The Effect of Eight Weeks of Water Training on Sirt1, Pgc-1α and Body Fat Percentage in Obese Men

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ABSTRACT

BACKGROUND AND OBJECTIVE: SIRT1 and PGC-1 α are two factors that increase the body's antioxidant capacity, which can improve inflammation and free radicals in obesity. The present study was conducted to determine the effect of eight weeks of water training on SIRT1, PGC-1 α and body fat percentage in obese men.

METHODS: In this quasi-experimental study, obese men (BMI = 30, waist-to-height ratio (WHtR) > 0.5) were selected voluntarily and were randomly divided into two groups of water training (n = 11) and control (n = 11). The training program included three sessions per week for eight weeks with training intensity of 60 to 80% of maximal heart rate. BMI, body fat percentage and weight of subjects were examined before and after the intervention. Arterial blood samples were used to measure SIRT1 and PGC-1 α during the pre-test and post-test stages.

FINDINGS: The assessments showed that PGC-1 α levels increased from 7.7 to 8.7 and SIRT1 levels increased from 11.3 to 12.6, and this increase was significant in the training group compared to the control group (p < 0.001). The results showed that body fat percentage decreased from 30.2 to 27.83 and weight decreased from 94.7 to 92.2, which was significant compared to the control group (p<0.001).

CONCLUSION: Based on the results of this study, water training can increase the levels of SIRT1 and PGC-1 α and decrease weight and body fat percentage.

KEY WORDS: SIRT1, PGC-1a, Training, Obesity.

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Introduction

Obesity is a chronic disease that is defined as the excess of fatty tissue (1). Obesity is associated with oxidative stress and inflammation, indicating an imbalance between production and the emergence of oxygen free radicals and the ability of the biological system to detoxify or repair their harmful effects. In this situation, the ability of the biological system to detoxify or repair the harmful effects of oxygen free radicals is not sufficient, and this will result in oxidative damage to the cells, tissues or organs of the body (2, 3). PGC-1 α and SIRT1 are important indices that play an important role in controlling obesity and metabolic disorders; these two indices increase the capacity of the antioxidant system to maintain the balance between inflammation and oxidative stress, and the antioxidant system of the body (4, 5).

SIRT1 is a major basic protein to confront with oxidative stress and homeostasis (6). In fact, sirtuins are involved in many vital functions, such as controlling the production of free radicals and oxidizing fats (7, 8), through histone deacetylation and several transcription factors, such as PGC-1a. SIRT1 suppression leads to systemic inflammation, increases oxidative stress and reduces aerobic metabolism (9). In addition, SIRT1 plays a role in the production or control of reactive oxygen species (ROS) via the FOXO pathway, and SIRT1 function decreases with the excessive increase in ROS (10-12). In addition to SIRT1, PGC-1a is also a key regulator of gluconeogenesis and fatty acid metabolism after activation with hepatocyte nuclear factor 4 alpha (HNF4 α), which is a transcription factor for the expression of gluconeogenesis genes and inhibition of glycolysis genes (13).

Moreover, PGC-1a is associated with SIRT1 so that after activation of SIRT1, PGC-1a is activated and regulates the expression of the genes that are involved in cell growth and energy metabolism (9 - 12). Studies have shown that regular exercise reduces excess body fat through increase in the levels and function of PGC- 1α and SIRT1 (14, 15), and improves body's antioxidant capacity, reduces inflammation caused by obesity, and they have the potential for improving physical fitness (3, 16, and 17). The results of the study by Casuso et al. showed that High-intensity interval training (HIIT) was associated with a greater increase in PGC-1 α and AMPK compared to fast-paced interval swim training (14). Vechetti-Junior et al. showed that the pathway of LRP130 / PGC-1a improves by training and reduces muscle atrophy (15). By performing regular exercise activities, SIRT1 and PGC-1a levels can be increased (8, 11). However, of all kinds of sports, the effect of water sports on PC-1 α and SIRT1 levels has been less studied. Water is an environment that applies the proper resistance to a person's body based on the needs of the body, it improves muscle activity and involvement of larger muscle groups to overcome the resistance and can be helpful in increasing mechanical pressure on muscles and bones (16).

Moreover, water sports, unlike other sports, cause the involvement of both upper and lower extremities and increase the metabolism in the body (16). Therefore, considering the advancement of therapeutic approaches to prevent obesity, as well as the importance of water sports as one of the most appropriate and least risky exercise models, as well as increased fat metabolism in this type of exercise due to the higher activity of the lower and upper extremities (16), this study was conducted to investigate the effect of eight weeks of water training on SIRT1, PGC-1 α and fat percentage in obese men.

Methods

After approval by the Ethics Committee of Ferdowsi University of Mashhad (IR.MUM.FUM.REC.1397.015), this quasi-experimental study was conducted on obese people who volunteered at the public and administrative centers of Mashhad through recall. People with BMI = 30, waist-to-height ratio (WHtR) > 0.5, lack of drug and alcohol addiction, lack of regular exercise activity for at least 6 months, no history of kidney, liver, and cardiovascular diseases, diabetes and or any kind of injury or physical impairment were included in the study. Before participating in the study, all the steps and methods were explained to the patients, and after getting completely informed and completing the health check questionnaire, written consent was obtained from them. After the recall, 22 eligible candidates were selected and the subjects were divided into control and training groups (11 subjects) according to weight, height and BMI. All subjects were advised to leave the exercise voluntarily whenever they could not continue or felt tired and exhausted.

Training protocol: The training group practiced for eight weeks (totally 24 sessions) and over 40 minutes in the four first and second sessions, and then, after four sessions, five minutes was added to the time of the main sessions (Table 1). Each exercise session had three stages: the first stage was matching with the water environment and warming up (15 minutes), which included stretching in all joints and major muscle groups, walking forward, back, sides, on the heel and paw, and jugging in the water. The second stage was the training session (30 minutes), which included weight transfer from front to back, fast walking in water, walking to the side and water squat.

The third stage was stretching, deep breathing and floating exercises (15 minutes). Exercise intensity was between 60% and 80% of maximum heart rate, and Polar heart rate monitor was used on the wrist area to control heart rate. Exercise in water was carried out at the shallow depth of an indoor swimming pool at a temperature of 26–28 °C (3, 4).

Table 1. Exercise program for experimental group

subjects											
Time	Warm	Warm Main		Total							
	up	training	down	time							
The first four	10	20	10	40							
sessions											
Four second	10	20	10	40							
sessions											
Four third	10	25	10	45							
sessions											
Four fourth	10	30	10	50							
sessions											
Four fifth	10	35	10	55							
sessions											
Four sixth	10	40	10	60							
sessions											

All subjects had the same diet during the study period. However, their diet was controlled by a 24-hour dietary recall questionnaire (17). The body composition was evaluated 72 hours before and after the training. For this purpose, height and weight were measured to estimate body mass index (BMI). Body fat density was evaluated by measuring the skin folds from the right side of the body using a SAEHAN-SH 5020 caliper (made in England) in the arms, thighs, and above the pelvis after 8 – 10 hours fasting.

The Jackson Pollock formula was used to estimate body fat percentage (18). Blood sampling was done from the brachial vein in early morning after 12 hours of fasting in two steps; 72 hours before the start of the training and 72 hours after the final session of the training, and samples were kept in test tubes containing EDTA. Blood samples were then centrifuged for 10 minutes at 3000 rpm and were used for further analysis. To measure the serum SIRT1, the Cusabio kit (made in China) with a sensitivity of less than 0.039 ng/ml (sensitivity<0.039 ng/ml), and inter – assay coefficient of variation (PIntra<8%) and intra – assay coefficient of variation (PIntra < 10%) were used. PGC-1 α was also measured using the Cusabio kit (made in China) with a sensitivity of less than 31.25 µg/ml (sensitivity < 31.25 pg/ml), and inter–assay coefficient of variation (PIntra<8%) and intra–assay coefficient of variation (PIntra<10%) were used. Both indices were determined by ELISA method.

Descriptive methods were used to calculate indices of central tendency, and dispersion. After confirming the normal distribution of data using Kolmogorov – Smirnov test, analysis of covariance and paired t-test were used to examine inter-group and intra-group variations, respectively, and p < 0.05 was considered significant.

Results

The results of intra-group variation test for changes in the weight of the subjects showed a significant difference between the two groups (p=0.001). In addition, the results of inter-group variation test for changes in weight levels for each of the groups showed that there was a significant decrease only in the training group before and after the training (p=0.001) (Table 2). The results of the inter-group variation test for changes in the BMI of the subjects showed that there was no significant difference between the two groups. In addition, the results of the intra-group variation test for changes in the BMI of the subjects for each of the groups showed that there was no significant difference in any of the groups before and after the training (Table 2).

The results of the inter-group variation test for fat percentage levels showed a significant difference between the two groups (p<0.001). In addition, the results of the intra-group variation test for fat percentage levels for each group showed that there was a significant decrease only in the training group before and after the training (p=0.001) (Table 2).

The results of the inter-group variation test for PGC-1 α and SIRT1 levels showed a significant difference between the two groups (p<0.001). In addition, the results of intra-group variation test for PGC-1 α and SIRT1 levels for each of the groups showed that there was a significant increase only in the training group before and after the training (p<0.001) (Table 2).

	A	0 0		1 0 /		0 1	
Group	Control			Experimental			
	experiment (Mean±SD)			experiment (Mean±SD)			P-value
Variable	Before	After	P 1	Before	After	P 2	
Weight (kg)	90.25±8.9	90.71±8.2	0.321	94.72±8.3	92.23±8.9	0.001*	0.001 #
BMI (kg/m2)	29.8±2.3	30.1±2.8	0.296	28.86±3.6	28.33±2.0	0.520	0.106
Fat percentage(%)	29.28±8.6	29.76±8.0	0.283	30.26±4.7	27.73±3.7	0.001*	0.000 #
PGC-1a (pg/ml)	7.59±1.6	7.37±1.6	0.128	7.7±1.9	8.57±1.9	0.000*	0.000 #
SIRT1 (ng/ml)	10.77±2.3	10.71±2.0	0.385	11.31±1.9	12.6±1.9	0.000*	0.000 #

Table 2. Comparison of changes in weight, BMI, fat percentage, PGC-1a and SIRT1 in groups

* Significant difference in the values in the training group before and after the training. # Significant difference between the two groups (p<0.05).

Discussion

The results of this study showed a significant increase in levels of PGC-1a and SIRT1 after eight weeks of training in water. Considering that few studies were conducted regarding water exercise and its effects on PGC-1a and SIRT1, a number of studies have previously examined the effects of various exercises on these indices, some of which are consistent with the present study (19-21) while some are not consistent with this study (22). It has been shown that highintensity interval training (HIIT) can increase PGC-1a and SIRT-1 (19, 20). Little et al. reported that HIIT for two weeks in 7 young men led to an increase in PGC- 1α expression in muscle cells and an increase in SIRT1 levels, but did not alter the level of PGC-1 α protein (19). In addition, Hoshino et al. stated that training for 4 weeks and 5 sessions per week on the treadmill would result in an increase in PGC-1 α (21).

In spite of the above results, the findings of some previous studies indicate that after exercise, PGC-1a and SIRT1 levels do not change significantly. After 6 weeks of HIIT training with 90% peak oxygen uptake, Gurd et al. showed that PGC-1a levels increased, but there was no change in SIRT1 levels (22). The causes of these contradictions may be associated with the type of exercise, the intensity of exercise, the type of subjects, and the level of readiness of subjects (19-22). In fact, the researchers pointed out that exercise training increases the expression and levels of SIRT1 by evacuating cellular charge through phosphate- and calcium-dependent pathways and the activity of calmodulin- and AMPK-dependent kinase enzymes (22). PGC-1a and SIRT1 have been reported to be associated with an increase in the activity of SIRT1, which increases the levels of PGC-1 α , and affects the metabolism of fat (23, 24). It has also been reported that SIRT1 is associated with ROS and inflammation, which, with an increase in inflammation and ROS, decreases SIRT1 and disrupts its activity. In fact, SIRT1

boosts FOXO activity and thus can control ROS (10 -12). Another reason for the increase in SIRT1 in this study is the reduction of ROS and obesity-induced inflammation because it has been confirmed that exercise improves the body's antioxidant capacity, which reduces inflammation and ROS in obesity (25, 26), which can be attributed to the increase in PGC-1 α (10-12). In the present study, after eight weeks of training in water, it was shown that PGC-1a and SIRT1 increased, and the increase in these indices in the body increased mitochondrial biogenesis and increased exercise performance (19), which leads to an increase in $VO2_{MAX}$, increase in the capacity and levels of β oxidation cycle, adaptation to less use of sugar deposits in body and the body's tendency to use more of stored body fat (27, 28). These factors can be considered as the reasons of weight loss and decrease in fat percentage in this study. Moreover, in the present study, decrease in fat percentage and weight after eight weeks of training in water was reported. The decrease in these anthropometric indices may indicate an increase in levels of PGC-1 α and SIRT1 in the training group, since these indices have a positive effect on fat metabolism and can increase it (29, 30). It is also possible to increase the antioxidant capacity of the body, because it affects fat metabolism and also strengthens the muscular system and improves both (29, 30), which has not been investigated in this study. The present study showed that eight weeks of water training increased levels of PGC- 1α and SIRT-1 and reduced fat percentage and weight. Therefore, this type of exercise can be used in exercise programs as a suitable training method for obese people.

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References

1.Ayissi VBO, Ebrahimi A, Schluesenner H. Epigenetic effects of natural polyphenols: A focus on SIRT1-mediated mechanisms. Mol Nutr Food Res. 2014;58(1):22-32.

Ochsendorf F. Infections in the male genital tract and reactive oxygen species. Hum Reprod Update. 1999;5(5):399-420.
 Hosseinzadeh S, Dabidi Roshan V, Mahjoub S, Taghipour Darzi M. The Interactive Effect of Lead Acetate and Endurance Training on the Brain-Derived Neurotrophic Factor and Malondialdehyde Levels in Rats Cortex. J Babol Univ Med Sci. 2012;14(2):7-15.[In Persian]

4.Afzalpour ME, Ghasemi E, Zarban A. Effects of an Intensive Resistant Training Sessions and Green Tea Supplementation on Malondialdehyde and Total Thiol in Non-Athlete Women. Zahedan J Res Med Sci. 2014;16(3):59-63.
5.Kumar R, Mohan N, Upadhyay AD, Singh AP, Sahu V, Dwivedi S, et al. Identification of serum sirtuins as novel noninvasive protein markers for frailty. Aging Cell. 2014;13(6):975-80.

6.Leibiger IB, Berggren PO. Sirt1: a metabolic master switch that modulates lifespan. Nat Med. 2006;12(1):34-6.

7.Hsu CH, Tsai TH, Kao YH, Hwang KC, Tseng TY, Chou P. Effect of green tea extract on obese women: a randomized, double-blind, placebo-controlled clinical trial. Clin Nutr. 2008;27(3):363-70.

8.Cantó C, Jiang LQ, Deshmukh AS, Mataki C, Coste A, Lagouge M, et al. Interdependence of AMPK and SIRT1 for metabolic adaptation to fasting and exercise in skeletal muscle. Cell Metab. 2010;11(3):213-9.

9.Gillum MP, Kotas ME, Erion DM, Kursawe R, Chatterjee P, Nead KT, et al. SirT1 regulates adipose tissue inflammation. Diabetes. 2011;60(12):3235-45.

10.Cantó C, Gerhart-Hines Z, Feige JN, Lagouge M, Noriega L, Milne JC, et al. AMPK regulates energy expenditure by modulating NAD+ metabolism and SIRT1 activity. Nature. 2009;458(7241):1056-60.

11.Gurd BJ, Little JP, Perry CG. Does SIRT1 determine exercise-induced skeletal muscle mitochondrial biogenesis: differences between in vitro and in vivo experiments? J Appl Physiol. 2012;112(5):926-8.

12.Menzies KJ, Singh K, Saleem A, Hood DA. Sirtuin 1-mediated effects of exercise and resveratrol on mitochondrial biogenesis. J Biol Chem. 2013;288(10):6968-79.

13.Rodgers JT, Puigserver P. Fasting-dependent glucose and lipid metabolic response through hepatic sirtuin 1. Proc Natl Acad Sci U S A. 2007;104(31):12861-6.

14.Casuso RA, Plaza-Díaz J, Ruiz-Ojeda FJ, Aragón-Vela J, Robles-Sanchez C, Nordsborg NB, et al. High-intensity high-volume swimming induces more robust signaling through PGC-1 α and AMPK activation than sprint interval swimming in m. triceps brachii. PLoS One. 2017;12(10):e0185494.

15.Vechetti-Junior IJ, Bertaglia RS, Fernandez GJ, de Paula TG, de Souza RW, Moraes LN, et al. Aerobic exercise recovers disuse-induced atrophy through the stimulus of the LRP130/PGC-1 α complex in aged rats. J Gerontol A Biol Sci Med Sci. 2016;71(5):601-9.

16.Littrell TR, Snow CM, editors. Bone density and physical function in postmenopausal women after a 12-month water exercise intervention. Abstract conference of Med Sci Sports Exerc; 2004.

17.McCance R. Food Standards Agency; AFRC Institute of Food Research McCance and Widdowson's The composition of foods. Cambridge: royed society of chemistry. 2002.

18. Jackson AS, Pollock ML. Practical assessment of body composition. Phys Sportsmed. 1985;13(5):76-90.

19.Little JP, Safdar A, Wilkin GP, Tarnopolsky MA, Gibala MJ. A practical model of low-volume high-intensity interval training induces mitochondrial biogenesis in human skeletal muscle: potential mechanisms. J Physiol. 2010;588(6):1011-22.

20.Vincent HK, Powers SK, Stewart DJ, Shanely RA, Demirel H, Naito H. Obesity is associated with increased myocardial Int J Obes Relat Metab Disord. 1999;23(1):67-74.

21.Hoshino D, Yoshida Y, Kitaoka Y, Hatta H, Bonen A. High-intensity interval training increases intrinsic rates of mitochondrial fatty acid oxidation in rat red and white skeletal muscle. Appl Physiol Nutr Metab. 2013;38(3):326-33.

22.Gurd BJ, Perry CG, Heigenhauser GJ, Spriet LL, Bonen A. High-intensity interval training increases SIRT1 activity in human skeletal muscle. Appl Physiol Nutr Metab. 2010;35(3):350-7.

23.Huang C-C, Wang T, Tung Y-T, Lin W-T. Effect of exercise training on skeletal muscle SIRT1 and PGC-1α expression levels in rats of different age. Int J Med Sci. 2016;13(4):260-70.

24.Little JP, Safdar A, Bishop D, Tarnopolsky MA, Gibala MJ. An acute bout of high-intensity interval training increases the nuclear abundance of PGC-1α and activates mitochondrial biogenesis in human skeletal muscle. Am J Physiol Regul Integr Comp Physiol. 2011;300(6):R1303-10.

25.Okoshi MP, Pagan L, Damatto R, Cezar M, Campos D, Lima A, et al. Physical training improves cardiac structure and function, antioxidant capacity, and exercise tolerance in spontaneously hypertensive rats. FASEB J. 2017;31(1 Suppl):838/6.

26.Otto S, Schumann U, Schulz S, Andress S, Graf T, Steinacker J, editors. High-intensity Strength-/Endurance Training increases the antioxidant Capacity in the Muscle of BRCA Mutation Carriers (BIJOU-Study). ONCOLOGY RESEARCH AND TREATMENT; 2017: KARGER ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.

27.Coggan AR, Kohrt WM, Spina RJ, Bier D, Holloszy J. Endurance training decreases plasma glucose turnover and oxidation during moderate-intensity exercise in men. J Appl Physiol (1985). 1990 Mar;68(3):990-6.

28. Mougios V. Exercise biochemistry: Human Kinetics; 2006.

29.Ghanbari-Niaki A, Saeidi A, Ahmadian M, Gharahcholo L, Naghavi N, Fazelzadeh M, et al. The combination of exercise training and Zataria multiflora supplementation increase serum irisin levels in postmenopausal women. Integr Med Res. 2018;7(1):44-52.

30. Tayebi SM, Saeidi A, Khosravi M. Single and Concurrent Effects of Endurance and Resistance Training on Plasma Visfatin, Insulin, Glucose and Insulin Resistance of Non-Athlete Men with Obesity. Ann Appl Sport Sci. 2016;4(4):21-31.