Study links primary insomnia to a neurochemical abnormality

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A research abstract that will be presented at SLEEP 2009, the 23rd Annual Meeting of the Associated Professional Sleep Societies, is the first demonstration of a specific neurochemical abnormality in adults with primary insomnia (PI), providing greater insight to the limited understanding of the condition's pathology.

Results indicate that gamma-aminobutyric acid (GABA), the most common inhibitory transmitter in the brain, is reduced by nearly 30 percent in individuals who suffer from primary insomnia for more than six months. These findings suggest that primary insomnia is a manifestation of a neurobiological state of hyperarousal, which is present during both waking and sleep at physiological and cognitive levels.

According to principal investigator Dr. John Winkelman of Brigham and Women's Hospital, at Harvard Medical School in Boston, Mass., the recognition that primary insomnia is associated with a specific neurochemical deficiency helps validate the often misunderstood complaint of insomnia.

"Recognition that insomnia has manifestations in the brain may increase the legitimacy of those who have insomnia and report substantial daytime consequences," he said. "Insomnia is not just a phenomenon observed at night, but has daytime consequences for energy, concentration and mood."

The study included 16 non-medicated individuals (eight of whom were women) with PI and 16 individuals (seven women) who were deemed normal sleepers. Global brain GABA levels were measured in both groups. PI was established through clinical interviews, sleep diary, actigraphy use and polysomnograpy.

Source: American Academy of Sleep Medicine (news : web)