NSA Concurrent Oral Session 4: Lipids

**Development and evaluation of foods enriched with omega-3 (ω3) fatty acids from fish oil**

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**Background**- Consumption of fish or fish oils rich in very long chain (VLC) ω3 reduce cardiovascular (CV) disease by acting on risk factors such as blood clotting, hypertension, blood triglycerides or loss of compliance in blood vessels.

**Objectives**- To determine whether a 1g/d target intake for VLC ω3 can be sustained using ω3 enriched processed foods; and if regular consumption of these foods can improve CV health.

**Design**- Overweight volunteers with high blood lipids were enrolled in a dietary intervention trial in Adelaide (n=50) and Perth (n=44) and were randomised to eat 8 serves/day from a selection of ω3 enriched foods (~125mg/serve) or matching control foods, which were substituted for equivalent foods in their regular diet.

**Outcomes**- VLC ω3 intake estimated by food compliance questionnaires was slightly below target (750mg/day) but DHA and total VLC ω3 content of erythrocytes increased by 45% and 35% respectively at 3 months and by 70% and 53% at 6 months. The increases were not accompanied, however, by changes in CV or inflammatory markers and there were no significant effects attributable to the ω3 enriched foods versus the control foods.

<table>
<thead>
<tr>
<th></th>
<th>Control foods</th>
<th>Omega-3 enriched foods</th>
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<tbody>
<tr>
<td></td>
<td>(n)</td>
<td>(n)</td>
</tr>
<tr>
<td>Small artery compliance1</td>
<td>6.8±0.6</td>
<td>7.29±0.5</td>
</tr>
<tr>
<td>Systolic blood pressure2</td>
<td>128±1.9</td>
<td>128±1.9</td>
</tr>
<tr>
<td>Diastolic blood pressure2</td>
<td>74±1.3</td>
<td>76±1.4</td>
</tr>
<tr>
<td>Plasma Triglycerides3</td>
<td>1.9±0.1</td>
<td>1.8±0.1</td>
</tr>
</tbody>
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**Conclusion**- Although regular consumption of ω3 enriched processed foods increased erythrocyte VLC ω3 to levels comparable to fish oil supplementation, the increase was insufficient to improve selected health parameters.

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**Dietary omega-3 fatty acid supply influences mechanisms controlling body weight and glucose metabolism**

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**Background**- Omega-3 polyunsaturated fatty acids are known to influence crucial membrane functions, eicosanoid metabolism and gene expression mechanisms.

**Objective**- To determine the influence of dietary omega-3 fatty acid supply on ingestive behaviour, body weight, adiposity and glucose tolerance.

**Design**- Female rats were fed with a ω3-linolenic acid (ALA) sufficient (CON) or deficient (DEF) diet throughout gestation and lactation. Three groups of male offspring were studied: (1) pups maintained on CON diet, from mothers on CON diet, CON (n=11); (2) pups maintained on DEF diet, from mothers on DEF diet, DEF (n=11); (3) pups maintained on CON diet from weaning (3 weeks of age), from mothers on DEF diet, DEF-CON (n=11). Food intake, body weight, fat and oral glucose tolerance were assessed in adult offspring. Brain gene expression of 3-week old and adult offspring was evaluated. Fatty acid profile of mothers’ milk was also analyzed. Statistical analysis by ANOVA; P<0.05 was considered significant.

**Outcomes**- CON-mothers showed a 15-fold increase of ALA content in milk compared with DEF-mothers. Relative to CON offspring, adult DEF-CON offspring consumed more food (P<0.05), were heavier, had a greater proportion of body fat and showed impairment in glucose tolerance; adult DEF animals had similar food intake, body weight, proportion of body fat, but showed impaired glucose tolerance. Two genes coding for proteins involved in glucose homeostasis (Pttg1; Pituitary tumor-transforming 1, Exoc7; exocyst complex component 7) were under-expressed in DEF weanlings but not in adult animals. The expression of genes coding for glucose transporter 4, insulin and leptin receptors and neuropeptide Y were not altered due to omega-3 deficiency.

**Conclusions**- Deficiency of omega-3 fatty acids from conception adversely affected glucose tolerance, assessed in adulthood. Exposure of DEF offspring to CON diet from weaning, however, caused more severe disruption of physiological mechanisms, possibly initiated by changes in the mechanisms controlling food intake.