congenital anomalies, late-onset sepsis (LOS) (57.8% vs. 23.8%; OR:4.40, 95%CI:4.0–4.84) and symptomatic PDA (56.0 vs. 30.6%; OR:2.9, 95%CI:(2.6–3.2)). After adjusting for all BPD predictive peri-
natal risk factors (BW, GA, Apgar scores, gender and congenital anom-
alias (AUC:0.8, 95%CI:0.79–0.81), the factors strongly associated with
BPD, other than BW and GA, were LOS (OR:2.54, 95% CI: 2.7–2.83) and symptomatic PDA (OR:1.54, 95% CI:1.38–1.73).

**Conclusion**

In this large cohort of VLBW/VLGA, the rate of BPD was
16% (15.4–16.1%), strongly associated with GA and BW but also with LOS and symptomatic PDA.

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**596** EARLY PREDICTION OF BRONCHOPULMONARY DYSPLASIA (BPD) BY AN EASILY AVAILABLE RISK SCORE

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**Background**

Early prediction of BPD is important for identifying high risk patients likely to benefit from preventive treatment approaches and for providing prognostic information. Therefore we aimed to develop a risk score for BPD based on early available clinical parameters.

**Patients** and methods: All infants born at the University Hospital of Lausanne < 32 weeks of gestation (WG) between 1998 and 2007 (n=956) were included. Patients diagnosed with RDS (n=223) were divided in two groups, either developing BPD or not. Independent risk factors for the development of BPD were searched by multivariate logistic regression analysis. The β-coefficients (β= log(OR)) derived from the fitted multivariate model were used to build a scoring system. An internal validation was performed using a two-fold cross-validation technique with two subgroups: two thirds of the patients were used as training set for model calibration and one third as prediction set.

**Results**

BPD-risk score was developed based on five covariates: intubation in the delivery room, early neonatal infection, duration of invasive mechanical ventilation in days, birth weight and gesta-
tional age, weighted according their β-coefficients. Area under curve (AUC) was 0.896. Sensitivity and specificity reached 82.7% and 82.6% with a score cut-off of -3 (range -25 to +17). Internal calibration proved a good prediction: AUC for the same cut-off was 0.882 for the training set and 0.927 for the prediction set.

**Conclusions**

A simple scoring system available within the first postnatal week can reliably predict the probability of developing BPD in infants born < 32 WG.

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**597** VALPOVIC ACID-MEDIATED PROTECTION AGAINST HYPEROXIC LUNG INJURY VIA HISTONE DEACETYLASE INHIBITION IN A NEONATAL RAT MODEL

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Epigenetic mechanisms might play an important role in development of BPD. The aim of this study was to evaluate the protective effect of valproic acid (VPA), an histone deacetylase inhibitor, in hyperoxic lung injury in neonatal rat model.

**Methods**

A total of 30 rat pups (0 days old) were divided equally into 3 groups: control, hyperoxia and hyperoxia+VPA groups. In hyperoxia groups, pups were maintained in 95% O₂ for 10 days while control group was maintained in room air. VPA was administered intraperitoneally once daily for the first 10 days of life.

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**598** EFFECTS OF CATALYTIC ANTIOXIDANT MNTBAP ON PULMONARY ANGIogenic AND OXidative GENE EXPRESSION TO HYPERoxia IN NEWBORN MICE

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**Background**

Development of lung injury during prolonged O₂ exposure is a complex process, associated with changes in expression of a number of genes important in the adaptive response to hyperoxia. MnTBAP is a compound with strong antioxidant properties.

**Objective**

To study the effects of MnTBAP on angiogenic and oxidative gene expression in C57BL6 neonatal mice following hyperoxia.

**Design** and methods: Newborn mice litters were randomized on postnatal day 4 to hyperoxia (> 95% O₂) (OX) or room air (RA) for 72 hrs during which they received MnTBAP (MN) 10mg/kg or saline (SL) daily by IP injection for 3 days and then were sacrificed. Whole lung angiogenic and oxidative gene expression profiling (84 related genes for each) was done by real-time, reverse transcription, quantitative PCR (n=4). Data was processed and analyzed using SA Biosciences PCR array data analysis web portal.

**Results**

Hyperoxia significantly upregulated peroxiredoxin 6 expression compared to room air exposed newborn mice. Treatment with MnTBAP downregulated the expression of myeloperoxidase and Pdxdh-6-r1. Hyperoxia downregulated the expression of angiogenic genes such as angioptin 1 & 2, TGF 1, TGF 3 and HGF; MnTBAP treatment during the hyperoxia exposure reversed this effect and these genes were upregulated.

**Conclusions**

The catalytic antioxidant MnTBAP reversed the effects of hyperoxia on angiogenic gene expression in newborn mice. The protective effects of antioxidants in newborn hyperoxia models need to be studied further to provide additional understanding of the management of bronchopulmonary dysplasia.