Exercise attenuates CTGF levels, delaying the onset of fibrosis in the aging left ventricle

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Int J Exerc Sci 2(1): S41, 2009. Aging is associated with a progressive increase in collagen (i.e., fibrosis) in the aging heart. Fibrosis is associated with a stiffening of the heart and a decrease in heart function. Previous data (Kwak, 2008) showed that matrix metalloproteinase (MMP) activity in the aging heart is decreased linked to an elevation TIMP-1 (tissue inhibitor of MMPs-1); while exercise training increased TIMP-1 and reduced MMP expression. Connective tissue growth factor (CTGF) is an upstream inhibitor of TIMP-1 and is implicated in diabetes and heart failure. We hypothesized that exercise would decrease CTGF levels, thus reducing fibrosis in the aged left ventricle. Young (3 months) and old (31 months) male Fischer 344 × Brown Norway F1 hybrid (F344BNF1) rats were used for the study. The rats were randomly assigned to one of the following experimental groups (n=10/group): young sedentary controls (YS), young exercise trained (YE), old sedentary controls (OS), and old exercise trained (OE). Rats walked quickly on a motor-driven treadmill for 45 min/day, 5 days/wk, for 12 wks. Heart levels of CTGF were determined via Immunohistochemistry (IHC). CTGF was expressed more in the old group compared to the young group, as well as the sedentary group compared to the exercise group. Future studies will determine if CTGF is downstream of angiotensin II, TGF-beta, and oxidative stress in regulating MMPs and TIMP-1. Supported by AHA (0855158F) and NIH (AR054084).