Journal of Clinical Toxicology
ISSN: 2161-0495
OPEN ACCESS Freely available online
Mini-Review

Metabolic Diseases and Oxidative Stress

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ABSTRACT

Metabolic syndrome is a public health problem that has been rapidly increasing from the last century. This condition is a cluster of pathophysiological malformation which includes cardiovascular disease, obesity, diabetes, hypertension, neurodegenerative diseases, and insulin resistance and obesity is a major modifiable causing factor for metabolic disease. The development and pathogenesis of metabolic syndromes is an imbalance between reactive oxygen species (ROS) and free radicals formation. Several clinical studies have reported that they can be triggered by the reactive oxygen species, genetic, environmental, lifestyle alterations, such as lack of physical inactivity, unhealthy dietary habits, alcohol consumption. Oxygen is essential for the living system at the same time oxygen may also be dangerous to the same biological system. ROS can injure cellular macromolecules leading to apoptosis and necrosis. ROS plays a vital role in physiological processes and a favorable role in the immune system. Low levels of ROS produced by mitochondrial respiration may continue oxidative damage to several metabolic syndromes. This review discusses the effect of oxidative stress on metabolic disease.

Keywords: Oxidative stress; Metabolic diseases; Reactive oxygen species

INTRODUCTION

The prevalence of metabolic syndrome is a combination of medical disorders leading to various complications that when occurring together increase the risk of developing obesity and diabetes including cardiovascular disease stroke, vascular inflammation, atherosclerosis, renal, liver, and heart disease. Abnormal glucose tolerance and hypertension combined with genetic susceptibility and metabolic syndrome are related to systemic complications that affect biological organs which induce hepatic steatosis, chronic obstructive pulmonary disease, malignant tumor, and degenerative joint disease. The impact of metabolic syndrome is considered an increase in morbidity and mortality as patients have a shortened lifespan compared with the general population this review focuses to present an update of the evidence supporting the role of metabolic syndromes oxidative stress and [1,2]. The World Health Organization (WHO) first developed its definition in 1998 for the syndrome and chose to call it the metabolic syndrome [3].

Oxidative stress is a well-recognized mechanism that has been rapidly evolving in many pathological conditions it occurs when there is an increase in the concentration of ROS or reduced activity of antioxidant defenses. Oxidative stress paves the way to the development of metabolic syndromes where treatments include reducing ROS production, reverse metabolic alteration such as hyperlipidemia, and improvement of insulin sensitivity and ROS induces cumulative oxidative damage eventually leading to cell death protein, membrane damage, and cellular dysfunction and lipid peroxidation. This review focuses to present an update of the evidence supporting the role of oxidative stress and metabolic syndromes [4].

Oxidative stress

Oxidative stress plays a vital role in metabolic syndromes it appears when there is an imbalance between the ROS and reactive nitrogen species [RNS] the highly reactive species is formed enzymatically in the cells. ROS is a well-recognized mechanism that plays an important role in homeostasis processes involving metabolism. Free radicals are defined as molecules that contain one or more unpaired electrons in molecular orbitals Free radical molecules are highly reactive chemical species in an increase, in interest on the oxygen-free radicals and collectively known as ROS and RNS. In both

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Received: February 16, 2021; **Accepted:** February 28, 2021; **Published:** March 10, 2021

Citation: Saraswathy R (2021) Metabolic Diseases and Oxidative Stress. J Clin Toxicol. 11(3):474.

experimental and clinical medicine, they are introduced by irradiation of UV, gamma, and X rays, they act as a pollutant in the atmosphere and metal-catalyzed reactive products [5].

High ROS are generated as an integrated process of normal cellular functions like phagocytosis, arachidonic acid metabolism, ovulation, fertilization during the pathological condition and their production multiplies several folds. During the recovery phase from other pathological noxious stimuli, the release of oxygen free radicals is reported and there are different pathways of ROS genesis. Oxygen biradical in nature accepts unpaired electrons to form a series of particularly reduced species known as ROS which include superoxide, hydrogen peroxide, etc. The major superoxide in the mitochondrial electron transport chain is important to source Adenosine triphosphate (ATP) in the cell and is a necessary life. The oxygen-free radical superoxide is formed prematurely during energy transduction which is the pathophysiology of various diseases [5,6]. The lipid hydroperoxides and nitric oxide are less reactive while limited enzymes like xanthine oxidate, tryptophan dioxygenase directly produce superoxide free radical. Some enzymes catalyze to produce hydrogen peroxide likely monoamine oxidase and L amino acid oxidase. The peroxynitrite, a high oxide is formed by the reaction of the nitric oxide free radical and superoxide radical [6].

Mitochondria, the main intracellular source of superoxide anion, involved in the aging process increased the target of free radical attack. ROS are produced during mitochondrial respiration under physiological conditions. Progressive oxidative injury to mitochondrial DNA may induce DNA strand breaks and progressive accumulation of oxidant damage induced more mitochondrial DNA mutations including proteins and lipid damages. Accumulation of these mtDNA changes may lead to damage of the respiratory chain complexes, leading to a vicious cycle, this toxic reaction is an increase in mitochondrial ROS development. This chain reaction has been elaborated in the oxidative damage during the aging process that causes growth reduction in the cellular functions as an outcome of a deficient supply of energy ROS plays a major role in biological systems [5].

ROS and RNS have a dual role either in a harmful or beneficial way to the living system. ROS helps in physiological processes and immune system by activating T lymphocytes and interleukin- production and also a beneficial role in the mediating neutrophils and macrophages phagocytosis, participate in Oxygen homeostasis, secondary messenger system, and signal transduction [5].

Oxidative stress and obesity

Obesity is the most common metabolic disease and a public health problem worldwide. Obesity is characterized as an excess of body weight that results in excessive accumulation of fat in adipose tissue. The impacts of obesity are considered as an increase in morbidity and mortality and this condition leads to the pathogenesis of several diseases like type 2 diabetes mellitus, cancer, hypertension, fatty liver diseases, and cardiovascular disease. Increased level of ROS, which plays an important role in the development of obesity oxidative stress, is harmful to both antioxidant protections. Recently it is found that obesity is similar to low grade chronic systematic inflammation in adipose tissue this condition regulates by activation of the innate immune system in adipose tissue which stimulates pro-inflammatory status and oxidative stress (OS), activates a systematic acute phase response.

Adipose tissue is endocrine organ storage for energy hemostasis this tissue generally comprises of adipocytes and some other cells like fibroblasts, fibroblastic preadipocytes endothelial, and immune cells. Secreting hormones and cytokines implement endocrine and autocrine action throughout the body. In the physiological and pathological condition ROS, give rise to OS and also irregular production of adipokines [6]. At first major adipose tissue has been recognized as the origin of pro-inflammatory cytokines involving tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1β, and IL-6. TNF-α is a critical cytokine that impacts the inflammatory response, the immune system, adipose cell apoptosis, lipid metabolism, rise in hepatic lipogenesis, insulin signaling, and produce OS[6].

TNF-α also elevates the connection of electron to produce superoxide anions. IL-6, classified by a wide variety of cells like adipocytes, endothelial cells β-pancreatic cells macrophages, and monocytes, control energy homeostasis and inflammation, impacting the change from acute to chronic inflammatory diseases like obesity and insulin resistance by producing the union of pro-inflammatory cytokines and negatively balancing inflammatory targets. In humans rise in serum IL-6 levels have been linked with a raised change of impaired glucose tolerance, diabetes mellitus, high blood pressure, and mainly obesity IL-6 can also subdue lipoprotein lipase activity, control appetite, and hypothalamic level.

Oxidative stress is lowered by maintaining a healthy lifestyle, having a balanced diet rich in antioxidative and physically active. Unfortunately, this is not so effective for obese having less intake of dietary antioxidants and low serum vitamin levels. Chronic oxidative stress in obesity has a progressive effect that satisfies the occurrence of end-organ damage. This incident is mostly studied in the cardiovascular system and liver in which chronic oxidative stress play a condemnatory role in the progress of atherosclerosis and nonalcoholic hepatic steatosis.

Diabetics Mellitus type 2 is the most common metabolic disorder where the oxidative stress, genes, and environmental risk factors contribute to the disease pathogenesis. T2DM is a major health problem that has been rapidly increasing from the last century. T2DM is mainly characterized by an increased level of insulin secretion in the body the impacts of T2DM are considered as an increase in morbidity and mortality The diabetic macro-vascular complication is associated with peripheral vascular, Cerebro vascular-disease, and heart disease. Excessive ROS development leads to microvascular complications includes nephropathy, neuropathy, and retinopathy. It mainly affects both macro-vascular and micro-vascular conditions. ROS and RNS have a dual role either in a functional and dysfunctional in the cellular system especially in tissues that aggravate diabetes such as muscle, adipose, and pancreatic cell.

Oxidative stress is a multifactorial process that results in the pathogenesis of T2DM overproduction of oxidative stress is harmful to dyslipidemia, insulin resistance, impaired glucose tolerance, and primarily caused hyperglycemia. Indication of oxidative stress in the pathogenesis of diabetes is not only by oxygen free radical but also due to antioxidation of glucose, impaired glutathione metabolism, lipid peroxide formation, and decreased ascorbic acid formation.

Diabetes causes an alteration in the activity of glutathione peroxidase and glutathione reductase they are found in the cell which metabolizes peroxide into water and glutathione disulfide converting into glutathione. The catalase manages hydrogen peroxide metabolism which in excess, causes major damage to lipids, RNA, and DNA. In catalyze deficiency, the beta-cell of the pancreas has an increasing amount of mitochondria, undergoes oxidative stress by excess ROS leading beta-cell dysfunction. Increases hydrogen peroxide production and downregulates enzymic catalase (CAT) gene expression. Superoxide, the primary ROS in metabolism, to molecular oxygen and peroxide, in other words, superoxide is dismutated into other compounds that are less toxic of superoxide dismutase(SODs). These antioxidant enzymes critically affect the various tissues and are affiliated in development problems in T2DM.

DISCUSSION AND CONCLUSION

In conclusion, the metabolic disease is a global prevalence and has been steadily increasing over the past 50 years and is today considered a major international health concern. Also, oxidative stress is one of the major risk factors of metabolic disease. Eventually, oxidative stress is also an early clinical condition of metabolic syndrome. The various mechanisms explaining the association between oxidative stress and metabolic syndrome are being identified. The association of oxidative stress appears conventional and self-reinforcing. This review highlights the reactive oxygen species and their effect on metabolic disease. Further study in-depth oxidative stress in metabolic syndrome will lead to the development of various further therapies in the future.

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