Effect of omega-3 fatty acid supplementation on blood pressure and heart rate variability following sympathoexcitatory stress in mild hypertensive and normotensive subjects

Elliott Carthy¹,², Leigh White¹, Fraser Russell¹,⁴, Mark Holmes¹,⁴, Anthony Leicht³, Peter Brooks¹,⁴, Philip Barker¹, Deborah Hitchen-Holmes¹, Chris Askew¹,⁴

¹Faculty of Science, Health, Education & Engineering, University of the Sunshine Coast, QLD, Australia, ²School of, Imperial College London, London, UK, ³Institute of Sport and Exercise Science, James Cook University, QLD, Australia, ⁴USC Inflammation and Healing Research Cluster, University of the Sunshine Coast, QLD, Australia

Background: Long chain omega-3 polyunsaturated fatty acids (n-3 FA) have known anti-hypertensive effects; however their mechanism of action remains unclear [Cicero et al., 2010; Cicero et al., 2009]. Resting hypertension has been associated with autonomic dysfunction, namely an increase in sympathetic and/or a decrease in parasympathetic activity at rest [Schroeder et al., 2003]. We hypothesised that n-3 FAs supplementation would increase vagal modulations and/or decrease sympathetic activity, contributing to their anti-hypertensive effects.

Methods: Mild hypertensive (HT: mean±SEM; blood pressure: 142.4±2.19/81.1±2.91mmHg; age: 51.5±2.94years; body mass index: 30.3±1.25kg/m²; n=16) and normotensive participants (NT: mean±SEM; blood pressure: 116.2±1.56/73.0±1.07 mmHg; age: 50.5±1.49years; body mass index: 25.8±0.59kg/m²; n=42) were supplemented with 2.52g/day of n-3 FA (docosahexaenoic acid: 0.84g; eicosapentaenoic acid: 1.68g; Omega Heart, Blackmores Pty Ltd.) or placebo (canola oil; Blackmores Pty Ltd.) for nine weeks. Participants were included if they were 30-85 years, on a low fish diet and not taking fish oil capsules or anti-hypertensive medication. Participant demographics are shown in Table 1. Participants provided 4 repeat morning measurements of resting BP, with readings 2-4 used for analysis. Subjects underwent an assessment of their resting heart rate variability (HRV) and BP initially, and at 3 weekly intervals. The omega-3 index was measured using gas chromatography-mass spectrometry. This study received approval from the University of the Sunshine Coast Human Research Ethics Committee (EC00297 A/08/167).

Results: There was a significant increase in the omega-3 index for the n-3 FA supplemented group (Before: 5.43±0.18%; After: 8.31±0.30%; p<0.05) compared to the placebo group (Before: 5.67±0.24%; After: 5.50±0.23%). A non-significant trend for reduction in systolic (SBP), diastolic (DBP) and mean arterial pressure (MAP) was observed for n-3 FA supplemented HT and NT participants when compared to placebo (SBP: -2.67±1.04 vs. -0.69±1.17mmHg; p=0.21; DBP: -1.68±0.82 vs. -0.71±0.89mmHg; p=0.43; MAP: -1.91±0.83 vs. -0.18±1.02mmHg; p=0.19). There were no significant main treatment effects for the HT or NT groups considering HRV throughout the intervention.

Conclusions: The significant increase in omega-3 index for the n-3 FA supplemented group confirmed incorporation of n-3 FA into red blood cell membrane phospholipids. The magnitude of this response was consistent with current recommendations for cardioprotection (>8.0%) [Harris, 2010]. There was a tendency for a greater overall reduction in BP in participants who were supplemented with n-3 FA, irrespective of their basal BP. However, there were no significant changes in HRV, forearm blood flow or reactive hyperemia in those supplemented with n-3 FA compared to placebo. The current results indicate that n-3 FA supplementation at a level needed for cardioprotection reduces BP slightly with no significant alteration of autonomic function for mild HT and NT participants. Supplementation with n-3 FA may be more effective (i.e. improve cardiac autonomic function) for those with a more severe hypertensive state.

References
Cicero AFG et al, Clinical and Experimental Hypertension 32:137, 2010
Cicero AFG et al, Current Vascular Pharmacology 7:330, 2009
Harris WS, Current Cardiology Reports 12:503, 2010
Schroeder EB et al, Hypertension 42:1106, 2003