Weight cycling during growth and beyond as a risk factor for later cardiovascular diseases: the 'repeated overshoot' theory

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In people trying to lose weight, there are often repeated cycles of weight loss and regain. Weight cycling is, however, not limited to obese adults but affects people of normal weight, particularly young women, who are unhappy with their appearance. Furthermore, the onset of a pattern of weight cycling is shifting towards younger ages, owing to the increasing prevalence of overweight and obesity in children and adolescents, and the pressure from the media and society for a slim image even for normal weight children. Although there is still controversy whether weight cycling promotes body fat accumulation and obesity, there is mounting evidence from large population studies for increased cardiovascular risks in response to a behavior of weight cycling. Potential mechanisms by which weight cycling contributes to cardiovascular morbidity include hypertension, visceral fat accumulation, changes in adipose tissue fatty acid composition, insulin resistance and dyslipidemia. Moreover, fluctuations in blood pressure, heart rate, sympathetic activity, glomerular filtration rate, blood glucose and lipids that may occur during weight cycling – with overshoots above normal values during weight regain periods – put an additional load on the cardiovascular system, and may be easily overlooked if humans or animals are studied during a state of relatively stable weight. Overshoot of those risks factors, when repeated over time, will stress the cardiovascular system and probably contribute to the overall cardiovascular morbidity of weight cycling.

Keywords: weight cycling; cardiovascular risk factor; hypertension

Introduction

In people trying to lose weight by dieting, there are often repeated cycles of weight loss followed by weight regain when the diet is interrupted, a phenomenon known as weight cycling or yo-yo dieting.[1] Indeed, weight maintenance is an enormous challenge for those who have voluntarily lost a significant amount of weight and therefore weight loss attempts are often not very successful in the long term.[2] Despite the poor efficiency of dieting in weight management, the prevalence of dieting has increased continuously in the last 50 years in parallel to the steadily increasing prevalence of overweight and obesity. In the US national surveys conducted between 1950 and 1966, about 14% of women and 7% of men reported that they were trying to lose weight. In the late eighties, this prevalence had increased to an estimated 40% of women and 25% of men trying to lose weight at any given time.[3] In a survey conducted during the mid-1990s in the US and involving over 100 000 adults aged 18 years and older, the prevalence of attempting to lose weight was 44% among women and 29% among men.[4]

Because dieting is so common and the likelihood of weight relapse is quite high, weight cycling is assumed to be highly prevalent too. However, studies reporting the prevalence of weight cycling in the general population have reported variable results.[5–8] Besides the question of recruiting different populations, one of the reasons is that there is no universally accepted definition of weight cycling but many possible variations on the same theme.[1] Weight fluctuations can be of different lengths or amplitudes (with cutoffs expressed differently as absolute or percentage changes), or with variable numbers of cycles ranging from one single large cycle to repeated weight cycles in unsuccessful dieters,[9] or in athletes who undergo seasonal or even weekly weight losses in order to make a weight category.[10] In a cohort of 46 224 normotensive women of the Nurses' Health Study II followed during 4 years, 78% of the women intentionally lost weight (between 2.25 and 4.45 kg) at least once, 41% had

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similar weight losses twice or more and 20.3% reported that they intentionally lost at least 4.5 kg three times within the past 4 years.[6] A recent epidemiological study assessed the prevalence of weight cycling in the general population of Finnish adults (25–64 years of age).[8] In this study, severe weight cycling, defined as a weight loss $\geqslant 5$ kg at least three times with regain, was reported by 10% of women and 7% of the men. Mild weight cycling, defined as a weight loss $\geqslant 5$ kg once or twice with regain, was reported by 19% of women and 11% of men. Thus, about 29% of women and 18% of men showed some degree of weight cycling. This underscores the high prevalence of weight cycling in the general population, particularly among women.

A history of weight cycling is not limited to adult obese subjects

Weight cycling typically occurs in association with weight loss induced by dieting. Because slimming prevalence increases with increasing body mass index (BMI),[3,11] it is often assumed that weight cycling is above all a problem of obese subjects. Indeed, a high prevalence of voluntary weight loss attempts has been reported in overweight and obese subjects in various studies. For example, in a survey of randomly selected subjects, 17 years of age and older in Denmark, 54% of overweight (BMI, 25 to <30 kg/m^2) and 77% of obese people (BMI $\geqslant 30$ kg/m^2) reported weight loss attempts at least once in their lives.[11] Weight cycling can also affect younger subjects. One of the reasons is that the increasing prevalence of overweight and obesity is particularly alarming among children and adolescents. Recent estimates (2001–2002) in 10–16 years youth from North American and European countries indicate a very high prevalence of overweight and obesity in North America, Great Britain and south-western Europe, with the highest prevalence in Malta (25.4 and 7.9%) and the US (25.1 and 6.8%).[12] In weight loss programs in obese adolescents, two types of weight fluctuations can be observed, that is, short-term mild fluctuations related to small deviations from the weight loss program, and long-term wider fluctuations owing to weight relapse. For example, severely obese adolescents (BMI 33.9 kg/m^2, 12–16 years of age) submitted to a multidisciplinary 9-month weight loss program,[13] with moderate dieting and physical activity during the week in a medical environment, but who had the possibility to return home during the week-end, often showed small weight gain during the week-end. Once the weight loss program was ended after 9 months, about one-third of the adolescents had regained more than 30% of the weight lost within 4 months,[14] and were thus likely to experience wide fluctuations of body weight over the long term.

However, a history of weight cycling is not limited to overweight and obese subjects. As illustrated in Figure 1, weight cycling may also affect people of normal weight, including younger subjects. In adult Danes, 25% of under-

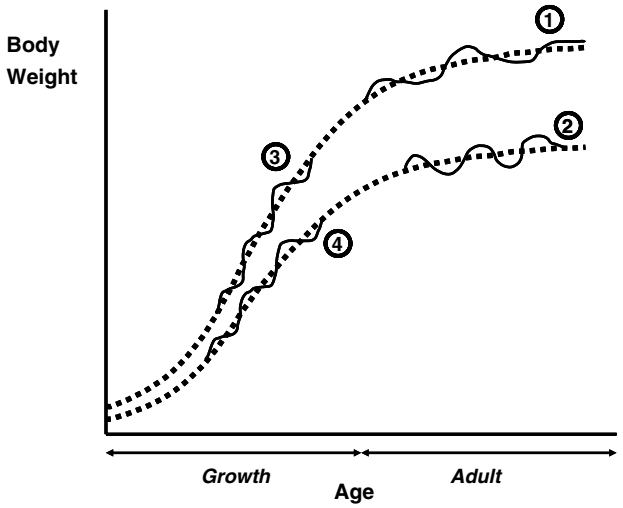

Figure 1 The four categories of intentional weight cycling: obese (1) and lean (2) adults, obese (3) and lean (4) children or adolescents.

weight (BMI <18.5 kg/m^2) and 38% of normal-weight subjects (BMI >18.5 and <25 kg/m^2) reported at least one weight loss attempt in their lives.[11] Prevalence of slimming was also higher in younger (<30 years) subjects compared to older (>50 years) subjects. Dieters often include people of normal weight who are unhappy with their appearance, particularly among young women. In a survey of 16 486 university students in 21 European countries (overall BMI of 20.5 kg/m^2 in women and 22.0 kg/m^2 in men), 44% of women and 17% of men were trying to lose weight. In spite of a low BMI, many students perceived themselves as overweight, especially among women.[15] Because a slim body image is widely promoted by the media and society, trying to lose weight has spread to children and adolescents in an attempt to conform with cultures that advocate '*slim is beautiful*'.[16] In a survey of 548 fifth- to 12th-grade girls in a working-class suburb in the north-eastern US, 47% of the girls reported wanting to lose weight because of magazine pictures.[17] In a cross-sectional mailed survey of 11 606 boys and girls aged 9–16 years, 46% of the girls and 27% of the boys reported making at least some effort to look like figures in the media.[18] Body dissatisfaction may also affect very young children. Girls from age 5 to age 8 exposed to images of the thin Barbie doll reported lower body esteem and greater desire for a thinner body shape than girls exposed to the larger Emme doll or to no doll, which could contribute to an increased risk of disordered eating and later weight cycling.[19]

Slimming is also common among athletes in sports with mandatory weight classes, such as weightlifting, martial arts,[20] boxing[21] and wrestling,[10,22–25] in sports where a small body weight is competitively beneficial, such as cycling and rowing,[26] or among performers, such as ballerinas, top models and entertainers, for whom a slim image is professionally an advantage.[17,27] Because competitive sports

activity may start at a young age, it is not surprising to observe weight cycling in adolescent athletes too.[28]

Another high risk group for weight fluctuations are people exposed to unintentional weight loss owing to recurrent chronic diseases with episodic remissions, such as cancer, alcoholism and gastrointestinal diseases, or simply to intermittent food availability. In developing countries mostly, fluctuations in food intake owing to food availability are not uncommon. For example, profound weight cycling due to an annual hungry season has been reported in rural African populations.[29] Scarceness in food supply can be irregular, with periods of severe food restriction related to unusual drought, war or political instability followed by periods of better food supply. This would lead to fluctuations in food intake and thereby in body weight. As children are often the most vulnerable to food availability and yet have high-energy demands, they are the most likely to suffer from fluctuations in food intake in a context of food insecurity.

Potential cardiovascular risk factors promoted by weight cycling

Intentional weight cycling has been implicated in many potential deleterious health consequences, such as obesity, disordered eating behavior and other psychological disorders, cancer, bone fracture risk, type 2 diabetes and hypertension. Although the topic of health consequences of weight cycling has been the source of considerable controversy,[30,31] several large population-based prospective studies have clearly reported an increased risk for all-cause and cardiovascular mortality in association with weight cycling, even after adjusting for preexisting disease.[32–36] The pathways from weight cycling to an increased cardiovascular mortality are not well understood. However, several cardiovascular risk factors associated with weight cycling have already been identified. These are as follows.

Enhanced weight gain

Although a larger weight gain after weight cycling has not been found systematically in all studies, some weight gain associated with weight cycling has been reported in obese women,[37] in young and middle-aged women in the Nurses' Health Study II,[38] in German non-smoking subjects from the general population,[7] in middle-aged Japanese men[39] and in elite athletes.[40] If these observations are confirmed, they are of major public health interest because there is growing evidence that weight gain is an independent risk factor for the development of type 2 diabetes,[41] hypertension,[42] cardiovascular diseases[43] and some types of cancer.[44,45]

Total body and visceral fat accumulation

It has been suggested that weight cycling may change body composition, by reducing lean body mass and/or increasing total body and visceral fat. However, human data are not fully convincing as only a few studies have shown differences in body composition between cyclers and non-cyclers, with a greater amount of total body fat[46] and a higher waist-to-hip circumference ratio[47] in cycling women. Animal data may be more conclusive, although not all studies have demonstrated excess body fat as a result of weight cycling.[48,49] Lim *et al*.[50] reported greater fat deposition in rats subjected to weight cycling (four cycles of 7 days of food restriction followed by 7 days of refeeding) than in control rats receiving the same overall energy supply as a constant food intake. Obese spontaneously hypertensive rats submitted to three cycles of very low calorie diet followed by *ad libitum* refeeding show higher retroperitoneal fat depots than *ad libitum* fed control rats despite a similar cumulative food intake.[51] Finally, we could demonstrate in young 6-week-old rats that repeated cycles of 3-day food restriction followed by 3-day refeeding led to greater epididymal and retroperitoneal fat depots as well as total body fat than a similar overall food intake given as a fixed daily portion.[52]

Preferential fat deposition could be due to a stimulation of lipogenic enzymes in white adipose tissue, as this has been consistently seen in rats submitted to a single cycle of starvation or restriction and refeeding.[53,54] Furthermore, this upregulation may be amplified by repeated cycles of weight loss and regain. Indeed, in rats submitted to either 1, 2 or 8 cycles of 3 days starvation followed by 3 days of refeeding, there was a progressive increase in mRNA levels of several key lipogenic enzymes in white adipose tissue, such as fatty acid synthase, acetyl-CoA-carboxylase, ATP citrate lyase and in the two enzymes providing nicotinamide adenine dinucleotide phosphate for fatty acid synthesis, namely malic enzyme and glucose-6-phosphate dehydrogenase.[55] In rats subjected to 2 cycles of 7-day 40% food restriction followed by 10-day *ad libitum* intake, key hepatic and white adipose tissue lipogenic enzymes show wide swings, with suppression during the period of food restriction and stimulation during refeeding.[56] Altogether, these studies suggest that accelerated fat gain during refeeding is mediated by an upregulated lipogenic activity in adipose tissue, which is further amplified by the number of starvation-refeeding cycles.

Finally, a simple redistribution of fat to the central areas without changes in body composition may be another possible link between weight cycling and cardiovascular diseases. Body fat distribution of the android type (prevalently abdominal) rather than of the gynoid type (prevalently gluteal) is more likely to be found in obese women with a history of weight cycling[57–59] and is known to be a major risk factor for metabolic and cardiovascular diseases. In non-obese women, controlled for age and parity, a higher waist-to-hip ratio (reflecting more central fat deposition) was significantly associated with a higher degree of weight cycling.[47] However, there are studies evaluating visceral fat by magnetic resonance imaging that could not find increased visceral fat in obese[60] and non-obese[61] cyclers, although in the latter study there was an increase in subcutaneous abdominal fat.

Alteration in the composition of tissue lipids

Weight cycling may not only lead to fat accumulation, but may also change the fatty acid composition of the accumulated fat. To address this question, Sea *et al*.[56] compared control rats maintained on *ad libitum* intake to rats submitted to a weight cycling protocol consisting of two cycles of 7-day 40% caloric restriction followed by 10 days of *ad libitum* refeeding. Rats were killed at different time points to measure various humoral and tissue parameters, and to perform an analysis of carcass fatty acid composition. Weight cycling led to a progressive enrichment of the proportion of saturated fatty acid (myristic acid 14:0, palmitic acid 16:0 and stearic acid 18:0) in the carcass lipids, whereas the proportion of the essential polyunsaturated fatty acids linoleic acid 18:2(n-6) and alpha-linolenic acid 18:3(n-3) decreased steadily, resulting in a pronounced decrease of the ratio of essential polyunsaturated to saturated fatty acids. The mechanisms of these changes could be related to a general catabolism of all fatty acids with preferential oxidation of essential fatty acids during the energy restriction period,[62] whereas the strong lipogenesis observed during the refeeding period allows the body to easily replenish the stores of saturated fatty acids, but not of essential fatty acids (mammals are unable to synthesize polyunsaturated fatty acids, in particular linoleic and alpha-linolenic acid, and must get them through the diet). Rapid weight loss in humans is also associated with accelerated depletion of α-linolenic acid.[63] If the animal observations of Sea *et al*. can be extended to weight cycling in humans, they could contribute to overall cardiovascular morbidity as a lower level of linoleic acid in human adipose tissue is associated with a higher risk for coronary heart disease.[64]

Insulin resistance and type 2 diabetes

In a 6-year follow-up of young and middle-aged female nurses from the Nurses' Health Study II, weight cycling was strongly associated with BMI and, furthermore, BMI at baseline had a strong association with the risk of developing diabetes, but weight cycling was not independently predictive of developing type 2 diabetes.[65] However, a direct association between weight cycling and fasting hyperinsulinemia has been demonstrated in lean and mildly overweight Japanese subjects.[66] In a cross-sectional analysis of 1932 middle-aged Japanese men with an average BMI of 22.7 kg/m^2, a positive association was found between fasting insulin concentration and a history of weight fluctuations (over the last $\sim$30 years). Individuals with larger weight fluctuations had higher fasting insulin, independently of BMI and other confounding factors (P-value for trend <0.001). When separating the participants into two groups (i.e. normal weight with a BMI <25 kg/m^2 and overweight with a BMI $\geqslant 25$ kg/m^2), this association remained statistically significant in the normal weight subgroup ($P=0.002$), whereas in the overweight subgroup the correlation became weaker ($P=0.077$). In another study of a second group of 664 middle-aged Japanese men investigating the relationship between weight cycling and the metabolic syndrome, a positive correlation was found between high fasting glucose and a history of weight cycling, and similarly the significance was only seen in the group of men with a BMI <25 kg/m^2.[39] Altogether, these observations suggest that weight cycling may promote insulin resistance in normal weight subjects and may be deleterious to health by promoting diabetes, either *per se*[39,66] or by favoring weight gain.[65]

Dyslipidemia

Alterations in blood lipids, characterized by a decrease in the cardioprotective high-density lipoprotein-cholesterol (HDL-C) or by an increase in the risk-promoting lipids, such as total plasma cholesterol, low-density lipoprotein-cholesterol and triglycerides, can have a strong deleterious impact on cardiovascular diseases. Olson *et al*.[67] investigated the impact of weight cycling on blood lipids in a cross-sectional study of 485 women with coronary risk factors undergoing coronary angiography. In this study, the Women's Ischemia Syndrome Evaluation study, 27% of the women reported a history of weight cycling, defined as voluntary weight loss of $\geqslant 4.5$ kg at least three times. They had significantly lower HDL-C levels (7%) than non-cyclers and showed a 'dose–response' effect, greater magnitudes of reported weight cycles being associated with significantly lower HDL-C. Similar decreases in HDL-C have been shown in men to be associated with an increased risk of cardiac events.[68] Alterations in blood lipids have also been reported in a cross-sectional study exploring the association of long-term body weight fluctuations with components of the metabolic syndrome in middle-aged Japanese men.[39] Hypertriglyceridemia and low HDL-C, but also hypertension and insulin resistance, were significantly associated with higher body weight variability, a significance that was evident in the group of men with BMI <25 kg/m^2, whereas significance was lost in the group of men with BMI $\geqslant 25$ kg/m^2. A significant increase in plasma triglycerides was also found in five young, non-obese (BMI 20.5 kg/m^2), healthy Japanese women who underwent in a well-controlled experimental trial two cycles of diet-induced weight loss of about 4.4 kg and *ad libitum* weight regain.[69]

Hypertension

Several studies in rodents have shown that weight cycling increases blood pressure during the weight regain period, but a long-lasting effect on blood pressure has not always been demonstrated. For example, in female rats undergoing two cycles of 3 weeks of 60% food restriction followed by 5 weeks of *ad libitum* refeeding, Miller *et al*.[70] observed that systolic blood pressure was increased during the first week of each refeeding period, but no longer 5 weeks after the beginning of refeeding. In contrast, Ernsberger *et al*.[51,71,72] could repeatedly demonstrate a sustained effect of refeeding on blood pressure, whereas others, using a comparable dietary

protocol, could not reproduce at all an effect of weight cycling on blood pressure.[73] A limitation of all these studies is that blood pressure was measured by tail cuff, a method that yields much more variable results than state of the art techniques by telemetry.[74] Furthermore, rats may not be the best model to study obesity-induced hypertension as, even within the same strain of rats (Sprague–Dawley), feeding rats a high-fat diet does not always produce hypertension,[75] whereas dogs[76,77] and rabbits[78] show consistent increase in blood pressure with diet-induced obesity.

The association of weight cycling and hypertension has been investigated in many human studies, often with positive results. Diagnosed hypertension was more frequently reported among male weight cyclers in a survey of Finnish adults of the general population.[8] In the EPIC study, short-term weight changes had a substantial impact on the risk of developing hypertension among obese subjects but not among non-obese subjects.[79] In a smaller study, when 96 android (i.e. central-type) obese women with a history of weight cycling, defined as diet-induced weight loss of $\geqslant$4.5 kg at least five times in the previous 5 years, were compared to 96 non-weight cycling controls matched for age, BMI and waist-to-hip ratio, a history of weight cycling was a strong predictor of hypertension.[58] Hypertension was also more frequent in non-obese Japanese men with a long history of weight variability.[39] Finally, in the experimental study of weight cycling in five non-obese Japanese women who went through two voluntary cycles of weight loss and regain, systolic and diastolic blood pressures were both significantly elevated at the end of the study, more than 100 days after the end of the second weight-loss period.[69]

Other investigators could only assess an indirect association between weight cycling and hypertension. In the Nurses Health Study II, weight cycling was strongly associated with body weight and body weight gain over the 4-year period of assessment, and both body weight and body weight gain were strong predictors for the development of hypertension.[6] However, weight cycling *per se* was not independently associated with hypertension but only through its adverse effect on body weight. Similar findings have been reported by Graci *et al*.[80] Observations of higher blood pressure with weight gain are not surprising, as there is a continuous positive relationship between BMI and blood pressure, even in the normal range of BMI.[81]

An additional potential risk factor, not yet well investigated in weight cyclers, could be related to the phenomenon of 'non-dipping', that is, the lack of a normal decrease in blood pressure during the night. Non-dipping of blood pressure is a known cardiovascular risk factor[82] and may hypothetically occur during the weight regain period, as overfeeding in rabbits[83] and dogs[84] has been shown to suppress the normal night–day difference in blood pressure. Studies with 24 h ambulatory monitoring of blood pressure are needed to test the hypothesis of a greater prevalence of non-dippers among weight cyclers.

In summary, there is growing evidence that many cardiovascular risk factors are promoted by weight cycling, either *per se* or indirectly via a small weight gain, thereby contributing to the increased cardiovascular morbidity and mortality of weight fluctuations. An interesting observation from the studies analyzed above is that the negative health consequences of weight cycling are readily seen in people of normal weight.[39,66,69] As the onset of a pattern of weight cycling is shifting towards younger ages and, as cardiovascular risk factors, even when only moderately present, act together in multiplicative way to lead slowly over many years to cardiovascular events, an increase in the prevalence of cardiovascular diseases associated with weight cycling is expected in the next few decades.

Additional mechanisms linking weight cycling to cardiovascular diseases: the 'repeated overshoot' theory

Many weight cycling studies investigate humans in cross-sectional studies with a single snapshot in a history of weight cycling, comparing cyclers and non-cyclers, or during a period of stable weight rather than longitudinally by a prospective study during weight cycling. Fluctuations of certain risk parameters, such as blood pressure, sympathetic hyperactivity or blood lipids, may thus be overlooked as those parameters may be fully normal in a situation of relatively stable weight or will add a background noise in the statistical analysis, obscuring the true fluctuations during weight cycling. Yet, fluctuations of several cardiovascular risk parameters do occur during fluctuations in food intake. Higher than normal values of blood pressure, heart rate, sympathetic activity, glucose, insulin, triglycerides and cholesterol, as well as glomerular hyperfiltration have all been described during rapid weight gain after a period of food restriction. Repeated overshoots of those variables could further enhance cardiovascular diseases as shown in Figure 2 and detailed below.

Fluctuations in blood pressure and heart rate

Caloric restriction decreases sympathetic activity and overfeeding increases sympathetic activity.[85] Fluctuations in food intake may thus lead to swings in sympathetic nervous activity, which in turn may contribute to swings in blood pressure. Indeed, Ernsberger *et al*.[86] reported in weight-cycled rats wide changes in 24-hour urinary catecholamine excretion, which paralleled weight fluctuations, that is, a decrease during food restriction and an increase during refeeding, and also paralleled blood pressure changes. An increase in sympathetic nervous activity during diet-induced weight gain may explain a large part of the hypertension of overfeeding.[87] Even, moderate fluctuations in food intake have been shown in rabbits to have small, but significant effects on blood pressure, and profound effects on heart

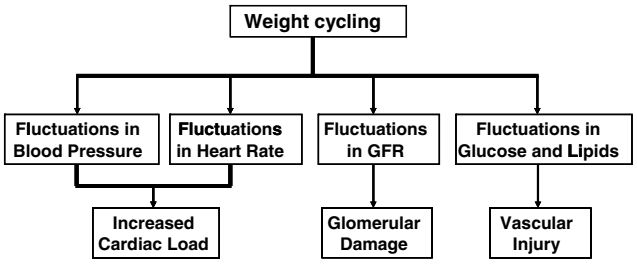

Figure 2 The 'repeated overshoot' theory: overshoot of some cardiovascular risks factors during the weight regain phase of weight cycling may contribute to overall cardiovascular morbidity and mortality even when the average values are normal. GFR, glomerular filtration rate.

rate.[88] In humans, significant fluctuations of blood pressure have been reported in the second of two weight cycles in the experimental study of Kajioka *et al*.[69] in young non-obese women. Repeated overshoots of blood pressure, heart rate and sympathetic activity during weight regain put an additional load on the heart and blood vessels, a load that is probably not compensated by lower values of these variables during weight loss. Increases in blood pressure and heart rate also elevate naturally the product of blood pressure and heart rate, the so-called double product, which raises myocardial oxygen demands.[89] Conceivably, fluctuations of all these hemodynamic variables with transient overshoots over control values during periods of higher food intake could have damaging cardiovascular effects. Indeed, blood pressure variability (although this has been demonstrated for fluctuations within a shorter time frame) is a cardiovascular risk factor independent of mean arterial pressure. In several clinical investigations, hypertensive target-organ damage was more advanced in patients with increased blood pressure variability.[90,91] These observations have been confirmed in large clinical studies, such as the PAMELA study[92] and the Syst-Eur trial.[93]

Fluctuations in renal function

Glomerular filtration rate (GFR) is modulated by food intake, increasing with feeding and decreasing during food restriction, with changes that are seen already within the first 24 h of an alteration of food intake.[94] Fluctuations in food intake associated with weight cycling may thus lead to fluctuations in GFR, and thereby to fluctuations in glomerular pressure. The long-term damaging effects of an increase in glomerular pressure have been well described,[95] and in the case of weight cycling may not be compensated by the protecting effect of a decrease in glomerular pressure during food restriction. Furthermore, glomerular hyperfiltration during weight regain may render the kidney more vulnerable to other cardiovascular risk factors, such as blood lipids or circulating inflammatory cytokines. For example, it has been suggested that hypercholesterolemia and glomerular hypertension act synergistically to cause glomerular sclerosis.[96] As blood lipids may overshoot during refeeding (see below), weight cycling may lead to a gradual loss of nephron function that worsens with time and exacerbates hypertension.

Fluctuations in plasma glucose, insulin and blood lipids

Fluctuations in plasma glucose, insulin, triglycerides and total cholesterol have been well described by Sea *et al*.[56] in an animal model of weight cycling, with undershoots during food restriction and overshoots during each refeeding interval. In their study in non-obese young women, Kajioka *et al*.[69] also reported wide fluctuations in plasma triglycerides during their two experimental weight cycles. Here again, fluctuations of plasma glucose, cholesterol and triglycerides with repetitive overshoots above control values may lead with time to greater vascular damage than average values with little fluctuations.

Conclusions and perspectives

Weight cycling is not limited to obese adults but affects people of normal weight, particularly young women, who are unhappy with their appearance. Furthermore, the onset of a pattern of weight cycling is shifting towards younger ages, owing to the increasing prevalence of overweight and obesity in children and adolescents, and the pressure from the media and society for a slim image even for normal weight children. Although there is still controversy whether weight cycling promotes body fat accumulation and obesity, many large-scale prospective studies have shown an association between weight fluctuations and cardiovascular morbidity and mortality. Higher prevalence of hypertension, accumulation of visceral fat, insulin resistance and dyslipidemia are more likely to occur in weight cyclers of normal body weight and may all contribute to cardiovascular risks. In addition, fluctuations of cardiovascular risk variables, such as blood pressure, heart rate, sympathetic activity, blood glucose and lipids, with probable repeated overshoots above normal values during periods of weight regain, put an additional stress on the cardiovascular system. As the prevalence of diet-induced weight cycling is increasing due to the opposing forces of an 'obesigenic' environment and the media pressure for a slim figure (that even targets children), weight cycling, particularly in girls and younger women, is likely to become a serious public health issue.

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