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Review

Dieting: proxy or cause of future weight gain?

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Summary

The relationship between dieting and body mass has a long and controversial history. This paper aims to help resolve this issue by making two key distinctions. The first is between dieting as a cause of weight gain/regain and as a proxy risk factor for identifying non-obese individuals prone to weight gain for reasons other than dieting. The second is between the body mass that is attained following one or more weight loss/regain cycles and the body mass that might have been reached had dieting never been undertaken. Evidence is reviewed on the relation between recent diet-induced weight loss and sustained weight loss (weight suppression), on the one hand, and weight regain, on the other hand. Furthermore, the reason that a history of dieting in non-obese individuals reflects a susceptibility to future weight gain is explained. It is concluded that (i) diet-induced weight loss hastens weight regain but a history of weight loss diets does not cause weight gain beyond that which would occur in the absence of dieting, and (ii) weight loss dieting in non-obese individuals does not cause future weight gain but is simply a proxy risk factor reflecting a personal vulnerability to weight gain and living in an obesogenic environment.

Keywords: Dieting, obesity, weight gain, weight regain.

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Introduction

Over the past 40 years, there has been a continuing controversy over the relationship between dieting, weight loss and weight gain. The major goal of this paper is to propose a distinction that could help resolve this controversy. Weight loss dieting has been viewed in the past both as a cause of weight regain (1,2) and as a proxy risk factor (3) for future weight gain that merely reflects the operation of variables that are actually responsible for weight gain (4). The first category applies to clinical populations whose diets usually result in significant weight loss (e.g. greater than 5% of starting body weight in obese individuals and often more in eating disordered individuals). In the second category are young, non-obese individuals who have been studied in the vast literature on restrained eating and dieting. These individuals report restricting their eating or dieting to lose weight, but evidence suggests that their efforts are much less likely to produce significant weight losses (5,6). I will argue that in these latter instances, dieting comprises a proxy risk factor that does not exert a causal influence itself but reflects the effects of predispositions that increase susceptibility to weight gain and lead to weight loss dieting in an attempt to limit or reverse weight gains. By clarifying when dieting helps cause weight gain (or regain) and when it constitutes a marker of susceptibility to weight gain from other causes, it is hoped that this perspective will bring greater order to a sometimes conflicting and confusing literature.

Weight loss dieting as a cause of weight regain and future additional weight gain

In this paper, dieting is defined as an intentional effort to create a negative energy balance for the purpose of losing weight, at least in part through the restriction of energy

intake. In considering the relationship between weight loss produced by dieting and subsequent weight change, it is critical to distinguish between weight loss as a cause of weight *regain* and weight loss as a cause of weight gain *beyond* the body weight that would have been attained in the absence of a weight loss. In the first case, there is broad consensus that significant weight loss produces changes in metabolism and food consumption that hasten the regain of lost weight. Weight loss appears to induce compensatory regulatory responses in the realms of both metabolism (1,7) and intake (8,9). One type of evidence for this conclusion is the regularity with which obese individuals who lose weight through lifestyle change programmes regularly regain it, regardless of the specific type of intervention used to induce weight loss. It appears that starting body weight (or body fat) represents a largely inflexible threshold beneath which long-lasting weight reductions are extremely difficult to achieve. A recent study (10) found a correlation of 0.96 between starting body mass index (BMI) and subsequent BMI among a group of obese individuals who dieted to lose weight and maintained an average weight loss of 5 kg over 3 years. This means that these individuals' BMIs at 3-year follow-up were almost entirely explained by their starting BMIs and suggest the operation of a powerful and highly calibrated system of subconscious biobehavioural regulation. The fact that the individuals in this study likely differed substantially in psychological characteristics thought to determine weight loss success (self-control, decision-making, impulsivity, body image, etc.) suggests that their regulatory responses to weight loss completely overshadowed these individual differences. Even among patients who underwent bariatric surgery and lost an average of 47 kg after 1 year, the correlation between starting and follow-up BMIs was very high ($r = 0.67$). Thus, not only does weight loss trigger mechanisms for weight regain, it appears that starting weight largely determines what body weight will be at various points following the loss.

The next pertinent question is whether repeated bouts of weight loss and regain (so-called weight cycling) produce weight gain beyond whatever weight would be gained by non-cycled individuals under otherwise similar conditions. Research has examined this question in relation to the effects of periods of weight loss and regain on both body composition and total weight gain. Most studies conducted in overweight or obese subjects found little evidence of effects of weight cycling on these outcomes (11–13). Also, some of the evidence relevant to the possible effects of weight cycling on weight gain is based upon correlations between self-reported history of cycling and BMI. Instances where cyclers have higher BMIs (14) could be due to this group having a predisposition towards accelerated weight gain, which preceded the first instance of weight cycling and which over time manifests itself in both more weight loss efforts and higher eventual BMIs. If this is the case, then the cycling pattern would not *produce* greater weight gain but would reflect unsuccessful efforts at preventing the additional weight gain to which cyclers were already predisposed. Finally, if weight cycling did cause excessive weight gain, it would do so in the form of a greater accumulation of fat relative to lean tissue. A review of weight cycling studies in obese humans (15) concluded that 'both natural and experimental weight cycling studies have failed to demonstrate permanent alterations of body composition or body fat distribution'. The work of Dulloo *et al.* (16) suggests that weight cycling may produce changes in body composition in favour of greater body fat in lean individuals but not in overweight or obese individuals.

One additional type of evidence is indirectly relevant to the question of whether repeated cycles of weight loss and regain cause additional future weight gain. This is whether such weight cycling has adverse effects on morbidity and mortality (perhaps via increased body fat). A recent review of the literature concluded that 'although weight regain following successful weight loss remains one of the most challenging aspects of body-weight regulation, evidence for an adverse effect of weight cycling appears sparse, if it exists at all' (17).

In summary, two types of dieting have been considered so far. Dieting-induced reductions in body weight do contribute to weight regain through both metabolic (loss of lean tissue and reduced metabolic rate per kilogram of lean body mass) and behavioural (e.g. increased hunger and reward value of food) routes. However, it is debatable whether losing weight via dieting produces weight regain beyond that which would have been gained if a diet had never been undertaken. Reviews of past behavioural weight loss studies indicate that most studies of long-term follow-up find that, despite substantial weight regain, the body weight of participants at 4- and 5-year follow-up is still below the weight at which they began the study (17,18). Because in the absence of intentional weight loss obese individuals could be expected to gain about 1 kg year^{-1} in an obesogenic environment, this means that former participants are still several kilograms lighter 4–5 years after a weight loss. Given the slope of the line characterizing weight regain, this means it would take at least several more years for participants to attain a body weight significantly higher than their initial BMI. This evidence again suggests that losing and regaining weight does not itself produce additional weight gain. The fact that some obese individuals eventually gain weight beyond the weight they were before they lost weight via dieting is probably due to the fact that already obese individuals would be expected to gain about 0.5–1.0 kg year^{-1} via the same processes that produced their obesity in the first place (18,19). As for repeated bouts of weight loss

and regain, although such cycles may affect the rapidity of weight regain or the composition of the tissue that is regained, there is little reason to believe that weight cycling causes future weight gain beyond that which would naturally occur from living in an obesogenic environment.

Weight suppression

Another possible, if less common, outcome of a weight loss diet is substantial weight loss that is maintained (rather than quickly regained) for some time after the weight loss. We have labelled sustained weight loss 'weight suppression', defined as a meaningful weight loss that is sustained for a minimum period of time (20). The maintenance of a weight loss for a specified period of time is what differentiates weight suppression from the domain of acute weight loss dieting reviewed earlier. Although weight regain appears to be a much more common outcome than long-term weight loss maintenance, there are still many people in the population who maintain a weight well below their highest weight ever. There are no absolute criteria defining this state in terms of the amount of weight loss required or the length of time it has to be maintained. Although arbitrary, in the past research, we defined it as a weight loss of at least 10 lb (or 4.5 kg) (21,22) that has been largely maintained for at least 6–12 months. In our studies, the weight losses typically occurred a year or more in the past, so such individuals would be considered weight suppressors. These studies found that women high in weight suppression, relative to those low in weight suppression, ate less food after a preload (21) and disliked sweet solutions (22). We concluded from these studies that weight suppressors may be showing adaptations to their prior weight loss that are helping them sustain their weight loss long-term. Behavioural adaptations to large, sustained weight losses have also been observed among members of the National Weight Control Registry, who have shown long-term maintenance of substantial weight losses with relatively little weight regain (23).

However, more recent research has shown that the dietary restraint and appetitive adaptation associated with weight suppression are not sufficient to counteract the pressures towards weight regain to which weight suppressors are susceptible. A wide variety of studies have prospectively examined the relationship between degree of weight suppression and future weight gain. These studies have consistently found that level of weight suppression at baseline predicts the amount of weight gained at later time points. This is true in non-clinical participants (2,7) and in those with either anorexia nervosa (24,25) or bulimia nervosa (26). Furthermore, the prediction of weight gain has spanned time periods ranging from 4 weeks (2) to 5 years (26) in bulimia nervosa and up to 18 years in anorexia nervosa (27). Even in a sample of college students where the average level of weight suppression was only 2.7 kg (2), weight suppression at the start of the academic year predicted the amount of weight gained 8 months later. The consistency of these findings across various groups of non-clinical and clinical populations, along with the consistent weight regain that follows weight loss in obese individuals, indicates that weight reduction below a previous high weight generates powerful forces that drive weight back towards its initial level. These results strongly suggest that a highest historical weight is not nearly as changeable as the thousands of dieting books and programmes imply. These forces are proportional to the percentage of weight loss; thus, weight suppression appears to act like a stretched rubber band. The further the band is stretched, the greater the force created towards restoring the rubber band to its initial state (26). This indicates that any short-term adaptations aimed at sustaining weight losses (e.g. choosing more low-calorie foods, increasing physical activity) are unlikely to prevent eventual weight regain to roughly the level reached before significant weight was lost. It appears that metabolic efficiency (1,7), consummatory changes (e.g., increased binge eating (28)) and increased reward value of food (29) contribute to this weight gain. Therefore, both acute and longer term weight losses induce responses that make eventual weight regain highly likely. Although results from the National Weight Control Registry show that it is occasionally possible to maintain major weight losses, the evidence from both the obesity treatment literature and the newly emerging weight suppression literature indicates that powerful and sustained pressures make weight regain back towards starting BMI highly likely in the vast majority of people across the BMI spectrum. The recent findings of Ferrannini *et al.* (10), reviewed earlier, suggest the same thing.

Weight loss dieting as a proxy of proneness to weight gain

Up to this point in the paper, I have been considering weight loss dieting as a potential cause of weight regain. It appears that the biobehavioural state created by weight loss does indeed causally contribute the weight regain that usually follows weight loss, although the weight regain may sometimes occur over years. However, current or past weight loss dieting undoubtedly also results from other influences that produce a positive energy balance. For instance, having a low basal metabolic rate (corrected for lean body mass) has been found to predict greater weight gain (30), as has a tendency towards eating disinhibition (31). The fact that well over half of individuals in many countries are overweight or obese means that most individuals living in obesogenic environments will experience

excessive weight gain – or more specifically, excessive fat gain – during their lifetimes. Given both the health risks and the psychosocial consequences (e.g. discrimination and stigma) associated with obesity, most individuals with a susceptibility towards weight gain will attempt to lose weight on one or more occasions. Therefore, it is reasonable to conclude that the act of going on weight loss diets could reflect the effects of a predisposition to weight gain stemming from personal (e.g. genetic susceptibility, emotional eating) or environmental (e.g. living in a neighbourhood with many fast-food restaurants) factors. Given the powerful and widespread fear of weight gain and obesity, it is not surprising that people prone to weight gain would start going on weight loss diets well before they become obese or even overweight. Therefore, the act of going on weight loss diets among those who are in or near the healthy weight range may represent an early warning sign that the dieter is experiencing weight gain that he or she wants to resist or reverse. Thus, while dieting that produces weight loss can cause weight regain, the act of going on weight loss diets might also be a marker of (and a consequence of) an already existing predisposition towards weight gain. From this perspective, the biobehavioural predisposition towards weight gain predates and gives rise to weight loss dieting.

Dieting in non-obese individuals could stem in part from the well-known socio-cultural emphasis on attaining a slim body shape (32), in addition to the stigma associated with obesity. This partly explains why so many *non-obese* people (and young women in particular) restrain their eating and go on weight loss diets. As reviewed earlier, dieting is unlikely to produce sustained reductions in weight. Furthermore, although losing and regaining weight apparently has little effect on susceptibility to excessive weight gain in the future, it also does nothing to reduce the strength of the enduring vulnerability to continue gaining weight in an obesogenic environment. Therefore, over time dieting to lose weight could become a proxy risk factor – i.e. a variable that predicts susceptibility to future weight gain because it is *correlated* with a causal risk factor, not because it *is* a causal risk factor (3).

We recently reviewed studies examining measures of restrained eating and dieting as prospective predictors of weight gain (4). We compared these two types of measures because of extensive evidence that restrained eating and dieting to lose weight, although correlated, have distinctly different associations with various outcomes such as laboratory-based eating regulation and binge eating (20,33). We reviewed 25 studies that contained 40 relevant analyses that used measures of either restrained eating or dieting to predict future weight gain among individuals who were mostly in the healthy weight range. Three-quarters of the analyses that used a measure of dieting (or past dieting frequency) significantly predicted future weight gain (with dieters gaining more than non-dieters), whereas only 5% of the analyses that used a measure of restrained eating did so. Although the reliable prediction of weight gain by dieting measures would be consistent with the view that dieting causes future weight gain, the preponderance of evidence reviewed earlier supports the view that dieting is simply a marker of – or a proxy risk factor of – other vulnerabilities that make certain non-obese individuals prone to accelerated weight gain.

Implications for the controversy over dieting

To summarize the conclusions of the foregoing review, it appears that dieting is relevant to weight gain in two very different ways. Dieting that results in weight loss does, in fact, appear to contribute to the weight regain that usually follows and this appears to be the case among both non-clinical and clinical populations. However, even substantial or repeated weight losses do not appear to cause absolute weight gain beyond the level that would otherwise occur in the absence of a weight loss. When individuals who have not yet developed a weight problem go on diets, however, the main significance of their dieting appears to be that they are predisposed to future weight gain.

The practice of dieting to lose weight has been a subject of intense and prolonged debate for decades (34–36). There are many facets to this controversy and reviewing all of them is beyond the scope of this paper. However, researchers who have suggested that dieting paradoxically promotes weight gain and contributes to the obesity epidemic (36–38) have contributed to strong anti-dieting sentiment in the general population that may be having counter-productive effects on reducing unhealthy eating and the weight gain it produces. The effort to curb and reverse obesity involves a battle between numerous obesogenic influences in the environment, on the one hand, and people's ability to resist and counteract these influences, on the other hand. Major changes will be needed in the broader food environment (39) but changes will also be needed in the attitudes, norms and customs that permit or encourage frequent, uninhibited consumption of food. Exaggerated claims about the dangers of dieting (or restricting food intake more generally) work against this latter goal by creating unwarranted fears of abstention and temperance in eating habits. The obesity epidemic has been with us for over 3 decades and it is true that the hundreds of diet programmes and diet books, and the billions of dollars spent on weight reduction appear to have had little lasting benefit. However, declaring that weight loss dieting is usually ineffective is quite different than suggesting that it is harmful or that it is actually helping fuel the obesity epidemic. Just as the vast majority of individuals who drink alcohol do so in a responsible

way, human beings have the potential for much better self-control of their food intake and body weight than has so far been demonstrated. Therefore, we need to guard against premature and ill-considered conclusions about the dangers of volitional eating restrictions when the much greater threat we face is a relentless epidemic of excessive consumption and obesity.

Conflict of interest statement

No conflict of interest was declared.

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