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Aerobic exercise training at maximal fat oxidation intensity improves body composition, glycemic control, and physical capacity in older people with type 2 diabetes



Yan Jiang ^a, Sijie Tan ^a, Zhaoyu Wang ^a, Zhen Guo ^a, Qingwen Li ^a, Jianxiong Wang ^{b, *}

^a Tianjin Physical Fitness Research Center, Department of Health and Exercise Science, Tianjin University of Sport, China ^b Faculty of Health, Engineering, and Sciences, University of Southern Queensland, Australia

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ABSTRACT

Background: Aerobic training has been used as one of the common treatments for type 2 diabetes; however, further research on the individualized exercise program with the optimal intensity is still necessary. The purpose of this study was to investigate the effects of supervised exercise training at the maximal fat oxidation (FATmax) intensity on body composition, glycemic control, lipid profile, and physical capacity in older people with type 2 diabetes.

Methods: Twenty-four women and 25 men with type 2 diabetes, aged 60–69 years. The exercise groups trained at the individualized FATmax intensity for 1 h/day for 3 days/week over 16 weeks. No dietary intervention was introduced during the experimental period. Whole body fat, abdominal fat, oral glucose tolerance test, lipid profile, and physical capacity were measured before and after the interventions.

Results: FATmax intensity was at $41.3 \pm 3.2\%$ VO₂max for women and $46.1 \pm 10.3\%$ VO₂max for men. Exercise groups obtained significant improvements in body composition, with a special decrease in abdominal obesity; decreased resting blood glucose concentration and HbA1c; and increased VO₂max, walking ability, and lower body strength, compared to the non-exercising controls. Daily energy intake and medication remained unchanged for all participants during the experimental period.

Conclusion: Beside the improvements in the laboratory variables, the individualized FATmax training can also benefit daily physical capacity of older people with type 2 diabetes.

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Introduction

Type 2 diabetes is a chronic disease which would lead to blindness, kidney failure, heart attacks, stroke, and lower limb amputation, if not treated properly.¹ Currently main treatments of type 2 diabetes include dietary management,² physical exercise,^{3,4} medicine,⁵ and bariatric surgery,⁶ with the major purposes of controlling blood glucose and decreasing body fat.⁷ Among these treatments, we focused on aerobic exercise treatment for type 2 diabetes in the present study. American Diabetes Association recommends the patients to engage in walking or cycling at least 150 min per week at moderate to vigorous intensity.³ The

* Corresponding author. Faculty of Health, Engineering, and Sciences, University of Southern Queensland, Toowoomba, QLD, 4350, Australia.

E-mail address: wangj@usq.edu.au (J. Wang).

commonly used exercise intensities are at 50%–70% maximal oxygen uptake (VO₂max) or at a heart rate (HR) of 110–140 bpm.⁸ However, these intensity ranges are too broad to determine a particular exercise intensity for individual patients. Therefore, further research on the most appropriate exercise program with the optimal intensity, mode, duration, and frequency is still necessary.⁴

During aerobic exercise with a gradually increased exercise intensity, fat oxidation rate will increase to the peak at a certain intensity and then begins to decline; and from this intensity onwards, carbohydrate oxidation takes precedent.⁹ The exercise intensity at the peak fat oxidation has been defined as the maximal fat oxidation rate (FATmax).¹⁰ FATmax is a low-to-moderate exercise intensity. Our team has reported that women with type 2 diabetes (63 ± 2 yrs) reach their FATmax at 37% VO₂max¹¹; while another study has shown that the FATmax intensity was at 34% VO₂max in women with type 2 diabetes (58 ± 7 yrs).¹² The studies of FATmax in older men with type 2 diabetes are scarce up to now. FATmax

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exercise training would be suitable to older people with type 2 diabetes. Firstly, aerobic exercise at a low-to-moderate intensity would be much easier to do and safer for older people.¹³ Secondly, deduced from the definition of FATmax, theoretically exercise at FATmax intensity would burn much more fat as energy, compared with other aerobic intensities ranges. In the literature, there are few studies of physical training at FATmax exercise intensity in people with type 2 diabetes.¹¹ In the present study, we employed a FATmax training program to investigate its effects on body fat, glycemic control, and physical capacity in daily life as a special focus in both older women and men with type 2 diabetes. The hypothesis was that FATmax training would improve the measured variables of the participants, compared to those of the non-exercising control group's responses.

Methods

Participants

The participants were 25 women and 29 men with type 2 diabetes, aged 60-69 years. They were recruited via local medical practitioners, following the diagnostic criteria: 1) a fasting plasma glucose concentration of 7.0 mmol/L or higher; or 2) a plasma glucose concentration of 11.1 mmol/L or higher two hours after a 75-g oral glucose tolerance test (OGTT).¹⁴ The history of their type 2 diabetes was 6-11 years. Exclusion criteria were heart disease, uncontrolled hypertension, pulmonary diseases, impaired renal or liver function, or inability to participate in exercise training due to orthopedic or neurological limitations. All participants took oral hypoglycemic drugs (metformin and/or sulfonylureas), but none of them were being treated with insulin. Other medicines included antihypertensive agents [ACE inhibitor, diuretic, or others. Fourteen (56%) participants from the exercise groups and 15 (63%) from the control groups] and lipid-lowering agents [statin, fibrate, or others. 7 (28%) from the exercise groups and 10 (42%) from the control groups]. Medicines and respective doses were not changed during the 16-week intervention. All female participants were postmenopausal. All participants were physically inactive (<60 min/ week of exercise) prior to the intervention. Before the baseline tests, the exact details of the study were explained to the participants and a written informed consent to the study was obtained from each of them. All methods and procedures of the present study were approved by the Ethics Committee of Tianjin University of Sport, China.

Study design

All participants underwent a 2-day baseline examination that included medical history, body composition, FATmax, glycated hemoglobin (HbA1c), OGTT, and lipid profile in Day 1; while the measurements of predicted VO₂max and physical capacity in Day 2. The participants were instructed to abstain from coffee and alcohol for 48 h before the tests. Then, the participants were allocated to the exercise group or control group. A third party who was not involved in the present study arranged participants' family names in an alphabetical order. Odd numbered participants were allocated to the exercise group and even numbered participants to the control group. Women and men were organised separately. Exercise groups took part in supervised exercise training at the individualized FATmax intensity. Control participants were required to maintain their habitual physical activity. Body composition, blood variables, predicted VO₂max, and physical capacity were measured again at the end of the experiments. No diet intervention was introduced during the experimental period for all participants.

Body composition

Each participant's body mass and height was measured (Omron ultrasonic weight METER HNH-219, Dalian, China) to calculate body mass index (BMI). Waist circumference was measured at the level of the umbilicus horizontally without clothing. Hip circumference was measured at the level of the greatest protrusion of the gluteal muscles with underwear. Waist-hip ratio was calculated. After overnight fast, body composition was measured using a dualenergy X-ray absorptiometry (DXA) (Prodigy Advance, GE Healthcare Lunar, USA). Total body fat (%) was determined as a portion of the total amount of fat in the entire body mass. Fat mass and fatfree mass were also calculated. To assess abdominal obesity, the abdominal subcutaneous and visceral fat areas at the umbilical level were estimated by a bioelectrical impedance analysis equipment (ORMRN, HDS-2000 DUALSCAN^R, OMRON Healthcare Co., Kyoto, Japan).

FATmax measurement

FATmax of each participant in the exercise groups was measured at baseline. The participants refrained from any vigorous physical activity 24 h and fasted for at least 10 h before the test, which was carried out in the morning between 8:00-10:00 to avoid circadian variance. A graded treadmill walking-running protocol, same for women and men, was used to measure the FATmax.¹¹ Briefly, there was a warm-up walking at 2.5 km/h with an incline of 1% for 3 min. Participants then stepped off the treadmill to do 3 min of static stretching and had a 2-minute sitting rest. The first stage of exercise was set at a speed of 3.5 km/h with an incline of 1% for 3 min. The speed was increased to 4.0 km/h for 3 min as the second stage, 4.5 km/h for 3 min as the third stage, and 5.0 km/h for 3 min as the fourth stage before the respiratory exchange ratio (RER) reached 1.0. Oxygen uptake (VO_2) and carbon dioxide production (VCO_2) were measured by using an open-circuit indirect gas analyser (Cortex Metalyzer 3B Gas Analyser, CORTEX Biophysik GmbH, Germany), which was calibrated with the standard gas before each test. Average gas values were recorded every 15 s during the exercise test until the RER reached 1.0, at which point the test was terminated. Fat oxidation rate was calculated following the stoichiometric equation with the assumption that the urinary nitrogen excretion rate was negligible:

Total fat oxidation = $1.67 \times VO_2 - 1.67 \times VCO_2$

with VO₂ and VCO₂ in litres per minute. The value 1.67 was derived from the volume of VO₂ and VCO₂ in oxidation of 1 gram of fat.¹⁵ The exercise intensity at which the highest rate occurred was defined as the FATmax.¹⁰ HR was recorded continuously during the test by an electrocardiogram. The corresponding HR at the FATmax intensity (FATmax HR)¹⁶ was recorded and then applied individually to control the intensity of the following exercise training.

Estimated maximal oxygen uptake

 VO_2 max was estimated by using a submaximal exercise test on a bicycle ergometer and VO_2 was measured using the same gas analyser as in the FATmax test. The HR and the VO_2 at the workload of 25 and 75 W were measured to develop the best-fit line of HR and VO_2 for each participant. VO_2 max was then predicted from the estimated maximal HR (220-age).¹⁷

Blood tests

A 6-ml fasting blood sample was collected to measure lipid

profile and HbA1c. The concentrations of triglyceride (TG), total cholesterol (TC), low-density lipoprotein-cholesterol (LDL-C), and high-density lipoprotein-cholesterol (HDL-C) were measured using an Automatic Biochemical Analyser Hitachi 7800 (Hitachi High-Technologies Corporation, Tokyo, Japan). HbA1c was measured by an enzymatic method (Bio-Rad Laboratories, Hercules, CA, USA). The OGTT was conducted after overnight fasting. The participant drank 75 g of glucose dissolved in 200 ml of water. The Roche Cobas C501 Chemistry Analyser was used to measure plasma glucose (hexokinase method) and insulin (radioimmunoassay) in the blood samples taken at baseline and two hours later. The reagent kits for glucose and insulin were purchased from the Roche Diagnostics Shanghai Limited (Shanghai, China). Insulin resistance was estimated by the homeostasis model assessment (HOMA-IR) = fasting insulin (μ U/mL) × fasting plasma glucose (mmol/L)/22.5.¹⁸

Physical capacity tests

Six tests were performed, in a random order, to measure lower body strength (30-s chair stand, the stand-up can be completed in one minute), upper body strength (30-s arm curl, women 5 lb and men 8 lb, the arm curl can be done in one minute), aerobic endurance (6-min walk, the distance covered), lower body flexibility (chair sit-and-reach, measure the distance between the figures to tip of toe), upper body flexibility (back scratch, measure the distance between the extended middle figures), and ability/dynamic balance (8-foot up-and go, record the time required to get up from seated position, walk 8 foot, turn, and return to seated position on the chair).¹⁹

Exercise training program

Exercise groups undertook 16 weeks of FATmax training, 1 h per day for 3 times per week. The training session consisted of a 10minute warm-up period which involved skeletal muscle stretches and joint movement. This was followed by walking or running with the intensity controlled at the individualized FATmax HR. The exercise duration was increased gradually: 20-25 min in week 1; 30-35 min in weeks 2-5, and 45 min in weeks 6-16. Short breaks of 1-2 min were allowed when it was necessary. Finally, there was a 5-minute cool-down period comprised of slow walking. All training sessions were supervised by the researchers and every participant wore a HR monitor (Polar Electro, Finland) during the training. An alarm on the HR monitor was set up at ± 5 beats of the FATmax HR to judge the moving speed. If the participant showed a sign of hypoglycemia, such as hunger, trembling, heart racing, or nausea, during training, exercise was stop immediately.

Dietary records

All participants were required to record a five-weekday dietary diary at the beginning and the end of the experimental period. The weight of the food and percentages of carbohydrate, fat, and protein in the food were estimated from the records. Daily energy intake was then calculated by multiplying the proportion of carbohydrate, fat, and protein consumed with their respective energy values (carbohydrate provides 4 kcal/g of energy, fat 9 kcal/g, and protein 4 kcal/g). After removing the highest and lowest values, the average of the other three days was reported as the daily energy intake.¹¹

Data analyses

All values were presented as $Mean \pm SD$. The independent samples *t*-test was run at baseline to compare possible differences

in measured variables between the exercise and control groups. Effects of FATmax training on the measured variables were detected using 2 times (before and after experiment) x 2 groups (exercise and control) factorial design, split plot analysis of variance (SPA-NOVA). The effect size (ES) of measured variables following intervention was calculated.²⁰ Within-group comparisons of interventional period were evaluated using the paired-samples *t*-test. Based on the data of body fat% change after the FATmax exercise training in diabetes patients from a previous study,¹¹ the sample size of the exercise group should be n = 10 when alpha = 0.05 for a two-tailed test and power = 0.80 (G*Power 3.1.9.4 statistical power analysis program). A p < 0.05 was regarded as statistically significant. All analyses were performed using the SPSS Version 22 for Windows (SPSS Inc. USA).

Results

Twenty-five participants of the exercise groups (11 women and 14 man) completed the 16-week FATmax training program. Due to time constraints, two participants from the exercise groups (one woman and one man) and three from the control groups (three men) discontinued the study. Therefore, the data of 25 participants of the exercise groups and 24 of the control groups (13 women and 11 men) were reported. In the trained women, the FATmax was 0.30 ± 0.07 g/min (range 0.22-0.44 g/min), occurring at the VO₂ of 12.21 ± 0.94 ml/min/kg and at $41.34 \pm 3.15\%$ VO₂max (range 34.55-44.98% VO2max). Estimated VO2max was 27.71 ± 4.14 ml/ min/kg. The average FATmax HR was 96 ± 12 bpm. In the trained men, the FATmax was 0.38 ± 0.12 g/min (range 0.27-0.60 g/min), occurring at the VO₂ of 13.99 ± 2.15 ml/min/kg and at 46.05 ± 10.34% VO₂max (range 31.16-67.20% VO₂max). Estimated VO_2max was 29.04 ± 5.18 ml/min/kg. The average FATmax HR was 95 ± 12 bpm.

There were no physical injuries or hypoglycemic events caused by the exercise training. Dietary records did not show significant changes before and after the experimental period within and between the exercise and control groups. Combined together, the average daily energy intake of female participants was 2145 ± 89 kcal/day at baseline and 2197 ± 94 kcal/day at the end of the study (p > 0.05); the data for male participants were 2345 ± 105 kcal/day and 2318 ± 96 kcal/day, respectively (p > 0.05).

There was no difference in the variables of body composition between the groups of both women and men at baseline. In female participants, the exercise group significantly decreased body mass, BMI, body fat%, and fat mass; while the control group increased body mass, BMI, abdominal subcutaneous fat area, and hip circumstance (Table 1). In the men's groups, the exercise group significantly decreased body mass, BMI, body fat%, fat mass, abdominal fat areas, waist circumference, and waist-to-hip ratio; while the control group had a significant increase in abdominal visceral fat area following the intervention (Table 2).

There was no difference in the blood variables between the groups of both women and men at baseline. For women, the exercise group significantly decreased their HbA1c and fasting blood glucose concentration and increased HDL-C concentration; while the control group increased their LDL-C concentration after 16 weeks of interventions (Table 3). In the men's groups, the exercise group obtained significant decreases in HbA1c, resting and 2-hr post-challenge blood glucose concentration; while increased their HDL-c concentration. The decrease in insulin resistance of the exercise group was very close to statistical significance (p = 0.052). There was no significant change in the blood variables of the control group (Table 4).

Both trained women and men significantly increased their

Table 1

Age and body composition variables of the participants (Women).

Variables	Exercise gro	pup(n = 11)		Control grou	P for time x				
	Before	After	P for within-group comparison	Effect size	Before	After	P for within-group comparison	Effect size	group
Age (years)	63.9 ± 6.1				62.6 ± 3.8			_	
Body height (cm)	156.2 ± 6.7	156.1 ± 6.7	0.053	0.01	157.5 ± 6.4	157.4 ± 6.3	0.104	0.02	0.591
Body mass (kg)	64.9 ± 7.7	$62.8 \pm 7.4^{*}$	<0.05	0.28	65.9 ± 5.6	$67.4 \pm 5.8^{*}$	<0.05	0.26	<0.01
BMI (kg/m ²)	26.6 ± 2.2	$25.8 \pm 2.1^{*}$	<0.05	0.37	26.7 ± 3.2	$27.3 \pm 3.0^{*}$	<0.05	0.19	<0.01
Fat%	34.2 ± 8.3	$31.4 \pm 7.4^{*}$	< 0.05	0.36	35.9 ± 6.2	36.2 ± 6.8	0.687	0.05	<0.05
Fat mass (kg)	22.4 ± 7.6	19.9 ± 5.7*	< 0.05	0.42	23.7 ± 5.0	24.4 ± 5.5	0.230	0.13	<0.01
Fat-free mass (kg)	42.5 ± 5.4	42.9 ± 5.6	0.379	0.07	42.2 ± 5.1	43.0 ± 5.7	0.320	0.15	0.764
Abdominal subcutaneous fat area (cm ²)	209.9 ± 58.3	3 192.7 ± 53.7	7 0.057	0.31	212.5 ± 46.4	220.8 ± 49.1*	<0.05	0.17	<0.01
Abdominal visceral fat area (cm ²)	85.6 ± 40.2	75.0 ± 27.6	0.081	0.30	88.2 ± 23.8	91.5 ± 21.0	0.321	0.15	<0.05
Waist circumstance (cm)	86.0 ± 13.0	81.9 ± 9.4	0.106	0.36	87.4 ± 8.9	91.9 ± 9.4	0.115	0.49	<0.05
Hip circumstance (cm)	99.9 ± 7.6	99.8 ± 8.4	0.979	0.01	97.8 ± 8.2	$100.2 \pm 8.2^{**}$	<0.01	0.29	<0.05
Waist-hip ratio	0.86 ± 0.09	0.82 ± 0.07	0.194	0.50	0.89 ± 0.07	0.92 ± 0.07	0.340	0.31	0.120

Note: All data are presented in mean \pm SD. BMI, body mass index. *p < 0.05; **p < 0.01.

Table 2

Age and body composition variables of the participants (Men).

Variables	Exercise group $(n = 14)$				Control grou	P for time x			
	Before	After	P for within-group comparison	Effect size	Before	After	P for within-group comparison	Effect size	group
Age (years)	63.9 ± 6.1				62.6 ± 3.8				
Body height (cm)	169.1 ± 6.2	169.0 ± 6.2	0.336	0.02	170.8 ± 4.7	170.6 ± 5.0	0.113	0.04	0.296
Body mass (kg)	76.8 ± 7.9	73.5 ± 7.1***	<0.001	0.44	77.3 ± 7.1	78.5 ± 8.4	0.171	0.15	<0.001
BMI (kg/m ²)	26.9 ± 2.1	25.7 ± 1.9***	<0.001	0.60	26.5 ± 2.1	27.0 ± 2.7	0.120	0.21	<0.001
Fat%	34.6 ± 4.6	31.2 ± 5.1**	<0.01	0.70	34.5 ± 6.4	35.7 ± 6.4	0.184	0.19	<0.01
Fat mass (kg)	26.7 ± 5.0	$23.0 \pm 4.7^{*}$	<0.001	0.76	26.8 ± 6.3	28.1 ± 6.0	0.099	0.21	<0.001
Fat-free mass (kg)	50.1 ± 5.0	50.5 ± 5.6	0.681	0.08	50.5 ± 5.4	50.4 ± 6.8	0.905	0.02	0.713
Abdominal subcutaneous fat area (cm ²)	214.4 ± 39.7	' 192.7 ± 40.6**	<0.01	0.54	213.7 ± 69.4	220.5 ± 68.1	0.090	0.10	<0.01
Abdominal visceral fat area (cm ²)	106.2 ± 33.5	91.8 ± 29.6	<0.05	0.46	103.9 ± 44.8	112.4 ± 47.1	<0.01	0.18	<0.01
Waist circumstance (cm)	96.3 ± 7.8	91.4 ± 7.9***	<0.001	0.62	93.9 ± 11.8	93.6 ± 9.4	0.807	0.03	<0.01
Hip circumstance (cm)	100.4 ± 4.6	100.0 ± 5.7	0.654	0.08	99.0 ± 6.0	100.2 ± 7.7	0.620	0.17	0.492
Waist-hip ratio	0.96 ± 0.06	0.91 ± 0.06***	<0.001	0.83	0.95 ± 0.08	0.94 ± 0.08	0.610	0.13	0.155

Note: All data are presented in mean \pm SD. BMI, body mass index. *p < 0.05; **p < 0.01; ***p < 0.001.

Table 3

Blood variables of the participants (Women).

Variables	Exercise grou	n = 11		Control group					
	Before	After	P for within-group comparison	Effect size	Before	After	P for within-group comparison	Effect size	P for time x group
HbA1c (%) Blood glucose (mr	6.90 ± 0.60 nol/L)	6.21 ± 0.64**	<0.01	1.11	6.84 ± 0.77	6.92 ± 0.73	0.597	0.11	<0.01
Fasting	8.57 ± 1.53	$7.98 \pm 1.40^{*}$	<0.05	0.48	8.57 ± 1.42	8.79 ± 1.25	0.316	0.16	< 0.01
2-hr post- challenge	12.75 ± 5.56	12.78 ± 6.45	0.979	0.01	11.90 ± 5.49	12.64 ± 5.31	0.296	0.14	0.520
Blood insulin (µU/	ml)								
Fasting	16.85 ± 9.64	16.15 ± 7.75	0.541	0.08	16.44 ± 6.17	16.89 ± 8.10	0.780	0.06	0.570
2-hr post-	45.10 ± 21.31	39.30 ± 19.55	0.351	0.28	46.94 ± 25.74	49.34 ± 28.99	0.552	0.09	0.210
challenge									
Insulin resistance	6.60 ± 4.15	5.75 ± 2.82	0.168	0.24	6.33 ± 2.64	6.67 ± 3.43	0.665	0.11	0.241
TG (mmol/l)	3.54 ± 1.24	2.99 ± 1.62	0.098	0.38	3.56 ± 0.97	3.42 ± 1.13	0.596	0.13	0.295
TC (mmol/l)	5.27 ± 0.65	5.47 ± 0.93	0.274	0.25	5.24 ± 0.99	5.43 ± 1.01	0.542	0.19	0.959
HDL-C (mmol/l)	1.60 ± 0.34	1.93 ± 0.32**	<0.01	1.00	1.56 ± 0.40	1.54 ± 0.29	0.802	0.06	<0.05
LDL-C (mmol/l)	3.27 ± 0.65	3.21 ± 0.81	0.748	0.08	3.30 ± 0.74	$3.68 \pm 0.60^{*}$	<0.05	0.56	0.067

Note: All data are presented in mean ± SD. HbA1c, glycated hemoglobin. TG, triglyceride. TC, total cholesterol. HDL-C, high-density lipoprotein-cholesterol. LDL-C, low-density lipoprotein-cholesterol. *p < 0.05; **p < 0.01.

Table 4	
Blood variables of the participants	(Men).

Variables	Exercise grou	n = 14			Control group	Control group $(n = 11)$				
	Before	After	P for within-group comparison	Effect size	Before	After	P for within-group comparison	Effect size	group	
HbA1c (%)	6.59 ± 0.57	6.21 ± 0.62**	<0.01	0.64	6.58 ± 0.88	6.62 ± 0.84	0.731	0.05	<0.01	
Blood glucose (n	nmol/L)									
Fasting	8.60 ± 0.99	7.73 ± 1.18*	<0.05	0.80	8.53 ± 1.20	8.96 ± 1.18	0.127	0.36	<0.01	
2-hr post- challenge	10.97 ± 3.46	8.66 ± 3.22*	<0.05	0.69	10.76 ± 3.17	11.75 ± 3.77	0.174	0.28	<0.05	
Blood insulin (µl	J/ml)									
Fasting	12.60 ± 6.00	11.28 ± 6.50	0.418	0.21	12.26 ± 8.00	11.61 ± 7.28	0.475	0.08	0.736	
2-hr post-	42.45 ± 30.63	30.99 ± 21.64	< 0.05	0.43	41.08 ± 14.67	47.80 ± 16.79	0.062	0.43	<0.01	
challenge										
Insulin resistance	e 4.77 ± 2.12	3.82 ± 2.03	0.052	0.46	4.80 ± 3.35	4.70 ± 3.10	0.817	0.03	0.195	
TG (mmol/l)	3.61 ± 1.16	3.13 ± 1.09	0.108	0.43	3.54 ± 1.31	3.42 ± 1.18	0.460	0.10	0.307	
TC (mmol/l)	5.22 ± 1.03	5.23 ± 0.87	0.995	0.01	5.25 ± 0.86	5.37 ± 1.16	0.644	0.12	0.717	
HDL-C (mmol/l)	1.46 ± 0.41	$1.84 \pm 0.46^{*}$	<0.05	0.87	1.49 ± 0.38	1.52 ± 0.43	0.733	0.07	0.081	
LDL-C (mmol/l)	3.19 ± 0.81	2.99 ± 0.73	0.174	0.26	3.17 ± 0.71	3.10 ± 0.46	0.789	0.12	0.652	

Note: All data are presented in mean ± SD. HbA1c, glycated hemoglobin. TG, triglyceride. TC, total cholesterol. HDL-C, high-density lipoprotein-cholesterol. LDL-C, low-density lipoprotein-cholesterol. *p < 0.05; **p < 0.01.

VO₂max, times of 30s chair stand, and 6-min walk distance after the 16-week FATmax training; while no change in these variables of the control groups. There was no change in the times of 30s arm curl, chair sit-and-reach performance, the time used to complete the 8-foot up-and go test for both trained women and men (Tables 5 and 6). In addition, the trained women improved their back scratch result (p < 0.05); while the controlled women got a worse result from their chair sit-and-reach test (p < 0.05) (Table 5).

Discussion

Main findings were that the 16-week FATmax training positively changed body composition; improved glycemic control; and increased VO₂max and physical capacity in the trained women and men. There were no favourable changes in these variables in the control groups. Daily energy intakes and medication and dosages remained unchanged for all participants during the experimental period. Therefore, the overall outcome supports the hypothesis that FATmax training is an effectively adjuvant therapy for managing type 2 diabetes in older people.

The 16-week FATmax training achieved significant changes in body composition of both women and men; especially, the significant decrease in abdominal obesity. Known as the 'sick fat',²¹ the fat depot around abdomen actively releases non-esterified fatty acid, cytokines, and adipokines, which can cause systemic inflammation and metabolic diseases.^{22,23} Related to the development of type 2 diabetes, there is an association between abdominal fat and insulin resistance in middle-aged people.²⁴ Aerobic training at other intensities, for example, 60–75% maximal HR training

decreased abdominal subcutaneous fat area²⁵ and 60–65% of the reserve HR training reduced visceral and subcutaneous adipose tissue in type 2 diabetes patients.²⁶ Referred to the FATmax training studies specially, our team has found the decrease in abdominal fat mass or area in overweight middle-aged women and older women with type 2 diabetes following 10–12 weeks of FATmax training.^{11,16} The present study provides further evidence that FATmax is an effective exercise intensity to decrease abdominal obesity, as well as whole body fat variables, for both older women and older men with type 2 diabetes.

The OGTT results showed significant decreases in resting blood glucose for both trained women and men, as well as the 2-hr postchallenge blood glucose for trained men only. The present study consolidated the decrease in resting blood glucose of older women with type 2 diabetes following FATmax training¹¹; and expanded the results to older men with type 2 diabetes. The decrease in blood glucose concentrations is in agreement with previous aerobic exercise studies in people with type 2 diabetes.^{27,28} HbA1c shows the average blood glucose level over the previous two to three months.²⁹ Both trained women and men decreased their HbA1c after the 16-week FATmax training. The results from trained women supported a recent FATmax training study.¹¹ Other aerobic training studies have also reported decreased HbA1c in type 2 diabetes patients^{25,30}; however, not all aerobic exercise studies reported significant change in this variable.³¹ Taken together, the decreased blood glucose concentrations and HbA1c indicate that FATmax is an effective exercise intensity to benefit glycemic control in older people with type 2 diabetes.

Another positive change in the blood tests was the increased

Table	5
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Maximal oxygen uptake and fitness capacity of the participants (Women).

Variables	Exercise group $(n = 11)$			Control g	Control group $(n = 13)$				
	Before	After	P for within-group comparison	Effect size	Before	After	P for within-group comparison	Effect size	P for time x group
Maximal oxygen uptake (ml/ min/kg)	27.7 ± 4.1	31.1 ± 5.2	<0.05	0.72	28.0 ± 4.0	27.5 ± 4.1	0.683	0.13	<0.05
30s arm curl with 5 Ib (times)	21 ± 3	24 ± 4	0.104	0.85	22 ± 5	23 ± 4	0.816	0.22	0.189
30s chair stand (times)	19 ± 5	$23 \pm 4^{***}$	<0.001	0.88	20 ± 3	19 ± 4	0.329	0.28	<0.001
6-min walk (m)	512 ± 72	$538 \pm 63^{*}$	<0.05	0.38	495 ± 69	491 ± 76	0.658	0.06	< 0.05
Chair sit-and-reach (cm)	3.9 ± 6.1	4.7 ± 4.5	0.443	0.15	5.4 ± 6.7	$7.9 \pm 6.2^{*}$	<0.05	0.39	0.283
Back scratch (cm)	-13 ± 11	$-14 \pm 10^{*}$	<0.05	0.10	-16 ± 13	-16 ± 14	0.637	0.01	0.061
8-foot up-and-go (s)	5.8 ± 1.4	6.4 ± 1.0	0.111	0.49	6.0 ± 1.8	6.3 ± 1.5	0.413	0.18	0.611

Note: All data are presented in mean \pm SD. *p < 0.05; ***p < 0.001.

Table 6

Maximal oxygen uptake and fitness capacity of the participants (Men).

Variables	Exercise	group (n = 1	<u>4)</u>	Control group $(n = 11)$			1)		
	Before	After	P for within-group comparison	Effect size	Before	After	P for within-group comparison	Effect size	group
Maximal oxygen uptake (ml/ min/kg)	29.0 ± 5.2	232.2 ± 6.7	<0.05	0.52	27.9 ± 3.3	27.2 ± 5.4	0.463	0.19	<0.05
30s arm curl with 8 lb (times)	29 ± 6	31 ± 7	0.071	0.31	28 ± 6	27 ± 7	0.322	0.15	0.051
30s chair stand (times)	21 ± 6	25 ± 7**	<0.01	0.88	22 ± 7	19 ± 7	0.089	0.65	<0.01
6-min walk (m)	537 ± 56	572 ± 52**	* <0.01	0.65	536 ± 68	543 ± 75	0.319	0.10	<0.05
Chair sit-and-reach (cm)	5.0 ± 7.6	4.7 ± 6.6	0.717	0.04	5.2 ± 10.0	5.9 ± 11.1	0.590	0.07	0.498
Back scratch (cm)	-17 ± 10	-18 ± 10	0.391	0.10	-15 ± 14	-14 ± 13	0.726	0.07	0.423
8-foot up-and-go (s)	5.4 ± 1.2	5.5 ± 1.5	0.590	0.07	5.7 ± 1.6	6.1 ± 1.8	0.358	0.23	0.495

Note: All data are presented in mean + SD. **p < 0.01.

HDL-c concentration after the 16-week FATmax training in both trained women and men. As a protective factor of cardiovascular disease, HDL-c concentration has also been increased after aerobic exercise training in older people with type 2 diabetes.^{28,32} The present FATmax study supports these previous studies.

In the studies of exercise training for people with type 2 diabetes, the improvements in laboratory variables are important. Furthermore, from the patients' point of view, it would be more important to evaluate the change in their daily physical activity capacity. People with type 2 diabetes $(59 \pm 8 \text{ yrs})$ showed poorer performance in 6-min walk, vertical jump, and upper body strength, compared with those of the aged-matched healthy people.³³ To the best of our knowledge, the present study is the first trial of the effects of FATmax training on physical capacity in daily life of older people with type 2 diabetes. We found that VO₂max, lower body strength and walking ability of both trained women and men were increased after the FATmax training, but no significant change in upper body strength and body flexibility. The main exercise mode employed in the present training program was walking, so it is reasonable to explain the non-change in upper body and flexibility. That is the limitation of the current FATmax training program. Future training program should add some upper body muscle training and flexibility training.

Conclusion

The 16-week FATmax exercise training program achieved improvements in body composition, blood glucose, lipid profile, as well as walking capacity and lower body strength in this group of older people with type 2 diabetes, compared to non-exercising controls. These results suggest, beside the improvements in the laboratory variables, the individualized FATmax training can also benefit daily physical capacity of older people with type 2 diabetes.

Conflicts of interest

The authors declare no conflict of interest.

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References

- 1. World Health Organization. Diabetes Fact Sheet No 312. WHO; 2016. http:// www.who.int/mediacentre/factsheets/fs312/en/.
- 2. Ajala O, English P, Pinkney J. Systematic review and meta-analysis of different dietary approaches to the management of type 2 diabetes. Am J Clin Nutr. 2013:97:505-516.
- 3. Colberg SR, Sigal RJ, Yardley JE, et al. Physical activity/exercise and diabetes: a

position statement of the american diabetes association. Diabetes Care. 2016;39:2065–2079.

- 4. O'Hagan C, De Vito G, Boreham CA. Exercise prescription in the treatment of type 2 diabetes mellitus: current practices, existing guidelines and future directions. Sport Med. 2013;43:39-49.
- 5. Qaseem A, Barry MJ, Humphrey LL, et al. Oral pharmacologic treatment of type 2 diabetes mellitus: a clinical practice guideline update from the american college of physicians. Ann Intern Med. 2017;166:279-290.
- Wu G, Cai B, Yu F, et al. Meta-analysis of bariatric surgery versus non-surgical treatment for type 2 diabetes mellitus. Oncotarget. 2016;7:87511-87522.
- 7. Boule NG, Haddad E, Kenny GP, et al. Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. J Am Med Assoc. 2001;286:1218-1227.
- Snowling NJ, Hopkins WG. Effects of different modes of exercise training on glucose control and risk factors for complications in type 2 diabetes patients. Diabetes Care. 2006;29:2518-2527.
- Brooks GA, Mercier J. Balance of carbohydrate and lipid utilization during exercise: the "crossover" concept. J Appl Physiol. 1994;76:2253-2261.
- 10 Jeukendrup AE, Achten J. Fatmax: a new concept to optimize fat oxidation during exercise? Eur J Sport Sci. 2001;1:1–5.
- 11. Tan S, Du P, Zhao W, et al. Exercise training at maximal fat oxidation intensity for older women with type 2 diabetes. Int J Sports Med. 2018;39:374-381.
- Suk MH, Moon Y, Park SW, et al. Maximal fat oxidation rate during exercise in 12. Korean women with type 2 diabetes mellitus. *Diabetes J.* 2015;39:328–334.
- 13. Hansen D, Dendale P, van Loon LLC, et al. The impact of training modalities on the clinical benefits of exercise intervention in patients with cardiovascular disease risk or type 2 diabetes mellitus. Sport Med. 2010;40:921-940.
- 14. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2013;36(Suppl 1):S67–S74.
- 15. Frayn KN. Calculation of substrate oxidation rates in vivo from gaseous exchange. J Appl Physiol. 1983;55:628-634.
- Tan S, Wang J, Cao L, et al. Positive effect of exercise training at maximal fat 16 oxidation intensity on body composition and lipid metabolism in overweight middle-aged women. Clin Physiol Funct Imaging. 2016;36:225-230.
- American College of Sports Medicine. ACSM's Guidelines for Exercise Testing and 17. Prescription. seventeenth ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006:144-145.
- 18. Matthews DR, Hosker JP, Rudenski AS, et al. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*. 1985;28:412–419.
- 19. Rikli RE, Jones CJ. Development and validation of criterion-referenced clinically relevant fitness standards for maintaining physical independence in later years. Gerontologidt. 2013;53:255-267.
- 20. Thomas JR, Salazar W, Landers DM. What is missing in P<.05? Effect size. Res Q
- Exerc Sport. 1991;62:344–348.
 21. Bays HE. "Sick fat," metabolic disease, and atherosclerosis. Am J Med. 2009;122: S26-S37.
- 22. Després J, Lemieux I. Abdominal obesity and metabolic syndrome. Nature. 2006:444:881-887.
- 23. Pedersen BK. The diseasome of physical inactivity and the role of myokines in muscle-fat cross talk. J Physiol. 2009;587:5559-5568.
- 24. de Mutsert R, Gast K, Widya R, et al. Associations of abdominal subcutaneous and visceral fat with insulin resistance and secretion differ between men and women: the netherlands epidemiology of obesity study. Metab Syndrome Relat Disord. 2018;16:54-63.
- 25. Sigal RJ, Kenny GP, Boule NG, et al. Effects of aerobic training, resistance training, or both on glycemic control in type 2 diabetes: a randomized trial. *Ann Intern Med.* 2007;147:357–369.
- 26. Bacchi E, Negri C, Zanolin ME, et al. Metabolic effects of aerobic training and resistance training in type 2 diabetic subjects: a randomized controlled trial (the RAED2 study). Diabetes Care. 2012;35:676-682.
- 27. Dincer S, Altan M, Terzioglu D, et al. Effects of a regular exercise program on biochemical parameters of type 2 diabetes mellitus patients. J Sport Med Phys Fit. 2016:56:1384-1391.
- 28. Mitranun W, Deerochanawong C, Tanaka H, et al. Continuous vs interval

training on glycemic control and macro- and microvascular reactivity in type 2 diabetic patients. *Scand J Med Sci Sport.* 2014;24:e69–e76.

- Coelho S. What is the role of HbA1c in diabetic hemodialysis patients? Semin Dial. 2016;29:19–23.
- 30. Hansen D, Dendale P, Jonkers RAM, et al. Continuous low- to moderateintensity exercise training is as effective as moderate- to high-intensity exercise training at lowering blood HbA_{1c} in obese type 2 diabetes patients. *Diabetologia*. 2009;52:1789–1797.
- 31. Church TS, Blair SN, Cocreham S, et al. Effects of aerobic and resistance training

on hemoglobin A1c levels in patients with type 2 diabetes: a randomized controlled trial. J Am Med Assoc. 2010;304:2253–2262.

- **32.** Zoppini G, Targher G, Zamboni C, et al. Effects of moderate-intensity exercise training on plasma biomarkers of inflammation and endothelial dysfunction in older patients with type 2 diabetes. *Nutr Metab Cardiovasc Dis.* 2006;16: 543–549.
- **33.** Ozdirenc M, Biberoglu S, Ozcan A. Evaluation of physical fitness in patients with type 2 diabetes mellitus. *Diabetes Res Clin Pract*. 2003;60:171–176.