

Special Issue on **Oxidative Stress in Age-Related Chronic Disease: From Bench to Bedside**

CALL FOR PAPERS

Aging is an irreversible biological process that may be associated with an increased incidence of chronic diseases, involving different organs and systems. Western societies are facing the unprecedented aging of the population (silver tsunami); this increasing number of older people will also need tailored healthcare for largely chronic diseases.

The causes of aging remain largely unknown; however, the free radicals theory of aging still provides a strong conceptual framework for experimental and clinical evidence on aging.

Oxidative stress and ROS generation are important molecular mechanisms for the maintenance of homeostasis. Namely, the integrity of the intracellular mitochondrial activity and of the endogenous antioxidant enzymatic defense system plays a role in shaping the aging process, leading to cell damage. As a result, oxidative intracellular damage impacts all age-related chronic conditions and, on a cellular basis, it is intertwined with biological aging, cellular survival, and longevity as well as the onset and progression of several age-related diseases such as cardiovascular diseases, cancer, neurodegeneration, metabolism, immune function, hematological diseases, lung diseases, and kidney failure.

Despite substantial correlative evidence to support it, studies in the last years have raised doubts over its importance. In particular, the expected elevation in oxidative damage does not impact linearly on lifespan and on the progression and worsening of age-related clinical conditions. All these findings raise fundamental questions over whether the free-radical damage theory remains useful for understanding the aging process and variations in lifespan and life histories.

Growing evidence indicates a correlation between oxidative damage and frailty status, instead of the mere chronological age. Frailty is a biological syndrome characterized by higher comorbidity and disability, due to decreased biological homeostasis, which put elderly subjects at higher risk when facing minor environmental stressors.

Thus, the free radical theory of aging seems to apply to biological age rather than chronological age, driving premature aging and higher comorbidity burden.

Since a major task for medicine and for social care sciences in modern societies is to delay or even treat the onset of chronic diseases, disability, and frailty, it is of key importance to design treatments that can prevent or reverse oxidative damage.

The deeper understanding of the cellular and molecular mechanisms of oxidative stress and its factual role in the development of age-related diseases will provide potential ways to target them and turn bench-to-bedside research into clinical strategies.

In this special issue, we invite authors to submit original research as well as review articles that focus on the current understanding of oxidative stress in age-related chronic disease, with an emphasis on identification and validation of new targets and pathways for the prognostic and therapeutic application.

Potential topics include but are not limited to the following:

- ▶ Therapeutic relevance of oxidative-stress targeting in aging and age-related diseases (Alzheimer's disease and neurodegeneration; psychiatric, chronic clinical conditions including depression and bipolar disorders; osteoporosis; renal diseases and end-stage renal failure; cardiovascular diseases, including atherosclerosis and heart failure; diabetes and related microvascular and macrovascular complications)
- ▶ Novel therapeutic targets in age-associated malignancies, including epigenetics (cancer and hematological malignancies)
- ▶ Metabolic dysfunctions and frailty in age-related chronic diseases (sarcopenia and frailty; nutrition and biological aging)
- ▶ Immunomodulation as an innovative approach to managing age-related chronic diseases

Authors can submit their manuscripts through the Manuscript Tracking System at <https://mts.hindawi.com/submit/journals/omcl/osacd/>.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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