

OBESITY

Fermentable carbohydrates increase satiety signals

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Supplementing the diets of rodents and humans with fermentable carbohydrates can reduce appetite and body weight gain; however, the mechanisms underlying this action were not known. Now, a new paper suggests that the effect of these carbohydrates is dependent on free fatty acid receptor 2 (FFAR2).

When the gut microbiota ferments non-digestible carbohydrates, they produce short-chain fatty acids (SCFAs) such as acetate and propionate, which signal via FFAR2. “We hypothesized the mechanism by which fermentable

carbohydrates protect against diet-induced obesity is by increased SCFA production and signalling via the FFAR2 in the colon,” explain corresponding authors Gary Frost and Gavin Bewick.

Ffar2^{-/-} mice and wild-type littermates were fed a high-fat diet containing inulin (a fermentable carbohydrate) or a control diet for 14 weeks. Wild-type mice, but not knockout mice, given inulin were protected against diet-induced obesity and had reduced adiposity. “Protection against diet-induced obesity by fermentable carbohydrates is dependent on FFAR2,” conclude Frost and Bewick.

The wild-type mice given inulin exhibited reduced food intake. The researchers found that the concentration of the anorectic gut hormone peptide YY was increased by 130% in the colons of wild-type mice given inulin, but no effect was seen in the knockout mice. Furthermore, immunofluorescent staining of cultured cells demonstrated that inulin

supplementation caused an increase in the density of cells that secrete peptide YY in wild-type mice, but not in *Ffar2*^{-/-} mice. This increase results in an increased satiety signalling potential of the gut.

“A number of important questions remain regarding the mechanisms by which fermentable carbohydrates improve metabolic health and more generally with respect to dietary constituents and their potential to remodel the gut and thereby influence physiology,” comment Frost and Bewick. For instance, exactly how FFAR2 affects cell fate is still not known. Reflecting on the wider implications of their research, Frost and Bewick wonder whether other microbial fermentation products or dietary components can remodel the gut. “If so, which cell types do they influence and can these systems be targeted either by diet or pharmaceutical means to change the architecture of the gut in order to treat a host of gut endocrine related disorders?”

Claire Greenhill

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