中文題目：*PNPLA3* 基因變異與 C 型肝炎患者接受抗病毒藥物後肝癌發生無關

英文題目：*PNPLA3* genetic variants do not impact on the development of HCC in HCV patients receiving anti-viral therapy

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Background: Patatin-like phospholipase domain-containing 3 (*PNPLA3*) genetic variants added the risk of hepatitis C virus (HCV) related liver fibrosis progression. Its role in hepatocellular carcinoma (HCC) development remains unlcear

Methods: 763 patients receiving pegylated interferon plus ribavirin combination therapy consecutively recruited. *PNPLA3* rs738409 genotype was tested for its association with HCC in the cohort.

Results: Fifty seven (8.6 %) patients developed HCC with a median follow-up period of 47.8 months (range: 6–129 months). Cox-regression analysis revealed that the strongest factor independently associated with HCC in the treatment cohort was liver cirrhosis (hazard ratio [HR]/ 95 % confidence intervals [CI]: 4.75/2.631-8.569, P<0.001), followed by non-sustained virological response (SVR) (HR/CI: 1.80/1.040-3.121, P=0.036), old age (HR/CI: 1.03/1.001-1.064, P=0.04), low platelet counts (HR/CI: 0.993/0.987-0.998, P=0.01) and high *r*-GT levels (HR/CI: 1.005/1.002-1.008, P=0.003). The risk of HCC development did not differ between patients with *PNPLA3* rs738409 GG or non-GG genotype (7.5 %vs. 8.8 %, Log-Rank test p=0.86). The risk of HCC development did not differ between the carriage of *PNPLA3* rs738409 GG or non-GG genotype in patients with (4.8 % vs. 6.4 %, Log-Rank test p=0.85) or without (12.0 % vs. 17.7 %, Log-Rank test p=0.61) SVR. *PNPLA3* genetic variants also did not determine HCC occurrence in each subpopulation ranging from low risk (SVR & non-cirrhosis) to high risk (non-SVR & cirrhosis) patients.

Conclusions: *PNPLA3* genetic variants do not impact on HCV related HCC