Biochimica et Biophysica Acta xxx (2016) xxx–xxx

Contents lists available at ScienceDirect

Biochimica et Biophysica Acta

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

Review

Damage to photosystem II by lipid peroxidation products

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ARTICLE INFO

Article history:
Received 22 July 2016
Received in revised form 7 October 2016
Accepted 8 October 2016
Available online xxxx

Keywords:
Lipid peroxidation
Photosystem II
Protein oxidation
Reactive oxygen species
Reactive carbonyl species

ABSTRACT

Background: Photosystem II proteins of higher plant chloroplasts are prone to oxidative stress, and most prominently the reaction center-binding D1 protein is damaged under abiotic stress. The reactive oxygen species produced under these stress conditions have been suggested to be responsible for the protein injury.
Scope of review: Recently, it has been shown that the primary and secondary products of non-enzymatic and enzymatic lipid peroxidation have a capability to modify photosystem II proteins. Here, we give an overview showing how lipid peroxidation products formed under light stress and heat stress in the thylakoid membranes cause oxidative modification of proteins in higher plant photosystem II.
Major conclusions: Damage to photosystem II proteins by lipid peroxidation products represents a new mechanism underlying photoinhibition and heat inactivation.
General significance: Complete characterization of photosystem II protein damage is of crucial importance because avoidance of the damage makes plants to survive under various abiotic stresses. Further physiological significance of photosystem II protein oxidation by lipid peroxidation product should have a potential relevance to plant acclimation because the oxidized proteins might serve as signaling molecules.
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1. Introduction

Reactive oxygen species (ROS) are produced as by-products of various cellular processes, including cellular respiration in mitochondria and photosynthesis in chloroplasts. When ROS formation is properly balanced by the antioxidant system, ROS play an important role in several cellular processes, particularly the defense against infection during phagocytosis and the cellular signaling associated with acclimation. However, when ROS formation in the cell exceeds the capacity of the antioxidant system, biomolecules (nucleic acid, proteins and lipids) are oxidized. Protein oxidation and lipid peroxidation are initiated by hydroxyl radicals ($HO^{\bullet}$) formed by metal-mediated reduction of hydrogen peroxide (H_2O_2) via Fenton reaction. Due to the highly positive standard redox potential of $HO^{\bullet}/H_2O$ redox couple ($E_0'(HO^{\bullet}/H_2O) = 2.3$ V, pH 7), $HO^{\bullet}$ is the most powerful oxidant of proteins and lipids [1]. The superoxide anion radical ($O_2^{\bullet -}$) is weakly reactive toward proteins and lipids; however, its protonated form $HO_2^{\bullet}$, known as hydroperoxyl radical, is considered to be more reactive. The higher ability of $HO_2^{\bullet}$ to abstract hydrogen from amino acid or polyunsaturated fatty acid is due to the more oxidizing power ($E_0'(O_2^{\bullet -}/H_2O_2)$ and $E_0'(HO_2^{\bullet}/H_2O_2)$ redox couples is 0.89 V and 1.06 V, respectively) and the lack of negative charge on the molecule. Apart from $HO^{\bullet}$ and $HO_2^{\bullet}$, singlet oxygen ($^1O_2$) formed by excitation energy transfer from triplet pigments to O_2 is known as potential oxidant of proteins and lipids [2].

2. General aspects of protein oxidation, cleavage and aggregation

Proteins are prone to oxidative stress due to their high abundance, association with pigments which serve as photosensitizers and coordination of metals which might serve as Fenton reagents. Several lines of evidence have been provided that oxidative modification and damage of the proteins occur in the stressed cells [3,4]. Indeed, the accumulation of oxidized protein is well correlated with cell aging and various diseases. In many studies that were intended to show protein oxidation, ROS produced through non-physiological processes were used for the model proteins such as bovine serum albumin. From those studies, it was shown that ROS cause oxidation of the protein backbone and the side chains of specific amino acid residues, which results in protein backbone cleavage (fragmentation), protein aggregation (protein-protein cross-linking), change in the protein hydrophobicity, and conformational change of the proteins such as protein unfolding, leading to dysfunction of the proteins [1,4].

When protein backbone is attacked by $HO^{\bullet}$ produced from H_2O by ionizing radiation ($H_2O \rightarrow HO^{\bullet} + H^{\bullet}$) or the Fenton reaction from H_2O_2 in the presence of Fe^{2+} [$Fe^{2+} + H_2O_2 \rightarrow Fe^{3+} + HO^{\bullet} + OH^-$], abstraction of the α-hydrogen atom of an amino acid takes place and a protein alkyl radical ($P^{\bullet}$) is produced [1] (Fig. 1A, reaction 1). In the presence of O_2, $P^{\bullet}$ reacts with O_2 and forms a protein peroxyl radical ($POO^{\bullet}$) (Fig. 1A,

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http://dx.doi.org/10.1016/j.bbagen.2016.10.005
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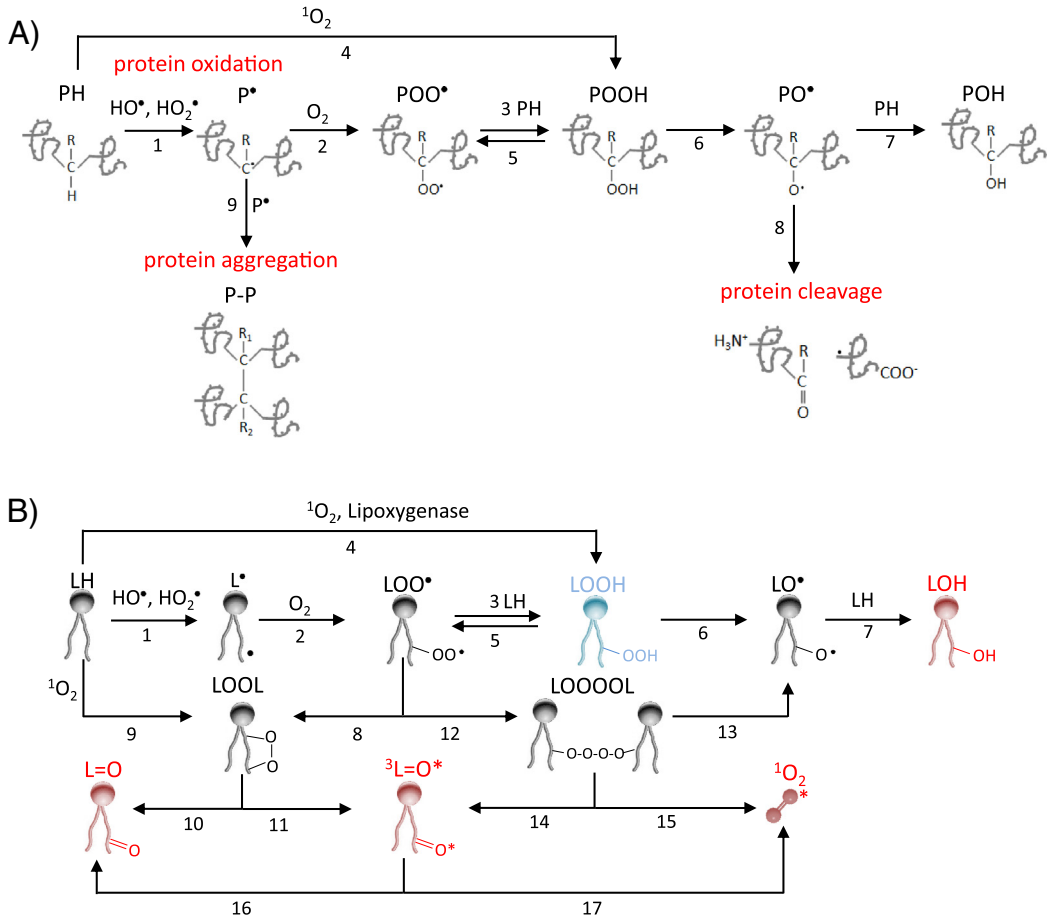

Fig. 1. Protein and lipid oxidative modification. (A) Protein oxidation, cleavage and aggregation occur by the following reactions: (1) formation of protein alkyl radical by hydrogen abstraction from amino acid by radical ROS, (2) formation of protein peroxyl radical by interaction of protein alkyl radical with O_2, (3) formation of protein hydroperoxide by hydrogen abstraction from another protein by protein peroxyl radical, (4) formation of protein hydroperoxide by addition of $^1O_2$ to double bond of amino acid, (5) oxidation of protein hydroperoxide to protein peroxyl radical, (6) reduction of protein hydroperoxide to protein alkoxyl radical, (7) formation of protein hydroxide by hydrogen abstraction from another protein by protein alkoxyl radical, (8) formation of fragments with carbonyl group and carbon-centered radical by β-scission of protein alkoxyl radical, (9) formation of protein-protein aggregates by recombination of protein alkyl radicals. (B) The primary (blue) and secondary (red) products of lipid peroxidation are formed by the following reactions: (1) formation of lipid alkyl radical by hydrogen abstraction from polyunsaturated fatty by radical ROS, (2) formation of lipid peroxyl radical by interaction of lipid alkyl radical with O_2, (3) formation of lipid hydroperoxide by hydrogen abstraction from an adjacent lipid by lipid peroxyl radical, (4) formation of lipid hydroperoxide by addition of $^1O_2$ to double bond of polyunsaturated fatty acid or by lipoxygenase, (5) oxidation of lipid hydroperoxide to lipid peroxyl radical, (6) reduction of lipid hydroperoxide to lipid alkoxyl radical, (7) formation of lipid hydroxide by hydrogen abstraction from an adjacent lipid by lipid alkoxyl radical, (8) formation of cyclic peroxide by cyclization of lipid peroxyl radical, (9) formation of cyclic peroxide by addition of $^1O_2$ to double bond of polyunsaturated fatty acid, (10) formation of reactive carbonyl species by decomposition of cyclic peroxide, (11) formation of triplet excited carbonyl by decomposition of cyclic peroxide, (12) formation of linear tetroxide by recombination of two lipid peroxyl radicals, (13) decomposition of linear tetroxide to lipid alkoxyl radical, (14) decomposition of linear tetroxide to triplet excited carbonyl, (15) decomposition of linear tetroxide to $^1O_2$, (16) de-excitation of triplet excited carbonyl to ground carbonyl, (17) formation of $^1O_2$ by energy transfer from triplet excited carbonyl to O_2. L and P stand for lipid and protein, respectively.

reaction 2), which oxidizes another protein, while a protein hydroperoxide (POOH) is formed (Fig. 1A, reaction 3). Alternatively, POOH is formed by the interaction of protein with $^1O_2$ (Fig. 1A, reaction 4). POOH might be re-oxidized to POO$^•$ (Fig. 1A, reaction 5) or reduced to a protein alkoxyl radical (PO$^•$) (Fig. 1A, reaction 6), depending on the reaction conditions and availability of supporting factors. PO$^•$ might subsequently either oxidize another protein forming a protein hydroxide (POH) (Fig. 1A, reaction 7) or undergo β-scission forming C-terminal and N-terminal fragments (Fig. 1A, reaction 8). Peptide bond cleavage is associated with the formation of carbon-centered radicals and carbonyl groups (C=O) on C-terminal and N-terminal fragments, respectively. When enough O_2 is not available after the first P$^•$ is formed, P$^•$ reacts with another P$^•$ to form protein aggregates with the nearby proteins (P-P) (Fig. 1A, reaction 9). In terms of the protein cleavage and aggregation, we realize that both processes are not competitive with each other but may proceed additionally under the oxidative stress conditions where O_2 is available. Both the protein cleavage and aggregation should cause irreversible damage to the proteins and inactivate their functions. The peptide bond cleavage is also known to occur when glutamyl, aspartyl and propyl side chains are attacked by ROS [1]. In particular, oxidation of propyl side chain is well correlated with the peptide bond cleavage in a quantitative manner. Because HO$^•$ used here is produced in a non-physiological way, it should be made clear whether the events described here really occur *in vivo* in the living cells under physiological oxidative stress conditions.

As already described above, the side chains of the amino acids can be oxidatively modified [4]. The side chain modification confers both the reversible and irreversible changes in the structure of polypeptides. A well-known case for the reversible oxidation of amino acid side chain and accompanying reversible structural change of proteins is oxidation of cysteine and methionine. Oxidation of sulfhydryl group of cysteine residues (-SH) leads to the formation of disulfide bridge (-S-S-) between the two cysteines. The -S-S- bridge is converted back to two -SH groups of two cysteine residues upon reduction. Methionine residues are converted to methionine sulfoxide under an oxidation condition, and further to methionine sulfone under a stronger oxidation condition. Methionine-sulfoxide reductase is known to reduce methionine sulfoxide in collaboration with the reduced type of thioredoxin and regenerate methionine residues, thus possibly working as a radical

Please cite this article as: P. Pospíšil, Y. Yamamoto, Damage to photosystem II by lipid peroxidation products, Biochim. Biophys. Acta (2016), http://dx.doi.org/10.1016/j.bbagen.2016.10.005

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