extracellular solution for 3 hours, then maintained for 24 hours in regular medium that contained BDNF. The morphologic changes of neurons dyed by acridine orange/ethidium bromide were observed. Mitochondria membrane potential (MEP) by JC-1 dye was assessed with laser scanning confocal microscope. Lactic acid dehydrogenase (LDH) in supernatant was detected by autobiochemical analyzer. BDNF was detected by immunocytochemistry and assessed by optical density.

**RESULTS:** There were some apoptotic and necrotic neurons in the seizure-like discharge group. Compared with the control group, MEP was significantly decreased and LDH level and BDNF expression were significantly increased in the seizure-like discharge group. Compared with the seizure-like discharge group, MEP was significantly increased and LDH level was significantly decreased in BDNF-treated group, but there was no significant difference on BDNF expression between them.

**CONCLUSIONS:** Seizure-like discharge could induce injury to hippocampal neurons and could upregulate BDNF expression in hippocampal neurons. BDNF could relieve the damage of neurons induced by seizure-like discharge, so BDNF has protective effects on hippocampal neurons.

## EFFECTS OF VITAMIN A ON LUNG DEVELOPMENT IN THE RAT FROM EARLY AGE TO ADULTHOOD

**Submitted by Ting-Yu Li**
Ting-Yu Li, Hua Wei
*Children's Hospital, Chongqing Medical University, Chongqing, China*

**INTRODUCTION:** Epidemiologic studies show that nutritional deficiency can alter lung development and have later adverse effects on lung function and respiratory health. Vitamin A is an important nutrient and is considered important in lung development and maturation. Additional studies are required to address whether vitamin A deficiency adversely affects lung development from early age to adulthood and whether such effects can be blocked or reversed.

**OBJECTIVE:** Our aim was to study the effect of vitamin A on lung development in the rat from early age to adulthood.

**METHODS:** Female rats were divided into control, marginal vitamin A deficiency (MVAD), and vitamin A intervention (VAI) groups. Control dams and pups were fed a normal diet (6500 U/kg vitamin A). MVAD rats were fed an MVAD diet (400 U/kg vitamin A). VAI rats were fed an MVAD diet until the birth of the pups and thereafter were fed with normal diet while the pups were given vitamin A through intragastric administration. All pups were killed at 8 weeks of age. Blood serum vitamin A levels were measured. Lungs were weighed and stained for light microscopy.

**RESULTS:** The vitamin A level of the MVAD group was lower than that of the control group. Lung weight of MVAD rats was lower than that of the controls. Morphometric measurements showed that the alveolar number in MVAD rats was less than that of the controls, and alveolar septa were thicker than those of the controls. All results in VAI group were better than those in the MVAD group and showed no difference from the controls.

**CONCLUSIONS:** Vitamin A status in early life can affect the lung development from early age to adulthood. Such effects can be reversed by dietary intervention after birth.

## MARGINAL VITAMIN A DEFICIENCY IN PREGNANCY CAN INDUCE MEMORY DEFICIT IN ADULT OFFSPRING

**Submitted by Ting-Yu Li**
Ting-Yu Li, Hong-Mei Huang, Chun-Ting Mao, Ying Liu, Ping Qu, Li Yang
*Children's Hospital, Chongqing Medical University, Chongqing, China*

**INTRODUCTION:** Vitamin A deficiency in pregnancy has a negative impact on the development of offspring; however, little is known about the effect of maternal marginal vitamin A deficiency (MVAD) on the function of the central nervous system in children later in postnatal life.

**OBJECTIVE:** We investigated whether MVAD during the gestational period can cause learning and memory impairment of adult offspring.

**METHODS:** There were 2 offspring groups: an experimental group that had MVAD only in pregnancy and a control group. Serum vitamin A was monitored by high-performance liquid chromatography. Both groups were trained by Marris water maze task at 8 weeks of age. The hippocampal CA1 long-term potentiation was detected by electrophysiologic technique, and the free calcium ion concentration in cells was examined by confocal laser scanning microscopy.

**RESULTS:** No significant difference in the serum vitamin A level was observed between the 2 groups; however, the escape latency of the experimental group (10.50 $\pm$ 1.58 seconds) was longer than that of the control group (8.75 $\pm$ 1.19 seconds) in the behavior test. Correspondingly, the changes of field excitatory postsynaptic potentials slope of the experimental group (29.5% $\pm$ 4.6%) was significantly less than that of the control group (57.5% $\pm$ 8.6%), and the lower relative intensity of fluorescence in cells was seen in the experimental group (85.8 $\pm$ 17.1) compared with the control group (113.6 $\pm$ 20.5) after the tetanus stimulation.

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