Dietary Maltodextrin May Enhance Adherence with Invasive *E.coli* and Promote CD

Introduction

Inflammatory bowel disease (IBD) affects approximately 1.3 million people in the United States. Crohn's Disease (CD) is a chronic and inflammatory form of IBD with an estimated prevalence of nearly 200 cases per every 100,000 adults, according to the Centers for Disease Control and Prevention (CDC).[1] The clinical manifestations of CDare varied and can often go undiagnosed for many years. The inflammatory nature of CD can trigger numerous long-term debilitating extra-intestinal symptoms including arthritis, dermatologic disorders, hypercoagulability, and various nutrient deficiencies.[2] Numerous contributing factors are known to influence the risk and pathophysiology of CD including genetics, bacterial and environmental factors. Mounting evidence suggests that intestinal dysbiosis and the presence of adherent-invasive *E.coli* bacterial strains may play a crucial role in disease pathogenesis, and that the ubiquitous intake of dietary maltodextrin may actually enhance *E.coli* adhesion and increase gut dysbiosis.

Adherent-Invasive *E.coli* in CD

Intestinal dysbiosis has been strongly associated with the development of CD as evidenced by the observation of thick biofilm formation on gut epithelium. [3] Adherent-invasive *E.coli* (AIEC), isolated from gut biopsies, has been frequently found in people with CD. In one study of 20 participants with CD and 28 healthy control subjects, researchers discovered approximately 100 *E.coli* colonies per biopsy from Crohn's participants, and *E.coli* counts were significantly higher in Crohn's participants, suggesting the implication of AICE in CD.[4] In a study of CD in the pediatric population, researchers found two AIEC bacterial strains (EC15, and EC10) in the mucosal biopsies of children with IBD, and these strains were able to survive inside macrophages while damaging intestinal barrier integrity.[5]

AIEC have been found in abundance adhering to ileal epithelial cells in greater numbers in people with CD, and appear to have varying levels of virulence depending on one's genetic SNP predisposition.[6][7] People with CD have demonstrated abnormal expression of the CEACAM6 receptor, a site where AIEC prefer to bind to ileal enterocytes.[6] Once the AIEC have adhered to the epithelium, the bacteria are able to colonize the gut mucosa, invade the epithelial cells and macrophages; subsequently the AIEC replicate inside of the occupied macrophages. In one study researchers found a 36.4% predominance of AIEC in the ileal mucosa of Crohn's participants, compared to only 6.2% in the control subjects.[6] These results strongly suggest the implication of AIEC in the pathogenesis of CD.

Maltodextrin Influences on Bacterial Growth and Adhesion

Polysaccharides, like maltodextrin, are common additives used to emulsify, stabilize or bulk up foods found in typical Western diets, which are also high in fat and sugar. For example, maltodextrin is added as a bulking agent in many no-calorie alternative sweeteners like Equal® (aspartame), and Splenda® (sucralose). These polysaccharides have been suggested as contributing factors for the development of bacterial associated gut disorders, specifically CD. There has also been a corresponding increase in the incidence of both CD and the consumption of maltodextrin suggesting a possible correlation.[8]

Some research has shown that maltodextrin actually enhances bacterial biofilm formation, including multiple species of *E.coli.* Researchers have also concluded that maltodextrin increased bacterial adhesion, which indicates that Western diets, commonly high in maltodextrin and other polysacchardes, may also promote AIEC, gut dysbiosis, and CD in susceptible individuals.[8] Evidence also has found that maltodextrin is metabolized by enzymes in the small intestine, including the enzyme maltase-glucoamylase, which is actually inhibited by high levels of maltodextrin. In diets where maltodextrin is consumed in large amounts, the small intestine could actually experience an accumulation of maltodextrin, which in turn may account for increased pathogenic bacterial imbalance in the small intestine; all of which may explain AIEC presence in CD.[8]

Conclusions

The ubiquitous use of maltodextrin as a dietary additive could potentially be contributing to the rising incidence of CD, particularly in younger populations. Abnormal bacterial microflora, specifically adherent-invasive *E.coli* has been linked in numerous studies to the development of inflammatory bowel disease. The consumption of maltodextrin, from common dietary sources including alternative, no-calorie, sweeteners appears to be strongly associated with the promotion of CD by stimulating gut dysbiosis leading to AIEC biofilm formation. Innovative dietary changes and nutritionally related interventions aimed at addressing bacteria dysbiosis, and AIEC specifically, are promising therapeutic options in the management and prevention of CD in at risk patient populations.

References

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