sultation room, they sometimes prefer to be on their own.

CONCLUSIONS: Four different preference profiles were uncovered. Caregivers recognize these profiles in daily practice. Because the goal of Q-methodology is to establish different patterns but not their prevalence, the distribution of profiles will be explored in a large follow-up survey. Additional use of these profiles in daily practice will be also explored, because rank-ordering the statements stimulated adolescents to talk about care issues.

EFFECT OF PERINATAL IRON DEFICIENCY ON BEHAVIORAL DEVELOPMENTS AND MYELINATION AROUND THE HIPPOCAMPUS IN RATS

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INTRODUCTION: Iron deficiency is a common nutritional disorder. The effect of iron deficiency on myelination of specific brain regions and their relevant behavior has not been well documented.

OBJECTIVE: The objective of this study was to determine the consequences of perinatal iron deficiency on behavioral and myelination of specific brain regions in rats.

METHODS: Sixteen dams were randomly assigned to iron-sufficient or low-iron diets during gestation and lactation. Thereafter, all offspring were fed the iron-sufficient diet and were assessed for behavioral and neurologic developments. Behavioral assessments included sensorimotor function tests, a recognition memory task, and a spatial learning task. The density of myelination around the hippocampus was measured by 2′,3′-cyclic nucleotide 3′-phosphohydrolase (marker of oligodendrocyte) by means of immunohistochemistry and quantified by analysis of integrated optical density. The regions of interest included the corpus callosum and the fimbria of the hippocampus.

RESULTS: Iron-deficient rats showed behavioral impairments in sensorimotor functions and recognition memory task but no significant differences were found in the spatial learning task. Iron-deficient rats had significantly reduced density of myelination than control rats in the corpus callosum but had no difference in the fimbria of the hippocampus.

CONCLUSIONS: This study shows that perinatal iron deficiency can significantly alter the behavioral outcomes in rat pups and can profoundly influence the development of myelination in specific brain regions.

EXPRESSION AND MODULATION OF AQUAPORIN 5 IN HYPEROXIA-INDUCED LUNG INJURY

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INTRODUCTION: Bronchopulmonary dysplasia (BPD) is a common disease that is caused by mechanical ventilation with persistent high-concentration oxygen in newborns, especially in preterm infants. One of the most important reasons is oxygen toxicosis. In physiologic conditions, liquid in the lung tissue is also transferred by aquaporins (AQPs), but the mechanism of aquaporins in hyperoxia-induced lung injury and lung dropsy is not clear.

OBJECTIVE: The objective of this study was to explore the expression and the modulation of AQP5 in hyperoxia-induced lung injury.

METHODS: Lung tissue was harvested after high-concentration oxygen exposure on the third, seventh, and 14th days in rats. The expression of AQP5 mRNA level and the location were detected by reverse-transcription polymerase chain reaction and immunohistochemistry, respectively, and compared with that in rats that were administered an injection of dexamethasone.

RESULTS: AQP5 was strongly labeled in alveolar epithelial cells. The expression of AQP5 in hyperoxia groups (3, 7, and 14 days) revealed a notable decline as compared with the control group, with no change even in the hyperoxia 14-day group. There was no difference between hyperoxia groups and hyperoxia + dexamethasone groups on AQP5 mRNA level.

CONCLUSIONS: The significant decrease of AQP5 expressed in hyperoxia-induced lung injury may be an important reason for abnormal water movement, which leads to pulmonary edema. Dexamethasone seems to have no effect in modulating AQP5 expression in acute lung injury.

PROTECTIVE EFFECT OF *N*-ACETYLCYSTEINE ON HYPEROXIA-INDUCED LUNG INJURY AND ITS INTERACTION WITH P38 MITOGEN-ACTIVATED PROTEIN KINASE SIGNALING PATHWAY

Submitted by Feng Xu
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INTRODUCTION: *N*-Acetylcysteine (NAC) is an effective oxidation inhibitor, but the protection of NAC in hyperoxia-induced lung injury is unknown.

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