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Series Introduction: Inspirations on asthma

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Perspective

Asthma continues to challenge physicians and scientists. After declining for years, the prevalence, morbidity and mortality of asthma have increased over the last two decades. In keeping with the importance of this “epidemic,” significant attention has been focused on the pathogenesis of this disorder. These investigations have revised our concepts of the abnormalities that underlie the asthmatic diathesis and our understanding of crucial aspects of its natural history. They have also provided exciting insights into the cellular and molecular events that regulate airway inflammation and inflammation in other tissues. It is clear from these studies that a genetic predisposition plus an appropriate life event(s) conspire to generate the asthmatic diathesis and that often early life (and possibly in utero) events play a crucial role in disease pathogenesis. It is also clear that inflammation is the cornerstone of asthma and that, in the majority of cases, Th2 dominated tissue responses are responsible for this disorder. Recently, the non-inflammatory structural alterations in the asthmatic airway have received renewed attention and the roles these structural changes may play in generating symptomatology and altering natural history have begun to be elucidated. The first Perspective in this series, by Gern, Lemanske and Busse, deals with the early life origins of the asthmatic diathesis, and the second article, by Oettgen and Geha, highlights recent insights into […]

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SERIES INTRODUCTION

Inspirations on asthma

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Perspective

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Asthma continues to challenge physicians and scientists. After declining for years, the prevalence, morbidity and mortality of asthma have increased over the last two decades. In keeping with the importance of this "epidemic," significant attention has been focused on the pathogenesis of this disorder. These investigations have revised our concepts of the abnormalities that underlie the asthmatic diathesis and our understanding of crucial aspects of its natural history. They have also provided exciting insights into the cellular and molecular events that regulate airway inflammation and inflammation in other tissues. It is clear from these studies that a genetic predisposition plus an appropriate life event(s) conspire to generate the asthmatic diathesis and that often early life (and possibly in utero) events play a crucial role in disease pathogenesis. It is also clear that inflammation is the cornerstone of asthma and that, in the majority of cases, Th2 dominated tissue responses are responsible for this disorder. Recently, the non-inflammatory structural alterations in the asthmatic airway have received renewed attention and the roles these structural changes may play in generating symptomatology and altering natural history have begun to be elucidated.

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The first Perspective in this series, by Gern, Lemanske and Busse, deals with the early life origins of the asthmatic diathesis, and the second article, by Oettgen and Geha, highlights recent insights into the importance of IgE and new anti-IgE-based therapies in atopy and the asthmatic response. The third article by Ray and Cohn highlights the large body of data that has now been generated regarding the cellular and molecular events that regulate and the effector functions of Th2 inflammatory responses and the fourth article by Lukacs and colleagues describes the organized chemokine network that is involved in the generation of airway inflammation. The last article in the series by Elias et al. deals with the structural changes and the concept of asthmatic airway remodeling, highlighting both our level of knowledge and the challenges for the future.

When the information presented in this series is looked at in composite, one can easily be impressed by the depth and breadth of our knowledge. It is also easy to see how these studies will serve as a springboard for future investigations and the foundation upon which new and powerful therapeutic interventions for asthma can be developed. It is important to keep in mind, however, that the relationship between atopy and asthma and the cause of the asthmatic epidemic are still poorly understood despite significant investigation. Perspective is also provided by the fact that William Osler stated in 1892 that asthma "in many cases is a special form of inflammation of the smaller bronchioles." It has taken us almost a century to fully embrace this concept and begin to carefully dissect this airway response. Although we have made impressive progress, we still have a long way to go.