Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Molecular Mechanisms Underlying Age-Related Ocular Diseases

CALL FOR PAPERS

In industrialised countries, the elderly population is predicted to increase from 25% to 40% by 2030. As a consequence, the functional decline associated with the ageing people creates a challenge to be met by the national healthcare systems. Among the age-related ocular pathologies, the WHO has identified some irreversible diseases, like age-related macular degeneration, diabetic retinopathy, and glaucoma as priority eye diseases, in terms of prevention of visual impairment and blindness. The lack of effective therapies for the prevention or cure of the diseases is mainly due to the insufficient understanding of these diseases at the molecular and cellular levels. In addition, novel cellular and animal models are needed to better understand the pathogenesis of age-related ocular diseases and to identify new biomarkers to predict the severity of these pathologies or the responsiveness to therapies.

Besides the aforementioned ocular diseases, ageing is a risk factor also for cataracts, retinal vein occlusion, ischemic optic neuropathy, corneal degeneration, and dry eye. Among the known factors inducing ageing and neurodegeneration, oxidative stress, inflammation, and impairment in protein clearance play a key role.

The purpose of this special issue is to publish high-quality research papers as well as review articles that shed light on the molecular and cellular responses to oxidative stress underlying the onset or progression of age-related ocular pathologies, thus helping to translate pioneering "bench to bedside" research into effective clinical strategies. Original high-quality contributions that are not yet published and are not currently under review by other journals or peer-reviewed conferences are sought. Ageing affects genetics, epigenetics, and biochemical and cellular pathways that regulate lifespan.

Potential topics include but are not limited to the following:

- Molecular and cellular mechanisms, proteins, and signalling cascades linked to inflammation, neurodegeneration, oxidative stress response, and cellular repair
- Changes in the gene expression or function of enzymes/structural proteins with a physiological role in the retina, optic nerve, or other ocular tissues
- Changes in mechanisms controlling gene expression at transcriptional or posttranscriptional level (including microRNAs)
- Alterations of protein folding or protein degradation pathways responsible for intracellular/extracellular deposits
- Alterations in the content or signalling cascade of growth factors (VEGF, BDNF, PlGF, etc.), with special interest in those endowed with a neuroprotective role
- Dysfunctions of mitochondria or other cellular organelles leading to derangement of essential biochemical processes in neurons
- Alterations of proteins/signalling controlling apoptosis, differentiation, and proliferation
- Molecules acting on the above targets that may be of pharmacological interest for ocular diseases, including those endowed with neuroprotective effects and/or targeting longevity pathways

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

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

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