Review Article

Effect of exercise on aging cardiac hypertrophy, role of oxidative pressure and some of the mitogen-activated protein kinases

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Abstract

Aging is an inevitable process, which is associated with the development of various diseases such as cardiac hypertrophy. Hypertrophy can occur in both pathological and physiological form. Both types can be divided into a variety of eccentric and concentric types. In the present review, we present the effects of aging and exercise on patological and physiological cardiac hypertrophy, oxidative stress and some of the mitogen-activated protein kinases with using 79 articles which accetable in pubmed and SID indexing which published during 1976-2016. If the age is associated with inactivity, leads to pathological heart hypertrophy. Meanwhile, the role of the protein family of kinases activated with mitogen and oxidative stress is important. Adolescence, if accompanied by low activity, can lead to increase oxidative stress through mitochondrial dysfunction. Oxidative stress can affect the activity of MAPKs. MAPKs have important role in wide variety of biological events, such as proliferation, differentiation, metabolism, mobility, survival and apoptosis. The tipping point of signal transduction and the regulation of these biological events begin initially by the four MAPK subunits, including extracellular signal regulated kinase (ERK1 / 2), c-Jun NH2-terminal kinase (JNK1, -2.3) kinase P38 (A, B, y, S) and large MAPKs (BMKs or ERK5s). This paper focuses on two types of ERK1 / 2 and P38 that play an important role in the development of cardiac hypertrophy. ERK1 / 2 and P38 amounts change with aging. These changes are associated with the development of pathological hypertrophy. Sports activities can control the pathological pathway of hypertrophy and can lead to physiological hypertrophy. Exercise can control or reduce oxidative stress, ERK1 / 2 and P38 and ultimately can affect cardiac hypothyroidism.

Keywords: Aging, Exercise, Cardiac hypertrophy, Mitogen activation protein kinase, Oxidative stress

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