and number of preceding reflux events 1 hour before an SRE was similar among the groups. The number of preceding acid reflux events 2, 3, and 4 hours prior to an SRE was significantly lower in the FH group compared to NERD and EE, 2 hours (5.57 ± 4.1 vs 15.64 ± 9.2, 5.57 ± 4.1 vs 14.42 ± 9.8 respectively, P < 0.05), 3 hours (8.80 ± 4 vs 25.60 ± 12.5, 8.80 ± 4 vs 21.19 ± 12.4 respectively, P < 0.01), and 4 hours (11.60 ± 4.4 vs 31.50 ± 15.9, 11.60 ± 4.4 vs 26.64 ± 15.7 respectively, P < 0.01).

Conclusion: Patients with FH demonstrated the highest proximal extent of SREs but a higher pH nadir and fewer reflux episodes prior to an SRE compared to NERD and EE. The study suggests that proximal extent of an acid reflux event is the most important physiological factor for symptom generation

Is There a Difference in the Prevalence of Helicobacter pylori Infection between Short-Segment and Long-Segment Barrett’s Esophagus?
Changcheng Wang, MD, Yuhong Yuan, PhD, Richard H. Hunt, MD, FRCP, FRCP(C). Division of Gastroenterology, McMaster University Health Science Centre, Hamilton, ON, Canada.

Purpose: Recent studies suggest that the length of Barrett’s esophagus (BE) is a risk factor for neoplastic progression. Patients with long-segment Barrett’s esophagus (LSBE) are at higher risk for development of dysplasia and adenocarcinoma of the esophagus when compared with short-segment Barrett’s esophagus (SSBE), which suggests that different pathophysiological mechanisms may be involved in SSBE and LSBE. To date, the role of H. pylori infection in the development of BE is still unclear. We aimed to investigate by meta-analysis if any difference exists in the prevalence of H. pylori infection between SSBE and LSBE.

Methods: Observational studies comparing the prevalence of H. pylori infection or cagA+ H. pylori strains in patients with SSBE (length <3 cm) and LSBE (length ≥ 3 cm) conducted in adult populations and published in all languages were identified through MEDLINE, EMBASE and Cochrane database searches until week 21, 2007. H. pylori infection required confirmation by histology and/or serology and/or RUT and/or culture. Primary outcome was the prevalence of H. pylori infection in SSBE and LSBE. Secondary outcome was the prevalence of cagA+ H. pylori strains in SSBE and LSBE. Studies were excluded if lacking raw data for outcome of interest or were duplicate publications. Summary effect size was calculated as odds ratios (OR) and 95% confidence intervals (CI) by the random-effects model using Review Manager 4.2.8.

Results: Of 458 citations, 13 studies met inclusion criteria. 825 patients with SSBE and 470 with LSBE were included for analysis. The prevalence of H. pylori infection was significantly higher in SSBE than in LSBE [39.6% (327/825) and 29.6% (139/470) respectively, OR = 1.49, 95% CI 1.02–2.18, P = 0.04], and homogeneity was seen between studies (P = 0.14). The results from 4 studies in H. pylori positive patients showed no difference in cagA+ H. pylori strains between SSBE and LSBE [36.7% (18/49) and 37.8% (14/37) respectively, OR = 0.51, 95% CI 0.68–3.04, P = 0.46], with homogeneity (P = 0.14).

Conclusion: The prevalence of H. pylori infection in LSBE is significantly lower than in SSBE, which suggests that H. pylori infection plays a different role, if any, in these two subtypes of BE and does not “protect” in patients with SSBE. Current evidence does not confirm a role for cagA+ in subtypes of BE although the sample size was small. Large prospective studies are needed to confirm these results in the future.

Omeprazole Induces a Transepithelial Leak in Gastric Mucosa
James M. Mullin, PhD*, Mary C. Valenzano, Marysue Whitty, Deborah Lurie, PhD, Vishal Jain, MD, Owen Tully, MD, Paul Allegretti, DO, Daniel Lazovich, DO, J. David Schmidt, MD, James J. Thornton, MD, Giancarlo Mercogliano, MD. Lankenau Institute for Medical Research, Wynnewood, PA; Mathematics, St. Joseph’s University, Philadelphia, PA; Medicine, Lankenau Hospital, Wynnewood, PA and Gastroenterology, Lankenau Hospital, Wynnewood, PA.

Purpose: Having observed a paracellular leak to oral sucrose in esophagitis patients, we tested if PPI therapy would decrease (paracellular) sucrose leak by allowing for mucosal healing.

Methods: 23 study subjects presenting to their primary care physician with symptoms of GERD were placed on an 8 week course of omeprazole (40 mg daily). Subjects were PPI and H-2 blocker naïve. All subjects reported symptom alleviation by 4 weeks. Before beginning esomeprazole therapy and at the end of therapy, subjects consumed at bedtime a solution of the disaccharide, sucrose (100 gms in 200 cc tap water), and collected an overnight urine specimen. Sucrose, as a disaccharide, can enter the blood-stream only by leaking paracellularly out of the upper GI lumen. Once in the blood, sucrose is filtered quantitatively into urine. Sucrose concentration in urine was measured by enzymatic/spectrophotometric means. The sucrose concentration × total urine volume yields the total amount of sucrose in the urine, which equals the the amount of sucrose which leaked paracellularly from the upper GI lumen.

Results: We observed that 17 of 23 patients exhibited a marked increase in sucrose leak by 8 weeks of omeprazole therapy (667% increase). Similar sucrose leak is observed after only 8 days of esomeprazole therapy. Considering all 23 patients, the Wilcoxon Signed Rank Test indicated a significant increase in sucrose leak (P = 0.007). In rat gastric mucosa, 200 μM omeprazole induces an immediate paracellular leak to D-mannitol, with a simultaneous decrease in transepithelial electrical resistance. Both phenomena indicate that omeprazole is likely producing a tight junction leak in gastric mucosa.

Conclusion: PPI therapy may be associated with a paracellular leak in the acid secreting region of the stomach. Future studies will examine the characteristics of this leak and the mechanism by which omeprazole induces the leak.

High Prevalence of Eosinophilic Esophagitis in Inflammatory Bowel Disease
Souheil Gebara, MD*, Robert M. Trudinger, MD, Saleha Khanum, MD, Sandra L. Hodges, RN, Chung-Ho Chang, MD, Neal S. Goldstein, MD. Pediatrics, William Beaumont Hospital, Royal Oak, MI and Anatomic Pathology, William Beaumont Hospital, Royal Oak, MI.

Purpose: Eosinophilic esophagitis has been reported in association with asthma and celiac disease but not with inflammatory bowel disease (IBD). We noted in our practice a frequent association between IBD (Crohn’s disease or ulcerative colitis) and eosinophilic gastrointestinal diseases (EG) especially eosinophilic esophagitis (EE).

Methods: Retrospective chart review of our outpatient pediatric gastroenterology practice. We looked at the pathology reports of our active patients with IBD and selected the ones with EE. Two pathologists confirmed the histological diagnosis of both IBD and EE. We reviewed the indications for the biopsies, age at diagnosis, gender, type of IBD, sequence of the diagnoses and medication history.

Results: We have 3800 active patients, out of these 357 had IBD and 151 had EE. We found 18 IBD patients who also had EG including 14 with EE. Thus the prevalence of EG in our IBD population is 5.0% and EE 3.9%. All EG patients were diagnosed simultaneously or after the diagnosis of the IBD. There was no relationship between EE and age, gender, type of IBD and medication history.

Conclusion: EE has been reported to have a prevalence of 1–4 per 10,000 children in the USA and thus the relative risk of EE in our IBD population ranges from 97 to 390 compared with the general population. In pediatric patients with IBD, we found a high prevalence of EE. A larger study should be helpful in confirming this observation and may improve our understanding of the pathophysiology of both diseases.