Hepatocellular carcinoma (HCC) is a common malignancy especially among middle-aged men in South-east Asia and sub-Saharan Africa. These regions of high prevalence are also endemic for hepatitis B virus (HBV) infection which is one of the aetiologic agents for HCC. Its prognosis is poor as a result of late presentation; hence various screening tests have been advocated among high risk individuals for early diagnosis at a stage when the disease is potentially curable.

A specific mutation in the codon 249 (AGG → AGT transversion) of the p53 tumour suppressor gene has been identified as a ‘fingerprint’ for exposure to aflatoxin, another aetiologic agent for HCC. High prevalence of this genetic aberration has been found in places of high exposure to aflatoxin which are also endemic for HBV infection and is presently being advocated as one of the biomarkers for aflatoxin exposure as well as a screening tool for early diagnosis of HCC. This study was carried out to determine the prevalence of this genetic mutation among adult HCC patients and controls at the University College Hospital (UCH), Ibadan as well as identify other possible aetiologic agents for HCC.

Sixty-one HCC patients and 60 non-liver disease control subjects matched for age and gender were recruited for the study. The p53 codon 249 status was determined by restriction fragment length polymorphism (RFLP). Hepatitis B surface antigen (HBsAg) and antibody to hepatitis C (anti-HCV) status were determined by third generation enzyme linked immunosorbent assay technique. Alcohol ingestion was estimated in grams/ day; with alcohol intake ≥ 40g/day for at least 10 years considered as being significant.

The prevalence of p53 codon 249 mutation was not significantly higher among HCC patients than in controls; 6.9% versus 0% (p = 0.119). The prevalence of HBsAg was 58.3% in the HCC patients.
versus 7.7% in controls (p = 0.002) and for anti-HCV, 6.0% in HCC patients and 11.5% in controls (p = 0.180). Twenty-two (38.6%) HCC patients drank significant amount of alcohol versus 13.3% of controls (p = 0.003). The crude odds ratio for HCC among those who were positive for HBsAg, anti-HCV and drank significant alcohol was 16.7 (95% CI = 5.276 – 52.857), 0.489 (95% CI = 0.115 – 2.073) and 4.086 (95% CI = 1.909 – 10.207) respectively.

It is therefore concluded that the prevalence of p53 codon 249 mutation is low among HCC patients at the UCH, Ibadan and so may not explain the molecular pathogenesis of HCC in this group of patients. HBV infection and alcohol play significant role in the aetiology of HCC among patients at the UCH, Ibadan whereas HCV and aflatoxin play less significant roles. Efforts should be made to prevent and reduce infection by these viruses as well as the long-term sequelae of these infections. Further search for the appropriate biomarker for HCC in this environment is necessary.