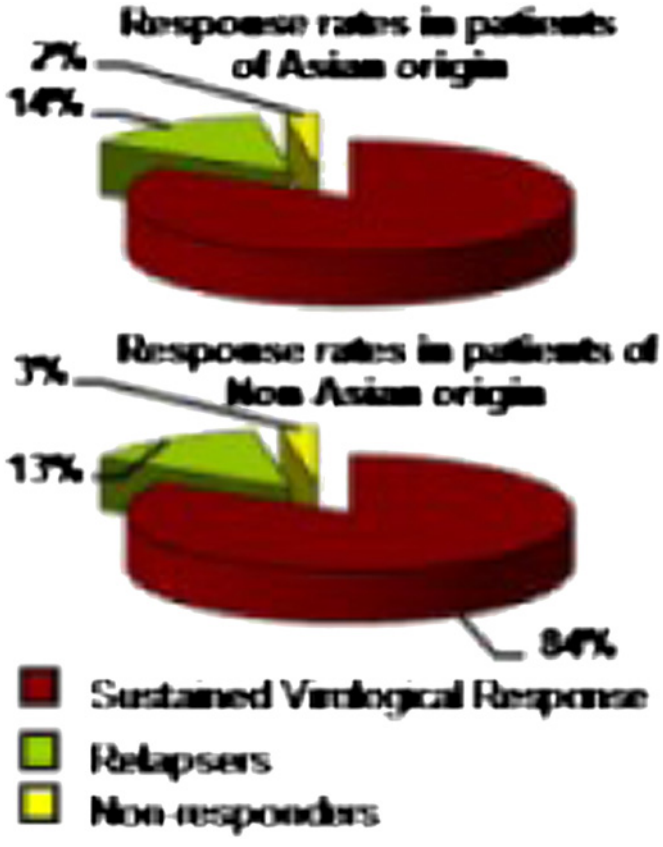
South East Asia, particularly Pakistan, and in Europe represents up to 45% of newly diagnosed infections.[1] Although it is well established that ethnicity impacts on response rates (sustained virological response (SVR) rates) for Hepatitis C virus (HCV) Genotype 1 infections, there is controversy over whether ethnicity impacts on SVR rates in HCV genotype 3 infections.[2 3] In this study we performed a retrospective analysis of patients with genotype 3 HCV infections who had undergone treatment in a busy District General Hospital over the last 4 years to assess whether ethnicity impacted on SVR rates.

Methods All patients treated with standard therapy (pegylated interferon and ribavirin) over the last 4 years were studied and factors which are known to influence response rates together with ethnicity were examined in patients with genotype 3 chronic hepatitis C infection.

Results In total 630 patients were undergoing/had undergone standard treatment for chronic hepatitis C infection in our hospital over the last 4 years. Of these 147 were identified as being HCV genotype 3 infections. 80 of these patients had completed a course of treatment and had at least 6 months of follow-up (for assessment of SVR rates). These patients were divided into those from South East Asia (43 patients) and European patients (37 patients). Of the patients of Asian ethnicity 36 achieved an SVR (83.7%), six relapsed (13.9%) and one was a non-responder (2.4%). Of the patients of European ethnicity 31 achieved an SVR (83.8%), five relapsed (13.5%) and one was a non responder (2.7%). There was no significant statistical difference in SVR rates in patients from South East Asia (83.7% SVR) compared with patients of European origin (83.8%) ($p<0.01$).

Conclusion In this retrospective cohort study SVR rates in patients with genotype 3 chronic hepatitis C infection were not affected by ethnicity. Those patients who did not achieve SVR and failed standard therapy were predominantly relapsers as opposed to non-responders.



Abstract PMO-145 Figure 1 Treatment response rates in patients with genotype 3 chronic hepatitis C infection of Asian and Non Asian Origin.

Competing interests None declared.

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PMO-146 THE SOLUBLE ANTAGONISTIC FORM OF IFN-INDUCIBLE-PROTEIN-10 (IP-10) IS A PREDICTOR OF CLINICAL OUTCOME DURING ACUTE HEPATITIS C INFECTION

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Introduction Several immunological parameters have been associated with clinical outcome of HCV infection. We have previously found that acute HCV-infected subjects progressing to chronicity had higher serum IP-10 levels than those who spontaneously resolved the infection, which is counterintuitive given the known pro-inflammatory role of this chemokine. IP-10 is subjected to physiological cleavage of two aminoacids by DPPIV (CD26), which produces an NH_2-truncated form of the protein. The cleaved form (referred to as short IP-10) antagonises the biologically active longer form by competitively binding to the common receptor without inducing signalling. Most IP-10 assays quantify the total IP-10 protein; however, recently developed assays also allow the distinction of the two forms. The aim of this study was to investigate the role of antagonistic short IP-10 in influencing the clinical outcome of acute HCV infection.

Methods We analysed 16 patients with acute HCV infection who met the following criteria: ALT>10× upper limit normal (ULN), exposure to HCV within previous 4 months and HCV-RNA(+). Plasma was collected from these patients longitudinally at 12 timepoints. We quantified the long and short forms of IP-10 in these samples. Results were then analysed and correlated with the clinical outcome (chronic vs resolver). The longitudinal nature of the dataset was taken into account through the use of a "mixed model" analytical strategy for repeated measures.

Results Our investigation confirmed that subjects who progressed to chronicity (n=11) had higher serum concentrations of total IP-10 ($p<0.001$) compared to patients who resolved spontaneously (n=5). However, these higher amounts of detectable IP-10 were ascribable to increased levels of the antagonistic form ($p=0.036$), while serum concentrations of biologically active long IP-10 form were comparable with subjects who spontaneously resolved the infection ($p=0.460$).

Conclusion This study reports a novel mechanism of chemokine antagonism during acute HCV infection and may represent an additional factor in the failure of the host immune response in eradicating HCV infection. The results also reveal that the short IP-10 is a predictor of clinical outcome after acute HCV infection and may be used to aid treatment decisions during acute HCV infection.

Competing interests None declared.