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Original Article

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The Association of GSDMB rs7216389 Polymorphism in Childhood Asthma

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ABSTRACT

BACKGROUND AND OBJECTIVE:
Asthma is a chronic airway disease with increasing cases in children, differing from adult-onset asthma in terms of its triggers and outcomes. This study focuses on the rs7216389 variant of the GSDMB gene, exploring its role in airway inflammation and its clinical and genetic links to pediatric asthma, with implications for improved diagnosis and targeted treatment.

METHODOLOGY:
A case-control study spanning 18 months was carried out at CMH Lahore and Children's Hospital,comprising 100 participants (50 with asthma and 50 healthy controls) aged 3–18 years. Genetic An evaluation was conducted on blood specimens, and data analysis was executed using SPSS (v25.0). A p-value of less than 0.05 was considered significant. Ethical clearance was secured, and Informed consent was acquired.

RESULTS:
Research has found that the GASDERMIN B variant rs7216389 functions as a possible genetic risk factor for asthma because it links to severe asthma in children. The T allele of rs7216389 was significantly more frequent in asthmatic children (60%) than in controls (40%). This research acknowledges industry challenges related to translating genetic discoveries into clinical practice, alongside descriptions of the therapeutic potential of genetic marker-based therapeutic approaches.The research provides comprehensive information on healthcare expenses and is distinctive in its scope.Symptoms encountered in pediatric asthma patients can help better understand the full impact of the disease.

CONCLUSION:
The rs7216389 polymorphism in the GSDMB gene shows a significant association with childhood asthma, underscoring its potential as a genetic marker for early risk identification and the development of personalized, targeted management strategies.

KEYWORDS: Asthma, Genetic Susceptibility, Case-Control Studies, Precision Medicine, Single Nucleotide Polymorphism (SNP), Genetic Association Studies.

INTRODUCTION

Childhood asthma is a common cause of concern worldwide [1]. World Health Organization(WHO) data have shown consistently increasing global incidence of asthma[2]. Clinically, it presents as dyspnea, coughing, wheezing, and a reduction in the FEV1/FVC ratio, typically worsening during these episodes [3]. Pneumonia-related illnesses and chronic obstructive pulmonary disease are expected to result in 4.5 million annual deaths throughout the world by 2030, and 90% of these deaths will occur in nations with limited and average incomes [4]. Pakistan has an asthma prevalence rate of 2.1% in adult citizens, while the incidence of asthma in children under 18 is 4.3% [5]. Despite extensive research, asthma has remained a challenge to manage and treat due to inadequate knowledge of its molecular and genetic basis. Key features of asthma include wheezing, airway constriction, hyperresponsiveness, and elevated IgE levels in response to airborne allergens [6]. The progression of asthma varies significantly based on age and gender. In children, it is usually atopic and marked by increased bronchial hyperresponsiveness, a lower FEV1/FVC ratio, elevated Ig E levels, and allergen exposure [6]. Adult asthma is relatively refractory and resistant to therapy [5]. Primary healthcare

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facilities report that one-fourth of their patients have asthma or COPD, according to data [7].

Research shows that T-cells, together with airway epithelium cells, control the development of childhood-onset asthma [8]. The 17q12-q21 chromosomal region, discovered four years ago, remains the main asthma-relevant region, encompassing a 6-Mb region [8]. The study of genetic correlations between 17q12-q21 and asthma primarily investigated European populations, yet researchers have also found substantial genetic links in Asian populations [9].

This study investigates the clinical and genetic determinants of pediatric asthma, emphasizing the role of the rs7216389 polymorphism in the GSDMB gene. It delves into the gene's regulatory functions, the biological implications of rs7216389 in the development of childhood asthma, and the expression patterns of GSDMB across various cell types. By identifying potential research directions and therapeutic strategies, this analysis aims to elucidate the role of GSDMB genetic variation in asthma pathogenesis.

METHODOLOGY

In accordance with clinical practice guidelines, this case-control study was conducted at CMH Lahore Medical and Dental College and Children's Hospital. The sampling technique employed was convenience sampling. Sampling was carried out after obtaining informed consent from the parents, child assent for minors and necessary administrative approvals.

The study spanned 18 months, from March 3, 2023, to May 21, 2024, following IRB approval (Case #.536/ERC/CMH/LMC) dated February 14, 2023, from the Office of Research, Innovation,and Commercialization (ORIC), CMH Lahore Medical College and Institute of Dentistry. The research included participants between 3 and 18 years of age who met the asthma criteria established by the Global Initiative for Asthma (GINA) 2019 guidelines. The research study followed the principles established by the Declaration of Helsinki.

Researchers recruited asthmatic participants based on wheezing, coughing, and dyspnea, along with a family history of asthma or exercise-induced symptoms, and healthcare testing to confirm the absence of lung conditions. Participants in the control group, aged 3 to 18, had no history of asthma or related symptoms such as coughing, wheezing, dyspnea, or allergies. The study excluded individuals with acute respiratory infections or congenital conditions, including chronic lung disease, cystic fibrosis, congenital lobar emphysema, or COPD. Additionally, participants without an asthma diagnosis but displaying asthma-like symptoms were not included. A 3 cc syringe wa used to draw 2 mL blood samples from each participant, which were stored at 4°C in vacutainer tubes containing EDTA anticoagulant. The scientists transferred these biological samples to the Department of Human Genetics and Molecular Biology at the University of Health Sciences, Lahore, for molecular analysis.

PCR and DNA sequencing were performed at the University of Health Sciences. The tetra-primer amplification refractory mutation system-polymerase chain (ARMS-PCR) was employed. A negative master mix and a positive control (master mix + successfully amplified DNA containing the target sequence) were set up for each experiment. PCR products were electrophoresed to verify the specificity. Automated Fluorescent DNA Sequencing was done, and the sequencingthesequencing data was analyzed using Chromas Lite software v2.01.

The researchers executed statistical analysis through SPSS Statistics 25.0 (IBM Corp., 2017). For both quantitative and qualitative variables, we reported means and standard deviations (SD), while percentages described the qualitative factors. Normality of the data was checked by the Shapiro-Wilk test. The research determined that significance was indicated by p-values below 0.05. The analysis produced frequencies for categorical data, while quantitative values received means with standard deviations. A Chi-square test was conducted in this study to assess statistical associations between categorical data.

RESULTS

The study included a total of 200 patients, equally divided into two groups: 100 diagnosed with asthma and 100 without asthma. There was a slight male predominance, with 104 (52.0%) males and 96 (48.0%) females.

Table-I: Distribution of Demographic variables.

Variable	Responses	n (%)
Age (years)	3-6	66(33)
	7-10	49(24.5)
	11-14	42(21.0)
	15-18	43(21.5)
Gender	Female	104(52.0)
	Male	96(48.0)
Socio economic status	Low	69(34.5)
	Middle	60(30.0)
	High	71(35.5)

This table elucidates the prevalence of a family history of asthma in the patient and control groups. A p value of less than 0.05 is considered significant and therefore there is an association between family history with Asthma/Non-Asthma groups.

Table-III shows the number of children experiencing respiratory infections annually in asthmatic patients and non-asthmatic individuals (controls). Among asthmatic patients, 38% of people often get infected (more than three times a year). Out of the non-asthmatic control group, 61% of patients have occasional infections.

The Chi-Square test results (Table-IV) demonstrate a significant association between asthma status and SNP variants. A p-value of <0.001 indicates a substantial difference in the distribution of SNP variants (C/C, T/C, T/T) and exposure to pets between asthmatic and non-asthmatic individuals.While The Chi-Square value = 0.474, p-value = 0.789 demonstrate a non-significant association between asthma status and exposure to pets.

Table-II: Association of Family History based on Asthma and Non-Asthma groups .

Variable	Categories	Family History		Total	Chi-square value	P-Value
		Yes n(%)	No n(%)			
Asthma/ Non-Asthma	No	79(79.0)	21(21.0)	100	39.494	≤0.001
	Yes	35(35.0)	65(65.0)	100		
Total		114(57.0)	86(43.0)	200		

Table-III: Association of Occurrence of childhood respiratory infections each year among 121both groups (Asthma/ Non-Asthma).

Variable	Categories	Asthmatic/ Non- Asthmatics		Total n(%)	Chi-square value	P-Value
		Yes n(%)	No n(%)			
Childhood Respiratory Infections	Rare (≤1/year)	25(25.0)	27(27.0)	52(26.0)	18.196	≤0.001
	Occasional (1-2/year)	61(61.0)	35(35.0)	96(48.0)		
	Frequent (≥3/year)	14(14.0)	38(38.0)	52(26.0)		
Total		100	100	200		

Table-IV: SNP Profile and Effect of Pet Exposure Comparison between Asthmatic and Non-Asthmatic Individuals.

Variable	Categories	Asthmatic/ Non- Asthmatics		Total n(%)	Chi-square value	P-Value
		Yes n(%)	No n(%)			
SNP Profile	T/T	24(24.0)	65(65.0)	89(44.5)	39.439	≤0.001
	C/C	12(12.0)	13(13.0)	25(12.5)		
	T/C	64(64.0)	22(22.0)	86(43)		
Exposure to pets	None	35(35)	39(39)	74(37)	0.474	0.789
	Occasional	33(330	29(29)	62(31)		
	Frequent	32(32)	32(32)	64(32)		

Table-V: Spirometry (FEV1) Comparison Between Asthmatic and Non-Asthmatic Groups.

Variable	Categories	Asthmatic/ Non- Asthmatics		Total n(%)	Chi-square value	P-Value
		Yes n(%)	No n(%)			
Spirometry (FEV1)	Normal (80-100%)	33(33.0)	96(96.0)	129(64.5)	86.77	≤0.001
	Mild obstruction (65-79%)	38(38.0)	3(3.0)	41(20.5)		
	Moderate to severe obstruction (50-65%)	29(20.0)	1(1.0)	30(15.0)		

Table-VI: Peak expiratory flow rate (PEFR) analysis and gender distribution in Asthmatic and Non-Asthmatic Groups.

Variable	Categories	Asthmatic/ Non- Asthmatics		Total n(%)	Chi-square value	P-Value
		Yes n(%)	No n(%)			
Peak Expiratory Flow Rate (PEFR)	Green Zone (80-100%)	12(12.0)	87(87.0)	99(49.5)	113.609	≤0.001
	Yellow Zone (50-79%)	55(55.0)	11(11.0)	66(33.0)		
	Red Zone (<50%)	33(33.0)	2(2.0)	35(17.5)		
Gender	Male	42(42.0)	62(62.0)	104(52)	8.013	0.005
	Female	58(58.0)	38(38.0)	96(48)		

The Chi-Square tests in Table-V indicate a highly significant association between asthma status and spirometry results, specifically FEV1 (Forced Expiratory Volume in 1 second). The Chi-Square value = 86.77 and p-value ≤0.001 demonstrates a significant association between asthma status and FEV1 scores.

The Chi-Square tests in Table VI establish a connection between asthma status and Peak Expiratory Flow Rate (PEFR). The Chi-Square value = 113.609 and (p-value<0.001) indicates a strong association between asthma status and PEFR measurements. A significant association was observed between asthma status and PEFR (χ^2 = 113.6, p < 0.001) reflecting the impact of asthma on lung function and expiratory flow. Similarly shows a significant relationship between asthma status and gender. The Chi-Square value= 8.013 with p-value=0.005) confirms that gender is meaningfully associated with asthma status and highlighting the importance of gender differences in understanding asthma status. The Odds ratio was calculated to be 2.55, with a 95% confidence interval depicting that females have a greater tendency to be asthmatic.

DISCUSSION

This study aims to establish the presence of the rs7216389 polymorphism in the GSDMB gene in children with asthma. It contributes to the growing body of studies on gene-environment interactions that influence childhood asthma, a condition affecting millions globally, in which susceptibility is shaped by a complex interplay of genetic and environmental factors [10]. Among the participants, 61% of individuals with asthma were female, whereas 62% of those in the non-asthmatic control group were male. As shown in Table 1.5, there is a significant association between gender and asthma status, with females exhibiting a higher prevalence of asthma. This disparity may be influenced by variations in gene expression, epigenetic modifications, and environmental exposures [11].

The present research found a strong association between asthma status and genotype distribution, with the T/C heterozygous genotype being most prevalent among asthmatics (64%) and a highly significant p-value (≤0.001), reinforcing the role of this SNP in disease susceptibility. Similar genotype patterns have been reported in European and Asian cohorts, where the T allele was associated with increased ORMDL3 expression, airway hyperresponsiveness, and early-onset asthma [12]. FEV1 is a well-established indicator of asthma severity, showing a strong positive correlation with the frequency of exacerbations and the intensity of clinical symptoms. Findings from our study reinforce the high test-retest reliability of FEV1 in assessing asthma-related morbidity [13]. Among the asthmatic participants, 67% had an FEV1 below the predicted value (PFEV1), compared to only 4% in the control group. This finding validates previous studies showing a higher prevalence of reduced pulmonary function in asthmatic individuals. These findings further emphasize the clinical basis of spirometry results as a reliable tool for diagnosing and monitoring asthma [14].

The asthma patients studied showed disease patterns that were strongly associated with environmental factors, particularly exposure to household pets. According to Xhang, the influence of pet exposure on asthma development depends on genetic predisposition and the timing of that exposure [15,16]. The significant correlation between asthma status and frequent respiratory infections (p≤0.001) also parallels earlier epidemiological studies that identified respiratory infections as both triggers and aggravating factors in children with a genetic predisposition [17].

Asthmatic immune responses are influenced by hereditary factors through the GSDMB gene on chromosome 17q12-21, which also influences both anti-inflammatory action and epithelial crypt cell function. Research shows that GSDMB controls TSLP, along with two other inflammatory cytokines, IL-33 and IL-25, which cause neutrophils and eosinophils to enter the bronchial airways and promote the inflammatory response. People with the T/C or T/T allele of the genotype show a stronger response to this effect [17]. Children with the rs7216389 polymorphism demonstrate elevated ORMDL3 gene expression in the respiratory tract according to various studies. This increases their susceptibility to endoplasmic reticulum (ER) stress and distorted calcium signalling, which contributes to asthma inflammation [18].

Genetic evidence indicates a significant association between the rs7216389 polymorphism and asthma susceptibility (p<0.001), supported by a Chi-Square value of 277.259. This genetic pleomorphism is an essential risk factor for childhood asthma, and studies in other regions have also associated it with the condition [19]. The T/C heterozygous genotype is most common in asthma patients (64%), suggesting that this genotype may increase susceptibility to asthma. This is further supported by the concept of gene-environment interactions, in which certain genotypes may predispose individuals to environmental factors such as allergens or smoke, contributing to the development of asthma [20].

Research has given abundant evidence that people with the rs7216389 polymorphism develop specific epigenetic modifications that alter the GSDMB locus methylation patterns, leading to elevated inflammatory cytokine production in response to allergens and pollutants [21]. Between populations, rs7216389 shows different allele frequencies, with European and Hispanic groups having a greater likelihood of carrying the risk-increasing Tallele than African and East Asian groups [22].

Experimental research indicates that the rs7216389 SNP regulates immune cell functions for dendritic cells and airway epithelial cells to manage inhalation of irritants through inhaled allergens [23]. The rs7216389 mutation may also affect the airway microbiome, with children carrying this variant showing a unique microbiome profile, including a higher frequency of harmful bacteria linked to more severe asthma [24].

The rs7216389 polymorphism is also associated with an earlier onset of asthma, suggesting that it may enhance the susceptibility to develop asthma at a younger age [25]. Moreover, it may affect the response to asthma therapies, particularly corticosteroids. Studies indicate that children with the T/T genotype of rs7216389 may exhibit reduced sensitivity to corticosteroids due to elevated synthesis of many cytokines involved in inflammation [25].

These findings carry significant clinical implications, indicating that genetic screening for rs7216389 and other asthma-associated SNPs could be integrated into routine assessments,particularly for children with a genetic predisposition [18]. Our study was conducted on a small sample size in two tertiary care hospitals in Lahore. The results may therefore vary in a different setting.

From a pharmacogenomics perspective, these results may pave the way for more personalized asthma treatments, enabling therapy plans tailored to genetic factors like the rs7216389 variant to enhance treatment efficacy, particularly in corticosteroid responsiveness. The ongoing advancement of technologies such as next-generation sequencing (NGS) will further enhance our understanding of asthma genetics, enabling more precise identification of risk factors and better patient stratification [19].

CONCLUSION

The study demonstrates that childhood asthma results from the combined influence of genetic and environmental factors, with the rs7216389 polymorphism in the GSDMB gene showing a significant association with disease susceptibility. The findings suggest that this genetic variant may serve as an important biomarker for early identification and risk assessment, while integrating genetic screening with environmental evaluation could enable personalized and more effective preventive and therapeutic strategies for childhood asthma. A comprehensive strategy is needed to implement asthma prevention and treatment based on research data. Integrating genetic screening, environmental assessments, and personalized interventions presents a promising approach. This precision medicine model focuses on addressing each patient's unique genetic and environmental profile, aiming to enhance outcomes and foster individualized care in the management of childhood asthma.

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Qudsia Umaira Khan: Substantial contributions to the conception and design of the work.
Mohd Saleh Bin Ahmad Kamal : The acquisition of data for the work.
Afreen Bano: Analysis and interpretation of data for the work.
Aimen Binte Asif : Drafting the work.
Ismail Mazhar: Reviewing it critically for important intellectual content.
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