relationship between LTA C+804A (resulting in a Thr26→Asp amino acid substitution) gene polymorphisms and CAE.
Methods: Sixty five patients with CAE (mean age 53±7 years) and 65 patients with normal coronary angiograms (mean age 51±7 years) were included in the study. The types of LTA polymorphisms were detected by the polymerase chain reaction method. For each polymorphic position, one of three possible patterns may be obtained: Normal genotype (CC), Heterozygous mutant genotype (CA), or Homozygous (AA) mutant genotype. Demographic characteristics and major risk factors for atherosclerosis were evaluated in the study groups.
Results: There was no significant difference with respect to age and gender between groups. Genotype distribution of coronary ectasia and control groups shown in Table 1. The frequency of the AA Homozygous mutant genotype was higher in CAE group than controls (9 (13.8%) vs 2 (3.1%), p = 0.027).
Between the two groups were compared according to the dominant genetic model (CA + AA vs. CC), The number of patients carrying at least one A mutant allele (CA + AA) was significantly higher in CAE than controls (51 (78.5%) vs 37 (56.9%), p = 0.009).
Conclusions: In this study, our data suggest that the lymphotoxin-alpha C804A gene polymorphisms may be assassed as a risk factor in the occurrence of CAE. However, further large-sized studies are required for determining relationship between LTA gene polymorphisms and CAE.

Table 1. Lymphotoxin-alpha C804A gene polymorphisms genotype frequencies

	CAE (n = 65)		Controls (n = 65)		P
	n:	%	n:	%	
CC homozygous normal genotype	14	21.5	28	43.1	0.009
CA heterozygous mutant genotype	42	64.6	35	53.8	0.142
AA Homozygous mutant genotype	9	13.8	2	3.1	0.027
CA + AA genotypes (Dominant genetic model)	51	78.5	37	56.9	0.009

PP-105
EVALUATION OF LYMPHOTOXIN-ALPHA C804A GENE POLYMORPHISMS IN PATIENTS WITH CORONARY HEART DISEASE

A. İçli[1], A. Altınbaş[2], Y. Türker[3], Ha. Yücel[4], S. Akçay[5], R. Sütçü[6], A. Arslan[7], F. Aksoy[2]. *[1]Department of Cardiology, Ahi Evran University Training and Research Hospital, Kırsehir, Turkey; [2]Department of Cardiology, Suleyman Demirel University, Isparta, Turkey; [3]Department of Cardiology, Duzce University, Düzce, Turkey; [4]Department of Cardiology, Isparta State Hospital, Isparta, Turkey; [5]Department of Cardiology, Celal Bayar University, Manisa, Turkey; [6]Department of Biochemistry, Suleyman Demirel University, Isparta, Turkey; [7]Department of Cardiology, Aksaray State Hospital, Aksaray, Turkey*

Background: Lymphotoxin-alpha (LTA), a pro-inflammatory cytokine, has been implicated in the pathogenesis of coronary atherosclerosis. LTA is found in atherosclerotic lesions and may contribute to these processes. Furthermore, LTA may also induce adhesion molecules and cytokines from vascular endothelial and smooth muscle cells. LTA has multiple functions in regulating the immune system and may contribute to inflammatory processes leading to coronary heart disease (CHD). We aimed to investigate relationship between LTA C+804A (resulting in a Thr26→Asp amino acid substitution) gene polymorphisms and CHD.
Methods: Sixty five patients with CHD (mean age 55±7 years) and 65 patients with normal coronary angiograms (mean age 51±7 years) were included in the study. The types of LTA polymorphisms were detected by the polymerase chain reaction method. For each polymorphic position, one of three possible patterns may be obtained: Normal genotype (CC), Heterozygous mutant genotype (CA), or Homozygous (AA) mutant genotype. Demographic characteristics and major risk factors for atherosclerosis were evaluated in the study groups.
Results: There was no significant difference with respect to age and gender between groups. Genotype distribution of CHD and control groups shown in the table. The frequency of the AA Homozygous mutant genotype was higher in CHD group than controls (13 (20%) vs 2 (3.1%), p = 0.003).
Between the two groups were compared according to the dominant genetic model (CA + AA vs. CC), The number of patients carrying at least one A mutant allele (CA + AA) was significantly higher in CHD than controls (52 (80%) vs 37 (56.9%), p = 0.005).
Conclusions: In this study, our data suggest that the lymphotoxin-alpha C804A gene polymorphisms may be assassed as a risk factor in the occurrence of CHD. However, further large-sized studies are required for determining relationship between LTA gene polymorphisms and CHD.

Table: Lymphotoxin-alpha C804A gene polymorphisms genotype frequencies

	CHD (n = 65)		Controls (n = 65)		P
	n:	%	n:	%	
CC homozygous normal genotype	12	18.5	28	43.1	0.002
CA heterozygous mutant genotype	40	61.5	35	53.8	0.375
AA Homozygous mutant genotype	13	20	2	3.1	0.003
CA + AA genotypes (Dominant genetic model)	52	80	37	56.9	0.005

PP-106
GENETIC POLYMORPHISMS OF THE HUMAN PLATELET ANTIGENS-1 IN ISOLE CORONARY ARTERY ECTASIA

A. İçli[1], A. Altınbaş[2], Y. Türker[3], Ha. Yücel[4], S. Akçay[5], F. Aksoy[2], R. Sütçü[6], H.A. Baş[2]. *[1]Department of Cardiology, Ahi Evran University Training and Research Hospital, Kırsehir, Turkey; [2]Department of Cardiology, Suleyman Demirel University, Isparta, Turkey; [3]Department of Cardiology, Duzce University, Düzce, Turkey; [4]Department of Cardiology, Isparta State Hospital, Isparta, Turkey; [5]Department of Cardiology, Celal Bayar University, Manisa, Turkey; [6]Department of Biochemistry, Suleyman Demirel University, Isparta, Turkey*

Background: Platelets play a key role in coronary artery disease pathogenesis, and increased platelets adhesion to vascular surfaces, activation and aggregation in acute coronary syndromes was previously documented Platelet adhesion and activation involve human platelet alloantigens (HPA), a complex of membrane glycoprotein (Gp) receptors with other cellbound factors on the platelet membrane surface. Several polymorphisms in the genes encoding platelet GPs have been associated with increased platelet adhesiveness and aggregation. These included HPA-1 (T196C, Leu33Pro), which are present on GPIIb/IIIa receptor complex. Conflicting results of an association between the HPA-1b allele and the risk of myocardial infarction and coronary artery disease have been reported. Coronary artery ectasia (CAE) is defined as local or generalized aneurysmal dilatation of the coronary arteries. Although clinical and pathological features have been previously described, the underlying pathophysiology has not been fully understood, the most frequent cause is coronary atherosclerosis. It is known that an expansive remodelling occurs in atherosclerotic coronary arteries due to plague rupture and increased plague burden particularly in early stages. The platelet function disorders