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## **ORIGINAL ARTICLE**

# Assessment of Genetic Polymorphisms of ACE lins / Del as a factor in Causing Heart Disease - CVD in a Population of Tabriz, Iran

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#### **ABSTRACT**

In this project, 120 persons without clinical symptoms of heart disease - cardiovascular and history of venous thrombosis, the population of the city of Tabriz were evaluated. To investigate the distribution of polymorphisms of ACE Ins / Del, one of the genetic factors that cause heart disease - cardiovascular reverse hybridization method was used for the rapid and accurate diagnosis. By experiments, Multiplex PCR and hybridization techniques on the test strip. The oligonucleotide probes containing allele-specific tape parallel lines.

Keywords: venous thrombosis, Gene polymorphism, mutation frequency, angiotensin enzyme, Tabriz

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#### **INTRODUCTION**

Heart disease - cardiovascular (CVD), refers to a group of multiple failure in which the heart, arteries and veins lose their normal function. Among these diseases, venous thrombosis (VTE), third heart failure - a common vessel in the world that year, 1 out of every 1000 people involved makes  $^1$ . The main trend is to safeguard the coagulation cascade by setting the exact reactions between the components of the vessel wall, platelets and plasma proteins, blood loss  $^2$ . Defects in the regulation of the activity of this cascade may lead to the formation of arterial and venous thrombosis and life-threatening adverse  $^3$ . Gene ACE on the long arm of chromosome 17 in 17q23, and a polymorphism in intron 16 of the creation of three genotypes of domestic / internal (II), internal / deletion (ID) and deletion (DD) leads  $^4$ . ACE gene contains 26 exons, 25 introns in human beings, they are separated from each other and the length of the gene  $^5$ .

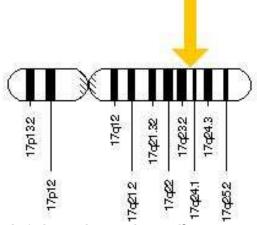


Fig 1: Gene ACE Location on Chromosome

#### **MATERIALS AND METHODS**

After obtaining the consent of the parties participating in the project, mixed with sodium EDTA peripheral blood samples were obtained from patients. Using saturated salt, DNA was extracted from the genome. The carriers were detected using reverse hybridization technique. In this technique the kit, CVD Strip Assay Austria product was used.

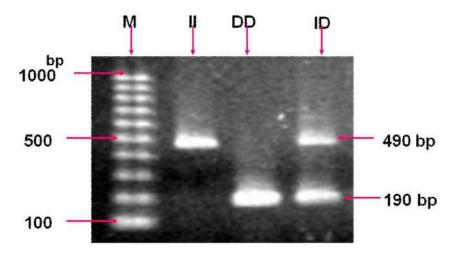


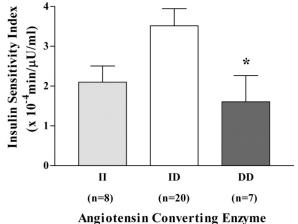
Figure 2: The band formed in genotypes II, ID, DD technique Multiplex PCR.

#### **RESULTS**

120 persons were examined in this project. According to information obtained, the most common allele causing heart disease - cardiovascular, ACE D frequency was 71%. ACE D allele homozygous state was approximately 29%.

#### **DISCUSSION AND CONCLUSION**

Heart Disease - Coronary sudden onset and is one of the major health problems in developing countries such as Iran. This group of diseases with a prevalence of 39% is the most common cause of death in the country. D allele was associated with venous thrombosis after orthopedic surgery, hypertension, and myocardial infarction associated.



Geneotype
Figure 3: Statistical Bioinformatics diagram of the enzymes involved in the genotype of angiotensin II, ID, DD.

## REFERENCES

- 1. Niu T, Chen X, Xu X (2002). "Angiotensin converting enzyme gene insertion/deletion polymorphism and cardiovascular disease: therapeutic implications". *Drugs* **62** (7): 977–93.
- 2. Roĭtberg GE, Tikhonravov AV, Dorosh ZhV (2004). "[Role of angiotensin-converting enzyme gene polymorphism in the development of metabolic syndrome]". *Ter. Arkh.* **75** (12): 72–7.
- 3. Vynohradova SV (2005). "[The role of angiotensin-converting enzyme gene I/D polymorphism in development of metabolic disorders in patients with cardiovascular pathology]". *Tsitol. Genet.* **39** (1): 63–70.

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- 4. König S, Luger TA, Scholzen TE (2006). "Monitoring neuropeptide-specific proteases: processing of the proopiomelanocortin peptides adrenocorticotropin and alpha-melanocyte-stimulating hormone in the skin". *Exp. Dermatol.* **15** (10): 751–61.
- 5. Sabbagh AS; Otrock ZK; Mahfoud ZR et al. (2007). "Angiotensin-converting enzyme gene polymorphism and allele frequencies in the Lebanese population: prevalence and review of the literature". *Mol. Biol. Rep.* **34** (1): 47–52.
- 6. Castellon R, Hamdi HK (2007). "Demystifying the ACE polymorphism: from genetics to biology". *Curr. Pharm. Des.* **13** (12): 1191–8.
- 7. Lazartigues E, Feng Y, Lavoie JL (2007). "The two fACEs of the tissue renin-angiotensin systems: implication in cardiovascular diseases". *Curr. Pharm. Des.* **13** (12): 1231–45.

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