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11

Clinical relevance of hepatitis B virus variants

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Abstract

The hepatitis B virus (HBV) is a global public health problem with more than 240 million people chronically infected worldwide, who are at risk for end-stage liver disease and hepatocellular carcinoma (HCC). There are an estimated 600,000 deaths annually from complications of HBV-related liver disease. Antiviral therapy with nucleos(tide) analogs (NA) targeting the HBV polymerase (P) can inhibit disease progression by long-term suppression of HBV replication. However, treatment may fail with first generation NA therapy due to the emergence of drug-resistant mutants, as well as incomplete medication adherence. The HBV replicates via an error-prone reverse transcriptase leading to quasispecies (QS). Due to overlapping open reading frames (ORF) mutations within the HBV P can cause concomitant changes in the HBV surface gene (S) and vice versa. HBV quasispecies diversity is associated with response to antiviral therapy, disease severity and long-term clinical outcomes. Specific mutants have been associated with antiviral drug resistance, immune escape, liver fibrosis development and

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