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ABSTRACT

The biology of appetite control – body composition, resting metabolic rate and the drive to eat

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An unresolved problem relevant for the control of food intake and obesity in the social environment is ‘what constitutes the Drive to Eat’. More than 50 years ago it was proposed that ‘the differences between the intakes of food must originate in the differences in energy expenditure’ and that this relationship could form the basis for appetite control (Edholm, Fletcher, Widdowson and McCance et al, 1955 p 297). However the zeitgeist of that time was focussed on the search for signals that would control food intake (satiety) and regulate body weight. This notion was exemplified by the adipocentric theory which, in turn, appeared to have been confirmed by the discovery of leptin in 1994. However this concept leaves unanswered many questions related to obesity and cannot account for the Drive to eat. Moreover, the attention directed to the study of adipose tissues may be misplaced. Over a 15 year period, we have investigated the drive and inhibition of eating by studying appetite control within an energy balance framework using a multi-level experimental platform (Caudwell et al, 2011). In several cohorts of obese subjects (male and female) it has been demonstrated that FFM, but not FM or BMI, has a positive relationship with self-determined meal size and daily EI (Blundell et al, 2011). This finding has been independently confirmed by several independent research groups (eg Weisse et al, 2013). FFM is the primary determinant of RMR and accounts for about 60% of the variance (FM accounts for approximately 7%). RMR has also been shown to be positively associated with meal size and daily EI (Caudwell et al, 2013) and with the profile of hunger across the day. An interpretation of these findings is that the body’s energy requirements contribute to the motivation underlying food intake, and suggest that the ‘regulation’ of food intake is not entirely under the control of adipose tissue, and that the model for control of food intake should be revised (Blundell et al, 2012). We propose that adipose tissue has a biologically inhibitory effect on EI but this effect weakens (permit-

ting overconsumption) as AT mass increases (and insulin and leptin sensitivity decrease). This formulation helps to explain why obese people continue to eat more than normal weight people and to experience strong hunger despite having large stores of energy in the body. These observations suggest that energy intake is driven in response to the body's energy requirements to maintain vital biological functions. This demand for energy should be considered as one of the drivers of appetite. We have extended these findings by objectively measuring key variables in the energy balance budget in a large heterogenous cohort of men and women (n = 399), and modelling their inter-relationships. FFM and RMR have emerged as strong predictors of EI. FM is negatively associated with EI and has both direct and indirect effects. The model indicates that EI (food intake behaviour) is influenced by biological, psychological and nutritional variables. This formulation has implications for understanding obesity in the social environment.

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