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Plant peroxisomes: Recent discoveries in functional complexity, organelle homeostasis, and morphological dynamics

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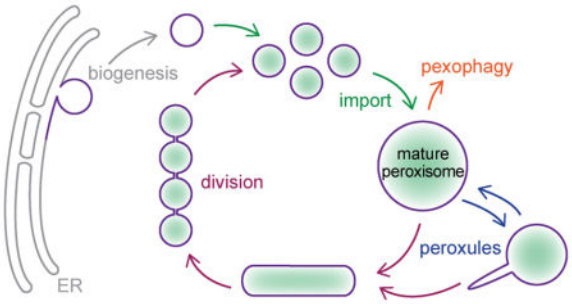
Abstract

Peroxisomes are essential for life in plants. These organelles house a variety of metabolic processes that generate and inactivate reactive oxygen species. Our knowledge of pathways and mechanisms that depend on peroxisomes and their constituent enzymes continues to grow, and in this review we highlight recent advances in understanding the identity and biological functions of peroxisomal enzymes and metabolic processes. We also review how peroxisomal matrix and membrane proteins enter the organelle from their sites of synthesis. Peroxisome homeostasis is regulated by specific degradation mechanisms, and we discuss the contributions of specialized autophagy and a peroxisomal protease to the degradation of entire peroxisomes and peroxisomal enzymes that are damaged or superfluous. Finally, we review how peroxisomes can flexibly change their morphology to facilitate inter-organellar contacts.

Graphical Abstract



Introduction

Peroxisomes are small organelles found in most eukaryotes that are delimited by a single lipid bilayer. Diverse metabolic activities are compartmentalized in plant peroxisomes (Table 1). Well-established peroxisomal activities include fatty acid β-oxidation, hormone production, and photorespiration. Peroxisomal pathways often include an oxidative step generating reactive oxygen species (ROS) as byproducts; peroxisomes therefore also house catalase and other ROS-inactivating enzymes.

Peroxisomes primarily proliferate by growth and division (Figure 1). The proteins required for peroxisome biogenesis, matrix protein import, and division are designated as peroxins (PEX proteins). Peroxisomal matrix proteins are imported into the organelle with the assistance of two interacting receptors, PEX5 for peroxisome-targeting signal type 1 (PTS1) proteins and PEX7 for PTS2 proteins (Figure 2). The cargo-loaded receptors dock at the peroxisomal membrane via interactions with PEX13 and PEX14 and release their cargo into the matrix by largely unknown mechanisms. Membrane-associated PEX5 is ubiquitinated, extracted from the membrane and retrotranslocated back to the cytosol for additional import rounds. After arrival in the matrix, the PTS2 domain is removed, whereas PTS1 proteins retain the signal (Figure 2).

Peroxisomal membrane proteins (PMPs) are inserted into peroxisomes either directly or via the ER membrane, from which pre-peroxisomes can bud and mature by post-translational import of matrix and additional membrane proteins into the organelle (Figure 1). PMPs include a subset of the PEX proteins necessary for import of matrix proteins into the organelle. Mature peroxisomes extend, tubulate, and divide by fission, which is considered to be the predominant mode of peroxisome proliferation (Figure 1).

Although key peroxins and core peroxisomal functions are largely conserved in eukaryotes, there are important differences among fungal, animal, and plant peroxisomes. These distinctions underscore the need for a thorough knowledge of peroxisome biology at all levels in the reference plant *Arabidopsis thaliana*, which is prerequisite for translation to crop plants and microalgae for future agricultural and biotechnological applications. In this article, we highlight recent discoveries that have deepened our understanding of the functional plasticity, biogenesis, degradation, and membrane dynamics of peroxisomes in plants.

Functional diversity of plant peroxisomes

The metabolic diversity and plasticity of peroxisomes is amazing (Table 1), and unexpected functions of plant peroxisomes continue to be discovered. For instance, peroxisomes house biosynthetic steps of phylloquinone (2-methyl-3-phytyl-1,4-naphtho-quinone or vitamin K_1), a vital co-factor for electron transfer in photosystem I. Phylloquinone biosynthesis begins in plastids with the synthesis of *o*-succinylbenzoate from chorismate. Peroxisomal enzymes catalyze the next three steps to the double-ring structure of naphthoquinone: i) activation via CoA esterification by acyl-activating enzyme 14, ii) ring cyclization by naphthoate synthase to yield the CoA thioester of 1,4-dihydroxy-2-naphthoate (DHNA) [1], and iii) hydrolysis of

DHNA-CoA by two functionally redundant peroxisomal thioesterases [2]. Phylloquinone biosynthesis is finalized in chloroplasts by DHNA prenylation and methylation. Interestingly, while most phylloquinone biosynthetic enzymes are of cyanobacterial origin, the thioesterases apparently originate from Lactobacillales by horizontal gene transfer [2]. Because none of the three peroxisomal enzymes of phylloquinone biosynthesis produces ROS, it remains to be elucidated why these steps were shifted from chloroplasts to peroxisomes during higher plant evolution.

The first enzyme in biotin synthesis (BioF) is peroxisomal in plants and fungi [3,4], while the subsequent steps are mitochondrial. Moreover, peroxisomal β-oxidation is required for synthesis of the BioF substrate, pimeloyl-CoA [3,4]. In addition, the enzymes catalyzing the final two steps of mevalonate biosynthesis, 5-phosphomevalonate kinase and mevalonate 5-diphosphate decarboxylase, were recently characterized as peroxisomal in *Arabidopsis* and *Catharanthus roseus* [5].

High amounts of photorespiratory H_2O_2 are produced during photosynthesis. Both host cells and pathogens can impinge on peroxisomal functions to modulate ROS homoeostasis [6]. For example, *Arabidopsis* LESION SIMULATING DISEASE1 interacts with catalase via a zinc finger domain and increases peroxisomal catalase activity to negatively regulate programmed cell death [7]. In addition, the Rab GTPase-activating protein RabGAP22 facilitates plant defenses against the soil-borne fungal pathogen *Verticillium longisporum. Verticillium* infection induces *Arabidopsis RabGAP22* expression and triggers RabGAP22 redirection from the nucleus to peroxisomes, where it forms a complex with the photorespiratory enzyme, serine:glyoxylate aminotransferase (AGT1) [8]. The *rabgap22-1* mutant displays elevated jasmonate (JA) levels, and it will be interesting to learn whether the RabGAP22-AGT1 complex interferes with peroxisomally localized JA biosynthetic enzymes.

Only a few membrane proteins that transport metabolic intermediates and co-factors across the peroxisomal membrane have been identified. One such transporter is PEROXISOMAL ATP binding cassette (ABC) TRANSPORTER 1 (PXA1), which transports various substrates into peroxisomes for β-oxidation, including fatty acids and lipophilic precursors of the hormones JA and auxin in *Arabidopsis* [9,10] and barley [11]. Whether transport by PXA1 is regulated and whether the import substrates are CoA esters or free fatty acids was long enigmatic. The α/β hydrolase, COMPARATIVE GENE IDENTIFICATION-58 (CGI-58), has emerged as a positive regulator of PXA1 [12]. CGI-58 interacts with PXA1 and promotes PXA1 functions in JA and auxin biosynthesis as well as lipid metabolism in non-seed vegetative tissues but not in germinating seeds [12]. In addition to its transport function, *Arabidopsis* PXA1 displays intrinsic thioesterase activity that is required for fatty acid transport and metabolism [13], implicating CoA esters rather than free fatty acids as PXA1 substrates. It remains to be resolved at which side of the membrane the CoA ester cleavage occurs and whether CGI-58 activates the transport and/or thioesterase activity of PXA1.

Peroxisomes also function in stomatal opening. The SUGAR-DEPENDENT1 lipase (SDP1) [14] and PXA1 transporter [9,15] are required not only for lipid mobilization during

germination but also contribute to stomatal opening [16]. Blue-light-induced stomatal opening is accompanied by reduced oil body volume in *Arabidopsis* guard cells, and *sdp1*, *pxa1*, and *cgi58* mutants all display slowed light-induced stomatal opening, presumably because impaired fatty acid catabolism reduces ATP production in these mutants and limits apoplast acidification [16]. These findings provide a rationale for the occurrence of stomatal oil bodies throughout the plant kingdom [16]. Moreover, *Arabidopsis* mutants deficient in peroxisomal NADP-dependent isocitrate dehydrogenase (pICDH) are compromised in light-induced stomatal opening [17]. pICDH is one of the few peroxisomal matrix sources of NADPH, which is needed for JA biosynthesis and the peroxisomal ascorbate-glutathione cycle. The stomatal defect of *picdh* is reversible by either an antioxidant such as ascorbate or a nitric oxide scavenger, suggesting that pICDH regulates peroxisomal H_2O_2 and/or NO levels and that peroxisomes are needed in guard cells not only for energy metabolism [16], but also for signaling [17].

In addition to pICDH, the oxidative pentose phosphate pathway (OPPP) is an alternative source of peroxisomal NADPH. The three OPPP enzymes are each encoded by multi-gene families, and the isoforms are located in different compartments. Another layer of subcellular complexity is added for the two peroxisomal isoforms (glucose-6-phosphate dehydrogenase and 6-phosphogluconolactonase), which can be targeted to peroxisomes or plastids depending on thioredoxin and redox balance [18,19]. Despite isoform redundancy and the high permeability of the peroxisomal membrane for small intermediates, the third peroxisomal OPPP enzyme, 6-phosphogluconate dehydrogenase isoform 2, is required for guided growth of pollen tubes within the style as well as successful pollen tube-ovule interaction and fertilization [20]. This finding provides a possible biochemical rationale for a similar requirement of the docking peroxin PEX13 for fertilization [21]. It will be interesting to learn whether the peroxisomal OPPP is essential for the production of nitric oxide, JA, or an unknown signaling molecule.

Calcium is implicated in regulating peroxisome functions. For example, calcium regulates *in vitro* dimerization and substrate specificity of the DEG15 protease that removes the N-terminal PTS2 from matrix proteins after entry into the peroxisome [22]. DEG15 dimerization is mediated by the calmodulin-like protein CML3 [23], a peroxisomal protein [24] that contributes to peroxisome metabolism, as evidenced by the slight β-oxidation defects of a *cml3* mutant [23]. However, unlike *deg15* mutants, which exhibit a complete block in PTS2 processing [25,26], *cml3* mutants process PTS2 proteins like wild type [23], suggesting that the β-oxidation defects in *cml3* might stem from additional to-be-discovered roles for CML3 (and presumably calcium) in the peroxisome.

Systematic large-scale plant peroxisome research

Given the metabolic plasticity and diversity of plant peroxisomes (Table 1), large-scale systematic approaches are needed to thoroughly characterize plant peroxisome functions. Proteome analyses combined with *in vivo* protein targeting validations are established for plant peroxisomes [27–30] and continue to be expanded, for instance to peroxisomes isolated from etiolated seedlings [31].

Most peroxisomal matrix proteins possess a C-terminal PTS1. Accurately predicting functional PTS1 sequences is challenging because non-canonical targeting signals in higher plants display high variability and few have been experimentally validated. The first plant-specific PTS1 prediction methods uncovered many non-canonical PTS1 examples and corresponding novel matrix proteins [32,33]. More quantitative assessments of plant peroxisomal protein import are beginning, and comparing *in silico* predictions, semi-quantitative *in vivo* targeting analyses, and *in vitro* PEX5 binding affinities of PTS1 variants reveals broad agreement [34]. The first plant-specific web server, PredPlantPTS1 (ppp.gobics.de), for predicting plant PTS1 proteins using these models is available [35]. Due to challenges posed by higher motif and position variability, plant-specific PTS2 prediction algorithms are not yet available.

These large-scale approaches have facilitated the discovery of new peroxisome functions (see above). Systematic phenotypic screening has revealed β-oxidation-related functions for several newly identified peroxisomal proteins [36,37]. Extending mutant screens to abiotic stress conditions combined with co-expression analyses uncovered *Arabidopsis* peroxisomal proteins involved in drought response, including the LON2 protease (see below) and peroxisomal hydroxypyruvate reductase, which is involved in photorespiration [38].

Peroxisome biogenesis

A core set of peroxins is conserved in plants, fungi, and mammals. Peroxisomal matrix proteins are imported into the organelle by PEX5 and PEX7, which are interdependent in plants and form a dual receptor complex; PEX5 requires PEX7 for stability [39], and PEX7 requires PEX5 for cargo delivery to the peroxisome [40–42]. Cargo-loaded receptors dock with PEX13 and PEX14 at the peroxisomal membrane (Figure 2), allowing PEX5 to enter the membrane to form a ligand-gated pore that transports cargo into the matrix [43]. Many questions regarding this process remain, including the oligomerization status of the receptors during import, the specific roles of the two docking peroxins, and the mechanism of cargo release. A novel covalent biotin label transfer method applied to *in vitro* peroxisomal import reveals that an N-terminally truncated version of PEX5, which lacks the PEX7-binding domain but retains PEX14-and PTS1-binding regions, initially docks in monomeric form to PEX14 in the peroxisomal membrane [44]. In addition to docking roles, *in vitro* studies suggest that PEX14-PEX5 interactions facilitate unloading of PTS2 cargo, but not PTS1 cargo, into *Arabidopsis* peroxisomes [45]. This result implicates the other (non-homologous) docking peroxin, PEX13, in PTS1 cargo release. Intriguingly, *PEX13* is an essential gene in *Arabidopsis* [21] whereas *PEX14* null alleles are viable and display some residual matrix protein import [46], indicating that PEX14 facilitates but is not absolutely required for matrix protein import in plants.

After cargo delivery, PEX5 and PEX7 are returned to the cytosol for further import rounds. PEX5 recycling is mediated by monoubiquitination and requires a suite of peroxisome-associated ubiquitination components (Figure 2): i) the ubiquitin-conjugating enzyme PEX4 tethered to the peroxisome by PEX22 [47], ii) three PMP RING domain ubiquitin-protein ligases (PEX2, PEX10, and PEX12 [48]), and iii) the PEX1-PEX6 heterohexameric ATPase tethered to the peroxisome by PEX26 [reviewed in 49]. Single *Arabidopsis* mutants

defective in several of these peroxins inefficiently remove PEX5 from the membrane after cargo delivery, triggering ubiquitin-dependent PEX5 degradation in *pex6* mutants [50,51] or PEX5 accumulation in the membrane in *pex4* [51,52] and *pex12* mutants [53]. PEX5 levels are restored in *pex2 pex6* or *pex4 pex6* double mutants [52,54], implicating PEX2 and PEX4 in polyubiquitinating PEX5 for proteasomal degradation when PEX5 retrotranslocation is slowed in *Arabidopsis*, as in yeast [reviewed in 55]. Little is known about PEX7 recycling in plants, but PEX7 recycling and matrix protein import are impaired when GFP-PEX7 is expressed in *Arabidopsis* [56]. These defects stem from excessive degradation of unmodified PEX7 and can be prevented by mutation of the RabE1c GTPase [56]. It will be interesting to learn whether RabE1c plays a role in receptor recycling in wild-type plants.

PEX5 retention in the peroxisomal membrane appears to impair peroxisome function. For example, decreasing PEX5 degradation in *pex6* mutants by mutating *PEX4* heightens physiological and molecular defects [52]. Conversely, increasing PEX5 degradation by elevating the growth temperature ameliorates *pex4* defects [51]. The ATP-dependence of *in vitro* PEX5-PEX14 docking [44] implies that ATP-driven release of endogenous PEX5 from the peroxisomal membrane is necessary for the docking complex to recruit new cargo-loaded PEX5 moieties, suggesting that docking complex impedance might underlie the detrimental effects of excessive membrane-associated PEX5. However, slightly reducing *PEX13* expression, which is expected to reduce PEX5 docking, ameliorates *pex4* and *pex6* physiological defects without notably improving matrix protein import [52], implying that excessive PEX5 retention in the membrane directly impairs peroxisome metabolism, perhaps by allowing leakage of some peroxisome contents.

How membrane proteins reach peroxisomes is less well understood. In *Saccharomyces cerevisiae*, PEX3 and PEX19 are needed for formation of pre-peroxisomes from the ER and PMP insertion. PEX19 is the receptor that delivers PMPs to PEX3 for insertion. In mammals, PEX16, which is conserved in plants, is needed to recruit PEX3 to the ER [reviewed in 57]. Overexpression of PEX16 derivatives modified to slow ER-to-peroxisome trafficking demonstrates that *Arabidopsis* PEX16 can recruit PEX3 to the ER in plant cells, as it does in mammals [58]. Although RNAi knockdown of *PEX3*, *PEX16*, or *PEX19* in *Arabidopsis* alters peroxisome morphology [59], genetic dissection of early steps in peroxisome biogenesis in plants has been slow because, unlike most *PEX* genes, *PEX3* and *PEX19* are duplicated in *Arabidopsis*, and mutants in these peroxins have not emerged from forward genetic screens. However, disrupting both *PEX19* isoforms results in embryonic lethality [60], and *PEX16* disruption confers embryonic defects [61] including loss of peroxisomes [62]. Future combinations of reporters that allow PMP visualization in mutants defective in various peroxins is expected to enhance understanding of PMP trafficking.

Matrix protein degradation and pexophagy

As peroxisomes compartmentalize oxidative reactions, peroxisomal proteins suffer oxidative damage and require turnover (Figure 1). The metabolic transition in germinating seedlings from the glyoxylate cycle to photorespiration is accompanied by the degradation of glyoxylate cycle enzymes [47,54,63,64]. This degradation is slowed when fatty acid β-

oxidation is impaired [63,64] and accelerated when catalase is disrupted [64], suggesting that peroxisomal H_2O_2 promotes matrix protein degradation.

Beyond individual matrix proteins, peroxisomes themselves can be degraded via pexophagy, a specialized form of autophagy [reviewed in 65]. In autophagy, cellular constituents are packaged into autophagosomes and sent to the vacuole for degradation. Because autophagy is not essential for life in *Arabidopsis* [reviewed in 66], peroxisomes can be examined in null mutants incapable of autophagy, including pexophagy. Autophagy-deficient seedlings accumulate peroxisomes under normal growth conditions [67,68]. Similarly, tobacco cell cultures degrade peroxisomes via pexophagy not only in carbon-starved but also in optimal growth conditions [69], reflecting pexophagy activity in non-stressed cells. However, glyoxylate cycle enzymes are only slightly stabilized in autophagy mutants [67,70], suggesting that additional pathways exist to degrade these enzymes.

Relationships between pexophagy and matrix protein turnover have been illuminated through studies of the peroxisomal LON2 protease. LON proteins are ATP-dependent homo-oligomeric proteases [reviewed in 71] that are cytosolic in prokaryotes and organellar in eukaryotes. Mutations in the *Arabidopsis* peroxisomal LON isoform (LON2) confer peroxisome-related defects [26,63] that stem from excessive pexophagy; preventing autophagy restores peroxisome numbers and function to *lon2* mutants [70,72]. Interestingly, glyoxylate cycle enzymes are destabilized in *lon2* mutants but are dramatically stabilized when LON2 and autophagy both are dysfunctional [70,72]. This synergy suggests complementary degradation mechanisms; these matrix proteins appear to be degraded by LON2 when autophagy is prevented and by pexophagy when LON2 is disabled. Intriguingly, a protease-dead LON2 variant can rescue the sparse peroxisome phenotype of *lon2* mutants but fails to efficiently degrade matrix proteins [72]. In contrast, an ATPase-dead LON2 variant fails to restore normal peroxisome numbers to *lon2* but degrades matrix proteins with normal kinetics [72]. These findings suggest that chaperone rather than protease activity of LON2 normally dampens pexophagy.

The recent demonstration of pexophagy in *Arabidopsis* [67,68,70,73] and tobacco cells [69] prompts the question of which proteins or signaling molecules target peroxisomes for destruction. It will be interesting to identify the LON2 substrates (and perhaps their post-translational modifications) that trigger pexophagy in *lon2* mutants and to learn whether these proteins also promote pexophagy in wild-type plants. Inactive catalase accumulates in peroxisomes of autophagy-defective mutants [68,73], but aggregated catalase is not the pexophagy signal, because pexophagy proceeds apace even in a mutant lacking the predominant seedling catalase isoform [68]. Ubiquitinated PMPs can target peroxisomes for pexophagy in mammalian cells [74], and yeast peroxisomes are directed to pexophagy in mutants (e.g., *pex6*) that accumulate polyubiquitinated PEX5 [75], but similar plant studies have not been reported. It will be interesting to discover how pexophagy is impacted in various *Arabidopsis pex* mutants.

Intriguingly, *Arabidopsis* PEX10 and PEX6 interact with AUTOPHAGY-RELATED8 (ATG8) [76], the ubiquitin-like protein that decorates the nascent autophagosome membrane to mediate autophagic cargo engulfment [reviewed in 66]. This interaction suggests that

certain peroxins might promote pexophagy in addition to their roles in matrix protein import. Strikingly, a single amino acid change (G93E) in PEX10 adjacent to the PEX10-ATG8 interaction domain [76] confers seedling lethality when homozygous and aberrant peroxisome morphology when heterozygous [77]. PEX10 carrying this G93E mutation still interacts with ATG8 in bimolecular fluorescence complementation experiments; the additional negative charge suggests that PEX10-ATG8 binding would be increased, perhaps causing excessive targeting of peroxisomes to the autophagy machinery [76]. It will be interesting to learn whether disabling autophagy abrogates the lethality of the *pex10-G93E* mutant.

Peroxisome morphology dynamics

Plastids, mitochondria, and peroxisomes were traditionally considered as physically separated spherical or rod-shaped organelles. However, these organelles physically interact, and peroxisomes can alter their shape to increase interaction surfaces between collaborating organelles. During photorespiration, recycling phosphoglycolate to phosphoglycerate requires intensive metabolic intermediate exchange between chloroplasts, peroxisomes, and mitochondria along with coordination of photorespiratory enzyme activities. An intimate physical association between these three compartments can be observed by transmission electron microscopy [78]. Peroxisomes adjacent to chloroplasts change from spherical to elliptical during photorespiration in *Arabidopsis* leaf mesophyll cells, increasing their interaction area and adhesion, determined by femtosecond laser technology, to the chloroplast surface [79]. This inter-organellar adhesion may provide contact sites that ensure efficient flow of photorespiratory metabolites between organelles. Interestingly, mutations in the RING domain of PEX10, but not other RING peroxins, can disrupt peroxisome-chloroplast interactions and confer photorespiration defects [77,80], again suggesting roles for PEX10 beyond PEX5 recycling.

Plant peroxisomes also form dynamic thin protrusions referred to as peroxules (Figure 1) that extend in response to H_2O_2 and hydroxyl radical stress and retract upon stress mitigation [81]. High light intensity induces ROS and increases peroxule-mitochondria interactions [82]. The tubulated peroxisomes become constricted and eventually undergo fission to increase peroxisome numbers [82]. Similarly, cadmium treatment induces cytosolic ROS and rapid PEX11a-dependent peroxule formation, followed by peroxisome elongation and division [83]. Beyond increasing surface and contact areas, peroxule-mediated inter-organellar contacts might assist in delivering enzymes from peroxisomes to their final subcellular destination. For example, the major triacylglycerol lipase involved in lipid reserve mobilization during seedling establishment in *Arabidopsis*, SDP1 (see above) [14], localizes to the peroxisomal membrane in young seedlings before traveling to its site of action on the oil body surface. This relocalization is accompanied by peroxule extensions from peroxisomes to oil bodies and facilitated by the core retromer complex [84], which is best known for roles in endosomal protein trafficking [reviewed in 85]. It will be interesting to learn if additional instances of peroxule dynamics involve inter-organellar protein trafficking or the retromer complex.

Conclusions and future directions

Although combining genetics and biochemistry with large-scale bioinformatics and proteomics has vastly improved our knowledge of plant peroxisome functions and dynamics, much remains to be discovered. The array of physiological functions of plant peroxisomes is amazingly wide compared to fungi and animals. Functions are unknown for several newly identified matrix proteins, and functional mechanisms need to be assembled from fragmentary information, such as the role of plant peroxisomes in biotic and abiotic stress responses. Several peroxisomal metabolite transporters remain to be identified, and deciphering the post-translational regulation that plant peroxisomes use to coordinate metabolite flow is only beginning. Our understanding of peroxisome biogenesis from the ER remains rudimentary, as neither pre-peroxisomes nor ER-derived vesicles delivering PEX proteins to mature peroxisomes have been imaged in plants or animals. Which proteins mediate pre-peroxisome budding from the ER and how cells decide between *de novo* biogenesis and fission of existing peroxisomes remain to be elucidated. It also is not known how peroxisomes obtain membrane lipids for expansion. Even matrix protein import is incompletely understood. The residual matrix protein import observed in *pex14* null mutants [46] implies important roles for the PEX13 docking peroxin, which might be explored using viable *pex13* partial loss-of-function alleles [53,86]. Moreover, some peroxins, such as PEX10, are implicated in processes in addition to matrix protein import [76,77,80] that may be plant-specific. We are only beginning to understand the roles of peroxules in peroxisome metabolism and inter-organellar communication. Now that it is clear that plant peroxisomes can be degraded by pexophagy, it is important to elucidate how sub-populations of “old” or dysfunctional peroxisomes are specifically identified, spatially separated, and targeted to the pexophagy machinery. As additional biological roles for the large collection of peroxisomal enzymes are uncovered, the importance of this fascinating organelle in diverse facets of plant biology becomes increasingly apparent.

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Abbreviations

AGT1, serine	glyoxylate aminotransferase
ATG	AUTOPHAGY-RELATED
ABC	ATP-binding cassette
CAT	catalase
CGI-58	COMPARATIVE GENE IDENTIFICATION-58

DHNA	1,4-dihydroxy-2-naphthoate
ER	endoplasmic reticulum
JA	jasmonate
OPPP	oxidative pentose phosphate pathway
PEX	peroxin
pICDH	peroxisomal NADP-dependent isocitrate dehydrogenase
PMP	peroxisomal membrane protein
PTS	peroxisome-targeting signal
PXA	PEROXISOMAL ABC-TRANSPORTER
RabGAP22	Rab GTPase-activating protein 22
ROS	reactive oxygen species
SDP	SUGAR DEPENDENT

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Papers of particular interest, published within the period of review, have been highlighted as:

• of special interest

•• of outstanding interest

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Highlights

- Plant peroxisomes house a diverse and expanding array of metabolic functions.
- Peroxisome biogenesis involves a suite of peroxin proteins.
- Plant peroxisomes can be degraded via pexophagy.
- Dynamic peroxules facilitate peroxisome interactions with other organelles.

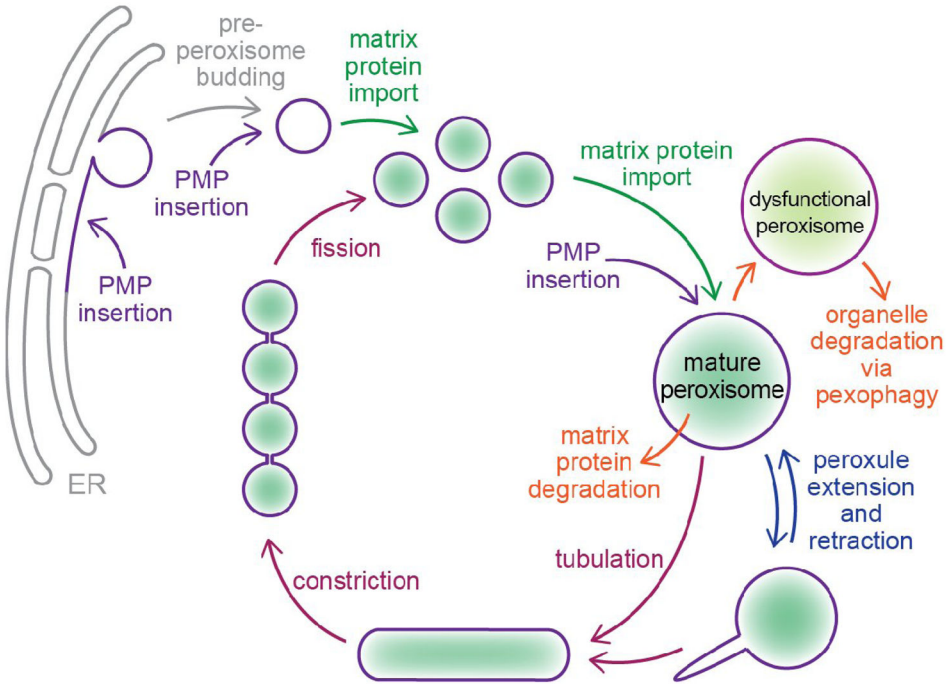

Figure 1. Peroxisome dynamics

Peroxisomal membrane proteins (PMPs) are inserted directly into peroxisomes or into the ER membrane from which pre-peroxisomes can bud. PMPs include a subset of the PEX proteins necessary for import of matrix proteins into the organelle (Figure 2). Mature peroxisomes extend and retract peroxules, tubulate, and divide by fission, which is considered to be the predominant mode of peroxisome proliferation. Peroxisomal quality control includes degrading damaged or obsolete matrix proteins and eliminating dysfunctional or superfluous peroxisomes via pexophagy, a specialized form of autophagy.

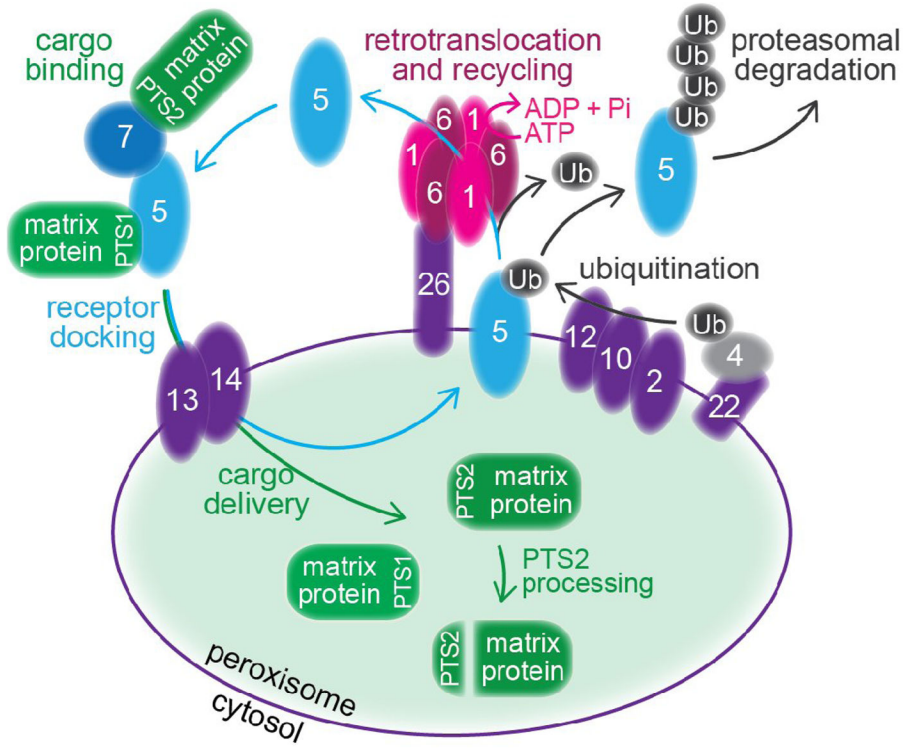

Figure 2. Import of matrix proteins into peroxisomes

Import of peroxisomal matrix proteins (green) is facilitated by peroxins (numbered ovals), some of which are integral membrane proteins (purple). The PEX5 and PEX7 receptors (blue) bind to PTS1 and PTS2 proteins in the cytosol, respectively, and dock at the peroxisomal membrane via interactions with each other and with PEX13 and PEX14. After cargo delivery into the matrix, the N-terminal PTS2 domain is cleaved. Membrane-associated PEX5 is ubiquitinated, presumably with the assistance of the PEX4 ubiquitin (Ub)-conjugating enzyme (gray) and the PEX2-PEX10-PEX12 ubiquitin-protein ligase complex. PEX5 retrotranslocation back to the cytosol for reuse requires the PEX1-PEX6 heterohexameric ATPase (pink). When retrotranslocation is impeded, PEX5 can be polyubiquitinated and degraded by the proteasome.

Table 1

Plant peroxisome functions.

Functions	Metabolites or proteins
Catabolic	
ROS detoxification	H_2O_2, $O_2^{\cdot-}$
Fatty acid β-oxidation	Straight-chain, branched, saturated, unsaturated fatty acids
Catabolism of primary metabolites	Purines, branched-chain amino acids
Catabolism of secondary metabolites	Polyamines
Defense compound metabolism	Indole glucosinolates
Biosynthetic	
ROS generation	H_2O_2, $O_2^{\cdot-}$, NO
Glyoxylate cycle	C_4 metabolite (succinate)
Photorespiration	C_3 metabolite and amino acids (glycerate, glycine, serine)
Hormone biosynthesis	Jasmonic acid (JA) and indole-3-acetic acid
Co-factor biosynthesis (and recycling)	Phylloquinone, biotin, NAD(P)H
Secondary metabolism	Polyamines, benzoic acid, isoprenoids (mevalonate)
Non-metabolic	
Protein processing and degradation	DEG15, LON2
Reversible phosphorylation	CPK1, GPK1, PP2A
Calcium signaling	CML3