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The Influence of Gut Microbiotaon Circadian Rhythm

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Abstract: The gut microbiota and the host circadian clock are deeply intertwined, with mounting evidence demonstrating their reciprocal influence on each other's activities. Disruptions in either system can trigger metabolic and inflammatory disturbances. Gut bacteria exhibit diurnal oscillations in abundance and function, driven by feeding cycles and host clock signals. In turn, microbial metabolites-such as short-chain fatty acids (SCFAs) and bile acids-entrain peripheral circadian clocks and modulate gene expression in intestinal and hepatic tissues. Circadian misalignment (from jet lag, shift work, or diet-induced dysbiosis) often disrupts microbial rhythmicity and has been linked to obesity, glucose intolerance, and systemic inflammation. Encouragingly, interventions like timed feeding (chrononutrition), probiotics, and even melatonin supplementation show promise in restoring synchrony to the gut–clock axis. In this review, We first outline core circadian regulators (e.g., the suprachiasmatic nucleus and clock genes such as CLOCK/BMAL1) and gut microbiota composition, emphasizing their homeostatic roles. Next, we discuss recent insights into bidirectional signaling between gut microbes and host circadian pathways. Finally, we highlight emerging therapeutic strategies and identify key open questions, including the need for longitudinal human studies and personalized approaches, as critical future research directions.

Keywords: Circadian clock, Gut microbiota, Diurnal oscillation, Metabolism

1. Introduction

Circadian rhythms are ~24-hour cycles that govern physiological processes ranging from sleep–wake patterns to metabolism [1]. In mammals, the master circadian clock resides in the suprachiasmatic nucleus (SCN) of the hypothalamus, which synchronizes peripheral clocks in virtually every organ [1-3]. This timing system is driven by transcriptional-translational feedback loops involving core clock genes (e.g., CLOCK, BMAL1, Period (PER), and Cryptochrome (CRY), which regulate rhythmic gene expression and behavior [1-3]. Under natural light–dark cycles, the SCN aligns peripheral oscillators via neural and hormonal signals, ensuring coordinated metabolic homeostasis [1]. The gastrointestinal tract is particularly sensitive to circadian regulation. For example, feeding during the active phase promotes optimal digestion and metabolism, whereas mistimed feeding disrupts metabolic balance [1, 3, 4]. In parallel, the gut hosts a complex microbiota—comprising trillions of microorganisms, predominantly Firmicutes and Bacteroidetes—that plays critical roles in host nutrition, immune function, and physiology [2]. Intriguingly, the gut

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microbiota itself exhibits diurnal oscillations in composition and activity [3,5]. Microbial processes such as nutrient fermentation, metabolite production, and even bacterial gene expression fluctuate across the day–night cycle [6,7]. These rhythms arise from both intrinsic microbial timekeeping mechanisms and extrinsic host cues (e.g., feeding patterns, bile acid secretion) [5, 8]. Consequently, the gut microbial community at 8 AM can differ markedly from that at 8 PM in both structure and function. These observations have given rise to the concept of the gut–clock axis: a bidirectional network linking the host circadian system with the gut microbiome [4, 9]. Disruptions to either system can propagate adverse effects. For instance, chronic circadian misalignment (e.g., due to shift work or jet lag) is associated with microbial dysbiosis and metabolic dysfunction [2, 4, 5]. Conversely, microbiome perturbations (e.g., from high-fat diets or antibiotics) can dampen circadian gene expression and exacerbate metabolic inflammation [6, 10]. Given the escalating prevalence of circadian disruption in modern lifestyles and the gut microbiota's pivotal role in metabolic regulation, elucidating their bidirectional interactions has become both urgent and clinically relevant. Understanding the influence of gut microbiota on circadian rhythm offers novel insights into metabolic disorders and suggests innovative chronotherapeutic strategies [4, 11]. This review's objective is to summarize the molecular and microbial mechanisms underpinning the gut–clock axis and examine how disturbances in microbial or circadian homeostasis impact health. Moreover, it also highlights therapeutic interventions that target this axis and proposes future research directions to fill current knowledge gaps.

2. Molecular and microbial mechanisms underpinning the gut-clock axis

2.1. Circadian clock machinery and peripheral rhythms

The mammalian circadian clock consists of core clock genes that generate self-sustained oscillations in gene expression [1-3]. In the canonical model, the transcription factors CLOCK and BMAL1 heterodimerize each other to activate target genes, including Per and Cry. As PER and CRY proteins accumulate, their feedback inhibits the CLOCK: BMAL1 complex, thereby repressing their own transcription by late day. Overnight, PER/CRY proteins are degraded, relieving inhibition and allowing a new CLOCK: BMAL1 cycle the next morning [1-3]. This molecular clock operates in virtually all cells. In the liver and intestine, it rhythmically regulates hundreds of metabolic genes involved in glucose and lipid handling [1, 12]. The SCN synchronizes these peripheral clocks to the external light cycle via signals like cortisol and feeding behavior. Notably, feeding is a strong zeitgeber (time cue) for the gut and liver clocks–food intake timing can reset peripheral clock phase even when the SCN is intact [13, 14]. For instance, mice fed only during the daytime (their normal rest period) show a phase shift in hepatic clock gene expression and dampened rhythm amplitude [13]. Thus, peripheral circadian oscillators are sensitive to metabolic cues.

2.2. Diurnal oscillations of the gut microbiota

Intriguingly, the gut microbiota also undergoes diurnal fluctuations in both composition and function [3, 5]. Pioneering studies by Thaiss et al. demonstrated that the relative abundances of major bacterial phyla oscillate throughout the day in mice, peaking at different times [3]. In conventionally raised mice on a regular feeding cycle, Bacteroidetes tend to increase during the active (dark) phase, while firmicutes rise during the resting (light) phase [5]. These dynamics result in time-of-day–specific microbial profiles. Functions of microbiota, such as the production of short-chain fatty acids (SCFAs) and other metabolites, also exhibit diurnal variation [6, 8]. For example, fecal SCFA

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concentrations are higher at certain times corresponding to when fermentable fiber is available in the gut after meals [6]. Gene expression in gut bacteria (e.g., pathways for nutrient utilization) oscillates in sync with feeding rhythms [7, 14]. Host feeding behavior is a primary driver of microbial rhythmicity. Nocturnal animals fed only at night display robust microbiome oscillations, whereas arrhythmic feeding blunts these cycles [3, 5]. In a study, restricting mice to feed during their inactive phase abolished the normal day–night differences in microbiota composition [3]. Likewise, germ-free mice (lacking a microbiome) on a regular feeding schedule do not show the typical diurnal variation in metabolic parameters, highlighting that microbes help program host rhythms [6]. Recent work indicates that signals from the host clock also shape microbial oscillations. Liang et al. found that genetic deletion of the core clock gene Bmal1 in mice markedly altered fecal microbiota rhythmicity, effectively dampening the daily oscillation of several bacterial families [5]. Interestingly, this effect depended on the host's sex– suggesting an interaction between circadian regulators, sex hormones, and microbiota composition [5]. Altogether, these data suggest that under normal conditions, the timing of feeding (dictated by the host's circadian schedule) and possibly rhythmic host secretions (bile acids, antimicrobial peptides, etc.) impose a daily structure on the gut microbiome [8, 9].

2.3. Microbial signals influencing host clocks

Communication along the gut–clock axis is bidirectional. Just as the host's clock influences microbes, the microbiota can send time cues to host tissues. Gut microbes produce a plethora of metabolites that enter circulation and can interact with host receptors. Key examples include SCFAs (such as butyrate and propionate) from fiber fermentation and secondary bile acids produced by microbial transformation of host bile [6, 7]. These metabolites have been shown to affect circadian clock gene expression in peripheral tissues. SCFAs appear to act as microbial messengers to the host clock [6, 7]. Butyrate and propionate can stimulate the production of hormones like peptide YY and GLP-1 and can also influence the epigenetic regulation of clock genes. In vitro, butyrate has been found to inhibit histone deacetylases, thereby affecting the expression of clock-controlled genes in intestinal cells [6]. In vivo, supplementation with SCFAs can shift the phase of peripheral clock gene expression. Tahara et al. demonstrated that administering SCFAs to mice could entrain the circadian clocks in the liver and kidney, effectively adjusting the timing of clock gene peaks [7]. Notably, germ-free mice (which lack SCFA-producing bacteria) exhibit blunted rhythms of certain clock genes, and reconstitution with SCFA-producing bacteria partially restores those rhythms [6,7]. These findings imply that microbial metabolites reinforce host circadian oscillations. Bile acids are another link: The host's primary bile acids are secreted in a circadian manner (peaking during active feeding periods), and gut bacteria convert these into secondary bile acids [2, 9]. These bile acids can activate nuclear receptors (like FXR and TGR5) that integrate into the hepatic clock gene network [2]. Disruption of normal bile acid rhythms (as occurs with microbiota changes) can desynchronize metabolic gene expression in the liver [2, 9]. Other microbially modulated molecules – for instance, tryptophan metabolites (e.g., indoles) and lipopolysaccharide – can engage receptors (AhR, TLR4) that crosstalk with clock gene regulators, thereby linking microbial composition to inflammatory and metabolic rhythmicity [9, 14]. Overall, these examples illustrate that gut bacteria are not passive recipients of host timing signals; they actively contribute timing cues back to the host.

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2.4. The gut–liver–brain axis

The gut–clock axis extends to multiple organ systems, notably the liver and brain. The liver, a key metabolic organ with a strong intrinsic clock, is directly exposed to intestinal blood flow carrying microbial metabolites. As discussed, rhythmic SCFAs and bile acids from the gut microbiota modulate the expression of liver clock genes and metabolic outputs [6, 12]. One illuminating example involves the circadian transcription factor NFIL3 (also known as E4BP4) in intestinal cells. Wang et al. showed that the microbiota controls the amplitude of Nfil3 expression in the intestinal epithelium via effects on the clock repressor REV-ERBα [12]. In conventional mice, microbiota-produced signals suppress REV-ERBα, de-repressing Nfil3 and promoting rhythmic lipid absorption. Germ-free mice have abnormally low Nfil3 levels and fail to absorb fat efficiently, remaining lean even on a high-fat diet [5, 12]. This exemplifies how microbial cues "hack" into the circadian clock system of the host to influence metabolism. The gut–liver axis is thus a central feature of the gut–clock interplay, with microbiota-driven entrainment of liver clock genes affecting glucose and lipid homeostasis [6, 12].

Microbiota–brain communication also occurs in a circadian context (the gut–brain axis). The gut microbiome can modulate levels of neurotransmitters and neuroactive compounds that affect brain function and behavior [9, 15]. Recent studies show that having a healthy microbiota is necessary for the regular daily changes in hormones like corticosterone (which is similar to cortisol in rodents) and for the body's ability to handle stress properly [9, 16]. In one striking experiment, mice raised germ-free or given antibiotics lost the normal daily rhythm in corticosterone secretion and exhibited exaggerated stress hormone spikes at the wrong time of day [9, 15]. This disruption was accompanied by altered clock gene expression in brain regions (such as the hypothalamus and hippocampus) that regulate the hypothalamic-pituitary-adrenal (HPA) axis [9, 15]. Transplanting a normal microbiota into these mice partially restored corticosterone rhythmicity and improved their stress response timing [9, 15]. Moreover, a specific probiotic strain (Lactobacillus reuteri) was identified that could normalize aspects of the stress hormone rhythm when given to microbiota-depleted hosts [9, 15]. These findings tie the gut microbiota to circadian regulation of the HPA axis and stress resilience. They suggest that microbial signals (possibly via the vagus nerve or bacterial metabolites that cross into circulation) influence circadian clock activity in the brain's stress centers. Collectively, these mechanistic insights reinforce that the gut microbiome is an integral part of the host's circadian system – capable of reinforcing, fine-tuning, or disrupting host clocks through a variety of chemical signals.

3. Disruptions of microbial and circadian homeostasis

3.1. Dysbiosis impairing circadian rhythms

The circadian clock and microbiome are intricately connected; thus, disruptions in one frequently affect the other. Such disruptions can arise from lifestyle (e.g., irregular light exposure or eating at odd hours) or from insults to the microbiome (e.g., poor diet or antibiotics). In this section, we explore how dysbiosis (microbial imbalance) and circadian misalignment can form a vicious cycle with significant health consequences. Alterations in gut microbiota composition – termed dysbiosis – are known to trigger metabolic and immune dysfunction [1, 4, 15]. Intriguingly, dysbiosis can also blunt the host's circadian clocks. Germ-free mice provide one extreme example: lacking microbes, they show markedly dampened circadian oscillations of clock genes and hormones in certain tissues [9, 17]. As noted earlier, germ-free mice have impaired rhythmic production of intestinal

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corticosterone and disrupted metabolic gene cycles [9]. More common forms of dysbiosis, as induced by diet or illness, likewise affect circadian regulation. A high-fat diet (HFD), for instance, not only shifts the microbiota toward an imbalanced state but also dampens the amplitude of core clock gene expression in the liver and adipose tissue [2, 6]. Leone et al. observed that mice on a Western-style HFD lost the normal diurnal variation in several gut bacteria and simultaneously exhibited "flat" circadian rhythms in hepatic Bmal1 and Per2 expression [6]. Notably, when those mice were treated with antibiotics to drastically reshape the microbiome, the rhythms of their liver clock genes were further altered, underscoring microbiota involvement in the effect [6]. Similarly, chronic low-grade dysbiosis is linked with systemic inflammation that can interfere with the suprachiasmatic nucleus and melatonin signaling [4], potentially weakening the central clock's output.

Disease states characterized by dysbiosis often show circadian disruptions. Patients with inflammatory bowel disease (IBD) commonly report disturbed sleep and irregular daily symptom cycles due to their typically altered gut microbiome. Recent studies on animals indicate a direct connection: when mice have colitis (which changes their gut bacteria), it can affect their gut's day-night activity patterns [10, 18]. Conversely, mice with genetically or environmentally disrupted circadian clocks develop shifts in their microbiota that favor pro-inflammatory taxa, which can exacerbate colitis severity [10, 14]. Voigt et al. showed that when mice were exposed to constant light, their disrupted circadian rhythms caused an increase in harmful microbes linked to inflammation; this was connected to more leaky intestines and higher levels of inflammation markers. [1, 15]. Thus, a dysbiotic microbiome can both result from and contribute to circadian rhythm disturbances, creating a self-reinforcing cycle.

Antibiotic usage is another acute way to induce dysbiosis, and studies have found that antibiotics can transiently affect host clock rhythms. Mice given broad-spectrum antibiotics lose the cyclical fluctuation of certain serum metabolites that normally follow a circadian pattern [2]. In one study, mice that were treated with antibiotics had about a 50% decrease in the changes of liver clock genes (with less variation in Rev-erbα and Dbp) compared to mice that were not treated [2]. Interestingly, reintroducing SCFA-producing bacteria (e.g., Clostridiales strains) restored some of these rhythms, again implicating microbial metabolites in maintaining robust clock function [7, 14]. These findings caution that disturbances to gut microbiota – whether by diet, disease, or drugs – can impair the very circadian mechanisms that maintain metabolic equilibrium.

3.2. Circadian misalignment and microbiota dysregulation

The converse scenario is equally important: disruption of the host's circadian system can cause microbiota imbalance. Human epidemiological studies have long noted that shift workers (who endure chronic circadian misalignment) have higher incidences of obesity, diabetes, and gastrointestinal disorders [4]. Recent research points to the gut microbiome as a mediator of these effects. In a controlled trial, healthy volunteers were subjected to a simulated shift-work schedule (with reversed sleep/wake and mealtimes) for a few days; their fecal microbiota showed decreased diversity and an increase in relative abundance of pro-inflammatory species compared to baseline [1, 2]. Moreover, the post– "jet lag" microbiome from these humans, when transplanted into germ-free mice, caused the mice to gain more weight and develop worse glucose intolerance than transplants from pre–jet lag microbiomes [3]. This elegant experiment by Thaiss et al. demonstrated that circadian disruption in humans can induce a dysbiotic microbiome capable of transmitting metabolic dysfunction to a host [3]. The dysbiosis observed included blooms of Firmicutes and loss of normal diurnal oscillation in bacterial populations [3]. Animal studies reinforce these findings. Chronic

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reversal of light–dark cycles in mice (mimicking rotating shift work) induces notable microbiota changes: overall bacterial load oscillations diminish, and specific taxa like Lactobacillaceae decrease while Clostridiales increase [10, 19]. Mice kept in constant light or constant darkness (both of which disrupt circadian cues) also develop an altered microbiome composition and metabolism. In one study, exposing mice to dim light at night led to loss of normal microbial rhythmicity and an overrepresentation of genera associated with obesity [19]. These mice became prone to weight gain and insulin resistance, suggesting the microbiome shifts were pathogenic. Indeed, 16S rRNA sequencing confirmed that dim-light exposure dampened the daily oscillation of several gut bacterial families and reduced overall microbial diversity [19]. Similarly, knockout mice lacking core clock genes (like Bmal1 or Per2) harbor different gut microbiota than wild-type mice, including higher levels of opportunistic pathogens and reduced beneficial SCFA producers [5, 14]. Such clock-disrupted mice often show metabolic phenotypes (e.g., hyperglycemia, hepatic steatosis) that can be partially ameliorated by co-housing or fecal transplants with healthy mice, implying a microbiota contribution.

Circadian misalignment can also increase gut permeability and promote metabolic endotoxemia, further perturbing the microbiome. Sleep-restricted mice, for instance, show elevated circulating lipopolysaccharide (LPS) and a shift in gut flora toward LPS-producing Gram-negative bacteria, indicating that disrupted sleep/circadian patterns can trigger mild endotoxemia and dysbiosis [9, 15, 20]. This low-grade inflammation in turn disturbs clock function in tissues (because cytokines like TNF-α can suppress clock gene expression), perpetuating a detrimental cycle [4]. Notably, circadian misalignment does not always produce identical microbiota changes – different studies have found somewhat varying taxa affected, likely due to differences in diet, light regime, or host genetics [1, 2, 21]. However, a consistent theme is the loss of normal rhythmic oscillations in the microbiome and a trend toward a pro-inflammatory, energy-harvesting microbial profile when circadian timing is chronically disrupted [3, 10]. Such profiles often include higher Firmicutes: Bacteroidetes ratios and increases in Clostridia and Enterobacteriaceae – changes also observed in obesity and diabetes.

3.3. Metabolic and inflammatory consequences

The convergence of circadian disruption and microbial imbalance has serious metabolic consequences. Human shift workers frequently develop obesity and insulin resistance, and experimental models have shown that circadian-misaligned microbiota directly leads to increased adiposity [3]. Mice experiencing “jet lag” not only had altered microbiota but also exhibited ~15% higher body weight and significantly worse glucose tolerance (elevated blood glucose ~20% higher after a standardized glucose load, $p < 0.05$) compared to mice on normal schedules [3]. These metabolic derangements were transmissible by the fecal microbiota, strongly implicating microbial mechanisms. Mice with disrupted circadian rhythms often have higher levels of inflammation in their intestines (such as IL-6 and MCP-1) and are more likely to develop colitis or react strongly to toxins [1, 15]. Voigt et al. found that mice with long-term disruptions to their circadian rhythms had higher levels of Toll-like receptor 4 in their colon and more NF-κB activity in their gut tissues, which suggests they were in a heightened state of inflammation [1, 15]. In a complementary study, Cui et al. found that mice exposed to abnormal light cycles experienced a leakier gut barrier and developed visceral hypersensitivity (a proxy for irritable bowel syndrome), along with shifts in their microbiota [1, 5, 19]. Restoring some rhythmicity — for example, via time-restricted feeding — attenuated these inflammatory phenotypes, again pointing to the microbiome’s role in mediating the pathology [13, 19]. In summary, disruptions of microbial and circadian homeostasis are often intertwined and mutually reinforced. Dysbiosis can dampen circadian signals, and circadian

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misalignment can foster dysbiosis; together they contribute to metabolic syndrome, gastrointestinal inflammation, and other disorders [1, 4, 15]. These insights underscore the importance of maintaining both a healthy microbiota and robust circadian rhythms. Even relatively short-term disturbances (like a week of shift work or a course of broad antibiotics) can perturb the gut–clock axis, though these changes may be reversible. Chronic disruptions pose a larger risk, potentially "locking in" an altered microbiome–circadian state that promotes disease. Recognizing individuals at risk (e.g., shift workers) and monitoring their microbiome and circadian markers could be a strategy to prevent long-term harm. Excitingly, this bidirectional fragility also presents opportunities for intervention – which we discuss next.

4. Conclusion

The evidence is now clear that the gut microbiota plays a pivotal role in tuning the host circadian rhythm. The relationship between circadian rhythm and gut microbiota is bidirectional: a well-synchronized circadian clock fosters a healthy cycling microbiome, and reciprocally, a balanced microbiome reinforces robust host rhythms. Disruption in either can send ripples through the other, contributing to a host of modern ailments from obesity and diabetes to inflammatory bowel disease and mood disturbances. The emerging picture of the gut–clock axis is one of intricate crosstalk. Microbial metabolites act as timing signals for peripheral clocks, and while the host's feeding–fasting cycles and hormonal rhythms serve as zeitgebers for microbial behavior. Appreciating this crosstalk opens new avenues for intervention. As mentioned, aligning lifestyle with our biological clock (regular light–dark exposure, daytime eating, sufficient sleep) and nurturing our microbiome (fiber-rich diets, possibly probiotics) are synergistic strategies that can restore harmony to the gut–clock axis. As research progresses, we anticipate that leveraging the gut–clock connection will lead to novel, holistic therapies–for example, microbiome-based supplements to aid shift workers or timed nutritional programs to complement cancer chronotherapy. The concept of "circadian hygiene" might soon encompass gut health guidelines, underscoring that to maintain our body's natural rhythms, we must also consider our symbiotic microbial partners. The old adage "timing is everything" may well extend to our microbes – and embracing that concept could revolutionize how we approach nutrition, medicine, and lifestyle in the 21st century.

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