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Review Article

Chronobiology of the Human Body: Integrating Circadian Rhythms, Health, and Environment

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Abstract

Biological timekeeping is a fundamental property of human physiology. Every organ system operates on an internal approximately 24-hour cycle, collectively studied under the discipline of chronobiology. The master clock, seated in the suprachiasmatic nucleus (SCN) of the hypothalamus, coordinates peripheral organ clocks through hormonal, neural, and metabolic pathways. At the cellular level, a self-sustaining transcription-translation feedback loop involving the clock genes CLOCK, BMAL1, PER, and CRY drives these oscillations. Environmental cues — principally light and meal timing — keep the internal clock aligned with the external world. When this alignment breaks down, whether through shift work, exposure to artificial light at night, or erratic eating habits, the consequences are wide-ranging: metabolic syndrome, type 2 diabetes, cardiovascular disease, immune dysfunction, cancer susceptibility, and psychiatric disorders all show links to circadian disruption. Individual variation in clock timing, known as chronotype, is substantially inherited and creates measurable health burdens when misaligned with social obligations. Practical applications of chronobiology — including timed drug administration, time-restricted eating, and bright light therapy — are increasingly demonstrating clinical value. This review surveys the current evidence across human chronobiology and argues that the temporal dimension of physiology deserves far greater attention in everyday clinical practice.

Keywords: *Circadian rhythm, chronobiology, chronomedicine, chronotype, zeitgeber.*

1. Introduction

For billions of years, life on Earth has been shaped by the regular alternation of day and night. Rather than simply reacting to these environmental shifts, living organisms evolved internal timing systems capable of anticipating them. In human beings, this biological clockwork governs virtually every measurable physiological variable — sleep-wake behaviour, body temperature, hormone secretion, immune activity, and cell division — across a cycle that runs close to 24 hours. This field of study is known as chronobiology.[1]

The discipline gained mainstream recognition in 2017 when Jeffrey Hall, Michael Rosbash, and Michael Young received the Nobel Prize in Physiology or Medicine for uncovering the molecular gears of the circadian clock.[2] Yet despite this landmark, temporal biology remains poorly represented in clinical training. Treatment schedules, drug dosing, and health guidelines continue to be designed with little regard for time of day, leaving a significant therapeutic opportunity largely untapped.[3]

Modern life has made this gap more pressing. Artificial lighting — particularly the blue-enriched output of LED screens — delays the biological clock by suppressing the evening melatonin signal. Roughly one in five workers in industrialised countries is engaged in shift work, which forces repeated conflict between the body clock and the demands of the working schedule.[4] Social jet lag, defined as the difference between the clock a person's biology prefers and the one their work schedule imposes, is thought to affect over two-thirds of the employed adult population.[5] Understanding how these disruptions harm health, and how circadian principles can be used therapeutically, is the central concern of this review.

2. Molecular Architecture of the Circadian Clock

2.1 The Core Feedback Loop

The molecular clock operates through a transcription-translation feedback loop (TTFL) that runs autonomously in virtually every cell of the body, requiring no external signal to keep time. The positive arm begins when two proteins — CLOCK and BMAL1 — join together and activate the Period (PER1, PER2, PER3) and Cryptochrome (CRY1, CRY2) genes.[6] The resulting PER and CRY proteins accumulate over the course of the day, are modified by kinases including CK1δ and CK1ε, and eventually return to the nucleus to switch off their own production. This negative feedback closes the loop with a periodicity of roughly 24 hours. A secondary stabilising loop, in which REV-ERBα/β and ROR receptors compete for control of BMAL1 expression, adds precision and robustness to the system.[7]

2.2 The Suprachiasmatic Nucleus and Light Input

Although every nucleated cell carries a functioning clock, the paired suprachiasmatic nuclei (SCN) — about 20,000 neurons sitting just above the optic chiasm in the anterior hypothalamus — act as the organism's master pacemaker.[8] Light information reaches the SCN from a specialised population of retinal ganglion cells expressing the photopigment melanopsin (OPN4), which are most sensitive to short-wavelength light near 480 nm.[10,11] The SCN then broadcasts timing signals to the rest of the body through the autonomic nervous system, the pineal melatonin rhythm, the adrenal cortisol surge, and through the behavioural rhythms of feeding and activity that entrain peripheral clocks by other routes.[9] Notably, even low-level light — around 10 lux at the cornea — is sufficient to suppress melatonin and shift the clock during the biological night.[12]

3. Peripheral Organ Clocks

A landmark 1998 experiment showed that ordinary fibroblasts maintain circadian gene expression for several cycles after a brief serum pulse, entirely without input from the SCN.[13] This established that the clock is not a central property of the brain but a feature of nearly every tissue. The SCN's role is coordination rather than sole generation of rhythm. When feeding is experimentally shifted to the middle of the night — mimicking the pattern of night-shift workers — the clocks in the liver, pancreas, kidney, and heart drift out of phase with the SCN over several days, creating a state of internal desynchrony that appears to drive much of the metabolic harm associated with shift work.[9]

Each organ clock serves tissue-specific functions. In the liver, circadian control extends to glucose and lipid handling, bile acid synthesis, and — importantly for pharmacology — the expression of roughly 80% of drug-metabolising enzymes, including most cytochrome P450 isoforms.[14,15] The heart clock regulates ion channels and the response to ischaemia-reperfusion injury; this partially explains why acute myocardial infarction, stroke, and sudden cardiac death cluster between 06:00 and 10:00 hours.[16,17] The immune system exhibits a daily separation of pro-inflammatory activity (peaking at night) and anti-inflammatory recovery (peaking in the morning), which has practical relevance: morning influenza vaccination has been shown in randomised trials to produce stronger antibody responses than afternoon administration.[18,19] Even the gut microbiome follows a 24-hour compositional rhythm driven by host feeding patterns; disrupting the host clock destabilises this ecosystem and worsens metabolic outcomes.[20]

4. The 24-Hour Physiological Timeline

Synthesising these findings into a practical clinical reference, Table 1 maps the major circadian events across the day alongside their physiological and clinical significance. The temporal patterning of symptoms is already familiar in several conditions — asthma worsens at night due to circadian amplification of airway inflammation, morning joint stiffness in rheumatoid arthritis corresponds to overnight cytokine accumulation, and angina peaks in the early hours.[21] Recognising these patterns can improve both diagnosis and the timing of treatment.

Table 1: Key circadian events across the 24-hour cycle and their clinical significance

Time	Key Circadian Event	Clinical Relevance
22:00–23:00	Melatonin onset; core body temperature falls	DLMO is the gold-standard clinical phase marker
23:00–02:00	Slow-wave sleep dominates; growth hormone surges	>70% of daily GH released here; critical for tissue repair
03:00–04:00	Temperature nadir; melatonin peaks	Lowest alertness; peak accident risk for night-shift workers
05:00–07:00	Cortisol awakening response; blood pressure rises	Peak incidence of myocardial infarction and ischaemic stroke
09:00–11:00	Testosterone peaks; cognitive performance optimal	Best window for learning, examinations, analytical work
13:00–15:00	Post-lunch alertness dip	Brief 10–20 min nap improves afternoon performance
15:00–17:00	Reaction time and muscle strength peak	Optimal for athletic performance and physical rehabilitation

17:00–18:00	Core temperature maximum; cardiovascular output peaks	Highest blood pressure of the day
21:00–22:00	Melatonin onset approaches; gut motility suppressed	Evening screen exposure maximally disrupts sleep onset

5. Environmental Zeitgebers and Clock Synchronisation

Zeitgebers — the German term for time-givers — are the external cues that anchor the internal clock to the 24-hour day. Light is by far the most powerful. Its phase-shifting effect depends on when it arrives relative to the internal clock: light in the early evening delays the clock, while light in the early morning advances it. This phase-response relationship explains jet lag and forms the scientific basis of light therapy.[22] The problem today is that indoor lighting environments have diverged dramatically from the conditions under which human circadian biology evolved. Most people now receive only 200–500 lux of light during the day — a small fraction of the 10,000 lux or more available outdoors — while being exposed to 50–500 lux of artificial light in the evening, more than enough to suppress melatonin.[23]

Food timing constitutes a second major zeitgeber, particularly for peripheral clocks. Experimentally shifting the feeding window to the biological rest phase can invert the phase of the liver, kidney, and heart clocks while leaving the SCN undisturbed, producing internal desynchrony through diet alone.[24] Physical activity also phase-shifts the clock, with morning exercise having the strongest clock-advancing effect.[25] These findings have practical implications: meal timing and exercise scheduling are accessible, low-cost tools for strengthening circadian alignment.

6. Chronotype and Individual Variation

People differ markedly in their preferred timing of sleep and wakefulness — a trait termed chronotype, assessed clinically through questionnaires such as the Morningness-Eveningness Questionnaire26 or the Munich Chronotype Questionnaire, or objectively via actigraphy and dim-light melatonin onset (DLMO) measurements. Twin studies place the heritability of chronotype at around 50%,27 and genome-wide studies in nearly 700,000 individuals have mapped hundreds of associated variants in and around the core clock genes.[28] A specific gain-of-function mutation in CRY1 has been shown to lengthen the free-running period and cause familial delayed sleep-wake phase disorder.[29]

Chronotype changes across the lifespan: it reaches its latest phase in the early twenties and progressively advances thereafter, with men trending later than women of the same age until around fifty.[30] When an individual's biological clock is systematically out of step with their social and occupational schedule — a state called social jet lag — the health consequences are tangible. Even one hour of social jet lag independently raises the odds of overweight by about one-third, and is associated with higher rates of depression, impaired cognition, and cardiometabolic risk markers.[5,31]

7. Circadian Disruption and Disease

7.1 Metabolic Consequences

Shift workers have a 40–60% higher risk of developing type 2 diabetes and metabolic syndrome compared with day workers, even after accounting for differences in sleep duration and diet.[4] Controlled laboratory studies that impose circadian misalignment on healthy volunteers — without changing caloric intake or total sleep time — reproduce key features of metabolic syndrome within a few weeks: insulin resistance, impaired glucose disposal, elevated triglycerides, and raised blood pressure.[32] The mechanisms are multiple and include clock-driven regulation of pancreatic beta-cell secretion, hepatic gluconeogenesis via CLOCK-BMAL1 target genes, and circadian variation in adipokine signalling.[33,34]

7.2 Cardiovascular Disease

Shift work raises the risk of coronary artery disease by approximately 40%.[35] One mechanism involves the loss of the normal nocturnal dip in blood pressure — a pattern seen in shift workers — which independently predicts adverse cardiovascular events. At the molecular level, the clock gene PER2 regulates endothelial nitric oxide synthase, linking the timekeeping machinery directly to vascular tone.[36] The Hygia Chronotherapy Trial, conducted in over 19,000 patients, found that taking antihypertensive medication at bedtime rather than in the morning substantially reduced the risk of major cardiovascular events, partly by restoring the nocturnal blood pressure dip.[37]

7.3 Cancer and Immune Function

Circadian control of cell division, DNA repair, and programmed cell death places the clock at the centre of cancer biology. BMAL1 directly targets WEE1, p21, and MDM2 — key regulators of the cell cycle and p53 pathway.[38] The International Agency for Research on Cancer designated shift work involving circadian disruption as a probable human carcinogen (Group 2A) in 2007.[39] Associations with colorectal, prostate, and endometrial cancer have since been strengthened through meta-analyses, involving mechanisms including melatonin suppression, impaired immune surveillance, and defective clock-gated DNA repair.[40]

7.4 Neuropsychiatric Disorders

In mood disorders — including major depression, bipolar disorder, and schizophrenia — circadian disruption is increasingly viewed as a core feature rather than a side effect of illness. Clock gene variants associate with mood disorder susceptibility, and chronotherapeutic interventions such as wake therapy and morning bright light produce rapid antidepressant effects through mechanisms distinct from conventional pharmacotherapy.[41] In Alzheimer's disease, sleep-dependent glymphatic clearance of amyloid-beta and tau from the brain interstitium is impaired by fragmented or phase-shifted sleep, suggesting that circadian disruption may accelerate neurodegenerative pathology.[42]

8. Chronomedicine: Clinical Applications

8.1 Chronopharmacology

Because the absorption, metabolism, and biological effect of most drugs vary with time of day, the timing of administration is a pharmacologically meaningful variable. Statins targeting HMG-CoA reductase are more effective when taken in the evening, when their target enzyme is most active. In colorectal cancer chemotherapy, oxaliplatin and 5-fluorouracil show two- to tenfold differences in maximum tolerated dose depending on when they are given, with circadian-optimised regimens achieving greater tumour response and lower toxicity.[43] Morning corticosteroids minimise adrenal suppression; bedtime aspirin better counters the early-morning surge in platelet aggregability that coincides with peak myocardial infarction risk.[3,15]

8.2 Time-Restricted Eating

Time-restricted eating (TRE) limits food intake to a consistent window of eight to twelve hours per day, aligned with the active phase of the body clock, without formally restricting calories. By reinforcing the daily feeding-fasting cycle, TRE strengthens the synchrony of peripheral metabolic clocks. Animal studies show protection against obesity, fatty liver, and cardiovascular dysfunction independently of caloric intake.[44] Human trials, particularly those using an early eating window, report improvements in insulin sensitivity, blood pressure, and circulating inflammatory markers that cannot be fully explained by any accompanying weight loss.[45]

8.3 Light Therapy

Morning bright light therapy — 20–30 minutes of exposure to 10,000 lux — is the first-line treatment for seasonal affective disorder, with response rates of 50–80%, and has well-documented benefits in delayed sleep-wake phase disorder, non-seasonal depression, shift work adaptation, and dementia-associated sleep disturbance.[46] Looking ahead, wearable actigraphy devices, personalised melatonin timing, and circadian-conscious building design offer scalable means of protecting population-level clock health.[47]

9. Discussion

Taken together, the evidence reviewed here points to a fundamental reframing: the human body is not just a biochemical machine but a time-keeping one, and health depends as much on temporal organisation as on the presence or absence of any particular molecule. Internal desynchrony — whether between different organ clocks, between the body and the environment, or between biology and social schedule — has consequences that can be measured from the gene to the population.

Several clinical implications stand out. Meal timing is an underused lever for strengthening peripheral clock synchrony that requires no expensive technology and is available to any patient. Evidence-based light hygiene — maximising morning daylight exposure and reducing evening short-wavelength light — is simple to recommend and biologically well-justified. And chronotype deserves recognition as a physiological trait: late chronotypes who are compelled to function on early schedules are not simply lazy; they are experiencing a genuine and measurable physiological burden.

A fair assessment must acknowledge the limitations of the field. Most mechanistic data come from nocturnal rodents, and direct extrapolation to diurnally active humans require caution. Clinical studies have predominantly enrolled young, healthy, Western men. The chronobiology of older adults, women at different hormonal stages, and populations across different genetic backgrounds and work patterns remains insufficiently characterised. These gaps are important targets for future research.

10. Conclusion

Chronobiology has grown from a niche academic interest into a clinically relevant discipline with direct implications for disease prevention and treatment. The circadian clock is not a passive timekeeper — it is an active regulator of physiology from the level of individual gene transcription to the coordination of whole-body function. When it is disrupted, the consequences span metabolic, cardiovascular, oncological, immunological, and psychiatric disease.[1,4,35] When it is respected, it offers a therapeutic dimension that conventional medicine has largely overlooked. Embedding temporal biology into clinical reasoning — asking not just what to do, but when — represents a practical and scientifically grounded opportunity to improve patient outcomes at relatively low cost.

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Authors' Contributions
Conceptualised and designed the review, performed the literature search, wrote and revised the manuscript, and approved the final version for submission.

Conflict of Interest
The author declares no conflict of interest.

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