


Chapter 14

ROS in Precision Medicine in Neurological Diseases: Personalized Approaches


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ABSTRACT

Reactive oxygen species (ROS), are a class of unstable chemicals produced by almost every subcellular organelle in cell. The ROS are highly reactive molecules that play essential roles in cell signaling and homeostasis but can also cause damage to cellular components when present in excess. The elevated levels of ROS, are causing damage to DNA, proteins, and lipids, promoting genetic instability, number of neurological illnesses, including, Alzheimer's, and Parkinson's disease. Precision medicine is an innovative approach to adapting medical treatment to the

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individual characteristics of each patient. This involves treating every patient as an individual and focusing on alternative healthcare treatment to every patient's unique characteristic. It relies on understanding how a person's genetic makeup, environment, and lifestyle affect their health and disease. The precision medicine enables early detection, prognostication, and monitoring of disease progression, allowing for timely and tailored interventions to mitigate ROS-mediated damage and improve patient outcomes.

1. INTRODUCTION:

Reactive oxygen species (ROS) may have initially appeared on Earth between 2400 to 3800 million years ago, along with the appearance of the first atmospheric oxygen molecules. From then on, ROS have become an essential part of life when oxygen is involved, (Mittler, 2017). ROS have gained attention as unique signal mediators that are involved in the progress, differentiation, development, and death of cells, (Sena & Chandel, 2012) (Zhang *et al.*, 2013). ROS are a class of unstable chemicals produced by almost every subcellular organelle in cell, (Sun *et al.*, 2020). These compounds include hydrogen peroxide (H_2O_2), singlet oxygen ($^1\text{O}_2$), hydroxyl radical (OH^\bullet), and superoxide (O_2^-) (5).

Production of ROS is carried out by a range of extracellular and intracellular processes, (Sena & Chandel, 2012) (Zhang *et al.*, 2013). In intracellular places such as complexes I and III of electron transport chain (ETC) produces ROS in the inner mitochondrial membrane, (Holmström KM, Finkel, 2014) (Quinlan *et al.*, 2013). Overall, in Complex I, 0.1–2% of ETC electrons leak to generate ROS, and this fraction rises as damage increases, (Li *et al.*, 2013). Oxygen free radicals are extremely flammable and can cause destruction of lipids (in membrane), proteins, and nucleic acids (DNA, RNA), between other biological components, (Alfadda & Sallam, 2012) (Bae *et al.*, 2011) (Dröge, 2002). According to reports, oxidative stress (OS), or an ROS, might eventually cause cell death, (Liu *et al.*, 2018). In order to prevent specific potentially harmful effects of ROS, the cells maintain ROS at a harmless level via numerous anti-oxidative processes. Unnecessary ROS is removed enzymatically by superoxide dismutase (SOD), superoxide reductase, catalase, glutathione peroxidase (GPX), glutathione reductase, peroxiredoxin (PRX) and thioredoxin (TRX), (Afonso *et al.*, 2007) (Moloney & Cotter, 2018), or scavenged by various antioxidants, (Oyewole & Birch-Machin, 2015). Any perturbation from the ROS equilibrium may trigger ROS signalling events, (Foyer & Noctor, 2013) (Nigolet-Spruyt *et al.*, 2016) (Mittler *et al.*, 2004). This imbalance can lead to a number of pathological illnesses, including diabetes, cancer, aging, atherosclerosis, Alzheimer's disease, and Parkinson's disease, (Guglielmotto, Tamagno, & Danni,

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