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Reactive oxygen species

The reactive oxygen species are the contributors of oxidative stress which lead to various diseases and disorders such as cardiovascular disease, cancer, aging, and various neurodegenerative diseases [14].

From: Toxicological Survey of African Medicinal Plants, 2014

Related terms:
Macrophages, Endothelium, Enzymes, Apoptosis, Antioxidants, Hydrogen peroxide, Cytokines, Mitochondrion, Oxidative stress, Neutrophil granulocyte

Learn more about Reactive oxygen species

Important Steps in Microbial Pa

Joshua Fierer, ... Jean-Claude Pechère†, in 2017

Inactivation of Reactive Oxygen Species
Reactive oxygen species damage DNA and phosphorylation. Bacteria may escape from reactive oxygen species by rapid detoxifica efficient DNA repair. Several bacterial path (SOD) and catalase, two enzymes that deg also express Rec-A enzymes to repair dam pathway is critical, as *rec*A mutants are avi

Oxidative Stress and Cardiac Muscle

Yasuhiro Maejima, ... Junichi Sadoshima, in Muscle, 2012

Definition of ROS
Reactive oxygen species (ROS) are derivatives of oxygen that are more reactive than molecular oxygen. A primary ROS is superoxide (O2–), which is formed by one-electron reduction of molecular oxygen.

the endocytic pathway of the host cell see of resistance to reactive oxygen species. Ir localize in phagosomes devoid of NADPH respiratory burst.[73]

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Hydrogen peroxide (H_2O_2) is produced by reduction of O2– through dismutation. Hydroxyl radical (OH^-) arises from electron exchange between O2– and H_2O_2 via the Harber–Weiss reaction or it is also generated by the reduction of H_2O_2 by the Fenton reaction. When generated under strictly regulated conditions, these ROS, in particular O2– and H_2O_2, may act as signaling molecules that mediate physiological processes, such as cell

Free Radical Damage and its Cardiac Metabolism in Health and Disease

Lionel H. Opie, in Cellular and Molecular Pathobiology of Cardiovascular Disease, 2014

Reactive Oxygen Species

Reactive oxygen species (ROS, also called oxygen free radicals) are a side-product of sites on mitochondrial complexes I and III of the electron transmitter chain (see later in text). In excess, ROS contribute to membrane damage by lipid peroxide formation and are part of the signaling sequence leading to apoptosis. Excess ROS are also derived from many complex sources besides damaged myocyte mitochondria, such as from uncoupled nitric oxide synthase in heart and endothelial cells, from xanthine oxidase and stimulation of membrane NADPH oxidase (by angiotensin II, endothelin, cytokines) and from neutrophils.[24] Excess ROS are formed particularly during the reperfusion phase of ischemic damage.[25]

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The rabbit heart contains only trace amounts of the enzyme xanthine

oxidase (as does the human heart) [37,38]. Therefore, this enzyme probably does not play a significant role in the generation of superoxide

Reactive Oxygen Species, Oxidative Stress, and Vascular Biology in Hypertension

Fatiha Tabet, Rhian M. Touyz, in Comprehensive Hypertension, 2007

SUMMARY

Reactive oxygen species are produced in the vasculature in a controlled and tightly regulated manner. Superoxide and H_2O_2 have important signaling properties, mainly through oxidative modification of proteins and activation of transcription factors that regulate vascular function. In such as NAD(P)H oxidase, NOS, xanthine SOD, altered thioredoxin and glutathione antioxidants, results in increased formatic on the vasculature. Factors that activate p poorly defined, but probably involve Ang cytokines.

Reactive oxygen species in hypertension c cell growth, extracellular matrix protein d metalloproteinases, inflammation, endoth tone. In experimental hypertension oxidat levels/activity is decreased. Clinical data ir oxidative excess. Although inconclusive at ROS bioavailability by decreasing product scavenging, may regress vascular remode reduce blood pressure in hypertensive pat can diffuse from vascular to parenchymal vascular oxidative excess contributes to ta of ROS formation will lead to end-organ

MICROCIRCULATION IN CHRONIC VENOUS INSUFFICIENCY

KEVIN BURNAND, SAID ABISI, in Venous Ulcers, 2007

OXIDATIVE STRESS IN VENOUS ULCERS

Reactive oxygen species are present in the ulcer tissue and exudate. Their release is probably mediated by inflammatory cells, fibroblasts, and endothelial cells, which are known to produce superoxidants. These cells are commonly present in chronic venous leg ulcers' environs, leading to hostile oxidative stress and consequent tissue destruction.[29]

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Oxidative stress is dependent on the presence of free ion radicals such as iron in the tissue around venous ulcers. Increased iron deposition may cause an elevation of toxic free radicals in venous ulcers' environs. Iron is toxic

The Pathophysiology of Cardiac Hypertrophy and Heart Failure

William E. Stansfield MD[1], ... Monte S. Willis MD, PhD[1], in Cellular and Molecular Pathobiology of Cardiovascular Disease, 2014

The GPIb-IX-V Complex

Robert K. Andrews[1], Michael C. Berndt[2], in Platelets (Third Edition), 2013

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TRAF4

Reactive oxygen species (ROS) are generated in response to ligand engagement of platelet GPIb-IX-V and GPVI. The orphan tumor necrosis factor (TNF) receptor-associated factor (TRAF) family member, TRAF4, selectively binds cytoplasmic sequences of GPIbβ and GPVI proximal to or overlapping the calmodulin-binding sequences (Section III.A.2) and may provide the link between redox pathways and early platelet signaling events, as TRAF4 is a binding partner for $p47^{phox}$ of the NADPH oxidase (Nox2) complex and is also involved in the assembly of other redox-relevant signaling proteins such as Hic-5 and Pyk2.[160] Elevated ROS could potentially enhance the prothrombotic state associated with cardiovascular and metabolic disorders and help explain the protective effect of dietary antioxidants in thrombotic diseases that is suggested by epidemiological studies.

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Redox and Cancer Part A

Daohong Zhou*[1], ... Douglas R. Spitz†[1], in Advances in Cancer Research, 2014

Abstract

stem cells for tumor progression and treatment are also discussed.

Reactive Oxygen Species and Oxidative Stress

Rhian M. Touyz, in Primer on the Autonomic Nervous System (Third Edition), 2012

Reactive oxygen species (ROS), intermediates of reduction–oxidation reactions, influence many physiological processes including host defense, hormone biosynthesis, fertilization, aging and cellular signaling. Increased ROS production (termed oxidative stress) has been implicated in various pathologies, including cardiovascular disease, cancer and neurological disorders. In the nervous system ROS have multiple actions in that they regulate neural activity and themselves act as neurotransmitters (gasotransmitters). ROS are generated throughout the brain and play an important role in activation of the sympathetic nervous system. Increased sympathetic excitation and diminished parasympathetic suppression of heart rate, cardiac contractility and vascular tone are associated with cardiovascular diseases such as hypertension and ischemic heart disease. Mechanisms responsible for regulating sympathovagal balance remain unclear, but superoxide (O_2^-) and hydrogen peroxide (H_2O_2) may be important. The present review discusses the biology of ROS and highlights the pathophysiological importance of ROS and oxidative stress in the autonomic nervous system.

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