Maternal Diabetes and Smoking: The Impact on Asthma Development

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**Objective:**
Explore phenotypic changes in airway smooth muscle (ASM) in response to maternal diabetes and environmental tobacco smoke.

**Hypothesis:**
Maternal diabetes and environmental tobacco smoke are two major risk factors for asthma.

**Background:**
Asthma is a chronic obstructive lung disease with both environmental and genetic components. Maternal diabetes and environmental tobacco smoke are two major risk factors.

**Objectives:**
1. Explore phenotypic changes in ASM in response to maternal diabetes and environmental tobacco smoke.
2. Investigate the role of maternal diabetes and environmental tobacco smoke in the development of asthma.

**Airway Remodeling-Asthma**
- Increased ASM mass
- Hypocontractility and hyperresponsiveness
- Inflammation
- ASM phenotypes
- Synthetic phenotype
- Proliferative
- Secretory
- Inflammation
- Constrictive phenotype
- Less proliferation
- Less secretion
- More concerned with contracting

**Which phenotype will co-exposure to hyperglycemia and nicotine favor?**

**Results**

- Figure 2: The effect of hyperglycemia and nicotine exposure on ASM proliferative phenotype marker COL1A1. Results normalized to LG condition. Data presented on log2 scale. n=3, different experiments.
- Figure 3: The effect of hyperglycemia and nicotine exposure on ASM contractile phenotype marker SM22. Results normalized to LG condition. Data presented on log2 scale. n=3, different experiments.

**Through an understanding of how early life environment impacts ASM function, we may be able to develop therapeutic strategies that limit ASM dysfunction in asthma.**

**Figure 4:** Hyperglycemia and nicotine exposure impact ASM proliferation rate. Results analyzed using 2-way ANOVA with Fisher's LSD post-test. Significant "main effect" of hyperglycemia and nicotine seen in asthma.