Does weight cycling present a health risk?1–3

Robert W Jeffery

ABSTRACT I review research on the hypothesis that weight fluctuations caused by unsuccessful dieting are hazardous to one’s health. Recent epidemiologic findings show that weight variability over time is associated with increased total and cardiovascular mortality (relative risk: 1.5–2.0), independent of a variety of possible confounding variables. Although these findings are consistent across studies, methodologic limitations of a lack of a uniform or standard definition of weight cycling, and the linking of weight variability to unsuccessful dieting raise serious questions about whether these findings should be interpreted as supporting the weight-cycling hypothesis. The absence of data identifying a plausible biological mediator for weight fluctuation per se as a health hazard is also a problem. It is concluded that, although epidemiologic data on weight variability and health are intriguing, they are at present insufficient to alter public health recommendations regarding weight control. Am J Clin Nutr 1996;63(suppl): 452S–5S.

KEY WORDS Obesity, dieting, weight cycling, health, healthy weight, body weight

INTRODUCTION

Obesity has long been of interest to health researchers because of its positive association with a variety of common health conditions, such as hypertension, diabetes, and coronary heart disease, and its association with premature mortality. These associations have formed the basis for the public health recommendation that persons who are substantially above average weight-for-height, particularly those who have health conditions that are responsive to weight loss, should lose weight (1).

Recently, however, several clinicians and scientists have begun to question whether these recommendations are well founded. In part, this concern stems from the fact that sustained weight loss is difficult to achieve (2). Also, it follows from recent epidemiologic data on the relation between weight variability and health. Concern about voluntary weight loss can be stated in the following question: Is a cyclical pattern of intentional weight losses followed by regains more hazardous to the health of overweight individuals than maintaining a high but stable weight? I review data related to this question.

WEIGHT CHANGE AND HEALTH

Historically, the focus of epidemiologic studies on weight has been on the ability of weight itself to predict future disease (3). Recently, however, several investigators have reexamined existing epidemiologic data sets to assess the association between weight change and health. The findings, which have been fairly consistent across studies (4–6), are illustrated in Figure 1. These data are taken from a report by Lee and Paffenbarger (5) on mortality risk in male Harvard alumni. About 12,000 healthy men of the average age of 58 y were followed for 10 y. Weight changes for a period of 10–15 y before the beginning of follow-up were assessed by self-report questionnaires. Through the use of stable weight as the reference category and with adjustment for smoking, physical activity, and body mass index (BMI), it was observed that both total and cardiovascular mortality were lowest in those maintaining stable weight. Weight loss and weight gain were each associated with increased risk.

Weight cycling, of course, requires weight change in both directions rather than in only one, and the first study to specifically address this issue was that published by Hamm et al in 1989 (7). This epidemiologic evaluation used data from a longitudinal cohort study of 2109 men aged 40–56 y in 1957 and followed mortality risk for 25 y. At the time of their first examination, these men were asked to recall their weights at age 20, 25, 30, 35, and 40 y. Through the use of these weights, they were classified into four different groups. Weight cycling was defined as a gain of ≥10% of body weight in one 5-y interval and a loss of 10% in another 5-y interval. Stable weight was defined as weights that never deviated by >5% from weight at the age of 20 y. Large weight gain was defined as a gain ≥10% of body weight between two time points with no loss of weight. “Other” was a catchall category that included all men not meeting any of the previous three definitions. On average, men in this group gained a modest amount of weight over time. As shown in Figure 2, men whose weights remained stable throughout life had the lowest all-cause and cardiovascular disease mortality. Those whose weights cycled had increased mortality, especially from cardiovascular disease. Those in the group with large weight gains and in the “other” category also had slightly increased mortality.

Lissner et al (8) took a somewhat different approach in analyses of Framingham data to define weight cycling. The Framingham study followed ~1400 men and 1800 women for

1 From the Division of Epidemiology, University of Minnesota School of Public Health, Minneapolis.
2 Supported by NIH grants HL41332 (NHLBI) and DK45361 (NIDDK).
3 Address reprint requests to RW Jeffery, Division of Epidemiology, University of Minnesota School of Public Health, 1300 South Second Street, Suite 300, Minneapolis, MN 55454-1015.

mortality in both men and women. Comparing the top and bottom tertiles of the weight-variability distribution, relative risks of 1.7 and 1.3 were noted for men and women, respectively, between the most and least variable weights for total mortality. Relative risks of 1.9 and 1.6 were noted for men and women, respectively, for coronary heart disease mortality.

A recent study by Blair et al (9) examined weight cycling with both categorical and variability approaches to defining weight cycling in participants in the Multiple Risk Factor Intervention Trial. In 3.8 y of follow-up of 10,529 men initially aged 35–57 y, whose weights were measured annually for 6–7 y, weight variability was shown to be positively associated with all-cause and cardiovascular disease mortality defined by either method. Results from the categorical method are shown in Figure 3. No weight change over the period of observation was associated with lowest mortality, sustained weight loss and sustained weight gain of ≥5% of initial body weight were each associated with elevated mortality, and weight changes of 5% in both directions (cycling) were associated with elevated mortality, regardless of whether the cycle pattern ended with a weight loss (cycle 1) or a weight gain (cycle 2). With weight variability over time as the definition of weight cycling, total mortality in those in the highest quartile of weight variability was about 1.5–2 times higher than that of men in the lowest quartile. Blair et al (9) also did subgroup analyses in which the data set was stratified by BMI. These analyses showed that weight variability was more strongly related to mortality in lean than in obese men (ie, adjusted relative risk of all-cause mortality was 1.20 in obese men with most versus least variable weights and 2.06 in lean men with most versus least variable weights).

**FIGURE 1.** Total and cardiovascular disease (CVD) mortality by weight change in male Harvard alumni (5).

12 y. In the analyses by Lissner et al, weights used to define cycling included measured weight at eight biennial examinations and recalled weight at the age of 25 y. Weight cycling was defined as the CV of body weight (ie, the SD of all the weight measurements divided by mean body weight over the entire time period). Statistical analyses controlled for absolute body weight, linear trend in body weight, and cardiovascular risk factors. Findings were that weight variability was positively associated with both total and coronary heart disease mortality.

**FIGURE 2.** Total and cardiovascular disease (CVD) mortality by weight cycling category in 40–56-y-old men (7).

**FIGURE 3.** Total and coronary heart disease (CHD) mortality by weight change category in middle-aged men (9). Cycle 1, the cycle pattern ended with a weight loss; cycle 2, the cycle pattern ended with a weight gain.
LIMITATIONS IN METHODOLOGY

Although it is tempting to interpret these epidemiologic studies as supporting the idea that intentional weight loss in the obese may not be wise, it is important to remember that these studies were not designed to answer this question and, thus, leave much to be desired in methodologic sophistication. One important issue is the definition of weight cycling. The common meaning of weight cycling is roughly equivalent to the clinical observation of yo-yo dieting, a phenomenon the salient characteristics of which are frequent oscillations over relatively short time intervals, often of low magnitude, that are caused by intentional changes in behavior. However, epidemiologic data sets generally lack the information needed to capture this meaning.

One issue is the spacing of measurements. In most epidemiologic studies, the weights on which weight variability have been based are spaced far apart, being at least 1 y and sometimes 5, 10 or more y between measurements. Because weight cycles associated with dieting are rarely of this duration, it is questionable whether the weight changes described in epidemiologic studies are measuring dieting-related cycling. A recent methodologic study by French et al (10), taken from the Iowa Women’s Health Study, illustrates this point. About 30,000 women of the average age of 68 y completed a questionnaire in which they reported their weights at ages 18, 30, 40, 50, 62, 64, 66, and 68 y, and also described their history of intentional weight losses. Reports of intentional weight loss were positively correlated with weight variability as calculated from the weights themselves. The strength of the association was weak, however ($r \sim 0.10$), suggesting that factors other than intentional weight loss contribute importantly to the measures of cycling used in epidemiologic studies.

Another methodologic issue is repetition. Although repetitive weight gains and losses are the prototypical cycling pattern, no epidemiologic study has yet evaluated cycling in terms other than a single gain and loss. This fact makes it difficult to assess whether cycles confer any greater risk than do single gains or losses.

A third issue is the heterogeneity of weight patterns captured by continuous measures of cycling such as the CV. Such measures have the analytic advantage of being able to place all study participants on a single continuum. However, there are many patterns of weight that contribute to variability in measures other than cyclical ones. An example of this is shown in Figure 4, which shows four patterns of weight actually observed in the Iowa Women’s Study through the use of reported weight at the ages of 18, 30, and 40 y. All four patterns yield high estimates of variability, but are obviously very different from each other, including patterns of weight gain only, weight loss only, weight loss followed by weight gain, and weight gain followed by weight loss.

A final key point that is missing from virtually all epidemiologic data sets is information about the causes of the weight change. Intentionality has been addressed in only one study on weight variability and mortality published to date. This was a study by Lissner et al (11) in which a Swedish database was used. These investigators found a positive association between the CV of body weight and mortality in a positive association between the CV of body weight and reported history of dieting but no association between mortality and history of dieting.

FIGURE 4. Weight patterns associated with high variability.

To summarize, given the intriguing nature of the data on weight changes and disease outcomes found in epidemiologic research, there is a clear need for better information on what exactly is being measured in these studies. The frequency and spacing of weight measures in these studies have been inadequate so far to the task of properly evaluating the differential significance of different patterns; furthermore, the causes of the weight changes have barely been explored.

BIOLOGICAL PLAUSIBILITY

The final question for the present review is biological plausibility. Mechanisms that have been proposed to relate weight cycling to ill health include the following: 1) decreases in metabolic rate, 2) increases in preference for and thus consumption of dietary fat, and 3) changes in body fat distribution toward a more abdominal type that in turn would adversely affect blood pressure, cholesterol, and glucose intolerance. Although the research literature on this topic is not extensive, it so far has not been supportive of any of these hypothesized mechanisms. Cross-sectional studies in obese and nonobese populations have failed to find significant relations between reported history of weight cycling and metabolic rate, body fat distribution, total cholesterol or its fractions, blood pressure, glucose tolerance, dietary fat intake, or steroid hormones (12–14).

Prospective studies of weight fluctuations have been similarly unsuccessful in showing that weight cycling adversely affects metabolic rate or cardiovascular risk factors. Jeeb et al (15), for example, studied 11 obese women over 18 wk that included three 2-wk weight-loss periods interspersed with three 4-wk periods of ad libitum diet and weight gain. They found that although basal metabolic rate declined during each of the weight-loss periods, it returned to the normal baseline after each period of weight regain with no net change over time. Wing et al (16) conducted a similar evaluation in which they
examined changes over time in cardiovascular risk factors in men and women who exhibited different weight-change patterns over a 30-mo period. Examining blood pressure, blood lipids, body fat distribution, and glucose intolerance, they found no evidence that individuals losing and regaining weight had adverse changes in risk factors compared with those who never lost weight.

CONCLUSIONS

Stable weight over time is associated with best health. All patterns of weight change other than stable weight—gains, losses, and both combined—appear to be associated with increased mortality risk. Epidemiologic data on weight variability and health are difficult to interpret regarding the weight-cycling hypothesis because the data allow crude definitions of cycling at best, and intentionality of weight losses has not been assessed at all. A key requirement for inferring causality from observational data is also lacking in the current research literature, namely biologic plausibility. To date, no mechanisms have been identified in either cross-sectional or prospective studies that might mediate an association between weight variability and ill health.

Although the association between weight variability and ill health is an intriguing research question, present data are insufficient to change public health recommendations about weight and weight loss. Maintaining a lean body weight throughout life is recommended. Weight loss in those who are obese and in those with obesity-related health conditions is also warranted.

REFERENCES