Editorial

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Endocrinology and Metabolism: Open Access

2021
Vol. 5 No. 2: e158

Controlling of Appetite and Regulating Metabolism

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Received: March 04, 2021; **Accepted:** March 18, 2021; **Published:** March 25, 2021

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Citation: Neuman D (2021) Controlling of Appetite and Regulating Metabolism. Endocrinol Metab Vol. 5 No.2: e158.

Description

A classic role of the gut microbiota is in digesting of carbohydrates and fermenting them into Short-Chain Fatty Acids (SCFAs). Germfree (GF) mice have different metabolic profiles than conventionally raised mice, including low concentrations of SCFAs, hepatic triacylglycerol and glucose. Interestingly, sub-therapeutic doses of antibiotics, which don't eliminate the gut microbial community but rather cause significant changes in its composition, because, increased levels of SCFAs and to weight gain in mice. The metabolic effects of the microbiome may further affect regulation of hormone production from cholesterol, peptides or amino acids. SCFAs are shown to stimulate release of 5-HT and thus the peptide YY, a hormone released after feeding involved in appetite reduction and decreasing of gut motility.

Few more hormones, mainly neuropeptides that have a task in controlling appetite and regulating metabolism, are likely affected by the gut microbiota. These include alpha-melanocyte-stimulating hormone, neuropeptide Y, agouti-related protein, ghrelin, leptin, insulin et al. Another effect of bacteria on metabolic hormones could be through production of somatostatin, which suppresses the discharge of the GI and pancreatic hormones.

Several pieces of evidence link the microbiota function to leptin levels. First, use of antibiotics (vancomycin) in rats leads to a dramatic decline (38%) in circulating leptin levels. Second, the abundance of several bacterial genera (e.g. Mucispirillum, Lactococcus, Bifidobacterium and Lactobacillus) positively correlates with circulating leptin concentrations in mice, while other bacterial genera (e.g. Allobaculum, Clostridium, Bacteroides and Prevotella) negatively correlate with leptin levels. These correlations may stem from bacteria affecting hormone levels, or the opposite way around. One proposed mechanism is that diet composition may impact leptin concentrations, which, in turn, may change the microbial community composition through inflammation and/or regulation of mucus production.

Recently, a study demonstrated that leptin may additionally influence the gut microbiota independently of diet. Another model proposes that L. plantarum specifically suppresses leptin by reducing adipocyte cell size in white fat tissue. This fits the finding that use of the probiotic L. plantarum during a gaggle of human smokers reduced their serum leptin levels.

Because leptin is involved in appetite inhibition, metabolism and behavior, deciphering its interconnections with bacteria is of great interest and will help us understand and perhaps control its many effects.

Ghrelin, another important appetite-regulating hormone, is negatively correlated with the abundance of Bifidobacterium, Lactobacillus and B. coccoides Eubacterium rectale group, and positively correlated with sort of Bacteroides and Prevotella species. Intake of oligofructose (a prebiotic that promotes growth of Bifidobacterium and Lactobacillus) decreases secretion of ghrelin in obese humans.

Insulin, the extremely important metabolic hormone involved in diabetes and metabolic syndrome, may provide another link between the microbiome and hormones. Significant variations in microbiome composition are observed in diabetes patients compared to healthy controls. Certain bacterial species are positively or negatively correlated with insulin levels. Transfer of the intestinal microbiota (including butyrate-producing microbiota) from lean donors to metabolic syndrome patients enhanced insulin sensitivity. The effect is perhaps going mediated by altering immune components. However, additional hormones also can be involved during this process.

One such example is glucagon-like peptide 1 (GLP1), associated with appetite control and insulin secretion. The intestinal microbiota are

recently implicated in lowering levels of the GLP1, and thereby slowing intestinal transit. Alterations of the microbiome through probiotics or bariatric surgery decrease adiposity and increase GLP1 levels in mice. this is often often primarily attributed to butyrate production by commensal bacteria, which can induce GLP1 production by intestinal L cells.

One interesting mechanism by which the microbiota affect peptide hormones is through autoantibodies. Fetissov et al. found that autoantibodies against peptide hormones involved in appetite control (including alpha-melanocyte-stimulating hormone, neuropeptide Y, agouti-related protein, ghrelin and leptin) exist in healthy humans and rats, and affect feeding and anxiety. In GF rats, levels of these autoantibodies are altered, suggesting a totally unique mechanism by which the microbiome can affect appetite. These findings have further implications for the potential role of the microbiota in eating disorders like anorexia. In support of this notion, differences in microbial composition are found between anorexic patients and healthy controls.

Conclusion

Finally, new correlations among the microbiota composition, hormonal levels and metabolism come from studies of gastric bypass surgery. Gastric bypass surgery has been shown to vary the intestinal microbiota composition.

While microenvironmental changes like reduced food intake and reduction of bile acids likely affect this new microbiota composition, variety of the compositional changes are likely because of alterations within the amount of intestinal hormones including elevation of glucose-dependent insulinotropic polypeptide (GIP), GLP1 and insulin following surgery. These alterations within the gut microbiome further contribute to reduced host weight and adiposity. Accordingly, transfer of the gut microbiota from RYGB-treated mice to non-operated GF mice resulted in weight loss and decreased fat mass within the recipient animals relative to recipients of the microbiota induced by sham surgery.