Perspective
Volume 8:5, 2022

Journal of Lung Diseases & Treatment

ISSN: 2472-1018 Open Access

The Airway Epithelium's Genetic Components and the Pathology of Asthma

Christiane Omer*

Department of Medicine, McMaster University, Hamilton, ON L8N 3Z5, Canada

Introduction

The symptoms of asthma include airway hyperresponsiveness, reversible airflow restriction, and mucus hypersecretion. Asthma is a heterogeneous inflammatory disease of the airways. Numerous comorbidities, such as rhinosinusitis, gastro-oesophageal reflux disease (GERD), obstructive sleep apnea (OSA), psychosocial dysfunction, and persistent infections, may have a negative impact on poorly controlled asthma. Around 262 million people worldwide were affected by asthma in 2019, and the prevalence has been rising. Due to their high exposure to allergens, such as tobacco smoke and air pollution, and the fact that their immune systems are not yet fully developed, children and adolescents have a much greater incidence of asthma. Asthma is a complicated illness with a number of risk factors contributing to its genesis, as has long been known. It was The existence of a genetic component in asthma was first established by a twin research.

In other words, genetic predispositions have a role in the development of asthma, and adult populations have heritability rates of up to 60%. The initial line of defence against pathogens and environmental irritants are the airway epithelial cells. Alarmin cytokines such TSLP, IL-33, and IL-25 are released as a result of any injury to the epithelium. These cytokines cause a number of immune cells and inflammatory pathways to become active. Histamine, prostaglandin (PG) D2, and leukotrienes are among the mediators released by activated mast cells and basophils that constrict airway smooth muscle and boost mucus output. SABA, LABA, and anti-leukotriene medications, among others, that target these mediators stop the growth of these pathways [1].

Description

Thymic stromal lymphopoietin (TSLP) is a cytokine that originates from epithelial cells and is produced as a result of exposure to outside stimuli such bacteria, viruses, smoke, and allergens. By attaching to the TSLP receptor on inflammatory cells such mast cells, dendritic cells, and eosinophils, TSLP causes allergic inflammation. In contrast to healthy controls, asthma patients have higher levels of TSLP expression, which is correlated with measurements of airway obstruction. In patients with moderate allergic asthma, an anti-human TSLP monoclonal antibody (AMG157/tezepelumab) that blocks the interaction of TSLP with its receptor has been found to drastically diminish allergen-induced airway responses and airway inflammation. Following clinical trials demonstrating a decrease in asthma exacerbations, improvement in lung function and asthma control, and reduction in asthma symptoms, tezepelumab has been licenced for treatment in severe asthma. between airway fractionated nitric oxide (FeNO) levels and blood eosinophils. The TSLP gene variations have been linked to increased risk and severity of asthma, according to genetic studies; however, the results are not always consistent [2].

Two single nucleotide polymorphisms (rs3806933 and rs2289276) found in the TSLP gene promoter region were positively associated with asthma susceptibility in children and adult asthmatics. This positive or direct association can be explained by a higher risk of asthma in people who have these SNPs. Birben et al. elaborated on the findings of this study and shown that the connection with these SNPs is sex-specific with the CC genotype of SNP. While rs2289276's CC genotype corresponded with greater eosinophil counts in female patients, rs3806933 was positively connected with asthma in the male population. Even though these two studies found a link between the TSLP polymorphisms and asthma, a study by Hunninghake et al. and colleagues found that two SNPs in the TSLP genomic region were negatively correlated with asthma, making people with those SNPs less likely to develop asthma. Moorehead et al. looked at the TSLP gene's SNP and its relationship to eosinophilia, asthma, and allergy illness. The authors hypothesised that the protective benefits they observed in rs1837253 carriers may be connected to alterations occurring in the production of TSLP's long isoform, which ultimately results in less of this protein being produced [3].

There has been a great deal of research done on the gene-environment connections of amino acid variations of Glutathione S-transferase Pi (GSTP1) and air pollution. In comparison to people with any Val-105 allele and those living in low air pollution locations, Ile-105 homozygotes at the Ile-105 locus have a greater chance of developing asthma. While conflicting evidence supports the idea that the val/val genotype acts as a protective genotype, other studies suggest that a val/val genotype at the ile105val locus may be an indication of harm from oxidative stress in asthmatic children. Children who have the minor allele for the GSTP1 SNPs rs1138272 or rs1695 (Ile105Val), as well as exposure to NO2 utilising land use regression and dispersion modelling, are discussed in discussions of gene-environment interactions. compared to major allele carriers, are more susceptible to harm from air pollution, and this susceptibility is highest in children with present asthma, prior asthma, and prior wheeze. Toll-like receptor 4 [TLR4] plays a minor function through its expression on alveolar macrophages, but Toll-like receptor 2 [TLR2] expression on human airway epithelial cells has been demonstrated to be involved in the response to air pollution particle [or particulate matter] exposure [4].

From birth to age 8 in asthmatic children, the rs4696480 and rs1898830 SNPs in the TLR2 gene augment the effect of exposure to particulate matter. In a similar manner, the TLR4 gene's SNPs rs2770150 TC, rs10759931 GG, rs6478317 GG, and rs1927911 TT alter the impact of exposure to When bearing specific genotypes of these two genes, asthmatics are more susceptible to the harmful effects of air pollution on children. Previous research on TGF-1 explains its function in airway remodelling, such as subepithelial fibrosis, and in potential airway inflammation through the release of inflammatory cytokines. Asthma risk is increased by prenatal exposure to traffic and tobacco smoke and the TGF-1 -509TT (rs4803457) genotype [5].

Conclusion

Because of the complicated interplay between hereditary and environmental variables, asthma is a diverse disease. Different effects of

***Address for Correspondence:** Christiane Omer, Department of Medicine, McMaster University, Hamilton, ON L8N 3Z5, Canada; E-mail: Christianeomer25@gmail.com*

Date of Submission: 01 September, 2022, Manuscript No. LDT- LDT-22-81085; **Editor Assigned:** 03 September, 2022, PreQC No. P-81085; **Reviewed:** 17 September, 2022, QC No. Q-81085; **Revised:** 23 September, 2022, Manuscript No. R-81085; **Published:** 01 October 2022, DOI: 10.37421/2472-1018.2022.8.163

single nucleotide polymorphisms on asthma susceptibility and severity can be seen throughout the genome. There are some particular variants that have a protective impact and reduce the risk when present, despite the fact that the majority of SNPs affecting asthma result in increased risk or susceptibility in the carriers. In accordance with the same pattern, asthmatic patients who have particular polymorphisms in their genome respond better to generic asthma medications, indicating that genetic predispositions should be taken into consideration when prescribing various asthma therapies. Our knowledge of the pathology, diagnosis, and treatment of this complex disease is improved by research into the genetics of asthma. finding a new song Individuals' nucleotide differences in their genetic makeup will not only aid in the discovery of new biomarkers and phenotypes but will also provide us insights into how to create effective and personalised treatments.

References

1. Thomsen, S. F., C. S. Ulrik, K. O. Kyvik and V. Backer et al. "Multivariate genetic analysis of atopy phenotypes in a selected sample of twins." *Clin Exp Allergy* 36 (2006): 1382-1390.
2. Dunican, Eleanor M. and John V. Fahy. "The role of type 2 inflammation in the pathogenesis of asthma exacerbations." *Ann Am Thorac Soc* 12 (2015): 144-149.
3. Shen, Ling X., James P. Basilion and Vincent P. Stanton Jr. "Single-nucleotide polymorphisms can cause different structural folds of mRNA." *Proc Natl Acad Sci* 96 (1999): 7871-7876.
4. Tam, Vivian, Nikunj Patel, Michelle Turcotte and David Meyre et al. "Benefits and limitations of genome-wide association studies." *Nat Rev Genet* 20 (2019): 467-484.
5. Demehri, Shadmehr, Mitsuru Morimoto, Michael J. Holtzman and Raphael Kopan. "Skin-derived TSLP triggers progression from epidermal-barrier defects to asthma." *PLoS Biol* 7 (2009): e1000067.

How to cite this article: Omer, Christiane. "The Airway Epithelium's Genetic Components and the Pathology of Asthma." J Lung Dis Treat 8 (2022): 163.