

The Body Has More Than One Clock

Absurd Health Institute | Frontier Medical Concept Paper 43 | Volume 9

Series: Hidden Clocks of Healing

Article type: Causal hypothesis and mechanistic argument; not an original clinical study

Corrective edition: 17 October 2026

The proposition we defend

Biological time is distributed. We argue that an organism can become ill partly because its component rhythms lose phase coordination even when average hormone, glucose or inflammatory measurements remain normal. Central and peripheral clocks need not be wholly broken; they can be out of step with one another.

Circadian time is distributed across central and peripheral tissues rather than dictated by one master signal alone. The hypothesis is that phase mismatches between immune, hepatic, gut and neural rhythms can contribute to disease even when each rhythm retains a normal average amplitude.

Why the mechanism deserves to be taken seriously

The suprachiasmatic nucleus responds to light while feeding, hormones, temperature and activity influence peripheral clock phases. Liver metabolic enzymes, immune-cell trafficking, bile-acid cycles and microbial oscillations are time-sensitive. A common clock time does not necessarily correspond to the same internal biological phase across individuals or organs.

The causal argument

The suprachiasmatic nucleus responds to light while feeding, activity and local signals influence peripheral clocks in liver, muscle and gut. Hormone secretion, substrate processing, immune recruitment and tissue repair exhibit time-dependent patterns. Suppose a hepatic clearance peak becomes misaligned with intestinal substrate entry. The average daily concentration may remain unchanged while the peak exposure and tissue response change. A similar mismatch might place an inflammatory signal in a phase of heightened tissue sensitivity. The relevant variable is the phase relationship between two processes, not an isolated “circadian score.”

Causal architecture under closer examination

A distributed clock system can become dysfunctional without any one rhythm disappearing entirely. A person may have a recognizable sleep-wake cycle while hepatic metabolic activity, intestinal substrate arrival and immune responsiveness have shifted relative to one another. In a feedback system, relative phase determines whether one organ's output arrives when another is prepared to process it. Average twenty-four-hour concentration may therefore be an inadequate metric. For instance, if production of a metabolite peaks when elimination is slow, transient exposure can rise even with unchanged daily production. If hormone sensitivity peaks after rather than during secretion, a normal daily hormone total may produce a weaker tissue response. These statements are mechanistic hypotheses, not proof that ordinary clinical fatigue comes from organ-clock mismatch. Demonstrating this requires repeated timed measurements. Phase, amplitude, waveform and internal coupling are different quantities; collapsing them into “circadian health” obscures the biology. Light, meals and physical activity can synchronize different components to different degrees, creating opportunities for both naturally occurring and experimentally induced mismatch.

A concrete physiological case

Consider two people with similar total sleep and caloric intake, one working regular daylight hours and the other alternating nights. At a single clinic appointment their laboratory results may be indistinguishable. Time-series sampling could reveal that insulin response, hepatic metabolism and immune signals peak at incompatible times in the shift worker. If phase misalignment predicts adverse outcomes beyond sleep debt, the multi-clock theory adds explanatory value.

Original conceptual illustration

A night-shift worker's sleep duration, feeding schedule and central circadian phase may disagree with the timing of liver metabolism. To isolate clock mismatch from ordinary sleep loss, investigators need more than a questionnaire asking when someone goes to bed. Repeated phase markers and functional outcomes would be necessary. The difficulty is precisely why circadian explanations must not be applied indiscriminately to chronic disease.

What established physiology already supports

Peripheral circadian oscillators, feeding-related phase shifts and circadian control of metabolism are established. The controversial synthesis is that inter-organ phase mismatch could become a disease-maintaining variable in its own right rather than a simple consequence of poor sleep.

The strongest counterargument—and the answer

The strongest rival is that irregular sleep, stress, diet or social schedule directly causes the measured problems without a special role for internal phase differences. The thesis must show an effect of relative timing after those factors are controlled. A one-off melatonin value or wearable sleep score cannot characterize whole-body coordination.

Sleep deprivation, shift work, diet quality and stress can themselves affect outcomes; circadian mismatch is often difficult to disentangle. Mouse clock experiments cannot show that an individual human symptom arises from phase discordance. A single melatonin measurement is rarely enough to characterize the entire system.

The discriminating experiment

Collect repeated time-resolved measures of core temperature, activity, selected hormonal and metabolic outputs, and in research settings peripheral clock markers. Estimate the phase angles among systems. Test whether phase misalignment predicts responses to standardized meals or inflammatory challenges better than rhythm amplitudes and sleep duration alone.

An informative analysis would repeatedly measure phase markers across tissues or validated proxies and determine whether phase relationships predict function better than sleep duration or exposure alone. If alignment has no independent relationship to outcomes, the broad disease model loses force.

Decisive comparisons and limits of inference

The strongest clinical application would emerge from a study showing that relative phase predicts a functional endpoint independently of sleep duration, meal composition and overall rhythm amplitude. Researchers could measure melatonin phase, glucose response, selected hormonal profiles and timed metabolite production under controlled feeding schedules. One prediction is that two people with comparable mean glucose and insulin might exhibit different postprandial responses because hepatic and peripheral tissue phases are misaligned. An alternative is that sleep loss itself impairs glucose control, making the clock-angle story unnecessary. The experiment must distinguish those routes, perhaps by comparing phase shifting under fixed sleep opportunity and meal energy. Researchers also have to avoid overinterpreting clock-gene markers from a single blood sample: peripheral phase estimates can vary by tissue and method. The proposed breakthrough is not a universal timing prescription, but a shift in the unit of analysis. Health may depend partly on coordination among time-dependent processes. A blood

test can accurately report how much of something is present yet omit whether it appeared at the right moment for the receiving tissue.

A second prediction from the original model

A clock-mismatch disorder should be defined by disagreement among measured biological phases, not by being awake at an unusual hour. The degree of mismatch should also forecast outcomes better than total sleep duration alone.

Why the hypothesis matters if correct

The practical consequence of multiple biological clocks is that a normal average measurement can conceal badly timed interactions. Hepatic processing may peak out of phase with intestinal substrate arrival; immune reactivity may rise during a period of reduced tissue resilience. The model argues for measuring relationships among rhythms rather than assigning health from sleep duration alone. A test would identify a phase mismatch that predicts a functional problem even after sleep loss, food composition and total exposure are controlled. It should also show that changing the relationship, not merely one average hormone value, changes outcome. If that independent effect cannot be demonstrated, ordinary circadian disruption remains a simpler explanation. The hypothesis is provocative because it treats coordination as a biological variable with its own causal influence. A body could possess individually functioning clocks and still become metabolically maladapted through their relative timing.

The clinical and conceptual consequence

The implication is that “normal” can be a misleading description of an average. Medicine may need to measure not only how much a process occurs but when it occurs relative to the organs that depend on it.

The proposal may sharpen questions about medication timing and shift work but should not be turned into universal sleep schedules or claims that synchronization cures autoimmune disease.

The proposal may sharpen questions about medication timing and shift work but should not be turned into universal sleep schedules or claims that synchronization cures autoimmune disease.

Source bibliography: verification pending

- Thaïss CA et al. Transkingdom control of microbiota diurnal oscillations promotes metabolic homeostasis. Cell. 2014.
- Chrono-tailored drug delivery systems: recent advances and future directions. Drug Deliv Transl Res. 2024.

Editorial and clinical boundaries

This paper advances a causal thesis and identifies observations that would support or defeat it. It does not establish a new diagnosis or treatment, and it should not be used to discontinue medically indicated care. Bibliographic entries inherited from the original edition have not all been independently verified.