American Research Journal of Cardiology

Volume 4, Issue 1, 1-6 Pages Research Article | Open Access ISSN (Online)- 2575-7601 DOI ; 10.21694/2575-7601.21001



Impact of aerobic versus resistance exercise training on glucose control and biomarkers of oxidative stress among Saudi patients with type 2 diabetes

Fadwah M. Al-Sharif *and Shehab M. Abd El-Kader**

*Department of Medical Laboratory Technology, Faculty of Applied Medical Sciences, King Abdulaziz University, Jeddah, 21589,Saudi Arabia.

**Department of Physical Therapy, Faculty of Applied Medical Sciences, King Abdulaziz University, Jeddah, 21589, Saudi Arabia.

ABSTRACT

Background: Type 2 diabetes (T2DM) is a chronic and progressive disease that is strongly associated with all-cause and cardiovascular mortality. Oxidative stress plays a key role in both initiation and complications of T2DM. There is limitation in clinical studies have addressed the ideal exercise intensity that efficiently modulates the insulin resistance and abnormal oxidative stress markers among type 2 diabetic patients.

Objective: The present study was designed to examine aerobic versus resisted exercise training effects upon insulin resistance and oxidative stress markers among type 2 diabetic patients.

Material and Methods: One hundred obese patients with type 2 diabetes mellitus, their age ranged from 45-57 years and their body mass index ranged from 30-36 kg/m2 were equally assigned into 2 groups: The first group received aerobic exercise training in the form of treadmill aerobic exercises where, the second group received resisted exercise training for 12 weeks.

Results: The mean values of Homeostasis Model Assessment-Insulin Resistance Index)HOMA-IR(, conjugated dienes (CD) and malondialdehyde (MDA) were significantly decreased, while the mean values of the quantitative insulin-sensitivity check index (QUICKI), glutathione peroxidase (GPx), superoxide dismutase (SOD) and glutathione (GSH) were significantly increased in patients of group (A) as a result of aerobic exercise training and group (B) as a result of resisted exercise training. There were significant differences between mean levels of the investigated parameters in group (A) and group (B) after treatment with more changes in patients received aerobic exercise training.

Conclusion: The current study provides evidence that aerobic exercise is more appropriate than resisted exercise training in modulating insulin resistance and oxidative stress among type 2 diabetic patients.

Keywords: Aerobic Exercise; Resisted Exercises; Type 2 Diabetes Mellitus; Oxidative Stress.

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 as a result 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 ideal exercise intensity that efficiently modulates the metabolic control and oxidative stress of T2DM patients, therefore this study was designed to examine aerobic versus resisted exercise training effects upon insulin resistance and oxidative stress markers among T2DM patients.



Materials and Methods

Subjects

One hundred obese T2DM patients, their age mean was 49.71 ± 6.28 year and their body mass index (BMI) mean was 32.87 ± 3.12 Kg/m2. Smoking, renal failure, heart failure, respiratory failure, hepatitis and pregnancy were the exclusion criteria. Participants were assigned into two groups; group (A) received aerobic exercise training on treadmill. While, group (B) received resisted exercise training. Informed written consent was signed by all participants.

Measurements

A. Measurement of insulin and insulin resistance: An insulin kit (Roche Diagnostics, Indianapolis, IN, USA) using a cobas immunoassay analyzer (Roche Diagnostics) was used to measure serum insulin. However, homeostasis model assessment (HOMA-IR). HOMA-IR = [fasting blood glucose (mmol/l) _ fasting insulin (mIU/ml)]/22.5 was used as a formula to assess insulin resistance [24]. While, the quantitative insulin-sensitivity check index (QUICKI) using the formula: QUICKI=1/[log(insulin) + log(glucose)] was used as a formula to assess insulin sensitivity [25]. **oxidant status:** Plasma level of malondialdehyde (MDA) was measured by the method described by Esterbauer et al [26]. Glutathione (GSH) level was measured by adopting the method described by Weckbecker and Cory[27], glutathione peroxidase (GPx) and superoxide dismutase (SOD) were was estimated according the method described by Masnini [28].

Procedures

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

2. Group (B) received resistance exercises on some 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

The baseline characteristics of the participants revealed no significant differences between both groups as shown in table (1).

	Group (A)	Group (B)	Significance
Age (year)	48.92 ± 6.15	49.75 ± 5.83	P <0.05
Gender (male/female)	31/19	34/16	P <0.05
BMI (kg/m ²)	32.73 ± 3.14	33.25 ± 3.11	P<0.05
Duration of diabetes (years)	13.87 ± 3.91	12.56 ± 4.12	P<0.05
SBP (mmHg)	145.16 ± 11.18	143.27 ± 10.32	P <0.05
DBP (mmHg)	89.14 ± 6.35	86.92 ± 7.64	P<0.05
Total Cholesterol (mg/dL)	205.37 ± 18.42	200.13 ± 16.25	P <0.05
HDL-C(mg/dL)	41.63 ± 6.71	44.53 ± 7.15	P <0.05
Triglycerides(mg/dL)	148.11 ± 12.57	144.74 ± 10.13	P<0.05
HBA1c (%)	8.61 ± 2.83	8.21 ± 2.54	P<0.05
Glucose (mmol/L)	5.44 ±1.28	5.26 ± 1.14	P <0.05
Insulin (pmol/L)	20.65 ±5.10	18.37 ± 4.88	P<0.05

Table (1): Baseline characteristics of all participants.

BMI: Body Mass Index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HDL-C: High density lipoprotein cholesterol; HBA1c: glycosylated hemoglobin.



B. Measurement of oxidative stress markers and anti-

The mean values of MDA, SOD and HOMA-IR were significantly decreased, while the mean values of GSH, GPx and QUICKI were significantly increased in patients of group (A) as a result of aerobic exercise training and group (B) as a result of resisted exercise training(Table 2 and 3). However,

there were significant differences between mean levels of the investigated parameters in group (A) and group (B) after treatment with more changes in patients received aerobic exercise training (Table 4).

	Mean +SD		Typhys	Significance
	Pre	Post	1-value	Significance
MDA (nM/mL)	0.30 ± 0.08	$0.17\pm0.05*$	7.24	P<0.05
GSH (nM/mL)	3.45 ± 0.74	$4.51 \pm 0.86*$	6.83	P<0.05
GPX (UI/mL)	2.86 ± 0.42	$3.62 \pm 0.57*$	6.92	P <0.05
SOD (UI/mL)	118.25 ± 17.63	98.85 ± 12.13*	7.43	P<0.05
QUICKI	0.123 ± 0.018	$0.155 \pm 0.024*$	6.85	P <0.05
HOMA-IR	5.17 ± 1.65	$3.95 \pm 1.48*$	6.27	P <0.05

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.

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.

	Mean +SD		Typha	Simifaanaa
	Pre	Post	1-value	Significance
MDA (nM/mL)	0.34 ± 0.07	0.25 ± 0.06	3.67	P<0.05
GSH (nM/mL)	3.21 ± 0.68	3.79 ± 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 ± 1.76*	4.62 ± 1.52	3.54	P <0.05

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.

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.



	Mean +SD		Tarahas	S::6
	Group (A)	Group (B)	1-value	Significance
MDA (nM/mL)	$0.17\pm0.05*$	0.25 ± 0.06	3.42	P<0.05
GSH (nM/mL)	$4.51\pm0.86*$	3.79 ± 0.72	3.61	P<0.05
GPX (UI/mL)	$3.62\pm0.57*$	3.36 ± 0.53	3.55	P <0.05
SOD (UI/mL)	98.85 ±12.13*	$108.45 \pm \! 13.44$	3.91	P<0.05
QUICKI	$0.142 \pm 0.021 *$	0.134 ± 0.023	3.43	P <0.05
HOMA-IR	$3.95 \pm 1.48*$	4.62 ± 1.52	3.77	P <0.05

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.

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.

Discussion

Oxidative stress (OS) plays a major role in pathogenesis of T2DM complications [31,32]. Therefore this study was designed to examine aerobic versus resisted exercise training effects upon insulin resistance and oxidative stress markers among 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.* reported that 5%-10% weight loss of patients with metabolic syndrome modulated insulin resistance [34].

Concerning oxidative stress, results of the present study 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. confirmed that 4 months of aerobic exercise improved oxidative stress markers among T2DM subjects [38]. However, Vinetti et al. found that 12 months of combined aerobic, resistance and flexibility training modulated oxidative stress among T2DM patients[39]. While, Farinha et al. mentioned that 3 months of treadmill exercise improved oxidative stress in women with metabolic syndrome [40]. Similarly, Nojima et al. stated that 12 months of aerobic exercise improved metabolic control and oxidative stress markers among T2DM patients [41]. Moreover, Gordon et al. 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 modulation of oxidative stress markers following exercise training may include improvement in glucose control[43-46], also improved insulin sensitivity in target tissues is the second possible mechanism for reduction in oxidative stress by aerobic and resistance exercise training [47,48].

Conclusion

Aerobic exercise is more appropriate than resisted exercise training in modulating insulin resistance and oxidative stress among type 2 diabetic patients.

Acknowledgment

This project was funded by the Deanship of Scientific Research (DSR), King Abdulaziz University, Jeddah, under grant no. (G-5-290-40). The authors, therefore, acknowledge with thanks DSR technical and financial support.

References

- 1. IDF Diabetes Atlas. http://www.Idf.Org/diabetesatlas/5e/the-globalburden; 2011.
- 2. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2010;33: S62–S69.
- Dandona, P., Mohanty, P., Chaudhuri, A., Garg, R., & Aljada, A. (2005). Insulin infusion in acute illness. The Journal of Clinical Investigation, 115, 2069–2072.
- Monnier, L., Mas, E., Ginet, C., Michel, F., Villon, L., Cristol, J. P., & Colette, C. (2006). Activation of oxidative stress by acute glucose fluctuations compared with sustained chronic hyperglycemia in patients with type 2 diabetes. Journal of the American Medical Association, 295, 1681–1687.
- Paravicini, T. M., & Touyz, R. M. (2008). Nadph oxidases, reactive oxygen species, and hypertension: clinical implications and therapeutic possibilities. Diabetes Care, 31(Suppl. 2), S170–S180.
- Ceriello A, Motz E. Is oxidative stress the pathogenic mechanism underlying insulin resistance, diabetes, and cardiovascular disease? The common soil hypothesis revisited. Arterioscler ThrombVasc Biol 2004;24:816–823
- 7. Robertson RP, Harmon J, Tran PO, Tanaka Y, Takahashi H. Glucose toxicity in b-cells: type 2 diabetes, good radicals gone bad, and the



glutathione connection. Diabetes 2003;52:581-587

- Matough FA, Budin SB, Hamid ZA, Alwahaibi N, Mohamed J. The role of oxidative stress and antioxidants in diabetes complications. Sultan Qaboos Univ J 2012;12:5e-18.
- Pan HZ, Zhang L, Guo MY, Sui H, Li H, Wu WH, Qu NQ, Liang MH, Chang D. The oxidative stress status in diabetes mellitus and diabetes nephropathy. Acta Diabetol 2010;47:71-6.
- Odum EP, Ejilemele AA, Wakwe VC. Antioxidant status of type 2 diabetic patients in Port Harcourt, Nigeria. Niger J Clin Pract 2012;15:55-8.
- Shi YC, Pan TM. Red mold, diabetes, and oxidative stress: A review. Appl Microbiol Biotechnol 2012;94:47-55.
- Lima V, Sampaio F, Bezerra D. Parameters of glycemic control and their relationship with zinc concentrations in blood and with superoxide dismutase enzyme activity in type 2 diabetes patients. Arq Bras Endocrinol Metab 2011;55:701-7.
- Likidlilid A, Patchanans N, Peerapatdit T, Sriratanasathavorn C. Lipid peroxidation and antioxidant enzyme activities in erythrocytes of type 2 diabetes patients. J Med Assoc Thai 2010;93:682-93.
- J. Lindstr"om, A. Louheranta, M.Mannelin et al., "The finnish diabetes prevention study (DPS): lifestyle intervention and 3- year results on diet and physical activity," Diabetes Care, vol. 26, no. 12, pp. 3230–3236, 2003.
- S. Kodama, S. Tanaka, K. Saito et al., "Effect of aerobic exercise training on serum levels of high-density lipoprotein cholesterol: a meta-analysis," Archives of Internal Medicine, vol. 167, no. 10, pp. 999–1008, 2007.
- T. Saito et al., "Lifestyle modification and prevention of type 2 diabetes in overweight Japanese with impaired fasting glucose levels: a randomized controlled trial," Archives of Internal Medicine, vol. 171, no. 15, pp. 1352–1360, 2011.
- J. Lindström, P. Ilanne-Parikka, M. Peltonen et al., "Sustained reduction in the incidence of type 2 diabetes by lifestyle intervention: follow-up of the Finnish Diabetes Prevention Study," The Lancet, vol. 368, no. 9548, pp. 1673–1679, 2006.
- S. S. Bassuk and J. E. Manson, "Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease," Journal of Applied Physiology, vol. 99, no. 3, pp. 1193–1204, 2005.
- N. Ahmadi, S. Eshaghian, R. Huizenga, K. Sosnin, R. Ebrahimi, and R. Siegel, "Effects of intense exercise and moderate caloric restriction on cardiovascular risk factors and inflammation," American Journal of Medicine, vol. 124, no. 10, pp. 978–982, 2011.
- C. A. Slentz, C. J. Tanner, L. A. Bateman et al., "Effects of exercise training intensity on pancreatic β-cell function," Diabetes Care, vol. 32, no. 10, pp. 1807–1811, 2009.
- M. F. Belotto, J.Magdalon, H. G. Rodrigues et al., "Moderate exercise improves leucocyte function and decreases inflammation in diabetes," Clinical and Experimental Immunology, vol. 162, no. 2, pp. 237–243, 2010.
- L. Bjork, N. T. Jenkins, S. Witkowski, and J. M. Hagberg, "Nitro-oxidative stress biomarkers in active and inactive men," International Journal of Sports Medicine, vol. 33, no. 4, pp. 279–284, 2012.
- 23. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2010;33(Suppl. 1):S62–9.
- Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta cell function from plasma FBS and insulin concentrations in man. Diabetologia 1985;28:412–9.
- Katz A, Nambi SS, Mather K, Baron DA, Follman DA, Sullivan F, et al. Quantitative insulin sensitivity check index: a simple, accurate method for assessing insulin sensitivity in humans. J Clin Endocrinol Metab 2000;85:2402–2410.

- Esterbauer H, Gebicki J, Puhl H, Jürgens G. The role of lipid peroxidation and antioxidants in oxidative modification of LDL. Free Radie Biol Med 1992;13:341-90.
- Weckbecker G, Cory JG. Ribonucleotide reductase activity and growth of glutathione-depleted mouse leukemia L 1210 cells in vitro. Cancer Lett 1988; 40:257-64.
- 28. Masnini M. Determination of superoxide dismutase activity with an electrochemical oxygen probe. Analyt Chim Acta 1988;211:195-204.
- 29. Deibert P, Konig D, Vitolins MZ, et al. (2007): Effect of a weight loss intervention on anthropometric measures and metabolic risk factors in preversus postmenopausal women. J Nutr, 6:31.
- Hagey AR, Warren MP. (2008): Role of exercise and nutrition in menopause. Clin Obstet Gynecol, 51:627–41.
- A. Ceriello, K. Esposito, L. Piconi et al., "Oscillating glucose is more deleterious to endothelial function and oxidative stress than mean glucose in normal and type 2 diabetic patients," Diabetes, vol. 57, no. 5, pp. 1349– 1354, 2008.
- L. E. Fridlyand and L. H. Philipson, "Reactive species and early manifestation of insulin resistance in type 2 diabetes," Diabetes, Obesity and Metabolism, vol. 8, no. 2, pp. 136–145,2006.
- 33. Bacchi E, Negri C, Targher G, Faccioli N, Lanza M, Zoppini G, et al: Both resistance training and aerobic training reduce hepatic fat content in type 2 diabetic subjects with nonalcoholic fatty liver disease (the RAED2 randomized trial). Hepatology 2013; 58: 1287–1295.
- 34. Angelico F, Loffredo L, Pignatelli P, Augelletti T, Carnevale R, Pacella A, Albanese F, Mancini I, Di Santo S, Del Ben M, Violi F. Weight loss is associated with improved endothelial dysfunction via NOX2-generated oxidative stress downregulation in patients with the metabolic syndrome. Intern Emerg Med 2012; 7: 219-227
- 35. G. Lazarevic, S. Antic, T. Cvetkovic, P. Vlahovic, I. Tasic, and V. Stefanovic, "A physical activity programme and its effects on insulin resistance and oxidative defense in obese male patients with type 2 diabetes mellitus," Diabetes and Metabolism, vol. 32, no. 6, pp. 583–590, 2006.
- R. S. Rector, S. O. Warner, Y. Liu et al., "Exercise and diet induced weight loss improves measures of oxidative stress and insulin sensitivity in adults with characteristics of the metabolic syndrome," American Journal of Physiology, vol. 293, no. 2, pp. E500–E506, 2007.
- H. K. Vincent, C. Bourguignon, and K. R. Vincent, "Resistance training lowers exercise-induced oxidative stress and homocysteine levels in overweight and obese older adults," Obesity, vol. 14, no. 11, pp. 1921– 1930, 2006.
- V. N. Oliveira, A. Bessa, M. L. Jorge et al., "The effect of different training programs on antioxidant status, oxidative stress, and metabolic control in type 2 diabetes," Applied Physiology, Nutrition, and Metabolism, vol. 37, no. 2, pp. 334–344, 2012.
- Vinetti G, Mozzini C, Desenzani P, Boni E, Bulla L, Lorenzettil, Romano C, Pasini A, Cominacini L, Assanelli D. Supervised exercise training reduces oxidative stress and cardiometabolic risk in adults with type 2 diabetes: a randomized controlled trial. Sci Rep. 2015 18;5:9238.
- 40. Farinha JB, Steckling FM, Stefanello ST, Cardoso MS, Nunes LS, Barcelos RP, Duarte T, Kretzmann NA, Mota CB, Bresciani G, Moresco RN, Duarte MM, Dos Santos DL, Soares FA. Response of oxidative stress and inflammatory biomarkers to a 12-week aerobic exercise training in women with metabolic syndrome. Sports Med Open. 2015;1(1):3.
- Nojima H, Watanabe H, Yamane K, Kitahara Y, Sekikawa K, Yamamoto H, Yokoyama A, Inamizu T, Asahara T, Kohno N. Effect of aerobic exercise training on oxidative stress in patients with type 2 diabetes mellitus.Metabolism,57, 2008: 170–176.
- 42. Gordon L; Morrison E; McGrowder D; Young R; Fraser Y; Zamora E; Alexander-Lindo R; Irving R. Effect exercise therapy on lipid profile and oxidative stress indicators in patients with type 2 diabetes. BMC Complement Altern Med. 2008; 8:21.



- 43. E. Vanninen, M. Uusitupa, O. Siitonen, J. Laitinen, E. Lansimies. Habitual physical activity, aerobic capacity and metabolic control in patients with newly-diagnosed type 2 (non-insulin-dependent) diabetes mellitus: effect of 1-year diet and exercise intervention. Diabetologia, 35 (1992), pp. 340–346
- J.T. Devlin, M. Hirshman, E.D. Horton, E.S. Horton Enhanced peripheral and splanchnic insulin sensitivity in NIDDM men after single bout of exercise. Diabetes, 36 (1987), pp. 434–439
- D. Jay, H. Hitomi, K.K. Griendling Oxidative stress and diabetic cardiovascular complications. Free Radic Biol Med, 40 (2006), pp. 183– 192
- S.N. Chugh, R. Dhawan, K. Kishore, A. Sharma, K. Chugh Glibenclamide vs gliclazide in reducing oxidative stress in patients of noninsulin dependent diabetes mellitus—a double blind randomized study. J Assoc Physicians India, 49 (2001), pp. 803–807
- 47. R. Elosua, L. Molina, M. Fito, A. Arquer, J.L. Sanchez-Quesada, M.I. Covas et al. Response of oxidative stress biomarkers to a 16-week aerobic physical activity program, and to acute physical activity, in healthy young men and women. Atherosclerosis, 167 (2003), pp. 327–334
- H. Urakawa, A. Katsuki, Y. Sumida, E.C. Gabazza, S. Murashima, K. Morioka et al. Oxidative stress is associated with adiposity and insulin resistance in men. J Clin Endocrinol Metab, 88 (2003), pp. 4673–4676

Citation: Prof. Shehab M. Abd El-Kader, Fadwah M. Al-Sharif, "Impact of aerobic versus resistance exercise training on glucose control and biomarkers of oxidative stress among Saudi patients with type 2 diabetes". American Research Journal of Cardiology, vol 4 no. 1, 2021, pp. 1-6.

Copyright © 2021 Prof. Shehab M. Abd El-Kader, etal. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

