

## Editorial Commentary on Oxidative stress

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### Editorial Commentary

Oxidative stress is the imbalance between the system damage caused by reactive oxygen species and the damage caused by the detoxifying intermediates of the biological system. When highly active molecules such as Reactive Oxygen Species (ROS) and reactive nitrogen species (RNS) are overexpressed capacity of the body, it will lead to an imbalance between the oxidation system and the antioxidant system, thereby damaging all components of the cell, including proteins, lipids, and DNA. For example, oxidative stress from oxidative metabolism causes base damage, as well as strand breaks in DNA. The relationship between oxidative stress and disease in humans has also been deeply investigated, such as atherosclerosis, Parkinson's disease, aging, cancer, respiratory muscle dysfunction, chronic obstructive pulmonary disease, diabetes, rheumatoid arthritis, osteoporosis, and so on. But free radicals are highly active and easily contact with other substances, making it difficult to measure their levels directly.

DROM and biological antioxidant potential (BAP) are used to evaluate the overall level of oxidative stress by measuring the total active oxygen metabolites and total antioxidant capacity in peripheral blood, respectively. Compared with detection of some free radical oxidative metabolites alone, DROM and BAP can evaluate the overall oxidative stress level more comprehensively. Hyperthyroidism is a common clinical syndrome characterized by thyroid production and excessive secretion of free T3 or T4. Thyroid hormones, as the main hormones in the body that control metabolisms and respiratory rate, are associated with oxidative stress damage not only in their enhancement of metabolic, but also

in their effects on antioxidant systems. High amount of thyroxine (TH) is produced in who is suffering with hyperthyroidism. Thyroxine accelerates energy metabolism by promoting intestinal glucose absorption and accelerating glucose oxidation and utilization, resulting in impaired glucose tolerance or aggravated diabetes. TH also accelerates the oxidative decomposition of lipids and proteins, which is the main cause of weight loss in patients with hyperthyroidism.

There is little and controversial data on oxidant stress and antioxidant capacity in hyperthyroidism. Erdamar et al. showed that the serum levels of malondialdehyde (MDA), nitrite, vitamin E and myeloperoxidase (MPO) activity were increased in patients with hypothyroidism, and the activity of SOD was the highest in patients with hyperthyroidism. However, there were no significant differences between the hyperthyroid patients and controls. By contrast, some other studies have shown that hyperthyroidism was characterized by increased levels of free radicals and peroxides, but decreased levels of antioxidant enzymes. However, no study has examined the relationship among DROM, BAP values and hyperthyroidism concurrently. The further research is going on to compare the differences of DROM, BAP and SOD levels between hyperthyroidism patients and normal controls, correlation among the three indicators levels, disease severity and metabolites of lipid and glucose in hyperthyroidism patients, to explore the role of three oxidative stress indicators in the evaluation of disease severity in hyperthyroidism patients. The originality of our study is the first time that we have investigated two comprehensive parameters (DROM, BAP) and the traditional antioxidant parameter (SOD) in patients with hyperthyroidism.

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