Childhood asthma: pathogenesis and phenotypes

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Complex interactions between viral infections, atopy, the microbiome, preterm birth, infant weight gain, environmental exposures and genetic susceptibility influence the development of wheezing illness and asthma in children https://bit.ly/3vK4HCF

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Abstract

In the pathogenesis of asthma in children there is a pivotal role for a type 2 inflammatory response to early life exposures or events. Interactions between infections, atopy, genetic susceptibility and environmental exposures (such as farmyard environment, air pollution and tobacco smoke exposure) influence the development of wheezing illness and the risk of progression to asthma. The immune system, lung function and the microbiome in gut and airways develop in parallel, and dysbiosis of the microbiome may be a critical factor in asthma development. Increased infant weight gain and preterm birth are other risk factors for development of asthma and reduced lung function. The complex interplay between these factors explains the heterogeneity of asthma in children. Subgroups of patients can be identified as phenotypes, based on clinical parameters, or endotypes, based on a specific pathophysiological mechanism. Paediatric asthma phenotypes and endotypes may ultimately help to improve diagnosis of asthma, prediction of asthma development and treatment of individual children, based on clinical, temporal, developmental or inflammatory characteristics. Unbiased, data-driven clustering, using a multidimensional or systems biology approach may be needed to better define phenotypes. The present knowledge on inflammatory phenotypes of childhood asthma has now been successfully applied in the treatment with biologicals of children with severe therapy-resistant asthma, and it is to be expected that more personalised treatment options may become available.