Oxidative Stress

1- Oxidative Stress in Cancer

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Abstract

Contingent upon concentration, reactive oxygen species (ROS) influence cancer evolution in apparently contradictory ways, either initiating/stimulating tumorigenesis and supporting transformation/proliferation of cancer cells or causing cell death. To accommodate high ROS levels, tumor cells modify sulfur-based metabolism, NADPH generation, and the activity of antioxidant transcription factors. During initiation, genetic changes enable cell survival under high ROS levels by activating antioxidant transcription factors or increasing NADPH via the pentose phosphate pathway (PPP). During progression and metastasis, tumor cells adapt to oxidative stress by increasing NADPH in various ways, including activation of AMPK, the PPP, and reductive glutamine and folate metabolism.

Keywords
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NF-KAPPA-BPENTOSE-PHOSPHATE PATHWAYREGULATORY T-CELLSDOUBLE-EDGED-SWORDNRF2 ACTIVATIONHEPATOCELLULAR-CARCINOMAHYDROGEN-PEROXIDEREDOX HOMEOSTASISREDUCTIVE CARBOXYLATIONMESENCHYMAL TRANSITION

2- Molecular mechanisms of oxidative stress in asthma

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Abstract

The lungs are exposed to reactive oxygen species oxygen (ROS) produced as a result of inhalation of oxygen, as well as smoke and other air pollutants. Cell metabolism and the NADPH oxidases (Nox) generate low levels of intracellular ROS that act as signal transduction mediators by inducing oxidative modifications of histones, enzymes and transcription factors. Redox signalling is also regulated by localised production and sensing of ROS in mitochondria, the endoplasmic reticulum (ER) and inside the nucleus. Intracellular ROS are maintained at low levels through the action of a battery of enzymatic and non-enzymatic antioxidants. Asthma is a heterogeneous airway inflammatory disease with different immune endotypes; these include atopic or non-atopic Th2 type immune response associated with eosinophilia, or a non-Th2 response associated with neutrophilia. Airway remodelling and hyper-responsiveness accompany the inflammatory response in asthma. Over-production of ROS resulting from infiltrating immune cells, particularly eosinophils and neutrophils, and a concomitant impairment of antioxidant responses lead to development of oxidative stress in asthma. Oxidative stress is augmented in severe asthma and during exacerbations, as well as by air pollution and obesity, and causes oxidative damage of tissues promoting airway inflammation and hyper-responsiveness. Furthermore, deregulated Nox activity, mitochondrial dysfunction, ER stress and/or oxidative DNA damage, resulting from exposure to irritants, inflammatory mediators or obesity, may lead to redox-dependent changes in cell signalling. ROS play a central role in airway epithelium-mediated sensing, development of innate and adaptive

immune responses, and airway remodelling and hyperresponsiveness. Nonetheless, antioxidant compounds have proven clinically ineffective as therapeutic agents for asthma, partly due to issues with stability and in vivo metabolism of these compounds. The compartmentalised nature of ROS production and sensing, and the role of ROS in homeostatic responses and in the action of corticosteroids and beta 2-adrenergic receptor agonists, adds another layer of complexity to antioxidant therapy development. Nox inhibitors and mitochondrial-targeted antioxidants are in clinical development for a number of diseases but they have not yet been investigated in asthma. A better understanding of the complex role of ROS in the pathogenesis of asthma will highlight new opportunities for more targeted and effective redox therapies.

Keywords
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AsthmaOxidative stressRedox signallingNADPH oxidaseMitochondriaAir pollution
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EXHALED BREATH CONDENSATEGROWTH-FACTOR-BETAENDOPLASMIC-RETICULUM STRESSNF-KAPPA-BEXTRACELLULAR-SUPEROXIDE DISMUTASEINDUCED AIRWAY HYPERRESPONSIVENESSOBSTRUCTIVE PULMONARY-DISEASEARYL-HYDROCARBON RECEPTORMUC5AC MUCIN EXPRESSIONSERUM URIC-ACID

4- Mitochondrial Dysfunction and Oxidative Stress in Alzheimer's Disease

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Abstract

Mitochondria play a pivotal role in bioenergetics and respiratory functions, which are essential for the numerous biochemical processes underpinning cell viability. Mitochondrial morphology changes rapidly in response to external insults and changes in metabolic status via fission and fusion processes (so-called mitochondrial dynamics) that maintain mitochondrial quality and homeostasis. Damaged mitochondria are removed by a process known as mitophagy, which involves their degradation by a specific autophagosomal pathway. Over the last few years, remarkable efforts have been made to investigate the impact on the pathogenesis of Alzheimer's disease (AD) of various forms of mitochondrial dysfunction, such as excessive reactive oxygen species (ROS) production, mitochondrial Ca2+ dyshomeostasis, loss of ATP, and defects in mitochondrial dynamics and transport, and mitophagy. Recent research suggests that restoration of mitochondrial function by physical exercise, an antioxidant diet, or therapeutic approaches can delay the onset and slow the progression of AD. In this review, we focus on recent progress that highlights the crucial role of alterations in mitochondrial function and oxidative stress in the pathogenesis of AD, emphasizing a framework of existing and potential therapeutic approaches.

Keywords
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mitochondriaoxidative stressfissionfusionmitophagyAlzheimer’s disease
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AMYLOID-BETA-PEPTIDEMILD COGNITIVE IMPAIRMENTAXONAL-TRANSPORTLIPID-PEROXIDATIONPROTEOMIC IDENTIFICATIONBRAIN PROTEINSMITOFUSIN 2COENZYME-Q10 SUPPLEMENTATIONPERMEABILITY TRANSITIONMOLECULAR-MECHANISMS

5- Oxidative Stress and Antioxidant Defense in Fish: The Implications of Probiotic, Prebiotic, and Synbiotics

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Abstract

In fish, like other organisms, the lack of balance between the production of reactive oxygen species (ROS) and antioxidant defense system (so-called oxidative stress) can cause DNA hydroxylation, protein denaturation, lipid peroxidation, apoptosis, and ultimately cell damage. To improve the antioxidant defense capability, different approaches such as the administration of synthetic antioxidants were practiced. During the past years, dietary approaches have been suggested as a promising way of increasing the antioxidant defense activity in different fish and shellfish species. Among them, microbial feed additives (including pre-, pro-, and synbiotics) showed promising effects in terms of affecting antioxidant enzymes activities. Their mechanism of action in influencing the antioxidant system is not fully understood. The present review briefly discussed the antioxidant defense activity in fish, influencing factors with special focus on dietary approaches and microbial feed additives. In addition, the proposed mechanism of action of microbial feed additives on the antioxidant system has been discussed.

Keywords
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6- Lifestyle, Oxidative Stress, and Antioxidants: Back and Forth in the Pathophysiology of Chronic Diseases

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Abstract

Oxidative stress plays an essential role in the pathogenesis of chronic diseases such as cardiovascular diseases, diabetes, neurodegenerative diseases, and cancer. Long term exposure to increased levels of pro-oxidant factors can cause structural defects at a mitochondrial DNA level, as well as functional alteration of several enzymes and cellular structures leading to aberrations in gene expression. The modern lifestyle associated with processed food, exposure to a wide range of chemicals and lack of exercise plays an important role in oxidative stress induction. However, the use of medicinal plants with antioxidant properties has been exploited for their ability to treat or prevent several human pathologies in which oxidative stress seems to be one of the causes. In this review we discuss the diseases in which oxidative stress is one of the triggers and the plant-derived antioxidant compounds with their mechanisms of antioxidant defenses that can help in the prevention of these diseases. Finally, both the beneficial and detrimental effects of antioxidant molecules that are used to reduce oxidative stress in several human conditions are discussed.

Keywords
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reactive oxygen speciesoxidative stressnatural antioxidantsneurological disorderscardiovascular diseasescanceragingantioxidant defense

Oxidative Stress

Keywords Plus

FREE-RADICALSHEAVY-METALSDNA-DAMAGENEURODEGENERATIVE DISEASESCOMPREHENSIVE EVALUATIONXANTHINE OXIDOREDUCTASECARDIOVASCULAR-DISEASENEUTROPHIL ACTIVATIONMOLECULAR-MECHANISMSREDOX REGULATION

7- Exposure to imidacloprid induce oxidative stress, mitochondrial dysfunction, inflammation, apoptosis and mitophagy via NF-kappaB/JNK pathway in grass carp hepatocytes

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Abstract

Imidacloprid (IMI) is a neonicotinoid compound widely used in agriculture production, causing surface water pollution and threatening non-target organisms. The aim of this study was to analyze the effects of IMI on grass carp (Ctenopharyngodon idellus) liver cell (L8824) injury. The L8824 cells were exposed to different doses of IMI (65 mg/L, 130 mg/L and 260 mg/L) for 24 h. Our results demonstrated that exposure IMI significantly sup-pressed the activity of anti-oxidant enzymes (SOD, CAT and T-AOC) and accumulated oxidase (MDA) levels, and promoting reactive oxygen species (ROS) generation in L8824 cells. Additionally, mitochondrial membrane potential (Delta psi m), mitochondria-derived ROS and ATP content and the MitoTracker Green indicated that IMI aggravated mitochondrial dysfunction, thereby inducing inflammation and enhancing pro-inflammatory genes (NF-kappaB, TNF alpha, IL-1 beta and IL-6) expressions. However, the addition of 2 mM N-acetyl-L-cysteine (NAC) can reverse these adverse effects of high-dose IMI-induced. Hence, ROS is the main factor of IMI-induced mito-chondrial dysfunction and inflammation. We further found that exposure to IMI induced apoptosis, which is characterized by promoting release of cytochrome c (Cyt-C), and increasing the expression of Bcl-2-Associated X (BAX), cysteinyl aspartate specific proteinases (Caspase 9 and 3), decreasing Bcl-2 level. Immunofluorescent staining, qRT-PCR and Western Blot results indicated that IMI exposure also activated mitophagy, which was demonstrated by the expression of mitophagy-related genes (BNIP3, LC3B and P62). Conversely, scavenging JNK by SP600125(10 mu M) alleviated the expression of mitochondrial apoptosis and

mitophagy-related gene induced by high-dose IMI. Therefore, these results of study demonstrated that IMI-induced oxidative stress to regulate mitochondrial dysfunction, thus causing inflammation, mitochondrial apoptosis and mitophagy in grass carp hepatocytes through NF-kappaB/JNK pathway.

Keywords
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Imidaclopridoxidative stressmitochondrial dysfunctionmitochondrial apoptosismitophagyNF-kappaB/JNK
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PERMEABILITY TRANSITIONSIGNALING PATHWAYINDUCED AUTOPHAGYDNA-DAMAGETNF-ALPHAJNKEXPRESSIONBNIP3INSECTICIDEHIPPOCAMPUS

8- Cadmium exposure triggers oxidative stress, necroptosis, Th1/Th2 imbalance and promotes inflammation through the TNF-alpha/NF-Kappa B pathway in swine small intestine

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Abstract

Cadmium (Cd) is a toxic environmental pollutant and induces toxic effects to organism. Nevertheless, the mechanism of Cd-induced toxicity in swine remains obscure. To explore this, 10 healthy 6-week-old weaned swine were placed into two groups stochastically, the Cd group was treated with a commercial diet containing 20 mg/kg Cd for 40 days. The results of histopathological and ultrastructural observations showed typical necrosis features and inflammatory cell infiltration in Cd group. Excessive Cd suppressed T-AOC and SOD activities, increased MDA content and ROS levels. Cd diet elevated the expression of RIPK1, RIPK3, and MLKL to activate the RIPK3-dependent necroptosis pathway. Results of Th1 and Th2 cytokines indicated that the levels of IL-4, IL6 and IL10 was increased, while the level of IFN-gamma was decreased, illustrating Th1/Th2 immune imbalance leads to aggravate inflammatory responses. Cd activated the TNF-alpha/NF-kappa B pathway and induced inflammatory responses via increasing the expression of HO-1, IL-1 beta, iNOS, COX2. Heat shock proteins were notably elevated in response to inflammatory reactions. And these effects were inhibited by necrostatin-1 (Nec-1) and N-acetylcysteine (NAC). Altogether, these data demonstrated that Cd induced necroptosis and inflammation to aggravate small intestine injury in swine by increasing the excessive accumulation of ROS and imbalanced Th1/Th2, respectively.

Keywords

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