Methods Studies were performed on 125 infants <29 weeks gestation who required mechanical ventilation at 7–14 days and received 24 days of iNO at 20–2 ppm. A control group of 19 infants did not receive iNO.

Results In NO-treated infants there was a significant dose-dependent increase of both urinary NOx and cGMP per creatinine (maximal 3.1- and 2-fold, respectively, at 10–20 ppm iNO) compared to off iNO. The ratio of cGMP to NOx, which may reflect efficiency of NO signalling via guanylate cyclase, was lower (mean 38%) at all doses of iNO compared to off NO. NOx and cGMP concentrations at both 2 ppm and off iNO were inversely related to severity of lung disease (Respiratory Severity Score) during the first month, and the NOx levels were lower in infants who died or developed BPD at term. NOx was higher in Caucasian compared to other infants at all iNO doses.

Conclusions Urinary NOx and cGMP are biomarkers of endogenous NO production and lung uptake of NOx, and levels at some doses reflect the severity of lung disease in infants. These results support a role of the NO-cGMP pathway in lung development and repair from injury.

PS-206 MATERNAL/NEONATAL VITAMIN D DEFICIENCY MAY BE A RISK FACTOR FOR DEVELOPMENT OF BRONCHOPULMONARY DYSPLASIA IN PRETERM INFANTS

Background and aims Vitamin D seems to play an important role in the pathogenesis of respiratory system diseases. The aim of this study was to evaluate the possible association between both maternal and neonatal 25-hydroxyvitamin-D (25-OHD) levels and the subsequent risk of bronchopulmonary dysplasia (BPD) development in preterm infants.

Methods Premature infants ≤32 gestational age and admitted to Neonatal Intensive Care Unit with a diagnosis of respiratory distress syndrome (RDS) between December 2012 and December 2013 were included in this prospective study. Blood for neonatal and maternal vitamin D levels were obtained from all infants and their mothers at the time of hospital admission. The maternal and neonatal demographic features, maternal vitamin D usage, maternal head cover status, birth season and neonatal morbidity and mortality were all recorded.

Results A total of 100 preterm infants were included and 31 of them developed BPD. The mean birthweight, gestational age, duration of ventilation and duration of oxygen supplementation were significantly higher in infants with BPD compared with those who did not develop BPD (p < 0.05). Both maternal (19 ± 2.2 vs 28.7 ± 7.6) and neonatal (7.1 ± 1.6 vs 14.8 ± 4.7) 25-OHD levels were significantly lower in infants with BPD (both p > 0.05). All of the infants with BPD had a 25-OHD level <10ng/ml that represented severe vitamin D deficiency (p < 0.05).

Conclusions This study suggested for the first time that maternal/neonatal vitamin D deficiency might be associated with increased risk of BPD in preterm infants.
population. DNA was extracted from whole blood and Guthrie card specimens. Surfactant single nucleotide polymorphism (SNP) genotyping was arranged using the Taqman technique. ACE gene primers were obtained for the previously known insertion/deletion polymorphism and PCR analysis was performed.

**Results**

236 infants born prematurely survived to 28 days postnatal age and contributed to genotype analysis. 106 infants did not develop BPD and 130 infants did develop BPD (54 mild, 29 moderate, 47 severe). Both gestational age and birth weight were significantly different between those infants who did and did not develop BPD and predicted BPD development with an area under the ROC of 0.88 and 0.82 respectively. We demonstrated using multifactorial statistical analysis that the inclusion of the ACE genotype to a predictive statistical model of BPD development improves the predictive potential of the model (area under ROC curve 0.88).

**Conclusion**

The presence of the ACE DD genotype is associated with a higher likelihood of developing BPD.

**PS-209  BCG VACCINATION CAN PREVENT HYPEROXIC LUNG INJURY?**

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**Introduction**

The aim of this study effects of BCG vaccine on the histopathologic and gene expression changes seen in hyperoxia induced newborn rat lung injury.

**Method**

Twenty-three rat pups were divided into four groups: air-exposed control group (n = 5), hyperoxia-exposed placebo group (n = 7), hyperoxia-exposed BCG-vaccinated group (n = 7), and air-exposed BCG-vaccinated group (n = 4). Neonatal hyperoxic lung injury model was established according to the previous studies. Measurement of alveolar surface area, quantification of secondary crest formation, microvesSEL count, evaluation of alveolar septal fibrosis, and smooth muscle actin immunostaining were performed to assess hyperoxia-induced changes in lung morphology. The gene expression level was evaluated by RT-PCR.

**Results**

The alveolar surface areas were significantly different between the oxygen exposed placebo group and oxygen exposed BCG vaccinated group (alveolar surface area: 0.29 ± 0.02 μm² and 0.52 ± 0.04 μm² p < 0.05 respectively). Number of crests and microvessel count was found to be significantly increased in the oxygen exposed BCG vaccinated group compared with the animals in the oxygen exposed placebo group (p < 0.05). Exposure to hyperoxia resulted in a significant decrease in mean alveolar surface area and number of crests formed compared with air-exposed animals (p < 0.05). The degree of fibrosis was found to be significantly increased in the oxygen exposed placebo group compared with the animals in the oxygen exposed BCG vaccinated group (degree of fibrosis: 1.88 ± 0.33 and 0.91 ± 0.66 p < 0.05 respectively). Immunostaining for SMA demonstrated hyperoxia-exposed animals with BCG vaccine in a significantly decrease in smooth muscle content compared with hyperoxia-exposed placebo animals (p < 0.05). The expression of VEGF, FGF1, IL13, NFkB1 and TNFα in the lungs of vaccinated animals was significantly higher than that of non-vaccinated animals (p < 0.05).

**Conclusion**

Our results suggest that BCG vaccination can be a new protective strategy against neonatal hyperoxic lung injury.