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What can we learn about predictors of atopy from birth cohorts and cord blood biomarkers?



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Key Messages

Birth cohorts can simultaneously assess many factors involved in atopy development, and will hopefully provide clinicians with information to prevent asthma and allergies.

- Family history contributes to atopic risk of a child.
- Several potential biomarkers have been evaluated in cord blood; however, there is no conclusive evidence suggesting one single predictive biomarker.
- Maternal exposures during pregnancy can contribute to the child's atopic outcomes.
- Numerous early life environmental exposures have been identified to either promote or prevent allergies and asthma, such as cigarette smoke exposure or the timing of food introduction.
- Birth cohort study designs have proven to be effective in evaluating several early life exposures at different time points prospectively; however, they have difficulty in reproducing data and often report conflicting findings.

INSTRUCTIONS

Credit can now be obtained, free for a limited time, by reading the review article and completing all activity components. Please note the instructions listed below:

- Review the target audience, learning objectives and all disclosures.
- Complete the pre-test.
- Read the article and reflect on all content as to how it may be applicable to your practice.
- Complete the post-test/evaluation and claim credit earned. At this time, physicians will have earned up to 1.0 AMA PRA Category 1 Credit™. Minimum passing score on the post-test is 70%.

Overall Purpose

Participants will be able to demonstrate increased knowledge of the clinical treatment of allergy/asthma/immunology and how new information can be applied to their own practices.

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Learning Objectives

At the conclusion of this activity, participants should be able to:

- Describe the use of birth cohorts to study the developmental origins of asthma and allergic disorders.
- Discuss the various risk factors and exposures that contribute to or limit atopic disease development in children.

Release Date: February 1, 2018 **Expiration Date:** January 31, 2020

Target Audience

Physicians involved in providing patient care in the field of allergy/asthma/immunology

Accreditation

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Designation

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Introduction

Atopic disorders such as allergy and asthma are affecting children at an increasing rate. The reason for this increase and the precise mechanisms behind the development of allergic disorders in children are unclear. Factors such as family history, prenatal exposures, and early childhood exposures are each known to be involved in atopy development in children. The use of large-scale birth cohorts has presented a useful opportunity to study the relations among risk factors, potential biomarkers in cord blood, early life exposures, and their associations with atopy. This review focuses on several different risk factors that contribute to allergic disease development, how birth cohorts evaluate these factors, and what clinicians can learn from these studies to help prevent, decrease, and/or revert the development of atopic disorders.

Importance of Family History

The influence of genetics and family history on allergy and asthma development has been well documented. It is clear maternal and

paternal atopic statuses contribute to overall atopic risk of the child; however, many studies have indicated their influence is not equal.²⁻⁴ Some studies have suggested maternal atopic status has a stronger bearing on childhood atopy; and maternal asthma, allergic rhinitis, and atopic dermatitis have each been positively corelated with offspring disease development of the same type.²⁻⁴ In comparison, only paternal asthma has been associated with asthma development in offspring.3 Differences between maternal and paternal effects can be a result of maternal exposures during pregnancy or the complex maternal-fetal interactions that occur in utero. Recent findings from the Isle of Wight Birth Cohort have suggested child sex-specific patterns, with paternal asthma associated with increased asthma in boys but not in girls (prevalence ratio [PR] 1.99, 95% confidence interval [CI] 1.42–2.79, P<.001) and maternal asthma associated with asthma in girls but not in boys (prevalence ratio 1.91, 95% CI 1.34–2.72, P = .003). This sex-specific inheritance pattern observation could be due to epigenetic imprinting, in which parental alleles are preferentially expressed in somatic cells of the offspring or to transgenerational genetic effects.⁴ Furthermore, inheritance patterns of allergy have been established in that at-risk

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