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The effects of 1-sarcosine – angiotensin II infusion on food intake, weight loss, energy expenditure and skeletal muscle UCP3 gene expression in rats

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Background – Previous work has shown that infusion of angiotensin II (Ang II) results in weight loss and that the effects of Ang II are mediated by its binding to angiotensin II type 1 (AT1) receptors. The mechanism by which Ang II causes weight loss is not fully understood. Here we report the effects of 1-sarcosine angiotensin II (1-Sar-Ang II), a potent AT1 receptor agonist, on weight loss, energy expenditure and on expression of uncoupling protein-3 (UCP3), inner mitochondrial membrane protein implicated in energy expenditure, in the skeletal muscle.

Objective – The aim of the present experiment was to determine the effects of 1-Sar-Ang II on weight loss, food intake, energy expenditure and skeletal muscle UCP3 gene expression.

Design – Twenty one female Sprague Dawley rats were housed in individual metabolism cages and maintained on a commercial rat chow diet. The rats were randomly allocated to one of three groups: (1) 1-Sar-Ang II group: subcutaneous infusion of 1-sarcosine angiotensin II (500 ng/kg body weight/min for 7 days; osmotic minipumps, Alzet model 2001), ad libitum food intake; (2) pair fed group: sham-operated and offered restricted amounts of food to match the food intake of animals in the 1-Sar-Ang II group; (3) control group: no infusion, ad libitum food intake. Body weights and fluid intake were monitored daily. After one week, the rats were killed and a small piece of quadriceps muscle was taken for analysis of UCP3 gene expression. Energy contents of food and whole body were determined by bomb calorimetry. Energy expenditure was estimated from food intake and change in whole body energy content.

Outcomes – Relative to the control, food intake and body weight were reduced in the 1-sar-Ang II group. Weight loss in the 1-Sar-Ang II group was ~50% higher than that in the pair fed group. Energy expenditure in the 1-Sar-Ang II group was ~30% higher than in the pair fed group but was not different from that in the control group. No differences between the 3 groups were observed in the expression UCP3 gene in skeletal muscle.

Conclusions – The results show that 1-sarcosine angiotensin II induces weight loss by decreasing food intake without an accompanying decrease in energy expenditure. Results do not support a role for UCP3 gene expression in skeletal muscle in the weight loss.

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Weight cycling, metabolic rate and eating behaviours in non-obese females

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Background & Objective – Previous studies state that repeated weight loss followed by weight gain (weight cycling) causes women to become metabolically efficient and therefore regain weight after dieting. Metabolic efficiency, referring to the body’s ability to survive with a low metabolic rate, has been observed in obese subjects, but not in non-obese weight dieters. The aim of this study is to investigate what effect long term weight cycling has on metabolic function and eating behaviour in non-obese group.

Design – Subjects were matched for body composition and grouped by previous weight fluctuations, (weight cyclers, WC) versus those who have remained weight stable (non-weight cyclers, NWC). Indirect calorimetry, 7-day nutritional intake and activity diaries, three-factor eating questionnaire and fat and fat free mass by dual Xray absorptiometry were measured.

Outcomes – There were no significant differences in resting metabolic rate adjusted for fat mass and fat free mass, however the WC did tend to be lower (4.8 ± 1.0 versus 5.0 ± 1.0 MJ/day). Reported dietary intake was not different. WC had higher levels of restrained eating and disinhibition than NWC (6.7 ± 3.7 vs 3.9 ± 2.9, P=0.04; and 7.8 ± 3.7 vs 5.1 ± 2.2 , P=0.03; respectively). Self reported daily activity was higher in the weight cyclers than the non-weight cyclers (1.7 ± 0.2 vs 1.5 ± 0.2, P=0.03).

Conclusions – Resting metabolic rate did not differ between groups, however will power, resistance to eating cues and daily activity levels did. Therefore, in order for subjects who have dieted to obtain the same body composition they must have compensatory psychological and physical behaviours.