RESEARCH PAPER

Ayurvedic Chrononutrition and Circadian Health: Influence of Meal Timing on Digestive and Metabolic Function, Sleep Quality and Metabolic Health

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ABSTRACT

Chrononutrition examines how the timing, regularity and distribution of food intake interact with circadian biology. Ayurveda has long treated time (kala), digestive readiness, regularity of meals and the relationship between food, sleep and daily routine as central determinants of health. Although these traditions use different explanatory frameworks, contemporary circadian physiology provides a plausible scientific basis for investigating temporal eating as a modifiable component of metabolic and sleep health. To critically integrate Ayurvedic principles of meal timing with modern evidence on circadian regulation of digestive and metabolic function, sleep quality and cardiometabolic outcomes.

Methods: A structured integrative review was undertaken using classical Ayurvedic sources and peer-reviewed human literature indexed in PubMed/MEDLINE and PubMed Central, emphasizing controlled feeding studies, randomized trials, mechanistic circadian experiments, prospective cohorts and systematic reviews/meta-analyses. Thirty core references were retained for synthesis. Numerical figures were generated only from published aggregate results; no synthetic patient-level data were created.

Human laboratory studies consistently demonstrate a time-of-day effect on glucose handling. Delaying lunch increased postprandial glucose exposure by 46% in a randomized crossover study, while a large randomized crossover experiment simulating late dinner produced an 8.3% higher glucose area under the curve and 6.7% lower insulin area under the curve.

Keywords: Ayurveda; chrononutrition; meal timing; circadian rhythm; time-restricted eating; metabolic health; sleep; glucose metabolism; Ahara Vidhi; circadian alignment

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1. Introduction

Human nutrition research has traditionally focused on what and how much people eat. During the last two decades, however, a third dimension—when food is consumed—has moved from a secondary behavioral variable to a mechanistically credible determinant of physiology. The circadian system organizes predictable 24-hour oscillations in sleep-wake behavior, endocrine secretion, autonomic activity, body temperature, gastrointestinal motility, glucose tolerance, lipid handling and

energy expenditure. Feeding is not merely an input into this system; it is also a potent timing cue for peripheral clocks. Consequently, identical meals can produce different metabolic responses when consumed at different biological times [4-7].

This temporal view of nutrition has notable parallels with classical Ayurveda. In the Ayurvedic literature, ahara (diet) is not considered solely according to ingredients. The suitability of food also depends on quantity, preparation, compatibility, the individual, season, place, digestive state and time of intake. The Charaka Samhita describes food-related factors under Ahara Vidhi and Ahara Vidhi Vishesha Ayatana, while the broader instruction to eat after the previous meal has been adequately processed places digestive readiness at the center of meal scheduling [1]. The Ashtanga Hridaya similarly emphasizes appropriate quantity, digestive capacity and avoidance of repeated intake before digestion is complete [2]. The Sushruta tradition also embeds diet within daily and seasonal regimens and recognizes that improperly timed or excessive intake can disturb physiological balance [3]. These concepts are prescriptive and arise from a different epistemological system than molecular chronobiology; nevertheless, they identify temporal regularity and digestive state as clinically meaningful features of eating behavior.

Modern circadian research gives a biological basis for asking whether such timing principles can be translated into measurable outcomes. The central circadian pacemaker in the suprachiasmatic nucleus is primarily synchronized by the light-dark cycle, whereas clocks in liver, pancreas, adipose tissue, skeletal muscle and gastrointestinal tissues are strongly influenced by feeding-fasting cycles [4-7]. When sleep-wake timing, light exposure and food intake are misaligned, central and peripheral rhythms can become internally desynchronized. Controlled laboratory circadian-misalignment protocols have shown reduced leptin, higher glucose and insulin, increased blood pressure and other adverse cardiometabolic changes [8]. Separate experiments demonstrate that the endogenous circadian phase itself modulates glucose tolerance, independent of behavior, and that misalignment adds a second metabolic burden [9]. These findings have made meal timing a potentially actionable target in obesity, diabetes prevention, shift-work medicine and sleep health.

The clinical literature, however, is not uniform. Earlier main meals have been associated with greater weight-loss success [10], and redistributing calories toward breakfast rather than dinner has produced favorable effects on weight, glucose, insulin, hunger and triglycerides in a randomized trial [11]. Delaying a standardized lunch impairs glucose tolerance and changes substrate oxidation [12], while later food intake relative to endogenous circadian timing is associated with higher body fat [13]. Yet not all time-restricted eating trials demonstrate clinically important advantages. Early time-restricted feeding can improve insulin sensitivity and other cardiometabolic markers even without weight loss [14,15], and a 10-hour eating window has shown favorable pilot results in metabolic syndrome [16]. In contrast, a noon-to-8-pm time-restricted eating intervention did not produce superior weight loss in the TREAT randomized trial [17], and a 12-month calorie-restriction trial found no statistically significant added benefit of an 8-am-to-4-pm eating window over calorie restriction alone [20]. These differences suggest that the biological timing of the eating window, the degree of energy restriction, adherence, baseline metabolic health and the comparator diet all influence outcomes.

Sleep introduces another layer. Meal timing can alter the relationship between metabolic physiology and the biological night, particularly when food intake overlaps with rising melatonin. Late eating also tends to cluster with evening chronotype, delayed sleep, irregular daily schedules and social jet lag. Controlled and observational studies suggest

that meals close to bedtime can modify sleep architecture or correlate with poorer sleep quality [22-24]. A recent scoping review found a rapidly expanding literature on chrono-nutrition and sleep, but also substantial heterogeneity in definitions and methods [26]. Thus, any modern Ayurvedic chrononutrition model should integrate food timing with sleep timing rather than treating them as independent lifestyle behaviors.

The purpose of this paper is to develop a rigorous, evidence-based synthesis of Ayurvedic temporal dietary principles and contemporary chrononutrition. The specific objectives are: (i) to identify Ayurvedic concepts relevant to meal timing without forcing one-to-one biomedical equivalence; (ii) to summarize human evidence linking meal timing with digestive and metabolic function, sleep and cardiometabolic risk; (iii) to present published numerical findings in transparent tables and graphs; and (iv) to propose a clinically testable modern Ayurvedic chrononutrition framework. The central hypothesis guiding the synthesis is that temporal regularity, adequate inter-meal recovery and avoidance of habitual late biological-night eating are shared operational themes that can be evaluated with modern biomarkers.

2. Literature Review

2.1 Ayurvedic temporal principles of eating

Ayurveda conceptualizes diet through a multidimensional framework. The Charaka Samhita does not reduce appropriate eating to a list of foods; it considers prakriti (nature of the substance), karana (processing), samyoga (combination), rashi (quantity), desha (habitat/place), kala (time), upayoga-samstha (rules of use) and upayokta (the consumer) [1]. Kala therefore operates at more than one level: time of day, season, stage of digestion, age and the temporal context of disease may all alter suitability. In the present paper, the relevant translation is not “Ayurveda prescribed a modern circadian diet,” which would be historically inaccurate, but rather that classical dietetics explicitly treated timing as a determinant of physiological response.

The instruction commonly summarized as jirne ashniyat—eat after the previous food has been digested—adds a dynamic criterion to meal timing. In practice, the decision to eat is linked to recovery of appetite and absence of signs suggesting incomplete digestion. This differs from a strict intermittent-fasting protocol because it is not defined by a fixed number of fasting hours. It also differs from calorie restriction because the primary variable is readiness for the next meal, not energy deficit. Yet the principle is relevant to modern studies of grazing, extended daily eating windows and frequent late snacks, because all concern the duration of the fed state and the opportunity for post-absorptive physiology to occur [1,2].

Matra, or appropriate quantity, is equally important. Classical texts repeatedly link excessive intake, insufficient intake and intake at an inappropriate time with impaired digestion and disturbance of physiological balance [1-3]. In a modern translation, quantity and timing cannot be separated: a large late meal presents a different metabolic challenge from a small early meal, and a 10-hour eating window may improve outcomes partly because it reduces spontaneous energy intake rather than because fasting itself exerts a unique effect. This issue is central to contemporary meta-analysis [27-29].

A second relevant theme is routine. Dinacharya describes a disciplined daily organization of waking, elimination, hygiene, physical activity, meals and sleep. Its significance for chrononutrition lies in regularity. Circadian systems respond not only to the mean time of an exposure but also to day-to-day variability. Modern observational work increasingly distinguishes meal timing, eating-window duration and eating regularity, while sleep science recognizes irregular sleep timing as a marker of circadian disruption. The conceptual bridge is therefore regular temporal patterning rather than a claim that traditional dosha time

blocks are directly equivalent to specific clock genes.

Ayurveda also links diet and sleep as foundational behaviors. Ahara and nidra are treated among major supports of health, making their interaction clinically relevant rather than incidental. A late meal can alter postprandial thermogenesis, glucose, insulin, gastrointestinal activity and autonomic state during a period when the organism is preparing for sleep. Conversely, short or delayed sleep can shift hunger hormones, food preferences and the timing of subsequent eating [24]. A modern Ayurvedic approach to chrononutrition should therefore examine the meal-sleep interval, not only breakfast, lunch and dinner clock time.

2.2 Circadian control of digestive and metabolic function

The mammalian circadian system is hierarchical. The suprachiasmatic nucleus coordinates daily timing using light as its dominant zeitgeber, while peripheral clocks respond to neural, hormonal, temperature and nutrient-related signals. Clock transcriptional networks interact with pathways controlling glucose production, insulin secretion, lipid synthesis, fatty-acid oxidation and mitochondrial function [4-6]. Feeding time can phase-shift peripheral metabolic rhythms even when the central pacemaker remains largely locked to the light-dark cycle. In healthy men, delaying all meals by five hours delayed the rhythm of plasma glucose and adipose PER2 expression without equivalently shifting the central melatonin rhythm, demonstrating that meal timing can selectively reset peripheral components of the human circadian system [7]. This separation is clinically important. When behaviors occur at an adverse circadian phase, the same nutrient load is handled under different hormonal and cellular conditions. In the classic circadian-misalignment experiment by Scheer and colleagues, subjects living on a 28-hour behavioral cycle developed higher postprandial glucose despite increased insulin, lower leptin, higher mean arterial pressure and a reversed cortisol pattern [8]. Morris and colleagues later showed that glucose tolerance is independently affected both by endogenous circadian phase and by circadian misalignment [9]. Thus, the evening disadvantage in glucose control cannot be attributed solely to behavioral choices made at night; it is partly embedded in circadian physiology.

The melatonin-insulin relationship is one mechanistic example. Melatonin rises before habitual sleep and signals biological night. Pancreatic beta cells express melatonin receptors, including MTNR1B, and glucose intake during high endogenous melatonin may be disadvantageous for insulin secretion. In 845 adults, a randomized crossover study simulating dinner one hour versus four hours before habitual bedtime found 3.5-fold higher melatonin in the late condition, 6.7% lower insulin area under the curve and 8.3% higher glucose area under the curve; the adverse glucose effect was stronger in carriers of the MTNR1B diabetes-risk allele [19]. This experiment supports the concept of “circadian-relative meal timing”: the metabolic meaning of 9 pm differs between individuals depending on sleep timing and melatonin phase.

Gastrointestinal physiology is also rhythmic. Gastric emptying, motility, digestive secretions, bile-acid metabolism and the intestinal microbiome show diurnal variation, although human evidence for an optimal digestive clock is less mature than evidence for glucose metabolism. From an integrative perspective, this is where caution is essential. Ayurvedic “digestive and metabolic function” is a broad functional construct and cannot be collapsed into glucose tolerance, gastric emptying or any single laboratory marker. A scientifically useful translation instead defines measurable domains—appetite return, gastrointestinal symptoms, postprandial glucose, insulin, lipids, substrate oxidation and bowel regularity—then tests how temporal dietary rules influence each domain.

2.3 Early versus late meals and energy distribution

Several human studies indicate that earlier energy exposure is metabolically favorable. In the Spanish weight-loss cohort, participants who ate their main meal later lost less weight despite broadly similar reported energy intake and expenditure, suggesting that meal timing may modify weight-loss efficiency or may mark other unmeasured behaviors [10]. Because this study was observational within a treatment program, causality cannot be assumed, but it provided a major stimulus for controlled chrononutrition trials.

A randomized 12-week study by Jakubowicz and colleagues compared two isocaloric 1400-kcal diets in women with overweight/obesity and metabolic syndrome. One group consumed 700 kcal at breakfast, 500 kcal at lunch and 200 kcal at dinner; the other reversed the breakfast and dinner energy loads. The high-breakfast group lost more weight and waist circumference, showed larger reductions in fasting glucose, insulin and HOMA-IR, had lower hunger, and experienced a 33.6% decrease in triglycerides, whereas triglycerides increased 14.6% in the high-dinner group [11]. Although the dietary distribution was extreme and the study population was specific, the trial directly demonstrates that calorie timing can alter cardiometabolic response even when total prescribed calories are matched.

Bandin and colleagues provided a more mechanistic test. Healthy young women underwent early-lunch and late-lunch conditions with standardized meals. Delaying lunch from 13:00 to 16:30 increased postprandial glucose area under the curve by 46%, reduced pre-meal resting energy expenditure and altered carbohydrate oxidation and cortisol-related rhythms [12]. This result is important because it isolates timing more tightly than observational studies. It also illustrates why a modern chrononutrition interpretation should focus on physiological phase rather than moralizing “late eating” as poor behavior.

Later circadian timing of food intake has also been linked with adiposity. McHill and colleagues measured food intake relative to endogenous melatonin onset and found that participants with higher body fat consumed a greater proportion of calories closer to the onset of melatonin, independent of absolute clock time and several behavioral covariates [13]. These findings are consistent with the distinction between clock time and biological time. Two people eating at 8 pm may not be metabolically equivalent if one has a 9-pm melatonin onset and the other a midnight onset. Late eating may influence both sides of energy balance. Vujovic and colleagues used an inpatient randomized crossover design in adults with overweight/obesity, holding caloric intake, diet composition, physical activity and sleep opportunity constant while shifting meals later. Late eating increased waking hunger, altered leptin/ghrelin-related appetite signaling, decreased waking energy expenditure and changed adipose-tissue gene expression in a direction consistent with greater lipid storage [21]. The strength of this study lies in its experimental control, although the intervention was short-term and cannot determine long-term weight change.

2.4 Time-restricted eating: circadian alignment versus energy deficit

Time-restricted eating (TRE) limits caloric intake to a consistent daily window without necessarily prescribing which foods or how many calories are consumed. It is attractive as a behavioral strategy because it simplifies dietary rules and creates a prolonged overnight fasting interval. From a circadian perspective, however, “TRE” is not one intervention. A 6-hour window ending at 2 pm differs biologically and socially from an 8-hour window beginning at noon. This distinction helps explain heterogeneous trial results.

Sutton and colleagues studied early time-restricted feeding in men with prediabetes using a tightly controlled crossover design. Participants consumed all food within a 6-hour window, with dinner before mid-afternoon, and

calories were matched to prevent weight loss. Early time restriction improved insulin sensitivity, beta-cell responsiveness, blood pressure and oxidative-stress markers, showing that at least some effects can occur independent of weight loss [14]. Jamshed and colleagues subsequently reported that an 8-am-to-2-pm schedule reduced 24-hour mean glucose by 4 ± 1 mg/dL and mean amplitude of glycemic excursions by 12 ± 3 mg/dL while altering circadian clock-gene expression and metabolic markers [15]. These experiments provide the clearest support for circadian alignment as a mechanism separate from simple calorie reduction.

In free-living adults with metabolic syndrome, Wilkinson and colleagues reduced a baseline eating window of at least 14 hours to a self-selected 10-hour window for 12 weeks. Participants lost approximately 3% of body weight, with reductions in waist circumference, blood pressure and atherogenic lipids and reports of more restorative sleep [16]. However, this was a small single-arm study, so regression to the mean, spontaneous calorie reduction and behavioral co-interventions cannot be excluded.

Larger randomized trials temper enthusiasm. The TREAT trial compared a noon-to-8-pm TRE schedule with a control schedule of three structured meals. Weight loss in the TRE group was modest and not significantly greater than control, and cardiometabolic benefits were limited [17]. Importantly, the eating window was shifted relatively late compared with early-TRE mechanistic studies. In a 12-month trial of 139 adults with obesity, Liu and colleagues compared calorie restriction plus an 8-am-to-4-pm eating window with calorie restriction alone. Both groups lost substantial weight, but the between-group difference was not statistically significant at 12 months [20]. These data show that timing cannot be evaluated independently of total energy intake and comparator intensity.

Meta-analyses provide a population-level estimate. Moon and colleagues synthesized 19 studies and reported modest reductions in body weight, fat mass, systolic blood pressure, fasting glucose and triglycerides with TRE [27]. Chang and colleagues concluded that TRE benefits in overweight/obesity were often strongly linked to spontaneous energy deficit, with circadian timing contributing additional but less consistently separable effects [28]. A later meta-analysis of 22 randomized trials found small reductions in BMI, body weight and fasting glucose and an increase in HDL cholesterol, while several other lipid and blood-pressure outcomes were not significantly changed [29]. The overall conclusion is therefore nuanced: TRE is a useful structure for some patients, but its benefit depends on timing, energy intake, adherence and metabolic phenotype.

2.5 Meal timing, sleep quality and the bidirectional sleep-food axis

Sleep and eating timing are bidirectionally related. Sleep restriction and circadian delay can increase appetite, shift food preference toward energy-dense foods and extend the opportunity to eat. Conversely, eating close to bedtime increases digestive and metabolic activity during the biological night and can influence sleep architecture, body temperature and autonomic physiology [24]. The practical implication is that an eating window cannot be evaluated without considering the sleep window.

St-Onge and colleagues showed experimentally that manipulating sleep and meal timing changed food intake and hormonal regulation in adults with overweight/obesity, supporting the idea that alignment of meals and sleep affects energy balance [24]. In healthy volunteers, Duan and colleagues compared a routine dinner five hours before bedtime with a late dinner one hour before bedtime. Late dinner altered sleep-stage distribution and EEG spectral characteristics, demonstrating that meal timing can affect sleep physiology even when total sleep opportunity is controlled [22]. The clinical magnitude and long-term relevance remain uncertain, but the study challenges the

assumption that a meal immediately before bed is metabolically and neurophysiologically neutral.

Observational studies provide complementary evidence. Chung and colleagues surveyed 793 young adults and found associations between eating closer to bedtime and poorer sleep-related outcomes, although cross-sectional design cannot establish direction of causality [23]. A 2024 systematic scoping review by Saidi and colleagues documented rapid growth in the field: among the included literature, 35 studies addressed meal timing, 84 irregular eating patterns and three eating frequency; within meal-timing studies, 13 examined breakfast skipping, 16 late eating and six earlier-versus-later meal schedules [26]. The review concluded that chrono-nutritional behaviors and sleep are linked, but definitions and measurement methods vary markedly.

For Ayurveda-informed research, this bidirectional axis is especially relevant because classical daily regimen integrates food and sleep within a single behavioral ecology. Rather than testing “early dinner” as an isolated rule, future trials should quantify the interval between the last caloric intake and sleep onset, bedtime regularity, chronotype, sleep duration and nocturnal awakenings. Such a design would allow traditional temporal advice to be examined with objective actigraphy, dim-light melatonin onset, continuous glucose monitoring and validated sleep questionnaires.

2.6 Chronotype, social timing and personalization

Chronotype describes an individual's preferred phase of sleep and activity, commonly ranging from morningness to eveningness. It is partly biological and partly shaped by age, light exposure, work schedules and social obligations. A systematic review by Teixeira and colleagues found that evening chronotypes were more likely to report late eating, breakfast skipping and less favorable dietary patterns; nearly half of the obesity-focused studies reported a stronger association between late chronotype and obesity [25]. Chronotype therefore acts both as a potential confounder and as a candidate effect modifier in chrononutrition research.

Personalization is necessary because a universal clock-time rule may be unrealistic or biologically imprecise. A shift worker cannot simply adopt the same meal clock times as a day worker. Chellappa and colleagues addressed this problem in a simulated night-work paradigm. Nighttime eating produced internal circadian misalignment and impaired glucose tolerance, whereas restricting food intake to daytime prevented these changes despite mistimed sleep [18]. The study provides a powerful proof of principle that behavioral timing can protect metabolism under circadian challenge, although real-world night workers face adherence, social and occupational constraints not reproduced in the laboratory.

Genetic variation may further modify response. The MTNR1B dinner-timing trial demonstrates gene-by-time interaction in glucose regulation [19]. This does not imply that routine genetic testing is currently needed for meal-timing advice; rather, it shows that biological response to “late dinner” is heterogeneous. Ayurveda's individualized orientation is conceptually compatible with this heterogeneity, but modern personalization should use measurable variables such as chronotype, sleep timing, metabolic status, medication schedule, pregnancy, age and work pattern rather than assuming that traditional constitution categories are established proxies for molecular chronotype.

A responsible modern Ayurvedic chrononutrition framework should therefore be flexible: maintain a consistent daily eating window; place a larger share of calories earlier in the individual's active phase when feasible; avoid substantial caloric intake near habitual sleep and high endogenous melatonin; allow sufficient time for postprandial digestion before the next meal; and preserve nutritional adequacy. These are hypotheses supported to different degrees by current evidence, not absolute prescriptions.

2.7 Meal spacing, digestive recovery and postprandial physiology

Meal timing is not only a question of the clock time of breakfast or dinner; the interval separating eating episodes also determines whether metabolism remains predominantly postprandial or returns toward a postabsorptive state. Repeated caloric exposure across a long waking day can prolong insulin secretion, substrate storage and gastrointestinal activity, whereas a defined daily eating window creates a longer overnight interval without energy intake [5,6,27-29]. Current evidence does not establish a universally optimal number of meals or a single ideal inter-meal interval, and the effects of meal frequency are difficult to separate from total energy intake and food quality. Nevertheless, the distinction between discrete meals and continuous grazing is biologically relevant when designing chrononutrition studies.

This issue provides one of the most useful, but also most easily overstated, points of dialogue with Ayurveda. The instruction commonly summarized as jirne ashniyat emphasizes eating when the preceding meal has been adequately digested [1,2]. In contemporary research this principle should not be converted into a fixed fasting duration, because classical digestive readiness includes subjective and contextual features that do not have a validated one-to-one biomarker. A testable translation would instead quantify the time since the preceding caloric event together with hunger, satiety, abdominal fullness, reflux or dyspepsia symptoms, bowel pattern and the return of postprandial glucose toward baseline. Continuous glucose monitoring can characterize recovery from glycemic excursions, while time-stamped dietary records can determine whether unplanned snacks extend the eating window into the biological night.

The metabolic switch associated with fasting intervals is also relevant but should be interpreted conservatively. Reviews of fasting and circadian biology describe coordinated changes in glycogen use, lipid oxidation, ketone production and cellular nutrient-sensing pathways as fasting lengthens [5,6]. These mechanisms provide biological plausibility for a daily fasting interval, yet they do not prove that the Ayurvedic concept of digestion is equivalent to a specific metabolic switch. Moreover, excessive fasting, poorly timed restriction or compensatory overeating may be inappropriate in children, pregnancy, frailty, eating disorders, or in people using glucose-lowering medication. Thus, a modern Ayurveda-informed model should operationalize meal spacing as an individualized behavioral exposure rather than a rigid rule.

Future clinical studies could add digestive endpoints to the metabolic outcomes that dominate current TRE research. A protocol might compare a regular three-meal schedule with a similar-energy schedule containing frequent caloric episodes, while holding diet composition and sleep opportunity constant. Outcomes could include continuous glucose metrics, fasting and postprandial insulin, appetite profiles, gastrointestinal symptom scales, actigraphy-derived sleep, and meal-to-bed intervals. Such designs would directly test whether structured meal spacing contributes independent benefit beyond calorie reduction and would give the classical emphasis on digestive readiness a scientifically falsifiable form.

Table 1. Functional mapping of selected Ayurvedic temporal-diet concepts to modern research constructs.

Ayurvedic concept	Modern research construct	Potential measurable variables	Interpretive caution
Kala (time/context)	Meal timing relative to clock time, circadian phase, season and sleep	Time-stamped food logs; melatonin phase; meal-sleep interval	Functional analogy only; classical kala is broader than circadian phase
Jirne ashniyat / eating after prior meal digestion	Inter-meal recovery; avoidance of continuous grazing	Inter-meal interval; hunger return; GI symptom score; CGM postprandial recovery	No single modern biomarker equals "digestion complete"
Matra (appropriate quantity)	Meal size and within-day energy distribution	Meal calories; % daily energy by meal; gastric load	Timing effects can be confounded by energy intake
Dinacharya (daily routine)	Regularity of meal and sleep timing	Day-to-day SD of first/last calorie and sleep timing	Regularity may itself be a circadian signal
Ahara-Nidra relationship	Bidirectional meal-sleep interaction	Last-calorie-to-bed interval; actigraphy; PSQI	Sleep and diet should be studied jointly

3. Materials and Methods

This paper used a structured integrative-review design rather than a de novo clinical trial. The review was designed to synthesize two evidence streams: classical Ayurvedic dietary principles relevant to temporal eating and contemporary human evidence on chrononutrition, circadian physiology, metabolic function and sleep.

Search strategy and sources. Classical concepts were identified from standard editions of the Charaka Samhita, Ashtanga Hridaya and Sushruta Samhita [1-3], focusing on passages concerning Ahara Vidhi, kala, quantity, digestion of the previous meal, daily regimen and the relationship of diet with sleep. Contemporary literature was identified through targeted searches of PubMed/MEDLINE and PubMed Central, supplemented by publisher records for bibliographic verification. Searches were updated through 10 August 2026. Search strings combined terms including "chrononutrition," "meal timing," "circadian," "early eating," "late eating," "dinner timing," "breakfast," "time-restricted eating," "time-restricted feeding," "glucose tolerance," "insulin sensitivity," "metabolic syndrome," "obesity," "sleep," "chronotype," "melatonin," "shift work" and "circadian misalignment."

Eligibility. Priority was given to human randomized controlled trials, controlled crossover feeding studies, circadian laboratory

experiments, prospective or well-characterized observational studies, systematic reviews and meta-analyses. Animal studies were used only indirectly through mechanistic reviews and were not used to generate clinical effect estimates. Studies were included when meal timing, daily eating-window duration, timing relative to sleep/circadian phase or distribution of energy across the day was a central exposure or intervention and when outcomes included glucose, insulin, body weight, lipids, blood pressure, energy expenditure, appetite, sleep or circadian markers. Studies focused exclusively on alternate-day fasting or multiday fasting without a meal-timing component were not emphasized. Thirty references were retained as the core evidence set for the present synthesis.

Data extraction and synthesis. For experimental studies, the extracted fields included population, sample size, intervention or timing contrast, duration, major metabolic or sleep outcomes and direction of effect. For systematic reviews/meta-analyses, the number of included studies or trials, pooled effect estimates and stated limitations were recorded where available. The synthesis was narrative because the interventions and outcomes were too heterogeneous for a valid new pooled analysis. No unpublished data, simulated participants or reconstructed individual-level datasets were used.

Graph construction. Figures were generated from numerical values explicitly reported in the cited publications. Figure 1 uses the reported percentage change in glucose area under the curve with late versus early meal timing in Bandin et al. [12] and Garaulet et al. [19]. Figure 2 uses the published changes in 24-hour mean glucose and mean amplitude of glycemic excursions with early time-restricted feeding from Jamshed et al. [15]. Figure 3 compares pooled body-weight differences reported by Moon et al. [27] and Qi et al. [29]. Figure 4 reproduces the number of studies across chrono-nutritional dimensions reported by Saidi et al. [26]. Figure 5 displays the published triglyceride changes in the high-breakfast and high-dinner arms of Jakubowicz et al. [11]. These plots are descriptive and should not be interpreted as a new meta-analysis.

Integrative mapping. Ayurvedic concepts were mapped to modern research constructs only when the mapping could be stated as a functional analogy. For example, kala was mapped to temporal exposure, and eating after digestion of the previous meal was mapped to inter-meal recovery/fasting interval. Agni was not treated as identical to metabolic rate, insulin sensitivity, mitochondrial function or the gut microbiome. This distinction was maintained to avoid category errors and retrospective overinterpretation.

4. Results

The 30-reference core evidence base comprised three classical Ayurvedic sources [1-3], mechanistic circadian reviews and experiments [4-9], human meal-timing and energy-distribution studies [10-13,19,21], time-restricted eating trials [14-17,20], a simulated night-work intervention [18], sleep-related studies [22-24], chronotype and sleep evidence syntheses [25,26] and systematic reviews/meta-analyses of time-restricted eating or breakfast omission [27-30]. The modern evidence base was heterogeneous in population, duration, eating window and outcome definition; consequently, findings were interpreted by domain rather than collapsed into a single effect estimate.

The first major result was a consistent direction of acute glycemic response: later eating tended to worsen glucose handling. Delaying lunch from 13:00 to 16:30 increased glucose area under the curve by 46% [12]. In the large dinner-timing crossover study, late simulated dinner increased glucose AUC by 8.3% and reduced insulin AUC by 6.7% while endogenous melatonin was 3.5-fold higher [19]. In simulated night work, daytime eating prevented the glucose intolerance and internal circadian misalignment observed with nighttime eating [18]. These findings triangulate across ordinary daytime meal delay, pre-sleep dinner timing and circadian inversion.

The second major result was that early TRE can produce metabolic effects without substantial weight loss under controlled conditions. Sutton et al. observed improvements in insulin sensitivity and blood pressure during early time-restricted feeding despite isocaloric intake [14]. Jamshed et al. reported a 4 mg/dL reduction in 24-hour mean glucose and a 12 mg/dL reduction in glycemic excursions [15]. These results support a time-of-day mechanism, although the studies were small and short.

The third result was that longer-term weight outcomes are more modest and inconsistent. Wilkinson et al. reported approximately 3% weight loss in a small single-arm 10-hour TRE study in metabolic syndrome [16]. The TREAT randomized trial found no significant superiority of a noon-to-8-pm TRE schedule for weight loss compared with structured meals [17]. Liu et al. found an 8.0-kg mean weight loss with TRE plus calorie restriction versus 6.3 kg with calorie restriction alone at 12 months, but the net difference was not statistically significant [20]. Meta-analytic estimates are correspondingly modest: Moon et al. reported a mean body-weight reduction of 0.90 kg [27], whereas Qi et al. reported a 0.48-kg reduction across 22 randomized trials [29]. Chang et al. emphasized that spontaneous energy deficit explains an important part of TRE-associated weight loss [28].

A fourth result concerned energy distribution. The high-breakfast intervention produced a 33.6% decrease in triglycerides compared with a 14.6% increase in the high-dinner group, alongside greater weight loss and better glycemic outcomes [11]. This supports the idea that the distribution of calories across the day may matter separately from eating-window duration.

Sleep-related findings were suggestive but less uniform than glycemic findings. Late dinner altered sleep-stage distribution and EEG spectral power in a controlled trial [22], and eating close to bedtime was associated with poorer sleep outcomes in young adults [23].

The broader scoping review showed that the field is dominated by studies of irregular eating patterns and meal timing, with relatively few studies isolating eating frequency [26]. Because sleep timing can influence eating and vice versa, causal direction remains difficult to establish in observational cohorts.

Finally, the integrative mapping showed meaningful conceptual convergence at the level of behavior: regular meal timing, avoidance of eating before recovery from the previous meal, appropriate quantity, and coordination of meals with the daily sleep-wake cycle are compatible with current chrononutrition hypotheses. However, there is not yet direct clinical evidence that a complete classical Ayurvedic temporal eating protocol improves circadian biomarkers more than a modern chrononutrition protocol. This remains a research question rather than an established conclusion.

Table 2. Representative human studies of meal timing and metabolic health.

Study	Population	Timing intervention/exposure	Principal finding	Key limitation
Garaulet et al. 2013 [10]	420 adults in weight-loss program	Earlier vs later main meal	Late eaters lost less weight	Observational timing within intervention; residual confounding

Study	Population	Timing intervention/exposure	Principal finding	Key limitation
Jakubowicz et al. 2013 [11]	93 women with metabolic syndrome	700-kcal breakfast vs 700-kcal dinner	Greater weight loss and glycemic benefit with large breakfast; TG -33.6% vs +14.6%	Population-specific; extreme energy distribution
Bandín et al. 2015 [12]	32 healthy women	Lunch 13:00 vs 16:30	Late lunch increased glucose AUC 46% and altered substrate oxidation	Short crossover study
Sutton et al. 2018 [14]	8 men with prediabetes	6-h early TRF vs control	Improved insulin sensitivity and BP without weight loss	Very small controlled trial
Jamshed et al. 2019 [15]	11 adults with overweight	08:00-14:00 vs 08:00-20:00	24-h glucose -4 mg/dL; MAGE -12 mg/dL	4-day intervention
Wilkinson et al. 2020 [16]	19 adults with metabolic syndrome	Self-selected 10-h TRE, 12 weeks	~3% weight loss; lower waist, BP and atherogenic lipids	Single-arm pilot
Lowe et al. 2020 [17]	116 adults with overweight/obesity	12:00-20:00 TRE vs structured meals	No significant superiority for weight loss	Late eating window; remote design
Chellappa et al. 2021 [18]	19 participants in simulated night work	Nighttime eating vs daytime-only eating	Daytime eating prevented glucose intolerance/internal misalignment	Laboratory shift-work simulation

Study	Population	Timing intervention/exposure	Principal finding	Key limitation
Garaulet et al. 2022 [19]	845 adults	Dinner 1 h vs 4 h before bedtime	Late dinner: glucose AUC +8.3%; insulin AUC -6.7%	OGTT simulation of dinner; genotype interaction
Liu et al. 2022 [20]	139 adults with obesity	TRE + calorie restriction vs calorie restriction	No significant added 12-month weight-loss benefit	Both groups received intensive calorie restriction
Vujović et al. 2022 [21]	16 adults completed both arms	Earlier vs later isocaloric meals	Late eating increased hunger, lowered energy expenditure, altered adipose pathways	Short inpatient crossover

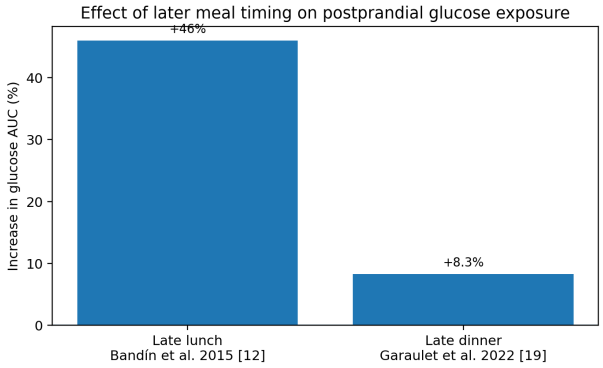

Figure 1. Published percentage increase in postprandial glucose area under the curve with later meal timing. Values are directly reported by Bandín et al. [12] and Garaulet et al. [19]; the studies differ in design and should not be pooled.

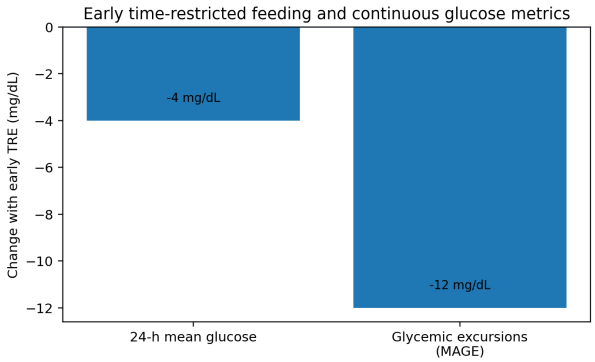

Figure 2. Change in continuous glucose-monitoring metrics during early time-restricted feeding versus control in Jamshed et al. [15]. Negative values indicate lower glucose exposure.

Table 3. Representative evidence linking meal timing and sleep.

Study	Design	Timing variable	Main sleep-related result
St-Onge et al. 2019 [24]	Experimental manipulation of sleep/meal timing	Food intake and hormonal regulation	Alignment of sleep and meals influenced intake/hormonal responses

Study	Design	Timing variable	Main sleep-related result
Chung et al. 2020 [23]	Cross-sectional survey, n=793 young adults	Meal proximity to bedtime	Closer eating to bedtime associated with less favorable sleep outcomes
Duan et al. 2021 [22]	Controlled dinner-timing study	Dinner 5 h vs 1 h before bed	Late dinner altered sleep-stage distribution and EEG power
Saidi et al. 2024 [26]	Systematic scoping review	Meal timing, irregular patterns, frequency	Evidence suggests chrono-nutrition/sleep association but marked heterogeneity

Table 4. Evidence-informed components of a modern Ayurvedic chrononutrition model.

Component	Current evidence level	Evidence basis
Avoid large habitual meals near sleep	Moderate-to-strong mechanistic support	Late dinner worsens glucose handling [19]; late eating affects appetite/energy expenditure [21]; sleep effects reported [22,23]
Prefer earlier/daytime energy loading when feasible	Moderate	High-breakfast and early-lunch evidence [11,12]; early TRE studies [14,15]
Use a consistent eating window	Moderate	Human TRE trials and meta-analyses [16,17,27-29]
Maintain adequate inter-meal intervals	Plausible; direct evidence limited	Consistent with Ayurveda [1,2] and shortened eating-window literature; needs dedicated trials
Personalize to chronotype and work schedule	Moderate	Chronotype review [25]; simulated night-work intervention [18]; MTNR1B interaction [19]
Do not treat timing as substitute for calorie/diet quality	Strong	Long-term RCT and meta-analysis show energy intake materially influences outcomes [20,28]

5. Discussion

The present synthesis indicates that meal timing is biologically consequential, but the clinically relevant question is more sophisticated than "early is good and late is bad." Human metabolism is rhythmic, food is a timing cue for peripheral clocks, and the biological response to a meal depends on its relationship to circadian phase, sleep, prior fasting duration and energy load [4-9]. The strongest evidence concerns glucose regulation: controlled studies repeatedly demonstrate poorer glucose handling when identical or standardized nutrient loads are moved later in the day or into the biological night [12,18,19]. This mechanistic consistency gives modern credibility to the broader Ayurvedic idea that kala and digestive state modify the effect of food [1-3].

At the same time, a direct equivalence between Ayurveda and chronobiology would be scientifically inappropriate. Agni is a classical functional construct encompassing aspects of digestion, transformation and tissue nutrition within Ayurvedic theory. It is not a synonym for resting metabolic rate, insulin sensitivity or mitochondrial oxidative capacity. Likewise, dosha time periods should not be presented as established surrogates for melatonin, cortisol or clock-gene phases. A modern Ayurveda research program should instead operationalize traditional instructions into testable behaviors and measure contemporary outcomes. For example, "eat after the previous meal is digested" could be translated into an intervention that avoids grazing and establishes a reproducible inter-meal interval, while collecting gastrointestinal symptom scores, hunger ratings, continuous glucose profiles and metabolomic markers.

The distinction between early and late time-restricted eating is particularly important. The favorable metabolic findings in Sutton and Jamshed used early windows that ended in the afternoon [14,15]. By contrast, the TREAT trial used a noon-to-8-pm window and did not demonstrate superior weight loss [17]. These studies should not be interpreted as contradictory tests of one identical intervention. They test different circadian exposures. Similarly, the 12-month trial by Liu et al. shows that when both groups receive substantial calorie restriction and weight-management support, adding an early 8-am-to-4-pm window may not yield a large additional weight-loss effect [20]. Thus, TRE may be most useful as a behavioral architecture that influences energy intake, eating regularity and circadian alignment rather than as a universally superior diet.

6. Conclusion

Ayurvedic chrononutrition can be formulated as a scientifically testable approach to temporal eating rather than as a literal translation of classical physiology into modern molecular terms. Classical Ayurvedic dietetics recognizes time of intake, adequate digestion of the previous meal, appropriate quantity and disciplined daily routine as determinants of dietary suitability [1-3]. Contemporary circadian science independently demonstrates that meal timing alters peripheral clocks, glucose tolerance, hormonal responses, substrate utilization, appetite, energy expenditure and the interaction between food intake and sleep.

Across the modern human evidence, the most reproducible signal is that substantial food intake late in the day or near the biological night is less favorable for glucose regulation than earlier intake. Early time-restricted feeding can improve glycemic and cardiometabolic markers, but its long-term benefits are modest on average and are not consistently greater than those achieved by equivalent calorie restriction. Therefore, meal timing should complement not replace diet quality, energy balance, physical activity and sleep health.

A practical evidence-based model emphasizes regular meal timing, avoidance of habitual late-night caloric intake, a meaningful interval between the final meal and sleep, sufficient time between meals, and a tendency to place a greater proportion of daily energy in the earlier

active phase when feasible. Personalization according to chronotype, work schedule, metabolic disease, medication use and life stage is essential. The next phase of research should directly compare Ayurveda-informed temporal eating protocols with contemporary chrononutrition interventions using objective circadian, metabolic, sleep and gastrointestinal outcomes. Such trials would provide the necessary evidence to determine whether traditional temporal dietary principles offer clinically meaningful benefits beyond established effects of calorie intake and conventional lifestyle modification.

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