynthesis and benzoic acid derivative biosynthesis [3,8,6] (Table 1). New functions of peroxisomes in the degradation of chlorophyll-derived phytol have been reported in Arabidopsis associated with α-oxidation [14], as well as in the degradation of pseudouridine, a modified nucleoside that occurs in noncoding RNAs and mRNAs [15]. The biosynthesis of benzaldehyde via β-oxidation, which acts in chemical communications as a sex-alarm pheromone, defends against arthropods and also acts as a pollinator attractant, is also associated with peroxisomes [17] (Table 1). In addition to ROS metabolism and fatty acid β-oxidation, peroxisomes from mammals and plant cells share other important processes such as ureide metabolism, polyamine and amino acid catabolism, biotin biosynthesis, among others (Table 1; [8,5,6,10]), mainwhile some others are exclusive of mammals cells such as bile acids and pyrimidine biosynthesis, prostaglandine degradation, between others [10].

Peroxisomes are highly dynamic organelles which can experience changes in abundance, shape, and protein content in response to the overproduction of H_2O_2. Plant peroxisome abundance is regulated by biogenesis, stress response-related proliferation, and pexophagy, which is a selective autophagic degradation of peroxisomes involved in removing obsolete and damaged peroxisomes [8,18]. Peroxines (PEXs) are proteins involved in peroxisome biogenesis and maintenance [3,8]. Peroxisomal membrane proteins (PMPs) imported into Arabidopsis require peroxin PEX19, which acts as a chaperone for PMPs in the cytosol, as well as PEX3 acting as the membrane anchor [8,5]. Peroxisomal matrix proteins containing the C- and N-terminal targeting signals PTS1 and PTS2, respectively, are imported by the soluble receptors PEX5 (for PTS1) and PEX7 (for PTS2) located in the cytosol [8,5]. Under stress conditions, peroxisome populations can increase through a process called proliferation, which starts with peroxisome elongation, followed by constriction, and ends with fission. This process is controlled by the PEX11 family of proteins, with five isoforms (PEX11a to PEX11e) being present in Arabidopsis, in addition to dynamin-related proteins (DRPs) and fission proteins (Fis1) [8]. Peroxisome proliferation is a rapid response to different abiotic stresses and is regulated by ROS (reviewed in Ref. [3]). However, NO has recently been reported to be also involved in regulating peroxisome proliferation in response to Cd, probably through the regulation of peroxisomal enzymatic antioxidants, such as CAT, and thus also regulates H_2O_2-dependent signalling [19]. GSNO also rescues peroxisome numbers and activity defects in *PEX1G843D* mild Zellweger syndrome fribroblasts [20]. Plant peroxisome proliferation could be a strategy to protect against ROS overflow in the cell as result of the highly efficient peroxisomal antioxidant defences, as evidenced by the link of abiotic stress tolerance and peroxisomal proliferation, such as during the protoplast transition from G0 to G1 [21]. In response to salt stress in Arabidopsis and *Oryza sativa,* peroxisome proliferation is associated with a reduction in both Na^+ accumulation and oxidative stress [22,23]. Peroxisome biogenesis and the induction of ROS production can regulate lignin, thus leading to the regulation of mesocotyl plasticity [24]. However, in quinoa plants, a negative correlation between peroxisome proliferation and the yields of plants exposed to heat, drought or both these conditions has been observed [25]. As mentioned above, excessive numbers of peroxisomes and obsolete or damaged organelles are eliminated in order to prevent metabolic disturbances and redox imbalances in the cell through selective autophagy (pexophagy) which is regulated by oxidative stress [18, 26–29]. Pexophagy, is especially frequent under biotic and abiotic stress conditions, but can also occur during cell differentiation and development and even under normal cellular conditions [18,28,30,31]. In plants, there are two main types of autophagy, microautophagy and macroautophagy, which in turn can be selective (pexophagy, chlorophagy, mitophagy, between others) or non-selective forms of authophagy [32]. Autophagy is a complex process which, in plants, requires more than 30 autophagy-related genes (ATG), thus suggesting a higher diversity of gene families, probably due to their sessile characteristics. The ubiquitin-like protein ATG8, which binds the growing

autophagosome membrane, is essential for selective cargo recruitment and autophagosome-tonoplast fusion [32]. However, specific peroxisomal autophagic receptors have not been clearly identified [18,31]. The signals determining plant peroxisome degradation have not either been fully established, although ROS and oxidative stress factors, such as CAT and peroxisomal oxidation, have been reported in different studies [18,26,28]. Thus, Calero et al. [28] have observed an increase in total carbonylated proteins in seedling extracts of Arabidopsis plants under treatment with 100 μM Cd over different periods of time (0–24 h); using the H_2O_2 biosensor HyperAs, the authors imaged a clear increase in H_2O_2 and peroxisomal oxidation after 4 h of Cd treatment [28]. Interestingly, peroxisome autophagy can negatively self-regulate due to the chaperone activity of the peroxisomal protease LON2 [33,34]. Therefore, plant adaptation to adverse conditions would require a fine-tuning regulation of peroxisome quality and abundance probably to accurate H_2O_2 homeostasis regulation.

This review updates our knowledge of the metabolism of peroxisomal reactive species, including ROS, RNS, NO, CRS and SRS, as well as their integration into the peroxisomal reactive species interactome (RSI), in addition to its role in the perception of environmental changes and the regulation of cell responses to promote resistance to different stress conditions. Particular attention will be paid to the role of H_2O_2 from peroxisomes in regulating the transcriptional responses of plant cells to different abiotic stress conditions and to the characterization of specific peroxisomal footprints.

2. Peroxisomal ROS metabolism

Reactive oxygen species (ROS) include different reduced derivatives of molecular oxygen which occur as a normal consequence of aerobic life through sequential O_2 reduction, giving rise to superoxide ($O_2^{\cdot-}$), hydrogen peroxide (H_2O_2), and hydroxyl (·OH) radicals. Other ROS include alkoxyl radicals (RO·), peroxyl radicals (ROO·), and electronically excited oxygen species singlet oxygen ($^1O_2$ (Table 2, [3,35])). The chemical reactivity and biological functions of ROS differ and, depending on their concentrations, can participate in signalling processes, as well as in cellular damage and death [35]. Peroxisomes, one of the main sources of cellular ROS production, are also some of the most oxidized cellular organelles. with a redox potential of approximately −360 mV, followed by chloroplasts, with a redox potential of −350 mV under light and of −306 mV under dark conditions; meanwhile, mitochondria yielded values of approximately −330 mV and −300 mV for the cytosol [36,37]. However, peroxisomes have a highly efficient antioxidant defence system to balance ROS levels, enabling these reactive species to regulate different metabolic pathways and signalling networks [3]. In plant peroxisomes, superoxide radicals are produced in the lumen during ureide and nucleic acid catabolism by xanthine oxidoreductase (XOR) and urate oxidase (UO) reactions, in sulfite oxidation catalysed by sulfite oxidase (SO), which also produced H_2O_2 and, in the peroxisomal membrane, via the NADH/NADPH-dependent electron transport chain (reviewed in Ref. [3]). Though less well understood than H_2O_2, $O_2^{\cdot-}$ reactivity probably affects the redox state of transition metal ion complexes in proteins and Fe–S clusters [35]. Superoxide accumulation is enzymatically regulated by superoxide dismutases (SODs), which dismutate superoxide to H_2O_2 and O_2. Different SODs, including CuZn-SOD, Mn-SOD and Fe-SOD, have been reported in plant peroxisomes depending on the plant species studied; thus pea leaf peroxisomes contain Mn-SOD and CuZn-SODs, while Arabidopsis contains only CuZn-SOD (CSD3) [38].

Table 2
Reactive Oxygen Species, Reactive Nitrogen Species, Reactive Sulfur Species and Reactive Carbonyl species.

Reactive Oxygen species	Reactive Nitrogen Species	Reactive Sulfur Species	Reactive Carbonyl Species
Superoxide radical, $O_2^{\cdot-}$	Nitric oxide, NO	Hydrogen sulfide, H_2S	Methylglyoxal, MGO
Hydrogen peroxide, H_2O_2	Nitrogen dioxide, NO_2	Sulfenic acid, RSOH	4-hydroxy-trans-2-onenal, 4-HNE
Hydroxyl radical, HO·	Dinitrogen trioxide, N_2O_3	Disulfide-S oxides, RS(O) 2SR	Malondialdehyde, MDA
Lipid peroxide, LOOH	Peroxynitrite, ONOO·	Polysulfide, RSSnR	Acroleina, C_3H_4O
Alcoxyl radical, RO·	Nitroxyl, HNO	Thiyl radical, RS	
Peroxyl radical RO_2·	Nitrite, NO_2^-	Disulfane H_2S_2	
Singlet oxygen $^1O_2$		Persulfide radical RSS·	

In photosynthetic tissue, peroxisomes are the largest source of H_2O_2, which is mainly associated with glycolate oxidase (GOX), a key enzyme in the photorespiration cycle [39]. Photorespiration is a compartmentalized process requiring physical contact between the chloroplast, peroxisome, mitochondrion and cytosol, in which GOX oxidizes glycolate, giving rise to glyoxylate and H_2O_2 [39]. Arabidopsis contain five glycolate oxidases, GOX1, GOX2, GOX3, HAOX1 and HAOX2 [40,41]. The induction of photorespiration-dependent H_2O_2 production occurs under different environmental stress conditions, including heat and drought in wheat [42], and drought in the sahariam plant *Oudneya africana* [43], heavy metals in pea and Arabidopsis leaves [44,45], high light and temperature in rice plants [46] and nonhost resistance in *Nicotiana benthamiana* and Arabidopsis [47,48]. Interestingly, the barley stripe mosaic virus (BSMV) Ƴb protein interacts with the host plant's GOX genes in order to inhibit GOX-derived H_2O_2 production, while Ƴb-GOX complexes accumulate near BSMV replication sites in vivo, thus facilitating BSMV infection [49].

Another source of H_2O_2 in peroxisomes is Acyl-coA oxidase (ACX), which is a key fatty acid β-oxidation enzyme. This process, which is mainly associated with seed germination, also plays an important role in the synthesis of the hormones, indole acetic acid (IAA), jasmonic acid (JA), ubiquinone, benzoic acid/benzaldehyde and phenylpropanoids, in green tissues [5,9,12]. In Arabidopsis, ACX is a family of six enzymes (ACX1-6) with overlapping specificities for Acyl-CoA substrates and different fatty acid chain lengths [50]. Upregulation of ACXs and other enzymes from the β-oxidation pathway has been reported in response to heat shock and drought conditions in wheat [51]. ACX3 upregulation has been observed in response to salinity and drought in cotton [52], and in *Camellia sinensis CsACX1* was upregulated by the insects *Trichoplusia ni* and *Empoasca (Matsumurasca) onukii and* the constitutive expression of *CsACX1* rescued wound-related JA biosynthesis in Arabidopsis mutant *acx1* [53]. The herbicide 2,4-D also upregulated ACX activity in pea leaves [54], as well as *ACX1* expression in Arabidopsis plants being in both cases involved in epinastic phenotype [55]. Polyamine catabolism is an additional peroxisomal source of H_2O_2 [13]. Copper amine oxidases (CuAOs) oxidatively deaminate polyamines to produce H_2O_2, which plays an essential role in plant responses to abiotic stresses and pathogen interactions [13]. In Arabidopsis plants, CuAOζ-dependent H_2O_2 is required for indole-3-butyric acid (IBA)-induced lateral root (LR) development [56]. Arabidopsis peroxisomal sarcosine oxidase (SOX; [57]) catalyses the degradation of l-pipecolate to Δ1-piperideine-6-carboxylate with H_2O_2 production and, to a lesser extent, the oxidation of sarcosine to glycine, formaldehyde, and H_2O_2 [57]. Finally, SODs are another potential source of H_2O_2 as described above.

Hydrogen peroxide can be accumulated in specific peroxisomal sites adjacent to those in contact with other organelles, such as chloroplasts or vacuoles, probably related to interorganelle cross-talk [58]. Hydrogen peroxide promotes the reversible oxidation of cysteines to sulfenic acid (-SOH) which is highly reactive and can be overoxidized to sulfinic acid (SO_2H) and, under excessive H_2O_2 accumulation conditions, to sulfonic acid (SO_3H), which irreversibly inactivates proteins [35,59]. Sulfenic acid also reacts with a free thiol group on the same or different protein molecule, giving rise to the formation of intramolecular

and intermolecular disulfides (S–S). Given their transient nature due to subsequent redox relay mechanisms, often involving peroxiredoxins and tioredoxins, these cysteine redox modifications are regarded as redox switch [59]. These redox switches regulate protein activity and constitute a basis for regarding H_2O_2 as an essential redox signalling molecule [35,59].

Peroxisomal catalase (CAT) can modulate H_2O_2 levels in peroxisomes, thus facilitating the regulation of its signalling function or as a pro-oxidant molecule. CAT is one of the most abundant peroxisomal proteins (10%–25%) and the most important defense against photorespiratory H_2O_2 [60]. Arabidopsis plants contain three CAT genes, *CAT1-CAT3*, with *CAT2* being the most active in green tissues (almost 80% of CAT activity). Arabidopsis *cat2* mutants, which accumulate H_2O_2 under high-light and ambient CO_2 conditions, present approximately 400 oxidized proteins containing methionine sulfoxide [61]. Interestingly, CAT interactome is very complex and several proteins have been shown to interact with this antioxidant. Thus, GOX physically interact with CAT2 in rice leaves, which are regulated by salicylic acid (SA) [62], indicating that CAT2 plays an important role in the regulation of photorespiratory-dependent H_2O_2 signalling. The connection between GOX and CAT also plays an important role in the regulation of stomatal movements in Arabidopsis plants [63]. A protective association between CAT and isocitrate lyase has also been observed in castor bean glyoxysomes, suggesting that glyoxylic acid and seed germination are regulated by CAT [64]. Likewise, CAT interacts with and regulates ACX2 and ACX3 activity and also modulates the SA-mediated regulation of JA biosynthesis in Arabidopsis plants infected by biotroph pathogens [65]. In the same plant species, the peroxisome-localized small heat shock protein Hsp17.6CII interacts with CAT2 in the peroxisome and enhances its activity to protect against abiotic stresses [66], while HSP90.9 interacts with CAT1 and enhances its activity [67]. A novel CAT2-interacting protein, leucine aminopeptidase 2 (LAP2), in Arabidopsis can lead to a mutual enhancement of both proteins in response to salt and osmotic stress conditions, while loss-of-function mutant *lap2-3* is hypersensitive to both abiotic stress [68]. Despite its peroxisomal location, there is increasing evidence of CAT's additional locations outside peroxisomes, although the reasons for this and the mechanism involved remain unclear [69]. The Arabidopsis salt overly sensitive 2 (SOS2) gene, which is required for salt tolerance, can interact with CAT2 and CAT3 and nucleoside diphosphate 2 in response to salt stress and H_2O_2 metabolism [70]. An Arabidopsis zinc finger protein lesion simulating disease 1 (LSD1) can interact with all three catalases in the cytosol to maintain catalase activity under hypersensitive cell death conditions [71]. Moreover, in Arabidopsis, no catalase activity 1 (NCA1), which interacts with all three catalases in the cytosol, is required for catalase activity during responses to multiple stresses, such as salt, cold, and alkaline, as well as for autophagy-dependent cell death [72,73]. Arabidopsis CAT3 also interacts with the cytosolic calcium-dependent kinase CDPK8 which boosts its activity [74]. CAT A in rice interacts with the plasma membrane-associated calcium-dependent kinase OsCPK10, which increases CAT activity [75]; in addition, CAT C interacts with the receptor-like cytoplasmic kinase STRK1 which phosphorylates and activates CAT C [76]. Changes in the location of Arabidopsisis CAT3 from the peroxisome to the nucleus in response to the cucumber mosaic virus (CMV) 2b protein have been reported to cause H_2O_2 accumulation and subsequent necrosis in infected Arabidopsis leaves [77,78]. However, CAT2 may also be translocated to the nucleus independently of any biotic factor, as reported recently by Al-Hajaya et al. (2022) [69], who demonstrated that CAT translocation to the nucleus is due to an alteration in the C terminal, which could affect CAT tetramerisation and thus its interaction with the peroxisomal importomer PEX5 [69]. Increasing evidence in mammalian cells suggests that oxidative stress at the cellular level might possibly regulate imports of CAT into the peroxisomes, thus enabling this protein to be relocated in the cell in order to increase extra-peroxisomal catalase activity [79]. Human PEX5 Cys-11 appears to act as a redox switch that modulates the import of peroxisomal matrix proteins such as CAT [79]. In mammalian cells, oxidative stress boosts the phosphorylation of PEX14 which disrupts the interaction of CAT with the Pex14-Pex5 importomer complex, thus boosting CAT accumulation in the cytosol [80]. However, in plant cells, the redox-dependent mechanism of CAT's location in the cytosol is not fully understood.

With a Km of around 43 mM, the affinity of CAT for H_2O_2 is low, which reduces its efficiency in controlling H_2O_2, suggesting that H_2O_2 diffuses into the cytosol under certain oxidative stress conditions [81]. However, peroxisomes have other defences against H_2O_2, mainly the ascorbate–glutathione cycle, which is composed of several enzymatic antioxidants. The peroxisomal matrix in Arabidopsis contains two ascorbate peroxidases (APX; APX3 and APX5), one monodehydroascorbate reductase (MDHAR1), one dehydroascorbate reductase (DHAR1), one glutathione reductase (GR1), as well as the non-enzymatic antioxidants, ascorbate (ASC) and glutathione (GSH), which control H_2O_2 levels in the peroxisomal matrix [5]. In pea leaves, MDHAR and APX activities are also associated with the peroxisomal membrane [82]. The higher affinity of APX, as compared to CAT, enables H_2O_2 leakage from peroxisomes to the cytosol to be regulated [83]. Therefore, both CAT and APX could contribute to the determination of the signalling role of peroxisomal H_2O_2.

The presence of other peroxidases in peroxisomes, such as peroxiredoxins, cannot be ruled out [84,85]. Peroxiredoxins are a family of thiol-based peroxide reductases with broad substrate specificity, ranging from hydrogen peroxide to alkyl hydroperoxide and peroxinitrite [86,87]. Recently, the orthologs AtTPX1 and AtTPX2 of peroxisomal peroxiredoxin 5 (PRDX5) from human cells were detected in Arabidopsis [88], although their location in peroxisomes has not been precisely established so far. A bioinformatics analysis of plant peroxisomal proteins has identified glutathione peroxidase 2 as a putative peroxisomal protein [89]. Additionally, using Strep-tagged recombinant proteins in Arabidopsis plants, the presence of three glutathione-S-transferases (GSTT1, GSTT2 and GSTT3) in peroxisomes has been reported [90]. GSTs, which require GSH, are involved in the detoxification of xenobiotic compounds and lipid peroxides. As these enzymes have been shown, in an in vitro analysis, to have high GSH-dependent peroxidase activity towards linoleic acid peroxide, they could protect the peroxisomal membrane against damage caused by fatty acid oxidation (Fig. 1) [90,91].

Other ROS, such as hydroxyl radical ·OH and $^1O_2$, can also be produced in peroxisomes through Haber-Weiss and Fenton type reactions [38,92] which require Fe/Cu ions, H_2O_2 and $O_2^{\cdot-}$ or ASC, thus promoting lipid peroxidation and carbonyl radicals, considered to be new players in the peroxisome-derived signalling network [38].

3. Peroxisomal metabolism of reactive nitrogen species (RNS)

Nitric oxide (NO), a highly reactive gaseous free radical, is a well-known signalling molecule in plants. Over the last twenty years, increasing evidence has demonstrated that NO is involved in a large number of plant processes, ranging from development and defence responses to both biotic and abiotic stresses; however, its metabolism, particularly in plant peroxisomes, has not been fully elucidated [93]. Peroxisomal NO production is associated with a NO synthase-like activity (NOS-l) [94], although neither the protein nor genes have so far been identified. In *Arabidopsis thaliana,* the conversion of IBA into IAA by β-oxidation is also involved in NO production [95], as well as polyamine catabolism [96], although no specific proteins have yet been identified. Xanthine oxidoreductase (XOR) appears to be another source of NO in animal cells [97] and in lupin roots the increase of NO production under phosphorus deficiency is associated to XOR, because its inhibitor allopurinol, markedly reduced NO production [98]. Other nitrogen-derived species, such as peroxynitrite ($ONOO^-$), resulting from the reaction of NO with $O_2^{\cdot-}$, as well as S-nitrosoglutathione (GSNO), which result of the combination of NO and GSH, and is considered a

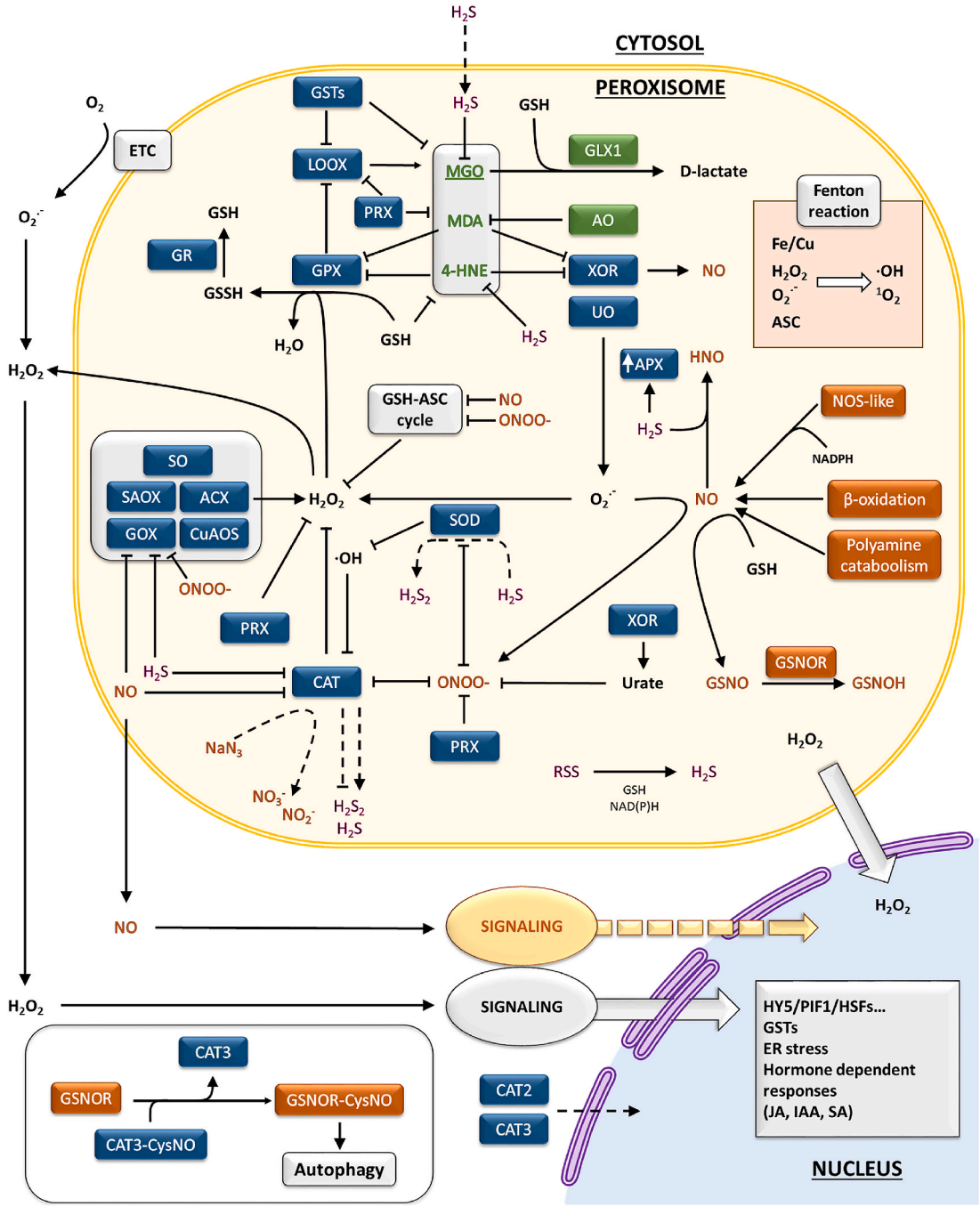

Fig. 1. Hypothetical model of reactive species interactome in peroxisomes and redox-dependent signalling network.The scheme shows a summary of the main endogenous sources of ROS, RNS, RSS, and RCS in peroxisomes and the interaction of different reactive species between themselves and with target molecules related to ROS and NO metabolism and peroxisomal dependent signalling transduction. ACX, acyl CoA oxidase; APX, ascorbate oxidase; ASC, ascorbate; AO, aldehyde oxidase; CAT, catalase; CuAOs, Copper amine oxidases; ER, endoplasmic reticulum; GLX1, Glyoxalase 1; GOX, glycolate oxidase; GR, glutathione reductase; GSH, reduced glutathione; GSSG, oxidized glutathione; GPX, glutathione peroxidase; GSNO; nitrosoglutathione; GSNOH, nitrosothiols; GSNOR, S-nitroso-glutathione reductase; GSNOR-CysNO, GSNOR *S*-nitrosylated; GST, glutathione-S-Transferase; HSFs, heat shock factors; 4-HNE, 4-hydroxy-(E)-2-nonenal; IAA; indolacetic acid; JA, jasmonic acid; LOOX, lipid hydroperoxide; MDA, malondialdehyde; MGO, methylglyoxal; NO, nitric oxide; NOS-like, nitric oxide oxidase like activity; NO_3^-, nitrate; NO_2^-, nitrite; $ONOO^-$, peroxinitrite; PRX, peroxiredoxin; RCS, reactive carbonyl species; RNS, reactive nitrogen species; ROS, reactive oxygen species; RSS, reactive sulfur species; SA, salicylic acid.; SO, sulfite oxidase.SOD, superoxide dismutase; UO, urate oxidase; XOR, xanthine oxidoreductase. Dashed lines represents reactions not demonstrated in vivo in plant cells.

cellular NO reservoir, have been detected in plant peroxisomes (Table 2) [99,100]. S-nitrosoglutathione reductase (GSNOR), which maintains a balance between NO and S-nitrosothiol levels, has been proteomically identified as a putative peroxisomal protein in plants [89]. Up to now, the principal mode of action of NO in plants has been through nitric oxide-derived post-translational protein modifications (PTMs) such as S-nitrosylation (or S-nitrosation), which forms a nitrosothiol in a Cys residue, leading to changes in the function, location, and/or stability of proteins [89,93]. $ONOO^-$, a strong oxidant molecule, induces PTMs through tyrosine nitration (NO_2-Tyr), thus affecting protein activity and/or stability. These modifications are also associated with NO_2 [89]. The peroxisomal targets of NO-dependent PTMs were obtained

following a proteomic bioinformatics analysis of PTMs including large-scale S-nitrosylation and nitration in Arabidopsis plants [89]. This analysis shows that approximately 8.4% of putative peroxisomal proteins can be targets of S-nitrosylation, while only 1.6% are targets of Tyr-nitration [89]. Most peroxisomal targets of S-nitrosylation are related to antioxidant defences (DHAR1, GR1, and CAT3), photorespiration (alanine-2-oxoglutarate aminotransferase 2, HPR1, alanine: glyoxylate aminotransferase2, glutamate: glyoxylate aminotransferase1), the glyoxylate cycle (isocitrate dehydrogenase, NAD-malate dehydrogenase 1 and 2, and citrate synthase 2); β-oxidation (ACX2,3 and 6, acyl-CoA synthase, 3-ketoacyl-CoA thiolase 3 and 4), the pentose phosphate cycle (6-phosphogluconate dehydrogenase family protein), the serine/threonine protein phosphatase 2A and nucleoside diphosphate kinase [89]. Additionally, a proteomic analysis of purified peroxisomes from pea leaves using the biotin switch method to identify S-nitrosylated proteins identified six targets in these organelles such as hydroxipyruvate reductase (HPR), GOX, serine:glyoxylate aminotransferase, aminotransferase 1, MDH and CAT, with CAT, MDH and GOX activity being negatively affected [99]. In general, PTMs dependent on NO and $ONOO^-$ are associated with the inhibitory effects of peroxisomal targets, mainly enzymes related to ROS metabolism and H_2O_2 homeostasis [24,89,99] thus suggesting that NO plays an important role in regulating H_2O_2-dependent signalling networks. It has been shown that NO regulates peroxisomal-dependent signalling and dynamics by using Arabidopsis mutants and treatments that alter general NO level [24] and regulates organelle metabolism by identifying NO targets in the organelle [99]. Direct signalling due to NO produced in peroxisomes however, is difficult to find out as the NO-source in plant peroxisomes has not yet been identified.

4. Metabolism of reactive carbonyl species (RCS) in peroxisomes

Reactive carbonyl species (RCS) are formed mainly through the oxidative degradation of lipid peroxides (LOOH), most of which are generated by the oxidation of polyunsaturated fatty acids by ROS, in particular ·OH, or in the reaction catalysed by lypoxigenases [101]. Oxidized fatty acids are decomposed to a variety of aldehydes and ketones (designated as "oxylipin carbonyls"). In plants, 19 different RCS, including 4-hydroxy-(E)-2-nonenal (HNE), acrolein, malondialdehyde (MDA) and methylglyoxal (MGO), have been identified using high-performance liquid chromatography (HPLC; Table 2) [101]. RCS participate in Michael addition reaction in which the electrophilic β carbon of an RCS molecule reacts with the thiol sulfur, amino nitrogen, or the imidazole τ-nitrogen atom to form a covalent bond and adding a carbonyl moiety to the protein, giving rise to protein carbonylation [102]. Additionaly, RCS can form a Schiff's base with an amino group [102]. Protein carbonylation can also take place directly through the reaction of proline, lysine, arginine, and threonine with ·OH produced in Fenton-type reactions [89,103]. The carbonylated protein proteome in peroxisomes, which has been analysed in different plant species under different oxidative stress conditions, includes proteins associated with antioxidant defences (CAT, GR, Mn-SOD), the glyoxylate cycle (isocitrate lyase and malate synthase), fatty acid β-oxidation (AIM1) and cell death (nitrilase) [89,102]. The increase in carbonylated proteins in peroxisomes has been observed under different stress conditions associated with an increase in H_2O_2, such as cadmium toxicity [104], treatment with Cu and ascorbate to induce Fenton-derived ·OH production [105], senescence processes [106], and treatment with the herbicide 2,4-dichlorophenoxiacetic acid [107]. Peroxisomal HNE-modified proteins, including nitrilase, nitrile-specifier protein 5, glycine-rich RNA-binding protein 7, and nucleoside diphosphate kinase, were analysed under combined salt and continuous light treatment in Arabidopsis plants [108]. Carbonyl groups are regarded as oxidative stress markers giving rise to the inactivation and further degradation of proteins [104]. In peroxisomes purified from pea leaves exposed to Cd treatment for 14 days, serine protease activities, capable of acting on carbonylated proteins, were identified [104]. In senescent pea leaves, CAT, GOX and glucose 6-P dehydrogenase (G6PDH) have been identified as some of the targets of serine proteases [109]. Peroxisomes can also use the cytosolic ubiquitin (Ub) 26S proteasome system to degrade ubiquitinated proteins in order to maintain peroxisomal quality. Plant peroxisomes contain several peroxins (PEXs), such as PEX4 [110], which can act as Ub-conjugating enzymes (E2s), while PEX2, PEX10, and PEX12 have been suggested to have in vitro E3 activity [31,111]. These proteins, which are involved in polyubiquitinating PEX5 for degradation by the cytosolic proteasome system, may participate in the ubiquitination of other peroxisomal proteins. The in silico analisis of peroxisomal ubiquitinated proteins in Arabidopsis has identified APX3, glutamate: glyoxylate aminotransferase, citrate synthase 3, 3-ketoacyl-CoA thiolase 3, alternative NAD(P)H dehydrogenase1, sulfite oxidase, 6-phosphogluconate dehydrogenase and cysteine proteinases, among others [89]. In fact, isocitrate lyase (ICL) and malate synthase (MLS) are stabilized in Arabidopsis mutants *pex4, pex22*, *pex2* and *pex10* (review in Ref. [31]). Ubiquitinated proteins would require the retrotranslocation of matrix proteins from the organelle in order to be proteasome targets (review in Ref. [31]. In peroxisomes from yeast, fungi and mammal cells, LON proteases, which have both chaperone and protease functions, can selectively degrade oxidatively damaged peroxisomal matrix proteins and may play a key role in the coordination of peroxisomal ROS metabolism [112,113]. In Arabidopsis plants, MLS and ICL have been reported to be LON2 substrates [31]. Alternatively, as previously observed in Arabidopsis roots exposed to H_2O. or methyl viologen, oxidized proteins could be removed by autophagy [114].

However, increasing evidence shows that RCS can act as a link between ROS stimuli and cellular responses which play a role in signalling processes in both plant and animal tissues (reviewed in Ref. [102]). However, specific information regarding signalling derived from peroxisomal RCS is not available for plants. Several enzymatic and non-enzymatic scavengers of RCS and carbonyls have been reported in plants, which include the low molecular weight GSH and the enzyme glutathione S-transferase (Fig. 1); both are present in peroxisomes and can efficiently trap RCS by forming an RCS-GSH conjugate [102]. The presence in Arabidopsis peroxisomes of glyoxalase [89,115], which detoxify methylglyoxal (MGO), strongly suggests that CRS are formed in peroxisomes (Fig. 1). Other enzymes involved in detoxifying CRS such as aldehyde oxidases may also participate in CRS detoxification in peroxisomes [102].

5. Metabolism of reactive sulfur species (RSS) in peroxisomes

The term reactive sulfur species (RSS) refers to sulfur-related molecules produced by the reaction of thiols with reactive oxygen and nitrogen species, including disulfide-S oxides [$RS(O)_2SR$], sulfenic acids (RSOH), thiyl radicals (HS·), disulfane (H_2S_2), polysulfides (H_2Sn) and persulfide radicals (RSS·) (Table 2) [116]. These RSS can indeed act either as potent oxidants or reductants and modulate the redox status of biological thiols and disulfides [116]. The chemical properties of RSS, such as nucleophilicity, electrophilicity, its pKa, and bond strengths, can be modulated by interactions with other redox-active systems, thus making RSS ideal for many roles in biological signalling [116,117]. H_2S is probably the most studied sulfur-derived molecule in animal and plant systems; rather than a toxic compound, at low concentrations, H_2S exerts beneficial effects and is now recognised as a key messenger equal in importance to NO and H_2O_2 in a huge number of physiologically important processes [117,118]. The endogenous production of H_2S in plant cells is associated with the biosynthesis and metabolism of cysteine in chloroplasts through the action of sulfite reductase and L-cysteine desulfurases (NifS-like and also present in mitochondria) and in the cytosol through L-cysteine (l-CDES) and D-cysteine (d-CDES) desulfhydrases (reviewed in Ref. [117]). Mitochondria are also a source of H_2S through O-acetylserine (thiol) lyase (OASTL) activity during the detoxification of cyanide by the action of β-cyanoalanine synthase (CAS)

(reviewed [117]). However, no specific source of H_2S has been identified in peroxisomes, although the identification in these organelles of putative protein targets of persulfidation suggests the presence of H_2S in peroxisomes, probably as a result of permeation from the cytosol. However, analyses in vitro using commercial protein carried out by Olson et al. (2017) [118] have shown that H_2S may be generated by CAT under hypoxia requiring NADPH, although this has yet to be demonstrated in vivo (Fig. 1). In mammals cells nonenzymatic production of H_2S could take place in a process that requires reducing equivalents such as NADPH and NADH and GSH, thus reducing RSS to H_2S and other metabolites (reviewed in Ref. [119]) (Fig. 1). With regard to its precise mechanism of action, H_2S shows a high affinity for metalloproteins by covalently binding to the metal and is also involved in the PTM persulfidation which oxidizes cysteine thiol groups to persulfide groups (R-S-SH; reviewed [117]). Persulfidation requires an oxidant, as H_2S cannot directly modify cysteine residues to form a persulfide group. Thus, the thiolate (HS-) can react with cysteine sulfenic acid (R–SOH) to form cysteine persulfide (R–SSH) [117]. The oxidative environment of peroxisomes, where reactive oxygen and nitrogen species are produced, may therefore be appropriate to form these oxidized intermediaries to enable persulfidation. Proteomic analysis carried out in Arabidopsis plants has identified 61 putative persulfidated proteins associated with peroxisomes including the antioxidants CAT3 and Cu–Zn-SOD3 (SOD3); fatty acid oxidation enzymes such as ACX1, 3, 4, and 6 and two enoyl-CoA hydratase/isomerase enzymes (AIM1/ECHIA) and sulfite oxidase [89,120]. Altogether, this means that persulfidation could regulate ROS production and ROS scavenging in peroxisomes and therefore determine peroxisomal H_2O_2-dependent signalling processes in response to stress conditions.

6. Peroxisomal reactive species interactome

Plants are exposed to continuous changes in their environment and are then prone to damage caused by abiotic and biotic factors. An effective buffer against environmental threats requires a perfect equilibrium between sensing, adaptation, defence, and repair systems to finally overcome damage and prevent plant death. Biological plasticity in response to changeable environmental conditions would provide resilience to different types of adverse factors. The chemical properties of reactive species constitute a good mechanism to integrate the perception of external signals by fine-tuning different biochemical reactions to facilitate metabolic flexibility [121] and signal transduction, giving rise to changes in gene expression. Peroxisomes can sense ROS/redox changes in the cell as a result of changes in their environment and thus trigger rapid and specific responses to environmental cues involving ROS-dependent signalling networks associated with different metabolic pathways and changes in peroxisomal dynamics [3,38]. However, the mechanisms involved have not yet been fully established.

The interaction (synergistic and antagonistic) between different reactive species has given rise to the concept of the reactive species interactome which was first developed in animal tissues as a new multilevel redox regulatory system that identifies the families of reactive species (ROS, RNS, RCS and RSS), their interactions with their respective downstream biological targets, mainly cysteine thiols in proteins (redox switches), as well other elements including membrane lipids and DNA [121]. Several examples of interaction between reactive species in animal and plant systems, such as the formation of $ONOO^-$ by the reaction of NO with $O_2^{\cdot-}$, have been reported (Fig. 1). As mentioned previously, although $ONOO^-$ promotes the Tyr nitration of proteins, there is a gap in the functional significance of this modification, which, in plants, is accepted as an irreversible reaction giving rise to protein inhibition and degradation by the proteasome [122]. In plant peroxisomes, as mentioned above, a proteomic analysis has identified several peroxisomal putative targets mainly related to ROS metabolism, with available data suggesting an inhibitory effect of this modification of antioxidant defences (CAT, MDAR, APX, CuZn,SOD) and ROS production (GOX) [89,99,123] (Fig. 1). Catalase could also be involved in RNS metabolism. Thus commercial CAT can oxidize sodium azide into at least nitrite (NO_2^-) and nitrate (NO_3^-), contributing to tyrosine nitration in the presence of H_2O_2 [124]. Peroxisomal SOD could regulate $ONOO^-$ accumulation by controlling $O_2^{\cdot-}$ availability, while CAT and peroxiredoxins could degrade $ONOO^-$, as reported in animal cells [125], thus playing a key modulatory role at the cross-roads between H_2O_2 and $NO/ONOO^-$-mediated signalling pathways (Fig. 1). Furthermore, urate, a metabolite of ureide metabolism in peroxisomes, is a well-known peroxynitrite scavenger [126,127] and may also contribute to the regulation of $ONOO^-$ in peroxisomes (Fig. 1). GSH can react with NO to form GSNO which acts as a NO reservoir and mediates protein S-nitrosation processes, but can also prevent excessive accumulation of S-nitrosylated proteins under oxidative stress conditions [128]. GSNOR, in turn, can be *trans*-nitrosylated by the non-canonical catalase CAT3 (described as a repressor of GSNOR1, ROG1), and degraded by autophagy (Fig. 1); thus, CAT3 positively regulates NO signalling, and, accordingly, Arabidopsis *rog1* mutants are more susceptible to NO than WT mutants [129]. Zhang et al. (2021) [128] have reported that glutathione is required to maintain GSNOR activity through denitrosylation and coordinates GSNOR activity with protein S-nitrosylation levels to ensure appropriate signalling strength involving salicilic acid (SA) pathways in response to H_2O_2 [129]). These results suggest that NO homeostasis and ROS/NO crosstalk are self-regulated (Fig. 1). The interaction between CAT and nitrate reductase (NR), the main source of NO in plants, has been described in cassava (*Manihot esculenta*) plants, as a mechanism of defence against the bacterium *Xanthomonas axonopodis* [130]. Both enzymes are coordinated by the transcription factor MeRAV5 which activates *MeNR* expression and inhibits MeCAT1 activity, thus regulating the H_2O_2/NO balance [130].

The carbonyl reactive species MGO, MDA, 4-HNE, and the enzymes GPX and PRX, which, in turn, regulate hydroxylipids, could act as a link between ROS, RNS, RSS, and RCS metabolism (Fig. 1). RCS could be oxidized by H_2O_2 and $ONOO^-$, giving rise to acetate, formate, and methyl radicals (reviewed in Ref. [131]). MGO can be detoxified in peroxisomes by glyoxalase (GLX1), requiring GSH as a cofactor, thus regulating not only oxidative damage but also GSH/GSSG homeostasis [132]. GLX, in turn, is sensitive to redox changes and is a target of sulfenilation [89]. Additionally, MDA and 4-HNE could inhibit XOR, glutathione peroxidases and peroxiredoxin activities, as reported in animal systems (reviewed in Ref. [131]) (Fig. 1). Aditionally, ·OH-dependent protein carbonylation can inactivate peroxisomal proteins associated to antioxidative defences, CAT and SOD, and in less extent malate dehydrogenase, which supply NADPH for metabolism and antioxidative defences as reported previously in peas, castor bean and Arabidopsis [104,105,108] (Fig. 1). All these reactions could take place in plant peroxisomes where all the actors mentioned are present, thus providing a fine tuned coordination of cell responses by cross-talk between different reactive species (Fig. 1).

H_2S plays a role in plant stress responses by enabling plants to adapt to adverse conditions and positively affects seed germination, root elongation, and survival under stress conditions (drought, salinity, hypoxia, heat, chilling and metal stress) by regulating defence responses through protein persulfidation [133,134]. Among the peroxisomal proteins identified as targets of persulfidation in Arabidopsis, there are two important antioxidants, SOD3 and CAT3; however, the effect of this PTM is unclear, with no effect being observed in SOD in the different plant species analysed [133]. H_2S donors negatively affect CAT activity, with increased APX and peroxidase in *Lycopersicum* plants observed in response to metal toxicity [133]. Recently, H_2S treatment has been reported to impact plant cellular redox homeostasis by repressing GOX activity probably by altering the levels of the major *GOX1* and *GOX2* transcripts, thus regulating photorespiration and peroxisomal H_2O_2 production and signalling [135]. Persulfidation of GOX may also be possible as proteomic analysis of persulfidated proteins from Arabidopsis has identified GOX as a target of this PTM, although its effect on

Table 3
Transcriptomic analyses related with ROS perturbed metabolism in plant peroxisomes . 2,4-D: 2,4-dichlorophenoxyacetic acid; ACX1: Acyl-CoA oxidase; AS: antisense; *Ath*: *Arabidopsis thaliana;* CAT: catalase; E3-SCF-FBOX: SKP1-CUL1-F-box E3 ligases complex; GOX1/2: glycolate oxidase 1/2; GR1: glutathione reductase 1; HL: high light; MAPK: mitogen-activated protein kinase; HSPs: heat shock proteins; *Nt: Nicotiana tabacum*; OXI1: oxidative signal inducible 1; SHR: short-root transcription factor.

Mutant/ Treatment	Stress	Main Results	Reference
CAT1-AS (*Nt*)	high light	HL induced photoinhibition in CAT1AS plants. Short HL exposure of CAT1AS plants induced tolerance to a subsequent severe oxidative stress	[161]
CAT2-AS *(Ath)*	high light	Photorespiratory H_2O_2 has a direct impact on transcriptional programs in plants	[162]
cat2 (Ath)	high light	Peroxisomal H_2O_2 plays a key role in the induction of small HSPs and regulation and biosynthesis of anthocyanin pathway	[163]
WT (Ath)	3-aminotriazole	Identification of novel H_2O_2-responsive genes involved in cell death process	[146]
cat2 (Ath)	different CO_2 conc. and photoperiod	Photorespiratory H_2O_2-dependent gene expression is significantly influenced by the photoperiod	[148]
cat2 (Ath)	Shift high CO_2/normal air	GR1 plays specific roles in intracellular H_2O_2 metabolism, in day length-linked control of phytohormones	[155]
cat2 (Ath)	Short/long day conditions	Increased peroxisomal availability of H_2O_2 induced resistance to biotic challenge, depending on both growth daylength and the isochorismate pathway of SA synthesis	[164]
cat2 (Ath)	Shift high CO_2/normal air	CO_2 and peroxisomal H_2O_2 have a key role in environmental responses	[165]
cat2 (Ath)	Shift high CO_2/normal air	H_2O_2 produced in peroxisomes induces transcripts involved in protein repair responses	[149]
cat2/gox1/gox2 (Ath)	high light, shift high CO_2/ normal air	GOX1 and not GOX2 attenuated the photorespiratory phenotype of *cat2* plants	[147]
cat2 (Ath)	high light, shift high CO_2/ normal air	SHR acts as a regulator of the photorespiratory redox homeostasis that integrates stress responses and development.	[157]
cat2/3; cat1/2/3	Long day	Peroxisomal H_2O_2 regulates plant growth, abiotic and biotic stress responses, probably through OXI1 and several MAPK cascades control in the catalase triple mutant	[166]
acx1 (Ath)	2,4-D	There is a peroxisomal footprint in early plant responses to 2,4-D being ACX1 a key sources of ROS. Peroxisomal (ACX1)-dependent signalling in plant responses to 2,4-D point to auxin signalling regulation and protein degradation associated with E3-SCF-FBOX as key processes in development of epinasty	[56]
35S:GOX2/gox2 (Ath)	Cd (100 μM)	Included in a meta-analysis with different transcriptomes related with peroxisomal H_2O_2-dependent signalling	[89]

enzymatic activity has not yet been established [120,135]. Persulfidation is believed to account for the protection against ROS/RNS, since persulfidated proteins react with ROS/RNS to form an adduct that may be restored by thioredoxin, thus recovering protein function [135]. However, in peroxisomes, the presence of thioredoxins has not been reported so far and the regulation of protein redox changes in these organelles needs to be elucidated. Interestingly, Olson et al. (2018) [136] have reported that commercial purified Cu,Zn-SOD from animal source can oxidize H_2S into polysulfides, probably as part of a mechanism to detoxify RSS (Fig. 1); therefore, SOD could be a key element in regulating ROS and RSS metabolism [136]. Analysis in vitro of purified CAT from animal tissues suggests that it can also act as a sulfide-sulfur oxido-reductase during normoxia and hypoxia, metabolizing H_2S with the participation of H_2O_2, and generating H_2S from dithiothreitol [118]. This reaction is inhibited by carbon monoxide and is potentiated by NADPH, suggesting that heme iron is the catalytic site with NADPH providing reducing equivalents [118]. Thus, ROS, RNS, and RSS metabolism could be regulated by catalase which itself can be regulated by all these reactive species [131]. Therefore, H_2S can cross-talk with and scavenge ROS and RNS, thus regulating their self-concentration and signalling-derived processes [134,137]. Interestingly, in rat vascular smooth muscle cells H_2S can directly react with MGO in a time- and concentration-dependent manner to limit its availability and prevent oxidative damage [138]. Interplay between H_2S and MGO regulates the thermotolerance in maize seedlings by regulating the activities of antioxidants such as CAT and APX, GR and the content of non-enzymatic antioxidants ascorbic acid, glutathione, thus regulating ROS accumulation [139]. In mammals tissues H_2S can also scavenge the aldehyde 4-HNE at physiological concentrations [131] (Fig. 1). Additionally, MDA and 4-HNE inhibit XOR activity in animal tissues through direct interactions with both oxidase and dehydrogenase forms, which can be prevented by GSH [131].

Interactions between the components of peroxisomal reactive species interactome, key biological targets of the antioxidant network and ROS sources are highly complex and contribute to the dynamic redox/ROS/NO homeostasis and plasticity of peroxisomes, enabling fine-tuned regulation of signalling networks associated with peroxisome-dependent H_2O_2 and perception of environmental cues and acclimation.

7. ROS perturbations in peroxisomes trigger retrograde signalling: transcriptomic analyses

ROS and redox changes play a decisive role in regulating plant development, especially in response to changes in the environment, at different levels, ranging from translation, protein post-translational modifications to transcription. Any environmental cue that enhances ROS production or down-regulates antioxidative enzymes would disrupt redox homeostasis and activate downstream ROS signalling [140]. Reactive species such as H_2O_2 participate in integrating external signal perception into the mechanisms available in the cell in order to regulate a complex cellular response to ensure viability under stressful conditions [35]. Signalling by H_2O_2 occurs through reversible oxidation of specific cysteines from proteins to sulfenic acid (RSOH) [35]. Due to their transient nature, these sulfur modifications, which can be reversibly reduced by thioredoxin and glutaredoxin pathways, are regarded as redox switches. These signalling events are coupled with metabolism, MAP kinase cascades, transcription factor regulation, transcriptomic changes, cytoskeleton changes, and the cell cycle, among others [35]. However, we can not rule out the possibility that H_2O_2 acts as a direct transducer due to its capacity to permeate membranes. Although the identification of retrograde signalling between chloroplasts, mitochondria and the nucleus have improved considerably thanks to the use of Arabidopsis mutants [141–143], little is known about peroxisome-dependent retrograde signalling. Peroxisomes, which are sources of signalling molecules, can play a key role in transcriptome adjustments, while the specific peroxisome-dependent imprint on the transcriptome response has been studied [144] (Table 3). In particular, the specificity of H_2O_2-dependent peroxisomal signalling has been demonstrated, mainly in relation to altered peroxisomal H_2O_2 levels induced either by chemical treatment with 3-aminotriazole (AT), a CAT inhibitor [145], or by disturbances in CAT activity under different stress conditions such as high or continuous light, CO_2 shifts and photorespiratory stress in Arabidopsis and *Nicotiana tabacum* plants (Table 3). H_2O_2-dependent signalling produced during peroxisomal

photorespiration in different photorespiratory mutants, such as Arabidopsis *gox1* and *gox2*, affected in glycolate oxidase, has also been analysed [88,146]. Furthermore, GOX-dependent signalling has been analysed in Arabidopsis *cat2* mutants [146]. Arabidopsis mutants lacking peroxisomal *CAT2* (*cat2-2*) displayed stunted growth and cell death lesions under ambient air, while the double mutant *cat2-2 gox1* showed an attenuated *cat2-2* photorespiratory phenotype. On the other hand, knocking out the virtually identical *GOX2* in the *cat2-2* background did not affect the photorespiratory phenotype, suggesting that GOX1 and GOX2 play distinct metabolic roles [146]. Transcriptomic analyses of both double Arabidopsis mutants show higher induction of transcripts in *cat2-2 gox1-1* as compared to *cat2-2,* mainly associated with glucosinolate and jasmonic acid biosynthesis, oxylipin metabolism, and wounding [146]. Meanwhile, transcripts, which are less induced by photorespiratory treatment in Arabidopsis mutants *cat2-2 gox1-1,* are associated with responses to high light, plant-pathogen interactions, salicylic acid-mediated signalling, ethylene biosynthesis, and programmed cell death regulation [146]. In contrast to *cat2-2 gox1-1*, the *cat2-2 gox2-1* transcriptional profile was largely comparable to that of *cat2-2* mutants [146]. Furthermore, H_2O_2 produced in peroxisomes in Arabidopsis *cat2* mutants induces transcripts involved in protein repair responses, heat shock proteins (HSPs) and proteins involved in ubiquitin-dependent protein degradation [147,148]; this suggests that peroxisomes are involved in acclimation and survival processes due to the induction of protein repair and protection processes under changing environmental conditions. Transcriptomic analyses of the triple mutant *cat1/2/3*, which shows redox disorders under control conditions, reveal strong disturbances in genes relating to growth regulation, plant responses to stress and Mitogen-activated protein kinases (MAPK) cascades [149].

In fact, several genes related to MAPK cascade pathways, such as *MPK11* and *MPK13*, as well as serine/threonine kinase oxidative signal inducible 1 (*OXI1*), are severely altered in the triple *cat* Arabidopsis mutant [149]. Recently, H_2O_2 produced by ACX1 has been shown to regulate genes related to IAA homeostasis, transport and signalling, which are involved in the epinastic phenotype in plant responses to the herbicide 2,4-D [55]. Furthermore, protein degradation by the E3-RING ubiquitin ligase and proteasome complex is also significantly regulated by ACX1-dependent H_2O_2 after short and long term exposure to the herbicide [55]. Recently, a bioinformatic analysis of most of the transcriptomic analyses shown in Table 3 has been carried out to identify common transcriptional footprints of plant peroxisome-dependent signalling under different conditions [88]. Peroxisomal footprints involve highly co-expressed genes, which are shared with transcriptomic responses to several abiotic stresses. Major late peroxisome-dependent gene clusters are associated with heat shock factors and proteins, as well as responses to ER stress and GSTs (Fig. 1), which are mainly involved in protein protection and detoxification [88]. At earlier stages, peroxisome-dependent genes are related to transcription regulation, hormone-dependent signalling and biosynthesis, particularly of JA, one of the phytohormones produced in this organelle (Fig. 1) [88]. Some processes, such as responses to stress and others, are maintained over time, suggesting that one of the main functions of peroxisomal retrograde signalling is to coordinate defence responses to prevent protein and cellular damage under adverse conditions [88]. The target genes HY5 and PIF1 are overrepresented in early peroxisome-dependent signalling, with HY5 being maintained later on (Fig. 1) [88]. In Arabidopsis plants, HY5 is a bZIP TF, which regulates a wide range of genes involved in plant responses to hormones and abiotic stresses such as cold and UV-B. The HY5 homolog (HYH) in the same species is associated with peroxisomal proliferation through activation of *PEX11b* via a phytochrome A-dependent pathway [158]). PIF1, which belongs to a small family of bHLH TFs, regulates the expression of multiple ROS-dependent genes. In Arabidopsis PIF1/PIF3 physically interact with HY5/HYH and regulate the transcription of some peroxisomal genes [159]; it has been suggested that the interaction PIF1/PIF3-HY5/HYH acts as a rheostat to fine-tune ROS-dependent signalling pathways [160]. Peroxisome-derived ROS signalling events would connect to MAPK cascades in the cytosol and transcription factors to regulate the expression of nuclear genes. However, the transducers, which relay signals from the peroxisome to the nucleus, have not been elucidated, although H_2O_2, as mentioned above, could also act as a direct transducer due to its ability to permeate membranes (Fig. 1). Specific peroxiporins have not been identified in plant peroxisomes, although H_2O_2 could use the porins described in plant peroxisomes (reviewed in Ref. [3]). The involvement of different PEX11 peroxins in yeast and mammalian cells in the formation of pores to transport H_2O_2 is under debate [161].

Different genetic studies using Arabidopsis double mutants that affect catalase activity and some components of the ASC–GSH cycle in the cytosol have demonstrated that light can regulate photorespiratory H_2O_2 peroxisome-dependent changes in redox homeostasis in the cytosol, giving rise to gene transcription regulation in the nucleus to promote acclimation or cell death, depending on the stimuli [146,153,156,162]. The 2A protein phosphatase subunit (PP2A-B'γ) could be an important player in controlling day length-dependent regulation of peroxisome-dependent H_2O_2 signalling, which is regulated by phytochrome A and repressed by IAA [163]. In turn, SA may modulate H_2O_2, subsequently conferring regulation on IAA [164].

In chloroplasts, the 3′-PHOSPHOADENOSINE 5′-PHOSPHATE (PAP) phosphatase SAL1 is one of the main players of retrograde signalling which acts as a sensor of ROS and oxidative stress in Arabidopsis [165], while HY5 acts as a transducer or effector [165]. The chloroplast GENOMES UNCOUPLED 1 (GUN1) is also involved in chloroplast retrograde signalling, which integrates several developmental and stress-related signals. Alternative oxidase 1 (AOX1a) has been used as a key marker of mitochondrial retrograde regulation, while the NAC transcription factor RAO2/Arabidopsis NAC domain-containing protein17 (ANAC017) has been identified as a master regulator of mitochondrial retrograde signalling [165]. ANA017 is located in the ER and, following activation by proteolysis, is translocated to the nucleus [165,166].

Several shared components between mitochondrial and chloroplast retrograde signalling pathways have been identified [165]. However, comparison of peroxisomal bioinformatics analysis with different transcriptomic data related to ROS showed no common genes with plastid retrograde signalling by GUN proteins [88,148] or with mitochondrial AOX1-dependent signalling [88,167]. However, as HY5 is common to peroxisomes and SAL1-dependent chloroplasts, it could act as a peroxisome transducer or effector, as has been reported with respect to chloroplasts [165], although this function in peroxisomes has not been demonstrated so far. This suggests that ROS signals derived from peroxisomes may differ from chloroplasts and mitochondria, although interconnections between ROS signals derived from different organelles cannot be ruled out [148,149]. Peroxisomal dynamic extensions termed peroxules, which are produce as a fast response to stress conditions invoving H_2O_2 production [168], may contribute to signal transduction by transferring H_2O_2 directly to the nucleus. This has been demonstrated in chloroplasts where stromules (dynamic structures similar to peroxules) can transfer H_2O_2 from chloroplasts to nuclei as part of a retrograde signalling process [169,170]. However, no direct evidence of a peroxule-nuclei contact site has been imaged so far. Interestingly, *PEX11a* RNAi Arabidopsis mutants which do not produce peroxules show disturbances in the expression of some genes regulated by ROS, such as *GST*, *CAT2*, *CuZn-SOD3 (CSD3)* and *redox responsive transcription factor1 (RRTF1),* during fast responses to cadmium exposure [168]. This fact suggests that peroxules could participate in transducing early ROS-dependent signalling against external stimuli.

Future perspective

Several questions should be answer in the future to improve our knowledge on peroxisomal contribution to redox biochemistry and

transcriptome regulation against environmental changes and development of resilience in plants. A deep characterization of peroxisomal reactive species interactome is required, such as identify H_2S production in peroxisomes, the sulfenylome and persulfidome of peroxisomal proteins and the interaction between all components, reactive species and their targets. The role of CAT in GSNOR transnitrosylation arise the question if other proteins could be transnitrosylated inside and outside of peroxisomes by this protein or others, and the functionality of this process. The existence of other transnitrosylases in peroxisomes is another question to be resolved. Deciphering peroxisomal NO sources and specific NO-dependent transcriptome is one of the more challenging issue in peroxisome and redox biology in plants. The identification of peroxisomal redox relay involved in retrograde signalling and the study of carbonylated and nitrated peroxisomal proteins and their function as signalling messengers are other exciting issues in cell biology that should be studied in depth in order to understand how peroxisome perceive, translate and regulate cell responses to environmental changes.

Submission declaration and verification

The manuscript has not been published previously and is not under consideration for publication elsewhere. The manuscript has been approved by all authors and responsible authorities from the CSIC and, if accepted, it will not be published elsewhere.

Authorship contribution statement

LMS conceived, design and written the manuscript. MCRM participated in writing and corrections and AMCA contribute with Tables and Figure edition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgement

This study was co-funded by the Spanish Ministry of Science, Innovation and Universities (MCIU), the State Research Agency (AEI) and European Regional Development Fund (FEDER) grants PGC2018-098372-B-I00, and PID2021-122280NB-I00, and Junta de Andalucía I + D + I grants P20-00364. The authors wish to thank Michael O'Shea for proofreading the manuscript.

References

[1] T. Gabaldón, Evolution of the peroxisomal proteome, Subcell. Biochem. 89 (2018) 221–233.
[2] D. Speijer, Oxygen radicals shaping evolution: why fatty acid catabolism leads to peroxisomes while neurons do without it: $FADH_2$/NADH flux ratios determining mitochondrial radical formation were crucial for the eukaryotic invention of peroxisomes and catabolic tissue, Bioessays 33 (2011) 88–94.
[3] L.M. Sandalio, M.A. Peláez-Vico, E. Molina-Moya, M.C. Romero-Puertas, Peroxisomes as redox-signaling nodes in intracellular communication and stress responses, Plant Physiol. 186 (2021) 22–35.
[4] C. Lismont, I. Revenco, M. Fransen, Peroxisomal hydrogen peroxide metabolism and signaling in health and disease, Int. J. Mol. Sci. 20 (2019) 3673.
[5] R. Pan, J. Liu, S. Wang, J. Hu, Peroxisomes: versatile organelles with diverse roles in plants, New Phytol. 225 (2020) 1410–1427.
[6] S. Reumann, B. Bartel, Plant peroxisomes: recent discoveries in functional complexity, organelle homeostasis, and morphological dynamics, Curr. Opin. Plant Biol. 34 (2016) 17–26.
[7] A.K. Werner, C.-P. Witte, The biochemistry of nitrogen mobilization: purine ring catabolism, Trends Plant Sci. 16 (2011) 381–387.
[8] Y.T. Kao, K.L. Gonzalez, B. Bartel, Peroxisome function, biogenesis, and dynamics in plants, Plant Physiol. 176 (2018) 162–177.
[9] D.A. Korasick, T.A. Enders, L.C. Strader, Auxin biosynthesis and storage forms, J. Exp. Bot. 64 (2013) 2541–2555.
[10] J.J. Smith, J.D. Aitchison, Peroxisomes take shape, Nat. Rev. Mol. Cell Biol. 1412 (14) (2013) 803–817.
[11] T.F. da Silva, V.F. Sousa, A.R. Malheiro, P. Brites, The importance of ether-phospholipids: a view from the perspective of mouse models, Biochim. Biophys. Acta 1822 (2012) 1501–1508.
[12] A. Block, J.R. Widhalm, A. Fatihi, R.E. Cahoon, Y. Wamboldt, C. Elowsky, S. A. Mackenzie, E.B. Cahoon, C. Chapple, N. Dudareva, G.J. Basset, The origin and biosynthesis of the benzenoid moiety of ubiquinone (coenzyme Q) in arabidopsis, Plant Cell 26 (2014) 1938–1948.
[13] W. Wang, K. Paschalidis, J.C. Feng, J. Song, J.H. Liu, Polyamine catabolism in plants: a universal process with diverse functions, Front. Plant Sci. 10 (2019) 561.
[14] W. Yang, P. Gutbrod, K. Gutbrod, H. Peisker, X. Song, A.L. Falz, A.J. Meyer, P. Dörmann, 2-Hydroxy-phytanoyl-CoA lyase (AtHPCL) is involved in phytol metabolism in Arabidopsis, Plant J. 109 (2022) 1290–1304.
[15] M. Chen, C.P. Witte, A Kinase and a glycosylase catabolize pseudouridine in the peroxisome to prevent toxic pseudouridine monophosphate accumulation, Plant Cell 32 (2020) 722–739.
[16] Z.J. Wright, B. Bartel, Peroxisomes form intralumenal vesicles with roles in fatty acid catabolism and protein compartmentalization in Arabidopsis, Nat. Commun. 111 (11) (2020) 1–13.
[17] X.Q. Huang, R. Li, J. Fu, N. Dudareva, A peroxisomal heterodimeric enzyme is involved in benzaldehyde synthesis in plants, Nat. Commun. 131 (2022) 1–15.
[18] A. Olmedilla, L.M. Sandalio, Selective autophagy of peroxisomes in plants: from housekeeping to development and stress responses, Front. Plant Sci. 10 (2019) 1021.
[19] L.C. Terrón-Camero, M. Rodríguez-Serrano, L.M. Sandalio, M.C. Romero-Puertas, Nitric oxide is essential for cadmium-induced peroxule formation and peroxisome proliferation, Plant Cell Environ. 43 (2020) 2492–2507.
[20] Y. Liu, C.M. Weaver, Y. Sen, G. Eitzen, A.J. Simmonds, L. Linchieh, O. Lurette, E. Hebert-Chatelain, R.A. Rachubinski, F. Di Cara, The nitric oxide donor, S-nitrosoglutathione, rescues peroxisome number and activity defects in PEX1G843D Mild Zellweger Syndrome Fibroblasts, Front. Cell Dev. Biol. 9 (2021) 2180.
[21] T.W.Y. Tiew, M.B. Sheahan, R.J. Rose, Peroxisomes contribute to reactive oxygen species homeostasis and cell division induction in Arabidopsis protoplasts, Front. Plant Sci. 6 (2015) 658. .
[22] P. Cui, H. Liu, F. Islam, L. Li, M.A. Farooq, S. Ruan, W. Zhou, OsPEX11, a peroxisomal biogenesis factor 11, contributes to salt stress tolerance in *Oryza sativa*, Front. Plant Sci. 7 (2016) 1357.
[23] D. Fahy, M.N.M.E. Sanad, K. Duscha, M. Lyons, F. Liu, P. Bozhkov, H.H. Kunz, J. Hu, H.E. Neuhaus, P.G. Steel, A. Smertenko, Impact of salt stress, cell death, and autophagy on peroxisomes: quantitative and morphological analyses using small fluorescent probe N-BODIPY, Sci. Rep. 71 (2017) 1–18, 2017.
[24] X. Zhao, Y. Niu, X. Bai, T. Mao, Transcriptomic and metabolic profiling reveals a lignin metabolism network involved in mesocotyl elongation during maize seed germination, Plants 11 (2022) 1034.
[25] L. Hinojosa, M.N.M.E. Sanad, D.E. Jarvis, P. Steel, K. Murphy, A. Smertenko, Impact of heat and drought stress on peroxisome proliferation in quinoa, Plant J. 99 (2019) 1144–1158.
[26] M. Shibata, K. Oikawa, K. Yoshimoto, M. Kondo, S. Mano, K. Yamada, M. Hayashi, W. Sakamoto, Y. Ohsumi, M. Nishimura, Highly oxidized peroxisomes are selectively degraded via autophagy in Arabidopsis, Plant Cell 25 (2013) 4967–4983.
[27] E.V. Tyutereva, K.S. Dobryakova, A. Schiermeyer, M.F. Shishova, K. Pawlowski, V. Demidchik, S. Reumann, O.V. Voitsekhovskaja, The levels of peroxisomal catalase protein and activity modulate the onset of cell death in tobacco BY-2 cells via reactive oxygen species levels and autophagy, Funct. Plant Biol. 45 (2018) 247–258.
[28] N. Calero-Muñoz, M. Exposito-Rodriguez, A.M. Collado-Arenal, M. Rodríguez-Serrano, A.M. Laureano-Marín, M.E. Santamaría, C. Gotor, I. Díaz, P. M. Mullineaux, M.C. Romero-Puertas, A. Olmedilla, L.M. Sandalio, Cadmium induces reactive oxygen species-dependent pexophagy in Arabidopsis leaves, Plant Cell Environ. 42 (2019) 2696–2714.
[29] L. Huang, L.J. Yu, X. Zhang, B. Fan, F.Z. Wang, Y.S. Dai, H. Qi, Y. Zhou, L.J. Xie, S. Xiao, Autophagy regulates glucose-mediated root meristem activity by modulating ROS production in Arabidopsis, Autophagy 15 (2019) 407–422.
[30] M. Luo, X. Zhuang, Review: selective degradation of peroxisome by autophagy in plants: mechanisms, functions, and perspectives, Plant Sci. 274 (2018) 485–491.
[31] D.S. Muhammad, K.A. Smith, B. Bartel, Plant peroxisome proteostasis—establishing, renovating, and dismantling the peroxisomal proteome, Essays Biochem. 66 (2022) 229–242.
[32] T. Avin-Wittenberg, F. Baluška, P.V. Bozhkov, P.H. Elander, A.R. Fernie, G. Galili, A. Hassan, D. Hofius, E. Isono, R. Le Bars, C. Masclaux-Daubresse, E.A. Minina, H. Peled-Zehavi, N.S. Coll, L.M. Sandalio, B. Satiat-Jeunemaitre, A. Sirko, P. S. Testillano, H. Batoko, Autophagy-related approaches for improving nutrient use efficiency and crop yield protection, J. Exp. Bot. 69 (2018) 1335–1353.
[33] L.M. Farmer, M.A. Rinaldi, P.G. Young, C.H. Danan, S.E. Burkhart, B. Bartel, Disrupting autophagy restores peroxisome function to an Arabidopsis *lon2* mutant and reveals a role for the LON2 protease in peroxisomal matrix protein degradation, Plant Cell 25 (2013) 4085–4100.
[34] P.G. Young, B. Bartel, Pexophagy and peroxisomal protein turnover in plants, Biochim. Biophys. Acta Mol. Cell Res. 1863 (2016) 999–1005.
[35] H. Sies, V.V. Belousov, N.S. Chandel, M.J. Davies, D.P. Jones, G.E. Mann, M. P. Murphy, M. Yamamoto, C. Winterbourn, Defining roles of specific reactive oxygen species (ROS) in cell biology and physiology, Nat. Rev. Mol. Cell Biol. 237 (2022) 499–515.

[36] A. Bratt, S. Rosenwasser, A. Meyer, R. Fluhr, Organelle redox autonomy during environmental stress, Plant Cell Environ. 39 (2016) 1909–1919.

[37] N. Smirnoff, D. Arnaud, Hydrogen peroxide metabolism and functions in plants, New Phytol. 221 (2019) 1197–1214.

[38] L.M. Sandalio, M.C. Romero-Puertas, Peroxisomes sense and respond to environmental cues by regulating ROS and RNS signalling networks, Ann. Bot. 116 (2015) 475–485.

[39] C.H. Foyer, A.J. Bloom, G. Queval, G. Noctor, Photorespiratory metabolism: genes, mutants, energetics, and redox signaling, Annu. Rev. Plant Biol. 60 (2009) 455–484.

[40] A. Baker, I. Graham, Plant Peroxisomes. Biochemistry, Cell Biology and Biotechnological Applications, Kluwer Academic Publishers, Dordrecht, The Netherlands, 2002.

[41] S. Reumann, The Photorespiratory Pathway of Leaf Peroxisomes, A. Baker and I. Graham, (Eds) Plant Peroxisomes. Biochemistry, Cell Biology and Biotechnological Applications, Kluwer Academic Publishers, Dordrecht, The Netherlands, 2002, pp 141-190.

[42] B.J. Walker, A. Vanloocke, C.J. Bernacchi, D.R. Ort, The costs of photorespiration to food production now and in the future, Annu. Rev. Plant Biol. 67 (2016) 107–129.

[43] S. Talbi, M.C. Romero-Puertas, A. Hernández, L. Terrón, A. Ferchichi, L. M. Sandalio, Drought tolerance in a Saharian plant *Oudneya africana*: role of antioxidant defences, Environ. Exp. Bot. 111 (2015) 114–126.

[44] M.C. Romero-Puertas, I. McCarthy, L.M. Sandalio, J.M. Palma, F.J. Corpas, M. Gómez, L.A. Del Río, Cadmium toxicity and oxidative metabolism of pea leaf peroxisomes, Free Radic. Res. 31 (1999) Suppl, 25-31.

[45] D.K. Gupta, L.B. Pena, M.C. Romero-Puertas, A. Hernández, M. Inouhe, L. M. Sandalio, NADPH oxidases differentially regulate ROS metabolism and nutrient uptake under cadmium toxicity, Plant Cell Environ. 40 (2017) 509–526.

[46] L.L. Cui, Y.S. Lu, Y. Li, C. Yang, X.X. Peng, Overexpression of glycolate oxidase confers improved photosynthesis under high light and high temperature in rice, Front. Plant Sci. 7 (2016) 1165.

[47] C.M. Rojas, M. Senthil-Kumar, K. Wang, C.M. Ryu, A. Kaundal, K.S. Mysore, Glycolate oxidase modulates reactive oxygen species–mediated signal transduction during nonhost resistance in *Nicotiana benthamiana* and Arabidopsis, Plant Cell 24 (2012) 336–352.

[48] A. Launay, S. Jolivet, G. Clément, M. Zarattini, Y. Dellero, R. Le Hir, M. Jossier, M. Hodges, D. Expert, M. Fagard, DspA/E-triggered non-host resistance against *E. amylovora* depends on the Arabidopsis GLYCOLATE OXIDASE 2 Gene, Int. J. Mol. Sci. 23 (2022) 4224.

[49] M. Yang, Z. Li, K. Zhang, X. Zhang, Y. Zhang, X. Wang, C. Han, J. Yu, K. Xu, D. Li, Barley stripe mosaic virus γb interacts with glycolate oxidase and inhibits peroxisomal ROS production to facilitate virus infection, Mol. Plant 11 (2018) 338–341.

[50] A.L. Schilmiller, A.J.K. Koo, G.A. Howe, Functional diversification of acyl-coenzyme A oxidases in jasmonic acid biosynthesis and action, Plant Physiol. 143 (2007) 812–824.

[51] A. Aprile, L. Havlickova, R. Panna, C. Marè, G.M. Borrelli, D. Marone, C. Perrotta, P. Rampino, L. De Bellis, V. Curn, A.M. Mastrangelo, F. Rizza, L. Cattivelli, Different stress responsive strategies to drought and heat in two durum wheat cultivars with contrasting water use efficiency, BMC Genom 14 (2013) 821.

[52] M.L. Shiraku, R.O. Magwanga, X. Cai, J.N. Kirungu, Y. Xu, T.G. Mehari, Y. Hou, Y. Wang, S.G. Agong, R. Peng, K. Wang, Z. Zhou, F. Liu, Functional characterization of *GhACX3* gene reveals its significant role in enhancing drought and salt stress tolerance in cotton, Front. Plant Sci. 12 (2021) 658755.

[53] Z. Xin, S. Chen, L. Ge, X. Li, X. Sun, The involvement of a herbivore-induced acyl-CoA oxidase gene, *CsACX1*, in the synthesis of jasmonic acid and its expression in flower opening in tea plant (*Camellia sinensis*), Plant Physiol. Biochem. 135 (2019) 132–140.

[54] D.M. Pazmiño, M. Rodríguez-Serrano, M.C. Romero-Puertas, A. Archilla-Ruiz, L. A. del Río, L.M. Sandalio, Differential response of young and adult leaves to herbicide 2,4-dichlorophenoxyacetic acid in pea plants: role of reactive oxygen species, Plant Cell Environ. 34 (2011) 1874–1889.

[55] M.C. Romero-Puertas, M.Á. Peláez-Vico, D.M. Pazmiño, M. Rodríguez-Serrano, L. Terrón-Camero, R. Bautista, A. Gómez-Cadenas, M.G. Claros, J. León, L. M. Sandalio, Insights into ROS-dependent signalling underlying transcriptomic plant responses to the herbicide 2,4-D, Plant, Cell Environ 45 (2022) 572–590.

[56] Y. Qu, X. Liu, X. Zhang, Y. Tang, Y. Hu, S. Chen, L. Xiang, Q. Zhang, Transcriptional regulation of Arabidopsis copper amine oxidase ζ (*CuAOζ*) in indole-3-butyric acid-induced lateral root development, Plant Growth Regul. 89 (2019) 287–297.

[57] A. Goyer, T.L. Johnson, L.J. Olsen, E. Collakova, Y. Shachar-Hill, D. Rhodes, A. D. Hanson, Characterization and metabolic function of a peroxisomal sarcosine and pipecolate oxidase from Arabidopsis, J. Biol. Chem. 279 (2004) 16947–16953.

[58] M.C. Romero-Puertas, M. Rodríguez-Serrano, F.J. Corpas, M. Gómez, L.A. Del Río, L.M. Sandalio, Cadmium-induced subcellular accumulation of $O_2^{\cdot-}$ and H_2O_2 in pea leaves, Plant, Cell Environ 27 (2004) 1122–1134.

[59] J. Huang, P. Willems, F. Van Breusegem, J. Messens, Pathways crossing mammalian and plant sulfenomic landscapes, Free Radic. Biol. Med. 122 (2018) 193–201.

[60] A. Mhamdi, G. Noctor, A. Baker, Plant catalases: peroxisomal redox guardians, Arch. Biochem. Biophys. 525 (2012) 181–194.

[61] S. Jacques, B. Ghesquière, P.J. De Bock, H. Demol, K. Wahni, P. Willems, J. Messens, F. Van Breusegem, K. Gevaert, Protein Methionine sulfoxide dynamics in *Arabidopsis thaliana* under oxidative stress, Mol. Cell. Proteomics 14 (2015) 1217–1229.

[62] Z. Zhang, Y. Xu, Z. Xie, X. Li, Z.H. He, X.X. Peng, Association-dissociation of glycolate oxidase with catalase in rice: a potential switch to modulate intracellular H_2O_2 levels, Mol. Plant 9 (2016) 737–748.

[63] S. Yamauchi, S. Mano, K. Oikawa, K. Hikino, K.M. Teshima, Y. Kimori, M. Nishimura, K. ichiro Shimazaki, A. Takemiya, Autophagy controls reactive oxygen species homeostasis in guard cells that is essential for stomatal opening, Proc. Natl. Acad. Sci. U.S.A. 116 (2019) 19187–19192.

[64] T. Yanik, R.P. Donaldson, A protective association between catalase and isocitrate lyase in peroxisomes, Arch. Biochem. Biophys. 435 (2005) 243–252.

[65] H.M. Yuan, W.C. Liu, Y.T. Lu, CATALASE2 coordinates SA-mediated repression of both auxin accumulation and JA biosynthesis in plant defenses, Cell Host Microbe 21 (2017) 143–155.

[66] G. Li, J. Li, R. Hao, Y. Guo, Activation of catalase activity by a peroxisome-localized small heat shock protein Hsp17.6CII, J. Genet. Genomics. 44 (2017) 395–404.

[67] Y. Wei, W. Liu, W. Hu, Y. Yan, H. Shi, The chaperone MeHSP90 recruits MeWRKY20 and MeCatalase1 to regulate drought stress resistance in cassava, New Phytol. 226 (2020) 476–491.

[68] Y. Zhang, L.F. Wang, T.T. Li, W.C. Liu, Mutual promotion of LAP2 and CAT2 synergistically regulates plant salt and osmotic stress tolerance, Front. Plant Sci. 12 (2021) 672672.

[69] Y. Al-Hajaya, B. Karpinska, C.H. Foyer, A. Baker, Nuclear and peroxisomal targeting of catalase, Plant Cell Environ. 45 (2022) 1096–1108.

[70] P.E. Verslues, G. Batelli, S. Grillo, F. Agius, Y.-S. Kim, J. Zhu, M. Agarwal, S. Katiyar-Agarwal, J.-K. Zhu, Interaction of SOS2 with nucleoside diphosphate kinase 2 and catalases reveals a point of connection between salt stress and H_2O_2 signaling in *Arabidopsis thaliana*, Mol. Cell Biol. 27 (2007) 7771–7780.

[71] Y. Li, L. Chen, J. Mu, J. Zuo, LESION SIMULATING DISEASE1 Interacts with catalases to regulate hypersensitive cell death in Arabidopsis, Plant Physiol. 163 (2013) 1059–1070.

[72] T. Hackenberg, T. Juul, A. Auzina, S. Gwizdz, A. Małolepszy, K. Van Der Kelen, S. Dam, S. Bressendorff, A. Lorentzen, P. Roepstorff, K.L. Nielsen, J.E. Jørgensen, D. Hofius, F. Van Breusegem, M. Petersen, S.U. Andersen, Catalase and NO CATALASE ACTIVITY1 promote autophagy-dependent cell death in Arabidopsis, Plant Cell 25 (2013) 4616–4626.

[73] J. Li, J. Liu, G. Wang, J.Y. Cha, G. Li, S. Chen, Z. Li, J. Guo, C. Zhang, Y. Yang, W. Y. Kim, D.J. Yun, K.S. Schumaker, Z. Chen, Y. Guo, A chaperone function of NO CATALASE ACTIVITY1 is required to maintain catalase activity and for multiple stress responses in Arabidopsis, Plant Cell 27 (2015) 908–925.

[74] J.J. Zou, X.D. Li, D. Ratnasekera, C. Wang, W.X. Liu, L.F. Song, W.Z. Zhang, W. H. Wu, Arabidopsis CALCIUM-DEPENDENT PROTEIN KINASE8 and CATALASE3 function in abscisic acid-mediated signaling and H_2O_2 homeostasis in stomatal guard cells under drought stress, Plant Cell 27 (2015) 1445–1460.

[75] M. Bundó, M. Coca, Calcium-dependent protein kinase OsCPK10 mediates both drought tolerance and blast disease resistance in rice plants, J. Exp. Bot. 68 (2017) 2963–2975.

[76] Y.B. Zhou, C. Liu, D.Y. Tang, L. Yan, D. Wang, Y.Z. Yang, J.S. Gui, X.Y. Zhao, L. G. Li, X.D. Tang, F. Yu, J.L. Li, L.L. Liu, Y.H. Zhu, J.Z. Lin, X.M. Liu, The receptor-like cytoplasmic kinase STRK1 phosphorylates and activates CatC, thereby Regulating H_2O_2 homeostasis and improving salt tolerance in rice, Plant Cell 30 (2018) 1100–1118.

[77] J. ichi Inaba, B.M. Kim, H. Shimura, C. Masuta, Virus-induced necrosis is a consequence of direct protein-protein interaction between a viral RNA-silencing suppressor and a host catalase, Plant Physiol. 156 (2011) 2026–2036.

[78] K. Murota, H. Shimura, M. Takeshita, C. Masuta, Interaction between Cucumber mosaic virus 2b protein and plant catalase induces a specific necrosis in association with proteasome activity, Plant Cell Rep. 36 (2017) 37–47.

[79] P.A. Walton, C. Brees, C. Lismont, O. Apanasets, M. Fransen, The peroxisomal import receptor PEX5 functions as a stress sensor, retaining catalase in the cytosol in times of oxidative stress, Biochim. Biophys. Acta Mol. Cell Res. 1864 (2017) 1833–1843.

[80] K. Okumoto, M. El Shermely, M. Natsui, H. Kosako, R. Natsuyama, T. Marutani, Y. Fujiki, The peroxisome counteracts oxidative stresses by suppressing catalase import via Pex14 phosphorylation, Elife 9 (2020) 1–52.

[81] C.H. Foyer, G. Noctor, Stress-triggered redox signalling: what's in pROSpect? Plant Cell Environ. 39 (2016) 951–964.

[82] A. Jiménez, J.A. Hernández, L.A. Del Río, F. Sevilla, Evidence for the presence of the Ascorbate-Glutathione cycle in mitochondria and peroxisomes of pea leaves, Plant Physiol. 114 (1997) 275–284.

[83] N. Kaur, S. Reumann, J. Hu, Peroxisome biogenesis and function, Arabidopsis Book 7 (2009), e0123.

[84] K.J. Dietz, Plant peroxiredoxins, Annu. Rev. Plant Biol. 54 (2003) 93–107.

[85] L. Verdoucq, F. Vignols, J.P. Jacquot, Y. Chartier, Y. Meyer, In vivo characterization of a thioredoxin h target protein defines a new peroxiredoxin family, J. Biol. Chem. 274 (1999) 19714–19722.

[86] K.J. Dietz, S. Jacob, M.L. Oelze, M. Laxa, V. Tognetti, S.M.N. De Miranda, M. Baier, I. Finkemeier, The function of peroxiredoxins in plant organelle redox metabolism, J. Exp. Bot. 57 (2006) 1697–1709.

[87] M.C. Romero-Puertas, M. Laxa, A. Mattè, F. Zaninotto, I. Finkemeier, A.M. E. Jones, M. Perazzolli, E. Vandelle, K.J. Dietz, M. Delledonne, S-Nitrosylation of peroxiredoxin II E promotes peroxynitrite-mediated tyrosine nitration, Plant Cell 19 (2008) 4120–4130.

[88] L.C. Terrón-Camero, M.Á. Peláez-Vico, A. Rodríguez-González, C. del Val, L. M. Sandalio, M.C. Romero-Puertas, Gene network downstream plant stress response modulated by peroxisomal H_2O_2, Front. Plant Sci. 13 (2022) 930721.

[89] L.M. Sandalio, C. Gotor, L.C. Romero, M.C. Romero-Puertas, Multilevel regulation of peroxisomal proteome by post-translational modifications, Int. J. Mol. Sci. 20 (2019) 4881.

[90] D.P. Dixon, T. Hawkins, P.J. Hussey, R. Edwards, Enzyme activities and subcellular localization of members of the Arabidopsis glutathione transferase superfamily, J. Exp. Bot. 60 (2009) 1207–1218.

[91] E. Sylvestre-Gonon, S.R. Law, M. Schwartz, K. Robe, O. Keech, C. Didierjean, C. Dubos, N. Rouhier, A. Hecker, Functional, structural and biochemical features of plant serinyl-glutathione transferases, Front. Plant Sci. 10 (2019) 608.

[92] A. Mor, E. Koh, L. Weiner, S. Rosenwasser, H. Sibony-Benyamini, R. Fluhr, Singlet oxygen signatures are detected independent of light or chloroplasts in response to multiple stresses, Plant Physiol. 165 (2014) 249–261.

[93] J. León, Á. Costa-Broseta, Present knowledge and controversies, deficiencies, and misconceptions on nitric oxide synthesis, sensing, and signaling in plants, Plant Cell Environ 43 (2020) 1–15.

[94] J.B. Barroso, F.J. Corpas, A. Carreras, L.M. Sandalio, R. Valderrama, J.M. Palma, J.A. Lupiánez, L.A. Del Río, Localization of nitric-oxide synthase in plant peroxisomes, J. Biol. Chem. 274 (1999) 36729–36733.

[95] M. Schlicht, J. Ludwig-Müller, C. Burbach, D. Volkmann, F. Baluska, Indole-3-butyric acid induces lateral root formation via peroxisome-derived indole-3-acetic acid and nitric oxide, New Phytol. 200 (2013) 473–482.

[96] S. Agurla, G. Gayatri, A.S. Raghavendra, Polyamines increase nitric oxide and reactive oxygen species in guard cells of *Arabidopsis thaliana* during stomatal closure, Protoplasma 255 (2018) 153–162.

[97] V.D. Antonenkov, S. Grunau, S. Ohlmeier, J.K. Hiltunen, Peroxisomes are oxidative organelles, Antioxidants Redox Signal. 13 (2010) 525–537.

[98] B.L. Wang, X.Y. Tang, L.Y. Cheng, A.Z. Zhang, W.H. Zhang, F.S. Zhang, J.Q. Liu, Y. Cao, D.L. Allan, C.P. Vance, J.B. Shen, Nitric oxide is involved in phosphorus deficiency-induced cluster-root development and citrate exudation in white lupin, New Phytol. 187 (2010) 1112–1123.

[99] A.P. Ortega-Galisteo, M. Rodríguez-Serrano, D.M. Pazmiño, D.K. Gupta, L. M. Sandalio, M.C. Romero-Puertas, S-Nitrosylated proteins in pea (Pisum sativum L.) leaf peroxisomes: changes under abiotic stress, J. Exp. Bot. 63 (2012) 2089–2103.

[100] F.J. Corpas, J.B. Barroso, O.N.O.O.– Peroxynitrite (, Is endogenously produced in arabidopsis peroxisomes and is overproduced under cadmium stress, Ann. Bot. 113 (2014) 87–96.

[101] J. Mano, M.S. Biswas, Analysis of reactive carbonyl species generated under oxidative stress, Methods Mol. Biol. 1743 (2018) 117–124.

[102] M.S. Biswas, J. Mano, Lipid peroxide-derived reactive carbonyl species as mediators of oxidative stress and signaling, Front. Plant Sci. 12 (2021) 720867.

[103] R. Shyama Prasad Rao, I.M. Møller, Pattern of occurrence and occupancy of carbonylation sites in proteins, Proteomics 11 (2011) 4166–4173.

[104] M.C. Romero-Puertas, J.M. Palma, M. Gómez, L.A. Del Río, L.M. Sandalio, Cadmium causes the oxidative modification of proteins in pea plants, Plant Cell Environ. 25 (2002) 677–686.

[105] A.T. Nguyen, R.P. Donaldson, Metal-catalyzed oxidation induces carbonylation of peroxisomal proteins and loss of enzymatic activities, Arch. Biochem. Biophys. 439 (2005) 25–31.

[106] H. Vanacker, L.M. Sandalio, A. Jiménez, J.M. Palma, F.J. Corpas, V. Meseguer, M. Gómez, F. Sevilla, M. Leterrier, C.H. Foyer, L.A. Del Río, Roles for redox regulation in leaf senescence of pea plants grown on different sources of nitrogen nutrition, J. Exp. Bot. 57 (2006) 1735–1745.

[107] I. McCarthy-Suárez, M. Gómez, L.A. Del Río, J.M. Palma, Role of peroxisomes in the oxidative injury induced by 2,4-dichlorophenoxyacetic acid in leaves of pea plants, Biol. Plant. (Prague) 553 (2011) 485–492..

[108] J. Mano, M. Nagata, S. Okamura, T. Shiraya, T. Mitsui, Identification of oxidatively modified proteins in salt-stressed Arabidopsis: a carbonyl-targeted proteomics approach, Plant Cell Physiol. 55 (2014) 1233–1244.

[109] S. Distefano, J.M. Palma, I. McCarthy, L.A. Del Río, Proteolytic cleavage of plant proteins by peroxisomal endoproteases from senescent pea leaves, Planta 2093 (209) (1999) 308–313, 1999.

[110] Y.T. Kao, B. Bartel, Elevated growth temperature decreases levels of the PEX5 peroxisome-targeting signal receptor and ameliorates defects of Arabidopsis mutants with an impaired PEX4 ubiquitin-conjugating enzyme, BMC Plant Biol. 15 (2015) 1–14.

[111] N. Kaur, Q. Zhao, Q. Xie, J. Hu, Arabidopsis RING peroxins are E3 Ubiquitin ligases that interact with two homologous ubiquitin receptor proteinsF, J. Integr. Plant Biol. 55 (2013) 108–120.

[112] S. Kumar, A. Kawałek, I.J. van der Klei, Peroxisomal quality control mechanisms, Curr. Opin. Microbiol. 22 (2014) 30–37.

[113] M. Nordgren, M. Fransen, Peroxisomal metabolism and oxidative stress, Biochimie 98 (2014) 56–62.

[114] Y. Xiong, A.L. Contento, P.Q. Nguyen, D.C. Bassham, Degradation of oxidized proteins by autophagy during oxidative stress in Arabidopsis, Plant Physiol. 143 (2007) 291–299.

[115] S. Quan, R. Switzenberg, S. Reumann, J. Hu, In vivo subcellular targeting analysis validates a novel peroxisome targeting signal type 2 and the peroxisomal localization of two proteins with putative functions in defense in Arabidopsis, Plant Signal. Behav. 5 (2010) 151–153.

[116] N. Lau, M.D. Pluth, Reactive sulfur species (RSS): persulfides, polysulfides, potential, and problems, Curr. Opin. Chem. Biol. 49 (2019) 1–8.

[117] C. Gotor, I. García, Á. Aroca, A.M. Laureano-Marín, L. Arenas-Alfonseca, A. Jurado-Flores, I. Moreno, L.C. Romero, Signaling by hydrogen sulfide and cyanide through post-translational modification, J. Exp. Bot. 70 (2019) 4251–4265.

[118] K.R. Olson, Y. Gao, E.R. DeLeon, M. Arif, F. Arif, N. Arora, K.D. Straub, Catalase as a sulfide-sulfur oxido-reductase: an ancient (and modern?) regulator of reactive sulfur species (RSS), Redox Biol. 12 (2017) 325–339.

[119] X. Cao, L. Ding, Z.Z. Xie, Y. Yang, M. Whiteman, P.K. Moore, J.S. Bian, A review of hydrogen sulfide synthesis, metabolism, and measurement: is modulation of hydrogen sulfide a novel therapeutic for cancer? Antioxidants Redox Signal. 31 (2019) 1–38.

[120] A. Aroca, J.M. Benito, C. Gotor, L.C. Romero, Persulfidation proteome reveals the regulation of protein function by hydrogen sulfide in diverse biological processes in Arabidopsis, J. Exp. Bot. 68 (2017) 4915–4927.

[121] M.M. Cortese-Krott, A. Koning, G.G.C. Kuhnle, P. Nagy, C.L. Bianco, A. Pasch, D. A. Wink, J.M. Fukuto, A.A. Jackson, H. Van Goor, K.R. Olson, M. Feelisch, The reactive species interactome: evolutionary emergence, biological significance, and opportunities for redox metabolomics and personalized medicine, Antioxidants Redox Signal. 27 (2017) 684–712.

[122] J. León, Protein tyrosine nitration in plant nitric oxide signaling, Front. Plant Sci. 13 (2022) 859374.

[123] F.J. Corpas, S. González-Gordo, J.M. Palma, Protein nitration: a connecting bridge between nitric oxide (NO) and plant stress, Plant Stress 2 (2021), 100026.

[124] K. Ogino, N. Kodama, M. Nakajima, A. Yamada, H. Nakamura, H. Nagase, D. Sadamitsu, T. Maekawa, Catalase catalyzes nitrotyrosine formation from sodium azide and hydrogen peroxide, Free Radic. Res. 35 (2009) 735–747.

[125] L. Gebicka, J. Didik, Catalytic scavenging of peroxynitrite by catalase, J. Inorg. Biochem. 103 (2009) 1375–1379.

[126] D.C. Hooper, G.S. Scott, A. Zborek, T. Mikheeva, R.B. Kean, H. Koprowski, S. V. Spitsin, Uric acid, a peroxynitrite scavenger, inhibits CNS inflammation, blood-CNS barrier permeability changes, and tissue damage in a mouse model of multiple sclerosis, Faseb. J. 14 (2000) 691–698.

[127] J.M. Alamillo, F. García-Olmedo, Effects of urate, a natural inhibitor of peroxynitrite-mediated toxicity, in the response of *Arabidopsis thaliana* to the bacterial pathogen *Pseudomonas syringae*, Plant J. 25 (2001) 529–540.

[128] T. Zhang, M. Ma, T. Chen, L. Zhang, L. Fan, W. Zhang, B. Wei, S. Li, W. Xuan, G. Noctor, Y. Han, Glutathione-dependent denitrosation of GSNOR1 promotes oxidative signalling downstream of H_2O_2, Plant Cell Environ. 43 (2020) 1175–1191.

[129] L. Chen, R. Wu, J. Feng, T. Feng, C. Wang, J. Hu, N. Zhan, Y. Li, X. Ma, B. Ren, J. Zhang, C.P. Song, J. Li, J.M. Zhou, J. Zuo, Transnitrosylation mediated by the non-canonical catalase ROG1 regulates nitric oxide signaling in plants, Dev. Cell 53 (2020) 444–457.e5.

[130] P. Wang, Y. Yan, Y. Bai, Y. Dong, Y. Wei, H. Zeng, H. Shi, Phosphorylation of RAV1/2 by KIN10 is essential for transcriptional activation of CAT6/7, which underlies oxidative stress response in cassava, Cell Rep. 37 (2021), 110119.

[131] E. Malard, S. Valable, M. Bernaudin, E. Pérès, L. Chatre, The reactive species interactome in the brain, Antioxidants Redox Signal. 35 (2021) 1176–1206.

[132] M. Hasanuzzaman, K. Nahar, M.S. Hossain, J. Al Mahmud, A. Rahman, M. Inafuku, H. Oku, M. Fujita, Coordinated actions of glyoxalase and antioxidant defense systems in conferring abiotic stress tolerance in plants, Int. J. Mol. Sci. 18 (2017) 200.

[133] J. Li, C. Shi, X. Wang, C. Liu, X. Ding, P. Ma, X. Wang, H. Jia, Hydrogen sulfide regulates the activity of antioxidant enzymes through persulfidation and improves the resistance of tomato seedling to copper oxide nanoparticles (CuO NPs)-induced oxidative stress, Plant Physiol. Biochem. 156 (2020) 257–266.

[134] A. Aroca, J. Zhang, Y. Xie, L.C. Romero, C. Gotor, Hydrogen sulfide signaling in plant adaptations to adverse conditions: molecular mechanisms, J. Exp. Bot. 72 (2021) 5893–5904.

[135] L. Wang, X. Mu, X. Chen, Y. Han, Hydrogen sulfide attenuates intracellular oxidative stress via repressing glycolate oxidase activities in *Arabidopsis thaliana*, BMC Plant Biol. 22 (2022) 1–12.

[136] K.R. Olson, Y. Gao, F. Arif, K. Arora, S. Patel, E.R. DeLeon, T.R. Sutton, M. Feelisch, M.M. Cortese-Krott, K.D. Straub, Metabolism of hydrogen sulfide (H_2S) and production of reactive sulfur species (RSS) by superoxide dismutase, Redox Biol. 15 (2018) 74–85.

[137] J. Hancock, M. Whiteman, Cellular redox environment and its influence on redox signaling molecules, React. Oxyg. Species 5 (2018) 78–85.

[138] T. Chang, A. Untereiner, J. Liu, L. Wu, Interaction of methylglyoxal and hydrogen sulfide in rat vascular smooth muscle cells, Antioxid, Redox Signal 12 (2010) 1093–1100.

[139] X.Y. Ye, X.M. Qiu, Y.Y. Sun, Z.G. Li, Interplay between hydrogen sulfide and methylglyoxal initiates thermotolerance in maize seedlings by modulating reactive oxidative species and osmolyte metabolism, Protoplasma 257 (2020) 1415–1432.

[140] G. Noctor, J.P. Reichheld, C.H. Foyer, ROS-related redox regulation and signaling in plants, Semin. Cell Dev. Biol. 80 (2018) 3–12.

[141] O. van Aken, Mitochondrial redox systems as central hubs in plant metabolism and signaling, Plant Physiol. 186 (2021) 36–52.

[142] L.-L. Gao, Z.-H. Hong, Y. Wang, G.-Z. Wu, Chloroplast proteostasis: a story of birth, life, and death, Plant Commun 4 (2022) 100424.

[143] C.H. Foyer, G. Hanke, ROS production and signalling in chloroplasts: cornerstones and evolving concepts, Plant J. 111 (2022) 642–661.

[144] S. Rosenwasser, R. Fluhr, J.R. Joshi, N. Leviatan, N. Sela, A. Hetzroni, H. Friedman, ROSMETER: a bioinformatic tool for the identification of

transcriptomic imprints related to reactive oxygen species type and origin provides new insights into stress responses, Plant Physiol. 163 (2013) 1071–1083.
[145] T.S. Gechev, J. Hille, Hydrogen peroxide as a signal controlling plant programmed cell death, J. Cell Biol. 168 (2005) 17–20.
[146] P. Kerchev, C. Waszczak, A. Lewandowska, P. Willems, A. Shapiguzov, Z. Li, S. Alseekh, P. Mühlenbock, F.A. Hoeberichts, J. Huang, K. Van Der Kelen, J. Kangasjärvi, A.R. Fernie, R. De Smet, Y. Van de Peer, J. Messens, F. Van Breusegem, Lack of GLYCOLATE OXIDASE1, but not GLYCOLATE OXIDASE2, attenuates the photorespiratory phenotype of CATALASE2-deficient Arabidopsis, Plant Physiol. 171 (2016) 1704–1719.
[147] G. Queval, E. Issakidis-Bourguet, F.A. Hoeberichts, M. Vandorpe, B. Gakière, H. Vanacker, M. Miginiac-Maslow, F. Van Breusegem, G. Noctor, Conditional oxidative stress responses in the Arabidopsis photorespiratory mutant *cat2* demonstrate that redox state is a key modulator of daylength-dependent gene expression, and define photoperiod as a crucial factor in the regulation of H_2O_2-induced cell death, Plant J. 52 (2007) 640–657.
[148] N. Sewelam, Y. Oshima, N. Mitsuda, M. Ohme-Takagi, A step towards understanding plant responses to multiple environmental stresses: a genome-wide study, Plant Cell Environ. 37 (2014) 2024–2035.
[149] T. Su, P. Wang, H. Li, Y. Zhao, Y. Lu, P. Dai, T. Ren, X. Wang, X. Li, Q. Shao, D. Zhao, Y. Zhao, C. Ma, The Arabidopsis catalase triple mutant reveals important roles of catalases and peroxisome-derived signaling in plant development, J. Integr. Plant Biol. 60 (2018) 591–607.
[150] S. Vandenabeele, K. Van Der Kelen, J. Dat, I. Gadjev, T. Boonefaes, S. Morsa, P. Rottiers, L. Slooten, M. Van Montagu, M. Zabeau, D. Inzé, F. Van Breusegem, A comprehensive analysis of hydrogen peroxide-induced gene expression in tobacco, Proc. Natl. Acad. Sci. U.S.A. 100 (2003) 16113–16118.
[151] S. Vandenabeele, S. Vanderauwera, M. Vuylsteke, S. Rombauts, C. Langebartels, H.K. Seidlitz, M. Zabeau, M. Van Montagu, D. Inzé, F. Van Breusegem, Catalase deficiency drastically affects gene expression induced by high light in Arabidopsis thaliana, Plant J. 39 (2004) 45–58.
[152] S. Vanderauwera, P. Zimmermann, S. Rombauts, S. Vandenabeele, C. Langebartels, W. Gruissem, D. Inzé, F. Van Breusegem, Genome-wide analysis of hydrogen peroxide-regulated gene expression in Arabidopsis reveals a high light-induced transcriptional cluster involved in anthocyanin biosynthesis, Plant Physiol. 139 (2005) 806–821.
[153] A. Mhamdi, G. Queval, S. Chaouch, S. Vanderauwera, F. Van Breusegem, G. Noctor, Catalase function in plants: a focus on Arabidopsis mutants as stress-mimic models, J. Exp. Bot. 61 (2010) 4197–4220.
[154] S. Chaouch, G. Queval, S. Vanderauwera, A. Mhamdi, M. Vandorpe, M. Langlois-Meurinne, F. van Breusegem, P. Saindrenan, G. Noctor, Peroxisomal hydrogen peroxide is coupled to biotic defense responses by ISOCHORISMATE SYNTHASE1 in a Daylength-Related Manner, Plant Physiol. 153 (2010) 1692–1705.
[155] G. Queval, J. Neukermans, S. Vanderauwera, F. van Breusegem, G. Noctor, Day length is a key regulator of transcriptomic responses to both CO_2 and H_2O_2 in Arabidopsis, Plant Cell Environ. 35 (2012) 374–387.
[156] C. Waszczak, P.I. Kerchev, P. Mühlenbock, F.A. Hoeberichts, K. Van Der Kelen, A. Mhamdi, P. Willems, J. Denecker, R.P. Kumpf, G. Noctor, J. Messens, F. Van Breusegem, SHORT-ROOT deficiency alleviates the cell death phenotype of the Arabidopsis catalase2 mutant under photorespiration-promoting conditions, Plant Cell 28 (2016) 1844–1859.
[157] J. Su, L. Yang, Q. Zhu, H. Wu, Y. He, Y. Liu, J. Xu, D. Jiang, S. Zhang, Active photosynthetic inhibition mediated by MPK3/MPK6 is critical to effector-triggered immunity, PLoS Biol. 16 (2018), e2004122.
[158] T. Orth, S. Reumann, X. Zhang, J. Fan, D. Wenzel, S. Quan, J. Hu, The PEROXIN11 protein family controls peroxisome proliferation in Arabidopsis, Plant Cell 19 (2007) 333–350.
[159] N. Kaur, J. Li, J. Hu, Peroxisomes and photomorphogenesis, Subcell. Biochem. 69 (2013) 195–211.
[160] D. Chen, G. Xu, W. Tang, Y. Jing, Q. Ji, Z. Fei, R. Lin, Antagonistic basic Helix-loop-helix/bZIP transcription factors form transcriptional modules that integrate light and reactive oxygen species signaling in Arabidopsis, Plant Cell 25 (2013) 1657–1673.
[161] C. Lismont, J. Koster, S. Provost, M. Baes, P.P. Van Veldhoven, H.R. Waterham, M. Fransen, Deciphering the potential involvement of PXMP2 and PEX11B in hydrogen peroxide permeation across the peroxisomal membrane reveals a role for PEX11B in protein sorting, Biochim. Biophys. Acta Biomembr. 1861 (2019), 182991.
[162] S. Vanderauwera, N. Suzuki, G. Miller, B. Van De Cotte, S. Morsa, J.L. Ravanat, A. Hegie, C. Triantaphylidès, V. Shulaev, M.C.E. Van Montagu, F. Van Breusegem, R. Mittler, Extranuclear protection of chromosomal DNA from oxidative stress, Proc. Natl. Acad. Sci. U.S.A. 108 (2011) 1711–1716.
[163] T. Li, K.P. Jia, H.L. Lian, X. Yang, L. Li, H.Q. Yang, Jasmonic acid enhancement of anthocyanin accumulation is dependent on phytochrome A signaling pathway under far-red light in Arabidopsis, Biochem. Biophys. Res. Commun. 454 (2014) 78–83.
[164] X. Li, M. Liao, J. Huang, Z. Xu, Z. Lin, N. Ye, Z. Zhang, X. Peng, Glycolate oxidase-dependent H_2O_2 production regulates IAA biosynthesis in rice, BMC Plant Biol. 21 (2021) 1–14.
[165] Y. Wang, J. Selinski, C. Mao, Y. Zhu, O. Berkowitz, J. Whelan, Linking mitochondrial and chloroplast retrograde signalling in plants, Philos. Trans. R. Soc. B. 375 (2020) 20190410.
[166] X. Meng, L. Li, I. De Clercq, R. Narsai, Y. Xu, A. Hartmann, D.L. Claros, E. Custovic, M.G. Lewsey, J. Whelan, O. Berkowitz, ANAC017 coordinates organellar functions and stress responses by reprogramming retrograde signaling, Plant Physiol. 180 (2019) 634–653.
[167] E. Giraud, L.H.M. Ho, R. Clifton, A. Carroll, G. Estavillo, Y.F. Tan, K.A. Howell, A. Ivanova, B.J. Pogson, A.H. Millar, J. Whelan, The absence of ALTERNATIVE OXIDASE1a in Arabidopsis results in acute sensitivity to combined light and drought stress, Plant Physiol. 147 (2008) 595–610.
[168] M. Rodríguez-Serrano, M.C. Romero-Puertas, M. Sanz-Fernández, J. Hu, L. M. Sandalio, Peroxisomes extend peroxules in a fast response to stress via a reactive oxygen species-mediated induction of the peroxin PEX11a, Plant Physiol. 171 (2016) 1665–1674.
[169] J.L. Caplan, A.S. Kumar, E. Park, M.S. Padmanabhan, K. Hoban, S. Modla, K. Czymmek, S.P. Dinesh-Kumar, Chloroplast stromules function during innate immunity, Dev. Cell 34 (2015) 45–57.
[170] A.S. Kumar, E. Park, A. Nedo, A. Alqarni, L. Ren, K. Hoban, S. Modla, J. H. McDonald, C. Kambhamettu, S.P. Dinesh-Kumar, J.L. Caplan, Stromule extension along microtubules coordinated with actin-mediated anchoring guides perinuclear chloroplast movement during innate immunity, Elife 7 (2018), e23625.