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Gene Technology

Opinion Article

Gene Expression in New-Borns Reveals Potential Causes of Autoimmune and Allergy Disease Risk

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DESCRIPTION

Infancy is a pivotal stage when physiological and developmental changes influence the etiology of later-life disorders. Genetic predisposition and early life exposures to allergens or bacteria play a role in the development of many complex diseases, including immunological and respiratory disorders. Nonetheless, little is understood about the early-life genetic regulation of gene expression and its relevance to a person's propensity for diseases as they get older, despite mounting evidence of its significance.

Studies on expression Quantitative Trait Loci (eQTLs) have shed light on how genetic polymorphisms affect gene regulation and how they relate to complex diseases. The majority of eQTLs have been identified in adult tissues, whereas eQTLs in perinatal tissues have just recently been investigated: eQTLs revealed in foetal placentas and foetal brains, for example, are enriched for genetic variations related with growth and neuropsychiatric symptoms, respectively. Disease development is regulated by both individual and cell-type-specific responses to environmental stimuli in addition to genetic diversity. Knowing how these stimuli interact with eQTLs can provide information on the conditions such as cell type, microorganism, or temperature under which genetic variations may affect disease. Unfortunately, most reQTL research up to this point has used adult samples, and our understanding of how neonatal immune gene expression is controlled in response to stimuli is still restricted.

Using purified cord blood samples from 152 newborns, we characterise the genetics of gene expression in the innate and adaptive arms of the neonatal immune system. To uncover *cis* regulatory mechanisms in these samples, instances of suspected Tran's gene regulation are examined using mediation analysis. We provide evidence that prevalent autoimmune and allergy illnesses have a genetic base that is shared by neonatal eQTLs and reQTLs, and many of this colocalization is cell type- or stimulation-specific. Lastly, we investigate the causal relationships between neonatal gene expression and risk of immune-mediated illnesses using Mendelian randomization. In conclusion, we stress the prenatal period's potential significance for understanding the genesis of immune-mediated illness.

We conducted a two-sample Mendelian Randomization (MR) study to find possible causal effects of neonatal gene expression on risk of autoimmune and allergy disease using *cis*-eQTLs as genetic tools, neonatal *cis*-eGene as exposure, and disease as outcome (the "Methods"). We evaluated the 52 eGenes for which three or more genetic instruments were accessible in addition to the illnesses described above for which we had GWAS transcript statistics available. We took into account genes whose significant causal effects on a disease without significant pleiotropic effects were detected by at least three of the four MR methods. The genetic control of gene expression in the innate and adaptive immune systems of newborns, as well as its connection to the genetics of autoimmune and allergy illnesses. Our findings revealed a significant genetic overlap between genetic variants affecting gene expression in neonatal immune cells and those linked to immunological-mediated disorders, shedding light on the potential early-life origins of disease. Last but not least, Mendelian randomization demonstrated that several variations in gene expression at birth may have causal impacts on the chance of developing autoimmune and allergic diseases. Asthma and allergic disease shared causative variations with the reQTL for IL13 in neonatal PHA-stimulated T cells, indicating that this reQTL may influence the risk of allergic disease through pathways that do include T cell activation and Interleukin 13 (IL-13).

As a pan-T cell mitogen, PHA may share intracellular signalling with allergen-mediated T cell activation. In addition to other sources, activated $CD4^+$ and $CD8^+$ T cells42 generate IL-13, which encourages the synthesis of Immunoglobulin E (IgE) by B cells. In mouse models, IL-13 has been demonstrated to elicit asthma symptoms such as airway hyper responsiveness, increased total serum IgE, and increased mucus production. In both mild and severe asthma, increased IL-13 expression is seen in sputum and bronchial biopsies, and it can be used as a biomarker for severe refractory asthma. Anti-IL-13 antibodies, such as tralokinumab

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and lebrikizumab, have been developed as IL-13-targeting therapies, although phase 3 clinical trials have shown inconsistent or moderate results in their ability to treat severe asthma exacerbations. Our findings support the use of IL-13 as a therapeutic target for asthma and point to the possibility of increased efficacy with treatment directed at particular genetic subgroups. The astonishing intricacy of the genetic control of gene expression in the innate and adaptive immune systems at birth, as well as its possible significance in the development of autoimmunity and allergy illness, are demonstrated by our work.