



The Interaction Between Chrononutrition, Gut Microbiota, and Glycemic Control in Type 2 Diabetes: A Narrative Review

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Abstract

Type 2 diabetes mellitus is influenced not only by dietary composition but also by the timing of food intake and circadian rhythms. This narrative review examines the emerging chrononutrition–gut microbiota–glycemic axis, highlighting how meal timing, microbial rhythmicity, and microbial metabolites may interact to influence glucose homeostasis and insulin sensitivity.

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KEYWORDS: Chrononutrition; Type 2 diabetes mellitus; Gut microbiota; Circadian rhythms; Meal timing; Glycemic control; Insulin sensitivity; Microbial metabolites; Time-restricted eating; Glucose homeostasis.

1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is one of the most prevalent chronic metabolic disorders worldwide and represents a major public-health challenge because of its association with cardiovascular disease, renal complications, neuropathy, retinopathy, and reduced quality of life. The pathophysiology of T2DM involves multiple interconnected abnormalities, including insulin resistance, impaired insulin secretion, altered hepatic glucose production, adipose tissue dysfunction, chronic low-grade inflammation, and disturbances in energy metabolism.

Dietary management is an essential component of diabetes prevention and treatment. Contemporary diabetes nutrition recommendations emphasize overall dietary quality, nutrient density, individualized energy intake, carbohydrate quality, and sustainable eating patterns rather than a single universally prescribed macronutrient distribution [1]. The 2026 American Diabetes Association (ADA) Standards of Care emphasize individualized medical nutrition therapy and recommend dietary patterns rich in vegetables, whole fruits, legumes, whole grains, nuts, seeds, and appropriate protein sources while minimizing highly processed foods, refined grains, sweets, and sugar-sweetened beverages [1].

Historically, nutritional research has primarily addressed what people eat and how much they eat. However, increasing evidence indicates that the timing of food intake may also influence metabolic physiology. Chrononutrition is an emerging field that examines the interaction between food intake and biological timing, including the timing, frequency, and duration of eating in relation to circadian rhythms [2,3]. Human metabolic processes do not remain constant throughout the 24-hour period. Insulin secretion, insulin sensitivity, glucose tolerance, hepatic glucose production, gastrointestinal activity, and energy metabolism exhibit circadian variation [2–5].

Experimental and clinical studies suggest that glucose tolerance is generally more favorable during the biological daytime than during the biological evening or night [3–5]. Consequently, consuming identical foods at different times may produce different postprandial glucose and insulin responses. In individuals with T2DM, disruption of normal diurnal patterns of insulin sensitivity and meal tolerance may further contribute to impaired glycemic control [6].

A second major development in metabolic research has been the recognition of the gut microbiota as an important metabolic organ. The intestinal microbial ecosystem interacts with dietary substrates and produces metabolites such as short-chain fatty acids (SCFAs), secondary bile acids, and other bioactive compounds that can influence glucose metabolism, insulin sensitivity, intestinal barrier integrity, inflammation, appetite, and incretin signaling [7–10].

Importantly, the gut microbiota is not metabolically static. Microbial composition and activity can fluctuate over the course of the day, and these oscillations are influenced by feeding-fasting cycles and host circadian rhythms [11,12]. Experimental studies have demonstrated that disruption of microbial rhythmicity can interfere with metabolic

homeostasis, suggesting that the microbiota may participate in the biological response to the timing of food intake [11].

The interaction between meal timing and gut microbial health has recently received increased attention. A 2025 review examining chrononutrition and gut health highlighted the potential effects of circadian misalignment and meal-timing variation on microbial composition, intestinal barrier function, and metabolic health [13].

These observations raise an important question: Could the gut microbiota represent a biological link between meal timing and glycemic regulation?

The potential relationship can be conceptualized as:

Circadian rhythm → meal timing → gut microbial rhythmicity → microbial metabolites → metabolic signaling → glycemic control

Although this pathway is biologically plausible, evidence directly demonstrating causality in humans with established T2DM remains limited.

Therefore, this narrative review aims to critically examine the interaction between chrononutrition, gut microbiota, and glycemic control in T2DM, with particular emphasis on circadian biology, meal timing, time-restricted eating, microbial metabolites, intestinal signaling, and personalized nutritional approaches.

2. CHRONOBIOLOGY AND METABOLIC REGULATION

2.1 Concept of circadian rhythms

2.1.1 Overview of circadian rhythms

Circadian rhythms are approximately 24-hour biological cycles that coordinate physiological and behavioral functions. They are generated by an endogenous molecular clock system and synchronized primarily by environmental cues such as the light-dark cycle and food availability [2,3].

The central circadian pacemaker is located in the suprachiasmatic nucleus of the hypothalamus, while peripheral clocks are present in metabolically active tissues including:

- Liver
- Pancreas
- Skeletal muscle
- Adipose tissue
- Gastrointestinal tract

These clocks regulate multiple metabolic processes, including glucose uptake, insulin secretion, hepatic gluconeogenesis, lipid metabolism, hormone secretion, and energy expenditure [2,3].

The molecular circadian system involves transcriptional-translational feedback loops involving clock genes such as CLOCK, BMAL1, PER, and CRY, together with nuclear receptors and metabolic regulators.

Consequently, metabolic pathways are temporally organized rather than continuously active at the same intensity throughout the day.

2.1.2 Circadian regulation of glucose metabolism

Glucose homeostasis is strongly influenced by circadian timing.

Insulin sensitivity and glucose tolerance generally show a daily rhythm, with metabolic responses often being more favorable earlier in the active period than later in the day [3–6].

During the biological daytime, glucose tolerance and insulin sensitivity are generally more favorable than during the biological night. Current diabetes standards recognize that glucose

metabolism follows a circadian pattern and that preliminary evidence suggests potential cardiometabolic advantages of appropriately earlier food consumption.

Several mechanisms may contribute:

1. Circadian regulation of pancreatic β -cell function.
2. Variation in peripheral insulin sensitivity.
3. Changes in hepatic glucose production.
4. Daily variation in incretin secretion.
5. Circadian regulation of skeletal muscle glucose uptake.
6. Changes in gastrointestinal motility and nutrient absorption.
7. Interaction between sleep and metabolic hormones.

When food intake occurs at a time that is poorly aligned with the biological clock, the resulting metabolic response may differ from that produced by the same meal consumed during the biological day. This provides the physiological basis for chrononutrition.

2.2 CHRONONUTRITION: CONCEPT AND IMPORTANCE

Chrononutrition refers to the study of the relationship between food intake and biological timing [2,3].

Three major components can be considered:

1. Timing- When meals and snacks are consumed during the 24-hour day.
2. Frequency- How frequently an individual consumes food or caloric beverages.
3. Feeding duration- The total period during which caloric intake occurs.

Chrononutrition therefore expands the conventional nutrition question: “What should be eaten?”

to:

“What should be eaten, in what quantity, and at what time?”

This does not imply that meal timing is more important than dietary quality. Rather, timing may represent an additional dimension of nutrition that interacts with food composition, energy balance, sleep, physical activity, and circadian biology.

2.3 MEAL TIMING AND GLYCEMIC CONTROL IN T2DM

2.3.1 Postprandial glucose response

Postprandial glucose excursions contribute substantially to overall glycemic exposure. The metabolic response to a meal is influenced by:

- Carbohydrate quantity
- Carbohydrate quality
- Dietary fiber
- Fat and protein content
- Gastric emptying
- Insulin sensitivity
- Physical activity
- Previous meal
- Time of day

Evidence suggests that the same meal may result in different glucose responses depending on when it is consumed [4–6].

A study examining people with T2DM demonstrated an impaired diurnal pattern of meal tolerance and insulin sensitivity, supporting the concept that metabolic responses are temporally regulated [6].

This is particularly relevant because repeated exposure to unfavorable postprandial glucose excursions may contribute to poor long-term glycemic control.

2.3.2 Late-night eating

Late-night eating has become an important area of chrononutrition research. During the biological night:

- Insulin sensitivity may be reduced.
- Glucose tolerance may decline.
- Melatonin secretion increases.
- Gastrointestinal physiology changes.
- Physical activity and energy expenditure are generally reduced.

Therefore, consumption of large meals during the biological night may produce a less favourable metabolic response than consumption earlier in the day [3–6].

A potential pathway can be proposed:

Late-night eating → circadian misalignment → reduced metabolic efficiency → greater postprandial glucose excursion → increased glycemic burden

However, late eating frequently occurs together with other behaviors such as sleep deprivation, sedentary activity, high-energy intake, and poor dietary quality. Therefore, the independent effect of meal timing must be interpreted cautiously.

2.4 TIME-RESTRICTED EATING AND TYPE 2 DIABETES

2.4.1 Definition

Time-restricted eating (TRE) is a dietary approach in which daily energy intake is confined to a predefined eating window, with fasting during the remaining hours.

TRE differs from conventional calorie restriction because its primary feature is temporal restriction of food intake, although spontaneous reductions in energy intake frequently occur.

Potential mechanisms include:

- Circadian alignment
- Prolonged fasting
- Reduced nighttime eating
- Improved insulin sensitivity
- Altered hepatic metabolism
- Changes in gut microbiota
- Improved metabolic flexibility

2.4.2 Evidence from human studies

Clinical studies have produced encouraging but heterogeneous results.

Parr et al. investigated a 9-hour eating window in adults with T2DM and reported improvements in measures of daily glycemic control, including reductions in glucose concentration and variability [14].

In a six-month randomized controlled trial, an eating window from 10:00 to 19:00 was compared with individualized dietetic guidance in adults with T2DM and overweight or obesity. The study directly evaluated HbA1c and other metabolic outcomes, providing evidence for the feasibility of TRE as a dietary strategy [15].

These findings suggest that TRE may be a useful strategy for some individuals with T2DM. Nevertheless, improvements may arise through multiple mechanisms, including reduced caloric intake, weight loss, improved dietary consistency, and circadian alignment.

Therefore, it remains difficult to determine how much of the benefit is attributable specifically to meal timing.

2.5 EARLY VERSUS LATE TIME-RESTRICTED EATING

The timing of the eating window may be as important as its duration.

Early time-restricted eating places most caloric intake earlier in the day, whereas later schedules shift food consumption toward the afternoon and evening.

Sutton et al. reported that early time-restricted feeding improved insulin sensitivity and other cardiometabolic outcomes in men with prediabetes, even in the absence of weight loss [16].

This finding is important because it suggests that metabolic improvements may occur independently of body-weight reduction.

However, extrapolation from prediabetes to established T2DM should be undertaken cautiously. The optimal eating window may depend on:

- Chronotype
- Sleep timing
- Medication regimen
- Physical activity
- Occupational schedule
- Cultural meal patterns
- Baseline metabolic status

Thus, a universal eating window may not be appropriate for every individual.

2.6 GUT MICROBIOTA AND METABOLIC HEALTH

The gut microbiota consists of a complex microbial ecosystem containing bacteria, archaea, fungi, viruses, and other microorganisms

The microbiota contributes to metabolic physiology through:

- Fermentation of dietary components
- SCFA production
- Bile acid transformation
- Vitamin metabolism
- Gut barrier regulation
- Immune modulation
- Gut hormone signaling
- Gut-brain communication

Changes in microbial composition and function have been associated with obesity, insulin resistance, metabolic syndrome, and T2DM [7–10].

However, the term dysbiosis should be interpreted carefully because there is no single microbial profile that universally defines T2DM.

Current research increasingly focuses on microbial function and metabolites rather than simply identifying bacterial taxa.

2.7 SHORT-CHAIN FATTY ACIDS AND GLYCEMIC REGULATION

SCFAs are among the most extensively studied microbial metabolites. The major SCFAs are:

- Acetate
- Propionate
- Butyrate

They are produced primarily through microbial fermentation of dietary fiber and resistant carbohydrates.

SCFAs can influence host metabolism through receptors including:

- GPR41
- GPR43

Potential metabolic effects include:

- Regulation of GLP-1 secretion
- Modulation of PYY
- Improvement of intestinal barrier integrity
- Regulation of inflammation
- Effects on hepatic metabolism
- Potential improvement in insulin sensitivity

Therefore, dietary fiber represents an important connection between dietary composition, gut microbiota, microbial metabolites, and glycemic regulation [7,8].

A chrononutrition perspective adds another layer: the timing at which fermentable substrates reach the gut may influence microbial activity and metabolite production.

2.8 BILE ACIDS AND METABOLIC SIGNALING

Bile acids are traditionally recognised for their role in lipid digestion, but they also function as metabolic signalling molecules.

Gut microbes transform primary bile acids into secondary bile acids, thereby modifying their biological activity.

Bile acids interact with receptors including:

- Farnesoid X receptor (FXR)
- Takeda G-protein receptor 5 (TGR5)

These pathways can influence:

- Glucose metabolism
- Lipid metabolism
- Energy expenditure
- GLP-1 secretion
- Insulin sensitivity

Because bile acid secretion and intestinal microbial metabolism are influenced by feeding patterns, bile acids represent a potential mechanistic bridge between chrononutrition and glucose regulation.

2.9 GUT MICROBIOTA AND GLP-1 SIGNALING

GLP-1 is an important incretin hormone involved in postprandial glucose regulation. It contributes to:

- Glucose-dependent insulin secretion
- Suppression of glucagon secretion
- Delayed gastric emptying
- Appetite regulation

Gut microbial metabolites can influence enteroendocrine signaling and may affect GLP-1 secretion [8,9].

This provides another possible pathway:

Food timing → microbial substrate availability → microbial metabolites → enteroendocrine signaling → GLP-1 → insulin response → glycemic control

Although mechanistically attractive, direct evidence demonstrating that this entire pathway mediates the benefits of chrononutrition in people with T2DM remains insufficient.

2.10 CIRCADIAN RHYTHMS OF THE GUT MICROBIOTA

An important development in microbiome research is the recognition that gut microbial communities exhibit diurnal oscillations.

Thaiss et al. demonstrated that the gut microbiota undergoes rhythmic fluctuations that are strongly influenced by feeding patterns and host circadian rhythms [11]. Their findings indicated that microbial rhythmicity contributes to metabolic homeostasis.

The intestinal epithelium itself also possesses circadian clock mechanisms that interact with microbial signals. Research has shown that disruption of communication between the intestinal circadian clock and microbiota can affect intestinal homeostasis and metabolic function [12].

This suggests that the gut microbiota can be viewed not simply as a microbial ecosystem but as a time-sensitive metabolic ecosystem.

2.11 CIRCADIAN MISALIGNMENT AND MICROBIAL DYSREGULATION

Circadian misalignment may result from:

- Shift work
- Irregular sleep schedules
- Late-night eating
- Social jet lag
- Frequent nighttime snacking
- Artificial light exposure
- Irregular feeding patterns

These factors may influence the temporal organization of gut microbial activity.

A 2025 review concluded that meal timing and circadian disruption may influence gut microbial composition, intestinal barrier integrity, and metabolic outcomes [13].

A proposed mechanism is:

Circadian disruption → altered feeding pattern → disrupted microbial rhythmicity → altered microbial metabolites → impaired metabolic signaling → insulin resistance

However, most mechanistic evidence comes from experimental models, and more human studies are required.

2.12 THE CHRONONUTRITION–GUT MICROBIOTA CONNECTION

The relationship between chrononutrition and gut microbiota is potentially bidirectional.

2.12.1 Effect of meal timing on microbiota

Meal timing may influence:

- Microbial nutrient availability
- Microbial abundance
- Microbial rhythmicity
- SCFA production
- Bile acid metabolism
- Intestinal barrier function

2.12.2 Effect of microbiota on host metabolism

Microbial metabolites may influence:

- Glucose metabolism
- Insulin sensitivity
- GLP-1 secretion
- Inflammation
- Hepatic metabolism
- Circadian clock pathways

Thus, the interaction may be represented as:

Meal timing ↔ Microbial rhythmicity ↔ Microbial metabolites ↔ Host circadian metabolism ↔ Glycemic control

This bidirectional relationship forms the central hypothesis of the present narrative review.

2.13 DIETARY QUALITY AS A MODERATOR OF CHRONONUTRITION

Meal timing should not be considered independently of dietary quality.

The 2026 ADA Standards of Care recommend individualized eating plans emphasizing nutrient-dense foods such as non-starchy vegetables, whole fruits, legumes, whole grains, nuts, seeds, and appropriate protein sources, while reducing refined and highly processed foods [1].

This is particularly important from a microbiome perspective. A fiber-rich dietary pattern provides substrates for microbial fermentation and SCFA production, whereas diets dominated by highly processed foods may provide a different microbial environment.

Therefore:

{Diet quality + meal timing + fasting duration}
may be more important than meal timing alone.

2.14 PERSONALIZED NUTRITION AND POSTPRANDIAL GLUCOSE

Not every individual responds identically to the same food or eating schedule.

Zeevi et al. demonstrated substantial inter-individual variability in postprandial glycemic responses and developed a predictive model incorporating dietary, anthropometric, lifestyle, and gut microbiota information [17].

This has important implications for chrononutrition.

Two individuals consuming the same meal at the same time may experience different glucose responses because of differences in:

- Gut microbiota
- Insulin sensitivity
- Physical activity
- Sleep
- Chronotype
- Medication
- Body composition
- Previous dietary exposure

Therefore, future chrononutrition may move away from a universal “best meal time” toward personalized meal timing.

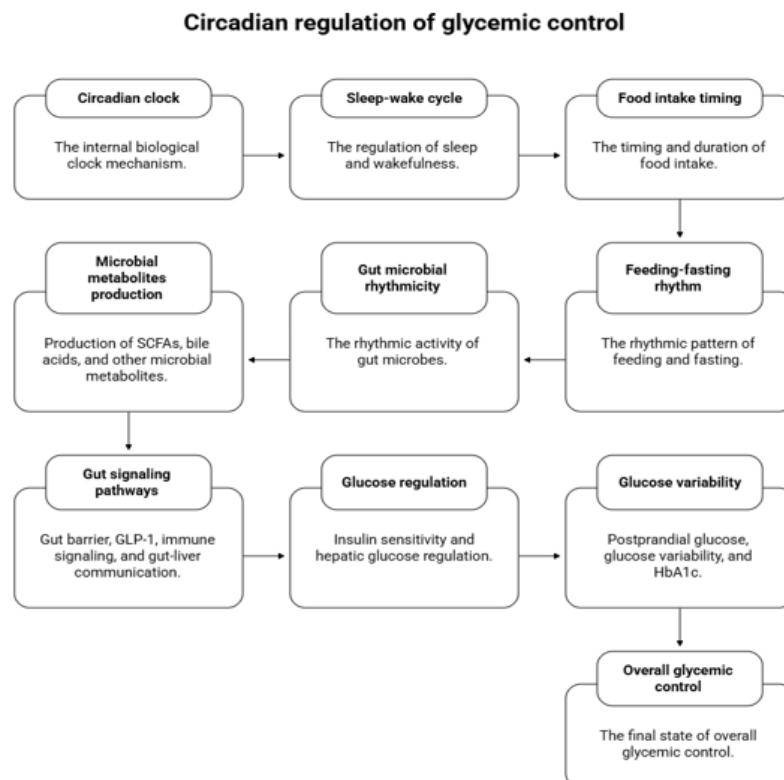


Fig 1. The proposed Chrononutrition–Microbiota–Glycemic axis

Table 1. Major components of Chrononutrition–Microbiota–Glycemic axis

Component	Major Features / Role
1. Chrononutrition (Dietary Timing)	• Meal timing and frequency • Time-restricted eating • Breakfast consumption • Late-night eating • Overall dietary quality
2. Circadian Rhythms	• Central circadian clock (SCN) • Peripheral clocks in organs and tissues • Hormonal rhythms, including melatonin and cortisol • Regulation of metabolism and behavior
3. Gut Microbiota	• Diurnal oscillations in microbial diversity • Rhythmic changes in microbial taxa • Functional gene expression • Microbial resilience and stability
4. Microbial Metabolites & Bioactive Molecules	• Short-chain fatty acids (e.g., butyrate, propionate) • Bile acid metabolites • Tryptophan metabolites (e.g., indoles) • Secondary metabolites • Modulation of host metabolism and immunity
5. Host Metabolic & Hormonal Regulation	• Glucose uptake and insulin sensitivity • Incretin hormones (GLP-1, PYY) • Inflammation and oxidative stress • Lipid metabolism and energy homeostasis
6. Glycemic Control Outcomes	• Fasting plasma glucose • Postprandial glucose • Insulin sensitivity (HOMA-IR, clamp) • Glycated hemoglobin (HbA1c) • Body composition and adiposity

3. DISCUSSION

The interaction between chrononutrition, gut microbiota, and glycemic control represents an emerging area at the intersection of nutritional science, endocrinology, microbiology, and circadian biology.

The conventional nutritional approach to T2DM has focused predominantly on food composition, carbohydrate quantity and quality, energy intake, and body-weight management. These factors remain fundamental. However, accumulating evidence suggests that the biological timing of food consumption may provide an additional layer of metabolic regulation [1–6].

The importance of timing is supported by the observation that glucose tolerance and insulin sensitivity fluctuate throughout the day. Therefore, identical meals can produce different postprandial responses depending on their timing [4–6].

The gut microbiota adds another level of complexity. Microbial communities exhibit temporal organization and respond to the availability of dietary substrates. Feeding-fasting cycles can therefore influence not only host metabolism but also the microbial ecosystem itself [11–13].

SCFAs provide a particularly interesting mechanistic pathway. Dietary fibers reaching the colon are fermented by microorganisms to produce acetate, propionate, and butyrate.

These metabolites can influence intestinal barrier integrity, enteroendocrine signaling, inflammation, and glucose metabolism [7,8].

Bile acid signaling provides another potential connection. Because gut microorganisms modify bile acid metabolism, changes in dietary timing may potentially influence bile acid signaling and downstream metabolic pathways. However, several limitations must be acknowledged.

First, many studies investigating chrononutrition and T2DM are relatively short-term. Second, TRE interventions vary substantially in eating-window duration and timing. Third, improvements in glycemia may occur partly because TRE reduces total energy intake or body weight. Fourth, microbiome research frequently identifies associations rather than demonstrating causal relationships.

Therefore, it would be premature to claim that modifying the gut microbiota through meal timing is an established treatment for T2DM.

The current evidence instead supports a more cautious conclusion:

Chrononutrition may influence glycemic physiology, and gut microbial rhythmicity is a plausible mediator that deserves further investigation.

The Synergy of Chrononutrition, Gut Microbiota, and Glycemic Control

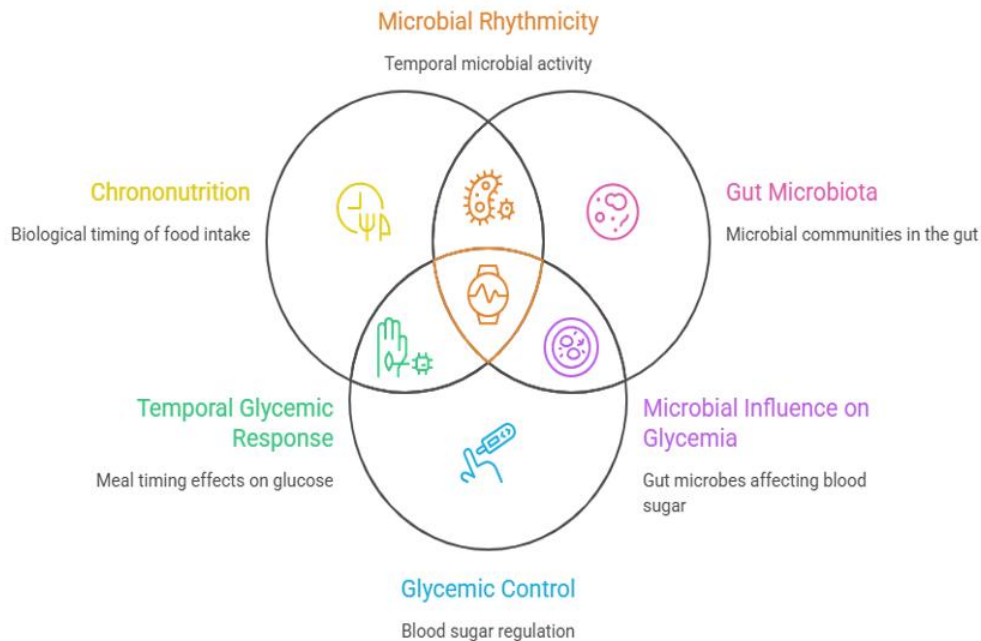


Fig 2. The synergic representation

4. RESEARCH GAPS

Despite increasing evidence linking chrononutrition and gut microbiota with metabolic health, a clear mechanistic understanding of how meal timing influences gut microbial function and glycemic control in individuals with established **type 2 diabetes mellitus (T2DM) remains lacking. The key research gaps are:**

1. Limited integrated human studies examining meal timing, gut microbiota, microbial metabolites, and glycemic outcomes together in T2DM.
2. Lack of standardized chrononutrition protocols, particularly regarding the optimal timing and duration of the eating window for individuals with T2DM.
3. Limited long-term evidence evaluating whether chrononutrition-induced changes in microbiota translate into sustained improvements in HbA1c, glucose variability, and other metabolic outcomes.
4. Inadequate investigation of microbial metabolites, particularly short-chain fatty acids and bile acids, as potential mediators between meal timing and glucose metabolism.
5. Limited attention to individual variability, including chronotype, sleep pattern, medication use, dietary composition, and baseline gut microbiota, which may modify the response to meal-timing interventions.

PRACTICAL IMPLICATIONS

The current evidence suggests that chrononutrition may eventually become an adjunct to conventional diabetes nutrition therapy.

Potentially useful principles include:

- Maintaining relatively consistent meal timing.
- Avoiding habitual large meals late at night.
- Reducing unnecessary nighttime snacking.
- Including adequate dietary fiber.
- Emphasizing minimally processed, nutrient-dense foods.
- Considering an individualized eating window when appropriate.
- Aligning food intake with the individual's sleep-wake schedule.
- Considering medication timing and hypoglycemia risk.
- Monitoring individual glucose responses when available.

Importantly, chrononutrition should not replace dietary quality. The 2026 ADA Standards emphasize individualized medical nutrition therapy and nutrient-dense dietary patterns rather than one universal diet [1].

Likewise, fasting or major changes in meal timing should not be considered automatically appropriate for every person with diabetes, particularly those taking glucose-lowering medications that may cause hypoglycemia.

5. CONCLUSION

Chrononutrition represents an important emerging dimension of nutritional management in type 2 diabetes mellitus. While traditional diabetes nutrition therapy has primarily focused on food composition, quantity, and overall dietary quality, increasing evidence suggests that the timing of food intake may also influence glucose metabolism through interactions with circadian biology.

The gut microbiota adds an important mechanistic dimension to this relationship. Microbial communities exhibit temporal fluctuations and respond to feeding-fasting cycles. Their metabolites, particularly SCFAs and bile acid derivatives, may influence intestinal barrier integrity, GLP-1 secretion, inflammation, insulin sensitivity, and glucose metabolism.

The evidence therefore supports a potential chrononutrition–gut microbiota–glycemic axis, in which: Circadian rhythm → meal timing → microbial rhythmicity → microbial metabolites → metabolic signaling → glycemic control.

Nevertheless, this relationship should currently be regarded as an emerging scientific framework rather than an established clinical pathway. Evidence directly demonstrating that microbiota changes mediate the glycemic benefits of chrononutrition in people with T2DM remains limited.

Future research should integrate continuous glucose monitoring, gut microbiome sequencing, microbial metabolomics, dietary timing, chronotype, sleep, and long-term clinical outcomes. Such approaches may ultimately support personalized nutritional strategies in which what is eaten, how much is eaten, when it is eaten, and how an individual's microbiota responds are considered together.

The future of diabetes nutrition may therefore move beyond a purely food-centered model toward a more comprehensive time–diet–microbiome–metabolism framework.

REFERENCES

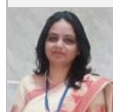
- American Diabetes Association Professional Practice Committee. 5. Facilitating positive health behaviors and well-being to improve health outcomes: Standards of Care in Diabetes—2026. *Diabetes Care*. 2026;49(Suppl 1).
- Henry CJ, Kaur B, Quek RYC. Chrononutrition in the management of diabetes. *Nutr Diabetes*. 2020; 10:6.
- Potter GDM, Skene DJ, Arendt J, Cade JE, Grant PJ, Hardie LJ. Circadian rhythm and sleep disruption: causes, metabolic consequences, and countermeasures. *Endocr Rev*. 2016;37(6):584-608.
- Morris CJ, Yang JN, Garcia JJ, et al. Endogenous circadian system and circadian misalignment impact glucose tolerance via separate mechanisms in humans. *Proc Natl Acad Sci U S A*. 2015;112(17).
- Garaulet M, Gómez-Abellán P. Timing of food intake and obesity: a novel association. *Physiol Behav*. 2014; 134:44-50.
- Yadav Y, et al. Impaired diurnal pattern of meal tolerance and insulin sensitivity in type 2 diabetes. *Diabetes*. 2023.
- Morrison DJ, Preston T. Formation of short chain fatty acids by the gut microbiota and their impact on human metabolism. *Gut Microbes*. 2016;7(3):189-200.
- Canfora EE, Jocken JWE, Blaak EE. Short-chain fatty acids in control of body weight and insulin sensitivity. *Nat Rev Endocrinol*. 2015; 11:577-591.
- Cani PD, Knauf C. How gut microbes talk to organs: the role of endocrine and metabolic signaling. *Gastroenterology*. 2016;150(7):1800-1807.
- Canfora EE, Meex RCR, Venema K, Blaak EE. Gut microbial metabolites in obesity, NAFLD and type 2 diabetes. *Nat Rev Endocrinol*. 2019; 15:261-273.
- Thaiss CA, Zeevi D, Levy M, et al. Transkingdom control of microbiota diurnal oscillations promotes metabolic homeostasis. *Cell*. 2014;159(3):514-529.
- Mukherji A, Kobiita A, Ye T, Chambon P. Homeostasis in intestinal epithelium is orchestrated by the circadian clock and microbiota cues transduced by TLRs. *Cell*. 2013;153(4):812-827.
- Bajaj P, Sharma M. Chrononutrition and gut health: exploring the relationship between meal timing and the gut microbiome. *Curr Nutr Rep*. 2025;14(1):79.
- Parr EB, Steventon-Lorenzen N, Johnston R, et al. Time-restricted eating improves measures of daily glycaemic control in people with type 2 diabetes. *Diabetes Res Clin Pract*. 2023; 197:110569.
- Parr EB, Radford BE, Hall RC, et al. Comparing the effects of time-restricted eating on glycaemic control in people with type 2 diabetes with standard dietetic practice: a randomised controlled trial. *Diabetes Res Clin Pract*. 2024; 217:111893.
- Sutton EF, Beyl R, Early KS, Cefalu WT, Ravussin E, Peterson CM. Early time-restricted feeding improves insulin sensitivity, blood pressure, and oxidative stress even without weight loss in men with prediabetes. *Cell Metab*. 2018;27(6):1212-1221.e3.
- Zeevi D, Korem T, Zmora N, et al. Personalized nutrition by prediction of glycemic responses. *Cell*. 2015;163(5):1079-1094.
- Kovatcheva-Datchary P, Nilsson A, Akrami R, et al. Dietary fiber-induced improvement in glucose metabolism is associated with increased abundance of *Prevotella*. *Cell Metab*. 2015;22(6):971-982.
- De Vadder F, Kovatcheva-Datchary P, Goncalves D, et al. Microbiota-generated metabolites promote metabolic benefits via intestinal gluconeogenesis. *Cell*. 2014;156(1-2):84-96.
- Vrieze A, Van Nood E, Holleman F, et al. Transfer of intestinal microbiota from lean donors increases insulin sensitivity in individuals with metabolic syndrome. *Gastroenterology*. 2012;143(4):913-916.e7.
- Zarrinpar A, Chaix A, Yooseph S, Panda S. Diet and feeding pattern affect the diurnal dynamics of the gut microbiome. *Cell Metab*. 2014;20(6):1006-1017.
- Thaiss CA, Levy M, Korem T, et al. Microbiota diurnal rhythmicity programs host transcriptome oscillations. *Cell*. 2016;167(6):1495-1510.e12.
- Cani PD, Amar J, Iglesias MA, et al. Metabolic endotoxemia initiates obesity and insulin resistance. *Diabetes*. 2007;56(7):1761-1772.
- Koh A, De Vadder F, Kovatcheva-Datchary P, Bäckhed F. From dietary fiber to host physiology: short-chain fatty acids as key bacterial metabolites. *Cell*. 2016;165(6):1332-1345.
- Kasubuchi M, Hasegawa S, Hiramatsu T, Ichimura A, Kimura I. Dietary gut microbial metabolites, SCFAs and

- bile acids in metabolic regulation. *Nutrients*. 2015;7(4):2839-2853.
26. Cani PD, Everard A, Duparc T. Gut microbiota, enteroendocrine functions and metabolism. *Curr Opin Pharmacol*. 2013;13(6):935-940.
 27. Sutton EF, Beyl R, Early KS, et al. Early time-restricted feeding improves insulin sensitivity and metabolic health. *Cell Metab*. 2018;27(6):1212-1221.
 28. Lowe DA, Wu N, Rohdin-Bibby L, et al. Effects of time-restricted eating on weight loss and other metabolic outcomes in adults with overweight or obesity. *JAMA Intern Med*. 2020;180(11):1491-1499.
 29. Liu D, Huang Y, Huang C, et al. Calorie restriction with or without time-restricted eating in weight loss. *N Engl J Med*. 2022; 386:1495-1504.
 30. Wilkinson MJ, Manoogian ENC, Zadourian A, et al. Ten-hour time-restricted eating reduces weight, blood pressure, and atherogenic lipids in patients with metabolic syndrome. *Cell Metab*. 2020;31(1):92-104.e5.
 31. St-Onge MP, Ard J, Baskin ML, et al. Meal timing and frequency: implications for cardiovascular disease prevention. *Circulation*. 2017;135(9).
 32. Garaulet M, Gómez-Abellán P, Alburquerque-Béjar JJ, Lee YC, Ordovás JM, Scheer FAJL. Timing of food intake predicts weight loss effectiveness. *Int J Obes (Lond)*. 2013; 37:604-611.
 33. Poggiogalle E, Jamshed H, Peterson CM. Circadian regulation of glucose, lipid, and energy metabolism in humans. *Metabolism*. 2018; 84:11-27.
 34. Longo VD, Panda S. Fasting, circadian rhythms, and time-restricted feeding in healthy lifespan. *Cell Metab*. 2016;23(6):1048-1059.
 35. Chaix A, Manoogian ENC, Melkani GC, Panda S. Time-restricted eating to prevent and manage chronic metabolic diseases. *Annu Rev Nutr*. 2019; 39:291-315.

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