

Impact of Aerobic Versus Resistance Exercise Training on Glucose Control and Biomarkers of Oxidative Stress among Saudi Patients with Type 2 Diabetes

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Abstract

Background: Oxidative stress is important in pathogenesis of diabetes complications. Limited research available regarding role of exercise in alleviation of oxidative stress in type 2 diabetes mellitus (T2DM).

Objective: The target of this study was compared response of insulin resistance and oxidative stress to aerobic versus resisted exercise training in T2DM patients.

Material and Methods: Eighty T2DM patients, the range of age ranged was 45 to 57 year and body mass index was 30 to 36kg/m². Smoking, renal failure, heart failure, respiratory failure, hepatitis and pregnancy were the exclusion criteria. Participants randomly assigned equally into group (A) received aerobic treadmill exercise and group (B) received resistance exercise for 3 months.

Results: Following aerobic and resistance exercises, there was significant reduction in the mean values of Homeostasis Model Assessment-Insulin Resistance Index (HOMA-IR), conjugated dienes (CD) and malondialdehyde (MDA), in addition to significant elevation in the mean values of the quantitative insulin-sensitivity check index (QUICKI), glutathione peroxidase (GPx), superoxide dismutase (SOD) and glutathione (GSH). However, significant differences found between both groups after training.

Conclusion: Aerobic exercise is superior to resistance exercise in modulating insulin resistance and oxidative stress in type 2 diabetes patients.

Keywords: Aerobic exercise; Diabetes mellitus; Resisted exercises; Oxidative stress

Abbreviations: T2DM: Type 2 Diabetes Mellitus; QUICKI: Quantitative Insulin-Sensitivity Check Index; SOD: Superoxide Dismutase; HOMA-IR: Homeostasis Model Assessment-Insulin Resistance Index

Introduction

Diabetes mellitus is a worldwide medical problem affects about 6% world population which is expected to reach more than 550 million 2030 [1]. Many system dysfunctions are associated with diabetes include renal, cardiac, eye, nerve and blood vessels [2]. Hyperglycemia induces oxidative stress and inflammation [3]. Oxidative stress is usually associated with diabetes because of abnormal glucose control [4-7]. However; oxidative stress is one of the main cause of diabetic complications [8-11]. In the other hand, metabolic control enhance antioxidant defense system in type 2 diabetes patients (T2DM) [12]. Hyperglycemia seems to cause an imbalance between oxidant and antioxidant systems in T2DM patients [13]. Physical activity reduces rate of mortality and morbidity in diabetic individuals [14-16]. Therefore, regular exercise is an effective therapeutic strategy for T2DM [17]. Aerobic exercise improves metabolic control and reduces the cardiovascular disease risk [18]. Physical activity ameliorate insulin resistance and oxidative stress [19-23]. Limited studies available regarding the more appropriate exercise modulates the metabolic control and oxidative stress of T2DM patients, so that the target of present study was compare response of insulin resistance and oxidative stress to aerobic versus resisted exercise training in T2DM patients..

Materials and Methods

Subjects

Eighty T2DM patients, the mean of age ranged was 49.71-±7.28 year and body mass index was 32.87±4.12kg/m². Smoking, renal failure, heart failure, respiratory failure, hepatitis and pregnancy were the exclusion criteria. Participants randomly assigned equally into group (A) received aerobic treadmill exercise and group (B) received resistance exercise for 3 months. Informed written consent was signed by all participants.

Measurements

Insulin and insulin resistance assessment: Serum insulin was measured using an insulin kit (Roche Diagnostics, Indianapolis, IN, USA) by a cobas immunoassay analyzer (Roche Diagnostics). However, formula used to assess insulin resistance was homeostasis model assessment (HOMA-IR). HOMA-IR=[fasting blood glucose (mmol/l)_fasting insulin (mIU/ml)]/22.5 [24]. While the formula used to assess insulin sensitivity was quantitative insulin-sensitivity check index (QUICKI) with the formula: QUICKI=1/[log(insulin)+log(glucose)] [25].

Oxidative stress markers and anti-oxidant status assessment: The method described by Esterbauer et al used to assess plasma level of malondialdehyde (MDA) [26]. While the method described by Weckbecker and Cory used to assess Glutathione (GSH) level [27]. In addition, the method described by Masnini used to measure

glutathione peroxidase (GPx) and superoxide dismutase (SOD) [28].

Procedures

Group (A): received treadmill aerobic exercise (Enraf Nonium, Model display panel Standard, NR 1475.801, Holand) following American College of Sports Medicine recommendation [29]. Participants conducted training intensity of 60-80% of maximum heart rate for 30 minutes, 3 sessions/week.

Group (B): received resisted exercises using resistance gym machines (Nautilus Sports/Medical Industries, Independence, VA). Participants conducted training intensity of 60 and 80% of their one maximal repetition weight (1-RM) for 30 minutes, 3 sessions/week [30].

Results

Participants' criteria before the study did not revealed significant differences between both groups as shown in Table 1. Following aerobic and resistance exercises, there was significant reduction in the mean values of Homeostasis Model Assessment-Insulin Resistance Index (HOMA-IR), conjugated dienes (CD) and malondialdehyde (MDA), in addition to significant elevation in the mean values of the quantitative insulin-sensitivity check index (QUICKI), glutathione peroxidase (GPx), superoxide dismutase (SOD) and glutathione (GSH) (Tables 2 & 3). However, significant differences found between both groups after training (Table 4).

Table 1: Participants criteria before the study. BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; HDL-C: High-Density Lipoprotein Cholesterol; HBA1c: Glycosylated Hemoglobin.

	Group (A)	Group (B)	Significance
Age (year)	48.92±7.15	49.75±7.83	P<0.05
Gender (male/female)	26/14	29/11	P<0.05
BMI (kg/m ²)	32.84±5.14	32.25±5.11	P<0.05
Duration of diabetes (years)	12.87±4.91	12.56±4.62	P<0.05
SBP (mmHg)	145.16±11.18	143.27±10.32	P<0.05
DBP (mmHg)	89.14±8.35	86.92±8.64	P<0.05
Total Cholesterol (mg/dL)	205.37±18.42	200.13±16.25	P<0.05
HDL-C(mg/dL)	41.63±6.81	44.53±7.15	P<0.05
Triglycerides(mg/dL)	148.11±16.57	144.74±17.13	P<0.05
HBA1c (%)	8.61±1.92	8.21±1.75	P<0.05
Glucose (mmol/L)	5.44±0.98	5.26±0.87	P<0.05
Insulin (pmol/L)	19.65±5.10	18.37±4.88	P<0.05

Table 2: Mean value and significance of MDA, GSH, GPX, SOD, QUICKI and HOMA-IR in group (A) before and at the end of the study. MDA: Malondialdehyde; GSH: Glutathione; GPX: Glutathione Peroxidase; SOD: Superoxide Dismutase; QUICKI: The Quantitative Insulin-Sensitivity Check Index; HOMA-IR: Homeostasis Model Assessment-Insulin Resistance Index; (*) indicates a significant difference, $P < 0.05$.

	Mean+SD		T-value	Significance
	Pre	Post		
MDA (nM/mL)	0.30±0.08	0.17±0.05*	7.24	$P < 0.05$
GSH (nM/mL)	3.45±0.74	4.51±0.95*	6.83	$P < 0.05$
GPX (UI/mL)	2.86±0.42	3.62±0.57*	6.92	$P < 0.05$
SOD (UI/mL)	118.25±17.63	98.85±12.13*	7.43	$P < 0.05$
QUICKI	0.119±0.017	0.155±0.024*	6.85	$P < 0.05$
HOMA-IR	5.17±0.95	3.95±0.67*	6.27	$P < 0.05$

Table 3: Mean value and significance of MDA, GSH, GPX, SOD, QUICKI and HOMA-IR in group (B) before and at the end of the study. MDA: Malondialdehyde; GSH: Glutathione; GPX: Glutathione Peroxidase; SOD: Superoxide Dismutase; QUICKI: The Quantitative Insulin-Sensitivity Check Index; HOMA-IR: Homeostasis Model Assessment-Insulin Resistance Index; (*) indicates a significant difference, $P < 0.05$.

	Mean+SD		T-value	Significance
	Pre	Post		
MDA (nM/mL)	0.34±0.07	0.25±0.06	3.67	$P < 0.05$
GSH (nM/mL)	3.21±0.68	3.84±0.72	3.45	$P < 0.05$
GPX (UI/mL)	2.95±0.47	3.36±0.53	3.71	$P < 0.05$
SOD (UI/mL)	121.34±18.12	108.45±13.44	4.52	$P < 0.05$
QUICKI	0.120±0.019	0.134±0.023	3.37	$P < 0.05$
HOMA-IR	5.31± 0.96*	4.32±0.74	3.54	$P < 0.05$

Table 4: Mean value and significance of MDA, GSH, GPX, SOD, QUICKI and HOMA-IR in group (A) and group (B) at the end of the study. MDA: Malondialdehyde; GSH: Glutathione; GPX: Glutathione Peroxidase; SOD: Superoxide Dismutase; QUICKI: The Quantitative Insulin-Sensitivity Check Index; HOMA-IR: Homeostasis Model Assessment-Insulin Resistance Index; (*) indicates a significant difference, $P < 0.05$.

	Mean+SD		T-value	Significance
	Group (A)	Group (B)		
MDA (nM/mL)	0.17±0.05*	0.25±0.06	3.42	$P < 0.05$
GSH (nM/mL)	4.51±0.95*	3.84±0.72	3.61	$P < 0.05$
GPX (UI/mL)	3.62±0.57*	3.36±0.53	3.55	$P < 0.05$
SOD (UI/mL)	98.85±12.13*	108.45±13.44	3.91	$P < 0.05$
QUICKI	0.155±0.024*	0.134±0.023	3.63	$P < 0.05$
HOMA-IR	3.95±0.67*	4.32±0.74	3.81	$P < 0.05$

Discussion

Oxidative stress is important in pathogenesis of diabetes complications [31,32]. The target of this study was compare response of insulin resistance and oxidative stress to aerobic versus resisted exercise training in T2DM patients. Concerning insulin resistance, both aerobic exercise and resisted exercise training significantly improved insulin resistance, these results agreed with Bacchi et al. stated that 4 months of aerobic and resistance exercises improved insulin sensitivity in T2DM with nonalcoholic

fatty liver [33]. However, Angelico et al. [34] reported that up to 10% weight loss of patients with metabolic syndrome modulated insulin resistance [34]. Concerning oxidative stress, our results proved that both aerobic and resistance exercises mean values of MDA, SOD and HOMA-IR were significantly decreased, while the mean values of GSH, GPx and QUICKI were significantly increased in T2DM patients. These results agreed with previous studies that stated that 6 months of aerobic exercise increased GSH in T2DM individuals [35,36]. However, another study reported that 24

weeks of resistance exercise ameliorated oxidative stress in obese individuals [37]. In addition, Oliveira et al. [38] confirmed that 4 months of aerobic exercise improved oxidative stress markers among T2DM subjects [38]. However, Vinetti et al. [39] found that 12 months of combined aerobic, resistance and flexibility training modulated oxidative stress among T2DM patients [39]. While Farinha et al. [40] mentioned that 3 months of treadmill exercise improved oxidative stress in women with metabolic syndrome [40]. Similarly, Nojima et al. [41] stated that 12 months of aerobic exercise improved metabolic control and oxidative stress markers among T2DM patients [41]. Moreover, Gordon et al. [42] proved that 3 months of Hatha yoga exercise improved glucose control, blood lipid profile and oxidative stress markers among T2DM patients [42].

The possible mechanisms for modulating oxidative stress following exercise training may include improvement in glucose control [43-46], also improved insulin sensitivity in target tissues is the second possible mechanism for reducing oxidative stress by aerobic and resistance exercise training [47,48]. The effects of aerobic versus resistance exercise to activate anti-aging genes that regulate insulin resistance and metabolic control is of specific interest to Type 2 diabetes. The anti-aging gene Sirtuin 1 (oxidative stress) is activated by exercise and calorie restriction with increased Sirtuin 1 levels in the blood plasma. Plasma sirtuin 1 levels have been shown to be reduced in NAFLD, obesity, diabetes and other chronic diseases. Aerobic and resistance exercise that maintain metabolic control is possibly linked to activation of Sirtuin 1 and the reversal of liver steatosis/early NAFLD [49-51].

Conclusion

Aerobic exercise is superior to resistance exercise in modulating insulin resistance and oxidative stress in type 2 diabetes patients.

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