

Rapid activation of specific genes readies the mammalian body for seasonal change

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The genes that regulate the process called photoperiodism—the seasonal responses induced in organisms by changing day length—have been found by researchers from the RIKEN Center for Developmental Biology, Kobe, and Kinki University, Osaka. Led by Koh-hei Masumoto and Hiroki R. Ueda from RIKEN, the researchers also discovered how

these genes can be activated within a single day. The work bears relevance to seasonal human disorders, such as winter depression, and symptoms associated with conditions such as bipolar disease.

Organisms need to alter body functions and behavior to accommodate seasonal changes in their environment. The measurement of day length is one obvious way of determining the time of year. To this end, the body uses its internal circadian clock, and against this background measures the extent and timing of light and dark.

The team noted that an increase in day length induces activity in the gene for thyroid stimulating hormone beta (TSH β) in the pars tuberalis (PT) region of the pituitary gland. TSH β plays a key role in the pathway that regulates photoperiodism in vertebrate animals. However, the detailed mechanism that links information about day length with induction of the production of TSH β is unknown.

Masumoto, Ueda and colleagues found the [genes](#) that stimulate the activity of the TSH β gene in mammals by observing the activity of genes in the PT of photoperiod-responsive mice under chronic 'short-day' (eight hours of light) and 'long-day' (16 hours) conditions. They identified 57 genes stimulated by short days and 246, including TSH β , by long days.

Then, the researchers placed chronic short-day mice into a long-day regime—they switched off the lights eight hours later—and observed that it took five days for TSH β to become fully active. They could, however, stimulate full activity of TSH β within a single 24-hour period if they subjected the mice to a short burst of light during a sensitive 'photo-inducible' period late at night. Thirty-four other long-day genes responded in the same way, including the transcription factor, Eya3, which seemed a likely candidate for regulating TSH β activity. In laboratory studies, the researchers determined that Eya3 and its partner

binding factor Six1 do indeed act together to activate TSH β . And this activity is enhanced by two other genes, Tef and Hlf.

“We are next planning to identify the upstream gene of Eya3,” Ueda says. “And we are also hoping to elucidate why the photo-inducible phase is late at night.”

More information: Masumoto, K., et al. Acute induction of Eya3 by late-night light stimulation triggers TSH β expression in photoperiodism. *Current Biology* 20, 2199–2206 (2010)

Provided by RIKEN

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