sulation 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.

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

**Submitted by Feng Xu**

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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-ACETYL-CYSTEINE 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.
EXPRESSION AND MODULATION OF AQUAPORIN 5 IN HYPEROXIA-INDUCED LUNG INJURY
Feng Xu and Liping Tan
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