Effect of Habitual Aerobic Exercise on Body Weight and Arterial Function in Overweight and Obese Men

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[3 figures and 1 table]

ABSTRACT

The effect of habitual exercise on vascular function, including central arterial distensibility and endothelial function, in obese subjects has not yet been clarified. We investigated whether aerobic exercise training affects central arterial distensibility and endothelial function in middle-aged overweight and obese men. Twenty-one overweight and obese men (age: 50 ± 2 years, body mass index (BMI): 30 ± 1 kg/m^2) completed a 12-week aerobic exercise intervention. Aerobic exercise training significantly reduced body weight and resulted in a significant decrease in BMI. After the weight-reduction exercise program, carotid arterial compliance (via simultaneous B-mode ultrasound and arterial applanation tonometry on the common carotid artery) significantly increased; meanwhile, the β-stiffness index, an index of arterial compliance adjusted for distending pressure, significantly decreased. Concentrations of plasma endothelin-1 (ET-1), a potent vasoconstrictor peptide produced by vascular endothelial cells, significantly decreased and plasma nitric oxide (NO) (measured as the stable end-product [nitrite/nitrate]), a potent vasodilator produced by vascular endothelial cells, significantly increased after the weight-reduction exercise program. In conclusion, weight reduction by aerobic exercise training in overweight and obese men increases central arterial distensibility. This increase may contribute to the improvement in endothelial function, as assessed by a decrease in ET-1 and an increase in NO, after exercise training-induced weight loss.

Keywords: exercise, arterial compliance, endothelial function, obesity

INTRODUCTION

The prevalence of obesity in middle-aged humans is increasing worldwide [1,2]. Obesity has been identified as an independent risk factor for cardiovascular morbidity and mortality [3,4]. It has been well established that regular aerobic exercise is an important strategy for preventing cardiovascular disease [5–7]. However, the effects of aerobic exercise training on vascular functions, i.e., central arterial distensibility and endothelial function, in overweight and obese adults are not known. Accordingly, the major aim of the present study was to examine whether aerobic exercise training affects central arterial distensibility in middle-aged overweight and obese men and, if so, whether the endothelial function, as assessed by endothelin-1 (ET-1) and nitric oxide (NO) concentrations, participates in the mechanism underlying the adaptation of central arterial distensibility to exercise training. We hypothesized that aerobic exercise training could induce weight reduction and an increase in central arterial distensibility in overweight and obese men, and that ET-1 and NO participate in the mechanism underlying this adaptation of central arterial distensibility to weight loss. To examine our hypothesis, we measured carotid arterial compliance; β-stiffness index, an index of arterial compliance adjusted for distending pressure; and plasma ET-1 and NO concentrations before and after a 12-week aerobic exercise intervention program in middle-aged overweight and obese men.

METHODS

Twenty-one middle-aged overweight and obese men participated in the study. Candidates who were current smokers or who were taking any medications were excluded. No subjects had apparent cardiovascular disease, as assessed by medical history and physical examination. The overweight and obese groups were

defined on the basis of the body mass index (BMI) (overweight group: BMI, 25 to <30 kg/m^2 and obesity group: BMI, ≥30 kg/m^2). This study was reviewed and approved by the Institutional Review Board at the University of Tsukuba. All potential risks and procedures of the study were explained to the subjects, and they provided written informed consent to participate in the study.

All overweight and obese men were studied before and after a 12-week training program. Participants were instructed to maintain current eating behaviors for the duration of the 12-week intervention. Dietary intake was assessed by 3-day weighed dietary records and dietary recall interviews for each subject at baseline and at the beginning of week 10 of the intervention by a skilled dietician. All measurements (excluding maximal oxygen uptake [VO_2max]) were obtained between 8 a.m. and 12 p.m. after abstinence from caffeine and an overnight fast. Subjects were studied under supine resting conditions in a quiet, temperature-controlled room (24–26°C). All measurements were performed after a resting period of at least 20 min.

The subjects performed exercise training in the forms of walking and jogging, for sessions of 40–60 min each (three days per week); subjects were supervised by two or three physical trainers. In the first two months, the exercise consisted only of walking, with the target Borg's scale ranging from 11 (light) to 13 (fairly hard). The distances walked were 3.5 km and 4.5 km in the first and second months, respectively. In the last month, subjects performed a combination of a 3.0-km brisk walk and a 1.0-km middle-intensity jog, with the target Borg's scale ranging from 13 (fairly hard) to 15 (hard). Subjects measured their heart rates (HRs) with portable heart-rate monitors (s-610i, Polar Electro OY, Oulu, Finland) while walking and jogging, and recorded the duration (min) and intensity (HR or the Borg's scale) during each exercise session.

VO_2max was determined during a graded exercise test using a cycling ergometer (818E, Monark, Stockholm, Sweden). After a 2-min warm-up in 30 W, the subject started with workload of 15 W each minute, until volitional exhaustion occurred. Pulmonary ventilation and gas exchange were measured, breath-by-breath, with an online data acquisition system (Oxycon Alpha System, Mijnhardt, Breda, The Netherlands). The measurements of individual VO_2max were obtained between 8 a.m. and 2 p.m.

DEXA (DPX-L, Lunar, WI, USA) was used to evaluate segmental body composition, which consisted of fat mass (FM) and fat-free mass (FFM). Transverse scans were used to measure FM and FFM, and pixels of soft tissue were used to calculate the ratio of mass attenuation coefficients at 40–50 ke V (low energy) and 80–100 ke V (high energy), using software version 1.3Z.

The visceral fat area and subcutaneous fat area (cm^2) were measured using computed tomography (CT) scans (Somatom AR.C; Siemens, Erlangen, Germany), as previously described [8]. Visceral and subcutaneous fat areas were calculated using a computer software program (FatScan, N2 System, Osaka, Japan).

The supine systolic blood pressure (BP) (SBP), diastolic BP (DBP), mean arterial pressure (MAP), and HR were recorded from the left arm using a semi-automated device (Dinamap, Johnson & Johnson, NJ, USA).

The combination of ultrasound imaging of a common carotid artery with the simultaneous applanation of tonometrically obtained arterial pressure from the contralateral carotid artery permits the noninvasive determination of dynamic arterial compliance. Subjects were studied under quiet resting conditions while in the supine position. Common carotid artery diameter was measured from the images derived from an ultrasound machine (EnVisor, Koninklijke Philips Electronics, Eindhoven, The Netherlands) equipped with a high-resolution (7.5-MHz) linear-array

transducer. Longitudinal images of the cephalic portion of the common carotid artery were acquired 1–2 cm proximal to the carotid bulb, with the transducer placed at a 90-degree angle to the vessel so that near and far wall interfaces were clearly discernible. These images were recorded on a computer recorder, for later offline analysis. The computer images were analyzed with the use of image analysis software. All image analyses were performed by the same investigator. Time-points that corresponded with maximum systolic expansion of the carotid artery and basal (minimum) diastolic relaxation were selected. The distances (or the diameter) between the vessel far-wall and near-wall boundary, corresponding to the interface of the adventitia and media, were then measured.

Cross-sectional compliance (CC) [9] was calculated from the change in cross-sectional area (dA) and local pulse pressure (dP), using the formula CC = dA/dP. dA was calculated as $dA = \pi \cdot ([D + dD] / 2)^2 - \pi \cdot (D/2)^2$. Pressure wave forms of the left common carotid artery were recorded with an applanation tonometry device (formPWV/ABI, Colin Medical Technology, Komaki, Japan) and calibrated by equating the carotid mean arterial and diastolic blood pressure to that of the brachial artery [10].

The β-stiffness index [11] was calculated using the equation $\beta = \ln (Ps / Pd) / ([Ds - Dd] / Dd)$, where Ds and Dd are the maximum and minimum arterial diameters and Ps and Pd are the highest and lowest blood pressures, respectively [10].

Each blood sample was placed in a chilled tube containing aprotinin (300 kallikrein-inactivating U/ml) and EDTA (2 mg/ml) and was then centrifuged at 2,000 *g* for 15 min at 4°C. The plasma was stored at –80°C until assayed. Plasma concentrations of ET-1 were determined using a sandwich-EIA Kit (Immuno-Biological Laboratories, Fujioka, Japan). The ET-1 assay was carried out as previously

described in our laboratory guidelines [7]. Plasma NO level (measured as the concentration of its stable end-product, nitrite/nitrate [NOx]) was determined using the methods followed in our laboratory [6]. Serum concentrations of cholesterol and triglycerides, as well as plasma concentrations of glucose, were determined using standard enzymatic techniques.

Data are expressed as means ± SE. To evaluate differences in the levels before and after the weight-reduction program in the subjects, the Student's *t*-test for paired values was used. The blood pressure-independent effect of weight loss on β-stiffness index was tested by using analysis of covariance (ANCOVA). Analyses were carried out using the statistical package for the social sciences (SPSS) version 16.0 for Windows. $P < 0.05$ was accepted as significant.

RESULTS

There was no difference between at baseline and at the follow-up for dietary intake (2221 ± 90 vs. 2163 ± 106 kcal/day; NS). Table 1 shows the characteristics of each of the overweight and obese men (age, 50 ± 2 years; BMI, 30 ± 1 kg/m^2) before and after the 12-week exercise training-induced weight-reduction program. Body weight, BMI, FM, body fat areas, serum concentrations of total cholesterol, LDL-cholesterol, and triglycerides, SBP, DBP, MAP, pulse pressure (PP), and HR significantly decreased after the intervention. Maximum oxygen uptake significantly increased after the exercise intervention (Fig. 1). The arterial compliance significantly increased and the β-stiffness index significantly decreased after the exercise training-induced weight-reduction program (Fig. 2). Moreover, ANCOVA revealed that the effect of the exercise intervention on β-stiffness index was statistically independent of age, SBP, DBP, and MBP ($F = 1.6$ and $P < 0.05$). Thus, weight reduction by aerobic exercise in overweight and obese men increased central

arterial distensibility. The plasma ET-1 concentration significantly decreased after the exercise training-induced weight-reduction program (Fig. 3A). The plasma concentration of NOx significantly increased with exercise training-induced weight loss (Fig. 3B).

DISCUSSION

In the present study, we determined central arterial distensibility and endothelial function in overweight and obese men before and after a 12-week aerobic exercise training-induced weight-reduction program. After the exercise program in which BMI markedly decreased, carotid arterial compliance significantly increased and β-stiffness index decreased. Thus, weight reduction by aerobic exercise induced an increase in central arterial distensibility among overweight and obese men. We also demonstrated that the aerobic exercise training-induced weight-loss program significantly decreased plasma ET-1 concentration and markedly increased plasma NOx—a stable end-product of NO concentration—among overweight and obese men, suggesting an improvement in endothelial function. Therefore, we suggest that increased central arterial distensibility via a 12-week aerobic exercise training-induced weight loss may also contribute to an improvement in endothelial function.

Reduced arterial distensibility has been implicated in the pathophysiology of cardiovascular disease and identified as an independent risk factor for cardiovascular disease [12-14]. Several studies have demonstrated that obese subjects have lower degrees of arterial distensibility [15-17]. Danias et al. [15] have shown that obese men have lower aortic elasticity than do age-matched non-obese men. Furthermore, it has been reported that aortic pulse-wave velocity (PWV), a traditional index of arterial stiffness, is higher in middle-aged overweight and obese adults than

age-matched normal weight adults [17]. Taken together, these findings clearly demonstrate that arterial distensibility in overweight and obese humans is lower than in normal-weight humans. In the present study, we demonstrate for the first time that aerobic exercise training-induced weight loss increased arterial compliance and decreased β-stiffness index in overweight and obese men. These findings suggest that weight reduction through habitual aerobic exercise induces an increase in central arterial distensibility in overweight and obese men, which may have beneficial effects *vis-à-vis* the prevention of cardiovascular disease.

Many studies have shown that regular aerobic exercise is associated with greater central distensibility [5,18,19]; however, it is not known whether aerobic exercise training influences arterial distensibility in overweight and obese adults. In the present study, we found that central arterial distensibility, as assessed by the carotid arterial compliance and the β-stiffness index, increased with aerobic exercise training in overweight and obese men. Thus, habitual aerobic exercise produces beneficial effects with respect to central arterial distensibility in overweight and obese adults. We suggest that regular aerobic exercise is an important strategy that can be applied for preventing the occurrence of vascular disease in overweight and obese humans.

Obesity is also strongly associated with endothelial dysfunction, which may play a role in the development of decreased arterial distensibility [5,20-23]. Vascular endothelial cells produce some vasoactive substances, i.e., ET-1 and NO [24,25]. ET-1 produced by vascular endothelial cells has a potent vasoconstrictor and proliferative activity on vascular smooth muscle cells [24,26]. Previous studies have reported that arterial distensibility was decreased by the intra-arterial infusion of ET-1 and increased by the administration of an ET-1 receptor antagonist [27,28]. NO produced by vascular endothelial cells has a potent vasodilator effect and plays an

important role in regulating platelet-vessel wall interactions and vascular resistance and growth [25]. Wilkinson et al. [29] reported that arterial distensibility decreased following intra-arterial infusion of a NO synthase inhibitor [29]. These findings suggest that endogenous ET-1 and NO participate in the regulation of arterial distensibility. In the present study, we demonstrated that plasma ET-1 concentration was decreased and plasma NOx concentration increased by aerobic exercise training-induced weight reduction in overweight and obese men. Taken together, these data suggest that the increase in central arterial distensibility via exercise training-induced weight loss may contribute to an improvement in endothelial function, as assessed by ET-1 and NO, following an aerobic exercise training-induced weight-reduction program in overweight and obese men.

This study, however, has several limitations. First, the present study may have measurement bias due to the lack of blinding. Second, there may be other confounders responsible for cardiovascular health. Third, it is not apparent whether weight loss or aerobic exercise had an impact on arterial function.

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FIGURE LEGENDS

Figure 1. Maximum oxygen uptake before and after a 12-week aerobic exercise intervention in overweight and obese men. Data are expressed as means ± SE.

Figure 2. Arterial compliance and β-stiffness index before and after a 12-week aerobic exercise intervention in overweight and obese men. Data are expressed as means ± SE.

Figure 3. Plasma endothelin-1 and nitrite/nitrate (NOx, the stable end-product of nitric oxide) concentrations before and after a 12-week aerobic exercise intervention in overweight and obese men. Data are expressed as means ± SE.

Figure 1

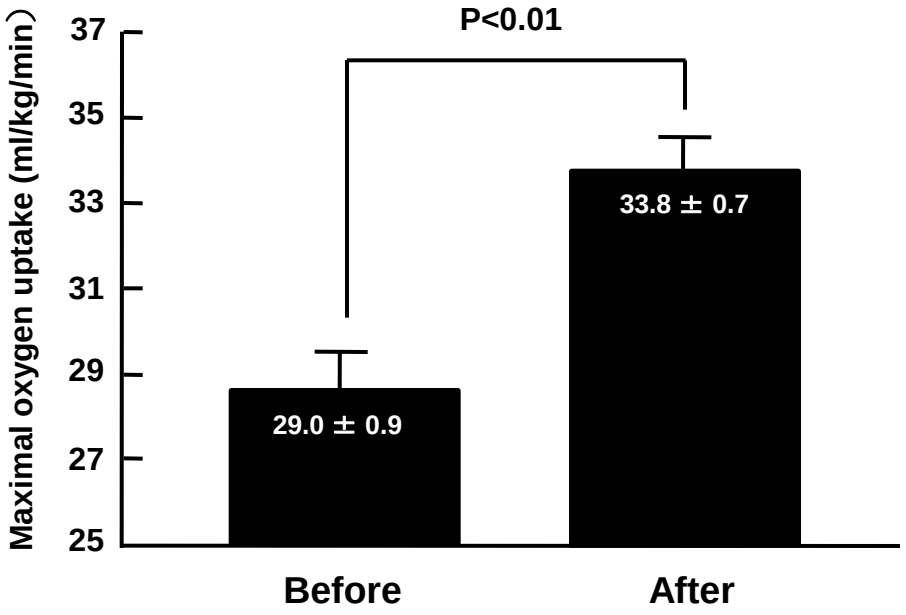
P<0.01
Maximal oxygen uptake (ml/kg/min)
37
35
33
31
29
27
25
33.8 ± 0.7
29.0 ± 0.9
Before
After

Figure 2

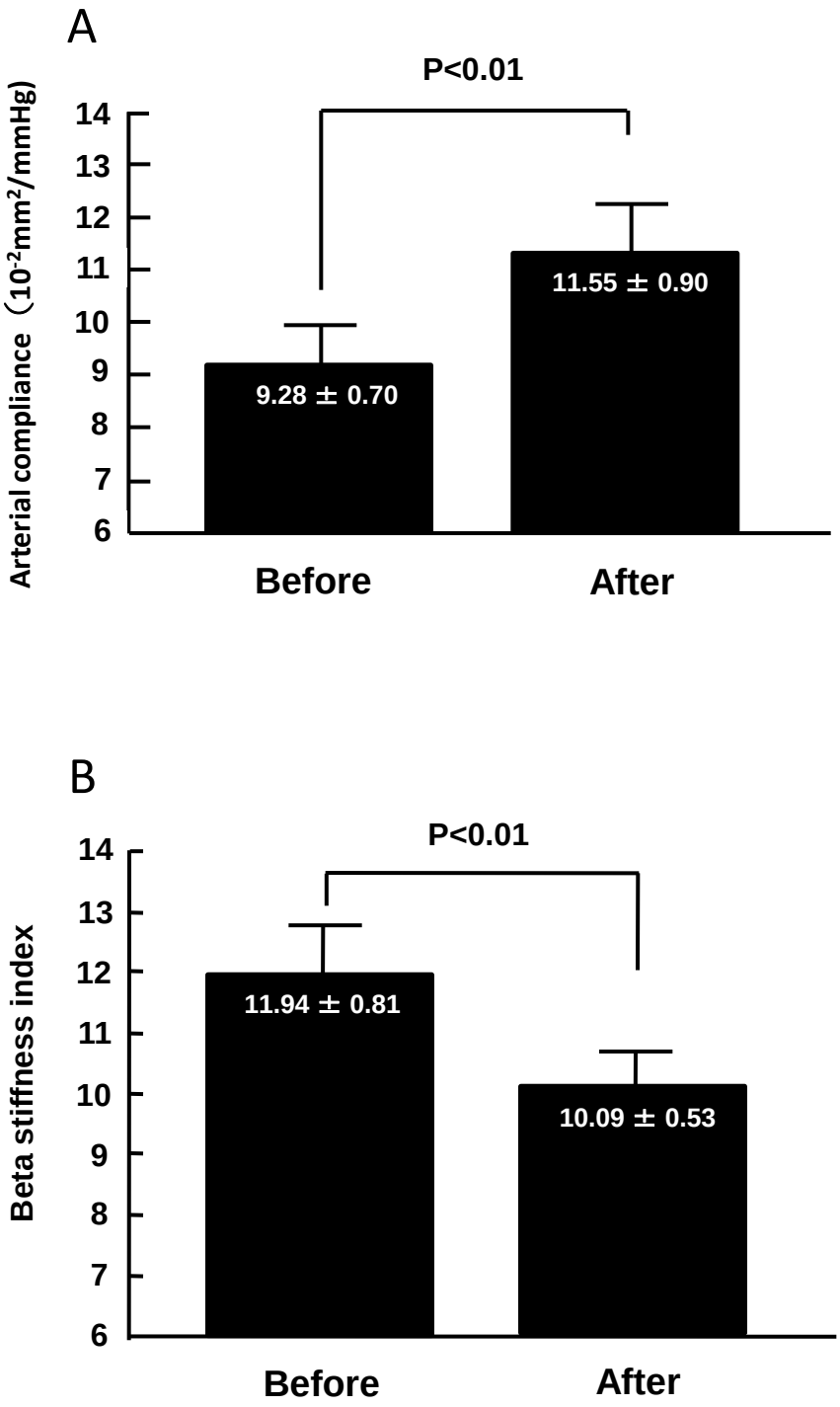
A
P<0.01
Arterial compliance（10-2mm2/mmHg)
14
13
12
11
10
9
8
7
6
11.55 ± 0.90
9.28 ± 0.70
Before
After
B
P<0.01
Beta stiffness index
14
13
12
11
10
9
8
7
6
11.94 ± 0.81
10.09 ± 0.53
Before
After

Figure 3

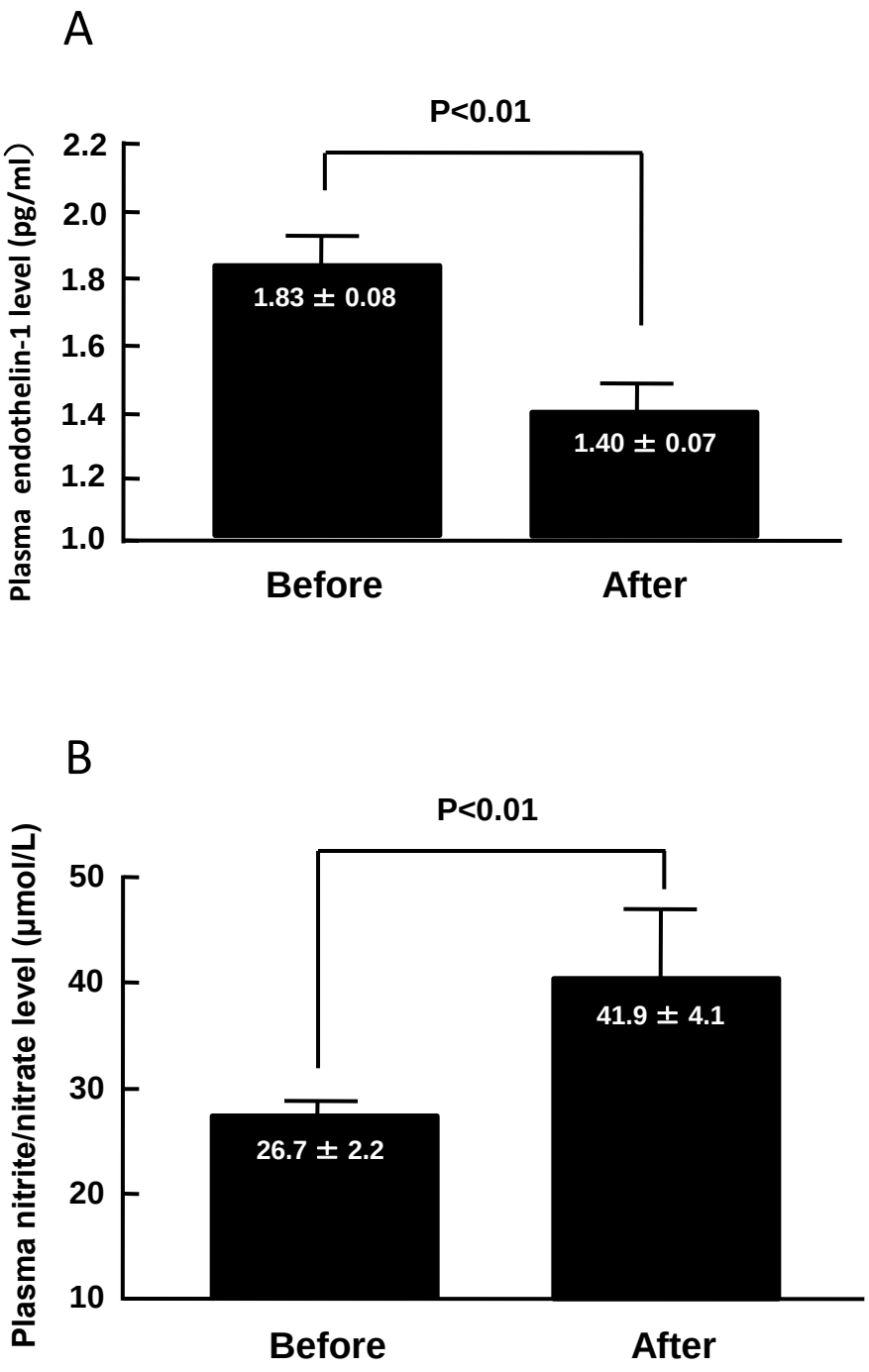
A
P<0.01
Plasma endothelin-1 level (pg/ml)
2.2
2.0
1.8
1.6
1.4
1.2
1.0
1.83 ± 0.08
1.40 ± 0.07
Before
After
B
P<0.01
Plasma nitrite/nitrate level (μmol/L)
50
40
30
20
10
41.9 ± 4.1
26.7 ± 2.2
Before
After

Table 1. Indivisual characteristics of overweight and obese men before and after the aerobic exercise-induced weight-reduction prog

subject No.	Age, yrs	Height, cm	Body Weight, kg		BMI, kg/m^2		Total FM, kg		Trunk FM, kg		Total LM, kg		Trunk LM, kg	
			pre	post	pre	post	pre	post	pre	post	pre	post	pre	post
1	56	183	83.2	79.3	25.0	23.8	20.2	16.2	12.5	9.5	60.5	60.3	26.2	26.3
2	45	159	64.7	65.0	25.6	25.7	19.6	18.3	11.2	10.8	55.3	55.7	25.3	26.1
3	47	175	81.9	77.3	26.8	25.3	23.9	18.2	15.3	11.9	62.5	61.6	27.1	27.9
4	61	164	73.6	67.5	27.4	25.1	19.3	13.0	12.8	8.2	58.1	58.7	25.3	26.8
5	41	175	84.5	82.1	27.5	26.7	25.3	22.8	16.0	14.4	55.0	56.1	24.6	24.3
6	59	177	86.5	83.3	27.6	26.6	26.5	23.3	18.7	16.7	48.6	49.8	21.4	22.4
7	53	162	72.8	72.1	27.7	27.5	22.1	20.7	15.1	13.8	48.2	48.9	22.9	22.7
8	39	173	84.6	81.5	28.2	27.2	29.5	26.1	18.9	16.6	52.3	52.5	22.8	23.0
9	58	168	82.3	78.6	29.1	27.8	28.5	22.4	18.2	14.8	53.4	53.4	23.5	23.9
10	63	155	71.5	69.4	29.6	28.8	21.0	19.6	12.2	11.4	46.8	47.0	21.0	20.6
11	61	165	82.6	77.2	30.2	28.2	27.1	20.9	16.2	12.4	52.5	53.1	24.2	24.1
12	57	175	93.5	88.9	30.4	28.9	31.9	29.8	20.0	18.5	54.5	56.3	23.0	23.8
13	50	178	97.3	93.0	30.6	29.2	30.4	26.4	18.4	16.6	60.3	61.7	27.1	28.9
14	42	164	83.0	82.3	30.7	30.5	29.6	28.0	18.6	17.4	60.8	59.2	26.1	25.6
15	36	174	93.1	93.2	30.8	30.8	29.0	29.4	17.4	18.4	50.1	50.9	21.7	23.4
16	46	175	95.0	93.1	30.9	30.3	32.1	31.0	19.3	19.2	53.2	53.7	24.2	24.7
17	56	178	101.2	95.3	32.0	30.1	35.0	28.6	22.9	18.7	42.6	42.5	18.6	18.0
18	61	174	97.5	94.7	32.3	31.4	27.6	25.1	18.2	16.3	65.9	65.0	29.4	28.3
19	34	169	93.5	91.0	32.8	31.9	34.2	33.4	20.5	20.2	55.6	52.3	25.8	23.6
20	40	157	91.2	87.6	37.0	35.5	37.4	34.2	21.9	20.0	51.3	50.7	23.1	22.3
21	38	174	117.6	111.0	39.1	36.9	45.9	41.5	26.8	23.8	67.4	65.1	34.0	31.4
Mean	50	170	87.2	84.0	30.1	29.0	28.4	25.2	17.7	15.7	55.0	55.0	24.6	24.7
SE	2	2	2.6	2.4	0.7	0.7	1.4	1.5	0.8	0.9	1.4	1.3	0.7	0.7
p value				**		**		**		**				

BMI = Body Mass Index; FM = Fat Mass; LM = Lean Mass; TFA = Total Fat Area; VFA = Visceral Fat Area; SFA = Subcutaneous Fat Area; T-CHO = Total-Cholesterol; HDL-CHO = HDL-Cholesterol; LDL-CHO= LDL-Cholesterol, FBG = Fasting Blood Glucose; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; MAP = Mean Arterial Pressure; PP = Pulse Pressure; HR = Heart Rate Significant difference before vs. after aerobic exercise intervention, *P < 0.05; **P < 0.01.

Table 1. (Continued)

TFA, cm^2		VFA, cm^2		SFA, cm^2		T-CHO, mg/dl		HDL-CHO, mg/dl		TG, mg/dl		LDL-CHO, mg/dl	
pre	post	pre	post	pre	post	pre	post	pre	post	pre	post	pre	post
290	205	151	90	139	115	204	204	46	54	178	153	123	119
272	242	146	126	125	115	252	230	38	32	263	201	161	158
348	316	165	144	184	172	172	167	52	54	92	54	102	102
275	172	134	80	141	91	235	257	110	125	51	63	115	120
376	300	199	164	177	136	266	219	33	36	480	291	156	125
394	364	236	223	158	141	234	203	53	54	94	75	162	134
344	329	202	191	142	138	235	200	47	43	160	122	157	133
384	351	216	198	169	153	181	164	41	36	132	135	114	101
437	383	199	197	238	186	250	184	51	49	143	99	170	115
286	266	141	125	144	140	239	221	52	57	317	160	136	132
421	269	200	122	221	147	204	176	59	62	84	85	128	97
480	427	276	257	203	170	242	241	44	50	138	105	171	170
348	316	214	203	134	113	244	231	56	60	294	118	130	147
499	384	206	129	293	255	241	239	47	47	241	203	145	152
417	431	200	215	218	216	201	215	51	49	127	227	124	121
350		160		190		164	178	38	42	162	165	94	103
480	381	306	215	173	165	312	238	45	53	410	133	201	159
525	412	249	187	275	225	255	211	67	58	140	160	160	121
455	437	140	159	315	279	220	180	55	46	195	76	126	118
627	540	322	266	305	274	368	358	48	49	138	127	293	283
735	497	309	165	426	332	228	192	46	46	189	136	144	119
416	351	208	173	208	178	236	215	51	52	192	138	148	135
26	21	13	12	18	15	10	9	3	4	24	13	9	9
	**		**		**		**				**		*

Table 1. (Continued)

FBG, mg/dl		SBP, mmHg		DBP, mmHg		MAP, mmHg		PP, mmHg		HR, bpm	
pre	post	pre	post	pre	post	pre	post	pre	post	pre	post
92	90	126	113	78	74	94	87	49	39	62	51
91	87	124	103	78	71	94	82	46	32	59	60
98	99	120	101	68	53	85	69	52	48	63	54
110	106	157	150	93	87	114	108	64	63	60	54
93	89	140	128	81	79	101	95	59	49	63	70
92	101	140	136	87	84	105	101	52	52	70	68
113	108	143	117	87	77	106	90	56	40	66	64
106	97	115	115	71	70	86	85	44	45	57	54
87	94	125	120	77	76	93	90	49	44	58	54
95	104	131	137	86	91	101	106	46	46	54	57
154	121	164	148	80	76	108	100	84	72	63	64
95	89	190	168	107	106	134	126	84	62	76	66
119	99	145	113	87	77	106	89	59	36	63	58
91	93	151	139	97	92	115	108	54	48	62	63
87	87	145	133	83	68	104	90	62	66	80	62
91	97	127	117	72	75	90	89	55	43	79	67
117	105	152	131	83	77	106	95	69	54	54	47
108	88	152	153	95	95	114	114	57	58	62	60
80	82	203	178	129	104	153	133	74	74	68	54
84	91	124	121	75	78	91	92	49	44	63	66
91	93	173	164	119	112	137	129	55	52	84	75
100 4	95 2	145 5	133 5 **	87 3	82 3 **	105 4	99 4 **	59 3	51 2 **	65 2	60 2 **