International Research Journal of Applied and Basic Sciences
© 2013 Available online at www.irjabs.com
ISSN 2251-838X / Vol, 4 (8): 2404-2407
Science Explorer Publications

Effect of 6 week aerobic training in morning and evening on angiotensin-converting enzyme in postmenopausal hypertensive women

SedighehNasiri[*1], Parvin Farzanegi[2] and Masume Habibian[2]

1. Department of Exercise Physiology, Science and Research branch, Islamic Azad University, Mazandaran, Iran.
2. Department of Exercise Physiology, Science and Research branch, Islamic Azad University, Mazandaran, Iran.
3. Department of Exercise Physiology, Science and Research branch, Islamic Azad University, Mazandaran, Iran.

[*]***Corresponding author email:*** Shabnamnasirii@yahoo.com

ABSTRACT: The present study designed to examine the effect a period of Exercise training in morning and evening on Angiotensin converting enzyme in post-menopausal women with hypertension. This study was Quasi-experimental study by using pretest – posttest model. The subjects were hypertensive menopausal women, aged 45-55 years. We selected 21 women that their hypertensive confirmed by physician. In this study blood samples were collected in two stages, before start training and end of training in sixth week. Subjects were randomly assigned in 3 equal group consist of: control group (n= 7), morning training group (n = 7) and evening training group (n = 7). Posttest, blood sampling was done 48 hours after last session training same the first condition and finally the results of 3 groups compare together.After 6 weeks, Angiotensin converting enzyme and levels significantly decreased in the morning and evening experimental group but no significant reductions were found in control group. There is no significant difference within the effect of two time of training (morning & evening) Angiotensin converting enzyme in hypertensive postmenopausal women.Exercise training might be considered an effective non-pharmacological intervention for attenuating or inhibiting hypertension and prevention of cardiovascular diseases by reduction of Angiotensin converting enzyme in hypertensive postmenopausal women.
Keywords: Postmenopausal women, Aerobic training, Hypertension, Angiotensinconverting enzyme

INTRODUCTION

Hypertension in women is often undiagnosed or inadequately treated, especially after menopause when cardiovascular risk increases. In premenopausal women, endogenous estrogens maintain vasodilation and thus contribute to blood pressure control. Aging and the loss of endogenous estrogen production after menopause are accompanied by increases in blood pressure, contributing to the high prevalence of hypertension in older women. Currently, about 75% of postmenopausal women in the United States are hypertensive (Barton and Meyer, 2009). The high prevalence of obesity, the lack of regular physical exercise, and dietary salt are important factors contributing to and aggravating postmenopausal hypertension. In view of the ongoing population aging throughout the world, diagnosis and treatment of hypertension in postmenopausal women are important to reduce the excess burden of associated cardiovascular disease and to improve outcomes of potentially fatal complications such as stroke and myocardial infarction.

More than 25% of the female adult world population ishypertensive (Kearney et al, 2005). Elevations in blood pressure in women arerelated to cardiovascular risk (vasan et al., 2001) with theprevalence of hypertension being particularly high amongwomen aged _60 years (Kearney et al, 2005). In the United States, about 75% ofpostmenopausal women are hypertensive (Ong et al., 2007). Hypertension isoften accompanied by other cardiovascular risk factors, egobesity, dyslipidemia, and diabetes mellitus (Chobanin et al., 2003). It is noteworthythat the prevalence of hypertension-related cardiovascularcomplications is greater in postmenopausal women than inage-matched men (Rosamond et al., 2007). Indeed, these complications represent theleading cause of death in women (Rosamond et al., 2007).Clinical studies have documented beneficial effects ofantihypertensive therapy on cardiovascular outcome (Chobanin et al., 2003) even inpatients about 80 years of age (Becket at al., 2008). Overall recognition, control, andtreatment of hypertension in postmenopausal women are stillpoor in primary care, and hypertension is often not beingtreated aggressively enough (Ong et al., 2007).Thus, further improvementsof medical and public health measures, awareness ofpatients and physicians, and improved information policiesare needed.Prior to menopause, blood pressure is

lower in womencompared with age-matched men (Burt et al., 1995). During the menstrualcycle, blood pressure levels are inversely related to circulatingestrogen concentrations and lower when 17-estradiollevels peak, reflecting the vasodilator activity of endogenous17-estradiol (Dubey et al., 2002). Similarly, increases of endogenousestrogen production during pregnancy contribute to maintenanceof normotension despite marked increases in plasma volume and cardiac output (Dubey et al., 2002). The first decade after menopauseis accompanied by an increase in blood pressure. In the seventh decade of life, theprevalence of hypertension among women is even higher thanin men, regardless of ethnic background (Burt et al., 1995). Specifically,there are pronounced increases in both systolic blood pressureand pulse pressure in postmenopausal women, whereas diastolicblood pressure remains at a similar level compared with age-matched men (Burt et al., 1995). Importantly, elevatedsystolic blood pressure is considered a more sensitive predictorof future cardiovascular events than diastolic bloodpressure (Chobanin et al., 2003). The role of endogenous estrogens in the pathogenesis ofhypertension is complex. Indeed, the effects of hormonalchanges after menopause are often masked by the presence ofother cardiovascular risk factors, eg, vascular aging, arterialstiffening, obesity, age-dependent changes in insulin sensitivity, and dyslipidemia (Mueck and Seeger, 2004). Cross-sectional studies indicatethat menopause increases the risk of hypertension by 2-fold, even after adjusting for factors such as age and body mass index (Amigoni et al., 2004). Both early onset of menopause and a longpostmenopausal period are associated with higher bloodpressure levels. Moreover, hypoestrogenemic premenopausalwomen diagnosed with the polycystic ovary syndromeand menstrual irregularities because of ovarian failure are data higher risk of developing hypertension, coronary arterydisease, and adverse cardiovascular events and experiencemenopause at an earlier age than healthy controls (Shaw et al., 2008). Interestingly,a recent report indicates that physiological estradiol treatment in patients with premature ovarian failureresults in significant reductions in blood pressure, whereas noeffects on blood pressure were observed using syntheticethinylestradiol (Langrish et al., 2009). Consistent with the blood pressure–loweringeffect of physiological estrogens in women with ovarianfailure, surgically induced menopause by bilateral oophorectomyincreases blood pressure within a few weeks (Mercuro et al., 2004). Inconclusion, evidence strongly supports the notion that endogenousestrogens contribute to a basal vasodilatory state.Therefore, the loss of endogenous estrogens facilitates thedevelopment of hypertension in postmenopausal women andincreases the cardiovascular risk associated with it.The renin-angiotensin-aldosterone system (RAAS) plays a vital role in cardiovascular homeostasis, including blood pressure and mineral balance (Wolny et al., 1997). It is also a well-known fact that excess activation of this system contributes to the elevation of blood pressure in some situations. Activation of the RAAS results in increased production of angiotensin I (AI), which is converted to angiotensin II (AII) by angiotensin-converting enzyme (ACE). AII is a potent vasoconstrictor and also stimulates aldosterone secretion, which increases sodium and water retention (Erhardt, 2005). AII and aldosterone are also implicated in other potentially deleterious effects on the cardiovascular system, including endothelial damage, sympathetic activation, collagen formation and decreased nitric oxide production (Erhardt, 2005). Together, these effects put a strain on the heart, which can eventually lead to myocardial infarction or heart failure. With the understanding that the RAAS plays a vital role in the regulation of blood pressure, two drug classes, ACE inhibitors and angiotensin type I receptor blockers (ARBs), were developed to inhibit the RAAS and thus provide a potentially beneficial therapeutic approach for the treatment of hypertension. In addition, there is a growing body of evidence showing that aerobic exercise training (EXT) can reduce a number of cardiovascular risk factors (Pereira et al., 2009; Berggren et al., 2008).Indeed, EXT has been recognized as an important non-pharmacological strategy to prevent obesity and related disorders. Furthermore, a recent study demonstrated that EXT induces effects on the RAS, such as decreased cardiac ACE and AII and increased cardiac ACE2 and Ang 1–7 (Fernandes et al., 2011) in healthy rats. In addition, Pereira et al (2009) using a genetic mouse model also showed a beneficial effect of aerobic exercise on cardiac RAS components.In the present study, it was hypothesized that exercise training alters the ACE2 level in untrained postmenopausal women.

MATERIAL AND METHODS

Twenty one sedentary, non-smoking women, diagnosed with hypertension by a physician (BP ≥ 140/90 mmHg) (Canadian Hypertension Education Program (CHEP), 2007), aged 45-65 years, were participate in this study.Participants must have an office baseline blood pressure of at least 140 mm Hg systolic and/or a diastolic blood pressure of at least 90 mm Hg. Patients must not have creatinine levels greater than 1.5 times the normal level, thereby excluding patients with secondary hypertension due to renal failure. Participants who were taking medications that affect blood pressure other than the study medications were excluded.The participants were instructed not to change their physical activity routines or dietary patterns during the course of the study. Women divided to three groups (n=7), control, morning and evening aerobic exercise protocol. The experimental groups consisted of aerobic exercise morning and evening for 6 weeks of aerobic exercise, three

times a week for 45 minutes at 50% of maximum heart rate. Before and after exercise training the serum ACE activity was determined by spectrophotometric method.

The following statistical tests were applied: the Student'st-test for independent samples to compare groups; repeatedmeasurest-test to compare pre- and post-training time pointsfor normally distributed variables;Parametrically distributed variables were expressed as means ±standard deviations, and non-parametrically distributedvariables were expressed as medians and interquartile ranges.The statistical significance level was set at $p<0.05$.All statistics were performed using SPSS statistical software (16 version.).

RESULTS

Subject characteristics showed in table 1.

The mean of ACE concentrations in postmenopausal women with blood pressure that participated in the morning training, afternoon training and control are presented in Table2.

Table 1.Participants characteristics

	Age ± SE (Yr.)	Weight ± SE(Kg)	Height ± SE (cm)
Control	53.2 ± 4.66	73.44 ± 9.38	157.56 ± 1.88
morning	54 ± 3.75	74.29 ± 9.1	158.22 ± 7.51
evening	53.8 ± 3.87	68.11 ± 12.53	156.22 ± 4.58

Table 2.Statistical description of the mean concentration of ACE (U/L) among the three groups

ACE concentrate	Control ± SE	morning ± SE	evening ± SE
Pre training	78.57 ± 4.69	79.43 ± 4.76	81.14 ± 4.1
Post training	77.71 ± 3.95	60.29 ± 3.73	61.71 ± 7.2

Results showed that the lowest and highest concentrations of ACE in morningtraining group are60.29 and 79.43 U/L in post and pre exercise, respectively. These results showed that ACE concentrations significantly decreased after six weeks of training in the morning group.

Table 3.Compare of ACE level in morning grouppre and post training

ACE	Mean	SD	df	t	sig
Pre training	79.43	4.76	6	6.75	0.001*
Post training	60.29	3.73			

*shows significance differences

Results showed that the lowest and highest concentrations of ACE in evening traininggroup are 61.71 and 81.14 U/L in post and pre exercise, respectively. These results showed that ACE concentrations significantly decreased after six weeks of training in the evening group.

Table 4.Compare of ACE level in evening group pre and post training

ACE	Mean	SD	df	t	sig
Pre training	81.14	4.1	6	8.82	0.000*
Post training	61.71	7.2			

*shows significance differences

Results showed that the lowest and highest concentrations of ACE in control group are 77.71 and 78.57 U/L in post and pre exercise, respectively; but ACE concentrations have not significance difference.

Table 5.Compare of ACE level in control group pre and post training

ACE	Mean	SD	df	t	sig
Pre training	78.57	4.69	6	0.779	0.466*
Post training	77.71	3.95			

*shows significance differences

DISCUSSION

Results showed that ACE concentration at the morning training group inpre and posttraining is different and in pre was higher than post raining, which represents a decrease ACE levels after 6 weeks of aerobic training; Statistical comparisons showed that ACE concentrations after a morning 6 week of aerobic training have significant difference at the 95% confidence level. Result of evening training group was similar to morning group and showed that ACE concentration reduce after exercise and have significance difference, while certain changes in ACE concentrations of control group did not show significance difference at the pre and post training. Results with expressed results by Pescatello et al (2007) opposite, that expressed ACE concentration remains unchanged during aerobic exercise, the Miura et al results indicate ACE levelsunchanged after a

period of intense training, but similar with the results of James et al (1986) that showed lower blood pressure and ACE level after aerobic training.

Woods et al (2002) found that ACE level not has change after 20 minutes of exercise, but showed significant increase after 40 min, as well as the results of Yau and colleagues (2011) showed that after 30 min of exercise, ACE levels increased significantly prior to exercise. Although numerous studies have examined the role of ACE in various age and sex groups that have hypertension, However, few studies have examined the impact of physical activity on ACE and therefore we cannot be certain conclusive on the decrease, increase or remain unchanged ACE level, But what the comes from the results of present study andother studies, ACE levels in postmenopausal women with hypertension who participated in the morning and evening aerobic training, showed a significant difference before and after exercise. Given the prominent role of ACE in hypertension and cardiovascular problems,these enzymes control can help to improve the health of humans thatstricken high blood pressure, so, exercise regularly and monitored by experts to be one of the best and cheapest ways to prevent and treat hypertension.

REFERENCES

Amigoni S, Morelli P, Parazzini F, Chatenoud L. 2000. Determinants ofelevated blood pressure in women around menopause: results from across-sectional study in Italy. Maturitas. 34:25–32.

Barton M., Dubey RK. 2009. Postmenopausal Hypertension : Mechanisms and Therapy. Hypertension; 54:11-18.

Beckett NS, Peters R, Fletcher AE, Staessen JA, Liu L, Dumitrascu D, Stoyanovsky V, Antikainen RL, Nikitin Y, Anderson C, BelhaniA,Forette F, Rajkumar C, Thijs L, Banya W, Bulpitt CJ. 2008. Treatment of hypertension in patients 80 years of age or older. N Engl J Med. 358:1887–1898.

Berggren JR, Boyle KE, Chapman WH, Houmard JA. 2008. Skeletal muscle lipid oxidation and obesity: influence of weight loss and exercise. Am J PhysiolEndocrinolMetab 294: E726–732.

Blood Institute sponsored Women's Ischemia Syndrome Evaluation. 2008.JClinEndocrinolMetab. 93:1276 –1284.

Burt VL, Whelton P, Roccella EJ, Brown C, Cutler JA, Higgins M, Horan MJ, Labarthe D. 1995. Prevalence of hypertension in the US adult population:results from the Third National Health and Nutrition Examination Survey.1988–1991.Hypertension. 25:305–313.

Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, IzzoJLJr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ. 2003. Seventhreport of the Joint National Committee on Prevention, Detection, Evaluation,and Treatment of High Blood Pressure. Hypertension. 42:1206–1252.

Dubey RK, Oparil S, ImthurnB, Jackson EK. 2002. Sex hormones and hypertension.Cardiovasc Res. 53:688 –708.

Fernandes T, Hashimoto NY, Magalhaes FC, Fernandes FB, Casarini DE. 2011. Aerobic Exercise Training-Induced Left Ventricular Hypertrophy Involves Regulatory MicroRNAs, Decreased Angiotensin-Converting Enzyme-Angiotensin II, and Synergistic Regulation of Angiotensin-Converting Enzyme 2-Angiotensin (1–7). Hypertension 58: 182–189.

James GD, Sealey JE, Muller F, Alderman M, Madhavan S, Laragh JH. 1986.Renin relationship to sex, race and age in a normotensive population. J HypertensSuppl;4:S387–389. [PubMed: 3553481].

Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. 2005.Global burden of hypertension: analysis of worldwide data. Lancet. 365:217–223.

Mercuro G, Zoncu S, Saiu F, Mascia M, Melis GB, Rosano GM. 2004.Menopause induced by oophorectomy reveals a role of ovarian estrogenon the maintenance of pressure homeostasis. Maturitas. 47:131–138.

Mueck AO, Seeger H. 2004.Effect of hormone therapy on blood pressure innormotensive and hypertensive postmenopausal women.Maturitas.49:189 –203.

Ong KL, Cheung BM, Man YB, Lau CP, Lam KS. 2007. Prevalence,awareness, treatment, and control of hypertension among United States adults 1999 2004. Hypertension.49:69 –75.

Pereira MG, Ferreira JC, Bueno CR, Mattos KC, Rosa KT. 2009. Exercise training reduces cardiac angiotensin II levels and prevents cardiac dysfunction in a genetic model of sympathetic hyperactivity-induced heart failure in mice. Eur J ApplPhysiol 105: 843–850. doi: 10.1007/s00421-008-0967-4.

Pescatello LS, Debbie T, Nancy R, Bruce E, Gregory JT, Carl MM, Valerie D, Paul DT. 2007. Dietary calcium intake and Renin Angiotensin System polymorphisms alter the blood pressure response to aerobic exercise: a randomized control design. Nutrition & Metabolism, 4:1.

Rosamond W, Flegal K, Friday G, Furie K, Go A, Greenlund K, Haase N, Ho M, Howard V, Kissela B, Kittner S, Lloyd-Jones D, McDermott M,Meigs J, Moy C, Nichol G, O'Donnell CJ, Roger V, Rumsfeld J, SorlieP, Steinberger J, Thom T, Wasserthiel-Smoller S, Hong Y. 2007. Heart diseaseand stroke statistics–2007 update: a report from the American HeartAssociation Statistics Committee and Stroke Statistics Subcommittee.Circulation. 115:e69–e171.

Shaw LJ, BaireyMerz CN, Azziz R, Stanczyk FZ, Sopko G, Braunstein GD, Kelsey SF, Kip KE, Cooper-Dehoff RM, Johnson BD, VaccarinoV,Reis SE, Bittner V, Hodgson TK, Rogers W, Pepine CJ. 2008. Postmenopausalwomen with a history of irregular menses and elevated androgen measurementsat high risk for worsening cardiovascular event-free survival:results from the National Institutes of Health–National Heart, Lung, andBlood Institute sponsored Women's Ischemia Syndrome Evaluation.J ClinEndocrinolMetab.93:1276 –1284.

Vasan RS, Larson MG, Leip EP, Evans JC, O'Donnell CJ, Kannel WB, Levy D. 2001.Impact of high-normal blood pressure on the risk of cardiovasculardisease.N Engl J Med. 345:1291–1297.

Wolny A, Clozel J, Rein J, Mory P, Vogt P, Turino M. 1997.Functional and biochemical analysis of angiotensin II forming pathways in the human heart. Circulation Research; 80:219-27.

Woods DR, Jones A, Sanders J, HurelS. 2002. Endocrine abstracts 3 P154.

Yau A, Moss AD, James LJ, Hynes J, Ashworth J, Evans GH. 2011. Serum angiotensin I-converting enzyme response to exercise; no differential effect of genotype. Br J Sports Med;45:A4 doi:10.1136/bjsports-2011-090606.11.