hours. There was a 5-7 day washout period between each injection. For energy expenditure assay rats were housed in a calorimetric chamber for 48 hours before injection.

Results: We observed that acute i.c.v. injections of ASP have a dose dependent effect on food intake. ASP injection interestingly diminishes food consumption by 20% to 50% ($P< 0.05$) at according to time and concentration over a 24 hour span. Furthermore, acute ASP injection affected energy substrate usage demonstrated by a lower RQ, and affected movement with a 49% decrease in total activity but there was no change in oxygen consumption (V_{O2}).

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A novel protein phosphatase CIPP mediates leptin-induced suppression of AMPK activity in the arcuate hypothalamus by inhibiting CaMKK activity

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Introduction: Leptin is an adipokine that regulates energy metabolism. We have previously shown that leptin inhibits food intake by suppressing the activity of AMP kinase (AMPK) in the arcuate (ARH) and paraventricular (PVH) hypothalamus, whereas it stimulates fatty acid oxidation in muscle by activating AMPK in this tissue. Here we found that a novel protein phosphatase, CIPP (CaMKK-AMPK cascade–inhibitory protein phosphatase), dephosphorylates and suppresses the activity of CaMKKβ in response to leptin, thereby inhibiting AMPK activity, in neuroblastoma cell line SH-SY5Y and in mouse ARH.

Methods: CIPP was found from the NCBI database and expression in SH-SY5Y cells and mouse ARH.

Results: CIPP belongs to the PP2C family and was expressed in human brain as well as SH-SY5Y cells and mouse ARH, but not in mouse or human skeletal muscle. CIPP was expressed in a part of NPY neurons in the ARH. CIPP dephosphorylated threonine residue of CaMKKβ, then suppressing its activity in vitro. CIPP siRNA suppressed leptin-induced inhibition of CaMKKβ and AMPK activity in SH-SY5Y cells. Leptin induced the phosphorylation of CIPP in a manner dependent on PI3-kinase and Akt as well as the recruitment of CaMKKβ to CIPP, thereby triggering the dephosphorylation of CaMKKβ and AMPK, in SH-SY5Y cells and mouse ARH.

Conclusion: Our results suggest that CIPP mediates inhibition of AMPK activity in the ARH by leptin via inhibition of CaMKKβ activity. Furthermore, the tissue distribution of CIPP may underlie the mechanism by which leptin reciprocally regulates AMPK activity in the ARH and skeletal muscle.

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Effect of bite size and oral processing time on satiation

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Introduction: In previous studies we showed a clear effect of viscosity on satiation/ad libitum food intake (1). So far, the underlying mechanism responsible for differences in satiation responses between liquids and solids is not clear. In other previous studies we found indications that oral processing time (OPT) and bite size could play a role (2). Therefore, our objective was to determine the effect of bite size and OPT on ad libitum food intake.

Methods: 22 healthy subjects participated in all 7 conditions of the study. Bite sizes were free or fixed to small bite sizes (approximately 5 gram) or large bite sizes (approximately 15 gram). OPT was free or fixed to 3 or 9 seconds. Subjects consumed chocolate custard with a peristaltic pump to control bite sizes. Sound signals indicated duration of OPT.

Results: Preliminary analyses show a significant effect of bite size ($P<0.05$). In the small bite size conditions, ad libitum intakes were 380±198 gram (3sec OPT) and 312±170 (9sec OPT). In the large bite size conditions, ad libitum intakes were much higher, 475±176 (3sec OPT) and 432±163 (9sec OPT) gram.

Conclusion: Larger bite sizes led to a significant higher food intake.

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The acute effects of vinegar supplementation on appetite and glycaemic response

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Introduction: Previous studies have reported acetate ingestion (delivered in vinegar) significantly increases satiety. However these studies failed to quantitatively assess effects on satiety and to account for the taste effects of vinegar on appetite. The present study therefore aims to address these methodological weaknesses.

Methods: Sixteen unrestrained eaters (three male, thirteen female) aged 22.2 [SD 3.0] years were recruited to this randomised single-blind cross-over study. Participants attended three study sessions where they consumed a standard breakfast alongside an unpalatable (Unpal) or more palatable (Pal) drink containing 25g vinegar or a drink without added vinegar (PL). Effects on appetite were assessed subjectively using visual analogue scales (VAS) and assessed quantitatively by providing a pre-weighed ad libitum pasta meal 3h post-prandially. Capillary blood samples were regularly collected by fingerprick to monitor the post-prandial glycaemic response.

Results: Pal and Unpal vinegar treatment significantly reduced subjective appetite ratings for the desire to eat ($p=0.036$) and hunger ($p=0.045$) and increased fullness ($p=0.000$) and also nausea ($p=0.001$) (repeated measures ANOVA) when compared to PL. However no significant effects on quantitative measures of appetite were observed, with a mean intake of 530 [SD 163]g, 497 [SD 154]g and 485 [SD 238]g of the pasta meal following PL, Pal and Unpal respectively. Vinegar treatment was also found to significantly lower the glycaemic response ($p=0.022$, repeated measures ANOVA).

Conclusion: While significant effects on appetite ratings were observed, actual intake 3h post-prandially was not significantly reduced. This suggests vinegar influences appetite only transiently, possibly due to increased nausea.

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