DOI 10.26773/smj.211007

ORIGINAL SCIENTIFIC PAPER

The Effects of Functional Exercise Training on Obesity with Impaired Glucose Tolerance

Sirikarn Suntisawee[1], Thanomwong Kritpet[1], Sitha Phongphibool[1] and Thep Himathongkam[2]

[1]Chulalongkorn University, Faculty of Sports Science, Bangkok, Thailand, [2]Theptarin Hospital, Bangkok, Thailand

Abstract

Obese individuals with impaired glucose tolerance (IGT) are at risk for developing overt diabetes and cardiovascular diseases (CVD). This study aimed to examine the effects of 12 weeks of a functional exercise training (FET) programme in obese individuals with IGT. Sixteen males and females university staff, aged 50.4±1.3 years (43 to 59 yrs) with mean BMI ≥25 kg/m^2 (WHO Asian Guidelines) and IGT were randomly divided into the functional exercise training (FET) group or the control (CON) group. Both groups underwent the baseline assessments, including anthropometric measurements, exercise capacity, oral glucose tolerance test (OGTT), and blood chemistry analysis. All testing was repeated at 12 weeks post-intervention. The FET groups engaged in the FET programme, and the CON group carried out normal daily physical activity, including walking. After the intervention, the FET group showed significant changes in exercise capacity, body weight (BW), BMI, waist circumference, triglycerides, fasting plasma insulin (FPI), 2 hrs glucose, and glucose AUC ($p<0.05$) while the CON group only exhibited an improvement in HDL-C ($p<0.05$). The study showed that the FET programme improves exercise capacity and alters cardiometabolic parameters. It can be an alternative form of exercise for managing obesity and improves glycaemic control in those at risk.

Keywords: *cardiorespiratory fitness, functional exercise, impair glucose tolerance, obesity*

Introduction

Impaired glucose tolerance (IGT) is a major predictor of type 2 diabetes (Alberti, 2007) and is a cardinal sign of insulin resistance (DeFronzo, & Abdul-Ghani, 2011). Those with impaired glucose tolerance (IGT) are at increased risk for developing overt type 2 diabetes and cardiovascular disease (CVD) (DeFronzo, & Abdul-Ghani, 2011). Additionally, epidemiological studies have shown an association between physical inactivity and IGT (Tapp et al., 2006). Physical inactivity alters functional capacity and normal metabolic action of insulin, including glucose transport, glycogen synthesis, and glucose oxidation (Venables, & Jeukendrup, 2009). Previous data have shown that most individuals with IGT are overweight, and up to 80% are obese (Hawley, 2004). Thus, being obese with IGT can further accelerate the risk of frank diabetes (Rai, Wadhwani, Sharma, & Dubey, 2019). The American Diabetes Association (ADA, 2002) recommends that overweight individuals with IGT undergo some kinds of lifestyle intervention to prevent the onset of type 2 diabetes.

Exercise training is often prescribed for blood glucose management (ADA, 2002; Diabetes Prevention Program, 2002). Swindell et al. (2018) showed that exercise training improves glucose transporter 4 (GLUT 4) translocation to the cell membrane, facilitating glucose transport into the cell. Regular exercise training lowers blood glucose and produces other benefits, such as increased fitness, weight reduction, improve physical function, and reduced risk for developing non-communicable diseases (NCD) (Rehn, Winett, Wisløff, & Rognmo, 2013). The investigators in the Diabetes Prevention Program (DPP) found that lifestyle intervention that included regular physical activity reduced the incidence of diabetes by 58% compared to the use of metformin in those with IGT (Sigal, Kenny, Wasserman, Castaneda-Sceppa, &

Correspondence:

T. Kritpet
Chulalongkorn University, Faculty of Sports Science, Rama I Rd. Wangmai District. Patumwan, Bangkok 10330, Thailand
Email: tkritpet@yahoo.com

White, 2006). The DPP study reinforced the importance of achieving ≥150 min/wk of physical activity at moderate intensity (e.g. walking) for preventing the onset of diabetes (DPP, 2002; Sigal et al., 2006). General aerobic activity such as walking can be monotonous for some people, leading to dropping out of activity participation (ACSM, 2018). Thus, adding a variety of exercise into the daily routine can add fun and promote social interaction that can make exercise more enjoyable.

We intended to investigate the effects of functional exercise training on exercise capacity, glucose metabolism, and metabolic profiles in obese individuals with IGT. The functional exercise is simple, economical, and can be carried out at a gym or at home without utilizing many pieces of equipment and can be an alternative form of exercise that complements general aerobic activity. This type of exercise trains the muscles to work together and prepares them for daily living activities (Silva-Grigoletto, Brito, & Heredia, 2014). Additionally, it strengthens the body's core and improves stability, which can result in better posture and balance (Lagally, Cordero, Good, Brown, & McCaw, 2009). This type of exercise simulates common movements that can be done at home or at work while using the upper and lower body simultaneously. Previous studies have shown that functional exercise training improved mobility in older adults (Whitehurst, Johnson, Parker, Brown, & Ford, 2005), and it significantly improved physical fitness components in male college students (Shaikh, & Mondal, 2012). However, little is known about its effects on the metabolic profiles in obese individuals.

This study aimed to investigate the effects of functional exercise training on exercise capacity, glucose metabolism, and metabolic profiles in obese individuals with IGT. We hypothesize that FET will produce favourable changes in exercise capacity, glucose metabolism, and metabolic profiles.

Methods

Participants

Sixteen obese males and females supporting staff from Nakhon Ratchasima Rajabhat University age 50.4±1.3 years (43 to 59 yrs) with impaired glucose tolerance (IGT) were recruited to participate in the study; they were randomly classified into experimental (n=8) or control (n=8) groups. The obesity classification was in accordance with WHO Asian guidelines: ≥25 kg/m² is considered obese (WHO, 2000) and IGT as classified by the American Diabetes Association (ADA, 2018) as having 2-hrs glucose ≥140 mg/dL^{-1} and ≤199 mg/dL^{-1}. The participants were contacted by the primary investigator and were invited to the orientation session where they signed informed consent, filled out the health questionnaire, underwent the screening process, performed oral glucose tolerance test (OGTT), exercise testing, and blood chemistry analysis. To be eligible for the study, the participants had to have impaired glucose tolerance, had not participated in any formal exercise for the previous six months, and be free from heart disease, hypertension, diabetes, orthopaedic, and neuromuscular problems. All the testing was performed at baseline and at 12 weeks which was similar to the previous studies by Whitehurst et al. (2005) and McNeilly et al. (2012). The study protocols and procedures were approved by the Research Ethics Review Committee for Research Involving Research Participants, Health Science Group (COA No. 230/2019), Chulalongkorn University, Thailand.

Anthropometric measurement

Bodyweight (kg) was assessed using the standard digital (Nagata BW-110, Taiwan). Height (cm) was measured using the standard stadiometer (Nagata BW-110, Taiwan). Bodyweight and height were measured to the nearest 0.01 kg and 0.01 cm, respectively. Body mass index (BMI) was calculated by dividing body weight in kilogram (kg) by height in metre-squared (m²). Waist circumference (cm) was measured at the horizontal plane at the iliac crest.

Exercise capacity

The participants underwent the Astrand maximal cycle test (ACSM, 2018) to assess their exercise capacity (Cateye-EC1600 Bicycle Ergometers, Japan). Each participant was briefed on the testing procedure, adjusted seat height, fitted with a wireless heart rate monitor (Polar model H7, Finland), and was allowed time to warm up on the cycle ergometer for three minutes with a resistance of zero watts. Following the warm up, the subject was instructed to pedal the cycle ergometer at 50 rpm for two minutes in the initial stage with a load of 100 watts (men) or 50 watts (women). After the initial stage, the workload of 50 watts (men) or 25 watts (women) was incrementally increased every three minutes until the participant reached volitional fatigue or was unable to maintain the instructed cadence. Testing was terminated in accordance with the standard guidelines (ACSM, 2018). The exercise capacity was calculated for maximal oxygen uptake (VO^2 max) and metabolic equivalent (MET) value.

OGTT and Blood chemistry analysis

After an overnight fast, a 75-g OGTT was performed on the participants, and blood samples were obtained at baseline plasma glucose and insulin and every 30 min interval for 120 min after an oral glucose load (Slentz et al., 2016). Glucose areas under the curve (AUC) was calculated using the trapezoidal principle. Early and total phase glucose tolerances were calculated as total area under the curve (tAUC) using the trapezoidal model (Matthews et al., 1985). The homeostasis model assessment of insulin resistance (HOMA) was calculated as described previously (Matthews et al., 1985; Vogeser et al., 2007). Blood samples taken at baseline were also analysed for HbA1C, total cholesterol (TC), triglycerides (TG), LDL-Cholesterol (LDL-C), HDL-Cholesterol (HDL-C). Insulin resistance was estimated by the homeostatic model assessment (HOMA-IR). Blood samples were analysed for glucose, HbA1C; the lipid profile was determined using hexokinase method was measured using a cobas6000 (c501) clinical chemistry analyser system and insulin was determined using electrochemiluminescence immunoassay; the ECLIA method was measured using a cobase411 insulin analyser. Blood samples were measured by the clinical laboratory (Lab Plus Professional Laboratory Ultimate Service, Theptarin Hospital, Thailand).

Exercise Programme

For the 12-week study, the participants were randomly assigned into two groups: functional exercise training (FET) and control (CON). The FET group engaged in the functional exercise training in a circuit manner; they had to complete three circuits of exercises in a session (Whitehurst et al., 2005). A circuit consisted of 12 exercises that had to be performed consecutively with 60 seconds of rest in between each exercise; the functional exercise programme details are described in Table 1 and Figure 1. Each exercise session consisted of a 10 minute warm up followed by 30 minutes of exercise session and concluded with a cool down (10 min). The participants engaged in supervised exercise sessions three times per week and performed the same exercise routine at home two times per week. All group exercise sessions were monitored and supervised by the primary investigator to ensure safety and proper technique. The task difficulty was increased by having participants balance on one leg, perform a choreographed movement, and add external hands weights. At every three-week increment, from week 4 to week 12, 2 water bottles filled with sand weighing 335 g, 500 g, and 750 g each, respectively, were added as an external weight to increase resistance while performing these exercises (illustrated in Figure 1). The participants were instructed to perform a warm up, cool down, and stretching for every exercise session.

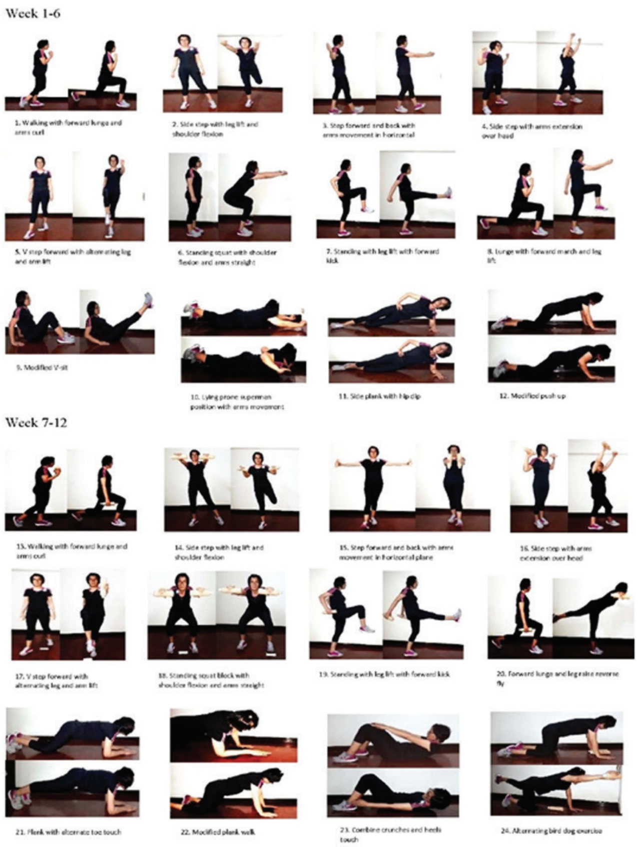

FIGURE 1. Description of Functional Exercise Training Programme

The participants in the CON group were instructed to continue their normal daily activity and were encouraged to engage in a walking routine on their own. All participants in both groups were educated on healthy diet. A text messaging group was set up for two-way communication to provide assistance and answer any questions.

Table 1. Description of Functional Exercise Training Programme

Warm up/Cool down	Week 1-6	Week 7-12	Remarks
Dynamic movement and stretching such as marching place, walking, shoulders and arms movement	1. Walking with forward lunge and arms curl	13. Walking with forward lunge and arms curl	• The participants were instructed to perform the movement in a controlled manner.
Static stretching of the upper and lower extremities	2. Sidestep with leg lift and shoulder flexion	14. Sidestep with leg lift and shoulder flexion	• A metronome was used to control the movement, and the pace was set at 100-110 beats/min.
	3. Step forward and back with arm movement in a horizontal plane	15. Step forward and back with arm movement in a horizontal plane	
	4. Sidestep with arm extension over head	16. Sidestep with arm extension over head	• Week 1-3: exercise with bodyweight alone.
	5. V step forward with alternating leg and arm lift	17. V step forward with alternating leg and arm lift	

(Continued on next page)

(continued from previous page)

Warm up/Cool down	Week 1-6	Week 7-12	Remarks
	6. Standing squat with shoulder flexion and arms straight	18. Standing squat block with shoulder flexion and arms straight	• Week 4-6: exercise with 2 water bottles filled with sand weighing 335 g each.
	7. Standing with leg lift with forward kick	19. Standing with leg lift with forward kick	
	8. Lunge with forward march and leg lift	20. Forward lunge and leg raise reverse fly	• Week 7-9: exercise with 2 water bottles filled with sand weighing 500 g each.
	9. Modified V-sit	21. Plank with alternate toe touch	
	10. Lying prone superman position with arms movement	22. Modified plank walk	• Week 10-12: exercise with 2 water bottles filled with sand weighing 750 g each.
	11. Side plank with hip dip	23. Combine crunches and heels touch	
	12. Modified push up	24. Alternating bird dog exercise	

Legend: The participants performed 12 exercises per circuit and completed 3 circuits of exercises (12 exercises equal 1 circuit). Each exercise was performed at 3 sets of 10 repetitions.

Final Testing

All testing was repeated after 12 weeks of intervention. Due to the quick change in glucose metabolism after the cessation of exercise, OGTT and blood sampling were conducted within 36 hours of the final exercise bout.

Statistical Analysis

Both groups baseline characteristics were analysed, and variables are presented as the mean ± SD. The dependent t-test was used to detect the intragroup differences over time for each variable. The extent of the change in variables was calculated by subtracting the baseline data with post-12 weeks of intervention. The differences in variables between the two groups (FET and CON) were compared using the independent t-test. Statistical significance was set at P<0.05. All statistical analyses were performed using SPSS statistical software version 23 (IBM SPSS Inc., Chicago, USA).

Results

Baseline measurements

Participants' characteristics in the FET and CON groups are presented in Table 2. The participants were similar in most variables at baseline. The FET group exhibited significantly higher FPG and glucose AUC at baseline than the CON group (p<0.05).

Table 2. Baseline characteristics of FET and CON groups

Variables	Total (n=16) Mean±SD	FET (n=8) Mean±SD	CON (n=8) Mean±SD
Age (yrs)	50.4±1.3	50.4±1.6	50.4±2.3
BW (kg)	70.5±2.3	68.4±2.5	72.7±3.9
Ht (cm)	159.3±1.8	156.9±2.2	161.7±2.7
BMI (kg.m^{-2})	27.7±0.5	27.7±0.5	27.7±0.8
Waist circumference (cm)	89.7±1.9	88.9±2.0	90.5±3.3
VO_2max (ml·kg·min^{-1})	29.3±1.3	29.8±1.5	28.8±2.2
VO_2max (MET)	8.7±0.4	8.5±0.4	9.0±0.8
Total cholesterol (mg.dL^{-1})	206.9±7.4	209.3±13.5	204.6±7.1
Triglycerides (mg.dL^{-1})	130.7±14.8	135.4±18.8	126.0±24.1
HDL-C (mg.dL^{-1})	51.9±3.0	50.9±5.2	52.9±3.4
LDL-C (mg.dL^{-1})	147.8±6.8	149.5±11.5	146.0±8.1
FPG (mg.dL^{-1})	91.3±2.5	97.3±3.6*	85.3±1.7
HbA1c (%)	5.6±0.1	5.6±0.2	5.5 ±0.2
FPI (pmol/L)	79.3±12.2	88.6±22.3	70.1±10.6
2-hrs glucose (mg.dL^{-1})	163.4±4.9	171.1±6.0	155.8±7.1
Glucose AUC	20935.3±651.9	22254.4±884.3*	19616.3±736.4
HOMA-IR	2.6±0.5	3.1±0.9	2.1±0.3

Legend: * Statistically significant between-group baseline training (p<.05); BW-Body weight; Ht-Height; FPG-fasting plasma glucose; glucose AUC-glucose area under the curve; HbA1c-Glycosylate haemoglobin; FPI-fasting plasma insulin and HOMA-IR-homeostasis model assessment of insulin resistance

Anthropometric variable and exercise capacity

After 12 weeks of intervention, the FET group showed a significant decrease in BW ($p<0.05$), BMI ($p<0.05$), and waist circumference ($p<0.05$). A significant improvement in functional capacity, as shown by the increase in VO2max and MET ($p<0.05$) were observed in this group. Conversely, no changes were observed in the previously mentioned variables in the CON group (Table 3).

Table 3. Changes in body weight, body mass index; BMI, waist circumference, VO_2max and lipid profiles at baseline and after 12 weeks of training in FET and CON groups

Variables	Within-group comparisons		
	Baseline (Mean±SD)	12 weeks (Mean±SD)	p-value
BW (kg)			
FET (n=8)	68.35±2.55	65.55±2.59*	0.008
CON (n=8)	72.69±3.92	72.50±3.94	0.780
BMI (kg.m-^{2})			
FET (n=8)	27.73±0.52	26.55±0.54*	0.005
CON (n=8)	27.65±0.80	27.60±0.82	0.847
Waist circumference (cm)			
FET (n=8)	88.88±2.02	84.31±2.53*	0.003
CON (n=8)	90.50±3.30	90.00±3.33	0.419
VO2max (ml/kg/min)			
FET (n=8)	29.83±1.51	33.55±1.28*	0.036
CON (n=8)	28.79±2.20	31.40±2.83	0.227
VO2max (MET)			
FET (n=8)	8.52±0.43	9.59±0.37*	0.036
CON (n=8)	8.97±0.81	9.48±6.11	0.931
Total cholesterol (mg.dL^{-1})			
FET (n=8)	209.25±13.50	204.25±12.50	0.515
CON (n=8)	204.63±7.10	218.50±8.66	0.093
Triglycerides (mg.dL^{-1})			
FET (n=8)	135.38±18.82	123.63±20.65*	0.046
CON (n=8)	126.00±24.11	115.88±26.89	0.190
HDL-C (mg.dL^{-1})			
FET (n=8)	50.88±5.17	53.45±5.46	0.096
CON (n=8)	52.88±3.42	59.00±3.60*	0.008
LDL-C (mg.dL^{-1})			
FET (n=8)	149.50±11.54	137.38±10.27	0.165
CON (n=8)	146.00±8.09	147.75±8.43	0.800

Legend: * Statistically significant within-group change after 12 weeks training ($p<.05$)

Blood chemistry

The FET group showed a significant decrease in triglycerides ($p<0.05$), FPI ($p<0.05$), 2-hrs glucose ($p<0.05$), and glucose AUC ($p<0.05$). The CON group showed a significant improvement in HDL-C ($p<0.05$) and 2-hrs glucose ($p<0.05$) at post-intervention (Table 3, 4).

Table 4. Changes in metabolic and glycaemic at baseline and after 12 weeks of training in FET and CON groups

Variables	Within-group comparisons		p-value
	Baseline (Mean±SD)	12 weeks (Mean±SD)	
FPG (mg.dL^{-1})			
FET (n=8)	97.25±3.61	95.63±2.12	0.509
CON (n=8)	85.25±1.73	90.13±2.97	0.070
HbA1c (%)			
FET (n=8)	5.61±0.15	5.53±0.16	0.613
CON (n=8)	5.51 ±0.17	5.49±0.12	0.722

(Continued on next page)

(continued from previous page)

Variables	Within-group comparisons		p-value
	Baseline (Mean±SD)	12 weeks (Mean±SD)	
FPI (pmol/L)			
FET (n=8)	88.63±22.27	50.31±8.62*	0.048
CON (n=8)	70.06±10.62	61.04±10.15	0.231
2-hrs glucose (mg.dL-1)			
FET (n=8)	171.13±5.99	112.38±9.96*	0.000
CON (n=8)	155.75±7.12	125.13±9.27*	0.007
Glucose AUC			
FET (n=8)	22254.38±884.28	18528.75±932.48*	0.003
CON (n=8)	19616.25±736.40	18442.50±1063.63	0.131
HOMA-IR			
FET (n=8)	3.13±0.86	1.72±0.32	0.060
CON (n=8)	2.13±0.35	1.97±0.33	0.387

Absolute changes

The FET group exhibited significant absolute changes in BW ($p<0.05$), BMI ($p<0.05$), waist circumference ($p<0.05$), 2-hrs glucose ($p<0.05$), and glucose AUC ($p<0.05$) when compared to the CON group (Table 5).

The Glucose AUC was significantly decreased with training at 12 weeks in the FET group ($p<0.05$) Figure 2.

Table 5. Absolute changes in body weight, body mass index; BMI, waist circumference, VO_2max, lipid profiles, metabolic and glycaemic after 12 weeks training between FET and CON groups

Variables	FET (n=8) (Mean±SD)	CON (n=8) (Mean±SD)	p-value
BW (kg)	-2.80±0.77*	-0.19±0.65	0.021
BMI (kg.m-2)	-1.18±0.30*	-0.05±0.25	0.011
Waist circumference (cm)	-4.56±1.05*	-0.50±0.58	0.005
VO2max (ml/kg/min)	3.73±1.44	2.62±1.98	0.657
VO2max (MET)	1.07±0.41	0.75±0.56	0.658
Total cholesterol (mg.dL-1)	-5.00±7.30	13.88±7.13	0.085
Triglycerides (mg.dL-1)	-11.75±4.85	-10.13±6.97	0.851
HDL-C (mg.dL-1)	2.88±1.49	6.13±1.66	0.168
LDL-C (mg.dL-1)	-12.13±7.82	1.75±6.65	0.198
FPG (mg.dL-1)	-1.63±2.34	4.88±2.29	0.067
HbA1c (%)	-0.09±0.17	0.09±0.15	0.440
FPI (pmol/L)	-38.28±16.02	-9.03±6.88	0.126
2-hrs glucose (mg.dL-1)	-58.75±7.33*	-30.63±8.12	0.022
Glucose AUC	-3725.63±856.78*	-1173.75±686.53	0.036
HOMA-IR	-1.42±0.63	-0.16±0.17	0.091

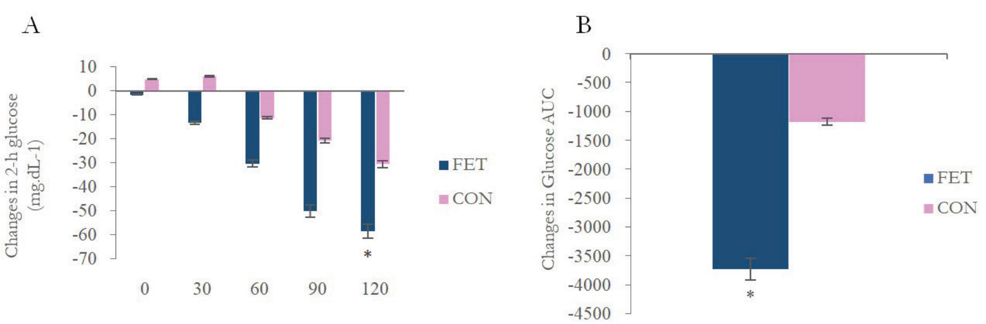

FIGURE 2. A. Training-induced changes in glucose values at each time point during OGTT; B. Change in glucose AUC following 12 weeks of training and glucose AUC. *Statistically significant (p<.05).

Discussion

This study shows that functional exercise training (FET) performed in a circuit manner resulted in improved exercise capacity, expressed in VO2 max and MET. The participants in the FET group significantly increased their exercise capacity by 1 MET from baseline ($p<0.05$); on the other hand, the CON group did not show a significant improvement in this parameter. It is conceivable that this change occurred as a result of repetitive physical training that induced physiological response in favour of cardiorespiratory endurance. Our findings are inconsistent with those of Whitehurst et al. (2005) that looked at the benefits of functional exercise training in older adults. Their findings showed that functional exercise performed in a circuit manner improved the timed walk test by 7.4% from baseline, indicating cardiorespiratory fitness improvement. The FET programme utilized large muscle groups for movement and was done continuously for a certain amount of time which elevated exercise heart rate and stimulated hemodynamic changes. When performed on a regular basis for 12 weeks, it caused physiological adaptation and improved fitness for this group.

Improvement in exercise capacity translates into a better quality of life in those with risk factors (e.g. impaired glucose tolerance) and/or chronic medical conditions such as heart disease, diabetes, hypertension, or obesity (E. Teixeira-Lemos, Nunes, F. Teixeira, & Reis, 2011). A previous study by Myers et al. (2002) showed that an improvement in fitness over time yielded a better prognosis and a marked reduction in the risk of death from all causes. The result of this 12 weeks study provides evidence that the FET programme can result in fitness gain when compared to the CON group that carried out the usual walking routine. The absolute change of 1 MET may not appear substantial, but the benefit is clinically significant. A meta-analysis conducted by Lee et al. (2011) shows that for each MET increase in exercise capacity is associated with a 15% reduction in risk of all-cause mortality and a 13% reduction in the future risk of CVD and CHD events. Fit individuals have lower all-cause and CVD mortality risk than unfit counterparts, regardless of adiposity classification and medical conditions. Thus, the improvement in exercise capacity showed in our FET group will result in a better prognosis for these individuals.

While the FET group's triglycerides concentration significantly reduced ($p<0.05$) at post-training, no significant change was observed in total cholesterol, HDL-C, and LDL-C. It is speculated that the change in triglycerides concentration may have been attributed to high energy expenditure during the exercise training. The FET group performed exercise in a circuit manner that requires major muscle groups to work in a coordinated fashion, which yielded high energy expenditure and higher fatty acid oxidation. Similarly, a study by Westcott (2012) showed that the reduction in triglyceride concentration is related to sufficient energy expenditure and previous level of physical activity, which is inconsistent with our findings. The participants in the FET group were sedentary upon entering the study; thus, engaging in a prescribed functional exercise training would have increased their activity level from sedentary to active, which may explain the observed reduction in triglycerides concentration.

In contrast, the CON group exhibited significant changes in HDL-C ($p<0.05$) at the end of the 12 weeks. It is widely accepted that HDL-C is inversely correlated with heart disease, and the improvement of HDL-C is related to the volume of physical activity and exercise (Durstine, Grandjean, Cox, & Thompson, 2002). The participants in the CON group were instructed to carry out their usual walking routine daily. It is possible that these individuals were walking in greater quantities, which resulted in the HDL-C change during the study. Our finding agrees with that of Koba et al. (2011), who showed that HDL-C change has a positive correlation with the amount of walking distance per week, and it increases in a dose-dependent manner.

In the current study, the body weight, BMI, waist circumference, fasting plasma insulin, 2-hrs glucose, and glucose AUC of the FET group were significantly decreased ($p<0.05$) at 12 weeks. However, when the absolute changes in these parameters were compared between the two groups, the FET group shows significant reductions in body weight, BMI, waist circumference, 2-hrs glucose, and glucose AUC ($p<0.05$). The reduction in 2-hrs glucose and glucose AUC (Figure 2) is postulated to be related to the body weight reduction and the decrease in waist circumference. Our result is consistent with that of McNeilly et al. (2012), in which the research group discovered that weight loss through moderate exercise training resulted in a reduction in blood biomarkers for cardiovascular risks. In our study, weight loss induces changes in many cardiometabolic parameters and improved insulin sensitivity which helps to lower the glucose appearance in the blood. The Diabetes Prevention Programme (DPP) (2002) showed that a 7% reduction in body weight from baseline has a significant impact on the glucose metabolism in prediabetes. The data from O'Gorman et al. (2006) showed that acute exercise training improves GLUT-4 response, which facilitates the glucose transport into the cell, which lowers blood glucose. Our participants in the FET group performed exercise for 12 weeks, which could have improved the GLUT-4 effectiveness that would result in lower 2-hrs glucose and glucose AUC ($p<0.05$).

Additionally, the fasting plasma insulin in the FET group significantly changed ($p<0.05$) at the end of the study, but the magnitude of change was not statistically significant compared to the CON group. It is speculated that functional exercise training exerted a certain effect on plasma insulin response. Our finding is supported by the study conducted by Rice, Janssen, Hudson, and Ross (1999), which concluded that physical training exerts a lowering effect on insulin concentrations in the plasma in obesity. Decreased plasma insulin concentration after physical training could be due to either decreased insulin secretion or an increase in peripheral clearance of insulin rate, or both (Eriksson et al., 1998; Pratley et al., 2000).

Limitation

The authors understand that the small sample size is a limitation of this study. It is difficult to find and recruit obese individuals with IGT that are not taking any medications or have other comorbidities. Despite the small sample size, the study was able to show the effects of 12 weeks of functional exercise training.

Conclusion

This study illustrates that 12 weeks of functional exercise training performed in a circuit manner is an effective means of inducing body weight change, increasing exercise capacity, and alters the cardiometabolic variables such as triglycerides HDL-C, 2-hrs glucose, and glucose AUC. It appears that the functional exercise training programme can be utilized as a cost-effective therapeutic means to help manage obesity and impaired glucose metabolism. Exercise training remains a cornerstone intervention for blood glucose management. The functional exercise training programme can be an alternative form of exercise for obese individuals.

Acknowledgements

The authors would like to thank all the participants for volunteering for the study. We would like to thank the 90th Anniversary of Chulalongkorn University Fund (Ratchadaphiseksomphot Endowment Fund, GCUGR1125631051D No. 51) and the Faculty of Sports Science Research Fund, G505_2, for providing funding support, which made this study possible. Lastly, we also like to thank the Foundation for Diabetes Care Development for partially funding this research.

Conflict of Interest

The authors declare the absence of conflict of interest.

References

Alberti, K. G. M. M. (2007). Screening and diagnosis of prediabetes: where are we headed?. *Diabetes, Obesity and Metabolism*, *9*, 12-16.

American College of Sports Medicine. (2018). *ACSM's Health-Related Physical Fitness Assessment Manual.* Fifth Edition.

American Diabetes Association. (2002). The prevention or delay of type 2 diabetes. *Diabetes Care*, *25*(4), 742-749.

American Diabetes Association. (2018). Standards of medical care in diabetes—2018 abridged for primary care providers. *Clinical diabetes: a publication of the American Diabetes Association*, *36*(1), 14.

Brill, P. A. (2008). Exercise your independence: functional fitness for older adults. In *Journal of Aging and Physical Activity*, *16*, S88-S89.

DeFronzo, R. A., & Abdul-Ghani, M. (2011). Assessment and treatment of cardiovascular risk in prediabetes: impaired glucose tolerance and impaired fasting glucose. *The American Journal of Cardiology*, *108*(3), 3B-24B.

Diabetes Prevention Program (DPP) Research Group. (2002). The Diabetes Prevention Program (DPP): description of lifestyle intervention. *Diabetes Care*, *25*(12), 2165-2171.

Durstine, J. L., Grandjean, P. W., Cox, C. A., & Thompson, P. D. (2002). Lipids, lipoproteins, and exercise. *Journal of Cardiopulmonary Rehabilitation and Prevention*, *22*(6), 385-398.

Eriksson, J., Tuominen, J., Valle, T., Sundberg, S., Sovijärvi, A., Lindholm, H., ... & Koivisto, V. (1998). Aerobic endurance exercise or circuit-type resistance training for individuals with impaired glucose tolerance?. *Hormone and Metabolic Research*, *30*(01), 37-41.

Hawley, J. A. (2004). Exercise as a therapeutic intervention for the prevention and treatment of insulin resistance. *Diabetes/Metabolism Research and Reviews*, *20*(5), 383-393.

Koba, S., Tanaka, H., Maruyama, C., Tada, N., Birou, S., Teramoto, T., & Sasaki, J. (2011). Physical activity in the Japan population: association with blood lipid levels and effects in reducing cardiovascular and all-cause mortality. *Journal of Atherosclerosis and Thrombosis*, *18*(10), 833-845.

Lagally, K. M., Cordero, J., Good, J., Brown, D. D., & McCaw, S. T. (2009). Physiologic and metabolic responses to a continuous functional resistance exercise workout. *The Journal of Strength & Conditioning Research*, *23*(2), 373-379.

Lee, D. C., Sui, X., Artero, E. G., Lee, I. M., Church, T. S., McAuley, P. A., ... & Blair, S. N. (2011). Long-term effects of changes in cardiorespiratory fitness and body mass index on all-cause and cardiovascular disease mortality in men: The Aerobics Center Longitudinal Study. *Circulation*, *124*(23), 2483-2490.

Matthews, D. R., Hosker, J. P., Rudenski, A. S., Naylor, B. A., Treacher, D. F., & Turner, R. C. (1985). Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*, *28*(7), 412-419.

McNeilly, A. M., McClean, C., Murrhy, M., McEneny, J., Trinick, T., Burke, C., Duly, E., McLaughlin, J., & Davison, G. (2012). Exercise training and impaired glucose tolerance in obese humans. *Journal of Sports Sciences, 30*(8), 725-732.

Myers, J., Prakash, M., Froelicher, V., Do, D., Partington, S., & Atwood, J. E. (2002). Exercise capacity and mortality among men referred for exercise testing. *New England Journal of Medicine*, *346*(11), 793-801.

O'gorman, D. J., Karlsson, H. K. R., McQuaid, S., Yousif, O., Rahman, Y., Gasparro, D., & Nolan, J. J. (2006). Exercise training increases insulin-stimulated glucose disposal and GLUT4 (SLC2A4) protein content in patients with type 2 diabetes. *Diabetologia*, *49*(12), 2983-2992.

Pratley, R. E., Hagberg, J. M., Dengel, D. R., Rogus, E. M., Muller, D. C., & Goldberg, A. P. (2000). Aerobic exercise training-induced reductions in abdominal fat and glucose-stimulated insulin responses in middle-aged and older men. *Journal of the American Geriatrics Society*, *48*(9), 1055-1061.

Rai, J. K., Wadhwani, J. L., Sharma, M., & Dubey, T. N. (2019). Prevalence of impaired glucose tolerance in different grades of obesity. *International Journal of Advances in Medicine*, *6*(5), 1520.

Rehn, T. A., Winett, R. A., Wisløff, U., & Rognmo, Ø. (2013). Increasing physical activity of high intensity to reduce the prevalence of chronic diseases and improve public health. *The Open Cardiovascular Medicine Journal*, *7*, 1.

Rice, B., Janssen, I., Hudson, R., & Ross, R. (1999). Effects of aerobic or resistance exercise and/or diet on glucose tolerance and plasma insulin levels in obese men. *Diabetes Care*, *22*(5), 684-691.

Shaikh, A. & Mondal, S. (2012). Effect of functional training on physical fitness components on college male students-A pilot study. *Journal of Humanities and Social Science, 1*(2), 1-5

Sigal, R. J., Kenny, G. P., Wasserman, D. H., Castaneda-Sceppa, C., & White, R. D. (2006). Physical activity/exercise and type 2 diabetes: a consensus statement from the American Diabetes Association. *Diabetes Care*, *29*(6), 1433-1438.

Silva-Grigoletto, M. E. D., Brito, C. J., & Heredia, J. R. (2014). Functional training: functional for what and for whom?. *Revista Brasileira de Cineantropometria & Desempenho Humano*, *16*(6), 714-719.

Slentz, C. A., Bateman, L. A., Willis, L. H., Granville, E. O., Piner, L. W., Samsa, G. P., ... & Kraus, W. E. (2016). Effects of exercise training alone vs a combined exercise and nutritional lifestyle intervention on glucose homeostasis in prediabetic individuals: A randomized controlled trial. *Diabetologia*, *59*(10), 2088-2098.

Swindell, N., Mackintosh, K., McNarry, M., Stephens, J. W., Sluik, D., Fogelholm, M., & Poppitt, S. D. (2018). Objectively measured physical activity and sedentary time are associated with cardiometabolic risk factors in adults with prediabetes: The Preview Study. *Diabetes Care*, *41*(3), 562-569.

Tapp, R. J., Dunstan, D. W., Phillips, P., Tonkin, A., Zimmet, P. Z., Shaw, J. E., & AusDiab Study Group. (2006). Association between impaired glucose metabolism and quality of life: results from the Australian diabetes obesity and lifestyle study. *Diabetes Research and Clinical Practice*, *74*(2), 154-161.

Teixeira-Lemos, E., Nunes, S., Teixeira, F., & Reis, F. (2011). Regular physical exercise training assists in preventing type 2 diabetes development: focus on its antioxidant and anti-inflammatory properties. *Cardiovascular Diabetology*, *10*(1), 12.

Venables, M. C., & Jeukendrup, A. E. (2009). Physical inactivity and obesity: links with insulin resistance and type 2 diabetes mellitus. *Diabetes/Metabolism Research and Reviews*, *25*(S1), S18-S23.

Vogeser, M., König, D., Frey, I., Predel, H. G., Parhofer, K. G., & Berg, A. (2007). Fasting serum insulin and the homeostasis model of insulin resistance (HOMA-IR) in the monitoring of lifestyle interventions in obese persons. *Clinical Biochemistry*, *40*(13-14), 964-968.

Westcott, W. L. (2012). Resistance training is medicine: effects of strength training on health. *Current Sports Medicine Reports*, *11*(4), 209-216.

Whitehurst, M. A., Johnson, B. L., Parker, C. M., Brown, L. E, & Ford, A. M. (2005). The benefits of a functional exercise circuit for older adults. *Journal of Strength and Conditioning Research, 19*(3), 647-651.

World Health Organization. (2000). The Asia-Pacific perspective: redefining obesity and its Treatment.