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Perspective

Epigenetics and Genetics of Allergic Diseases

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DESCRIPTION

Allergic diseases such as allergic rhinitis, asthma, and food allergies are a group of immune-mediated diseases that affect children and adults. There is strong evidence for the concept that allergic diseases are influenced by a combination of gene-environment interactions known to be mediated through epigenetic mechanisms. Environmental factors such as allergens, air pollution, and microbes can undoubtedly contribute to the etiology of allergic diseases. All of these factors directly or indirectly regulate DNA methylation, histone modifications, and non-coding microRNAs. Assessing genetic predisposition and epigenetic risk in allergic diseases is not yet fully elucidated.

Allergic diseases are on the rise in the western world, and known allergic protective and driving factors such as microbial and dietary exposure, pollution and smoking mediate their effects through changes in the epigenetic landscape. The involvement of epigenetic modifications in allergic disease critically evaluates findings from epigenome. Advances in new technologies such as epigenome editing and DNAzymes enable targeted modification of the epigenome, providing new therapeutic tools in the future. Type II Helper T cells (Th2) cytokines may also be synthesized by innate lymphocyte type 2.

Allergic diseases have a wide range of clinical manifestations and conditions. A major group of them are mediated by immunoglobulin E and are commonly referred to as atopic diseases. These include allergic bronchial asthma (predominant manifestation in the lungs and lower respiratory tract), allergic rhinitis (hay fever; upper respiratory tract), allergic conjunctivitis (ocular), (extrinsic) atopic dermatitis (eczema) and food allergy (upper and lower gastrointestinal tract). All patients exhibit an individual spectrum of these diseases throughout their lives, but certain patterns are readily recognizable. It describes a prototypical sequence of symptoms beginning with atopic eczema or food allergy and then transitioning to respiratory disease, during which one surface barrier dysfunction leads to a dysfunctional epithelial barrier of the other. Several diseases can coexist, such as eczema with food allergies and eczema with bronchial asthma. Since this co-occurrence may already occur before the first actual food intake, the early postnatal stage appears to be critical for the development of IgE-mediated allergy. Although IgE-mediated food allergies appears to be increasing rapidly in Western countries, currently affecting about 3-5% of children and about 1-3% of adults, and are geographically distributed. Typical childhood food allergies, such as Cow's Milk allergy (CMA), also persist into old age. There is a strong sex difference in favor of males in the early onset of asthma and other allergic diseases (including food allergies) that reverses after puberty and throughout adulthood, with higher prevalence in females.

Asthma and allergy genetics were dominated by Genome-Wide Association Studies (GWAS) for more than a decade, starting with the first GWAS on asthma exploring genetic susceptibility for elevated total IgE, allergic sensitization, atopic dermatitis, and allergic rhinitis as well as food allergy. Common genetic traits for common diseases have been largely identified. However, the lack of heritability in asthma and allergy remains high, and techniques currently in use have reached resolution limits, so increasing patient numbers in GWAS studies will not advance knowledge of genetic susceptibility.

On the other hand, the analysis of epigenetic modifications in allergic diseases has recently received considerable interest. Because epigenetic modifications may mediate environmental influences on the development or protection of allergic diseases, represent a new class of biomarkers, and provide new therapeutic targets. Epigenetics, including DNA methylation, post-translational histone modifications, nucleosome occupancy, and small and long noncoding RNAs, may hold the key to explaining the high degree of plasticity of immune responses over a lifetime.

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