

Exercise to Promote Healthy Mitochondria and Improve Lifestyle of Aging Society

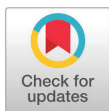
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Mitochondria produce ATP that provides readily releasable energy to cells [1]. In order to maintain proper function, the mitochondrial network undergoes fission to separate damaged mitochondrial fragments from the healthy, and fusion to reduce mitochondrial dysfunction [2,3]. Dynamin-related proteins that are known to regulate mitochondrial fusion include Mitofusin 1 (Mfn1), Mitofusin 2 (Mfn2) and Optic atrophy gene 1 (Opa1), whereas the major effector of fission is Dynamin-related protein 1 (Drp1) which has been most frequently associated with pathological conditions [4].

During aging, mitochondrial performance declines as the balance between fusion and fission is often compromised [5,6]. This imbalance has been implicated in neurodegenerative diseases [6], sarcopenic muscles [2] and various other pathological conditions [2,4]. Exercise has been reported to increase mitochondrial biogenesis, remove damaged mitochondria, stimulate mitochondrial function [5,7] and delay aging associated decline in physical fitness [8-10] and cognitive function [11]. Exercise in the elderly has also been reported to improve mitochondrial efficiency as observed with the increase in Ser637 phosphorylation in Drp1 [12].

Therefore, promoting exercise is a promising strategy to improve lifestyle of the current aging society, requiring further research to better understand the mechanisms of mitochondrial dynamics.

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