Orginal Article Open Access
Iranian Journal of Diabetes and Obesity (IJDO)

# Improvement of Glucose Homeostasis in Response to Short-Term Aerobic Training in Middle-Aged Men with Abdominal Obesity

Reza Naseri Rad[1], Mojtaba Eizadi[2*], Morteza Ghasemi[3]

[1]MS.C, Department of Exercise Physiology, Faculty of Physical Education and Sport Sciences, Islamic Azad University, Islamshahr Branch, Islamshahr, Iran.
[2]Assistant Professor, Department of Exercise Physiology, Faculty of Humanities Sciences, Islamic Azad University, Saveh Branch, Saveh, Iran.
[3]Assistant Professor, Department of Nursing, Faculty of Medical Sciences, Islamic Azad University, Arak Branch, Arak, Iran.

## Abstract

**Objective:** Overweight and obesity is associated with insulin resistance and is the most important risk factor of type 2 diabetes (T2DM). In present study, we assessed glycemic profile and insulin resistance response to a short term aerobic training in middle-aged men with abdominal obesity.

**Materials and Methods:** The subjects included 28 abdominally obese (waist circumference≥102cm) middle-aged men (39 ± 5 year) that were divided into exercise (n=14) or control (n=14) groups by randomly. Exercise subject were completed a short-term aerobic training at 55-70 % of maximal heart rate (6 weeks, 3 times / weekly) and control subjects remained no training. Pre-training and post-training of anthropometrical markers, fasting glucose, hemoglobin (HbA1C), insulin and insulin resistance were measured of 2 groups and compared by independent – paired t test (SPSS, Version 22.0).

**Results:** Aerobic exercise resulted in a significant decrease in glucose level (114 ± 13 versus 101 ± 11 mg/dL, *P:* 0.009) and HbA1C (6.14 ± 1.11 versus 4.91 ± 1.23, *P:* 0.021) in exercise group. But no significant changes were observed in insulin (8.31 ± 4.12 versus 8.29 ± 3.21, *P:* 0.119) and insulin resistance (2.34 ± 0.51 versus 2.07 ± 0.59, *P:* 0.073) in exercise groups.

**Conclusion:** Based on our finding, aerobic training independent of insulin function is associated with improved glucose in middle-aged obese men and this improvement can be attributed to other changes caused by exercise that requires further study in this area.

**Keywords**: Aerobic exercise, Obesity, Glucose, Insulin function

QR Code:

**Citation:** Naseri Rad R, Eizadi M, Ghasemi M. Improvement of Glucose Homeostasis in Response to Short-Term Aerobic Training in Middle-Aged Men with Abdominal Obesity. IJDO 2023; 15 (2) :110-118

**URL:** http://ijdo.ssu.ac.ir/article-1-794-en.html

10.18502/ijdo.v15i2.12969

**Article info:**
**Received:** 09 January 2023
**Accepted:** 08 May 2023
**Published in June 2023**



**Corresponding Author:**
**Mojtaba Eizadi,** Department of Exercise Physiology, Faculty of Humanities Sciences, Islamic Azad University, Saveh Branch, Saveh, Iran.
**Tel:** (98) 353 728 0226
**Email:** izadimojtaba2006@yahoo.com
**Orcid ID:** 0000-0003-1989-692X



[ DOI: 10.18502/ijdo.v15i2.12969 ]

## Introduction

In addition to psychological and social problems, obesity, especially abdominal obesity, has been associated with the prevalence of chronic diseases like cardiovascular diseases, diabetes, respiratory and liver diseases, and cancers (1). Although the molecular mechanisms underlying the relationship between obesity and its associated diseases have not been entirely grasped, it is inferred from scientific sources and research findings that increased adipose tissue mass, which is a hallmark of obesity, plays a significant role in metabolic disorders. In addition to genetics and inactivity, obesity is also one of the most important environmental factors that cause insulin resistance syndrome. In this regard, it can be mentioned that insulin level is balanced and insulin resistance is reduced following weight loss or reduction of body fat levels (2).

Insulin resistance is defined as an inadequate response of tissues such as liver, adipose tissue and skeletal muscle, as insulin-sensitive tissues to systemic insulin (3). Furthermore, the decrease in the number of insulin-receptor proteins in the presence of obesity in insulin-sensitive tissues provides the basis for increased insulin resistance (4). In addition, the activation of inflammatory pathways in immune cells by the greater influence of inflammatory cytokines and fatty acids or lipotoxins has led to the disruption of insulin-signaling pathways, which in turn result in increased insulin resistance, especially in healthy or obese-induced disease populations (5). Researchers have also suggested inflammatory processes as a mediator between obesity and insulin resistance (6). In contrast, increased adipose tissue leads to morphological and physiological changes including sequestration or secretion of macrophages and release of several pro-inflammatory cytokines. Moreover, some of these changes directly or indirectly affect insulin resistance and sensitivity by regulating insulin symptoms and molecules involved in lipid and glucose metabolism (7).

In addition to obesity, an inactive lifestyle is associated with a high risk of increased insulin resistance and cardiovascular diseases. Moreover, hyperglycemia or high blood glucose is the major consequence of insulin resistance (8). Therefore, providing short- or long-term exercise training to improve insulin resistance and inflammatory profile, especially in healthy or diseased obese individuals has always been the focus of health science researchers. However, the findings are often contradictory in this regard depending on the type of activity in terms of duration, frequency, and intensity of the training program, the measurement tool, and the studied population. For example, some studies have reported a decrease in insulin resistance and blood glucose following aerobic exercises (9). Moreover, there was significant increase in insulin sensitivity and improvement in blood glucose in the absence of inflammatory profile change after 6 months of moderate-intensity physical activity (10). Other studies have also suggested that a significant reduction in body weight is required for improved insulin resistance (11). In contrast, in a relatively recent study, 8 months of aerobic exercise did not alter insulin resistance but improved blood glucose in obese adolescents (12). Despite the above-mentioned evidence, it is also questionable whether improving blood glucose or glycosylated hemoglobin (HbA1C) in response to external interventions such as exercise programs is directly dependent on insulin changes or insulin resistance or whether exercise training in the absence of their changes can also affect blood glucose or HbA1C. Therefore, our study aimed determining the effect of 6 weeks of aerobic training on fasting glucose, insulin, and insulin resistance in a group of inactive males with abdominal obesity.



[ DOI: 10.18502/ijdo.v15i2.12969 ]

## Materials and Methods

Twenty eight sedentary middle-aged (35–45 years) obese men with abdominal obesity (Table 1) participated in this experimental study and were randomly divided into exercise (aerobic training/6 weeks, n=14) and control (no-training, n=14). The experimental protocol and potential risks were explained to participants verbally and informed consent was obtained. Ethics approval was given by Ethics Committee of Islamic Azad University, Saveh Branch, Iran .

The participants who had weight stable (±1kg) for 3 months before study and engaged in exercise less than once per week were included in the study. Athletes, smokers, and individuals with alcohol consumption, persons with a known diagnosis of diabetes, absence of restraining orthopedic/ neuromuscular diseases, asthma, arthritis, cancer, hypertension, heart attack, bronchitis or chronic cough and other chronic diseases were excluded. Exclusion criteria were also included medications that alter lipid and carbohydrate metabolism and inability to exercise. In this study, although the aim was to assess the impact of aerobic training in the absence of calorie restriction on dependent variables, but individuals were asked to use the food recall questionnaire 3 days before blood sampling. Each individual's records the type of food consumed 3 days before the first blood sample (pre-training) and repeats the same diet program 3 days before the second blood sample (post-training).

### Anthropometry

Height and Body weight were measured to the nearest 0.1 cm and the nearest 0.1 kg, respectively when subjects were in a fasting state. Obesity measured by body mass index ($Kg/m^2$). Body fat percentage and visceral fat was determined using body composition monitor (OMRON, Finland). Hip and abdominal circumferences were measured in the most condensed part using a non-elastic cloth meter after normal expiration. Bout measurements were made every 1 minute and average of two measurements used for analysis. Baseline variables for anthropometric and clinical marker are reported in Table 1.

### Training protocol

Aerobic training lasted 6-weeks (3 sessions/ weekly). Each session was supervised by an exercise physiologist. In each exercise session, they performed warm-up for 5-10 min, followed by a 15 – 30 min running, at a intensity of 55-70% of HRmax followed by a 5-10 min cooling-down (Table 2). The principle of overload in the training protocol was designed based on increasing the volume and intensity of training in 2 weeks. After 2 weeks of training, 5 minutes was added to the

**Table 1. The baseline Clinical and anthropometrical characteristics of the subjects**

| Variables | Exercise group | Control group | *P* |
|---|---|---|---|
| **Age (year)** | 40.7 (± 4.51) | 41.2 (± 3.83) | 0.681 |
| **Height (cm)** | 174 (± 5.11) | 175 (± 4.23) | 0.552 |
| **Weight (kg)** | 101.5 (± 10.14) | 102.5 (± 9.28) | 0.361 |
| **Abdominal circumference (cm)** | 111 (± 5.24) | 113 (± 3.97) | 0.492 |
| **Body mass index (kg/m2)** | 33.50 (± 3.44) | 33.49 (± 2.35) | 0.411 |
| **Body fat (%)** | 34.03 (± 4.10) | 34.11 (± 3.21) | 0.544 |
| **Visceral fat** | 13.6 (± 0.91) | 13.8 (± 0.74) | 0.285 |
| **Glucose (mg/dL)** | 114 (± 13) | 116 (± 11) | 0.726 |
| **Insulin ( µ IU/ml )** | 8.31 (± 4.12) | 9.01 (± 2.21) | 0.323 |
| **Insulin resistance (HOMA-IR)** | 2.34 (± 0.51) | 2.58 (± 0.72) | 0.542 |
| **HbA1C (%)** | 6.14 (± 1.11) | 6.21 (± 1.06) | 0.721 |

Data represented by independent sample T-test.

**Table 2. Distribution of exercise intensity during exercise intervention**

| Weeks | Exercise intensity (%HRmax) | Time of running |
|---|---|---|
| **1 -2** | %55 ≤ intensity ≤ %60 | 3 × 5 minute |
| **3 - 4** | %60 ≤ intensity ≤ %65 | 2 × 10 minute |
| **5 - 6** | %65 ≤ intensity ≤ %70 | 2 × 15 minute |



[ DOI: 10.18502/ijdo.v15i2.12969 ]

running time and 5% to exercise intensity (HRmax). In each session, main exercise was running at mentioned intensity. In addition, exercise intensity was controlled using the Polar heart rate tester (H10: USA). Control subjects were instructed to maintain their habitual activities. The subjects were instructed to maintain usual diet throughout of the study.

### Laboratory assays

A venous blood sample was drawn from the cubital vein of all the subjects after a 10-12 h overnight fast (pre-training). All participants were asked to avoid doing any heavy physical activity for 48 h before sampling. After the last exercise session, participants rested for 48 h, then fasting blood samples were taken same to pretest (post-training). Serums were immediately separated and stored at -76° until the assays were performed. Glucose was assessed by oxidase method (Pars Azmoon kit-Tehran). Insulin was determined by ELISA method (Demeditec, made Germany) and intra- assay and inter-assay coefficient of the method were 2.6% and 2.88 respectively. NycoCard kit used to determine HbA1C by Boronate affinity assay made in England. Insulin resistance index (HOMA-IR) index was calculated by the formula:

HOMA1-IR= fasting plasma insulin (µU/ ml) x fasting plasma glucose (mmol/L)/ 22.5 (13).

### Data analysis

Statistical analyses were performed through statistical software package (SPSS, Version 22.0, SPSS Inc, IL, USA). The Kolmogorov-Smirnov was applied to assess the variables with normal distribution. Independent student t test was used for comparison of variables between groups at baseline. To determine the impact of exercise training on variables, the delta between pre and post values of each variable were compared by independent t test between groups. Paired T-test used to assess the intra-group changes of variables in each group. A criterion alpha of $P \leq 0.05$ was used for statistical comparisons.

## Ethical considerations

This study was approved by Research Department of Azad University of Saveh Branch, Saveh, Iran. (Project Code: 1181287110600).

## Results

Baseline variables for anthropometric and clinical marker are reported in Table 1. No significant differences were observed in anthropometrical indexes between groups at baseline (*P*> 0.05). In addition, no differences were observed between groups for glucose (*P:* 0.726), insulin (*P:* 0.323), insulin resistance (*P*: 0.542) and HbA1C (*P:* 0.721) at baseline (Table 1).

To determine the change of each variables, first the delta of each variable (pre to post difference) in each group was assessed, then delta of each variable were compared by independent T-test between groups. Findings from independent T-test showed significant difference in delta for glucose (*P*: 0.014) and HbA1C (*P*: 0.019) between groups. Actually, findings from intra-group changes indicates that aerobic training resulted a significant decrease in glucose when compared with pre-test in exercise group (*P*: 0.009), but this variable remained no change in control subjects (*P*: 0.236). In addition, aerobic training resulted a significant decrease in HbA1C when compared with baseline levels in exercise group (*P*: 0.021) but this variable remained no change in control subjects (*P*: 0.413) (Table 3).

In contrast, delta comparison of other variables showed no significant differences between groups with regard to serum insulin (*P*: 0.411) and insulin resistance (*P*: 0.263). Actually, intra-group changes showed no significant differences between pre and post-training of insulin (*P*: 0.119) and insulin resistance (*P*: 0.073) in exercise group (Table 4). There were also no differences in these variables in the control group.



[ DOI: 10.18502/ijdo.v15i2.12969 ]

**Table 3. Anthropometrical indexes before and after intervention of 2 groups**

| Variables | | Baseline | After | *P* | Change | *P* |
|---|---|---|---|---|---|---|
| **Weight** | Exercise group | 101.5 (± 10.14) | 97.21 (6.28) | 0.001 | 4.29 | 0.021 |
| | Control group | 102.5 (± 9.28) | 102.3 (± 6.58) | 0.231 | 0.2 | |
| **AC** | Exercise group | 111 (± 5.24) | 106 (5.14) | 0.001 | 5 | 0.014 |
| | Control group | 113 (± 3.97) | 113 (± 4.13) | 0.411 | 0 | |
| **BMI** | Exercise group | 33.50 (± 3.44) | 32.08 (3.28) | 0.011 | 1.42 | 0.033 |
| | Control group | 33.49 (± 2.35) | 33.43 (± 2.88) | 0.213 | 0.03 | |
| **Body fat** | Exercise group | 34.03 (± 4.10) | 31.06 (2.28) | 0.07 | 2.97 | 0.028 |
| | Control group | 34.11 (± 3.21) | 34.28 (± 2.41) | 0.423 | - 0.17 | |
| **Visceral fat** | Exercise group | 13.6 (± 0.91) | 11.08 (1.24) | 0.001 | 2.52 | 0.009 |
| | Control group | 13.8 (± 0.74) | 13.6 (± 0.79) | 0.234 | 0.2 | |

AC, abdominal circumference; BMI: body mass index. Data represented by paired sample T-test.

**Table 4. Pre and post-training of diabetes markers of 2 groups**

| Variables | | Baseline | After | *P* | Change | *P* |
|---|---|---|---|---|---|---|
| **Glucose** | Exercise group | 114 (± 13) | 101 (± 11) | 0.009 | 3 | 0.014 |
| | Control group | 116 (± 11) | 118 (± 13) | 0.236 | - 2 | |
| **Insulin** | Exercise group | 8.31 (± 4.12) | 8.29 (± 3.21) | 0.119 | 0.02 | 0.411 |
| | Control group | 9.01 (± 2.21) | 9.11 (± 2.29) | 0.411 | - 0.10 | |
| **Insulin resistance** | Exercise group | 2.34 (± 0.51) | 2.07 (± 0.59) | 0.073 | 0.27 | 0.236 |
| | Control group | 2.58 (± 0.72) | 2.65 (± 0.68) | 0.279 | - 0.07 | |
| **HbA1C** | Exercise group | 6.14 (± 1.11) | 4.91 (± 1.23) | 0.021 | 1.23 | 0.019 |
| | Control group | 6.21 (± 1.06) | 6.29 (± 0.93) | 0.413 | - 0.08 | |

Data represented by paired sample T-test

## Discussion

In the present study, despite a significant decrease in fasting glucose but insulin levels and insulin resistance did not change in response to six weeks of aerobic training. In addition, decreased HbA1C in response to aerobic training is also one of the main findings of the present study. In this regard, Balducci et al (2010) noted that 12 months of aerobic training (2 sessions/weekly) leads to a significant reduction in HbA1C and insulin resistance (14). Some recent studies have repeatedly proposed the hypothesis that glucose and insulin have pro-inflammatory and anti-inflammatory properties, respectively (15). Studies addressing adults have revealed that insulin resistance is an independent predictor of coronary heart disease, hypertension, cancer, heart attack and T2D. Moreover, the increased insulin function and insulin sensitivity are important protective factors against all of the mentioned clinical diseases (16).

It is well-acknowledged that dietary-induced obesity and its complications such as metabolic syndrome are one of the most central health problems in the world nowadays. The impairment of glucose tolerance and the prevalence of T2D, especially in obese individuals increase with age (17). In this respect, more than 45% of older people meet the criteria for T2D. Multiple risk factors for T2D such as increased adipose tissue, decreased exercise, and inactivity that are associated with increased insulin resistance or beta-cell dysfunction are manifested with increasing age (18). The development of T2D, especially in obese individuals is associated with the inability of beta-cells to compensate for insulin resistance (19). Impairment of insulin secretion has also been observed in healthy older adults (20). In response to obesity, especially visceral obesity, insulin sensitivity decreases and is closely linked to dyslipidemia and systemic hypertension. The mentioned features are associated with the phenomenon of metabolic syndrome and all of the mentioned features are associated with increased cardiovascular risk factors (21). To put in a nut shell, the overall consequence of these metabolic disorders is elevated blood glucose or hyperglycemia and related diseases in obese individuals.

Although obesity increases the risk of insulin resistance, T2D and other cardiovascular abnormalities (22), it has been revealed that



[ DOI: 10.18502/ijdo.v15i2.12969 ]

physical fitness level, as compared with body weight is a more accurate predictor of mortality from cardiovascular diseases (21). It has been hypothesized that increased cardiovascular fitness leads to increased insulin function and decreased body fat tissue concurrently (23); however, some studies have reported that increased cardiovascular fitness, independent of changes in body composition and body weight changes insulin function or insulin sensitivity (24). In this regard, some studies have introduced VO2max as a predictor of insulin concentration (25). As some studies have indicated that cardiovascular fitness affects insulin function far above fat percentage levels, widespread efforts have been made to perform regular exercise trainings to improve insulin function in individuals susceptible to insulin resistance syndrome or T2D (26). Evidence suggests that exercise may delay or prevent the development of T2D in susceptible individuals (27). In addition, it has been suggested that the metabolic benefits of exercise are short-lived (28).

However, researchers have also mentioned that aerobic exercise with different time durations is associated with a significant improve in insulin sensitivity in obese or T2D (29). Exercise training has been shown to increase insulin sensitivity in insulin-resistant obese individuals or those with a family history of T2D and T2D patients. The beneficial effects of exercise and physical activity on improving insulin sensitivity in healthy adults and elderly individuals have also been reported by other studies (30). In another study, weight loss induced by 6 months of exercise training ranged from improved insulin sensitivity in the elderly individuals to normal values in the young individuals (31). On the basis of this evidence, it is likely that the beneficial roles of exercise training on blood glucose may be mostly attributed to improvements in insulin sensitivity than to changes in insulin resistance. The mentioned finding requires further examination. It is also possible that decrease in blood glucose in the study population is rooted in altering or improving cardiovascular risk factors such as triglyceride or HDL as well as decreasing body fat percentage. Lack of measurement of cardiovascular risk factors is limitation of the present study. Reduced blood glucose in response to aerobic training in the absence of altered insulin resistance may be due to changes of glucose transporters. In this regard, exercise training has been suggested to reduce blood glucose, independent of insulin resistance, by increasing the number of glucose transporters such as GLUT4 in skeletal muscle and adipose tissues (32). Clinical studies have also revealed that regular exercise training is able to improve glycemic profile or decrease blood glucose by enhancing AMPK activity in skeletal muscle (33), enhancing GLUT4 translocation and expression (34), decreasing hepatic glucose release induced by decreased phosphoenolpyruvate carboxykinase expression (35), weight loss (36), and maintenance of beta-cell mass (37), or other unknown factors affecting insulin sensitivity.

Finally, despite the lack of measurement of inflammatory or anti-inflammatory mediators affecting insulin function and glycemic profile as limitations and weaknesses of the study, improvement of glucose and insulin in response to 6 weeks of aerobic training in the absence of insulin and insulin resistance change are the strengths of the present study. Improved glucose and reduced HbA1C are reported in the present study, while Garcia et al (2022) noted that exercise training longer than 24 weeks with at least 60 min exercise in the form high-intensity concurrent may serve as a supportive therapy to glycemic profile and metabolic control (38). Improved HbA1C may also be attributed to weight loss or improvement in anthropometric parameters in response to aerobic training in obese individuals studied. As Chou et al (2012) based on their findings collected from 90,958 individuals between 2008 and 2019 have pointed out that normal exercise training,



[ DOI: 10.18502/ijdo.v15i2.12969 ]

coupled with a normal WHR was significantly associated with lower HbA1c among non-diabetic people in Taiwan (39). Yan et al (2022) also stated based on their Data of 2559 participants were included (1273 females and 1286 males) from the 2015-2019 Korea National Nutrition and Health Nutrition Examination that HbA1c were more likely to be controlled when walking or resistance exercises were performed for ≥5 days a week in male or women (40).

## Conclusions

Aerobic training for 6 weeks in the absence of significant changes in insulin resistance improved blood glucose and HbA1C in obese men. Based on the available evidence, exercise training is likely to indirectly affect blood glucose levels by affecting other hormonal mediators or lipid profiles. In addition, despite no significant decrease insulin resistance in response to exercise training but this decrease is considerable from a clinical perspective.

## Acknowledgments

We are particularly grateful to all participants who participated in the study.

## Funding

The authors report no conflicts of interest.

## Conflict of Interest

The research was supported by Islamic Azad University, Saveh Branch, Saveh, Iran.

## References

1. Alexandraki K, Piperi C, Kalofoutis C, Singh J, Alaveras A, Kalofoutis A. Inflammatory process in type 2 diabetes: The role of cytokines. Annals of the New York Academy of Sciences. 2006;1084(1):89-117.
2. LeRoith D, Taylor SI, Olefsky JM, editors. Diabetes mellitus: a fundamental and clinical text. Lippincott Williams & Wilkins; 2004.
3. Schenk S, Saberi M, Olefsky JM. Insulin sensitivity: modulation by nutrients and inflammation. The Journal of clinical investigation. 2008;118(9):2992-3002.
4. Taniguchi CM, Emanuelli B, Kahn CR. Critical nodes in signalling pathways: insights into insulin action. Nature reviews Molecular cell biology. 2006;7(2):85-96.
5. Xu H, Barnes GT, Yang Q, Tan G, Yang D, Chou CJ, et al. Chronic inflammation in fat plays a crucial role in the development of obesity-related insulin resistance. The Journal of clinical investigation. 2003;112(12):1821-30.
6. Haider DG, Schindler K, Prager G, Bohdjalian A, Luger A, Wolzt M, Ludvik B. Serum retinol-binding protein 4 is reduced after weight loss in morbidly obese subjects. The Journal of Clinical Endocrinology & Metabolism. 2007;92(3):1168-71.
7. Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. The Journal of Clinical Endocrinology & Metabolism. 2004;89(6):2548-56.
8. Ford ES. Does exercise reduce inflammation? Physical activity and C-reactive protein among US adults. Epidemiology. 2002:561-8.
9. Ping L, Xia L, Li-xin W. Effects of exercise interference on the serum resistin and insulin sensitivity in patients with impaired glucose tolerance. Beijing Sport Univ. 2007;10:22-3.
10. Monzillo LU, Hamdy O, Horton ES, Ledbury S, Mullooly C, Jarema C, et al. Effect of lifestyle modification on adipokine levels in obese subjects with insulin resistance. Obesity research. 2003;11(9):1048-54.
11. Jung SH, Park HS, Kim KS, Choi WH, Ahn CW, Kim BT, et al. Effect of weight loss on some serum cytokines in human obesity: increase in IL-10 after weight loss. The Journal of nutritional biochemistry. 2008;19(6):371-5.
12. Jones TE, Basilio JL, Brophy PM, McCammon MR, Hickner RC. Long-term exercise training in overweight adolescents improves plasma peptide YY and resistin. Obesity. 2009;17(6):1189-95.
13. McAuley KA, Williams SM, Mann JI, Walker RJ, Lewis-Barned NJ, Temple LA, et al. Diagnosing insulin resistance in the general population. Diabetes care. 2001;24(3):460-4.
14. Balducci S, Zanuso S, Nicolucci A, Fernando F, Cavallo S, Cardelli P, et al. Anti-inflammatory effect of exercise training in subjects with type 2 diabetes and the metabolic syndrome is dependent on exercise modalities and independent of weight loss. Nutrition, Metabolism and Cardiovascular Diseases. 2010;20(8):608-17.
15. Gustafson B, Hammarstedt A, Andersson CX, Smith U. Inflamed adipose tissue: a culprit underlying the metabolic syndrome and



[ DOI: 10.18502/ijdo.v15i2.12969 ]

atherosclerosis. Arteriosclerosis, thrombosis, and vascular biology. 2007;27(11):2276-83.
16. Facchini FS, Hua N, Abbasi F, Reaven GM. Insulin resistance as a predictor of age-related diseases. The Journal of Clinical Endocrinology & Metabolism. 2001;86(8):3574-8.
17. Chang AM. Halter JB. Aging and insulin secretion. Am J Physiol Endocrinol Metab. 2003;284:E7-12.
18. Bloem CJ, Chang AM. Short-term exercise improves β -cell function and insulin resistance in older people with impaired glucose tolerance. The Journal of Clinical Endocrinology & Metabolism. 2008;93(2):387-92.
19. Weyer C, Bogardus C, Mott DM, Pratley RE. The natural history of insulin secretory dysfunction and insulin resistance in the pathogenesis of type 2 diabetes mellitus. The Journal of clinical investigation. 1999;104(6):787-94.
20. Chang AM, Smith MJ, Galecki AT, Bloem CJ, Halter JB. Impaired β -cell function in human aging: response to nicotinic acid-induced insulin resistance. The Journal of Clinical Endocrinology & Metabolism. 2006;91(9):3303-9.
21. Timar O, Sestier F, Levy E. Metabolic syndrome X: a review. The Canadian journal of cardiology. 2000;16(6):779-89.
22. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. Pediatrics. 1998;101(Supplement_2):518-25.
23. Sinha R, Dufour S, Petersen KF, LeBon V, Enoksson S, Ma YZ, et al. Assessment of skeletal muscle triglyceride content by 1H nuclear magnetic resonance spectroscopy in lean and obese adolescents: relationships to insulin sensitivity, total body fat, and central adiposity. Diabetes. 2002;51(4):1022-7.
24. Eliakim A, Scheett TP, Newcomb R, Mohan S, Cooper DM. Fitness, training, and the growth hormone→ insulin-like growth factor I axis in prepubertal girls. The Journal of Clinical Endocrinology & Metabolism. 2001;86(6):2797-802.
25. Gutin B, Yin Z, Humphries MC, Hoffman WH, Gower B, Barbeau P. Relations of fatness and fitness to fasting insulin in black and white adolescents. The Journal of pediatrics. 2004;145(6):737-43.
26. Dietz WH. Childhood weight affects adult morbidity and mortality. The Journal of nutrition. 1998;128(2):411S-4S.
27. Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. New England journal of medicine. 2002;346(6):393-403.
28. Dela F, Larsen JJ, Mikines KJ, Ploug T, Petersen LN, Galbo H. Insulin-stimulated muscle glucose clearance in patients with NIDDM: effects of one-legged physical training. Diabetes. 1995;44(9):1010-20.
29. Houmard JA, Tanner CJ, Slentz CA, Duscha BD, McCartney JS, Kraus WE. Effect of the volume and intensity of exercise training on insulin sensitivity. Journal of applied physiology. 2004;96(1):101-6.
30. Short KR, Vittone JL, Bigelow ML, Proctor DN, Rizza RA, Coenen-Schimke JM, Nair KS. Impact of aerobic exercise training on age-related changes in insulin sensitivity and muscle oxidative capacity. Diabetes. 2003;52(8):1888-96.
31. Kahn SE, Larson VG, Schwartz RS, Beard JC, Cain KC, Fellingham GW, et al. Exercise training delineates the importance of B-cell dysfunction to the glucose intolerance of human aging. The Journal of Clinical Endocrinology & Metabolism. 1992;74(6):1336-42.
32. Brozinick Jr JT, Etgen Jr GJ, Yaspelkis 3rd BB, Kang HY, Ivy JL. Effects of exercise training on muscle GLUT-4 protein content and translocation in obese Zucker rats. American Journal of Physiology-Endocrinology And Metabolism. 1993;265(3):E419-27.
33. Pold R, Jensen LS, Jessen N, Buhl ES, Schmitz O, Flyvbjerg A, et al. Long-term AICAR administration and exercise prevents diabetes in ZDF rats. diabetes. 2005;54(4):928-34.
34. Banks EA, Brozinick Jr JT, Yaspelkis III BB, Kang HY, Ivy JL. Muscle glucose transport, GLUT-4 content, and degree of exercise training in obese Zucker rats. American Journal of Physiology-Endocrinology And Metabolism. 1992;263(5):E1015-20.
35. Chang SP, Chen YH, Chang WC, Liu IM, Cheng JT. Merit of physical exercise to reverse the higher gene expression of hepatic phosphoenolpyruvate carboxykinase in obese Zucker rats. Life sciences. 2006;79(3):240-6.
36. Kibenge MT, Chan CB. The effects of high-fat diet on exercise-induced changes in metabolic parameters in Zucker fa/fa rats. Metabolism-Clinical and Experimental. 2002;51(6):708-15.
37. Király MA, Bates HE, Yue JT, Goche-Montes D, Fediuc S, Park E, et al. Attenuation of type 2 diabetes mellitus in the male Zucker diabetic fatty rat: the effects of stress and non-volitional exercise. Metabolism. 2007;56(6):732-44.
38. García-Hermoso A, Ezzatvar Y, Huerta-Uribe N, Alonso-Martínez AM, Chueca-Guindulain MJ, Berrade-Zubiri S, et al. Effects of exercise training on glycaemic control in youths with type 1 diabetes: A systematic review and meta-analysis of randomised controlled trials. European Journal of Sport Science. 2022:1-2.
39. Chou YH, Cheng YY, Nfor ON, Chen PH, Chen CH, Chen HL, et al. Effects of aerobic and resistance exercise on glycosylated hemoglobin (HbA1c) concentrations in non-diabetic Taiwanese



[ DOI: 10.18502/ijdo.v15i2.12969 ]

individuals based on the waist-hip ratio. Plos one. 2022;17(5):e0267387.
40. Yun I, Joo HJ, Park YS, Park EC. Association between Physical Exercise and Glycated Hemoglobin Levels in Korean Patients Diagnosed with Diabetes. International Journal of Environmental Research and Public Health. 2022;19(6):3280.



[ DOI: 10.18502/ijdo.v15i2.12969 ]