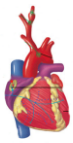

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The effects of physical exercise on myocardial telomere regulating proteins, survival Pathways, and apoptosis In 204 patients with coronary artery disease and 6 patients with dilated cardiomyopathy

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Abstract:

Introduction:

Telomerase is a ribonucleo protein involved [1-8-14-15] in maintaining telomere [1-8-14] length in stem cells and immoral and actively dividing cells. Accumulation of cellular[23] damage with advancing age leads to atherothrombosis and associated cardiovascular disease.

Methods:

Regular exercise has been shown to improve [8-10-11-20-21-22] control of Lipid abnormalities, diabetes mellitus, hypertension, and obesity, with the greatest benefit realized by sedentary individuals who begin to exercise. This research has indicated that response to exercise may be mediated in large part by variation in genes.

Results:

We sought to determine whether the benefit of training for vasodilation in the skeletal muscle vasculature of patients with CAD[9-16-17-18-19] is likely to be caused at the molecular level primarily by increased nitric oxide (NO)[1-2-3-4-5-7-8-11-12-13-14] production or decreased inactivation of NO[11-13]. Some animal studies support a mechanism whereby training increased vascular NO levels by sustained transcriptional activation of the endothelial no synthesis (ENOS) gene, presumably due to shear stress[10-11-12].

Discussion:

Long and short term voluntary physical exercise upregulates cardio[4-2-1] telomere regulating proteins, there by induces antisenescent and protective effects for example to prevent Doxorubicin toxicity and has beneficial cardic effects are mediated by(TERT) telomere reverse transcriptase, endothelial nitrous oxide synthesis (ENOS), insulin growth factor I (IGF-1).

Key words:

Concurrent training, shear stress, aging, telomere regulating protein (TRP).
Telomere repeat binding factor [1-8-14-16] (TRBF I), (TRBF II).
Chaperon protein,ubiqutin proteasome pathway[23].
Endothelial cell nitrous oxide synthesis [1-8-14] (ECNOS).
Coronary artery disease, impedance cardiography[19].

Introduction:

Telomere [1-8] regulating proteins affect cellular senescence, survival and regeneration.
Telomeres are important cellular structures whose integrity is essential for maintaining cell viability. There is now clear evidence that links Telomeres and their associated proteins to cancer, cardiovascular disease and aging. therefore understanding the function of this biological system is attracting much attention, in an effort to used the information as a means to combat cancer and age – related disease. Without any doubt in 6 of/our patients with dilated cardiomyopathy and 204 patients with CAD long term[16-17-18-21] physical exercise up-regulated cardiac telomere-stabilizing proteins and there by induced antisenescent, regeneration and survival of endothelial cells happened, and had protective effects.
These benefiticial [8-11-14-16] cardiac effects are mediated by telomere reverse Transcriptase (TRT), telomere repeat binding factor (TRF), insulin like growth factor I (IGF1), endothelial cell nitrous oxide synthesis (ECNOS) did great help for collateralization re - epithelialization of the coronary artery.

Methods:

Regular exercise and loss of 300 – 500 k/cal per day, (anaerobic exersice), resistance exercise (maximal repitation) with erobic exercise, tractions, or loss of 2000, 2500 k/cal a week have/ shown to improve control of diabetes mellitus, hypertension obesity, psychotic problems, lipid abnormalities, body mass index (BMI) , pulmonary capacity, muscular strength, vo_2 max, homocysteine levels, glucose transferase 4 protein (GLUT4 P), coronary artery disease, atrial neutrouretic peptide, regulate heat shock protein, blance between chaperon protein and ubiquitin protea some pathway, elongation of telomers. Recent research has indicated that response to exercise may be mediated in large part by variation in genes. The purpose of this study was to investigate the underlying molecular, physiological mechanisms of the protective cardiac effects of physical exercise. 204 patient[16-17-18-19] with coronary artery disease (mean age 60±6 years, mean weight 74±12 kg and mean body mass index 26±4), and 6 patient with dilated[22] cardiomyopathy admitted in our clinic from aug 2002 tile july 2008. They participated in 3 month concurrent training, 3 times/ week for 60 to 80 minutes at 70% to 85% of (MHR)* and 40% to 60% of 1RM**. Investigation variables were assessed at baseline and at the end of the protocol by the impedance cardiography method.

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*MHR = 1 Repeat maximum

**1RM = 1 Repeatation maximum

Discussion:

Telomerase [1-13-4] is a ribonucleo protein involved in maintaining telomere length in stem cells and immortal and actively dividing cells. Accumulation of cellular damage with advancing age leads to atherothrombosis and associated cardiovascular disease. Aging[1-8] is also characterized by shortening of the DNA component of telomeres, the special genetic segments located at the end of eukaryotic chromosomes that protect them from end to end fusion. By inducing genomic instability replicative senenscence and apoptosis.

shortening of the telomeric[1-14-15] DNA is thought to contribute to organismal aging in this review, we discuss experimental and human studies that have linked telomere and associated proteins to several factors which influence cardiovascular risk (fg, estrogens, oxidative stress, hypertension, diabetes[15-25], psychological stress) as well as to neovascularization and the pathogenesis of atherosclerosis and heart disease are whether telomere shortening is caused cardiovascular disease and whether therapies targeting the telomere may find application in treating these disorders (eg. cell telomerization to engineer blood vessels of clinical value for bypass surgery and to facilitate cell based myocardial regeneration strategies), are aorto coronary bypass graft[16-17-18-19-21-22-24] (ACBG) or angioplastry can save the damage endothelial walls? are these procedures can able to correct telomeres? we must accept that CAD is a generalized arterial (vascular) disease.

Results:

This article briefly summerised strategies presently being used to elucidate genes and genetic effects that may be mediated or influenced by exercise[18-21-22] and severs to illustrate the importance of considering the effect of exercise[15-16]when investigating / genes, related to health or other physiological outcomes.

A significant training effect was documented by an decrease in heart rate at rest. Stroke volume (SV)[19] increased/from 60± 8 to 81±13ml/beats ($p<0.05$).

stroke index (SI) increased from 33± 4 to 44± 6 ml/ beat m^2 ($p<0.05$). Cardiac[19]out put (CO) increased from 4± 1 to 5±1 lit/min ($p<0.05$). Cardiac[19] index (CI) increased from 2± 0.5 to 3±0.5 lit/min/m^2 ($p<0.05$). Systemic vascular resistance (SVR) decreased from 1782± 361 to 1540± 294 dynes/sec/cm^5 ($p<0.05$). Systemic[19] vascular resistance index (SVRI) decreased from 3212± 662 to 2751± 558 dynes/sec/m^2/cm^5 ($p<0.05$).

Conclusion:

We conclude that concurrent training may improve myocardial hemodynamic and ribonucleo protein responses in patients with coronary artery disease and cardiomyopathy.

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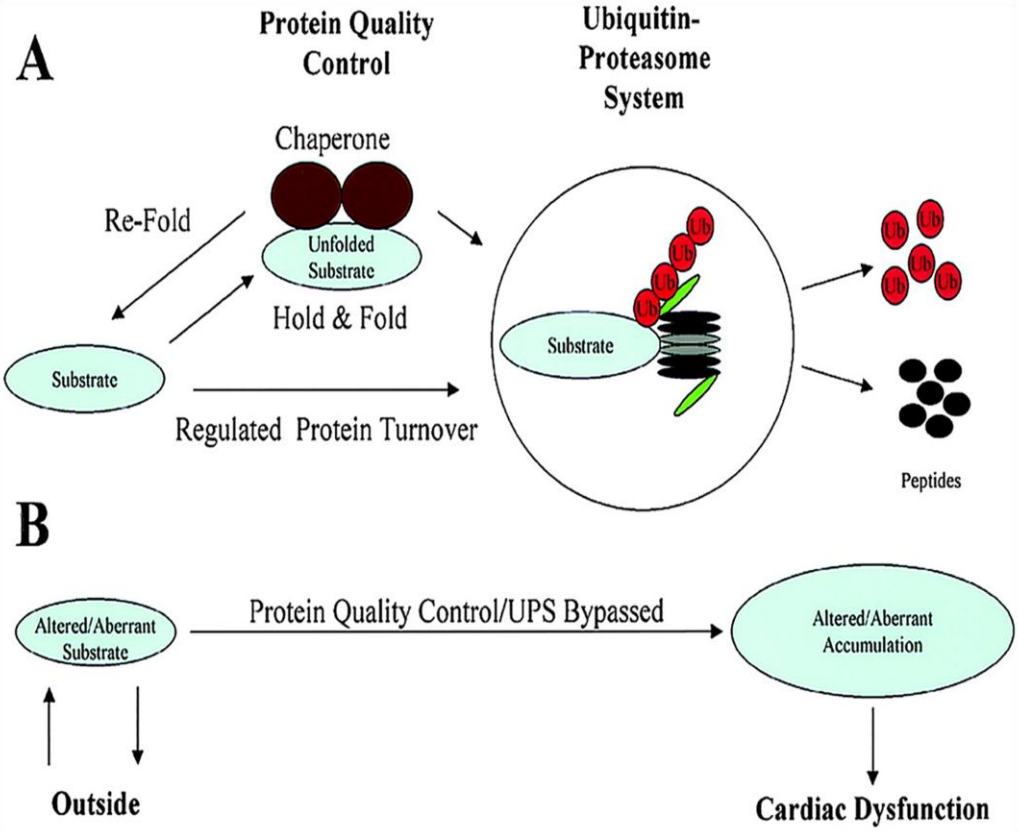
Relationship between protein folding and the ubiquitin (Ub)-proteasome system (UPS) in protein quality control and the consequences of defects in these processes



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