Aging is a complex process, characterized by a gradual decrease in physiological functions that is often accompanied by many illnesses. Mitochondria are considered to have a significant influence on aging due to their critical role in the regulation of bioenergetics, oxidative stress, and cell death. Numerous studies have shown that mitochondrial bioenergetic deterioration is an important factor in aging and age-related disorders (age-related macular degeneration, Parkinson's diseases, Alzheimer's disease, etc.). One of the causes of aging is the cumulative damage from reactive oxygen species (ROS) to mitochondrial DNA which reduces the maintenance of adequate energy supply during aging that is required for cellular repair, homeostasis mechanisms (proteostasis, mitophagy, etc.), and mitochondrial biogenesis.

Additionally, the evidence from recent studies highlights the importance of nicotinamide adenine dinucleotide (NAD) metabolism in aging. NAD+ is a coenzyme that participates in various energy metabolism pathways including glycolysis, -oxidation, and oxidative phosphorylation and is a required cofactor for important enzymes such as poly(ADP-ribose) polymerases (PARPs) and sirtuins. Age-associated decline in NAD+ levels has been reported in various tissues and cells. Therapies (nicotinamide mononucleotide, nicotinamide ribose, etc.) that increase NAD+ levels have been shown to delay cellular aging process. Furthermore, epigenetic modification of genetic materials has been shown to greatly influence the oxidative stress and mitochondrial dysfunction during aging. The efforts towards minimizing oxidative stress and maintaining mitochondrial energy metabolism for productive aging are at the forefront of current aging research, and newer aspects (mechanisms and therapeutic options) are continuously emerging.

We invite investigators to submit preclinical and clinical original research and review articles encompassing various aspects of mitochondrial oxidative stress and bioenergetics in regulating longevity.

Potential topics include but are not limited to the following:

- The implications of altered NAD+ metabolism in aging
- Altered mitochondrial bioenergetics in aging
- Role of mitochondrial oxidative stress and biogenesis in cellular senescence and longevity
- Epigenetic regulation of mitochondrial biogenesis and oxidative metabolism

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

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