

Abstract

Worldwide, primary liver cell carcinoma (PLC) is one of the most common tumours. Epidemiological evidence has implicated hepatitis B virus (HBV) in its aetiology and the mechanisms whereby HBV could operate at the genomic level have been investigated using the techniques of molecular biology. The resemblance of certain features of HBV to the retroviruses has also suggested mechanisms whereby malignant transformation may take place, but as yet there is no clear evidence for HBV being directly oncogenic. This has suggested to some that it is the persistent inflammatory reaction caused by HBV infection that is instrumental in causing PLC. We believe, however, that HBV can act independently of this mechanism and that the failure so far to show this at the molecular level may be due to technical reasons.