

# Impact of Oxidative Stress on Aging and Age-Related Degeneration

# Andrea Sanders<sup>\*</sup>

Department of Molecular Biology & Genetics, Johns Hopkins University, Baltimore, United States of America

# DESCRIPTION

Aging is a multifaceted process influenced by a combination of genetic, environmental and metabolic factors. Among the leading contributors to aging and age-related degeneration is oxidative stress, a biological condition where the production of harmful molecules called Reactive Oxygen Species (ROS) overwhelms the body's ability to neutralize those using antioxidants. Over time, oxidative stress damages cellular components such as DNA, proteins and lipids, leading to a decline in cellular function, tissue degeneration and the onset of age-related diseases. Understanding the role of oxidative stress in aging is important for exploring interventions that may slow down or even reverse the aging process.

### What is oxidative stress?

Oxidative stress refers to an imbalance between the production of ROS and the body's ability to counteract their harmful effects through antioxidants. ROS, also known as free radicals, are highly reactive molecules containing oxygen. While ROS are a natural by-product of cellular metabolism especially during the process of energy production in the mitochondria their excessive accumulation can cause significant damage to cells and tissues [1].

#### The role of oxidative stress in aging

Oxidative stress plays a pivotal role in aging through its direct impact on cellular structures and functions. As ROS accumulate, they cause damage to critical cellular components, including DNA, proteins and lipids, which impairs the ability of cells to function properly. Over time, this damage leads to cellular dysfunction, tissue degeneration and the progressive decline in physiological function associated with aging [2,3].

# DNA damage and cellular senescence

ROS can cause damage to DNA by inducing mutations, strand breaks and cross-linking. DNA damage that is not adequately repaired can lead to the activation of pathways that induce cellular senescence, a state in which cells stop dividing and lose their ability to function optimally. Senescent cells accumulate in tissues as we age, releasing pro-inflammatory molecules that contribute to tissue dysfunction and chronic inflammation, a phenomenon known as inflammation. Senescent cells also lose their regenerative capacity, reducing the body's ability to repair damaged tissues, which contributes to the physical decline seen in aging [4].

# Mitochondrial dysfunction

Mitochondria, the energy-producing organelles in cells, are a major source of ROS production. As cells age, mitochondrial function declines, leading to increased ROS production and reduced energy output. Mitochondrial DNA (mtDNA) is particularly susceptible to oxidative damage due to its proximity to ROS production sites and its limited repair mechanisms. Damaged mitochondria further exacerbate ROS production, creating a vicious cycle of oxidative damage, mitochondrial dysfunction and cellular aging. This mitochondrial dysfunction is a key factor in the loss of cellular energy production and is linked to conditions such as muscle weakness, neurodegeneration and cardiovascular disease in older adults [5].

# Lipid peroxidation and membrane damage

ROS can also attack lipids in cellular membranes, leading to a process known as lipid peroxidation. This results in the breakdown of lipid molecules, which compromises the integrity of cell membranes, leading to cellular dysfunction and death. Lipid peroxidation is particularly damaging to neurons, which are highly dependent on membrane integrity for proper signaling. As a result, oxidative stress-induced lipid peroxidation has been implicated in neurodegenerative disorders, including Alzheimer's disease and Amyotrophic Lateral Sclerosis (ALS) [6].

# Oxidative stress and age-related diseases

The cumulative effects of oxidative stress contribute significantly to the onset and progression of various age-related diseases.

Correspondence to: Andrea Sanders, Department of Molecular Biology & Genetics, Johns Hopkins University, Baltimore, United States of America, E-mail: sanders26@gmail.com

**Received:** 27-Nov-2024, Manuscript No. JASC-24-26955; **Editor assigned:** 29-Nov-2024, PreQC No. JASC-24-26955 (PQ); **Reviewed:** 13-Dec-2024, QC No. JASC-24-26955; **Revised:** 20-Dec-2024, Manuscript No. JASC-24-26955 (R); **Published:** 26-Dec-2024, DOI: 10.35248/2329-8847.24.12.387

Citation: Sanders A (2024). Impact of Oxidative Stress on Aging and Age-Related Degeneration. J Aging Sci. 12:387.

**Copyright:** © 2024 Sanders A. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

Some of the well-documented connections between oxidative stress and age-related diseases include:

### Cardiovascular diseases

The cardiovascular system is particularly vulnerable to oxidative stress, as ROS can damage endothelial cells lining blood vessels, promoting atherosclerosis (the build-up of plaque in arteries). Oxidative stress also promotes the oxidation of Low-Density Lipoprotein (LDL) cholesterol, which is a key step in the development of atherosclerosis. Over time, oxidative damage to blood vessels can lead to hypertension, stroke and heart attacks [7].

#### Neurodegenerative disorders

Neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease and ALS are strongly linked to oxidative stress. The brain consumes a significant amount of oxygen and is particularly susceptible to oxidative damage due to its high metabolic activity. ROS-induced damage to neurons and glial cells contributes to the loss of cognitive function and motor control seen in these conditions. In Alzheimer's disease, for example, oxidative stress is believed to play a role in the accumulation of beta-amyloid plaques, which disrupt neural communication [8].

#### Diabetes

Oxidative stress is a key factor in the development of type 2 diabetes and its complications. ROS can impair insulin signalling, leading to insulin resistance, a hallmark of type 2 diabetes. Additionally, oxidative damage to pancreatic beta cells, which produce insulin, can further exacerbate glucose regulation problems. The chronic high blood sugar levels seen in diabetes also promote the production of ROS, contributing to complications such as diabetic neuropathy, retinopathy and kidney disease.

#### Cancer

Oxidative stress can induce mutations in DNA, promoting the initiation and progression of cancer. While ROS-induced DNA damage is a natural defence mechanism to trigger apoptosis in damaged cells, chronic oxidative stress can overwhelm these protective mechanisms, allowing damaged cells to proliferate uncontrollably. Moreover, tumors often experience high levels of oxidative stress, which can contribute to the development of aggressive and treatment-resistant cancer cells [9].

#### Stress management

Chronic psychological stress has been linked to increased oxidative stress. Practices such as meditation, mindfulness and yoga can help reduce stress and, in turn, lower ROS production, promoting overall health and longevity [10].

# CONCLUSION

Oxidative stress is a central player in the aging process and contributes to the onset and progression of numerous agerelated diseases. By damaging key cellular components such as DNA, proteins and lipids, oxidative stress accelerates cellular aging and tissue degeneration. Understanding the mechanisms behind oxidative stress and adopting lifestyle practices that reduce its impact such as a healthy diet, regular exercise and stress management offers potential strategies for promoting healthy aging and reducing the risk of age-related degeneration.

# REFERENCES

- Jarrett SG, Boulton ME. Consequences of oxidative stress in agerelated macular degeneration. Mol Aspects Med. 2012;33(4): 399-417.
- 2. Tan BL, Norhaizan ME, Liew WP, Sulaiman Rahman H. Antioxidant and oxidative stress: A mutual interplay in age-related diseases. Front Pharmacol. 2018;9:1162.
- Salminen A, Ojala J, Kaarniranta K, Kauppinen A. Mitochondrial dysfunction and oxidative stress activate inflammasomes: Impact on the aging process and age-related diseases. Cell Mol Life Sci. 2012;69:2999-3013.
- Yildirim Z, Ucgun NI, Yildirim F. The role of oxidative stress and antioxidants in the pathogenesis of age-related macular degeneration. Clinics (Sao Paulo). 2011;66:743-746.
- Luo J, Mills K, le Cessie S, Noordam R, van Heemst D. Ageing, age-related diseases and oxidative stress: What to do next?. Ageing Res Rev. 2020;57:100982.
- 6. Liguori I, Russo G, Curcio F, Bulli G, Aran L, Della-Morte D, et al. Oxidative stress, aging and diseases. Clinical interventions in aging. 2018:757-772.
- Migliore L, Coppedè F. Environmental-induced oxidative stress in neurodegenerative disorders and aging. Mutat Res. 2009;674(1-2): 73-84.
- Bokov A, Chaudhuri A, Richardson A. The role of oxidative damage and stress in aging. Mech Ageing Dev. 2004;125(10-11): 811-826.
- 9. Tisi A, Feligioni M, Passacantando M, Ciancaglini M, Maccarone R. The impact of oxidative stress on blood-retinal barrier physiology in age-related macular degeneration. Cells. 2021;10(1):64.
- 10. Martin I, Grotewiel MS. Oxidative damage and age-related functional declines. Mech Ageing Dev. 2006;127(5):411-423.