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**PRESS RELEASE** (2026/08/25)

# Pressure-eyes at night: Researchers find new mechanism that causes increase in internal eye pressure, and a possible new target for glaucoma treatment

Norepinephrine-induced RHOB is a key regulator of circadian intraocular pressure rhythm

**Fukuoka, Japan**—Glaucoma is an eye disease that causes progressive damage to the optic nerve, leading to loss of sight. One major risk factor is an increase in intraocular pressure (IOP), or the pressure inside the eye. This pressure fluctuates throughout the day and is known to rise at night. However, the detailed molecular mechanisms on why this happens have not been fully understood.

Using human cells and mouse models, researchers at Kyushu University have found a new molecular pathway that explains why IOP increases at night. The neurotransmitter norepinephrine (also known as noradrenaline) increases the levels of a molecule called RHOB in the eye's drainage system called the trabecular meshwork. This weakens the eye's "cleaning function," resulting in increased IOP. The team expects these findings will lead to the development of new approaches for the early detection of glaucoma, as well as new treatments for controlling IOP by targeting RHOB. Their results were published in the journal *Communications Biology*.

IOP is maintained by balancing fluid production and fluid drainage within the eye. As with many bodily functions, it is regulated by the circadian clock. In the case of the eye, IOP increases at night. Elevated IOP is a key factor in identifying the onset of glaucoma, but because pressure tends to be lower during the day, regular checkups can potentially miss these warning signs.

Because the circadian clock regulates IOP, disruptions to a person's internal clock can lead to increased risk of glaucoma. This is why there is a higher risk of glaucoma in the elderly whose body clocks are desynchronizing.

"Previous studies have found that signals from the sympathetic nervous system contribute to the nighttime rise in eye pressure, but that underlying process was not well understood," explains first author of the study, Associate Professor Keisuke Ikegami from Kyushu University's Faculty of Agriculture. "Most of the fluid in the eye is drained through a tissue called the trabecular meshwork. These cells also help keep the drainage pathway clear by taking up and removing small particles and waste. We also know that norepinephrine is a chemical that is released by the sympathetic nervous system. We decided to investigate how norepinephrine can change the function of fluid drainage in the eye and whether it can

explain why eye pressure increases at night."

The team began by exposing human and mouse trabecular meshwork cells to norepinephrine and compared changes in their genetic activity. They identified 18 genes that increased in both systems and focused on one called RHOB. RHOB is a molecule that is involved in controlling cell shape, movement, and intracellular transport.

Norepinephrine increased RHOB in the trabecular meshwork cells, and when RHOB was removed from human cells, their ability to take up and clear particles increased. In contrast, increasing RHOB reduced the cleaning activity and fluid movement in the eye. Testing in mice, the team used eye drops that inhibit a chemical pathway that controls RHOB activity, the RHO-ROCK pathway. The results showed that the eye drops reduced the nighttime rise in eye pressure.

While these results are not intended for immediate clinical application, they have identified the RHOB pathway as a new potential target for suppressing nocturnal increase in IOP. While ROCK inhibitors are used in glaucoma treatments today, further verification is needed to determine the most effective time of day for administration and how they alter IOP rhythm.

"Loss of vision from glaucoma occurs slowly, so early detection is crucial. We hope our work will lead to new treatment regimens and strategies for administering medicines to achieve the greatest effect," concludes Ikegami.

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For more information about this research, see "Norepinephrine-induced small GTPase RHOB mediates nocturnal intraocular pressure rise in mice," Keisuke Ikegami, Takumi Takahashi, Yuto Katamoto, Atsuro Oishi, Akihide Yoshimi, Miki Nagase, Atsuya Miki, Shinobu Yasuo & Satoru Masubuchi, in *Communications Biology,* https://doi.org/10.1038/s42003-026-10710-1

**About Kyushu University**

Founded in 1911, Kyushu University is one of Japan's leading research-oriented institutions of higher education, consistently ranking as one of the top ten Japanese universities in the Times Higher Education World University Rankings and the QS World Rankings. Located in Fukuoka, on the island of Kyushu—the most southwestern of Japan's four main islands—Kyushu U sits in a coastal metropolis frequently ranked among the world's most livable cities and historically known as Japan's gateway to Asia. Its multiple campuses are home to around 19,000 students and 8,000 faculty and staff. Through its VISION 2030, Kyushu U will "drive social change with integrative knowledge." By fusing the spectrum of knowledge, from the humanities and arts to engineering and medical sciences, Kyushu U will strengthen its research in the key areas of decarbonization, medicine and health, and environment and food, to tackle society's most pressing issues.

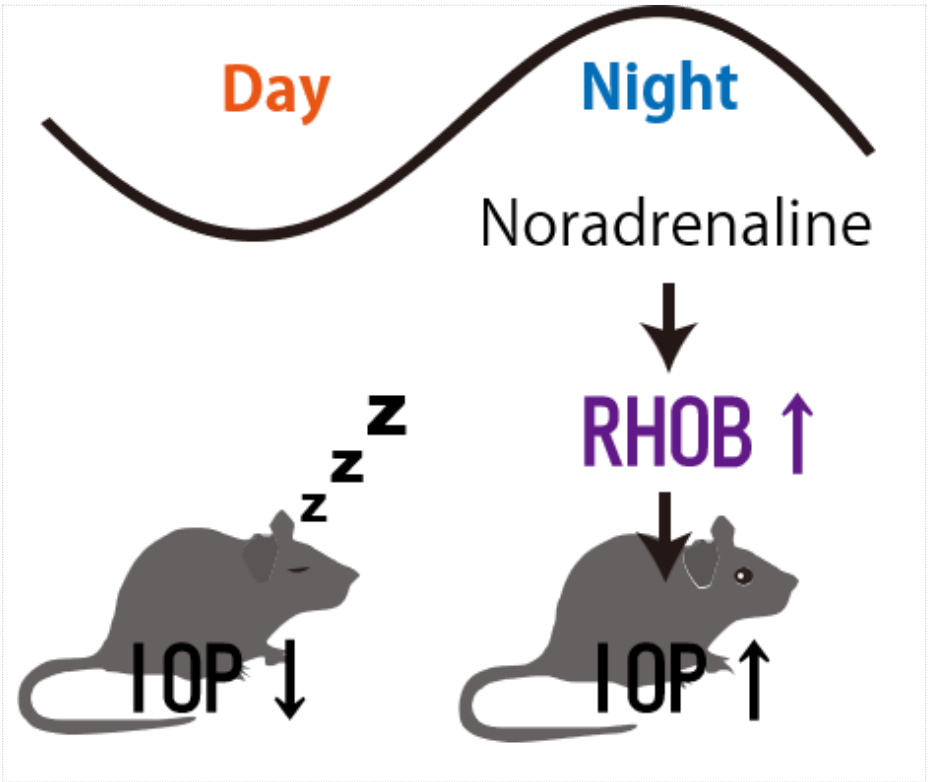


Fig. 1. Graphical abstract of the research results.
Using mice, researchers found that norepinephrine released from the sympathetic nervous system increases RHOB levels and suppresses the eye's drainage system, thereby contributing to the rise in intraocular pressure at night. (Keisuke Ikegami/Kyushu University)

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