Results During infection, control mice experienced a mild self-limiting colitis, with minimal weight loss. Expression of *Adamdec1* was upregulated in the colon and this normalised with resolution. *Adamdec1*$^{-/-}$ mice were more susceptible to *C. rodentium* infection: they demonstrated dramatic weight loss ($p < 0.001$), a more severe colitis and a reduced survival at the higher dose (67% vs 0%, $p = 0.009$). Serum levels of TNF, IL-1β and IL-6 were significantly lower in the knock-out mice ($p < 0.05$). Impaired survival was associated with positive cultures of the organisms from the spleen ($p = 0.02$).

Conclusion By analysing the transcriptome of macrophages from CD patients we have identified a novel molecule involved in mucosal immunity. Further work is underway to elucidate the precise role of ADAMDEC1 in the immune response. Individuals with grossly attenuated expression levels may be at an increased risk of developing CD as a consequence of an impaired ability to handle enteric bacterial pathogens.

Disclosure of Interest None Declared

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Gastroduodenal free papers

OC-020 THE MURINE GASTRIC MICROBIOME IS INFLUENCED BOTH BY HELICOBACTER FELIS INFECTION AND SOMATIC DELETION OF NF-KAPPAB FAMILY MEMBERS

doi:10.1136/gutjnl-2013-304907.020

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Introduction The development of gastric atrophy in C57BL/6 mice infected with *Helicobacter felis* is differentially regulated by signalling involving NF-κB1 and NF-κB2. After infection, more severe atrophy developed in *Nfkb1*$^{-/-}$ mice than wild-type (WT), whilst *Nfkb2*$^{-/-}$ mice were protected from atrophic gastritis. Previous studies have also shown that the development of *H. pylori* induced gastric pathology was delayed in INS-Gas mice maintained in germ free, compared to conventional animal house conditions. Consequently we hypothesised that the different phenotypes observed in *H. felis* infected mice lacking specific NF-κB proteins may be influenced by altered gastric microbiomes. We have therefore quantified the abundance of specific bacterial phyla in *Nfkb1*$^{-/-}$, *Nfkb2*$^{-/-}$ and C57BL/6 mice with and without *H. felis* infection.

Methods Groups of 3 C57BL/6, *Nfkb1*$^{-/-}$ and *Nfkb2*$^{-/-}$ mice aged 6 weeks were infected with *H. felis* by gavage. Animals were euthanased at 12 weeks and gastric antral DNA was extracted. Total bacterial load and relative abundance of α-*Proteobacteria*, γ-*Proteobacteria*, *Bacteriodetes*, *Firmicutes* and *Actinobacteria* were determined by qPCR of 16S rDNA. *H. felis* colonisation was quantified by qPCR for *Flaa,* samples were normalised to murine *Gapdh*.

Results Untreated WT mice had 3.4 and 2.6 fold greater universal bacterial transcripts than *Nfkb1*$^{-/-}$ and *Nfkb2*$^{-/-}$ mice respectively. *Actinobacteria* abundance was 6.0 fold greater in untreated *Nfkb1*$^{-/-}$ mice *cf* WT and 7.0 times greater in *Nfkb2*$^{-/-}$ mice. α-*Proteobacteria* were 9.0 times more abundant in untreated *Nfkb1*$^{-/-}$ mice *cf* WT. *H. felis* infection of WT mice resulted in 5.6 and 16.7 fold increases in α-*Proteobacteria* and γ-*Proteobacteria* respectively *cf* uninfected mice. γ-*Proteobacteria* were more abundant in all infected groups, but significantly more so in *Nfkb2*$^{-/-}$ mice than others. This correlated with a 30 fold higher abundance of *H. felis* (Phylum: γ-*Proteobacteria*) in infected *Nfkb2*$^{-/-}$ compared to WT mice. Infected *Nfkb2*$^{-/-}$ mice also had a 3.3 fold greater abundance of *Actinobacteria cf* infected WT mice. No statistically significant differences were observed in the abundance of *Firmicutes* or *Bacteroidetes*.

Conclusion Constitution of the murine gastric antral microbiome is influenced both by *Helicobacter felis* infection and somatic deletion of NF-κB family members. Since deletion of NF-κB1 and NF-κB2 have been reported to alter susceptibility to *H. felis* induced gastric atrophy, these data support the hypothesis that specific differences in microbiota may be responsible for, or reflect this altered susceptibility. Further studies are needed to determine whether specific organisms influence the development of gastric pathology either individually or within complex communities.

Disclosure of Interest None Declared

OC-021 A RETROSPECTIVE AUDIT OF PNEUMOCOCCAL & INFLUENZA VACCINATION IN COELIAC DISEASE

doi:10.1136/gutjnl-2013-304907.021

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Introduction Patients with coeliac disease (CD) are at increased risk of mortality from sepsis. In particular, the risk of sepsis with encapsulated organisms such as Pneumococci is higher[1]. This may be a consequence of hyposplenism. Current Department of Health Green Book guidelines as well as Coeliac UK recommend vaccination against Pneumococci and influenza. We aimed to investigate whether or not patients with CD received vaccination against Pneumococci and influenza.

Methods CD patients were identified through a register of 1000 ICD-10 classified patients currently under follow up at Royal Liverpool and Broadgreen University Hospitals NHS Trust. A random sample of 250 patients was selected from the register. Electronic case notes were interrogated for demographic details as well as the most recently confirmed comorbidities. Patients of all ages were included in the study. Patients aged over 65 and patients with certain comorbidities (e.g. chronic obstructive airways disease, ischaemic heart disease, liver cirrhosis etc) were analysed separately as these patients should already be immunised against pneumococci and influenza regardless of their coeliac disease diagnosis. Immunisation status was obtained by contacting individual patient's general practitioners. The proportion of vaccinated patients were analysed according to the age groups < or > 65.

Results A total of 250 patients were included out of which we were able to obtain records for 198 patients. Of the 198 patients, 32% of patients had osteopenia and 13% of patients had one or more concurrent autoimmune illness (e.g. thyroid disease, pernicious anaemia, Sjogren's syndrome). 129 patients were < 65 years of age (M: F ratio 1: 2, median age-50, range 19–64) and 69 were > 65 (M: F ratio 1: 2, median age-73, range 65–84).

In the < 65 category, we found 10 with no comorbidities necessitating vaccination which left 119 patients in the main study arm; 23 (19.3%) were vaccinated against Pneumococci and 42 (35%) against influenza.

Abstract OC-021 Table 1 Vaccination uptake in < and > 65 age groups.

	Percentage of patients who had received the vaccination	
Age	Pneumococcal vaccine	Influenza vaccine
< 65	19.3%	35%
> 65	72.5%	82.6%

Conclusion Very few patients with CD are vaccinated against preventable causes of sepsis such as Pneumococci. Gastroenterologists, GPs and patients need to be aware of the increased risk of sepsis and the need to administer vaccines against preventable infections in CD patients. Vaccination status should be routinely obtained during follow up visits. Due to the lack of a simple clinical test for