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Abstract

Eating is often described as a simple response to hunger, but everyday food choices are shaped by a much richer conversation between the gastrointestinal tract and the brain. The gut–brain axis integrates mechanical distension, nutrients, gut hormones, microbial metabolites, vagal signals and central reward pathways to influence hunger, satiation, satiety and food preference. This narrative review presents a human-centred view of how that communication may help explain why people sometimes eat when they are not physiologically hungry, stop eating before energy needs are fully restored, or find highly palatable foods unusually difficult to resist. Particular attention is given to ghrelin, glucagon-like peptide-1 (GLP-1), peptide YY (PYY), cholecystokinin (CCK), the vagus nerve and the intestinal microbiota. Current evidence suggests that gut microbes and their metabolites may modify appetite signalling, although human studies remain heterogeneous and do not support the idea that a single 'good' or 'bad' microbiome determines eating behaviour. The gut–brain axis is therefore best understood as a flexible biological network interacting with sleep, stress, food environment, learning and reward.

Keywords: gut–brain axis; microbiota–gut–brain axis; eating behaviour; appetite; satiety; gut hormones; GLP-1; ghrelin; PYY; vagus nerve; food reward; nutrition.

1. Introduction

Why do we feel hungry at one time of day but not another? Why can the smell or sight of food trigger interest even after a meal, while stress or illness can make food unappealing? These everyday experiences show that eating behaviour is not controlled by the stomach alone. It is the outcome of continuous communication among the gastrointestinal tract, nervous system, endocrine signals, immune pathways, microbial communities and the brain. The term gut–brain axis describes this two-way communication network. Recent work places the axis at the centre of research on appetite and food intake because it connects basic physiological needs with motivation, reward and learned behaviour.

The modern perspective is useful because it replaces a simple 'hungry versus full' model with a dynamic one. The gut reports what has entered the digestive tract, how much is present and which nutrients are being processed. The brain combines these signals with memories, sensory cues, emotions, habits and the availability of food. Eating behaviour therefore reflects both homeostatic regulation—maintaining energy balance—and hedonic regulation, in which food can be rewarding even when energy needs are already met.

This review discusses the gut–brain axis as a new lens for understanding eating behaviour. It focuses on established gut hormones, neural communication and emerging evidence concerning the microbiota, while also highlighting an important limitation: much of the mechanistic literature comes from animal models, and effects observed in experimental settings do not automatically translate into predictable changes in human eating.

2. How the Gut Talks to the Brain

Communication begins as soon as food reaches the gastrointestinal tract. Stretch receptors detect distension, while enteroendocrine cells sense nutrients and release signalling molecules. These messages can reach the brain through the vagus nerve, circulating hormones and metabolites, and neuroendocrine pathways. Brainstem and hypothalamic networks then integrate incoming information with higher brain regions involved in motivation and reward.

A meal can therefore be viewed as a conversation rather than a one-way event. The gut does not merely receive food; it actively reports the meal back to the brain. This helps explain why the same amount of food can produce different experiences of hunger or fullness depending on nutrient composition, eating speed, previous meals, physiological state and environmental cues.

Figure 1. Conceptual map of gut-brain signalling in eating behaviour

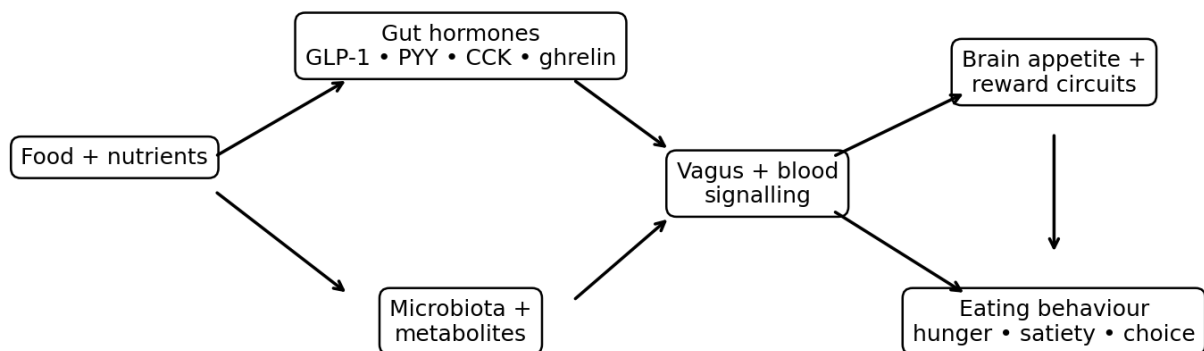


Figure 1. Conceptual map of gut–brain signalling. This is an educational schematic, not a quantitative effect-size model.

3. Gut Hormones: The Chemical Language of Appetite

Several gut-derived hormones are central to appetite research. Ghrelin is commonly described as an orexigenic, or hunger-promoting, signal, while GLP-1, PYY and CCK generally contribute to satiation or satiety. These hormones do not act in isolation; they interact with one another and with glucose, insulin, leptin and neural signals. Brain-imaging research shows that gut-related signals can influence regions involved in appetite, motivation and food-related processing.

Signal	Main source	Typical role	Behavioural relevance
Ghrelin	Stomach and upper gut	Promotes hunger	Helps signal energy need and can increase food motivation.
GLP-1	Intestinal enteroendocrine cells	Promotes satiation/satiety	Links nutrient arrival with reduced food intake and

			metabolic regulation.
PYY	Distal intestine	Promotes satiety	Helps extend the post-meal period before hunger returns.
CCK	Small intestine	Promotes satiation	Responds to nutrients and contributes to meal termination.

Figure 2. Major gut-hormone signals and appetite

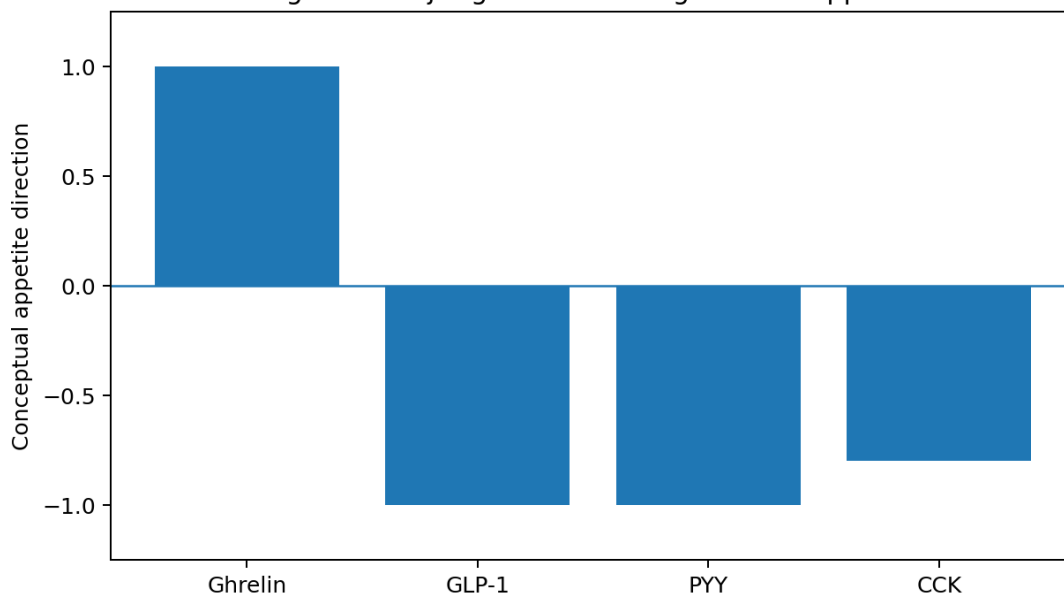


Figure 2. Conceptual direction of major gut-hormone appetite signals; not a measurement of hormone concentrations or effect sizes.

4. From Gut Microbes to Food Motivation

The microbiota adds another layer to the gut–brain conversation. Intestinal microorganisms metabolize dietary components and generate compounds such as short-chain fatty acids, bile-acid derivatives and other signalling molecules. These products can interact with intestinal cells, immune pathways and neural or endocrine signalling. Recent reviews describe microbial metabolites as important potential mediators of appetite regulation, while emphasizing that the field is still developing.

The most interesting possibility is that microbes may influence eating behaviour indirectly rather than acting like a hidden controller of food choice. Changes in

microbial metabolism can alter the chemical environment of the gut, which may affect hormone secretion, nutrient sensing and communication with the nervous system. However, human evidence is not yet strong enough to say that a particular bacterial species will reliably make a person crave or avoid a particular food. A systematic review of 37 studies found associations between the gut microbiome and appetite, appetite hormones, food intake or weight-related outcomes in many studies, but probiotic and synbiotic findings were inconsistent.

This distinction matters because microbiome research can easily be oversimplified in popular health discussions. Eating behaviour is shaped by biology, but it is also shaped by culture, family routines, food availability, stress, sleep, learning and personal preference. The microbiome is one participant in that network, not a single master switch.

5. Homeostatic Hunger Meets Food Reward

A useful way to understand eating behaviour is to separate homeostatic and hedonic influences. Homeostatic eating is closely tied to energy needs and physiological signals that promote or reduce food intake. Hedonic eating involves pleasure, reward, attention and learned associations. In real life, these systems overlap. A person may be physiologically satisfied but still interested in dessert because its taste, smell, social meaning or previous associations activate reward-related processes.

This perspective makes 'willpower' an incomplete explanation for eating behaviour. Biological signals can change the perceived value of food, while the environment can repeatedly expose people to highly rewarding food cues. The brain must continuously decide whether a food cue is worth acting on, and that decision is influenced by both internal state and context.

6. Gut–Brain Signalling and Modern Eating Environments

Modern food environments can challenge biological systems that evolved to protect against energy shortage. Foods that combine intense sweetness, saltiness, fat, aroma and texture may be especially attention-grabbing. Large portions and constant availability can also reduce the natural boundaries between meals. The gut–brain axis does not create these environmental pressures, but it helps explain why repeated exposure can interact with physiology and learning.

Sleep and stress are additional modifiers. Poor sleep, psychological stress and irregular routines can change appetite, food choice and meal timing, making the overall system even more complex. For this reason, interventions that focus only on a single nutrient or a single hormone may overlook the broader behavioural context.

7. Factors That Can Shape Eating Behaviour Through the Gut–Brain Network

Factor	Gut-level signal	Brain/behavioural influence	Practical interpretation
Meal size/composition	Distension and nutrient sensing	Changes satiation and post-meal satiety	Meal quality and pace can influence fullness.
Dietary fibre	Fermentation and microbial metabolites	May affect satiety-related signalling	Fibre-rich foods support a broader gut environment.
Highly palatable foods	Nutrient and sensory inputs	Can engage reward and learned cues	Eating is not governed by hunger alone.
Stress	Changes gut function and appetite signalling	May alter cravings or appetite	Emotional state can modify eating patterns.
Sleep disruption	May alter endocrine/metabolic regulation	Can affect appetite and food choice	Regular sleep supports healthy appetite regulation.
Microbiota composition	Metabolites and host signalling	Potential effects on appetite pathways	Associations are promising but not deterministic.

8. Clinical and Public-Health Perspective

Understanding the gut–brain axis may broaden how clinicians and educators talk about eating behaviour. Instead of framing appetite as a simple test of self-control, it may be more helpful to consider sleep, stress, meal patterns, food environment, digestive symptoms, medications and metabolic health alongside dietary intake. This does not mean every appetite change is caused by the microbiome, nor that microbiome-targeted products are universally effective.

Gut-hormone research has already influenced modern metabolic therapeutics. GLP-1-based medicines demonstrate that manipulating physiological appetite pathways can change food intake and metabolic outcomes. At the same time, pharmacological effects should not be confused with proof that ordinary foods or supplements can reproduce

the same results. Current evidence supports continued research rather than simplistic promises.

9. What the Evidence Still Cannot Tell Us

Three questions remain particularly important. First, causality is difficult to establish: does a change in microbiota cause altered eating behaviour, or does a change in diet reshape the microbiota? In many human studies, both processes may occur at the same time. Second, individual variation is substantial. Age, genetics, medication exposure, habitual diet and health status can all influence gut microbial communities and appetite signalling. Third, animal studies are valuable for mechanism but cannot by themselves establish how a pathway operates in everyday human life. The most useful future research will combine detailed dietary records, objective metabolic measures, microbiome profiling, brain imaging and long-term behavioural follow-up.

10. A Humanized View: Eating Is a Conversation

The most important shift offered by the gut–brain axis is conceptual. Eating is not simply a message sent from the brain to the stomach, nor is it simply a response to an empty stomach. It is an ongoing conversation. The gut senses the meal, hormones carry information, nerves transmit rapid signals, microbes transform nutrients, and the brain interprets all of this alongside memories, emotions and the surrounding food environment.

Seeing eating behaviour in this way can reduce blame and increase curiosity. A person who struggles with appetite is not necessarily lacking discipline; their behaviour may reflect many biological and environmental signals arriving at the same time. That does not remove personal agency, but it places behaviour in a more realistic physiological context. The future of nutrition may therefore depend less on searching for a single perfect diet and more on understanding how whole patterns of eating, sleep, stress, activity and food environments interact with individual biology.

11. Conclusion

The gut–brain axis provides a powerful framework for understanding eating behaviour as a dynamic interaction between physiology and experience. Gut hormones such as ghrelin, GLP-1, PYY and CCK communicate information about nutrient availability and meal completion, while the vagus nerve and circulating signals connect the gastrointestinal tract with brain networks that regulate appetite, satiety and reward. The microbiota adds another layer by producing metabolites that may influence intestinal, endocrine, immune and neural pathways. Current human evidence is promising but heterogeneous, so microbiome–behaviour relationships should be interpreted

cautiously. Overall, eating behaviour is best viewed as the product of several interacting systems rather than a simple matter of hunger or willpower. A more complete understanding of these systems may support future nutritional and clinical strategies that are more individualized, biologically informed and compassionate.

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