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Diabetes Research and Clinical Practice 56 (2002) 115–123

DIABETES RESEARCH AND CLINICAL PRACTICE

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Combined aerobic and resistance exercise improves glycemic control and fitness in type 2 diabetes

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Received 19 March 2001; received in revised form 28 August 2001; accepted 13 November 2001

Abstract

We investigated the effect of an 8 week circuit training (CT) program, combining aerobic and resistance exercise, on indices of glycemic control, cardiorespiratory fitness, muscular strength and body composition in 16 subjects (age 52 ± 2 years) with type 2 diabetes using a prospective randomised crossover protocol. Submaximal exercise heart rate and rate pressure product were significantly lower after training ($P < 0.05$), whilst ventilatory threshold increased (11.8 ± 0.7 vs 13.8 ± 0.6 ml kg^{-1} min^{-1}, $P < 0.001$). Muscular strength also increased with training (403 ± 30 to 456 ± 31 kg, $P < 0.001$), whilst skinfolds (148.7 ± 11.5 vs 141.1 ± 10.7 mm, $P < 0.05$), % body fat (29.5 ± 1.0 vs 28.7 ± 1.1%, $P < 0.05$) and waist:hip ratio (99.2 ± 1.5 vs 97.9 ± 1.4%, $P < 0.05$) significantly decreased. Concurrently, peak oxygen uptake ($P < 0.05$) and exercise test duration ($P < 0.001$) increased following training, whilst glycated hemoglobin ($P < 0.05$) and fasting blood glucose ($P < 0.05$) decreased. CT is an effective method of training that improved functional capacity, lean body mass, strength and glycemic control in subjects with type 2 diabetes.

Keywords: Exercise training; Diabetes; Peak oxygen uptake; Maximal voluntary contraction; Anthropometry; Glycated hemoglobin

1. Introduction

Type 2 diabetic patients are at increased risk of all cause premature mortality, and death from cardiovascular disease [1], and many consider physical activity to be an integral component of diabetes management [2]. Traditionally, exercise programs in type 2 diabetes have involved 'aerobic' training regimens, consisting of repetitive large muscle group exercise aimed at improving cardiorespiratory fitness. These programs improve peak oxygen uptake ($\dot{V}O_2$peak) [3], skeletal muscle capillary density [4] and, at a cellular level, GLUT-4 transporter expression and muscle glycogen synthase concentration [5], but have an inconsistent effect on body composition and skeletal muscle strength [6–11]. Recent reports suggest

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0168-8227/02/$ - see front matter
PII: S0168-8227(01)00368-0

that resistance exercise modalities that increase muscle mass may improve glycemic control and insulin resistance [12–14]. In addition, we recently reported, in the same group of patients as those studied here [15] and also in a group with heart failure [16], that combined aerobic and resistance exercise improves endothelial vasodilator function and may therefore increase blood flow and glucose uptake in active muscle beds [5,17]. It has therefore been proposed that both aerobic and resistance exercise have beneficial effects in subjects with type 2 diabetes, possibly through different mechanisms. We therefore proposed that a combination of these modalities would be an effective strategy for improving body composition, functional capacity, muscular strength and glycemic control in type 2 diabetes.

2. Methods

2.1. Subjects and screening measures

Sixteen subjects (14 male, 2 female), aged 52 ± 2 (SE) years, were recruited. The following were excluded: smokers, those with renal impairment or proteinuria, hepatic impairment, gout or hyperuricemia, hypercholesterolemia (total cholesterol > 6.0 mmol l^{-1}) or hypertension (systolic BP > 160 mmHg). Five were taking ACE inhibitors, 2 lipid lowering therapy, 2 aspirin and all but one were taking an oral hypoglycemic drug (metformin 3, gliclazide 3, glipizide 1, combined therapy 8). Medications were not altered during the course of the trial. The study protocol was approved by Royal Perth Hospital Ethics Committee and subjects gave written informed consent. Data from these subjects was recently published in an article which emphasised the impact of exercise training on vascular function [15].

2.2. Experimental design

Subjects were assigned to an 8 week exercise training program or a non-training period by random allocation. They were instructed not to undertake any formal exercise or change their level of general physical activity during these periods. Experimental measures were assessed at entry, after 8 weeks and, following cross-over, 16 weeks after entry. These measures included respiratory gas exchange assessment at submaximal steady-state and peak workloads during an incremental bicycle ergometer test, muscular strength measurement and anthropometric assessment of body composition. Antecubital venous blood was drawn for determination of fasting blood glucose, glycated hemoglobin and plasma lipid concentrations. Familiarisation exercise test and strength assessments were undertaken during a 2 week lead-in period preceding randomisation.

2.3. Experimental measurements

2.3.1. Anthropometric assessment

Body weight and height were measured prior to each exercise test and body mass index was calculated. Skinfolds were measured using spring loaded calipers (Harpenden, England) at 8 standard sites [18]; triceps, biceps, subscapulare, supraspinale, iliocristale, mid-abdominal, anterior thigh and medial calf. All sites were measured in triplicate, with the median score recorded. Body fat (%) was calculated from these measures [19]. Muscle girths were similarly recorded at the following standard sites using an anthropometric steel tape (Lufkin); relaxed arm, flexed arm, waist, hip, thigh. The sum of relaxed arm, flexed arm and thigh were calculated to assess muscularity, with waist and hip measures used to determine waist:hip ratio. All trained and untrained anthropometric measures for each subject were performed by the same investigator.

2.3.2. Exercise testing and respiratory gas exchange variables

Exercise testing was undertaken on an electronically braked bicycle ergometer (Orival 400, Lode), with initial resistance set at either 20 W ($n = 2$) or 60 W ($n = 14$) depending upon baseline functional capacity, with 20 W step-wise increments every 3 min. Heart rate and rhythm were continuously recorded by 12-lead electrocardiogram and blood pressure was measured manually during the last 30 s of each 3 min stage using a mercury sphygmomanometer. Subjects reported

their rate of perceived exertion (RPE) on the 15 point Borg scale at the end of each 3-min stage.

The rate of oxygen use during exercise was calculated from minute ventilation ($\dot{V}_E$), measured using mass flow ventilometry, and simultaneous mixing chamber analysis of expired gas fractions (Vmax, Sensormedics). Gas analysers and flow probes were calibrated before each test. $\dot{V}O_2$ and $\dot{V}CO_2$ were recorded during the final 40 s of each stage of the test and at peak exercise and expressed in $l\,min^{-1}$ and relative to body weight ($ml\,kg^{-1}\,min^{-1}$). Rate pressure product (RPP) was calculated at the end of each stage of exercise as the product of HR and SBP. The ventilatory threshold (T_{VENT}) was assessed by two investigators using a combination of breakpoints in the relationship between $\dot{V}O_2$ and $\dot{V}CO_2$ (the V-slope method) and a systematic increase in the $\dot{V}_E/\dot{V}O_2$ without a concomitant increase in $\dot{V}_E/\dot{V}CO_2$.

2.3.3. Assessment of muscular strength

Maximal isotonic voluntary contractile strength (MVC_7) was assessed for seven distinct muscle groups using the 1 repetition maximum (1 RM) technique and pin-loaded weight stack resistance equipment (Pulsestar, Cheshire, England), with minimum 2.5 kg increments. These machines were also used during the exercise training program. The seven resistance exercises consisted of: dual seated leg press, left and right hip extension, pectoral exercises, shoulder extension, seated abdominal flexion and dual leg flexion. Subjects were instructed in correct lifting technique, to avoid Valsalva maneuver and hand gripping. MVC_7 was calculated as the sum of strength measures on each apparatus.

2.3.4. Assessment of biochemical parameters

Plasma lipids and glucose were measured by standard techniques using a Hitachi 917 analyser (Tokyo, Japan). Glycated hemoglobin was measured using a Biorad Variant HPLC system (Hercules, CA).

2.4. Exercise training regimen

The exercise intervention was structured and supervised by an experienced exercise physiologist in a dedicated gymnasium at Royal Perth Hospital. The 8 week training regimen consisted of three 1 h sessions of whole body exercise each week. Each of these sessions commenced and concluded with a 10-min warm-up/cool-down and stretching period.

The conditioning phase of each session involved 'circuit' training (CT); i.e. a combination of cycle ergometry, treadmill walking and resistance (weights) training. An exercise 'circuit' consisted of seven resistance exercises alternated with eight aerobic exercise (cycling) stations, each performed for 45 s, at which point a timer sounded and subjects had 15 s to move to the next station. To conclude the circuit, subjects spent 5 min walking on a treadmill. The active recovery (aerobic cycling exercise) between resistance stations was designed to maintain exercise HR within the training zone and facilitate changes in cardiorespiratory fitness. Intensity and duration of the exercise training were progressively increased throughout the 8 weeks of the program, as individually tolerated, initially by increasing the number of exercise circuits from 1 to 3 and then by increasing resistance, cycling and treadmill load.

Resistance training intensity commenced at 55% of pre-training MVC, as determined from initial 1 RM strength tests, and increased to 65% by week 4 of the program. During resistance exercise, subjects were instructed to perform one complete lift every 3 s so that 15 repetitions of an exercise were performed in 45 s. Cycle ergometry and treadmill walking commenced at 70% of the peak HR observed during the initial incremental exercise test and increased to 85% by week 6.

2.5. Treatment and analysis of data

Results are expressed as means $\pm$ SE except for plasma triglycerides which are expressed as geometric means with 95% confidence limits. The responses after exercise training were compared to non-training responses using Student's paired t-tests. $P < 0{\cdot}05$ was considered significant.

3. Results

Six of the 16 subjects were randomised to receive exercise training during the first 8 weeks, 10 during the last 8 weeks. All completed 24 exercise sessions and the exercise regimen was well tolerated, with no adverse events. No significant adverse events occurred during exercise testing procedures or the training sessions.

3.1. General effects of exercise training

There were no significant differences in plasma total, HDL or LDL cholesterol, or triglycerides following training (Table 1). Similarly, mean arterial pressure was unchanged. Resting HR was significantly lower following training (70 ± 3 vs 66 ± 3 beats min^{-1}, $P < 0.05$) as were glycated hemoglobin (8.5 ± 0.4 vs $7.9 \pm 0.3\%$, $P < 0.05$) and fasting blood glucose (12.0 ± 0.5 vs 9.8 ± 0.5 mmol l^{-1}, $P < 0.05$). Some of these data were included, in summary form, in our recent publication regarding the effects of exercise training on vascular function in these subjects [15], because they provide a common basis for interpretation of both studies.

Table 1
Subject characteristics following the trained and untrained periods

	Untrained	Trained
Plasma lipids (mmol l^{-1})		
Total cholesterol	4.6 ± 0.2	4.6 ± 0.2
LDL-C	2.4 ± 0.2	2.5 ± 0.2
HDL-C	1.0 ± 0.1	1.1 ± 0.1
Triglycerides	2.14 (1.48–3.08)	1.97 (1.46–2.64)
Glycated hemoglobin (%)	8.5 ± 0.4	7.9 ± 0.3*
Fasting blood glucose (mmol l^{-1})	12.0 ± 0.5	9.8 ± 0.5*
Mean arterial pressure (mmHg)	102 ± 3	104 ± 2
Resting heart rate (beats min^{-1})	70 ± 3	66 ± 3*

Values are means $\pm$ SE except for plasma triglycerides which are expressed as geometric means with 95% confidence limits. Exercise training significantly decreased glycated hemoglobin, fasting blood glucose and resting heart rate (*$P < 0.05$). No significant differences were evident for other variables. This table was previously published in a study of the same patients which reported vascular function outcomes [15].

Table 2
Anthropometric and muscular strength characteristics following the trained and untrained periods

	Untrained	Trained
Body weight (kg)	88.7 ± 4.4	88.7 ± 4.4
BMI	29.6 ± 3.4	29.6 ± 3.3
Waist:Hip (%)	99.2 ± 1.5	97.9 ± 1.4*
Sum of 8 skinfolds (mm)	148.7 ± 11.5	141.1 ± 10.7*
Body fat (%)	29.5 ± 1.0	28.7 ± 1.1*
Sum of 3 segment girths (mm)	122.6 ± 2.5	123.4 ± 2.8
Muscle strength:		
Sum of 7 max contractions (kg)	403 ± 30	456 ± 31**

Values are means $\pm$ SE. Exercise training significantly decreased the sum of skinfolds, body fat and waist:hip (*$P < 0.05$). Muscular strength increased (**$P < 0.001$). No significant differences were evident for other variables.

3.2. Anthropometric assessment and muscular strength

Anthropometric and strength data are presented in Table 2. Exercise training significantly enhanced muscular strength from 403 ± 30 to 456 ± 31 kg ($P < 0.001$). Body weight and segment girths did not significantly change after training, although the sum of skinfolds (148.7 ± 11.5 vs 141.1 ± 10.7 mm, $P < 0.05$), % body fat (29.5 ± 1.0 vs $28.7 \pm 1.1\%$, $P < 0.05$) and waist:hip ratio (99.2 ± 1.5 vs $97.9 \pm 1.4\%$, $P < 0.05$) significantly decreased.

3.3. Peak exercise test data

Exercise training was associated with significant increase in $\dot{V}O_2$peak, from 23.1 ± 1.2 to 24.8 ± 1.4 ml kg^{-1} min^{-1} ($P < 0.05$, Fig. 1), which was also evident when $\dot{V}O_2$peak data were expressed in absolute terms 2.0 ± 0.1 to 2.2 ± 0.2 l min^{-1} ($P < 0.01$). Exercise test duration improved from 12.6 ± 1.2 to 14.8 ± 1.3 min ($P < 0.001$, Fig. 1). Peak HR (158 ± 4 vs 159 ± 4 beats min^{-1}, $P =$

NS), RPP (35964 ± 1770 vs 35840 ± 1446, beats min^{-1} mmHg, $P = NS$) and RPE (15.9 ± 0.5 vs 15.7 ± 0.5, $P = NS$) did not significantly differ after training.

3.4. Sub-maximal exercise test data

All subjects completed all exercise test workloads up to, and including, 60 W. Prior to training, 14 subjects completed 80 W, 11 completed 100 W, 9 completed 120 W and 3 completed 140 W. Following training, 15 subjects completed 80 W, 12 completed 100 W, 11 completed 120 W and 9 completed 140 W. HR and RPP were significantly lower after training at all workloads through 60–120 W (Table 3). RPE was lower at 80, 100 and 120 W, submaximal $\dot{V}O_2$ at 100 and 120 W. The T_{VENT} occurred at a higher value following training, both in absolute terms (11.8 ± 0.7 vs 13.8 ± 0.6 ml kg^{-1} min^{-1}, $P < 0.001$) and as a proportion of $\dot{V}O_2$peak (51.3 ± 2.0 vs $56.6 \pm 1.0\%$, $P < 0.005$).

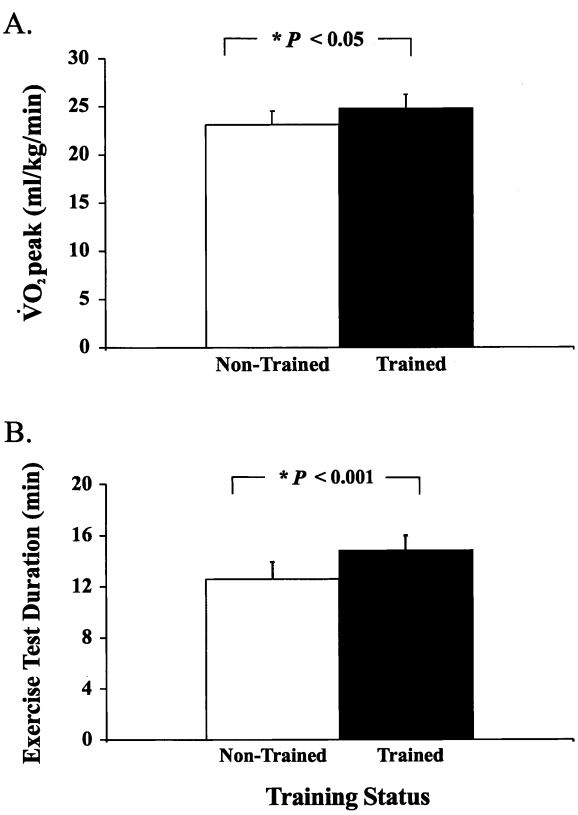

Fig. 1. (A) Peak oxygen uptake ($\dot{V}O_2$peak), and (B) exercise test duration following 8 weeks of inactivity (□) or 8 weeks of circuit training (■) in subjects with type 2 diabetes. Values are means ± SE. Both $\dot{V}O_2$peak ($P < 0.05$) and test duration ($P < 0.001$) were enhanced after circuit training.

3.5. Effect of order of administration

Fig. 2 depicts trained and untrained $\dot{V}O_2$peak data according to order of administration of exercise training; those who received exercise training first compared to those who trained second. The effect of exercise training on $\dot{V}O_2$peak was not different between these subgroups ($P = NS$). Fig. 3 presents a similar analysis of the strength data. The difference between trained and untrained MVC_7 was, on average, less in those trained first, although not significantly so ($P = NS$), suggesting that, while there might have been some persistence of the training effect, it was not significant. In subjects who trained first ($n = 6$), glycated hemoglobin was elevated, on the average, from post-training levels of 8.0 ± 0.5 to $8.4 \pm 0.6\%$ 8 weeks following the cessation of training ($P = NS$). Similarly, fasting blood glucose rose from post-training levels of 10.2 ± 1.1 to 11.5 ± 1.5 mmol l^{-1} 8 weeks following the cessation of training ($P = NS$). Although there could have been some carry-over effect of training on these parameters, the data from those trained first and those trained second were pooled, and any such effect would have only tended to decrease the documented beneficial effects of training.

4. Discussion

The present study examined the influence of CT, a novel exercise prescription involving aerobic and resistance modalities, in subjects with type 2 diabetes. The rationale behind this combined method of training was that aerobic exercise increases cardiovascular fitness and skeletal muscle capillary density and reduces body fat whilst resistance exercise improves muscle mass and strength. The principle findings of this study are that CT improved maximal and sub-maximal exercise ca-

Table 3
Submaximal exercise test data before and after training

	Exercise test workload (W)			
	60	80	100	120
Heart rate (beats min^{-1})				
Untrained	116 ± 4	126 ± 4	134 ± 3	144 ± 4
Trained	111 ± 4*	122 ± 5***	127 ± 4**	135 ± 4***
Rate pressure product (beats min^{-1} mmHg)				
Untrained	20 711 ± 1363	25 271 ± 1725	28 983 ± 1729	32 837 ± 2251
Trained	18 774 ± 1183**	23 041 ± 1782**	26 132 ± 1728**	29 626 ± 1659*
Rate perceived exertion				
Untrained	10.9 ± 0.5	12.3 ± 0.4	13.1 ± 0.4	15.0 ± 0.6
Trained	10.3 ± 0.4	11.5 ± 0.5**	11.9 ± 0.4**	12.8 ± 0.4***
$\dot{V}O_2$ *(ml kg^{-1} min^{-1})*				
Untrained	14.7 ± 0.5	17.4 ± 0.5	19.9 ± 0.8	22.7 ± 1.2
Trained	14.5 ± 0.5	17.3 ± 0.3	18.6 ± 0.7*	20.8 ± 0.9*

Values are means ± SE. *$P<0.05$, **$P<0.01$, ***$P<0.001$ for difference between untrained and trained data.

pacity, body composition, muscular strength and glycemic control in type 2 diabetic subjects.

Recent studies suggest that training programs which involve a resistive exercise component, that is moderate intensity weight-lifting exercises, may be of particular benefit in type 2 diabetes, due to an effect of increasing insulin action [12–14]. Increases in muscle mass have been associated with benefits in terms of glycemic control [20] as skeletal muscle represents the largest mass of insulin-sensitive tissue. In the present study skinfolds, % body fat and waist:hip ratio decreased, muscle girths and body weight did not change and muscular strength improved, suggesting that lean body mass increased. In addition, both glycated hemoglobin and fasting blood glucose decreased. CT was also associated with improved aerobic capacity, independent of changes in body weight which, in turn, is associated with enhanced glucose uptake [5], possibly due to improved blood flow [15,16,21] and enhanced tissue exposure to insulin and glucose.

In the present study, peak oxygen uptake and treadmill exercise time to exhaustion increased, whilst submaximal RPP and HR decreased following training. In addition, the percent of $\dot{V}O_2$peak at which the T_{VENT} occurred significantly increased after training, indicating that subjects could exercise at higher submaximal exercise intensities prior to the onset of blood lactic acid accumulation. These findings indicate that aerobic capacity improved following CT, a result of clinical importance as studies indicate that physical inactivity is associated with glucose intolerance [22,23]. Previous studies of aerobic training in middle aged and elderly type 2 diabetics have found no change [24] or a modest increase (8%)

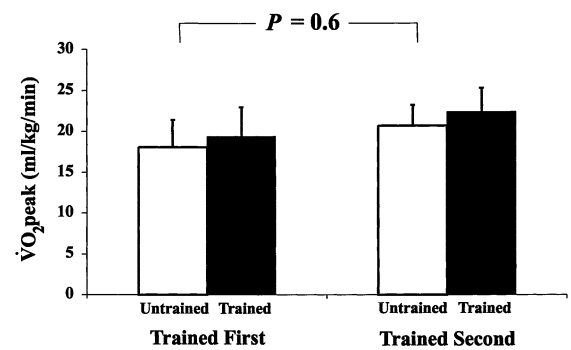

Fig. 2. Peak oxygen uptake ($\dot{V}O_2$peak) response following 8 weeks of inactivity (□) and 8 weeks of circuit training (■) in the group trained first and the group trained second. Values are means ± SE. The effect of exercise training on $\dot{V}O_2$peak was not different between these subgroups (P = NS). The order of training did not influence $\dot{V}O_2$peak when untrained or trained results were compared.

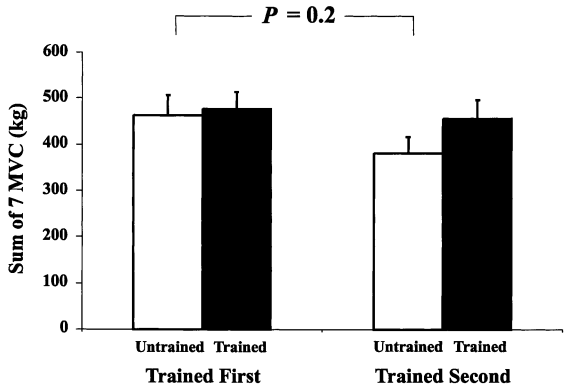

Fig. 3. Muscular strength assessed as the sum of maximal voluntary contractions for 7 muscle groups (MVC_7) following 8 weeks of inactivity (□) and 8 weeks of circuit training (■) in the group trained first and the group trained second. Values are means ± SE. The difference between trained and untrained MVC_7 was, on average, less in those trained first, although not significantly so (P = NS), suggesting that persistence of the training effect on this parameter was not significant.

[25] in $\dot{V}O_2$peak using training interventions of similar duration to the present study. However greater increases have been evident with longer periods of training [25,26]. These studies have not demonstrated improvements in skeletal muscle strength whereas strength did increase following training in the present study. Moderate resistance training in type 2 diabetes has previously been associated with strength increases that are comparable to those found in the present study [27,28], but were not associated with improvements in aerobic capacity. This is the first study, to our knowledge, to report significant improvements in peak and submaximal aerobic capacity, as well as strength, in type 2 diabetes subjects following exercise training involving a resistance component [28,29]. Unlike many previous studies, all subjects undertook preliminary exercise testing during the 2 week run-in period preceding the initial experimental measures to ensure that they were familiarised with the test procedures and that a learning effect did not influence the results. The increases in strength (13%) and $\dot{V}O_2$peak (7%) described in this study reflect the moderate levels of resistance and aerobic exercise prescribed. Higher intensity training which is exclusively resistance or aerobic in nature may result in greater improvements in strength and $\dot{V}O_2$peak respectively, but does not offer the combined benefit of CT.

It is possible from the results of this cross-over trial to determine whether the effects of CT on strength, glycemic control and $\dot{V}O_2$peak persisted after the cessation of exercise. The data suggest that improvement in strength might have been more persistent than that in $\dot{V}O_2$peak, although the effect on neither was fully sustained. Similarly, 8 weeks after training both glycated hemoglobin and resting blood glucose exhibited trends towards baseline levels in those subjects who trained first. It is therefore likely that a regular regimen of exercise is needed to preserve the benefits of CT, a conclusion consistent with previous findings indicating that the effects of exercise on glycemic control rapidly subside [20].

In summary, we suggest that the CT exercise modality is beneficial for subjects with type 2 diabetes, since it is well tolerated, combines the beneficial effects of aerobic conditioning and skeletal muscle strength training and improves body composition and glycemic control. Furthermore it involves rotation between large muscle groups which minimises local muscle fatigue and spreads the benefits of exercise training to a larger mass of skeletal muscle [20]. Although the exercise program here was tightly regimented and supervised for the purpose of documenting its effects, the principles of such a program should be generally applicable to diabetic subjects able to undertake exercise and should be safely adaptable to a home based program.

Acknowledgements

This study was supported by the Heart Foundation (Australia) and Medical Research Fund of Western Australia (MEDWA).

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