SATIETY

Beyond Weight Management

Appetite Regulation, Food Structure and Metabolic Health

A critical review of gut–brain signaling, glycemic regulation, food architecture, body composition and healthy aging

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EDITORIAL NOTE

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Abstract

Satiety has traditionally been discussed as a mechanism for weight management: foods that produce greater fullness are expected to reduce subsequent energy intake and, ultimately, body weight. That proposition is biologically plausible, but incomplete. Satiety is not a single hormonal response, nor is it synonymous with reduced calorie intake. It emerges from the integration of sensory perception, oral processing, gastric distension and emptying, intestinal nutrient sensing, enteroendocrine signaling, postabsorptive metabolism, learned expectations, reward processing and the surrounding food environment. Consequently, the metabolic significance of a satiating food depends not only on how full a person feels, but also on how that state of satiety was generated.

This distinction has become increasingly important. Protein can enhance postprandial fullness and stimulate satiety-associated hormones such as glucagon-like peptide-1 (GLP-1) and peptide YY (PYY), yet greater subjective satiety does not invariably translate into lower total daily energy intake or improved cardiometabolic outcomes. Dietary fibre may promote fullness through volume, viscosity, gastric and intestinal mechanisms and microbial fermentation, while simultaneously exerting effects on glycemic control, lipid metabolism and cardiovascular risk that extend well beyond appetite regulation. Food texture and eating rate can materially alter energy intake even when nutrient composition is similar. Conversely, excessive satiation may be undesirable in older or nutritionally vulnerable individuals in whom preservation of energy and protein intake is a priority.

This review therefore argues that satiety should be understood as a systems-level property of foods, meals and dietary patterns rather than as a surrogate endpoint for weight loss. The most relevant future applications lie at the intersection of appetite regulation, postprandial metabolic control, nutrient density, preservation of lean mass, food structure and long-term dietary adherence. In the era of GLP-1-based pharmacotherapy, this broader perspective becomes particularly relevant: nutritional strategies should not attempt simply to reproduce pharmacological appetite suppression, but to engineer better metabolic nutrition around adequate protein, fibre, food structure, micronutrient density and preservation of functional tissue.

Keywords satiety; satiation; appetite; metabolic health; protein; dietary fibre; GLP-1; PYY; glycemic control; food matrix; eating rate; healthy aging; body composition; functional foods

CENTRAL THESIS

Satiety is not a surrogate for weight loss. Its health relevance depends on the physiological route by which it is generated, the nutritional quality of the meal, the population in which it occurs and the behavioural outcome that follows.

Key messages

- Satiation (meal termination) and satiety (post-meal suppression of hunger) are related but distinct physiological constructs.
- Greater subjective fullness does not necessarily reduce total daily energy intake, body weight or cardiometabolic risk.
- Protein, fibre, food structure, energy density, viscosity and eating rate can influence satiety through different and partly independent mechanisms.
- The metabolic benefits of protein- and fibre-rich foods cannot be reduced to their effects on appetite alone.
- In older, frail or pharmacologically appetite-suppressed populations, maximizing satiety may be nutritionally counterproductive.
- Future product development should focus on “satiety architecture”: the integrated design of nutrient density, food structure, digestive kinetics and eating behaviour.

1. Introduction: satiety has been asking the wrong question

For decades, nutritional discussions surrounding satiety have tended to begin with an apparently straightforward question: Will this food make people eat less? From a weight-management perspective, the logic is compelling. If a food increases fullness after consumption, delays the return of hunger and reduces subsequent energy intake, repeated exposure might facilitate a negative or less positive energy balance. This concept has underpinned a substantial body of research into high-protein diets, viscous fibres, low-energy-density foods and meal replacements.

But there is a conceptual problem. Satiety is a physiological state; weight change is a long-term outcome. They are related, but they are not interchangeable.

The distinction is not semantic. Controlled studies repeatedly demonstrate that subjective appetite ratings and actual energy intake can dissociate. A meal can make participants report feeling significantly fuller without producing a meaningful reduction in food intake later in the day. In the NewStart randomized trial, for example, a higher-protein breakfast produced greater reported satiety than a low-protein breakfast, yet this did not translate into significant differences in energy intake, body composition, lipid profile or glucose tolerance over the intervention period (Dalgaard et al., 2025).

That observation fundamentally changes how satiety should be interpreted. Rather than treating fullness as a proxy for weight loss, the scientifically more useful question is: what physiological, nutritional and behavioural processes are being modified when a food generates satiety, and what consequences do those processes have for health?

Once the problem is formulated in this way, satiety becomes much larger than weight management. It intersects with postprandial glycemia, insulin secretion, gastrointestinal physiology, food choice, meal frequency, protein adequacy, preservation of skeletal muscle, dietary fibre intake, gastrointestinal microbial metabolism and the physical structure of foods. Some of those processes may contribute to weight control. Others have metabolic value even when body weight changes little or not at all.

At the same time, greater satiety is not universally desirable. An older adult with declining appetite, a patient at risk of malnutrition or an individual struggling to consume sufficient protein represents a very different physiological problem from an otherwise healthy person living in a highly obesogenic food environment. In the former situation, excessive satiation may be counterproductive. Satiety should therefore not be treated as an inherently beneficial property. It is beneficial when the magnitude, timing and nutritional consequences of the response are appropriate for the individual and the metabolic objective.

2. Satiation is not satiety

A rigorous discussion must begin by separating two concepts that are frequently used interchangeably. Satiation describes the processes occurring during a meal that contribute to meal termination. It influences how much food is consumed before eating stops. Satiety, in contrast, describes the suppression of hunger and eating after a meal has ended. It influences the interval before the next eating episode and potentially the amount consumed subsequently.

This distinction is central to modern appetite research. Methodological frameworks developed for the evaluation of foods specifically distinguish effects on meal termination from effects on post-meal appetite and subsequent consumption (Blundell et al., 2010).

A food could therefore produce strong satiation by slowing eating or occupying considerable gastric volume without generating unusually prolonged post-meal satiety. Conversely, a relatively modest meal might produce prolonged metabolic or enteroendocrine signals that delay subsequent hunger.

The traditional satiety cascade conceptualizes appetite control as a sequence of overlapping processes. Before nutrients are absorbed, visual, olfactory, cognitive and oral sensory information already modifies expectations about fullness. During consumption, chewing, texture, eating rate and gastric filling provide additional information. Once nutrients reach the intestine, receptors and enteroendocrine cells detect carbohydrates, amino acids, peptides, fatty acids and other components. After absorption, circulating nutrients and metabolic signals continue to influence the central nervous system.

The result is not an on/off signal. Satiety is an integrated biological computation. This helps explain why measuring a single hormone—or even several hormones—is insufficient to characterize the satiating effect of a food.

3. The gut–brain architecture of appetite regulation

3.1 Gastric signals: volume matters, but volume is not everything

As food enters the stomach, gastric distension activates mechanosensitive pathways that contribute to sensations of fullness. Gastric emptying subsequently determines how rapidly nutrients are delivered to the small intestine and therefore influences both gastric distension and intestinal nutrient sensing.

Slower gastric emptying has often been associated with prolonged fullness. GLP-1 and several other gastrointestinal peptides can slow gastric emptying, providing one plausible mechanism by which gut-derived signals influence meal size and post-meal appetite (Hellström & Näslund, 2001).

Yet it would be an oversimplification to state that slower gastric emptying equals greater satiety. Appetite regulation arises from multiple interacting signals rather than a single gastric mechanism. This is important for food formulation: increasing viscosity, volume or gastric residence time may influence appetite, but none guarantees reduced energy intake independently of sensory properties, nutrient delivery and later compensatory eating.

3.2 Enteroendocrine signaling

The intestinal tract is a major endocrine organ. Nutrient exposure stimulates release of several peptides involved in appetite regulation, including cholecystokinin (CCK), GLP-1 and PYY. Ghrelin, generated predominantly in the stomach and strongly associated with hunger signaling, generally falls following food consumption.

GLP-1 occupies a particularly important position because it links nutrient ingestion with glucose-dependent insulin secretion, gastric physiology and central regulation of food intake. Its physiological actions are far broader than satiety alone (Müller et al., 2019).

Protein-rich meals provide a useful example of this integrated response. Human experiments have shown higher postprandial GLP-1 and PYY concentrations following high-protein meals compared with macronutrient-matched alternatives, while meta-analysis indicates that acute protein ingestion can suppress appetite, reduce ghrelin and increase CCK and GLP-1. Importantly, longer-term intervention evidence is much less definitive. Again, physiology and clinical outcome should not be conflated. An acute increase in GLP-1 is evidence of a biological response; it is not evidence, by itself, of long-term weight reduction.

3.3 The ileal and colonic contribution

Satiety signaling does not end in the proximal small intestine. When nutrients or fermentable substrates reach more distal regions of the gastrointestinal tract, additional endocrine and neural pathways can be activated.

Dietary fibres are particularly interesting because their physiological effects depend strongly on their physicochemical characteristics. Viscosity can modify gastric emptying and nutrient diffusion. Fermentability

determines the extent to which intestinal microorganisms convert substrates into metabolites including short-chain fatty acids. These metabolites may, among other effects, interact with enteroendocrine pathways involved in GLP-1 and PYY secretion (Akhlaghi, 2024).

However, the term fibre describes a heterogeneous group of compounds rather than a single biological intervention. Systematic reviews show substantial variability in appetite outcomes depending on fibre type, dose, viscosity, fermentability, food vehicle and experimental methodology (Clark & Slavin, 2013). The scientifically defensible statement is therefore not simply that fibre increases satiety, but rather that specific fibres, delivered in appropriate matrices and quantities, may modify several mechanisms involved in appetite regulation.

4. Satiety beyond caloric restriction

The strongest argument for looking beyond weight management arises from the observation that many dietary characteristics associated with increased satiety also affect metabolism through pathways that are partly independent of appetite. The challenge is to distinguish correlation from causation.

A high-fibre food may increase fullness and improve postprandial glycemic control. It would be tempting to say that satiety improved glycemic control. Usually, that is not what happened. Rather, the fibre produced both effects through overlapping but distinct physiological mechanisms.

A more accurate model is therefore that satiety is often one visible manifestation of a broader metabolic response to the physical and nutritional characteristics of food.

5. Postprandial glycemia: where metabolic health separates from appetite

Postprandial metabolism is increasingly recognized as an important dimension of metabolic health. The speed with which digestible carbohydrate becomes available, the composition and order of the meal, fibre viscosity, gastric emptying and the physical structure of the carbohydrate source can all alter glucose appearance in the circulation.

Higher-quality carbohydrate patterns characterized by greater dietary fibre and whole-grain intake have been associated in systematic reviews and intervention evidence with better cardiometabolic outcomes. A major series of systematic reviews and meta-analyses found substantial health advantages associated with higher fibre and whole-grain consumption (Reynolds et al., 2019). In people with diabetes, higher fibre intake has also been associated with improvements in several measures of glycemic control and cardiometabolic risk (Reynolds et al., 2020).

Some of the same fibres can enhance fullness. But the metabolic value should not be attributed exclusively—or even primarily—to feeling full. Viscosity may slow nutrient accessibility. Whole-grain structures may alter digestion kinetics. Fermentation may modify colonic metabolism. Replacing refined carbohydrates with fibre-rich foods changes the overall nutrient matrix. Satiety can accompany these changes without being their sole causal mediator.

6. Protein: perhaps the clearest example of why satiety is bigger than weight loss

Protein is generally considered the macronutrient with the greatest acute satiating capacity, although effect size varies substantially between studies, populations, protein sources and experimental conditions. Reviews and meta-analyses support an overall enhancement of fullness and suppression of appetite following higher-protein meals.

Its relevance, however, extends far beyond appetite. Dietary protein provides indispensable amino acids and supports skeletal muscle protein turnover. During energy restriction, preserving lean tissue becomes particularly important because weight loss does not consist solely of adipose tissue.

A recent systematic review and meta-analysis concluded that enhanced protein intake can materially improve preservation of muscle mass during intentional weight loss in adults with overweight or obesity (Kokura et al., 2024). This creates an intriguing nutritional intersection. A properly formulated higher-protein meal may simultaneously increase meal-related satiety, improve protein adequacy, support retention of lean tissue during energy restriction and reduce the fraction of the diet occupied by foods providing substantial energy but limited nutritional value.

These outcomes should not be collapsed into a single mechanism. The preservation of lean tissue is biologically valuable even if appetite changes are modest. Conversely, a strong satiety response is not necessarily useful if it suppresses total protein or micronutrient intake.

7. Satiety and healthy aging: a necessary paradox

The phrase satiety-enhancing nutrition usually sounds beneficial. In healthy aging, however, the situation is more complicated. Older adults are at increased risk of declining appetite and insufficient energy or protein intake. Altered gastrointestinal physiology, delayed gastric emptying, changes in hormonal responses, sensory decline, medication use, disease and psychosocial factors can all contribute to what has historically been termed the anorexia of aging.

At the same time, older adults may require particular attention to protein intake because anabolic responsiveness of skeletal muscle can decline with age. Protein therefore creates a nutritional paradox: it is nutritionally valuable for muscle preservation but also strongly satiating.

The implication is profound. There is no universally optimal level of satiety. For an individual with obesity and frequent snacking, prolonged satiety may be advantageous. For a frail older adult struggling to consume adequate calories and protein, the same degree of satiety could be undesirable. Future functional nutrition therefore needs to move away from the simplistic objective of maximizing satiety toward the more sophisticated objective of optimizing appetite in relation to nutritional need.

8. Fibre: fullness, glycemic physiology and cardiovascular relevance

Dietary fibre may represent the clearest example of a food component whose benefits extend far beyond appetite. Different fibres can influence stool physiology, gastrointestinal transit, nutrient absorption, fermentation, microbial ecology, bile-acid metabolism, glycemic response and lipid metabolism. Their capacity to influence satiety depends strongly on properties such as viscosity, hydration, gel formation, fermentability and integration into the food matrix.

The broader health literature is persuasive. Systematic analyses consistently associate higher fibre exposure with improved health outcomes, including lower cardiometabolic risk, while intervention evidence in individuals with diabetes demonstrates improvements in glycemic parameters and several cardiovascular risk markers.

This creates an important distinction for food science. A fibre ingredient may be selected because it increases viscosity and fullness. But its value should ideally be evaluated across appetite, postprandial glycemia, gastrointestinal tolerance, fermentation, lipid metabolism, dietary displacement and long-term adherence. A food producing spectacular short-term fullness but poor gastrointestinal tolerance is unlikely to be successful

nutritionally or commercially. Likewise, an isolated fibre cannot automatically be assumed to reproduce the physiological effects observed in epidemiological studies of fibre-rich dietary patterns. Food context matters.

9. Food structure: calories do not arrive in the body as numbers

Traditional nutritional models frequently reduce foods to nutrient composition: grams of carbohydrate, protein, fat and fibre per serving. Human eating behaviour does not operate on a nutrition label. Foods have structure. They must be bitten, chewed, lubricated, swallowed, disintegrated and digested. Their physical characteristics determine how rapidly energy can be consumed and how quickly nutrients become accessible to digestive enzymes.

This has important implications for satiety. Controlled research shows that manipulating food texture can alter eating rate and energy intake even when foods are otherwise broadly comparable nutritionally. In a randomized crossover study, texture-driven differences in eating rate materially changed energy intake across both minimally processed and ultra-processed meals (Teo et al., 2022).

Eating rate therefore deserves to be considered a metabolic property of food design. A rapidly consumed food allows substantial energy to enter the gastrointestinal tract before the full set of oral, gastric and intestinal feedback mechanisms has developed. A food requiring greater oral processing extends sensory exposure per calorie and gives satiation signals more time to interact with ongoing consumption. This does not mean that every soft food is metabolically undesirable or every hard food beneficial. But texture is not merely a sensory attribute; it can influence ingestive behaviour.

10. The ultra-processed food lesson

One of the most influential modern demonstrations of the importance of food structure came from the controlled inpatient trial conducted by Hall and colleagues. Participants were exposed to ultra-processed and unprocessed diets under tightly controlled conditions. Despite the diets being designed to match several presented nutritional characteristics, the ultra-processed condition resulted in substantially greater spontaneous energy intake and weight gain (Hall et al., 2019).

This experiment should not be interpreted as proving that all ultra-processed foods cause overeating, nor that processing level alone explains the effect. Instead, it demonstrates something more nuanced and scientifically useful: nutrient composition on paper is not sufficient to predict eating behaviour. Energy density, eating rate, texture, palatability, food form and the accessibility of nutrients can alter the way calories are consumed.

For the food industry, the implication is potentially transformative. The next generation of metabolically intelligent foods should not be designed only by changing ingredient lists. They should be engineered at the level of food architecture.

11. Energy density, volume and the physiology of “enough”

Humans eat food by weight and volume as well as by energy. Foods containing substantial water, intact plant structures or other low-energy-density components can provide considerable oral and gastric volume for relatively modest energy delivery. This creates one of the most practical pathways through which satiety can contribute to energy regulation.

But there is an important difference between reducing energy density by incorporating physiologically meaningful foods and merely replacing nutrients with noncaloric bulk. The metabolic value of vegetables, legumes, fruit, whole grains or high-quality protein-containing foods cannot be reduced to their effects on gastric volume. Their nutrient density and physiological characteristics matter simultaneously. Thus, the ideal

formulation is not simply one that occupies the stomach. It is one that makes the calories consumed nutritionally worthwhile.

12. Satiety and diet quality: the neglected behavioural pathway

Another route by which satiety may influence metabolic health is behavioural rather than purely endocrine. Repeated hunger between meals can encourage additional eating opportunities. In modern food environments, those opportunities frequently involve readily available energy-dense foods. A meal pattern that produces an appropriate degree of sustained fullness may therefore reduce the behavioural pressure to snack.

Yet this outcome is far from automatic. Humans eat for reasons other than homeostatic hunger. Reward, habit, stress, environmental cues, social context and food availability can override gastrointestinal satiation and satiety signaling. This is one reason why laboratory measurements of hunger do not always predict real-world eating.

Satiety should therefore be viewed as one component of appetite control rather than the final arbiter of food intake. The practical objective should be to reduce unnecessary physiological hunger without pretending that physiology can eliminate the psychological and environmental dimensions of eating.

13. The GLP-1 era changes the conversation

The extraordinary clinical impact of GLP-1 receptor agonists and related incretin therapies has made appetite biology visible to a much wider audience. GLP-1 biology demonstrates that appetite regulation, gastric physiology and glucose metabolism are deeply interconnected. Physiological GLP-1 contributes to glucose-dependent insulin secretion, gastrointestinal regulation and appetite control, while pharmacological receptor stimulation can produce effects far larger and more sustained than those generated physiologically by ordinary foods (Müller et al., 2019).

This distinction is crucial. A yoghurt, protein drink, fibre ingredient or structured meal should not be described as a nutritional equivalent of pharmacological GLP-1 receptor agonism simply because it produces an acute increase in endogenous GLP-1. Physiological secretion and pharmacological receptor activation differ substantially in magnitude, duration, pharmacokinetics and systemic effects.

The food industry's legitimate opportunity lies elsewhere. Rather than trying to create a fictional “natural Ozempic”, food science can design nutrition that works intelligently around human appetite physiology.

14. Satiety during pharmacological weight loss: the emerging nutritional problem

The rise of potent anti-obesity pharmacotherapy has also exposed a second issue. When energy intake declines substantially, nutritional quality becomes more—not less—important. Weight loss inevitably includes some loss of fat-free tissue. This does not mean that GLP-1 therapies uniquely destroy muscle; loss of some lean tissue accompanies substantial weight loss through multiple approaches.

It does mean that the objective of obesity treatment should not be reduced to making the number on the scale fall. The emerging clinical objective is better described as fat loss with preservation of metabolically and functionally valuable tissue.

Here, nutritional product development has an important role. Foods designed for people experiencing pharmacologically reduced appetite may need to combine high nutritional density, adequate high-quality protein, appropriate fibre, micronutrient adequacy, good gastrointestinal tolerance and manageable portion

sizes. Under these circumstances, maximizing satiety would be the wrong formulation goal. The consumer is already experiencing strong appetite suppression. The challenge becomes delivering sufficient nutritional value before satiation terminates the meal.

15. Why “high protein” does not automatically mean metabolically superior

Protein deserves further qualification. Higher-protein diets can increase fullness and assist preservation of lean mass during weight loss. Yet protein source, accompanying nutrients and the food replacing or being replaced by the protein all matter.

Thirty grams of protein cannot be evaluated independently of its food matrix. A protein-rich legume meal introduces fibre, resistant starch, minerals and plant bioactives. A dairy matrix introduces calcium and other nutrients. A highly processed protein-containing snack may contain very different amounts of fat, sodium, fibre or energy.

Consequently, comparisons based only on protein grams risk missing the actual nutritional substitution occurring within the diet. This is particularly relevant to commercial product development. A claim such as “high protein for satiety” addresses only one dimension of the food. The more sophisticated objective is protein adequacy within an overall metabolically appropriate matrix.

16. The same problem applies to fibre

Adding an isolated fibre to an otherwise rapidly consumed, energy-dense product does not necessarily transform the product into the metabolic equivalent of an intact fibre-rich food. The food matrix determines hydration, viscosity, mastication, nutrient accessibility and gastrointestinal delivery.

Systematic reviews of fibre and appetite show precisely why simple ingredient-level generalizations fail: effects are highly heterogeneous across fibre types and experimental conditions (Clark & Slavin, 2013). This does not diminish the value of functional fibres. It means they need to be selected rationally, and their physicochemical characteristics should match the intended physiological outcome.

17. A more useful concept: Satiety Architecture

The accumulated evidence supports a broader concept that may be more useful than the traditional search for a single satiety ingredient. That concept is Satiety Architecture.

Satiety Architecture describes the combined effect of nutrient composition, protein quality and quantity, fibre chemistry, food volume, energy density, viscosity, physical structure, oral processing requirements, eating rate, gastric behaviour, intestinal nutrient release, sensory characteristics and the temporal pattern of consumption.

None of these variables operates independently. A well-designed food can use several simultaneously. For example, a metabolically intelligent meal may combine a meaningful protein fraction with viscous or structurally intact fibre, moderate energy density and a texture that discourages extremely rapid consumption. The advantage of this systems approach is that no exaggerated biological claim is required. The physiological burden is shared among several modest mechanisms rather than being attributed to a single fashionable ingredient.

PROPOSED FRAMEWORK

SATIETY ARCHITECTURE = nutrient composition + food matrix + physical structure + oral processing + digestive kinetics + endocrine response + eating behaviour + population-specific nutritional need.

18. Satiety per calorie versus satiety per gram of nutrition

One of the most promising conceptual shifts would be to stop asking which foods produce the greatest fullness and instead ask: what nutritional value is delivered for each unit of energy required to achieve comfortable satiety?

This produces a fundamentally different formulation philosophy. A food containing high-quality protein, fibre and micronutrients might generate moderate but sustained fullness while supplying meaningful nutrition. Another product might generate strong gastric fullness through volume alone but provide relatively little nutritional value. From a metabolic-health perspective, these are not equivalent.

The future of satiety research should therefore integrate nutrient density with appetite outcomes.

19. Subjective satiety is useful—but it is not enough

Visual analogue scales remain valuable tools for appetite research. They allow participants to report hunger, fullness, desire to eat and prospective consumption across time. But these measurements capture perception; they do not measure behaviour directly.

Methodological guidance in appetite research has long emphasized the importance of distinguishing subjective appetite ratings from subsequent food consumption (Blundell et al., 2010). This issue is more than methodological housekeeping. If a test product produces a statistically significant improvement in fullness but participants eat exactly the same amount later, the biological effect may be real but its practical significance for energy regulation is uncertain.

Likewise, a reduction in energy consumed at the next meal does not prove a reduction in 24-hour intake because compensation may occur later. And a one-day reduction in intake does not establish sustained weight loss. The hierarchy of evidence therefore matters.

20. Measuring satiety properly

A robust evaluation of a satiety-oriented food should ideally distinguish at least four levels of evidence.

The first is mechanistic evidence: gastric behaviour, gastrointestinal hormones, glycemia or other physiological responses. The second is subjective appetite: hunger, fullness and desire to eat. The third is behaviour: meal size, subsequent ad libitum consumption, eating frequency and total daily energy intake. The fourth is chronic outcome: sustained dietary intake, body composition and relevant metabolic parameters.

Each level answers a different question. The mistake occurs when evidence from one level is used to claim an outcome from another. A rise in GLP-1 does not prove weight loss. Greater fullness does not prove reduced calorie intake. Reduced calorie intake at lunch does not prove lower daily intake. Lower daily intake over several days does not prove sustained fat loss. And weight loss does not necessarily demonstrate improved diet quality. This hierarchy should be explicit in both academic research and product communication.

Evidence level	Primary measurements	What it can support
1. Mechanistic	Hormones, gastric emptying, glycemia	Biological plausibility
2. Subjective appetite	Hunger, fullness, desire to eat	Perceived appetite response
3. Behavioural	Meal size, ad libitum intake, daily intake	Actual eating behaviour
4. Chronic outcomes	Body composition, diet quality, metabolic markers	Long-term clinical relevance

21. Regulatory science has reached the same conclusion

Regulatory thinking in Europe reflects this scientific distinction. EFSA guidance addressing appetite ratings, energy intake, weight management and blood-glucose responses treats these as separate claimed physiological effects requiring appropriate substantiation rather than assuming that evidence for one automatically establishes another. Human intervention evidence, appropriate comparators and relevance of study duration are central considerations.

The importance of this framework extends beyond regulatory compliance. It represents good scientific reasoning. A company demonstrating greater fullness after consuming a product should not automatically communicate weight loss. A product reducing postprandial glycemic response should not automatically claim improved long-term glycemic control. A product stimulating endogenous GLP-1 should certainly not imply pharmacological equivalence to a GLP-1 receptor agonist.

Scientific precision is not an obstacle to innovation. It is what makes innovation defensible.

22. Designing the next generation of satiating foods

The most promising functional foods will probably not be those producing the largest possible appetite suppression. They will be those producing the right amount of satiety for the right population while improving the nutritional quality of the calories that remain.

For weight management, this might mean meals providing adequate protein and fibre, lower energy density and a physical structure that reduces rapid consumption. For metabolic health, emphasis might shift toward slowly available carbohydrate, viscous fibre, protein quality and food matrices that moderate postprandial nutrient delivery. For healthy aging, the strategy may need to be almost the reverse: nutrient-dense foods that deliver substantial high-quality protein and micronutrients without producing excessive early satiation. For consumers undergoing GLP-1-based treatment, small-volume, nutrient-dense, high-protein foods with appropriate fibre and excellent tolerability may become increasingly relevant.

There is therefore no single “satiety food”. There are different nutritional problems requiring different appetite profiles.

23. Personalization will matter

Individual variability in appetite responses is substantial. Body composition, sex, age, habitual diet, metabolic phenotype, sleep, physical activity, medication, gastrointestinal physiology and learned eating behaviour can all modify responses to foods. This variability partly explains why mean effects observed in controlled trials do not necessarily predict what every individual will experience.

Future satiety science is therefore likely to move toward phenotyping. Instead of asking whether protein or fibre is satiating on average, researchers may increasingly ask: who responds, under what metabolic circumstances, at what time of day, within which food matrix, and with what downstream consequence? This is a far more sophisticated research question than the historical search for universally satiating ingredients.

24. The conceptual error of maximizing a biomarker

Nutrition science repeatedly encounters the temptation to optimize what is easy to measure. If GLP-1 is associated with satiety, maximize GLP-1. If viscosity delays gastric emptying, maximize viscosity. If fullness predicts lower intake, maximize fullness. Biological systems rarely behave so simply.

A biomarker is useful only to the extent that it meaningfully represents the outcome of interest. This is particularly relevant to satiety because several physiological processes that suppress appetite can become undesirable when excessive. Nausea can reduce food intake. Severe gastric distension can reduce food intake. Illness can reduce food intake. None represents desirable nutritional satiety.

The objective therefore cannot be appetite suppression at any cost. The objective must remain health.

25. From “eat less” to “eat appropriately”

Perhaps the most important evolution in this field is linguistic. Traditional weight-management nutrition asks consumers to eat less. A metabolic-health framework asks them to eat appropriately.

Appropriate eating means consuming sufficient—not excessive—energy, adequate protein, adequate fibre and micronutrients, and foods whose physical and nutritional characteristics support physiological regulation rather than continually challenging it. Satiety is useful because it can make appropriate eating easier. But it is the means, not the endpoint.

26. Implications for the food and nutraceutical industries

This broader interpretation creates substantial opportunities for innovation, but it also raises the scientific bar. The first generation of satiety products often relied on a relatively simple proposition: add protein or fibre, demonstrate greater fullness, and position the product for weight control. The next generation should be more ambitious.

Development programs should integrate food technology, gastrointestinal physiology, nutrition, metabolic endpoints and consumer behaviour from the beginning. Formulation should investigate not simply what ingredients are present, but how quickly the product is consumed, how it hydrates, how its structure changes during mastication, how nutrients are released, how the matrix behaves in the stomach, how glucose and insulin respond, whether the fibre is fermented, whether gastrointestinal tolerance is acceptable, whether the consumer compensates at later meals, and whether the product improves the nutritional composition of the diet over time.

That is a fundamentally different R&D philosophy.

27. Satiety as a property of systems, not ingredients

No single nutrient controls human appetite. Protein matters. Fibre matters. Energy density matters. Texture matters. Eating rate matters. Gastric mechanics matter. GLP-1, PYY, CCK and ghrelin matter. Reward matters. Environment matters. But their effects occur simultaneously.

The enduring lesson from decades of satiety research is therefore not the discovery of one dominant mechanism. It is the realization that appetite regulation is distributed across a biological system. Food design should reflect that complexity.

28. A critical note on causality

The statement that greater satiety improves metabolic health should be used cautiously. There is little justification for treating subjective satiety itself as an independent causal determinant of cardiometabolic health.

A more defensible formulation is: foods and dietary patterns that generate appropriate satiety frequently possess nutritional and structural characteristics that can also improve metabolic health. Satiety may facilitate

adherence and energy regulation, while the underlying nutrients and food matrix exert additional metabolic effects.

This distinction prevents a common causal error. Fibre is not healthy because it makes people full. Protein is not metabolically valuable because it suppresses appetite. Whole foods are not advantageous solely because they are eaten more slowly. These characteristics interact, but they retain biological effects independent of appetite perception.

29. Conclusions

Satiety deserves to be rescued from the narrow conceptual territory of weight-loss marketing. It is one expression of a far more complex physiological system linking sensory perception, oral processing, gastrointestinal mechanics, endocrine signaling, nutrient metabolism, brain function and eating behaviour.

Increasing satiety can support weight management, but the relationship is neither linear nor guaranteed. Acute fullness can occur without reductions in daily energy intake, body weight or cardiometabolic risk. The greater opportunity lies elsewhere.

Protein-rich foods may combine appetite regulation with preservation of lean tissue. Appropriately selected dietary fibres may combine fullness with improved glycemic and cardiovascular physiology. Food texture and structure can slow eating and alter spontaneous energy consumption independently of simple nutrient labeling. And in older or nutritionally vulnerable populations, excessive satiation may be counterproductive, demonstrating that appetite suppression cannot itself be considered an unconditional nutritional benefit.

The GLP-1 era reinforces rather than overturns these principles. Powerful pharmacological appetite suppression makes nutritional quality and preservation of lean tissue increasingly important, not less important.

The future objective should therefore not be to formulate foods that make people eat as little as possible. It should be to create foods that allow people to become comfortably satisfied after consuming the amount and type of nutrition that best supports their physiology. That difference may sound subtle. It represents a profound change in nutritional thinking.

Satiety is bigger than weight management because human metabolism is bigger than body weight.

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