STRONG CHILDREN'S RESEARCH CENTER

Summer 2015 Research Scholar

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ABSTRACT

Title: ***Neonatal Oxygen Exposure leads to Altered Alveolar Epithelial Type-II cell pools, affecting the Response to Influenza A Virus at 2 week and 4 weeks of Age***

Background: Premature infants, specifically those at low birth weight, often have underdeveloped lungs that are physiologically and structurally immature, making them susceptible to sequelae as developing children and young adults. In addition, children born prematurely often display reduced pulmonary lung function and lung capacity, placing them at increased risk for numerous disorders such as bronchopulmonary dysplasia (BPD), respiratory viral infections and asthma. Such changes have been attributed to life-saving early-life exposure to oxygen that reprograms the development of the lungs, eyes and brain. Oxygen supplementation during the neonatal period influences lung development, alters respiratory abilities, and hinders host defenses of the lung,

infection for analysis. Manual counting and quantification was conducted using SP-C and ABCA3 staining, two different genes expressed by alveolar type II cells. Western blotting for SP-C was also conducted and electronically quantified.

Hypothesis: Neonatal oxygen exposure leads to a rapid expansion and then pruning of alveolar type II cells. At 2 weeks of age, hyperoxia exposed mice possess a heightened number of type II cells. The ability of type II cells to secrete various antiviral components and act as immune cells subsequently protects mice from respiratory infections such as IAV as 2 weeks of age.

analogous type II cell populations. At this time point morbidity and mortality outcomes due to IAV infection were very similar. These similar outcomes were presumably due to their relative numbers of type II cells, subsequently generating immune responses of comparable magnitudes. Taken together this data suggests that early life oxygen exposure disrupts the proper number of type II cells required to defend the lung against respiratory infection, providing protection at 2 weeks of age and increasing susceptibility as the mice grow into adulthood.