



American College of Neuropsychopharmacology

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Sleeping Too Little May Contribute to Depression

Research Shows Depression-Related Brain Changes in Animals with Restricted Sleep

WAIKOLOA, HI (December 6, 2011) – Disrupted sleep is often a symptom associated with mood disorders, like depression and bipolar disorders, but researchers now believe that sleep problems may be a contributing factor in causing these disorders. Scientists presenting at the American College of Neuropsychopharmacology annual meeting today reported significant changes in brain structure and responses in sleep-deprived rats, which mimic the changes seen in the brains of people experiencing depression.

Researchers led by Peter Meerlo, PhD, University of Groningen in the Netherlands, looked at how sleep loss impacts specific neural circuits and chemical responses that are associated with depression. They found that after one week of only four hours of sleep a day, the rats demonstrated reduced sensitivity in the areas of the brain that regulate neurotransmitters and hormones associated with mood disorders. Changes appeared in the hypothalamus-pituitary-adrenal axis, areas of the brain that control reactions to stress and regulate mood and emotions. After one month of restricted sleep, the hippocampus – the area of the brain associated with memory and cognitive function – had decreased volume and produced fewer new cells.

“For a long time, sleep problems have been viewed as a consequence of depression. What we’re showing is that in some cases, sleep problems may be actually causing depressive symptoms,” said Meerlo. “We may need to think about addressing sleep problems independent of the mood disturbances in patients with depression.”

Rats received an injection of a serotonergic agent, which causes an acute change in hormone levels, body temperature and other physiological processes that are under control of serotonin. Dr. Meerlo and his team chose this procedure to test their hypothesis about sleep loss and depression in laboratory animals, as it has been used in clinical research settings with patients with depression. Measuring the magnitude of these physiological changes in response to the injection is an indication of the sensitivity of the serotonergic system, which regulates mood. At one week and one month, researchers observed a diminished response similar to that seen in people with depression, a sign that their brains were less efficient in transmitting serotonin signals.

Researchers also looked at the animals’ ability to recover from lost sleep. Following a week of sleep restriction, animals were allowed unlimited sleep for one week to test how their systems

recovered. Researchers saw that an entire week of recovery sleep was needed to increase the sensitivity of their serotonin 1A receptor back to original levels.

“The slow rate of recovery is one of the most surprising and important outcomes of this study,” said Meerlo. “It argues against the idea that our brains can quickly recover from sleep loss. You might feel subjectively more rested after recovery sleep, but there still may be traces of the impact of sleep loss in your brain,” said Meerlo.

Meerlo cautioned that mood disorders are complex and potentially caused by many factors including genes, stress and sleep. The role of sleep in people with mood disturbances may vary from person to person.

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ACNP, founded in 1961, is a professional organization of more than 900 leading scientists, including four Nobel Laureates. The mission of ACNP is to further research and education in neuropsychopharmacology and related fields in the following ways: promoting the interaction of a broad range of scientific disciplines of brain and behavior in order to advance the understanding of causes, prevention and treatment of diseases of the nervous system including psychiatric, neurological, behavioral and addictive disorders; encouraging scientists to enter research careers in fields related to these disorders and their treatment; and ensuring the dissemination of relevant scientific advances in these disorders.