Prebiotics and Mucosal Barrier Function

Dear Editor,

In the January issue of The Journal of Nutrition, Dr. Ten Bruggencate and co-workers reported the results of a nutritional intervention study aimed at investigating the effect of fructooligosaccharides on intestinal barrier function in human volunteers consuming a low calcium diet (1). The study did not show changes in intestinal barrier function as assessed by the CrEDTA test (a validated method to measure intestinal permeability), because the test outcome was identical during both placebo and fructooligosaccharide feeding periods (see Table 1). However, contrary to the evidence presented in the paper, the title of the article and the conclusions in the Abstract and Discussion sections indicate that fructooligosaccharides did affect intestinal barrier function in healthy males.

The assumption made by the authors to support their conclusion is that fecal mucin excretion is a marker of mucosal irritation. Because fecal mucin excretion was increased during the fructooligosaccharide feeding period, the authors presume that fructooligosaccharides caused mucosal irritation. This concept is incorrect, however, and not based on the scientific evidence. All dietary fibers (2–7), and even other indigestible substrates (8), can increase small intestinal and colonic mucin secretion to a variable extent. This is a consequence of both short-chain fatty acid production (6) and mechanical stimulation (4) by increased fecal mass (both effects were documented during the fructooligosaccharide feeding period in the trial), but it has never been suggested that this reflects mucosal irritation by the fiber. On the contrary, experimental data have shown that the effect of fibers on mucin is beneficial for transit time (lubricant effect of mucus, see references 2 and 7) and improves intestinal barrier function (4,5). When considering specifically the variable measured in Ten Bruggencate’s study, fecal excretion of mucin, it is also important to bear in mind that this is directly related with fecal weight and fecal water content (7,9). During the fructooligosaccharide feeding period, subjects experienced increased fecal output in terms of wet and dry weight of feces (Table 1) and, thus, fecal mucin excretion was also increased.

There is ample experience with physiological stimulants of mucin secretion other than fibers. For instance, certain probiotic bacteria were shown to stimulate mucin secretion by intestinal epithelial cells, which resulted in protection against subsequent invasion by pathogens (10). Likewise, we have found that stimulation of mucus secretion by epidermal growth factor protects against luminal aggressions both in the upper and lower intestinal tract (11,12). Neither probiotic lactobacilli nor epidermal growth factor are mucosal irritants, even if they stimulate mucin secretion.

In summary, the study by Ten Bruggencate and co-workers demonstrates that dietary fructooligosaccharides do not affect intestinal barrier function in healthy men on a low calcium diet, as proven by the CrEDTA test. The authors should consider revision of their conclusion, which is not supported by the data presented in the paper.

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Literature Cited


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