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Oxidative Stress and Associated Diseases: Toxicological and Pharmacological Issues

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Abstract

During the process of oxygen metabolism, REOs) like superoxide, hydrochloric acid, or hydroxyl radical are made and can cause oxidative damage when they combine with bio-molecules. Damage from oxidative stress to the inflammatory signalling cascade &chemoattractant production. Hydrogen peroxide, at low concentrations, can control cellular signalling and even encourage cell proliferation, but at higher quantities it can cause apoptosis and even necrosis. Oxidative stress, ageing, carcinogenesis, and other age-related and oxidative-related diseases (OSRDs) have all been linked to ROS, and There is more and more evidence that ROS are involved in the death and damage of cells. Antioxidants, on the other hand, are thought to dampen the immune response and boost the body's own cellular defences in the face of tissue damage. The study's authors sought to clarify the relationships between OSRDs & the many diseases previously related to them. The treatment of OSRD has been shown to be successful with a variety of medications, such as pentaerythritol tetranitrate, resveratrol, hypertension converting enzyme (ace) inhibitors, angiotensin receptor baddies, lovastatin, nebivolol, or carvedilol, as well as antioxidants that target mitochondria and drugs made from plants, are some of the drugs that are used to treat high blood pressure (singly or in combination).

Keywords:oxidative, stress, diseases, reactive

INTRODUCTION

When there is an overabundance of ROS in the system without the need for a corresponding drop in their levels, the condition is referred to as "oxidative stress" (OS). Oxidative stress causes chemical changes in biomolecules that can have an impact on their structural and functional. More free radicals are being made, and the antioxidant system is getting weaker, or even both may be the root of this stress. One of the ROS created during oxygen metabolism is the hydroxyl radical, which is also formed together with superoxide and hydrogen peroxide. However, when these radicals get into touch with biomolecules, oxidative damage may result. In the face of a pathogen assault,

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oxidative damage might be advantageous. Although ROS are generally detrimental to cell health, there are situations in which they can be beneficial. During normal cellular mitochondrial function, for instance, fewer ROS are generated and serve as the signalling molecules. The oxidant level and typical biological antioxidants need to be in equilibrium. Toxic oxidative stress can occur if the previously indicated equilibrium is disturbed (Fig. 1). This discord develops naturally with age (as an example), but it also contributes to the aetiology of some diseases and appears as a symptom of others. There are both enzyme-based and non-enzyme-based antioxidants, such as vitamin E, glutathione, and ascorbate (vitamin C), are naturally present in the human body and participate in this process. Ascorbic acid (AA) & reduced glutathione are two of the body's most significant natural antioxidants for protecting against ROS and keeping the oxidative balance in check (GSH). It appears that AA and GSH are interacting via ascorbate-glutathione cycle [2]. Before, it was recognised that mitochondrial respiration makes a gradient of protons and O2•, which might also act as just a signalling mechanism during oxidative stress or alkaline-induced cell death.

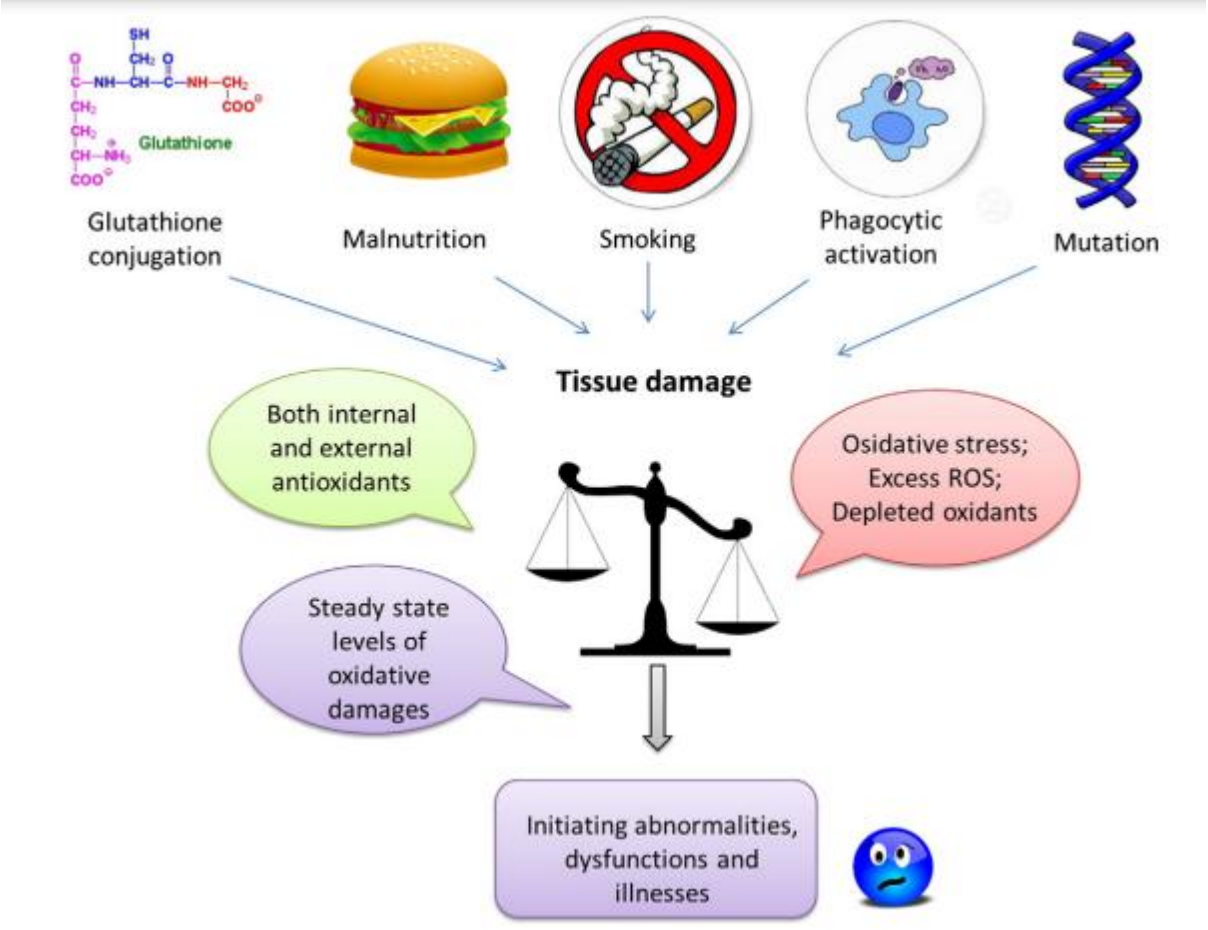

Fig. 1:Induction of oxidative stress & damage as a result; ROS stands for reactive oxygen species; AOX for antioxidants.

Chronic inflammation and the generation of chemoattractants both include oxidative stress as a

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prominent upstream link in their signalling pathways. Hydrogen peroxide is turned into the highly reactive hydroxyl radical when it comes in contact with transition metals. Most of the damage that oxidation does to proteins, lipids, carbs, and nucleic acids is caused by the hydroxyl radical. In addition to being a signalling molecule, hydroxyl radical can also cause inflammation by turning on NF-B, which is a key transcription factor (B-cell activation gene product nuclear factor kappa light-chain-enhancer). While H2O2 in small doses can modulate cell signalling & drive cell development, higher doses can cause apoptosis and even necrosis.

Oxidative stress related diseases (OSRDs)

In this part, we wanted to give some background information on a number of OSRDs that are related to the idea of oxidative and also the effects of oxidative on cells that can be fixed by antioxidants. Molecular manipulations of oxidative stress are connected with well-known diseases, as shown in Figure 2 and as follows:

Neurodegenerative diseases

All aerobic cells can be damaged by oxidative stress, but the mammalian brain is more vulnerable. The brain only has a small percentage (10%) of the antioxidants found in the liver, yet it uses 20% of the body's oxygen. Both iron and ascorbate are present in the human brain, with higher amounts of the former in some locations. The cumulative body of research up to this point [3] established that oxidative stress is especially bad for brain cells.

Pathological apoptosis has been linked to ageing and neurodegenerative diseases like Alzheimer's, Parkinson's, sclerosis, and amyotrophic lateral sclerosis (ALS). Neurodegenerative disease probably comes about because of a mix of environmental and genetic factors. In addition to redox biometals (Cu and Fe), oxidative stress and free radical generation play a part in redox processes. These metals have direct interactions with beta-peptides, the primary components of betaamyloid, one of Alzheimer's disease's first symptoms [4].

Around 16 million people around the world are living with Alzheimer's disease, the most prevalent neurological disorder worldwide. Protein aggregates in the form of external amyloid (A) plaques & intracellular tau tangles are hallmarks of this disease, which causes gradual neuronal degeneration. Malondialdehyde was found in higher concentrations in the brains of Alzheimer's patients & 4-hydroxynonenal in their cerebrospinal fluid (CSF) and brain tissue than healthy controls. Higher concentrations of protein carbonyl moieties have been found in the hippocampus, parietal, and frontal cortices, but not in cerebellum. AD samples have more hydroxylated guanine than samples from people the same age who don't have AD. More protein or lipid peroxidation is seen in the

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hippocampus and the cortex of transgenic animal models of Alzheimer's disease [5], corroborating results from human brains showing that this occurs before the formation of plaques and tangles.

Parkinson's vascular dementia is the second greatest common disease that causes nerve cells to die. It happens when protein-synuclein builds up inside the substantia nigra and changes the way dopaminergic neurons with in substantia nigra work. Parkinson's disease manifests itself in the ventral striatum of it's own sufferers, there are less polyunsaturated free fatty amino acid and more lipid peroxidation markers [6]. Oxidative damage to proteins in the form of nutrient carbonyls [7] can also be seen in the brains of people with Parkinson's disease compared to people without the disease, and there is some proof that reactive oxygen and nitrogen species in the brains of people with PD contribute to a nitration or nitrosylation from certain proteins [8]. In the PD brain, the amount of 8-hydroxydeoxyguanosine has gone up [9], and the prevalent mitochondrial DNA removals in the dopaminergic neurons that are still alive in the substantia nigra have also gone up. It is thought that oxidative stress caused these deletions [10].

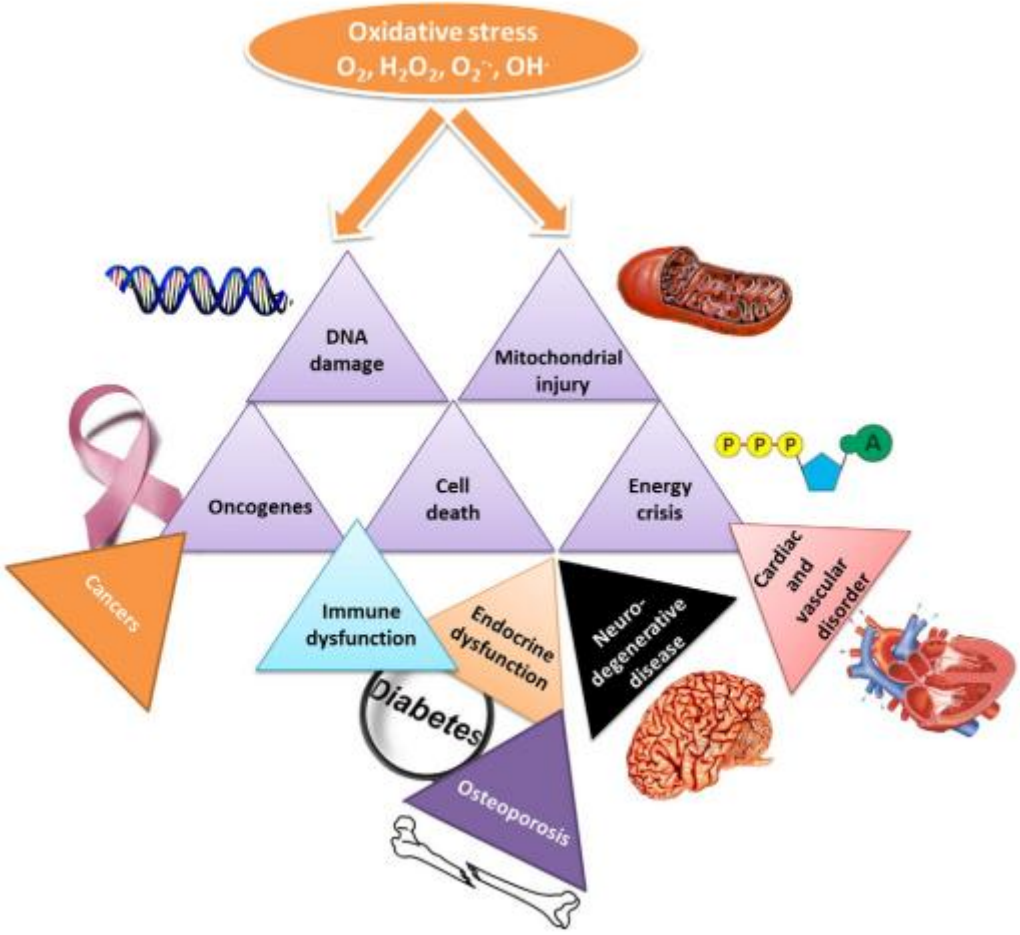

Fig. 2. A theory that simplifies the relationships between oxidative stress and subsequent cell death or dysfunctions is presented.

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Vascular diseases

Oxidative stress is central to cardiovascular disease. Blood pressure, high cholesterol, diabetes, or smoking all cause the arterial wall to have more ROS are all things that can make you more likely to get cardiovascular disease (Fig. 3).

Once superoxide (O2•) reacts to nitric oxide (NO), it makes peroxynitrite (ONOO), which may stop NO from being made. Most people think that heart diseases are caused by not having enough NO in the blood. Oxidative stress inside the blood vessels has been linked to atherogenesis. These include turning on redox-sensitive transcription factors, which start the interpretation of proinflammatory genes, but also causing oxidative stress, peroxidation, but also mitochondrial and nuclear DNA damage [11].

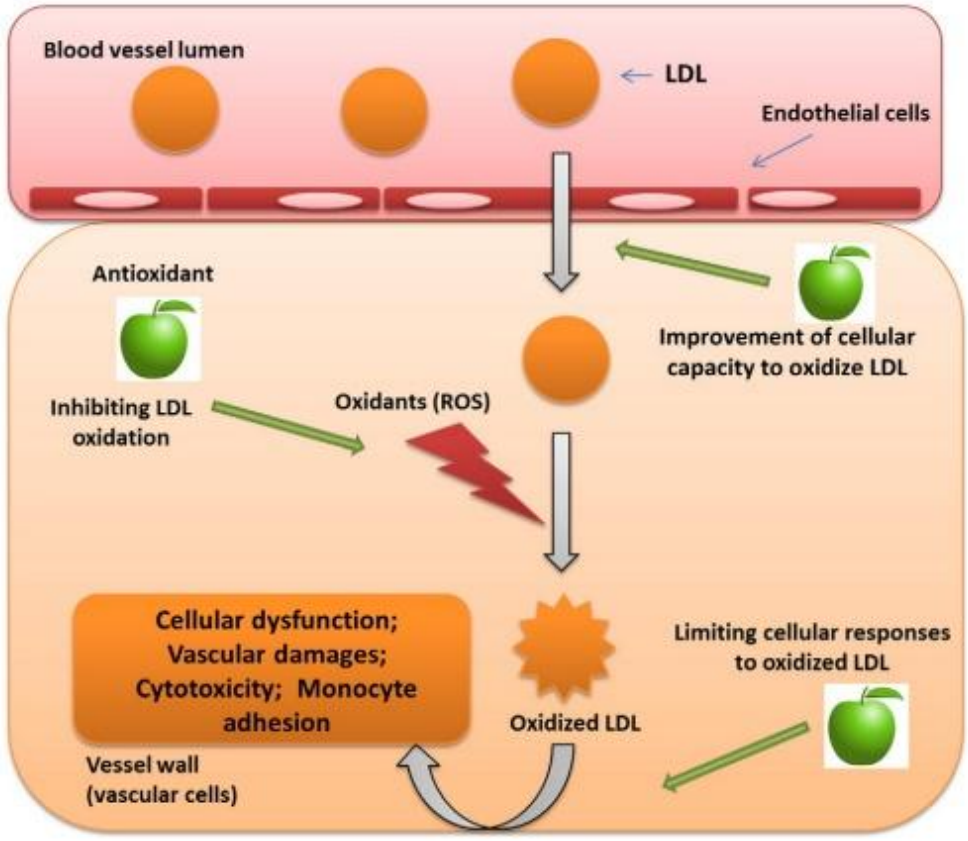

Fig. 3. Function of oxidative stress in atherosclerosis development and progression and antioxidants' interference

Cancer

Increased iron levels in the body increase the risk of numerous diseases, such as cancer [12]. Iron-mediated production of ROS leading to DNA & lipid harm appears to originate from a disruption of iron's normal physiological function of transporting oxygen to tissues. Researchers have found a

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link between a rise in ROS and a change in the structure of DNA and the start of cancer. When ROS are made more by ligand-independent transcriptional activation of the receptor tyrosine kinase, cancer can spread and tumours can grow faster. Oxidative phosphorylation (OPP) could indeed make the vascular endothelial growth component transcription factor hypoxia-inducible factor 1 more stable. This factor is important for angiogenesis. Even though antioxidants may provide some chemopreventive advantages, it is still not clear what role they play in cancer treatment, particularly during the initial stages of carcinogenesis. On the one hand, there really are anti-cancer drugs with antioxidant properties like curcumin, isoflavones, and resveratrol. These drugs fight cancer by changing epigenetic procedures like DNA demethylation, chromatin remodeling, as well as RNA interference. But there are anti-cancer drugs like piperlongumine that go straight for the antioxidant enzymes in cancer cells. Antioxidants have been shown in the literature to improve cell survival after separation from the extracellular matrix; however, they may have a dual role by either protecting DNA from oxidative damage or keeping tumours alive by promoting cell survival via metabolic rescue [13].

LITERATURE REVIEW

Shukla et al. (2011) [14], As far as we can tell, oxidative stress contributes to every form of neurodegenerative illness. Reactive oxygen species are one of the main things that cause oxidative stress (ROS), or mitochondria are one of the main places in the body where ROS are made. There is a lot of evidence in the tissues of people with neurodegenerative diseases that mitochondria have morphological, biochemical, but also molecular abnormalities. However, it is still not clear whether oxidative causes neurodegeneration or is just a symptom of it. Oxidative stress has been linked to a number of neurodegenerative diseases, such as Alzheimer's, Parkinson's, Huntington's, or amyotrophic lateral sclerosis. The last part of the study talks about how oxidative stress makes the neurodegenerative enzymatic cyclin-dependent kinase 5 make too much of itself (Cdk5).

Ma (2010) [15] Researchers looked at important transcription factors that control transcriptional reactions to oxidative stress. They focused on recent advances inside the signalling pathways and processes by which oxidative stress stimulates transcription factors, as well as the effects of this control on the advancement of disease as well as chemical toxicity. Oxidative stress turns on a lot of genes that either stop ROS from being harmful or are essential for detecting oxidants and getting rid of them. The amount of oxidative controls how certain transcription factors are expressed, how they look, how stable they are, where they are in the nucleus, and how well they bind to DNA.

Jomova et al. (2011) [16] Long-term and short-term arsenic dangers were discussed in relation to cancer, cardiovascular illness (hypertension and atherosclerosis), neurological disorders,

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gastrointestinal issues, diseases and degenerative diseases, effects upon reproductive health, skin abnormalities, and other difficulties. Antioxidant defences against exposure to arsenic are also discussed. Air, water, or soil all contain trace amounts of the metalloid element as. Sometimes inorganic arsenic can be harmful, but organic arsenic is usually preferable. Chemical oxidative stress: a compared of vitamin C (ascorbic), vitamin E (-tocopherol), and carvacrol with glutathione peroxidase and the antioxidative enzyme catalase and catalase.

Liu et al. (2009) [17] Researchers looked at the role of reactive oxygen compounds (ROS) throughout chronic Cd stress but also carcinogenesis. They also found direct evidence that free radicals are made in intact living creatures after an acute Cd overload. Electron spin resonance spectra show that In living organisms, Cd breaks down into superoxide radicals, hydrogen peroxide, or hydroxyl radicalsThus, redox-sensitive transcription factors are activated, and there are wide-ranging changes in ROS-related gene expression (such as NF-kB, AP-1, and Nrf2). Acute Cd poisoning is associated with oxidative stress, which is well-known. Acute Cd poisoning changes the expression of more genes than chronic Cd exposure does. Most likely, this is because of the development of adaptations, like metallothionein and glutathione, that reduce the oxidative stress caused by Cd.

Wells et al. (2009) [18] investigated the influence of oxidative stress on teratogenesis, neurodevelopmental problems, and the advancement of cancer. Long-term adverse effects may include teratogenesis, developmental delays, and even illness. Pathways that control the balance of ROS in embryos, Animal studies have shown that enzymes involved in bioactivating endogenous substrates or xenobiotics into oxygen radicals intermediates, as well as antioxidant properties enzymatic reactions that clean it up ROS and enzymes that fix oxidative DNA damage, can be altered to mitigate the negative effects of thalidomide, crystal meth, phenytoin, benzo[a]pyrene, and other chemicals during pregnancy. Some preventive therapies have been shown to make it more likely that bad developmental effects will happen. This shows how complicated development is and how careful researchers need to be when looking into therapeutic interventions in humans.

Roberts et al. (2009) [19] showed that damage to the brain caused by anaesthetics changes genes that control how oxidative stress affects development. Dopamine-producing neurons are particularly vulnerable to the degenerative effects of Parkinson's disease. Both inflammation and oxidative have been associated with the onset and development of the condition. When oxidative stress is present, dopamine neurons exhibit higher levels of the endogenous neurotoxic 3,4-dihydroxyphenylacetaldehyde. These discoveries have important implications for the search for novel indicators of the pathogenesis of Parkinson's disease & the creation of new treatments for the

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disorder. The aggressive phenotype of inflammatory breast carcinoma is linked to a proinflammatory microenvironment, which may be caused by oxidative and nitrite stress.

Patra et al. (2011) [20] Researchers looked into the ways that lead and cadmium cause oxidative damage, as well as how antioxidant supplements can be used to treat oxidative damage and pathotoxicity. Oxidative stress has been linked to the pathophysiology of pathotoxicity caused by lead and cadmium. Scientists have looked into the a variety of therapies which use antioxidants to reduce the damage to the body resulting from oxidation during or after exposure to such dangerous substances. People have thought that oxidative is at least partly to blame for how some diseases and poisons affect animals. When a cell comes into contact with xenobiotics, this makes more free radicals and reactive oxygen species than it can get rid of. This leads to oxidative stress and harms lipids, proteins, and DNA.

Galaris et al. (2008) [21] looked at how Fe homeostasis is controlled at the cellular but also system level, choosing to focus on how iron overload diseases develop and how to treat them. The authors look into the position of iron throughout ROS-induced toxic effect and, as a result, the molecular processes and physiological properties of ROS- but also iron-mediated signaling. Iron is a key part of many important biological and metabolic processes, like how red blood cells move oxygen around the body and how respiration turns oxygen into water. Iron's bioavailability is typically low, but pathological buildup of any metal in tissues boosts ROS generation or has largely deleterious effects connected to oxidative stress even when the metal itself has a poor bioavailability.

Mechanisms involved in creation of OSRD

Antioxidants play a crucial role in the prevention of chronic diseases by inhibiting the reactions of free radicals, which would otherwise transform polyunsaturated fatty acids into lipid peroxides, thereby amplifying the initial oxidative damage [22]. Arterial endothelial macrophages, for instance, take in oxidised low population lipoprotein to promote atherogenesis. As with other DNA oxidation products, 8-hydroxydeoxyguanosine is detected in the human urinary tract after oxidative stress exposure. To varying degrees, oxidative pathways contribute to age-related degeneration, the onset of cardiovascular disease, and cancer [23-25]. Diabetes is one of the numerous modern health problems thought to be related to oxidative stress [26]. Since a lack of b cells results in insulin insufficiency or insulin's inappropriate action in different organs, it stands to reason that antioxidant therapy, by preventing b cells from apoptosing, can maintain b cell function [27]. Cancer is another example where telomeres are maintained at a constant length by the telomerase enzyme, whereas in normal somatic cells, telomere length steadily decreases with each cell cycle. Remarkably, H2O2 can shorten telomeres, which in turn inhibits cell proliferation and differentiation. The opposite is

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true: moderate amounts of reactive oxygen species (ROS) can hinder cell growth, while low levels can stimulate cell division. Evidence suggests that free radicals, which can be produced as by-products of faulty metabolism, can become crucial parameters in the development of late complications or secondary disorders. Examples from the literature include the fact that both moms and infants who experience a natural birth have higher levels of oxidative stress than those who have a caesarean section [28]. RDS, bronchopulmonary dysplasia, newbornhemolytic illness, hypoxic-ischemic encephalopathy, and SIDS are just few of the neonatal conditions linked to oxidative stressors. Stress-induced premature senescence is another type of accelerated senescence that is not caused by telomeric signals. Oxidative stress is a key factor in this type of senescence.

Utilization of traditional medicinal plants for the prevention and treatment of OSRD

Since the Middle Ages, people have used aromatic and medicinal plants to treat a wide range of diseases, including some that only recently seemed to be linked to oxygen radicals (Fig. 5). Analyzing the results of a range of these investigations demonstrates that the bulk of the previously mentioned plants have an antioxidative effect [29]. There appears to be a strong association among the antioxidative stress capabilities of Iranian herbal remedies & the therapeutic or preventative effects of these plants against a wide range of disorders [30]. The accumulated and organised information on medicinal plants used in the prevention and treatment of OSRD based on their application in traditional medicine.

CONCLUSION

During oxidation, reactive oxygen species (ROS) like superoxide, hydrogen peroxide, and the hydroxyl radical are created. When they interact with biomolecules, they can cause severe oxidative damage. ROS are known to cause cell damage and death, which links them to a wide range of degenerative changes, such as tissue breakdown, cancer, ageing, and other OSRD (e.g. tissue degradation, carcinogenesis, ageing and other OSRD.) Critical factors include degenerative disorders, such as Alzheimer's and Parkinson's, as well as diabetes, cardiovascular disease, cancer, ageing, decreased bone, inflammatory bowel, and obesity that are all at least partly caused by high levels of free radicals that damage tissue. Hyperglycemia, metabolic imbalance, and oxidative stress may all affect how fast diabetic neuropathy gets worse. Antioxidants have been shown to help treat many diseases, which suggests that oxidative may be a cause of some of these diseases. Antioxidants can fight the free radical effects by stopping them from turning polyunsaturated fats into lipid peroxides. If this happened, the initial oxidation would be made worse. Some of the most important medicines for preventing or treating OSRD are mostly involved in the body's different oxidative pathways. Resveratrol, angiotensin-converting protease inhibitors and blockers,

angiotensin receptor blockers, furosemide, nebivolol, carvedilol, PETN, or antioxidants that target the mitochondria are among examples. MitoQ is indeed a quinone functional group that can only be found in mitochondria, in which it reduces the production of free radicals and oxidative damage to mitochondria without changing how well the mitochondria breathe. Ocular surface vascular disease (OSRD) can be treated or prevented with the use of antioxidant-rich plants. There are a small number of scholarly articles that discuss the synergistic or synergistic-like effects of plant-based medicines. One such example is the combination of R. canina, U. dioica, & T. vulgare extracts that have been subjected to a pulsed electromagnetic field and contain selenium, carotene, flavonoids, & urea.In addition, OSRD can also be treated with traditional treatments such oleogum resins and Magliasa.

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