A recent epidemiological study clearly demonstrated that inflammatory bowel disease (IBD), especially ulcerative colitis, is a rapidly emerging disease in the Asia Pacific region (1). A systematic review of the global incidence and prevalence of IBD before publication of the Asia Pacific data (2) confirmed the worldwide nature of IBD. In time-trend analyses, it showed that 75% of Crohn disease studies and 60% of ulcerative colitis studies reported statistically significant increases in incidence (2). The emergence of IBD in the Asia Pacific region suggests strong environmental influence on the pathogenesis of IBD including socioeconomic, lifestyle and dietary changes.

The age-standardized incidence of ulcerative colitis in the Asia Pacific region ranged from 0.24 to 7.47 per 100,000 population, with the lowest incidence in Bangkok and the highest in Australia. Even highly westernized Asian countries, such as Hong Kong, have an ulcerative colitis incidence of 1.30 per 100,000 population (1). The incidence of Crohn disease in Australia was nearly double that of ulcerative colitis; however, in the Asian countries, ulcerative colitis was generally more prevalent than Crohn disease. In a recent Asia Pacific epidemiology study of IBD incidence (1), the distribution of ulcerative colitis location in Australia and in Asia were similar, with approximately one-third each being proctitis, left-sided colitis, and extensive or total colitis (1). The Asia Pacific epidemiology study did not include South Korea, Japan or India, which tend to have a higher incidence of IBD than other Asian countries. However, in the current issue of the Journal, Park et al (3) (pages 125 to 130) reported a retrospective analysis of disease location at diagnosis in 240 ulcerative colitis patients diagnosed at Asan Medical Centre, Seoul, South Korea. Approximately 19% of patients had an atypical distribution of disease, including patchy or skip lesions or, less commonly, rectal sparing. Such atypical distribution was more common in patients reported to demonstrate appendiceal orifice inflammation. Skip lesions were more common in the proximal colon. While such atypical distribution inevitably raises the question of Crohn’s colitis, 67% of ulcerative colitis patients showing atypical distribution at diagnosis had reverted to the typical distribution of lesions at follow-up colonoscopy. The ulcerative colitis disease course and outcome of individuals with atypical disease location was similar to those with more classical disease location.

While such atypical distribution of ulcerative colitis has been sporadically reported in the West (4,5), including 27% of ulcerative colitis patients showing discontinuous inflammation of the appendiceal orifice in a prospective study, rectal sparing is considered to be unusual in the West after meticulous pathological examination of colectomy specimens (6). This is not surprising given that in the study by Park et al (3), follow-up colonoscopy showed that the distribution of ulcerative colitis had reverted to the classical in the majority of patients. However, several reports from Asia, especially Japan, Korea and India, with higher incidence of ulcerative colitis in Asia, have reported atypical distribution of ulcerative colitis (7-9) despite the contrary data in the recent Asia Pacific epidemiology study. Although single-centre and retrospective, the study by Park et al (3) substantiates these previous findings. It is important to recognize that the Asia Pacific region is a vast, inhomogeneous area with multiple cultures, lifestyles, environment, diet and genetics. For example, three regions of mainland China had ulcerative colitis incidences of 2.05, 0.42 and 0.41 per 100,000 population (1), suggesting unique environmental influences in specific regions.

The study by Park et al (3) and previous studies encourage us to obtain additional epidemiological and environmental data collected prospectively from geographical regions with emerging IBD. It will be important to understand whether disease presentation varies in different geographical areas based on colonic microbiome, dietary habits and genetics. This may shed light on its pathogenesis and provide more evidence to answer the question whether ulcerative colitis is ‘one disease or many’. This discussion may be even more relevant in the case of Crohn disease. It especially points to the challenges of epidemiological studies that attempt to distinguish between ulcerative colitis and Crohn’s colitis.

REFERENCES