X Qin. How to explain recent multiple reports on the decline of inflammatory bowel disease in Canada.**Can J Gastroenterol Hepatol 2014;28(11):620.***To the Editor:*

I read with great interest multiple recent reports regarding the decrease of inflammatory bowel disease (IBD) in Canada, including the study by Bitton et al (1) showing the significant decline in the annual incidence of IBD including both Crohn disease (CD) and ulcerative colitis (UC) in Quebec from 2001 to 2008, and another by Leddin et al (2) showing the remarkable decline in both CD and UC in Nova Scotia between 1996 and 2009. These declines are unlikely to reflect the natural trends in IBD incidence, and do not correlate with progress in modernization. In fact, a decline and plateau in the incidence of IBD was also observed during the late 1970s and early 1980s in multiple studies from many countries including Canada, United States, Germany, United Kingdom, Denmark, Sweden, Israel and Japan. These studies were collectively discussed in an article I wrote regarding a unified hypothesis for the etiology of IBD (3). However, those declines were followed again by a striking increase in IBD in these countries and all over the world thereafter. How do we explain this change? The answer may provide valuable clues as to the cause and root mechanism of IBD, which would be critical for the prevention and cure of the disease.

Evidence I collected over the past decade made me suspect that dietary chemicals, such as the widely used artificial sweeteners saccharin and sucralose, may be important causative factors for IBD through inhibition on gut bacteria and the resultant impaired inactivation of digestive proteases, as well as damage of mucus layers and underlying gut tissue (3). The decline in IBD during the latter 1970s and early 1980s was in accordance with decreased consumption of saccharin after the finding in 1977 by studies in Canada reporting that saccharin may cause cancer in animals. The new global increase in IBD may be related to the increased consumption of sucralose, which was first approved in Canada in 1991 followed by many other

countries all over the world (3,4). There has been constant dynamic change in the production and marketing of dietary chemicals, such as artificial sweeteners, over time. Additionally, significant variation in consumption among different countries and ethnicities, even among different regions within the same country and different age groups within the same population, has been observed. Intriguingly, many of the peculiar changes in IBD, such as the recent decrease in CD but increase in UC in children in Sweden (5), as well as the shared trend of change of pediatric IBD in Sweden with the general population IBD in Denmark but not pediatric IBD in Norway (6), can also be easily explained by the pattern of consumption of some artificial sweeteners and the united hypothesis published earlier (3). Here, I advocate investigating a possible link between dietary chemicals and IBD.

Xiaofa Qin MD PhD
GI Biopharma Inc
Westfield, New Jersey,
USA

REFERENCES

1. Bitton AM, Vutcovici V, Patenaude M, Sewitch, S, Suissa, Brassard P. Epidemiology of inflammatory bowel disease in Quebec: Recent trends. *Inflamm Bowel Dis* 2014;20:1770-6.
2. Leddin D, Tamim H, Levy AR. Decreasing incidence of inflammatory bowel disease in Eastern Canada: A population database study. *BMC Gastroenterol* 2014;14:140.
3. Qin X. Etiology of inflammatory bowel disease: A unified hypothesis. *World J Gastroenterol* 2012;18:1708-22.
4. Qin X. What made Canada become a country with the highest incidence of inflammatory bowel disease: Could sucralose be the culprit? *Can J Gastroenterol* 2011;25:511.
5. Qin X. How to explain the discordant change of ulcerative colitis and Crohn disease in adjacent or even the same regions and time periods. *J Pediatr Gastroenterol Nutr* 2013;57:e30.
6. Qin X. Why pediatric inflammatory bowel disease (IBD) in Sweden shared similar trend of change as general population IBD in Denmark but not pediatric IBD in Norway? *Scand J Gastroenterol* 2014;49:1268-9.