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IN BRIEF

VIRAL HEPATITIS

IL-32: A new proinflammatory cytokine involved in HCV-related liver inflammation and fibrosis

Moschen, A. R. *et al. Hepatology* doi:10.1002/hep.24285

IL-32 is a proinflammatory cytokine that activates p38 MAPK and NFκB and thus induces the production of proinflammatory cytokines such as IL-1β and TNF. Moschen *et al.* investigated the role of IL-32 in HCV infection and found positive associations with levels of steatosis, inflammation, fibrosis and alanine aminotransferase both *in vitro* and *in vivo*. They also found that expression of IL-32 is regulated by proinflammatory stimuli.

VIRAL HEPATITIS

High predictive accuracy of an unbiased proteomic profile for sustained virologic response in chronic hepatitis C patients

Patel, K. *et al. Hepatology* doi:10.1002/hep.24284

Only half of patients with chronic HCV infection achieve a sustained virologic response with current standard-of-care therapy. Accurate predictors of host response to treatment are therefore needed. Data from the study by Patel *et al.* demonstrate that a serum-based protein signature can accurately predict treatment response to the current standard-of-care in most patients with chronic HCV infection.

VIRAL HEPATITIS

Occult and previous hepatitis B virus infection are not associated with hepatocellular carcinoma in US patients with chronic hepatitis C.

Lok, A. S. *et al. Hepatology* doi:10.1002/hep.24257

Previous studies have indicated that prior infection with HBV might increase the risk of hepatocellular carcinoma (HCC) in patients with chronic hepatitis C. However, this study in the USA investigated the prevalence of previous or occult HBV infection in patients with chronic hepatitis C who did or did not have HCC and found that it was not an important factor in HCC development.

VIRAL HEPATITIS

MicroRNA-122 antagonism against hepatitis C virus genotypes 1–6 and reduced efficacy by host RNA insertion or mutations in the HCV 5' UTR

Li, Y. P. *et al. Proc. Natl Acad. Sci. USA* doi:10.1073/pnas.1016606108

MicroRNA-122 (miR-122) stimulates HCV replication. It is thought that miR-122 interacts with two adjacent sites downstream of stem loop I within the HCV 5' untranslated region (UTR). Li *et al.* have demonstrated that miR-122 antagonism has a potent antiviral effect against HCV genotypes 1–6. They also found that recombinant HCV, with mutations in the 5' UTR region, was not affected by miR-122 antagonism.