Effects of Weight Cycling on Coronary Risk Factors

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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.

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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 examinations (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 studies 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) 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 categorial 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±SD                         | Mean±SD                         | Mean±SD                         | Mean±SD                         |
| SBP (mmHg)                     |                                 |                                 |                                 |                                 |
| at age 35                      | 122.9 ± 13.0                    | 122.0 ± 12.6                    | 123.3 ± 13.1                    | 124.3 ± 14.2                    |
| at age 40                      | 125.7 ± 13.0                    | 124.1 ± 13.5                    | 126.7 ± 12.9                    | 127.8 ± 11.2                    |
| age 40 - age 35                | 2.8 ± 12.3                      | 2.1 ± 12.2                      | 3.3 ± 12.3                      | 3.5 ± 13.3                      |
| DBP (mmHg)                     |                                 |                                 |                                 |                                 |
| at age 35                      | 75.4 ± 9.9                      | 74.3 ± 9.4                      | 75.3 ± 10.4                     | 80.2 ± 9.3*                     |
| at age 40                      | 78.7 ± 8.9                      | 77.9 ± 8.9                      | 79.0 ± 9.2                      | 80.1 ± 7.8                      |
| age 40 - age 35                | 3.3 ± 9.1                       | 3.7 ± 9.2                       | 3.7 ± 8.7                       | -0.1 ± 10.3                     |
| total cholesterol (mg/dl)      |                                 |                                 |                                 |                                 |
| at age 35                      | 179.4 ± 35.2                    | 175.0 ± 34.0                    | 179.4 ± 33.8                    | 196.5 ± 41.9*                   |
| at age 40                      | 193.9 ± 32.6                    | 194.2 ± 34.5                    | 193.0 ± 31.4                    | 196.1 ± 31.0                    |
| age 40 - age 35                | 14.5 ± 23.2                     | 19.2 ± 22.6                     | 13.6 ± 20.9                     | -0.3 ± 28.2*                    |
| HDL cholesterol (mg/dl)        |                                 |                                 |                                 |                                 |
| at age 35                      | 47.5 ± 12.1                     | 47.5 ± 12.3                     | 47.8 ± 11.8                     | 46.2 ± 13.1                     |
| at age 40                      | 54.5 ± 14.4                     | 55.9 ± 16.1                     | 53.0 ± 13.1                     | 55.3 ± 11.6                     |
| age 40 - age 35                | 6.9 ± 11.8                      | 8.2 ± 13.1                      | 5.1 ± 10.6                      | 9.0 ± 10.4                      |
| BMI (kg/m²)                    |                                 |                                 |                                 |                                 |
| at age 21                      | 21.1 ± 2.0                      | 20.5 ± 1.6                      | 21.5 ± 2.1*                     | 22.3 ± 2.4*                     |
| at age 35                      | 22.9 ± 2.6                      | 22.1 ± 2.4                      | 23.3 ± 2.6*                     | 24.3 ± 2.7*                     |
| at age 40                      | 23.3 ± 2.8                      | 22.6 ± 2.6                      | 23.8 ± 2.7*                     | 24.5 ± 3.0                      |
| Cereals 40 - age 35            | 0.4 ± 1.0                       | 0.5 ± 1.0                       | 0.4 ± 1.0                       | 0.2 ± 1.1                       |
| smoking (cigarettes/day)       | 16.0 ± 12.7                     | 15.7 ± 12.7                     | 16.0 ± 12.7                     | 16.5 ± 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\(^{1}\) |
|---------------------|----|------|-----------------|
| **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.
\(^{1}\) 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\(^{1}\) |
|----------------------|----|------|-----------------|
| \(\Delta\)SBP (mmHg) |    |      |                 |
| non-cycler           | 88 | 2.3  |                 |
| one cycler           | 90 | 3.3  | 0.576           |
| frequent cycler      | 22 | 3.8  | 0.598           |
| \(\Delta\)DBP (mmHg) |    |      |                 |
| non-cycler           | 88 | 3.2  |                 |
| one cycler           | 90 | 3.3  | 0.869           |
| frequent cycler      | 22 | 2.4  | 0.694           |
| \(\Delta\)total cholesterol (mg/dl) |    |      |                 |
| non-cycler           | 88 | 17.2 |                 |
| one cycler           | 90 | 13.7 | 0.266           |
| frequent cycler      | 22 | 7.4  | 0.074           |
| \(\Delta\)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 \(\Delta\)HDL cholesterol, adjusted for interaction between smoking status and weight cycling category. Further for \(\Delta\)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.
\(^{1}\) compared with non-cycler
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.
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.\(^8\) 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.\(^9\) 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,\(^9\) 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\(^9,20\) 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,\(^3\) 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 concerning 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 class\(^21,22\). 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 weight\(^24,26\) 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 habits\(^29\), while others have reported the influence of lifestyle such as drinking habits on total cholesterol or HDL cholesterol\(^30\). 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\(^5,8,3\). 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 weight\(^2\). 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.

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