Scores were considered useful if AUROC was >0.7 and excellent if >0.8.

**Results:** 332 patients with severe AH were treated with prednisolone 40 mg/day. At first day of treatment, patients had the following characteristics (results in medians): male gender 55.5%, ascites 74.2%, encephalopathy 24.3%, age 50.5 years (95% CI: 49.2–51.7), alcohol consumption 100 g/day (80–100), prothrombin time 20.9 sec (20.3–21.8), INR 1.9 (1.85–2), AST 109 IU/l (102–117), albumin 25 g/l (24.1–26), bilirubin 15.6 mg/dl (13.4–18), creatinine 0.86 mg/dl (0.8–0.9).

At first day of treatment with steroids, median of prognostic functions were: ABIC 8.3 (8.8–8.5), Glasgow 9 (8–9), Lille 0.31 (0.25–0.37), Maddrey 60.3 (56.2–63.4) and MELD 22.5 (21.2–23.6). The AUROC (0.84) of the Lille score was significantly higher than the AUROC of other scores: ABIC (0.76, p = 0.008), Glasgow (0.68, p = 0.0004), Maddrey (0.65, p < 0.00001) and MELD (0.7, p = 0.0002). The AUROC for ABIC score was higher than for Glasgow (p = 0.003), Maddrey (p = 0.0001) and MELD (p = 0.002). The diagnostic accuracy of the scores using baseline variables was not improved by their evolution between 1st and 7th day of treatment: ABIC 0.68, Glasgow 0.65, Maddrey 0.65, MELD 0.64. We compared the ability of the Lille, Glasgow and ABIC scores to classify patients according to their proposed cut-offs (9 for the ABIC and Glasgow scores, 0.45 for the Lille score). Percentage of patients correctly classified by these cut-offs was higher for the Lille score than for the ABIC (79.2% vs. 72%, p = 0.01) and of the ABIC and Glasgow scores, 0.45 for the Lille score). Percentage of patients correctly classified by these cut-offs was higher for the Lille score than for the ABIC (79.2% vs. 72%, p = 0.01) and of the ABIC and Glasgow scores, 0.45 for the Lille score). Percentage of patients correctly classified by these cut-offs was higher for the Lille score than for the ABIC (79.2% vs. 72%, p = 0.01) and of the ABIC and Glasgow scores, 0.45 for the Lille score).

**Conclusion:** Lille score than for the ABIC (79.2% vs. 72%, p = 0.01) and of the ABIC and Glasgow scores, 0.45 for the Lille score.

**Background and Aims:** Hepatic enzyme CYP2E1 is involved in the metabolism of a number of exogenous and endogenous substances (i.e. ethanol, drugs and chemical carcinogens). Being polymorphic, CYP2E1 gene can give different xeno-metabolic capabilities in a population and it is well known that inadequate or no enzymatic deactivation of xenobiotics could induce an increased susceptibility to disease and cancer. In particular, one of the 5′-flanking region polymorphisms, able to differentiate CYP2E1 gene transcriptional activity, is caused by the appearance/disappearance of Rsal and Pstl restriction sites, which generates two different alleles, namely *C1(Rsal+/Pstl−) and *C2(Rsal−/Pstl+)* respectively, reported to be in complete linkage disequilibrium.

**Methods:** To confirm the existence of a correlation between some particular CYP2E1 genotypes/haplotypes and hepatocarcinoma, we determined CYP2E1 Pstl/Rsal genotypes/haplotypes by RFLP-PCR in a cohort of central western Sicily hepatocarcinoma patients and in a population of healthy students from the same geographic area.

**Results:** In hepatocarcinoma patients, modal genotype association was Rsal+/Pstl−, corresponding to CYP2E1 *C1/*C1 haplotype, whereas the Rsal+/Pstl+ association, equivalent to CYP2E1 *C1/*C2 haplotype, resulted to have the lowest frequency both in patients and in controls. Moreover, both in patients and in controls, non-canonical genotype associations were frequent and arose from a no-linkage disequilibrium between the two polymorphic sites. Other authors reported this finding as a rare occurrence. Thus, from analysis of only one restriction site, Rsal+ genotype was approximately 1.5-fold more frequent in patients than in controls, and the non-canonical Rsal+ genotype was found relatively frequent in patients. Moreover, HuH7 and HAA2T transformed hepatocarcinoma cell lines also showed the Rsal+ genotype

**Conclusions:** These results suggest that the presence in CYP2E1 genotype of at least one allele with an Rsal I restriction site is correlated with hepatocarcinoma. As this site is known a consensus sequence for some specific CYP gene transcription factors, like HNF-1, it may be supposed that a single nucleotide polymorphism can alter the possibility of HNF-1 to bind CYP2E1 promoter. This could determine a marked change in the transcriptional activity of the gene, incompetence in xenobiotic metabolism or in toxic substance deactivation and an increased susceptibility to neoplastic diseases, such as hepatocarcinoma.