doi:10.5505/sakaryamj.2013.87609

Lipid Peroxidation and Antioxidant Enzymes

Lipid Peroksidasyonu ve Antioksidan Enzimler

Gönül Gürol, Ali Aslan

Sakarya University Medical Faculty, Department of Physiology, Sakarya, Türkiye

Abstract

The role of reactive oxygen species (ROS) in the pathologic and physiologic effects on health has been emphasized in recent years. Production of ROS by mitochondria is generally thought to be the main cause of oxidative stres (OS). ROS are neutralized by various antioxidant defense mechanisms such as catalase, superoxide dismutase and glutathione peroxidase, vitamin C, vitamin E, vitamin A, pyruvate, glutathione, taurine and hypotaurine. Biomarkers of oxidative damage associated with human diseases could be used to diagnose and manage the diseases.

Keywords: lipid peroxidation, oxidative stres, antioxidants

Aplication: 28.01.2013 **Accepted:** 20.03.2013

Özet

Reaktif oksijen türlerinin (ROS) sağlık üzerindeki patolojik ve fizyolojik etkileri son yıllarda vurgulanmaktadır. Mitokondri tarafından ROS üretiminde oksidatif stresin ana neden olduğu genellikle düşünülmektedir. ROS katalaz, superoksid dismutaz, glutatyon peroksidaz, vitamin C, Vitamin E, vitamin A, pruvat, glutatyon, taurin, hiptaurin gibi antioksidan savunma mekanizmaları tarafından nötralize edilmektedir. Hastalıklarla ilişkili olan oksidatif hasar biyobelirteçleri hastalıkların teşhis ve idaresinde kullanılmaktadır.

Anahtar Kelimeler: lipid peroksidasyonu, oksidatif stres, antioksidanlar

Başvuru Tarihi: 28.01.2013 **Kabul Tarihi:** 20.03.2013

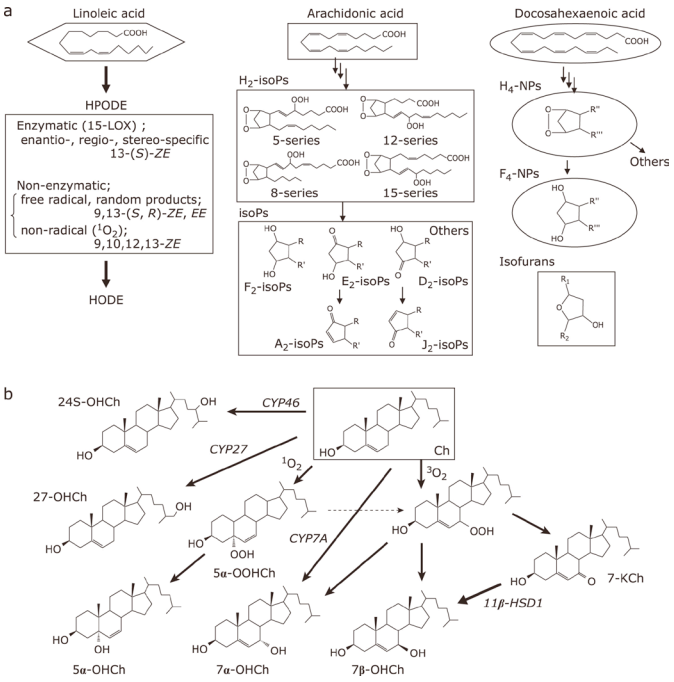
Giriş

Interest in the physiologic and pathologic effects of Reactive oxygen species (ROS) on health is growing. The most common ROS include superoxide, anion (O2-), hydrogen peroxide (H202), peroxyl (ROO-) radicals, and reactive hydroxyl (OH-) radicals. Oxygen free radicals may be implicated in the many clinical disorders such as carcinogenesis, and aging, apoptosis, neurologic and psychiatric diseases, cardiovascular diseases, respiratory distress syndrome, atherosclerosis, joint diseases, asthma and normal physiologic processes in humans. Basal levels of ROS production in cells could be related to several physiological functions including cell proliferation, apoptosis and homeostasis. However, ROS overproduction above basal levels can lead to oxidize DNA, lipids, sugars and proteins[1,2,3,4]. Production of reactive oxygen species (ROS) by mitochondria is generally thought to be the main cause of oxidative stress (OS)[5]. Carbon centered radicals quickly reacts with oxygen,

Yazışma Adresi/Corresponding to: Gönül Gürol, Sakarya University Medical Faculty, Department Of Physiology, Sakarya, Türkiye
GSM : 02642751010, e-mail: gonulgurol@gmail.com

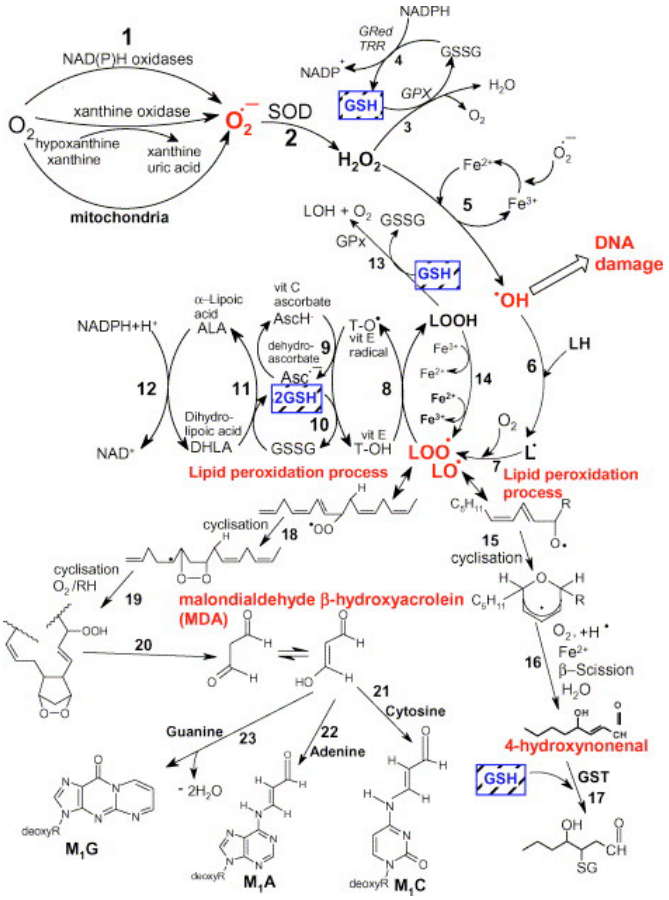
peroxyl radical form. This peroxyl radical initiator radical for lipid peroxidation [6]. Lipid peroxidation proceeds by 3 distinct mechanisms: (i) free radical-mediated oxidation, (ii) free radical independent non-enzymatic oxidation, and (iii) enzymatic oxidation. Both PUFA and cholesterol are oxidized by enzymatic and non-enzymatic pathways (Figure 1 a and b) [7].

Figure 1: Oxidation products of PUFA and cholesterol (8).



OS is caused by an imbalance between free radicals and antioxidants [8]. Most of the free radicals are neutralized by cellular antioxidant defence system (enzymes and non-enzymatic molecules). Enzymatic antioxidant defense systems is copper–zinc super oxide dismutase (Cu–Zn SOD), catalase (Cat), Selenium dependent glutathione peroxidase (GPx), glutathione reductase (GR). The non-enzymatic antioxidant defense system includes ascorbic acid (vitamin C), alfa-tocopherol (vitamin E), vitamin A, glutathione (GSH), melatonin, üric acid, albumin, haptoglobin, Cysteine, Ceruloplasmin, Transferrin, and Lactoferrin, Ferritin, Selenium, Oksipurinol, Ubiquinone, bilirubin, mannitol, Lipoic acid, Hemopeksin etc. Among the these antioxidants, SOD and its two isozymes, and catalase have a significant role. SOD spontaneously dismutates (O2-) anion to form O2 (Figure 2) and H2O2, while catalase converts H2O2 to O2 and H2O [6].

Figure 2: Pathways of ROS formation, the lipid peroxidation process and the role of antioxidants (10).



There is a relationship between OS and human diseases but the relationship between OS and the onset and progression of disease processes is not fully established. OS is thought to be the relationship between pathological conditions can be divided into two groups: (i) the first group involves diseases characterised by pro-oxidants shifting the thiol/disulphide redox state and impairing glucose tolerance—the so-called "mitochondrial oxidative stress" conditions (cancer and diabetes mellitus); (ii) the second group involves disease characterised by "inflammatory oxidative conditions" and enhanced activity of either NAD(P)H oxidase (leading to atherosclerosis and chronic inflammation) or xanthine oxidase-induced formation of ROS (implicated in ischemia and reperfusi-

on injury). The process of ageing is to a large extent due to the damaging consequence of free radical action (lipid peroxidation, DNA damage, protein oxidation) [9;10].

In the research studies, lipid peroxidation products are potential validated biomarkers for OS status in its related diseases. Among these markers are 'primary' products such as hydroperoxides, or 'secondary' products such as malondialdéhyde (MDA), 4-hydroxynonénal (4-HNE) and isoprostanes [11]. Table 1 summarises most representative biomarkers of oxidative damage associated with human diseases [10]. However, measuring OS can be difficult due to the presence of complex endogenous systems for correction and repair antioxidant defense systems [12].

The findings of data indicate that OS in pathophysiology of diseases, associated with lipid peroxidation, may induce the changes of hemostasis in the person. Altogether these findings suggest that the body to be taken to increase the quality of life in the name of naturel antioxidants nutrition required. However it is also necessary to protect the body from external factor such as ionizing radiation, smoking, toxins can trigger the production of ROS.

Table 1: Biomarkers of oxidative damage associated with some human diseases

Disease/biomarker	
Cancer MDA GSH/GSSG ratio NO_2-Try 8-OH-dG Cardiovascular disease HNE GSH/GSSG ratio Acrolein NO_2-Try F_2-isoprostanes Acrolein Rheumatoid arthritis F_2-isoprostanes GSH/GSSG ratio Alzheimer's disease MDA HNE GSH/GSSG ratio F_2-isoprostanes NO_2-Try AGE	Parkinson's disease HNE GSH/GSSG ratio Carbonylased pnoteins Iron Level Isehemia/reperfusion F_2-isoprostanes GSH/GSSG ratio Atheroselerosis MDA HNE Acrolein F_2-isoprostanes NO_2-Try Diabetes mellitus MDA GSH/GSSG ratio S-glutathionylated proteins F_2-isoprostanes NO_2-Try AGE
Abbreviations: MDA, malondialdehyde, HNE, 4-hydroxy,-2-nonenal,AGE, advanced, glycation and products, 8-OH-d, 8-hydroxy-20-deoxyguanosine, GSH, reduced glutathione, GSSG, oxidised glutathione, NO_2-Try, 3-nitro-tyrosine	

Kaynaklar

1. Sangameswaran B Balakrıshnan Br, Chumbhale Deshraj And Jayakar B. In Vıtro Antıoxıdant Actıvıty Of Roots Of Thespesıa Lampas Dalz And Gıbs. Pak. J. Pharm. Sci. 2009;22:4;368-372
2. Cross CE, Halliwell B, Borish ET, Pryor WA, Ames BN, Saul RL et al. Oxygen radicals and human disease. Ann Intern Med. 1987;107(4):526-45.
3. Ho YS, Magnenat JL, Gargano M, Cao J. The nature of antioxidant defense mechanisms: a lesson from transgenic studies. Environ Health Perspect. 1998;106:5:1219-28.
4. Mohsenzadegan M, Mirshafiey A. The immunopathogenic role of reactive oxygen species in Alzheimer disease. . Iran J Allergy Asthma Immunol. 2012;11(3):203-16.
5. Sorce S, Krause KH. NOX enzymes in the central nervous system: from signaling to disease. Antioxid Redox Signal. 2009;11(10):2481-504.
6. Gümüştaş K, Atukeren P. Oksidatif ve nitrozatif stresin psikiyatrik bozukluklar ile ilişkisi. İÜCerrahpaşa Tıp Fakültesi Sürekli Tıp Eğitimi Etkinlikleri. Türkiye'de sık karşılaşılan psikiyatrik hastalıklar. Sempozyum Dizisi 2008;62;329-340.
7. Yoshida Y, Umeno A, Shichiri M. Lipid peroxidation biomarkers for evaluating oxidative stress and assessing antioxidant capacity in vivo. J Clin Biochem Nutr. 2013;52(1):9-16.
8. Vetrani C, Costabile G, Di Marino L, Rivellese AA. Nutrition and oxidative stress: a systematic review of human studies. nt J Food Sci Nutr. 2012.
9. D. Harman. Aging—A theory based on free-radical and radiation-chemistry J. Gerontol.1956;11:298–300
10. Valko M, Leibfritz D, Moncol J, Cronin MT, Mazur M, Telser J. Free radicals and antioxidants in normal physiological functions and human disease. Int J Biochem Cell Biol. 2007;39(1):44-84.
11. Michel F, Bonnefont-Rousselot D, Mas E, Drai J, Thérond P. Biomarkers of lipid peroxidation: analytical aspects. Ann Biol Clin (Paris). 2008;66(6):605-20.
12. Blumberg J. Use of Biomarkers of Oxidative Stress in Research Studies. American Society for Nutritional Sciences. J. Nutr. 2004;134: 3188S–3189S,