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Establishment of the mouse model for neural tube defects induced by maternal nutrient inositol deficiency

Jianhua Wang, Jin Guo, Xiuwei Wang, Huixuan Yue, Shen Li, Fang Wang, Zhen Guan, Zhiqiang Zhu, Bo Niu and Ting Zhang
Capital Institute of Pediatrics, China

Emerging findings from murine and epidemiological studies showed that periconceptional maternal nutrient inositol deficiency is one of the risk factor of neural tube defects (NTDs), but the mechanism is unclear. We established a maternal inositol deficiency NTDs mouse model for further using on the study of inositol deficiency related NTDs. C57BL/6 mice were injected with Li₂CO₃ (250 mg/kg-400 mg/kg body weight) which inhibit the key enzyme necessary for inositol synthesis and recycling (IMPase) on gestation day 7.5 (GD7.5). The myo-inositol levels were measured on 0 h, 4 h, 8 h and 16 h after injection respectively. With the increase in Li₂CO₃ doses, both the incidence of absorbed embryos and the rate of growth retardation increased. Rates of NTD embryos in each Li₂CO₃ group were different. The group treated with 350 mg/kg Li₂CO₃ had the highest rate of NTD (26.2%) and lowest lethality. The phenotype of NTDs included encephalocele (88.8%), anencephaly (7.4%) and spina bifida (3.7%). The inositol level of the pregnant mouse decreased significantly after the 350mg/kg Li₂CO₃ injection, and reached to the lowest inositol level 8 hours after Li₂CO₃ injection ($P < 0.01$). Serial transverse sections of brain in GD13.5 NTD embryos displayed obvious constrictive and deformed third, fourth and lateral ventricle due to over proliferation. The treated non-NTDs group also showed obvious hyper proliferation in the brain tissue and displayed serious changes of the neuroepithelial cells. The maternal inositol deficiency induced NTDs mouse model was successfully established, which could provide the animal model for further investigate the mechanism of inositol deficiency on NTDs occurrence.

Biography

Jianhua Wang is currently working at Capital Institute of Pediatrics, China. His research interests are pediatric nutrition, epidemiological and clinical studies, etc.

fywjh@163.com

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