# Chapter 9
# Oxidative Stress in Cardiovascular Disorders: Mechanisms and Therapeutic Implications

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## ABSTRACT

*Cardiovascular disorders (CVDs) include atherosclerosis, cardiac failure, hypertension, myocardial infarction, and oxidative stress, which are increasingly recognized as essential factors for the onset and advancement of these conditions. he imbalance between the formation of reactive oxygen species (ROS) and the antioxidant defense system is the molecular basis of oxidative stress, which leads to oxidative damage to lipids, proteins, and DNA. This potential damage promotes inflammatory responses, disrupts endothelial function, and speeds up atherogenesis. The nitric oxide synthase uncoupling, mitochondrial dysfunction, and NADPH-oxidizing enzymes are important pathways that enhance ROS production. Antioxidants including polyphenols,*

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*vitamins E and C, and others have shown conflicting outcomes in clinical studies. These novel methods aim to improve natural antioxidant defenses, alter redox-sensitive signaling pathways, and target specific ROS sources.*

## 1. INTRODUCTION

Oxidative stress arises when ROS generation exceeds antioxidant neutralization. (Das & Roychoudhury, 2014) This state is crucial to the genesis and progression of many illnesses, including cardiovascular, neurodegenerative, and cancer. Both endogenous and external sources create reactive oxygen species like hydroxyl radicals, hydrogen peroxide, and superoxide anion. Peroxisomes, NADPH oxidase along with xanthine oxidase activities contribute to ROS, although mitochondria are the main generators, especially via the electron transport chain.(Bhattacharyya et al., 2014; Dumanović et al., 2021; Poljsak et al., 2013a) Environmental contaminants, cigarette smoke, radiation, UV light, and chemicals are exogenous.(Dumanović et al., 2021; Poljsak et al., 2013a) ROS are needed for signaling and pathogen defense, but excessive buildup may damage DNA, proteins, and lipids. The body uses enzymatic as well as non-enzymatic antioxidant defenses to combat ROS. ROS are converted into water and oxygen by enzymatic antioxidants such SOD, glutathione peroxidase, and catalase. Antioxidants include vitamins C and E, and glutathione scavenge free radicals. Maintaining redox homeostasis requires an equilibrium between the production of ROS and antioxidant defenses to avoid oxidative stress and cellular malfunction.(Das & Roychoudhury, 2014; He et al., 2017) When ROS levels rise, key regulatory mechanisms including the Nrf2 signaling route upregulate antioxidant gene expression for protection of organisms from damage caused by oxidation. Oxidative stress overwhelms the body's defenses, causing cellular and systemic damage. Oxidative stress causes lipid peroxidation, which damages membranes and causes apoptosis. Protein oxidation affects enzyme performance, whereas DNA oxidation may cause mutations and cancer.(Poljsak et al., 2013b) Systemic oxidative stress is mainly associated with cardiovascular disorders like atherosclerosis, hypertension, and myocardial infarction. Neurodegenerative illnesses like Alzheimer's and Parkinson's depend on it for neuronal cell death. The vicious circle of prolonged oxidative stress along with inflammatory responses worsens disease development. Denham Harman's 1950s “free radical theory of aging,” relating free radicals to aging and age-related disorders, changed the notion of oxidative stress. Over the decades, studies have linked oxidative stress to cardiovascular disease, cancer, and neurodegeneration. The antioxidants discovery and endogenous antioxidant systems have helped us understand how oxidative stress causes illness and create treatments to combat it. Today, researchers are studying the molecular underpinnings of oxida-