

## NSA Poster Presentations: Friday 13 August 2004

### Effect of sucrose feeding on genes associated with liver fat metabolism

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**Background** - High sucrose intake causes marked derangements in liver metabolism, characterised by increased lipogenesis, elevated triglyceride accumulation and hepatic insulin resistance. To date little is known of the molecular mechanisms governing these alterations in lipid homeostasis.

**Objectives** - This study aimed to investigate the alterations in the expression of the peroxisome proliferator activated receptor (PPAR) family of transcription factors and key genes necessary for lipid oxidation and synthesis.

**Design** - Sprague Dawley rats were ad-libitum fed a high sucrose (30%) (n=8) or high starch diet (n=8) for 4 weeks after which the mRNA level of PPAR $\alpha$ , PPAR $\delta$ , peroxisome proliferator activated receptor gamma co-activator beta (PGC-1beta) and genes important in lipid oxidation and synthesis was measured in the liver.

**Outcomes** - Sucrose feeding markedly lowered the gene expression of PPAR $\alpha$  (50% lower). However, the gene expression of PPAR $\delta$  and PGC-1beta remained unaltered by sucrose feeding. Analysis of genes important in lipid synthesis (lipogenesis) revealed that malonyl-CoA decarboxylase (MCD) was increased 1.5 fold by sucrose feeding.

**Conclusion** - These results support previous findings that lipogenic activity is increased by sucrose feeding, however these alterations are not due to increased PPAR $\alpha$  gene expression. Further work is necessary to characterise the sucrose-dependent transcriptional control of MCD and the impact of sucrose on genes necessary for the oxidation of lipids.

### Short term energy restriction (using meal replacements) improves reproductive parameters in polycystic ovary syndrome

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**Background**- Polycystic ovary syndrome (PCOS) is a common endocrine condition affecting women of reproductive age. Weight loss improves the reproductive and metabolic dysfunction associated with PCOS. However, it is unclear what extent of adiposity reduction is required to restore reproductive function.

**Objective**- To assess the relative effects of energy restriction and weight loss on changes in reproductive parameters and to assess the effectiveness of meal replacements as a weight loss strategy in overweight women with PCOS.

**Design**- Overweight women with PCOS (n = 34; age = 32.6  $\pm$  5.1 years; weight = 96.0  $\pm$  19.5 kg; mean  $\pm$  SD) followed a weight loss intervention (two meal replacements, low fat snacks and evening meal daily) for 8 weeks. Fasting weight, waist circumference, body composition (assessed by bioelectrical impedance analysis), blood pressure (BP) and venous testosterone, sex hormone binding globulin (SHBG) and free androgen index (FAI) were assessed fortnightly.

**Outcomes**- The intervention resulted in a reduction in weight (5.6  $\pm$  2.4 kg, 6%), waist circumference (6.1  $\pm$  2.5 cm), body fat (4.1  $\pm$  2.2 kg) and systolic BP (8.4  $\pm$  11.1 mmHg) (p < 0.001). There was no change in SHBG but a significant reduction in testosterone (0.3  $\pm$  0.7 nmol/L, p = 0.05) and FAI (3.1  $\pm$  4.6 nmol/L, 16.8%) (p = 0.001). This change in FAI occurred from week 0 – 2 (2.4  $\pm$  4.2 nmol/L, p = 0.002) and corresponded with a weight loss of 2.4  $\pm$  1.0 kg (2.5%) with no further changes in FAI occurring from week 2 – 8. The change in FAI from week 0 – 2 or week 0 – 8 did not correlate with the change in weight, waist circumference or fat mass.

**Conclusions**- Reproductive parameters improved after only 2 weeks of weight loss. Therefore, these data suggest that reproductive function can be restored with either a small degree of weight loss or by acute energy restriction. The implication is that only short term energy restriction may be required to improve reproductive function. This requires further investigation.