

3 days of hyperoxia exposure, minimized at 7 days, and increased from 14 days. Immunohistochemistry for AQP1 was seen primarily in microvascular endothelia cells around bronchus and alveolus and interstitial cells; the positive regions were similar between the room air group and the hyperoxia group, AQP1 protein expression in the lungs was significantly decreased at 3 days of hyperoxia exposure, minimized at 7 days, but increased at 14 days. The dynamic changes of AQP1 protein level coincided with the changes of AQP1 mRNA expression. **CONCLUSIONS:** Hyperoxic lung injury may induce regulative imbalance of aquaporin expression. It may be 1 of the reasons for lung edema caused by hyperoxic lung injury.

### **BASIC FEATURES OF HUMAN METAPNEUMOVIRUS CHINESE ISOLATE PROTEINS**

**Submitted by Xiaodong Zhao**

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**INTRODUCTION:** Human metapneumovirus (hMPV), initially described in 2001, is an enveloped RNA virus of the genus *Metapneumovirus*, subfamily Pneumovirinae, family Paramyxoviridae.

**OBJECTIVE:** We sought to clarify the basic features of hMPV proteins.

**METHODS:** Rabbits were immunized with inactivated virions of hMPV Chinese isolate, CHN05-01, to yield anti-hMPV antiserum. Antiserum was used as primary antibody to detect hMPV proteins by Western blotting. NetNglyc 1.0 server, NetOglyc 3.1 server, and the NetPhos 2.0 server were applied for predicting potential glycosylation and phosphorylation sites of proteins of prototype virus of subtype A, CAN97-83.

**RESULTS:** The highest reactive titer of the antiserum with hMPV antigens reached 1:500 in enzyme-linked immunosorbent assay. Potential glycosylation sites of G protein and phosphorylation sites of P protein were greatest among all hMPV proteins. G protein was shown as a narrow band with molecular weight between 55 and 72 kd (~68 kd), indicating that its glycosylation level is consistent and remarkably different from that of CAN99-80 and CAN99-81. F1 subunit of fusion protein displayed molecular weight between 40 and 55 kd (~48 kd), which is consistent with previous reports.

**CONCLUSIONS:** Basic features of 2 major membrane proteins of Chinese hMPV isolate were clarified, which will benefit future studies on protein function and the pathogenesis of this virus.

### **APOPTOSIS OF ALVEOLAR TYPE II CELL AND C-JUN N-TERMINAL KINASE SIGNAL TRANSDUCTION INDUCED BY OXIDATIVE STRESS**

**Submitted by Lu Zhongyi**

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**INTRODUCTION:** Alveolar epithelial apoptosis has been described in the early stages of bronchopulmonary dysplasia. The production of reactive oxygen species during hyperoxia is thought to contribute to alveolar epithelial apoptosis, but the molecular mechanisms of oxidative stress-induced alveolar epithelial cell death is unclear.

**OBJECTIVE:** The objective of this study was to explore the role of the c-Jun N-terminal protein kinase (JNK) pathway in the apoptosis of alveolar epithelial cells that is induced by oxidative stress.

**METHODS:** Primary cultured rat alveolar type cells were treated with 500  $\mu$ M hydrogen peroxide ( $H_2O_2$ ) at various time intervals (0, 1, 3, 6, and 9 hours), whereas some cells were pretreated with a specific JNK inhibitor (SP600125). Mitochondrial membrane potential (MMP) change, cell survival, and apoptotic ratios were measured by fluorescence microscopy, 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide assay, and flow cytometry analysis, respectively. The expression of phosphorylated JNK and Bax was detected by Western blot.

**RESULTS:**  $H_2O_2$  treatment resulted in cell apoptosis and a decrease of MMP and cell viability in a time-dependent manner. Meanwhile, the JNK was activated and peaked at 30 minutes, and the Bax expression level was increased. Pretreated SP600125 enhanced cell viability and decreased apoptotic ratios after  $H_2O_2$  treatment. The expression of Bax declined after using SP600125 compared with cells that were treated with  $H_2O_2$  only.

**CONCLUSIONS:** High levels of oxidative stress induced cell apoptosis in a time-dependent manner. The mechanisms of oxidative stress-induced cell apoptosis involves JNK activation, Bax upregulation, and MMP decrease. JNK activation could improve the expression of Bax and play a proapoptotic role in the regulation of apoptosis that is induced by oxidative stress.

### **EFFECTS OF MESENCHYMAL STEM CELL TRANSPLANTATION ON CARDIAC FUNCTION, STRUCTURE, AND ELECTROPHYSIOLOGY IN RABBITS WITH DILATED CARDIOMYOPATHY**

**Submitted by Tian Jie**

# BASIC FEATURES OF HUMAN METAPNEUMOVIRUS CHINESE ISOLATE PROTEINS

Xiaodong Zhao and Huang Lu  
*Pediatrics* 2008;121;S157  
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