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Obesity And Oxidative Stress : A Comprehensive Review

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Abstract

Dyslipidemia is a term used to describe a general elevation of fats in the body's bloodstream. More specifically, it denotes an elevation of triglycerides and/or total cholesterol or low-density lipoprotein (LDL) cholesterol, and/or a decrease in high-density lipoprotein (HDL) cholesterol. and can accumulate as a result of an imbalance between lipoprotein uptake and secretion in the body's tissues. ***(Ofori, 2023 ; Bays et al.,2024)*** *These changes can occur as a result of genetic abnormalities, secondary factors such as diet, alcohol, drugs, and certain medical conditions, Plasma or serum is the only known physiological fluid that can be used to reliably detect dyslipidemia and abnormal lipid levels.* ***(Berberich & Hegele, 2022 ; Chen et al.,2023 ; Yanai et al.,2023).***

These pieces of evidence undoubtedly suggest the importance of managing dyslipidemia in preventing many diseased condition such as diabetes ***(Cheng, et al.,2025)****, atherosclerosis* ***(Laurindo et al.,2025****, cardiovascular diseases.* ***(Zakai et al.,2022 ; König et al.,2023 ; Krohn et al.,2023)****and alzahaimar* ***(Kuroda et al., 2025).***

Obesity now affects many millions of people worldwide, and the incidence of this condition continues to increase. Modern lifestyles, characterized by high-calorie foods and minimal physical activity, are conducive to the development of obesity. Simply put, people are eating more high-fat, energy-dense foods and are getting less physical activity than in years past ***(Balwan & Kour, 2021 ; S□rensen et al.,, 2022).***

Fat intake, particularly saturated and trans fat, is a major dietary contributor to dyslipidemia, and research suggests that this type of diet in obese individuals can lead to an atherogenic lipoprotein profile, impaired vascular function, and an increase in inflammatory and thrombotic markers ***(Kim et al.,, 2021 ; Magriplis et al.,2022).*** *In line with the recent ATP III guidelines, there is also interest in triglyceride levels and the prevalence of low HDL in relation to obesity and the metabolic syndrome* ***(Kosmas et al.,2023).*** *It is widely recognized that obesity greatly increases the risk of type II diabetes, and in turn, diabetes is associated with a major dyslipidemic state characterized by increased LDL, VLDL, cholesterol, and triglycerides* ***(Hariharan et al.,2022).***

Both obesity and dyslipidemia are states of increased oxidative stress, signified by increased production of free radicals, oxidation of lipids, and increased levels of lipid peroxidation products. Increased fatty acid delivery to adipose tissue in obesity results in increased free fatty acid efflux to the liver ***(Blagojevic et al.,2022).*** *Increased oxidative stress and peroxidation of these fatty acids can result in lipoperoxidation and subsequent production of oxidized, atherogenic LDL. Increased production of inflammatory adipocytokines and cytokines in obesity can up-regulate hepatic production of very-low-density lipoproteins (VLDL) and impair reverse cholesterol transport, also increasing atherogenic oxidized LDL* ***(Čolak & Pap, 2021 ; Blagojevic et al.,2022).*** *The net result is the production of atherogenic dyslipidemia and increased lipid peroxidation.*

Key Words: *Obesity, Oxidative Stress, Dyslipidemia, Cardiovascular diseases, Free Radical*

INTRODUCTION

Hyperlipidemia is a condition that incorporates various acquired and inherited diseases, it is a metabolic disorderwhich have been described as an elevated level of lipid mainly in TAG and LDL in the body **(Hill & Bordoni, 2020; Hill and Bordoni, 2021).** Abnormal lipid metabolism is the dominant causing of chronic diseases including obesity, diabetes, and cardiovascular **(Martin et al., 2014)** and cerebrovascular diseases **(Duan, Y et al., 2022).**

It is a well-known fact that obesity and a high-fat diet can lead to dyslipidemia. High intake of fat, especially saturated fats, will increase cholesterol and triglyceride production in the liver and therefore increase the secretion of very low- density lipoprotein (VLDL), which is the main carrier of triglycerides. Obesity also decreases the removal of LDL cholesterol in the body due to the small number of LDL receptors **(Stadler & Marsche, 2020 ; Henning, 2021 ; Lustig et al.,2022).**

Obesity is linked to a range of adverse health outcomes. The basic cause of obesity is the imbalance between energy intake and energy expenditure, where accumulation of fat tissue, especially the

accumulation of visceral fat tissue, is a possible primary cause of dyslipidemia and metabolic syndrome **(Althunibat, 2019).**

Excessive lipid accumulation in the liver triggers a cascade of events, including increased oxidative stress, inflammation, and mitochondrial dysfunction **(Rivas et al., 2023).** These mechanisms contribute to the progression of non-alcoholic fatty liver disease (NAFLD), a common manifestation of dyslipidemia-induced hepatic dysfunction. Diet with high-fat consumption causes imbalance of oxidative stress, change in lipid metabolism, and microbial dysbiosis, resulting hyperlipidaemia **(Yang, 2019).** ROS is the major oxidative factor, which could be dramatic increase in liver under high- fat diet (HFD) **(Banerjee, 2019).** Recent observational studies and animal models have also provided evidence to suggest that oxidative stress is intimately linked with both obesity and its associated changes to lipid metabolism. Studies measuring oxidative stress markers have consistently shown an increase in oxidative damage in the macromolecular components of adipose tissue in obese subjects **(Čolak & Pap, 2021).** This appears to be due to an increase in oxidative stress within the adipocytes themselves as well as increased infiltration of macrophages. The source of oxidative stress in obesity is thought to be a result of excess nutrients causing increased mitochondrial and endoplasmic reticulum stress **(Lemmer et al.,, 2021).** Results from animal models suggest that increased oxidative stress is causative in the development of insulin resistance and dyslipidemia **(Ghowsi et al.,2021).**

Dyslipidemia and obesity, individually and collectively attributable to insulin resistance, are basic risk factors for the development of impaired oxygen handling with raised formation of reactive oxygen species, primarily reduced oxygen and peroxynitrite. Factors related to obesity, particularly those centered within the visceral fat pad, contribute to an exaggerated production and reduced scavenging of reactive oxygen and oxygen species **(Čolak & Pap, 2021 ; Abot et al.,, 2022).**

With the advancing comprehension of the various syndromes linked to insulin resistance, type 2 diabetes, dyslipidemia, and hypertension, commonly known as the metabolic syndrome, there has been a growing interest in the potential impact that an altered intravascular environment can have on atherosclerosis **(Zhang et al.,2022 ; Zhou et al.,2023 ; Noothi et al.,, 2023).**This review will comprehensively explore the the deleterious impact of dyslipidemia and obesity on oxidative stress and the potential damage caused **.**

Obesity and Dyslipidemia

Obesity is a state of excess adiposity that implies the accumulation of triglycerides in adipose tissue, which results in adipocyte hypertrophy and hyperplasia. In this state, there is a positive energy balance in which energy intake exceeds energy expenditure, leading to increased fat stores **(Kojta et al.,, 2020 ; Ahmed et al.,, 2021 ; Koenen et al.,, 2021).** Overweight and obesity are labels for a range of weight that is greater than what is generally considered healthy for a given height.

High blood pressure can also be a result of obesity and occurs when the force of the blood is too high against the artery walls. High blood pressure can also lead to heart disease, kidney failure, and stroke. A type of diabetes can also develop as a result of obesity. Type 2 diabetes is when the blood has high levels of sugar due to either the pancreas not producing enough insulin or the body not reacting to the insulin. This can cause serious health complications. **(Oh & Cho, 2020 ; Matsushita et al.,2022 ; Saiz et al.,2022)**

Causes of Obesity

Obesity is influenced by a complex interplay of genetic, environmental, and behavioral factors. Environmental factors, such as the obesogenic food environment and sedentary lifestyle, have been extensively studied **(Mainieri et al., 2023).** Additionally, emerging research **(Sankararaman et al., 2023)** highlights the impact of the gut microbiome on obesity development. An increase in weight and fat mass, particularly central obesity, is associated with insulin resistance and low HDL-C levels. An increase in TG synthesis by the liver results in the secretion of VLDL, which is a precursor of LDL-C. High TG levels in the blood can also cause acute pancreatitis. Factors peculiar to high TG and low HDL-C levels are often associated with metabolic syndrome **(Ahmed et al., 2021).**

Endocrine disruptors in the form of several industrial pollutants have been shown to promote adiposity, as have some medications. Additionally, it is thought that genetic factors significantly influence susceptibility to obesity when exposed to an "obesigenic" environment. Unfortunately, those prone to obesity are at a higher risk due to the obesigenic environment present in the modern world. These factors, combined with the now widespread epidemic of obesity, have the potential to make obesity a problem of greater inequalities in the future. **(Gupta et al.,2020 ; Amato et al.,, 2021 ; Kumar et al.,2020 ; Egusquiza & Blumberg, 2020).** The risk factors for being overweight (BMI 25–29.9) or obese (BMI ≥30) can be

viewed in a variety of ways. Slightly simplifying the matter, it can be said that the primary cause of obesity is the prolonged imbalance between energy intake (calories consumed in food and drink) and energy expenditure (calories expended through basal metabolism, physical activity and thermogenesis) **(Jehan et al.,2020 ; Hall et al.,2022).** This is a direct result of changes in the global environment that have increased the availability of high energy food (often high in fat and sugars) and reduced the necessity for physical activity. This has been further compounded in genetically predisposed individuals who have been targeted by such an environment. **(Godde et al.,2021 ; Ben Hassen & El Bilali, 2022).**

Focusing on the health consequences of obesity, it has gained major importance in the current decade due to its enormous rate at which its prevalence is increasing and the substantial role it plays in the development of chronic disease. The emergence of chronic disease is of major concern due to its associated disability and economic burden **(Sarma et al.,2021 ; Malik & Hu, 2022).**

Oxidative Stress

Oxidative stress stems from an imbalance between the production of reactive oxygen species (ROS) and the ability of biological systems to readily detoxify the reactive intermediates or to repair the resulting damage. ROS can lead to increased lipid peroxidation, protein alteration, and DNA damage, and can cause a significant impact on the pathogenesis of several diseases **(Demirci-Cekic et al.,202 ; Jelic et al.,2021).**

Mechanisms: ROS are chemically reactive molecules containing oxygen. At the core of many radical and non- radical species is an oxygen molecule, which confers the high reactivity. Free radicals can be formed from direct univalent reductions of oxygen, and all known ROS are either radicals or compounds that are easily converted to radicals **(Tvrdá & Benko, 2020 ; Ponnampalam et al.,2022).** An example of a radical oxygen species is the superoxide anion (O2-), and hydrogen peroxide (H2O2) is an example of a non-radical species **(Costa et al.,2021).** H2O2 is highly stable and can diffuse through cell membranes. Various radicals can interact with transition metals to form more radical species capable of more damage, such as the hydroxyl radical **(Liu et al., 2021).**

Oxidative stress is often implicated as the cause of many diseases, including atherosclerosis and other coronary artery diseases, but it is rarely proven to be the cause of disease in vivo. However, oxidative stress induced experimentally after exposure of laboratory animals to H2O2 in water **(Khudair et al., 2001)** for different periods are claimed to be responsible for diabetes and altering insulin signaling **(Poznyak et al.,2020 ; Yaribeygi et al.,2020),** atherosclerosis **(Diab et al., 2005),** cardiovascular dysregulation **(Abdul-Katum and Khudair, 2008; (Ali and Khudair , 2019a), intestinal injury (Aziz and Khudair , 2022) and DNA damage (Ali and Khudair , 2019b).**

Sources of ROS

Increasing attention has been paid to endogenous sources of ROS, including the mitochondrial respiratory chain, xanthine oxidase, lipoxygenases, and monoamine oxidase but mainly to nicotinamide adenine dinucleotide phosphate (NADPH) oxidases (NOXs) **(Kaludercic N., 2016 ; Dan Dunn e al., 2015 ; Battelli et al., 2014 ; Raimondi et al., 2020 ; Kim et al., 2008 ; Selivanov et al., 2011).**

Approximately 1-2% of the oxygen consumed by healthy cells is converted to superoxide. superoxide production in the electron transport chain during oxidative phosphorylation in the inner mitochondrial membrane **(Dourmap et al.,2020).** However, mitochondrial superoxide production is increased under certain conditions such as disease states or with increased membrane potential and high O2 tension **(Mailloux, 2020).** This pathway is important in endothelial cell damage, where the mitochondrial superoxide can cause inactivation of NO and subsequent peroxynitrite formation, leading to impaired vasodilatory mechanisms **(Janaszak-Jasiecka et al.,2023).**NADPH oxidase is a group of membrane-bound enzymes found mainly in phagocytes, where it is a major generator of superoxide anions. The enzyme is made up of 5-6 subunits, its catalytic subunit is membrane-bound and is called Nox2. Another subunit called p22phox stabilizes it, and any other subunit is known to be dependent on the cell type **(Andrés et al.,2023; Amadio et al.,, 2024).**

NOX proteins are a family of enzymes whose distinguishing feature is the production of ROS following specific physiological stimuli. To date, seven membrane-crossing enzymes have been identified, namely, NOX1–5 and dual oxidases 1 and 2 (DUOX1 and DUOX2, respectively). Exploring respiratory bursts in neutrophile, the $gp91_{phox}$ catalytic subunit (NOX2) was identified as the first NOX enzyme. **(Gabig et al., 1978 ; Harper et al., 1985).** Later, other NOX complexes (NOX1,3–5 and DUOX1/2) were discovered and named according to their catalytic domain. **(Suh et al., 1999 ; Kikuchi et al., 2000 ; Banfi et al.,**

2001 ; De Deken et al., 2000) In fact, the term "NOX" denotes the transmembrane catalytic domain, but it is commonly used to refer to the entire multiprotein enzyme complex. **(Vermot et al., 2021)**
Uncoupling of NO synthase Nitric oxide is synthesized from the guanidino nitrogen of L-arginine in a reaction catalyzed by NO synthase **(Wu et al.,2021).** There are three isoforms of the enzyme, which are found in neuronal tissue (nNOS), endothelial cells (eNOS), and as an inducible form (iNOS). The reaction results in the stoichiometric production of NO and citrulline, except under certain conditions, e.g., airway epithelial cells, or in the presence of free radicals, where the NOS enzyme can lead to NO and superoxide anion formation **(Jomova et al.,2023 ; Jomova et al.,2024).** Xanthine oxidase Xanthine oxidase and xanthine dehydrogenase are forms of the same enzyme, xanthine oxidoreductase. The enzyme can be converted to xanthine oxidase in a reversible reaction under certain conditions such as ischemia and increased cytokine production. This conversion is due to enzyme cleavage from proteoglycan linkage **(Bortolotti et al.,, 2021).** Next, xanthine dehydrogenase is converted in an irreversible reaction to xanthine oxidase; this is due to specific modification of the enzyme, which increases its O2 and free radical dependency **(Kusano et al.,, 2023).** The last step is transcriptional upregulation of the enzyme in certain disease states. At present, it is clear that endothelial cells and epithelial cells, to a smaller extent, are the most important non-parenchymal source of xanthine oxidase **(Czyzynska-Cichon et al.,2024 ; Allameh et al.,2023).**

Role of oxidative stress in Disease Development

Disturbance in the balance between ROS production and cellular antioxidant defenses can lead to an excessive ROS buildup, causing oxidative stress. This stress damages essential cellular components, including lipids, proteins, and DNA, potentially culminating in oxidative cell death **(An et al,.2024).** The role of oxidative stress on the development of diet-induced diseases is of paramount importance. It has been broadly implicated through in vitro and in vivo research in the development of atherosclerosis, NAFLD, and the metabolic syndrome **(Masenga et al.,2023).** It has been shown that the key oxidative modifications to lipoproteins are the peroxidation of LDL and the acetylation of scavenger receptor types A and CD36. These processes lead to the increased uptake of oxidized LDL in macrophages and the subsequent foam cell formation, a hallmark of early atherosclerosis **(Orekhov et al.,2020).**
Oxidized lipids were also discovered to impair the function of the LDL receptor in vitro, potentially creating an even more atherogenic environment while promoting foam cell formation. In humans, carotid intima medial thickness was found to be associated with elevated levels of oxidized LDL, malondialdehyde, and conjugated dienes, both in patients with type 2 diabetes and in healthy middle-aged men without diabetes **(Schwedhelm et al.,2022 ; Zhang et al.,2022).** The most compelling evidence of oxidative stress being linked to the progression of lipid disorders and atherosclerosis stems from recent groundbreaking clinical trials, which have demonstrated the effectiveness of antioxidant therapy in reducing cardiovascular events **(Wang and Kang, 2020 ; Forman and Zhang, 2021).** Additionally, the production of oxidative stress in the form of the F2 isoprostane compounds has been shown to lead to the development of hepatocellular damage and the increased lipid peroxidation seen in non-alcoholic fatty liver disease **(Arroyave-Ospina et al., 2021; Smirne et al.,2022).** In the development of insulin resistance, a key factor in the pathogenesis of the metabolic syndrome, oxidative stress has been shown to cause the inhibition of the tyrosine kinase insulin pathway through increased expression of C-protein phosphatase 2A. This causes a decreased glucose transport in individuals' adipocytes and a downstream effect in impairing the action of insulin on the suppression of hepatic glucose production **(Yaribeygi et al.,2020 ; Singh et al., 2022).** Consequently, oxidative stress on nervous tissue may seriously damage the brain via several interacting mechanisms, including an increase in intracellular free Ca^{2+}, release of excitatory amino acids, and neurotoxicity **(Halliwell et qal., 1992 ; Bambrick et al., 2004).** Other important sources or modulators of oxidative stress, include reactive nitrogen species (RNS), including nitric oxide (NO) and peroxynitrite which can particularly be extremely reactive with proteins, lipids, nucleic acid and other molecules in further altering structure and/or functionalities leading to detrimental effects for the brain **(Lee et at., 2004 ; Smith et al., 1999 ; Turrens et al., 2003).** Cells with an accumulation of oxidized products such as aldehydes and isoprostanes, protein carbonyls, and base adducts from DNA oxidation can be seriously altered (10). Consequently, the considerable ROS formation increased by the electron transport system within the mitochondria under stressful conditions and in aging constitutes a risk for developing Alzheimer's disease (AD), when no efficient antioxidant system is available. Thus, mitochondria function as both the source and target of toxic ROS since

mitochondrial dysfunction and oxidative stress are important in aging and neurodegenerative diseases, particularly AD **(Clarke et al., 2015 ; Beal et al., 1998).**

Relationship between Obesity, and Oxidative Stress

Adipose tissue is now recognized to be an active endocrine organ, synthesizing various substances which have effects on lipid metabolism **(Richard et al., 2020).**Several adipocytokines are now known to have important roles in the pathophysiology of dyslipidemia. Leptin has effects on food intake and energy expenditure, but also has an important role in lipid metabolism. It acts on the central nervous system and also on peripheral tissues to increase fatty acid oxidation and inhibit triglyceride synthesis, and in doing so lowers triglyceride stores in adipose tissue and skeletal muscle **(Pereira et al.,2021 ; Obradovic et al.,2021).** It also has a paradoxical effect of increasing VLDL production and secretion. In states of increased leptin resistance, such as obesity, these effects on lipid metabolism are attenuated **(Stadler & Marsche, 2020).** Low levels of adiponectin, an anti-inflammatory and insulin sensitizing adipokine, are associated with obesity and insulin resistance. Adiponectin has been shown to increase beta- oxidation of fatty acids in liver and skeletal muscle, whilst decreasing hepatic glucose production and increasing insulin-induced suppression of de-novo lipogenesis in the liver **(Muratsu et al.,2022 ; Castela et al.,2023).** The relationship between obesity and dyslipidemia is well established. Obesity can result in increased levels of various lipoproteins, and these changes are strongly related to the degree of obesity, more so than the absolute body weight. Increased levels of triglycerides and decreased HDL cholesterol are common lipid alterations in obesity **(Stadler & Marsche, 2020 ; Vekic et al.,, 2023).** Abdominal obesity is more strongly associated with dyslipidemia than is lower body obesity. This pattern of lipid changes is similar to that seen in insulin resistance, and there is strong evidence that obesity is associated with insulin resistance, and that insulin resistance is a major causative factor in the dyslipidemia of obesity **(Zhu et al.,2022).**

A. Interactions and Associations

Although it is tempting to think that simply obesity leads to dyslipidemia, which in turn might cause increased oxidative stress, the issue appears to be more complex than this. Obesity can cause lipid peroxidation and increased oxidative stress in the absence of dyslipidemia **(Blagojevic et al.,2022).** In an animal model of obese dyslipidemic rats, there was increased oxidative damage to proteins, which was prevented by treatment aimed at preventing the dyslipidemia **(Khutami et al.,2022).** This suggests that it is not simply the lipid abnormality, rather the metabolism of lipids that leads to increased oxidative stress. Increased oxidative stress also leads to degradation of HDL cholesterol and impairs its reverse cholesterol transport function. Excess oxidized LDL in the vessel wall, if not cleared, can also induce macrophage foam cell formation and enhance atherosclerosis **(Poznyak et al.,2020 ; Ouyang et al.,, 2023).** Erythrocytes from patients with increased oxidative stress in the form of glycation or lipid peroxidation deposit more cholesterol ester to LDL in the plasma, which will lead to enhanced atherosclerosis. This process is known as cholesterol cholesteryl-ester exchange, and it increases the LDL cholesterol concentration in erythrocytes and the subsequent transfer to the vessel wall **(Duan et al.,, 2023 ; Tutino et al.,2024).**

The striking feature of atherogenic dyslipidemia is also a feature of insulin resistance and the metabolic syndrome, which suggests that oxidative stress may have a pathogenic role in the link between obesity, dyslipidemia, and oxidative stress **(Čolak & Pap, 2021).** ROS is the major oxidative factor, which could be dramatic increased in liver under high-fat diet (HFD) **(Banerjee, 2019).** The endogenous antioxidant molecules such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) play important roles in alleviating tissue damage caused by free radicals **(Samarghandian, 2013 ; Qin, et al., 2022).** Besides, great correlation between oxidative stress parameters and breast and intestinal cancer has been recorded **(Abd-AlHameed and Al-Ani, 2023; Mahdi et al., 2024).**

B. Shared Pathophysiological Mechanisms

Oxidative stress in obesity can increase the production of inflammatory markers and activation of pro-inflammatory transcription factors such as NF-κB and AP-1, which can increase the production of inflammatory genes. Increased oxidative stress can also impair insulin signaling and PPAR activity and increase the production of VLDL **(Čolak & Pap, 2021 ; Masenga et al.,2023).** All of these mechanisms are shared with the induction of atherogenic dyslipidemia in obesity and suggest that oxidative stress plays

a central role in the pathogenesis of cardiovascular atherosclerosis. In dyslipidemia, increased LDL-C and LDL-C/triglyceride ratio are significantly associated with increased oxidative stress and decreased antioxidant enzyme activity **(Patel & Kashfi, 2022 ; Mohammadshahi et al., 2023),** Oxidative modifications to LDL, such as lipid peroxidation and the presence of oxidized LDL, are key steps in early atherogenesis and the promotion of these processes increases CVD risk **(Gianazza et al.,2021).** Increased lipid peroxidation in dyslipidemia can also shift the balance between prostacyclin and thromboxane production to a pro-thrombotic state and can decrease NO availability, both of which are vasoactive mediators that have significant effects on atherosclerosis and plaque stability **(Zhao et al.,2021).**

C. Obesity and Inflammation

There is now a growing body of evidence to suggest that obesity is a state of chronic inflammation. This state is typified by high levels of circulating inflammatory markers such as CRP and a sustained increase in the production of inflammatory chemicals by adipose tissue **(Khanna et al.,, 2022).** Inflammatory pathways have been suggested to link obesity with insulin resistance **(Wu & Ballantyne, 2020).** Macrophages are thought to be recruited to adipose tissue and become activated by excess nutrients. This causes these cells to synthesize and release cytokines which can block insulin signaling and create a state of insulin resistance in nearby adipocytes **(Cai et al.,, 2022).** If macrophage infiltration into adipose tissue is inhibited in animal models, the development of obesity and insulin resistance can be retarded. Alternative activation of macrophages has also been shown to increase production of catecholamines, which can increase fat accumulation in visceral adipose tissue **(Lin et al.,2022 ; Li et al.,2023).** Inflammatory changes which typify adipose tissue in obesity have also been suggested to increase the production of very low density lipoproteins, which causes dyslipidemia **(Guria et al.,2023).**

Measurement Methods

Methods for assessing oxidative stress can be divided into in vivo and in vitro measurements. A common in vivo method for assessing oxidative stress is the measurement of F2 isoprostanes, which are formed from the random oxidation of tissue phospholipids by oxygen radicals **(Murphy et al.,2022).** Measurement of F2 isoprostanes in urine or plasma is thought to provide an integrative index of lipid peroxidation in vivo and is now considered the most reliable and validated method for estimating oxidative stress **(Morrow et al.,, 2005) (Graille et al.,2020 ; Granick et al.,2021).** In vitro measurements of oxidative stress, mainly lipid peroxidation, have been used to estimate the presence of oxidative stress within a system **(Demirci-Cekic et al.,2022).**

The measurement of individual oxidized molecules may provide information on specific pathways of oxidative damage and may be useful for identifying which antioxidants are most likely to be depleted in a particular disease state **(Murphy et al.,2022).** However, the site- specific nature of many oxidants and the rapid metabolism of many oxidized molecules means that detection methods for individual oxidized molecules may need to be tailored to each specific molecule **(Qian et al.,, 2021).** Methods of detecting antioxidants and antioxidant enzymes provide information on the overall redox status of a system. In general, in vivo measurements of oxidative stress correlate poorly with each other and with measures of tissue damage **(Menzel et al.,2021 ; Murphy et al.,2022).**

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