surfactant, varespladib, both or nothing (overall p=0.016; Dunnett post-hoc between cultures treated with varespladib and varespladib+surfactant against untreated cultures p=0.01). FFA are higher in untreated cultures (394±82 µM), than in surfactant-(219±70 µM) and in varespladib-treated ones (142±51 µM). Combined treatment reduced FFA to 206±47 µM (overall p=0.017; Sidak post-hoc p=0.036 and p=0.023 for the varespladib and combined treatment against control cultures).

Conclusions The joined administration of varespladib and poractant-α significantly reduce sPLA2 activity and FFA production. Surfactant+varespladib affect sPLA2 pathway significantly more than the surfactant alone.

Abstract 59
THE PEDIATRIC ALIEN STUDY: INCIDENCE AND OUTCOME OF THE ACUTE RESPIRATORY DISTRESS SYNDROME IN CHILDREN

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Introduction The incidence and outcome of the acute respiratory distress syndrome (ARDS) in children is not well known, especially under current ventilatory practices. The goal of this study was to determine the incidence, etiology and outcome of ARDS in the pediatric population in the setting of lung protective ventilation.

Method A 1-year, prospective, multicenter, observational study in 12 geographical areas of Spain covered by 21 pediatric intensive care units (PICUs).

Results Data on ventilatory management, gas-exchange, hemodynamics, and organ dysfunction were collected. A total of 146 mechanically ventilated patients fulfilled the ARDS definition, representing an incidence of 3.9/100,000 population ≤15 years of age/year. Pneumonia, sepsis and respiratory syncytial virus-related infection were the most common causes of ARDS. At the time of meeting ARDS criteria, mean PaO2/FiO2 was 99±41 mmHg, mean tidal volume was 7.6±1.8 ml/kg predicted body weight, mean plateau pressure was 27±6 cmH2O, and mean PEEP was 8±2.9 cmH2O. Overall ARDS PICU and hospital mortality was 26% (95%CI: 19.6–33.7) and 27.4% (95%CI: 20.8–35.1), respectively. At 24 h, after assessment of oxygenation under standard ventilatory settings, 118 (80.8%) patients continued to meet ARDS criteria (PaO2/FiO2 104±36 mmHg; PICU mortality 30.5%) whereas 28 patients (19.2%) had a PaO2/FiO2 >200 mmHg (PICU mortality 7.1%) (p=0.014).

Conclusions This is the largest study to estimate prospectively the pediatric population-based ARDS incidence and the first incidence study performed during the routine application of lung protective ventilation in children. Our findings support a lower ARDS incidence and mortality than those reported for adults.

Abstract 60
TABLE 1

<table>
<thead>
<tr>
<th>OXYGENATION INDEX</th>
<th>BASELINE</th>
<th>1h</th>
<th>3h</th>
<th>6h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>79 (52)</td>
<td>46 (18)</td>
<td>52 (30)</td>
<td>64 (27)</td>
</tr>
<tr>
<td>PLV</td>
<td>102 (49)</td>
<td>6 (11)*</td>
<td>12 (6)*</td>
<td>32 (15)</td>
</tr>
<tr>
<td>PFC-aero</td>
<td>71 (45)</td>
<td>23 (11)*</td>
<td>10 (2)*</td>
<td>18 (18)*</td>
</tr>
</tbody>
</table>

* vs. CONTROL; # vs. PFC-aero. One-way ANOVA

Conclusion Both PFC administration techniques show pulmonary efficacy in RDS. Future research should focus on the PFC aerosol delivery efficiency.

Abstract 61
ASSOCIATION OF VITAMIN D RECEPTOR GENE POLYMORPHISMS AND BRONCHOPULMONARY DYSPLASIA

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Background and aims Vitamin D is considered as an important regulator of fetal lung development and innate immune system. Its functions involved in susceptibility and resistance to infections and pulmonary diseases may be important for the occurrence of bronchopulmonary dysplasia (BPD). The aim of the study was to investigate the relationship between Vitamin D receptor gene polymorphism and BPD in preterm infants.

Methods Fok I, Bsm I, Apa I, and Taq I polymorphisms in the Vitamin D Receptor (VDR) gene were genotyped using restriction fragment length polymorphism in 109 preterm infants (47 with BPD, 62 without BPD) born at gestational age ≤32 weeks and admitted to NICU at Ege University Hospital.

Results The univariate analysis showed Ff (OR=3.937, p=0.022, 95% CI 1.22–12.69) and ff (OR=5.238, p=0.004, 95% CI 1.69–16.23) genotypes of Fok I polymorphism were associated with increased risk of BPD, whereas tt genotype of Taq I polymorphism; was associated with a protective effect against BPD (OR=0.30, p=0.04, 95% CI 0.098–0.894). In a multivariate logistic regression analysis of the model including variant FokI genotype with significant PDA, clinical and culture proven sepsis, mechanical ventilation and surfactant treatment; variant Fok I genotype increased the risk of BPD (OR=4.115, CI=1.080–15.696, p=0.038) independent from these factors. Taq I, Bsm I and Apa I polymorphisms did not have any effect in the same model.

Conclusion FokI polymorphism was associated with increased frequency of BPD after adjusting for multiple confounders. VDR gene polymorphisms may be suitable for prediction of infants at high risk for BPD.