

Circadian Rhythms, Sleep Patterns, and the Regulation of Cravings for Sweet and Refined-Carbohydrate Foods

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Abstract

Cravings for sweet and refined-carbohydrate foods are commonly reported and can contribute to excess energy intake, although terminology and measurement vary across studies. This narrative review examines how sleep duration, sleep architecture, circadian timing, and meal timing are associated with appetite, food reward, and cravings for sweet and refined-carbohydrate foods. PubMed, Scopus, and Web of Science were searched for English-language peer-reviewed human studies published primarily between January 2021 and August 2026, and selected foundational studies were included for historical and mechanistic context. Experimental sleep restriction has been associated with changes in appetite-regulating hormones and with heightened responses of neural reward circuits to palatable food, although hormonal responses are heterogeneous across protocols, sexes, and populations. Observational studies link shift work and social jetlag to poorer diet quality and altered eating behavior, and controlled trials indicate that late meal timing can increase hunger independent of energy content. Most of the available evidence concerns hunger, appetite, or highly palatable food intake rather than carbohydrate-specific craving, experimental samples are often small, and observational designs cannot establish causality. Overall, the evidence supports an association between circadian and sleep disruption and an increased drive toward sweet and highly palatable foods, and the proposed integrative framework linking homeostatic, reward-related, and behavioral pathways may guide future research.

Keywords: circadian rhythms, sleep restriction, food craving, appetite, chrononutrition, social jetlag, meal timing, food reward, refined carbohydrates

1. Introduction

Cravings for sweet and highly palatable foods are common and can influence dietary intake, food choice, and energy consumption. Although food cravings are often discussed in relation to individual choice or self-control, contemporary research indicates that they are also influenced by biological and environmental factors, including sleep duration, circadian phase, meal timing, endocrine regulation, and neural reward processing (Akhlaghi & Kohanmoo, 2025). Accordingly, chronobiology provides a useful framework for examining craving as a multidimensional phenomenon rather than exclusively as a behavioral outcome. Hunger and food craving should be distinguished conceptually. Hunger represents a relatively general motivation to

consume food, whereas craving refers to an intense desire for a specific food or food category. These states overlap but are not interchangeable. Sleep and circadian disruption may affect both homeostatic appetite and reward-related food motivation, and studies vary considerably in whether they measure hunger, appetite, craving, food preference, or actual food intake.

Foundational controlled experiments in the early 2000s provided some of the first evidence that severe sleep curtailment could alter appetite-related physiology and subjective hunger. Early studies reported changes in leptin and ghrelin alongside increased appetite for calorie-dense foods (Spiegel et al., 2004). Subsequent research has expanded this framework to include food-reward processing, free-living dietary behavior, circadian misalignment, social jetlag, chronotype, and meal timing. More recent evidence also demonstrates substantial heterogeneity across studies, indicating that appetite responses to insufficient sleep cannot be explained by leptin and ghrelin alone (Akhlaghi & Kohanmoo, 2025; Alfikany et al., 2025).

Two features of modern life give the topic its urgency. Short sleep has become habitual for a substantial fraction of the working population, and schedules that detach waking time from the internal biological clock, from rotating shifts to weekend delays in sleep timing, now affect hundreds of millions of people worldwide (Baek & Yoon, 2024). Artificial light, 24-h commerce, and screen-based work extend wakefulness into hours when the circadian system expects rest, while the food environment supplies palatable carbohydrates at every point of this extended day. The collision of a delayed behavioral schedule with an unchanged internal temporal order may create conditions that favor increased craving, and the question of how circadian rhythms and sleep patterns relate to the drive toward sweet and refined-carbohydrate foods takes on both scientific and applied significance.

The review concentrates on the chronobiological axis of appetite regulation. Macronutrient composition of the diet is included in the discussion as context, since the primary analytical focus is on the timing of sleep and food intake and on the hormonal and neural consequences of their disruption. The aim of the work is to synthesize evidence published primarily between 2021 and 2026 on the associations among circadian rhythms, sleep duration and architecture, meal timing, and cravings for sweet and refined-carbohydrate foods, and to propose an integrative framework as a hypothesis-generating basis for future research.

2. Review Method

This work was conducted as a targeted narrative review with analytical synthesis. PubMed, Scopus, and Web of Science were searched for English-language peer-reviewed human studies published primarily between January 2021 and August 2026. Search terms included combinations of sleep restriction, sleep deprivation, circadian misalignment, circadian rhythm, social jetlag, chronotype, chrononutrition, meal timing, food craving, appetite, food reward, carbohydrate intake, sugar intake, leptin, and ghrelin. Reference lists of relevant reviews and original studies were also examined. Selected foundational studies published before 2021 were included when necessary to provide historical or mechanistic context.

Priority was given to randomized controlled studies, crossover experiments, systematic reviews and meta-analyses, and observational investigations using established sleep or dietary measures. Studies were considered relevant when they examined at least one relationship between sleep or circadian timing and appetite, craving, food preference, eating behavior, dietary intake, or appetite-related endocrine measures. Editorials, conference abstracts, preprints without peer review, and materials from social media platforms were excluded. Because this is a narrative rather than a systematic review, the search was designed to identify representative and methodologically informative evidence rather than to provide exhaustive study enumeration or pooled effect estimates. No formal meta-analysis or quantitative risk-of-bias synthesis was conducted.

The narrative format carries known constraints, and 2 measures limited their influence. Quantitative results

appear in the text, together with their effect sizes and probability values wherever the source reported them, anchoring qualitative statements to numbers. The selection of core publications favored systematic reviews and randomized designs over single observational reports, which reduces the weight of any individual study in the final synthesis.

Terminology follows accepted usage in chronobiology. Circadian misalignment denotes a mismatch between the endogenous circadian system and the behavioral cycles of sleep, wakefulness, and eating. Social jetlag refers to the shift of the sleep midpoint between workdays and free days. Craving refers to a motivational state directed toward a specific food category and is measured using visual analog scales, validated questionnaires, or behavioral choice paradigms. Carbohydrate-dense foods are products in which refined starches or sugars provide the majority of energy. Because terminology varies across the included studies, this review uses the phrase sweet and refined-carbohydrate foods primarily for foods high in added sugars and/or refined starches and uses highly palatable foods when that broader category more accurately reflects the evidence. These terms should not be interpreted as implying that all carbohydrate-rich foods have equivalent physiological or behavioral effects.

3. Evidence Synthesis

3.1. Circadian Organization of Energy Metabolism

The mammalian circadian system operates as a hierarchy of oscillators. The suprachiasmatic nucleus of the hypothalamus generates a rhythm of about 24 h and synchronizes peripheral clocks located in the liver, pancreas, adipose tissue, skeletal muscle, and gut. At the molecular level, the rhythm arises from transcription-translation feedback loops involving the genes *CLOCK*, *BMAL1*, *PER*, and *CRY*, whose products oscillate in cells throughout the body. Light is the dominant environmental timing signal for the central circadian pacemaker, whereas meal timing represents an important timing cue for peripheral metabolic clocks (Akhlaghi & Kohanmoo, 2025).

Glucose tolerance, insulin sensitivity, gastric emptying, diet-induced thermogenesis, and the release of the hormones cortisol, leptin, and ghrelin all show diurnal variation biased toward the processing of food in the biological daytime. Subjective hunger itself carries an endogenous rhythm that peaks in the biological evening, a pattern documented in protocols that separate the circadian component from the effects of fasting and behavior (McHill et al., 2022). The endogenous evening rise in hunger may interact with environmental food availability and learned eating patterns, potentially increasing vulnerability to energy-dense food intake later in the day.

Sleep occupies a defined position within this architecture. Slow-wave sleep in the first half of the night accompanies the nadir of cortisol and a surge of growth hormone; melatonin secretion frames the biological night, and the leptin curve reaches its acrophase during habitual sleep hours, which suppresses hunger across the overnight fast. When behavior remains aligned with the internal order, hormonal signals of hunger and satiety operate within their evolved range. Misalignment redistributes food intake toward phases of low metabolic preparedness, and this redistribution alters the hedonic value of food, since reward circuits are also under circadian control. These 2 levels, endocrine and neural, jointly determine when and toward what the drive to eat intensifies.

Peripheral tissues deserve attention in their own right, since the drive to eat forms at their intersection. The clock of the liver schedules glycogen storage and gluconeogenesis, the clock of the pancreas gates insulin secretion, adipose tissue times leptin release, and the gastrointestinal tract coordinates motility, enzyme secretion, and the anticipatory rise in ghrelin before habitual meals. Coherence among these local programs depends on a shared phase, and food delivered at an unaccustomed hour reaches organs whose programs point

elsewhere, thereby lowering processing efficiency and distorting the feedback the brain receives about the meal. Altered eating patterns under disordered schedules may partly reflect this internal desynchronization, although direct evidence linking peripheral-clock disruption to cravings for specific macronutrients remains limited.

Cortisol and melatonin deserve separate mention because both hormones mark the boundaries of the biological night and both respond to its violation. Sleep restriction elevates evening cortisol, and elevated evening cortisol has been associated with reduced insulin sensitivity and may contribute to appetite for energy-dense foods, adding a stress-endocrine channel to the leptin-ghrelin axis. Melatonin secretion, in turn, overlaps with late meals in people who eat close to bedtime, and glucose tolerance during the melatonin phase declines, so that identical food produces a larger glycemic excursion at night than in the morning. The temporal geometry of hormone release thus makes the clock time of behavior an important metabolic variable, although neither hormone has been shown to directly generate cravings for sweet foods.

3.2. Sleep Duration, Sleep Restriction, and Appetite Hormones

In early laboratory protocols in which time in bed was shortened to about 4 h for consecutive nights, circulating leptin declined while ghrelin rose, and participants reported stronger hunger together with increased appetite for energy-dense foods (Spiegel et al., 2004). Later work reports heterogeneous hormonal responses that depend on protocol, sex, and population, and a recent comprehensive review concluded that sleep deprivation affects both homeostatic and non-homeostatic regulation of appetite, with a prominent role assigned to the food reward system (Akhlaghi & Kohanmoo, 2025; Alfikany et al., 2025). Figure 1 summarizes appetite-related responses reported after sleep restriction together with the principal modifiers of these responses; the pattern of reduced leptin and elevated ghrelin describes early findings rather than a universal physiological response.

The 2 hormones at the center of this literature perform complementary duties. Leptin, secreted by adipose tissue in proportion to energy stores, informs the hypothalamus of long-term sufficiency and restrains intake, while ghrelin, released by the stomach in anticipation of meals, rises before habitual eating times and initiates the search for food. Their receptors converge on hypothalamic circuits that integrate these signals with input from reward structures, and the balance between the 2 messengers sets the background tone of appetite on which individual meals are decided. Sleep enters the system because the secretion of both hormones follows a 24-h profile anchored to habitual rest, so that any disruption of the sleep schedule also distorts the hormonal message.

Appetite-related responses to sleep restriction: observed patterns and modifiers

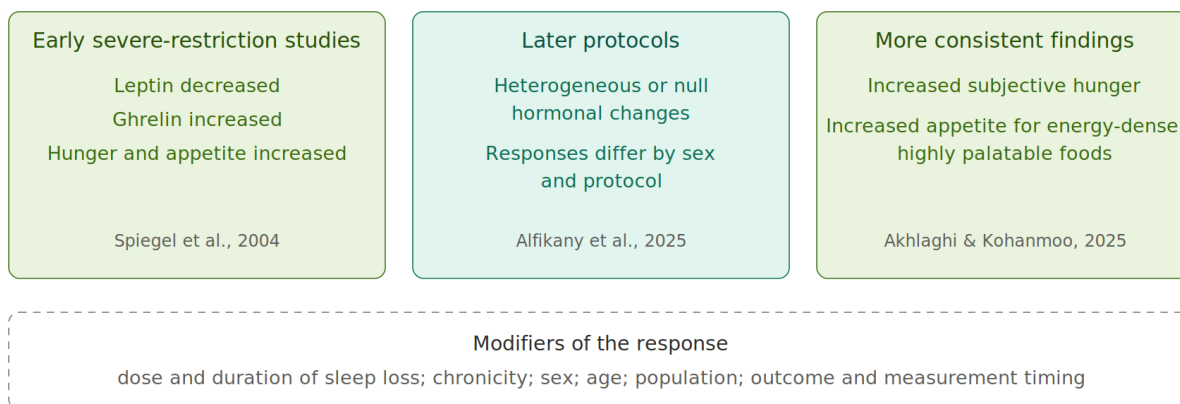


Figure 1. Appetite-related responses reported after sleep restriction and principal modifiers of these responses, compiled by the author based on Spiegel et al. (2004), Akhlaghi and Kohanmoo (2025), and Alfikany et al. (2025)

The hormonal picture contains nuances that complicate a simple 2-hormone account. A systematic review of human experimental studies published before December 2024 examined whether sex modifies the endocrine response to sleep curtailment and found divergent leptin dynamics in men and women across 8 studies with a combined sample of 302 participants (Alfikany et al., 2025). Heterogeneity of protocols, from a single night of total deprivation to a week of partial restriction, explains why several individual trials failed to detect hormonal change even as behavioral measures of appetite moved in the expected direction. The endocrine response, in other words, depends on the dose of sleep loss, on its chronicity, and on the characteristics of the population under study. A narrative synthesis published in 2026 treats the question of whether sleep curtailment stimulates appetite, motivation to eat, and dietary intake as only partly settled and emphasizes methodological considerations for future studies, which reinforces the heterogeneity documented in the hormonal literature (Tan et al., 2026).

Evidence from free-living conditions strengthens the case for external validity. In a randomized crossover trial, 3 days of sleep limited to 5 h, compared with 8 h, changed the dietary behavior of healthy adults outside the laboratory, with shifts in appetite and intake recorded under the everyday constraints of work and family life (Tajiri et al., 2023). The convergence of laboratory and field data indicates that the endocrine consequences of short sleep translate into real food choices.

Dose and reversibility complete the endocrine picture. Effects on appetite have been reported after a single night of total deprivation, can grow across consecutive nights of partial restriction, and tend to recede when recovery sleep restores habitual duration, a temporal profile that suggests a reversible regulatory response. Whether habitual weekly cycles of weekday restriction and weekend recovery produce a corresponding rhythm of craving has not been directly established and remains a question for prospective studies.

3.3. Hedonic and Cognitive Mechanisms of Craving

Endocrine signals explain the intensity of hunger, while the selectivity of craving, its fixation on sweets and refined starches, belongs to the domain of neural reward processing. Neuroimaging work conducted over the past 2 decades indicates that acute sleep loss can amplify the responses of the amygdala and ventral striatum to images of high-calorie food and can weaken the activity of prefrontal regions responsible for evaluative control. Reviews of this literature converge on a 2-part formula: sleep loss increases the incentive value of energy-dense foods and reduces the capacity to resist them (Akhlaghi & Kohanmoo, 2025).

A conceptual distinction between hunger and craving structures this domain of research. Hunger designates a general readiness to eat that a wide range of foods can satisfy, whereas craving refers to a directed motivational state whose object resists substitution, so that a craved food category is not readily replaced by an alternative of similar energy content. The distinction matters because sleep loss and misalignment may act on the 2 states through partly separate channels, with hunger influenced through the endocrine route and the selectivity of craving shaped through the hedonic route; a short night may therefore increase both the overall amount of desired food and the share of that desire directed at highly palatable items.

Behavioral studies provide the connecting evidence linking neural findings to everyday eating. In a cross-sectional sample of young adults, greater social jetlag was associated with higher scores for emotional eating and loss of control over eating, along with reduced intuitive eating, a pattern that links circadian instability to eating styles through which cravings convert into consumption (Vrabec et al., 2022). Emotional eating deserves attention in this context because highly palatable foods are a frequent object of affect-driven intake, and factors that erode top-down control may channel behavior toward them.

Reward sensitivity itself obeys a daily rhythm. The response of dopaminergic circuits to palatable stimuli intensifies throughout the waking day and peaks in the evening, the phase at which the endogenous rhythm of hunger also peaks. The alignment of the 2 curves concentrates vulnerability in the hours after dinner, and sleep restriction deepens the trough of control at exactly this phase, since the prefrontal cost of a long day accumulates toward night. Evening craving for sweets may therefore follow a partly predictable circadian pattern, and earlier sleep onset is one candidate lever through which its amplitude could be reduced, pending interventional confirmation.

Measurement of craving has matured alongside the mechanistic work. Contemporary studies employ visual analog scales administered at fixed circadian phases, trait and state questionnaires of food craving, progressive ratio tasks that quantify the effort a participant will expend for a palatable item, and portion selection paradigms. The multiplicity of instruments complicates comparisons across studies, yet it also demonstrates that craving admits to objective quantification, placing the construct on the same methodological footing as hunger or satiety.

Hormonal and hedonic pathways act synergistically rather than sequentially, and this synergy has a defined direction: ghrelin sensitizes reward circuits to food cues, reward circuits amplify the behavioral consequences of ghrelin elevation, and prefrontal fatigue removes the remaining brake. A self-reinforcing loop, in which each short night raises the probability of an eating episode focused on highly palatable food and each such episode degrades the subsequent night, is plausible on this basis, although the full cycle has not been demonstrated and remains a hypothesis.

3.4. Models of Circadian and Sleep Misalignment

Research on circadian disruption employs several complementary models, each of which isolates a different facet of the phenomenon while pointing toward the same behavioral outcome. Table 1 summarizes these models together with their defining characteristics, the type of available evidence with representative studies, and the principal reported associations and limitations.

Table 1. Models of sleep and circadian misalignment and their associations with eating behavior

Model	Characteristics	Evidence type and representative studies	Reported associations and key limitations
Acute and short-term sleep restriction	Total or partial sleep loss over 1 to several nights in laboratory protocols	Experimental; Spiegel et al. (2004); Alfikany et al. (2025)	Early studies: reduced leptin, elevated ghrelin, increased hunger; later studies heterogeneous; small samples
Chronic sleep restriction	Habitual sleep shorter than 6 to 7 h in the population and laboratory settings	Observational and laboratory; Akhlaghi & Kohanmoo (2025)	Associated with higher energy intake through snacking and with excess body weight; confounding possible
Sleep fragmentation	Repeated awakenings with preserved total sleep duration	Limited experimental evidence	Altered sleep architecture; evidence linking fragmentation to refined-carbohydrate craving remains limited
Shift work and circadian misalignment	Systematic wakefulness and eating during the biological night	Experimental crossover; Qian et al. (2023), N = 7; McHill et al. (2022)	Increased acylated ghrelin and hunger under controlled intake; very small samples limit generalization
Social jetlag	Divergence of sleep timing between workdays and free days, mismatch with chronotype	Observational; Islam et al. (2022); Vrabec et al. (2022); Baek & Yoon (2024)	Associated with poorer diet quality, meal skipping, and emotional eating; cross-sectional design precludes causal inference
Late meal timing	Displacement of the main share of energy intake to evening and night hours	Experimental crossover; Vujović et al. (2022)	Increased hunger and ghrelin-to-leptin ratio; participants with overweight or obesity; craving not directly measured

Forced desynchrony represents the most rigorous of the laboratory approaches. In a 32-day inpatient protocol, sleep, wakefulness, and meals were distributed across all phases of the circadian cycle, which allowed the investigators to separate the contribution of chronic circadian disruption from that of concurrent sleep restriction, and both factors independently influenced subjective hunger, appetite, and food preference (McHill et al., 2022). Such designs are directly relevant to rotating shift schedules, in which workers pass through a comparable sequence of mismatched phases every week.

Field-anchored experiments confirm that these effects survive contact with real occupational populations. In a randomized crossover study of chronic night shift workers, a 3-day protocol with a 12-h inversion of behavioral cycles raised 24-h concentrations of acylated ghrelin by about 17%, $p = 0.009$, and increased hunger at breakfast by about 14%, $p = 0.04$, under full control of dietary intake (Qian et al., 2023). The sample comprised 7 chronic night shift workers, which limits generalization, yet the finding indicates that years of shift experience do not abolish the endocrine response to misalignment; craving for specific food categories was not measured in this protocol.

Social jetlag extends the model to the general population. Among Japanese workers, later chronotype and greater weekday-to-weekend shifts in sleep timing showed associations with weaker adherence to national dietary guidelines (Islam et al., 2022). A nationwide Korean analysis of 11,430 workers found that social jetlag of 120 min or more corresponded to lower diet quality scores, with an odds ratio of 1.27 for falling into the poorest quartile of the national healthy eating index, and the deficit concentrated in meal skipping, low intake of fruits and vegetables, and unrestricted consumption of saturated fat (Baek & Yoon, 2024). Meal skipping merits emphasis in the context of craving, since a skipped breakfast after a delayed weekend night may predispose toward compensatory intake later in the day. Most of this evidence is observational and cannot establish causality, and definitions and cutoffs of social jetlag vary between studies.

Age amplifies the relevance of the weekend model. Students and young adults carry the largest average values of social jetlag because late chronotypes concentrate in the second and third decades of life, while institutional schedules remain anchored to the morning, and the same age window forms long-term dietary habits. Findings that link jetlag in this group to emotional eating and weakened control over intake are cross-sectional, yet they point to a potentially sensitive period in which temporal disorder and eating style may consolidate together (Vrabec et al., 2022).

Quantification of social jetlag follows an accepted convention: the absolute difference between the sleep midpoint on free days and the midpoint on workdays, expressed in minutes or hours. The measure captures a weekly oscillation in the sleep schedule that resembles a flight across time zones, repeated every 7 days, and thresholds of 60, 90, and 120 min serve as markers of rising severity in the literature, although these cutoffs are conventions that differ across studies rather than validated thresholds. Values of 120 min or more, present in 12.1% of the Korean working sample, define the group in which dietary consequences are most consistently observed (Baek & Yoon, 2024).

Chronotype adds an individual dimension to these population patterns. Evening-oriented individuals experience greater social jetlag under standard work schedules, and this group also shows a dietary profile characterized by more snacking, later dinners, and higher intake of sweetened products, associations documented among working adults alongside the effects of jetlag itself (Islam et al., 2022). Later chronotype is best regarded as a potential modifying factor within these associations, and late chronotypes represent a population segment for which the mechanisms reviewed here may carry particular practical relevance.

Sleep fragmentation completes the series of models. Repeated awakenings reduce the proportion of slow-wave sleep even when total sleep duration remains within the recommended range. Evidence specifically linking fragmentation to altered appetite and, in particular, to refined-carbohydrate craving remains limited, and the model is included for completeness rather than as an established pathway. It matters for populations, from parents of infants to patients with sleep apnea, whose nights are long on paper and shallow in fact, and it cautions against reducing sleep health to a single number of hours.

3.5. Chrononutrition and the Timing of Food Intake

The timing of food intake serves as an input to the circadian system and an output of behavior, and this dual role defines the field of chrononutrition. The strongest causal evidence on meal timing comes from a randomized controlled crossover trial in which participants consumed identical meals either early or late in the waking day under strict control of nutrient composition, physical activity, sleep, and light exposure. Late eating increased hunger, with $p < 0.0001$, raised the 24-h ratio of ghrelin to leptin, reduced waking energy expenditure, and shifted gene expression in adipose tissue toward adipogenesis (Vujović et al., 2022). The design excluded caloric differences by construction, which permits the conclusion that the clock time of eating, taken alone, modifies hunger and its hormonal correlates. Participants were adults with overweight or obesity, and craving for specific food categories was not a measured outcome, so extension of these findings to carbohydrate-specific craving remains indirect.

Time-restricted eating approaches the same relationship from the interventional side. A systematic review of randomized controlled trials with daily fasting windows of 14 h or longer and durations of at least 8 weeks assessed effects on objective and subjective sleep parameters and found improvement of subjective sleep quality in 1 of 6 included trials, with the remaining trials showing no deterioration (Bohlman et al., 2024). The bidirectional reading of these data matters for practice: consolidating food intake into the earlier part of the day leaves sleep intact while removing the late-night eating window during which snacking on highly palatable foods tends to concentrate.

A parallel concept of eating jetlag has entered the literature to describe the weekday-to-weekend shift of meal timing that accompanies the shift of sleep. The 2 displacements correlate without coinciding, since a person can hold meals steady across the week while sleeping late on free days, and studies that separate them attribute independent dietary consequences to each. For the analysis of craving, the concept adds a practical target: stabilizing weekend meal times offers a partial defense for individuals whose social circumstances block the stabilization of sleep itself.

Interpretation of time-restricted eating requires caution on 2 points. Adherence in free-living conditions declines over time, and a fasting window imposed regardless of chronotype can conflict with social meal patterns, generating stress that offsets the metabolic gains. The reviewed trials do not identify a universally optimal eating window, and prescriptions of a specific interval between the last meal and sleep would exceed the evidence; the available data indicate only that consistent eating windows were not accompanied by deterioration of sleep quality (Bohlman et al., 2024).

The distribution of energy across the day supplies a further variable. Physiological considerations favor earlier intake, since insulin sensitivity is higher in the biological morning and declines toward evening, yet direct evidence that morning-loaded eating reduces evening craving is mixed, and conclusions about the optimal caloric distribution should remain provisional pending controlled trials with craving-specific outcomes.

Breakfast behavior illustrates how timing variables may interlock. Delayed or skipped morning meals occur more often among late chronotypes and after weekend shifts of sleep, and observational data associate meal skipping with poorer overall diet quality (Baek & Yoon, 2024). Whether breakfast regularity itself reduces later intake of sweets has not been established by the evidence reviewed here, and mechanistic explanations through ghrelin accumulation or morning insulin sensitivity remain hypotheses; breakfast timing is best characterized as an area of mixed evidence awaiting controlled trials.

Regularity of meals emerges as a third element beside clock time and distribution. Erratic eating schedules weaken the entrainment of peripheral clocks by food and reproduce, in miniature, the desynchronization observed in shift work. Population data linking social jetlag to meal skipping and poorer diet quality support the interpretation that a stable meal rhythm serves as a zeitgeber whose loss has measurable dietary consequences (Baek & Yoon, 2024). For the regulation of craving, a plausible working hypothesis follows from this physiology: meals of predictable timing, placed within the biological day, may narrow the windows during which hormonal and hedonic pressures toward highly palatable foods coincide.

3.6. Carbohydrate Specificity of the Evidence

A dedicated statement on carbohydrate specificity is warranted. Across the reviewed literature, outcome measures more often concern hunger, appetite, total energy intake, or preference for highly palatable foods that combine carbohydrate, fat, salt, and sugar than refined-carbohydrate craving measured as a distinct construct. Findings of increased hunger after sleep restriction or late eating should therefore not be read as direct proof of increased sugar craving, and studies reporting preference shifts toward energy-dense foods capture a broader category than sweets alone. The current evidence is stronger for changes in hunger, food reward, and highly palatable food consumption than for a unique biological mechanism that selectively generates cravings for refined carbohydrates, and future studies that measure carbohydrate-specific craving with validated instruments are required before more precise claims can be made. Studies that measure category-specific craving directly remain rare; in a laboratory study of 143 non-diabetic adults published in 2026, self-reported daytime sleepiness was examined in relation to pre-meal cravings for sweet, salty, and starchy foods across a 14-h protocol with standardized meals, with insulin sensitivity assessed as a moderating factor, and designs of this precision indicate the direction in which craving research is moving (Romaker et al., 2026).

4. Discussion

The reviewed evidence can be organized into a proposed conceptual framework rather than a proven mechanism. Figure 2 groups the relevant pathways into 3 broader domains: homeostatic appetite regulation, reward-related mechanisms, and behavioral and circadian timing. Within this framework, insufficient sleep and circadian misalignment may shift homeostatic signals toward hunger, may amplify reward responses to palatable food while weakening prefrontal control, and may open a late eating window that places intake at the circadian phase of elevated endogenous hunger. Increased drive toward sweet and highly palatable foods is positioned at the hypothesized point of convergence, and the dashed arrows of the framework denote proposed associations rather than established causal links.

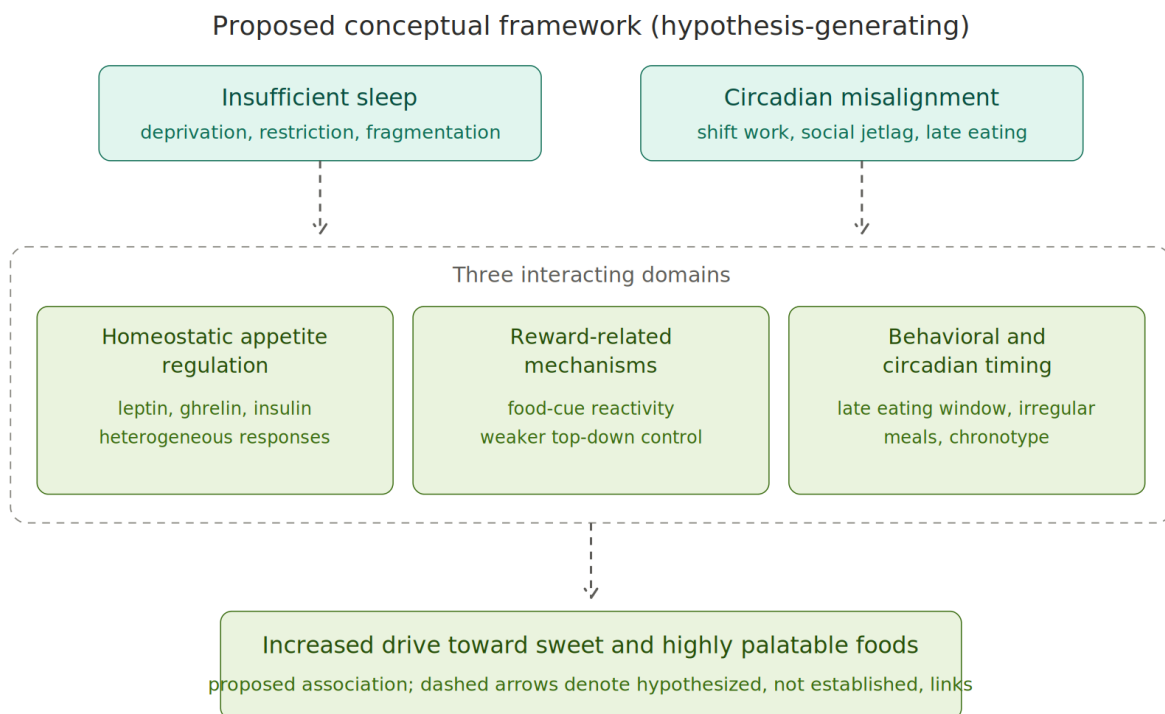


Figure 2. Proposed conceptual framework linking insufficient sleep and circadian misalignment with the drive toward sweet and highly palatable foods; dashed arrows denote hypothesized associations, compiled by the author

The framework carries conceptual significance because it dissolves an apparent competition between explanations. Endocrine accounts of overeating after short sleep and hedonic accounts centered on reward circuits have at times been presented as rivals, yet the material reviewed here suggests that they may operate as connected stages, in which hormonal change sensitizes neural valuation and behavioral schedule determines when the sensitized system meets food. The framing may also help explain why interventions aimed at willpower alone show modest durability: they address the final step of a longer sequence.

The period covered by this review contributed advances of a specific kind. Earlier work had established the basic endocrine response to sleep loss, and the studies of 2021 to 2026 extended the finding in 3 directions: into chronic shift workers whose long occupational history might have produced adaptation, into free-living conditions where competing demands could have diluted the effect, and into the isolated manipulation of meal timing under full control of energy content. Each extension closed an avenue of doubt, and their combination moved the field from demonstration toward specification, from the claim that temporal disorder alters appetite

toward measurement of how much, in whom, and through which pathway.

The public health relevance of the framework warrants a careful statement. Short sleep and social jetlag each show associations with excess body weight at the population scale, and the pathways assembled here suggest plausible behavioral intermediaries. The full sequence from circadian disruption through craving and carbohydrate intake to caloric surplus and obesity has not been demonstrated as a chain, and each link currently rests on evidence of different strength. An account of obesity that includes the temporal dimension of behavior complements, without displacing, accounts centered on the food environment and physical activity, and it directs preventive attention toward work schedules, school start times, and the cultural norms that govern bedtime.

The evidence base combines methodological strengths with limits that constrain causal language. Randomized, crossover studies using full dietary control (for example, the late-eating and shift-worker studies) found evidence that manipulation of eating time alters human appetite hormones (Qian et al., 2023; Vujović et al., 2022). However, sample sizes in studies of this kind tend to be small ($N = 7$ in the shift worker study), and replication of this work is limited due to the cost of inpatient chronobiology. However, cross-sectional associations between social jetlag and dietary quality are subject to potential reverse causation from diet to sleep, and residual confounding from occupation, income, and psychological distress, even statistically adjusting for those variables (Baek & Yoon, 2024; Islam et al., 2022). Definitions of craving vary across studies, from visual analog ratings of desire to composite questionnaires of eating style, complicating quantitative synthesis. For these reasons, the relationships described in this review warrant the cautious language of association, and claims of proven causality would outrun the evidence. Additional limitations include the narrative-review format itself, heterogeneous sleep protocols and craving definitions, cultural differences among study populations, sex and age differences, chronotype as an uncontrolled modifier in several designs, and the scarcity of carbohydrate-specific outcome measures.

Sex differences add a further layer of complexity, since leptin responses to sleep curtailment diverge between men and women (Alfikany et al., 2025), and chronotype interacts with age, occupation, and culture in shaping social jetlag. Future research would profit from interventional trials that manipulate sleep extension or meal timing in habitual short sleepers and measure craving with harmonized instruments, from studies that examine sleep architecture beyond total duration, and from work in populations whose meal cultures differ from the Western pattern that dominates the current literature.

Practical implications should be framed as hypotheses for intervention research rather than as clinical guidance. Sleep of adequate duration and stable timing may attenuate hormonal and hedonic amplifiers of craving, and alignment of meals with the biological day may narrow the behavioral pathway. Programs of dietary change that target sugar reduction have a plausible rationale for attending to sleep hygiene, and bedtime and mealtime represent candidate targets for future controlled trials of craving management.

Bidirectionality deserves an explicit statement in the model. Sleep may shape eating through the pathways described, and eating may shape sleep in return, since late intake of large meals can disturb the night through glycemic oscillations and delayed digestion, which would close a loop capable of self-perpetuation. The loop is consistent with the clinical observation that disorders of sleep and of the eating schedule co-occur, and it implies that interventions may gain power when applied to both ends at once, a prediction open to direct experimental test.

Recommendations stratified by an individual's temporal profile represent a direction for future research rather than a validated algorithm. A rotating shift worker, an evening chronotype with pronounced weekend jetlag, and a parent with fragmented nights face different configurations of the same 3 domains, and intervention studies that tailor sleep and meal-timing strategies to such profiles would test the framework directly. The framework in Figure 2 is offered as hypothesis-generating, and its use as a diagnostic or clinical tool awaits

empirical validation.

5. Conclusion

The reviewed evidence supports an association between the state of the circadian system, the sufficiency of sleep, and the drive toward sweet and highly palatable foods. Experimental sleep restriction has been associated with reduced leptin and elevated ghrelin in early studies, with heterogeneous hormonal responses in later work, and with more consistent increases in subjective hunger and in reward responses to energy-dense food. Circadian misalignment produced by shift work and by late meal timing increases hunger and appetite-related hormone ratios under controlled conditions, while social jetlag shows observational associations with poorer diet quality. These findings converge from laboratory, occupational, and population methods, yet association predominates over demonstrated causation in most of the evidence.

Uncertainty deserves equal emphasis. Carbohydrate-specific craving has rarely been measured as a distinct outcome, samples in experimental chronobiology remain small, definitions of craving and of social jetlag vary across studies, and observational designs admit confounding and reverse causation. The integrative framework proposed here is hypothesis-generating and requires testing in interventional trials with harmonized craving instruments, larger and more diverse samples, and carbohydrate-specific outcome measures. Priorities for future research include sleep-extension and meal-timing trials in habitual short sleepers, studies of sleep architecture beyond total duration, and work in populations outside the Western meal cultures that dominate the current literature.

Declarations

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Data Availability. No new data were created or analyzed in this review; all data discussed are available in the cited publications.

Ethics Statement. Not applicable; this work is a review of previously published studies and involved no human or animal subjects.

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