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Macronutrient Balance as a Determinant of Sustainable Eating Behavior and Sugar Craving Regulation

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Abstract

Added sugar accounts for a large share of contemporary food environments, and public guidance tends to frame the problem around a single ingredient and personal discipline. Both frames leave gaps in the mechanism. This work synthesizes evidence on how the structure of a diet, meaning the balance of protein, fiber, fat quality, and carbohydrate quality, shapes the glycemic and satiety response and, through that response, the intensity of sugar cravings and the durability of eating behavior. Peer-reviewed studies, systematic reviews, meta-analyses, and official guidance were retrieved from major databases and organized by thematic block. The synthesis shows that identical carbohydrate loads produce divergent blood glucose responses depending on food matrix, fiber content, protein, fat, and degree of processing. Protein and viscous fiber extend satiety and blunt glucose peaks through gut hormone signaling. Cravings emerge from dopamine-linked reward pathways and habit loops shaped by the environment. Sleep restriction and chronic stress move appetite hormones toward hunger and toward the seeking of energy-dense food. An integrative model links macronutrient balance to a smoother glycemic and hormonal response, weaker craving, and eating patterns that hold without reliance on constant self-control, with sleep and stress acting as modulators of the entire loop. Reading cravings as biological phenomena lowers self-blame and shifts attention to meal composition and daily rhythm. The contribution of this synthesis lies in combining glycemic physiology, food structure, reward neurobiology, and lifestyle modifiers within one conceptual framework, a set of domains that earlier reviews have examined in isolation. The model provides a testable account of personalized nutrition and longitudinal work on dietary sustainability.

Keywords: macronutrient balance, sugar cravings, glycemic response, satiety, appetite hormones, eating behavior, sleep, stress

1. Introduction

Free sugars account for a growing share of daily energy intake in industrialized food supplies. Guidance from the World Health Organization sets a ceiling of 10% of total energy from free sugars for adults and children, with added benefit below 5% (World Health Organization, 2015). Large-scale evidence connects high sugar intake with cardiometabolic disease across a wide span of outcomes (Y. Huang et al., 2023). Public messaging built on these findings often narrows the issue into a pair of familiar claims. One version treats sugar as a uniform hazard to be removed. Another version treats craving as a failure of willpower.

Each version captures a fragment and misses the mechanism. Sugar within a whole fruit moves through digestion along a slower route than sugar released from a soft drink, because fiber, water, and micronutrients change absorption. The number on a label describes quantity, and it says little about how the body will meet that quantity. Craving intensity shifts with sleep, stress, meal timing, and the reward value of available food, which places much of the experience outside conscious control. A frame that reduces the problem to an

ingredient or to a character strength cannot account for these moving parts.

Dietary architecture offers a fuller frame. The balance of protein, fiber, fat quality, and carbohydrate quality shapes how quickly glucose enters the blood and how long fullness lasts, and that pair of responses feeds back into craving and the stability of eating patterns. Craving reads as an output of biology and habit, shaped by hormones and by environment. Food quality and meal structure carry weight that a single nutrient count cannot capture, since the same gram of sugar behaves along different paths depending on the food that carries it.

Relevant evidence has grown along separate channels. Glycemic response, satiety signaling, reward neurobiology, and the influence of sleep and stress each carry a mature body of work, while an integrated account aimed at the durability of eating behavior stays thin across the available sources. Reviews of glycemic control seldom incorporate reward neurobiology, work on craving seldom addresses food structure, and evidence on sleep and stress sits apart from both, so no framework has yet linked meal composition to craving and to long-term adherence through one causal chain. This gap leaves parallel explanations that share no common variable and combine into no single line of guidance. The aim of the analysis is to draw these streams into a single model. The work is organized around 3 tasks. The first task examines how food structure and macronutrient balance shape the glycemic and satiety response. The second task reviews the neurobiology of craving. The third task weighs sleep and stress as modulators of that system. The scientific contribution of the analysis lies in the conceptual integration of these domains, since the model specifies how meal composition reaches craving through defined physiological steps and where lifestyle factors enter that sequence. The practical context includes popular sugar-reduction programs, where reports of shifts in energy, sleep, and craving accompany changes in meal structure, and the argument below rests on peer-reviewed evidence.

Conceptual Contribution of This Review

Unlike previous reviews that examine glycemic control, appetite regulation, or food reward independently, this review proposes an integrated conceptual framework linking macronutrient balance, food structure, neurobiology, appetite hormones, and lifestyle modifiers into a unified model of sustainable eating behavior. By synthesizing these previously separate domains, the review provides a mechanistic explanation for how meal composition influences sugar cravings and long-term dietary adherence.

2. Methods

The work follows a narrative review design with a structured search component. Literature came from PubMed, MEDLINE, Scopus, Web of Science, and Google Scholar. Search terms combined sugar cravings, macronutrient balance, glycemic index, satiety, leptin, ghrelin, glucagon-like peptide-1, food matrix, sleep restriction, and stress eating, joined through Boolean operators. Reference lists of retrieved reviews were scanned for additional sources that met the criteria. The search identified 96 records across the 5 databases, and removal of duplicates followed by screening of titles and abstracts reduced the pool to 13 sources that entered the synthesis.

Inclusion covered peer-reviewed original studies, systematic reviews, meta-analyses, monographs, and official documents from health authorities such as the World Health Organization. Priority went to work published between 2021 and 2026, with a few foundational older studies kept where they anchor a concept. Exclusion removed non-peer-reviewed web content and social media, including video platforms and short-form clips, because such material lacks the vetting expected for an academic reference base. Screening favored human data where available, with animal models cited where they establish a mechanism. Study quality was not appraised through a formal risk-of-bias instrument, and the evidence was synthesized qualitatively, with

greater weight given to systematic reviews, meta-analyses, and randomized designs than to observational reports.

Synthesis proceeded by thematic grouping. Retrieved sources were sorted into blocks covering food structure and glycemic response, macronutrient balance and satiety, reward neurobiology, degree of food processing, and lifestyle modulators. Within each block, findings were compared for direction and consistency, and points of disagreement were noted for discussion. The narrative format supports integration across disciplines that seldom share journals, from food science to behavioral neuroscience to sleep medicine. That breadth comes with a trade-off in systematic completeness. A narrative design remains susceptible to selection bias, since the choice of sources reflects reviewer judgment, and the search made no claim to capture every eligible publication. The effect directions reported below reflect the weight of the retrieved evidence, and individual variation in glycemic and hormonal responses is treated as a boundary condition throughout.

3. Results

3.1. Food matrix and the glycemic response

Carbohydrate quantity alone is an incomplete predictor of blood glucose. Foundational work on the glycemic index established that foods carrying the same carbohydrate load can drive divergent blood glucose responses, with structure, fiber, protein, fat, and processing all shaping the curve (Jenkins et al., 1981). Sugar embedded in an intact food matrix travels through digestion along a slower route than sugar freed from a refined product. This early insight moved the field beyond viewing carbohydrate as a single quantity and toward a view of carbohydrate as a delivery problem, where timing and packaging matter as much as amount.

Food form, texture, and matrix govern oral processing, gastric emptying rate, and nutrient absorption rate (Forde & Bolhuis, 2022). A whole fruit requires chewing, provides fiber and water, and releases its sugars over time. A blended or refined version of the same carbohydrate content clears the stomach faster and produces a sharper rise in blood glucose. Particle size, cell-wall integrity, and starch structure all enter this calculation, since intact plant cells shield their contents from rapid enzymatic attack. When processing ruptures those cells and grinds the material fine, the same sugars reach the bloodstream on a compressed schedule.

The healthfulness of a food tracks its structure alongside its nutrient totals. A soft, energy-dense, fast-clearing product asks little of the digestive system and delivers a steep glucose load, while a firm, fiber-rich, water-heavy food spreads that load across a longer window. This distinction reframes sugar as a component whose behavior depends on its surroundings, and it explains why two foods with matched sugar content can produce opposite metabolic signatures. The glycemic response then becomes a readout of whole-food architecture, and it sets the stage for the satiety effects that follow.

3.2 Fiber, macronutrient balance, and satiety

Fiber reshapes the postprandial curve through physical and hormonal routes. A systematic review of randomized crossover trials found that fiber added to starchy foods lowers the glycemic and insulinemic response in the hours after a meal, with viscous soluble fibers showing the strongest effect (Tsitsou et al., 2023). Viscosity slows gastric emptying and forms a barrier to glucose diffusion at the intestinal wall, which flattens the glucose peak and softens the insulin demand that follows. Insoluble and fermentable fibers add a further layer, since gut microbes convert them into short-chain fatty acids that feed back onto appetite and metabolism.

Protein anchors satiety from a different angle. The protein leverage framework treats protein appetite as a driver that organizes total intake, since animals and people adjust how much they eat to reach a protein target (Raubenheimer & Simpson, 2023). A meal low in protein can push overall consumption upward as the body

chases that target, which links dilute, protein-poor food environments to overeating. Protein also triggers release of glucagon-like peptide-1 and peptide YY and lowers ghrelin, which lengthens the interval before hunger returns, and its digestion carries a higher thermic cost than carbohydrate or fat.

Fat quality contributes through slower gastric emptying and through signals that support fullness, and the source and structure of carbohydrate influence whether a meal delivers a gentle rise or a steep one. Gut-derived hormones tie these effects together. Signaling through glucagon-like peptide-1, peptide YY, ghrelin, leptin, and cholecystokinin along the gut-brain axis coordinates hunger and fullness across a meal and across the day (Liu et al., 2025). These messengers reach the hypothalamus and brainstem through the bloodstream and the vagus nerve, and they translate the chemistry of a meal into the sensation of having eaten enough.

A meal balanced across these components tends to smooth the glycemic curve, extend fullness, and lower the drive to eat sweet foods in the hours that follow. Protein and viscous fiber blunt the glucose peak, quality fat slows delivery, and an intact carbohydrate matrix spreads the sugar load, so the four levers act together on one outcome. The interactions among these components are shown in Table 1, which maps each element of dietary structure to its glycemic and satiety effects and to the resulting direction of craving.

Table 1. Dietary structure components and their effect on glycemic and satiety response

Dietary component	Mechanism	Glycemic response	Satiety response	Net effect on craving
Protein	Slows gastric emptying, raises GLP-1 and PYY, lowers ghrelin	Blunts glucose rise	High and prolonged	Reduces
Fiber, soluble and insoluble	Raises viscosity, slows glucose absorption, feeds gut microbiota	Lowers peak and rate of rise	Increases through volume and viscosity	Reduces
Fat quality, unsaturated	Slows gastric emptying, supports satiety signaling	Moderate blunting	Moderate to high	Tends to reduce
Carbohydrate quality and structure, whole against ultra-processed	Intact matrix slows digestion and absorption	Lower peak for whole, sharp peak for processed	Whole higher, processed lower	Whole reduces, processed heightens
Food matrix, water and micronutrients	Alters energy density and rate of sugar release	Lowers at equal sugar content	Increases at low energy density	Reduces

Note. Conceptual synthesis based on glycemic-index and food-structure literature and on satiety-regulation data. Effect directions summarize the overall weight of the literature and may vary between individuals, since glycemic and satiety responses to the same food differ across people.

3.3. The neurobiology of craving

Craving carries a neurochemical signature. Intermittent intake of large sugar loads activates dopamine-linked reward pathways involved in motivation and food seeking, and animal models show behavioral and neurochemical patterns that resemble addiction-like responses (Avena et al., 2008). These mechanisms resemble the neurobiology of addiction without establishing sugar as a clinically recognized addictive substance, since the evidence rests on animal models and current clinical classification holds no diagnostic category for sugar dependence. The overlap helps explain why cravings can feel strong even after a person

has eaten enough energy. The reward value of a food then operates on a track separate from raw caloric need.

Newer synthesis extends the account. A recent review of sugar and the brain's reward system included dopamine release in the mesolimbic system, receptor changes in the system, and downstream effects related to cravings, mood, and dependence (Qin et al., 2025). Chronic exposure to highly rewarded foods may lead to a desensitization of the dopaminergic response, requiring greater intake of the food in order to obtain the same reward, leading to a self-reinforcing cycle. Craving also recruits prefrontal control-related areas, the amygdala, and the hypothalamus, tapping into systems related to motivation, emotion, and energy balance.

Distinct from hunger, food cravings are governed by hormonal and neuropsychobiological mechanisms which do not align with the body's energy needs (J. Huang et al., 2023). Visual and olfactory cues can trigger cravings in a fed state, and the strength of that response tracks with appetite hormones and prior exposure. Reading craving as a neuroendocrine process, tied to appetite hormones and to reward circuitry, moves the concept past a label of weak self-control. The strength of a craving signals biology, habit loops, environment, and the reward value of nearby food, and that reframing carries direct consequences for how eating behavior is managed.

3.4. Degree of food processing

Processing level changes outcomes even when nutrient guidelines hold constant. A randomized crossover trial compared a diet built from ultra-processed foods with a diet built from minimally processed foods, both following the same healthy dietary guidelines, and recorded differences in weight and cardiometabolic markers between the arms (Dicken et al., 2025). The finding marks structure and processing as active variables that operate beyond the numbers on a nutrition label. Two diets can match on paper for macronutrients and still diverge in physiological effects once texture, matrix, and eating rate are taken into account.

Ultra-processed products carry high energy density, soft textures, and fast eating rates, a combination that raises intake and dampens satiety signaling. Their engineering prioritizes palatability and shelf life, the same features that make them convenient, while also stripping away the structural friction that slows digestion. The broader picture on sugar and health supports caution about high intake. An umbrella review of 73 meta-analyses found that high dietary sugar intake was associated with a wide range of cardiometabolic and other outcomes and endorsed limits on free and added sugars (Y. Huang et al., 2023). Processing level and nutrient composition interact across most of this evidence, since heavy processing tends to accompany higher sugar and lower fiber, and the 2 factors reach satiety through overlapping routes, which leaves their separate contributions hard to isolate outside controlled trials.

These results place refined sugar and heavy processing on the same side of the ledger, since both remove the structural features that slow digestion and support fullness. A whole food resists rapid breakdown and arrives with fiber, water, and micronutrients, while an ultra-processed product delivers a concentrated, fast-absorbing load with a soft mouthfeel that invites more. The processing lens therefore complements the matrix lens from the opening blocks, and it points toward whole-food structure as a lever for both glycemic control and satiety.

3.5. Sleep and stress as modulators

Sleep loss tilts appetite hormones toward hunger. A systematic review of sleep restriction reported shifts in leptin and ghrelin and in related appetite and weight-regulating hormones, with effects that vary by sex and by context (Alfikany et al., 2025). Lower leptin weakens the satiety signal, higher ghrelin strengthens the hunger signal, and the combination raises appetite and pushes preference toward carbohydrate-rich, energy-dense food. A night of short sleep can load the next day with stronger cravings that owe more to hormones than to any lapse in resolve. The evidence separates acute sleep restriction from chronic sleep deprivation. Acute

restriction across 1 or 2 nights shifts leptin and ghrelin within days, while chronic short sleep sustains milder hormonal changes alongside altered food choice, daytime fatigue, and a longer eating window, so the 2 patterns overlap without matching in physiological effect.

Stress recruits the same hormonal machinery. Glucocorticoid excess from chronic stress blunts appetite-suppressing hormone signaling and shifts eating toward hedonic, reward-driven choices (Kuckuck et al., 2023). Cortisol shifts food choice toward sweet and fat-rich alternatives, which suggests that stress-induced craving aligns with the reward circuitry described above and converts emotional load into a physiological push toward energy-dense food. These effects appear additive, since both stress and short sleep drive appetite systems in the same direction.

Erratic meal patterns and low caloric intake add their own pressure, since they produce wide fluctuations in blood glucose that induce hunger and trigger craving. Regularity of meal timing works in the opposite direction and stabilizes glycemia and the hormonal signals that track it (Raubenheimer & Simpson, 2023). These lifestyle inputs sit upstream of the meal itself, and they set the baseline against which the body interprets any given food. The effects of these modifiers on appetite hormones and cravings are summarized in Table 2.

Table 2. Lifestyle modifiers, appetite hormones, and sugar craving

Factor	Mechanism and hormones	Direction of shift	Effect on eating behavior	Supporting evidence
Sleep deprivation	Leptin lower, ghrelin higher	Stronger hunger signaling	Greater preference for carbohydrate-rich, energy-dense food	Alfikany et al., 2025
Chronic stress	HPA-axis activation, cortisol higher	Shift toward reward eating	Heightened craving for sweet and fatty food	Kuckuck et al., 2023
Irregular meals and under-eating	Wide glucose swings, rising hunger	Compensatory overeating	Episodes of strong craving	Liu et al., 2025
Regular meal timing	Stabilized glycemia and satiety signaling	Smoother fluctuations	Steadier craving, sustainable behavior	Raubenheimer & Simpson, 2023

Note. Hormonal directions under sleep restriction are generalizations, and the table does not reproduce numeric data from primary sources. Physical activity sits outside the conceptual model, since exercise acts on appetite and glycemia through energy expenditure and insulin sensitivity, a pathway that runs parallel to the dietary structure examined here, and its inclusion would widen the scope beyond meal composition and its immediate modulators.

3.6. An integrative model

The blocks above combine into a single loop. Macronutrient balance shapes a smoother glycemic and hormonal response, which in turn can lower craving through a weaker reward drive and a slower return of hunger. Lower craving supports eating patterns that hold without dependence on constant self-control. Sleep and stress act at the middle of the chain, altering glycemic and hormonal responses and the cravings that follow. The loop treats sustainable eating as an outcome of physiology and daily structure, with each stage feeding the next.

Figure 1 presents this model. The design places macronutrient balance and food structure at the entry point, routes them through the glycemic and satiety response, links that response to reduced craving, and then to durable eating behavior. Sleep, stress, and meal timing act on several stages of the chain at once, and the

degree of food processing enters at the level of dietary structure and of the glycemic response. A feedback arrow returns from sustainable eating behavior to dietary structure, since stable patterns reinforce the food choices that produced them. The value of the diagram lies in its causal ordering, which shows craving as a downstream product of structure and physiology, and locates the lifestyle modifiers at the points where they exert their pull.

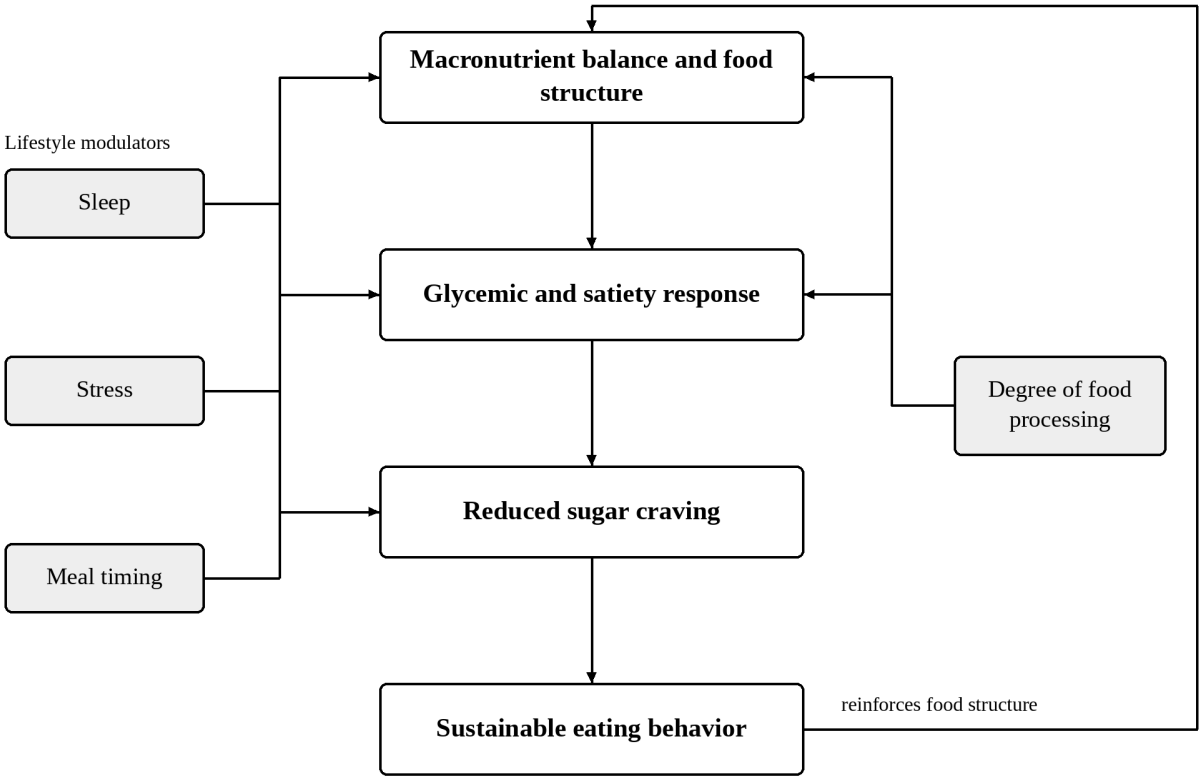

Figure 1. Integrative model of macronutrient balance and sugar craving

4. Discussion

The synthesis reframes a familiar debate. A pair of popular positions, one casting sugar as a uniform toxin and one casting craving as weak character, each describes a fragment of a larger mechanism. The evidence points toward dietary architecture as the organizing variable, since the same sugar produces different physiological and behavioral effects depending on the company it keeps in a meal. This view absorbs the grain of truth in each popular position while supplying the mechanism that both leave out, and it turns a moral question about discipline into a design question about meals.

The model departs from earlier dietary frameworks in the placement of its causal center. Glycemic index frameworks rank foods by their blood glucose response and stop at the metabolic readout. Energy balance frameworks count intake against expenditure and treat craving as noise around that account. Food reward

frameworks describe dopamine signaling without connecting it to meal composition. The framework advanced here links these levels, since it treats dietary structure as the input, the glycemic and satiety response as the mediating step, craving as the behavioral output, and sleep, stress, and meal timing as modulators that act across the sequence. This ordering yields testable predictions about which meal structures lower craving for which individuals, and it supplies clinicians with a mechanism they can explain to patients, which advances the field beyond parallel accounts that share no common variable.

4.1. Practical implications

Practical implications follow from the satiety and glycemic findings. Meals built around protein, viscous fiber, quality fat, and intact carbohydrate sources flatten glucose peaks and extend fullness, which lowers the pull toward sweet food in the hours that follow. A structure of this kind offers a durable route, since it works with hunger biology in place of fighting it through restriction alone. Whole-food sweeteners such as honey or dates still deliver sugar, and their matrix and dose shape the response, which keeps portion and context in the picture even for foods that feel more natural.

Reading craving as biology carries a behavioral payoff. When a craving registers as a hormonal and reward-linked signal, self-blame loses its footing, and attention moves toward sleep, stress, meal timing, and the surrounding food environment. This shift matters for adherence, because programs that lean on willpower tend to fail at the moments when hormones and cues run highest. A person who understands the biology can act on the causes, adjusting sleep and meal structure, in place of straining against a signal that the body keeps regenerating. Clinical settings offer 3 immediate applications. Obesity management can use meal structure to lower craving pressure alongside energy targets. Diabetes prevention can draw on the same structural levers to flatten postprandial glucose in people with insulin resistance. Nutrition counseling can move the conversation from restriction toward composition and daily rhythm, which gives patients a route that holds without sustained willpower.

Traditional eating patterns illustrate the model in a concrete setting. Home cooking, fermented foods, warm meals, regular timing, and low reliance on packaged snacks assemble many of the structural features that flatten the glycemic curve and support fullness, and they do so without singling out any one ingredient as the enemy. Such patterns show how meal structure and daily rhythm can carry the load that ingredient-by-ingredient rules struggle to bear, and they align long-standing practice with the mechanisms drawn out above.

4.2. Limitations

Limitations temper the conclusions. The retrieved evidence spans heterogeneous designs, and several mechanistic claims draw on animal models that do not map cleanly onto human eating. Individual glycemic responses vary among people to the same food, limiting universal prescriptions (Y. Huang et al., 2023). Much of the lifestyle evidence comes from short-term laboratory work, and long-term field data remain sparse. Differences in measurement and reporting across studies add further caution, and a narrative review cannot rule out selective coverage.

4.3. Future directions

Future work can move in several directions. Personalized nutrition can match meal structure to individual glycemic and hormonal profiles, so that guidance fits the person in front of it. Continuous glucose monitoring offers a research tool for capturing individual responses in daily life, and it can test which meal structures flatten the curve for a given individual. Longitudinal designs can test whether structure-based eating sustains over years and whether it reduces craving and improves cardiometabolic markers over time. Trials that manipulate

sleep and stress alongside diet can quantify how much these modulators move the craving response, and they can show where meal design meets the limits set by lifestyle.

5. Conclusion

Dietary structure organizes the craving response more than any isolated nutrient. Balance across protein, fiber, fat quality, and carbohydrate quality shapes the glycemic and hormonal response to a meal, and that response influences how strong sugar cravings become and how long eating patterns persist. Cravings reflect biology and habit, shaped by reward circuitry and appetite hormones, and modulated by sleep and stress. Framing the problem this way shifts the practical target from ingredient elimination to meal composition and daily rhythm, offering a steadier path for eating behavior. The available data support this conceptual framework while individual responses and environmental factors continue to influence dietary behavior, so the model stands as an organizing account without settling as the single explanation. Continued work on personalized responses and long-term sustainability will test how far the model extends and show where meal structure ends, and other factors begin.

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