glutamine levels (r=0.78, p=0.002). *Myo*-inositol concentration decreased significantly by 0.7 (±0.7) mMol/l between scans and this correlated with the mean difference in ADC (r=0.59, p<0.04).

Conclusion These results show that hyperammonia can be derived from nitrogenous substrates in the colon and can *directly* drive changes in brain water distribution as a mechanism for cerebral oedema development. Since cerebral astrocytes contain glutamine synthetase, our MRS data suggests intracerebral formation of glutamine from ammonia. Developments in therapy for hepatic encephalopathy need to focus on amelioration of colonic ammonia formation.

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P12 MEASURING THE EFFECTIVENESS OF A MULTIDISCIPLINARY NASH CLINIC

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Introduction Non-alcoholic fatty liver disease (NAFLD) and specifically non-alcoholic steatohepatitis (NASH) are associated with both increased liver-related and cardiovascular morbidity and mortality. A multi-disciplinary, individualised approach targeting the complex pathogenesis of the disease has been employed in a tertiary/secondary care setting. The aims of this study were (1) to investigate the effectiveness of a multi-disciplinary NASH clinic by assessing the change in liver disease markers and risk factors for liver-related and cardiovascular mortality over time, and (2) to identify factors that influence patient response to treatment.

Method This was a retrospective survey of current clinical practice. NASH/NAFLD was defined histologically or NAFLD by echogenic liver on ultrasound with raised aminotransferase values in absence of a significant alcohol history or other hepatic co-morbidities. Interventions included: lifestyle advice; dietetic input; exercise classes; pharmacological therapy; bariatric surgery. Clinical and anthropometric data were collected including serum ALT, BMI, HBA1c, systolic blood pressure, total and HDL cholesterol values and analysed for the cohort overall and for patients who were obese, diabetic, hypertensive and dyslipidaemic respectively. Responders to treatment were defined as those with >10% decrease in ALT over the study period. Univariate and multivariate analysis were conducted to analyse baseline factors influencing patient response.

Results 145 patients were included with median follow-up of 12.5 months (range 3–44 months). Overall improvement was seen in ALT (−15%, $p=7\times10^{-6}$), BMI (−1.5%, $p=6\times10^{-6}$) and total cholesterol (−4.1%, p=0.006). BMI improved by >10% in 8%, by >7% in 16% and by >5% in 23% of patients. Patients categorised by abnormal baseline ALT, baseline obesity, baseline hypertension and baseline dyslipidaemia had improvements in ALT (−19%, $p=1\times10^{-7}$), BMI (−2.4%, p=0.001), systolic BP (−5.4%, $p=3\times10^{-4}$) and total cholesterol (−5.5%, p=0.002) respectively. Patients with type 2 diabetes made up a higher proportion of those who did not respond or who progressed compared to those who responded on univariate analysis (p=0.02), but this was not significant on multivariate analysis. Moreover, patients with diabetes did not have a significant decrease in ALT (−8%, p=0.06).

Conclusion The management framework adopted by the multidisciplinary NASH clinic is effective at reducing ALT overall. Cardiovascular risk factors were improved overall. Diabetic patients had a poor ALT response. These data support the use of a multidisciplinary NASH clinic, but long-term outcome data are awaited.

P13 THE PERFORMANCE VALIDITY OF BREATH SAMPLE ANALYSIS IN THE DIAGNOSIS OF HEPATIC ENCEPHALOPATHY IN PATIENTS WITH CIRRHOSIS

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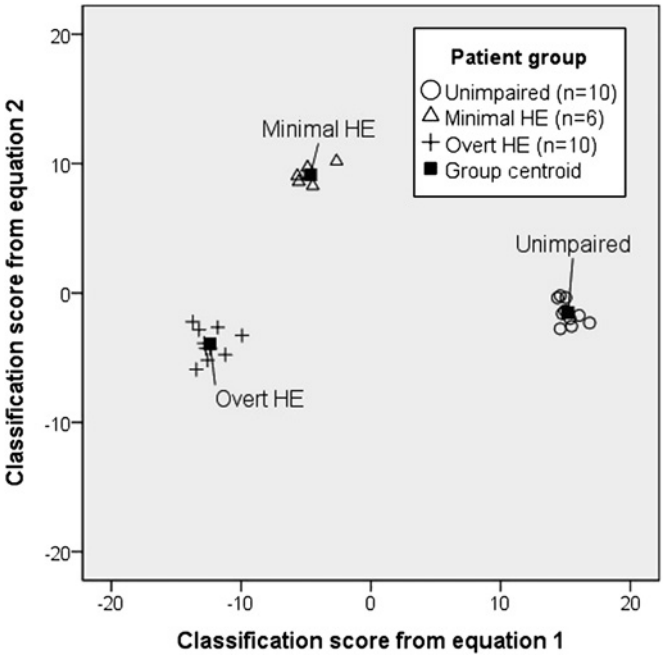
Introduction Hepatic encephalopathy (HE) has a detrimental effect on patients' health-related quality of life and a significant negative impact on survival. Nevertheless, there is no diagnostic gold standard so the condition is often poorly diagnosed and managed.

Aim The aim of this study was to evaluate the performance validity of breath sample analysis for volatile organic compounds (VOCs) in the diagnosis of hepatic encephalopathy (HE) in patients with cirrhosis.

Method The patient population comprised 26 individuals (17 men, 9 women) of mean (range) age 60 (45 to 75) years, with biopsy-proven cirrhosis secondary to alcohol (n=21), or non-alcoholic steatosis, hepatitis C, autoimmune hepatitis, haemochromatosis or cryptogenic



Abstract P13 Figure 1 Classification scores for the model used to predict HE.