Oxidants and Antioxidants in Medical Science, 2022
VOL 11, NO. 4, PAGE 01-02

COMMENTARY

Open Access

Note on Oxidaive Stress Related Diseases

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ARTICLE HISTORY
Received: 07-Apr-2022, Manuscript No. EJMOAMS-22- 59920;
Editor assigned: 11-Apr-2022, PreQC No. EJMOAMS-22- 59920 (PQ);
Reviewed: 25-Apr-2022, QC No. EJMOAMS-22-59920;
Revised: 30-Apr-2022, Manuscript No. EJMOAMS-22- 59920 (R);
Published: 09-May-2022.

Description

Oxidative stress has been described as dangerous because oxygen-free radicals attack biological molecules such as lipids, proteins, and DNA. However, oxidative stress also plays a key role in adapting to physiologic conditions and regulating intracellular signal transduction.

The role of oxidative stress has been implicated in many conditions, including atherosclerosis, inflammatory conditions, certain cancers, and the aging process. In many cases, this follows the recognition of increased amounts of free radical damage products, especially the markers of lipid peroxidation, in body fluids. It is important to remember, however, that lipid peroxidation is the inevitable correlation of cell death from any cause. In many cases peroxidation is a secondary factor, and this does not directly indicate the important role of oxidative stress in this disease. If the primary role of oxidative stress in a particular area is to be maintained, there must be a reasonable way in which free radical production may occur or a decrease in antioxidant defences.

In addition, evidence of oxidative stress should be seen before the onset of tissue damage and the addition of an antioxidant state early should prevent or significantly reduce tissue damage. Atherossteosis can be considered as an example of a process that has evidence of the role of oxidative stress. Hypercholesterolemia is internationally recognized as a major risk factor for atherosclerosis. However, in any concentration of plasma cholesterol, there is still a significant difference in the occurrence of cardiovascular events. One of the major advances in the study of atherogenesis has been the recognition that oxidative modification of low-density lipoprotein may be a significant step in the formation of atherosclerotic plaque.

The formation of foam cells from macrophages found in monocyte at the beginning of atherosclerotic lesions is not caused by native Low-density lipoprotein but only after the conversion of low-density lipoprotein by various chemical reactions such as oxidation. LDL oxidation is a process initiated and distributed by free radicals or one of several enzymes, and is believed to occur mainly in the arterial wall in a small area where antioxidants may be depleted. A number of mechanisms may be involved, including the production of ferrous metal ions for hydroxyl radicals, the production of active oxygen species by enzymes such as myeloperoxidase and lipoxygenase, and the direct conversion of active nitrogen species. Because oxidation of Low-density lipoprotein is primarily a free radical mediated process inhibited by antioxidants, depletion of antioxidants may be a risk factor for coronary heart disease.

In immunocytochemical studies, antibodies against oxidized Low-density lipoprotein contaminate atherosclerotic lesions but not normal arterial tissue. Low-density lipoprotein extracted from animal and human wounds has been shown to be oxidized and is rapidly absorbed by macrophage scavenger receptors. In young survivors of myocardial infarction, a correlation between increased Low-density lipoprotein exposure to oxidation and the degree of coronary atherosulinosis was indicated, while the presence of ceroid, a product of lipid peroxidation, was shown in advanced atherosclerotic plaques.

Controlling and preventing oxidative stress

- Berries
- Cherries
- Citrus fruits
- Prunes
- Dark leafy greens
- Broccoli
- Carrots
- Tomatoes

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Vitamin E (i.e., α-tocopherol) and Vitamin C (i.e., ascorbic acid) are antioxidants thought to have a protective effect by reducing or preventing oxidative damage.

Conclusion

There is overwhelming evidence that oxidative stress occurs in cells due to normal body processes and environmental interactions, and that complex web defense systems plays a key role in protecting against oxidative damage.