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REVIEW ARTICLE

Before and after Pregnancy with Asthma

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ABSTRACT
Asthma is a chronic respiratory problem and very common chronic disease that occurs in all age groups. Epigenetics was first recognized in cell differentiation but has been shown to play an important role in the regulation of a wide variety of genes, which includes the genes involved in the inflammatory immune response. Asthma in pregnant women can be appearing more than non-pregnant women and that maybe penetrate in embryonic respiratory system from mother. The critical issue is whether the potential adverse effects of a given asthma medication on embryo or fetal development outweigh the substantial potential benefit of improving the mother's asthma and There is a long-recognized, stronger association of maternal than paternal asthma with the disease in their offspring. In allergic reactions, tonsil can be influenced and it could be hyperplasia and show tonsillitis.
Key Words: *Asthma, Pregnancy, Neonatal, Tonsil*

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INTRODUCTION

Asthma is a chronic respiratory problem characterized by recurring attacks of impaired breathing, of varying intensities. Asthma is a very common chronic disease that occurs in all age groups. The definition of asthma has four cardinal components are bronchoconstriction, symptoms, airway inflammation, and airway hyper-responsiveness. Its high prevalence has significant health costs, which are even higher in the most severe disease forms. Given the high asthma prevalence and lung cancer incidence and mortality in developed countries, this association would have important public health implications (1-3). Indeed the mainstays of treatment, in the form of inhaled corticosteroids, b2 adrenoceptor agonists and cholinergic antagonists, were first used clinically but none of drugs prevent asthma. Since even patients with mild asthma have evidence for inflammation of the large and small airways, and the severity of the inflammation often correlates with the severity of the disease. Allergic asthma is a main public health problem that involved specially children and reduces life quality. Therefore prevention, recognition and treatment of asthma are necessary for all populations (4-6).

EPIGENETICS OF ASTHMA

Asthma is common inflammation and allergic reaction that appear in respiratory tract. Epigenetics was first recognized in cell differentiation but has been shown to play an important role in the regulation of a wide variety of genes, which includes the genes involved in the inflammatory immune response. Epigenetic modifications can alter the structure of DNA itself, such as DNA methylation, or alter the structure of chromatin through alterations to scaffolding proteins, such as histones. Once established, these changes in DNA methylation and histone modifications can be maintained through many cell divisions, leading to a state whereby specific gene expression patterns are determined by the epigenetic profile (7-10). Chronic inflammatory diseases of the lung, including asthma, are the second largest cause of mortality worldwide after cardiac conditions. Asthma and allergy are complex diseases with many influencing risk factors. However, as with many diseases asthma shows complex and non-Mendelian patterns of inheritance and many gene loci are thought to be involved. Candidate genome wide association studies (GWA), looking for single nucleotide polymorphisms (SNPs), have provided valuable insight into the genetic architecture of asthma by providing evidence for the involvement of novel genes in asthma and emphasized the importance of distinct asthma subjects associated with specific genotypes. In addition to Mendelian inheritance, heritable changes to gene expression can be caused by epigenetic changes (11-

15). These epigenetic modifications may help to explain the patterns of inheritance seen in asthma and explain how they interact with environmental factors. Epigenetic mechanisms help to explain many of the characteristics of asthma development and inheritance including maternal inheritance and how environmental impacts can alter the immune response, leading to inappropriate signaling and allergy. This may be particularly valid when accounting for the effects of environmental stressor such as cigarette smoke and air pollution on enhancing the risk of asthma in children exposed in utero. There are a diverse number of epigenetic mechanisms which are involved in the regulation of gene expression, generally silencing gene expression by preventing interactions of DNA binding proteins with the DNA sequence. Although these do not alter the DNA sequence the changes in DNA structure can be inherited across generations (16-19).

Asthma in Pregnant Women

Previous research has supported an association between poorly controlled asthma and adverse health effects in non-pregnant and pregnant women. Bronchodilator and anti-inflammatory agents are now an integral part of asthma management protocols (20-23). Asthma in pregnant women can be appearing more than non-pregnant women and that maybe penetrate in embryonic respiratory system from mother. That is a risk factor for child that has an asthmatic mother (24-26).

PREVENTION OF ASTHMA IN WOMEN

The effect of reduction in food and house dust mite allergen exposure in infancy in preventing allergy and asthma for infants at high risk based on family predisposition (27, 28). Allergic diseases could be reduced for at least the first 8 years of life and recommended this for consideration in high-risk infants (29, 30). The influence of breast-feeding on development of atopy and asthma differs by sex and by maternal and paternal atopy and is most significant among subjects at lower baseline risk. Intervention measures, initiated shortly before birth and applied only during the first year of life, included avoidance of house dust mites, pets, and environmental tobacco smoke, encouragement of breast-feeding and delayed introduction of solids (31-33). studies showed that the intervention measures were effective in preventing asthma in children who did not develop atopy by age 1 year, but the specific measure could not be identified (34-36).

ASSOCIATED RISK MEDICATION USE DURING PREGNANCY AND ASTHMA

The decision to treat a pregnant asthmatic woman with anti-inflammatory and bronchodilator medications involves a discussion of the benefits of each drug and their potential risks of adverse effects. The benefits of the appropriate use of asthma medications are better control of the underlying disease and fewer complications during pregnancy (37-40). There is a secondary gain for the fetus she is carrying since the best environment for growing a healthy baby is a healthy mother. The critical issue is whether the potential adverse effects of a given asthma medication on embryo or fetal development outweigh the substantial potential benefit of improving the mother's asthma (41-44).

SEX DIFFERENCES OF NEONATAL AND ASTHMA

Chronic maternal asthma is associated with reduced growth of the female fetus and normal growth of the male fetus. The mechanisms that control the differential effects of maternal asthma on the fetus have not been fully elucidated but alterations in placental function may play a role (45-48). There are sex-specific alterations in placental gene expression in the presence of maternal asthma. Given that many of the identified genes in the female placentae were associated with or involved in cellular growth and tissue development, these may contribute to the sexually dimorphic difference in fetal growth in response to maternal asthma (49-53).

FETAL ORIGINS OF ASTHMA

Fetal growth restraint has been associated with a number of respiratory outcomes, including inconsistent associations between low birth weight and asthma. Several studies have reported associations that are consistent with greater fetal dimensions or growth trajectories being associated with lower risk of asthma outcomes in children (54-57). Susceptibility to persistent inflammation, may interact with reduced airway size at birth to result in persistent wheeze and asthma. Preterm birth is associated with chronic lung disease and asthma-like symptoms during childhood. There are also evidence studies that extremely preterm birth is independently associated with asthma symptoms and treatment through to early adulthood. Risk factor for development of asthma and allergy, possibly through differences in early bowel bacterial colonization in infants born vaginally and by caesarean section. Some reports have been

published, some of which were included in a meta-analysis that concluded there was a small increased risk of asthma associated with caesarean section delivery (58-61).
There is a long-recognized, stronger association of maternal than paternal asthma with the disease in their offspring. This could be ascribed to intrauterine influences on developmental biology of the fetus but can also be explained by genetic imprinting or more closely shared environment between children and their mothers during the postnatal period. Given the high heritability of asthma, there is clearly an association between maternal and child asthma through inheritance of shared asthma candidate genes (62-64). Pre-pregnancy obesity has been reported to be associated with asthma in young children. Based on observations that maternal age of menarche was associated with atopic outcomes in their adult offspring. There has been interest in potential hormonal influences on asthma development in the fetus. Maternal pre-pregnancy obesity has been suggested in preliminary observations to be associated with an increased risk of asthma in the offspring and there is interest in pregnancy weight gain as a potential risk factor for asthma. A fascinating finding in recent years regarding materno-fetal origins of asthma has been the link between stress in mothers and asthma in their children. The association was initially observed in the postnatal period. At least the best example of an epigenetic effect on asthma comes from the observation that grand maternal smoking during the pregnancy of the mother increases the risk of asthma in the grandchild. Recent studies have suggested that folic acid supplementation in pregnancy cause small increases in asthma risk for the offspring (65-68).

RISK FACTORS FOR THE CHILD

Important recent reviews of asthma are categorized asthma as a "largely developmental disease". Much of asthma has its origins in early life when immune and respiratory system development is altered via the combination of environmental exposures overlaid on top of genetic predispositions (69, 70). Additionally, now is emphasized the increasingly recognized importance of the innate immune function in the risk for childhood asthma. Suggested risk factors for childhood asthma include those in the purely genetic category such as inherited allelic differences, those that are a result of genetic-environment interactions in early life and those that are largely a result of environmental exposures and conditions (71-73). The environmental risk factors can be further divided into those that are likely to cause the conditions necessary for asthma. Those that appear to trigger asthma by unmasking underlying immune/inflammatory dysfunction (74, 75). Maternal and childhood asthma are significant health concerns. The Th2-skewed environment of the pregnancy can increase the severity and duration of asthmatic symptoms for the pregnant women where treatment options maybe limited given concerns for fetal safety. It is also one that is associated with an elevated risk of several other chronic diseases and conditions interconnected via underlying immune-dysfunction (76-78). Because multiple chronic diseases are associated with asthma, there is greater benefit in prevention of childhood asthma via risk reduction. For existing cases of childhood asthma, treatment options should be evaluated not only in the context of the presenting case of asthma but also with an understanding of the comorbid health risks (79-81). Ironically as drugs used to treat allergies and asthma have increased along with the prevalence of this disease, our presently required safety testing for drugs and chemicals is not designed with the capability of identifying the risk of asthma. This safety testing gap needs to be closed with the application of asthma- and early-life-relevant safety testing (82-86).

Tonsilar hyperplasia and asthma in children

Prevalence and severity of asthma have recently increased. According to "hygiene hypothesis", infections give rise to a Th1predominant immune response which inhibits development of asthma and allergic diseases, known to be associated with Th2 originated cytokines (87, 88). Atopic diseases characterized by dominance of Th2 mechanisms and production of Ig-E with exposure to common environmental allergens are increasing in prevalence in world. Increase associated with improved life standards, hygienic environmental conditions and reduction in early childhood infections and infectious diseases might have a protective role against development of asthma and atopic diseases. The differences in cytokine secretion by Th1and Th2 cells determine which disease process they mediate. Recent studies have shown an inverse association between exposure to intracellular infections including tuberculosis, measles and hepatitis A and atopic disorder (89-92). Since many intracellular infections generate predominantly Th1immune responses and since Th1and Th2 immune mechanisms are to a degree mutually antagonistic, it can be envisaged that exposure to certain infections may repress atopic disorder. tonsilar hyperplasia as the proliferation of lymphoid elements in the tissue and a possible etiological factor as bacterial infections. It is demonstrated that a positive correlation between tonsil size and infectious burden. Tonsil size to recurrent and chronic infections of the tonsils has relation. Incidence of tonsillitis in asthma group was significantly less when compared to control group. A positive correlation existed between tonsil size and

incidence of tonsillitis; thus tonsil sizes were significantly smaller in the asthma group. Recurrent tonsillitis is associated with a declined in the prevalence of asthma by inducing a Th1predominant immune response. The finding that tonsilar hyperplasia as a consequence of recurrent tonsillitis is observed less in the group of asthmatic children proves our thesis. But other studies showed in allergic reactions, tonsil can be influenced and it could be hyperplasia and show tonsillitis (93-96).

CONCLUSION

Asthma is a multi-factorial chronic respiratory disease that occurs in all age groups and reduces quality of life with healthy risk for people. In Incidence and Prevalence of asthma Genetic, Epigenetic, Environment, individual and public factors have effect and play an important role to beginning, continue persistent, cure and recurrent of asthma. Genetic involved inflammatory and allergy immune response genes for example; mediators and Cell factors, cytokines, migration factors and chemokines, activation and inhibition factors, recognition molecules, intermediate molecules, suppressing and regulation activating and genes of Immune system. Parallel of Genetic is Epigenetic that has important effect in gene expression and their effects. Environment is main factor allergic asthma and does this function with two ways. One of this does with influence of Environmental agents on Epigenetic and its mechanism. Second, Environmental agents could trigger of respiratory tract and Immune system so continue allergic responses that lead to asthma. Some of these are public and effect many people, but some of these are individual and have effect on one person. For example physiological and pathological conditions of each person have effect for any treatment of asthma. Therefore, for treatment of asthma and prevention of recurrent, should be attention to all factors and correlation of these that are necessary for any action.

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