

**STRONG CHILDREN'S RESEARCH CENTER**

**Summer 2019 Research Scholar**

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**ABSTRACT**

**Title:** *Role of AMPK in Neonatal Hyperoxia-induced Cardiovascular Disease*

**Background:** Premature infants needing the most supplemental oxygen have higher risk of cardiovascular disease due to poorly understood mechanisms. A mouse model was established in which exposure to hyperoxia from birth to postnatal day (PND) 0 to 4 causes pulmonary hypertension and heart failure. These effects are preceded by the failure to expand cardiomyocytes (CMs) lining the pulmonary vein (PV) and left atrium (LA). Gene expression profiling shows that fatty acid synthesis is suppressed in the atria of mice exposed to neonatal hyperoxia on PND4. Enzymes needed for fatty acid synthesis such as Fatty acid synthase (FASN) may be responsible for the hyperoxia-induced suppression of fatty acid synthesis.

**Objective:** To determine if AMPK is responsible for the hyperoxia-induced loss of fatty acid synthesis genes and CM proliferation in the PV and LA.

**Methods:** *Cell Proliferation Analysis* HL-1 cells plated at equal density in 96 well plates were also treated with increasing doses of the AMPK activators Metformin (Dosages: 0, 0.75, 1.5, 3, 6 and 12mM) and AICAR (Dosages: 0, 0.5, 1, 2mM)