HBV GENOTYPE **F** AND PRECORE STOP CODON MUTATION PREDOMINATE IN AN ARGENTINIAN BLOOD DONOR POPULATION. *Paul0* H *C Franfa, UFRJ, Rio de Janeiro, Brazil;* **Jorge** *E Gonzalez, Silvina Munni, Dr C. G. Malbran, National Reference Lab / lNEI ANLIS, Buenos Aires, Argentina; Lnrissa H Brandiio, IDMT, Rio de Janeiro, Brazil; Vera Gouvea, UFRJ, Rio de Janeiro, Brazil; Emin Sablon, Barf 0 M Vanderborght, Innogenetics* ***W,*** *Gent, Belgium*

HBV-infected individuals carrying HBeAg-negative variants due to HBV precore polymorphisms often present low viremia, are more likely to have silent and persistent infections, and are subsequently at increased risk for cirrhosis and hepatocellular carcinoma. Although distinct precore mutations distributed according to geographic locality and viral genotype have been reported, epidemiological data from South America, and Argentina in particular, concerning precore mutations, genotypes, and polymerase gene polymorphisms are still scarce. We therefore surveyed their prevalence in 75 asymptomatic blood donors (HBsAg and anti-HBc positive individuals) throughout Argentina (15 cities). HBV genotypes were identified by a reverse hybridization genotyping assay (INNO-LiPA HBV Genotyping); precore promotor (1762 and 1764 nucleotides) and codon 28 polymorphisms by INNO-UPA HBV PreCore; and HBV polymerase gene (codons 108, 204, and 207) by the INNO-LiPA HBV DR assay (all Innogenetics, Gent, Belgium).

The observed prevalence for the HBV genotypes was 64% (48/75) for genotype F, 17.3% (13/75) for each of genotypes A and D, and 1.3% (1175) for genotype C. Only wild-type polymorphisms at polymerase codons 180, 204, and 207 were found. An extremely high proportion of stop codon mutation (UAG) at the precore codon 28 (66.7%; 50/75) was observed in this symptomless population. Wild-type codon (UGG) was present in 29.3% (22/75) of the samples, and the remaining 4% (3/75) gave a mixed pattern (UGG and UAG in the same sample) by LiPA. The combination of A at 1762nt and G at 1764nt precore promotor was found in 58.7% (44/75) of the samples. The T 1762nt / A 1764nt profile was observed in 28% (21/75) of samples. Only one sample showed A at both promotor positions examined. Mixed precore promotor polymorphisms were found in 4% (3/75) of samples. A correlation between genotype F and high prevalence of precore stop codon mutation was found ($p<0.05$). Previously reported associations between genotype A with wild-type codon 28 and genotype D with mutant-type codon 28 were also observed. In conclusion, HBV genotype F and precore stop codon mutation 28 predominated among a geographically broad Argentinian blood donor population. In countries like Argentina, where genotype F prevails, special attention should be given to the detection of HBeAgnegative variants.

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