Oxidative Stress

1-Oxidative stress in poultry production

By Oke, OE (Oke, O. E.) [1] , [2] ; Akosile, OA (Akosile, O. A.) [1] ; Oni, AI (Oni, A. I.) [1] ; Opowoye, IO (Opowoye, I. O.) [3] ; Ishola, CA (Ishola, C. A.) [3] ; Adebiyi, JO (Adebiyi, J. O.) ; Odeyemi, AJ (Odeyemi, A. J.) [1] ; Adjei-Mensah, B (Adjei-Mensah, B.) [2] ; Uyanga, VA (Uyanga, V. A.) [1] ; Abioja, MO (Abioja, M. O.) [1] **(provided by Clarivate) Source POULTRY SCIENCE Volume 103 Issue 9 DOI 10.1016/j.psj.2024.104003 Article Number 104003 Published SEP 2024 Early Access JUL 2024 Indexed 2024-08-13 Document Type Article**

Abstract

Oxidative stress (OS) OS) is a major concern that impacts the overall health of chickens in modern production systems. It is characterized by an imbalance between antioxidant defence mechanisms and the production of reactive oxygen species (ROS). ROS). This literature review aims to provide a comprehensive overview of oxidative stress in poultry production, with an emphasis on its effects on growth performance, immune responses, and reproductive outcomes. This review highlights the intricate mechanisms underlying OS and discusses how various factors, including dietary components, genetic predispositions, and environmental stressors can exacerbate the production of ROS. Additionally, the impact of oxidative stress on the production performance and physiological systems of poultry is examined. The study also emphasizes the relationship between oxidative stress and poultry diseases, highlighting how impaired antioxidant defenses increase bird's susceptibility to infections. The review assesses the existing approaches to reducing oxidative stress in chickens in response to these challenges. This includes managing techniques to lower stress in the production environment, antioxidant supplements, and nutritional interventions. The effectiveness of naturally occurring antioxidants, including plant extracts, minerals, and vitamins to improve poultry resistance to oxidative damage is also examined. To improve the antioxidant defenses of poultry under stress conditions, the activation of cellular homeostatic networks termed vitagenes, such as Nuclear Factor Erythroid 2-Related Factor 2 (Nrf2) is necessary for the synthesis of protective factors that can counteract the increased production of ROS and RNS. Future studies into novel strategies for managing oxidative stress in chicken production would build on these research advances and the knowledge gaps identified in this review.

Keywords

Author Keywords

oxidative stresspoultryclimate changeperformancereactive oxygen species

Keywords Plus

HEAT-STRESSBROILER-CHICKENSREACTIVE OXYGENGROWTH-PERFORMANCEVITAMIN-ELIPID-PEROXIDATIONANTIOXIDANT STATUSCARCASS TRAITSFREE-RADICALSHEAT-SHOCK-PROTEIN-70 EXPRESSION

Oxidative Stress

2-Oxidative Stress in Health and Disease

By Reddy, VP (Reddy, V. Prakash) [1] Source BIOMEDICINES Volume 11 Issue 11 DOI 10.3390/biomedicines11112925 Article Number 2925 Published NOV 2023 Indexed 2023-12-23 Document Type Review

Abstract

Oxidative stress, resulting from the excessive intracellular accumulation of reactive oxygen species (ROS), reactive nitrogen species (RNS), and other free radical species, contributes to the onset and progression of various diseases, including diabetes, obesity, diabetic nephropathy, diabetic neuropathy, and neurological diseases, such as Alzheimer's disease (AD), amyotrophic lateral sclerosis (ALS), and Parkinson's disease (PD). Oxidative stress is also implicated in cardiovascular disease and cancer. Exacerbated oxidative stress leads to the accelerated formation of advanced glycation end products (AGEs), a complex mixture of crosslinked proteins and protein modifications. Relatively high levels of AGEs are generated in diabetes, obesity, AD, and other I neurological diseases. AGEs such as Ne-carboxymethyllysine (CML) serve as markers for disease progression. AGEs, through interaction with receptors for advanced glycation end products (RAGE), initiate a cascade of deleterious signaling events to form inflammatory cytokines, and thereby further exacerbate oxidative stress in a vicious cycle. AGE inhibitors, AGE breakers, and RAGE inhibitors are therefore potential therapeutic agents for multiple diseases, including diabetes and AD. The complexity of the AGEs and the lack of well-established mechanisms for AGE formation are largely responsible for the lack of effective therapeutics targeting oxidative stress and AGE-related diseases. This review addresses the role of oxidative stress in the pathogenesis of AGE-related chronic diseases, including diabetes and neurological disorders, and recent progress in the development of therapeutics based on antioxidants, AGE breakers and RAGE inhibitors. Furthermore, this review outlines therapeutic strategies based on single-atom nanozymes that attenuate oxidative stress through the sequestering of reactive oxygen species (ROS) and reactive nitrogen species (RNS).

Keywords

Author Keywords

oxidative stressAlzheimer's diseasediabetesreactive oxygen speciesreactive nitrogen species4-hydroxy-trans-2-nonenal (HNE)lipid peroxidationnanozymesreceptors for advanced glycation end products (RAGE)

Keywords Plus

GLYCATION END-PRODUCTSALZHEIMERS-DISEASECROSS-LINKS-NITROSYLATIONREACTIVE OXYGENDNABRAINRISKMETABOLISMNANOZYMES

Oxidative Stress

3-Mechanisms of oxidative stress-induced sperm dysfunction

By Wang, YT (Wang, Yutao) [1] ; Fu, X (Fu, Xun) [1] ; Li, HJ (Li, Hongjun) [1] (provided by Clarivate) **Source FRONTIERS IN ENDOCRINOLOGY Volume 16 DOI 10.3389/fendo.2025.1520835 Article Number 1520835 Published FEB 5 2025 Indexed 2025-02-23 Document Type Review**

Abstract

Oxidative stress plays a pivotal role in male infertility by impairing sperm function through various molecular mechanisms. This review explores the impact of excessive reactive oxygen species (ROS) on spermatozoa, particularly focusing on lipid peroxidation, DNA fragmentation, and protein oxidation. Lipid peroxidation damages sperm membranes, reducing fluidity and motility. ROS-induced DNA fragmentation compromises genetic integrity, potentially leading to infertility and adverse offspring outcomes. Protein oxidation alters key structural proteins, impairing sperm motility and the ability to fertilize an egg. The primary sources of oxidative stress in sperm include leukocyte activity, mitochondrial dysfunction, and environmental factors such as smoking and pollution. Despite the presence of natural antioxidant defenses, spermatozoa are particularly vulnerable due to limited repair mechanisms. The review highlights the importance of early intervention through antioxidant therapies and lifestyle changes to mitigate the detrimental effects of oxidative stress on male fertility. Further research is essential to enhance therapeutic approaches and improve reproductive outcomes.

Keywords

Author Keywords

oxidative stresssperm dysfunctionreactive oxygen speciesDNA fragmentation and male infertilitymale infertility

Keywords Plus

GLUTATHIONE-PEROXIDASE 4SUPEROXIDE-DISMUTASELIPID-PEROXIDATIONTYROSINE PHOSPHORYLATIONDNA FRAGMENTATIONMALE-INFERTILITYSEMEN QUALITY8-HYDROXY-2'-DEOXYGUANOSINEEXPRESSIONFERTILITY

Oxidative Stress

4-Oxidative Stress in Liver Pathophysiology and Disease

By Allameh, A (Allameh, Abdolamir) [1] ; Niayesh-Mehr, R (Niayesh-Mehr, Reyhaneh) [1] ; Aliarab, A (Aliarab, Azadeh) [1] ; Sebastiani, G (Sebastiani, Giada) [2] , [3] ; Pantopoulos, K (Pantopoulos, Kostas) [3] , [4] (provided by Clarivate) **Source ANTIOXIDANTS Volume 12 Issue 9 DOI 10.3390/antiox12091653 Article Number 1653 Published SEP 2023 Indexed 2024-02-04 Document type Review**

Abstract

The liver is an organ that is particularly exposed to reactive oxygen species (ROS), which not only arise during metabolic functions but also during the biotransformation of xenobiotics. The disruption of redox balance causes oxidative stress, which affects liver function, modulates inflammatory pathways and contributes to disease. Thus, oxidative stress is implicated in acute liver injury and in the pathogenesis of prevalent infectious or metabolic chronic liver diseases such as viral hepatitis B or C, alcoholic fatty liver disease, non-alcoholic fatty liver disease (NAFLD) and non-alcoholic steatohepatitis (NASH). Moreover, oxidative stress plays a crucial role in liver disease progression to liver fibrosis, cirrhosis and hepatocellular carcinoma (HCC). Herein, we provide an overview on the effects of oxidative stress on liver pathophysiology and the mechanisms by which oxidative stress promotes liver disease.

Keywords
Author Keywords
ROSliver diseasefibrosishepatitisNAFLDNASHhepatocellular carcinoma

Keywords Plus
NONALCOHOLIC FATTY LIVERADENINE-DINUCLEOTIDE PHOSPHATEENDOPLASMIC-RETICULUM STRESSSINUSOIDAL ENDOTHELIAL-CELLSHEPATIC STELLATE CELLSTISSUE GROWTH-FACTOROXYGEN SPECIES ROSHEPATOCELLULAR-CARCINOMATGF-BETAKUPFFER CELLS

Oxidative Stress

5-Overview of oxidative stress and inflammation in diabetes

By Sibony, RW (Weinberg Sibony, Roni) [1] ; Segev, O (Segev, Omri) [2] ; Dor, S (Dor, Saar) [1] ; Raz, I (Raz, Itamar) [3] , [4] (provided by Clarivate) **Source JOURNAL OF DIABETES Volume 16 Issue 10 DOI 10.1111/1753-0407.70014 Article Number e70014 Published OCT 2024 Indexed 2024-10-28 Document Type Review**

Abstract

The global prevalence of diabetes has increased significantly, leading to various complications and a negative impact on quality of life. Hyperglycemia hyperglycemic-induced oxidative stress (OS) and inflammation are closely associated with the development and progression of type 2 diabetes mellitus (T2D) and its complications. This review explores the effect of T2D on target organ damage and potential treatments to minimize this damage. The paper examines the pathophysiology of T2D, focusing on low-grade chronic inflammation and OS and on their impact on insulin resistance. The review discusses the role of inflammation and OS in the development of microvascular and macrovascular complications. The findings highlight the mechanisms by which inflammatory cytokines, stress kinases, and reactive oxygen species (ROS) interfere with insulin signaling pathways, leading to impaired glucose metabolism and organ dysfunction. Lifestyle interventions, including a balanced diet and exercise, can help reduce chronic inflammation and OS, thereby preventing and controlling T2D and its associated complications. Additionally, various antioxidants and anti-inflammatory agents show potential in reducing OS and inflammation. Some anti-diabetic drugs, like pioglitazone, metformin, and glucagon-like peptide-1 (GLP-1) agonists, may also have anti-inflammatory effects. Further research, including randomized controlled trials, is needed to evaluate the efficacy of these interventions.image

HighlightsChronic inflammation from poor nutrition, unhealthy lifestyles, and toxin exposure increases the risk of chronic diseases and diabetes complications. OS, ROS, and inflammation contribute to insulin resistance and beta-cell dysfunction. A healthy lifestyle, proper weight, regular exercise, and an antioxidant-rich diet including flavonoids, carotenoids, curcumin, gallic acid, and vitamins can help prevent OS and ROS, thus preventing diabetes complications. Anti-diabetic medications also play a role in reducing inflammation. image

Keywords

Author Keywords

inflammationinsulin resistanceOStype 2 diabetes mellitus

Keywords Plus

VITAMIN-E SUPPLEMENTATIONGLYCEMIC CONTROLCARDIOVASCULAR-DISEASEINSULIN-RESISTANCEPHYSICAL-ACTIVITYNITRIC-OXIDEGALLIC ACIDGREEN TEAMELLITUSRISK

Oxidative Stress

6-Microplastics and Oxidative Stress-Current Problems and Prospects

By Kadac-Czapska, K (Kadac-Czapska, Kornelia) [1] ; Osko, J (Osko, Justyna) [1] ; Knez, E (Knez, Eliza) [1] ; Grembecka, M (Grembecka, Malgorzata) [1] (provided by Clarivate) **Source ANTIOXIDANTS Volume 13 Issue 5 DOI 10.3390/antiox13050579 Article Number 579 Published MAY 2024 Indexed 2024-06-03 Document Type Review**

Abstract

Microplastics (MPs) are plastic particles between 0.1 and 5000 mu m in size that have attracted considerable attention from the scientific community and the general public, as they threaten the environment. Microplastics contribute to various harmful effects, including lipid peroxidation, DNA damage, activation of mitogen-activated protein kinase pathways, cell membrane breakages, mitochondrial dysfunction, lysosomal defects, inflammation, and apoptosis. They affect cells, tissues, organs, and overall health, potentially contributing to conditions like cancer and cardiovascular disease. They pose a significant danger due to their widespread occurrence in food. In recent years, information has emerged indicating that MPs can cause oxidative stress (OS), a known factor in accelerating the aging of organisms. This comprehensive evaluation exposed notable variability in the reported connection between MPs and OS. This work aims to provide a critical review of whether the harmfulness of plastic particles that constitute environmental contaminants may result from OS through a comprehensive analysis of recent research and existing scientific literature, as well as an assessment of the characteristics of MPs causing OS. Additionally, the article covers the analytical methodology used in this field. The conclusions of this review point to the necessity for further research into the effects of MPs on OS.

Keywords
Author Keywords
microplasticsplasticoxidative stressreactive oxygen specieshuman health risk

Keywords Plus
POLYSTYRENE MICROPLASTICSSUPEROXIDE-DISMUTASEANTIOXIDANT ENZYMESLIPID-PEROXIDATIONHYDROGEN-PEROXIDEFREE-RADICALSDNA-DAMAGEMAP KINASEIN-VITRONANOPARTICLES

Oxidative Stress

7-Oxidative stress and mitochondrial impairment: Key drivers in neurodegenerative disorders

By Wen, P (Wen, Pei) [1] ; Sun, ZX (Sun, Zhixin) [1] ; Gou, FT (Gou, Fengting) [1] ; Wang, JJ (Wang, Jingjing) [1] ; Fan, Q (Fan, Qing) [1] ; Zhao, DM (Zhao, Deming) [1] ; Yang, LF (Yang, Lifeng) [1] (provided by Clarivate) **Source AGEING RESEARCH REVIEWS Volume 104 DOI 10.1016/j.arr.2025.102667 Article Number 102667 Published FEB 2025 Early Access JAN 2025 Indexed 2025-02-09 Document Type Review**

Abstract

Mitochondrial dysfunction and oxidative stress are critical factors in the pathogenesis of neurodegenerative diseases. The complex interplay between these factors exacerbates neuronal damage and accelerates disease progression. In neurodegenerative diseases, mitochondrial dysfunction impairs ATP production and promotes the generation of reactive oxygen species (ROS). The accumulation of ROS further damages mitochondrial DNA, proteins, and lipids, creating a vicious cycle of oxidative stress and mitochondrial impairment. This review aims to elucidate the mechanisms by which mitochondrial dysfunction and oxidative stress lead to neurodegeneration, and to highlight potential therapeutic targets to mitigate their harmful effects.

Keywords
Author Keywords
Mitochondrial dysfunctionROSOxidative stressNeurodegenerative diseases

Keywords Plus
INCREASED LIPID-PEROXIDATIONALZHEIMERS-DISEASE NEURONSDYNAMIN-RELATED PROTEIN-1TRANSGENIC MOUSE MODELAMYLOID-BETASUPEROXIDE-DISMUTASEHUNTINGTONS-DISEASEPARKINSONS-DISEASEREACTIVE OXYGENSYNAPTIC DAMAGE

Oxidative Stress

8-Oxidative Stress and Skin Diseases: The Role of Lipid Peroxidation

By Li Pomi, F (Li Pomi, Federica) [1] ; Gammeri, L (Gammeri, Luca) [2] ; Borgia, F (Borgia, Francesco) [3] ; Di Gioacchino, M (Di Gioacchino, Mario) [4] ; Gangemi, S (Gangemi, Sebastiano) [5]
(provided by Clarivate) Source ANTIOXIDANTS Volume 14 Issue 5 DOI 10.3390/antiox14050555 Article Number 555 Published MAY 7 2025 Indexed 2025-06-06 Document Type Review

Abstract

Lipid peroxidation (LPO) is a biochemical process through which lipids are subjected to a peroxidation reaction in the presence of free radicals. The process can cause alterations in biological membranes and the formation of substances harmful to the body that can form aggregates with proteins and nucleic acids. Malondialdehyde (MDA) and 4-hydroxynonenal (4-HNE) are the main products of LPO. These compounds have cytotoxic and genotoxic properties and contribute to the pathogenesis of various diseases. This research focuses on the correlation between LPO and skin diseases. For some skin diseases, such as psoriasis, vitiligo, and alopecia, LPO products have been shown to have a clear role in the pathogenesis of the disease. Lipid aldehydic products like MDA and 4-HNE can enhance inflammation by stimulating pro-inflammatory genes and producing cytokines. Furthermore, these products can stimulate cell death and increase oxidative stress. For other diseases (atopic dermatitis, urticaria, pemphigus, and melanoma), the role of LPO is unclear, even if the levels of LPO biomarkers are elevated in proportion to the severity of the disease. LPO can also be exploited to counteract the proliferation of neoplastic cells. Therefore, enhancing LPO would play an adjuvant role in the therapy of neoplastic diseases such as melanoma. In particular, the therapeutic implication resulting from the role of LPO products in the cytotoxicity induced by photodynamic therapy used for the adjuvant treatment of melanoma could be of interest in the future.

Keywords
Author Keywords
lipid peroxidationoxidative stress4-hydroxynonenalmalondialdehydeskinmelanomaferroptosisvitiligopsoriasisatopic dermatitis

Keywords Plus
ANTIOXIDANT ENZYME-ACTIVITYPHOTODYNAMIC THERAPYPEMPHIGUS FOLIACEUSATOPIC-DERMATITISALOPECIA-AREATAMELANOMAEXPRESSIONGROWTHMALONDIALDEHYDEINFLAMMATION

Oxidative Stress

9-Reactive Oxygen Species Signaling and Oxidative Stress: Transcriptional Regulation and Evolution

By Hong, YH (Hong, Yuhang) [1] ; Boiti, A (Boiti, Alessandra) [1] ; Vallone, D (Vallone, aniela) [1] ; Foulkes, NS (Foulkes, Nicholas S.) [1] (provided by Clarivate) **Source ANTIOXIDANTS Volume 13 Issue 3 DOI 10.3390/antiox13030312 Article Number 312 Published MAR 2024 Indexed 2024-04-03 Document Type Review**

Abstract

Since the evolution of the aerobic metabolism, reactive oxygen species (ROS) have represented significant challenges to diverse life forms. In recent decades, increasing knowledge has revealed a dual role for ROS in cell physiology, showing they serve as a major source of cellular damage while also functioning as important signaling molecules in various biological processes. Our understanding of ROS homeostasis and ROS-mediated cellular signaling pathways has presumed that they are ancient and highly conserved mechanisms shared by most organisms. However, emerging evidence highlights the complexity and plasticity of ROS signaling, particularly in animals that have evolved in extreme environments. In this review, we focus on ROS generation, antioxidative systems and the main signaling pathways that are influenced by ROS. In addition, we discuss ROS's responsive transcription regulation and how it may have been shaped over the course of evolution.

Keywords
Author Keywords
reactive oxygen speciescellular signalingDNA repairtranscriptional regulationvertebrate evolution

Keywords Plus
NF-KAPPA-BMAP KINASEHYDROGEN-PEROXIDEREDOX REGULATIONROS HOMEOSTASISNITRIC-OXIDEDNA-DAMAGEVITAMIN-CCELLANTIOXIDANT

Oxidative Stress

10-Flavonoids and their role in oxidative stress, inflammation, and human diseases

---

By Jomova, K (Jomova, Klaudia) [1] ; Alomar, SY (Alomar, Suliman Y.) [2] ; Valko, R (Valko, Richard) [2] ; Liska, J (Liska, Jan) [3] ; Nepovimova, E (Nepovimova, Eugenie) [4] , [5] ; Kuca, K (Kuca, Kamil) [5] , [6] ; Valko, M (Valko, Marian) [7] (provided by Clarivate) **Source CHEMICO-BIOLOGICAL INTERACTIONS Volume 413 DOI 10.1016/j.cbi.2025.111489 Article Number 111489 Published MAY 25 2025 Early Access MAR 2025 Indexed 2025-04-11 Document Type Review**

Abstract

Oxidative stress and chronic inflammation are important drivers in the pathogenesis and progression of many chronic diseases, such as cancers of the breast, kidney, lung, and others, autoimmune diseases (rheumatoid arthritis), cardiovascular diseases (hypertension, atherosclerosis, arrhythmia), neurodegenerative diseases (Alzheimer's disease, Parkinson's disease, Huntington's disease), mental disorders (depression, schizophrenia, bipolar disorder), gastrointestinal disorders (inflammatory bowel disease, colorectal cancer), and other disorders. With the increasing demand for less toxic and more tolerable therapies, flavonoids have the potential to effectively modulate the responsiveness to conventional therapy and radiotherapy. Flavonoids are polyphenolic compounds found in fruits, vegetables, grains, and plant-derived beverages. Six of the twelve structurally different flavonoid subgroups are of dietary significance and include anthocyanidins (e.g. pelargonidin, cyanidin), flavan-3-ols (e.g. epicatechin, epigallocatechin), flavonols (e.g. quercetin, kaempferol), flavones (e.g. luteolin, baicalein), flavanones (e.g. hesperetin, naringenin), and isoflavones (daidzein, genistein). The health benefits of flavonoids are related to their structural characteristics, such as the number and position of hydroxyl groups and the presence of C2--C3 double bonds, which predetermine their ability to chelate metal ions, terminate ROS (e.g. hydroxyl radicals formed by the Fenton reaction), and interact with biological targets to trigger a biological response. Based on these structural characteristics, flavonoids can exert both antioxidant or prooxidant properties, modulate the activity of ROS-scavenging enzymes and the expression and activation of proinflammatory cytokines (e.g., interleukin-1beta (IL-1 beta), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha)), induce apoptosis and autophagy, and target key signaling pathways, such as the nuclear factor erythroid 2-related factor 2 (Nrf2) and Bcl-2 family of proteins. This review aims to briefly discuss the mutually interconnected aspects of oxidative and inflammatory mechanisms, such as lipid peroxidation, protein oxidation, DNA damage, and the mechanism and resolution of inflammation. The major part of this article discusses the role of flavonoids in alleviating oxidative stress and inflammation, two common components of many human diseases. The results of epidemiological studies on flavonoids are also presented.

Keywords
Author Keywords
FlavonoidsOxidative stressInflammationChronic diseasesTherapy

Keywords Plus

Oxidative Stress

NF-KAPPA-BCELL-CYCLE ARRESTLIPID-PEROXIDATIONALZHEIMERS-DISEASEBLOOD-PRESSURECANCER CELLSNITRIC-OXIDEIN-VITROEXPERIMENTAL COLITISINSULIN-RESISTANCE

Oxidative Stress

11-Gut Microbiota Dysbiosis, Oxidative Stress, Inflammation, and Epigenetic Alterations in Metabolic Diseases

By Abdolmaleky, HM (Abdolmaleky, Hamid Mostafavi) [1] , [2] ; Zhou, JR (Zhou, Jin-Rong) [1]
(provided by Clarivate) Source ANTIOXIDANTS Volume 13 Issue 8 DOI 10.3390/antiox13080985 Article Number 985 Published AUG 2024 Indexed 2024-09-15 Document Type Review

Abstract

Gut dysbiosis, resulting from an imbalance in the gut microbiome, can induce excessive production of reactive oxygen species (ROS), leading to inflammation, DNA damage, activation of the immune system, and epigenetic alterations of critical genes involved in the metabolic pathways. Gut dysbiosis-induced inflammation can also disrupt the gut barrier integrity and increase intestinal permeability, which allows gut-derived toxic products to enter the liver and systemic circulation, further triggering oxidative stress, inflammation, and epigenetic alterations associated with metabolic diseases. However, specific gut-derived metabolites, such as short-chain fatty acids (SCFAs), lactate, and vitamins, can modulate oxidative stress and the immune system through epigenetic mechanisms, thereby improving metabolic function. Gut microbiota and diet-induced metabolic diseases, such as obesity, insulin resistance, dyslipidemia, and hypertension, can transfer to the next generation, involving epigenetic mechanisms. In this review, we will introduce the key epigenetic alterations that, along with gut dysbiosis and ROS, are engaged in developing metabolic diseases. Finally, we will discuss potential therapeutic interventions such as dietary modifications, prebiotics, probiotics, postbiotics, and fecal microbiota transplantation, which may reduce oxidative stress and inflammation associated with metabolic syndrome by altering gut microbiota and epigenetic alterations. In summary, this review highlights the crucial role of gut microbiota dysbiosis, oxidative stress, and inflammation in the pathogenesis of metabolic diseases, with a particular focus on epigenetic alterations (including histone modifications, DNA methylomics, and RNA interference) and potential interventions that may prevent or improve metabolic diseases.

Keywords
Author Keywords
gut dysbiosismicrobiotamicrobiomeoxidative stressinflammationepigenetictransgenerationalmetabolic diseases

Keywords Plus
HOSTDIETHOMEOSTASISBACTERIALCELLSACID

Oxidative Stress

12-Mitochondria in oxidative stress, inflammation and aging: from mechanisms to therapeutic advances

---

By Xu, XY (Xu, Xieyang) [1] , [2] , [3] ; Pang, Y (Pang, Yan) [1] , [2] , [3] ; Fan, XQ (Fan, Xianqun) [1] , [2] , [3] **(provided by Clarivate) Source SIGNAL TRANSDUCTION AND TARGETED THERAPY Volume 10 Issue 1 DOI 10.1038/s41392-025-02253-4 Article Number 190 Published JUN 11 2025 Indexed 2025-06-18 Document Type Review**

Abstract

Mitochondria are the energy production centers in cells and have unique genetic information. Due to the irreplaceable function of mitochondria, mitochondrial dysfunction often leads to pathological changes. Mitochondrial dysfunction induces an imbalance between oxidation and antioxidation, mitochondrial DNA (mtDNA) damage, mitochondrial dynamics dysregulation, and changes in mitophagy. It results in oxidative stress due to excessive reactive oxygen species (ROS) generation, which contributes to cell damage and death. Mitochondrial dysfunction can also trigger inflammation through the activation of damage-associated molecular patterns (DAMPs), inflammasomes and inflammatory cells. Besides, mitochondrial alterations in the functional regulation, energy metabolism and genetic stability accompany the aging process, and there has been a lot of evidence suggesting that oxidative stress and inflammation, both of which are associated with mitochondrial dysfunction, are predisposing factors of aging. Therefore, this review hypothesizes that mitochondria serve as central hubs regulating oxidative stress, inflammation, and aging, and their dysfunction contributes to various diseases, including cancers, cardiovascular diseases, neurodegenerative disorders, metabolic diseases, sepsis, ocular pathologies, liver diseases, and autoimmune conditions. Moreover, we outline therapies aimed at various mitochondrial dysfunctions, highlighting their performance in animal models and human trials. Additionally, we focus on the limitations of mitochondrial therapy in clinical applications, and discuss potential future research directions for mitochondrial therapy.

Keywords

Keywords Plus

CYCLIC GMP-AMPACTIVATED PROTEIN-KINASEAGE-RELATED-CHANGESREACTIVE OXYGENNLRP3 INFLAMMASOMESKELETAL-MUSCLEGASDERMIN-DCELL-DEATHCOMPLEX-IDNA-DAMAGE

Oxidative Stress

13-Oxidative stress in Alzheimer's disease: current knowledge of signaling pathways and therapeutics

---

By Dhapola, R (Dhapola, Rishika) [1] ; Beura, SK (Beura, Samir K.) [2] ; Sharma, P (Sharma, Prajjwal) [1] ; Singh, SK (Singh, Sunil K.) [2] ; Harikrishnareddy, D (Harikrishnareddy, Dibbanti) [1]
(provided by Clarivate) Source MOLECULAR BIOLOGY REPORTS Volume 51 Issue 1 DOI 10.1007/s11033-023-09021-z Article Number 48 Published DEC 2024 Indexed 2024-01-22 Document Type Review

Abstract

Alzheimer's disease's pathophysiology is still a conundrum. Growing number of evidences have elucidated the involvement of oxidative stress in the pathology of AD rendering it a major target for therapeutic development. Reactive oxygen species (ROS) generated by altered mitochondrial function, dysregulated electron transport chain and other sources elevate aggregated A beta and neurofibrillary tangles which further stimulating the production of ROS. Oxidative stress induced damage to lipids, proteins and DNA result in neuronal death which leads to AD. In addition, oxidative stress induces apoptosis that is triggered by the modulation of ERK1/2 and Nrf2 pathway followed by increased GSK-3 beta expression and decreased PP2A activity. Oxidative stress exaggerates disease condition by interfering with various signaling pathways like RCAN1, CREB/ERK, Nrf2, PP2A, NF kappa B and PI3K/Akt. Studies have reported the role of TNF-alpha in oxidative stress stimulation that has been regulated by drugs like etanercept increasing the level of anti-oxidants. Other drugs like pramipexole, memantine, carvedilol, and melatonin have been reported to activate CREB/RCAN1 and Nrf2 pathways. In line with this, epigallocatechin gallate and genistein also target Nrf2 and CREB pathway leading to activation of downstream pathways like ARE and Keap1 which ameliorate oxidative stress condition. Donepezil and resveratrol reduce oxidative stress and activate AMPK pathway along with PP2A activation thus promoting tau dephosphorylation and neuronal survival. This study describes in detail the role of oxidative stress in AD, major signaling pathways involving oxidative stress induced AD and drugs under development targeting these pathways which may aid in therapeutic advances for AD.

Keywords
Author Keywords
Alzheimer's diseaseOxidative stressSignaling pathwaysCREBNrf2Drugs

Keywords Plus
AMYLOID-BETA PEPTIDEFREE-RADICALSMITOCHONDRIAL DYSFUNCTIONPROTEASOMAL DEGRADATIONINFLAMMATIONRCAN1ACIDANTIOXIDANTSCALCINEURINACTIVATION

Oxidative Stress

14-Oxidative stress and inflammation in the pathogenesis of neurological disorders: Mechanisms and implications

---

By Dash, UC (Dash, Umesh Chandra) [1] ; Bhol, NK (Bhol, Nitish Kumar) [2] ; Swain, SK (Swain, Sandeep Kumar) [3] ; Samal, RR (Samal, Rashmi Rekha) [4] ; Nayak, PK (Nayak, Prabhat Kumar) [5] ; Raina, V (Raina, Vishakha) [1] ; Panda, SK (Panda, Sandeep Kumar) [1] ; Kerry, RG (Kerry, Rout George) [2] ; Duttaroy, AK (Duttaroy, Asim K.) [6] ; Jena, AB (Jena, Atala Bihari) [7] (provided by Clarivate) **Source ACTA PHARMACEUTICA SINICA B Volume 15 Issue 1 Page 15-34 DOI 10.1016/j.apsb.2024.10.004 Published JAN 2025 Early Access FEB 2025 Indexed 2025-02-27 Document Type Article**

Abstract

Neuroprotection is a proactive approach to safeguarding the nervous system, including the brain, spinal cord , peripheral nerves, by preventing or limiting damage to nerve cells , other com- ponents. It primarily defends the central nervous system against injury from acute and progressive neuro- degenerative disorders. Oxidative stress, an imbalance between the body's natural defense mechanisms and the generation of reactive oxygen species, is crucial in developing neurological disorders. Due to its high metabolic rate and oxygen consumption, the brain is particularly vulnerable to oxidative stress. Excessive ROS damages the essential biomolecules, leading to cellular malfunction and neurodegenera- tion. Several neurological disorders, including Alzheimer's, Parkinson's, Amyotrophic lateral sclerosis, multiple sclerosis , ischemic stroke, are associated with oxidative stress. Understanding the impact of oxidative stress in these conditions is crucial for developing new treatment methods. Researchers are exploring using antioxidants , other molecules to mitigate oxidative stress, aiming to prevent or slow down the progression of brain diseases. By understanding the intricate interplay between oxidative stress and neurological disorders, scientists hope to pave the way for innovative therapeutic and preventive ap- proaches, ultimately improving individuals' living standards. 2025 The Authors. Published by Elsevier B.V. on behalf of Chinese Pharmaceutical Association and Institute of Materia Medica, Chinese Academy of Medical Sciences. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

Keywords

Author Keywords

NeurodegenerationOxidative stressROSNeuroprotectionMitochondrial dysfunctionAging

Keywords Plus

AMYOTROPHIC-LATERAL-SCLEROSISCORONARY-HEART-DISEASEMITOCHONDRIAL DYSFUNCTIONNEURODEGENERATIVE DISEASESABNORMAL INTERACTIONIONIZING-RADIATIONLIPID-PEROXIDATIONMUTANT HUNTINGTINALPHA-SYNUCLEINAMYLOID-BETA

Oxidative Stress

15-Hypoxia, oxidative stress, and the interplay of HIFs and NRF2 signaling in cancer

By Bae, T (Bae, Taegeun) [1] ; Hallis, SP (Hallis, Steffanus Pranoto) [2] ; Kwak, MK (Kwak, Mi-Kyoung) [1] , [2] , [3] (provided by Clarivate) **Source EXPERIMENTAL AND MOLECULAR MEDICINE Volume 56 Issue 3 Page 501-514 DOI 10.1038/s12276-024-01180-8 Published MAR 2024 Early Access MAR 2024 Indexed 2024-03-27 Document Type Review**

Abstract

Oxygen is crucial for life and acts as the final electron acceptor in mitochondrial energy production. Cells adapt to varying oxygen levels through intricate response systems. Hypoxia-inducible factors (HIFs), including HIF-1 alpha and HIF-2 alpha, orchestrate the cellular hypoxic response, activating genes to increase the oxygen supply and reduce expenditure. Under conditions of excess oxygen and resulting oxidative stress, nuclear factor erythroid 2-related factor 2 (NRF2) activates hundreds of genes for oxidant removal and adaptive cell survival. Hypoxia and oxidative stress are core hallmarks of solid tumors and activated HIFs and NRF2 play pivotal roles in tumor growth and progression. The complex interplay between hypoxia and oxidative stress within the tumor microenvironment adds another layer of intricacy to the HIF and NRF2 signaling systems. This review aimed to elucidate the dynamic changes and functions of the HIF and NRF2 signaling pathways in response to conditions of hypoxia and oxidative stress, emphasizing their implications within the tumor milieu. Additionally, this review explored the elaborate interplay between HIFs and NRF2, providing insights into the significance of these interactions for the development of novel cancer treatment strategies.

In our daily lives, oxygen is vital for survival, but its levels can vary due to environmental shifts or within our bodies, such as in diseases like heart disease or cancer. However, excess oxygen can also be detrimental, leading to a condition called oxidative stress. Cells have evolved systems to adapt to these fluctuating oxygen levels, with key roles played by proteins named hypoxia-inducible factors (HIFs) and nuclear factor erythroid 2-related factor 2 (NRF2). These factors aid cells' survival by activating different and overlapping genes that can enhance oxygen supply or shield against damage. This study discovered that HIFs and NRF2 can occasionally collaborate to aid cancer cells' growth and treatment resistance. The key discoveries suggest that targeting these pathways could be a novel approach to cancer treatment, especially in tumors that have adapted to low oxygen conditions.This summary was initially drafted using artificial intelligence, then revised and fact-checked by the author.

Keywords

Keywords Plus

INDUCIBLE FACTOR 1-ALPHATRANSCRIPTION FACTOR NRF2ENDOTHELIAL GROWTH-FACTORSTEM-CELLSMESENCHYMAL TRANSITIONMULTIDRUG-RESISTANCEFACTORS HIF-1-ALPHATUMOR ANGIOGENESISCARCINOMA-CELLSDOWN-REGULATION

Oxidative Stress

16- Oxidative Stress: The Role of Antioxidant Phytochemicals in the Prevention and Treatment of Diseases

---

By Muscolo, A (Muscolo, Adele) [1] ; Mariateresa, O (Mariateresa, Oliva) [1] ; Giulio, T (Giulio, Torello) [2] ; Mariateresa, R (Mariateresa, Russo) [1] (provided by Clarivate) **Source INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES Volume 25 Issue 6 DOI 10.3390/ijms25063264 Article Number 3264 Published MAR 2024 Indexed 2024-04-12 Document Type Review**

Abstract

Oxidative stress, characterized by an imbalance favouring oxidants over antioxidants, is a key contributor to the development of various common diseases. Counteracting these oxidants is considered an effective strategy to mitigate the levels of oxidative stress in organisms. Numerous studies have indicated an inverse correlation between the consumption of vegetables and fruits and the risk of chronic diseases, attributing these health benefits to the presence of antioxidant phytochemicals in these foods. Phytochemicals, present in a wide range of foods and medicinal plants, play a pivotal role in preventing and treating chronic diseases induced by oxidative stress by working as antioxidants. These compounds exhibit potent antioxidant, anti-inflammatory, anti-aging, anticancer, and protective properties against cardiovascular diseases, diabetes mellitus, obesity, and neurodegenerative conditions. This comprehensive review delves into the significance of these compounds in averting and managing chronic diseases, elucidating the key sources of these invaluable elements. Additionally, it provides a summary of recent advancements in understanding the health benefits associated with antioxidant phytochemicals.

Keywords
Author Keywords
antioxidant phytochemicalsfree radicalschronic diseasehealth benefitspolyphenols

Keywords Plus
TOTAL PHENOLIC CONTENTSCANCER CELL-GROWTHSECONDARY METABOLITESBIOACTIVE COMPOUNDSBIOAVAILABILITYCAPACITIESPOLYPHENOLSEGCGRESVERATROLCURCUMIN

17-Oxidative Stress and Redox Imbalance: Common Mechanisms in Cancer Stem Cells and Neurodegenerative Diseases

By Selvaraj, NR (Selvaraj, Nikhil Raj) [1] ; Nandan, D (Nandan, Durga) [1] ; Nair, BG (Nair, Bipin G.) [1] ; Nair, VA (Nair, Vipin A.) [1] ; Venugopal, P (Venugopal, Parvathy) [1] ; Aradhya, R (Aradhya, Rajaguru) [1] (provided by Clarivate) **Source CELLS Volume 14 Issue 7 DOI 10.3390/cells14070511 Article Number 511 Published MAR 29 2025 Indexed 2025-04-18 Document Type Review**

Abstract

Oxidative stress (OS) is an established hallmark of cancer and neurodegenerative disorders (NDDs), which contributes to genomic instability and neuronal loss. This review explores the contrasting role of OS in cancer stem cells (CSCs) and NDDs. Elevated levels of reactive oxygen species (ROS) contribute to genomic instability and promote tumor initiation and progression in CSCs, while in NDDs such as Alzheimer's and Parkinson's disease, OS accelerates neuronal death and impairs cellular repair mechanisms. Both scenarios involve disruption of the delicate balance between pro-oxidant and antioxidant systems, which leads to chronic oxidative stress. Notably, CSCs and neurons display alterations in redox-sensitive signaling pathways, including Nrf2 and NF-kappa B, which influence cell survival, proliferation, and differentiation. Mitochondrial dynamics further illustrate these differences: enhanced function in CSCs supports adaptability and survival, whereas impairments in neurons heighten vulnerability. Understanding these common mechanisms of OS-induced redox imbalance may provide insights for developing interventions, addressing aging hallmarks, and potentially mitigating or preventing both cancer and NDDs.

Keywords
Author Keywords
oxidative stressredox imbalancecancer stem cellsneurodegenerative diseasesreactive oxygen speciesmitochondrial dysfunctionoxidative phosphorylationferroptosisautophagyantioxidant

Keywords Plus
ERYTHROID 2-RELATED FACTOR-2AMYOTROPHIC-LATERAL-SCLEROSISAMYLOID-BETA PEPTIDEBLOOD-BRAIN-BARRIERMITOCHONDRIAL DYSFUNCTIONALZHEIMERS-DISEASETHERAPEUTIC IMPLICATIONSTUMOR MICROENVIRONMENTSUPEROXIDE-DISMUTASESIGNALING PATHWAYS

Oxidative Stress

18-Antioxidant Defense System in Plants: Reactive Oxygen Species Production, Signaling, and Scavenging During Abiotic Stress-Induced Oxidative Damage

By Rao, MJ (Rao, Muhammad Junaid) [1] ; Duan, MZ (Duan, Mingzheng) [2] ; Zhou, CX (Zhou, Caixia) [1] , [3] ; Jiao, JJ (Jiao, Jiejie) [4] ; Cheng, PW (Cheng, Peiwen) [1] , [3] ; Yang, LW (Yang, Lingwei) [1] , [3] ; Wei, W (Wei, Wei) [1] , [3] ; Shen, QY (Shen, Qinyuan) [1] , [3] ; Ji, PY (Ji, Piyu) [1] , [3] ; Yang, Y (Yang, Ying) [1] , [3] ; **(provided by Clarivate) Source HORTICULTURAE Volume 11 Issue 5 DOI 10.3390/horticulturae11050477 Article Number 477 Published APR 29 2025 Indexed 2025-05-31 Document Type Review**

Abstract

Plants face various abiotic stresses in their natural environments that trigger the production of reactive oxygen species (ROS), leading to oxidative stress and potential cellular damage. This comprehensive review examines the interplay between plant antioxidant defense systems and ROS under abiotic stress conditions. We discuss the major enzymatic antioxidants, including superoxide dismutase, catalase, reductases, and peroxidases, as well as non-enzymatic antioxidants, such as ascorbic acid, glutathione, polyphenols, and flavonoids, which play crucial roles in ROS detoxification. This review elaborates on different types of ROS, their production sites within plant cells, and their dual role as both damaging oxidants and key signaling molecules. We discuss how various abiotic stresses-including heat, cold, drought, flooding, salinity, and heavy metal toxicity-induce oxidative stress and trigger specific antioxidant responses in plants. Additionally, the mechanisms of ROS generation under these abiotic stress conditions and the corresponding activation of enzymatic and non-enzymatic scavenging systems are discussed in detail. This review also discusses recent advances in understanding ROS signaling networks and their integration with other stress-response pathways. This knowledge provides valuable insights into plant stress-tolerance mechanisms and suggests potential strategies for developing stress-resistant crops by enhancing antioxidant defense systems. Moreover, the strategic ROS modulation through priming, exogenous antioxidants, nanoparticles, or genetic tools can enhance plant resilience. Integrating these methods with agronomic practices (e.g., irrigation management) offers a sustainable path to climate-smart agriculture. Our review reveals that ROS accumulation can be detrimental; however, the coordinated action of various antioxidant systems helps plants maintain redox homeostasis and adapt to environmental stress.

Keywords
Author Keywords
antioxidantsabiotic stressROS productionoxidative damageROS signalingROS scavengingredox homeostasisclimate-smart agriculture

Keywords Plus
CYTOSOLIC ASCORBATE PEROXIDASEDROUGHT STRESSHYDROGEN-PEROXIDEGLUTATHIONE-REDUCTASEHIGH-TEMPERATUREGENE-EXPRESSIONCADMIUM STRESSMEDICAGO-TRUNCATULAGLYOXALASE SYSTEMSABSCISIC-ACID

Oxidative Stress

19-Unraveling the AMPK-SIRT1-FOXO Pathway: The In-Depth Analysis and Breakthrough Prospects of Oxidative Stress-Induced Diseases

By Guan, GQ (Guan, Guangqi) [1] ; Chen, YX (Chen, Yaoxing) [1] ; Dong, YL (Dong, Yulan) [1]
(provided by Clarivate) Source ANTIOXIDANTS Volume 14 Issue 1 DOI 10.3390/antiox14010070 Article Number 70 Published JAN 2025 Indexed 2025-01-30 Document Type Review

Abstract

Oxidative stress (OS) refers to the production of a substantial amount of reactive oxygen species (ROS), leading to cellular and organ damage. This imbalance between oxidant and antioxidant activity contributes to various diseases, including cancer, cardiovascular disease, diabetes, and neurodegenerative conditions. The body's antioxidant system, mediated by various signaling pathways, includes the AMPK-SIRT1-FOXO pathway. In oxidative stress conditions, AMPK, an energy sensor, activates SIRT1, which in turn stimulates the FOXO transcription factor. This cascade enhances mitochondrial function, reduces mitochondrial damage, and mitigates OS-induced cellular injury. This review provides a comprehensive analysis of the biological roles, regulatory mechanisms, and functions of the AMPK-SIRT1-FOXO pathway in diseases influenced by OS, offering new insights and methods for understanding OS pathogenesis and its therapeutic approaches.

Keywords
Author Keywords
AMPK-SIRT1-FOXO pathwayoxidative stressreactive oxygen speciesantioxidant mechanism

Keywords Plus
ACTIVATED PROTEIN-KINASEFOXO TRANSCRIPTION FACTORSSUPEROXIDE-DISMUTASESKELETAL-MUSCLEALZHEIMERS-DISEASEENERGY-METABOLISMSIRTUIN 1AMPKRESVERATROLCELLS

Oxidative Stress

20-From imbalance to impairment: the central role of reactive oxygen species in oxidative stress-induced disorders and therapeutic exploration

---

By Afzal, S (Afzal, Sheryar) [1] ; Manap, ASA (Manap, Aimi Syamima Abdul) [1] ; Attiq, A (Attiq, Ali) [2] ; Albokhadaim, I (Albokhadaim, Ibrahim) [1] ; Kandeel, M (Kandeel, Mahmoud) [1] , [3] ; Alhojaily, SM (Alhojaily, Sameer M.) [1] (provided by Clarivate) **Source FRONTIERS IN PHARMACOLOGY Volume 14 DOI 10.3389/fphar.2023.1269581 Article Number 1269581 Published OCT 18 2023 Indexed 2023-11-12 Document Type Review**

Abstract

Increased production and buildup of reactive oxygen species (ROS) can lead to various health issues, including metabolic problems, cancers, and neurological conditions. Our bodies counteract ROS with biological antioxidants such as SOD, CAT, and GPx, which help prevent cellular damage. However, if there is an imbalance between ROS and these antioxidants, it can result in oxidative stress. This can cause genetic and epigenetic changes at the molecular level. This review delves into how ROS plays a role in disorders caused by oxidative stress. We also look at animal models used for researching ROS pathways. This study offers insights into the mechanism, pathology, epigenetic changes, and animal models to assist in drug development and disease understanding.

Keywords
Author Keywords
oxidative stressepigenetic marksreactive oxygen speciesanimal modelsdrug discovery

Keywords Plus
HYDROPEROXIDE T-BHPALZHEIMERS-DISEASEDNA METHYLATIONAMYLOID-BETAEPIGENETIC INSTABILITYBUTHIONINE SULFOXIMINEGLUTATHIONE DEPLETIONCARBON-TETRACHLORIDEACATALASEMIC MICEINDUCED APOPTOSIS

Oxidative Stress

21-Isoliquiritigenin alleviates cerebral ischemia-reperfusion injury by reducing oxidative stress and ameliorating mitochondrial dysfunction via activating the Nrf2 pathway

By Lan, XB (Lan, Xiaobing) [1] , [4] ; Wang, Q (Wang, Qing) [2] , [3] ; Liu, Y (Liu, Yue) [1] ; You, Q (You, Qing) [1] ; Wei, W (Wei, Wei) [1] ; Zhu, CH (Zhu, Chunhao) [1] , [4] ; Hai, DM (Hai, Dongmei) [1] ; Cai, ZY (Cai, Zhenyu) [1] ; Yu, JQ (Yu, Jianqiang) [1] , [5] ; Zhang, J (Zhang, Jian) [1] , [4] ; **(provided by Clarivate) Source REDOX BIOLOGY Volume 77 DOI 10.1016/j.redox.2024.103406 Article Number 103406 Published NOV 2024 Early Access OCT 2024 Indexed 2024-11-08 Document Type Article**

Abstract

Cerebral ischemia-reperfusion injury (CIRI) refers to a secondary brain injury that occurs when blood supply is restored to ischemic brain tissue and is one of the leading causes of adult disability and mortality. Multiple pathological mechanisms are involved in the progression of CIRI, including neuronal oxidative stress and mitochondrial dysfunction. Isoliquiritigenin (ISL) has been preliminarily reported to have potential neuroprotective effects on rats subjected to cerebral ischemic insult. However, the protective mechanisms of ISL have not been elucidated. This study aims to further investigate the effects of ISL-mediated neuroprotection and elucidate the underlying molecular mechanism. The findings indicate that ISL treatment significantly alleviated middle cerebral artery occlusion (MCAO)-induced cerebral infarction, neurological deficits, histopathological damage, and neuronal apoptosis in mice. In vitro, ISL effectively mitigated the reduction of cell viability, Na+-K+ATPase, and MnSOD activities, as well as the degree of DNA damage induced by oxygen-glucose deprivation (OGD) injury in PC12 cells. Mechanistic studies revealed that administration of ISL evidently improved redox homeostasis and restored mitochondrial function via inhibiting oxidative stress injury and ameliorating mitochondrial biogenesis, mitochondrial fusion-fission balance, and mitophagy. Moreover, ISL facilitated the dissociation of Keap1/Nrf2, enhanced the nuclear transfer of Nrf2, and promoted the binding activity of Nrf2 with ARE. Finally, ISL obviously inhibited neuronal apoptosis by activating the Nrf2 pathway and ameliorating mitochondrial dysfunction in mice. Nevertheless, Nrf2 inhibitor brusatol reversed the mitochondrial protective properties and anti-apoptotic effects of ISL both in vivo and in vitro. Overall, our findings revealed that ISL exhibited a profound neuroprotective effect on mice following CIRI insult by reducing oxidative stress and ameliorating mitochondrial dysfunction, which was closely related to the activation of the Nrf2 pathway.

Keywords

Author Keywords

NeuroprotectionIsoliquiritigeninCerebral ischemia-reperfusion injuryMitochondrial dysfunctionNrf2 pathway

Keywords Plus

TRANSCRIPTION FACTOR NRF2ARTERY OCCLUSIONCELL-DEATHNEUROINFLAMMATIONMODULATIONPROTECTSMODELSSTROKEHEALTHTARGET

Oxidative Stress

22-The Role of Quercetin, a Flavonoid in the Management of Pathogenesis Through Regulation of Oxidative Stress, Inflammation, and Biological Activities

---

By Alharbi, HOA (Alharbi, Hajed Obaid A.) [1] ; Alshebremi, M (Alshebremi, Mohammad) [1] ; Babiker, AY (Babiker, Ali Yousif) [1] ; Rahmani, AH (Rahmani, Arshad Husain) [1] (provided by clarivate) **Source BIOMOLECULES Volume 15 Issue 1 DOI 10.3390/biom15010151 Article Number 151 Published JAN 2025 Indexed 2025-02-01 Document Type Review**

Abstract

Quercetin, a flavonoid found in vegetables and fruits, has been extensively studied for its health benefits and disease management. Its role in the prevention of various pathogenesis has been well-documented, primarily through its ability to inhibit oxidative stress, inflammation, and enhance the endogenous antioxidant defense mechanisms. Electronic databases such as Google Scholar, Scopus, PubMed, Medline, and Web of Science were searched for information regarding quercetin and its role in various pathogeneses. The included literature comprised experimental studies, randomized controlled trials, and epidemiological studies related to quercetin, while editorials, case analyses, theses, and letters were excluded. It has been reported to have a wide range of health benefits including hepatoprotective, antidiabetic, anti-obesity, neuroprotective, cardioprotective, wound healing, antimicrobial, and immunomodulatory effects, achieved through the modulation of various biological activities. Additionally, numerous in vitro and in vivo studies have shown that quercetin's efficacies in cancer management involve inhibiting cell signaling pathways, such as inflammation, cell cycle, and angiogenesis, activating cell signaling pathways including tumor suppressor genes, and inducing apoptosis. This review aims to provide a comprehensive understanding of the health benefits of quercetin in various pathogeneses. Additionally, this review outlines the sources of quercetin, nanoformulations, and its applications in health management, along with key findings from important clinical trial studies. Limited clinical data regarding quercetin's safety and mechanism of action are available. It is important to conduct more clinical trials to gain a deeper understanding of the disease-preventive potential, mechanisms of action, safety, and optimal therapeutic dosages. Furthermore, more research based on nanoformulations should be performed to minimize/overcome the hindrance associated with bioavailability, rapid degradation, and toxicity.

Oxidative Stress

23-Several lines of antioxidant defense against oxidative stress: antioxidant enzymes, nanomaterials with multiple enzyme-mimicking activities, and low-molecular-weight antioxidants

---

By Jomova, K (Jomova, Klaudia) [1] ; Alomar, SY (Alomar, Suliman Y.) [2] ; Alwasel, SH (Alwasel, Saleh H.) [3] ; Nepovimova, E (Nepovimova, Eugenie) [4] ; Kuca, K (Kuca, Kamil) [4] , [5] ; Valko, M (Valko, Marian) [6] **(provided by Clarivate) Source ARCHIVES OF TOXICOLOGY Volume 98 Issue 5 Page 1323-1367 DOI 10.1007/s00204-024-03696-4 Published MAY 2024 Early Access MAR 2024 Indexed 2024-04-10 Document Type Review**

Abstract

Reactive oxygen species (ROS) and reactive nitrogen species (RNS) are well recognized for playing a dual role, since they can be either deleterious or beneficial to biological systems. An imbalance between ROS production and elimination is termed oxidative stress, a critical factor and common denominator of many chronic diseases such as cancer, cardiovascular diseases, metabolic diseases, neurological disorders (Alzheimer's and Parkinson's diseases), and other disorders. To counteract the harmful effects of ROS, organisms have evolved a complex, three-line antioxidant defense system. The first-line defense mechanism is the most efficient and involves antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx). This line of defense plays an irreplaceable role in the dismutation of superoxide radicals (O2 center dot-) and hydrogen peroxide (H2O2). The removal of superoxide radicals by SOD prevents the formation of the much more damaging peroxynitrite ONOO- (O2 center dot- + NO center dot -> ONOO-) and maintains the physiologically relevant level of nitric oxide (NO center dot), an important molecule in neurotransmission, inflammation, and vasodilation. The second-line antioxidant defense pathway involves exogenous diet-derived small-molecule antioxidants. The third-line antioxidant defense is ensured by the repair or removal of oxidized proteins and other biomolecules by a variety of enzyme systems. This review briefly discusses the endogenous (mitochondria, NADPH, xanthine oxidase (XO), Fenton reaction) and exogenous (e.g., smoking, radiation, drugs, pollution) sources of ROS (superoxide radical, hydrogen peroxide, hydroxyl radical, peroxyl radical, hypochlorous acid, peroxynitrite). Attention has been given to the first-line antioxidant defense system provided by SOD, CAT, and GPx. The chemical and molecular mechanisms of antioxidant enzymes, enzyme-related diseases (cancer, cardiovascular, lung, metabolic, and neurological diseases), and the role of enzymes (e.g., GPx4) in cellular processes such as ferroptosis are discussed. Potential therapeutic applications of enzyme mimics and recent progress in metal-based (copper, iron, cobalt, molybdenum, cerium) and nonmetal (carbon)-based nanomaterials with enzyme-like activities (nanozymes) are also discussed. Moreover, attention has been given to the mechanisms of action of low-molecular-weight antioxidants (vitamin C (ascorbate), vitamin E (alpha-tocopherol), carotenoids (e.g., beta-carotene, lycopene, lutein), flavonoids (e.g., quercetin, anthocyanins, epicatechin), and glutathione (GSH)), the activation of transcription factors such as Nrf2, and the protection against chronic diseases. Given that there is a discrepancy between preclinical and clinical studies, approaches that may result in greater pharmacological and clinical success of low-molecular-weight antioxidant therapies are also subject to discussion.

Keywords

Oxidative Stress

Author Keywords

Antioxidant enzymesLow-molecular antioxidantsROSOxidative stressChronic diseaseEnzyme mimics

Keywords Plus

EXTRACELLULAR-SUPEROXIDE DISMUTASEPEROXIDASE-LIKE ACTIVITYCATALASE-LIKE ACTIVITYDOSE VITAMIN-CHYDROGEN-PEROXIDEFREE-RADICALSLIPID-PEROXIDATIONCANCER PREVENTIONALPHA-TOCOPHEROLPROSTATE-CANCER