ORIGINAL CONTRIBUTION

Effects of Weight Cycling on Coronary Risk Factors

Takashi Itoh¹, Seichi Horie², Ken Takahashi¹, and Toshiteru Okubo¹

Interest in the health hazards of weight loss and regain (weight cycling) is growing. This study examined the relationship between weight cycling during ages 21 to 35 and changes in coronary risk factors (systolic blood pressure, diastolic blood pressure, total cholesterol, and HDL cholesterol) during ages 35 to 40. We followed up 264 blue-collar workers from the time they were 21 to 40 years of age working for one company. Analysis of covariance was applied including the number of weight cycles during ages 21 to 35 as the independent variable and the changes in coronary risk factors between age 35 and 40 as the dependent variables. The covariates were adjusted for body mass index (BMI) at age 35, increase in BMI between ages 35 and 40, value of coronary disease risk factors at age 35, and smoking status at age 40. The results showed that increase in systolic and diastolic blood pressure was not significantly related to the number of weight cycles. For total and HDL cholesterol, interaction was seen between smoking status and the number of weight cycles, and the effects of weight cycle on cholesterol levels were examined by smoking status. The change in total cholesterol among frequent cyclers was not significantly different from that among non-cyclers. Though the increment of HDL cholesterol in frequent cyclers was significantly larger than that in non-cyclers for non-smokers, an inverse relationship was seen for heavy smokers (not significant). Our results showed the effects of weight cycling on changes in coronary risk factors only for HDL cholesterol based on analysis by smoking status. As the present study was conducted on a small population, interaction between weight cycling and smoking status on HDL cholesterol should be confirmed using a larger population. *J Epidemiol*, 1996 ; 6 : 55-62.

Weight cycling, weight variability, BMI, coronary risk factor, smoking

Weight reduction diets have become popular in accordance with growing health concerns in recent years. However, many people who successfully reduce weight often regain it.^{1,2)} The long-term health consequences of such weight changes are not well understood. Interest in the health consequences of weight loss and regain (weight variability) is on the rise.

A relationship between the long-term effects of weight variability and mortality in general or due to cardiovascular disease has been reported. As for general mortality, some have reported positive relationship of weight variability with an increase in mortality,^{3,4)} while others have not.^{5,6)} Many researchers have reported that weight variability is related to increase in mortality or morbidity from cardiovascular disease.^{3-5,7)}

To investigate the health effects of weight variability, several short-term or cross-sectional studies examined the relationship between changes in coronary risk factors and weight variability. For blood pressure and lipids, no relationship has been found.⁸⁻⁹⁾ For glucose tolerance, some researchers reported significant relationships¹⁰⁾, but others did not.⁸⁻⁹⁾

Some researchers examined the relationship between body fat or energy expenditure (e.g. resting metabolic rate) and weight variability. The hypothesis that weight variability affects body composition by increasing total body fat has been denied in all studies,^{8,11-14)} except one.¹⁵⁾ Regarding energy expenditure, some found that weight variability decreases resting metabolic rate,^{11,15)} but others reported negatively.^{8,13,16)}

Received November 30, 1995 ; accepted February 9, 1996.

¹Department of Environmental Epidemiology, Institute of Industrial Ecological Sciences, University of Occupational and Environmental Health, Kitakyushu, Japan ; ²Keihin Health Service Center, Nippon Kokan Hospital, Kawasaki-ku, Kawasaki, Japan
Address for correspondence : Takashi Itoh, Department of Environmental Epidemiology, Institute of Industrial Ecological Sciences, University of Occupational and Environmental Health, 1-1 Iseigaoka Yahatanishi-ku Kitakyushu, 807 Japan.

Although many long-term studies have observed an association of weight variability with negative health consequences, studies investigating the mechanism of the effects have failed to show a causal relationship. One explanation for this is that, in most studies, information on weight variability was based on each subject's recall and may therefore have been misclassified. A second explanation is that the effects of weight variability may be persistent but weak, and cross-sectional studies may fail to detect the effects.

The purpose of the present study was to demonstrate the effect of *weight cycling* (i.e., pattern of weight change caused by weight loss and gain, one aspect of weight variability) on coronary risk factors by examining the relationship between weight cycling during ages 21 to 35 and changes in coronary risk factors during ages 35 to 40, based on objective information on weight cycling.

METHODS

Subjects

This follow-up study was done with the cooperation of a company whose workers underwent periodic health examination (all workers must undergo periodic health examination every year under Japanese law) at the Keihin Health Service Center of the Nippon Kokan Hospital in Japan. The population consisted of 264 blue-collar male workers who joined the company between April 1968 and December 1971, at the ages of 18 to 21 years, and who worked at least ten years afterwards for the company. They were followed up for at least 20 years. Twenty-six workers retired during the follow-up period and were excluded from this study (follow-up rate 90.2%). No one retired due to death. None of the subjects had a disease for which he was receiving medication at the time of retirement.

In the follow-up period, thirty individuals had at least one of the following diseases: cerebrovascular disease, diabetes mellitus, cancer, coronary heart disease, hypertension, hypercholesterolemia, hypertriglyceridemia, liver dysfunction, thyroid disease, hyperuricemia. They were excluded from the following analyses because their risk factors may have been affected by medication. In addition, eight workers whose biological data for the periodic health examination were not available were excluded from the following analyses. As a result 200 of 264 subjects were analyzed in the present study.

Measures

Data on height and weight were obtained from health examination records at the company. Health examinations were conducted every year at almost the same season for each worker. Height and weight were measured at every examination. Height in standing position (to the nearest 0.1cm) was measured and weight (to the nearest 0.5kg) was measured using a calibrated platform scale. Subjects were measured in light clothing and without shoes.

Body mass index (BMI: weight in kilograms divided by the

square of the height in meters) was used as the index for relative weight. One weight cycle was defined as a cycle in which BMI consistently increased 5% or more from a point of any duration subsequent to an event with a decrease of 5% or more (this decreasing period did not include any increasing period). The number of weight cycles for each subject during the period from age 21 to 35 was counted.

Four coronary risk factors - systolic blood pressure (SBP), diastolic blood pressure (DBP), total cholesterol, and high density lipoprotein (HDL) cholesterol - were measured at ages 35 and 40. Blood pressure was taken on the left arm, sitting, after the subject had been seated for five minutes, using a standard mercury sphygmomanometer and a 14cm cuff. Systolic and fifth-phase diastolic blood pressure were measured to the nearest 2mmHg. Blood samples were taken from cubital veins and analyzed for total cholesterol and high density lipoprotein (HDL) cholesterol. Fasting was not required of subjects. Cholesterol and HDL cholesterol levels were quantified by enzymatic methods with an automatic analyzer (RX-30, NIHONDENSI). Investigation of smoking status (number of cigarettes) began at the examination in 1988. Thus, we used smoking status only at age 40, because smoking information for all subjects was available at age 40 (mean age of subjects in 1988 was 37.6). Information on status of health guidance in the health examination during ages 21 to 39 by health nurses was obtained from health examination records. This information revealed whether health guidance on obesity, hypercholesterolemia, or hypertension was or was not done, but time of length or content of guidance was not available.

Analysis

To examine the effects of weight cycling, the difference between values of the four coronary risk factors measured at ages 35 and 40 was used as the dependent variable. The independent variables were the number of weight cycles during ages 21 to 35. Because factors other than weight cycling may influence change in coronary risk factors, we also corrected for the following potential confounders: BMI at age 35, the difference between BMI at ages 35 and 40, level of the risk factors at age 35, smoking status at age 40, and status of health guidance during ages 35 to 39.

Descriptive statistics on dependent variables were calculated and the mean of these variables was compared between each weight cycling group, using t-test. The analysis-of-covariance model (the GLM procedure, SAS for Windows ver.6.08)¹⁷⁾ was applied to analyze change in each risk factor by the number of weight cycles, taking potential confounders into consideration. In this analysis, smoking status was used as categorical data (no smoking: non smoker, smoking ≤ 20 cigarettes per day: light smoker, smoking ≥ 21 cigarettes per day: heavy smoker). Status of health guidance was coded as "1" if the individual had received health guidance at least once during ages 35 to 39, and "0" if otherwise.

RESULTS

Subjects were grouped into three categories based on number of weight cycles during ages 21 to 35 (no weight cycle group : “non-cycler”, one weight cycle group : “one cycler,” two or more weight cycle group : “frequent cycler”). Twenty-three individuals had received health guidance at least once during ages 35 to 40. The mean of risk factors was calculated at ages 35 and 40, and differences between levels of risk factors at ages 35 and 40 were also calculated (Table 1). The mean of SBP, DBP, and total cholesterol increased with the number of weight cycles. However, significant differences between non-cyclers and frequent cyclers ($p<0.05$) were seen only for mean DBP at age 35, mean total cholesterol at age 35, and change in total cholesterol from age 35 to 40. BMI was significantly related to the number of weight cycles : the mean of BMI at any age was highest in the frequent cycler group and lowest in the non-cycler group. The number of smoking cigarettes per day was not different among weight cycling groups.

We compared the means of the four coronary risk factors at age 35 among the three weight cycling groups by adjusting for BMI at age 35 and smoking status at age 40 (Table 2). The differences in levels of coronary risk factors among weight

cycling groups decreased by the adjustment and none of them was statistically significant.

Using analysis of covariance, we examined the effects of weight cycling on change (i.e. increment) for each coronary risk factor, while controlling for BMI at age 35, the difference in BMI between age 35 and age 40, measurement value of this risk factor at age 35, smoking status at age 40, and status of health guidance during ages 35 to 39. In this analysis of covariance for increment in total cholesterol, interactions between weight cycling and smoking status at age 40, between health guidance and the difference of BMI, between health guidance and weight cycling, were significant ($p=0.034$, 0.004 , 0.009 , respectively). Significant interaction between weight cycling and smoking status at age 40 was found ($p=0.031$) in the model for increment in HDL cholesterol. All the above significant interaction terms were included in the analysis model. Mean increment in total cholesterol was almost significantly different between non-cyclers and frequent cyclers ($p<0.10$), as shown in Table 3. However, the direction was that weight cycling is associated with a decrease in the increment of total cholesterol. There was no significant difference in the amount of change for the other risk factors.

In order to adjust for the effect of interaction between

Table 1. The unadjusted mean of coronary risk factors and other characteristics by weight cycling group

	All (N=200)	Non-cycler (N=88)	One cycler (N=90)	Frequent cycler (N=22)
	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD
SBP (mmHg)				
at age 35	122.9 $\pm$ 13.0	122.0 $\pm$ 12.6	123.3 $\pm$ 13.1	124.3 $\pm$ 14.2
at age 40	125.7 $\pm$ 13.0	124.1 $\pm$ 13.5	126.7 $\pm$ 12.9	127.8 $\pm$ 11.2
age 40 - age 35	2.8 $\pm$ 12.3	2.1 $\pm$ 12.2	3.3 $\pm$ 12.3	3.5 $\pm$ 13.3
DBP (mmHg)				
at age 35	75.4 $\pm$ 9.9	74.3 $\pm$ 9.4	75.3 $\pm$ 10.4	80.2 $\pm$ 9.3*
at age 40	78.7 $\pm$ 8.9	77.9 $\pm$ 8.9	79.0 $\pm$ 9.2	80.1 $\pm$ 7.8
age 40 - age 35	3.3 $\pm$ 9.1	3.7 $\pm$ 9.2	3.7 $\pm$ 8.7	-0.1 $\pm$ 10.3
total cholesterol (mg/dl)				
at age 35	179.4 $\pm$ 35.2	175.0 $\pm$ 34.0	179.4 $\pm$ 33.8	196.5 $\pm$ 41.9*
at age 40	193.9 $\pm$ 32.6	194.2 $\pm$ 34.5	193.0 $\pm$ 31.4	196.1 $\pm$ 31.0
age 40 - age 35	14.5 $\pm$ 23.2	19.2 $\pm$ 22.6	13.6 $\pm$ 20.9	-0.3 $\pm$ 28.2*
HDL cholesterol (mg/dl)				
at age 35	47.5 $\pm$ 12.1	47.5 $\pm$ 12.3	47.8 $\pm$ 11.8	46.2 $\pm$ 13.1
at age 40	54.5 $\pm$ 14.4	55.9 $\pm$ 16.1	53.0 $\pm$ 13.1	55.3 $\pm$ 11.6
age 40 - age 35	6.9 $\pm$ 11.8	8.2 $\pm$ 13.1	5.1 $\pm$ 10.6	9.0 $\pm$ 10.4
BMI (kg/m²)				
at age 21	21.1 $\pm$ 2.0	20.5 $\pm$ 1.6	21.5 $\pm$ 2.1*	22.3 $\pm$ 2.4*
at age 35	22.9 $\pm$ 2.6	22.1 $\pm$ 2.4	23.3 $\pm$ 2.6*	24.3 $\pm$ 2.7*
at age 40	23.3 $\pm$ 2.8	22.6 $\pm$ 2.6	23.8 $\pm$ 2.7*	24.5 $\pm$ 3.0
Cereals 40 - age 35	0.4 $\pm$ 1.0	0.5 $\pm$ 1.0	0.4 $\pm$ 1.0	0.2 $\pm$ 1.1
smoking (cigarettes/day)				
	16.0 $\pm$ 12.7	15.7 $\pm$ 12.7	16.0 $\pm$ 12.7	16.5 $\pm$ 13.6

* $P<0.05$, compared with non-cycler by t-test

Table 2. Adjusted* mean of coronary risk factors at age 35 by weight cycling group

	N	Mean	P value [§]
SBP (mmHg)			
non-cycler	88	123.1	—
one cycler	90	122.9	0.925
frequent cycler	22	122.7	0.893
DBP (mmHg)			
non-cycler	88	75.8	—
one cycler	90	74.8	0.484
frequent cycler	22	78.1	0.307
total cholesterol (mg/dl)			
non-cycler	88	178.0	—
one cycler	90	177.1	0.858
frequent cycler	22	190.0	0.146
HDL cholesterol (mg/dl)			
non-cycler	88	47.6	—
one cycler	90	48.2	0.753
frequent cycler	22	47.0	0.839

*adjusted for BMI at age 35 and smoking status at age 40.

[§]compared with non-cycler

weight cycling and smoking status for increment in total cholesterol and HDL cholesterol, further analysis was done by group categorization of smoking status (Table 4). In the light

smoker group, mean increment in total cholesterol in one cyclers was significantly less than in non-cyclers. The change was not significantly different among the different cyclers in

Table 3. Adjusted* mean increment in coronary risk factors between age 35 and 40 by weight cycling group

	N	Mean	P value [§]
Δ SBP (mmHg)			
non-cycler	88	2.3	—
one cycler	90	3.3	0.576
frequent cycler	22	3.8	0.598
Δ DBP (mmHg)			
non-cycler	88	3.2	—
one cycler	90	3.3	0.869
frequent cycler	22	2.4	0.694
Δ total cholesterol (mg/dl)			
non-cycler	88	17.2	—
one cycler	90	13.7	0.266
frequent cycler	22	7.4	0.074
Δ HDL cholesterol (mg/dl)			
non-cycler	88	7.5	—
one cycler	90	5.5	0.264
frequent cycler	22	9.7	0.434

*For each risk factor, adjusted for BMI at age 35, difference in BMI between age 35 and age 40, measurement value of the risk factor at age 35, smoking status at age 40, and status of health guidance. Further for Δ HDL cholesterol, adjusted for interaction between smoking status and weight cycling category. Further for Δ total cholesterol, adjusted for interactions between smoking status and weight cycling category, between health guidance and difference of BMI, and between health guidance and weight cycling category

[§] compared with non-cycler

Figure 1. Effect of the number of weight cycles and smoking on Δ HDL cholesterol

other smoking categories. The mean increment in HDL cholesterol in the non-smoker group was significantly higher in the frequent cyclers than in non-cyclers. However, that in the heavy smoker group was less for the frequent cyclers than for non-cyclers, although the difference in change was not significant. In effect, the trend for heavy smokers was opposite to that for non-smokers (Table 4 and Figure 1).

Since interactions between status of health guidance and the difference of BMI, and, between health guidance and weight cycling were found in the model for increment in total cholesterol, additional analysis was done by status of health guidance (not shown). In the group without any health guidance

($n=177$), mean increment in total cholesterol was not significantly different among the different cyclers. In the group receiving at least one health guidance ($n=23$), the mean increment was lower in one cyclers as well as frequent cyclers compared with non-cyclers ($p=0.065$, 0.055 , respectively).

DISCUSSION

The purpose of this research was to demonstrate the effect of weight cycling on coronary risk factors by examining the relationship between weight cycling during ages 21 to 35 and change of coronary risk factors during ages 35 to 40.

Table 4. Adjusted* mean increment between age 35 and age 40 in total cholesterol and HDL cholesterol by weight cycling group classified by smoking status

	Non-smoker (N=61)		Light smoker (≤ 20 /day) (N=86)		Heavy smoker (≥ 21 /day) (N=53)	
	Mean	p value [§]	Mean	p value [§]	Mean	p value [§]
Δ total cholesterol (mg/dl)						
non-cycler (N=88)	22.8	—	21.0	—	16.8	—
one cyclist (N=90)	12.3	0.621	4.4	0.001	17.9	0.854
frequent cyclist (N=22)	-2.4	0.203	14.9	0.638	15.6	0.882
Δ HDL cholesterol (mg/dl)						
non-cycler (N=88)	3.0	—	9.4	—	9.6	—
one cyclist (N=90)	8.5	0.066	3.8	0.043	4.7	0.102
frequent cyclist (N=22)	14.3	0.009	13.2	0.506	3.5	0.193

*adjusted for BMI at age 35, the difference in BMI between age 35 and age 40, and measurement value of the risk factor at age 35, and status of health guidance. Further for Δ total cholesterol, adjusted for interactions between health guidance and difference of BMI, and between health guidance and weight cycling category

[§] compared with non-cycler

However, the present study did not show any positive relationship between weight cycling and four coronary risk factors except for HDL cholesterol based on analysis divided by smoking status. Jeffery et al.⁹ reported that, using maximum weight fluctuation (difference between maximum BMI and minimum BMI since adulthood) as the index of weight cycling, its relationship with coronary risk factors was negative. Lissner et al.,⁶ using the standard error of the regression estimate of BMI for a certain period as the index of weight cycling, examined its relationship with changes in coronary risk factors per year (in a multiple regression analysis), and reported that the index of weight cycling did not have any significant relationship with the risk factors. They did not include smoking status in their analysis model and their reports are in accord with our results based on the model which was not divided by smoking status.

Rebuffe-Scrive et al.,⁸ by categorizing subjects into two groups by frequency of weight cycling, examined the cross-sectional relationship between weight cycling and serum lipid level and reported that total cholesterol was not significantly different between cyclers and non-cyclers. They postulated that, because it was shown that intraabdominal fat stores start to increase only at a certain degree of obesity,¹⁸ their subjects with a mean BMI of 25.0 may have not reached the threshold necessary to start filling their intraabdominal fat deposits and, therefore, have accumulated fat in the subcutaneous abdominal region. The mean BMI of our subjects in each weight cycling category was below 25.0 so their inference may also apply to our case.

The results of the present study depend substantially on the definition of weight cycling and the motivation for weight loss. In preceding reports which examined the relationship between mortality by cardiovascular disease and weight cycling, coefficient of variation (CV) or standard error of the regression estimate of BMI for a certain period was often used as the index of weight cycling. However, problems in using these indices have been pointed out,^{19,20} e.g. individuals who continually gain weight over the study period have a high CV despite the fact that they have undergone no weight cycles in conventional terms. Thus, we used the number of weight cycles during a specified age period as the index. We defined one weight cycle as a cycle in which BMI consistently increased 5% or more from a point of any duration subsequent to an event with a decrease of 5% or more (this decreasing period must not include any increasing period). This criterion was determined referring to Hamm's report,⁵ which defined the no-weight-change group as 5% or less BMI change and showed a significant relationship between coronary death and weight cycling. However, the time span for one weight cycle was not defined in the present study (mean time span was 3.3 years and time span within 5 years was 93%). In order to exclude the influence of the long span cycler, the same analyses were repeated excluding subjects whose time span was 6 y or more. The results obtained were almost the same. Further studies con-

cerning the definition of weight cycling are necessary because different definitions may influence results in varying degrees.

In the present study, it was not investigated whether weight loss was intentional or not. Rather, the health guidance record was examined, revealing that only fifteen cyclers (13% of all cyclers) had received health guidance about their weight just before their weight cycle. This proportion was higher in frequent cyclers than in one cyclers. These fifteen individuals probably reduced weight intentionally. In order to eliminate the influence of intentional weight loss, the same analyses were repeated excluding these fifteen individuals. The results were found to be almost the same even though the number of frequent cyclers was decreased.

The age of the subject is an important determinant factor of chronic disease. Previous studies which showed significant association between mortality by cardiovascular disease and weight cycling were conducted on subjects older than those of the present study. Our subjects were so young that a longer follow-up may be necessary for our cohort. A further follow-up of the current cohort may clarify the relationship between weight cycling and its health effects. Some researchers have reported that BMI is related to education and social class²¹⁻²³. In this study, the subjects' education and social class were almost the same. We thus assume this influence to be very small.

It is necessary to regard smoking status as an important confounder because many reports have shown that smoking status influences weight²⁴⁻²⁶ and cholesterol^{27,28}. We found significant interaction between smoking status and weight cycling in the models of increment in HDL cholesterol. It was shown that the influence of weight cycling differed according to smoking status. This relationship has never been shown in the preceding studies. Some researchers have reported that the increase in serum cholesterol during 20~30 years of age is not caused by changes in dietary habits²⁹, while others have reported the influence of lifestyle such as drinking habits on total cholesterol or HDL cholesterol³⁰. The interaction between smoking status and weight cycling may be influenced by factors such as dietary and drinking habits, which were not accounted for this study.

Systolic and diastolic blood pressure were not significantly different between the cycler and non-cycler groups, coinciding with some reports^{6,8,9}. Thus we conclude that weight cycling does not affect blood pressure in young male adulthood, but further study is necessary to clarify the influence of factors, e.g., definition of cycling, age of subjects, and motivation of weight loss, which may distort the effects of weight cycling.

The following limitations need to be considered in this research. We selected subjects of almost the same social class and occupation so that the influence of these two factors on BMI could be minimized. However, this resulted in the limitation that the results of the present report cannot be directly applied to the general population. Second, we were able to use smoking information only at age 40 because there were no

smoking data for many subjects before this age. Prior change in smoking status may have influenced the results. There is a report that change in smoking status strongly affects change in weight²¹. Thus, smoking information at only one point in time may be insufficient when considering the influence of smoking. To estimate the influence of smoking, analyses were repeated using smoking data at age 37 (150 data were available), but the results did not change substantially. Third, only samples where fasting was not required of subjects were available for the measurement of serum lipid level in this study. This study used data obtained in the past when subjects for the present study did not realize whether they were cyclers or non-cyclers. Thus, it was unlikely that systematic differences in fasting time existed between cyclers or non-cyclers. Their fasting times were probably random in each group. Variance in level of coronary risk factors would be larger than those in data based on fixed fasting time, and could have masked the difference, if any, between two groups. Therefore, the present results may have underestimated the effect of weight cycling.

The present study did not support our hypothesis that weight cycle during ages 21 to 35 was related to change of coronary risk factors during ages 35 to 40. Interaction between weight cycling and smoking status was found in the model of increment in total cholesterol and HDL cholesterol. It was also shown that the effect of weight cycling differed according to smoking status. Although the mechanism for these observations is not clear at this stage, the results appear to indicate that weight cycling and other potential confounders such as lifestyle interact to influence coronary risk factors. As the present study was conducted on a small population, these interactions warrant further investigation on a larger population.

ACKNOWLEDGMENTS

The authors thank Prof. Masato Ikeda, Dr. Hideo Ookawa, and Dr. Tsutomu Housuyama for their helpful suggestions and all the occupational health nurses at the Keihin Health Service Center for their assistance in performing this study.

REFERENCES

1. Stunkard AJ, Penick SB. Behavior modification in the treatment of obesity. The problem of maintaining weight loss. *Archs Gen Psychiat* 1979 ; 36 : 801-806.
2. Sohar E, Sneh E. Follow-up of obese patients : 14 years after a successful diet. *Am J Clin Nutr* 1973 ; 26 : 845-848.
3. Lissner L, Odell PM, D'Agostino RB, Stokes JIII, Kreger BE, Belanger AJ, Brownell KD. Variability of body weight and health outcomes in the Framingham population. *N Engl J Med* 1991 ; 324 : 1839-1844.
4. Lissner L, Collins G, Blair S, Brownell K. Weight fluctuation in the Multiple Risk Factor Intervention Trial population. *Am J Epidemiol* 1989 ; 130 : 845.
5. Hamm P, Shekelle RB, Stamler J. Large fluctuations in body weight during young adulthood and twenty-five-year risk of coronary death in men. *Am J Epidemiol* 1989 ; 129 : 312-318.
6. Lissner L, Andres R, Muller DC, Shimokata H. Body weight variability in men : metabolic rate health and longevity. *Int J Obesity* 1990 ; 14 : 373-383.
7. Lissner L, Bengtsson C, Lapidus L, Larsson B, Bengtsson B, Brownell K. Body weight variability and mortality in the Gothenburg Prospective Studies of men and women. In : Björntorp P, Rossner S, eds. *Obesity in Europe 88 : Proceedings of the First European Congress on Obesity*. Libbey, London, 1989 : 55-60.
8. Rebuffé-Scrive M, Hendler R, Bracero N, Cummings N, McCarthy S, Rodin J. Biobehavioral effects of weight cycling. *Int J Obesity* 1994 ; 18 : 651-658.
9. Jeffery R, Wing R, French S. Weight cycling and cardiovascular risk factors in obese men and women. *Am J Clin Nutr* 1992 ; 55 : 641-644.
10. Holbrook TL, Barrett-Connor E, Wingard D. The association of lifetime weight and weight control patterns with diabetes among men and women in an adult community. *Int J Obesity* 1989 ; 13 : 723-729.
11. Steem SN, Oppliger RA, Brownell KD. Metabolic effects of repeated weight loss and regain in adolescent wrestlers. *JAMA* 1988 ; 260 : 47-50.
12. Wadden TA, Bartlett S, Letizia KA, Foster GD, Stunkard AJ, Conill A. Relationship of dietary history to resting metabolic rate, body composition, eating behavior, and subsequent weight loss. *Am J Clin Nutr* 1992 ; 56 : 203S-208S.
13. van Dale D, Saris W. Repetitive weight loss and weight regain : effects on weight reduction, resting metabolic rate, and lipolytic activity before and after exercise and/or diet treatment. *Am J Clin Nutr* 1989 ; 49 : 409-416.
14. van der Kooy K, Leenen R, Seidell JC, Deurenberg P, Hautvast GAJ. Effects of a weight cycle on visceral fat accumulation. *Am J Clin Nutr* 1994 ; 60 : 861-863.
15. Manore MM, Berry TE, Skinner JS, Carroll SS. Energy expenditure at rest and during exercise in nonobese female cyclical dieters and in nondieting control subjects. *Am J Clin Nutr* 1991 ; 54 : 41-46.
16. Melby CL, Schmidt WD, Corrigan D. Resting metabolic rate in weight cycling collegiate wrestler compared with physically active, noncycling control subjects. *Am J Clin Nutr* 1990 ; 52 : 409-414.
17. PROC GLM. In : SAS Institute Inc., *SAS/STAT User's Guide*, version 6, fourth edition, volume 2, SAS Institute Inc., Cary, NC, 1989 : 891-996.
18. Kvist M, Sjöström L, Tylén U. Adipose tissue volume determination in women by CT : technical considerations. *Int J Obesity* 1986 ; 10 : 53-67.
19. Wing RR. Weight cycling in humans : a review of the literature. *Ann Behav Med* 1992 ; 14 : 113-119.

20. French SA, Jeffery RW, Folsom AR, Williamson DF, Byers T. Weight variability in a population-based sample of older women : reliability and intercorrelation of measures. *Int J Obesity* 1995 ; 19 : 22-29.
21. Kahn HS, Williamson DF, Stevens JA. Race and weight change in US women : the roles of socioeconomic and marital status. *Am J Public Health* 1991 ; 81 : 319-323.
22. Leigh JP, Fries JF, Hubert HB. Gender and race difference in the correlation between body mass and education in the 1971-1975 NHANES I. *J Epidemiol Community Health* 1992 ; 82 : 821-826.
23. Burke GL, Savage PJ, Manolio TA, Sprafka JM, Wagenknecht LE, Sidney S, Perkins LL, Liu K, Jacob DR Jr.. Correlates of obesity in young black and white women. *Am J Public Health* 1992 ; 82 : 1621-1625.
24. Williamson DF, Madans J, Anda RF, Kleinman JC, Giovino GA, Byers T. Smoking cessation and severity of weight gain in national cohort. *N Engl J Med* 1991 ; 324 : 739-745.
25. Troisi RJ, Heinold JW, Vokonas PS, Weiss ST. Cigarette smoking, dietary intake, and physical activity : effects on body fat distribution-the Normative Aging Study. *Am J Clin Nutr* 1991 ; 53 : 1104-1111.
26. Taylor CB, Jatulis DE, Winkleby MA, Rockhill BJ, Kraemer HC. Effects of life-style on body mass index change. *Epidemiology* 1994 ; 5 : 599-603.
27. Berns MAM, de Vries JHM, Katan MB. Increase in body fatness as a major determinant of change in serum total cholesterol and high density lipoprotein cholesterol in young men over a 10year period. *Am J Epidemiol* 1989 ; 130 : 1109-1122.
28. Wannamethee G, Shaper AG. Blood lipids: the relationship with alcohol intake, smoking, and body weight. *J Epidemiol Community Health* 1992 ; 46 : 197-202.
29. Berns MAM, de Vries JHM, Katan MB. Determinants of the increase of serum cholesterol with age : a longitudinal study. *Int J Epidemiol* 1988 ; 17 : 789-796.
30. Hubert HB, Eaker ED, Garrison RJ, Castelli WP. Life-style correlates of risk factor change in young adults : an eight-year study of coronary heart disease risk factors in the Framingham offspring. *Am J Epidemiol* 1987 ; 125 : 812-830.