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# Management and Understanding of Asthma during Adulthood

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**Abstract**

Basic research has made significant progress in understanding the link between the innate and acquired type II immune responses in asthma and the role of the airway epithelium. New information is emerging regarding the etiology and heterogeneity of severe asthma. In the field of pediatric asthma, important translational clinical trials are underway to treat allergies to improve asthma outcomes and to improve drug delivery to optimize asthma management. Additionally, there is growing data on the use of biologic agents to treat severe asthma. This paper describes the most notable advances in understanding and managing asthma.

**Keywords:** Asthma; Childhood asthma; Understanding and treatment of asthma

## Introduction

In recent years, great advances have been made in understanding and treating asthma. Improved understanding of disease mechanisms through basic scientific research over the past two decades has led to the development of highly specific therapeutics that better target the dysregulated immune processes behind asthma, leading to the development of new and effective therapies [1]. Together, translational clinical research is enabling researchers to address the diverse biology of asthma and target mechanisms beyond the allergic response and type II immune response to meet the needs of more asthma patients. These changes ushered in an exciting era in asthma research, with a significant increase in new interventions entering clinical trials.

Viral respiratory infections are the most common triggers of acute asthma in children and adults. Although asthma patients are not susceptible to viral infections, they have more severe symptoms and these infections cause periods of exacerbation and poor control of asthma [2]. Given this observed susceptibility to the effects of viral infections. It has been suggested that asthma patients may have defective antiviral immunity due to genetic or acquired factors. fulfill. Airway epithelia release IFN in response to viruses as part of the initial innate immune response to infection. They induce IFN-stimulated genes, leading to the production of antiviral proteins.). Her impaired IFN response in asthma remains a controversial topic in the literature [3]. We originally showed that rhinovirus 16 (RV-16) replication was increased in asthmatic bronchial epithelial cells (BECs) compared to healthy controls. This was due to impairment of IFN-β, and addition of exogenous IFN-β to the cultures induced apoptosis and decreased viral replication [4]. This indicated that the expression of IFN-β-stimulated antiviral genes was comparable in primary BECs from asthmatic and non-asthmatic patients. They suggested that IFN-β might reduce his expression of inflammatory cytokines such as CXCL-10, RANTES and interleukin (IL)-6, suggesting that IFN-β may contribute to the inflammatory response. It suggests that it may shorten the duration [5].

This was proposed by Contoli et al. expanding on type III IFN-λ; he found that asthmatic BECs were defective in his IFN-λ induction. This correlated with RV-induced asthma exacerbations and viral load in experimentally infected human volunteers [6]. This lack of IFN-λ in his primary BEC was reviewed by Parsons et al. They showed that this was not due to underexpression of MDA5 and Toll-like receptor 3 (TLR3), but due to the subsequent failure to activate his type I and III IFN immune responses to RV infection [7]. These defective innate responses were identified by Baraldo et al. It was also demonstrated in asthmatic children and in atopic patients without asthma, regardless of atopic status. However, other studies did not find changes in IFN-β10 or λ11 in asthmatic epithelia.

Kichiku and others investigated this process further [8]. We recently found that BECs from children with asthma showed decreased IFN-β release and increased proinflammatory cytokine production compared with healthy control cells. Exogenous IFN-β restored apoptosis, suppressed viral replication and improved wound healing, but did not suppress increased proinflammatory cytokine production.

Inadequate IFN responses and susceptibility to viral infection may be present only in some asthmatic patients, reflecting airway pathology or disease severity [9]. In 86 adults with inadequately controlled asthma despite treatment with moderate to high dose inhaled corticosteroids (ICS), peripheral blood monocytes (PBCs) for exposure to a single her RV strain were evaluated. PBMC) responses were examined [10]. They found that those who showed impaired PBMC release of IFN-α to RV-1B had elevated airway neutrophils and received the highest doses of ICS [11, 12]. Responses to infection may vary according to the inflammatory features of asthma, suggesting that her treatment with high doses of ICS may increase the susceptibility of asthma to viral infections. This is an area of great interest and may have significant implications for individually tailored IFN-enhancing therapies.

## The Role of the Airway Epithelium in Asthma

BECs are recognized as important contributors to the innate immune response, but are thought to have a limited contribution to the adaptive immune response. Normally, TH1-derived her IFN-γ inhibits TH2 cell differentiation, whereas TH2-derived IL-4 promotes TH2 differentiation. It is not known whether airway epithelial cells have the same her TH1/TH2 program as lymphocytes. BEC recognize IL-4 and express a receptor for its structural homolog, IL-13 [13]. Airway epithelial cells also express her IFN-γ receptors. Several studies have shown antagonistic patterns of regulation of IL-4 and IFN-γ,

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thereby translating TH1/TH2 antagonism directly into epithelial gene regulation. IL-4 and IFN-γ induced distinct transcription factor hubs or clusters that exist in antagonistic and polarized gene regulatory networks [14]. Moreover, the IL-4-dependent induction of IL-24 observed in rhinitis patients was downregulated by IFN-γ. Therefore, IL-24 may be a unique biomarker for allergic inflammation and TH2-polarized epithelia.

Clearly, preventing asthma or altering the risk of developing asthma in utero would have enormous benefits for society and for those who suffer from asthma throughout their lives [14]. Vitamin D has long been associated with asthma pathogenesis and immunity and two important studies were conducted to determine whether vitamin D3 supplementation affects the development of asthma in childhood. The first study, conducted in Europe, recruited 623 females aged 24 weeks and followed 581 infants aged 3 to her 28 years. Women were randomized to receive either 400 IU or 2400 IU of vitamin D as a regular supplement. The primary aim was that the supplement reduced persistent wheezing by age 3 years [15]. In the intervention group, 20% of the control group had wheezing lasting 16 minutes, but this was not a significant difference. No, but other secondary outcomes such as troublesome episodes of lung problems decreased. A second study, reported in the same journal, was a larger, three-center US study that followed 806 children to age 3 years but recruited women whose children were at high risk for asthma. . ) vitamin D and control groups received 400 IU [16]. By selecting the high-risk group, more children (218/806) developed persistent wheezing, with an absolute reduction in persistent wheezing of 6%. , but none were significant. Interestingly, most effects may have occurred in the first year or two and then faded. In both studies, people who took additional supplements were more likely to get enough vitamin D. The results observed in both studies were similar, and although the studies may have been too weak to detect such complex findings, the results suggest that vitamin D supplementation alters asthma risk. The case has not been proven.

It has long been known that the risk of developing asthma during childhood is complex, and that this risk varies by gender [17]. The importance of gender was further emphasized31. It was found that 12.7% experienced initial transient wheezing and 13.1% experienced persistent wheezing. Compared with no/rare wheezing, maternal asthma, infant bronchiolitis, and atopic dermatitis were associated with persistent wheezing in both boys and girls, whereas paternal asthma was associated only with boys. Being black or Hispanic was a predictor only for girls, which was associated with persistent wheezing (odds ratio [OR] 4.27; 95% confidence interval [KI ] 2.33-7.83). The strong association between paternal asthma and boys' risk is indeed underscored, confirming the role of other environmental and social factors previously mentioned [18]. These studies indicate that paternal genetic factors appear to play an important role in the development of persistent asthma in boys and highlight which factors need further investigation.

## Sublingual Dust Mite Immunotherapy and Asthma

The close relationship between childhood asthma and dust mite allergy has led to a number of interventions to reduce the effects of asthma exposure [19]. The advent of sublingual immunotherapy has reversed academic interest in desensitization, it opened up as a treatment to a wider population with less disruption and potentially lower risk compared to subcutaneous desensitization protocols was administered to 834 her HDM sensitizers with asthma inadequately controlled with ICS alone [20]. Subjects were randomized to receive either placebo or her two doses of sublingual HDM. The primary endpoint was reduction in time to first moderate-to-severe exacerbation. Both doses reduced the risk of asthma exacerbation (hazard ratio 0.72; 95% CI 0.52-0.99), but did not change asthma control scores or quality of life [21]. Although the effects observed were modest, they indicate that chronic allergen exposure plays a direct role in exacerbation risk and, when reduced, modifies asthma.

## Conclusion

Important studies are currently underway that reveals important links between the innate immune response in asthma and the traditional acquired type II immune response. We expect this to be one of the next areas where the development of targeted therapies will lead to important new therapies. Because this domain is important for the pathogenesis of pediatric asthma and the interaction between viral infection and allergic sensitization, the development of therapeutics specifically targeting acute asthma and potentially affecting disease progression in childhood is expected.

Clinical trials of biologics may progress, but at this stage they are still most effective in patients with severe disease, allergic disease or a dysregulated type II immune response. Its use in early disease in the form may alter the course of asthma.

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