CCL20 production, with a sixfold increase after 24 h at MOI 1 (p=0.01). The Δ*cagE* (p<0.01) and Δ*slt* (p<0.01) but not Δ*cagA* (p=0.18) inactivations reduced CCL20 induction by multiple *Hp* strains, indicating CagA-independent, *cag*PAI-dependent signalling.

Conclusion *Hp* induces CCL20 production by gastric epithelial cells in a *cag*PAI-dependent manner. Higher gastric CCL20 levels in *Hp*+ patients correspond to increased gastric infiltration of $CCR6^+$ Tregs and to the proportions of these cells in the peripheral blood. We speculate that CCL20/CCR6 is the main homing system for Tregs to the stomach in *Hp* infection and thus central to pathogenesis. Migration assays are being performed in vitro prior to in vivo studies in mouse models.

Competing interests None declared.

PWE-174 PROTON PUMP INHIBITORS (PPI) USE AND *CLOSTRIDIUM DIFFICILE* INFECTION: GUILTY OR INNOCENT BYSTANDER?

doi:10.1136/gutjnl-2012-302514d.174

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Introduction PPIs have been implicated in predisposing to *Clostridium difficile* infection by causing hypochlorhydria. A 5–10% asymptomatic toxin burden in the community is also reported. The previously voluntary reporting of *C difficile* infection in UK became mandatory for all age-groups in April 2007. Aim: To compare the pattern of PPI prescribing & *C difficile* rates in Rotherham (industrial town in Yorkshire; pop 250k) and England (50mill.) over a 7-year period to identify any association at a community level.

Methods Retrospective population study of seven consecutive years (2004/2005–2010/2011) on data of annual PPI prescriptions & *C difficile* incidence (both/100k pop.) in Rotherham primary care trust (PCT) and all England PCTs combined obtained from our microbiology department, Rotherham PCT, NHS Information Centre and UK Health Protection Agency. PPI prescribing trends and *C difficile* rates (irrespective of PPI use) in each year was compared between the two groups as % difference (Rotherham vs England: + =higher %, − =lower %).

Results (Abstract PWE-174 table 1) PPI prescription: Prescription rates have risen steadily in both cohorts over the study period. Rotherham had higher rate throughout study period but gap with England in % terms has steadily narrowed. *C difficile* rates: Rotherham rates were much higher till 2006/2007, reversed dramatically in 2007/2008, continuing to fall for next 2 years. England rates peaked in 2007/2008 and fell steadily from 2008/2009 with hardly any gap in 2010/2011.

Abstract PWE-174 Table 1 PPI prescribing and *C difficile* rates in Rotherham (R) vs all-England (E)

	Rates/year/100 k population						
	2004/2005	2005/2006	2006/2007	2007/2008	2008/2009	2009/2010	2010/2011
R PPI	53 566.0	60 217.0	66 974.0	75 948.0	80 554.0	86 641.0	92 829.0
E PPI	39 715.6	45 501.1	51 505.0	59 543.6	66 800.4	73 876.3	80 377.4
R/E difference (%)	+26	+24	+23	+22	+17	+15	+13
R *C difficile*	120.0	150.4	144.4	88.0	54.0	38.0	40.4
E *C difficile*	80.8	94.0	102.6	111.0	72.2	51.2	43.4
R/E difference (%)	+33	+38	+29	−26	−34	−35	−1

Conclusion Strict hand hygiene in hospital and microbiologist-controlled prudent antibiotic use in hospital and community from 2002/2007 seem to have resulted in a marked fall in *C difficile* rates in Rotherham from 2007/2008. We presume that similar measures, gradually introduced in the rest of England during 2007, account for the more widespread but less steep fall in the England infection rates from 2008/2009.·The 7-year community level data suggests a mere association rather than true cause-effect relation between *C difficile* rates and PPI use in the past. Any potential risk from PPI use seems to be offset by rigorously applied hand hygiene (secondary care) and careful antibiotic prescribing (primary and secondary care) as evidenced by falling infection rates, despite rising PPI prescribing, since 2007/2008.

Competing interests None declared.

PWE-175 DUODENAL EOSINOPHILIA AND EARLY SATIETY IN FUNCTIONAL DYSPEPSIA (FD): CONFIRMATION OF A POSITIVE BIOMARKER ASSOCIATION FOR FD IN AN AUSTRALIAN COHORT

doi:10.1136/gutjnl-2012-302514d.175

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Introduction Functional dyspepsia (FD), defined by unexplained pain or discomfort centred in the upper abdomen, affects 15% of the population. Diagnosis and treatment of FD based on the symptom-based Rome III criteria remains challenging. Recently, subtle eosinophilic inflammation in the duodenum has been implicated in the pathophysiology of FD in adults based on a Swedish case-control study.[1] Specifically, increased eosinophils in early satiety and postprandial distress have been replicated in the UK,[2] in paediatric dyspepsia in the USA[3] and in post-infectious FD in Japan,[4] but the association remains controversial. The aim of this study was to characterise upper gastrointestinal tract pathology in an Australian cohort and correlate this with available clinical data in FD cases and controls.

Methods Patients prospectively referred for an upper gastrointestinal endoscopy (n=55; mean age, 49.6 years; 61.8% female) were entered to the study (with informed consent), stratified to FD cases (n=33) and controls (n=22) by Rome II criteria and completed a validated Bowel Symptom Questionnaire. Two blinded independent observers assessed the eosinophil count in five high power fields, in the duodenal bulb (D1) and second part (D2). *H pylori* status was assessed by gastric histology. Associations with clinical symptoms were assessed by Mann–Whitney U test.

Results Cases and controls were demographically similar. There was a significant increase in eosinophils in D1 vs D2 for both cases (p=0.0003) and controls (p=0.008). Abdominal pain was associated with eosinophilia in both D1 (p=0.03) and D2 (p=0.007). Duodenal eosinophilia was significantly increased in subjects experiencing early satiety, (p=0.015). This association remained after exclusion of coeliac disease (n=2) and *H pylori* (n=9, 16%) (p=0.04) in D2 but not in D1. Subjects who "felt food staying in their stomach" similarly had increased D2 eosinophilia (p=0.002); this remained significant on exclusion of coeliac disease and *H pylori* (p=0.004). Smoking was also associated with eosinophilia in D2 (p=0.008).

Conclusion Data supporting subtle duodenal eosinophilia in subsets of FD has become credible. The potential role of duodenal eosinophils as biomarkers has implications for diagnosis and therapeutic trials.

Competing interests None declared.