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Original article

Three-month exercise and weight loss program improves heart rate recovery in obese persons along with cardiopulmonary function

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Summary
Objective: Heart rate recovery (HRR) after exercise is an independent risk factor for cardiovascular disease and mortality, and it is well known to be modifiable by weight loss. We investigated whether HRR was mainly improved by better cardiopulmonary function or by alteration of the metabolic profile.
Methods: The weight loss program included 2 h of group exercise per week and individual dietary instruction by a qualified dietician every week. Clinical assessment (including HRR) was done before and after the 3-month program.
Patients: The subjects were 125 obese persons without a past history of stroke, cardiovascular events, or use of medications who participated in and completed our exercise plus weight loss program.
Results: HRR (35.61 ± 12.83 to 45.34 ± 13.6 beats/min, $p < 0.0001$) was significantly faster after the program. The change in HRR was significantly correlated ($p < 0.05$) with the changes in body weight, body mass index, percent body fat, waist circumference, hip circumference, resting heart rate, peak exercise heart rate, exercise time, maximal work load, physical working capacity divided by body weight (PWC75%HRmax/weight), subcutaneous fat area, visceral fat

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area, low-density lipoprotein cholesterol, and leptin. Multivariate analysis showed that the change in HRR was significantly correlated ($p < 0.05$) with the changes in resting heart rate, peak exercise heart rate, and PWC75%HRmax/weight.

Conclusions: Our data demonstrated that HRR can be improved in obese subjects by a 3-month exercise and weight loss program. Improvement in cardiopulmonary function by exercise seems to be the main contributor to the increment of HRR.

Introduction

Obesity and metabolic syndrome are mainly caused by the lack of regular exercise and excessive intake of calories on a daily basis. Obesity has been proven to be an important risk factor for cardiovascular disease and for all-cause mortality [1—8].

Many studies have demonstrated the efficacy of improved physical fitness and weight loss for preventing cardiovascular disease, as well as the cost-effectiveness of exercise programs [9—15]. The extent of heart rate recovery (HRR) after exercise has been shown to be an independent risk factor for cardiovascular disease and mortality in healthy adults [16—20].

Furthermore, an association has been found between HRR and the metabolic syndrome as well as between HRR and each of the components of this syndrome [21,22]. It is well known that HRR after exercise can be modified by weight loss, but the mechanism underlying the improvement of HRR is unclear. We investigated whether HRR was mainly influenced by improvement of cardiopulmonary function or the metabolic profile.

Subjects

The subjects were 125 obese persons (including 27 who fulfilled the criteria for metabolic syndrome [23]) who were not on medications, had no past history of stroke or cardiovascular events, and participated in our weight loss program (5 of the 130 initial participants dropped out because of orthopedic problems). Inclusion criteria were a body mass index (BMI) above 25 kg/m^2, and/or a percent body fat (impedance method) greater than 25% for men and 30% for women. The full quota of the program was 30 individuals, and this program was held twice per year.

There were 14 men and 111 women aged 22—71 years (mean age: 53.1 ± 11.4 years). Written informed consent for this study was obtained from all subjects. Approval of the study was also obtained from the Research Ethics Committee of Yokohama City Sports Medical Center.

Methods

Weight loss program

All subjects were instructed by a qualified dietician to reduce the daily intake to 25 kcal/kg of their ideal body weight [$height^2 \times 22$ (kg/m^2)] and were followed every week. Before and during the program, the daily calorie intake was calculated from a questionnaire based on the Dietary Reference Intake for Japanese (2005) produced by the Japanese Ministry of Health, Labor, and Welfare. The weekly exercise program was 60 min of group aerobics and 4 strength-training exercises (abdominal crunch, prone arm leg extension, push ups, and squats), with 3 sets of 30 repetitions of each exercise being done under a trainer's supervision. All subjects were also advised to perform 30 min of walking at least 5 times a week.

Medical examination

Medical examinations were done before and after the weight loss program. Body weight (weight) and the percentage of body fat (Fat%) were examined by using a body composition analyzer (MC-190, Tanita Co., Tokyo, Japan). The BMI was calculated as body weight (kg)/height $(m)^2$. Waist circumference was measured by using a plastic belt-type measuring tape at the navel while the subject was standing and expiring.

The subcutaneous fat area and visceral fat area were measured manually by tracing (Nature View imaging system, Hitachi Medico Co., Tokyo, Japan) on magnetic resonance images (Intera 1.5 T, Koninklijke, Philips Electronics N.V., Eindhoven, the Netherlands).

Fasting blood levels of high-density lipoprotein cholesterol (HDL-chol), low-density lipoprotein cholesterol (LDL-chol), triglycerides (TG), glucose, insulin, and hemoglobin A_{1c} (HbA_{1c}) were measured using a Roche INTEGRA 400 plus (Roche International Ltd., Basel, Switzerland), while leptin was measured by RIA (Mitsubishi Chemical Medience Co., Tokyo, Japan). Homeostasis model assessment of insulin resistance (HOMA-R) was performed by using Matthews's equation (insulin $\times$ glucose/405) [24].

Symptom-limited maximal graded exercise testing was done on an electronic bicycle ergometer (The Multi Exercise Test System, ML-1800, Fukuda-Denshi, Tokyo, Japan) using a ramp protocol [25]. Following peak exercise, the subjects cooled down for 1.5 min at 60 rpm (1 min with a 20 W load and 30 s without a load).

The electrocardiogram was recorded and the systolic/diastolic blood pressures (SBP/DBP) were measured automatically every minute before, during, and after exercise test. HRR was defined as the difference between the heart rate at peak exercise and that at 2 min after the finish of exercise [20,26]. The physical working capacity at 75% of maximum HR was divided by body weight (PWC75%HR max/weight) to assess aerobic capacity [27]. To calculate 75% of maximal HR, the age-related maximal HR was determined as (220-age) beats per minutes.

All parameters were compared between before and after the weight loss program by using the paired *t*-test. Correlations of HRR with each parameter were examined by

Table 1 Physical and biochemical characteristics of the subjects before and after the exercise and weight loss program ($n = 125$).

	Before	After
Weight (kg)	70.9 ± 11.4	66.0 ± 10.9*
Body mass index (kg/m^2)	28.3 ± 3.5	26.3 ± 3.4*
Fat%	38.0 ± 6.2	34.0 ± 6.6*
Waist circumference (cm)	99.2 ± 9.0	93.2 ± 9.3*
Hip circumference (cm)	101.0 ± 6.5	97.5 ± 6.0*
Systolic blood pressure (mmHg)	133.5 ± 20.5	124.0 ± 19.2*
Diastolic blood pressure (mmHg)	82.5 ± 13.0	75.8 ± 12.6*
Resting heart rate (min^{-1})	85.6 ± 14.7	76.3 ± 13.1*
Peak exercise heart rate (min^{-1})	149.0 ± 16.9	150.0 ± 16.0
Exercise time (min)	7.2 ± 1.8	7.9 ± 1.7*
Maximal work load (W)	127.7 ± 34.6	139.5 ± 36.1*
PWC75%HRmax/weight (W/kg)	1.2 ± 0.4	1.5 ± 0.4*
Heart rate recovery at 2 min	37.4 ± 12.8	46.3 ± 13.5*
Subcutaneous fat area (cm^2)	310.6 ± 85.3	255.7 ± 83.4*
Visceral fat area (cm^2)	123.7 ± 51.9	94.5 ± 46.4*
High-density lipoprotein cholesterol (mg/dl)	53.4 ± 11.5	52.9 ± 10.8
Low-density lipoprotein cholesterol (mg/dl)	129.2 ± 27.7	114.1 ± 24.6*
Triglycerides (mg/dl)	130.4 ± 82.2	93.3 ± 55.9*
Fasting glucose (mg/dl)	104.9 ± 15.1	98.0 ± 9.4*
Fasting insulin (μU/ml)	10.8 ± 7.8	7.4 ± 5.7*
Hemoglobin A1c (%)	5.4 ± 0.5	5.2 ± 0.3*
Leptin (ng/ml)	14.4 ± 6.9	7.3 ± 4.3*
Insulin resistance (HOMA-R)	2.9 ± 2.2	1.8 ± 1.5*

PWC75%HRmax/weight, physical working capacity divided by body weight.
* $p < 0.05$.

Table 2 Predictors of the change in heart rate recovery after completing the program ($n = 125$).

	β	p
Change in weight (kg)	0.421	0.5798
Change in body mass index (kg/m^2)	−2.405	0.2368
Change in Fat%	−0.138	0.7241
Change in waist circumference (cm)	0.260	0.2809
Change in hip circumference (cm)	0.057	0.8886
Change in resting heart rate (min^{-1})	−0.181	0.0368*
Change in peak exercise heart rate (min^{-1})	0.342	0.0011*
Change in exercise time (min)	−0.607	0.7977
Change in maximal work load (W)	−0.018	0.9088
Change in PWC75%HRmax/weight (W/kg)	14.200	0.0002*
Change in subcutaneous fat area (cm^2)	−0.018	0.5299
Change in visceral fat area (cm^2)	0.014	0.7059
Change in low-density lipoprotein cholesterol (mg/dl)	−0.023	0.4951
Change in leptin (ng/ml)	0.053	0.7338

PWC75%HRmax/weight, physical working capacity divided by body weight.
* $p < 0.05$.

univariate and multivariate regression analysis. A probability value <0.05 was considered significant. Statistical analysis was done with a Hitachi FRORA 310W computer (Hitachi Electronics Co., Tokyo, Japan), using the Stat View 5.0 program (SAS Institute, Inc., Cary, NC, USA).

Results

The extent of HRR (35.61 ± 12.83 to 45.34 ± 13.6 beats/min, $p < 0.0001$) showed a significant increase after the program. Daily calorie intake was decreased significantly by the dietary program (from 1723.9 ± 380.6 to 1393.7 ± 262.4 kcal/day, $p < 0.0001$). The body weight, BMI, Fat%, waist circumference, hip circumference, SBP, DBP, resting heart rate, subcutaneous fat area, visceral fat area, LDL-chol, TG, fasting glucose, fasting insulin, HbA_{1c}, leptin, and HOMA-R were all significantly decreased after the program ($p < 0.0001$) (Table 1). The exercise time, maximal work load, and PWC75%HRmax/weight were significantly increased after the program ($p < 0.0001$) (Table 1). The change in HRR was significantly correlated ($p < 0.05$) with

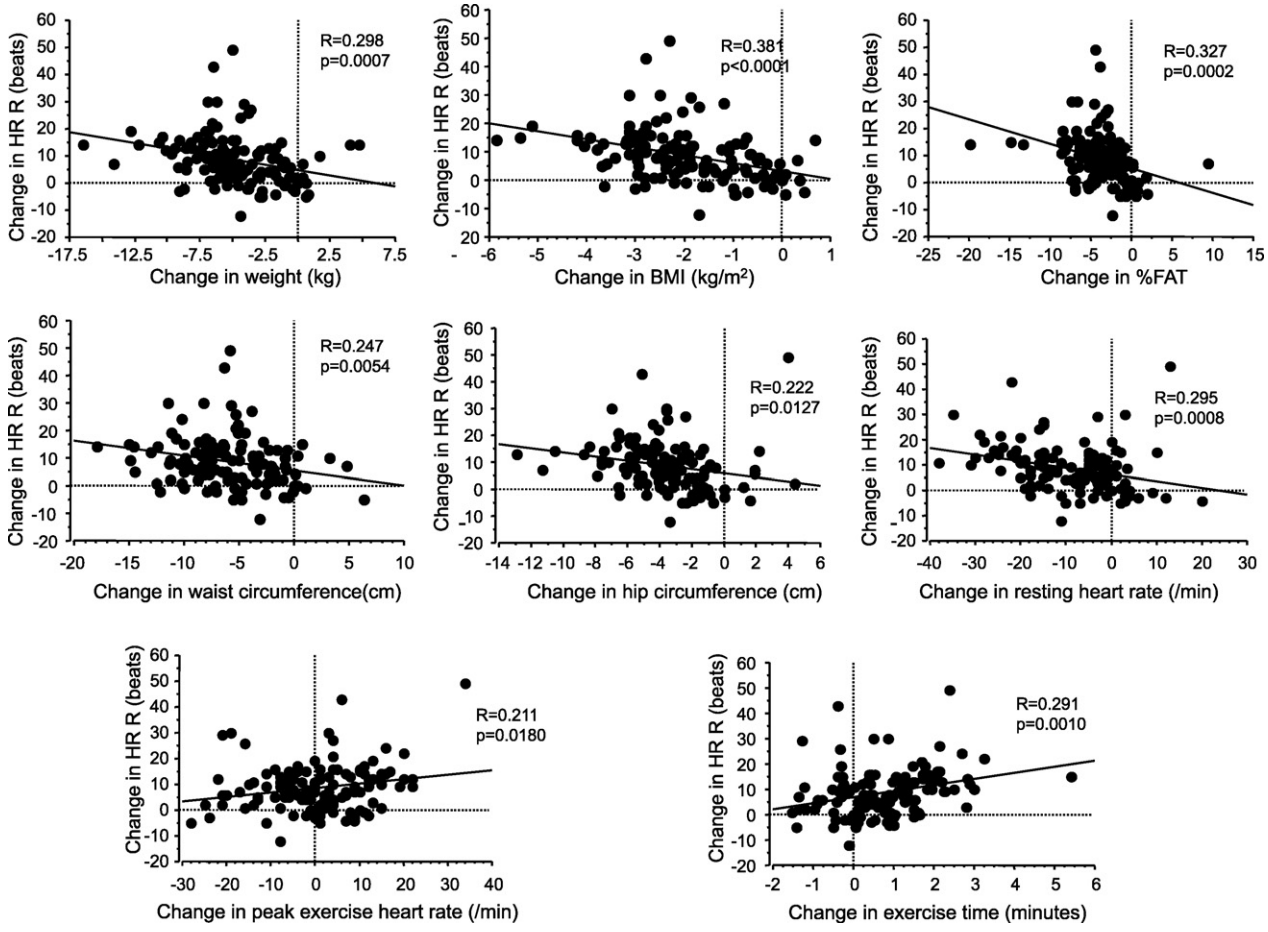

Figure 1 Correlations between the change in heart rate recovery and the changes in weight, body mass index (BMI), %Fat, waist circumference, hip circumference, resting heart rate, peak exercise heart rate, and exercise time.

the changes in weight, BMI, Fat%, waist circumference, resting heart rate, exercise time, maximal work load, PWC75%HRmax/weight, subcutaneous fat area, visceral fat area, LDL-chol, and leptin (Figs. 1 and 2). According to multivariate analysis, however, the change in HRR was only correlated with the changes in resting heart rate, peak exercise heart rate, and PWC75%HRmax/weight (Table 2).

Discussion

The important finding of this study was that the strong predictors of an improvement in HRR were all markers of cardiopulmonary function (resting heart rate, peak exercise heart rate, and PWC75%HRmax/weight). On the other hand, a number of metabolic parameters (weight, BMI, Fat%, waist circumference, hip circumference, visceral fat area, LDL-chol, and leptin) were found to be weak predictors that only showed a significant association in univariate analysis. Other metabolic parameters (TG, fasting glucose, fasting insulin, HbA_{1c}, and HOMA-R) were also significantly improved by the program, but were not predictors of the change in HRR. Recently, Brinkworth et al. reported an improvement in HRR after a weight loss program that only involved dieting without any change in physical activity [28]. They found a good correlation of the change in HRR with the reduction of metabolic parameters (weight, BMI, waist circumference, TG, glucose, and the TG/HDL-chol ratio). Furthermore, they found that the change in fasting blood glucose had the strongest independent correlation with the improvement in HRR. Accordingly, they concluded that their results reflected a change of the sympathetic/parasympathetic balance that occurred along with alterations of glucose metabolism associated with weight loss. Furthermore, Kim et al. reported improvement in HRR after a weight loss program that only involved exercise without any change in calorie intake [29]. They investigated 20 middle-aged obese men with metabolic syndrome and 20 men without this syndrome. They showed improvement in HRR after 12 weeks of exercise training in both groups of men. Their hypothesis was the same as ours, i.e. the change in HRR appears to be related to a change in resting HR, regardless of weight loss and improvement in cardiovascular fitness. The differences in the predictors of the improvement of HRR between our study and that of Brinkworth et al. may be related to the different types of weight loss program (Brinkworth et al. employed diet alone and our program included diet plus physical exercise). Differences in the profile of the subjects may also be involved, since Brinkworth et al. studied men with metabolic syndrome and we mainly studied women (including subjects without metabolic syndrome). Furthermore, the mean BMI (28.3) of our study subjects would be referred to as

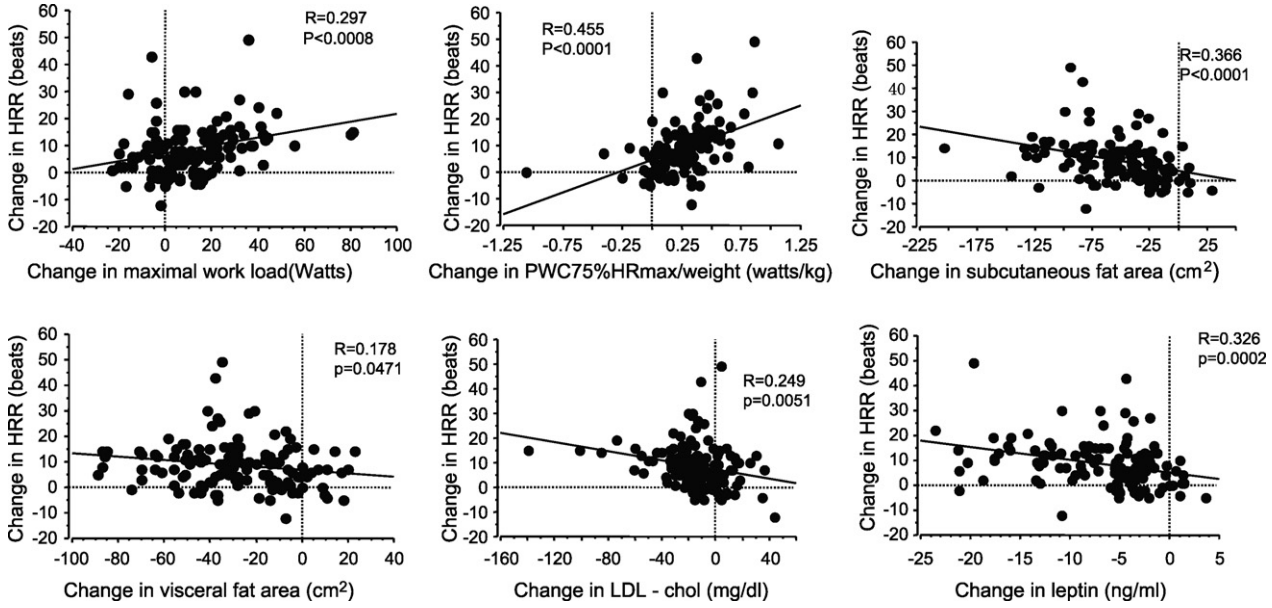

Figure 2 Correlations between the change in heart rate recovery and the changes in maximal work load, physical working capacity divided by body weight (PWC75%HRmax/weight), subcutaneous fat area, visceral fat area, low-density lipoprotein (LDL)-cholesterol, and leptin.

''overweight'' rather than ''obese'' in the USA or Europe. Such differences in the subjects may have influenced the results. It is widely accepted that the improvement in HRR caused by improved cardiopulmonary fitness is due to increased vagal tone along with an increase of peak VO_2 [30]. Furthermore, previous studies have shown that physical training not only improves HRR in ordinary people but also in athletes [31]. On the other hand, several studies have demonstrated that weight loss increases vagal tone in obese people [32,33]. Therefore, the change in HRR in our subjects may have been related to increased vagal tone that was mainly due to improvement of cardiopulmonary function by exercise rather than improvement of metabolic factors including weight loss. However, we definitely do not deny the usefulness of improving the metabolic profile to achieve better HRR.

Limitations

The main limitations of this study are the small number of subjects and the lack of evaluation of other adipose tissue-related factors (including adiponectin, resistin, highly sensitive C-reactive protein [34], etc.). A further limitation was that we used PWC75%HRmax/weight as the marker of cardiopulmonary function and a substitute for peak VO_2 because of the limited time available for testing. In the future, investigation of other parameters and longitudinal clinical outcome observation should be performed. Additionally, this study did not have a control group, because the program investigated was a routine program at our sports medical center. In the strict sense, it is therefore not possible to decide whether the observed changes were attributable to the program or were natural changes.

Conclusions

Our data demonstrate that HRR can be improved in obese subjects by a 3-month exercise and weight loss program. Improvement in cardiopulmonary function by exercise seems to be the main contributor to improvement in HRR.

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