Dietary Xylooligosaccharide Downregulates IFN-γ and the Low-Grade Inflammatory Cytokine IL-1β Systemically in Mice

Dietary carbohydrates improve growth conditions for distinct populations of bacteria that may affect mucosal and systemic immunity. In this study, we fed in a parallel experiment a 10% xylooligosaccharide (XOS)–supplemented diet or a control diet to 2 groups of male C57BL/6NTac mice for 10 wk from weaning. We found that the XOS diet significantly increased Bifidobacterium throughout the intestine compared with control-fed mice, with the highest proportions found in the ileum after XOS feeding (P <0.001). In the intestinal epithelium, most innate immune-related genes were unaffected by XOS feeding, whereas expression of interleukin 1β (Il1β) (P <0.01) and interferon γ (Ifnγ) (P <0.05) was significantly less in blood from XOS-fed mice than from control-fed mice. In vitro treatment of blood with propionate significantly decreased Il1β (P <0.01), Ifnγ (P <0.01), and interleukin 18 (Il18) (P <0.001) expression, supporting our hypothesis that increased production of short-chain fatty acids (SCFAs) in the gut, which are transported across the intestine and into the systemic compartments, results in downregulation of low-grade inflammatory cytokines. The defensin regenerating islet-derived protein 3γ (RegIIIγ) was significantly more highly expressed in the small intestine (P <0.01) in XOS-fed mice compared with control-fed mice, suggesting only minor contact between bifidobacteria and epithelial cells. In support of this, the SCFA-induced sodium/hydrogen exchanger isoform 3 expression tended to be greater in the XOS group than in the control group (P = 0.06), indicating an indirect SCFA-mediated antiinflammatory effect of XOS. In conclusion, XOS feeding decreases systemic inflammation, and this effect is most likely caused by higher SCFA concentrations as a result of an increased bifidobacterial saccharolytic fermentation in the entire gut and not only in the large intestine.

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