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Effects of exercise on cardioprotection – down-regulation of Ras homolog gene family member A
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Background – Oxidative stress plays a role in developing cardiovascular disease (CVD) and regular exercise is believed to be cardioprotective. However, the molecular mechanisms are poorly understood. Recent data suggest that exercise-induced alteration in the heart and vascular endothelial cells (ECs) may provide cardioprotection.

Objective – To investigate the effects of exercise and/or antioxidant supplement on myocardial and vascular endothelium gene expression.

Design – Male rats (12 per group) were divided into four groups: i) exercise (90 min at 70% VO2 max/d, 4 d per week); ii) antioxidant-treated; iii) antioxidant and exercise-treated and iv) control. The exercise group underwent 14 weeks of endurance running on treadmill. The supplement group received Vitamin E (1000 IU/kg diet) and α-lipoic acid (1.6 g/kg diet) mixed with rat chow. After 14 weeks, rats were killed and myocardial and coronary artery ECs were isolated from the hearts. cDNA microarray analysis (27K rat genome) was performed using purified EC RNA.

Outcome – 2-way ANOVA revealed that expression levels of 35, 40 and 40 genes were altered for group i, ii, and iii respectively compared to control and differentially expressed genes were analysed using the KEGG pathway database and hierarchical cluster analysis. Most notably one gene that is involved in cardioprotection, Ras homolog gene family member A (RhoA), was down-regulated by the effect of exercise (P <0.02), an effect being confirmed by real-time PCR.

Conclusion – Previous studies have revealed that gene expression level of RhoA was increased in patients with CVD. RhoA has also been shown to regulate important cell functions which contribute to CVD. Therefore, this pathway might be one of the mechanisms to explain the positive effect of exercise-induced cardioprotection.

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Dairy intake and resistance exercise during energy restriction in abdominal obesity: effects on endothelial function and blood pressure.
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Background – Sustained weight loss benefits endothelial function and the metabolic profile in abdominal obesity.

Objectives – To determine, (i) the specific impact of serves of dairy with, and without resistance exercise (RE), on C-reactive protein levels, micro albumin to creatinine ratio (ACR), stiffness index (SI), reflective index (RI), mean arterial pressure (MAP) and plasma lipids, (ii) whether insulin resistance at baseline modified these responses.

Design – 36 abdominally obese European Australians (20M, 16F), aged (mean ± SD) 57 ± 8.9 y were randomised to one of three dairy groups while consuming an energy deficient diet for 12 weeks: 3 serves/d (3S, ~1311 mg calcium & 50 IU vitamin D/day, n = 13), 5 serves/d (5S, ~2124 mg calcium & 100 IU vitamin D/day, n = 12) and 5 serves/d plus RE (5S+RE, n = 11). Blood pressure and non-invasive endothelial function (PT-1000, Micro Medical, UK) was recorded at weeks 0 and week 12. All measurements were made in the supine position, after an overnight fast and following a mandatory 1h rest. Blood samples and timed urines were also collected. The change over 12 weeks (Δ) was analysed using multivariate 2x2 ANOVA with appropriate covariates (age, gender, machine, Δfat and Δfat free mass). Diet effects, HOMA-group effects (group 0 < 1.52 vs. group 1 = ≥ 1.52) and their interaction were examined.

Outcomes – Data are mean ± SE. ΔMAP showed a significant diet x HOMA-group interaction (P = 0.048), where HOMA-group modified the direction of change only on the 3S diet, while ΔMAP decreased in both groups on the other diets. ΔRI was significantly higher on the 5S+RE diet (3S: ~ 2.4 ± 3.11; 5S: -6.1 ± 3.21; 5S-RE: +6.5 ± 3.38 %, P = 0.034), and ΔSI showed a similar trend between diets (P = 0.084). There was no statistical difference in any other plasma or urinary biomarker.

Conclusion – The inclusion of 5 serves of dairy per day in an energy restricted diet lowered resting MAP, RI and SI irrespective of HOMA scores at the start. The addition of RE to 5 serves of dairy preserved the blood pressure effects, but impaired both vascular tone (RI) and arterial stiffness (SI).