



Scholars Research Library

Annals of Biological Research, 2015, 6 (4):9-13
(<http://scholarsresearchlibrary.com/archive.html>)



The effect of one period of resistance training exercise on hormone sensitive lipase and lipid profiles levels in women with type 2 diabetes

Omid Norouzi*, Maghsoud Peeri and Mohammad Ali Azarbayjani

Department of Exercise Physiology, Faculty of Physical Education and Sports Sciences, Islamic Azad University, Central Tehran Branch, Tehran, Iran

ABSTRACT

One of considered factor in diabetes disease is increase on insulin level, which is the most important mechanism of insulin adjustment measure through HSL (hormone sensitive lipase). There are conflicting studies about the effect of exercise training on HSL. Therefore, the aim of this investigation is to was to investigate the effect of 8 weeks of resistance exercise training on HSL levels and lipid profile levels in patient with type 2 diabetes. Twenty four adult women with type 2 diabetes were divided randomly into 2 groups: Training (n=12) and control (n=12). The subjects of training group practiced in 8 weeks resistance trainings, 3 sessions a week, with intensity 50 – 80 % 1RM. HSL levels and some metabolic parameters such as lipid profiles, glucose, insulin and HOMA-IR were measured by ELISA method before and after intervention. ANCOVA test was used to analyse between-group data and paired t-test for analyzing inter-group data. After 8 weeks resistance training, LDL, insulin and HOMA-IR levels significantly decreased in training group as compared to control group (respectively $P= 0/04$, $P= 0/002$, $P= 0/006$) While HSL, cholesterol, TG and HDL-C levels didn't significantly change. The results of this research indicates that after one period of resistance exercise trainings, HSL levels didn't significantly change while LDL levels, insulin and HOMA-IR significantly change. Therefore resistance training can be used as an effective factor to improve insulin sensitivety in type 2 diabetes patients.

Keywords: resistance training, hormone sensitive lipase, lipid profile, type 2 diabetes.

INTRODUCTION

Type 2 diabetes is the most common diabetes which was appeared by persistent increase of blood Glucose, disorder in Glucose, protein and fat metabolism (1). Diabetes and cardio-vascular disease have approximately same risk factors which involved high-blood pressure, obesity, insulin resistance, increase triglyceride (TG) and decrease high density lipoprotein-C (HDL-C) (2). It has been predicted that, in 2029, 438 milion people will sufer from type 2 diabetes throughout the world (3). The researches showed that insulin and glucose level adjustment andadaptions is the most important factor to treat type 2 diabetes (4). hormone sensitive lipase (HSL) is the most important mechanism to adjust insulin measure (5).

HSL is a protein consists of 768 amino acids which has a key role in analyzing lipids such as: triasil gliserol, diacylglycerol, monoacylglycerol and cholesterol (5). HSL is responsible for returning fat from fatty cells i.e break triglycerides to fatty acids (6). Insulin makes HSL inactive in fat cells as lipoprotein lipase (LPL) in muscle cells (7). HSL controls analysis and release TG and in response to massages (like catecholamines derived from neurons,

glucagon and gonadotropin) increase cyclic Adenosine Mono Phosphate (CAMP) in-cell and fatty acid of blood plasma (6). Insulin decreases the density of free fatty acids in plasma, therefore restrains HSL levels (7). The finding of researches showed that people with metabolic syndrome have insulin resistance, and hyperinsulinemia and less HSL (8). Also clinical importance of this enzyme function hasn't been demonstrated because in epidemiological conducted studies which signifies the correlation of this enzyme activity with type 2 diabetes has been obtained different and conflicting results (6,7).

Since the researches showed HSL has a key factor to adjustment and adaptations lipids, Therefore the survey of environmental factors on this enzyme activity is important. The results of researches about survey of exercise training effect on HSL levels are conflicting. In some researches hasn't been observed increase (9,10,11,12,13,14), and in some researches hasn't been observed any change (15,16,17). One period of aerobic training in healthy people increased HSL levels (9,12). Also Louche *et al.*, (2013) declared in their studies that 8 weeks endurance training increased HSL levels (13). While, Alsted *et al.*, (2009) didn't observed any significant change on HSL levels after one period of aerobic training in healthy young men (15). As well Higa *et al.*, (2012) didn't observed any significant change on HSL levels after a swimming period (17). Regarding to the importance of type 2 diabetes and potential role of resistance training to increase insulin sensitivity and few and conflicting investigations about the effect of training exercise and especially resistance training on HSL levels, the aim of this investigation was to survey the effect of one resistance training period on HSL serum density in women with type 2 diabetes.

MATERIALS AND METHODS

This research was done as half-experimental and in pre test and post test format. Twenty eight adult women with type 2 diabetes voluntarily participated in investigation. The subjects arrival terms to investigation involved at least one year type 2 diabetes history, the absence of any other diseases except type 2 diabetes and non consumption of tobacco and alcohol. Also the subjects didn't have special diets and insulin prescription in treatment phase and before this investigation they didn't have any regular performance training with weights. The subjects of this research during research period, took only 2 metformin capsule daily basis of doctors prescription, as ethical considerations, there isn't the possibility of stopping drugs. After the surveys written consent was taken from subjects. The subjects were asked to keep their normal diets during the research and the study method was approved by the local ethical committee. Then qualified subjects ($24 = n$) randomly divided into to groups: training ($47/75 \pm 7/04$ years, $n = 12$) and control group ($49/08 \pm 6/48$ years, $n = 12$). Table 1 shows physical and functional characteristics of research subjects before and after training.

Every training session involved 10– 15 minutes warm up, main training, 10 minutes cool down. The resistance group subjects practiced in 8 weeks resistance trainings, 3 days every week, 60 minutes everyday with the intensity of 50-80% maximum one repetition (18,19). Every training session involved 3 sets and every sets involved 8 stops. The activity time had been considered 45– 60 seconds in each stop and 30– 60 seconds rest time between stops and 120– 180 seconds rest time between 2 sets. Every stop respectively involved chest press, knee extension, leg curl, lat pull down, arm curl, seated rowing, heel raise, arm extension (18,19).

Blood sampling was done 48 hours before and after training period at 8- 9 A.M before fasting. HSL levels were measured by special human kits, from body composition analyser made in America. Triglyceride concentration, total cholesterol and HDL-C was measured by enzymatic method and with Pars Azmoun Company kits made in Iran. Farid Wald *et al.*'s formula was used to determine LDL-C density. Insulin levels was used by ELISA method and with special human kits (Mercodia AB, Uppsala Sweden Company). To evaluate HOMA-IR, this computational formula was used:

$$\text{HOMA-IR} = [(\text{Insulin(U/I)} \times \text{blood glucose (mmol/l)}) / 22:5]$$

Statistical analysis was performed by SPSS software 18 version. Relevant t-test was used to compare inter-group differences and analytic ANCOVA test was used to compare between– groups differences. All data was presented as mean \pm standard deviation and significant tests levels was considered $P < 0/05$.

RESULTS

Body and functional characteristics of experiential and control groups was presented in table 1. The inter-group study showed that weight Mean in training group has significantly decreased ($P= 0/03$), While in control group body fat percentage has significantly increased ($P= 0/02$). In training group, HDL levels has significantly increased from 39 ± 8.75 to 42.75 ± 8.03 ($P= 0/01$). While in control group significant change wasn't observed as compared to the baseline ($P= 0/76$). In training group, cholesterol levels (9.12%), TG (7.39%) and LDL-C (16.45%) decreased but they didn't significantly change (respectively $P= 0/10$, $P= 0/46$ and $P= 0/08$). As well in training group, insulin levels and HOMA-IR significantly changed (respectively $P=0/007$ and $P=0/003$). The survey results shows that HSL levels in both training and control groups didn't significantly changed (respectively $P= 0/04$ and $P= 0/72$). The results of this study showed that LDL, insulin and HOMA-IR levels in training group as compared to control group significantly changed (respectively $P= 0/04$, $P= 0/002$ and $P= 0/006$) While HSL levels, cholesterol, TG, HDL-C and glucose didn't significantly differ in training group as compared to control group.

Table 1 Anthropometric, metabolic parameters, and HSL levels before and after 8 weeks of training programs

Groups/ characteristics	Control		Resistance training	
	Baseline	After 8wk	Baseline	After 8wk
Weight (kg)	84.2 ± 5.86	81.3 ± 7.5	90.9 ± 9.6	83.2 ± 14.6 *
BMI (kg/m ²)	28.1 ± 4.4	26.7 ± 3.9	27.8 ± 3.6	27.5 ± 3.2
PBF (%)	27.29 ± 5.11	29.43 ± 6.02 *	29.85 ± 3.7	27.13 ± 6.2
TC (mg/dl)	176.91 ± 25.56	178.08 ± 16.46	187.08 ± 53.15	170 ± 34.37
TG (mg/dl)	184.83 ± 61.98	182.83 ± 25.20	195 ± 50.91	180.58 ± 74.81
HDL (mg/dl)	39.16 ± 10.46	40.29 ± 7.47	39 ± 8.75	42.75 ± 8.03 *
LDL (mg/dl)	100.78 ± 22.10	101.22 ± 13.25	109.08 ± 44.86	91.13 ± 23.78 †
Glucose (mg/dl)	207.50 ± 77.24	257.33 ± 82.70	257.92 ± 83.95	216.08 ± 43.04
Insulin (mU/l)	16.19 ± 9.19	18.68 ± 3.82	17.22 ± 11.95	8.68 ± 3.82 *†
HOMA-IR	8.07 ± 3.65	12.27 ± 9.48	10.65 ± 7.22	4.59 ± 2.23 *†
HSL (ng/ml)	4.11 ± 1.30	4.36 ± 1.45	4.18 ± 1.49	4.08 ± 1.52

The values are presented as mean ± standard deviation

* $P \leq 0.05$; significantly different from baseline

† $P \leq 0.05$; significantly different from aerobic group

DISCUSSION

In this investigation, we found that 8 weeks resistance training led to decrease insulin density and insulin resistance as well increase HDL-C density, but no significant change wasn't observed on HSL levels, cholesterol, triglyceride. As well between-group survey showed that LDL-C levels has significant decrease in experiential group as compared to control group. There is a few information about exercise effects especially resistance exercise on HSL plasma concentration in type 2 diabetes patients. Some of researches reported in their research that HSL levels increased, which is uncounter with this research results (9,10,11,12,13,14). Tongjian et al., (2012) studied about the effect of 20 weeks exercise training with the moderate intensity (40- 50% maximum heart rate) and exercise with high intensity (50- 75% maximum heart rate) on HSL gene expression levels of adipose tissue in 30 obese women, and reported increase of HSL gene with high intensity (12). Also Watt et al., (2003) declared that HSL activity within 10 minutes exercise in human skeletal muscle increased as compared to rest time had more increase, and after 120 minutes cycling decreased (9). While Higa et al., (2012) didn't observed any significant change on HSL levels after a period of swimming (17). As well Cho et al., (2011) reported that after 12 weeks aerobic training with low intensity (40- 50% VO_2 max) and with high intensity (70- 75% VO_2 max) dint show a significant change in adult women (16). This research results is concurrent with Alsted et al., (2009), Cho et al., (2011) and Higa et al., (2012) studies results (15,16,17). Unconcurrent results in above researches with this research may relates to some factors. One of these factors is the kind of used exercise training which has different effects on metabolic systems (15,16). Because the research results showed that the expression of HSL in oxidizer muscles is more than glycolytic muscle (9,12,13). One of other effective factors on HSL levels changes is subject's age factor (17). In mice 24 months old was observed decrease on HSL levels that one of its decrease reason is increase in TG content which along aging (17). This research results shows that, perhaps more training time, intensity and subjects is required to make clear exercise effect on HSL in type 2 diabetes. As well regarding to researches, a clear mechanism of HSL changes in body after exercise training especially resistance exercise hasn't been yet expressed in people with type 2 diabetes and more researches on this field are necessary.

One of other important findings of this investigation is significant increase on HDL levels in training group and significant decrease on LDL levels in training group as compared to control group, while cholesterol and TG levels

didn't significantly change in both experiential and control groups. This investigation results have had concurrent with some researches. Casella– Filho et al., (2011) showed that short time exercise didn't change LDL levels in people with metabolic syndrome (20). Regarding to involved mechanisms to decrease LDL measure we can say that one of important factor to increase lipolysis is stimulating adrenoceptors which decreased in exercise training and finally led to Lipolysis increase which led to increase nonesterified cholesterol measure of LDL particles and decrease these particles protein (21). This issue increases LDL particles diagonal and decreases their Concentration. Therefore, decrease LDL particles after exercise training can indicates the positive effect of exercise on cardio-vascular system (22).

One of other important findings of this investigation was significant increase on HDL levels in experiential group. The most common lipid disorders in diabetic involved increase TG and decrease HDL-C levels, recent parameter is the risk of cardi-vascular disease (23). While increase TG levels is along increase LDL levels Which is known as atherogenic factor (23). Dyslipidemia is along body fat increase especially visceral fat, there are reliable evidences that exercise with high intensity has positive significant effect on plasma lipid levels (22). The researches results showed that plasma lipid levels in obese people with metabolic syndrome has conflicting results (24,25,26,27). It seems that regular resistance training with approximately high intensity can significantly change the total improvement of lipoprotein characteristics (26). The researches show that, HDL-C mechanism changes is complicated after training. Enzymes like lipoprotein lipase and cholestery lester transfer protein (CETP) has a key role to change HDL-C levels (27). Lipoprotein lipase by hydrolysis triglyceride plasma is the most important factor to change HDL-C levels. HDL-C concentration increase after training may related to CETP activity levels (24). CETP is responsible for carrying fat in HDL-C molucul and other lipoproteins. Change on CETP levels is a permissions for changes on HDL-C catabolism, and finally changes HDL-C levels too (24,27).

Howere in this research was tried to prevent the effectiveness of some factors on research results by choosing approximately same subjects, but one of this research limitations was few subjects and this issue approximately may related to non significant difference of between-groups Differences. There is no doubt that controled performance of these protocols may approximately answer some ambiguities on HSL mechanism changes.

CONCLUSION

Regarding to the results of this investigation significant increase on HDL levels, insulin, insulin resistance and significant decrease on LDL levels and non change on cholesterol, triglyceride and HSL levels was observed after 8 weeks resistance training. Therefore it can be said, resistance training can as an effective factor was used to improve type 2 diabetes complications in people with type 2 diabetes.

REFERENCES

- [1] Giacco F, Brownlee M., *Circ Res.*, **2010**, 107(9), 1058-70.
- [2] Diakoumakou O, Hatzigeorgiou G, Gontoras N, Boutsikou M, Kolovou V, Mavrogeni S, Giannakopoulou V, Kolovou G., *Cholesterol*, **2014**, 109263.
- [3] Rizos CV, Elisaf MS., *World J Cardiol.*, **2014**, 6(7), 517-30.
- [4] Gosmanov AR, Gosmanova EO, Dillard-Cannon E., *Diabetes Metab Syndr Obes.*, **2014**, 7, 255–264.
- [5] Zechner R, Langin D., *Cell Metabolism*, **2014**, 20(2), 199-201.
- [6] Osório J., *Nat Rev Endocrinol.*, **2014**, 10(8), 445.
- [7] Alsted TJ, Ploug T, Prats C, Serup AK, Høeg L, Schjerling P, Holm C, Zimmermann R, Fledelius C, Galbo H, Kiens B., *J Physiol.*, **2013**, 591(Pt 20), 5141-55.
- [8] Albert J. S., Yerges-Armstrong L. M., Horenstein R. B., Pollin T. I., Sreenivasan U. T., Chai S, Blaner W. S., Soren Snitker, O'Connell J. R., Gong D, Breyer R. J., Ryan A. S., McLenithan J. C., Shuldiner A. R., Sztalryd C, Damcott C. M., *N Engl J Med.*, **2014**, 370, 2307-2315.
- [9] Watt MJ, Heigenhauser GJ, Spriet LL., *J Physiol.*, **2003**, 547(Pt 1), 301-8.
- [10] Trepp R, Flück M, Stettler C, Boesch C, Ith M, Kreis R, Hoppeler H, Howald H, Schmid JP, Diem P, Christ ER., *Am J Physiol Endocrinol Metab.*, **2008**, 294(6), E1127-34.
- [11] Lira FS, Rosa JC, Pimentel GD, Tarini VA, Arida RM, Faloppa F, Alves ES, do Nascimento CO, Oyama LM, Seelaender M, de Mello MT, Santos RV., *Lipids Health Dis.*, **2010**, 9, 109.
- [12] Tongjian You, Xuewen Wang, Rongze Yang, Mary F. Lyles, Dawei Gong, Barbara J. Nicklas., *Journal of Sport and Health Science.*, **2012**, 1(3), 184-190.

- [13] Louche K, Badin PM, Montastier E, Laurens C, Bourlier V, de Glisezinski I, Thalamas C, Viguerie N, Langin D, Moro C., *J Clin Endocrinol Metab.*, **2013**, 98(12), 4863-71.
- [14] Hashimoto T, Sato K, Iemitsu M., *J Appl Physiol*, **2013**, 115(2), 260-7.
- [15] Alsted TJ, Nybo L, Schweiger M, Fledelius C, Jacobsen P, Zimmermann R, Zechner R, Kiens B., *Am J Physiol Endocrinol Metab.*, **2009**, 296(3), E445-53.
- [16] Cho JK, Lee SH, Lee JY, Kang HS., *Int J Sports Med.*, **2011**, 32(6), 468-75.
- [17] Higa TS, Bergamo FC, Mazzucatto F, Fonseca-Alaniz MH, Evangelista FS., *Braz J Med Biol Res*, **2012**, 45(10), 988-994.
- [18] Dunstan DW, Daly RM, Owen N, Jolley D, De Courten M, Shaw J, Zimmet P., *Diabetes Care.*, **2002**, 25(10), 1729-36.
- [19] Hordern MD, Dunstan DW, Prins JB, Baker MK, Singh MA, Coombes JS., *J Sci Med Sport.*, **2012**, 15(1), 25-31.
- [20] Casella-Filho A, Chagas AC, Maranhão RC, Trombetta IC, Cesena FH, Silva VM, Tanus-Santos JE, Negrão CE, da Luz PL., *Am J Cardiol.*, **2011**, 107(8), 1168-72.
- [21] Otocka-Kmieciak A, Orłowska-Majdak M., *Postepy Hig Med Dosw (Online).*, **2009**, 63, 668-77.
- [22] D'hooge R, Hellinckx T, Van Laethem C, Stegen S, De Schepper J, Van Aken S, Dewolf D, Calders P., *Clin Rehabil.*, 2011, 25(4), 349-59.
- [23] Ozder A., *Lipids in Health and Disease*, **2014**, 13(1), 183.
- [24] Thompson PD, Rader DJ., *Arterioscler Thromb Vasc Biol.*, **2001**, 21(7), 1097-8.
- [25] Kwon H.R., Han Kyung A., Ku Yun H., Ann Hee J., Koo B-K., Kim Ho C., Min Kyung W., *Korean Diabetes J.*, 2010, 34(2), 101-110.
- [26] Jorge ML, de Oliveira VN, Resende NM, Paraiso LF, Calixto A, Diniz AL, Resende ES, Ropelle ER, Carnevalheira JB, Espindola FS, Jorge PT, Geloneze B., *Metabolism.*, **2011**, 60(9), 1244-52.
- [27] Balducci S, Zanuso S, Cardelli P, Salvi L, Mazzitelli G, Bazuro A, Iacobini C, Nicolucci A, Pugliese G., *Diabetes Care.*, **2012**, 35(6), 1347-54.