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Review

Adaptive response to oxidative stress: Bacteria, fungi, plants and animals

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ABSTRACT

Reactive oxygen species (ROS) are continuously produced and eliminated by living organisms normally maintaining ROS at certain steady-state levels. Under some circumstances, the balance between ROS generation and elimination is disturbed leading to enhanced ROS level called "oxidative stress". The primary goal of this review is to characterize two principal mechanisms of protection against oxidative stress — regulation of membrane permeability and antioxidant potential. The ancillary goals of this work are to describe up to date knowledge on the regulation of the previously mentioned mechanisms and to identify areas of prospective research and emerging directions in investigation of adaptation to oxidative stress. The ubiquity for challenges leading to oxidative stress development calls for identification of common mechanisms. They are cysteine residues and [Fe,S]-clusters of specific regulatory proteins. The latter mechanism is realized via SoxR bacterial protein, whereas the former mechanism is involved in operation of bacterial *OxyR* regulon, yeast H_2O_2-stimulon, plant NPR1/TGA and Rap2.4a systems, and animal Keap1/Nrf2, NF-κB and AP-1, and others. Although hundreds of studies have been carried out in the field with different taxa, the comparative analysis of adaptive response is quite incomplete and therefore, this work aims to cover a plethora of phylogenetic groups to delineate common mechanisms. In addition, this article raises some questions to be elucidated and points out future directions of this research. The comparative approach is used to shed light on fundamental principles and mechanisms of regulation of antioxidant systems. The idea is to provide starting points from which we can develop novel tools and hypothesis to facilitate meaningful investigations in the physiology and biochemistry of organismic response to oxidative stress.

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Abbreviations: AHP1, alkylhydroperoxide reductase; AHR, aryl hydrocarbon receptor; ARE, antioxidant response element; 2CPA, 2-Cys peroxiredoxin-A; CRD, cysteine rich domain; Cul3, Cullin 3; DPH, diphenylhexatriene; EpRE, electrophile response element; ERK, extracellular signal-regulated kinases; JA, jasmonic acid; G6PDH, glucose-6-phosphate dehydrogenase; GSH1, γ-glutamylcysteine synthase; GSH2, glutathione synthase; GPX2(3), glutathione peroxidases; GSH, glutathione; HIF-1α, hypoxia inducible factor alfa; HPI and HPII, hydroxyperoxidases; IkB, inhibitory protein of NF-κB; IkK, IkB kinase; Kelp1, Kelch-like ECH-associated Protein 1; MAP-kinases, mitogen-activated protein kinases; MDR, multidrug resistance; MPT, mitochondrial permeability transition pore; PR-1 gene, pathogenesis-related gene; NES, nuclear exporting sequence (NES); NF-κB, nuclear factor-κB; NOS, NO-synthase; NPR1, nonoexpressor of pathogenesis-related gene 1; Nrf2, NF-E2-related factor 2; OmpF, outer membrane protein F; PERK, PKR-like endoplasmic reticulum kinase; PKC, protein kinase C; PPAR, peroxisome proliferator-activated receptor; RNS, reactive nitrogen species; ROS, reactive oxygen species; SA, salicylic acid; SAR, systemic acquired resistance; SOD, superoxide dismutases; soxR, superoxide response; TMA-DPH, trimethylammonium diphenylhexatriene; TRX2, thioredoxins 2; TRR1, thioredoxin reductase 1; TSA1, thioredoxin peroxidases 1; VCAM-1, vascular cell adhesion molecule 1; bZip, basic domain/leucine zipper transcription factors.

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1. Introduction

The generation of reactive oxygen species (ROS) is an inevitable aspect of life under aerobic conditions. ROS are continuously produced as side-products of certain metabolic pathways and also by some specific systems under fine cellular control. At the same time, ROS are degraded via several mechanisms, both, specific and non-specific. Two different processes, generation and degradation of ROS, usually are under delicate cellular control and very low ($<10^{-8}$ M) steady-state ROS concentrations are maintained (Halliwell and Gutteridge, 1989). However, under some circumstances, the balance between ROS production and elimination is disturbed leading to their enhanced steady-state level called "oxidative stress". Its development is either the reason, or common event of many pathological states, including aging. Therefore, it is clear why oxidative stress investigation has become increasingly popular not only from basic points of view, but also from different applied aspects including medicine, sport science, toxicology and environmental science. Cell survival of oxidative stress challenge depends on physiological state, intensity and nature of stress. The possibility to enhance protective potential is among the critical issues. Therefore, this review will deal with two well-characterized mechanisms of increase of cellular tolerance to oxidative stress: (*i*) permeability of cellular membranes to reactive species and (*ii*) up-regulation of antioxidant and associated enzymes. However, before the analysis of enhancement of tolerance, I will briefly discuss the key aspects of ROS metabolisms, their effects on cells and their consequences.

Over 90% of oxygen consumed by living organisms is used to produce energy by oxidative phosphorylation with operation of the electron-transport chain *via* a four-electron mechanism leading to ATP and water production. Consecutive addition of one by one electron to oxygen molecule also finally leads to water production (but no ATP is produced) via intermediate ROS forms (Fig. 1). The latter include free radicals such as superoxide anion ($O_2^{\bullet -}$) and hydroxyl radical ($^{\bullet}OH$) and also non-radical reactive species, such as hydrogen peroxide (H_2O_2) and singlet oxygen. They seem are the best

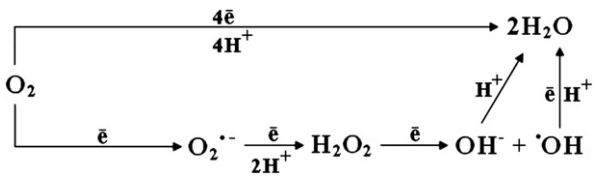

Fig. 1. Different ways of oxygen reduction in biological systems. The upper part of the scheme shows four-electron reduction of oxygen molecule in electron transport chain. The bottom part demonstrates the consecutive addition of one electron to oxygen molecule with the formation of intermediate products – reactive oxygen species – superoxide anion radical, hedrogen peroxide and hydroxyl radical. To the end, partially reduced forms may accept four electrons and combining with protons give water molecule. In both cases the maximally reduced state of oxygen is reached in water molecule.

studied among all reactive species. Although very important, reactive nitrogen species (RNS) such as nitric oxide ($^{\bullet}NO$), nitric peroxide (peroxynitrite, $OONO^{\bullet -}$) and their derivatives have garnered less attention. Along with ROS, $^{\bullet}NO$ and other gaseous compounds such as carbon monoxide (CO) and hydrogen sulfide (H_2S) play important regulatory functions (Li and Moore, 2007).

The main part of ROS (usually over 90%) in living organisms is produced by electron-transport chains — mitochondrial, endoplasmic reticulum, plasmatic and nuclear membranes, and photosynthetic system (Starkov, 2008). In addition, minor ROS amounts are generated by some enzymes such as oxidases, through autooxidation of different molecules. The oxidases known producing ROS are NADPH oxidase, lipoxygenases, cyclooxygenases, and xanthine oxidase (Puddu et al., 2008), whereas molecules entering autooxidation may be exogenous and endogenous origination like neurotoxin 5-hydroxydecanoate (Rodriguez-Pallares et al., 2009) or catecholamines (Callaway et al., 2003) and many other compounds.

Fig. 1 demonstrates ROS metabolism. One electron reduction of molecular oxygen finally yields water and can be realized spontaneously. However, certain enzymes can enhance the speed of the transformations many-fold. Superoxide dismutases (SODs; EC 1.15.1.1) speed-up the dismutation of superoxide anion to molecular oxygen and hydrogen peroxide. Catalases (EC 1.11.1.6) decompose hydrogen peroxide, yielding water and molecular oxygen, and peroxidases (EC 1.11.1.x), using some reductants and peroxides as cosubstrates, produce water and reduced products. The hydroxyl radical is the most active oxidant among the previously mentioned ROS. It is short-lived and, therefore, has a short diffusion distance. Probably for this reason living organisms have not developed specific enzymatic systems for its detoxification. The prevention of its production is the most efficient way to protect cells against deleterious $^{\bullet}OH$ effects. Low molecular mass compounds such as vitamins C and E, glutathione (GSH), uric acid and some others form a group of low molecular mass antioxidants some of which can directly neutralize $^{\bullet}OH$.

Interaction of ROS with cellular components depends on many circumstances. However, place of formation, nature of ROS and target molecules are among main determinants. Chemical activity of ROS is enhanced in the order: $O_2^{\bullet -}$–H_2O_2–$^{\bullet}OH$. In fact, all cellular constituents can be modified by ROS. However, several types are especially susceptible. The enzymes containing [Fe–S]-clusters such as aconitase (E.C. 4.2.1.3), 6-phosphogluconate dehydratase (E.C. 4.2.1.12), and fumarate hydratase (E.C. 4.2.1.2), as well as ones possessing active thiol groups (e.g. glyceraldehyde-3-phosphate dehydrogenase (E.C. 1.2.1.12)) are particularly sensitive to ROS (Busi et al., 2006; Bouton, 1999; Hancock et al., 2006). Most ROS-modified molecules are subjected to degradation, but some may be accumulated in the cell. However, only few types of molecules modified by ROS can be repaired: this includes DNA, and oxidized cysteine and methionine residues in proteins that may be reduced to their initial forms (Lushchak, 2007).

As mentioned previously, ROS are continuously produced and eliminated and, therefore, their concentration is a dynamic parameter

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