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There may be certain conditions under which animals can forgo sleep without serious consequences, even though scientists have considered it an activity that is absolutely indispensible, new research suggests.

Image: Matthew Thimgan and Cassandra VanDunk

When flies are starved, they are able to stay awake for long periods of time without suffering the negative outcomes of sleep deprivation, including cognitive impairment, according to a study published online today (August 31) in *PLoS Biology*.

The paper demonstrates "that in *Drosophila*, when there isn't food in the environment, they're able to increase waking time without any of the deleterious effects of sleep loss," said neuroscientist Jerry Siegel of the Center for Sleep Research at the University of California, Los Angeles, who did not participate in the study.

There seems to be "this ability to reduce sleep when it's adaptive for the animals to do that," Siegel added. "If there's food available and it can be found by exerting greater effort, then it's highly adaptive for them to suppress sleep."

By and large, sleep appears to be a necessary part of life, and sleep deprivation can lead to serious impairments in cognitive function and even death. But under certain conditions, the amount of sleep an animal needs appear to vary. Starvation, for example, is known to be arousing, with starved animals being more active and sleeping less, but until now, no one could say why.

Recently neuroscientist Paul Shaw of the Washington University School of Medicine noticed that in addition to staying awake more, starved flies were also less susceptible to the effects of sleep deprivation. They didn't, for example, sleep more after they ate to make up for the sleep they lost while hungry.

Comparing starved flies to flies that were forced to be awake for similar amounts of time by a brief physical jolt every 10 seconds, Shaw and his colleagues found that starved flies didn't seem to get sleepy or suffer the same cognitive impairments as normal sleep-deprived flies.

Furthermore, they used flies that lacked a functional copy of the canonical clock gene cycle, making them extremely sensitive to sleep loss, even dying within just 10 hours of being deprived of sleep. When these flies were starved, however, they survived nearly three times as long.

Indeed, parallels in vertebrates point to another interesting case of sleep deprivation, Siegel said -- that of migrating birds and marine mammals. Some whale species "reduce sleep for long periods of time, for a month or more, well beyond the duration of sleep loss that's lethal [experimentally], and yet these animals are quite active and responsive," he said. "So what we have assumed in the past -- that there's a fixed need for sleep and the more you get the better -- may be the wrong way of looking at sleep."

As for what mechanism may underlie these unexpected effects of starvation, the team suspected that metabolism genes may play a role. Along with the fact that starvation keeps flies awake and alert, sleep deficits in another species -- humans -- can lead to an increased risk of the metabolic disorders obesity and diabetes, as well as coronary disease.

Sure enough, mutations in fat metabolism genes appeared to affect flies' ability to withstand the effects of sleep loss. *Brummer (bmm)* flies, which are fat and resistant to starvation, were particularly affected by sleep loss, sleeping more afterwards to make up for the loss and showing greater signs of cognitive impairment. *Lipid storage droplet 2 (Lsd2)* flies, on the other hand, are lean and were able to stay awake without showing signs of sleepiness or cognitive decline.

"Our hypothesis is that fats themselves play a role in regulating sleep," Shaw said. They are, after all, signaling molecules, he said, and it's possible "that lipids themselves are somehow signaling to the brain that you should be sleepy or are initiating cascades that either result in impairment or can protect you from impairment."

While these results are currently limited to flies, the authors are "providing a blueprint for how we can potentially counteract the effects of sleep deprivation in humans," agreed endocrinologist and fat cell biologist Perry Bickel of the University of Texas Health Science Center, who was not involved in the research. Indeed, mice knockouts of the mammalian homologs of both *bmm* and *Lsd2* already exist, he said. "That will get us that much closer to understanding what's going on in humans."

M.S. Thimgan, et al., "The perilipin homologue, lipid storage droplet 2, regulates sleep homeostasis and prevents learning impairments following sleep loss," *PLoS Biology*, 8: e1000466, 2010.

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