


# Chapter 2


## Antioxidant Defense Mechanisms: Enzymatic and Non-Enzymatic

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### ABSTRACT

*Reactive oxygen species (ROS) are highly reactive molecules generated as a by-product of normal metabolism and through exposure to external factors, such as radiation, pollutants, and certain medications. Overproduction of ROS leads to oxidative stress. Stress can damage vital biomolecules like DNA, proteins, and lipids, leading to chronic diseases. This chapter discusses the response of cells to oxidative stress. Roles and regulatory mechanisms of superoxide dismutase, catalase, the glutathione system, peroxiredoxin, and thioredoxin, in ROS defense have been discussed. The benefits of non-enzymatic antioxidants such as vitamins C and E, and minerals like selenium, and zinc have been highlighted. The synergistic interactions between enzymatic and non-enzymatic components have been detailed. This chapter highlights the potential of antioxidants to prevent chronic diseases, such as cardiovascular disorders, cancer, and neurodegeneration. The chapter concludes by discussing the intricacies of antioxidant supplementation, including challenges*

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*and future directions.*

## 1. INTRODUCTION TO OXIDATIVE STRESS

Oxidative stress is a state of imbalance between the generation of free radicals and antioxidant defense mechanisms of the body (Betteridge, 2000). Free radicals are atoms or molecules with unpaired electrons, and this characteristic makes them highly reactive (Shields et al. 2021). Due to their high reactivity, they can accept electrons from other compounds to become stable. As a result, the attacked molecule loses an electron and becomes a free radical, triggering a cascade of events that eventually harm the living cell. While free radicals can be of various types, this chapter will specifically focus on reactive oxygen species (ROS) and their implications in biological systems. Free radicals derived from oxygen are byproducts of metabolic reactions (Phaniendra et al. 2015). ROS refers to oxygen-containing reactive species like superoxide ( $O_2^{\bullet-}$ ), hydrogen peroxide ( $H_2O_2$ ), hydroxyl radical ( $OH^{\bullet}$ ), singlet oxygen ( $^1O_2$ ), peroxy radical ( $LOO^{\bullet}$ ), alkoxy radical ( $LO^{\bullet}$ ), lipid hydroperoxide (LOOH), peroxynitrite ( $ONOO^-$ ), hypochlorous acid (HOCl), and ozone ( $O_3$ ) (Li et al. 2016). Cellular ROS can be produced endogenously in the mitochondria, the intracellular site for oxidative phosphorylation. Other organelles associated with ROS production are the endoplasmic reticulum, where misfolded protein aggregation causes 'ER stress' and subsequently increases ROS, and the peroxisome, which contains several peroxisomal enzymes involved in the generation and metabolism of ROS (Hong et al. 2024; Phaniendra et al. 2015). Additionally, by producing ROS, metabolic enzymes like NADPH oxidases (NOX), nitric oxide synthases, lipoxygenases, xanthine oxidases, and cyclooxygenases can contribute to oxidative stress (Hong et al. 2024; Villalpando-Rodriguez & Gibson, 2021). Extracellular factors that generate ROS include UV radiation, industrial solvents, pollution, alcohol, tobacco smoke, ionizing radiations, chronic infections, inflammatory disorders, and nonsteroidal anti-inflammatory drugs (Bhattacharyya et al. 2014; Phaniendra et al. 2015). At moderate or low levels, ROS can be favorable by involving in several physiological activities like killing invading pathogens, wound healing, tissue repair, cell signaling pathways, mitogenic responses, and redox regulation (Bhattacharyya et al. 2014; Phaniendra et al. 2015). On the other hand, an accumulation of ROS in the cell can cause RNA and DNA damage, membrane phospholipid peroxidation, and protein oxidation, which can alter these biomolecules beyond repair and cease their function (Juan et al. 2021). Free radical-induced oxidative stress has been linked to several diseases (Figure 1) like cancers (lung, breast, prostate, colorectal, and bladder), cataract development, premature aging, neurodegenerative disorders (Parkinson's disease, Alzheimer's disease, and multiple sclerosis), rheumatoid ar-

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