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CHANGES IN BLOOD LIPID LEVELS INDUCED BY DIFFERENT DIETARY FAT TYPES ARE NOT INFLUENCED BY PRE-SUPPLEMENTATION WITH FISH OIL

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Background/Aims: Although, the health benefits of long chain *n*-3PUFA are well known, the effects of n-3PUFA status on blood lipid modulation by background dietary fats is not well understood. Therefore, this study aimed to determine the effects of diets high in either saturated fatty acids (SFA) or *n*-6PUFA following pre-supplementation with *n*-3PUFA.
Methods: This was a randomised, controlled, parallel, dietary intervention trial involving 22 healthy adults aged 18 to 65 years. Participants consumed 2.4 g *n*-3PUFA daily for 4 weeks and then were randomized to one of 2 diets, enriched either with SFA or *n*-6PUFA combined with 2.4 g *n*-3PUFA daily for at least 10 days. Blood samples and anthropometric measurements were collected after an overnight fast, at baseline, after 4 weeks and post-intervention. Blood samples were assessed for lipid [total, low density lipoprotein (LDL) and high density lipoprotein (HDL) cholesterol and triglyceride] levels.
Results: Pre-supplementation with *n*-3PUFA decreased plasma triglycerides ($p = 0.006$) and increased HDL cholesterol ($p = 0.032$) and body mass index (BMI; $p < 0.001$) significantly. After the SFA and *n*-6PUFA diets no further change was observed in plasma triglycerides, HDL cholesterol or BMI. The SFA diet caused an increase in total and LDL cholesterol ($p = 0.008$ and $p = 0.013$, respectively), while the *n*-6PUFA diet caused a decrease in total and LDL cholesterol ($p = 0.003$, both).
Conclusions: Pre-supplementation with *n*-3PUFA does not influence changes in blood lipid levels induced by type of background dietary fat.
Funding source(s): Hunter Medical Research Institute (HMRI) and Coordenação Nacional de Desenvolvimento Científico e Tecnológico (CNPq, Brazil).

IS COGNITIVE IMPAIRMENT IN POSTMENOPAUSAL WOMEN ATTRIBUTABLE TO POOR CEREBRAL PERFUSION? BASELINE RESULTS OF THE RESFEM STUDY

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Background/Aims: Postmenopausal women suffer disproportionately from dementia, which we hypothesise may be partly attributable to loss of cerebrovascular benefits of oestrogen. We are currently testing whether a 14 week supple-mentation with resveratrol (a phytoestrogen) can improve cerebral perfusion and cognition in postmenopausal women. Herein, we present baseline evaluation of these parameters.
Methods: Eighty postmenopausal women aged 45-85 years underwent cognitive testing; domains of executive function, semantic, verbal and visuospatial working memory were scored individually and as a composite. Transcranial Doppler ultrasound was used to record basal blood flow velocity in the middle cerebral artery (MCA) and a pulsatility index (PI), reflecting stiffness of the vessel, was calculated. Cerebrovascular responsiveness (CVR) to the cognitive testing, which reflects the ability of a brain region to vasodilate in response to demands, was expressed as the percentage change in mean blood flow velocity from the basal level recorded for 30 sec before tests to the peak velocity attained during testing
Results: Using Pearson's correlation, we found that the composite cognitive score correlated with PI ($r = -0.291$, $p = 0.017$), the basal mean blood flow velocity ($r = 0.34$, $p = 0.005$) and CVR to the test battery ($r = 0.301$, $p = 0.017$) in the MCA.
Conclusions: Our baseline assessment of post-menopausal women shows that cognitive performance is linked to cerebrovascular function, at rest and during activation. Therefore, optimising cerebral perfusion may help to attenuate cognitive decline in this at-risk population.
Funding source(s): Hunter Medical Research Institute 3D Healing Project Grant.

FEASIBILITY OF A RANDOMISED CONTROLLED TRIAL OF FISH OIL SUPPLEMENTATION IN PEOPLE WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Background/Aims: To determine the feasibility of undertaking a dietary supplementation trial to evaluate fish oil as an adjunct therapy for chronic obstructive pulmonary disease (COPD).
Methods: A double-blind, randomised controlled parallel trial was undertaken to compare the effects of fish oil versus corn oil placebo (six 1 g capsules/day orally for 16 weeks) on respiratory function, dyspnoea, functional exercise capacity and well-being. The following a priori feasibility criteria were evaluated: ability to recruit 40 participants within 52 weeks, 80% participant retention rate, and a moderate sized effect in at least three outcome measures.
Results: Of 267 potential participants, 101 declined to participate and 91 were unable to be contacted. Of those willing to participate 62 were excluded at the telephone screening, the most common reasons being participants were taking fish oil ($n = 20$), did not have COPD ($n = 10$), were participating in another study ($n = 7$) or, at visit 1, had a significant bronchodilator response ($n = 13$). Only13 were enrolled (7 in the initial 52 weeks), 9 of whom completed the 16 week intervention. Participants withdrew from the study due to illness ($n = 2$), injury unrelated to the study ($n = 1$) or an adverse event ($n = 1$). There was one moderate sized change (impulse oscillometry, effect size -0.56).
Conclusions: This preliminary trial did not meet the pre-determined key feasibility criteria, which raises doubts as to whether we can ascertain the efficacy of omega-3 supplementation in COPD using this approach.
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DOES CAFFEINE CONSUMPTION DURING 50 H OF SLEEP DEPRIVATION ALTER GLUCOSE METABOLISM, HUNGER AND SATIETY RATINGS?

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Background/Aims: Sustained operations are common in military and emergency services. In these operations it is not always possible to obtain sleep, resulting in sleep deprivation. Caffeine is a widely used fatigue countermeasure. However, the impact of caffeine on glucose metabolism, hunger, and satiety during sleep deprivation is largely unknown.
Methods: In this double-blinded laboratory based study, participants were assigned to either a caffeine ($n = 12$, 4F, 22.5 ± 3.3 y, 21.7 ± 1.5 kg/m^2) or placebo condition ($n = 12$, 5F, 22.5 ± 2.5 y, 22.3 ± 2.1 kg/m^2). The protocol included one baseline sleep (22:00h–08:00h), 50h sleep deprivation and a daytime recovery sleep (10:00h–19:00h). Caffeine (200 mg) or placebo gum was chewed for 5min at 01:00h, 03:00h, 05:00h and 07:00h during each night of sleep deprivation. Meal timing and composition were controlled throughout the study; breakfast composition ≈1611 kJ; 16% protein, ≈73% carbohydrate and 3% total fat. Interstitial continual glucose monitors captured 2h post-breakfast levels, at 5min intervals. Hunger/satiety scales were administered at 10:00h after 26h of sleep deprivation.
Results: Relative to baseline (6.1 ± 0.5 mmol/L) sleep deprivation led to increased mean glucose 2h post breakfast at 24h sleep deprived (6.5 ± 0.5 mmol/L, $p < 0.001$) and 48h sleep deprived (7.1 ± 0.5 mmol/L, $p < 0.001$). There was no difference between placebo and caffeine conditions ($p =$