The effects of exercise on caspase-independent mitochondrial proteins in regards to age-related apoptosis

Sean M. Courtney, Jong Hee Kim, Brandon R. Macias, Claire N. Canon, John M. Lawler

Texas A & M University

Int J Exerc Sci 2(1): S39, 2009. Exercise may have protective factors in reducing oxidative stress, mitochondrial dysfunction and mitochondrial caspase-dependent apoptosis with aging. It is presently unclear whether the caspase-independent apoptosis via EndonucleaseG (EndoG) and Apoptosis Inducing Factor (AIF) translocation from the mitochondria to the nucleosome is effected by exercise in aging skeletal muscle. It is understood that in aging skeletal muscle EndoG and AIF do translocate from the mitochondria to the nucleosome. We hypothesize that exercise will attenuate the translocation of EndoG and AIF from the mitochondria to the nucleosome in aging white gastrocnemius muscle. Twenty-four Fischer Brown Norway rats were randomly assigned to four groups, young sedentary, old sedentary, young exercisers and old exercisers. The exercise consisted of treadmill training. The protein expression of EndoG and AIF were analyzed using western blot assays. In the old sedentary group, EndoG increased 86.4 % in the soluble fraction, but there was no change in the young groups. EndoG protein levels in the nucleosome fraction of young exercisers decreased 49 % when compared to young sedentary controls and old sedentary increased by 86.5 % when compared to young sedentary controls. With AIF changes in the soluble fraction were neglible. Protein levels of AIF in the nucleosome fraction increased 64 % in the old sedentary group compared to young sedentary controls. The data indicates that exercise was a protective factor against caspase-independent apoptosis by decreasing the translocation of EndoG and AIF to the nucleosome in aged skeletal muscle.