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

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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.

OBJECTIVE: The objective of this study was to explore the protective effect of NAC on hyperoxia-induced lung injury and change of p38 mitogen-activated protein kinase (MAPK) expression caused by NAC treatment.
METHODS: Forty Wistar rats were randomly assigned to room air (A), hyperoxia injury (B), hyperoxia + NAC (C), hyperoxia + SB203580 (D), or hyperoxia + NAC + SB203580 (E). The lung wet/dry ratio, pathology, and location and quantity of p38 protein were detected.
RESULTS: Although pathologic changes in group B included severe alveolar edema with inflammatory cell aggregation and red blood cell leakage, the lung micrographic pictures in groups C, D, and E were improved significantly compared with group B; p38-positive cells increased in group B compared with that in group A and labeled in many types cells in lung tissue, especially in infiltrative inflammatory cells. In groups C, D, and E, the positive cells remarkably decreased compared with those in group B; the quantity of p38 MAPK was higher in group B than in group A, and p38 expression in groups C, D, and E decreased significantly compared with group B but was higher than that in the control group. There was no significant difference of p38 quantity among the 3 groups.
CONCLUSIONS: Reactive oxygen species activated phospho-p38 MAPK signaling pathway, and NAC and SB203580 treatments reduced the extent of hyperoxia-induced lung injury, as evidenced by reduction of the wet/dry ratio and lung pathology. NAC may exert a protective effect on hyperoxia-induced lung injury through attenuation of reactive oxygen species–induced p38 MAPK activation.

STUDY OF PULMONARY SURFACTANT AND SURFACTANT PROTEIN IN RATS WITH LIPOPOLYSACCHARIDE-INDUCED ACUTE LUNG INJURY

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INTRODUCTION: The abnormal metabolism of pulmonary surfactant (PS) may have some relationship to acute lung injury (ALI).
OBJECTIVE: The objective of this study was to examine the alteration trend of PS and surfactant-associated protein (SP) in rats with lipopolysaccharide (LPS)-induced ALI.
METHODS: Fifty-six adult Wistar rats were randomly divided into the normal saline (NS) group and the ALI group. The levels of mRNA of surfactant protein A (SP-A) and SP-B were measured by reverse-transcription polymerase chain reaction during intravenous LPS administration at 1, 3, 5, and 7 hours. The content and component of PS in the bronchoalveolar lavage fluid (BALF) were measured by high-performance liquid chromatography. In addition, lung dry/wet weight ratio, the protein content of BALF, alveolar oxygen partial pressure, and histologic changes were detected.
RESULTS: Compared with the NS group, the ALI group developed severe lung damage; edema, hemorrhage, and inflammation were found. Total phospholipids in BALF at 1, 3, 5, and 7 hours were lower than those in the NS group; phosphatidylcholine at 3, 5, and 7 hours was lower than that in the NS group, whereas lysophosphatidylcholine at 1, 3, 5, and 7 hours was higher than that in the NS group. The expression of SP-A and SP-B mRNA at 3, 5, and 7 hours was less than that in the NS group.
CONCLUSIONS: The changed metabolism of PS may be responsible for the pathogenesis of ALI. It is mainly demonstrated by the decrease in total phospholipids and phosphatidylcholine and the decreased expression of SP-A and SP-B mRNA. Decrease in content and change in components of PS may play an important role in severe hypoxemia in ALI.

EXPRESSION CHANGE OF AQUAPORIN 1 IN HYPEROXIC LUNG INJURY

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INTRODUCTION: Bronchopulmonary dysplasia (BPD) is a disease that is caused by prolonged high-concentration oxygen therapy, and its typical pathologic character is edema of pulmonary alveolus. Aquaporins play an important role in the fluid transition.
OBJECTIVE: The objective of this study was to examine the expression change of aquaporin 1 (AQP1) in hyperoxia-induced lung injury and the mechanism of action in lung edema.
METHODS: Thirty-two juvenile Wistar rats were randomly divided into breathing room air ($n = 8$) and hyperoxia exposure ($O_2 > 95\%$; $n = 8$ at 3, 7, and 14 days, respectively). The distribution of AQP1 in the lung tissues and its mRNA expressions were detected by immunohistochemistry and reverse-transcription polymerase chain reaction.
RESULTS: Light microscopic findings in the hyperoxia group included edema, hemorrhage, and extensive inflammatory cells. The lung wet/dry ratio, the protein content in bronchoalveolar lavage fluid, and the lung leak index in the hyperoxia group were significantly higher than those in room air group. The expression of AQP1 mRNA in the lungs was significantly decreased at

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