Effects of Intentional Weight Cycling on Non-Obese Young Women

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This study investigated the effects of intentional weight cycling in non-obese, young women (n = 5; mean age, 24.6 years; mean body mass index [BMI], 20.5 kg/m^2). During the first 30 days, the subjects lost more than 4 kg with energy restriction alone (first diet period). In the following 14 days, they regained more than the weight they had lost by eating ad libitum (free-living period). In the subsequent 30 days, they once again lost more than 4 kg with only energy restriction (second diet period). Height, weight, waist and hip circumferences, body composition, lipid profiles, thyroid hormones, systolic and diastolic blood pressures (SBP, DBP), and resting energy expenditure (REE) were examined. Measurements were taken at the beginning of the study (day 0), at the end of each diet period (day 30 and day 74), the end of the free-living period (day 44), and on day 180. The mean change in each variable from baseline (day 0) was used and controlled for the baseline value, baseline weight, and change in weight (△weight). Statistical tests were performed to determine the significance of the mean changes in the variables. By day 180, there were significant decreases in the subjects' lean body mass ($P < .01$), serum triiodothyronine (T_3) ($P < .001$), serum total thyroxine (T_4) ($P < .001$), and REE ($P < .001$), and significant elevations of SBP ($P < .05$) and DBP ($P < .001$). The lipid profiles had not changed except for increased triglycerides (TG). These results suggest that weight cycling through energy restriction alone may have negative health consequences in non-obese, young women.

WEIGHT CYCLING OR yo-yo dieting is defined as a repeated cycle of weight loss and regain. This phenomenon is often observed in obese people who lose weight, fail to maintain the lower weight, and then bounce back to their former weight or higher. Recently, because a slim body image is widely promoted by the media and society, dieting for weight loss has spread among people who are not overweight, especially young women.[1] Weight cycling is also prevalent in normal weight female dieters who use inappropriate dieting techniques, such as crash dieting or fasting; it is a serious public health issue.[2] Although many studies have been conducted examining the effects of weight cycling, most subjects represented obese populations.[3-6] Few studies have focused on young, non-obese women, the age group more likely to try various diets and experience weight cycling.

The retrospective or observational studies that have been performed looking at the potential effects of weight cycling on health have several limitations.[7-9] First, the existing data sets were not designed to investigate the effects of weight cycling. Because recorded weights were separated by months or even years, little was known about weight fluctuation between assessments. Second, these studies did not attempt to distinguish intentional from unintentional weight loss or discuss the reason for the weight loss. Therefore, a real cycle of weight gain followed by weight loss in a well-controlled experimental trial is needed to clarify the health consequences of weight cycling.

In the late 1980s and early 1990s, some studies were conducted to investigate whether the cycle of weight loss and regain affects metabolic outcomes, specifically a decrease in metabolic rate[6,10,11] and a changed body composition[12,13] with a redistribution of fat to the abdomen that, in turn, would adversely affect cardiovascular risk factors.[4,14,15] Although these studies contributed a great deal to the understanding of weight cycling, some of the work had conflicting results. However, in a recent review of studies regarding weight change and metabolic rate, Astrup et al[16] demonstrated that weight-reduced subjects have a lower energy expenditure than control subjects, and Weinsier et al[17] indicated that acute and chronic energy restriction induced a decrease in body weight and metabolic rate. These findings suggest that thyroid hormone levels may be useful for documenting subjects' metabolic state in our controlled study.

The purpose of this study was to clarify the effect of intentional weight cycling through energy restriction alone in non-obese, young, healthy women. To achieve this, we examined subjects' body composition, body fat distribution, lipid profiles, thyroid hormones, blood pressure, and resting energy expenditure (REE) over a 180-day period.

MATERIALS AND METHODS

Subjects

The subjects were 5 healthy, non-obese women aged 22 to 34 years (mean age, 24.6 years). All subjects were nonsmokers and were not taking any medications including the oral contraceptive pill. Subjects were prohibited from performing their regular exercise during the data-collecting period from day 0 to day 180. The study was approved by the appropriate institutional review boards of Nagoya University, and each subject gave her written consent after a full explanation of the procedures and risks.

Study Design

The study design is presented in Fig 1. During the first 30 days, the subjects lost more than 4 kg with energy restriction alone (first-diet period). In the following 14 days, they regained more weight than they had lost by eating ad libitum (free-living period). During the next 30 days, they once again lost more than 4 kg with only energy restriction (second diet period). They were permitted an energy consumption of less than 5,016 J (1,200 kcal)/d and were supervised by a dietitian during each diet period. Each subject cooked nearly all of her meals by

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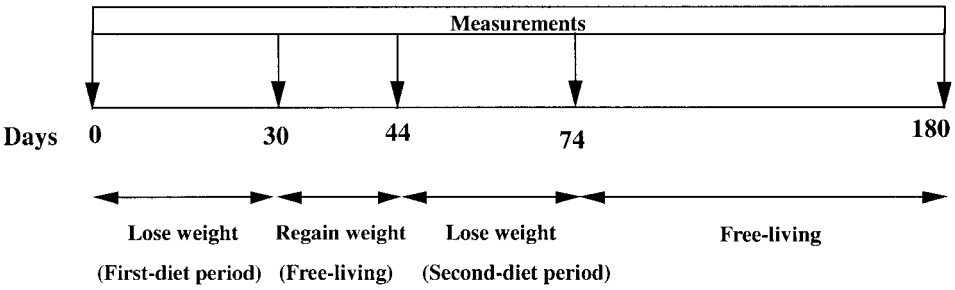

Fig 1. Study design. Measurements including height, weight, body composition, waist and hip circumferences, lipid profiles, thyroid hormones, blood pressure, and REE were taken 5 times over 180 days.

herself. They made rough estimates of their energy intake according to the dietitian's directions, which focused on a nutritional balance except for the energy intake. Dehydration, diuretic drugs, and laxatives were prohibited. The subjects recorded their weight, food intake, work schedule, and their physical and mental conditions daily during the 180 days of the study. Subjects could consult with physicians at anytime, and a psychologist followed this study throughout. Measurements were taken at the beginning of the study (day 0), at the end of each diet period (day 30 and day 74), and at the end of the free-living period (day 44). Furthermore, to clarify the long-term effects of weight cycling, we also took measurements at day 180. Subjects fasted overnight (at least 12 hours) prior to each measurement day. All measurements were made between 9 AM and 12 PM and were performed at the same time each day for a given subject.

Anthropometry

Anthropometry measurements were made with the subjects in bathing suits and without shoes. Height was measured with an electronic stadiometer to the nearest 0.1 cm. Waist circumference was measured at the minimal abdominal girth, and hip circumference was measured at the greatest protrusion of the gluteal muscles. Waist-to-hip ratio (WHR) was then determined by dividing the waist circumference by the hip circumference. Body weight and body fat were determined with the BODPOD Body Composition System (Life Measurement Instruments, Concord, CA), which uses air displacement to measure body volume.[18] Body density was calculated as weight divided by body volume, and percent body fat was estimated by the Brozek equation.[19] Fat mass (FM) was determined from the percent body fat, and lean body mass (LBM) was calculated by subtracting FM from total body weight. Height and weight data were used to calculate the body mass index (BMI, kg/m^2).

Lipid Profiles and Thyroid Hormones

Venous blood was drawn after overnight fasting and the following parameters were determined 5 times (day 0, 30, 44, 74, and 180): total cholesterol (TC) and triglycerides (TG) were measured by an enzymatic method using commercial kits (Shinotest, Tokyo, Japan); high-density lipoprotein (HDL) cholesterol was measured by a selective inhibition method using commercial kits (First Chemical, Tokyo, Japan); low-density lipoprotein (LDL) cholesterol was calculated by Friedewald's formula[20]; triiodothyronine (T_3), serum total thyroxine (T_4), and thyroid-stimulating hormone (TSH) were determined by chemiluminescent immunoassay. Lipoprotein lipase (LPL) was determined only 3 times (days 0, 74, and 180) by enzyme immunoassay.

Blood Pressure

Sitting systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured at the right upper arm after at least 10 minutes of rest using an electronic sphygmomanometer (COLIN, 203RVII, Aichi, Japan). The measurements were repeated after 5 minutes, and the average of the 2 measurements was used in the analysis.

Resting Energy Expenditure

The REE was obtained with an indirect calorimeter (Metavain, VMB-002N, Tokyo, Japan).[21] This device measures the concentration of expired oxygen with a stabilized zirconia oxygen sensor having a coefficient of variation (CV) of ± 0.1%.[22] All measurements were conducted at the National Institute for Longevity Sciences, Metabolic Testing Laboratory. The laboratory was maintained at 67° to 72°F, and the noise level was minimized. After a 12-hour fast, the subjects' REEs were assessed before other measurements were taken. Strenuous physical activity was prohibited 24 hours prior to the REE measurement. Subjects rested at least 15 minutes in the supine position before measuring REE for 15 minutes or longer or until a CV of less than 0.5% was observed for at least 10 minutes. Subjects were instructed to remain awake, but relaxed, and to refrain from voluntary skeletal muscle activity during the test.

Statistical Analysis

To evaluate the effects of weight cycling, the mean change in each variable was controlled for the baseline value (day 0), baseline weight, and change in weight (Δweight) using a general linear model. Statistical tests were completed with Student's paired *t* test to determine the significance of the mean changes in the variables. A 2-tailed *P* value less than .05 was considered statistically significant. Data were analyzed using the Statistical Analysis System version 6.12 (SAS Institute, Cary, NC).[23]

RESULTS

Anthropometry

Subjects' measurements at the beginning of the study are shown in Table 1. Mean height was 159.4 cm, mean weight was 52.1 kg, and mean BMI was 20.5 kg/m^2. The changes in mean weight and percent body fat over the course of the study are shown in Table 2. Weight losses and weight gains were significant when compared with baseline weight except on day 180. The proportion of FM and LBM in weight loss or weight gain is also shown in Table 2. The mean changes in FM and LBM, controlled for the baseline values, baseline weight and Δweight, are shown in Fig 2. The FM was significantly lower

Table 1. Subject Characteristics at the Beginning of the Study

Subject No.	Age (yr)	Height (cm)	Weight (kg)	Fat (%)	BMI
1	21	159.2	50.40	23.8	19.9
2	21	154.4	49.40	21.1	20.7
3	22	161.2	58.02	29.3	22.3
4	25	155.9	47.70	22.6	19.6
5	34	166.0	54.85	20.3	19.9

Table 2. Mean Change in Body Weight, Percent Body Fat and the Proportion of FM Versus LBM

Days	Weight (SE) (kg)	Body Fat (SE) (%)	Proportion of FM *v* LBM in Weight Loss and Weight Gain
0 (baseline value)	52.07 (1.90)	23.6 (1.7)	
			−4.42 (72% FM *v* 28% LBM)
30 (first diet)	47.65 (1.93)	19.1 (2.2)	
			5.61 (55% FM *v* 45% LBM)
44 (free living)	53.26 (1.80)	22.9 (1.3)	
			−4.55 (59% FM *v* 41% LBM)
74 (second diet)	48.71 (1.79)	19.5 (1.8)	
			2.94 (95% FM *v* 5% LBM)
180 (free living)	51.65 (1.94)	23.8 (1.9)	

than its baseline value on day 30 ($P < .001$) and day 74 ($P < .001$). The LBM was significantly lower than its baseline value on day 30 ($P < .001$), day 74 ($P < .01$) and day 180 ($P < .01$). Waist and hip circumferences and WHR were not significantly different between baseline values and day 180.

Lipid Profiles and Thyroid Hormones

Table 3 shows the mean changes in lipid profiles and thyroid hormones, which were controlled for baseline values, baseline weight, and Δweight. These variables were dependent on changes in body weight, that is, they declined on day 30 and day 74, and increased on day 44 and day 180. For TC, LDL, HDL, and LPL, there were no significant differences between baseline values and day 180, whereas, TG on day 180 was significantly higher than its baseline value ($P < .05$). Throughout the study, T_3 and T_4 were significantly lower than their baseline values ($P < .001$). Although TSH was significantly lower than its baseline value ($P < .001$) on day 30, there was no significant difference between the baseline value and day 180.

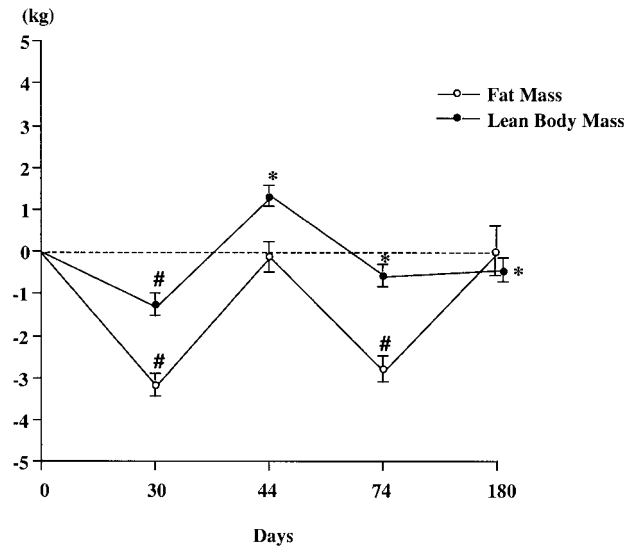

Fig 2. Mean change in FM (○) and lean body mass (●) controlled for baseline values, baseline weight, and Δweight. Error bars are SE of individual changes. *$P < .01$, #$P < .001$ *v* baseline value.

Blood Pressure

The mean change in SBP and DBP, controlled for their baseline values, baseline weight, and Δweight, are presented in Fig 3. The SBP was significantly lower than its baseline value ($P < .001$) on day 74 and significantly higher than its baseline value ($P < .05$) at day 180. The DBP was significantly lower than its baseline value ($P < .05$) on day 74 and significantly higher than baseline value on day 44 and day 180 ($P < .01$).

Resting Metabolic Rate

The mean change in REE, controlled for its baseline value, baseline weight, and Δweight are shown in Fig 4. On day 30 and day 74 (the end of the first and second diet periods), the REEs were significantly lower than the baseline value ($P < .001$). On day 44 (end of free-living period), REE was higher than the baseline value ($P < .001$). On day 180, however, REE had not returned to the baseline value and had even become significantly lower than its baseline value ($P < .001$). The mean change in REE per kilogram LBM was similarly changed; there was a significant difference between the baseline value and day 180 ($P < .01$).

DISCUSSION

In this study, we investigated the effects of intentional weight cycling on body composition, body fat distribution, lipid profiles, thyroid hormones, blood pressure and REE in young, non-obese, healthy women. Although several studies have demonstrated that weight cycling did not change body composition,[24,25] these were cross-sectional studies that looked

Table 3. Mean Change From Baseline Values in Lipid Profiles and Thyroid Hormones

	Day 30 (first diet)	Day 44 (free living)	Day 74 (second diet)	Day 180 (free living)
TC	−20.0 ± 11.6	6.20 ± 1.50†	−13.4 ± 1.33‡	0.80 ± 3.22
TG	−2.40 ± 1.27	17.8 ± 0.68‡	−4.60 ± 3.33	14.8 ± 3.86*
LDL	−18.8 ± 7.73	−7.40 ± 3.04	−9.20 ± 1.05‡	−5.20 ± 2.59
HDL	−0.80 ± 1.34	10.0 ± 3.61*	−3.40 ± 1.46	3.00 ± 2.18
LPL			4.20 ± 38.3	−26.2 ± 40.9
T_3	−50.8 ± 0.61‡	−16.0 ± 0.94‡	−49.0 ± 0.88‡	−31.8 ± 0.83‡
T_4	−1.16 ± 0.10‡	−0.44 ± 0.04‡	−1.60 ± 0.02‡	−1.38 ± 0.02‡
TSH	−0.55 ± 0.05‡	−0.12 ± 0.10	−0.72 ± 0.45	−0.55 ± 0.36

NOTE. Controlled for baseline value, baseline weight, and Δweight. Values are mean ± SE.

*$P < .05$, †$P < .01$, ‡$P < .001$ *v* baseline value (day 0).

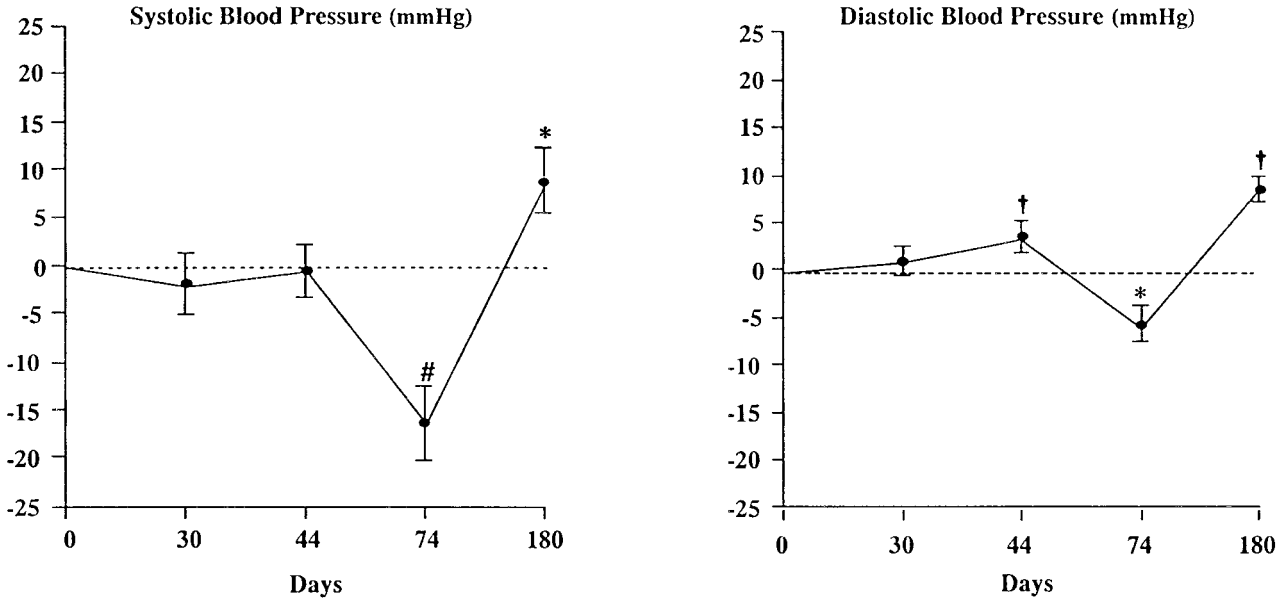

Fig 3. Mean change in SBP and DBP controlled for baseline values, baseline weight, and Δweight. Error bars are SE of individual changes. *$P < .05$, †$P < .01$, #$P < .001$ *v* baseline value.

at obese people. In contrast, our study was conducted to clarify the longitudinal effects of weight cycling on body composition by intentionally causing weight loss and weight gain, with the focus on non-obese, young women. By day 180 at the end of the study, FM was not significantly changed, however, LBM was significantly decreased compared with its baseline value.

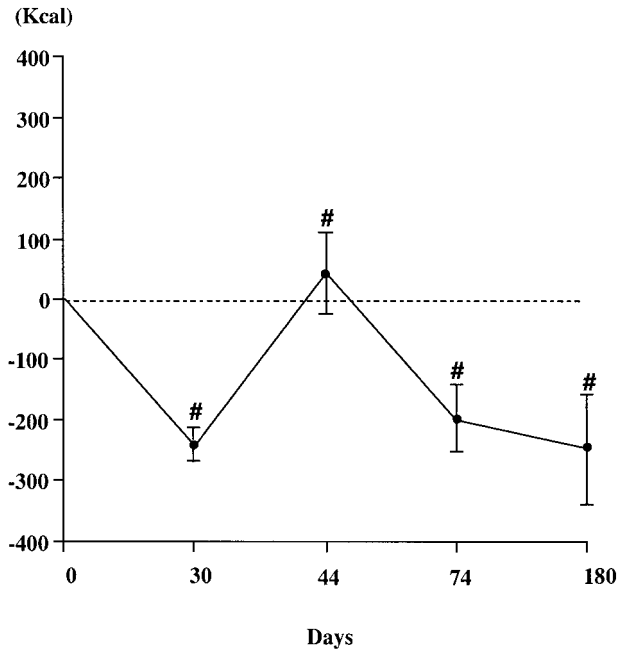

Fig 4. Mean change in REE controlled for baseline value, baseline weight, and Δweight. Error bars are SE of individual changes. #$P < .001$ *v* baseline value.

Weight loss regimens with energy restriction alone accelerate the loss of LBM compared with energy restriction in combination with exercise training.[26,27] In this study, the subjects lost their weight with energy restriction alone, and they were prohibited from participating in their regular exercise during the 180 days of the study. If the subjects were to lose weight with energy restriction plus exercise training, the increasing loss of LBM might be prevented. Our results suggest that the proportion of lost LBM increased, and the rebuilding of lost LBM decreased due to weight cycling through energy restriction alone.

In our study, body fat distribution as determined by WHR, was not significantly different between the baseline parameters and day 180. Rodin et al[28] reported that a higher degree of weight cycling was associated with a higher WHR. They defined the degree of weight cycling by a "weight cycling index" based on frequency and amount of reported weight loss. We considered several possible reasons for the difference in WHR between these 2 studies. First, our subjects returned to baseline weight by day 180, and their weights were never classified as obese. If their final weights had greatly overshot their baseline weights or if BMI had reached the over weight range by day 180, they may have redistributed body fat shifting fat from peripheral locations toward the trunk or accumulating an intra-abdominal fat depot. Second, our subjects did not have a history of weight cycling prior to this study; a single weight cycle might be insufficient to induce redistribution of their body fat.

The lipid profile parameters changed in accordance with weight loss or weight gain, however, there were no significant differences between their baseline values and day 180 except for TG. Even though TG was significantly higher than the baseline value, it was still within normal range. These results

may indicate another reason why the WHR was not increased on day 180, since it is well known that increased WHR is strongly associated with risk factors for cardiovascular disease.[29] Similarly, 2 studies found no evidence that weight cycling increased cardiovascular risk or changed the WHR.[4,30]

Studies using recorded weights did not allow us to determine clear associations between weight cycling and its effects on blood pressure,[31,32] although Ernsberger and Nelson[33] found sustained hypertension in cycled rats: repeated starvation and refeeding increased blood pressure. This phenomenon probably can be explained by the mechanism called diet-induced hypertension in which a low energy intake results in decreased norepinephrine secretion to avoid excessive energy expenditure and, as a consequence, norepinephrine sensitivity in peripheral vessels increases to maintain blood pressure. Even if norepinephrine secretion returns to normal with increased energy intake after dieting, norepinephrine sensitivity remains elevated. This mechanism may lead to elevated blood pressure because of an increase in systemic vascular resistance.[34] This correlation between norepinephrine and weight change is not conclusive. Rosenbaum et al[35] reported urinary norepinephrine decreased significantly during and after weight loss, but Rio et al[36] did not detect that association. Ernsberger et al[37] found urinary excretion of norepinephrine, epinephrine, and dopamine had a several fold change in weight cycling rats. These changes paralleled changes in blood pressure, decreasing during caloric restriction and rebounding during refeeding. At day 180, the subjects' SBPs and DBPs were significantly higher than their baseline values. They experienced severe energy restriction during weight loss, followed by a period of ad libitum food intake (free-living period). This sort of intentional weight loss and gain is similar to the caloric restriction and refeeding process in animal studies. Although norepinephrine secretion was not measured in this study, our results suggest that diet-induced hypertension could have occurred in subjects.

Most studies concerned with the adverse effects of weight cycling are investigating whether or not weight cycling causes a reduction in REE, since a resistance to weight loss may lead to a low REE. A cross-sectional study in wrestlers has shown an association between weight cycling and decreased REE.[38] A longitudinal investigation of 8 obese subjects observed a reduction in REE after 1 weight cycle,[39] while other studies could not detect the association between weight cycling and a reduced REE.[6,10,11] Thus, studies assessing the effects of weight cycling on REE present contrasting findings.

We found that the mean change in REE on day 180 was 15.8% lower than its baseline value. Both T_3 and T_4 paralleled changes in REE. Nevertheless, although our subjects' weights bounced back to their baseline values by day 180 and energy restriction was not imposed after day 74, REE, T_3 and T_4 were still lower than baseline values. Additionally, the decreased LBM at day 180 may explain the subsequent reduction in REE, because REE correlates closely with LBM, but not FM.[40] Although the decreased levels of thyroid hormones and LBM may contribute to the decreased REE, other factors are probably involved in this relationship, for example, the sympathetic nervous system. The decline in catecholamine secretion after weight loss has been described as correlating with the decline in REE, because catecholamine secretion contributes to the regulation of REE.[35] This study did not examine hormones other than thyroid hormones, although the decrease of both LBM and the thyroid hormones might be associated with decreased REE.

This study was conducted in targeted subjects who were non-obese, young women. Most human studies have used self-reports by the subjects on weight cycling history. However, our study was conducted as a well-controlled experimental trial with measured, consistent weight cycles over 180 days. Both immediate and long-term effects of the weight cycling could be measured and the mean change of variables compared. Weight cycling through energy restriction alone induced a change in body composition, elevated blood pressure, and decreased REE. These findings suggest that weight cycling has truly negative health consequences.

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