Table 3 Factors associated with sustained virological response in patients with chronic hepatitis C who underwent 48 weeks of pegylated inferferon-α plus ribavirin therapy

	UVA			MVA	
	SVR	Non-SVR	*p*	OR (95 % CI)	*p*
Number	74 (38 male, 36 female)	45 (31 male, 14 female)	0.06		
Age (years)	55.4 ± 10.1	58.2 ± 10.0	0.122		
WBC (/mm^3)	5,043 ± 1,695	5,248 ± 1,363	0.247		
Hb (g/dL)	14.3 ± 1.5	14.4 ± 1.6	0.504		
Plt (×10^4/mm^3)	18.2 ± 4.6	16.9 ± 6.0	0.186		
TP (g/dL)	7.5 ± 0.6	7.6 ± 0.5	0.292		
Alb (g/dL)	4.2 ± 0.4	4.1 ± 0.4	0.575		
AST (U/L)	47.5 ± 27.9	66.5 ± 50.0	0.049	1.012 (0.997–1.027)	0.108
ALT (U/L)	66.4 ± 47.9	80.0 ± 62.9	0.286		
T-bil (mg/dL)	0.7 ± 0.3	0.9 ± 0.4	0.101		
T-chol (mg/dL)	178.1 ± 36.8	174.3 ± 37.7	0.717		
AFP (ng/mL)	7.1 ± 7.8	14.1 ± 18.8	0.062		
HCV RNA (log IU/mL)	6.3 ± 0.7	6.3 ± 0.5	0.753		
IFNL3 rs8099917 (TT/non-TT)	70:4	30:15	<0.0001	17.25 (3.34–89.13)	0.001
Histological activity score (A0-A1/A2-A3)	45:20	24:15	0.454		
Fibrosis score (F1–F2/F3-F4)	57:8	27:12	0.023	0.239 (0.072–0.798)	0.02
IFN-λ_3 (pg/mL)	17.3 ± 31.7	11.8 ± 14.9	0.262		
IP-10 (pg/mL)	458.0 ± 404.9	504.7 ± 364.0	0.208		
MIP-1α (pg/mL)	13.1 ± 36.1	4.2 ± 5.6	0.026	0.66 (0.457–0.956)	0.028
MIP-1β (pg/mL)	195.7 ± 204.3	154.9 ± 81.5	0.865		
RANTES (pg/mL)	18,125 ± 8,076	16,597 ± 7,946	0.187		
PDGF-BB (pg/mL)	3,931 ± 1,846	3,312 ± 1,803	0.079		

Alb albumin, *AFP* α-fetoprotein, *ALT* alanine aminotransferase, *AST* aspartate aminotransferase, *CI* confidence interval, *Hb* hemoglobin, *HCV* hepatitis C virus, *IFN-λ3* interferon-λ$_3$, *IP-10* interferon-γ-inducible protein 10, *MIP-1α* macrophage inflammatory protein 1α, *MIP-1β* macrophage inflammatory protein 1β, *MVA* multivariate analysis, *OR* odds ratio, *PDGF-BB* platelet-derived growth factor BB, *Plt* platelets, *RANTES* regulated on activation, normally T cell expressed, and secreted, *T-bil* total bilirubin, *T-chol* total cholesterol, *TP* total protein, *UVA* univariate analysis, *WBC* white blood cells

function was reported for PDGF-BB, the level of which is reported to be increased in patients with advanced/fibrosis stages of HBV infection [32, 33]. These reports support the notion that IFN-λ$_3$ is related to liver inflammation and fibrosis. As well as in B-CH patients, a positive correlation was observed between serum IFN-λ$_3$ levels and inflammation (AST levels) and fibrosis markers (FIB-4 score and APRI). Secondly, we examined whether serum IFN-λ$_3$ and chemokines are involved or not involved in the SVR to PEG-IFN-α plus RBV therapy for C-CH patients. We confirmed that *IFNL3* genotypes, fibrosis score, and MIP-1α are associated with SVR in this cohort, but failed to do so with IP-10 and serum IFN-λ$_3$. Several studies showed that pretreatment IP-10 levels could be a predictor of SVR in PEG-IFN-α plus RBV therapy for C-CH [34], the significance of which became stronger in combination with *IFNL3* genotypes [35, 36]. One of the reasons why the IP-10 levels failed to be significant in this study may be a bias for the enrollment of patients from multiple hospitals and medical centers.

In summary, serum IFN-λ$_3$ levels are increased in patients with chronic HCV infection regardless of the *IFNL3* genotype, the level of which is associated with liver inflammation and fibrosis. The biological role and clinical impact of IFN-λ$_3$ in patients with chronic HCV infection need to be investigated further.

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Conflict of interest The authors declare that they have no conflict of interest.

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