Background
Gastroparesis is a condition characterized by gastric retention in the absence of physical obstruction. As part of an ongoing project aimed at determining the pathogenesis of reactive gastropathy, we hypothesized that patients with gastroparesis, in whom potentially noxious substances (e.g., non-steroidal anti-inflammatory drugs, NSAID) have a prolonged contact with the gastric mucosa, would have a greater prevalence of chemical gastropathy than subjects with normal gastric motility. To our knowledge the prevalence of H pylori in these patients has only been addressed in three studies, which concluded that no apparent relationship between H pylori gastritis and delayed gastric emptying existed. To provide a broader picture of the changes occurring in the gastric mucosa in patients with gastroparesis we designed a study that, in addition to testing our hypothesis regarding the prevalence of reactive changes, would also provide insights into the most common mucosal abnormalities diagnosed in gastric biopsy specimens from these patients.

Methods
This study was conducted at Miraca Life Sciences. We analyzed electronic data from the Miraca database, which includes demographic and clinical information for each patient, a summary of the endoscopic findings or the entire endoscopic report, the site of origin of each specimen, and the histopathology report for each biopsy. To identify the records for eligible patients, we extracted data for subjects who had esophagogastroduodenoscopy (EGD) with at least one gastric biopsy submitted to Miraca Life Sciences between January 1, 2008 and June 30, 2012. Patients with a history or diagnosis of upper gastrointestinal cancer or surgery and those with chronic conditions known to affect gastric motility (e.g., scleroderma) were excluded.

Results
There were 641,705 unique patients with gastric biopsies. Of these, there remained 3,940 unique patients with a reasonably certain diagnosis of gastroparesis and 575,895 unique control patients with neither evidence nor suspicion of gastroparesis. Reactive gastropathy, which was at the core of our study hypothesis, was significantly, but only slightly so, more prevalent in patients with gastroparesis (5.76%, or 18.9%) than in controls (97,857 patients, or 17.0%), with OR of 1.14 (95% CI: 1.04 – 1.25; p<0.05). In contrast, H pylori gastritis was present in 62,110 (10.8%) but only in 180 (3.9%) patients with gastroparesis (OR 0.32, 95% CI: 0.045 – 0.81; p<0.001). Intestinal metaplasia was also less common in patients with gastroparesis than in controls (2.8% versus 3.9%; OR 0.82; 95% CI: 0.58 – 0.89; p<0.001). Chronic inactive gastritis and H pylori-negative active gastritis were equally prevalent within the two groups.

Conclusions
One possibility is that stasis makes the gastric environment unfavorable to the colonization by H pylori. Such changes could involve the balance between acid and alkali content or the bacterial ecology, with H pylori being able to colonize the stomach and compete with H pylori.

A final and highly speculative conjecture is that H pylori infection, which is usually acquired during early childhood, could be protective against the development of gastroparesis, which develops later in life.

References