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Contents

Oxidative stress, aging and antioxidants

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Abstract

The term oxidative stress is defined as a direct consequence of the increased production of the so-called free radicals accompanied with the decreased activity of the antioxidant defense system against them. The “free radicals theory of aging” was firstly proposed by Denham Harman in 1950. According to this concept, free radicals attack the susceptible cell structures continuously over the life, thus impairing their physiological functions. Moreover, it was suggested that the increased oxidative stress plays a crucial role in the pathogenesis of various chronic diseases associated with aging, such as cancer, neurodegenerative diseases, atherosclerosis, etc. Later, in 1970, Harman expanded the previous concept introducing the role of the endogenously generated reactive species in mitochondria in the oxidative stress phenomena. The findings that the mitochondrial DNA damage in mammals increases with age further supported the mitochondrial theory of aging. Therefore, it was concluded that the intrinsic generation of free radicals is an inevitable consequence of the consumption of oxygen and ATP production by mitochondria. Later on, Halliwell and Gutteridge renamed this theory in “oxidative stress theory of aging”, suggesting that aging and age-related diseases are not only caused by free radicals, but also by other reactive oxygen species. According to their theory, organisms age because of an accumulation of oxidative damage to the critical cellular components. For example, the DNA is continuously exposed to the oxidative modification. Consequently, reactive species may form a variety of DNA adducts via direct attacks or indirectly by initiating autocatalytic lipid peroxidation. The situation is further complicated knowing that the moderate levels of radicals are not only beneficial, but also essential, for exerting their functions in signaling pathways, inflammation, or immune response to pathogens. There are various enzyme systems whose functions are to remove and/ or repair DNA modifications in order for cells to maintain the necessary level of oxidative stress. Moreover, the oxidative damage can be worsened by a decreased efficiency of antioxidant defense mechanisms. The first line of endogenous defenses against reactive oxygen species includes antioxidant enzymes. The most significant ones are glutathione peroxidase (GPX), catalase (CAT), and superoxide dismutase (SOD). Genetic factors may affect about 25% of the variation in human life span. Among the antioxidant enzymes, the biggest role in longevity can be attributed to SOD2 and GPX genes. Up todate, there are more than 80 publications supporting the role of genetic variations in the regulation of the response to the oxidative stress. Finnaly, nowadays, in addition to the natural defense systems, it is generally believed that dietary antioxidants are able to increase the antioxidant protection, thus improving health and increasing cellular resistance to stress. While, there is growing evidence that changes in lifestyle (such as, moderate exercise, increased intake of dietary antioxidants, and

reduced calorie intake) could improve health and increase longevity, the role of the synthetic antioxidant supplements in controlling optimal levels of oxidative stress remains still controversial.

Keywords
Aging; Antioxidant enzymes; Free radicals; Oxidative stress.