

New insights into gene underlying circadian rhythms

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A genetic modification in a "clock gene" that influences circadian rhythm produced significant changes in the length and magnitude of cycles, providing insight into the complex system and giving scientists a new tool to further investigate how circadian rhythm is regulated.

The study, published in *Proceedings of the National Academy of Sciences*

of the United States of America, was co-authored by Kazuhiro Shimomura, DVM, research assistant professor in The Ken & Ruth Davee Department of Neurology.

Circadian rhythms influence organisms' daily sleeping and feeding patterns, as well as contribute to the measurement of day length that predicts seasonal changes crucial for migration or reproduction; While these patterns are easily observable, decoding the genetics behind the behavior has been difficult.

Previous studies have demonstrated that mammals have a biological timer called a circadian oscillator that uses a series of genetic feedback loops, but the mechanisms of this system are complicated and not fully understood.

In the current study, scientists generated mouse models with small changes to the gene *Per2*, cutting out a section of genetic code. That caused the *Per2* gene to code for more PER2 proteins than usual. The mice in the study lengthened circadian period and displayed more robust oscillations in the cycle, showing that normal levels of the PER2 [protein](#) are important for regulating those rhythms.

According to the authors, defining how an accumulation of PER2 proteins changes circadian rhythm has the potential to make mouse models a useful research tool for future circadian rhythm studies, helping to provide insight into the potential causes of human circadian [rhythm](#) disorders.

More information: Seung-Hee Yoo et al. Period23'-UTR and microRNA-24 regulate circadian rhythms by repressing PERIOD2 protein accumulation, *Proceedings of the National Academy of Sciences* (2017). [DOI: 10.1073/pnas.1706611114](https://doi.org/10.1073/pnas.1706611114)

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