Using two science news articles, I have gone in and picked out excerpts relating to research, including researcher names.

I then went into PubMed and found the primary research article using the researcher name(s) and added that citation with abstract and Medical Subject Headings (MeSH).

I highlighted major MeSH terms and bolded record terms that described research concepts that would be useful to use for finding similar articles.

Your Gut Bacteria May Be Controlling Your Appetite

The microbes in your stomach seem to hijack a hormone system that signals the brain to stop eating

By Brian Handwerk
smithsonian.com
November 24, 2015

https://www.smithsonianmag.com/science-nature/gut-bacteria-may-be-controlling-your-appetite-180957389/#aSzoHcz4v3xGg0WK.99
Hear that little voice in your head telling you to skip a second slice of pumpkin pie? It might be coming not from your conscience, but from the masses of bacteria in your stomach.

Experiments in mice and rats suggest that certain microbes living in your body as part of the gut microbiome have ways of letting the brain know when they've received enough nutrients to reach their goal—creating a billion more of their kind. Those signals seem to turn hunger on and off in their hosts.

Serguei Fetissov  *Cell Metabolism*

Gut Commensal E. coli Proteins Activate Host Satiety Pathways following Nutrient-Induced Bacterial Growth.

Abstract
The composition of gut microbiota has been associated with host metabolic phenotypes, but it is not known if gut bacteria may influence host appetite. Here we show that regular nutrient provision stabilizes exponential growth of E. coli, with the stationary phase occurring 20 min after nutrient supply accompanied by bacterial proteome changes, suggesting involvement of bacterial proteins in host satiety. Indeed, intestinal infusions of E. coli stationary phase proteins increased plasma PYY and their intraperitoneal injections suppressed acutely food intake and activated c-Fos in hypothalamic POMC neurons, while their repeated administrations reduced meal size. ClpB, a bacterial protein mimetic of α-MSH, was upregulated in the E. coli stationary phase, was detected in plasma proportional to ClpB DNA in feces, and stimulated firing rate of hypothalamic POMC neurons. Thus, these data show that bacterial proteins produced after nutrient-induced E. coli growth may signal meal termination. Furthermore, continuous exposure to E. coli proteins may influence long-term meal pattern.

MeSH terms
- Adenosine Triphosphate/biosynthesis
- Amygdala/metabolism
- Animals
- Electrophysiological Phenomena
- Endopeptidase Clp
- Escherichia coli/growth & development*
- Escherichia coli/metabolism
- Escherichia coli Proteins/metabolism*
- Feeding Behavior
- Female
- Gastrointestinal Tract/microbiology*
- Glucagon-Like Peptide 1/metabolism
- Heat-Shock Proteins/metabolism
“This is something that I think could possibly be important to help understand the problem of binge eating,” he says. “If people are constantly snacking so that there is no long interval between meals, it may be that the body doesn’t receive a good satiety signal. So that could help to explain why some people would eat continually.”

The results also speak to the fascinating possibility that the trillions of microbes we house inside our gut could be influencing our bodies and minds in many more unforeseen ways.

“Here we see a bacterial protein that appears to inhibit appetite by stimulation of neurons in the brain,” Fetissov notes. “But you can imagine that other bacteria can produce other proteins that can influence not only other appetite pathways but entirely different pathways. We may find out that human behavior is in some part very much influenced by gut bacteria.”
Abstract
Numerous studies have connected the gut microbiome with diet-induced obesity; however, mechanistic explanations for the host-microbial interactions are needed. Perry et al. (2016) present studies suggesting that microbially produced acetate (MPA) increases post-prandial insulin release via a sequential and integrated gut, brain, and pancreatic signaling network promoting energy retention.

Sean Davies explained at the American Chemical Society national meeting in March.

Special microbes make anti-obesity molecule in the gut

DENVER, March 22, 2015 — Microbes may just be the next diet craze. Researchers have programmed bacteria to generate a molecule that, through normal metabolism, becomes a hunger-suppressing lipid. Mice that drank water laced with the programmed bacteria ate less, had lower body fat and staved off diabetes — even when fed a high-fat diet — offering a potential weight-loss strategy for humans.

The team will describe their approach in one of nearly 11,000 presentations at the 249th National Meeting & Exposition of the American Chemical Society (ACS), the world’s largest scientific society, taking place here through Thursday.

One advantage to microbial medicine would be that it's low maintenance, says Sean Davies, Ph.D. His goal is to produce therapeutic bacteria that live in the gut for six months or a year, providing sustained drug delivery. This is in contrast to weight-loss drugs that typically need to be taken at least daily, and people tend not to take their medications as directed over time. “So we need strategies that deliver the drug without requiring the patient to remember to take their pills every few hours,” Davies says.

Microbial metabolism of dietary components to bioactive metabolites: opportunities for new therapeutic interventions. REVIEW
Zhang LS, Davies SS. BY SAME RESEARCHER
Abstract
Mass spectrometry- and nuclear magnetic resonance-based metabolomic studies comparing diseased versus healthy individuals have shown that microbial metabolites are often the compounds most markedly altered in the disease state. Recent studies suggest that several of these metabolites that derive from microbial transformation of dietary components have significant effects on physiological processes such as gut and immune homeostasis, energy metabolism, vascular function, and neurological behavior. Here, we review several of the most intriguing diet-dependent metabolites that may impact host physiology and may therefore be appropriate targets for therapeutic interventions, such as short-chain fatty acids, trimethylamine N-oxide, tryptophan and tyrosine derivatives, and oxidized fatty acids. Such interventions will require modulating either bacterial species or the bacterial biosynthetic enzymes required to produce these metabolites, so we briefly describe the current understanding of the bacterial and enzymatic pathways involved in their biosynthesis and summarize their molecular mechanisms of action. We then discuss in more detail the impact of these metabolites on health and disease, and review current strategies to modulate levels of these metabolites to promote human health. We also suggest future studies that are needed to realize the full therapeutic potential of targeting the gut microbiota.

MeSH terms
- Animals
- Diet*
- Disease Susceptibility
- Energy Metabolism*
- Fatty Acids, Volatile/metabolism
- Gastrointestinal Microbiome
- Gastrointestinal Tract/metabolism*
- Gastrointestinal Tract/microbiology*
- Homeostasis
- Humans
- Indoles/metabolism
- Metabolome*
- Metabolomics
- Methylamines/metabolism
- Microbiota*
- Translational Medical Research
- Tryptophan/metabolism
- Tyrosine/metabolism

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How Gut Bacteria Tell Their Hosts What to Eat

By Knvul Sheikh on April 25, 2017

By suppressing or increasing cravings, microbes help the brain decide what foods the body “needs”
Gut microbes have also been shown to influence diet and behavior as well as anxiety, depression, hypertension and a variety of other conditions. But exactly how these trillions of tiny guests—collectively called the microbiome—influence our decisions on which foods to stuff into our mouths has been a mystery.

Now neuroscientists have found that specific types of gut flora help a host animal detect which nutrients are missing in food and then finely titrate how much of those nutrients the host really needs to eat. “

Ribeiro  PLOS Biology

Commensal bacteria and essential amino acids control food choice behavior and reproduction.
Leitão-Gonçalves R, Carvalho-Santos Z, Francisco AP, Fioreze GT, Anjos M, Baltazar C, Elias AP, Itskov PM, Piper MDW, Ribeiro C.

Abstract
Choosing the right nutrients to consume is essential to health and wellbeing across species. However, the factors that influence these decisions are poorly understood. This is particularly true for dietary proteins, which are important determinants of lifespan and reproduction. We show that in Drosophila melanogaster, essential amino acids (eAAs) and the concerted action of the commensal bacteria Acetobacter pomorum and Lactobacilli are critical modulators of food choice. Using a chemically defined diet, we show that the absence of any single eAA from the diet is sufficient to elicit specific appetites for amino acid (AA)-rich food. Furthermore, commensal bacteria buffer the animal from the lack of dietary eAAs: both increased yeast appetite and decreased reproduction induced by eAA deprivation are rescued by the presence of commensals. Surprisingly, these effects do not seem to be due to changes in AA titers, suggesting that gut bacteria act through a different mechanism to change behavior and reproduction. Thus, eAAs and commensal bacteria are potent modulators of feeding decisions and reproductive output. This demonstrates how the interaction of specific nutrients with the microbiome can shape behavioral decisions and life history traits.

MeSH terms
- Acetobacter/genetics
- Acetobacter/growth & development
- Acetobacter/physiology*
- Acetobacteraceae/genetics
- Acetobacteraceae/growth & development
- Acetobacteraceae/physiology
- Amino Acids, Essential/administration & dosage
- Amino Acids, Essential/analysis
- Amino Acids, Essential/deficiency
- Amino Acids, Essential/metabolism*
- Animals
- Animals, Genetically Modified
- Appetite Regulation
“The findings show there is a unique pathway that has coevolved between animals and the resident bacteria in their gut, and there is a bottom-up communication about diet,” says Jane Foster, who is a neuroscientist at McMaster University in Ontario and not associated with the study.

Although the research does not specify the exact mechanism of communication, Ribeiro thinks it could take different forms. Strong evidence from the study indicates that microbially derived metabolites carry information from the gut to the brain, telling the host whether it needs a particular kind of food. “One of the big evolutionary mysteries is why we lost the ability to produce essential amino acids,” he says. “Maybe these metabolites gave animals more leeway to be independent of these nutrients and to deal without them sometimes.”