# Exercise training and autonomic nervous system activity in obese individuals

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**ABSTRACT**

AMANO, M., T. KANDA, H. UE, and T. MORITANI. Exercise training and autonomic nervous system activity in obese individuals. *Med. Sci. Sports Exerc.*, Vol. 33, No. 8, 2001, pp. 1287–1291. **Purpose:** This study was designed to investigate the effects of 12 wk of exercise training on autonomic nervous system (ANS) in 18 obese middle-aged men ($N = 9$) and women ($N = 9$) (age: 41.6 ± 1.2 yr; BMI: 27.3 ± 0.4 $kg \cdot m^{-2}$; %fat: 29.6 ± 1.3%, mean ± SE). **Methods:** Each subject participated in an aerobic exercise training at anaerobic threshold (AT), consisting of 30 min/session, 3 times/wk, for 12 consecutive weeks. The ANS activities were assessed by means of power spectral analysis of heart rate variability (HRV) at resting condition before, at 5 wk, and after the exercise program. **Results:** The exercise training resulted in a significant decrease in body mass, BMI, and % fat ($P < 0.01$) but not in lean body mass ($P > 0.05$) together with a significant increase in the AT $\dot{V}O_2$ ($P < 0.01$). Our power spectral data indicated that there were significant increases in the low-frequency component associated with the sympathovagal activity (0.03–0.15 Hz, 348.5 ± 66.8 vs 694.7 ± 91.5 $ms^2$, $P < 0.01$), the high-frequency vagal component (0.15-0.4 Hz, 146.3 ± 30.4 vs 347.7 ± 96.5 $ms^2$, $P < 0.05$), and the overall autonomic activity as evaluated by total power (0.03-0.4 Hz, 494.8 ± 88.5 vs 1042.4 ± 180.9 $ms^2$, $P < 0.01$) of HRV after the training. **Conclusions:** Twelve weeks of exercise training has significantly improved both the sympathetic and parasympathetic nervous activities of the obese individuals with markedly reduced ANS activity, suggesting a possible reversal effect of human ANS functions. These favorable changes may also have an influence on the thermoregulatory control over the obesity. **Key Words:** OBESITY, AEROBIC EXERCISE TRAINING, AUTONOMIC NERVOUS SYSTEM ACTIVITY, HEART RATE VARIABILITY

Obesity is highly prevalent in developed countries and is associated with an increased risk for the development of hypertension, cardiovascular disease (CVD), diabetes mellitus, and other chronic diseases. Obesity is a significant independent predictor of cardiovascular risk (10), and CVD is the leading cause of morbidity and death in Japan, according to the research in the Japan Ministry of Health and Welfare (11).

Power spectral of heat rate variability (HRV) has been proved as a simple, useful noninvasive method for analyzing the autonomic mechanisms through the measurement of instantaneous beat-to-beat variations in R-R interval length (1,13,18). In general, the high-frequency component of HRV (≥0.15 Hz) is associated solely with the parasympathetic nervous system (PNS) activity, and the low frequency of HRV (<0.15 Hz) is associated with both the sympathetic nervous system (SNS) activity and PNS activity (1,21). The alterations of HRV are observed in the patients with coronary heart disease (CHD) (8), which is one of the most prevalent forms of CVD, and lower HRV has been identified as a risk factor for cardiac sudden death (CSD) (16) and all-cause mortality (12,27). Lower HRV is thought to reflect the cardiac autonomic outflow in which the modulation of the heart rate by cardiac parasympathetic activity is decreased (24) and susceptibility of the myocardium to arrhythmia generation is increased (28).

It is well known that HRV declines (5,7,9) and both the SNS and PNS activities are reduced with the age (5,7,9,20). Also, the reduction of both functions is accompanied by a change of the vagosympathetic balance with advancing age according to our recent paper (20).

Moreover, several previous cross-sectional studies have indicated that HRV at resting is greater (5,6,7) and ANS activity, especially PNS activity, is higher (5,7) in physically active populations compared with less active populations. However, there has been little agreement as to whether the intervention of exercise training would evoke changes in ANS activity (2,4,25).

Furthermore, the ANS plays an important role in metabolism, and obesity is associated with a relative or absolute reduction in the activity of the thermogenic component of the SNS (3). We have recently demonstrated that obese women manifested significantly lower SNS activity against various physiological perturbations, such as cold exposure (13), food intake (14), and capsaicin intake (15).

All of the above-mentioned work strongly suggests that obesity might be linked with not only higher risk for CHD but also reduced ANS activity, together with depressed SNS activity associated with thermogenesis. However, there was no study, at least to our knowledge, investigating the possibility of whether or not exercise training can modify the ANS activities of obese middle-aged individuals. Therefore, this study was designed to investigate the effects of 12 wk of exercise training on the ANS in obese middle-aged

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TABLE 1. Physical characteristics of subjects ($N = 18$).

| | Before | After |
|---|---|---|
| Age (yr) | 41.6 ± 1.2 | |
| Height (cm) | 164.2 ± 2.5 | |
| Weight (kg) | 74.1 ± 2.6 | 70.3 ± 2.9** |
| Body mass index (kg · m$^{-2}$) | 27.3 ± 0.4 | 25.9 ± 0.5** |
| % fat | 29.6 ± 1.3 | 26.6 ± 1.3** |
| Fat mass (kg) | 21.7 ± 0.9 | 18.6 ± 1.0** |
| Lean body mass (kg) | 52.4 ± 2.5 | 51.7 ± 2.6 N.S. |

Value are mean ± SE.
** $P < 0.01$; paired *t*-test for comparison before and after the 12-wk exercise training program.

individuals. Our data will shed some light upon the unique role of exercise training in human ANS functional trainability and/or reversibility in these obese middle-aged individuals and may provide important information in the preventive role of exercise in enhancing reduced SNS activity associated with thermogenic control of obesity individuals in general.

## METHODS

**Subjects.** Eighteen obese individuals volunteered to participate in this study. Subject selection criteria were: sedentary, over 25 for body mass index (BMI) on the basis of the guidelines defined by the Japan Society for the Study of Obesity, nonsmoker, the absence of hypertension, cardiovascular disease, or diabetes mellitus. Percentage of body fat was directly determined by means of hydrostatic weighing technique (A&D AD-4323B, Fukuda Denshi ELLIPSE 1000, Tokyo, Japan). Characteristics of the subjects are presented in Table 1. All the subjects gave their written informed consent before training experiment.

**Exercise training program.** Each subject performed an aerobic exercise training cycling on an electronically controlled cycle ergometer (Combi 232CXL, Tokyo, Japan) consisting of 30 min/session (5-min warming up, 20-min anaerobic threshold (AT) work, and 5-min cooling down), 3 times/wk for 12 consecutive weeks. We used gas exchange parameters (SensorMedics $V_{max}$ 29, Yorba Linda, CA) to determine AT, and our methods and procedures for determining gas exchange parameters on online have been fully described in our previous communication (17). The determination of AT was made during the progressive exercise test using respiratory gas exchange parameters, i.e., the nonlinear increase in min volume ($\dot{V}_E$) and $CO_2$ production ($\dot{V}CO_2$), abrupt increase in the fraction of $O_2$ in expired air and respiratory exchange ratio (R) and systematic increase in $\dot{V}_E$/oxygen uptake ($\dot{V}O_2$) without any increase in $\dot{V}_E/\dot{V}CO_2$ (29). The subject adjusted the cycling load keeping the heart rate values between 105 and 130 bpm according to each individual AT intensity by monitoring heart rate using an ECG monitor (Nihon Koden WEB-5000, Tokyo, Japan). Subjects performed the progressive exercise stress test every week so as to adjust the training heart rate. They also received three sessions of educational consultation for their nutrition (one group session for 60 min and two individual sessions for 15 min each) at the beginning of 12-wk exercise program.

**Electrocardiogram (ECG).** For assessment of ANS activity, ECG was taken while the subject was in the supine position on a bed in a quiet room. The subject was instructed to relax and not to move or sleep during measurements. The subject was asked to avoid strenuous activity on the day before testing. After the subject rested at least 15–20 min, a $CM_5$ lead ECG was continuously recorded on a computer for 5 min. During the testing, the subject breathed in synchrony with a metronome at 15 times·min$^{-1}$ (0.25 Hz) to ensure that respiratory-linked variations in heart rate did not overlap with low-frequency heart rate fluctuations (below 0.15 Hz) from other sources. ECG measurements were conducted before the exercise program started, during the 5th week, and after the 12-wk exercise program.

**R-R interval power spectral analysis.** We used a power spectral analysis of HRV to measure the ANS activity at the resting condition. Our R-R interval power spectral analysis procedures have been fully described elsewhere (13–15,18,19). An analog output of the ECG monitor (Life Scope, Nihon Koden) was digitized via a 13-bit analog-to-digital converter (Trans Era HTB 410, UT) at a sampling rate of 1 kHz. The digitized ECG signal was differentiated, and the resultant QRS spikes and the intervals of the impulses (R-R intervals) were stored sequentially on a hard disk for later analysis.

The off-line analysis conducted with the assist of a personal computer. The stored R-R interval data were converted into equal spaced samples with an effective sampling frequency of 2 Hz (23). Then the DC component and trend were completely eliminated by digital filtering for the bandpass between 0.03 and 0.5 Hz as described elsewhere (13–15,18,19). The root mean square value (RMS) of R-R interval was calculated as representing the average amplitude. After passing through the Hamming-type data window, the power spectral analysis by means of fast Fourier transform was then performed on consecutive 240-s time series of R-R interval data obtained during the test. To evaluate the ANS activity in each subject of present study, we analyzed low (LO) frequency (0.03–0.15 Hz), high (HI) frequency (0.15–0.4 Hz), and total power (TOTAL) (0.03–0.4 Hz) by integrating the spectrum for the respective bandwidth.

**Statistics.** All statistical analyses were performed using a commercial software package (SPSS version 10.0.5 J for Windows, SPSS Inc., Chicago, IL). A paired *t*-test was performed to compare before and after the 12-wk exercise program for comparing pre- and post-values in body composition and cardiorespiratory parameters. A one-way analysis of variance (ANOVA) by a least significant difference test was used for comparisons of our time course changes in HRV power spectral parameters. $P$ values $< 0.05$ were considered to be statistically significant. Data are expressed as mean ± SE.

## RESULTS

The changes in body composition after the exercise training are shown in Table 1. The exercise training resulted in a significant ($P < 0.01$) reduction in body mass (74.1 ± 2.6

TABLE 2. The changes in aerobic capacity ($N = 18$).

| | Before | After |
|---|---|---|
| $\dot{V}O_2$ AT ($mL^{-1} \cdot min^{-1}$) | 1137.2 ± 55.6 | 1239.3 ± 61.2** |
| $\dot{V}O_2$ AT/body mass ($mL \cdot kg^{-1} \cdot min^{-1}$) | 15.5 ± 0.6 | 17.7 ± 0.7** |
| $\dot{V}O_2$ AT/lean body mass ($mL \cdot kg^{-1} \cdot min^{-1}$) | 21.8 ± 0.9 | 24.2 ± 0.9** |

Value are mean ± SE.
** $P < 0.01$; paired *t*-test for comparison before and after the 12-wk exercise training program.

vs 70.3 ± 2.9 kg), BMI (27.3 ± 0.4 vs 25.9 ± 0.5 $kg \cdot m^{-2}$) and percentage of body fat (29.6 ± 1.3 vs 26.6 ± 1.3%), respectively. These significant changes were accompanied with nearly identical lean body mass (52.4 ± 2.5 vs 51.7 ± 2.6 kg, $P > 0.05$).

The changes in aerobic capacity at AT with training are presented in Table 2. $\dot{V}O_2$ AT (1137.2 ± 55.6 vs 1239.3 ± 61.2 $mL \cdot min^{-1}$), $\dot{V}O_2$ AT per body mass (15.5 ± 0.6 vs 17.7 ± 0.7 $mL \cdot kg^{-1} \cdot min^{-1}$), and $\dot{V}O_2$ AT per lean body mass (21.8 ± 0.9 vs 23.9 ± 0.9 $mL \cdot kg^{-1} \cdot min^{-1}$) were significantly increased after the exercise training ($P < 0.01$), respectively.

We examined the differences of all frequency components of HRV between male and female subjects before exercise training. There were no significant differences between them (male vs female; TOTAL: 618.7 ± 133.5 vs 370.9 ± 107.8; LO: 452.6 ± 110.8 vs 244.3 ± 71.5; HI: 166.1 ± 36.3 vs 126.6 ± 50.2; $P > 0.05$). In the present study, our major objective was to evaluate the possible trainability and/or reversibility of the ANS activity of the obese individual with less emphasis of the gender difference. We therefore combined male and female data to obtain stronger statistical inference.

Figure 1 shows typical sets of raw R-R interval and the corresponding power spectral data obtained from a subject at rest before, during the 5th week, and after the completion of exercise training program. It can be readily seen that R-R variability and frequency components in the power spectra had increased dramatically as training progressed. This figure clearly shows that both LO and HI frequency components representing sympathovagal and respiration-linked parasympathetic activities, respectively, increased during the time course of 12-wk exercise training program.

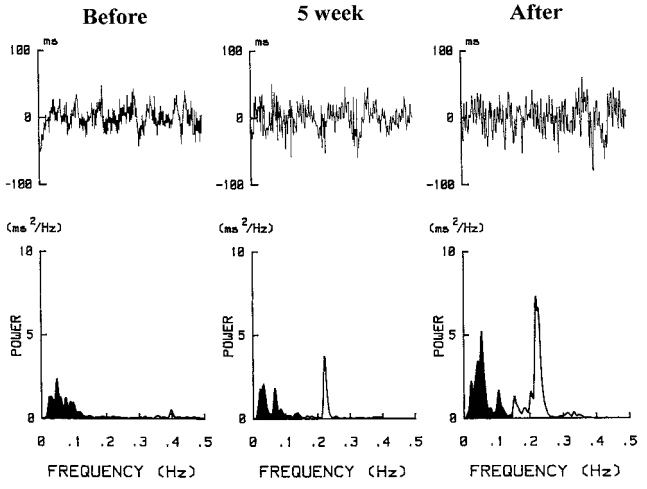


**FIGURE 1—Example of ECG R-R interval changes and the corresponding power spectra for a subject before, at 5 wk, and after the exercise program.**

Figure 2 represents group data with respect to the time course changes in HR and the R-R spectral parameters (LO, HI, and TOTAL) during the training period. As shown in Figure 2A, HR significantly decreased during the 5th week of the exercise program (70.5 ± 2.4 vs 64.4 ± 1.9 bpm, $P < 0.05$) and after completion of the exercise program (70.5 ± 2.4 vs 63.2 ± 2.3 bpm, $P < 0.01$). The significant increases were observed in the LO (348.5 ± 68.8 vs 694.7 ± 91.5 $ms^2$, $P < 0.01$) and HI (146.3 ± 30.4 vs 347.7 ± 96.5 $ms^2$, $P < 0.05$) frequency components, as well as in the TOTAL power (494.8 ± 88.5 vs 1042.4 ± 180.9 $ms^2$, $P < 0.01$) of HRV after 12 wk of exercise program (Figure 2B, C, D).

## DISCUSSION

Heart rate variability decreases (5,7,9) and both sympathetic and parasympathetic nervous activities decline with age (5,7,9,20). When comparing the aging process with the pharmacological blockade experimental data, we reported earlier that the aging process followed the changes of ANS activities, closely resembling those induced by propranolol and atropine infusion (20). These results indicate that not only parasympathetic but also sympathetic nervous system activities are withdrawn and that vagosympathetic balance significantly alters with the age.

Earlier cross-sectional studies indicated that HRV power at rest was greater in physically active populations compared with less active populations (5,6,8). The previous exercise intervention studies have also demonstrated that HRV was increased after exercise training in healthy middle-aged and older men (25). Some interventional studies, however, have indicated that exercise training did not improve ANS activity at rest (2,4). These differences in data might have resulted from different intensity, duration, frequency, and length of the exercise training, different populations used in the studies, and the sensitivity of the HRV method or the R-R interval power spectral analysis method.

Our data clearly indicated that the reduced ANS in middle-aged obese individuals was regained by AT level exercise training with a significant reduction in fat mass without losing lean body mass. Our AT-level exercise program with the educational consultation for their diet successfully reduced body fat and supported the previous study (30) that exercise training has an important role in the reducing body fatness in addition to the improvement of aerobic capacity.

On the other hand, ANS plays an important role in metabolism, and according to previous studies, the alteration or reduction of sympathetic nervous activity *per se* has been widely believed to contribute to the pathogenesis of obesity (3,22). We have recently examined the effects of capsaicin on the SNS activity and diet-induced thermogenesis in lean and obese young women (15). Capsaicin increases energy metabolism by catecholamine secretion from the adrenal medulla through sympathetic activation via the central

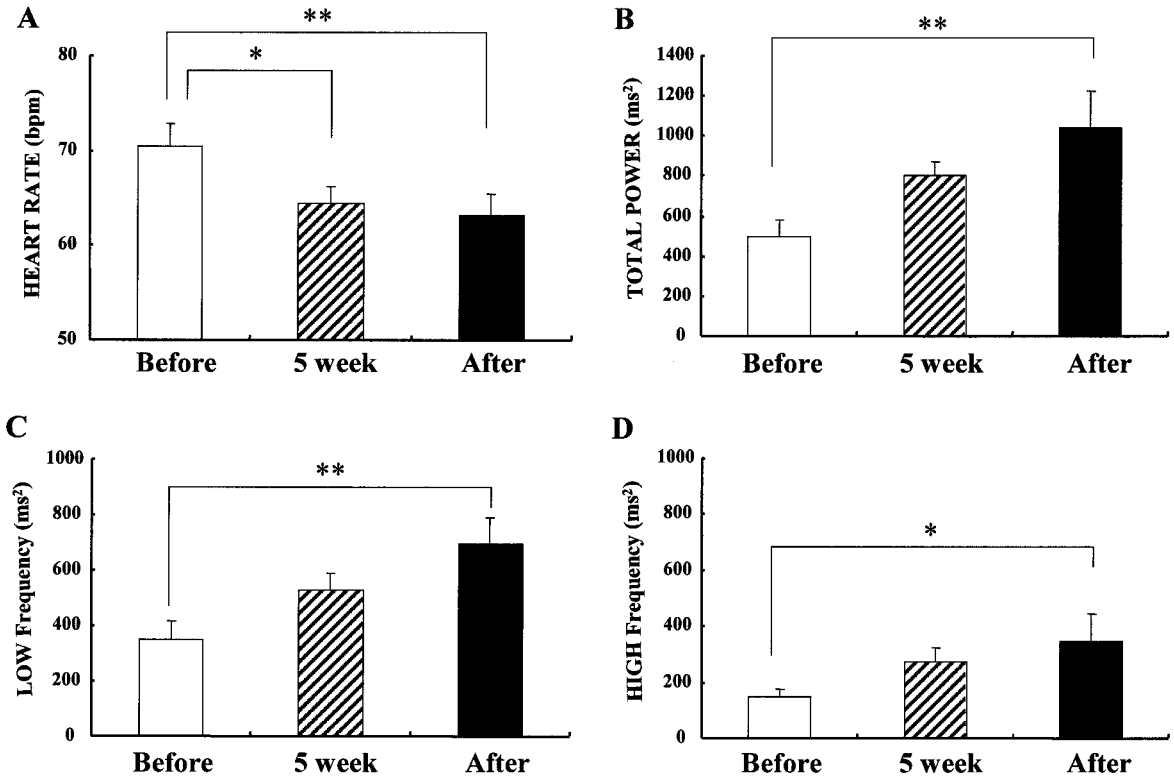


**FIGURE 2—Group data with respect to the heart rate and the R-R spectral parameter before, at 5 wk, and after the exercise program. Heart rate (A), total power (B), low frequency (C), and high frequency (D). Results are expressed as mean ± SE. * $P < 0.05$; ** $P < 0.01$; ANOVA followed by a least significant difference test for comparisons of our time course changes.**

nervous system. Our data demonstrated that obese young women process much reduced the LO frequency component of HRV responsiveness to capsaicin-containing spicy food, as well as a lower capacity to enhance energy metabolism after food ingestion (14).

We have also shown that the LO frequency component of HRV selectively increased against external thermogenic perturbation such as acute cold exposure in nonobese individuals (13). Our recent work demonstrated that subjects with the heterozygotes of $Trp^{64}$ polymorphism of the$\beta$-$_3$ adrenergic receptor, responsible for the control of lipolysis and thermogenesis through ANS activity, manifested a significantly lower LO frequency power than normal subjects (26). With all these data taken into account, the LO frequency component of HRV may reflect the SNS activity modulating energy metabolism, although it is associated with both the SNS and PNS activity (1,21). Our present data indicated that the LO frequency component was significantly increased after exercise training. These data suggest that exercise training increased the SNS activities and that these favorable changes may have an influence on the energy balance and thermogenesis related to obesity. Thus, the exercise training in obese individuals may result in not only fat loss and the reduction of cardiovascular risk (16) but also in the improvement of the autonomic nervous system of thermoregulatory control over the obesity.

In summary, our 12-wk exercise training program has significantly improved both the sympathetic and parasympathetic nervous activities of the obese individuals with markedly reduced ANS activity, suggesting a possible reversal effect of human ANS functions. These favorable changes may also have an influence on the thermoregulatory control over the obesity.

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