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SINGLE NUCLEOTIDE POLYMORPHISMS IN MTTP GENE AS RELEVANT HOST FACTORS IN HEPATITIS C VIRUS INFECTION

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Hepatitis C virus (HCV) infection can influence the serum levels of glucose and lipids, resulting in a high prevalence of insulin resistance and hepatic steatosis. The aim of this study was to evaluate the influence of the MTTP -164T/C (rs1800804), -400A/T (rs1800803) and H297Q (rs2306985) single nucleotide polymorphisms (SNPs) on insulin and lipid levels, and on steatosis. This work was supported by the São Paulo Research Foundation (FAPESP), grant #2016/19690-5. Genotyping was undertaken in 236 HCV monoinfected Brazilian patients. 56.4% were female and the mean age was 55.5 years. The frequencies of mutated alleles in the -164T/C, -400A/T, H297Q, I128T and Q95H SNPs were 0.30, 0.41, 0.50, 0.30 and 0.09 respectively. The genotype distributions of these SNPs were in Hardy-Weinberg equilibrium ($p \geq 0.05$). Linkage disequilibrium analysis were performed and the results have demonstrated strong linkage disequilibrium between -164T/C and I128T (D'=0.97). The -164T/C SNP was associated with high insulin levels in the three genetic models studied (TT vs TC vs CC; TT vs TC/CC; TT/TC vs CC, $p<0.05$) (Figure 1), these associations were not observed in -400A/T SNP. Elevations in insulin levels were also associated with H297Q SNP in the dominant model (HH vs HQ/QQ, $p=0.049$) (Figure 1). Polymorphisms in the MTTP gene seems to have important implications in insulin levels. No association was observed between lipid levels alterations and these SNPs. This study identified a significant interaction between the presence of mutated alleles in the MTTP -164T/C and -400A/T SNPs and HCV genotype 3 ($p=0.004$ and $p=0.032$, respectively) (Table 1 and 2), suggesting that host factors in conjunction with viral factors may increase the risk of developing steatosis in patients with chronic hepatitis C. Based on these findings, HCV association with host factors may influence the development of HCV-related comorbidities such as insulin resistance and hepatic steatosis.

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