non-type II DM cases (p>0.01), except fasting and non-fasting glucose levels as expected (p<0.0001). In regard to the healthy controls (mean age: 33.9±7.3), 70 were males and 80 were females.

The mitochondrial DNA 16189 T>C polymorphism was determined in all cases. Overall frequency of polymorphic C allele was 0.19 in our study population including patients and healthy controls. The polymorphic C allele was found in 18 of the 70 MetS patients and in 24 of the 150 healthy controls. The frequency of this allele was not statistically significant between patients and controls (p=0.066). Also, there was no association of type II DM with the mtDNA 16189 T>C polymorphism (Table 1) (p=0.082). Distribution of mtDNA 16189 alleles did not differ in both sexes (p>0.01).

In order to determine the effect of the mtDNA 16189 T>C polymorphism on disease associated variables, statistical studies were performed, as noted in the Materials and Methods section. Means and standard deviations of these variables among 16189 C or T allele carriers are summarized in Table 2, indicating statistical significance. As seen in this table, lower levels of LDL and sup-cortisol were found in polymorphic C allele carriers in MetS patients (p=0.003 and p=0.0367, respectively). However, there was no significant association with other variables tested (p>0.01).

**DISCUSSION**

A common cluster of several risk factors for cardiovascular diseases such as dyslipidemia, hypertension, hyperglycemia has been termed syndrome X by Reaven. This syndrome, which is caused by both genetic and environmental factors, was subsequently named metabolic syndrome. Of these genetic factors, the mtDNA 16189 T>C polymorphism has been identified as a hot spot for the determination of insulin resistance, obesity and development of type II DM. This region is close to the replication origin of mtDNA and may play an important role in the maintenance of copy number per cell as well as mitochondrial transcription. In addition, mitochondrial single-stranded DNA-binding protein (mtSSB) has been shown to bind with lower affinity to the mtDNA 16189C variant than to the 16189T variant. Higher incidence of this polymorphic variant has been found to be associated with type II DM, higher fasting insulin, insulin resistance and β-cell function in Asian populations. However, others failed to find associations between metabolic phenotypes and this variant. Despite these findings, it is still questionable if this variant has a direct effect on the pathogenesis of type II DM or metabolic syndrome. An explanation for this inconsistency might be that the prevalence of the T16189C polymorphism is higher in Asia than in Europe and that the power of a polymorphism to influence disease outcome is dependent on its frequency in the population.

In this study we evaluated a total of 220 subjects (70 patients and 150 healthy controls) in terms of