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.

CONCLUSIONS: MVAD in pregnancy causes learning and memory impairment of adult offspring.

EFFECTS OF MARGINAL VITAMIN A DEFICIENCY ON LONG-TERM POTENTIATION IN YOUNG RATS

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

INTRODUCTION: Vitamin A is an essential micronutrient for brain development. Marginal vitamin A deficiency (MVAD) remains a subclinical public health problem in children, but little is known about the mechanism by which it affects brain development beginning from embryonic period and early postnatal period.
OBJECTIVE: The objective of this study was to study the effects of MVAD on the hippocampal CA1 long-term potentiation (LTP) in young rats.
METHODS: The MVAD group was fed a vitamin A–deficient diet (400 IU/kg vitamin A), and the control group was fed a vitamin A–sufficient diet (6500 IU/kg vitamin A) at 3 weeks before coitus. Serum vitamin A was assessed by high-performance liquid chromatography. Hippocampal CA1 LTP was detected by electrophysiologic technique, and the ultrastructure of synapses was observed by electron microscope.
RESULTS: The changes of field excitatory postsynaptic potentials slope (25.4% ± 2.01%) in MVAD rats aged 7 weeks was much lower than that in the control group (57.5% ± 8.6%). The changes of slope of field excitatory postsynaptic potentials induced by MVAD in young rats could be replenished after addition of retinoic acid (RA); however, LTP impairment was observed again after addition of RA antagonist into the solution of the control group. No differences of LTP were found after addition of $FeSO_4$ or $ZnSO_4$. The curvature of the synaptic interface of the MVAD group was less than that of the MVAD group that was supplemented with RA and of the control group.
CONCLUSIONS: MVAD during the embryonic and early postnatal period can directly impair the hippocampal CA1 LTP of young rats.

EFFECT OF BCG VACCINATION ON SPLENIC DENDRITIC CELL DEVELOPMENT IN NEONATAL BALB/C MICE

Submitted by Enmei Liu
Liu Enmei[a], Liu Chunhua[a], He Yun[b], Liu Wei[b], Li Xin[c], Jiang Liping[b], Yang Xiqiang[b]
[a]Division of Respiratory Disorders, [b]Department of Clinical Immunology, and [c]Department of Core Laboratory, Children's Hospital, Chongqing Medical University, Chongqing, China

INTRODUCTION: As an immunoregulator, Mycobacterium BCG has the potential to be applied in allergic disease such as asthma prevention in clinic. Previous studies showed that neonatal BCG vaccination promoted mouse splenic T helper 1 development.
OBJECTIVE: The objective of this study was to investigate further the impact of BCG vaccination on dendritic cell (DC) development in neonatal mice.
METHODS: Neonatal and adult BALB/C mice were divided into 2 groups: the control group and the BCG-treated group in which BALB/C mice were inoculated with 1×10^5 colony-forming units of BCG intraperitoneally. After 4 weeks, splenic cells were isolated and co-stimulatory molecules and major histocompatibility complex molecules were analyzed by flow cytometry on CD11c-positive cells.
RESULTS: $CD11c^+CD8\alpha^+$ and $CD11c^+CD8\alpha^-$ DCs were found in spleen cells of BALB/C mice. In comparison with the control group, the percentage of $CD8\alpha^-$ DCs was significantly decreased (45.00 ± 14.14 vs 67.00 ± 8.27) and that of $CD8\alpha^+$ DCs was strikingly increased (55.00 ± 14.14 vs 33.00 ± 8.27) in BCG-treated neonatal mice. In contrast, the percentage of $CD8\alpha^-$ DCs markedly increased from 57% to 70% and that of $CD8\alpha^+$ DCs noticeably decreased from 43% to 30% in adult mice that were vaccinated. BCG vaccination upregulated the expression of co-stimulatory molecules on DC in adult and neonatal mice.
CONCLUSIONS: Our results indicate that development of T cells was induced by BCG vaccination through an effect on DC differentiation and maturation in BALB/C mice, possibly not only by DC phenotype but also by cytokines.

IMPACT OF ZINC SUPPLEMENTATION ON RESPIRATORY AND GASTROINTESTINAL INFECTIONS: A DOUBLE-BLIND, RANDOMIZED TRIAL AMONG URBAN IRANIAN SCHOOLCHILDREN

Submitted by Nahid Masoodpoor
Nahid Masoodpoor, Shokoofeh Darakhshan, Darakhshan Darakhshan, Seyed Taghi Tabatabaei, Seyed Abualghasem Mosavat
Ali-Ebn-Abitaleb Medical Hospital, Rafsanjan, Iran

INTRODUCTION: In addition to inhibiting growth, mild zinc deficiency is probably associated with reduced resistance to infection in children, but it has been difficult to establish this link; however, children with severe

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Ting-Yu Li, Hong-Mei Huang, Chun-Ting Mao, Ying Liu, Ping Qu and Li Yang

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