P50 APOLIPOPROTEIN E AND LOW-DENSITY, APOLIPOPROTEIN B ASSOCIATED LIPOVIRAL PARTICLES IN CHRONIC HEPATITIS C INFECTION: EVIDENCE FOR GENOTYPE-SPECIFIC MODULATION OF LIPID PATHWAYS
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Introduction Hepatitis C virus (HCV) co-opts the VLDL assembly, maturation, degradation, and secretory machinery of hepatocytes. Infectious low density particles have been termed lipoviral particles (LVP). LVPs in vivo are triglyceride (TG) rich and contain at least viral RNA, HCV core protein and the VLDL components apolipoprotein B (apoB) and apoE. ApoE is a constituent of infectious HCV particles produced in cell culture, and production of infectious particles is dramatically impaired from cells in which apoE expression has been genetically silenced.

Aim To examine the relationship between LVP and apoE in vivo.

Method Fasting plasma samples were obtained from 39 chronic HCV genotype (G) three patients and 51 HCV G1 patients. LVP were measured using iodixanol density gradient ultracentrifugation as recently described.1 ApoE levels were determined by an automated immunonephelometric method. Demographic data were measured in all patients.

Results were measured in all patients.

HOMA-IR, again contrasting to G1 infection1. In the whole cohort, there was no change in IDO expression at week 12 vs baseline (ΔCt=10.2±0.23 vs ΔCt=9.76±0.59, p=NS), although serum Kyn/Tyr increased significantly (p<0.001).

Conclusion This study suggests that while serum apoE quantity is a positive determinant for LVP quantity in HCV G1 infection, it is a negative determinant in HCV G3 infection. Furthermore, LVP quantity in HCV G3 is largely based on the paucity of HDL quantity and its components, rather than the parameters associated with TRL levels as in HCV G1. These differences highlight that interaction with host lipoprotein metabolism is important for HCV infection in different genotypes, but in genotype specific ways.

REFERENCE

P52 MACROPHAGE INFLAMMATORY PROTEIN-1 α/CC CHEMOKINE LIGAND 3 AND TUMOUR-ASSOCIATED MACROPHAGES IN HEPATITIS C VIRUS-RELATED HEPATOCELLULAR CARCINOMA: RELATION TO TUMOUR PROGRESSION AND ANGIOGENESIS
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Introduction Hepatitis C virus (HCV) is a major risk factor for development of hepatocellular carcinoma (HCC), however, the mechanism of hepatocarcinogenesis in HCV infection is still undefined. Chemokines, which can induce the migration of leucocytes and activate inflammatory/immune responses, have recently been implicated in the regulation of tumour growth.

Aim Therefore, the aim of the present work was to study the role of macrophage inflammatory protein-1 α/CC chemokine ligand 3 (MIP-1α/CCL3), a potent macrophage chemoattractant, in the
P51 Interferon-induced fatigue in hepatitis C infected patients is associated with increased expression of indoleamine 2,3-dioxygenase in peripheral blood mononuclear cells
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