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Is "Yo-Yo" Dieting or Weight Cycling Harmful to One's Health?

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Introduction

Since the 1980's when obesity became prevalent, "yo-yo dieting", or weight cycling, has emerged. Weight cycling involves losses and gains in weight, which can either be intentional, such as through dieting then subsequent regain of lost weight, or unintentional, as through chronic disease states. Studies have yet to determine the health effects of such weight losses and regains. Weight cycling is difficult to study, as studies are mostly retrospective and rely on patients' recall of weight changes. There are also no consistent outcome measures or definitions of weight cycling, so quantifying its prevalence is difficult. In the Nurses' Health Study (> 45,000 women), 1.4% of the subjects were classified as severe weight cyclers (loss of 20+ lbs. three or more times during the four year study) and 18.9% were moderate cyclers (loss of 10+ lbs. three or more times)(1). Most studies have been conducted on overweight, mildly, or moderately obese individuals. Few are conducted on the morbidly obese. Likewise, there are few studies on the effects of weight cycling in never-before overweight individuals who do not need to lose weight. This review will examine the recent literature on weight cycling in different groups and determine whether detrimental health effects exist. If so, then losing weight could have serious consequences as a treatment option for the obese, many of whom regain the weight they lose and weight cycle. It could also have health implications for normal weight individuals who weight cycle for their profession (actors) or for athletics (wrestlers, boxers).

Metabolic Changes

Metabolic changes in weight cycling stem from the "set-point" theory, which maintains that the body has a feedback mechanism to maintain its fat stores and will regain any weight it loses (2). An adaptive down-regulation of resting metabolic rate (RMR), resting energy expenditure (REE), and thyroid hormones is thought to cause this regain. This theory is important to the treatment of obesity, for if an obese person faces metabolic factors which counter their efforts to lose weight, their treatment plan should change.

A 1995 clinical trial (Leibel, *et al.*) on 18 obese individuals (average age 29 years) matched with never-before obese controls showed that a 10-20% decrease in body weight in both groups was significantly correlated to a decrease in REE (3), thus supporting the set-point theory. However, this study did not examine REE with respect to weight cycling and did not examine REE after allowing energy expenditure to stabilize post-loss. The more recent 2000 study (Weinsier, *et al.*) of 24 overweight postmenopausal women matched with never-before overweight women measured RMR and serum triiodothyronine (T₃) (2). Unlike the retrospective 1995 study, this one induced weight cycling in a controlled in-patient environment. RMR and T₃ were measured not only after weight loss of at least 10kg or upon reaching a BMI=25, but also during a 10-day period of energy stabilization after ceasing caloric restriction. Initially, T₃ and RMR levels decreased in both the control and the overweight groups. However, during the energy stabilization phase, both groups experienced a rebound to pre-weight loss levels of RMR and T₃. These results suggest that the hypometabolic-hypothyroid state induced by the weight loss is actually transient and that it normalizes upon return to energy stabilizing conditions. The hypometabolic-hypothyroid state induced in the Leibel, *et al.* study could have been seen because the measurements were taken during the transient

phase and not during the energy stabilization phase. Furthermore, subjects in the 2000 Weisner, *et al.* study were re-weighed four years later. The overweight group still regained 87% of their lost weight compared to 1.7% for the controls (1). The hypometabolic-hypothyroid state was therefore not responsible for the subsequent weight gain, dispelling the set-point theory. Whether this type of metabolic rebound is also seen in weight cycling of obese or younger individuals needs to be studied. The Nurses' Health Study of 224 young and middle-aged women may provide a possible explanation for weight regain. The study found a significant correlation between weight cyclers and greater weight gain, lower physical activity, and a higher prevalence of binge eating (4).

What about metabolic changes in never before overweight individuals who weight cycle? A 2002 study (Kajioka, *et al.*) examined the metabolic effects of weight cycling on five healthy, non-obese Japanese women. The average age of these women was 24.6 (range 22-34 years). They experienced two cycles of caloric restriction and weight loss with allowed time for weight regain after each restriction. After 106 days post cycling, the women had regained on average 87.5% of the weight they lost. Immediately after the 2nd cycle, they were found to have significant decreases in lean body mass, T₃, REE, systolic and diastolic blood pressures, and triglycerides. At 106 days later, all outcome measures remained decreased, except for systolic and diastolic blood pressures and triglycerides, which all rose significantly from baseline (5). The most notable difference between this study and the Weisner, *et al.* study is that the Weisner study involved postmenopausal women. Also, the diets of the two studies were likely dissimilar as the women were from different countries. However, while this study was small and involved women from one ethnicity, it does indicate that weight cycling may have deleterious effects in young, normal weight women. Perhaps the set-point theory applies to individuals who do not need to lose weight.

Cardiovascular Effects

Coronary heart disease was one of the first harmful effects that researchers thought weight cycling caused, though the exact mechanisms are unknown. Hyperinsulinemia and hypertension induced by weight cycling are possible mediators, but studies have shown contradictory results on this theory (6). One of the latest retrospective studies published in 2004 (Graci, *et al.*) studied 459 obese individuals (340 women, 119 men) in Italy. This study found that in subjects of either gender, weight cycling and weight regain were not associated with harmful effects on body composition (BMI and fat percentage), fat distribution (waist circumference), or cardiovascular risk factors (glucose, insulin, insulin resistance, HDL, triglycerides, total cholesterol, and blood pressure levels). Rather, a significant positive correlation was found between the maximum absolute amount of weight regained following a single diet episode and cardiovascular risk factors (7). However, in another 2000 Italian study (Guagnano, *et al.*), the risk of hypertension was found positively correlated to abdominal waist-to-hip ratio and weight cycling. The sum of the weight regained was also a risk factor. This study is comparable to the Graci, *et al.* study in that it also involved obese individuals (258 women with similar age range)(8). However, the Graci, *et al.* study excluded patients on hypertensive drugs and only included mildly hypertensive individuals in their

analysis. Perhaps individuals with more severe hypertension do have a positive correlation with previous episodes of weight cycling.

Chronic Disease and Mortality

The effects of weight cycling on chronic disease and mortality have been even more difficult to elucidate. The Wannamethee, *et al.* 2002 study of 5608 British men showed an increased association of mortality with weight cycling. However, the correlation was explained to a large extent by lifestyle factors and preexisting disease (9). Unfortunately, the study excluded the amount by which the correlation was attenuated. It also did not separate intentional from unintentional weight loss. The Iowa Women's Health Study of 33,832 women aged 55-69 showed higher relative risk for MI, stroke, and type 2 diabetes (10). Though the relative risk was adjusted for and somewhat attenuated by demographics and health behavior, preexisting disease and poor health were not taken into account. Therefore, the correlations could stem from the fact that these individuals had chronic disease at the study's inception. Field, *et al.* researched 46,634 young and middle-aged women from the Nurses' Health Study. While weight cycling was strongly associated with BMI, it was not found to predict type 2 diabetes (11). This opposite result from the Iowa study could be due to the fact that the Iowa study involved a less healthy population than the younger Nurses' Health Study.

Osteoporosis

Few studies have examined the relationship between weight cycling and bone density. The Fogelholm, *et al.* 1997 study showed that premenopausal obese female weight cyclers (ages 29-46) had lower spine and distal radius bone mineral densities than a control group (12). A 2002 study by Gallagher, *et al.* showed that overweight/obese sedentary women (ages 27-40) did not have significantly lower total-body bone mineral content or density or total femur bone mineral density (13). Total-body bone mineral density is correlated with distal radius bone mineral densities, so the findings between the two studies differ for this region. Gallagher, *et al.* did not examine spine densities, however. A more recent study by Fogelholm, *et al.* of 74 premenopausal women (ages 30-45) who underwent controlled weight reduction and subsequent weight regain showed clinically small and reversible effects on bone mineral content and density. The authors state that they could not attribute such effects to either DXA scan artifacts or limitations or to weight cycling (14). Further research needs to be conducted in men and these different groups of women, especially since these preliminary studies show some decrease in bone density.

Immunocompetence and Cancer

As with osteoporotic studies, studies on immunocompetence, cancer, and weight cycling are very few. The largest study thus far is the Iowa study, which showed a correlation between severe weight cyclers and an increased risk of lung cancer. There was no increased risk for breast, colon, or endometrial cancer (10). Natural killer (NK) cell function has also been examined, as NK cells are easily measured and are often seen as the initial line of defense against primary tumor cells and metastases (15). Shade, *et al.* showed a decrease in NK cell function and the number of circulating NK cells with an increase in weight loss episodes for postmenopausal women (16). Further research needs

to be conducted examining the interplay between obesity, weight cycling, and the immune system before any recommendations can be made.

Conclusion

Clinicians should not deter overweight/obese individuals from losing weight for fear of weight cycling as studies have not definitively concluded if there are harmful metabolic effects to weight loss and regain. However, the benefits to overweight/obese individuals of losing weight have been well documented. Young women who are within normal weight range should not intentionally lose unnecessary weight, however, as the Japanese study indicated potentially deleterious health effects of weight cycling. Certainly, more controlled studies need to be done on this population to confirm or deny this finding.

No definitive association was found between weight cycling, stroke and MI. Hypertension and hyperinsulinemia occurred in some studies and insofar as these are mediators to MI or stroke, weight cycling would be considered harmful. However, MI and stroke are also exacerbated by other factors, so the amount by which weight cycling contributes to these morbidities is difficult to delineate. The mechanisms by which weight cycling could cause MI and stroke are also unknown. This uncertainty may explain the variability in findings of cardiovascular risk factors with weight cycling. Nevertheless, several studies did find an association between weight regain amounts in overweight/obese individuals and these risk factors, so every effort should be made to minimize such regain in these groups.

Weight cycling does not appear to directly increase mortality. Mortality is influenced by many other factors. Studies need to determine first whether weight cycling causes other morbidities which may lead to an increase in mortality. Weight cycling also probably does not influence the development of type 2 diabetes unless it causes a large amount of weight regain that significantly influences waist circumference. More studies need to be done to determine patterns of weight regain after weight loss in different groups of individuals. These studies will be important in the treatment of obesity as certain “crash” diets that cause large weight losses within short time periods can lead to more weight regain than other types of diets.

Finally, no conclusive recommendations can be made with regards to osteoporosis, immunocompetence, and weight cycling, as these fields of research are still in their incipient stages.

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