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Dietary protein level affects ω-3 PUFA deficiency-induced hypertension

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Background – Our previous studies have demonstrated that dietary ω-3 PUFA deficiency from the perinatal period leads to hypertension in later life. However, in more recent studies we have been unable to replicate this observation. These later studies contained minor modifications to the diets including a reduction in the protein level which may have interacted with ω-3 PUFA deficiency.
Objective – We examined the effect of altering the level of dietary protein (casein), from the perinatal period, on blood pressure, food intake, body weight and body composition in ω-3 PUFA deficient animals, compared with ω-3 PUFA sufficient animals.
Design – Female Sprague Dawley rats were placed on one of four semi-synthetic experimental diets one week prior to mating. Diets were either deficient (10% safflower oil; DEF) or sufficient (7% safflower oil, 3% flaxseed oil; SUF) in ω-3 PUFA and contained 20% or 30% w/w casein (DEF20, SUF20, DEF30, SUF30). Offspring were weaned at 3 weeks and maintained on their mother's diet for the duration of the experiment.
Outcomes – DEF30 animals had a lower body weight at weaning than SUF30 animals, but had a higher food intake in the subsequent weeks and more rapid body weight growth. These differences were not observed in the DEF20/SUF20 groups. At 12 and 18 weeks of age, no differences in blood pressure were observed based on diet. By 24 weeks of age, hypertension was present in DEF30 animals; other groups did not have blood pressure differences. DEF30 animals also had a higher percentage of body fat.
Conclusion – Our results indicate that ω-3 deficient hypertension is dependent on other dietary factors including dietary protein levels.

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Dietary polyunsaturated fatty acids and sex interact to determine myocardial membrane fatty acid composition

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Background – Clinical and experimental studies have shown that dietary polyunsaturated fatty acids (PUFA) modulate cardiac function under pathological conditions, and that this is, at least in part due to modulation of the membrane fatty acid composition. Sex-based differences are evident in cardiovascular function and disease (including cardiac hypertrophy) and these differences have been largely attributed to sex steroids, however the cellular and molecular mechanisms underlying sex differences remain unresolved.
Objective – To determine whether there are sex-differences in diet-induced modulation of myocardial membrane fatty acid composition in a murine model of cardiac hypertrophy.
Design – Female and male cardiac angiotensinogen over-expressing transgenic (TG) and wild type (WT) mice were fed a fish oil-derived omega-3 PUFA diet (FO) or an omega-6 PUFA rich contrast diet (CTR) for four weeks (n=8 / group) and studied at age 18 weeks. Myocardial membrane PUFA was analysed by gas chromatography.
Outcomes – Dietary FO was associated with a smaller hypertrophic index (heart mass normalized to body mass) in hearts of males (mean ± SEM, TG CTR/FO 5.0 ± 0.3 vs 4.4 ± 0.2 mg/g, $P<0.01$) but not females (TG CTR/FO 4.7 ± 0.1 vs 4.6 ± 0.2 mg/g, $P>0.05$), and this was coupled with a greater myocardial membrane omega-3: omega-6 PUFA ratio in male mice compared to female mice (TG FO 5.8 4± 0.14 vs 5.42 ± 0.18, $P<0.01$). Overall (ie in both diet groups), relative to male hearts, female myocardial membranes had a smaller proportion of total saturated fatty acids, characterised by less myristic and stearic acids; a greater proportion of total omega-6 PUFA, namely, linoleic, eicosadienoic, arachidonic and adrenic acids; and greater proportion of total PUFA (3-way ANOVA, $P<0.01$).
Conclusion – This study indicates that variations in membrane fatty acid composition may underlie or modify sex-dependent differences in this animal model of cardiac hypertrophy.