

Morphometric effects of maternal zolpidem administration on rat embryo

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Introduction. Zolpidem can affect the developing embryo via the activating of the GABA A receptors. One of the important organs that expressed this receptors is fetal brain. However it is used now as a sedative even by the pregnant women. So its effect on embryo is questioned here.

Materials and Methods. Female pregnant rats were randomly divided into 4 groups.

Control with saline injection intra peritoneal (i.p); Treated with 5, 10, 20 mg/kg, i.p. injection of zolpidem. Zolpidem was injected at 1st post mating day. On 18th day of pregnancy, fetuses were excluded and processed for anatomical characters such as body weight, body size and head size and the end data was analyzed.

Results. Our morphometric results show a significant decrease in the body weight, body size and size of the head of embryo related to the dosage.

Conclusion. GABA A receptors are expressed during development of rat brain nervous system and have multiple roles in development of mammalian nervous system. zolpidem has agonistic effect on this receptors. Based on this fact, zolpidem causes disorder function of this receptor and then in some disorders in the developing fetal brain. As a result of this, there will be some malformation in the rat embryo.

Keywords: Zolpidem; GABA A receptors; Fetal brain; Developing embryo

Distinct pathway of oxidative stress induced apoptosis in human neuroblastoma cell line: miR-182 and FOXO1 are involved

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It has been suggested that excess accumulation of Reactive Oxygen Species (ROS), termed oxidative stress may lead to neuronal death resulting in neurodegenerative disorders such as Parkinson's and Alzheimer's disease. However, the molecular mechanism of the oxidative stress-associated apoptosis is far to be elucidated. Recently it has been investigated that miRNAs are extensively involved in neural death. MicroRNAs are small oligonucleotides, approximately 22 nucleotides in length that regulate gene expression. It has been elucidated that they can bind to the 3' untranslated regions (3' UTR) of mRNA targets inducing their degradation or translational inhibition. In the current study using Stem Loop real time PCR and SK-N-MC neuroblastoma cell line, we investigated that oxidative stress dramatically reduced miR-182 which is a robust inhibitor of FOXO1. Thus, under oxidative stress FOXO1 has the opportunity to be translated leading to FOXO1 over expression. Finally, pro-apoptotic gene targets of FOXO1 e.g. Bim and Bax are up-regulated leading to apoptosis. To further confirm such events, we also demonstrated that