



Clinical Group

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Xiaofa Qin*

GI Biopharm Inc, 918 Willow Grove Road, Westfield, NJ 07090, USA

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*Corresponding author: Xiaofa Qin, GI Biopharma Inc 918 Willow Grove Road, Westfield, NJ 07090, USA, Tel: 908-463-7423; E-mail: xiaofa_qin@yahoo.com

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Short Communication

More Pronounced Reduction of Gut Bacteria in Crohn's Disease than Ulcerative Colitis may have just Reflected Intimate Connection Rather than Difference of these two Diseases-A Different View on Findings Published in Gut

Short Communication

I read with great interest a paper published recently in Gut [1], as its finding that patients with Crohn's disease (CD) have more pronounced reduction and dysbiosis in gut bacteria than ulcerative colitis (UC) is just as what I have predicted during the last fifteen years [2,3]. However, in contrast to the notion of the authors of the Gut paper that the more pronounced reduction in gut bacteria in CD suggests CD and UC are two different diseases, my prediction came from the perception of the profound intimate connections between UC and CD. As we know, countries and places have a high incidence of UC usually also have high rate of CD. However, it always showed a pattern that UC emerged first, followed by CD, then CD showed a trend of catching up or even overpassing the incidence of UC. From my finding that digestive proteases such as trypsin and chymotrypsin can be inactivated by unconjugated bilirubin but not conjugated bilirubin and information gathered after that on inflammatory bowel disease (IBD) including UC and CD, I perceived that the primary cause for both UC and CD may be the impairment in the inactivation of digestive proteases due to the reduction in gut bacteria, thus β -glucuronidase enriched in certain kinds of gut bacteria that is needed for catalyzing the deconjugation of biliary bilirubin. UC and CD only differ in that the ubiquitous existence of bacteria in the colon makes infiltration of bacteria, recruitment of neutrophils and formation of crypt abscess as the main characteristic feature of UC, while CD is formed at places with less bacteria, such as in the small intestine, or more striking reduction in gut bacteria as evidenced by the result of newly published Gut paper, along with industrialization, modernization and westernization, resulting in increased infiltration of particles and antigens from gut lumen and the recruitment and reaction of macrophages

and lymphocytes and formation of granulomas as the main characteristic features of CD. This notion is also in accordance with the continue increase of the so-called colonic CD over time [3], such as the increase of colonic CD in Stockholm County, Sweden from 15% during 1959-1964 to 32% during 1980-1989, and further to 52% during 1990-2001, and the decrease of ileocaecal CD from 58% during 1959-1964 to 41% during 1980-1989, and to 28% during 1990-2001 [4,5], as well as the similar changes observed in Cardiff, UK [6]. Thus I believe UC and CD are just two manifestations of the same morbidity, rather than two different diseases, as first described in the original paper published in 2002 [2] then discussed in more detail in a paper published in 2012 with a unified hypothesis on the etiology of IBD, including the cause and mechanism of IBD as well as the relationship between UC and CD [3] and many other papers with explanations from such a view on different aspects of IBD such as epidemiological, immunologic, therapeutic, clinical, pathological, genetic, dietary, microbial, etc [7]. Thus the newly revealed greater reduction and dysbiosis in gut bacteria in CD as I predicted and discussed time and again during the last fifteen years would be just another piece of evidence of the intimate essential connection between UC and CD hiding behind the superficial diversity and differences as perceived by the authors of the paper. In my opinion, we should put more effort to find out the essential nature and primary cause of IBD but not be bewildered by the phenomenal complexity.

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