

# Role of Circadian Rhythms in Bone Remodeling and Metabolism

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## ABOVE THE STUDY

The concept that biological processes follow a daily rhythm is well established, yet its implications for bone biology have only recently gained serious attention. Circadian rhythms endogenous, approximately 24-hour cycles regulated by molecular clocks are now recognized as key modulators of bone remodeling and metabolism. In my view, integrating circadian biology into skeletal research represents an important and overdue shift, one that may fundamentally reshape how we approach bone health and disease management.

Bone remodeling is a continuous and tightly coordinated process involving osteoblast-mediated bone formation and osteoclast-driven resorption. Emerging evidence suggests that these activities are not constant throughout the day but instead exhibit distinct circadian patterns. For instance, markers of bone resorption tend to peak during the (night), while bone formation markers often follow a different temporal profile. This rhythmicity is thought to be governed by core clock genes such as *CLOCK*, *BMAL1*, *PER*, and *CRY*, which regulate transcriptional programs in bone cells and synchronize them with systemic physiological cycles.

At the cellular level, both osteoblasts and osteoclasts possess intrinsic circadian clocks. These clocks influence gene expression related to differentiation, activity, and lifespan of these cells. Disruption of clock genes in experimental models has been shown to impair bone formation, alter bone mass, and even affect skeletal architecture. This indicates that circadian regulation is not merely a peripheral influence but a central determinant of bone homeostasis.

One of the most compelling aspects of circadian control in bone biology is its integration with metabolic processes. Bone is increasingly recognized as an endocrine organ, interacting with energy metabolism through hormones such as osteocalcin. Circadian rhythms regulate not only bone cell activity but also systemic factors like cortisol, melatonin, and parathyroid hormone, all of which have well-documented effects on bone turnover. For example, cortisol follows a diurnal pattern with peak levels in the morning, and its dysregulation such as in

chronic stress or shift work can lead to increased bone resorption and reduced bone formation.

Lifestyle factors that disrupt circadian rhythms are becoming increasingly common in modern society, and their impact on bone health should not be underestimated. Shift work, irregular sleep patterns, and exposure to artificial light at night can all lead to circadian misalignment. In my opinion, this represents an underappreciated risk factor for bone diseases such as osteoporosis. Epidemiological studies have begun to link circadian disruption with decreased bone mineral density and increased fracture risk, although more research is needed to establish causality and underlying mechanisms.

Another intriguing dimension is the role of feeding-fasting cycles and their synchronization with the circadian clock. Nutrient intake influences bone metabolism, and the timing of (food) may be just as important as its composition. Time-restricted feeding and other chrononutrition strategies are being explored for their potential to optimize metabolic health, and it is plausible that such approaches could also benefit bone remodeling by aligning nutrient availability with peak periods of osteoblastic activity.

From a therapeutic perspective, the circadian dimension opens new possibilities for optimizing treatment strategies. Chronotherapy the timing of medication administration to align with biological rhythms has shown promise in other fields and could be applied to bone diseases. For example, administering anti-resorptive drugs at times when bone resorption is highest may enhance their efficacy. Similarly, understanding the circadian dynamics of hormone secretion could inform the timing of interventions involving vitamin D, calcium, or anabolic agents.

However, several challenges remain. The complexity of circadian regulation, influenced by genetic, environmental, and behavioral factors, makes it difficult to establish universal guidelines. Moreover, individual variability in circadian rhythms often referred to as chronotype means that personalized approaches may be necessary. Advances in wearable technology and biomarker monitoring could help tailor interventions to individual circadian profiles.

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**Received:** 23-Apr-2025, Manuscript No. BMRJ-25-41396; **Editor assigned:** 25-Apr-2025, PreQC No. BMRJ-25-41396 (PQ); **Reviewed:** 09-May-2025, QC No. BMRJ-25-41396; **Revised:** 16-May-2025, Manuscript No. BMRJ-25-41396 (R); **Published:** 23-May-2025. DOI: 10.35841/2572-4916.25.13.339.

**Citation:** Johansson E (2025). Role of Circadian Rhythms in Bone Remodeling and Metabolism. J Bone Res. 13:339.

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In conclusion, circadian rhythms play a fundamental role in regulating bone remodeling and metabolism, influencing both cellular activity and systemic factors. Recognizing and harnessing this temporal dimension offers a promising avenue for improving bone health and treating skeletal disorders. In my

opinion, future research and clinical practice should move toward a more time-aware approach, where not only what we treat but also when we treat becomes a critical component of effective care.