World Journal of Sport Sciences 3 (2): 100-106, 2010
ISSN 2078-4724

ACE and ACE Gene Polymorphism and Some Cardiac Parameters of Fencing, Basketball and Endurance Players

[1]*H.A. Heshmat,* [2]*A.I. Azab and* [3]*A.M. El-Tantawy*

[1]Department of physiology, Fac. Vet. Med., Zagazig University, Egypt
[2]Department of Competitive and Aquatic Sports, Faculty of Physical Education, Menofia University, Egypt
[3]Department of Curriculum and Teaching Methods,
Faculty of Physical Education, Mansura University, Egypt

Abstract: The aim of this study was to know the ACE genotype, ACE serum concentration and some cardiac parameters for players of different activities (fencing, basketball and endurance) and to compare between the different activities in respect to some cardiac parameters as well as the relationship between the ACE serum concentration in all activities and some cardiac parameters at the rate of five players per each activity. The average age (y) of the players (fencing 24 ± 1,0 basketball 24 ± 1.1, endurance 23 ± 1.2) while the average weight (kg) (fencing 71 ± 1.0, basketball 76 ± 6,0, endurance 86 ± 9.0) and average length (cm) of the players (fencing 170 ± 2.0, basketball 179 ± 8.0, endurance 169 ± 7.0). The results indicated an increased ACE level in case of ACEDD (fencing and basketball) ACEII, ID (endurance). Echo (PWT, SWT and LV mass) showed higher values in ACEDD compared with II, ID while BMI increased in case of ACEDD compared with II, ID. There is a slight increase in ACEDD BP compared with II and ID as well as HR that increased slightly in genotype DD than II and ID. In conclusion ACE genotype is responsible for ACE levels in our subjects and ACEDD exerts an influence on cardiac posterior wall thickness and inter-ventricular septum together with blood pressure and HR. So ACE gene may exert an influence on cardiac size through the action of ACE.

Key words: ACE %Angiotensin converting enzyme %ACE genotype % Echocardiography

INTRODUCTION

Andreas [1] stated that ACE is a key component of the renin-angiotensin system thought to be important in the pathogenesis of hypertension. Tiret *et al.* [2] reported that the I allele was characterized by lower ACE levels and D allele by higher ACE levels.

Renin-angiotensin system (RAS), produced in the kidney, Renin cleaves angiotensinogen to yield angiotensin I, acted upon by ACE, angiotensin I is converted into Ang II (angiotensin II), whose effects are mediated predominantly through two specific human receptors (AT1, AT2). Stimulation of AT1 receptor by Ang II mediates a hypertensive response through primary vasoconstriction and salt and water retention secondary to adrenal aldosterone release. Payne and Montgomery [3] reported that polymorphism of the ACE gene has been described in which the absence (deletion or Dallele) rather than the presence (insertion or I allele) of a 287 base pair markers in intron 16 is associated with significantly higher ACE levels in the circulation Rigat *et al.* [4] as well as in tissue systems Costerousse *et al.* [5] including the human myocardium Jan Danser and Schalekamp [6].

Echocardiography is a safe and painless diagnostic procedure that uses high frequency sound waves to take dynamic pictures of the heart; sound waves emitted from a transducer penetrate the chest wall then rebound off from within the heart so that various cardiac structures can be obtained. Its function is to measure the size of the cardiac chambers, how well the heart valves are working and how forcefully the heart muscle is contracting. Robergs and Roberts [7]. The effect of physical activity and cardiovascular system has been a topic of interest for physiologists, however, cardiac dimensions in relation to physical activity is relatively new. Graf *et al.* [8] added that the regulation of myocardial mass is based on complex interactions of physical activity and genetic determinants.

Corresponding Author: A.I. Azab, Department of Competitive and Aquatic Sports,
Faculty of Physical Education, Menofia University, Egypt.

Sports activity cause basic changes in the energy required for metabolism and to ensure the excess production in muscle and cardiac contraction due to physical exertion and the hormones working to accumulate energy in the physical activity are stress hormones including growth hormone, or enzymes expressed by genes.

This storms the researchers to try knowing fencing, basket ball and endurance physical activity and its effect on cardiac dimensions characteristics in ACE and ACE gene polymorphism.

MATERIALS AND METHODS

15 players of different activities are as follows (5 fencing players, 5 basket ball players and 5 endurance players).

Venous blood (EDTA) was obtained from all participants, for genomic DNA. and serum ACE, also HR, B/P, BMI, Echo (PWT, SWT and LV mass) are measured. Each participant underwent a medical history and physical examination. None had hypertension or any chronic medical illness.

Echocardiography two dimensional Echo is recorded for PWT, SWT and LV mass were performed, the I/D ACE polymorphism was identified with polymerase chain reaction using 3 primers. ACE activity in serum was determined using commercial kit. Pulse rate was determined using pulse meter, B/P using sphygmomanometer, BMI using Tanita body analyzer.

Statistical Analysis: The values are expressed as percentage and the data were presented as the mean +/- SD 5 subjects per group were tested and the values were averaged +/-SD. Comparisons between groups were analyzed by Kruskal Wallis Test, Spearman's rho Correlation between concentration of ACE and other variables in fencing, basketball, endurance activity. P values <0.05 were defined as statistically significant.

RESULTS AND DISCUSSION

Table 1 indicated that there was no significance between the three groups (fencing, basketball and endurance) in the three variables (age, weight and height).

Table 2 revealed ACE conc., PWT,SWT, LV mass, Pulse rate, B/P and BMI of Fencing, Basketball and Endurance Players.

Table 3 revealed significance in ACE for fencing players, cardiac dimension PWT, SWT, Pulse rate, SYST. B/P, BMI for fencing players and LV mass, DIAST. B/P for basketball players.

Table 4 revealed significant correlation in PWT, SWT, B/P SYST, DIAST and BMI of fencing players. As for basketball the significance was in PWT, SWT, LV mass, B/P SYST, DIAST and BMI, in case of endurance players the significance was in SWT, PULSE rate and BMI.

Our results show that in subjects (fencing, basketball players) with DD genotype in whom serum

Table 1: Ages, weights and heights of fencing, basketball and endurance players

	Fencing N=5		Basketball N=5		Endurance N=5		
Variables	mean	SD	mean	SD	mean	SD	Sig.
Age (y)	24	1	24	1.1	23	1.2	N.S
Weight (Kg)	71	1	76	6	68	9	N.S
Height (Cm)	170	2	179	8	169	7	N.S

Table 2: Means and standard deviation of the variables in selected activities

	Fencing		Basketball		Endurance	
Variables	mean	SD	mean	SD	mean	SD
ACE concentration u/l	52.800	0.277	50.600	0.699	21.400	3.435
PWT mm	8.800	0.158	8.680	0.311	8.320	0.130
SWT mm	8.760	0.207	8.600	0.158	8.200	0.158
LV mass/g	251.200	6.723	251.800	4.550	237.200	3.347
PULSE rate c/m	72.800	1.304	72.600	1.673	70.200	1.483
B/P SYST mm HG	123.200	2.280	123.200	1.304	117.600	1.517
B/P DIAST mm HG	77.400	2.408	78.000	3.162	72.400	2.302
BMI	24.800	0.837	23.600	1.140	20.000	1.581

Table 3: Kruskal Wallis Test between activities

Variables		GROUPS	N	Mean Rank	Chi-Square	Asymp. Sig.
ACE Concentrations u/l		Fencing	5	11	9.534*	0.009
		Basketball	5	10		
		Endurance	5	3		
		Total	15			
Echocardiography	PWT mm	Fencing	5	11.6	8.859*	0.012
		Basketball	5	9		
		Endurance	5	3.4		
		Total	15			
	SWT mm	Fencing	5	11.7	9.855*	0.007
		Basketball	5	9.2		
		Endurance	5	3.1		
		Total	15			
	LV mass/g	Fencing	5	10.4	9.397*	0.009
		Basketball	5	10.6		
		Endurance	5	3		
		Total	15			
PULSE rate c/m		Fencing	5	10.2	5.947*	0.051
		Basketball	5	9.7		
		Endurance	5	4.1		
		Total	15			
B/P	SYST mm HG	Fencing	5	10.6	9.585*	0.008
		Basketball	5	10.4		
		Endurance	5	3.0		
		Total	15			
	DIAST mm HG	Fencing	5	9.8	7.174*	0.028
		Basketball	5	10.5		
		Endurance	5	3.7		
		Total	15			
BMI		Fencing	5	12	10.486*	0.005
		Basketball	5	8.9		
		Endurance	5	3.1		
		Total	15			

Table 4: Correlation between concentration of ACE and other variables in fencing, basketball, endurance activities

	fencing		Basketball		Endurance	
variables	Spearman's rho	Sig. (2-tailed)	Spearman's rho	Sig. (2-tailed)	Spearman's rho	Sig. (2-tailed)
PWT mm	0.624*	0.300	0.500*	0.391	0.051	0.935
SWT mm	0.873*	-0.100	0.600*	0.285	0.500*	0.391
LV mass/g	0.285	-0.600	0.500*	0.391	0.205	0.741
PULSE rate c/m	0.219	0.667	0.103	0.870	0.564*	0.322
B/P SYST mm hg	0.493*	-0.410	0.564*	0.322	-0.224	0.718
B/P DIAST mm Hg	0.541*	0.369	-0.500*	0.391	-0.100	0.873
BMI	0.541*	0.369	0.667*	0.219	0.600*	0.285

ACE is elevated, cardiac dimensions post wall thickness (PWT mm) and septal wall thickness together with left ventricular mass (LV mass/g.) calculated, were elevated than endurance players. Previous studies have demonstrated that cardiac ACE activity is correlated with cardiac ACE mRNA levels and that elevated cardiac ACE activity may result in increased cardiac angiotensin II levels, hence the increase in cardiac dimension was reported by Jan Danser and Schalekamp [6].

Serratosa *et al.* [9] reported that increased cardiac dimensions in the more predominantly the disciplines were and that high dynamic disciplines with a high static component show thicker through inside the normal range walls. Jonathan *et al.* [10] stated that in some highly trained athletes, the thickness of the left ventricular wall and other cardiac dimensions may increase as a consequence of exercise training and resemble that found in cardiac diseases associated with left ventricular hypertrophy and that athletes with a wall

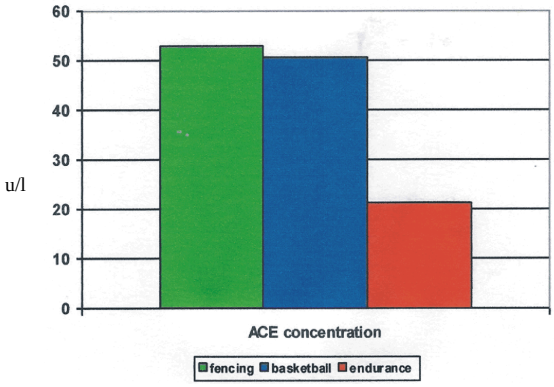

Fig. 1: ACE conc, Fencing, Basketball and Endurance Players

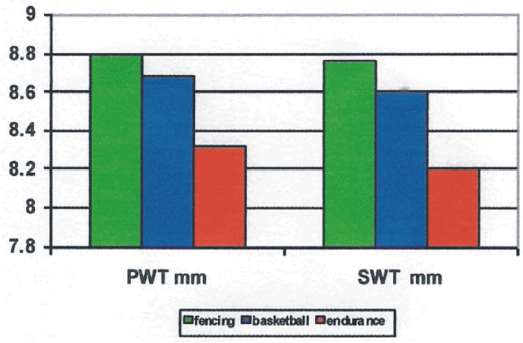

Fig. 2: PWT, SWT. Fencing, Basketball and Endurance Players

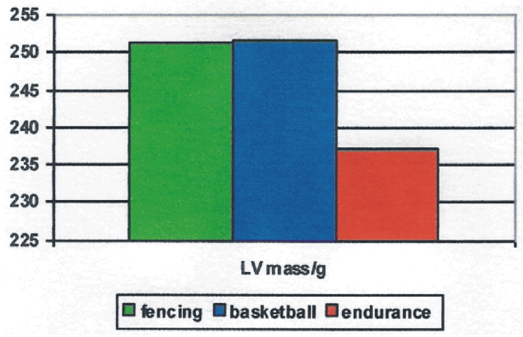

Fig. 3: LV mass, Fencing, Basketball and Endurance Players

thickness of more than 16 mm and a non dilated left ventricular cavity are likely to have primary forms of pathologic hypertrophy such as hypertrophic cardiomyopathy.

Andreas *et al.* [1] reported angiotensin converting enzyme level close to our results for II and DD genotype 24 and 58 U/l and that post wall thickness was 8.1 ±1.3 and 8.9±1.9mm for II and DD genotypes. They added that the correlation between ACE levels and posterior wall thickness suggests that this effect

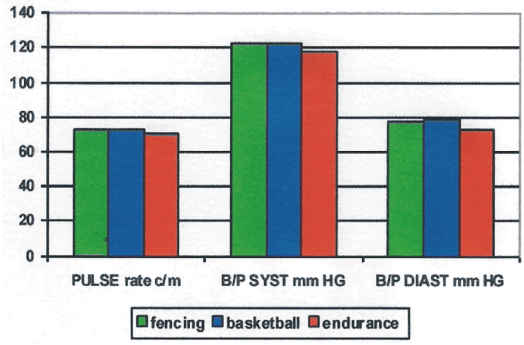

Fig. 4: PULSE rate c/m, B/P SYST mm HG, B/P DIAST mm HG. Fencing, Basketball and Endurance Players

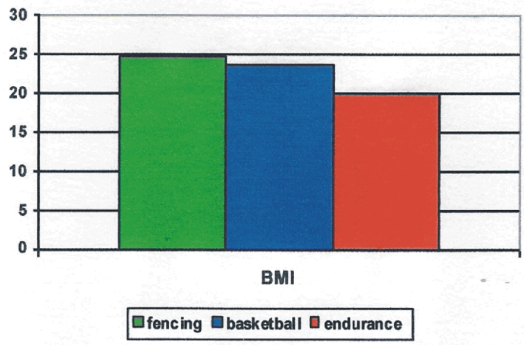

Fig. 5: BMI. Fencing, Basketball and Endurance Players

is exerted by angiotensin converting enzyme. Tiret *et al.* [2] used evidence from combined segregation and linkage analysis and showed that the I allele was characterized by lowered ACE levels as shown by our data in Tables 2 and 3, De Maria *et al.* [11], Kanalis and Hickson [12] suggested that exercise is a potent stimulus for LV growth hypertrophy, with increase in LV mass noted in response to both endurance and isometric training.

Montgomery *et al.* [13] reported that they have confirmed the ACE genotype effect on LV growth. Indra and Annapoorani [14] stated that LV mass is normally proportional to skeletal muscle man and that the former should thus be indexed for the latter, when this is done the small increase in LV mass in ACEII subjects increase in LV mass in ACEDD subjects is in excess of the increase in lean body mass. Linz *et al.* [15] assumed the increasing LV growth through either increasing activity of the cellular growth factor on the angiotensin type receptor or increased degradation of growth inhibiting kinins.

Myerson *et al.* [16] showed that cardiac growth responses are influenced by exercise intensity and type, leading to large variations in LV mass in those

participating in different sports and with different prospective training schemes. Dubach *et al.* [17] reported that even modest training changes may alter cardiac growth stimuli or responses.

Tables (2-4) indicated an increase in BMI in case of ACEDD compared with ID and II. Also there were a slight increase in B/P and HR in case of ACEDD compared with ID and II. Robergs and Roberts [7] stated that a normal BMI has been classified as 20 for men and 25 for women. These classifications of overweight are based on BMI measurements corresponding to approximately 20% or more. The increased BMI in case of fencing, basketball players compared to endurance players indicated that the increase in adipose tissue was accompanied by decrease in lean body mass.

As for the slight increase in B/P and HR in case of ACE DD compared with ACEID,II indicated that the more the musculature and the thickness of cardiac wall, septum and LV mass the more the increase in B/P and HR, Froelicher [18] reported a variety of factors affect heart rate including age, gender, physical stature, emotions, type of food consumed, also body temperature, environmental factors and exercise and in case of this work the most probable cause might be exercise and fitness and we cannot excluded other causes to affect HR and blood pressure. Hashimoto *et al.* [19] reported that the increase in blood pressure noticed in subjects having ACEDD genotype might results from the conversion of angiotensin I into angiotensin II by ACE, also due to inactivation of bradikinin, which results in increasing intraglomerular pressure due to increasing different arteriolar resistance, as for, Lewis *et al*. [20] added that the ACE gene polymorphism is related to the serum and cellular levels of ACE and accounts for half of individual variation in the serum ACE level, and the increase ACE level might elevate blood pressure.

CONCLUSION

- ACE genotype is responsible for ACE levels.
- ACE DD exerts an influence on cardiac posterior wall thickness and septum, also LV mass suggests that ACE is indeed responsible.
- ACE affects HR, B/P and BMI.
- ACE gene exerts an influence on cardiac size and development through the actions of ACE.
- It is recommended to use ACE to detect genotype.

REFERENCES

1. Andreas, B., K. Hans, K. Margit, B. Jurgen, M. Marianne, F. Hans-Dieter, B. Albert, S. Herbert and L. Friedrich, 1997. Angiotensin-Converting Enzyme and Angiotensinogen Gene Polymorphisms, Plasma Levels, Cardiac Dimensions: A Twin Study. Scientific Contribution Hypertension, 29:165-170.
2. Tiret, L., B. Rigat, S. Visvikin, C. Breda, P. Corvol, F. Cambien and F. Soubrier, 1992. Evidence from combined segregation and linkage analysis that a variant of the angiotensin 1-converting enzyme (ACE) gene controls plasma ACE levels. Am. J. Hum. Genet., 51: 197-205.
3. Payne, J. and H. Montgomery, 2003. The renin angiotensin system and physical performance. Biochemical Soc. Transaction, 31: 1286.
4. Rigat, B., C. Hubert, F. Alhenc-Gelas, F. Cambien, P. Corvol and F. Soubrier, 1990. An insertion/deletion polymorphism in the angiotensin I converting enzyme gene accounting for half the variance of serum enzyme levels. J. Clin. Invest., 86: 1343-1346.
5. Costerousse, O., J. Allegrini, M. Lopez and F. Alhenc-Gelas, 1993. Angiotensin I-converting enzyme in human circulating mononuclear cells: genetic polymorphism of expression in T-lymphocytes. Biochem. J., 290: 33-40.
6. Jan Danser, A. and M. Schalekamp, 1996. Heart., 76: 28-32.
7. Robergs, R. and S. Roberts, 1997. Exercise physiology, Mosby, U.S.A.
8. Graf, C., F. Diet and H. Predel, 2001. Correlations of the Renin Angiotensin System (RAS) Gene polymorphisms with cardiac growth factors Endothelin-1 and Angiotensin in High Performance Athletes, 6 Ann. Cong. ECSS, Cologne.
9. Serratosa, L., F. Morateand and A. Barriata, 2001. Training specific cardiac adaptations: high vs. moderate and low dynamic disciplines. 6 Ann. Cong. ECSS, Cologne.
10. Jonathan, M., G. Ute and D. Gerald, 2000. Exercise training and myocardial remodeling in patients with reduced ventricular function: one-year follow-Up with magnetic resonance imaging. Am. Heart J., 139: 252-261.
11. De Maria, A., A. Neumann, G. Lee, W. Fowler and D. Mason, 1978. Alterations in ventricular mass and performance induced by exercise training in man evaluated by echocardiography. Circulation, 57: 237.

12. Kanakis, C. and R. Hickson, 1980. Left ventricular response to program of lower -limb strength training. Chest., 78: 618-621.
13. Montgomery, H., P. Clarkson and C. Dollery, 1997. Association of angiotensin-converting enzyme gene I/D polymorphism with change in left ventricular mass in response to physical training. Circulation, 96: 741-747.
14. Indra, M. and P. Annapoorani, 2008. Relationship between the ace gene polymorphism and angiographic coronary artery disease. Ethnobotanical Leaflets, 12: 643-646.
15. Linz, W., G. Wiemer and B. Scholkens, 1996. Role of kinins in the pathophysiology of myocardial ischaemia *in vitro* and *in vivo* studies. Diabetes, 45: 851-858
16. Myerson, S., H. Montgomery and M. Whittingham, 2001. Left ventricular hypertrophy with exercise and ACE gene insertion/deletion polymorphism: a randomized controlled trial with losartan. Circulation, 103: 226-230.
17. Dubach, P., J. Myers and G. Dziekan, 1997. Effect of exercise training on myocardial remodeling in patients with reduced left ventricular function after myocardial infarction of MRI. Circulation, 95: 2060-2067
18. Froelicher, V., 1993. Exercise and Heart 3 rdEd. Mosby, U.S.A.
19. Hashimoto, Y., A. Futamura, H. Nakarai and K. Nakahara, 2001. Relationship between response of -glutamyl transpeptidase to alcohol drinking and risk factors for coronary heart disease. Atherosclerosis, 158: 465-470.
20. Lewis, E., L. Hunsicker, R. Bain and R. Rohde, 1993. The Collaborative Study Group. The effect of angiotensin-converting-enzyme inhibitors in diabetic nephropathy. N. Engl. J. Med., 329: 1456-1462.