CHRONONUTRITION AS A FACTOR IN THE REGULATION OF METABOLISM AND HUMAN HEALTH (LITERATURE REVIEW)

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ABSTRACT

Background. The global epidemic of obesity and metabolic disorders necessitates a reassessment of traditional dietary approaches. Chrononutrition, which investigates the impact of meal timing on health, is emerging as a novel paradigm, as the misalignment ("chronodisruption") of internal biological clocks is an independent risk factor for the development of type 2 diabetes mellitus and cardiovascular diseases.

Aim. To synthesize and analyze current scientific data on the impact of meal timing on metabolic processes and key human health metrics, with a particular focus on the clinical application of the Time-Restricted Eating (TRE) strategy and personalized approaches.

Materials and Methods. A search of publications was conducted in the PubMed, Cochrane Library, and Google Scholar bibliographic databases using the keywords *chrononutrition*, *circadian rhythm*, *meal timing*, and *time-restricted eating*. The analysis included reviews, meta-analyses, and randomized controlled trials involving human subjects published from 2015 to 2025. The research was carried out as a private initiative of the authors, without grant support and state registration of the topic.

Research Ethics. This work is a literature review; no original research was conducted. Only those publications whose authors confirmed adherence to international ethical standards, particularly the provisions of the World Medical Association Declaration of Helsinki (1964–2024), and had obtained approval from ethics committees were selected for analysis.

Results. The body's metabolic efficiency, including insulin sensitivity and the diet-induced thermogenesis, peaks in the morning and declines in the evening. The TRE strategy demonstrates clinical efficacy in improving cardiometabolic outcomes, including body weight and blood pressure. Early TRE (eTRE) has been found to be significantly more effective for improving insulin sensitivity than late TRE (lTRE), and these benefits may be independent of weight loss. The effectiveness of these approaches depends on individual chronotype and requires adaptation for specific groups (e.g., shift workers, the elderly). The underlying mechanisms of chrononutrition effects include the modulation of gut microbiome diurnal rhythms and epigenetic regulation.

Conclusions. Meal timing represents a powerful, independent determinant of metabolic health. The integration of chrononutrition principles, particularly shifting caloric intake to the first half of the day and implementing eTRE, is a scientifically substantiated strategy for the prevention and management of metabolic disorders, opening a new era in dietetics.

Keywords: *endocrinology, circadian rhythms, time-restricted eating, insulin resistance, chronotype.*

Abbreviations

CCGs – Clock-Controlled Genes.
DIT – Diet-Induced Thermogenesis.
eTRE – early Time-Restricted Eating.
lTRE – late Time-Restricted Eating.
RCTs – Randomized Controlled Trials.
SCN – SupraChiasmatic Nuclei.
T2DM – Type 2 Diabetes Mellitus.
TIR – Time In Range.
TRE – Time-Restricted Eating.
TTFL – Transcription-Translation Feedback Loop.

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Introduction

The global epidemic of obesity and its associated metabolic disorders, such as T2DM and cardiovascular diseases, remains one of the most acute public health challenges of the 21st century. Traditional dietary approaches, which for decades have focused on the quantity of calories consumed and the qualitative composition of macronutrients,

have failed to halt the rising prevalence of these diseases. This has indicated an urgent need to find new paradigms, as emphasized by Johnston J.D. et al. (2016) [1]. Recent scientific research has provided compelling evidence that the circadian system, or the internal biological clock, plays a fundamental role in metabolic regulation, as detailed by Reinke H. & Asher G. (2019) [2].

Against this backdrop, chrononutrition has emerged and is rapidly developing – a scientific discipline that studies the third, previously underestimated, dimension of nutrition: timing, as noted by Raji O. et al. (2024) [3]. It is based on the premise that not only what we eat, but also when we eat, has a crucial impact on metabolism and health, a central thesis in the work of Zarrinpar A. et al. (2016) [4]. The modern lifestyle, characterized by round-the-clock access to food and the prevalence of shift work, leads to a state known as "chronodisruption", a concept introduced by Panda S. et al. (2002) [5]. This state represents a misalignment between internal biological rhythms and external cues (*Zeitgebers*), particularly meal timing. This 'internal misalignment' between the central and peripheral clocks is considered an independent risk factor for the development of insulin resistance, obesity, and T2DM, as summarized by Mentzelou M. et al. (2024) [6].

Thus, understanding the mechanisms by which meal timing modulates metabolic processes opens new therapeutic and preventive opportunities. This review aims to systematize and analyze current scientific data regarding the impact of meal timing on metabolic processes, hormonal regulation, and key human health metrics. Particular emphasis is placed on the clinical application of chrononutrition strategies, such as TRE, and on emerging areas of research, including the personalization of approaches based on individual chronotype, application in specific populations, and the in-depth study of the fundamental mechanisms underlying these effects.

Materials and Methods

A comprehensive search of scientific publications was conducted in the international bibliographic databases PubMed, Cochrane Library, and Google Scholar. The search strategy included the following keywords: *chrononutrition*, *circadian rhythm*, *meal timing*, *time-restricted eating*, *metabolism*, *postprandial glucose*, *insulin sensitivity*, *cortisol*, and *melatonin*. The analysis included systematic reviews, meta-analyses, and randomized controlled trials involving human subjects, published between January 1, 2015 and June 30, 2025. Animal studies were considered exclusively to explain fundamental biological mechanisms. Exclusion criteria included publications irrelevant to the review topic, case reports, and letters to the editor.

Research Ethics

This work is a literature review; therefore, no original research involving human subjects or animals was conducted. Only publications whose authors confirmed adherence to international and national ethical standards in conducting their research were selected for analysis. Specifically, statements confirming compliance with the provisions of the World Medical Association Declaration of Helsinki (1964–2024) and approval from local ethics committees, a mandatory requirement for clinical research, were verified.

Results

Fundamentals of circadian biology and metabolism

A complex hierarchical system of biological clocks underlies the diurnal organization of human physiology. At the apex of this hierarchy is the central, or master, clock, located in the SCN of the anterior hypothalamus. The SCN functions as the body's master conductor, generating an endogenous rhythm with a period of approximately 24 hours, as noted by Poggiogalle E. et al. (2018) [7]. This central clock synchronizes with the external environment, primarily with the light/dark cycle. Light is the most potent *Zeitgeber* (from German, "time giver") for the SCN [3]. However, the SCN is merely the tip of the iceberg, as virtually every cell in the body, especially in metabolically active organs such as the liver, pancreas, and skeletal muscles, possesses its own autonomous molecular clock, known as a peripheral clock. The SCN coordinates the function of these numerous peripheral clocks through a complex network of neural and humoral signals, ensuring the coherent operation of all bodily systems [7].

At the molecular level, each cellular clock operates based on a TTFL. As described in the work of Panda S. et al. (2002) [5], the heterodimer of transcription factors CLOCK and BMAL1 forms the positive limb of the loop, activating transcription of the negative limb genes – Period (PER) and Cryptochrome (CRY). Throughout the day, PER and CRY proteins accumulate in the cytoplasm, return to the nucleus, and inhibit CLOCK:BMAL1 activity, thereby switching off their own transcription in a cycle that takes approximately 24 hours [5]. These clock genes regulate the diurnal expression of thousands of other genes – CCGs – that

directly govern key metabolic pathways, as studied in detail by Reinke H. & Asher G. (2019) [2].

While light is the primary synchronizer for the central clock, meal timing is the most potent synchronizer for peripheral clocks [7]. Food intake triggers a cascade of signals that "reset" the clocks in the liver and adipose tissue, allowing the body to synchronize metabolic activity with nutrient availability [4]. When an individual consumes food at a time misaligned with the central clock, such as late at night, misalignment occurs. This "internal misalignment" between central and peripheral rhythms acts as a chronic stressor on the metabolic system, forcing organs to perform tasks at a time when their genetic program is set for rest. Over time, this state can lead to insulin resistance and other metabolic diseases [6]. As illustrated in the conceptual framework (*Fig.*), aligned environmental and nutritional cues promote metabolic homeostasis, whereas circadian misalignment (chronodisruption) acts as a critical driver toward obesity, dyslipidemia, and metabolic syndrome.

Diurnal dynamics of hormones and their impact on metabolism

The endocrine system is the primary executive mechanism by which the circadian system regulates metabolism. The levels of most hormones exhibit distinct 24-hour oscillations, enabling the body not merely to react to changes but also to anticipate them, preparing for cyclical events. The body's physiology operates on the principle of a "metabolic efficiency curve", which peaks in the first half of the day and declines in the evening. Disruption of these rhythms, particularly through sleep deprivation or chronodisruption, has a significant impact on the hormonal profile and metabolism, as noted by Kim T.W. et al. (2015) [8].

Cortisol, known as the "activity hormone", begins to rise in the latter half of the night, reaching its peak early in the morning. This morning surge mobilizes energy reserves by stimulating gluconeogenesis and prepares the body for daytime activity and efficient nutrient assimilation [7]. In contrast, the synthesis of melatonin, the "sleep hormone", is stimulated by darkness and suppressed by light. Its concentration increases in the evening and peaks at night, inducing sleep [8]. Importantly, melatonin also directly affects metabolism by inhibiting insulin secretion from pancreatic β-cells. Consequently, food intake, especially high-carbohydrate meals, against a backdrop of high melatonin levels, leads to a significant, albeit transient, impairment of glucose tolerance.

Peripheral tissue insulin sensitivity is also subject to circadian rhythms: it peaks in the morning and gradually decreases throughout the day, reaching a minimum in the evening. This is confirmed by clinical studies that have demonstrated that an identical meal elicits a significantly higher glycemic and insulinemic response at 20:00 than at 08:00, as shown by Leung G.K.W. et al. (2019) [9]. Similarly, DIT – the energy expended on the digestion and assimilation of nutrients – also has a pronounced diurnal rhythm. In a study by Rich-

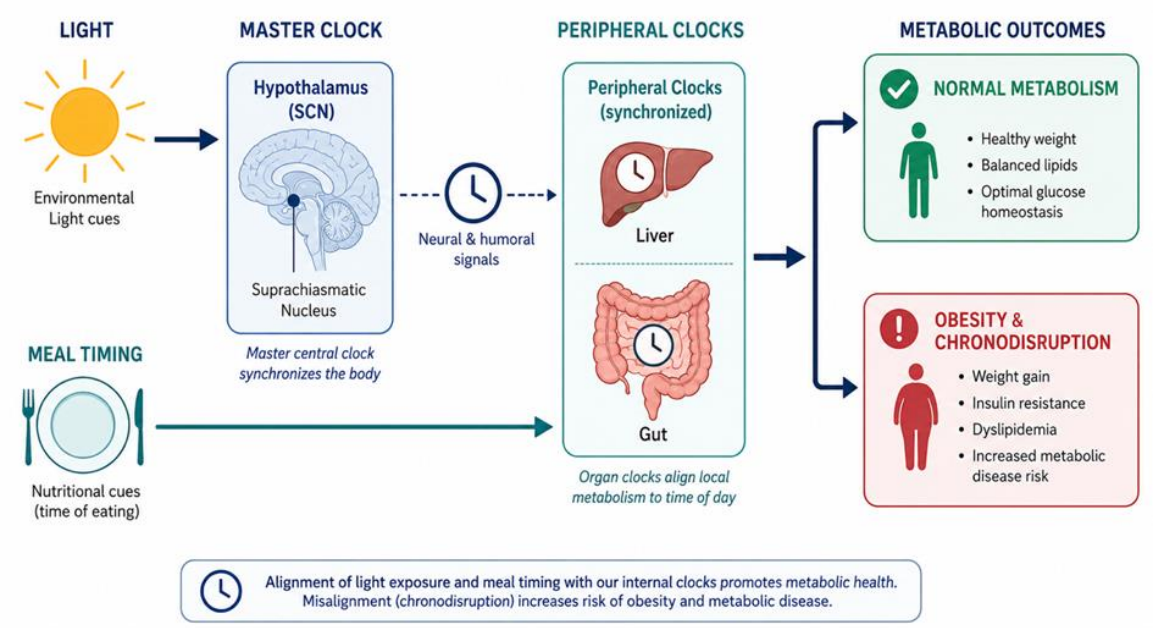

Fig. The interplay between central and peripheral circadian clocks, light exposure, meal timing, and metabolic outcomes.

ter J. et al. (2020) [10], it was established that an isocaloric meal consumed for breakfast induced a twofold higher DIT than the same meal consumed for dinner. This implies that the body expends significantly more energy processing food in the morning.

Even appetite hormones are subject to diurnal fluctuations. Ghrelin levels, the "hunger hormone", typically rise before anticipated meals, whereas leptin, the "satiety hormone", peaks at night, helping to suppress appetite during sleep, as noted by Martchenko A. et al. (2020) [11]. The combination of reduced insulin sensitivity, melatonin-induced suppression of insulin secretion, and low DIT results in a "double metabolic hit" with evening food intake. This creates conditions for hyperglycemia and hyperinsulinemia, which stimulate lipogenesis and promote the accumulation of visceral fat [7].

Discussion

Time-restricted eating as a clinical strategy

Given the importance of meal timing, practical dietary strategies have been developed, among which Time-Restricted Eating (TRE) has gained the most popularity. TRE is a dietary strategy that limits daily food consumption to a specific "eating window", typically ranging from 4 to 10 hours, followed by an extended fasting period, as noted by Ganesan K. et al. (2018) [12]. The key concept of TRE is not primarily conscious caloric restriction. However, this often occurs involuntarily, but rather the restoration and reinforcement of circadian synchronization between the central and peripheral clocks. An extended overnight fasting period allows peripheral clocks to synchronize with the central rhythm, thereby restoring metabolic homeostasis [7].

Numerous RCTs and their meta-analyses confirm the clinical efficacy of TRE for improving metabolic health. Specifically, meta-analyses, such as the one conducted by Chen W. et al. (2023) [13], demonstrate that TRE is an effective strategy for modest reduction s in body weight and fat mass. However, some studies indicate that without additional caloric control, the effect of TRE on weight loss may not differ from a control group, highlighting the importance of circadian mechanisms rather than just passive calorie reduction, as pointed out by Yuan X. et al. (2022) [14]. Beyond its impact on weight, systematic reviews document the positive effects of TRE on key cardiometabolic markers, including reductions in systolic blood pressure, fasting glucose, insulin, and triglycerides, as summarized in the meta-analysis by Liu L. et al. (2022) [15]. In contrast, data regarding the impact of TRE on systemic inflammatory markers, such as C-reactive protein, are ambiguous. As noted in the review by Mulas A. et al. (2023) [16], most studies do not find a significant impact, suggesting that the anti-inflammatory effect may be primarily a consequence of weight loss rather than a specific effect of the timing regimen.

TRE demonstrates particularly significant results in populations with pre-existing metabolic disorders, indicating that this strategy may be more than just a weight-loss method, but rather a "circadian alignment therapy". Key evidence for this comes from the study by Sutton E.F. et al. (2018) [17], in which men with prediabetes followed an eTRE protocol with a 6-hour eating window (dinner before 15:00) under eucaloric conditions. Importantly, even without weight loss, they exhibited significant improvements in insulin sensitivity pancreatic β-cell function, reduced blood pressure, and markers of oxidative stress. This study was the first to clearly demonstrate that the metabolic benefits of TRE are not solely a consequence of weight loss but rather result from a direct circadian mechanism [17].

These findings were confirmed and expanded in a recent meta-analysis by Nam T. et al. (2025) [18], which included RCTs involving patients with T2DM and impaired fasting glycemia. The analysis showed that TRE leads to clinically significant improvements in glycemic control, including reductions in fasting glucose, glycated hemoglobin (HbA1c), and, importantly, an increase in Time in Range (TIR). An increase in TIR indicates better glycemic stability throughout the day and is a robust advantage of TRE [18]. Long-term observations by Rastogi S. et al. (2024) [19] also confirm that an early dinner is an effective strategy for improving glycemic control and weight in patients with T2DM.

Recent data emphasize that not only the duration but also the timing of the "eating window" itself is crucial. A comparison of the effectiveness of eTRE versus lTRE restriction was the focus of a network meta-analysis by Liu J. et al. (2023) [20]. It was established that although weight loss may be similar between the two approaches (e.g., an 8:00 to 16:00 window vs. a 12:00 to 20:00 window), eTRE is significantly more effective at improving insulin sensitivity and reducing insulin resistance. This is explained by the fact that eTRE synchronizes food intake with the morning peak of the body's metabolic efficiency. The benefits of

an early window are evident even in specific groups, as demonstrated by Jamshed H. et al. (2022) [21], where an early window (8:00–14:00) led to significantly greater weight loss compared to a late window (12:00–18:00) in adults combining TRE with resistance training.

Personalized approaches in chrononutrition

Accumulating data indicate that the effectiveness of chrononutrition strategies can substantially depend on individual biological characteristics and lifestyle, necessitating a transition from general recommendations to personalized approaches. One of the key individual factors is chronotype – an innate predisposition of an individual to specific diurnal activity, which determines one's preferences for sleep and wake times. Research indicates that the evening chronotype ("owls") is an independent risk factor for the development of metabolic disorders. As demonstrated by Al-Lesani A. et al. (2025) [22], individuals with an evening chronotype exhibit a direct correlation between higher dinner calorie intake and a higher body mass index, as well as between a longer "eating window" duration and higher fasting glucose levels. "Owls" are more likely to skip breakfast, consume the majority of their daily calories in the evening, and have a higher risk of developing T2DM compared to "larks". An important exacerbating factor is "social jetlag" – a mismatch between the "owl's" biological rhythm and a socially imposed schedule, which intensifies circadian misalignment.

Specific populations whose lifestyles create extreme conditions for the circadian system require special attention. Shift workers, particularly those on night shifts, are a model of chronic chronodisruption. They are forced to be active and eat at night – during a period when their circadian system is programmed for sleep and restoration. This leads to a significant increase in the risk of obesity, metabolic syndrome, and T2DM, as nocturnal food intake occurs against a backdrop of physiologically low glucose tolerance. Chrononutrition strategies for this group should aim to minimize harm, for example, by consolidating food intake during the daytime, even on night shift days, and choosing light, low-glycemic snacks at night.

For athletes, balancing body composition optimization maintaining high physical performance is key. Systematic reviews and meta-analyses show that combining TRE with regular training leads to a significant reduction in fat mass, while most physical performance metrics, such as aerobic endurance and strength, do not deteriorate. As noted in their review by Tinsley G.M. & Paoli A. (2019) [23], TRE can be an effective tool for athletes. Potential mechanisms include adaptive changes in energy substrate utilization, particularly more efficient fat oxidation, which may be beneficial for endurance sports, as indicated by Parr E.B. et al. (2020) [24].

In older adults, the risk of sarcopenia – the progressive loss of muscle mass and function – increases. To combat this, consuming sufficient protein is crucial. At first glance, TRE might seem incompatible with these recommendations. However, studies show that with proper planning, an 8-hour TRE window comfortably allows for 2–3 meals with adequate protein portions. There is also a hypothesis that the fasting periods within TRE may stimulate autophagy, which improves the quality of muscle cells, potentially slowing their aging, as discussed in the work by Chaix A. et al. (2019) [25].

Advanced interplay mechanisms: from the epigenome to the microbiome

The effects of chrononutrition are mediated by a complex network of molecular mechanisms that translate a behavioral intervention into stable physiological changes. Two of these mechanisms that are drawing increasing attention are interactions with the gut microbiome and epigenetic modulation.

The gut microbiome, composed of trillions of microorganisms, is not a static system. Its composition and functional activity exhibit distinct diurnal oscillations that synchronize with the host's circadian rhythms. The most potent synchronizer of microbial rhythms is not light but rather meal timing, as demonstrated by Thaiss C.A. et al. (2016) [26]. The relationship between meal timing, the microbiome, and host metabolism is bidirectional. On one hand, regular daytime food intake, as with TRE, creates a predictable "feast-famine" cycle for the microbes, which restores their diurnal rhythmicity. On the other hand, a rhythmic microbiota produces metabolites, such as short-chain fatty acids, which enter the bloodstream and act as signaling molecules that influence clock gene expression in the host's peripheral organs, particularly the liver [26]. Thus, TRE restores the rhythmicity of the microbiome, which, in turn, generates biochemical signals that reinforce the host's circadian rhythms.

Epigenetics studies the mechanisms that regulate gene activity without altering the DNA sequence itself, particularly through DNA methylation and histone modifications. A key point is that

the enzymes performing these epigenetic modifications use metabolites produced during metabolism (e.g., Acetyl-CoA) as substrates and cofactors. As noted by de Oliveira Melo N.C. et al. (2024) [27], the levels of these metabolites in the cell fluctuate throughout the day and change sharply after food intake. Thus, meal timing directly impacts the availability of "building blocks" for epigenetic marks. Eating at a biologically appropriate time provides these substrates when the cellular machinery is ready to use them correctly to maintain the normal rhythm of metabolic gene expression. Conversely, eating late at night can lead to aberrant epigenetic "marking" and disruption of the diurnal gene expression program, a profound mechanism by which chrononutrition can have long-term effects on metabolic health [27].

Practical implications and future directions

Based on the analyzed data, multilevel practical strategies can be formulated to mitigate the risks of metabolic diseases through chrononutrition. For the general population, the core guidelines involve shifting the primary daily caloric load to the first half of the day and scheduling the last meal at least 3–4 hours before sleep onset, aligning food intake with the physiological peaks of insulin sensitivity and diet-induced thermogenesis. The clinical implementation of TRE, particularly within an 8-10- hour eating window in the form of eTRE, serves as an effective, accessible strategy for weight management and improving insulin sensitivity.

However, successful implementation requires strict personalization. For shift workers, interventions should focus on harm minimization by consolidating food consumption during daylight hours and avoiding high-glycemic or heavy meals overnight. For older adults, special clinical caution must be maintained to ensure adequate dietary protein intake within the restricted eating window to prevent the development or exacerbation of sarcopenia.

Conclusions

Meal timing is a potent, independent determinant of metabolic health, acting via the synchronization of peripheral cellular molecular clocks with the master central clock and endogenous endocrine rhythms.

Restricting the daily eating window through early time-restricted eating aligns nutrient availability with the morning peak of human metabolic efficiency, providing distinct therapeutic and preventive benefits for systemic insulin resistance and sustained metabolic stability, which can occur independently of passive calorie restriction or weight loss.

Future research must prioritize long-term (over 1 year) randomized controlled trials to evaluate the sustainability of TRE on hard clinical endpoints, directly compare different eating window schedules, and establish targeted chrononutrition protocols optimized for individual chronotypes, age-specific groups, and severe chronodisruption models such as shift work.

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Authors' Contributions

Contribution / Authors	A	B	C	D	E	F
Zavgorodnii I.V.	+	+			+	+
Lysak M.S.		+	+	+	+	+
Merkulova T.V.					+	+
Bilychenko N.P.					+	+
Litovchenko O.L.					+	+

Notes: A – concept; B – design; C – data collection;
D – statistical processing and interpretation of data;
E – writing or critical editing of the article;
F – approval of the final version for publication and agreement to be responsible for all aspects of the work.

Declarations

Conflict of interest is absent.

The authors of the manuscript state that in the process of conducting research, preparing, and editing the text of this manuscript, no generative AI tools or services were used to perform any of the tasks listed in the Generative AI Delegation Taxonomy (GAIDeT, 2025). All conceptual stages of work, data analysis, and literature synthesis were carried out exclusively by the authors without the involvement of artificial intelligence.

A generative AI tool (ChatGPT, OpenAI) was utilized exclusively for the formal creation and technical visualization of the conceptual graphic diagram (Fig. 1). The scientific accuracy, pathways, and structure depicted in the diagram were fully developed, verified, and approved by the authors.

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