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Editorial

Prenatal Cigarette Smoke Exposure and Development of Asthma in Progeny

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Tobacco cigarette smoking is the primary preventable cause of morbidity and mortality, causing the premature death of over 7 million individuals every year. The World Health Organization estimates that the adult tobacco smoking rate is 14.8% in Australia, 21.9% in the US, 20.8% in the UK, 29.4% in Europe and 25.2% in China in 2016 [1]. Cigarette smoke (CS) contains over 4000 chemicals, 93 of which have been regarded as harmful or potentially harmful. The components of cigarette smoke are absorbed through the lungs into the bloodstream and thus have the potential to cause diseases in almost all human organ systems and decreases overall health status [2]. Maternal smoking during pregnancy causes a range of negative pregnancy outcomes, including: miscarriage, low birth weight, preterm birth, and perinatal death. Furthermore, there have also been links between maternal smoking and adverse neurobehavioral, cardiovascular, respiratory, endocrine and metabolic outcomes in the offspring, which can persist into adulthood [2]. Since rapid organ growth occurs *in utero*, the vulnerability to certain toxins is increased and the exposure to these toxins can potentially interrupt organ development. The inflammatory response and oxidative stress induced by inhaled CS may play a key yet common role in all organ disorders by both direct and *in utero* exposure.

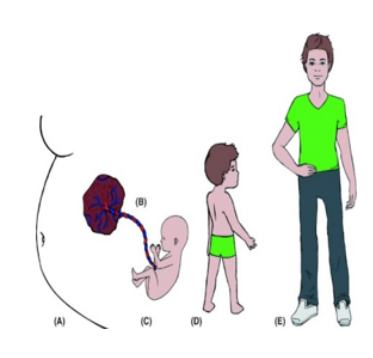

Figure 1: *Fetal growth and maternal factors (A), placenta (B), umbilical cord and fetal factors (C) in relation to development of asthma, and allergic disease in childhood (D) and adolescence (E).*

Gestational exposure to a wide range of environmental pollutants such as CS and polycyclic aromatic hydrocarbons affect the maturation and function of the respiratory system and contribute to the development of pulmonary disease in children [3,4], including higher

risk for allergic asthma, airway remodeling, and impaired lung function, and the effects are similar if either parent smoked [5]. Asthma is a highly prevalent respiratory disease affecting ~300 million patients worldwide and 24 million in the United States alone including 10 million children. Recent studies suggest that lung functions of children are more likely to be compromised if the fetuses were exposed to tobacco smoke during gestation, particularly during the third trimester. Moreover, some of changes in the lung function induced by gestational CS exposure may be permanent. Prenatal exposure to environmental tobacco smoke (ETS) affects lung responses in mice characterized by exacerbated allergic asthma, impaired alveolar development, suppressed angiogenesis and proangiogenic factors and increased expression of antiangiogenic factor [6]. It is recognized that asthma arises in the context of a complex interaction between genetic factors and the evolving immune system of the infant and the environment to which it is exposed, which now includes its' *in utero* exposure [7]. ETS is a mixture of the smoke given off by the burning end of tobacco products (side-stream smoke) and the smoke exhaled by smokers. *In utero* ETS exposure may affect normal fetal lung development decreasing pulmonary function and promoting childhood asthma [7,8]. Although asthma is multi-factorial in origin, it is generally associated with inappropriate immunological responses to common environmental allergens. In addition to maternal smoking even exposure to ETS during pregnancy are risk factors for childhood asthma, and the decrease in lung function of the offspring may be permanent. The cellular/molecular mechanisms by which *in utero* CS exposure promotes childhood asthma are currently unknown. We and other investigators have shown that prenatal ETS exposure exacerbates the allergen-induced Th2 polarization and other parameters of allergic asthma.

A number of environmental factors related to asthma development have been reported to be associated with differential DNA methylation. Several studies suggest a possible epigenetic mechanism underlying the life-long effect of prenatal environmental exposure including maternal tobacco smoking on altered immune regulation and development of asthma. We postulate that prenatal exposure of parental tobacco smoking might program epigenetic modifications of immune or/and genotoxicant detoxification genes, which could be programmed *in utero,* retained into childhood, and contribute to the development of childhood asthma [9]. It is hoped that clarifying the prenatal epigenetic program of immune and detoxification genes for asthma development may provide potentially reversal strategies of DNA methylation for the early prediction and prevention of prenatal CS exposure associated asthma.

Key Messages

- Tobacco cigarette smoking is the primary preventable cause of morbidity and mortality, causing the premature death of over 7 million individuals every year.
- Maternal smoking during pregnancy causes a range of negative pregnancy outcomes.
- Gestational exposure to CS contributes to the development of pulmonary disease in children.
- Prenatal and early life environmental exposure is important contributor in the development of childhood asthma and allergic disease.
- Prenatal CS exposure might program epigenetic modifications of immune or/and detoxification genes, which could be programmed *in utero,* retained into childhood, and contribute to the development of childhood asthma.

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