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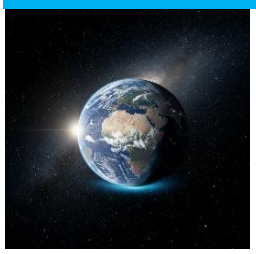

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Eat, sleep and feel better - the impact of intermittent fasting on sleep and general well-being

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Abstract

Background

Intermittent fasting (IF) is a dietary pattern based on alternating fasting and feeding periods. It is gaining attention for its potential effects on sleep, health, and well-being. Current evidence on these links remains limited and inconsistent.

Objective

To analyze and synthesize existing studies on intermittent fasting and evaluate its potential impact on sleep outcomes and overall well-being, including possible underlying mechanisms.

Methods

A systematic literature search was conducted using PubMed and Google Scholar. Articles published between 2015 and 2025 were reviewed. Keywords included: *circadian rhythm, intermittent fasting, sleep, quality of life*.

Results

Intermittent fasting shows small but noticeable improvements in perceived well-being, including better mood, reduced inflammation, and enhanced cognitive functioning. Its effects on sleep remain modest and inconsistent: some studies report slight improvements in subjective sleep quality, while objective measures reveal minimal or no change, with occasional worsening of sleep efficiency. In terms of health, IF supports metabolic regulation by improving insulin sensitivity, glucose control, and lipid profile.

Conclusions

Intermittent fasting appears to exert variable effects on sleep and well-being and also supports metabolic health and weight control. Current research is not yet sufficient to define clear clinical guidance. IF may benefit some individuals, although its safety and effectiveness vary. More long-term, well-designed studies are needed to identify protocols that provide meaningful benefits with minimal risk.

Keywords

intermittent fasting; circadian rhythm; sleep; quality of life; metabolic diseases; inflammation;

List of abbreviations: ADF, Alternate-Day Fasting; AMPK, AMP-Activated Protein Kinase; BHB, Beta-Hydroxybutyrate; CGM, Continuous Glucose Monitoring; CK1, Casein Kinase 1; EODF, Every-Other-Day Fasting; GSK3, Glycogen Synthase Kinase 3; HbA1c, Glycated Hemoglobin; HDL, High-Density Lipoprotein; IER, Intermittent Energy Restriction; IF, Intermittent Fasting; ISI, Insomnia Severity Index; LDL, Low-Density Lipoprotein; MGBA, Microbiota–Gut–Brain Axis; REM, Rapid Eye Movement; ROS, Reactive Oxygen Species; SBP, Systolic Blood Pressure; SCN, Suprachiasmatic Nucleus; T2D, Type 2 Diabetes; TG, Triglycerides; TRE, Time-Restricted Eating; TRF, Time-Restricted Feeding;
VLDL, Very Low-Density Lipoprotein.

Introduction

Today's times often require food restrictions such as working hours, religious practices, or health conditions like obesity or diabetes. In the past, some of the great fathers of medicine, including Hippocrates as one of the foremost figures, wrote about fasting as a remedy for illnesses. A dietary pattern composed of intervals of eating and fasting is called intermittent fasting (IF).[1] The health benefits of this dieting style appear to be multiple. It improves

metabolic health by regulating glucose metabolism and increasing adiponectin levels, which are associated with better insulin sensitivity.[2,3] Combining caloric deficiency with IF is an efficient method for obtaining fat loss. It should be noted that short-term fasting is less effective than medium- and long-term fasting.[2] Moreover, evidence from animal research indicates that intermittent fasting, with or without aerobic exercise, may benefit mental health by influencing both neurotransmitter activity and the microbiota–gut–brain axis.[4,5]

Some IF types are shown in Figure 1.

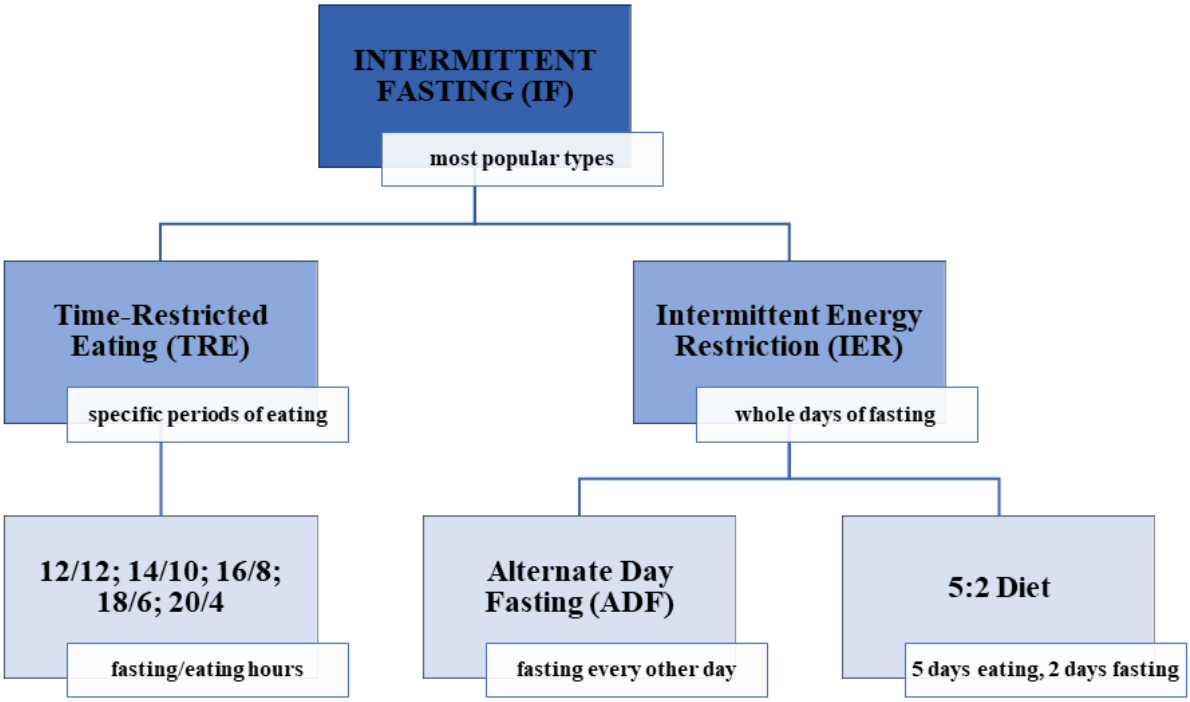

Figure 1. Most common types of IF.

Sleep periods are typically included within fasting intervals, as caloric intake does not occur during sleep. Many adults do not sleep according to the recommended amount of seven hours per day by public health authorities. Diet can directly influence sleep (for example, caffeine by prolonging the length of sleep induction), and insufficient sleep can affect diet (for example, causing hormonal changes in releasing appetite-regulating hormones (leptin and ghrelin), resulting in increased food intake and an excessive calorie diet.[6,7] The rising prevalence of insufficient sleep and circadian rhythm misalignment, which are linked to an increased risk of obesity and metabolic and mental disorders, has led to growing scientific interest in the potential effects of intermittent fasting on these outcomes.

Well-being is a term that is not strictly defined. Some divide it into subjective (related more to happiness, satisfaction, and finding meaning in life) and objective well-being (measuring

quality of life with material resources, food as an example).[8] Some consider physical and mental health, although others do not want to use medical concepts so that it can be understood as something personal and not too medically dependent. Sometimes, even the definitions of mental health are not quite clear.[9] Associating healthy dietary habits with maintaining good physical health is already well-described in the literature, but exploring the relationship between mental health and food is becoming an emerging matter with conducted studies, such as the investigation using data from the UK Biobank.[10]

Despite growing interest in the effects of dietary patterns on disease management and mental health, evidence on the impact of intermittent fasting on sleep quality and well-being is limited. This study aimed to review the existing literature to evaluate the association between these components, explain the mechanisms underlying this association, and highlight current findings and research gaps.

The role of Intermittent Fasting in modulating Circadian Rhythm and Sleep

The circadian rhythm is an endogenous, self-sustained oscillation of approximately 24 h that regulates a wide range of metabolic, behavioral, and physiological processes, thereby regulating the pace of human life and supporting efficient sleep. Situated in the hypothalamus, the suprachiasmatic nucleus (SCN) serves as the master pacemaker of the circadian rhythm. SCN neurons possess intrinsic molecular clocks that generate their own circadian oscillations. However, they are organized into a dense network and communicate with each other, which allows the formation of a stable circadian rhythm at the organismal level. The SCN also enables the synchronization of peripheral clocks present in nearly all tissues.[11]

The circadian rhythm is entrained by light and dark stimuli. It can also be modulated by external time cues originating from peripheral tissues. Feeding signals have been identified as dominant timing cues for peripheral clocks in various tissues, including clocks in the liver and those regulating metabolic pathways.[12]

In individuals who consume food outside the physiological feeding window, the function of peripheral clocks may be disrupted, which can, in turn, disturb the overall circadian rhythm and lead to metabolic dysregulation.[13]

Food intake causes a rapid rise in nutrient levels from their basal state, which induces acute expression of clock genes that modulate the circadian rhythm. Moreover, food intake activates

the mTOR signaling pathway, leading to the phosphorylation of casein kinase 1 (CK1) and glycogen synthase kinase 3 (GSK3), which regulate the stability of essential regulators of circadian rhythm-period circadian proteins (PERs).[14]

In individuals following a 16:8 intermittent fasting diet aligned with the 24-hour light–dark cycle, improvements in the oscillation of circadian rhythm gene expression have been observed. This coordination enables the reprogramming of molecular mechanisms of energy metabolism, resulting in the restoration of circadian rhythmicity.[15]

The role of Fasting in regulation of Oxidative Stress and Anti-Inflammatory Mechanisms

Reactive oxygen species (ROS) are physiologically generated as by-products of oxidative metabolism. They are a source of genomic mutations and diseases. During fasting, both oxidative metabolism and protein synthesis are moderately reduced. Fasting acts as a mild metabolic stress that promotes cellular and systemic adaptations to periods of food deprivation. In response, cells enhance DNA repair pathways and activate antioxidant enzymes, such as Superoxide Dismutase (SOD), Catalase (CAT) and Glutathione Peroxidase (GPx). Autophagy and mitophagy are then initiated, leading to the elimination of dysfunctional mitochondria, which are major sources of ROS.[16]

Fasting activates Sirtuin 3 (SIRT3), which stabilizes mitochondrial membrane potential and attenuates the NLRP3 inflammasome pathway, thereby decreasing the production of ROS. This mechanism may underlie the anti-inflammatory effects observed in fasting and fasting-mimicking diets.[17]

Periods of fasting generally lead to increased availability of free fatty acids in the hypothalamic region, accompanied by reductions in circulating glucose and amino acid levels. These changes lead to further activation of the AMP-activated protein kinase (AMPK) and sirtuin pathways and inhibition of the mTOR signaling pathway, thereby stimulating autophagy.[18]
The main pathways involved in these processes are summarized in Figure 2.

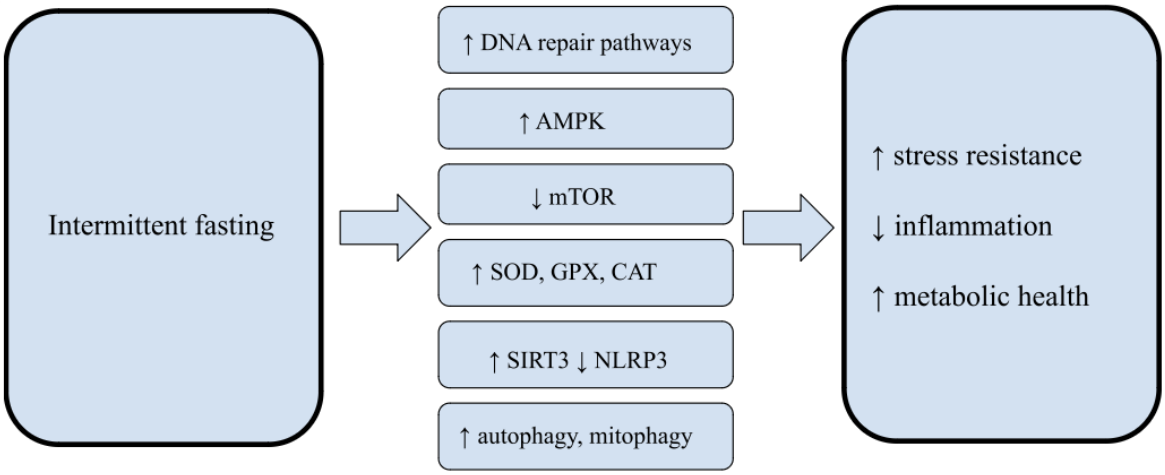

Figure 2. Summary of the cellular and molecular effects of intermittent fasting.

Intermittent Fasting and Glucose Homeostasis

Insulin is a key hormone regulating glucose metabolism. Regardless of the time of day or night, a postprandial increase in blood glucose levels induces a rise in circulating insulin levels. However, tissue sensitivity to insulin fluctuates throughout the day and night. This phenomenon results from the autonomous circadian rhythm of insulin secretion and the nocturnal pulsatile increase in growth hormone levels, which suppresses insulin release. Consequently, the insulin response is attenuated at night; therefore, late-night food intake induces a greater postprandial rise in blood glucose and insulin levels. Restricting daytime food intake during intermittent fasting enables the synchronization of postprandial hormonal responses with endogenous circadian rhythms of nutrient metabolism.[12]

It has been demonstrated that following IF for a period longer than one month leads to improved insulin sensitivity and reduced serum insulin levels, thereby contributing to the regulation of glucose metabolism. Furthermore, IF enhances hypothalamic leptin sensitivity in individuals with obesity. By reducing leptin resistance, IF improves central perception of satiety signals and consequently decreases excessive food intake.[18]

IF restores autophagic flux in pancreatic islets, enhances β-cell survival, and increases nuclear expression of NEUROG3, a pancreatic regeneration marker. Moreover, IF promotes glucose-stimulated insulin secretion, leading to glucose tolerance.[19] Fasting protocols have been shown to reduce HbA1c levels and promote weight loss by increasing the body's sensitivity to satiety signals within a limited eating window.[20]

The impact of Intermittent Fasting in modulating sleep's quality

Intermittent fasting has gained significant attention as a dietary approach with potential health benefits extending beyond weight management. Various studies have investigated IF's influence on sleep, but the results remain inconsistent.

Over the past few years, intermittent fasting has been suggested to potentially improve sleep quality.[21] One proposed mechanism involves improvement of circadian rhythmicity.[22] IF may strengthen the peripheral circadian rhythm by limiting food intake during the evening and nighttime. This, in turn, can restore the homeostatic nature of the internal clock, which may have beneficial effects on sleep in individuals with irregular sleep patterns.[21] Another way in which intermittent fasting may benefit sleep is through weight reduction. Weight loss has been shown to enhance various sleep parameters, such as sleep quality, duration, and the risk of developing obstructive sleep apnea.

Time-restricted eating appears to have questionable effects on sleep quality, particularly when weight loss is modest (2–3%), suggesting that clinically significant reductions (>5%) may be required for meaningful improvements.[23] However, across various experiments, daily assessment tools such as the myCircadianClock app and visual analog scales have detected improvements in subjective sleep quality, highlighting the importance of measurement frequency. Alternate-day fasting has shown transient sleep improvements in "poor sleepers" (baseline ISI (Insomnia Severity Index) = 12) during the weight-loss phase, which did not persist during weight maintenance, potentially influenced by high dietary protein intake.[24–27] Overall, the effects of intermittent fasting on sleep appear modest and may depend on baseline sleep quality, weight loss magnitude, and assessment methods.

The only study reporting changes in sleep duration was the 16-week, 10-hour TRE (Time Restricted Eating) trial conducted by Gill and Panda. Participants completed a subjective self-assessment survey, responding to the question "Do you feel you are getting enough sleep?" on a scale of 1 to 10 at baseline and post-intervention. The study reported a 1.5-point increase in perceived sleep duration relative to baseline. However, these findings are limited by methodological concerns, as the study did not employ a validated instrument to measure sleep

duration, nor did it report the actual increase in minutes of sleep. These limitations should be taken into account when interpreting the observed effects.[28]

Time-restricted eating has also been postulated to improve sleep by reducing insomnia, potentially through abstaining from fatty and acidic foods 2–5 h before bedtime, thereby decreasing nighttime heartburn and acid reflux.[29,30]

The effects of time-restricted eating on insomnia were assessed in two trials using the Insomnia Severity Index (ISI).[31,32] Participants had no clinically significant insomnia at baseline, and 8–12 weeks of TRE did not alter ISI scores, likely due to the absence of baseline sleep disturbances and minimal weight loss.

Alternate-day fasting (ADF) combined with a low-carbohydrate, high-protein diet was evaluated in a 24-week trial.[25] Overall, ISI scores remained unchanged in participants with obesity. In a sub-analysis, "poor sleepers" experienced a temporary reduction in ISI to 8 at week 12 following 5% weight loss, but by week 24, ISI returned to 9. "Good sleepers" (baseline ISI = 0-7) showed no changes, consistent with their normal baseline sleep.

Evidence from four trials suggests that TRE may negatively affect sleep parameters. In particular, the 8-hour TRE study by Lowe et al.[31] reported that sleep latency and efficiency worsened in TRE groups compared to controls by the end of the study, indicating a longer timeto fall asleep and lower sleep efficiency.

Taken together, these findings indicate that TRE and ADF have minimal impact on sleep outcomes. The limited weight loss achieved in these trials likely contributed to the lack of significant improvements in sleep metrics.

A recent controlled study examined the impact of meal timing on sleep architecture while accounting for Ramadan-related lifestyle changes such as altered sleep–wake cycles, caloric intake, and meal composition.[33] Rapid-eye movement (REM) sleep is a paradoxical sleep state defined as activity similar to wakefulness, rapid-eye-movement, and lack of muscle tone. REM sleep is a constitutional brain function, evolutionary conserved across species, including human, mouse, bird, and even reptiles.[34] REM sleep proportion decreased during fasting both within and outside Ramadan compared with baseline but returned to normal afterward.[33] Although the mechanisms underlying reduced REM sleep during fasting remain unclear, the proposed explanations include nocturnal elevations in cortisol and insulin level, as well as increased body

temperature due to nighttime eating and early morning awakenings for the predawn meal (Suhur), which typically coincide with peak REM sleep.[35] The clinical implications of these changes are not yet established.

Impact of Intermittent Fasting on general well-being

The relationship between intermittent fasting and well-being has become an important topic in health and behavioral sciences. Although further investigations are needed, it is a promising lead to believe that intermittent fasting may moderately reduce or even diminish symptoms of depression.[36] This effect may be mediated by biological mechanisms involved in mood regulation, stress response, and brain function.

Studies conducted in animals have shown that intermittent fasting, either alone or in combination with aerobic exercise, improves markers of depression and anxiety. In addition, it increases the levels of neurotrophins, dopamine, serotonin, and glutamine, enhances insulin sensitivity, and decreases corticosterone levels.[5] It is worth mentioning that intermittent fasting affects not only neurotransmitters but also the microbiota–gut–brain axis (MGBA).[4]

Intermittent fasting appears to have anti-inflammatory effects.[37] It can also influence neuroplasticity and cognitive function, promoting neuronal development.[38,39] These findings have led researchers to believe that IF may protect the brain, slow down its aging processes, and potentially prevent Alzheimer's disease.[40]

Intermittent fasting has been shown to induce the production of ketone bodies, particularly β-hydroxybutyrate (BHB), which can serve as an alternative energy source for the brain.[40,41] These ketones not only provide metabolic fuel but also modulate cellular signaling pathways that enhance neuroplasticity, reduce oxidative stress, and regulate inflammatory responses, all of which are critical for maintaining neuronal health.[40,42] Experimental studies have suggested that BHB may improve cognitive function, mood, and resilience to neurodegenerative processes, highlighting a potential mechanism through which IF contributes to mental well-being.[42] Moreover, IF-induced ketone production has been associated with protection against neurodegenerative diseases, such as Alzheimer’s and Parkinson’s, by promoting neuronal survival and supporting synaptic functions.[40,42]

Potential negative effects of Intermittent Fasting on well-being

Fasting is generally classified as either short-term, including IF, or prolonged, defined as lasting longer than eight days. Some studies have indicated that short-term fasting may lead to an increase in negative affect (depression, anxiety, anger, irritability, insomnia, fatigue, tension) and a decrease in positive sensations.[43]

Although only a handful of studies have been conducted on the impact of intermittent fasting on mental disorders, healthcare professionals should exercise caution when recommending IF to their patients because of its potential risks. Further analyses on larger populations should be conducted to deepen our understanding of the effects of IF on well-being.[44]

Potential clinical applications of IF in obesity, diabetes and older adults

Intermittent fasting limits weight gain[45,46], helps decrease visceral obesity, and enables long-term weight loss through lower calorie intake combined with moderate physical activity.[46] It has been shown to be as effective in reducing body weight as conventional calorie-restriction diets.[45] For example, TRF and ADF have demonstrated efficacy in reducing abdominal (central) obesity, and ADF reduces waist circumference by an average of 4.00 cm (weighted mean difference), primarily in adults with obesity aged ≥ 40 years.[46] The reduction of adiposity, particularly from visceral and truncal fat, occurs as a result of relatively small energy deficits[1] and the redistribution of visceral fat to subcutaneous fat.[47] A possible pathway for weight loss may involve a decrease in oxidative stress and inflammation (resulting in symptom improvement), the induction of a ketogenic state (shifting the main energy source from glucose to fatty acids and ketones), and the modulation of circadian rhythms.[46] Intermittent fasting may also support weight loss by altering gut microbiota composition, particularly by increasing Parabacteroides distasonis and Bacteroides thetaiotaomicron, which are associated with improved metabolic function.[48] Properly applied IF appears to be beneficial for metabolic regulation in obesity. It improves glucose metabolism[46], increases HDL levels, and reduces LDL, VLDL, TG, and total cholesterol levels.[49]For instance, sustained 5:2 IER leads to beneficial changes in the lipid profile.[46] The aforementioned small energy deficits may improve the levels and sensitivity of leptin and adiponectin, thereby enhancing appetite control.[1] Individual intermittent fasting methods act through different mechanisms: Every-Other-Day

Fasting (EODF) stimulates the conversion of beige fat within white adipose tissue, leading to increased energy expenditure, while TRF works mainly by reducing appetite and enhancing fat burning. Moreover, IF, especially TRF, improves muscle function in obesity by preventing the shift from slow- to fast-twitch fibers, restoring calcium signaling and AMPK activity, and reducing mitochondrial abnormalities and intramuscular fat infiltration. It should be highlighted that isocaloric IF works well for diet-induced obesity but may be ineffective for genetically driven obesity.[46] Additionally, the extra weight loss achieved with IF is more pronounced among people with morbid obesity.[47]

Intermittent fasting may serve as an effective, non-pharmacological option for individuals with type 2 diabetes and help prevent the disease in the prediabetic population.[50] It leads to a reduction in insulin resistance[1,45], improvement of lipid metabolism (without strict calorie restriction) and weight loss.[50] Another short-term advantage of IF is a marked decrease in HbA1c levels.[51,52] Combining IF with conventional treatment may result in a synergistic effect. With IF, it may be possible to decrease the amount of medication within only 12 weeks. This type of diet may even eliminate the need for insulin supplementation[50] or lower the overall daily insulin dosage in patients with insulin-treated type 2 diabetes.[52] The 5:2 MR eating plan can enhance blood sugar control and support short-term weight loss more effectively than metformin or empagliflozin, making it a promising option for early type 2 diabetes management.[51] However, if the goal is to discontinue or reduce medication, this diet should be followed under medical supervision. IF can be useful for patients with type 2 diabetes in managing comorbidities (e.g., hypertension) and may help lower the risk of cardiovascular complications. It is also worth noting that intermittent fasting is simple and likely the least expensive of all therapeutic tools.[50] It does not require major changes to usual eating patterns, exclusion of specific food groups, or daily monitoring of caloric intake.[45] TRE can be successfully practiced even by individuals leading a normal professional life. Regular glucose monitoring is crucial for patients following IF[50] because this dietary approach may increase the risk of hypoglycemia.[46,48] Continuous Glucose Monitoring and reducing insulin doses on fasting days help limit these episodes. Nevertheless, the most important aspect regarding the safety of this therapy is educating patients and healthcare staff on adjusting medication doses during fasting.[52]

IF is also beneficial for older adults. It can lead to weight loss, decreased visceral adipose tissue and waist circumference, and lower levels of C-reactive protein, plasma insulin, and systolic

blood pressure (SBP) in this population. Verbal memory, walking speed, and quality of life can be improved as well.[16,53] In overweight patients, TRE may exert potential anti-inflammatory effects by significantly reducing TNF-α and IL-1β levels. Adherence to this type of diet is not easy for older adults, mainly due to hunger, fatigue, limited social flexibility regarding meal timing, and a sense of restriction.[53]

Clinical risks associated with intermittent fasting

Research on the potential negative effects of intermittent fasting remains limited, largely because most studies assess its impact only over relatively short periods.[45] This type of diet is not universally applicable and should be approached with caution, particularly in clinical settings.[50] In patients with liver cirrhosis, IF may induce a hypercatabolic effect.[47] In individuals with eating disorders, it may worsen underlying psychological issues or strengthen unhealthy eating behaviors. Pregnant women may experience fatigue and dehydration,
with possible consequences for fetal development.[50]
The benefits and risks of IF vary as shown in Table 1.

Clinical population	Benefits	Risks
Obesity	weight loss, reduced waist circumference, better appetite control, improved glucose metabolism and lipid profile, support of muscle function, improvement of gut microbiota composition	
Type 2 diabetes	improved glucose and lipid metabolism (without strict calorie restriction), decreased HbA1c levels, weight loss, lower risk of cardiovascular complications, decreased medication need, reduced treatment costs	hypoglycemia

Elderly	weight loss, lower levels of C-reactive protein, plasma, insulin, and SBP; better verbal memory, walking speed, quality of life	difficulty adhering to intermittent fasting
Liver cirrhosis		hypercatabolic effect
Eating disorders		worsening underlying psychological issues, strengthening unhealthy eating behaviors
Pregnant		fatigue and dehydration

Table 1. Benefits and risks of using IF in different clinical populations.[1,16,46–48,50–53]

Discussion and Conclusions

Intermittent fasting, particularly time-restricted eating protocols, represents a promising but still incompletely validated intervention approach that influences sleep and overall well-being. Recent systematic reviews of randomized clinical trials focusing on TRE's impact on sleep emphasize that conclusions are constrained by the heterogeneity of intervention protocols (varying eating window lengths and IF patterns), diversity of sleep assessment methods (subjective questionnaires, which are less objective than actigraphy or polysomnography), and relatively small sample sizes in primary studies.[21,54,55] Many available studies are pilot trials or secondary analyses in which sleep was not a predefined primary endpoint, thereby increasing the risk of post-hoc bias and weakening causal inference.[21,56]

A closer look at methodological aspects of the literature reveals several interconnected weaknesses.

The first is the lack of intervention standardization: TRE may involve eating windows of 6, 8, 10, or 12 h, while other forms of IF (e.g., alternate-day fasting or the 5:2 protocol) differ substantially in their influence on circadian–metabolic rhythms. Such protocol variability hinders direct comparison and reduces meta-analytic power.[57]

The second weakness lies in the dominance of subjective sleep measures – many studies rely on self-reported questionnaires (e.g., PSQI), which are prone to placebo effects and perceptual bias. Studies employing objective measures (actigraphy, polysomnography) remain relatively scarce, though they provide more robust evidence.[21,56,58]

A third issue concerns the short duration of interventions: most trials lasted only several weeks to months, preventing assessment of long-term sustainability, potential adaptive attenuation, or delayed adverse outcomes.[21,59]

Another critical gap is the underrepresentation of diverse demographic groups regarding sex, age, and ethnicity – and the lack of subgroup analyses assessing whether TRE effects are homogeneous across populations.

Furthermore, there is insufficient exploration of the interaction between TRE and chronotype; preliminary data suggest a chronotype-dependent modulation of effects, but this requires validation in adequately powered studies.[35]

Based on the current state of evidence, several research gaps and methodological recommendations can be delineated.

First, randomized controlled trials (RCTs) are needed in which sleep parameters and well-being indicators are defined as primary endpoints. Such trials should be sufficiently powered to detect clinically meaningful changes in total sleep time, sleep efficiency, and quality-of-life outcomes.

Second, future studies need to change their methodology; for example, they must combine objective sleep measures (actigraphy and, where feasible, polysomnography) with validated subjective questionnaires to provide a comprehensive assessment of intervention effects. Interventions should be extended over longer periods (≥6–12 months), with follow-up assessments to evaluate sustainability and adaptation. Trials must also ensure demographic diversity (sex, age, ethnicity), include subgroup analyses (e.g., chronotypes), and rigorously control for confounders, such as light exposure, physical activity, caffeine and alcohol intake, and sleep/work regularity (e.g., shift work).

Another important direction involves integrating metabolic, hormonal, and microbiome data with objective sleep metrics and clinical well-being outcomes to elucidate mediating mechanisms and potential prognostic biomarkers. Furthermore, TRE could be investigated as

an adjunctive therapy for insomnia (e.g., in combination with CBT-I), as the phase advance and increased total sleep time might theoretically enhance the efficacy of standard treatments.[60]

In summary, the current evidence suggests that TRE/IF may beneficially influence certain aspects of sleep and well-being; however, the strength of the evidence is insufficient for definitive clinical recommendations. Larger, better-designed, and longer-term studies are required to determine which populations and IF parameters yield the greatest benefit with minimal risk. Until such evidence becomes available, TRE should be regarded as a promising but experimental dietary strategy to be considered individually and under professional supervision, especially in populations with chronic conditions.

Disclosure

Author's contributions

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Conflicts of Interest

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