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Animal Models: Explore the Oxidative Stress Accumulation, Mitochondrial Dysfunction, and Aging Mechanism

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Message from the Guest Editor

The hypothesis that Reactive Oxygen Spices (ROS), containing free radicals generated in living organisms, contribute to aging progression have been proposed. As most of intracellular ROS is produced in mitochondria, it is important to understand how the mtDNA and proteins in the organelles are damaged, and they contribute aging progression. The mtDNA damages impair the translational capacity and, consequently, this results in insufficient protein supply in the organelle. Thereby, the increased levels of ROS can results in a vicious cycle that generates further mtDNA damages. However, further investigation using animal models need to be performed before the conclusion concerning the oxidative stress, mitochondrial dysfunction and ageing. Some recent studies reported the aging-related phenotypes seen in artificial conditions have not always observed in normal aging. Once oxidative damages are accumulated in a mitochondrion, the damaged parts are separated by fission and subsequently removed by mitophagy. It still remains to be studied how mitochondrial dynamics and mitophagy influences aging-related phenomena in animal models.

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