

Letter to the Editor

Dieting. Makes you fat?

Two articles have been published in Nutrition Society journals explicitly responding to the theory that ‘dieting makes you fat’.

Of these one reported results that evidently contradicted a rationale of the theory (Prentice *et al.* 1991). The other displays results from nine studies from seven centres, of which eight conclude that dieting increases the risk of weight gain, warns that ‘a great deal of effort is required to counter Cannon’s paradox’ and proposes as an alternative explanation that (being) fat makes you diet (Hill, 2004).

Dieting Makes You Fat is the title of a popular book now out of print (Cannon & Einzig, 1984), so readers may be perplexed.

One origin of the theory was a reading of the classic ‘Minnesota experiment’ (Keys *et al.* 1950), in which thirty-six male objectors to military service were kept on a diet of around 50% usual energy intake in a controlled environment. After 24 weeks average weight loss was 24%, and average RMR had dropped by 39%.

Some of the subjects were then retained and observed. With free access to food they gorged, up to and sometimes beyond their original body weight; but were not followed up long-term. The theory proposes that this ‘rebounding’ effect is part of a whole syndrome that is not merely ‘psychological’, and is the result of an adaptive drive with an evolutionary rationale and identifiable physiological and biochemical analogues.

Some studies of metabolic and other responses during and after energy restriction conducted after Keys are cited in a UK report on obesity, which commented that: ‘the body is able to adapt to changes in energy intake’ (Royal College of Physicians, 1983). More recent studies formed the basis of a judgement that ‘rigid restraint/periodic disinhibition’ eating patterns are a possible cause of obesity (World Health Organization, 2003).

Such tentative conclusions await the results of adequately conducted controlled trials. But trials designed to identify relevant adaptive mechanisms would need to be comparably demanding with that of Keys and colleagues, and it is not obvious who would want to fund the studies and who would want to endure them. Many questions remain unanswered in the literature for such reasons. The study using modern techniques of measurement whose results seemed to refute one aspect of the theory, that a physiological explanation is permanent depression of metabolic rate (Prentice *et al.* 1991), may not have been sufficiently restrictive or conducted for long enough to induce relevant changes such as in composition of lean tissue, and I believe was not followed up long-term.

‘Dieting makes you fat’ is a general theory proposed as the best fit with the facts. After the book was written, Hugh Sinclair, who at one stage in his remarkable career bred pigs, reported that the most effective way to fatten animals is to starve them before penning and feeding them (H. Sinclair, unpublished results). Further, health professionals in the South report that children undernourished as infants tend to gorge; in Brazil this is known

as *fome histórica* or ‘historic hunger’ (R. Bittar, unpublished results).

The theory is guided by the principle that: ‘in biology, nothing makes sense except in the light of evolution’ (Dobzhansky, 1973). Thus, the body is evolved to adapt to periods of energy restriction as if these are periods of scarcity or famine, by means of mechanisms that after the restriction is over, trigger hunger, inhibit satiety and preferentially conserve body fat. ‘From an evolutionary point of view it makes sense that the body energy stores are defended during times of famine... and that in times of food surplus the essential requirements of the body can be met rapidly’ (van Baak, 2004). Thus the tendency for stunted children exposed to energy-dense diets to become unusually fat, findings that ‘help to clarify the greater susceptibility to obesity (and related disorders) evidenced in countries under nutritional transition’ (Martins *et al.* 2004). Indeed, it is hard to see how *Homo sapiens* could have evolved and survived without some such adaptive mechanisms.

As indicated, the theory does not only apply to dieting, which might be described as voluntary human energy restriction undertaken with the intention of losing body fat. It applies to all forms of energy restriction, including *in utero* and/or during infancy, or experienced for whatever reason, including famine, starvation and fasting. It can be seen as explaining the anorexia–bulimia syndrome and the ‘Barker hypothesis’ (Barker, 1998). It applies to laboratory, domestic and agricultural animals. It does not propose that one session of dieting (etc.) is bound to have this effect; its focus is substantial, regular, sustained energy restriction of any type.

Further, the proposed adaptive drive has the effect of storing excess fat and so becomes pathogenic, and thus in effect maladaptive, only in conditions when, after the period of energy restriction, the subjects have free access to food and drink *ad libitum*, in particular when the food and drink is energy-dense, and most of all when the subjects are sedentary and therefore can be described as being in unnaturally low energy balance. It is only recently in human history that these three conditions have often been met, but these are now the typical human circumstances in most countries in the world.

In a sentence, the thesis, which fits with a range of facts and observations, is that sustained or regular energy restriction followed by feeding *ad libitum*, in particular of energy-dense diets and most of all when subjects are physically inactive, causes additional deposition of body fat with its sequelae.

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