Campylobacter pylori infection is now recognized as a major factor in gastroduodenal pathology. Studies in developed countries have shown that asymptomatic *C pylori* infection is frequent and that the prevalence increases with age. Few data are presently available in developing countries. The aim of the study was to determine the prevalence of the infection in 3 countries on 2 continents by a population-based study using serology. One thousand sera were collected. In Vietman and Algeria, adult sera were collected in blood banks; sera from children were collected at outpatient clinics. In the Ivory Coast, sera from adults and children were collected by cluster sampling. Sera from a French health center were also included as a control.

An ELISA with a sonicata as antigen was used. It was standardized with sera from patients known to be *C pylori* positive or negative resulting in a specificity of 96% and a sensitivity of 84%.

In all 3 developing countries, an increased prevalence with age was noted. The major difference with data from developed countries was the very high prevalence of the infection; 75% or higher from the third decade on. Children were contaminated early in life (= 50% by age 10) except in Vietnam, in contrast to that seen in developed countries. No difference was noted between men and women in any country. The place of residence (urban or rural) studied in Vietnam was not associated with *C pylori* infection.

We conclude that, with the given prevalence, *C pylori* is probably a mild pathogen. However, it could very well contribute to gastric atrophy already observed in a large proportion of the population by age 30 and to gastric discomfort which is common in these countries.

***Campylobacter pylori* in Chronic Duodenal Ulcer and the Zollinger-Ellison Syndrome.** A. Fich, N. J. Talley, R. G. Shorter and S. F. Phillips, Mayo Clinic, Rochester, Minnesota.

Since *Campylobacter pylori* is present in nearly all patients with chronic duodenal ulcer (CDU), it has been hypothesized that *C pylori* plays a pathogenetic role. We postulated that, if *C pylori* is pathogenic in the usual form of CDU, it would be infrequent in patients with duodenal ulcer of specific etiology, such as the Zollinger-Ellison syndrome (ZES).

Gastric tissue was available from 12 patients with ZES and 18 patients with CDU, all of whom had undergone elective gastric resections. Antral tissues were cut (5 μm thickness) and stained by hematoxylin-eosin and Giemsa. All slides were reviewed by an independent experienced pathologist who was unaware of the diagnosis. Chronic and active chronic gastritis was graded from 0 (no inflammatory cells) to 4 (very dense cellular infiltrate). *C pylori* were recognized morphologically by the presence of typically curved, rod-shaped organisms.

The prevalence of antral *C pylori* in patients with ZES was significantly lower than that in patients with CDU ($p < 0.05$). Moreover, chronic antral gastritis scores were significantly higher in patients with CDU compared to ZES patients ($p < 0.05$).

	Z-E Syndrome n = 12	Chronic Duodenal Ulcer n = 18
Age (mean + SEM)	43.8 ± 3.4	48.6 ± 3.4
Sex M/F	6/6	9/9
Prevalence of *C. pylori*	4/12	16/18
Gastric score	1.5 ± 0.2	2.4 ± 0.2

We confirm that CDU is strongly associated with the presence of *C pylori* in antral mucosa. In contrast, the prevalence of *C pylori* in duodenal ulcer secondary to Zollinger-Ellison syndrome was markedly less and was, in fact, similar to the prevalence reported in healthy volunteers. These data support the hypothesis of a role for *C pylori* in the induction of CDU.

Can *C pylori* Be Eradicated? Long-Term Followup on 28 Successfully Treated Patients. B. J. Marshall, S. H. Caldwell, S. R. Hoffman, H. F. Frierson, R. L. Guerrant and R. W. McCallum, University of Virginia at Charlottesville.

C pylori does not usually respond to single agent antibiotic therapy and many investigators have reported recurrence after apparent clearance of the bacterium. The purpose of this study was to identify criteria which could be used to define bacteriologic cure.

114 patients with biopsy proven *C pylori* were treated with at least 14 days of bismuth subsalicylate concurrently with an antibiotic, either metronidazole 1-1.5 g/day, amoxicillin 2 g/day or erythromycin 2 g/day. Endoscopy with multiple gastric biopsies for histology, culture and CLO test and/or 14C urea breath test were performed 28 days after therapy. All patients were then asked to return for repeat evaluations at 3, 6, and 9 months after initial clearance.

At assessment 28 days after therapy there were 58 patients cleared of *C pylori*. In 28 patients, at least one subsequent study has been performed at times ranging from 1 to 10 months after initial clearance. Specifically, 22 patients have been evaluated for at least 3 months, 9 for 6 months, and 5 for 9 months, representing a total of 125 patient months of follow-up. No patient has been found to be reinfected.

We conclude: 1) Eradication of *C pylori* is achievable. 2) Endoscopic and/or breath test evidence of *C pylori* clearance four weeks after therapy is predictive of long-term cure. 3) Reinfection with *C. pylori* is apparently uncommon in the United States.

***Campylobacter pylori* Gastritis After Ulcer Surgery.** S. H. Caldwell, B. J. Marshall, M. W. Plankey, H. F. Frierson, S. R. Hoffman, R. W. McCallum, University of Virginia at Charlottesville.

Campylobacter pylori (CP) infection is present in over 90% of duodenal ulcer (DU) patients (pts) and

over 70% of gastric ulcer (GU) pts. It is suspected that infection plays a permissive role in peptic ulceration. Acid-reducing surgery is effective therapy for peptic ulcer and post-operative relapse rates of 0.8–20%. Little is known about the course and significance of the infection after surgery. Our goal was to study this process in pts who have had acid-reducing surgery at UVa for peptic ulcer since 1980.

21 pts out of 209 identified through chart review met criteria and agreed to participate in the study. Of the original 209, 70 were excluded due to age over 75 yr, 52 due to death, Zollinger-Ellison syndrome or medical problems, 29 could not be reached, 37 were uninterested. Of the 21, 11 had parietal cell vagotomy (PCV), 8 had vagotomy and antrectomy (VA) and 2 had vagotomy and pyloroplasty (VP). All pts underwent C14 urea breath test and serologic testing and 12 (7 PCV, 4 VA, 1 VP) underwent endoscopy and biopsy. 3 pts who were CP+ have been treated and had repeat biopsy. All pts completed symptom and history questionnaires.

13 pts were CP+ (62%). 8 of 13 with antrums in situ were CP + and 4 of 8 with antrectomy were CP +. By original diagnosis, 10 of 13 DU pts were CP+ after surgery, 3 of 6 GU were CP+ after surgery and 1 of 2 with either channel ulcer or DU and GU were CP+. The mean time since surgery for CP+ was 44 mo. and for CP− it was 42 mo. Among CP+ pts, 9 of 13 underwent endoscopic biopsy and all had histologic gastritis. 3 of 8 CP− pts had biopsy and all had normal gastric histology or had evidence of bile effect (foveolar hyperplasia) without inflammation. Repeat biopsy in the 3 CP+ pts treated with antibiotics showed marked histologic improvement. Symptom scores were similar in CP+ and CP− but more CP+ were regularly using medications (61 vs 38%). 4 pts (19%), all PCV, had suspected or proven ulcer relapse and all were CP+.

We conclude: 1) CP is common after ulcer surgery regardless of type of surgery or time since surgery. 2) In this setting, CP is associated with histologic gastritis and eradication of the organism heals the gastritis. 3) Though this is a small study, it suggests that the success of surgery for peptic ulcer may relate not only to the traditionally accepted acid reduction but also to the presence or absence of CP.

***Campylobacter pylori* in Dyspeptic Elderly Patients.** T. O'Riordan, A. Tobin and C. O'Morain, Meath/Adelaide Hospitals, Dublin, Ireland.

The association between *Campylobacter pylori* (CP) and gastritis is well established. As there is evidence that CP infection increases with age we reviewed the clinicopathological records of 119 consecutive patients aged 65 to 85 years (mean 71.1 years) on whom gastroscopy had been performed for dyspeptic symptoms. The male/female ratio was 7:6 and the ratio if inpatient to day case patients was 1:3. All patients had 2 antral biopsies—1 was assessed for histological evidence of gastritis and the other was independently assessed for CP using microscopy gram stain culture and Warthin-Starry stain. 36 patients (30.2%) had duodenal ulceration, of whom 32 had an associated CP positive gastritis. 5 patients (4.2%) had a benign gastric ulcer and all had an associated CP positive gastritis. 49 patients (41.2%) had antral gastritis without ulceration, of whom 38 were CP positive and 11 were CP negative. 91% of CP negative gastritis patients had a history of recent ingestion of NSAIDs compared to 29% of CP positive gastritis patients. 3 patients with gastritis had a history of recent alcohol abuse (2 were CP positive). 10 patients (8.4%) had normal antral mucosa but had evidence of reflux oesophagitis (one of these patients was CP positive). 19 patients (15.9%) had normal antral mucosa and normal endoscopic findings and two of these were CP positive.

We conclude that CP infection is associated with the majority of cases of symptomatic gastritis in the elderly and that a CP positive gastritis is associated with the majority of duodenal ulcers in this age group. The most important cause of CP negative gastritis in this population appears to be NSAID ingestion.

***C pylori*-Related Non-Ulcer Dyspepsia Improves After *C pylori* Eradication.** L. George, R. Taylor, S. Noonan, P. Cole, L. Hyland, A. Morgan, J. Lenne, D. Moore-Jones, S. Brandl and T. Borody, Centre for Digestive Diseases, Sydney University, Sydney, Australia.

Controversy continues as to whether C pylori (CP) can contribute to the etiology of non-ulcer dyspepsia (NUD). Since blinded "triple therapy" results (Borody T et al, Gastroenterology 1988;94:A43) are not yet available, we reviewed symptoms retrospectively in NUD patients at least 6 mons after successful (−ve at 8 weeks rebiopsy) triple therapy. Records identified 62 NUD patients at least 6 months post-triple therapy for CP + ve gastritis. Only those with no endoscopic evidence of oesophageal inflammation or ulceration were included. Symptoms of abdominal pain (AP), nausea (N) and eructation (E) were scored on a linear analog scale.

Changes in symptom scores at 6 months or more post-therapy were seen in pain, nausea and eructation.

	Before Therapy	After Therapy	'p*'
AP −	6.2	2.5	<0.001
N −	2.0	0.8	<0.001
E −	3.4	2.0	0.01

Globally 60/62 patients stated that the positive symptom improvement warranted the 4 week therapy. These results need to be interpreted in light of retrospective methodology used.

We conclude that 1) treatment of CP-related NUD appears to relieve abdominal pain, nausea and eructation and 2) randomised trials are warranted in CP-related NUD.