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| **A pilot study assessing the wound repair capacity of the upper airway epithelium of children born prematurely** |
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| **Introduction/Aim:** Premature birth is the leading cause of mortality among children under 5 years of age; primarily from lung complications. The airway epithelium protects the body from the external environment, eliciting rapid repair when injured to reinstate barrier integrity. However, in some disease states this mechanism is potentially defective. In this study we aimed to characterise the repair mechanisms of the airway epithelial cells in preterm infants.  **Methods:** Upper airway nasal epithelial cells (NECs) obtained by cytological brushing of the nasal turbinate from 10 children born very preterm (25-31 weeks gestational age) at 1.07-1.22yrs corrected age (8 males; 3 BPD) and 6 full-term children (2.4-6.5yr; 4 males) were used to establish primary cell cultures. Twenty four hours prior to wounding, cell monolayers were cultured in restricted media and subsequently wounded (Essen CellPlayer® WoundMaker®). Monolayers were washed to remove cellular debris and media replenished every 48h. Time-lapse images were captured and analysed every 30 minutes with the IncuCyte® ZOOM. Wound recovery was calculated by manual tracing of the new wound edge at each time interval and comparing the wound width to that of the originally created wound edge. Calculated values were then expressed as a percentage of total wound recovery over the period to achieved full repair.  **Results:**  Healthy full-term NECs were found to fully repair wounds by 36 hrs. In contrast, the preterm NECs only completed 37.5% repair on average at the same time point. At 72 hours post injury, two preterm NECs exhibited significantly dysregulated repair (20-30% repair), five achieved 50-80% wound closure, 1 >90 repair with 2 achieving full repair.  **Conclusion:** Data generated suggests that there is an inherently delayed ability of preterm infant upper airway AEC to successfully repair which may contribute to their increased susceptibility to environmental pathogens.  **Grant Support:** Perpetual IMPACT Philanthropy Grant:IPAP2017/1355  **Declaration:** No conflict of interest |