The Effect of Circuit Resistance Training With Varying Intensity on Selected Inflammatory Markers in Obese Men

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Received: 08 May 2021; Accepted: 26 Oct. 2021

Abstract- Obesity and associated chronic inflammation lead to insulin resistance. The aim of this study was to evaluate the effect of varying intensity circuit resistance training on metabolic and inflammatory markers in obese men. In a semi-experimental trial, 44 obese men were selected and randomly divided into four groups, including 1) Control (n=11), 2) Low-intensity circuit resistance training (n=11), 3) Moderate-intensity circuit resistance training (n=11), and 4) High-intensity circuit resistance training (n=11). Resistance training was performed at different intensities, including 1) High-intensity circuit resistance training (80% 1RM), 2) Moderate-intensity circuit resistance (60% 1RM), and 3) Low-intensity circuit resistance training (40% 1RM), three sessions per week for 12 weeks. Serum levels of Dectin-1, TLR2, TLR4, MyD88 were measured using an ELISA kit. Data were analyzed with covariance analysis at $P<0.05$. Twelve weeks of moderate and high-intensity circuit resistance training significantly reduced weight, body mass index, serum levels of Dectin-1, TLR2, TLR4, MyD88, and HOMA-IR ($P=0.001$). The reduction of weight, body mass index, serum levels of Dectin-1, TLR2, TLR4, MyD88, and HOMA-IR were significant in obese men in the high-intensity training group compared to low-intensity training ($P=0.001$). It seems that circuit resistance training, especially high-intensity circuit resistance training, can be used as an option to reduce the inflammatory and metabolic complications associated with obesity.

Acta Med Iran 2021;59(12):733-739.

Keywords: Circuit resistance training; Chronic inflammation; Insulin resistance; Obesity

Introduction

Type 2 diabetes mellitus (T2D) is a global epidemic with high morbidity and mortality rates (1). Risk factors such as obesity, unhealthy eating habits, and a sedentary lifestyle increase the risk of T2D (2,3). Both obesity and T2D are associated with increased oxidative stress and inflammatory markers, which also have detrimental effects on the immune system (4).

Dectin-1 is a member of the C-type lectin receptor family that is highly expressed in macrophages and dendritic cells (5). Dectin-1 is an innate immune receptor involved in various cellular responses, including chronic inflammatory conditions such as autoimmunity and T2D (5,6). Activation of Dectin-1 induces the production of pro-inflammatory cytokines, chemokine (6), and reactive oxygen species (7). Dectin-1 is suggested to have a role in obesity-associated inflammation and insulin resistance, making it a therapeutic target in the treatment of chronic inflammation in insulin-resistant individuals (8). Immune cell receptors such as Toll-like receptors (TLRs), including types 2 and 4, are mainly expressed in adipocytes and adipose tissue macrophages, which are important molecules in inducing insulin resistance due to inflammation (9). Stimulation of TLRs on macrophages produces proinflammatory cytokines. MyD88 signaling has also been reported to be a vital component of passage-like receptor signaling (9).

Macrophages also express high levels of dectin-1, the stimulation of which, along with TLR, further increases the production of TLR-stimulated proinflammatory cytokines so that the secretion of Dectin-1 increases

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TLRs and increases pro-inflammatory markers (10). Data from previous studies suggest that dectin-1 may be an important indicator in the development of insulin resistance and inflammation due to obesity because Dectin-1 is expressed on macrophages and plays an important role in obesity and insulin resistance by modulating their function and phenotype. Inhibition of Dectin-1 has been shown to improve glucose homeostasis and insulin sensitivity. In addition, macrophages without Dectin-1 have anti-inflammatory properties that are associated with improved insulin sensitivity in adipose tissue (9,11,12).

On the other hand, one of the ways to control diabetes and related diseases is to do physical activity. Recently, resistance training has been mentioned as a suitable way to improve health and increase muscle mass. Resistance training has been introduced as a treatment program by the American Heart Association and the American College of Sports Medicine (9,11,12). Studies have shown that muscle hypertrophy resulting from resistance training is associated with decreased levels of inflammatory markers and cytokines (13). In addition, high-intensity resistance training may be able to compensate for the decrease in bone mass and age-related muscle strength in the elderly (14). There are different models of resistance training. One of these training models is circuit resistance training (CRT). Traditional resistance training increases strength, lean mass, bone density and also improves blood lipids but has no significant effect on cardiorespiratory endurance. Circuit resistance training, however, also improves cardiorespiratory endurance and can be a good form of exercise for improving both strength and cardiorespiratory endurance (13,14).

Finding the right intensity for this type of training to improve insulin resistance and finding the main mechanisms involved in insulin resistance due to inflammation can provide new insight into therapeutic goals for obese people. Considering the beneficial effects of circuit resistance training and reducing training time compared to other training, examining the mechanism and appropriate intensity of this type of training in improving insulin resistance and the inflammatory mechanism is important for community health, especially obese people. Therefore, the aim of the present study was to investigate the effect of varying intensity circuit resistance training on metabolic and inflammatory markers in obese men.

Materials and Methods

The present study is an applied study, and its method is a semi-experimental one, which was performed as a pretest-posttest with a control group. The statistical population of this study consists of obese male volunteers in Tehran who were selected through a call in public and administrative centers. Accordingly, after initial clinical evaluations, including history, history of cardiovascular disease, clinical and diagnostic examinations, 50 subjects were selected based on inclusion criteria to participate in the study. Finally, according to Morgan's table, 44 subjects (mean age 23-35 years, body mass index 30-34) were invited. Inclusion criteria include the following: Aged 23-35 years, no chronic diseases according to the medical history questionnaire (cardiovascular disease, diabetes, various cancers, and kidney and gastrointestinal disorders or any injury or problem that limit participation in physical activity), Body mass index (BMI) range 30-34, and waist-to-height ratio (WHtR)> 0.5 and no history of training in the last six months, no history of sleep disorders, non-smoking and no use of supplements, alcohol, caffeinated substances or drug treatment. In addition, the criteria for excluding the subjects from the study are the absence of more than one session in the training program, the occurrence of accidents, injuries, disruptive diseases, or the occurrence of any interfering factor that affects the effective participation of the subjects in the training sessions. In a separate session after the medical examination, the purpose of the study and how it was to be conducted were explained to the subjects. Then, in another session, after determining a maximum repetition, the subjects were homogeneously divided into four groups based on the maximum strength: 1) Control (n=11), 2) Low-intensity circuit resistance training (n=11), 3) Moderate-intensity circuit resistance training (n=11) and 4) High-intensity circuit resistance training (n=11). The training groups completed their research period according to the protocol. The control group also carried out their daily lives during the 12-week research period and were prohibited from participating in regular activities. After filling in the personal information questionnaire and signing the consent form, each of the subjects came to the test site the next day to perform the tests. At the beginning of the session, anthropometric characteristics, including height, weight, and three fat-percentage areas of the subjects, were measured. Subjects' height (in centimeters) using a German height gauge with an accuracy of 0.1 cm and their body weight (in kilograms) using a German digital scale with an accuracy of 0.1 kg were registered. After measuring the height and weight of the subjects, their body mass index was calculated using the formula (height square meter/weight (kg)=(body mass index) BMI). In the second session, all subjects were tested for determining a maximum

repetition. After two days, subjects were referred to the laboratory, and blood samples were taken to assess serum dectin-1, TLR2, TLR4, MyD88 levels, and insulin resistance. The experimental groups then performed the 12-week training program. In the end, anthropometric and blood sampling features were obtained again.

Diet status was recorded by recording food intake for three days (2 days a week and one day on the weekend) before training and after the study to assess changes in normal diet over time. Each food was individually entered into the 10 Plus Diet Analysis (Cengage, Boston, MA, USA), and the total energy intake, as well as the amount of energy obtained from proteins, fats, and carbohydrates, were calculated. Analysis of dietary data showed no significant difference in protein, fat, and carbohydrate intake, and calorie intake between groups before and after the training period.

Circuit resistance training protocol

The circuit resistance training protocol consisted of 8 movements of the upper torso and lower torso (squat, forearm, chest press, knee expansion, knee contraction, barbell shoulder, leg press, armpit wire from behind), which were circuit and performed in different intensities as follows (13,14).

1) High-intensity circuit training group: 3 sets of 10 repetitions with 80% one-repetition maximum (1RM)
2) Moderate-intensity circuit training group: 3 sets of 13 repetitions with 60% 1RM
3) Low-intensity circuit training group: 3 sets of 20 repetitions with 40% 1RM

The training volume was calculated based on the formula presented by (15) (Amount of weight×number of repetitions×number of sets=training volume). The rest between sets was 2 minutes and was inactive. Maximum repetition (1RM) of the subjects was calculated using the Brzezinski equation: The method of measuring a maximum repetition is that first, the person warms up with a lightweight and then chooses a weight with which they can do up to 10 repetitions. If the weight is light and the number of repetitions is more than 10, after a brief rest, more weight is selected so that less than ten repetitions can be performed. The amount of weight and the number of repetitions in each movement are recorded and then placed in the formula.

One maximum repetition=displaced weight (kg)/0.0278-number of repetitions to fatigue (×0.0278)

To evaluate the biochemical variables, blood sampling was performed after 12-14 hours of fasting before and 12 weeks after the intervention (48 hours after the last training session). At each stage, the laboratory attendants from the antecubital vein of the left-hand 5cc blood were taken at rest in a sitting position. Blood samples were stored at -80° C after centrifugation and serum separation until the tests were carried out. In order to prevent the effect of circadian rhythm, blood sampling was performed at a specific time of day (8.30 to 9.30) in the morning. Insulin resistance after measuring fasting glucose and insulin concentration was calculated using the homeostasis model (HOMA-IR) and according to the formula. Serum levels of Dectin-1, TLR2, TLR4, MyD88 were measured using an ELISA kit.

The Shapiro-Wilk test was also used to check the normality of the data distribution. For analyzing the data, covariance analysis was used with SPSS-25, and the significance level was $P \leq 0.05$.

Results

The effect of varying intensity circuit resistance training on weight

The results of the analysis of covariance show that there is a significant difference between the mean weight in varying training intensities after removing the pre-test effect (F=13.612 and *P*=0.000). This significance in the training groups with an intensity of 60% (*P*=0.003) and training with an intensity of 80% (*P*=0.000) compared to the control group and the training group with an intensity of 80% compared to the training group with an intensity of 40% (*P*=0.001) (Table 1).

The effect of varying intensity circuit resistance training on BMI

Additionally, there is a significant difference between the mean BMI in the varying intensity of training after removing the pre-test effect (F=13.396 and *P*=0.000). This significance in the training groups with an intensity of 60% (*P*=0.003) and training with an intensity of 80% (*P*=0.000) compared to the control group and the training group with an intensity of 80% compared to the training group with an intensity of 40% (*P*=0.002) (Table 1).

The effect of varying intensity circuit resistance training on dectin-1

The results show that there is a significant difference between the mean of dectin-1 in varying training intensities (*P*=0.000, F=25.396). There was a significant difference between training groups with intensity 40% (*P*=0.05), 60% intensity training (*P*=0.002), and 80%

intensity training (*P*=0.000) compared to the control group, and 80% intensity training group compared to the training group with the intensity of 40% (*P*=0.000) and exercise with the intensity of 60% (*P*=0.008) (Table 1).

The effect of varying intensity circuit resistance training on TLR-2

The results show that there is a significant difference between the mean of TLR-2 in varying training intensities (*P*=0.000, F=268.342). Bonferroni's post hoc test showed that between training groups with an intensity of 40% (*P*=0.000), 60% intensity training (*P*=0.000), and 80% intensity training (*P*=0.000) compared to the control group and 60% intensity training group compared to the training group with an intensity of 40% (*P*=0.000) and 80% (*P*=0.000), there was a significant difference between the training group with an intensity of 40% and the training group with an intensity of 80% (*P*=0.000) (Table 1).

The effect of varying intensity circuit resistance training on TLR-4

The results show that there is a significant difference between the mean of TLR-4 in varying training intensities (*P*=0.000, F=14.270) there was a significant difference between training groups with intensity 60% (*P*=0.000) and 80% intensity training (*P*=0.000) compared to the control group and 40% intensity training group compared to the 60% (*P*=0.017) and 80% intensity training group % (*P*=0.008) (Table 1).

The effect of varying intensity circuit resistance training on MyD88

The results show that there is a significant difference between the mean of MyD88 in varying training intensities (F=202.50 and *P*=0.000). This significance was in the training group with an intensity of 40% (*P*=0.002), training with an intensity of 60% (*P*=0.000), and training with an intensity of 80% (*P*=0.000) compared to the control group and the training group with Intensity 40% compared to the training group with an intensity of 60% (*P*=0.026) and training with an intensity of 80% (*P*=0.000) and training group with an intensity of 60% compared with an intensity of 80% (*P*=0.003) (Table 1).

The effect of varying intensity circuit resistance training on HOMA-IR

The results show that there is a significant difference between the mean of HOMA-IR in varying intensity of training (F=458. 46 and *P*=0.000). This significance was in the training group with the intensity of 40% (*P*=0.003), training with the intensity of 60% (*P*=0.001), and training with the intensity of 80% (*P*=0.000) compared to the control group and training group with 40% intensity compared to the training group with 60% intensity (*P*=0.001) and 80% intensity training (*P*=0.000) (Table 1).

Table 1. The results of the covariance analysis test for comparing the variables of the study in different groups

Variable	Factor	Sum of Square	df	Mean of Square	F	*P*
	pre-test	6.157	1	6.157	1.541	0.223
Weight	group	163.158	3	54.386	13.612	0.000
	Error	135.850	34	3.996	-	-
	pre-test	35.203	1	35.203	70.818	0.000
BMI	group	19.977	3	6.659	13.396	0.000
	Error	16.901	34	0.497	-	-
	pre-test	449/0	1	449/0	6/0	445/0
Dectin-1	group	971/56	3	990/18	396/25	000/0
	Error	686/21	29	748/0	-	-
	pre-test	982/1734	1	982/1734	485/0	016/0
TLR-2	group	72/2880724	3	574/960241	342/268	000/0
	Error	29/103774	29	42/3578	-	-
	pre-test	016/0	1	016/0	014/0	906/0
TLR-4	group	593/47	3	864/15	270/14	000/0
	Error	24/32	29	112/1	-	-
	pre-test	89/3975	1	89/3975	235/0	632/0
MyD88	group	03/2549378	3	68/849792	202/50	000/0
	Error	32/490899	29	563/16927	-	-
	pre-test	226/0	1	226/0	407/2	132/0
HOMA-IR	group	085/13	3	362/4	458/46	000/0
	Error	723/2	29	094/0	-	-

Discussion

The results of the present study showed that 12 weeks of low, moderate, and high-intensity circuit resistance training significantly reduced Dectin-1 in obese men. Also, the decrease of Dectin-1 in the high-intensity training group compared to low-intensity and moderate-intensity training was significant. Dectin-1 protein can be regulated by different types of cytokines such as interleukin 4 (IL-4) and Granulocyte-macrophage colony-stimulating factor (GM-CSF) as well as by microbial components (16). Little information is available about the response of dectin-1 to training and the mechanisms by which changes in Dectin-1 levels occur after training. Dectin-1 also requires the regulatory factor 5 interferon (IRF5) to respond to the immune system, and IRF5 is essential for differentiation from macrophages M1 AT, which play a major role in obesity-induced insulin resistance (17,18).

In addition, Dectin-1 has been reported to be activated by vimentin (intermediate filament expressed in mesenchymal cells) (19). Therefore, it is possible that the effects of training on Dectin-1 changes occur through Vimentin. Dectin-1 may be a major factor in the development of inflammation and insulin resistance associated with obesity. Because Dectin-1 is expressed in macrophages and dendritic cells, Dectin-1 is thought to modulate macrophage function and phenotype and contributes to the development of obesity and insulin resistance (20). The results of the present study on changes in insulin resistance in relation to changes in Dectin-1 levels can help justify our findings. Contrary to the findings of our study, the results of Ruffino *et al.*, (2016) showed that after eight weeks of the moderate-intensity training program (45 minutes of walking, three times a week), Dectin-1 increased significantly in sedentary women (21). Differences in training method, place of measurement, and duration of training can lead to different results.

The results of the present study also show that 12 weeks of moderate and high-intensity circuit resistance training caused a significant decrease in TLR2 and TLR4 levels in obese men. Decreased TLR-2 levels were significantly different in all three training groups, but decreased TLR-4 levels were significantly higher in the high-intensity and moderate-intensity training groups than in the low-intensity training group. These findings suggest that training intensity is important in altering TLR2 and TLR4 levels in obese individuals. The decrease in TLR2 and TLR4 levels in the present study is consistent with the results of previous studies (22-25). The exact physiological mechanisms of TLR changes are not yet known. However, some of the signals involved, including anti-inflammatory cytokines, stress hormones, and heat shock proteins, appear to play a role in training-induced TLR2 and TLR4 changes (26). Also, in connection with the identification of mechanisms involved in the effects of training on the reduction of TLR2 and TLR4, it can be argued that training can lead to increased levels of anti-inflammatory cytokines, heat shock proteins (HSPs), and glucocorticoids which inhibit TLR4. TLR4 is known to induce the release of cytokines (27,28). Increased levels of glucocorticoids as a result of training have been proven in various studies (23). It seems that the resistance training in the present study inhibits the TLR4 receptor and decreases the levels of inflammatory cytokines by increasing the levels of heat shock proteins. Intense training causes fundamental changes in blood monocyte subtypes and alters the cell surface expression of their receptors, such as TLRs. Intense physical activity preferably induces proinflammatory monocytes and increases the ratio of pro-inflammatory monocytes to classical monocytes (29). Contrary to the findings of our study, some studies have reported no significant change in TLR2 and TLR4 after resistance training in sedentary elderly women and obese postmenopausal women (30,31). It seems that the difference with the above findings is due to the type of subject. Another finding of the present study shows that 12 weeks of low, moderate, and high-intensity circuit resistance training caused a significant decrease in MyD88 in obese men. There was also a significant difference in MyD88 levels between the three training groups. Obesity is associated with an increase in MYD88 protein. A decrease in MyD88 in this study is consistent with the results of Fernandez-Gonzalo *et al*., (2014), who showed that the response of TLR4 signaling pathways decreases after an eccentric training program through dependent pathways independent of the myeloid-88 differentiation factor (32). Signaling independent of MyD88 is via TRIF and is used by TLR4 and TLR3. Recognition of ligands by TLRs enhances the activity of MyD88-dependent pathway adapter proteins or non-MyD8 and TLR4-independent downstream signaling pathways and induces inflammatory reactions (33). TLR4 initiates its response by forming a complex with differential factor 2 myeloid (MD-2), which activates MyD88-dependent and non-dependent cascades (34). The results of the present study show that the intensity of training is an important factor in changes in MyD88

levels in obese people and shows the need to design an appropriate protocol for obese people.

In addition, the results of the present study show that 12 weeks of low, moderate, and high-intensity circuit resistance training significantly reduced HOMA-IR in obese men. Also, the decrease in HOMA-IR in the high-intensity training group and moderate-intensity training compared to low-intensity training was significant. Some studies have shown that resistance training is effective in reducing insulin resistance (34). The results of the present study show the appropriate intensity of resistance training for optimal changes in insulin resistance in obese subjects. High-intensity and long-term training may increase insulin sensitivity and glucose reabsorption through skeletal muscle activity, possibly through increased skeletal muscle mass, increased glucose transport to muscle, or decreased fatty acid synthesis. According to these observations, the reason for the decrease in insulin resistance in this study is probably a decrease in factors such as weight and body mass index in these people after training; Whereas obesity can increase the production of pro-inflammatory agents involved in the pathogenesis of insulin resistance by creating subclinical inflammatory conditions; With training-induced weight loss, a reduction in the pathogenesis of insulin resistance can be observed. Circuit resistance training was one of the strengths of the present study. This is because this type of training can have different responses and adaptations than other training programs, despite the performance limitations. There were some limitations in the present study, including the lack of measurement of other inflammatory and anti-inflammatory agents (Nuclear factor-kappa B (NF-κB), Interleukin 6 (IL-6), Tumour necrosis factor α (TNFα), and Interleukin-10 (IL-10). This is a research weakness suggested to future studies to measure these indicators in obese individuals.

In summary, the results of the present study showed that resistance training reduced Dectin-1, inflammatory factors, and insulin resistance in obese men, and high-intensity circuit resistance training was more effective. Therefore, this type of training, especially high-intensity circuit resistance training, can be used as an option to reduce the inflammatory and metabolic complications associated with obesity.

Acknowledgments

This study was approved by the ethics committee of Sport Sciences Research Institute of Iran and registered under number IR.SSRC.REC.1398.074. The support of all those who have collaborated in this research is greatly appreciated.

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