IL-26 is overexpressed in chronically HCV-infected patients and enhances TRAIL-mediated cytotoxicity and interferon production by human NK cells

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Résumé en anglais	Objective Interleukin-26 (IL-26) is a member of the IL-10 cytokine family, first discovered based on its peculiar expression by virus-transformed T cells. IL-26 is overexpressed in chronic inflammation (rheumatoid arthritis and Crohn's disease) and induces proinflammatory cytokines by myeloid cells and some epithelial cells. We thus investigated the expression and potential role of IL-26 in chronic HCV infection, a pathology associated with chronic inflammation.Design IL-26 was quantified in a cohort of chronically HCV-infected patients, naive of treatment and its expression in the liver biopsies investigated by immunohistochemistry. We also analysed the ability of IL-26 to modulate the activity of natural killer (NK) cells, which control HCV infection. Results The serum levels of IL-26 are enhanced in chronically HCV-infected patients, mainly in those with severe liver inflammation. Immunohistochemistry reveals an intense IL-26 staining in liver lesions, mainly in infiltrating CD3+ cells. We also show that NK cells from healthy subjects and from HCV-infected patients are sensitive to IL-26. IL-26 upregulates membrane tumour necrosis factor (TNF)-related apoptosis-inducing ligand (TRAIL) expression on CD16− CD56bright NK cells, enabling them to kill HCV-infected hepatoma cells, with the same efficacy as interferon (IFN)-α-treated NK cells. IL-26 also induces the expression of the antiviral cytokines IFN-β and IFN-γ, and of the proinflammatory cytokines IL-1β and TNF-α by NK cells. Conclusions This study highlights IL-26 as a new player in the inflammatory and antiviral immune responses associated with chronic HCV infection.

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