population. DNA was extracted from whole blood and Guthrie card specimens. Surtarget 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 neonatal 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.

**Results** 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 and 0.52 ± 0.04 m² 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, NFLB1 and TNNF 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.

These beneficial effects may be interpreted with its immunomodulatory effects on proinflammatory-antiinflammatory cytokine balance and expression of growth factors.