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**Abstract Title:**

**The impact of maternal hypoxia-induced intrauterine growth restriction on postnatal airway smooth muscle thickness and contractility in mice**

**Level of experience (i.e. Honours student, Research Assistant, First year PhD, Second year PhD, Third year PhD, Postdoc):**

**Honours student**

**ASMR Member (YES / NO):**

**NO**

**\**Please note: you must be an ASMR member to be considered for an award. If eligible for an award, please be sure to keep available for the night of the Gala Dinner (Friday June 8th).***

**Please nominate 1-2 subject areas relevant to your submitted abstract:**

|  |  |
| --- | --- |
| Physiology  Reproductive and developmental biology  Cell biology  Biotechnology  Molecular biology  Psychology  Animal models | Biochemistry  Immunology  Clinical research  Genetic medicine  Pathology  Dentistry  Other (please specify): Respiratory biology |

**The impact of maternal hypoxia-induced intrauterine growth restriction on postnatal airway smooth muscle thickness and contractility in mice**

Authors: Darshinee Kowlessur1, 2, Peter Noble2, Kimberley Wang1

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**Background:** Asthma is an obstructive airway disease that produces considerable morbidity for the affected individual, including breathing difficulty and need for medical treatment. Asthma is highly prevalent in children; it is more prevalent in males up to the age of 14, and thereafter is more dominant in females. A primary structural abnormality in asthma is an increase in the thickness of the airway smooth muscle (ASM) layer, which is considered a likely cause of the ensuing functional defects, notably excessive narrowing of the airway passages and airflow limitation. Since the thickness of the ASM layer is increased early in life (in children with asthma) and even before diagnosis, it is possible that the ASM is thickened prior to birth. An association between intrauterine growth restriction (IUGR) and asthma has been demonstrated, although the mechanism is unknown.

**Aims and hypothesis:** The aim of the study is to examine the impact of maternal hypoxia-induced intrauterine growth restriction (IUGR) on ASM thickness and contractility in trachea from mice and establish whether there are any differences between male and female offspring. We hypothesize that hypoxia-induced IUGR increases the thickness of the ASM layer and therefore increases force production.

**Methods:** Pregnant mice will be exposed in a hypoxic chamber from gestational day 11 to 17.5, during the pseudoglandular-canalicular stages of fetal lung development, coinciding with the period of peak airway development. A second group of pregnant mice will be housed under normoxic conditions throughout pregnancy (Control group). Weights of offspring (males and females) will be recorded until 8 weeks of age at which point they will be tracheostomized. The trachea will then be studied in an organ bath to measure force (g), and subsequently fixed and wax embedded for morphometry. The thickness of the ASM layer will be measured on haematoxylin and eosin stained sections.

**Significance:** Findings will increase our understanding of the underlying mechanism leading to the increased susceptibility of IUGR-affected individuals to future asthma development.