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T1:PS.170

The effects of oleoylethanolamide on feeding behaviour involve hypothalamic oxytocin neurons.

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Oleoylethanolamide (OEA) is the monounsaturated analogue of the endocannabinoid anandamide. Differently from anandamide, which causes overeating and stimulate lipogenesis by activating CB1 receptors, OEA decreases food intake and body weight gain in rats and mice through a cannabinoid receptor-independent mechanism. The effects of OEA on feeding are behaviourally selective and are due to the prolongation of feeding latency and post meal interval. A large body of evidence indicate that they are mediated by the activation of peripheral PPAR-alpha receptors, but the central mechanisms downstream to this activation that are still unclear.

Data obtained mapping brain c-fos mRNA levels revealed that the systemic administration of OEA evokes highly localized increase of c-fos transcription in the nucleus of the solitary tract (which is in accordance with the peripheral action of OEA), the paraventricular nucleus (PVN) and the sopraoptic nucleus (SO). The magnocellular components of both nuclei release oxytocin, one of the anorectic hypothalamic neuropeptides. During feeding, magnocellular oxytocin neurons, especially those in the SO, become strongly activated indicating their imminent role in meal termination. We hypothesized that oxytocin neurons, might play a key role in regulating energy intake after OEA administration.

In agreement with our hypothesis, we found that OEA enhances the gene expression of oxytocin in both areas and that its anorexiant action can be prevented by pretreatment with a selective oxytocin receptor antagonist. Our data suggest that oxytocin release in the PVN and SO nuclei may be responsible for the mediation of the effects induced by OEA on feeding.

T1:PS.172

Do overweight subjects show consistency on *adlibitum* food consumption and self-reported food intake in laboratory intervention studies? Effect of a protein-containing liquid preload on voluntary food intake at a subsequent meal

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Background: Recent studies indicate that certain types of milk protein (eg whey) may be more satiating than others (eg Casein). However, most studies on this topic have been carried out in normal-weight individuals and their replication might not be feasible in overweight/obese subjects.

Methods: Six healthy men and two women, BMI 27-32 kg/m², aged 18-45 yrs, not dieting and weight-stable during the previous three months were studied. Women were studied during the first phase of their menstrual cycle.

The day of the study participants consumed a standardized breakfast providing 10% of their daily energy requirements. In a randomized cross-over design, 150 minutes after breakfast, participants consumed either a 250 kcal (40% of energy from whey-protein) or a control non-energy flavoured liquid preload (400 ml), with each being repeated on two occasions. Ninety minutes later, adlibitum food intake was assessed at lunch. Visual analogue scales for subjective appetite were scored every 30 minutes. Food intake during the remaining of the day was also reported. Treatments were repeated within a week to assess consistency of results.

Results: Eating at lunch and during the remainder of the day was consistent for the repeated exposure of each arm of the study. Energy intake at lunchtime was 115 kcals lower after whey-protein, representing 46% of the preload energy content. No significant differences in appetite ratings were observed between treatments.

Conclusions: Our results in overweight/obese subjects participating in a laboratory study showed consistency in ad-libitum and self-reported food intake.

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T1.PS 171

Variation in postprandial peptide YY $_{\rm 3.36}$ status following ingestion of high carbohydrate, high fat and high protein meals in obese females

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Aim: This study investigates the effect of macronutrient composition of meals on postprandial peptideYY $_{3,36}$ (PYY $_{3,36}$) response, in obese hyperinsulinemic females. **Methods:** Eight obese females consumed three iso-energetic meals of different

Methods: Eight obese females consumed three iso-energetic meals of different macronutrient composition, a high carbohydrate (HC) (60% CHO, 20% protein, 20% fat), a high fat (HF) (30% CHO, 20% protein, 50% fat) and a high protein (HP) (30% CHO, 50% protein, 20% fat), on three separate occasions, 1 month apart. PYY_{3.36}, insulin and glucose were measured before, and 15, 30, 60, 120 and 180min following each meal.

Results: PYY₃₋₃₆ levels increased significantly following the three meals with the HC meal resulting in a sustained postprandial increase in PYY level throughout the experimental period. Comparing the three meals, the HF meal induced a significantly higher increase in postprandial PYY₃₋₃₆ levels, at 15 and 30min as compared to the HP (p <0.05). whereas the postprandial increase following the HP meal became significantly higher than that of the HF meal at 120min. Postprandial increase in PYY was highest in the first hour following the HF meal, while that of HP meal was delayed by one hour.

Conclusion: Increasing both protein and fat content of a meal may induce an immediate and prolonged increase in PYY_{3.36}, resulting in increased satiety and its maintenance for a longer period of time.

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T1:PS.173

Baseline leptin levels affect the response of leptin to 6 months of aerobic exercise training.

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There is inconsistency regarding whether aerobic exercise training (ET) causes a reduction in leptin in weight stable humans. We speculate this may be due to the variability of leptin levels within the studied populations. The purpose of this study was to examine whether baseline leptin levels affect the response of leptin to ET

Ninety seven previously sedentary individuals underwent a 6 month progressive and supervised ET program (60-85% VO2max, 45 minutes per day, 4 days per week). Blood was sampled for the measurement of leptin prior to ET, 24 and 48 hours after completion of the final ET session. All participants were instructed to maintain normal eating habits in order to maintain weight.

ET resulted in a small reduction in body mass (80.47 ± 18.03 vs 79.42 ± 17.34 kg, p<0.01). Leptin was reduced 24 hours post, but returned to baseline values 72 hours post (Pre: 13.51 ± 12.27 , 24hr: 12.14 ± 12.34 , 72hr: 12.98 ± 11.40 ng/ml).

We compared the lowest 10% (n=9) of baseline leptin to the highest 10% (n=9). There was a significant time X baseline leptin interaction whereby the highest 10% did not follow the typical pattern of increasing leptin between 24 to 72 hour post-training. We expanded the subpopulation to the lowest (n=24) and highest quartiles (n=24) of baseline leptin. Despite the larger sample size the time X baseline leptin interaction disappeared These data suggest that ET is successful in reducing leptin levels only in those whose baseline levels are the highest.