INTRODUCTION

Chronic hepatitis comprises a group of disorders with inflammation of the liver that has been present for more than 6 months. The most common cause is chronic viral hepatitis related to viral hepatitis B,C or D ***[Willson 1995].***

Hepatitis B is a cause of acute and chronic hepatitis throughout the world. More than 5% of the world population are chronic carriers of the virus. Acute infection with HBV leads to the development of chronic hepatitis in about 5% of infected adult patients, in addition as many as 1% of patients with acute hepatitis B develop fulminant hepatitis. Therefore, hepatitis B causes significant morbidity and mortality [***Boyer & Strauss 1994].***

The presence of HBV in serum has previously been determined by an evaluation of the immunological markers. With these procedures, the primary indicators of infectious viral particles have been HBsAg and HBeAg. However, these antigens can exist in a free form, as the HBeAg can be detected in blood when free of viral particles, hence the correlation with infectious particles is not absolute ***[Robinson 1995].***

After cloning HBV genome, molecular hybridization techniques have been established for detection of HBV-DNA in the serum and liver tissue. HBV-DNA is presently the

most sensitive marker of viral replication and infectivity, which was previously related to the presence of HBeAg in serum and HBcAg in liver cells. HBV-DNA is usually analyzed by different hybridization techniques and it has become a valuable part of the routine diagnosis in chronic hepatitis B, providing a more reliable estimation of viral replication and contagiousness and a better parameter for severity and prognosis of chronic infection ***[Wirth et al 1991; Wirth & Zabel 1992, and Willson 1995].***

Cautions should be taken to clarify those so called normal individuals who have no symptoms of hepatitis B, no HBsAg in the serum and normal transaminases, but have HBV replication in their bodies with low levels of HBV-DNA ***[Luo et al 1992 and Fong et al 1993],*** and it has become apparent that many HBeAg-negative patients still have small amount of circulating viral DNA ***[Buti et al 1993; Soni et al 1994 and Willson 1995].***

Also, HBV-DNA is a more sensitive and precise marker of viral replication and infectivity if compared with HBeAg, especially in Mediterranean countries, where a frequent variant form of HBV is characterized by severe chronic active hepatitis and the presence of HBV-DNA without HBeAg in the serum, because of a mutation that prevents synthesis of HBeAg in infected hepatocytes [pre-core mutant]. ***[Friedman 1995 and Akarca & Lok 1995]***