European Respiratory Society
Annual Congress 2012

Abstract Number: 1209
Publication Number: P4126

Abstract Group: 7.5. Neonatology and Paediatric Intensive Care
Keyword 1: Lung injury **Keyword 2:** Neonates **Keyword 3:** Biomarkers

Title: The association of NQO1 $C^{609}T$ polymorphism and the susceptibility of bronchopulmonary dysplasia in preterm neonates

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Body: Introduction: The development of bronchopulmonary dysplasia (BPD) involves genetic and environmental factors. Supplemental oxygen can damage the pulmonary epithelium through the generation of reactive oxygen species (ROS). The NAD(P)H:quinone oxidoreductase (NQO1) enzyme is involved in ROS detoxification. The encoding gene is subjected to the inactivating sense polymorphism ($C^{609}T$). Individuals homozygous for the mutant allele (T/T) completely lack NQO1 activity, whereas heterozygotes (C/T) have low enzyme activity. We conducted a case-control study to investigate the potential role of NQO1 inborn polymorphism in BPD susceptibility. Patients/Methods: The study enrolled 119 Greek premature neonates with gestational age ≤32 weeks; 42 developed BPD (case group) and 77 did not develop BPD (control group). Genomic DNA was extracted from peripheral blood of all neonates. NQO1 genotyping was performed by real-time PCR. The NQO1 gene status was also evaluated in respect to gender. Results: A different distribution of NQO1 genotypes was found between BPD neonates and controls, with a higher frequency of variant genotypes (heterozygotes C/T and homozygotes T/T) in BPD population (50% in BPD vs 28.6% in no BPD neonates, p=0.046). Thus, a significantly higher frequency of the mutant T allele was observed in BPD neonates as compared to controls (0.27 vs 0.17, p=0.058). According to gender, we observed no genotypic differences. Conclusion: The higher incidence of the C/T and T/T variant genotypes among neonates who developed BPD suggests that the NQO1 $C^{609}T$ inborn polymorphism probably constitutes an early biomarker of BPD development.