Editorial

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Antioxidant

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Editorial

Antioxidants are compounds that, when present in trace quantities, prevent or slow the oxidation of a substrate. Endogenous antioxidant defences include both non-enzymatic (e.g., uric acid, glutathione, bilirubin, thiols, albumin, and nutritional factors such as vitamins and phenols) and enzymatic (e.g., Superoxide Dismutases, Glutathione Peroxidases GSHPx, and catalase). In a healthy person, endogenous antioxidant defences complement reactive oxygen species activity, except for the 1% regular leak described earlier. Food is the most essential source of antioxidants, with all of them belonging to the phenol band.

Nutritional antioxidants work in a variety of ways and in various compartments, but they are mostly free radical scavengers, meaning they specifically neutralise free radicals, they lower peroxide levels and help to restore oxidised membranes. They reduce reactive oxygen species production by quenching iron, and short-chain free fatty acids and cholesteryl esters neutralise reactive oxygen species through lipid metabolism. Antioxidant plasma levels (micronutrients, enzymes, and other antioxidants) may be used to estimate the body's antioxidant defence, bearing in mind that the circulating compartment only expresses the movement between organs and tissues. Since tissue biopsies are necessary, the tissue levels of different antioxidants are restricted to study protocols.

In whole blood and plasma, vitamin C is the most effective water-soluble antioxidant and serves as the first line of protection against free radicals. It is an active lipid peroxidation inhibitor that also regenerates vitamin E in lipoproteins and membranes. Plasma ascorbic acid and isoprostanes have been shown to have a clear inverse relationship. Isoprostanes are a group of prostaglandin isomers that, unlike classic prostaglandins, are generated by a free radical-catalyzed mechanism rather than an enzymatic action of the prostaglandin-H-synthase from Arachidonic Acid. As a result, isoprostanes are an outstanding biomarker of lipid peroxidation for ageing studies and offer an ideal estimation of oxidative damage to cellular lipids. In animal models it is found that chronic vitamin C therapy would reduce elevated levels of isoprostanes. The mixture of Ascorbic Acid and Tocopherol is especially effective at preventing oxidation. Vitamin C prevents tocopheroxyl radical-mediated proliferation by quickly reducing tocopheroxyl radicals in membranes and LDL to recycle tocopherol. Vitamin E is present in cell membranes and circulating lipoproteins as a lipid-soluble vitamin. It works directly with a variety of oxygen radicals to protect against oxidative damage. Vitamin C facilitates regeneration, which helps its antioxidant function. Vitamin E is believed to play a part in atherosclerosis prevention by inhibiting the oxidative alteration of LDLs. In animals low in vitamin E, the formation of isoprostanes increases drastically. Furthermore, vitamin E supplementation has been shown to prevent the development of isoprostanes in both humans and animal models. Tocopherol is the most studied and quantitatively the most important source of vitamin E in humans. Tocopherol, on the other hand, despite being the most abundant source of vitamin E in the American diet, has gained less recognition. A-tocopherol is a somewhat less powerful antioxidant in terms of electron-donating propensity than tocopherol, but it is superior in detoxifying electrophiles such as reactive nitrogen oxide organisms. Tocopherol, on the other hand, has received less attention despite being the most abundant vitamin E source in the American diet. In terms of electron-donating propensity, tocopherol is a somewhat less effective antioxidant than tocopherol, but it is better at detoxifying electrophiles including reactive nitrogen oxide species.

Carotenoids are antioxidants that are lipid soluble. Isoprostane levels are adversely associated with carotenoids in the blood. Carotenoids are inversely linked to inflammation, atherosclerosis, cardiovascular disease, sarcopenia, and death, while being positively related to physical health. Carotenoids supplementation has been shown to improve antioxidant status and reduce lipid peroxidation. The most well-known and researched carotenoid is carotene, a powerful antioxidant capable of rapidly quenching singlet oxygen. All of the carotenoids, including carotene, cryptoxanthin, lycopene, and lutein/zeaxanthin, have been linked to inflammation. Recently, a correlation was discovered between low carotene levels in the blood and an increased risk of death.

Vitamins C, E, and carotenoids have been shown to work together to prevent lipid peroxidation. Higher antioxidant levels in the blood are linked to increased strength and physical activity, meaning that oxidative damage can play a role in the onset of the disabling phase.

Mammalian hormone, melatonin that is primarily produced in the pineal gland and is derived from serotonin. Melatonin has been shown to lead to the reduction of oxidative damage in both the lipid and aqueous environments of the cell, in addition to its well-known function in controlling the circadian rhythm. Melatonin acts as an antioxidant by growing the expression and function of glutathione peroxidase, superoxide dismutase, and NO synthetase. Melatonin levels are particularly high in mitochondria and the cell nucleus, where large oxidation reactions take place.

The relationship between oxidant and reducing forces is fragile. As a result of their physical effects, trace elements with antioxidant properties, such as copper and selenium, can become highly pro-oxidant both in vivo and in vitro this is also true of vitamins A, C, and E, which can turn pro-oxidant in some circumstances.

In therapeutic settings, vitamin E can also serve as a pro-oxidant in isolated lipoprotein suspensions, such as parenteral nutrition solutions. On cultured vascular cells exposed to parenteral nutrition containing different sources and amounts of selenium, the pro-oxidant effects of selenium were studied. Nakamura and colleagues proposed in a recent study that vitamin C could be important in preventing the pro-oxidant role of vitamin E in LDL oxidation.