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Diet in the etiology of inflammatory bowel disease

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The two predominant forms of inflammatory bowel disease (IBD), namely Crohn's disease (CD) and ulcerative colitis (UC), can have a significant impact on patients' quality of life and incur major costs in terms of time off work and therapies. The precise etiology of IBD is unknown, but is probably due to a combination of: genetic, immunological and environmental factors. The role for the latter is strongly supported by rises in the incidence of IBD in the 20th century, and the emergence of both CD and UC in countries in the East (1). Dietary changes over time and according to region may be the environmental factor explaining these incidence patterns.

Recently, there has been much research on habitual diet in the etiology of IBD, including carbohydrates, fatty acids, alcohol and dietary patterns (2-8). The article published in the current issue of the journal reports a meta-analysis of dietary carbohydrate in IBD, and for CD and UC separately (9). The methodology and analyses are well conducted, and the authors reported no significant associations with odds ratios close to unity. Importantly, the researchers investigated effects according to the study design, namely case-control or cohort investigations, and again found no associations. This distinction in reviewing results according to methodology is important as cohort studies minimize firstly recall biases for dietary intake, and secondly selection biases of the participants studied. In cohort investigations, subjects report their current diet before a diagnosis of IBD, compared to in a case-control investigation where patients diagnosed with IBD have to recall their diet prior to symptoms. If the latter is many months or years previously, recall is impaired and current diet may be reported, which has been altered by symptoms. Furthermore, in a cohort investigation, as future cases and controls are drawn from the same population and have

comparable characteristics, there is less selection bias than in retrospective case-control work.

This meta-analysis does not provide support for carbohydrates influencing the development of IBD. The role of diet now needs to be further studied for other nutrients with plausible mechanisms. In addition to ensuring the correct choice of study design, there are other methodological issues that need to be considered. For example, measuring habitual diet can be challenging, and the availability of biomarkers as accurate measurement of dietary intake are beneficial if available. For some nutrients such as fatty acids these exist, but not for all dietary components. The authors correctly comment that such epidemiological work needs to be completed in many regions around the world, and that sub-group analyses are required. For example, in Crohn's disease the etiology of small-bowel and colonic Crohn's disease may differ and justify individual assessment.

The impact of IBD on the lives of sufferers is such that further etiological work on IBD is urgently required. Ideally, such research should be of a prospective design, using a valid biomarker of dietary intake, according to disease site, with consistent findings from studies in many regions. Such work may lead to dietary changes to actually prevent IBD, but also inform modifications to assess in patients in order to improve clinical outcomes and quality of life.

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