Original Research

The Effect of Four-Week Vibration Training on Adiponectin and Fibrinolysis Markers Levels in Overweight Women

Tahereh Rashidi¹, Behrouz Baghaiee², Ramin Forouzandeh¹, Manizheh Noruzian^{1*}

- 1- Department of Exercise Physiology, Faculty of Physical Education and Sport Science, Kharazmi University, Karaj, IRAN
- 2- Department of Physical Education and Sports Science, Jolfa Branch, Islamic Azad University, Jolfa, IRAN

ABSTRACT

Sedentary lifestyle is one of the factors causing pathophysiological problems such as the impairments in coagulation and fibrinolysis systems. The present study aimed to examine the effect of 4-week whole-body vibration training on tissue plasminogen activator (tPA), plasminogen activator inhibitor type 1 (PAI-1), and adiponectin in overweight women. The research was an applied quasi-experimental study with an experimental group and a control group using pretest-posttest design. In this interventional study, 45 employed women with the age range of 25-40 years and body mass index of 25-29.5 kg/m² were introduced by the National Olympic Academy. The participants who met the inclusion criteria were then randomly assigned to two experimental and control groups. The vibration training was performed for 4 weeks. Paired and independent T-test was used to statically analyses. The tPA level significantly increased in the vibration training group (P = 0.01), and there was a significant difference between the tPA levels of the two groups in the post-test phase (p = 0.01). Furthermore, PAI-1 value also decreased significantly in the vibration training group (P = 0.004), and the tPA / PAI-1 ratio also dropped significantly after 4 weeks of vibration training (P = 0.03). With 4 weeks of vibration training, adiponectin increased meaningfully (P = 0.02), and there was a significant difference between the two groups in this regard in the post-test phase (P = 0.01). Vibration training decreases tPA / PAI-1 so that the vibration training plays a critical role in increasing adiponectin, enhancing blood flow of the muscles and adipose tissues, and decreasing fat percentage.

Keywords: Adiponectin, Plasminogen activator, Plasminogen activator inhibitor, Vibration, Overweight.

Introduction

Sedentary is considered as a risk to individuals' health, particularly women's health status [1]. According to the US National Center for Health Statistics (2008), more than 55.2 percent of women population (N= 116 million persons) are overweight based on their body mass index (BMI). Also, 27.9% of all American women have less than 10 minutes of training per week [1]. In Iran, the rates of participation in sports for women are not satisfactory as well. Surveys by Tondnevis et al. (2001) show that 45.8% of women do not exercise [2]. Ehsani also estimated Iranian women's participation in sports to be 33% [3].

The sedentary lifestyle is one of the factors aggravating pathophysiological conditions leading to different problems such as cardiovascular diseases. Research has revealed a relationship between obesity, overweight, hypertension, and blood lipid with mortality and cardiovascular diseases, and thus with atherothrombosis [4-6]. In this regard, it has been found that impaired hemostatic balance leads to thrombosis or clot formation and vessel closure[7]. This imbalance probably induces thrombosis and myocardial infarction [8]. The fibrinolysis system is a critical physiological mechanism consisting of the combination of proteolytic enzymes and is to break down fibrin layers in the blood vessels [9]. It is generally regarded as a cardio-protective mechanism against cardiovascular problems [10]. Existing evidence suggests that patients with cardiovascular diseases have impaired fibrinolytic activity, which results in decreased plasma tPA or increased PAI-1 levels [11]. Endothelial dysfunction, high resting heart rate, overweight, and hypertension play significant roles in the pathogenesis of cardiovascular diseases[12].

Plasminogen activator inhibitors (PAIs) reduce the fibrinolysis process as the PAI-1 is the major inhibitor of tPA in the blood [13]. The tPA is synthesized, stored, and released as the first plasminogen activator in vascular endothelial cells, sympathetic nerve, and skeletal muscle [14] and is the major activator of adrenaline, vasopressin, training, venous occlusion, and shear stress [13]. Tissue plasminogen activator converts plasminogen into plasmin that causes clot breakdown [7]. PAI-1 is synthesized and released by vascular endothelial cells, activated platelets, macrophages, hepatocytes, and adipose tissues [15] and is positively associated with hypertension [16]. For the thrombus formation, the endothelial is the major source of tPA synthesis and release [17]. According to the previous studies, weight gain leads to an increase in TNF- α and thus PAI-1, and TNF-a is one of the factors activating the PAI-1 [18]. The other studies, however, have revealed that adiponectin inhibits the expression of PAI-1 [19]. In contrast, some researchers believe that adiponectin level decreases as a result of weight gain and BMI [20].

Accordingly, exercises and physical activity are of great importance. Cross-sectional studies have shown lower endothelial function in passive individuals than in trained individuals [21]. Researchers have also claimed that sub-maximal/maximal exercises promote the performance of the fibrinolysis system and fibrin breakdown, which has a significant impact on the prevention of cardiovascular diseases [22]. Vibration training is one of the exercises that have attracted the attention of a large number of researchers and individuals.

Whole-body vibration (WBV) training is a mechanical sinusoidal oscillation, which transmits the acceleration force to the body and thus generates an active training force [23]. This occurs through recalling the muscle contraction reflection, called "tonic vibration reflex" [24]. The effect of WBV on human's fibrinolytic capacity has been less addressed in research. A study on animals showed that pulsatile vibration on the spinal cord increases shear stress in vascular endothelial cells, which raises plasma levels of tPA and thus fibrin breakdown operations during and after periodic acceleration. It is assumed that pulsatile stress increases shear stress in vascular endothelial cells, resulting in the tPA release [25]. WBV indirectly causes the changes in blood glucose and cholesterol levels and is directly associated with the improved cardiovascular system by increasing the blood flow [24]. The cardiovascular effects of vibration training are mild; however, such a training has a significant positive effect on blood flow, blood pressure, heart rate, and maximal oxygen consumption [24]. Increased blood flow is also associated with increased endothelial function and hormones such as testosterone and epinephrine [24]. High concentrations of catecholamine and testosterone [26], as well as high blood flow, promote the fibrinolysis potential. Activation of the sympathetic nervous system is recorded at various WBV frequencies [27]. Lee (2004) also found that WBV had a significant effect on body fat reduction in overweight individuals [28]. In Boyle et al. (2010) study, an increase in tPA levels and a decrease in PAI-1 levels were observed in the three training; however, the changes in the training and vibration groups were greater than the other two groups. According to them, such changes may be due to increased fibrinolytic responses to the increased shear stress of vessels as a result of increased blood flow, enhanced muscle activity, and raised catecholamine concentrations during the WBV training [29]. Kent et al. (1994) also documented that hand vibration activated blood platelets in healthy individuals [30]. During the activation process, alpha-platelet granules secrete PAI-1 into the plasma and surrounding tissues, and it seems likely that the WBV promotes platelet activation and subsequently the PAI-Irelease. Bellia et al. (2014) also reported that eight weeks of vibration training significantly increased adiponectin [31].

Weight gain is associated with changes in PAI-1 and tPA, as well as an increased risk of cardiovascular disease, therefore, it is necessary to use new methods of training in this regard [4, 11]. Since studies have found that vibration has beneficial cardiovascular effects as well as reducing inflammation [13], the present study aimed to examine the effect of 4-week WBV training on tPA, PAI-1, and adiponectin levels in overweight women.

Materials and methods

The research was an applied semi-experimental study with an experimental group and a control group using pretest-posttest design. In this interventional study, 45 employed women with the age range of 25-40 years and body mass index of 25-29.5 kg/m² were introduced by the National Olympic Academy. Inclusion criteria were no illness, sedentary lifestyle, ability to perform sports, and no participation in a training program for at least six months before the study period. Meeting the aforementioned criteria, 20 individuals were allowed to participate in this study (these individuals were selected based on past articles as well as research funding). After explaining the research objectives, filling out the Physical Activity Preparation Questionnaire and the Health Status Scale, and obtaining the participants' written informed consent to take part in the research, the subjects were randomly assigned to one of two experimental and control groups. All the subjects were provided with the necessary information. The experimental and control groups were asked to pursue their diet program; however, the control group were requested to continue their previous lifestyle until the end of the study. Before applying the training protocol, factors such as participants' blood pressure, body mass index (In Body, South Korea), visceral fat, resting heart rate, and aerobic capacity (Maximum oxygen consumption) were measured. The participants were then divided into two vibration and control groups, where the training group had vibration training for four weeks (three sessions per week). Their training program consisted of warm-up exercises, exercise protocol, and cool-down exercises, and the vibration training sessions were supervised by the research team (Bosco Model developed in Germany). Blood samples were taken after 12 hours of being fast before starting the training protocol and at the end of the training protocol after 48 hours of resting and fasting in two stages, namely before the vibration training and after 4 weeks of training. The blood sample was then centrifuged at 1 rpm for 5 min, and the plasma was placed at -40 °C in a fridge to be later utilized for the detection of plasma factors using ELISA (BioTek, USA) and in vitro methods. Zellbio GmbH kits made in Germany were used for testing tPA and PAI-1, and Adipogen.co-South Korea was employed for testing adiponectin. Lipid profiles were also measured using Pars Test kits.

Vibration Training Protocol

During four weeks, the vibration group, in addition to their daily activities, practiced their specific protocol three sessions per week, with each session lasting for 14 minutes, and the control group only had their daily activities. The research protocol was based on the protocol proposed by Juang Hu (2014) [32]. In this protocol, the experimental group should warm up before each session for 10 to 12 minutes, and they then had WBV starting at a frequency of 25 Hz which was enhanced by 5 Hz per week until it reached 40 Hz. In each session, seven positions were repeated twice, there were 30 seconds of vibration exercises and 2 seconds of resting for each participant. The training exercises were performed in pairs. Seven different body postures were static; lunge, squat with the knees flexed at 120, squat with the knees flexed at 100, wide stance squat, claves, deep claves and gentle push up (Figure 1).



Figure 1. Somebody postures 122

Kolmogorov-Smirnov test was used to normalize the data distribution, and independent sample t-test was used to compare the means of the control and experimental groups. Paired sample t-test was also run to compare four weeks of training, and Pearson correlation test was also used to examine the relationship between variables. Data were considered significant at 0.05. All tests were run with SPSS software version 22.

Results

Table 1 presents some physiological features of both groups. According to this table, there was a significant decrease in weight, body mass index, and body fat percentage of the vibration training group (P = 0.03, P = 0.04, & P = 0.04, respectively). No significant change, however, was observed for the other variables including systolic and diastolic blood pressure and heart rate.

N 1	Table 1. Ph	lysiological	leatures of both groups	5	
Markers	Group		Mean ± SD	Р	
Age (years old)	Ex	Pre	39.11±6.79	.99	
		Post	39.11±6.79		
	Con	Pre	39.11±6.79	.99	
		Post	39.11±6.79	.,,,	
Wight (Kg)	Ex	Pre	70.01±6.38	03	
	-	Post	68.01±1.3		
	Con	Pre	69.68±8.15	78	
		Post	70.21±5.23	.70	
Height (cm)	Ex	Pre	161.33±6.51	99	
		Post	161.33±6.51	.))	
	Con	Pre	160.44±7.79	00	
		Post	160.44±7.79	.77	
BMI (kg/m ²)	Ex	Pre	26.93±2.56	04	
		Post	25.31±1.19	.04	
	Con	Pre	27.28±2.28	72	
		Post	28.55±2.33	.12	
Fat(%)	Ex	Pre	33.19±3.33	04	
		Post	31.71±3.38	.04	
	Con	Pre	35.33±3.03	27	
		Post	35.65±2.92	.57	
SBP (mmg)	Ex	Pre	117.84±4.11	24	
		Post	116.8±4.34	.24	
	Con	Pre	116.72±4.31	20	
		Post	118.11±5.01	.29	
DBP (mmg)	Ex	Pre	77.66±3.31	1	
		Post	75.77±3.28	.1	
	Con	Pre	78.33±4.3		
		Post	78.13±4.74	.37	
HR (beat/min)	Ex	Pre	62.44±11.88	14	
		Post	63.77±9.67	.14	
	Con	Pre	66.48±9.05	66	
		Post	66±8.74	.00	

Table 2 also shows a significant increase in tPA level of the vibration training group (P = 0.01); however, no significant change was observed in the control group in this case. Moreover, there was a significant difference between the two groups in the post-test phase in terms of the tPA level (p = 0.01).

The PAI-1 level significantly decreased in the vibration training group (P = 0.004); however, there was no significant difference for the control group and between the groups in the post-test phase.

The tPA / PAI-1 ratio significantly decreased after 4 weeks in vibration group (P = 0.03). There was also a significant difference between the experimental and control groups in terms of tPA / PAI-1 ratio in the posttest phase (P = 0.03).

Furthermore, adiponectin significantly increased after 4 weeks in vibration group (P = 0.02), and there was a significant difference between the two groups in the post-test phase in this regard (P = 0.01).

TG level did not significantly change after in vibration group (P = 0.7) and control group (P = 0.42). There was no significant difference between the two groups in this case (P = 0.07).

Cholesterol level also experienced no meaningful change in the experimental and control groups (P = 0.33 & P = 0.54, respectively), and no significant difference was also noticed between the two groups (P = 0.75).

HDL level did not significantly change in the experimental group (P = 0.73) and control group (P = 0.11). Further, there was a significant difference between the two groups in the post-test phase regarding the HDL level (P = 0.01).

LDL levels in experimental and control groups did not change significantly (P = 0.45 & P = 0.82, respectively), and there was no significant difference between the two groups in terms of LDL (P = 0.21).

$ \begin{array}{ c c c c c c } \mbox{Markers} & \mbox{Group} & \mbox{Mean \pm SD} & \mbox{P} \\ \hline \mbox{Intra} & \mbox{Between} \\ \hline \mbox{Intra} & \mbox{Intra} & \mbox{Between} \\ \hline \mbox{Intra} & \mbox{Between} \\ \hline \mbox{Intra} & \mbox{Intra} & \mbox{Intra} \\ \hline \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} \\ \hline \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} \\ \hline \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} \\ \hline \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} \\ \hline \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} \\ \hline \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} \\ \hline \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} & \mbox{Intra} \\ \hline \mbox{Intra} & Int$	Table 2. Markers Change in experimental and control groups								
$\begin{array}{ c c c c c c c c c c c c c c c c c c c$	Markers	Group		Mean \pm SD	Р				
$\begin{array}{c c c c c c c c c c c c c c c c c c c $					Intra	Between			
$\begin{array}{ c c c c c c c c c c c c c c c c c c c$	tPA	Ex	Pre	299.66±216.62	.012				
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$			Post	386.11±229.40	_	01			
$\begin{array}{ c c c c c c c c c c c c c c c c c c c$		Con	Pre	210.55±189.66	.81	.01			
$\begin{array}{c c c c c c c c c c c c c c c c c c c $			Post	224.35±160.22					
$\begin{array}{ c c c c c c } \hline Post & 3.63 \pm 2.51 \\ \hline Con & Pre & 3.9 \pm 2.79 & .11 \\ \hline Post & 2.37 \pm 1.6 \\ \hline Post & 12.2 \pm 2.3 & .02 \\ \hline Post & 13 \pm 8.72 & .02 \\ \hline Post & 13 \pm 8.72 & .01 \\ \hline Post & 12.1 \pm 2 \\ \hline Post & 12.1 \pm 2 \\ \hline PA/PAI-1 & Ex & Pre & 293.35 \pm 216.87 & .03 \\ \hline Post & 382.47 \pm 230.4 & .03 \\ \hline Post & 382.47 \pm 230.4 & .00 \\ \hline Post & 221.98 \pm 160.08 & . \\ \hline TG & Pre & 141.33 \pm 98.8 & .7 \\ \hline Post & 131.77 \pm 85.53 & .07 \\ \hline Post & 131.77 \pm 85.53 & .07 \\ \hline Post & 134.33 \pm 61.75 & .07 \\ \hline Con & Pre & 118.22 \pm 30.87 & .42 \\ \hline Post & 134.33 \pm 61.75 & .07 \\ \hline Cholesterol & Ex & Pre & 212.88 \pm 31.85 & .33 \\ \hline Post & 203.66 \pm 45.56 & .54 \\ \hline Post & 197.62 \pm 36.02 & .75 \\ \hline HDL & Ex & Pre & 48.33 \pm 4.97 & .73 \\ \hline Post & 47.72 \pm 3.75 & .11 \\ \hline Post & 50.34 \pm 4.77 & .11 \\ \hline Post & 50.34 \pm 4.77 \\ \hline LDL & Ex & Pre & 125.71 \pm 7.72 & .45 \\ \hline Post & 128.44 \pm 6.46 & \\ \hline Con & Pre & 122.32 \pm 7.21 & .82 \\ \hline Post & 120.57 \pm 4.97 & .21 \\ \hline \end{array}$	PAI-1	Ex	Pre	5.71±1.96	.04				
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$			Post	3.63±2.51	_	22			
$\begin{array}{ c c c c c c c c c c c c c c c c c c c$		Con	Pre	3.9±2.79	.11	.22			
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$			Post	2.37±1.6	_				
$ \begin{array}{c c c c c c c c } (\mu g/ml) & \hline Post & 13 \pm 8.72 \\ \hline Con & Pre & 12.4 \pm 3.7 & .31 \\ \hline Post & 12.1 \pm 2 \\ \hline Post & 12.1 \pm 2 \\ \hline Post & 382.47 \pm 230.4 \\ \hline Post & 382.47 \pm 230.4 \\ \hline Con & Pre & 293.35 \pm 216.87 & .03 \\ \hline Post & 382.47 \pm 230.4 \\ \hline Con & Pre & 206.65 \pm 189.42 & .78 \\ \hline Post & 221.98 \pm 160.08 \\ \hline TG & Ex & Pre & 141.33 \pm 98.8 & .7 \\ \hline Post & 131.77 \pm 85.53 \\ \hline Con & Pre & 118.22 \pm 30.87 & .42 \\ \hline Post & 134.33 \pm 61.75 \\ \hline Cholesterol & Ex & Pre & 212.88 \pm 31.85 & .33 \\ \hline Post & 134.33 \pm 61.75 \\ \hline Cholesterol & Ex & Pre & 212.88 \pm 31.85 & .33 \\ \hline Post & 203.66 \pm 45.56 \\ \hline Con & Pre & 192.55 \pm 35.26 & .54 \\ \hline Post & 197.62 \pm 36.02 \\ \hline HDL & Ex & Pre & 48.33 \pm 4.97 & .73 \\ \hline Post & 47.72 \pm 3.75 \\ \hline Con & Pre & 50.47 \pm 5.7 & .11 \\ \hline Post & 50.34 \pm 4.77 \\ \hline LDL & Ex & Pre & 125.71 \pm 7.72 & .45 \\ \hline Post & 128.44 \pm 6.46 \\ \hline Con & Pre & 122.32 \pm 7.21 & .82 \\ \hline Post & 120.57 \pm 4.97 \\ \hline \end{array}$	Adiponectin	Ex	Pre	12.2±2.3	.02				
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	(µg/ml)		Post	13±8.72	_	01			
$\begin{array}{ c c c c c c c c c c c c c c c c c c c$		Con	Pre	12.4±3.7	.31	.01			
$\begin{array}{c c c c c c c c c c c c c c c c c c c $			Post	12.1±2	_				
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	tPA/PAI-1	Ex	Pre	293.35±216.87	.03	0.01			
$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$			Post	382.47±230.4	_				
$ \begin{array}{c c c c c c c c c c c c c c c c c c c $		Con	Pre	206.65±189.42	.78	0.01			
$\begin{array}{c c c c c c c c c c c c c c c c c c c $			Post	221.98±160.08	_				
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	TG	Ex	Pre	141.33±98.8	.7				
$ \begin{array}{c c c c c c c c c c c c c c c c c c c $			Post	131.77±85.53	_	07			
$\begin{tabular}{ c c c c c c c c c c c c c c c c } \hline Post & 134.33\pm61.75 \\ \hline Post & 212.88\pm31.85 & .33 \\ \hline Post & 203.66\pm45.56 & .54 \\ \hline Post & 203.66\pm45.56 & .54 \\ \hline Post & 197.62\pm36.02 & & .75 \\ \hline Post & 197.62\pm36.02 & & & .75 \\ \hline Post & 197.62\pm36.02 & & & & .75 \\ \hline Post & 47.72\pm3.75 & & & & & & .01 \\ \hline Post & 50.47\pm5.7 & .11 & & .01 \\ \hline Post & 50.34\pm4.77 & & & & .01 \\ \hline Post & 50.34\pm4.77 & & & .11 \\ \hline Post & 128.44\pm6.46 & & & & .21 \\ \hline Post & 128.44\pm6.46 & & & & .21 \\ \hline Post & 120.57\pm4.97 & & & & .21 \\ \hline \end{tabular}$		Con	Pre	118.22±30.87	.42	.07			
$\begin{array}{c c c c c c c c c c c c c c c c c c c $			Post	134.33±61.75					
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Cholesterol	Ex	Pre	212.88±31.85	.33				
$\begin{array}{c c c c c c c c c c c c c c c c c c c $			Post	203.66±45.56	75	75			
$\begin{array}{c c c c c c c c c c c c c c c c c c c $		Con	Pre	192.55±35.26	.54	.75			
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$			Post	197.62±36.02					
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	HDL	Ex	Pre	48.33±4.97	.73				
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$			Post	47.72±3.75		01			
$\begin{array}{c c c c c c c c c c c c c c c c c c c $		Con	Pre	50.47±5.7	.11	.01			
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$			Post	50.34±4.77					
$\begin{tabular}{ c c c c c c c c c c c c c c c c c c c$	LDL	Ex	Pre	125.71±7.72	.45				
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$			Post	128.44±6.46	_	21			
Post 120.57±4.97		Con	Pre	122.32±7.21	.82	.41			
			Post	120.57±4.97					

Discussion

The present study examined the effect of a four-week WBV training on the plasma levels of tissue plasminogen activator and inhibitor, the ratio of plasma levels for the tissue plasminogen activator and

inhibitor, systolic and diastolic blood pressure, high and low-density lipoproteins, body fat percentage, lipoprotein, lipid profile of blood cholesterol, triglyceride, resting heart rate, and adiponectin in overweight women.

In this study, after 4 weeks of WBV training, there was a significant increase in the plasma levels of tissue plasminogen activator in overweight women. Manzel et al. (2008) also studied the effect of exercises on tPA, fibrinolysis, and blood coagulation, and found a significant increase in the tPA level [33]. Ghazalian et al. (2001) assessed the impact of five-week WBV training on coagulation and fibrinolytic factors. The results of this study showed that high frequency had a greater effect on increasing the tPA [34]. Some possible mechanisms of increased plasma tPA levels are autonomic nervous system stimulation, sympathetic involvement, and the release of catecholamine (i.e., epinephrine and norepinephrine). As with other exercises, with high cardiovascular and respiratory pressure, vibration training increases the plasma tPA levels as an important driver of fibrinolysis [21]. Studies on animals have proposed that decreased tPA activity increases fibrin deposition and thus disrupts the function of various organs, eventually leading to vascular thrombosis [35]. Since muscles are regarded as a source of tPA release, vibration can enhance it by activating the muscles. On the other hand, a decrease in the PAI-1 also increases the tPA.

The findings of the present study showed that vibration significantly reduces PAI-1 as well as tPA / PAI-1 ratio. Ghazalian et al. (2014) also reported a significant decrease in PAI-1 level caused by high-intensity vibration in healthy men; however, low-intensity vibration had no significant effect on PAI-1 [34]. Various factors affect the PAI-1 decrease induced by vibration. Vibration training enhances shear stress [34]. According to some studies, the shear stress enhanced by exercises could lead to a decrease in inflammation [36]. Inflammatory factors such as Tumor necrosis factor alpha (TNF- α) also increase PAI-1, so a vibration-induced decrease in TNF-a may decrease the PAI-1.

Other studies have indicated that adiponectin decreases inflammation as well as TNF- α level [19]. In the present study, vibration significantly increased adiponectin. Baghaiee et al. (2018) reported a significant increase in adiponectin as a result of exercises among middle-aged men [37]. Bellia et al. (2014) also documented a significant increase in adiponectin induced by vibration [31]. The changes in blood adiponectin concentration have an inverse relationship with body fat mass, and its positive changes are associated with weight loss and muscle mass gain. Thus the effect of vibration on fat mass might be one of the reasons for the variations in adiponectin causes fatty acid oxidation is unknown and this might be correlated with the variations of the adiponectin expression in adipose tissue. Adipose tissue can detect energy balance and lipid content as energy storage and consequently modifies the adiponectin gene expression [40]. A huge number of the fatty acids required by the working muscles are provided with a 3- to 4-fold increase in lipolysis [41, 42]. Vibration doubles the amount of blood flow to adipose tissue and increases the blood flow to the working muscles by 10 times. Decreasing body fat and improving body composition caused by disrupting the balance between energy intake, consumption, and negative caloric balance may lead to an increased adiponectin concentration.

Conclusion

In the present study, the participants' body fat percentage, body mass index, and body weight significantly decreased, and the HDL level increased meaningfully. Vibration seems to improve the human body composition through increasing blood flow. Finally, vibration training reduces tPA / PAI-1 ratio; hence, it plays a critical role in increasing adiponectin, enhancing blood flow to muscles and adipose tissue, and decreasing body fat percentage.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

Acknowledgment

This work was supported by Kharazmi University.

Reference:

- 1. Dummer, T.J., et al., *Is overweight and obesity in 9–10-year-old children in Liverpool related to deprivation and/or electoral ward when based on school attended?* Public health nutrition, 2005; **8**(6): 636-641.
- 2. F, T., Exercise at leisure in Iranian women. Harakt, 2001; 12(12): 77-82.
- 3. mohmmadd, E., *Investigation and analysis of deterrent factors and participation rate of women in Tehran in recreational sports activities.* Research in sport science, 2007; **17**(4): 63-88.
- 4. Lavie, C.J., R.V. Milani, and H.O. Ventura, *Obesity and cardiovascular disease: risk factor, paradox, and impact of weight loss.* Journal of the American College of Cardiology, 2009; **53**(21): 1925-1932.
- 5. Alessi, M.-C. and I. Juhan-Vague, *PAI-1 and the metabolic syndrome: links, causes, and consequences.* Arteriosclerosis, thrombosis, and vascular biology, 2006; **26**(10): 2200-2207.
- 6. Nobakht, F. and F. Meamarbashi, *Gene transfer technology to improve athletic performance*. Journal of Advanced Sport Technology, 2018; **2**(1): 41-43.
- 7. Ouchi, N., et al., *Adipokines in inflammation and metabolic disease*. Nature reviews immunology, 2011; **11**(2): 85.
- 8. Hou, B., et al., *Tumor necrosis factor* α *activates the human plasminogen activator inhibitor-1 gene through a distal nuclear factor* κB *site.* Journal of Biological Chemistry, 2004; **279**(18): 18127-18136.
- 9. Aso, Y., Plasminogen activator inhibitor (PAI)-1 in vascular inflammation and thrombosis. Front Biosci, 2007; **12**(8): 2957-2966.
- 10. Bouchard, L., et al., *Contribution of genetic and metabolic syndrome to omental adipose tissue PAI-1 gene mRNA and plasma levels in obesity.* Obesity surgery, 2010; **20**(4): 492-499.
- 11. Sironi, A., et al., *Impact of increased visceral and cardiac fat on cardiometabolic risk and disease*. Diabetic Medicine, 2012; **29**(5): 622-627.
- 12. Alessi, M.-C., et al., *Plasminogen activator inhibitor 1, transforming growth factor-beta1, and BMI are closely associated in human adipose tissue during morbid obesity.* Diabetes, 2000; **49**(8): 1374-1380.
- 13. Park, H.S., J.Y. Park, and R. Yu, *Relationship of obesity and visceral adiposity with serum concentrations of CRP*, *TNF-α and IL-6*. Diabetes research and clinical practice, 2005; **69**(1): 29-35.
- 14. Bruunsgaard, H., et al., *Ageing, tumour necrosis factor-alpha (TNF-α) and atherosclerosis.* Clinical & Experimental Immunology, 2000; **121**(2): 255-260.
- 15. Mazurek, T., et al., *Human epicardial adipose tissue is a source of inflammatory mediators*. Circulation, 2003; **108**(20): 2460-2466.
- 16. Atalar, F., et al., *The role of mediastinal adipose tissue 11β-hydroxysteroid d ehydrogenase type 1 and glucocorticoid expression in the development of coronary atherosclerosis in obese patients with ischemic heart disease.* Cardiovascular diabetology, 2012; **11**(1): 115.
- 17. Dellas, C. and D.J. Loskutoff, *Historical analysis of PAI-1 from its discovery to its potential role in cell motility and disease*. Thrombosis and haemostasis, 2005; **93**(04): 631-640.
- 18. Gazioglu, S.B., et al., *PAI-1 and TNF-α profiles of adipose tissue in obese cardiovascular disease patients.* International journal of clinical and experimental pathology, 2015; **8**(12): 15919.
- 19. Chen, Y., et al., *Adiponectin inhibits TNF-α-activated PAI-1 expression via the cAMP-PKA-AMPK-NF-κB axis in human umbilical vein endothelial cells.* Cellular Physiology and Biochemistry, 2017; **42**(6): 2342-2352.
- 20. Yang, W.S., et al., *Plasma adiponectin levels in overweight and obese Asians*. Obesity research, 2002; **10**(11): 1104-1110.
- 21. Baynard, T., et al., *Fibrinolytic markers and vasodilatory capacity following acute exercise among men of differing training status.* European journal of applied physiology, 2007; **101**(5): 595-602.
- 22. McMurray, R.G. and L. Bo Andersen, *The influence of exercise on metabolic syndrome in youth: a review*. American Journal of Lifestyle Medicine, 2010; **4**(2): 176-186.
- 23. Flegal, K.M., et al., *Excess deaths associated with underweight, overweight, and obesity.* Jama, 2005; **293**(15): 1861-1867.
- 24. Yule, C.E., Does whole-body vibration training affect arterial stiffness, cognitive ability, and quality of life in chronic stroke?: a thesis presented in partial fulfilment of the requirements for the degree of Master of Sport and Exercise in Exercise Prescription and Training at Massey University, Manawatū, New Zealand. 2015, Massey University.
- 25. Adams, J.A., et al., *Periodic acceleration: effects on vasoactive, fibrinolytic, and coagulation factors.* Journal of applied physiology, 2005; **98**(3): 1083-1090.
- 26. Goto, K. and K. Takamatsu, *Hormone and lipolytic responses to whole body vibration in young men.* The Japanese journal of physiology, 2005; **55**(5): 279-284.

- 27. Ando, H. and R. Noguchi, *Dependence of palmar sweating response and central nervous system activity on the frequency of whole-body vibration*. Scandinavian journal of work, environment & health, 2003; **29**(3): 216-219.
- 28. Lee, K.W. and G.Y. Lip, *Effects of lifestyle on hemostasis, fibrinolysis, and platelet reactivity: a systematic review.* Archives of internal medicine, 2003; **163**(19): 2368-2392.
- 29. Boyle, L.J. and P.R. Nagelkirk, *The effects of whole body vibration and exercise on fibrinolysis in men.* European journal of applied physiology, 2010; **110**(5): 1057-1061.
- 30. Kent, P., G. Williams, and R. Kester, *Platelet activation during hand vibration*. British journal of surgery, 1994; **81**(6): 815-818.
- 31. Bellia, A., et al., *Effects of whole body vibration plus diet on insulin-resistance in middle-aged obese subjects.* International journal of sports medicine, 2014; **35**(06): 511-516.
- 32. Huh, J.Y., et al., *Irisin in response to acute and chronic whole-body vibration exercise in humans*. Metabolism, 2014; **63**(7): 918-921.
- 33. Menzel, K. and T. Hilberg, *Coagulation and fibrinolysis are in balance after moderate exercise in middle-aged participants*. Clinical and Applied Thrombosis/Hemostasis, 2009; **15**(3): 348-355.
- 34. Ghazalian, F., et al., *Effects of whole-body vibration training on fibrinolytic and coagulative factors in healthy young men.* Journal of research in medical sciences: the official journal of Isfahan University of Medical Sciences, 2014; **19**(10): 982.
- 35. Gebbink, M.F., et al., *Physiological responses to protein aggregates: fibrinolysis, coagulation and inflammation (new roles for old factors).* FEBS letters, 2009; **583**(16): 2691-2699.
- 36. Sallam, N. and I. Laher, *Exercise modulates oxidative stress and inflammation in aging and cardiovascular diseases*. Oxidative medicine and cellular longevity, 2016; **2016**.
- 37. Baghaiee, B., P. Karimi, and K. Ebrahimi, *Effects of a 12-week aerobic exercise on markers of hypertension in men.* Journal of cardiovascular and thoracic research, 2018; **10**(3): 162.
- 38. Lin, E., et al., *Increases in adiponectin predict improved liver, but not peripheral, insulin sensitivity in severely obese women during weight loss.* Diabetes, 2007; **56**(3): 735-742.
- 39. Mazzali, G., et al., Interrelations between fat distribution, muscle lipid content, adipocytokines, and insulin resistance: effect of moderate weight loss in older women. The American journal of clinical nutrition, 2006; 84(5): 1193-1199.
- 40. Fatouros, I., et al., Leptin and adiponectin responses in overweight inactive elderly following resistance training and detraining are intensity related. The Journal of Clinical Endocrinology & Metabolism, 2005; **90**(11): 5970-5977.
- 41. Siahkouhian, M., *Multifunction device for estimating cardiorespiratory fitness in the Step Tests.* Journal of Advanced Sport Technology, 2018; **2**(1): 2-6.
- 42. Siahkouhian, M., R. Shams, and R. Gholizadeh, A New Fitness Dependent Maximal Protocol for Determination of Heart Rate Deflection Point. Journal of Advanced Sport Technology, 2019; **3**(2): 9-18.

Corresponding Author: Manizheh Noruzian, Department of Exercise Physiology, Faculty of Physical Education and Sport Science, Kharazmi University, Karaj, Iran (email: tahereh.rashidi1991@gmail.com)

چکیدہ فارسی

تاثیر چهار هفته تمرین ویبریشن بر سطوح آدیپونکتین و نشانگرهای فیبرینولیز در در زنان دارای اضافه وزن طاهره رشیدی^۱، بهروز بقایی^۲، رامین فروزنده^۱، منیژه نوروزیان^{۱*} ۱- گروه فیزیولوژی ورزش، دانشکده تربیت بدنی و علوم ورزشی، دانشگاه خوارزمی، کرج، ایران

۲- گروه تربیت بدنی و علوم ورزشی، واحد جلفا، دانشگاه آزاد اسلامی، جلفا، ایران

سبک زندگی بی تحرک یکی از عواملی است که باعث ایجاد مشکلات پاتوفیزیولوژیکی نظیر اختلالات سیستم انعقادی و فیبرینولیز می شود. مطالعه حاضر با هدف بررسی تأثیر ۴ هفته تمرین ویبریشن تمام بدن بر فعال کننده پلاسمینوژن بافتی (TPA) ، مهار کننده فعال کننده پلاسمینوژن نوع ۱ (PAI-1) و آدیپونکتین در زنان دارای اضافه وزن انجام شد. پژوهش حاضر یک مطالعه نیمه تجربی کاربردی با یک گروه آزمایش و یک گروه کنترل با (PAI-1) و آدیپونکتین در زنان دارای اضافه وزن انجام شد. پژوهش حاضر یک مطالعه نیمه تجربی کاربردی با یک گروه آزمایش و یک گروه کنترل با استفاده از طرح پیش آزمون-پس آزمون بود. در این مطالعه مداخله ای ، ۴۵ زن شاغل با دامنه سنی ۲۵-۴۰ سال و شاخص توده بدنی 7۹–۵/۵ کیلوگرم بر متر مربع توسط آکادمی ملی المپیک معرفی شدند. شرکت کنندگانی که معیار ورود به مطالعه را داشتد، به طور تصادفی در دو گروه تجربی در گروه تربری قرار گرفتند. تمرین ویبریشن به مدت ۴ هفته انجام شد. برای تجزیه و تحلیل آماری از آزمون تی زوجی و مستقل استفاده شد. سطح TPA در روم توردی و مستقل استفاده شد. سطح TPA در رود به مطالعه را داشتد، به طور تصادفی در دو گروه تجربی در گروه تمرین ویبریشن به طور معناداری افزایش یافت (۲۹۰۰–1)، و بین دو گروه از نظر سطح TPA در مرحله پسآزمون تفاوت معنی داری وجود داشت (۲۰۱۰ گروه ویبریشن به طور معناداری افزایش یافت (۲۰۰۱–1)، و بین دو گروه از نظر سطح TPA در مرحله پسآزمون تفاوت معنی داری وجود داشت (۲۰۱۰–1). علاوهبر این، مقدار ۲۹–14 نیز در گروه ویبریشن به طور معناداری کاهش یافت (۲۰۰۱–1)، و بین دو گروه از نظر سطح TPA در مرحله پسآزمون تفاوت معنی داری وجود داشت (۲۰۰۱–1). علاوهبر این، مقدار ۲۹–14 نیز در گروه ویبریشن به طور معناداری کاهش یافت (۲۰۰۱–1)، و بین دو گروه از نظر سطح ۲۹۰ در مرحله پسآزمون تفاوت معنی داری وجود از موبریشن از مان می دارد باز ۲۹ دارد کردند و به معاداری کاهش یافت (۲۰۰۰–1). و نسبت ۲۹–14 دارد بیز پس در گروه تمرین ویبریشن به طور معناداری کاهش یافت (۲۰۰۱–2)، و بین دو گروه از نظر سطح ۲۹۰ در مرحله پسآزمون تفاوت معن داری و نور تون دارد و باز دارد (۲۰۰۱–2). و نسبت (۲۰۰۱–2)، و نورد دو کردن و و بردن از دارد و باز دارد و مو معنی داری کاهش یافت (۲۰۰۰–2)، و نورد دارد و بازمون مان و بازمون یا در در دری و و در دارد و بود مود و بود دو رد و و و دری و و

واژه های کلیدی: آدیپونکتین ، PAI-1 ،tPA ، ویبریشن ، اضافه وزن