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Ovariectomy does not exacerbate the negative effects of sleep deprivation on synaptic plasticity in rats

Vahid Hajali [a,b], Vahid Sheibani [a,*], Saeed E. Mahani [a,c], Zahra Hajializadeh [c], Mohammad Shabani [a], Hamzeh P. Aliabadi [c], Hakimeh Saadati [a,d], Khadijeh Esmaeilpour [a]

[a] Neuroscience Research Center, Kerman University of Medical Sciences, Kerman, Iran
[b] Quchan Higher Health Education Center, Mashhad University of Medical Sciences, Mashhad, Iran
[c] Department of Biology, Faculty of Science, Shahid Bahonar University of Kerman, Iran
[d] Faculty of Medicine, Ardebil University of Medical Sciences, Ardebil, Iran

HIGHLIGHTS

- Regardless of reproductive status, SD impaired the spatial memory.
- Regardless of reproductive status, SD impaired the hippocampal LTP.
- Regardless of reproductive status, SD did not inhibit the hippocampal BDNF expression.
- Plasma corticosterone levels were decreased after SD in experimental groups.

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ABSTRACT

In our previous work, we found that female rats showed more cognitive impairment than male rats following 72 h sleep deprivation (SD). Here, we compared the intact female with ovariectomized (OVX) rats to assess the potential modulatory effects of endogenous female sex hormones against the 48 h SD-induced cognitive and synaptic modulations. The multiple platform method was applied for SD induction and spatial performances were determined using Morris water maze (MWM) task. Early longterm potentiation (E-LTP) was evaluated in area CA1 of the hippocampus and PCR and western blotting assays were employed to assess hippocampal BDNF gene and protein expression. To reveal any influence of sleep loss on stress level, we also measured the plasma corticosterone levels of animals. Regardless of reproductive status, SD significantly impaired short-term memory and LTP, but did not significantly change the BDNF expression in the hippocampus. The corticosterone levels were decreased in both intact and OVX female rats following SD. These findings suggest that depletion of female sex steroid hormones does not lead to any heightened responsivity of female animals to the negative effects of SD on cognitive and synaptic functions.

1. Introduction

Over the preceding century, the normal sleep time per 24 h has been decreased by 1.5 h and this decline seems to continue to rise [1]. A large body of evidence from both human and rodent studies suggests that sleep has a key role in certain types of learning and memory [2]. A primary hypothesis was that post-training sleep contributes to the consolidation of new information into long-term (reference) memory [3]. Accordingly, sleep deprivation (SD) following a learning task causes subsequent memory impairment in both humans [4] and rodents [5]. However, as post-training sleep, sleep prior to learning can also facilitate memory consolidation by promoting the efficiency of related neuronal systems to process new information and encode new memories [3]. The studies in humans show that total SD for a single night impairs different kinds of subsequent memory, including episodic, motor, procedural, and working memory in humans [6,7]. Several reports in animal studies also show that one to five days of SD before a training task leads to impairments in hippocampus-dependent memories, such as spatial memory in MWM [8,9], working memory in Y maze [10], and emotional memory in plus-maze discriminative avoidance tasks [11]. This evidence is consistent with the fact that the hippocampus is extremely vulnerable to sleep loss [12–14]. In parallel with these alterations in cognitive function, a variety of cellular and molecular correlates involved in membrane excitability and synaptic plasticity within the hippocampus, including long-term potentiation (LTP) [15] and neurogenesis [14], along with the expression of various plasticity-

* Corresponding author at: Quchan Center of Higher Health Education, Imam Khomeini Avenue, Quchan, Iran.
E-mail addresses: Vhajali@yahoo.com, Hajaliv@mums.ac.ir (V. Hajali).

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