

**Conclusion** Escalating PIFN2a monotherapy is associated with HCV clearance and a reduction in liver related mortality in this small RCT. The differences from HALT-C and EPIC, and similarity to COPILOT may relate to marked differences in methodology (specifically the omission of therapy in the control arm), cirrhosis stage or sample size. These findings warrant further investigation of PIFN2a for patients with advanced cirrhosis for whom there is no other treatment and where transplantation is associated with graft infection and rapid progression to cirrhosis.

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Abstract P68 Table 1 Results

	SVR n (%)	Liver-related morbidity	Liver-related mortality	All-cause mortality
Intervention n=17	3 (18)	2 (12)	0	0
Control n=22	0	6 (26)	5 (23)	6 (27)
p Value	>0.001	0.23	0.035	0.019

**P69 HEPATITIS DELTA RNA LEVEL AND GENOTYPE, AND HEPATITIS B SURFACE ANTIGEN TITRE PREDICT RESPONSE TO PEG-INTERFERON IN THE TREATMENT OF CHRONIC HEPATITIS DELTA VIRUS**

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**Introduction** Hepatitis Delta Virus (HDV) infection is associated with more severe liver disease in individuals with hepatitis B virus (HBV). The only treatment for HDV shown to be of benefit is  $\alpha$ -interferon, but predictors of response to therapy are not known.

**Aim** To evaluate the efficacy of PEG-interferon  $\alpha$  2a (PEG-IFN) therapy and factors predicting viral response in patients with chronic HBV/HDV co-infection.

**Method** Between 2005 and 2010, 14 patients (71% female, median age 32, 57% Black African, 36% Caucasian, 7% Oriental, 79% HBeAg -ve, 100% HDV RNA +ve, 64% HDV genotype 1, 29% HDV genotype 5, 7% HDV genotype 6, 43% cirrhotic) were treated with PEG-IFN 180 mcg/week for a median of 48 weeks. The median follow-up post treatment was 16.5 months. A retrospective analysis was undertaken to assess clinical and virological factors predictive of outcome. HDV RNA was measured by an in-house real-time quantitative PCR assay (range  $6.4 \times 10^2$  to  $6.4 \times 10^7$  copies/ml), and genotyping was performed by comparison of nucleotide sequences of HDV RNA with previously reported sequences of HDV genotypes 1–8. HBV DNA was measured using the Roche COBAS Ampliprep/TaqMan assay. Hepatitis B surface antigen (HBsAg) titres were measured using the Abbott Architect assay.

**Results** In response to PEG-IFN, 64% cleared HDV RNA by end of treatment (EOT), 1 patient with genotype 1 HDV relapsed and 54% remain HDV RNA -ve beyond 24 weeks post-treatment (sustained virological response; SVR); 2 patients subsequently cleared HBsAg. Baseline HDV RNA was significantly higher in non-responders compared to those with SVR ( $2.1 \times 10^6$  vs  $1.3 \times 10^4$  copies/ml,  $p=0.003$ ) and predicted treatment response (AUROC=1.0,  $p=0.003$ ). A HDV RNA of  $>1.96 \times 10^5$  copies/ml predicted treatment failure ( $p=0.001$ , positive predictive value 100% and sensitivity 100%). There was a strong correlation between HDV RNA level and HBsAg titre ( $r=0.82$ ,  $p<0.001$ ). Baseline HBsAg titre was significantly higher in non-responders compared to those with SVR (10 067 vs 5820 IU/ml,  $p=0.007$ ) and also predicted treatment response (AUROC 0.95,

$p=0.007$ ). A cut-off HBsAg titre of  $>9000$  IU/ml predicted treatment failure ( $p=0.021$ , positive predictive value 100% and sensitivity of 67%). All patients with HDV genotype non-1 achieved SVR, compared with only 25% of those with HDV genotype 1 ( $p=0.02$ ). Responders were similar to non-responders with respect to gender, age, liver histology staging and pre-treatment ALT.

**Conclusion** PEG-IFN was an effective treatment for chronic HDV, with 54% achieving SVR. Levels of HDV RNA  $>1.96 \times 10^5$  copies/mL and HBsAg titres  $>9000$  IU/ml predicted treatment failure, whereas HDV genotype non-1 predicted long-term viral clearance.

**P70 CAN ANTIVIRAL THERAPY FOR HEPATITIS C REDUCE THE PREVALENCE OF HCV AMONG INJECTING DRUG USER POPULATIONS? A MODELLING ANALYSIS OF ITS PREVENTION UTILITY**

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**Introduction** HCV antiviral treatment (peginterferon and ribavirin) is effective for individual patients, but few active injecting drug users (IDUs) are treated.

**Aim** We considered the utility of antiviral treatment for reducing HCV transmission amongst active IDUs.

**Method** An HCV transmission model amongst IDUs was developed, incorporating HCV antiviral treatment. We projected the chronic prevalence reductions resulting from different treatment rates over 5–40 years. Treatment efficacy was varied for three genotype scenarios (mixed, genotype 1, and genotype 2/3) and assumed to result in IDUs becoming susceptible (75%) or resistant/immune (25%). Two models were considered with treatment non-responders either allowed (unrestricted model) or not allowed (restricted model) to be retreated with the same success rates.

**Results** In the unrestricted model with mixed genotype, annually treating 10 infections per 1000 IDUs results in a relative decrease in HCV prevalence over 10 years of 31%, 14% or 7% for baseline (untreated endemic chronic infection) prevalences of 20%, 40% or 60%, respectively. Prevalence reductions are lower (by ~25% at this treatment level) for populations with all genotype 1 and similarly higher for genotype 2/3 populations. Reduction of prevalence to negligible levels within 20 years could be achieved by treating 21 infections per 1000 IDUs annually with 20% baseline prevalence, increasing to 53 or 99 in the 40% and 60% prevalence situation. Restricting retreatment does not alter the short-term (<5 year) projections with low treatment (<20 per 1000 IDUs annually), but reducing prevalence to negligible levels takes longer and becomes impossible at high prevalences (>55%). Lastly, the HCV free life years gained from treating active IDUs are projected to be higher than from treating non-IDUs for prevalences below 60%.

**Conclusion** Despite the possibility of re-infection, modest rates of HCV treatment amongst active IDUs could effectively reduce transmission. Evaluating and extending strategies to treat HCV among active injectors is warranted.

**P71 ANTIVIRAL TREATMENT OF RECURRENT HEPATITIS C AFTER LIVER TRANSPLANTATION—THE NEWCASTLE EXPERIENCE**

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**Introduction** Recurrence of hepatitis C (HCV) post liver transplantation is universal and may follow a rapidly progressive course,