Does dieting make you fat?

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Dieting Makes You Fat, the title of a 1980s book on weight control, is a popularised paradox, conveying a conclusion that is consistent with personal experience and the reported failure of most dietary approaches in the treatment of obesity. Few studies have been designed specifically to test this association. Yet there are prospective data showing that baseline dieting or dietary restraint increases the risk of weight gain, especially in women. Metabolic adaptations and the disinhibited eating of restrained eaters have been the most commonly cited explanations for such weight gain. Dietary restraint has also been implicated in the development and persistence of binge eating. The present paper critically evaluates the evidence supporting this paradox and reaches a rather different conclusion.

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

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included show that baseline dieting or dietary restraint was associated with increased risk of weight gain, sometimes exclusively in women. The magnitude of risk is rather modest, amounting to an approximate greater gain of 0·5 kg per year or, in predictive models, a doubling of modest, amounting to an approximate greater gain. The magnitude of risk is rather associated with increased risk of weight gain, sometimes included show that baseline dieting or dietary restraint was exclusively in women. The magnitude of risk is rather modest, amounting to an approximate greater gain of 0·5 kg per year or, in predictive models, a doubling of modest, amounting to an approximate greater gain.

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 7 d 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 they would start on a 7 d 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.

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### Table 1. Prospective studies showing the relationship between dieting or dietary restraint and weight change

<table>
<thead>
<tr>
<th>Authors</th>
<th>Cohort</th>
<th>Time period</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>French et al. (1994)</td>
<td>Healthy Worker Project: 3553 adults in smoking-cessation and weight-control worksite intervention</td>
<td>2 years</td>
<td>Baseline dieters gained approximately 1 kg more than non-dieters (women only)</td>
</tr>
<tr>
<td>McGuire et al. (1999)</td>
<td>National Weight Control Registry: 714 successfully maintaining weight losers</td>
<td>1 year</td>
<td>Baseline dieting one of several predictors of weight gain</td>
</tr>
<tr>
<td>Korkeila et al. (1999)</td>
<td>Finnish Twin Cohort: 7729 members of twin pairs</td>
<td>6 and 15 years</td>
<td>Baseline dieters had two times the risk of major weight gain (&gt;10 kg) than non-dieters (younger men and women aged 30 years only)</td>
</tr>
<tr>
<td>Juhaeri et al. (2001)</td>
<td>Atherosclerosis Risk in Communities (ARIC) study: 10 554 white and African American adults</td>
<td>6 years</td>
<td>Baseline dieters gained approximately 0·5 kg/year more than non-dieters</td>
</tr>
<tr>
<td>Drapeau et al. (2003)</td>
<td>Quebec Family Study: subsample of 75 adults</td>
<td>6 years</td>
<td>Baseline TFEQ, dietary restraint associated with weight gain (women only)</td>
</tr>
<tr>
<td>Klesges et al. (1989)</td>
<td>Young adults (n 65)</td>
<td>8 d (US Thanksgiving holiday)</td>
<td>Baseline dietary restrained gained more weight than non-restrained</td>
</tr>
<tr>
<td>Klesges et al. (1991)</td>
<td>Young adults (n 305)</td>
<td>2 years</td>
<td>Baseline dietary restraint not related to weight change</td>
</tr>
<tr>
<td>Klesges et al. (1992)</td>
<td>Adults in study of cardiovascular risk (n 287)</td>
<td>1 year</td>
<td>Baseline dietary restraint a predictor of weight gain (women only)</td>
</tr>
<tr>
<td>Stice et al. (1999)</td>
<td>Adolescent girls (n 692)</td>
<td>3 years</td>
<td>Baseline dietary restraint (two times risk) and dieting (three times risk) predictors of obesity onset</td>
</tr>
</tbody>
</table>

TFEQ, three-factor eating questionnaire.
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, and as barriers to sustained weight loss.

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 restrained individuals only hint at what might lead to weight gain. There is vigorous debate as to whether different restraint factors ranging from negative effect, through social processes, to beliefs about the energy content of food.

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 scales measure the same psychological tendency, and how this relates to weight-loss dieting. Dieting itself varies between studies in terms of its intensity, duration and application (Hill, 2002). Moreover, dieting is commonly part of a weight-loss package making partitioning of component contributions to weight change very difficult.

Perhaps most telling is a closer examination of the studies summarised in Table 1. Take, for example, the Finnish Twin cohort study (Korkia & et al. 1999). Detailed inspection of these twins showed that dieting aggregated in families, suggesting a familial predisposition to gain weight. The most parsimonious explanation is that this predisposition accounted for higher levels of dieting and ultimately overwhelmed weight-loss attempts. Similarly, examination of the study relating adolescent dieting to obesity onset (Stice et al. 1999) shows that exercise for weight control also predicted obesity onset. Indeed, all weight control methods were positively correlated with the girls’ BMI at the start of the study.

Cannon’s paradox is unlikely to be a paradox after all. 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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