expression of CD39 has been associated with T-reg phenotypic stability under inflammatory conditions.

**Aim** To investigate the frequency and phenotypic stability of CD39+ Tregs in AIH.

**Method** 24 AIH patients (23 ANA/SMA+, 1 LKM-1+; 12 females, median age: 15 years) and 24 healthy subjects (16 females, median age: 35 years) were studied. The phenotype of circulating Tregs was assessed by flow cytometry using monoclonal antibodies to CD4, CD25, CD127, CD39 and CD73, an ectonucleoside triphosphate diphosphohydrolase that in mice is expressed by Tregs and works in tandem with CD39. The frequency of IFNγ, IL10 and IL17-producing cells within Tregs was determined by intracellular staining. Analysis was performed at baseline and after exposure to anti-CD3/CD28 T-cell expander or to the pro-inflammatory cytokines IL1β and IL6 (IL1β+IL6).

**Results** At baseline, CD39+ Tregs were less numerous in AIH (8.75±0.77) than HS (11.98±1.04, P=0.019) and displayed a trend towards higher CD127 expression (7.41±5.25 vs 2.75±1.53, P=0.15) and reduced FOXP3 mean fluorescence intensity (1230±260 vs 997±232, P=0.09). CD73 expression on CD39+ Tregs did not differ between the two groups. Exposure to T-cell expander increased the frequency of IFNγ+CD39+ Tregs in AIH (from 7.37±2.15 to 24.3±13.57, P=0.043) but not in HS (12.4±2.9 to 6.6±2.4, P=NS). Although the frequency of IFNγ+CD39+ Tregs augmented after treatment with IL1β+IL6 in both AIH (from 7.57±2.15 to 72.6±9.42, P<0.001) and HS (12.39±2.94 to 56.51±16.12, P<0.001), the increase was higher in the former than in the latter (10-fold vs fivefold). IL1β+IL6 increased the frequency of CD127+CD39+ Tregs in AIH (from 4.98±2.25 to 19.6±11.43, P=0.05) but not in HS (2.7±1.3 to 1.3±0.8 P=NS). No change in the frequency of IL10+ and IL17+CD39+ Tregs was noted upon T-cell expander or IL1β+IL6 stimulation in AIH and HS.

**Conclusion** Compared to HS, Tregs from AIH patients display lower CD39 expression and are more prone to become activated upon exposure to pro-inflammatory stimuli, a finding which indicates reduced phenotype stability. A decrease in CD39 expression and in phenotypic stability may contribute to impaired Treg suppressive function in AIH.

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**BASL abstracts**

**P102 CLEVER-1 MEDIATES THE TRANSMIGRATION OF B CELLS ACROSS HUMAN HEPATIC SINUOSIDAL ENDOTHELIUM**

doi:10.1136/gutjnl-2011-300857a.102

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**Introduction** Lymphocytes are recruited via the unique hepatic sinusoidal channels during chronic inflammatory liver diseases. This low shear vascular bed is lined by hepatic sinusoidal endothelium (HSEC) which lacks certain conventional adhesion molecules leading us to look for novel receptors involved in lymphocyte recruitment. HSEC express several scavenger receptors including CLEVER-1 which we have recently shown mediates regulatory T cell recruitment to HSEC. B cells have been implicated in the pathogenesis of liver disease and driving liver fibrosis.

**Aim** B cells must be recruited from the peripheral circulation into liver tissue but the molecular mechanisms that mediate this process are not known. Our aim was to study if CLEVER-1 plays a role in this process.

**Method** We used isolated HSEC in flow adhesion assays to study the functional role of CLEVER-1 in lymphocyte subset recruitment. Immunofluorescent staining and confocal microscopy were used to characterise the transmigration of lymphocytes across HSEC under conditions of flow. Time lapse video recordings and Image J software was used to compare T cell and B cell recruitment via HSEC monolayers under conditions of flow.