PS-279 PROLONGED OXIDATIVE STRESS AND INCREASED INCIDENCE OF NEONATAL MORBIDITIES AFTER EARLY POSTNATAL EXPOSURE TO OXIDANTS IN INFANTS LESS THAN 29 WEEKS GESTATIONAL AGE

Background The antioxidant defenses are poorly developed in preterm infants. Oxygen and parenteral nutrition (PN) which is contaminated with peroxides are two major sources of oxidants. 

Objective To assess the effect of early oxygen (on day 7 and 28) and the PN duration on oxidative stress markers at 36 weeks post menstrual age (PMA) and on the incidence of neonatal morbidities.

Design/methods A prospective observational study including 120 infants less than 29 weeks gestational age without major congenital anomalies. Consent for blood sample at 36 weeks PMA was obtained for 51 infants. GSH and GSSG (nmol/mg protein) were measured by capillary electrophoresis and were used for redox potential (mV) calculation using Nernst equation, and expressed as mean (± sem). BPD was defined as the need of O2 supplement at 36 weeks PMA. ROP that required either laser or anti-VEG treatment and NEC grade 2 or higher according to Bell criteria were included. Students’s t test or Chi squared were used as appropriate, * = p < 0.05, ** = p < 0.01.

Results FiO2 ≥ 25% on day 7 and 28 of life and PN duration > 14 days resulted in higher GSSG concentration, more oxidised redox potential at 36 weeks PMA and increased the incidence of BPD, ROP and NEC.

Conclusions Early life exposure to oxidants is associated with prolonged oxidative stress and higher incidence of neonatal morbidities. These results suggest that strategies targeting judicious O2 use and either decreasing the duration or using safer formulation PN will help decreasing the incidence of BPD, ROP and NEC.

Abstract PS-279 Table 1

<table>
<thead>
<tr>
<th>FiO2 &lt; 25% on day 7</th>
<th>GSH (µmol/mL)</th>
<th>GSSG (µmol/mL)</th>
<th>Redox potential</th>
<th>BPD or NEC</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.6 (0.5)</td>
<td>0.18 (0.02)</td>
<td>-198 (2)</td>
<td>26/54</td>
<td>25/65</td>
</tr>
<tr>
<td>7.4 (0.6)</td>
<td>0.29 (0.04)</td>
<td>-191 (2)</td>
<td>46/50</td>
<td>65/50</td>
</tr>
<tr>
<td>P</td>
<td>NS</td>
<td>*</td>
<td>**</td>
<td>NS</td>
</tr>
</tbody>
</table>

S/P-280 SURFACE FILM FORMATION IN VITRO BY INFANT AND THERAPEUTIC SURFACTANTS: ROLE OF SURFACTANT PROTEIN B

Background Essential surfactant properties include transfer to gas-liquid interface, reduction of surface tension and film replenishment during respiratory cycles.

Objective To compare component-specific film formation properties of infant and therapeutic surfactants.

Design/methods Using a multilayer fluorescence assay, we compared maximal fluorescence (Max), time to reach Max (tMax) and phospholipid concentration for 1/2 maximal signal (1/2Max) for calf surfactant (CAL), porcine surfactant (POR), bovine surfactant (BER), colostrum lipids (COL), with surfactant from immature infants with RDS. Dose-response studies were performed for addition of SP-B, albumin and budesonide.

Results Max and 1/2Max values for CAL were higher/similar to those of rat surfactant. There were significant differences between CAL and other therapeutic surfactants for Max (CAL > COL > POR > BER) whereas 1/2Max were similar except for COL.

In surfactant from 39 infant tracheal aspirates, 1/2Max was inversely correlated with SP-B content (p = 0.001). Addition of SP-B to samples with low endogenous content (<0.1%) decreased 1/2Max in a dose-dependent way. Addition of 1.25% SP-B to BER (SP-B content 0.04%) increased Max by 324%. Addition of albumin to CAL (0.75 µg/g PL) increased 1/2Max by 110% and reduced Max by 13%. By contrast, addition of budesonide to CAL at 2% and 10% increased Max by 51 ± 26% and 93 ± 19%, with no effect on 1/2Max.

Conclusions This assay reveals differences in film formation efficiency for therapeutic surfactants reflecting differences in SP-B content and lipid composition. Film formation by infant surfactant is strongly influenced by SP-B content. The findings support the key physiological role of SP-B and the safety of surfactant as anti-inflammatory drug vehicle.