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Satellite of the Congress of the Society of the Study of Ingestive Behaviour, Maastricht, The Netherlands

Control of food intake in man

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The Society for the Study of Ingestive Behaviour meets each year to discuss and highlight recent developments in our understanding of all aspects of controls of eating and drinking. The aim of this satellite was to focus on key issues in human food intake control: palatability, central and peripheral signalling, dietary restraint, macronutrients, energy density, heritability and the significance of animal models for research in man, and to bring together researchers approaching these issues from different perspectives. The presentations including live discussions are reflected in the following papers.

The traditional view of palatability as part of a homeostatically driven motivational system indicating nutritional deficit does not fit with the common observation of palatability leading to short-term over-consumption. If anything, palatability exerts a stronger stimulatory effect on eating when sated, and over-consumption induced by palatability may contribute to obesity. Differential responsivity to palatability may be a component of the obese phenotype, perhaps through sensitisation of the neural structures related to hedonic aspects of eating. Together, Yeomans, Blundell and Leshem conclude that these disparate data clearly indicate that palatability is not a simple reflection of need state, but acts to promote intake through a distinct hedonic system that has inputs from a variety of other systems, including those reflecting need. Potential developments of novel therapies for obesity based on modulation of hedonic rather than homeostatic controls are discussed.

'Dieting makes you fat' deals with the reported failure of most dietary approaches in the treatment of obesity. Baseline dieting or dietary restraint increases risk of weight gain, especially in women, probably due to metabolic adaptations and disinhibited eating. Dietary restraint has also been implicated in the development and persistence of binge eating. Hill critically evaluates the evidence supporting this paradox and reaches the conclusion that it is not dieting that makes you fat, but that being fat makes you (more likely to) diet. The assertion that dieting makes you fat fails to recognise that people who successfully control their weight are often misclassified

as non-dieters and that obesity causes dieting rather than *vice versa*. With regard to the same issue, Lowe & Timko state that 'dieting' can refer to a variety of behavioural patterns that are associated with different effects on eating and body weight. Depending on what one means by the term, dieting can be quite harmful, merely ineffective or actually beneficial. The paper considers examples of all three.

The role of several physico-chemical properties of carbohydrate and fat molecules, which appear to influence the short-term satiating properties of these macronutrients, is discussed by French, concluding that long-term substantiation of these findings expressed either in terms of food intake or body weight is required to make clear recommendations regarding dietary composition to aid satiety. Tome continues on the role of protein, listing the most commonly accepted conclusions: (1) the existence of an aversive response to diets deficient or devoid in protein or at least in one essential amino acid; (2) the existence of a mechanism allowing organisms to reach the minimum requirement for N and essential amino acids by increasing a low-protein diet intake; (3) a decrease in the intake of a high-protein diet associated with different processes including the high satiating effect of protein.

Yeomans' main conclusion from the contribution on alcohol was that there is minimal evidence for any compensatory reduction in food intake in response to energy ingested as alcohol. In the long-term, energy ingested as alcohol is additive to energy from other sources, suggesting that moderate alcohol consumption results in long-term passive over-consumption alongside

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short-term active over-consumption of energy through appetite stimulation. Despite the consistency of enhanced energy intake after moderate alcohol intake, evidence of an association between alcohol in the diet and obesity remains contentious, and confounding effects are likely. Continuing with energy-density effects, Westerterp-Plantenga concludes that changing energy density of food due to changes in macronutrient composition affects energy intake; however, when energy density is only affected by dilution with water, it does not relate to energy intake anymore. Moreover, in the long-term, dietary restraint and portion size are tools to counteract energy density effects.

On central and peripheral issues, Hellström, Geliebter, Näslund, Thelin Schmidt, Yahav, Hashim and Yeomans examine evidence of defects in the signalling network underlying hunger, satiety and metabolic status, including the hormonal signals leptin and insulin from energy stores and cholecystokinin, glucagon-like peptide-1, ghrelin and peptide YY3-36 from the gastrointestinal tract as well as neuronal influences via the vagus nerve from the digestive tract. They conclude that understanding the host of neuropeptides and peptide hormones through which hunger and satiety operate should lead to novel therapeutic approaches for obesity, and potential therapeutic strategies are highlighted.

At this point, the significance of animal studies for research on man is relevant, and highlighted by Thibault,

Woods and Westerterp-Plantenga. Then de Castro shows how recent evidence suggests that genes exert multiple and subtle influences on the control of food intake in human subjects. Tremblay, Pérusse and Bouchard state that from a clinical standpoint, the effect of gene–environment interactions on energy balance implies that some individuals are more susceptible to body-weight gain or loss than others because of genetic differences. This opens new perspectives in predictive medicine.

Overall, the papers arising from this special satellite meeting provide excellent summaries of the current state of knowledge of these diverse, but interrelated, aspects of the control of human appetite, and also highlight key recent findings. The success of the meeting is reflected in the quality and breadth of the following papers, and we would like to thank all the speakers and the members of the audience who joined in the discussions for their contributions.

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