Does dieting make you fat?

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'Dieting makes you fat'. This extraordinary proposal was the title of a book published in the UK in 1983. Written with ample expert consultation, Cannon & Einzig (1983) dedicated it to 'the scores of millions of people in the West who are fatter than they want to be, who have tried dieting, who have found that dieting does not work, and who want to know why.' A blend of personal experience, science and contemporary opinion, this was among the first books on non-dieting weight control. In many ways it was a book of its time. Publication coincided with the advent of an increasingly vocal anti-diet movement, the early signs of increasing adult obesity, and a change in focus from food and eating to physical activity and exercise.

The book's legacy is not its content, but a four-word paradox. It suggests that a behaviour intended to facilitate weight loss actually has the opposite effect. As such, it describes a bleak causal association congruent with an increasingly negative depiction of dieting. Research has associated dieting with negative psychological states, such as increases in depression and anxiety (Warren & Cooper, 1988). Dieters have deficits in certain aspects of cognitive performance (Green & Rogers, 1995). Dieters are also at increased risk of eating disorders (Patton et al. 1999), and even increased morbidity and mortality (Blair et al. 1993).

The evidence supporting these conclusions has been reviewed several times (e.g. Brownell & Rodin, 1994; French & Jeffery, 1994; Gregg & Williamson, 2002). While not fully absolved, dieting appears to have been given a broad safety certificate. Nevertheless, the well-documented failure of most dietary approaches in the treatment of obesity, in conjunction with personal experience of the difficulties of energy-deficit diets, has done little to enhance the reputation of dieting.

Nor has the diet industry done much to raise consumer confidence. Two reports published recently, intended for the diet industry itself, make interesting, if expensive, reading. Retailing at nearly $2000 and $5000 respectively, they reveal an annual turnover of $39 billion in the US diet industry (MarketData, 2002), and €93 billion in the equivalent European industry in 2002 (Datamonitor, 2003). Headline statements include, 'In 2002, 231 million Europeans attempted some form of diet. Of these only 1% will achieve permanent weight loss.' A degree of cynicism appears justified. The size of the diet industry has grown commensurate with the rise in population obesity. The previous quotation confirms that the products sold by the diet industry are known to be ineffective. Indeed, a newcomer to this area may be justified in concluding that industry is contributing to the problem. From a purely business perspective, a diet industry that marketed products yielding safe and sustainable weight loss would commit long-term financial suicide. Moreover, the diet industry is one of several major and global concerns, including agriculture, pharmaceuticals, and the food industry itself, that make a profit out of obesity (Nestle, 2003). For the diet industry to regain the trust of consumers (Datamonitor, 2003), a great deal of effort is required to counter Cannon's paradox.

Dieting and weight change

Cross-sectional studies say little about the causal association between dieting and weight change. Prospective studies can be more informative, but few have been designed specifically to test this association. Table 1 summarises the outcomes of investigations that have used data collected from special cohorts or that have addressed this association directly. All but one of the nine studies...
et al. investigate obesity onset was that by Stice et al. risk compared with non-dieters. In fact, the only study to 0·5 kg per year or, in predictive models, a doubling of modest, amounting to an approximate greater gain of associated with increased risk of weight gain, sometimes exclusively in women. The magnitude of risk is rather included show that baseline dieting or dietary restraint was associated with increased risk of weight gain, sometimes modestly, amounting to an approximate greater gain of 0·5 kg per year or, in predictive models, a doubling of risk compared with non-dieters. In fact, the only study to investigate obesity onset was that by Stice et al. (1999). On the face of it, this represents the clearest empirical evidence that dieting facilitates weight gain.

In terms of mechanisms to account for the failure of dieting to bring about weight loss (and that potentially also explain weight gain), two have been prominent. One concerns the alterations in metabolic rate and energy requirements associated with energy-deficient diets. Much has been made in the popular press regarding the degree and persistence of these physiological responses and their potential to facilitate post-dieting weight rebound and gain. Prentice et al. reviewed the issue in 1991 and reached conclusions that still hold more than 10 years later:

1. BMR is rarely suppressed by more than 20%;
2. very-low-energy diets suppress metabolic rate more than moderate energy-deficit diets;
3. physical exercise probably has a protective effect;
4. after dieting BMR returns to a level commensurate with the new body size.

**Dietary restraint**

The second explanatory mechanism is inferred by the laboratory work on dietary restraint pioneered by Herman, Polivy and their colleagues. Take one of their recent studies as an example. A group of female undergraduate students was recruited to a study ostensibly about the effects of food deprivation on taste perception (Urbszat et al. 2002). Half of the participants were assigned to a diet condition and told that immediately after the study they would start on a 7d low-fat, energy-reduced diet. Along with the diet plan was a list of forbidden foods. Participants were presented with three plates of cookies (one of the forbidden foods) that they had to taste and rate, and were left alone for 10 minutes with the instruction, ‘help yourself … we have tons.’ Participants then completed a dietary restraint scale and the cookie plates were re-weighed.

The outcome is summarised in Fig. 1. Unrestrained female subjects were unresponsive to the diet manipulation. Restrained eaters ate least in the no-diet condition, but significantly more (than all three other groups) in the anticipated diet condition. This ‘last supper effect’ as a response to anticipated deprivation is just one example of the breakdown of dieting that has been described in this elegant and replicable research scenario. Disinhibition, or the loss of inhibition, is the term that describes this self-regulatory failure. The cognitive explanatory model borrowed from the addictions literature is the abstinence violation effect. Rigid and dichotomous thinking, in this

![Fig. 1. Food intake by unrestrained and restrained eaters in a no-diet condition (●) and faced with starting a diet after the study (■). (Re-drawn from Urbszat et al. 2002.)](https://www.cambridge.org/core/terms)
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case about eating, fosters catastrophising of a single lapse or the prospect of prolonged food deprivation. The ‘what-the-hell’ reasoning is familiar to most people in relation to a range of different substances. One of the triumphs of the dietary restraint literature has been the capacity to show the disinhibiting effects of a variety of factors ranging from negative effect, through social processes, to beliefs about the energy content of food.

The value of this literature has been augmented by the proposed association between dieting and binge eating (Polivy & Herman, 1985). Disinhibited eating by restrained eaters has been argued as a laboratory analogue of binge eating: periods of dietary restriction alternating with episodes of uncontrolled overeating. Dieting is also implicated in the persistence of binge eating. Fairburn’s cognitive model of maintenance, used in cognitive behavioural therapy for the disorder, places dieting at the centre, albeit rigid and intense dieting (Fairburn, 2002). Empirical research support, however, is a mixed affair with no clear support for an association, organising the studies either by methodology (cohort vs. prospective studies) or clinical group (eating disorders, obesity). The one exception is research looking at binge-eating onset in adolescent girls.

Two studies using statistical modelling have shown that dieting is one of several factors that are predictive of binge-eating psychopathology onset (Stice & Agras, 1998; Stice, 2001). A third study has shown moderate dieters to be five times more likely than their non-dieting peers to develop an eating disorder, in nearly all cases partial syndrome bulimia nervosa (Patton et al., 1999).

Convincing evidence?

Any consideration of dieting engenders a huge emotional response, as it is clouded by people’s own experience of failure and anticipated accusations of blame for their overweight (Hill, 2003). Accusing the diet industry of contributing to obesity may be one such expression. But just how convincing is the evidence presented earlier? Relating dieting to binge-eating onset is poor support for the contention that dieting leads to weight gain or obesity onset. Weight is an uncertain factor in this small literature. Indeed, Stice argues that dieting is merely a proxy for more important predictive variables in relation to eating disorders, let alone weight gain (Stice, 2002).

Laboratory studies of disinhibited eating by highly restrained individuals only hint at what might lead to weight gain. There is vigorous debate as to whether different restraint processes, to beliefs about the energy content of food.

The most obvious conclusion from this review is not that dieting makes you fat, but that being fat makes you (more likely to) diet. That the research literature fails to substantiate the success claimed by some weight-loss products should not lead us to reinforce a causal association that is naive and inaccurate. 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. Most importantly, it underplays the roles of biology and the environment in the determination of weight gain and as barriers to sustained weight loss.

References


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