# Inducing non-REM sleep in mice by novel optogenetical control technique

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Recently, optogenetics, which controls the activity of neuron using the light-activated protein, has been getting a lot of attention. This light-activated protein works like a switch of neurons by sensing specific color of light. This time, Associate Professor Akihiro YAMANAKA and Dr. Tomomi Tsunematsu from National Institute for Physiological Sciences (NIPS), succeeded in suppressing only the activity of the orexin neurons in the mice's brains (hypothalamus) when the optical switch was on, using the light-activated protein, halorhodopsin (eNpHR). This flipping on and off the optical switch led mice into sleep and wakefulness. Those mice fell into non-REM sleep (slow-wave sleep) only when the halorhodopsin-expressed orexin neurons were exposed to the light. It is reported in the *Journal of Neuroscience* published by the Society for Neuroscience in USA (July 20, 2011, Eastern Standard Time, USA) .

It has been known the orexin neuron is related to awaking of the brain so far. However, the details have not been clarified whether sleep can be actually induced, if so, what sort of sleep it is, when only the activity of the orexin neuron related to awaking is suppressed even for a short time. Associate Professor Akihiro YAMANAKA and the collaborators made transgenic mice introduced the light-activated protein called halorhodopsin (eNpHR) into their orexin neuron, which can suppress the activity of neuron when exposed to the orange light. By use of the optical switch onto these mice, they suppressed the activity of orexin neuron for one minute, and succeeded in the artificial inducement of sleep. There are two sorts of sleep, one is non-REM sleep which is deep sleep rarely dreaming, and another is REM sleep which rich in dreams. Their

experiments selectively induced only the non-REM sleep among two.

Associate Professor Akihiro YAMANAKA says"Narcolepsy, a sickness of abnormal sleep causes sudden sleep attack and muscle weakness, cataplexy, because the orexin neuron disappears for the long term. These mice reproduced sudden sleep similar to narcolepsy when suppressed the activity of the orexin neuron by the optical switch. However, the reproduced sleep was only non-REM sleep, and it did not become sleep onset REM sleep as a characteristic symptom of narcolepsy. It is expected that it would provide clues as to the underlying cause of narcolepsy by examining such a difference."

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