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GUT MICROBIOTA

Gas-induced GLP1 release

New research shows that hydrogen sulphide (H_2S), a microbial gas produced in the colon by sulphate-reducing bacteria (SRB), can directly stimulate the release of glucagon-like peptide 1 (GLP1; an incretin hormone involved in glucose homeostasis and appetite regulation) from enteroendocrine L cells in the gastrointestinal tract. The findings support the increasingly recognized role of the gut microbiota and its metabolites as key regulators of host metabolism.

As high concentrations of H_2S are produced in the vicinity of GLP1-secreting L cells, Jeffrey Gagnon and colleagues were interested in potential crosstalk between H_2S and GLP1. The team treated mouse L cells (the GLUTag cell line) with a H_2S donor (NaHS or GYY4137) and found that GLP1 secretion was increased approximately twofold compared with vehicle-treated cells.

To confirm the findings *in vivo*, C57BL/6 male mice were fed a diet low in fermentable carbohydrate with or without the prebiotic chondroitin sulphate for 4 weeks. Chondroitin sulphate treatment increased the abundance of the SRB *Desulfovibrio piger* in faeces and faecal and colonic levels of H_2S. Moreover, this increase in H_2S-producing bacteria was accompanied by enhanced GLP1 and insulin secretion, improved oral glucose tolerance and reduced food consumption.

Although absolute proof that H_2S-producing bacteria can stimulate GLP1 secretion will require confirmatory studies in germ-free mice given a defined probiotic that contains SRB, Gagnon is optimistic about the findings so far. "The compound we used, chondroitin sulphate, is a relatively affordable natural health product that is currently used in the treatment of arthritis," he explains. "If future work can demonstrate a similar enhancement of GLP1 levels in humans, chondroitin sulphate could become a useful tool to treat obesity and type 2 diabetes mellitus."

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ORIGINAL ARTICLE Pichette, J. *et al.* Hydrogen sulfide and sulfate prebiotic stimulates the secretion of GLP-1 and improves glycaemia in male mice. *Endocrinology* http://dx.doi.org/10.1210/en.2017-00391 (2017)

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