ORIGINAL ARTICLE

The Relative Benefits of Endurance and Strength Training on the Metabolic Factors and Muscle Function of People With Type 2 Diabetes Mellitus

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ABSTRACT. Cauza E, Hanusch-Enserer U, Strasser B, Ludvik B, Metz-Schimmerl S, Pacini G, Wagner O, Georg P, Prager R, Kostner K, Dunky A, Haber P. The relative benefits of endurance and strength training on the metabolic factors and muscle function of people with type 2 diabetes mellitus. Arch Phys Med Rehabil 2005;86:1527-33.

Objective: To compare the effects of a 4-month strength training (ST) versus aerobic endurance training (ET) program on metabolic control, muscle strength, and cardiovascular endurance in subjects with type 2 diabetes mellitus (T2D).

Design: Randomized controlled trial.

Setting: Large public tertiary hospital.

Participants: Twenty-two T2D participants (11 men, 11 women; mean age ± standard error, 56.2±1.1y; diabetes duration, 8.8±3.5y) were randomized into a 4-month ST program and 17 T2D participants (9 men, 8 women; mean age, 57.9±1.4y; diabetes duration, 9.2±1.7y) into a 4-month ET program.

Interventions: ST (up to 6 sets per muscle group per week) and ET (with an intensity of maximal oxygen consumption of 60% and a volume beginning at 15min and advancing to a maximum of 30min 3×/wk) for 4 months.

Main Outcome Measures: Laboratory tests included determinations of blood glucose, glycosylated hemoglobin (Hb A_{1c}), insulin, and lipid assays.

Results: A significant decline in Hb A_{1c} was only observed in the ST group (8.3%±1.7% to 7.1%±0.2%, $P=.001$). Blood glucose (204±16mg/dL to 147±8mg/dL, $P<.001$) and insulin resistance (9.11±1.51 to 7.15±1.15, $P=.04$) improved significantly in the ST group, whereas no significant changes were observed in the ET group. Baseline levels of total cholesterol (207±8mg/dL to 184±7mg/dL, $P<.001$), low-density lipoprotein cholesterol (120±8mg/dL to 106±8mg/dL, $P=.001$), and triglyceride levels (229±25mg/dL to 150±15mg/dL, $P=.001$) were significantly reduced and high-density lipoprotein cholesterol (43±3mg/dL to 48±2mg/dL, $P=.004$) was significantly increased in the ST group; in contrast, no such changes were seen in the ET group.

Conclusions: ST was more effective than ET in improving glycemic control. With the added advantage of an improved lipid profile, we conclude that ST may play an important role in the treatment of T2D.

Key Words: Hyperglycemia; Insulin resistance; Physical endurance; Rehabilitation.

THE EFFECTIVENESS OF physical exercise for the treatment of type 2 diabetes mellitus (T2D) has long been recognized.[1,2] Endurance training (ET) has been advocated as the most suitable form of exercise,[3,4] with many positive metabolic effects, such as improvements in lipid profile,[5] reduced body fat,[5] and decreased blood glucose (BG) levels.[5] ET also appears to be effective in improving insulin resistance in patients with T2D[6,7] and in obese subjects without diabetes.[8] By comparison, only limited information is available on the effect of strength training (ST) on T2D.[9-12] Reports on the effects of ST on glycemic control in patients with T2D have been controversial. For example, a 2-month trial with 11 patients with T2D reported that ST had no effect on glucose metabolism,[11] whereas another study[13] found only small improvements (0.5% difference in glycosylated hemoglobin [Hb A_{1c}] vs the control group) in patients with T2D after a 5-month resistance training program. In a third study,[12] 8 T2D patients who had participated in a 3-month circuit of progressive resistance training showed significant improvement ($P<.05$) in Hb A_{1c} that was associated with a significant increase in muscle tissue, as measured by magnetic resonance imaging. Two recent studies support the benefits of ST on glycemic control. First, Dunstan et al[10] reported a significant improvement of Hb A_{1c} (15%) after high-intensity resistance training in older T2D patients. After 6 months of resistance training in combination with a moderate weight loss diet, there was a 15% reduction in Hb A_{1c}. Second, Castaneda et al[9] showed improved metabolic control (Hb A_{1c} decreased from 8.7% to 7.6%, $P=.01$) by progressive resistance training in 31 Latino patients with T2D. Erikson et al[12] reported no significant changes in lipid levels with a moderate-intensity and high-volume resistance training program. Similarly, serum lipids and lipoproteins remained unchanged in the study of Dunstan,[10] whereas Castaneda[9] reported a trend toward a reduction in serum triglyceride (TG) levels within the progressive resistance training group compared with control subjects ($P=.08$).

One possible explanation of the positive effects of ST on insulin resistance (IR) may be the increase in the number of glucose transporter (GLUT) proteins. In skeletal muscle

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cells, GLUT4 is thought to be responsible for insulin- and contraction-stimulated glucose transport[14] in skeletal muscle. An increase in GLUT4 has also been observed after ST by Tabata et al.[15] In addition, increasing total muscle mass will ultimately result in an increase in total insulin-mediated glucose uptake. Another possible underlying mechanism for improved glucose uptake could be an increased number of insulin receptors in the muscle cell.

In contrast to ST, ET has different effects on skeletal muscles, the cardiovascular system, and the autonomic nervous system. ET increases skeletal muscle capillarization and blood flow, muscular GLUT4 levels, hexokinase, and glycogen synthase activities. In contrast to ST, the adaptations in skeletal muscle as a result of ET involve an increase in the capacity for aerobic metabolism made possible by an adaptive increase in mitochondrial content as well as a number of other enzymatic adaptations that may contribute to the altered metabolic response to exercise in the trained state.[16-18]

Abnormal insulin secretion, diminished glucose effectiveness, and both peripheral and hepatic IR are the primary pathogenic factors that lead to T2D,[6] which is a serious, chronic disease associated with hyperglycemia, obesity, and the metabolic syndrome.[19] In addition to obesity, hyperglycemia alone impairs insulin secretion and causes IR and thus makes the pathogenesis of T2D even more complex.[19,20] Hyperinsulinemia and IR are associated with several atherogenic changes that increase the risk of development of coronary heart disease.[21] These include dyslipidemia, especially abnormalities in total cholesterol (TC) with high levels of low-density lipoprotein cholesterol (LDL-C) and TG; obesity; and hypertension. Such abnormalities contribute to the risk of micro- and macrovascular complications.[6,21]

The skeletal muscle is responsible for up to 40% of total body weight. ST may induce beneficial changes in IR via muscle mass development. Skeletal muscle tissue is the major site of insulin-mediated glucose uptake and strongly influences IR, which is characterized by a decrease in glucose uptake into the skeletal muscle tissue in patients with T2D.[6] Because IR is an important modifiable risk factor for atherosclerosis, we studied the potential beneficial effects of ST versus ET on IR, muscular mass, and oxygen consumption ($\dot{V}O_2$) in patients with T2D.

METHODS

Study Population

We randomized 43 patients from our diabetes outpatient department—22 men (mean age ± standard error [SE], 56.5±0.9y; range, 51–69y) and 21 women (mean age, 57.4±0.9y; range, 50–70y)—between September 2000 and May 2002 who had T2D and no complications or comorbid conditions. The patients were consecutively divided into 2 groups (ST vs ET); none from either group was involved in organized ET programs. One subject did not complete the study because of health reasons unrelated to the investigation and 3 subjects did not complete the study because of private reasons. All participants had a fasting glucose concentration of 126mg/dL or greater (≥7.0mmol/L) and met the World Health Organization criteria for the diagnosis of T2D. Only patients between the ages of 50 and 70 years were accepted for the study. No limitations were given for body weight or body mass index (BMI). All demographic data are shown in table 1.

A physician performed physical examinations on all subjects before the study. Subjects were excluded if they had rapidly progressive or terminal illness, myocardial infarction, uncontrolled arrhythmias, third-degree heart blockage, elevated blood pressure (>200/100mmHg on therapy), nephropathy (microalbuminuria >20μg/min albumin excretion), severe peripheral or autonomic neuropathy, or diabetic proliferative retinopathy. Other exclusion criteria were severe musculoskeletal and neurologic abnormalities. Mild peripheral neuropathy was not considered a contraindication.

Table 1: Subject Characteristics and Treatment Regimens at Baseline

Characteristics and Regimens	Strength Training	Endurance Training	*P*
Sex (male/female)	11/11	9/8	
Age (y)	56.4±1.1	57.9±1.4	NS
Duration of diabetes (y)	8.83±3.5	9.2±1.71	NS
Treatment regimens			NS
Antidiabetic drug therapy			
Sulphonylurea	11	11	NS
Metformin*	15	13	NS
Insulin therapy	4	3	NS
Lipid-lowering drug therapy			
Statins†	8	7	NS
Antihypertensive drug therapy			
3 or more different antihypertensive medications	14	13	NS

NOTE. Values are n or mean ± SE.
Abbreviation: NS, not significant.
*Biguanide.
†A hyrdoxymethyl glutaryl coenzyme A reductase inhibitor.

All participants were told to continue their current medications during the study. Medications (especially sulphonylureas) were modified only to avoid hypoglycemia. All participants received specific recommendations to keep their energy intake unchanged during the 4-month training period.

The Ethics Committee at the Confraternitaet Hospital, Vienna, approved the study protocol. The purpose, nature, and potential risks of the study were explained to the participants before obtaining their written consent.

Training Program

We tried to define comparable training units for both groups. A unit is defined as an organizational unit for both training groups where training occurs. To do this, we took comparable training units of top athletes for each training group. A top weight-lift body builder, for example, does 30U per muscle group per week, whereas a top endurance athlete trains for 10 to 12 hours a week. For our study, we took 15% to 20% of these training units (repetitions by sets) for each group.

Endurance training. Systematic ET was performed on a cycle ergometer on 3 nonconsecutive days of the week. During the first 4 weeks, ET participants trained for 15 minutes per session, 3 times a week. Exercise sessions were increased by 5 minutes every 4 weeks. The total exercise time per week, excluding warmup and cool down, was 90 minutes during the last 4 weeks.

Heart rate (HR) was monitored continuously throughout the training period.[a] Based on the linear correlation between $\dot{V}O_2$ and heart rate, training was controlled by a heart rate according to 60% of $\dot{V}O_2$max. This was derived from ergometry by using the following formula[22]:

$$HR = HRrest + (HRmax - HRrest) \times 0.6 \pm 5 \text{ beats/min}$$

where HRrest was heart rate after a break of 5 minutes, in supine position.

Strength training. Twenty-two subjects participated in a 4-month systematic ST program on 3 nonconsecutive days of the week. A brief warmup of 10 minutes of moderate cycling with very low intensity was performed before each training session. Participants were instructed in correct exercise techniques and supervised throughout the entire training period by a professional instructor and an experienced physician. During the first 2 weeks, the weight was kept at a minimal level in order for participants to learn the exercise techniques, adapt the muscles to training, and prevent muscle soreness. From the third week, the training was aimed at hypertrophy and began with 3 sets per muscle group per week. One set consisted of 10 to 15 repetitions without interruption, until severe fatigue occurred and further repetitions were impossible. The training load was systematically adapted to keep the maximal possible repetition per set between 10 and 15. When more than 15 repetitions were successfully performed at a given weight, the weight was increased by an amount that permitted approximately 10 repetitions to be performed. The number of sets for each muscle group was systematically increased from 3 at the beginning of the program to 4, 5, and finally 6 sets per week at the end of the program. The ST program consisted of exercises for all major muscle groups. Exercises to strengthen the upper body included bench press (pectoralis), chest cross (horizontal flexion of the shoulder joint), shoulder press (trapezius, latissimus dorsi), pull downs (back muscles), biceps curls, triceps extensions, and exercises for abdominal muscles (situps). Lower-body exercises included leg press (quadriceps femoris), calf raises, and leg extensions (biceps femoris).

Testing

Dynamometry. Maximal strength of a muscle was determined by 1 repetition maximum (1-RM in kilograms) by using the Concept 2 Dyno.[b] One repetition maximum is defined as the maximal strength that a muscle group is able to generate with a single contraction. Resistance is created in direct response to the patient's effort. After each completed lift, a monitor shows how much weight was lifted. The Concept 2 Dyno has 3 basic positions for the determination of muscle strength using the 1-RM. A maximum of 3 tests are allowed to avoid muscle fatigue. The 3 representative exercises include bench press, rowing, and leg press, all performed in a seated position.

Spiroergometry: Vo_2peak. All subjects underwent a cycling test on an electrically braked cycle ergometer[c] to the point of exhaustion. Heart rate was continuously monitored via an electrocardiogram, with blood pressure measured in the final minute of each work level. Exercise started with a work load of 50W and was increased stepwise by 25W every 2 minutes until exhaustion. During ergometry, expired air was collected by a facemask using the Vmax 229[d] to analyze ventilation and gas fractions. Respiratory gas exchange ($\dot{V}O_2$ and carbon dioxide production [$\dot{V}CO_2$]) was measured breath-by-breath. Vo_2peak (in mL/min) was reached if the following criteria were met: a respiratory exchange ratio (RER; RER=$\dot{V}CO_2/\dot{V}O_2$) greater than 1.0 and ventilatory equivalent to 30 or more. The following parameters were determined: Vo_2peak, Vco_2peak, RER at maximal exercise, maximal power (in watts), and maximal heart rate.

Anthropometric Measurements

Body mass index. Each participant had her/his body weight[e] (to the nearest 0.1kg) and height (to the nearest 0.1cm) recorded while wearing light indoor clothes but no shoes. BMI was calculated as weight divided by height squared (in kg/m^2).

Fat mass. The same person took all skinfold measurements with calipers[f] and recorded them to the nearest 0.1mm. To minimize interobserver variation, the same experienced instructor assessed each patient's skinfold. A mean of 3 measurements was considered to be representative. Measurements were taken at 10 different body sites (bucca, chin, chest, mid-axillary-suprailiac, thigh, abdomen, triceps, subscapula, calf, knee). Percentage of body fat (%BF) was then estimated by using sex-appropriate equations.[23]

$$BF(\%) = BW \times \sqrt{\{[(\text{sum of mean values of the 10 skinfold measurements} - 40)/20 \times BS \times .739/BW] - .003\}} \times 100$$

where body weight (BW) is measured in kilograms and body surface (BS) is equal to $.007184 \times BW^{.425} \times height^{.725}$.

Lean body mass. Lean body mass (LBM) was calculated by total weight minus fat mass.

Laboratory Determinations

Blood glucose, Hb A_{1c}, insulin, and lipid assays. Venous blood was drawn after overnight fasting. TC, high-density lipoprotein cholesterol (HDL-C), and TG were determined with commercially available kits.[g] LDL-C was calculated by using the Friedewald equation.[24] Routine fasting blood glucose (FBG) levels, Hb A_{1c}, and fasting plasma insulin levels were measured with standard techniques.

Insulin resistance. The degree of IR was estimated by the homeostasis model assessment (HOMA). In particular, an IR score was computed with the following formula: fasting plasma glucose (in mmol/L) and fasting serum insulin (in μU/mL) divided by 22.5, as previously described.[25]

Blood Pressure Measurements

Blood pressure measurements were always taken by the same person with a standard sphygmomanometer after the subject remained seated for 5 minutes of quiet rest. Resting blood pressures were recorded twice daily and averaged from 5 separate days in the first and last weeks of training.

Statistical Analysis

Data were analyzed with SPSS, version 10.0.[h] All parameters were described by mean values ± standard error of the mean (SEM). We used 2-way analysis of variance to assess differences between groups at the same time and multivariate analysis of variance to assess significant differences in changes of the same variables before and after training. We used Pearson product moment correlation coefficients to compare changes in LBM and %BF with the changes in metabolic parameters before and after ST. P values less than .05 were considered statistically significant. All P values correspond to 2-sided hypotheses. Because of the exploratory nature of the study, no correction for multiple testing was applied.

RESULTS

At study entry, both exercise groups had similar profiles for all parameters examined except for TG and FBG. Participants who undertook ST had both higher TG baseline levels (229±25mmol/L vs 146±14mmol/L, P=.01) and FBG baseline levels (204±16 vs 160±9, P=.04) than the ET group.

After the 4-month ST period, there were highly significant changes in the maximum strength of all muscle groups, as well as a highly significant increase of LBM. In addition, there were significant changes in glycemic control, TC, and TG between

both groups (table 2). No hypoglycemic episodes were reported during or after training for both groups.

Glycemic Control and Lipid Profile

Indices of glucose, lipid profile, and insulin regulation are shown in table 2. After 4 months of ST, insulin sensitivity (HOMA) significantly improved ($P=.04$), whereas after 4 months of ET no significant changes in insulin sensitivity were seen. The difference in changes after 4 months between ST and ET programs was statistically significant for FBG ($P<.01$), Hb A_{1c} ($P<.05$), fasting plasma insulin ($P<.05$), and IR ($P<.01$) levels. The percentage change in metabolic parameters is shown in figure 1.

Body Composition

The effectiveness of ST and ET in participants with T2D was shown by alterations in body composition (table 3).

Cardiorespiratory Endurance and Muscle Strength

Muscle strength data and cardiorespiratory data are provided in table 4. Peak Vo_2 improved by 8% for ET and by 1% for the ST group, neither of which was significant. Maximum workload improved significantly (by 12%) for both groups (ST, $P<.01$; ET, $P<.01$). The improvement in maximum strength (1-RM) of all muscle groups subjected to ST was highly significant (22%–48% of initial levels). The percentage change in the ET group ranged from no improvements (0%) in bench press up to 15% in leg press. Both ST and ET produced significant improvements in hemodynamic regulation and performance (in watts) but an improvement of Vo_2 was only seen after ET. Antihypertensive medication was not altered during the trial. A significant reduction from baseline was measured for systolic (SBP) and diastolic blood pressure (DBP) after 4 months of ST or ET (see table 2).

Changes in Medications

After 4 months of training, the antidiabetic medication in ST participants was reduced by 12.2% for sulphonylurea and unchanged for metformin, and for the subgroup of 3 ST patients receiving insulin therapy, the insulin dose was decreased by an average of 1.0U/d. For ET participants, sulphonylurea therapy was reduced by 1.7% from baseline, metformin therapy was unchanged and the mean insulin dose was decreased 2U/d after the training period. These changes were not statistically significant. Fifteen subjects were taking lipid-lowering therapy, which was not altered during the exercise period.

Relation between changes in LBM and percentage of body fat and metabolic parameters after 4 months of ST. The change in LBM did not correlate significantly with the change in Hb A_{1c} ($r=.05$, $P<.8$) or in BG ($r=.33$, $P<.1$). There was a tendency toward a negative correlation between FBG and changes in percentage of body fat ($r=-.42$, $P<.06$), although no correlation with Hb A_{1c} was seen ($r=-.18$, $P<.4$). There was a strong correlation between change in LBM and changes in TC ($r=.44$, $P<.05$) and TG ($r=.46$, $P<.05$).

Table 2: Glycemic Control, Lipid Profile, and Blood Pressure Values at Baseline and After 4 Months of ST or ET

Measures	ST	ET	P*
Blood glucose (mg/dL)			
Before	204±16	160±9	.04
After	147±8	159±10	
Difference	−57	−1	.002
P†	<.001	NS	
Plasma insulin (pmol/L)			
Before	130.9±17.9	105.12±18.84	NS
After	118.4±18.2	125.58±23.34	
Difference	−12.5	20.46	.04
P†	NS	NS	
Hb A_{1c} (%)			
Before	8.3±1.7	7.7±0.3	NS
After	7.1±0.2	7.4±0.3	
Difference	−1.2	−0.3	.04
P†	.001	NS	
HOMA-IR			
Before	9.1±1.5	6.8±1.4	NS
After	7.2±1.2	8.4±1.9	
Difference	−2.0	1.5	.009
P†	.04	NS	
Cholesterol (mg/dL)			
Before	207±8	194±8	NS
After	184±7	191±7	
Difference	−23	−3	.03
P†	<.001	NS	
HDL-C (mg/dL)			
Before	43±3	51±4	NS
After	48±2	52±16	
Difference	5	1	NS
P†	.004	NS	
LDL-C (mg/dL)			
Before	120±8	108±9	NS
After	106±8	102±9	
Difference	−14	−6	NS
P†	.001	NS	
TC (mg/dL)			
Before	229±25	146±14	.01
After	150±15	145±15	
Difference	−79	−1	.002
P†	.001	NS	
SBP (mmHg)			
Before	138±3	141±5	NS
After	119±3	121±3	
Difference	−19	−20	NS
P†	<.001	.002	
DBP (mmHg)			
Before	84±2	87±2	NS
After	76±2	74±2	
Difference	−8	−13	NS
P†	<.001	<.001	

NOTE. Values are mean ± SE.
Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure.
*Difference between groups at baseline and the difference in changes after 4 months of ST or ET.
†Difference in each group before and after 4 months of strength or endurance training.

DISCUSSION

We found significant improvements in long-term glycemic control, as shown by reduced Hb A_{1c} levels and an improved IR estimated by HOMA, in participants with diabetes on ST. The effects of ET on the respective parameters, however, were only moderate.

Maximum strength (1-RM) of all muscle groups increased after 4 months of ST in contrast with no improvements after 4 months of ET, with the exception of a small increase in leg press. Additional improvements were observed in Vo_2peak after ET, whereas no such changes were seen in ST. The latter findings were predictable from the specificity of the training

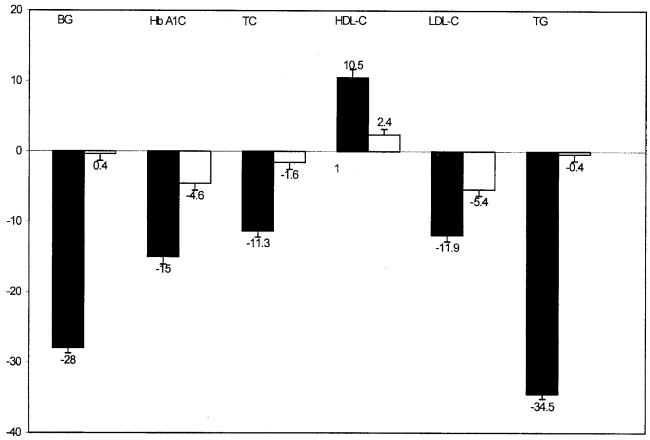

Fig 1. Percentage change in metabolic parameters after 4 months of ST (black) or ET (white). Whiskers represent standard deviation.

stimulus and show that the training was adequate in both groups. It is therefore evident that the specific training stimulus was sufficient for both training groups.

Moreover, significant improvements in long-term glycemic control, as determined by reduced Hb A_{1c} and improved IR, as estimated by HOMA, were associated with ST, whereas the effects of ET were only moderate. Two recent studies by Dunstan[10] and Castaneda[9] and colleagues support these benefits of ST on glycemic control.

Both training programs had very positive effects on blood pressure. We showed that ST was associated with a significant improvement in IR, with a concomitant increase in muscle mass and muscle strength as well as decrease in body fat mass. The strong association observed between muscle size and glycemic control support the importance of muscle tissue in IR in T2D. We also found improvements in the atherogenic lipid profile after 4 months of ST, whereas the effects of ET on metabolic parameters were only moderate. In the ST group, we observed a significant reduction of TG, TC, and LDL-C and a significant increase in HDL-C levels. Importantly, these observations were made in the presumed absence of dietary changes during the training period. The positive alteration in the lipid profiles of our participants, therefore, must be largely due to the changes in body composition as a result of ST.

Table 3: Physical Characteristics of Participants with T2D Before and After ST or ET

Measures	ST	ET
BMI (kg/m^2)		
Before	31.3±0.9	33.9±1.3
After	30.9±0.9	33.5±1.3
Difference %	−1.1	−1.1
Body weight (kg)		
Before	91.3±2.9	96.7±4.5
After	90.2±2.8	95.4±4.5
Difference %	−1.1	−1.1
LBM (kg)		
Before	49.4±1.8	51.9±2.5
After	52.6±1.7*	52.9±2.7
Difference %	+6.5	+2
Percentage body fat (%)		
Before	44.5±0.8	46.3±0.8
After	40.5±1.1*	44.5±0.8*
Difference %	−9.1	−3.4
Fat mass (kg)		
Before	39.6±1.4	44.8±2.3
After	35.8±1.7*	42.5±2.1*
Difference %	−9.7	−5.3

NOTE. Values are mean ± SEM.
Abbreviation: Difference %, difference in percentage before and after 4 months of ST or ET.
*Significant difference of $P<.001$ in each group before and after training.

Table 4: Muscle Strength and Cardiorespiratory Endurance Before and After ST or ET

Measures	ST	ET
Vo_2peak (mL·kg^{-1}·min^{-1})		
Before	20.71±1.1	16.33±1.1
After	20.95±1.4	17.82±1.2
Difference %	+1	+8
Bench press (kg)		
Before	52.3±3.1	40.4±3.8
After	67.4±4.0	40.2±3.5
Difference %	+29†	0
Leg press (kg)		
Before	113.6±7.8	93.2±8.7
After	167.9±9.7	107.2±10.2
Difference %	+48†	+15†
Pull sitting (kg)		
Before	56.9±3.6	47.4±3.8
After	69.7±4.0	49.7±4.2
Difference %	+22†	+5*
Heart rate max (bpm)		
Before	149±4	145±4
After	155±4	146±4
Difference %	+4	+1
Performance (W)		
Before	132±9	106±9
After	147±10	118±10
Difference %	+12†	+12*

NOTE. Values are mean ± SEM.
Abbreviations: Heart rate max; maximum heart rate using a cycle ergometer; Performance, maximum workload using a cycle ergometer.
*Significant difference of $P<.01$ in each group before and after training.
†Significant difference of $P<.001$ in each group before and after training.

The effectiveness of ET was shown by a 12% improvement in cardiorespiratory endurance and an 8% increase in Vo_2peak and an improvement of 15% in maximum strength in leg press. The changes in leg press in the ET group were because of increased intramuscular synchronism and an improvement in movement coordination. In contrast to ST, ET resulted in only moderate changes in both glycemic control (4.5% reduction in Hb A_{1c}, 1.53% increase in IR), and lipid profile (5.4% decrease in LDL, 2.4% increase in HDL, TG values did not change from baseline). The changes in the lipid profile are in good agreement with those reported by Ligtenberg et al,[26] but are less pronounced than those reported by Mosher[27] and Campaigne[28] and colleagues, who found a 14% decrease of LDL accompanied by moderate, but not statistically significant, improvements in long-term glycemic control after 3 months of ET in patients with T2D.

The decrease in blood pressure observed with ET in T2D patients[29] and non-T2D patients[30,31] is a well-recognized phenomenon. Of the few articles available on ST, 2[11,12] reported

no beneficial changes in blood pressure as a result of ST, but this was possibly because of the short-term nature of the programs. Positive effects on blood pressure, however, have been reported in 2 other studies on ST,[9,10] in which the programs were 4 or 6 months in duration. In both studies, significant reductions in SBP and DBP ($P<.05$) were observed.

Much of the caution surrounding ST is based on the reported acute elevation in blood pressure caused by this form of exercise.[32] However, we found no extreme increases of blood pressure during or after training and we support the findings of earlier investigations,[33,34] that high-intensity ST does not negatively affect blood pressure. Furthermore, there were no recorded hypoglycemic episodes in either the ST or ET groups during or after training periods.

A limitation to this study is that participants randomized to the ST group had higher baseline levels for FBG and TG than did patients randomized to the ET group. It must be mentioned that values that are high and beyond the normative physiologic range can be reduced more easily than can values that lie closer to the normative range. Although FBG and TG in the ST group were higher and beyond the physiologic range at study entry, after the 4-month training period the values in the ST group were closer or nearly equal to normative values than in the ET group. Because ET has been advocated as the most suitable exercise mode, with many positive metabolic effects in T2D patients, and because we wanted to compare the effects of ST with the most effective exercise mode, we used ET as the control group. We also wanted to compare the effects of ST with a second control group—a group without training—but our Ethics Committee had reservations about that group because exercise is known to improve health in these patients and the committee felt that it would be unethical not to recommend exercise. A second limitation is the use of HOMA for determination of IR. We did not use the criterion standard in the assessment of insulin sensitivity, that is, the glucose clamp[35]; however, other investigators have reported that HOMA-IR is strongly related to clamp-measured IR in diabetic subjects.[36] All patients were training naive, which meant that we had to start with low intensity and low volume. But then we increased ET intervention so that it matched the ST intervention (3 sets at beginning, advancing to a maximum of 6 sets). The 8% improvement in cardiorespiratory endurance is comparable to the 8% increase reported by Campaigne et al[28] after a 3-month aerobic training program in patients with T2D. Unlike some other studies, in which increases in Vo_2peak of up to 27% after different aerobic training programs could be measured,[37,38] cardiorespiratory endurance in our patients was not significantly lower than that measured by Lynch[39] (10%) or Ross[40] (8% in women) and colleagues in patients without T2D. However, absolute levels remained lower posttraining in subjects with T2D than those observed in sedentary patients without T2D undergoing similar training programs.[29,41]

This may add to the difficulty in prescribing an exercise training program in this population. The results after ST, with an increase of 30% to 50% in muscle strength, are similar to the results of Hurley et al.[42] A 4-month trial on Nautilus exercise machines 3 to 4 time a week with 11 untrained men resulted in a significant 30% to 50% increase in muscle strength but did not result in a significant change in Vo_2peak.

Nevertheless, whether there are changes in glycemic and metabolic control, all favorable changes observed after ST support the usefulness of ST in the treatment of T2D. With the added benefit of protection against the development of cardiovascular diseases observed after ST, we consider ST to be equal to ET in the treatment of T2D.

CONCLUSIONS

ST was better than ET in all metabolic parameters measured in regard to an improvement in metabolic parameters. The positive changes observed in the muscular system coincided with highly significant improvements in metabolic control that resulted in a decreased atherogenic lipid profile. With the advantage of an improved lipid profile, we recommend ST for the treatment of T2D.

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Suppliers

a. Continuous heart rate monitoring; Polar Electro Oy, Professorintie 5, FIN-90440 Kempele, Finland.
b. Concept 2 Ltd, Vermont House, Unit 5 Nottingham South & Wilford Ind. Est, Ruddington Ln, Wilford, Notts, NG11 7HQ, UK.
c. Ergo-metrics 900; Ergoline GmbH, Köhlershohner Str, D-53578 Windhagen, Germany.
d. SensorMedics Corp, 22705 Savi Ranch Pkwy, Yorba Linda, CA 92887-4645.
e. Seca Deutschland, Medizinische Waagen und Messysteme, Postfach 76 11 80, Hamburg 22061, Germany.
f. Model Caliper GMP; Siber Hegner Maschinen AG, Zürich, Switzerland.
g. Roche Diagnostics, Sandhoferstrasse 116, DE-68305, Mannheim, Germany.
h. SPSS Inc, 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.