Complex homeostatic attributes of the forebrain glucose-monitoring neuronal network

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To study involvement of the forebrain glucose-monitoring (GM) neuronal network in the control of homeostasis, series of electrophysiological and behavioral experiments have been conducted in the rodent and macaque monkey. Single neuron recordings in anesthetized rats and alert primates elucidated characteristic activities of forebrain GM cells during: (1) microelectrophoretic administration of chemicals (such as the diabetes inducing streptozotocin (STZ) or the primary cytokine interleukin 1beta (IL-1)); (2) gustatory stimulation, as well as (3) performing behavioral tasks. A single microinjection of STZ into various forebrain sites of Wistar rats caused type 2 diabetes like metabolic disturbances, and that of IL-1 was followed by hypophagia, hyperthermia, and metabolic alterations as well. These findings - along with results of our most recent fMRI studies in the rhesus monkey - indicate that intact functioning of the forebrain GM neuronal network is indispensable to protect the maintenance of homeostasis against the impact of environmental challenges.

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A cognitive-experimental approach to reducing food cravings and modifying eating behaviour

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Food cravings have been identified as an important precursor to binge eating, itself a risk factor for obesity and eating disorders. Based on converging evidence that visual and olfactory images are key components of food cravings, this paper used a cognitive-experimental approach to suppress such cravings and modify subsequent eating behaviour. In each of 4 experiments, 90 undergraduate women (18-30 years) underwent a craving induction procedure involving a combination of chocolate deprivation and exposure to chocolate cues. They then performed either a visual, auditory or olfactory imagery task. Participants rated their chocolate craving intensity after the craving induction, and again after the imagery task. Following the imagery task, participants also completed one of four behavioural measures of food craving: a so-called chocolate tasting task (Exp. 1), latency to begin a tasterating task (Exp. 2), speed of chocolate consumption (Exp. 3), and a salivation measure (Exp. 4). As predicted, the visual and olfactory imagery tasks were consistently superior to the auditory imagery task in reducing participants' craving for chocolate. However, these self-reported reductions in craving did not correspond to any objective behavioural indicator. A behavioural index of craving that is completely devoid of food or eating connotations could perhaps prove more successful. Nevertheless, imagery-based techniques in the visual and olfactory domain offer potential scope for reducing food cravings in the context of problem eating behaviour. doi:10.1016/j.appet.2008.04.126

Ratings of fullness after ad libitum meals are not predicted from ratings of fullness during interrupted meals in pre-school children

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This study was conducted to determine whether ratings of fullness during meals, interrupted at fixed increments, predict fullness during an ad libitum meal in pre-school children. Demonstration of such a relationship would enable ratings of fullness to predict ad libitum intake, thereby greatly facilitating measurements of the satiating effects of foods. Eleven kindergarten students (four girls, seven boys; ages 5-6 years) were tested in five, 1-h sessions, during their usual lunch time. After training them to rate fullness with pictures of varying sized portions of foods with a child-friendly scaling device [Appetite 47:233-243], children rated how full they felt after consuming each of 15, 15-ml portions of strawberry yogurt shake to a maximum of 450 cal, for four sessions. During the fifth session, they consumed fifteen 1-ml portions of the strawberry yogurt shake and then ate ad libitum from 800 g of yogurt shake served in a closed container. After the second session, 8 of the 11 children reliably increased ratings of fullness per unit eaten (*p*'s < 0.05 on both days). However, the fullness ratings predicted for the ad libitum meal size, from the equations for ratings on the last two interrupted meals, were only marginally correlated (Pearson's r = 0.45, p = 0.26, Spearman's r = 0.61, p = 0.11). Physiological or environmental contextual controls of ratings may be different than during interrupted, than during ad libitum, uninterrupted, meals, and one cannot rely completely on one, to predict the other.

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Orosensory stimulation during modified sham feeding increases intake of sweet solutions more in women with bulimia than women with anorexia nervosa

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Although it is possible that abnormal meal size in humans is due to altered responsiveness of orosensory excitatory controls of eating, there is no direct evidence for this because food ingested in a test meal stimulates orosensory excitatory and postingestive inhibitory controls. We adapted the modified sham feeding (MSF) technique to measure orosensory excitatory control of intake of a series of sweetened solutions in the absence of postingestive negative feedback of ingested solution. Previously presented data showed that women with bulimia nervosa (BN) sipped more solution than women without an eating disorder in this procedure. In the present study, 14 women with anorexia nervosa (AN) were randomly presented with cherry Kool Aid® solutions sweetened with one of five concentrations of aspartame in a closed opaque container fitted with a straw. They were instructed to sip as much as they wanted of the liquid during 1-min trials and to spit the fluid into another opaque container, for 15 solutions per subject. Across all subjects, sweetener presence increased intake. Women with BN sipped more, and those with AN sipped less solution than controls. Over three trials, intake increased in women with BN, decreased in AN, and was stable in controls. Results further validate this MSF procedure, its ability to distinguish among eating disorder diagnoses, and hypotheses that women with BN have increased orosensory excitation, while non-binge eating women with AN, do not. doi:10.1016/j.appet.2008.04.128

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