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EDITORIAL

Maltodextrin, Modern Stressor of the Intestinal Environment

A critically important new study by Laudisi et al[1] shows that consumption of the food additive maltodextrin, incorporated into many processed foods, leads to the promotion of intestinal inflammation. These findings suggest that this broadly used food additive could be a risk factor for chronic inflammatory diseases.

Maltodextrin is a polysaccharide derived from starch hydrolysis and used as a thickener and filler in processed food. Maltodextrin is regarded as inert and generally is regarded as safe by the US Food and Drug Administration. However, multiple recent studies have shown detrimental roles played by maltodextrin in the intestinal environment, suggesting that this broadly used food additive may play a role in the rapid increased incidence of chronic inflammatory disorders, such as inflammatory bowel disease and metabolic syndrome. In this issue of *Cellular and Molecular Gastroenterology and Hepatology*, Laudisi et al[1] add a new line to this list of evidences, with the investigation of the impact of maltodextrin on murine models of colitis. By using 2 different models of colitis, the investigators found that consumption of maltodextrin exacerbated intestinal inflammation in a dose-dependent manner. Mechanistically, such detrimental effects of maltodextrin were linked to activation of endoplasmic reticulum stress and subsequent alterations of the protective mucus layer. Importantly, pharmacologic inhibition of endoplasmic reticulum stress was sufficient to prevent maltodextrin-induced intestinal inflammation and associated depletion in the mucus layer.[1]

With the appreciation that the intestinal microbiota plays a central role in intestinal inflammatory diseases, Laudisi et al[1] also investigated mucosa-associated microbiota after maltodextrin consumption. Although they did not observe any major alterations in microbiota composition, suggesting that maltodextrin-induced detrimental effects are microbiota-independent, more subtle effects on the microbiota or microbial metabolites cannot be excluded and are deserving further studies.

Indeed, previous work has shown that maltodextrin favors biofilm formation by Crohn's disease–associated adherent-invasive *Escherichia coli* bacteria by modulating bacterial gene expression[2] as well as microbiota encroachment (microbiota penetration of the normally sterile inner mucus layer) in the mouse distal colon.[3]

Importantly, in addition to the use of a murine model of colitis, Laudisi et al[1] next investigated the impact that maltodextrin may have on a wild-type host (ie, without genetic susceptibility or induced colitis). Although maltodextrin did not induce visible levels of intestinal inflammation after 45 days of treatment, longer exposure led to the development of low-grade intestinal inflammation, characterized by a subtle but nonetheless consistent increase in intestinal inflammatory markers. This treatment was also associated with alterations of the protective mucus layer, ultimately leading to metabolic abnormalities, as previously reported in other models of low-grade intestinal inflammation.[4]

Altogether, these findings show that maltodextrin detrimentally impacts the intestinal environment by promoting depletion of the protective mucus layer and favoring the development of intestinal inflammation. Interestingly, another study recently showed that the artificial sweetener Splenda, which contains both sucralose and maltodextrin, impacts the intestinal microbiota in a manner that promotes Crohn's-type disease in genetically susceptible hosts.[5]

Moreover, studies investigating the effects of maltodextrin on the central nervous system and behavior may shed light on yet another mechanism by which maltodextrin promotes metabolic abnormalities. Indeed, a recent study found that Splenda alters neuronal activity in the ventromedial hypothalamus and hippocampus of rats, 2 areas of the brain known to play an important role in food intake, food preference, obesity, and energy homeostasis.[6]

To conclude, these recent results, together with previous reports, suggest that consumption of the food additive maltodextrin may be a risk factor for the inflammatory bowel disease–prone population, as well as a factor promoting chronic low-grade intestinal inflammation leading to metabolic abnormalities in the general population. Mechanistic studies are now needed to understand the mechanism by which maltodextrin detrimentally impacts the intestinal environment, since this compound is readily metabolized to glucose in the intestinal tract. Moreover, these findings support the concept that US Food and Drug Administration testing of food additives should be performed in disease-prone and -resistant models designed to detect chronic and-low grade inflammation, as well as to asses the impact on the gut microbiota.

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