

## GENES OF PREDISPOSITION TO MISSED ABORTION IN KAZAKH POPULATION

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**Introduction:** Missed abortion (MA) in early stages of pregnancy occurs in 10-15% miscarriages and are caused by chromosomal abnormalities in the fetus, parental chromosomal anomalies, maternal thrombophilic, anatomic, endocrine, and immunological disorders (Branch et al., 2010). Application of genetic association studies focusing on pre-selected gene of predisposition with potential pathological effect to MA show limitations to the small study size and ethnic heterogeneity.

**The aim of the study** - to investigate the input of genetic polymorphism of folate metabolism's genes (MTHFR, MTRR, MTR), fibrinolysis system of hemostasis (PLANH-1) in pathogenesis of MA in Kazakh population

**Materials and methods:** There were examined 102 women with MA in the first trimester of pregnancy (basic group) and 105 women with normal reproductive anamnesis (control group). All tested women were Kazakhs and comparable in age and somatic anamnesis. The SNP polymorphisms of rs1801131 and rs1801133 of MTHFR, rs1801394 MTRR, rs1805087 MTR genes, rs7242 of PLANH-1 gene were analyzed by PCR-real time method.

**Results:** Comparative statistical analysis revealed a statistically significant differences by decreasing the frequency of the genotypes 1298CC (MTHFR) and 66GG (MTRR) in basic group from 20,6±4,0 and 18,6±3,9 to 9,5±2,9 and 5,7±2,3 in control group ( $p<0.05$ ,  $x_2=4,9$ ; 8,1). Significant changes in the frequency of rs7242 PLANH-1 were found ( $p<0.05$ ,  $x_2=4,1$ ). The most important contribution in MA was determined for unfavorable genotypes 1298CC (MTHFR) and fibrinolysis system of hemostasis 4G/4G (PLANH-1). It was found that the carriage of haplotypes with 2 and more homozygous weak genotypes of MTHFR, MTRR, MTR and PAI-1 genes increase the risk of MA in 2,0 to 8,3 times.

**Conclusion:** We observed the significant genetic input of functionally weak genotypes 677TT and 1298CC (MTHFR), 66GG (MTRR), 2756GG (MTR) and 4G/4G (PAI-1) in MA in Kazakh population, suggesting that this genotypes cause hyper coagulation and endothelial dysfunction, which leads to disruption of normal trophoblast's differentiation and invasion.