Today’s work pressure has lead people to curtail their sleep. However, all of the consequences of sleep deprivation are not clear. Histone acetylation is a key epigenetic mechanism responsible for controlling gene expression. Increased histone acetylation increases gene expression; reduced histone acetylation results in reduced gene expression. We hypothesized that as compared to spontaneous wakefulness, sleep deprivation may produce a significant increase in histone acetylation in the basal forebrain (BF) region. We chose the BF region because it is a critical brain region responsible for regulating various brain functions including sleep, vigilance, attention, learning and memory.

Methods: Male Sprague-Dawley rats were divided: The spontaneous wakefulness group was euthanized, two hours after dark onset, when the animals are maximally active. The sleep deprived group was kept awake for 6 hr and then euthanized. Sleep deprivation began at the onset of light period (during their normal sleep period) and was performed by “gentle handling” technique. The animals were kept awake by a slight touch of a brush or hands or introducing novel objects into the cage. Upon euthanization, the brains removed and processed for acetylated histone H3 immunohistochemistry in the BF region.

Results: Our initial results suggest that there was significant increase in the number of cells expressing acetylated histone H3 in the BF of sleep deprived animals as compared to the spontaneously awake animals.

Conclusion: Our results suggest that sleep deprivation causes increased histone acetylation in the BF. Increased histone acetylation may affect gene expression that may have long term consequences.