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Introduction Identification of factors that influence response to treatment are important for individualised care in the era of direct acting antivirals. Interferon-γ inducible protein-10 (IP10) is one of hundreds of interferon stimulated genes (ISGs). Paradoxically, high ISGs, notably IP10 are associated with poor treatment response. Although HCV hijacks the VLDL pathway in its life cycle, another paradox is that high LDL cholesterol is a marker of favourable outcome. We have shown that infectious HCV LVP correlate with insulin resistance and that high LVP ratio (LVP ratio = LVP/LVP + non LVP) is associated with lower EVR.[1]

Aim To evaluate the interactions between LVP, IP10 and lipid profiles.

Methods Fasting samples were collected from 71 consecutive HCV G1 patients. LVP and LVP ratio were determined as previously described.[1] Fasting lipids, apoB and apoE were measured using automated enzymatic methods. IP10 was measured using a commercially available ELISA (R&D Systems, Abingdon, UK).

Results Univariate analysis demonstrated a relationship between IP10 and LVP ratio (r=0.31, p=0.028) but not between total viral load and IP10 (r=0.005, p=0.973). IP10 levels inversely correlated with LDL cholesterol (r=-0.302, p=0.017) and apoB (r=-0.295, p=0.015). IP10 levels also strongly positively correlated with apoE (r=0.444 p<0.001). ApoE positively correlated with LVP ratio (r=0.498, p=0.001) but not with total viral load (r=0.035, p=0.826). When apoE and IP10 were included in the multivariate model as predictors of LVP ratio, IP10 was no longer a significant correlate with LVP ratio (p=0.446) suggesting that the relationship between IP10 and LVP ratio is mediated via apoE.

Conclusion This data indicates there is a close link between ISGs and lipoprotein metabolism. High IP10 correlates with apoE which is an exchangeable lipoprotein that transfers from HDL to triglyceride-rich lipoprotein remnants where it mediates clearance via LDLR, a candidate HCV receptor. ApoE is important in HCV replication/assembly and is a constituent of infectious LVP. Hence the effect of the endogenous interferon response on lipids is favourable to the virus by promoting LVP. This provides a possible explanation for the interferon paradox.

Competing interests None.

Keywords apolipoprotein E, cholesterol, hepatitis C virus, interferon gamma inducible protein 10, lipoviral particle

REFERENCES
1. Bridge SH *et al. Gut* 2010.

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OC-109 **THE 'INTERFERON PARADOX' AS A PREDICTOR OF NON-RESPONSE TO ANTI-VIRAL THERAPY: NOVEL ASSOCIATION WITH APOLIPOPROTEIN E AND HEPATITIS C VIRUS (HCV) LIPOVIRALPARTICLES (LVP).**

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