Jundishapur J Chronic Dis Care. 2018 October; 7(4):e83364. doi: 10.5812/jjcdc.83364.

Published online 2018 December 25. **Research Article**

The Effect of 8 Weeks Aquatic Exercise Program on Plasma Visfatin Level and Some Blood Factors in Obese Men

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Received 2018 August 15; **Revised** 2018 September 15; **Accepted** 2018 October 01.

Abstract

Background: As a novel adipokine, visfatin is associated with obesity exerting an insulin mimetic effect. Nevertheless, it is not clear whether an aquatic exercise program causes variations in the adipose tissue and lipids in the blood reduce plasma visfatin or not.
Objectives: The present research aims to investigate the influence of an eight-week aquatic exercise program on plasma visfatin level and some blood factors in non-athletic obese men.
Methods: This study was conducted as a semi experimental design with a pretest-posttest and with a control group. After a public call, the subjects who met the research criteria were selected. Then, 24 eligible, healthy, non-athletic obese men ($30 \leq BMI \leq 32$), aged 35 - 45 years, were selected randomly and assigned into an experimental and a control group, each comprising of 12 subjects. The experimental group was subjected to an intervention period of eight weeks, three days per week, at an intensity of 60% - 70% of maximum heart rate for 40 to 55 minutes. The independent *t*-test and the paired *t*-test were employed to determine variations in the variables. For the purposes of statistical analysis, the software SPSS-21 was used at the level of significance of $P < 0.05$.
Results: The results showed that body mass index or BMI ($P < 0.003$), the ratio of waist to hip ($P < 0.012$), and body fat percentage ($P < 0.001$) in the experimental group, decreased significantly subsequent to aquatic exercise training. The results obtained from the independent *t*-test also demonstrated that HOMA-IR ($P = 0.001$) and visfatin plasma ($P = 0.001$) was significantly reduced in the experimental group in comparison with the control group.
Conclusions: It appears that the aquatic exercise program with weight loss induced changes in adipose tissue decreased plasma visfatin and insulin resistance in non-athletic obese men.

Keywords: Aquatic Exercise Program, Visfatin, Non Athletic Obese Men

1. Background

Obesity and numerous factors in the metabolic syndrome are closely associated due to the differential secretory role of the adipose tissue (1). In addition, obesity manifests itself by macrophage infiltration into the adipose tissue, thus, inducing a state of low-grade inflammation in the course of numerous complications, e.g. atherosclerosis and type 2 diabetes (2). Therefore, cytokines and adipokines such as IL-6, IL-8, tumor necrosis factor (TNF)-a, and visfatin may exert a substantial effect on the pathogenesis of insulin resistance and cardiovascular disease (1).

As a new adipokine, visfatin has been identified in visceral fat tissue by Fukuhara et al. (3), representing a novel adipocytokine, which preferentially expressed more in the VAT or the visceral adipose tissue than the SAT or the subcutaneous adipose tissues in humans (3). Visfatin is identical with the gene product previously documented pre-B cell colony–enhancing factor (3), a 55-kDa protein comprising of 491 amino acids while lacking the translocational signal specific to the secretory protein (3).

The expression of the visfatin gene enhances in the course of differentiation in the adipocytes being cultured (4). The level of plasma visfatin rises as obesity develops and visceral fat accumulates in KKAy obese mice (3). The quantity of plasma visfatin closely correlates with the VAT area in adult men (3), whose result generally indicates that visfatin is a suitable surrogate marker for accumulation of visceral fat inducing the metabolic syndrome (MS) in humans (5).

The response of visfatin to exercise training has been investigated by Choi et al. (6). Nevertheless, other possible associations among variations in visfatin and factors of the metabolic syndrome concerning obesity, heart disease, and diabetes are yet to be clarified (6). Physical exercise can be considered as a metabolic and neuroendocrine stressor

mobilizing lipids for energy representing a key treatment for obesity and diabetes (7-9).

Moreover, exercise enhances cytokine and protein secretion from adipose tissues such as leptin, adiponectin, and interleukins, all substantially influencing metabolism (10, 11). Subsequent to the training of supervised endurance (6-12), exercise of acute endurance (13, 14), and prolonged rowing (15), the concentration of systemic visfatin among patients with diabetes and obesity was reduced. Nevertheless, the conditions stayed unchanged among competitive male athletes.

Much research into the effect of exercise on visfatin mRNA and concentration of plasma have led to findings, which have been inconsistent and contradictory. For instance, Frydelund-Larsen et al., showed that the expression of visfatin mRNA rises subsequent to an exhaustion exercise staying high 24 h after exercise (13). Nevertheless, plasma visfatin was not affected (16) or decreased (12-17) subsequent to exercise training. It remains to be clarified whether aquatic exercise training caused variations in adipose tissue reducing plasma visfatin.

2. Objectives

The present research investigates the effect of training of aquatic exercise on plasma visfatin and factors of cardiovascular risk among obese men who were nonathletic.

3. Methods

3.1. Subjects

The present semi experimental research was performed using a pretest-posttest and a control group. The statistical population comprised of overweight and obese men in Gachsaran.

Subsequent to a public call, 24 non-athletic obese men (BMI $\leq$ 30 and aged 35 - 45 years old) participated as subjects in the present study. The inclusion criteria included men were not regularly performing exercise training at enrolment. Men suffering from hypertension, heart disease, diabetes, and pulmonary diseases in need of orthopedic treatment and neurological limitations to physical exercise were excluded. The subjects were required to fill out a personal health and medical history questionnaire (PAR-Q), as a screening tool being given verbal and written instruction as to how the experiment proceeded. After obtaining written informed consents from the subjects, they were randomly assigned to either the experimental group or the control group, each comprising of 12 members.

3.2. Exercise Training

The training protocol was undertaken for eight weeks, each week being of three sessions, and each session lasting for 60 to 70 minutes. The experimental group undertook the protocol such that subsequent to heating and water adaptation, the subjects of the experimental group conducted stretching, walking back and forth, and sides on heel and paw, followed by weight transfer from front to back, fast walking in water and scooting, and finally, conducting stretching, deep breathing, and floating in the water. All these activities were performed at a temperature ranging between 26°C to 28°C in the shallow part of a covered pool. The control group only performed their daily tasks in the course of the research with no intervention.

3.3. Anthropometrics and Body Composition Measurements

Heights and weights of the subjects were measured to obtain the body mass index (BMI; kg/m^2). The circumference of the waist was specified through obtaining the minimum circumference (narrowest torso section above umbilicus) and the maximum hip circumference, while the subjects stood with heels together. The waist to hip ratio (WHR) was obtained through division of waist by hip circumference (cm) (ACSM, 2005) (18). The subcutaneous body fat was obtained at the three sites of abdominal, chest, and thigh by using a Lafayette caliper. The body fat percentage was given by the formula proposed by Jackson and Pollock (JACKSON and POLLOCK, 1985) (19). All the subjects were fasting for a minimum of 12 hours and a fasting blood sample was taken using venipuncture. The level of plasma visfatin was taken in duplicate with reference to enzyme-linked immunosorbent assay (ELISA) kits (Uscn Life Science Inc, Wuhan, China). HDL-c, LDL-c, and serum cholesterol triglycerides were assayed using automated techniques. Glucose of plasma was determined using the enzymatic (GOD-PAP, Glucose Oxidase-Amino Antipyrine) colorimetric method (Pars Azmoun, Tehran, Iran). The intra and inter-assay coefficients of variation for glucose were less than 1.3% with a 1 mg/dL sensitivity. The level of serum insulin was taken by a radioimmunoassay (RIA) and the index of insulin resistance was obtained with reference to homeostasis model assessment (HOMA-IR) that correlates satisfactorily with the euglycemic hyperinsulinemic clamp among diabetic individuals. SPSS software (version 21, SPSS, Inc., Chicago, IL) was employed for statistical analysis with values being represented as mean $\pm$ standard deviation (SD). The present study utilized the Kolmogorov-Smirnov test to investigate group homogeneity in the variables (Table 1). Subsequently, the independent *t*-test and the paired *t*-test were utilized to establish variations among the variables with P values lower than 0.05 being considered statistically significant.

Table 1. Kolmogorov-Smirnov Test for Normality of Data

Variable	LDL		HDL		TC		TG		Glu		Ins		Ho		Vis	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Z	0.768	0.624	1.10	0.741	1.28	0.914	0.830	1.11	1.09	1.25	1.53	1.35	0.765	0.654	1.12	0.659
P value	0.59	0.831	0.176	0.642	0.075	0.374	0.496	0.196	0.181	0.87	0.134	0.63	0.16	0.112	0.161	0.778

Abbreviations: Ho, HOMA-IR; Vis, visfatin; Ins, insulin; Glu, glucose.

4. Results

Tables 2 and 3 depict the anthropometric and biochemical group characteristics with values being represented as mean and standard deviation. The results emanating from the present research suggest that body mass index, body fat percent, body weight, and WHR fell ($P < 0.05$) subsequent to aquatic exercise training, Nevertheless, no significant changes were observed among these variables within the control group. In addition, the results from the independent *t*-test revealed a significant difference in insulin resistance ($P = 0.001$) and plasma visfatin and LDL-c (decreased) ($P < 0.05$) and HDL-c (rising) between the experimental and the control groups following an eight-week aquatic exercise training.

5. Discussion

The results emanating from the present research suggest that an eight-week aquatic exercise program is capable of significantly reducing levels of visfatin, body fat percentage, and waist to hip ratio. Numerous studies have also examined the effect of sport exercises on the levels of visfatin plasma reporting a reduction of visfatin serum concentrations.

Seo et al. document significant reductions in the levels of visfatin after 12 weeks of combined trainings among obese women whose finding is in agreement with the results emanating from the present research (20).

Choi et al. investigated the effect of 12 weeks of combined training on levels of visfatin among obese and overweight women suggesting a significant decrease in visfatin (6).

Azimi et al. additionally, report a significant decrease in levels of visfatin after eight weeks of aerobic training among men with type 2 diabetes (21). The reduction in visfatin plasma levels can be explained by decreasing fat percentage, especially visceral fat mass, among the subjects of the present research (taking the high body mass index into consideration).

Nevertheless, the results from the recent study are not in agreement with those of Ghanbari and Fathi (22). This inconsistency can be explained by the fact that the metabolic needs of these trainings can also be considered. One-bout trainings do not significantly alter levels of visfatin plasma while subject to the duration of sport activity, this will significantly reduce visfatin (13).

Research also suggests that levels of visfatin plasma significantly correlated with visceral fat mass, weight, and body mass index. Berndt et al. report a positive association between visfatin plasma and body fat percentage, being independent of other factors, e.g. weight and body mass index. Nevertheless, research is yet to establish the function of visfatin in the incidence of metabolic syndrome and fat disorder (23).

For instance, Fukuhara et al. have proven that visfatin is an determining factor in the development and formation of the metabolic syndrome (3), a finding which is not supported by Kloting and Kloting (24). The results emanating from the present research demonstrate that aquatic exercise training is effective in the improvement of blood lipoproteins (LDL-c and HDL-c). The present study reports a positive and significant correlation between levels of primary visfatin plasma and triglyceride.

Moreover, Sun et al. reported a positive and significant correlation between the levels of primary visfatin plasma and triglyceride being independent of fat percentage and age (25). Consistent with the results from research conducted by Sun et al. (25) and Davutoglu et al. (26), the present research suggests that visfatin can be independently involved with the metabolism of triglyceride within the human body as visfatin gene expression and polymorphism is related to blood triglyceride and cholesterol levels. Nevertheless, this is subject to further research. Rising triglyceride levels are indicative of the metabolic syndrome, which supports Fukuhara et al.'s results (3). Thus, visfatin can also be considered as an effective factor in the development of metabolic syndrome whose finding is subject to further research.

The results emanating from the present research demonstrate that an eight week aquatic exercise training, in addition to reducing levels of visfatin plasma improved insulin resistance among the research subjects. The research also demonstrates that the precise control of blood glucose caused a reduction in the levels of visfatin (27).

Research documenting decreases in the levels of vis-

Table 2. Anthropometric Characteristics of the Subjects Before and After Intervention (Mean ± SD)

Variable/Group	Pre-Test	Post-Test	T	P Value
Height (cm)			-1.214	0.306
Control	1.73 ±.64	1.73 ± 0.64		
Experimental	1.75 ±.71	1.75 ± 0.71		
Body weight (kg)			-3.797	0.001*
Control	91.24 ± 11.76	90.83 ± 11.94		
Experimental	92.82 ± 12.34	89.96 ± 10.54		
BMI (kg/m^2)			-3.230	0.003*
Control	30.51 ± 2.54	30.38 ± 2.54		
Experimental	30.08 ± 3.21	29.40 ± 2.87		
WHR			-2.704	0.012*
Control	98.12 ± 2.99	98.78 ± 6.77		
Experimental	98.81 ± 3.16	97.68 ± 7.63		
FB%			-3.767	0.001*
Control	28.24 ± 3.58	28.44 ± 3.89		
Experimental	28.57 ± 3.99	26.77 ± 2.47		

fatin after sports interventions, weight loss, decreased glucose plasma levels, as well as decreased insulin and body mass index have been observed (6).

Variations in insulin and blood glucose levels (quantity and time interval) may be associated with the reduction in the levels of visfatin among subjects subsequent training interventions (27).

Insulin drops ensuing pancreatic beta cellular dysfunction can be compensated by variations in visfatin concentration due to the insulin-like function of visfatin and in accordance with the theory that the presence of an anabolic message (insulin) inhibits another anabolic message; hence, the necessity of increasing the level of visfatin considering exercise therapies in insulin resistance or reducing blood glucose levels is diminished, thus, decreasing the levels of visfatin (28).

The findings from the present research are also in agreement with those obtained by Lee et al., who concluded that the implementation of three months of training significantly decreased the visfatin plasma concentration and improved insulin resistance (29).

Having regard to the visfatin relationship, exercise-induced changes in blood insulin can be considered as one of the causes for serum visfatin reduction. Two characteristics of duration and intensity of training affect insulin response to exercise. It can be inferred from the present research that the intensity of exercise is capable of reducing visfatin.

While the widespread distribution of visfatin is of widespread distribution among numerous body cells and tissues, e.g. adipose tissue, white blood cells and macrophages, the mechanisms regulating visfatin cell secretion are not completely established. Nevertheless, there is research to suggest that gene expression and levels of visfatin plasma are affected by such factors as obesity and overweight, diabetes, glucose levels, insulin levels, and plasma levels of blood lipids (6, 28-30).

Limitations in the present research include the lack of measurement of inflammatory factors. This is due to the fact that visfatin has been shown to be effective in the development of metabolic diseases and has a close relationship with inflammatory agents such as TNF-α and IL-6 (25). Thus, further research is recommended on the effect of aquatic exercise training on levels of visfatin plasma along with the measurement of inflammatory factors to clarify the effect of these activities on these factors with the visfatin mechanism approach.

Generally, the results emanating from the present research indicate that performance of aquatic exercises can be effective in reducing levels of visfatin plasma in non-athlete men owing to reduced body fat mass with and these changes being independent of the improvement of blood lipids.

5.1. Conclusions

The results from the present research demonstrated that an eigh-week aquatic exercise training improved cardiovascular risk factors and decreased variations in the adi-

Table 3. Metabolic Characteristics of the Subjects Before and After Intervention (Mean ± SD)

Variable/Group	Pre-Test	Post-Test	T	P Value[a]
LDL (mg/dL)			-7.41	0.001
Control	136.05 ± 12.73	138.83 ± 13.73		
Experimental	137.21 ± 11.4	126.92 ± 10.4		
HDL (mg/dL)			2.835	0.008
Control	41.44 ± 7.43	41.11 ± 6.87		
Experimental	40.74 ± 6.87	43.74 ± 7.93		
TG (mg/dL)			-3.239	0.003
Control	202.47 ± 11.57	203.98 ± 1187		
Experimental	203.27 ± 12.11	196.43 ± 8.48		
TC (mg/dL)			-3.751	0.001
Control	217.97 ± 13.66	219.13 ± 13.84		
Experimental	218.43 ± 12.75	209.76 ± 10.11		
Glucose ($mg.dL^{-1}$)			-4.329	0.001
Control	100.60 ± 7.84	102.93 ± 8.98		
Experimental	101.00 ± 8.92	95.13 ± 6.76		
Insulin ($\mu U.mL^{-1}$)			-3.335	0.002
Control	11.66 ± 2.43	12.21 ± 3.11		
Experimental	12.86 ± 3.72	10.90 ± 2.78		
HOMA			-4.966	0.001
Control	2.90 ± 0.76	2.94 ± 0.54		
Experimental	2.95 ± 0.89	2.58 ± 0.26		
Visfatin (ng/mL)			-6.026	0.001
Control	18.34 ± 2.76	18.48 ± 3.12		
Experimental	18.89 ± 3.14	16.51 ± 2.23		

[a] $P < 0.05$, pre-training vs. post-training values.

pose tissue, decrease plasma visfatin, and some inflammatory factors in obese and overweight men. Nevertheless, the present research was limited owing to factors including the genetic capabilities of the subjects on cellular modifications and adaptation, motivation level, psychological stress, and lifestyle. Thus, it is recommended that further research be conducted to elucidate the mechanisms responsible for the effect of exercise on the levels of visfatin.

Acknowledgments

Since the findings of the present study are based on the outcomes of a Master's thesis; this is to appreciate all subjects who have worked with the researchers sincerely.

Footnotes

Authors' Contribution: Study concept and design: Amin Mohammadi and Uones Karimi. Acquisition of data: Uones Karimi. Analysis and interpretation of data: Ali Khajehlandi and Amin Mohammadi. Drafting of the manuscript: Amin Mohammadi and Uones Karimi. Critical revision of the manuscript for important intellectual content: Amin Mohammadi. Statistical analysis: Ali Khajehlandi.

Conflict of Interests: None declared.

Ethical Considerations: This research was approved by the Research Council of Islamic Azad University and obtained a Master's degree.

Financial Disclosure: The findings of the present study are based on the outcomes of a Master's thesis in Master degree.

Funding/Support: None declared.

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