type 2 and transferrin saturation more than 38% was significantly greater than that in the controls.

**HFE mutations, transferrin saturation and Ferritin values (Table 6)**

Subjects with even one mutation (C282Y or H63D) had higher transferrin saturation compared to those with no such mutations. This seems to apply to both diabetics (49± 8,6 vs 44,5± 5,4, p<0,01) and controls (49,3± 7,3 vs 42,6± 3,3 p<0,01). Ferritin was higher in control subjects carrying a mutation compared to those with no such mutation (99± 43 vs 68± 29, p<0,01). However, no such difference was detected among diabetics.

**Hereditary hemochromatosis among patients with type 2 diabetes**

The frequency of the C282Y allele was very low in the Greek population tested (0.0075) and homozygotes (C282Y/C282Y) were not detected in either group. We found 4 compound heterozygotes C282Y/H63D in the diabetics and none in the control group. This difference did not reach statistical significance (p=0.06).

**DISCUSSION**

The allele frequency for the C282Y mutation in the Hellenic population was much lower (0.0075) than the one reported in people of Celtic origin. Analogous findings to our study were reported from southern Europe. The allele frequency for the H63D mutation (0.115) was comparable to the one in other populations. In our subjects, homozygotes for hemochromatosis mutations were not detected, possibly because of the low frequency of C282Y mutation in our group. Based on these data, we cannot draw conclusions as to whether hereditary hemochromatosis is more prevalent among patients with diabetes type 2 in our population. Mutations of the transferrin receptor and the ferroportin 1 have recently been reported in Italian people. These mutations may account for at least a fraction of HH patients in whom the C282Y/C282Y or C282Y/H63D genotypes are not detected. We assume that the Greek population is genetically closer to the southern Italian population in terms of hereditary hemochromatosis causing mutations, and most likely a greater percentage of HH patients carry mutations other than the C282Y and H63D.

We did not detect any differences in the frequencies of C282Y or H63D alleles between type 2 diabetics and controls. This finding does not support the view that these mutations are in linkage disequilibrium for diabetes type 2. The discordance between our findings and those of other studies might be attributed to the different genetic background of the tested subjects.

It seems that the presence of these mutations even in the heterozygous state increases the iron load of the organism. The iron load could render peripheral tissues, and especially the liver, less sensitive to the action of insulin and consequently speed up the progression of the natural course of diabetes.

The mean serum ferritin level was found to be higher in patients with diabetes type 2 in accordance with other studies. This finding could either represent an elevated iron body stores in diabetes type 2 or simply reflect inflammation without true iron load, or else reflect a decreased clearance of glycosylated ferritin. Patients with diabetes type 2 seem to have higher transferrin saturation compared to the general population. This difference is even greater in women. The