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Journal of Sleep Disorders & Therapy

Short Communication

The Transcriptomic Response of Brain to Sleep Deprivation

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DESCRIPTION

Sleep is a vital biological function that can be observed in organisms as diverse as fruit flies to humans. It allows for molecular replenishment and detoxification, as well as neuronal downscaling and memory consolidation, in an inactive state. Human sleep and wake are synchronized with other circadian rhythms and occur at optimal periods during the 24 hour circadian cycle. The proper time and duration (or even quality) of the sleep/wake cycle is a challenge in modern life. Work and lifestyle routines, as well as inappropriate light and food exposure, are examples. In humans, insufficient and irregular sleep has been linked to a variety of negative health effects, but the underlying molecular pathways are still unknown [1]. The progress of transcriptome has been quantified sleep deprivation to determine molecular correlates of sleep demand and to identify molecular pathways related to sleep functions. Early investigations of rat cerebral cortex transcript expression discovered that numerous genes were elevated during spontaneous or induced wakefulness (i.e., sleep deprivation) compared to their levels during sleep. Immediate early genes, metabolism, neurotransmission, and synaptic function genes, and genes coding for heat shock proteins or chaperones were among them [2]. These investigations also discovered genes involved in intracellular calcium homeostasis and glutamate signaling, suggesting that sleep may play a role in neuroprotection. Thousands of genes that are up or down regulated as a result of sleep loss.

Over 500 genes in human blood were impacted by extending awake to 60 hours, including genes involved in the immune system, stress response, and DNA damage repair, with many genes down regulated after sleep loss and then recovering with sleep. Using transcriptomics, researchers have lately attempted to find genetic correlates of sleep loss-sensitive and sleep loss-resistant phenotypes in humans [3]. In comparison to sleep loss-sensitive patients, subjects resistant to sleep loss showed a drop in the amplitude of expression of rhythmic transcripts, according to this microarray investigation. Such findings may one day allow for the prediction of an individual's ability to perform under high sleep. The comparison of circadian genes (i.e., genes with a circadian expression profile) identified from two human gene expression datasets that include four conditions: 40 hours of Total Sleep Deprivation (TSD) following sufficient sleep (8.5 hours per night), 40 hours of TSD following insufficient sleep (5.7 hours per night), and sleeping either in phase with melatonin, a marker of the central hypothalamic clock and biological night, or sleeping 12 hours out of phase with melatonin.

Researchers used the NGS platform to conduct transcriptome analysis in brain samples from control and Rapid Eye Movement Sleep Deprivation (REMSD) rats inside the mouse model. Genes involved in chromatin assembly, methylation, learning, memory, synaptic transmission control, neural plasticity, and neurohypophysial hormone production had their expression altered. Transcripts encoding histone subtypes and key actors in chromatin remodeling were found to be altered. Two small non-coding RNAs, RMRP and BC1, as well as mRNAs that transcribe neurotransmitters including OXT, AVP, PMCH, and LNPEP, were down regulated. At least some of these alterations are likely to affect REMS regulation and may contribute to the repercussions of REMS loss. As a result of the findings of this study, critical epigenetic regulators and neural plasticity genes linked to REMS and its loss have been identified. This study gives context and opens doors to understanding their individual functions in the complex behavioural network, particularly in regard to long-term REMS-related alterations [4].

CONCLUSION

The use of a combination of mouse and human transcriptome data to uncover genes and pathways that are impacted by sleep and influence health is proving to be a powerful technique. Recent transcriptome studies are beginning to discover probable molecular mechanisms and pathways that may underpin these effects, paving the way for more research into the creation of biomarkers for sleep disruption and potential intervention vectors. Pathways linked to glucose and lipid metabolism, immunological and inflammatory responses, hormone signaling, cellular signaling and cell cycle regulation, and transcription and translation regulation have all been discovered. However, study in this new field has only recently begun. In addition, data from additional human tissues/cells and other molecular technologies

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Received: 05-Apr-2022, Manuscript No. JSDT-22-17196; **Editor assigned**: 08-Apr-2022, PreQC No. JSDT-22-17196 (PQ); **Reviewed:** 22-Apr-2022, QC No. JSDT-22-17196; **Revised:** 29-Apr-2022, Manuscript No. JSDT-22-17196 (R); **Published:** 06-May-2022, DOI: 10.35248/2167-0277.22.S3.005.

Citation: Marco A (2022) The Transcriptomic Response of Brain to Sleep Deprivation. J Sleep Disord Ther. S3:005.

will be valuable (e.g., proteomics, cistromics, and metabolomics). Disrupted circadian rhythms are also thought to be linked to unfavorable health consequences such as metabolic disorders (obesity, type 2 diabetes), cardiovascular disease, and cancer.

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