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Chrononutrition: How Meal Timing Impacts Metabolism and Disease Risk

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Abstract
Chrononutrition, the study of how meal timing affects health, has emerged as a critical component in understanding the regulation of metabolism and the development of chronic diseases. This review explores the interplay between circadian rhythms and dietary patterns, highlighting how aligning food intake with the body's internal clock can optimize metabolic processes and reduce the risk of disorders such as obesity, type 2 diabetes, and cardiovascular disease. Evidence from both observational and interventional studies suggests that irregular eating habits, such as skipping breakfast and consuming late-night meals, disrupt circadian alignment and impair glucose regulation, lipid metabolism, and hormonal balance. Time-restricted eating (TRE) and other chrono-aligned dietary interventions show promising results in improving insulin sensitivity, weight management, and systemic inflammation. As lifestyle-related metabolic diseases continue to rise, chrononutrition provides a novel framework for dietary recommendations that incorporate not only what and how much we eat, but also when we eat. This article synthesizes current evidence, discusses mechanistic insights, and outlines practical implications and future research directions.

Keywords: Chrononutrition**,** Circadian Rhythms**,** Metabolic Health

1. Introduction

Nutrition science has long focused on the quantity and quality of dietary intake, emphasizing caloric balance, macronutrient distribution, and nutrient density as primary determinants of health. However, a growing body of research has highlighted a third, often overlooked, dimension of diet: meal timing. This concept lies at the heart of chrononutrition, an emerging field that examines the interaction between nutrition and circadian biology—specifically, how the timing of food intake influences metabolic regulation and disease risk.

The human body operates on a roughly 24-hour cycle known as the circadian rhythm, which is governed by a central clock located in the suprachiasmatic nucleus (SCN) of the hypothalamus (Albrecht, 2012). This master clock is primarily synchronized by environmental light-dark cycles and orchestrates various physiological processes including hormone secretion, body temperature regulation, sleep-wake cycles, and feeding behavior. In addition to the central clock, peripheral clocks exist in nearly every tissue and organ, including the liver, pancreas, muscle, and adipose tissue, where they play a key role in metabolic homeostasis (Takahashi, 2017).

Unlike the central clock, peripheral clocks are most strongly influenced by food intake and meal timing (Sahar & Sassone-Corsi, 2009). When food is consumed at inappropriate biological times—such as late at night—this can lead to circadian misalignment, where internal biological rhythms become desynchronized from each other and from environmental cues. Circadian misalignment has been increasingly recognized as a contributor to metabolic dysfunction and chronic diseases (Reutrakul & Knutson, 2015).

Epidemiological studies have shown that irregular eating patterns—such as skipping breakfast, prolonged daily eating windows, and late-night meals—are associated with increased risk of obesity, insulin resistance, type 2 diabetes, dyslipidemia, and cardiovascular disease (St-Onge et al., 2017; Pot, 2018). For example, habitual breakfast skippers tend to have higher body mass index (BMI) and worse glycemic control, while individuals who consume the bulk of their calories earlier in the day show improved weight management and metabolic profiles (Jakubowicz et al., 2013; Almoosawi et al., 2016).

Time-restricted eating (TRE)—a form of intermittent fasting in which food intake is limited to a specific time window (typically 8–12 hours)—has emerged as a promising chrono-aligned dietary intervention. Human and animal studies have demonstrated that TRE can lead to improvements in weight loss, insulin sensitivity, blood pressure, lipid metabolism, and inflammatory markers, independent of calorie reduction (Chaix et al., 2019; Sutton et al., 2018).

Despite these encouraging findings, challenges remain. Chrononutrition research is still in its early stages, with variability in study designs, populations, and endpoints. Furthermore, inter-individual differences—including age, sex, chronotype (i.e., one's natural sleep-wake cycle), and social or occupational constraints—may affect how individuals respond to chrono-aligned dietary strategies.

Given the increasing prevalence of lifestyle-related metabolic diseases and the global burden of obesity and type 2 diabetes, the integration of chronobiological principles into nutritional guidelines holds considerable promise. This review aims to provide a comprehensive synthesis of the current evidence on chrononutrition, with an emphasis on the biological mechanisms underlying the relationship between meal timing and metabolism, the role of chrono-disruption in disease pathogenesis, and the therapeutic potential of timing-based dietary interventions.

2. Circadian Rhythms and the Biological Clock

The concept of circadian rhythms—from the Latin circa diem, meaning "about a day"—refers to the near-24-hour cycles in physiology and behavior that are driven by an internal timekeeping system. These rhythms regulate a multitude of biological functions including sleep-wake cycles, hormone secretion, body temperature, blood pressure, and importantly, metabolic processes such as glucose regulation, lipid metabolism, and energy expenditure (Bass & Takahashi, 2010).

2.1 The Central Clock: Suprachiasmatic Nucleus (SCN)

At the helm of the circadian system is the suprachiasmatic nucleus (SCN), a bilateral cluster of neurons located in the anterior hypothalamus. The SCN serves as the master circadian pacemaker, synchronizing peripheral clocks throughout the body and coordinating physiological rhythms in accordance with the environmental light-dark cycle (Reppert & Weaver, 2002).

Light signals received through the retina are transmitted to the SCN via the retinohypothalamic tract, which enables the SCN to align internal time with the external day-night cycle. In turn, the SCN regulates hormonal rhythms such as melatonin (produced by the pineal gland, promoting sleep) and cortisol

(produced by the adrenal glands, promoting wakefulness and energy mobilization), both of which exhibit marked diurnal patterns (Roenneberg & Merrow, 2016).

2.2 Peripheral Clocks and Food as a Zeitgeber

In addition to the central SCN clock, virtually all tissues in the body possess autonomous circadian clocks, collectively referred to as peripheral oscillators. These clocks are composed of transcriptional-translational feedback loops involving core clock genes such as CLOCK, BMAL1, PER, and CRY as can be seen in figure 1, which regulate rhythmic gene expression and cellular function in a tissue-specific manner (Takahashi, 2017).

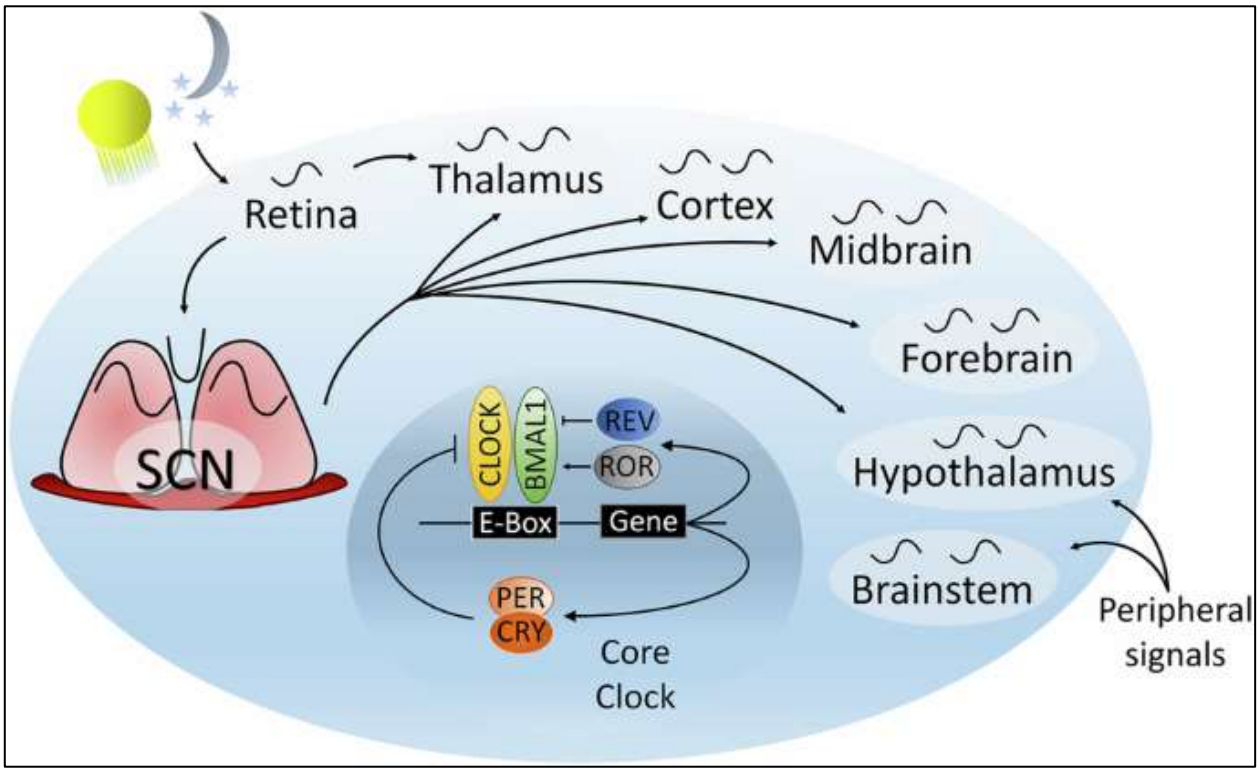

Figure 1 The Circadian Brain (Mendoza, 2025)

While the SCN is entrained primarily by light, feeding–fasting cycles are the dominant zeitgeber (time cue) for peripheral clocks, especially in metabolically active organs like the liver, pancreas, and adipose tissue (Sahar & Sassone-Corsi, 2009). Studies in rodents have shown that restricting food intake to the inactive (light) phase can uncouple peripheral clocks from the SCN, leading to metabolic disturbances even without changes in total calorie intake (Hatori et al., 2012).

2.3 Molecular Mechanisms Linking Clocks and Metabolism

Circadian clocks regulate the expression of metabolic genes involved in glucose transport, insulin secretion, lipid biosynthesis, and mitochondrial function (Green et al., 2008). For example:

- In the liver, clock genes modulate gluconeogenesis and bile acid synthesis.
- In the pancreas, they influence β-cell function and insulin secretion.
- In adipose tissue, circadian rhythms affect lipolysis and adipokine release (e.g., leptin).

Disruption of these rhythms—via shift work, jet lag, or irregular eating—can lead to circadian misalignment, impairing metabolic regulation. For instance, mice with a liver-specific deletion of BMAL1 exhibit impaired glucose tolerance and elevated triglycerides, even on a normal diet (Lamia et al., 2008).

2.4 Circadian Misalignment and Health Consequences

Circadian misalignment occurs when behavioral cycles (e.g., sleep, food intake) are desynchronized from the internal biological clock. This is common in modern societies due to nighttime light exposure, late meals, social jet lag, and shift work. Epidemiological studies have linked circadian misalignment to increased risk of:

- Obesity
- Insulin resistance
- Type 2 diabetes
- Cardiovascular disease
- Mood disorders (Reutrakul & Knutson, 2015; Zimmet et al., 2019)

Such findings underscore the importance of aligning food intake and lifestyle behaviors with the circadian system to support metabolic health.

3. The Metabolic Implications of Meal Timing

Meal timing plays a critical role in regulating metabolic health by influencing circadian rhythms, hormonal secretions, energy balance, and substrate utilization. Increasing evidence suggests that not just what we eat, but when we eat can significantly impact body weight, glucose homeostasis, lipid metabolism, and inflammatory processes (Sutton et al., 2018; Wehrens et al., 2017).

3.1 Morning vs. Evening Caloric Distribution

Multiple studies indicate that consuming a higher proportion of calories earlier in the day—such as during breakfast or lunch—can confer metabolic benefits, while consuming more calories late in the evening is associated with metabolic disturbances. This aligns with the diurnal rhythm of insulin sensitivity, which is highest in the morning and progressively declines throughout the day (Morris et al., 2015).

In a pivotal study by Jakubowicz et al. (2013), overweight women who consumed a high-calorie breakfast and a low-calorie dinner lost significantly more weight and showed greater improvements in insulin sensitivity and triglyceride levels compared to those consuming the opposite meal distribution, despite identical caloric intake. Similarly, a randomized crossover study by Al-Naimi et al. (2004) demonstrated that glucose tolerance is substantially impaired in the evening due to reduced insulin responsiveness.

Late-night eating is particularly problematic. Studies in shift workers—who often eat during the biological night—have consistently shown increased risks of obesity, type 2 diabetes, and cardiovascular disease (Gan et al., 2015). These effects are likely mediated through circadian misalignment, decreased thermic effect of food at night, and unfavorable alterations in appetite-regulating hormones such as ghrelin and leptin (Romon et al., 1993; Scheer et al., 2009).

3.2 Meal Skipping and Irregular Eating Patterns

Irregular eating patterns, including skipping breakfast or eating at inconsistent times, have been associated with increased body fat, poor glycemic control, and adverse lipid profiles. Breakfast skipping, in particular, has been linked to a higher risk of type 2 diabetes and metabolic syndrome (Uzhova et al., 2018; Odegaard et al., 2013).

A consistent meal schedule helps reinforce circadian synchronization of peripheral clocks. In contrast, erratic eating disrupts this alignment, leading to metabolic desynchrony and suboptimal nutrient processing. For example, irregular meal timing has been shown to impair postprandial glucose control and increase oxidative stress (Almoosawi et al., 2016).

3.3 Time-Restricted Eating (TRE)

Time-restricted eating (TRE) is a dietary strategy that confines food intake to a fixed daily window, typically 8–12 hours, and aligns eating with the circadian rhythm. Unlike traditional caloric restriction, TRE does not necessarily reduce calorie intake, yet it has been shown to improve metabolic health.

In rodent models, TRE prevents obesity, insulin resistance, hepatic steatosis, and inflammation—even when animals consume high-fat diets—provided that food intake is limited to the active phase (Hatori et al., 2012). In humans, early time-restricted feeding (eTRF)—where meals are consumed earlier in the day—has been associated with improved insulin sensitivity, reduced blood pressure, and lower oxidative stress (Sutton et al., 2018).

A study by Wilkinson et al. (2020) found that adults with metabolic syndrome who followed a 10-hour TRE regimen for 12 weeks showed reductions in weight, fasting glucose, LDL cholesterol, and blood pressure. These benefits occurred even in the absence of prescribed caloric restriction, suggesting that aligning food intake with circadian rhythms can independently modulate metabolic pathways.

3.4 Hormonal and Molecular Mediators

Meal timing affects the secretion and sensitivity of key metabolic hormones such as:

- Insulin – Higher sensitivity in the morning, diminished later in the day (Morris et al., 2015).
- Leptin – Exhibits nocturnal peaks; late-night eating may suppress its satiety signal (Salgado-Delgado et al., 2010).
- Ghrelin – Increases before meals and decreases after; irregular eating may desynchronize its rhythm (Cummings et al., 2001).
- Cortisol – Peaks in the morning; meal timing influences its diurnal curve (Wehrens et al., 2017).

These hormones interact with circadian transcription factors, influencing metabolic gene expression and nutrient partitioning. For instance, insulin regulates circadian genes such as PER2 and BMAL1, while clock genes modulate gluconeogenic enzymes and lipogenic pathways (Green et al., 2008; Panda, 2016).

4. Chrononutrition and Chronic Disease Risk

Emerging research in chrononutrition has illuminated the role of meal timing in modulating the risk of chronic diseases such as obesity, type 2 diabetes, cardiovascular disease, and certain cancers. Chronically misaligned eating patterns—particularly those that conflict with our circadian rhythms—may act as independent risk factors for these conditions, even when caloric intake and nutrient quality remain constant (Reutrakul & Knutson, 2015; Zimmet et al., 2019).

4.1 Obesity and Weight Regulation

Irregular eating schedules and nighttime eating have been consistently associated with increased adiposity and impaired energy balance. Disruption of the feeding–fasting rhythm impairs the diurnal pattern of energy expenditure and decreases diet-induced thermogenesis, which may contribute to weight gain (Garaulet et al., 2013).

In a study by Garaulet et al. (2013), individuals who consumed lunch after 3:00 PM lost significantly less weight than early lunch eaters, despite consuming similar calories and engaging in similar physical activity. Delayed eating was associated with altered expression of circadian clock genes in adipose tissue, suggesting a mechanism by which timing alone can influence obesity outcomes.

Nighttime eating has also been shown to increase cravings for calorie-dense foods, suppress satiety hormones, and reduce fat oxidation (McHill et al., 2017). These effects may be compounded by sleep deprivation, which often co-occurs with late eating, creating a vicious cycle of weight gain and metabolic

dysregulation.

4.2 Type 2 Diabetes and Insulin Resistance

Meal timing significantly affects glucose metabolism and insulin sensitivity, both of which exhibit strong circadian variation. Glucose tolerance is highest in the morning and diminishes throughout the day, with the poorest glycemic control occurring at night (Morris et al., 2015).

Nighttime eating has been linked to elevated postprandial glucose and insulin levels, as well as increased risk of insulin resistance. In a study involving shift workers, eating meals during the biological night resulted in 17% higher glucose and insulin responses compared to meals consumed during the day (Leproult et al., 2014).

Additionally, chronic circadian misalignment—such as that seen in rotating shift workers—has been associated with increased HbA1c levels, higher incidence of metabolic syndrome, and a significantly elevated risk of developing type 2 diabetes (Gan et al., 2015).

4.3 Cardiovascular Disease (CVD)

Chrononutrition also plays a vital role in cardiovascular health. Irregular meal timing, breakfast skipping, and late-night eating are all associated with hypertension, increased LDL cholesterol, and systemic inflammation (Crispim et al., 2011; Uzhova et al., 2017).

One of the key mechanisms is circadian disruption of blood pressure regulation and endothelial function. Normally, blood pressure follows a diurnal pattern, dipping during sleep. Eating late at night has been shown to blunt this nocturnal dip and elevate nighttime blood pressure, a known risk factor for cardiovascular events (Yamashita et al., 2015).

A large prospective cohort study from the PESA project found that individuals who skipped breakfast or consumed high-calorie dinners had a higher prevalence of non-coronary atherosclerosis, independent of traditional risk factors (Uzhova et al., 2017).

4.4 Cancer Risk and Circadian Disruption

The International Agency for Research on Cancer (IARC) classifies shift work involving circadian disruption as a probable carcinogen (Group 2A). Disrupted circadian rhythms can lead to dysregulation of cell proliferation, DNA repair, and apoptosis—key processes in tumorigenesis (Haus & Smolensky, 2013).

Late-night eating may compound this risk by altering melatonin secretion, which has anti-cancer properties, and by enhancing insulin resistance and systemic inflammation. In women, night shift work has been associated with increased breast cancer risk, and similar associations have been observed for prostate and colorectal cancer (Parent et al., 2012).

While the mechanisms remain under investigation, the interaction between meal timing, circadian gene expression, and cancer-related pathways is an emerging area of concern and interest in chronomedicine.

5. Mechanisms Linking Meal Timing to Metabolic Health

The connection between when we eat and how our body processes food is mediated by complex physiological mechanisms. These mechanisms involve the molecular circadian clock, hormonal regulation, energy metabolism, and gut microbiota. Understanding these biological pathways provides critical insights into how disrupted eating patterns can promote metabolic disorders and disease risk.

5.1 The Molecular Circadian Clock and Peripheral Clocks

At the core of chrononutrition lies the circadian clock system. The central clock in the suprachiasmatic nucleus (SCN)of the hypothalamus synchronizes peripheral clocks in tissues such as the liver, adipose tissue, pancreas, and gut (Bass & Takahashi, 2010).

These peripheral clocks regulate the timing of metabolic processes including glucose production, lipid metabolism, and hormone secretion (Sahar & Sassone-Corsi, 2009). When food intake is mistimed—such as eating late at night or irregularly—the synchronization between the central and peripheral clocks is disrupted (Panda, 2016). This desynchrony leads to metabolic inflexibility, impaired glucose tolerance, increased fat storage, and systemic inflammation.

For example, animal studies have shown that mice fed during their inactive phase (light phase for nocturnal mice) develop obesity and insulin resistance even without an increase in caloric intake (Hatori et al., 2012).

5.2 Hormonal Regulation: Insulin, Cortisol, and Melatonin

Hormones that regulate metabolism follow strong circadian patterns:

- Insulin sensitivity is highest in the morning and decreases throughout the day (Morris et al., 2015).
- Cortisol, a key regulator of glucose metabolism, peaks in the early morning and declines toward evening (Kalsbeek et al., 2014).
- Melatonin, produced at night, impairs insulin secretion and reduces glucose tolerance (Cipolla-Neto & Amaral, 2018).

Eating at night, when melatonin levels are elevated and insulin sensitivity is reduced, leads to poorer postprandial glucose control. This increases the risk of developing insulin resistance and type 2 diabetes (Qian & Scheer, 2016).

Moreover, chronic mistiming of meals can blunt the diurnal rhythm of cortisol, contributing to metabolic syndrome features such as abdominal obesity, hypertension, and dyslipidemia.

5.3 Energy Metabolism and Nutrient Processing

Meal timing influences not just how much energy we burn, but when we burn it. Studies have shown that diet-induced thermogenesis—the increase in energy expenditure after eating—is higher in the morning compared to the evening (Romon et al., 1993).

Late-night eating reduces energy expenditure, promotes fat storage, and impairs lipid metabolism. In addition, consuming high-fat or high-sugar foods during the biological night has been shown to impair mitochondrial function and promote oxidative stress (Kinouchi et al., 2018).

Thus, even when caloric intake remains stable, eating at the "wrong" time leads to less efficient energy utilization and greater adiposity.

5.4 The Role of Gut Microbiota

The gut microbiota also exhibits circadian rhythms, influencing digestion, immune function, and metabolic health (Thaiss et al., 2014).

Feeding patterns strongly modulate the composition and function of gut microbes. Disruption of feeding–fasting cycles (such as irregular meal timing or eating at night) leads to dysbiosis—an imbalance in microbial populations—linked to obesity, insulin resistance, and inflammatory diseases (Leone et al., 2015).

Interestingly, time-restricted feeding in mice restores diurnal microbial oscillations and improves metabolic outcomes even without reducing calorie intake (Zarrinpar et al., 2014). This suggests that maintaining regular meal timing could be a powerful tool for modulating gut health and preventing metabolic diseases.

6. Time-Restricted Eating and Intermittent Fasting: Chrononutrition in Practice

One of the most practical applications of chrononutrition research is the implementation of time-restricted eating (TRE) and intermittent fasting (IF) protocols. These strategies aim to restore alignment between

eating patterns and circadian rhythms, offering promising avenues for improving metabolic health and reducing disease risk.

6.1 What is Time-Restricted Eating (TRE)?

Time-Restricted Eating (TRE) involves consuming all daily calories within a consistent window of 6–12 hours, typically during the daytime, and fasting for the remaining hours, including overnight (Panda, 2016). Unlike calorie-restricted diets, TRE does not necessarily alter what or how much people eat but focuses on when eating occurs.

Animal models have shown that TRE can prevent obesity, insulin resistance, and hyperlipidemia—even when animals are fed a high-fat, high-sugar diet—simply by restricting food intake to an 8–12-hour window (Chaix et al., 2014).

Human studies also support the benefits of TRE. In one trial, overweight individuals who restricted eating to an 8-hour window (e.g., 10:00 AM–6:00 PM) experienced reductions in body weight, blood pressure, and markers of oxidative stress without explicitly reducing caloric intake (Gabel et al., 2018).

6.2 Intermittent Fasting (IF) Patterns and Their Chrononutritional Implications

Intermittent fasting refers to patterns that cycle between periods of eating and fasting, such as:

- Alternate-day fasting (ADF)
- 5:2 fasting (two non-consecutive fasting days per week)
- Periodic prolonged fasting

These strategies not only reduce caloric intake but also reinforce periods of fasting that enhance metabolic switching (Mattson et al., 2017). When timed properly—preferably earlier in the day—they can synchronize with circadian biology, promoting improvements in glucose metabolism, lipid profiles, autophagy, and cellular repair processes.

However, the timing of eating and fasting cycles is crucial. Early Time-Restricted Feeding (eTRF), where the eating window is aligned earlier in the day (e.g., 7:00 AM–3:00 PM), has been shown to improve insulin sensitivity, blood pressure, and oxidative stress more effectively than late-day eating patterns (Sutton et al., 2018).

6.3 Mechanisms Underlying the Benefits of TRE and IF

Several mechanisms explain how TRE and IF exert their metabolic benefits:

- Restoration of circadian rhythms: TRE reinforces the natural light-dark cycle, enhancing the amplitude of circadian gene expression (Chaix et al., 2014).
- Improved insulin sensitivity: Fasting periods allow insulin levels to fall, reducing hyperinsulinemia and promoting fat oxidation.
- Enhanced autophagy and cellular repair: Fasting activates autophagy, a critical process for clearing damaged cells and maintaining tissue health (Madeo et al., 2019).
- Reduction of oxidative stress and inflammation: Restricting eating to the active phase reduces oxidative stress, systemic inflammation, and mitochondrial dysfunction (Longo & Panda, 2016).

7. Challenges and Future Directions in Chrononutrition Research

Despite substantial progress, the field of chrononutrition faces several notable challenges that must be addressed to fully harness its therapeutic potential. One of the primary limitations is the lack of long-term human studies. Much of the existing research consists of short-term interventions, often lasting only a few weeks or months, and involving relatively small sample sizes (Pot et al., 2016). Although these studies consistently demonstrate improvements in weight management, insulin sensitivity, and cardiovascular

markers, the long-term sustainability, adherence, and effects on hard clinical outcomes—such as type 2 diabetes incidence, cardiovascular events, or mortality—are largely unknown. Large, multi-center randomized controlled trials with extended follow-up periods are urgently needed to confirm the lasting benefits and practical viability of meal timing interventions.

Another major challenge is the considerable variability in study designs across chrononutrition research. There is heterogeneity regarding the definition of early versus late eating windows, the duration of the eating window, population demographics (age, sex, ethnicity, and baseline metabolic health), and whether individual chronotype was considered (Templeman et al., 2023). This diversity complicates the comparison of study outcomes and hampers the development of standardized clinical guidelines. Future research would greatly benefit from harmonized protocols, clearer definitions, and uniform reporting standards to enable more meaningful synthesis and meta-analysis of data across studies.

Implementing chrononutrition principles into real-world settings also presents significant obstacles. Modern lifestyles often conflict with ideal meal timing patterns. Work schedules—especially those involving shift work—social commitments, cultural eating traditions, and family obligations make strict adherence to early or consistent eating windows challenging for many individuals (Dashti et al., 2019). Additionally, natural variations in chronotype, with some individuals having a biological predisposition toward eveningness, suggest that a "one-size-fits-all" approach is unlikely to be effective. Practical, flexible, and personalized strategies that accommodate individual life circumstances while still aligning as closely as possible with circadian biology are needed for successful public health implementation.

Finally, while mechanistic studies in animal models have provided profound insights into how circadian disruption impairs metabolic processes, translating these findings to humans remains a challenge. Ethical and technical limitations restrict the depth of mechanistic exploration possible in human subjects. More human-based studies incorporating advanced tools such as continuous glucose monitoring, wearable circadian trackers, transcriptomics, and metabolomics are needed to elucidate how specific meal timing patterns impact molecular pathways in diverse populations. Integrating these approaches with personalized medicine frameworks may eventually allow tailored chrononutrition interventions optimized to an individual's genetic background, microbiome, and chronotype.

Looking forward, the field of chrononutrition holds immense promise for reshaping dietary recommendations and chronic disease prevention strategies. Addressing the current research gaps, embracing personalized approaches, and developing flexible, sustainable interventions will be essential steps in realizing the full potential of meal timing as a cornerstone of metabolic health.

8. Conclusion

Chrononutrition represents a rapidly growing field that highlights the critical role of meal timing in metabolic regulation and disease risk. A growing body of evidence underscores that not only what and how much we eat, but also when we eat, can profoundly influence key metabolic processes, circadian biology, and long-term health outcomes. Early eating patterns, time-restricted eating (TRE), and other meal timing strategies have demonstrated significant potential to improve glucose metabolism, lipid profiles, body weight regulation, and reduce the risk of chronic diseases such as type 2 diabetes and cardiovascular disorders.

Despite these promising findings, challenges remain. Long-term, large-scale human studies are still needed to confirm the sustained benefits of chrononutritional interventions across diverse populations. In addition, real-world barriers—including social, cultural, occupational, and biological factors—must be

considered when designing practical, flexible meal timing strategies. A deeper understanding of individual chronotypes, genetic influences, and the molecular mechanisms linking circadian rhythms with metabolism will be essential for the development of personalized chrononutrition guidelines.

Looking ahead, integrating chrononutrition principles into public health strategies and clinical practice could provide a powerful tool for combating the global burden of metabolic diseases. Future research should focus on refining intervention models, embracing personalized approaches, and ensuring that chrononutritional advice is both scientifically grounded and adaptable to the complexities of modern life. By aligning our eating behaviors with our intrinsic biological clocks, we may unlock new opportunities for optimizing health, enhancing quality of life, and promoting healthy aging.

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