risk of UC but not CD. Future studies focusing on potential mechanisms, which mediate this association are warranted.

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Incidence and Phenotype of Inflammatory Bowel Disease From 13 Countries in Asia-Pacific: Results From the Asia-Pacific Crohn's and Colitis Epidemiologic Study 2011-2013

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Background: The Asia-Pacific Crohn's and Colitis Epidemiology Study, initiated in 2011, aimed to determine the incidence and phenotype of inflammatory bowel disease (IBD) in Asia-Pacific. We herein present incidence and phenotype data from 2011 through 2013 for 12 countries or areas in Asia (Brunei, China, Hong Kong, India, Indonesia, Macau, Malaysia, Philippines, Singapore, Sri Lanka, Taiwan, Thailand) and Australia. **Methods:** We performed a prospective, population-based study of IBD incidence in predefined catchment areas using a web-based database. New cases were diagnosed based on standard criteria and ascertained from multiple sources. Endoscopy, pathology, and pharmacy records were searched for completeness of case capture. Age-standardized incidence was calculated with 95% confidence interval (CI). Crude incidence in different regions within Asia was pooled together using a random effect model. **Results:** We identified 1,572 new IBD patients (2011-2012, n=419; 2012-2013, n=1,153; 131 from Australia) including 1,057 (67%) ulcerative colitis (UC) and 515 (33%) Crohn's disease (CD). The mean annual incidence for IBD per 100,000 was 1.68 (95% CI, 1.59-1.77) in Asia and 22.28 (95% CI, 18.48-26.71) in Australia. The three countries within Asia with the highest incidence per 100,000 was India (9.31; 95% CI, 8.38-10.31), China (Guangzhou) (3.30; 95% CI, 2.68-4.06) and Hong Kong (2.58; 95% CI, 2.20-3.03). Within five regions of China, IBD incidence varied from 0.49 to 3.30 per 100,000. Pooled incidence of UC and CD within East Asia (China, Macau, Hong Kong, Taiwan) was 1.12 (95% CI, 0.77-1.48) and 0.33 (95% CI, 0.17-0.49), respectively. Pooled incidence of UC and CD within South East Asia (Brunei, Malaysia, Singapore, Indonesia, Thailand, Philippines) was 0.38 (0.21-0.55) and 0.30 (95% CI, 0.22-0.38), respectively **(Table 1).** Ratio of UC:CD was 2.21 in Asia and 0.64 in Australia. Median time from symptom onset to diagnosis was 3 months [interquartile range (IQR), 2-10] for UC and 7 months (IQR 2-19) for CD. Stricturing, penetrating and perianal CD at diagnosis was common (21%, 10% and 17%, respectively) in Asia **(Table 2). Conclusion:** Robust large-scale comparative epidemiologic IBD data from newly industrialized countries are emerging. Incidence of IBD varies throughout Asia with a higher incidence in East than South-East Asia. Complicated CD at diagnosis emains prevalent in Asia. Emergence of IBD in Asia will result in the need for specific health-care resources.

		Mean Annual Age-adjusted Incidence	
	Total	UC	CD
Australia*	22.28 (18.48 - 26.71)	9.32 (6.95 - 12.32)	12.96 (10.08 - 16.48)
India#	9.31 (8.38-10.31)	5.40 (4.70-6.18)	3.91 (3.31-4.57)
China (Guangzhou)	3.30 (2.68 - 4.06)	1.99 (1.53 - 2.59)	1.31 (0.92 - 1.85)
Hong Kong	2.58 (2.20 - 3.03)	1.34 (1.10 - 1.67)	1.23 (0.96 - 1.59)
Macau	2.38 (1.46 - 3.86)	1.32 (0.74 - 2.47)	1.06 (0.42 - 2.35)
China (Daqing)	1.77 (1.16 - 2.59)	1.64 (1.06 - 2.43)	0.13 (0.02 - 0.47)
Taiwan#	1.33 (1.18-1.48)	1.17 (1.03-1.32)	0.16 (0.11-0.22)
China (Xiangshan)	1.26 (0.64 - 2.51)	0.92 (0.42 - 2.09)	0.34 (0.07 - 1.38)
China (Kunming)	1.17 (0.93 - 1.46)	1.10 (0.87 - 1.39)	0.06 (0.02 - 0.17)
China (Chengdu)	0.55 (0.31 - 0.96)	0.42 (0.21 - 0.80)	0.13 (0.04 - 0.44)
China (Xian)	0.49 (0.36 - 0.68)	0.44 (0.32 - 0.62)	0.05 (0.02 - 0.15)
East Asia		**1.12 (0.77 - 1.48)**	**0.33 (0.17 - 0.49)**
Sri Lanka	1.69 (1.32 - 2.13)	1.17 (0.87 - 1.55)	0.52 (0.33 - 0.79)
Singapore	1.01 (0.80 - 1.27)	0.61 (0.45 - 0.82)	0.40 (0.27 - 0.59)
Malaysia	0.82 (0.43 - 1.47)	0.68 (0.32 - 1.31)	0.14 (0.03 - 0.53)
Indonesia	0.74 (0.40 - 1.41)	0.50 (0.22 - 1.10)	0.25 (0.08 - 0.78)
Brunei	0.62 (0.13 - 2.64)	0.21 (0.01 - 2.15)	0.41 (0.05 - 2.38)
Thailand (Bangkok)	0.55 (0.41 - 0.75)	0.25 (0.16 - 0.39)	0.31 (0.20 - 0.47)
Thailand (Chiangmai)	0.48 (0.29 - 0.80)	0.23 (0.11 - 0.49)	0.25 (0.11 - 0.52)
Philippines	0.31 (0.04 - 1.45)	0.15 (0.00 - 1.22)	0.16 (0.00 - 1.24)
South East Asia		**0.38 (0.21 - 0.55)**	**0.30 (0.22 - 0.38)**

*Australia included data from 2010 and 2012

#Crude incidence

Table 1 Mean Age-adjusted Annual Incidence per 100,000 from 2011-2013

	UC	CD
Age, median months (IQR)	43 (31-54)	34 (22-49)
Sex, M (%)	57.9%	59.8%
Time of symptom onset to diagnosis, median months (IQR)	3 (2-10)	7 (2-19)
Smoking		
Current	8.9%	11.6%
Never	74.6%	77.1%
Ex	16.5%	11.3%
Family History	2.4%	5.3%
CD disease location, n (%)		
L1: Terminal ileum	-	31.9%
L2: Colon	-	24.4%
L3: ileocolon	-	43.8%
L4: Upper gastrointestinal	-	6.20%
CD disease behavior, n(%)		
B1: inflammatory	-	70.9%
B2: stricturing	-	21.2%
B3: penetrating	-	9.80%
p: perianal	-	17.4%
UC disease extent, n(%)		
E1: proctitis	32.8%	-
E2: distal	34.4%	-
E3: extensive	32.8%	-

Table 2 Disease Characteristics of IBD Patients

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Fruit Consumption May Protect Against the Development of Intestinal Inflammation via Modification of Microbial Composition

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Background: Patients with ulcerative colitis (UC) undergoing proctocolectomy with ileal pouch-anal anastomosis commonly develop pouchitis. As pouchitis is inflammation developing in a previously normal small bowel we hypothesize that it may represent the small intestinal inflammation characterizing Crohn's disease (CD). CD pathogenesis involves environmental factors. We thus asked whether diet and the microbiome had a role in pouch inflammation. **Methods**: Patients recruited at the Comprehensive Pouch Clinic were prospectively followed-up. Pouch behavior was determined clinically and defined as normal pouch (NP) or pouchitis. All patients completed Food Frequency Questionnaires (FFQs). Fecal samples were collected and analyzed for microbial composition (16S rRNA gene pyrosequencing). Microbial diversity was calculated using Shannon diversity index. P values were corrected for multiple comparisons using false discovery rate (FDR<0.1). **Results**: A total of 172 pouch patients (89 [52%] females, average age 44.9±14 years, mean time since ileostomy closure 9.1 [range 0-30.4] years) were recruited. At the beginning of follow up, 39 (22.6%) patients had NP. Within 1 year of follow up, 5 (12.8%) of these developed pouchitis. Higher (>1.45 servings/day), compared to lower fruit consumption was associated with significantly less development of pouchitis (3.8% vs. 30.8%, respectively, log rank test, p=0.03). Fecal microbial analysis was performed in 81 patients (NP [n=22], pouchitis [n=53], familial adenomatous polyposis [n=7]). Fruit consumption correlated with microbial diversity (r=0.37, p=0.001), and with the abundance of several microbial groups. After adjustment for pouch behavior and use of antibiotics, fruit consumption remained positively correlated with *Faecalibacterium* (r=0.27, p=0.01), *Lachnospira* (r=0.31, p=0.005) and two un-annotated genera from the *Lachnospiraceae* and *Ruminococcaceae* families (r=0.24 p=0.03; r=0.28, p=0.01, respectively). Significant decrease in fruit consumption was noticed in 10 patients who developed active disease over time (Δ=-1.4±1.7 s/d, p=0.02). Corresponding decrease in microbial diversity was noticed as well (Δ=-0.6±1.2, p=0.17). **Conclusions**: Fruit consumption of pouch patients significantly modified microbial composition, favoring expansion of Firmicutes, specifically *Faecalibacterium*, *Lachnospiraceae* and *Ruminococcaceae*. Significant decrease in fruit consumption was associated with the development of active disease and with a decrease in microbial diversity. Thus, fruit consumption may be protective against intestinal inflammation, possibly by altering microbial composition. Therefore, dietary intervention may contribute to prevention of intestinal inflammation.

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Association of Environmental Exposures With the Composition and Diversity of the Human Gut Microbiome in Healthy First Degree Relatives (FDR) of Crohn's Patients

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The nature of the intestinal microbiome community is influenced by host and environmental factors. The specific influences of environmental exposures on the microbiome have yet to be fully defined. To date, only a few studies have assessed the complex interactions between host and environment and gut microbiome. The objective of this study was to investigate if demographic and environmental variables are associated with fecal microbiota composition