ABSTRACT
The data presented here differs from certain reports in other regions. In half of the isolates, the occurrence of anti-HBe phenotype could not be explained by any of these previously described mutations.

INTRODUCTION
The purpose of this study was to evaluate the frequency of mutations in the core promoter (CPR) and precore regions of hepatitis B virus in anti-HBe positive Brazil carriers.

Methods:

The study was performed in five Brazilian carriers and nine non-carriers matched for age, sex, and geographic location. The study was conducted in the Brazilian population of the city of Rio de Janeiro (Rio Grande do Sul, Brazil). The study was carried out within the study period of 1999 to 2014.

Results:

The frequency of the two loci showed higher frequency in anti-HBe positive Brazil carriers than in non-carriers. The frequency of the two loci was higher in anti-HBe positive Brazil carriers than in non The presence of surface antigens in serum that persist for more than six months can lead to chronic hepatitis B infection, which can cause a wide range of liver disease, including carrier-state, CAH, cirrhosis, and HCC. Although some studies have linked the A1896 mutation to an increase in clinical symptoms of human mitosis caused by HBV, others have shown alterations in the precore region that prevent this process from being repeated. In addition to the A1896 mutation, there have been multiple point mutations in the precore region that cause initiation failure or premature termination, as well as deletions and insertions of nucleotides leading to frameshifts. The regulation of the precore and core genes' transcription and expression has been the subject of extensive research. Mutations in the core promoter, such as the double mutation at positions 1762 and 1764 that changes AGG to TGA, have been proposed to mediate down-regulation of HBeAg production. Such mutations can prevent or reduce the transcription of prekord mRNA and HBE synthesis and are observed in chronic carriers that exhibit anti-HBe positive status. The precore and core promoter regions of HBV isolates (genotypes A, D, and F) from anti-HBe positive Brazilian patients are characterized by the mutations in this study.

CONCLUSION
Remarkable conclusions This was a very different pattern of core promoter and precore mutations of HBV's caused by anti-HBe Brazilian carriers: isolates A1896 and A1762-1764 had hardly any TGA (17%) mutation in them, while those from genotype D showed none (p 0.05) and other points that were frequently associated with the common point mutation—places 1727, 1740, 175 and 1773 and in the hypervariable region (nt 1751–1765). On the other observation Until recently, it was widely assumed that HBV genotypes are responsible for the outcome of hepatitis B infection. However, recent research has revealed recombination events between different HBS genotypses, which could explain geographical differences in the natural history of this disease. Central and South America are the only regions where genotypic A, D and F co-circulate at a large scale. This may explain how HBRs may affect the core promoter and precore mutations. Nucleotide sequencing of the whole genome of many HBAs caused

significantly South American H