

The Impact of Passive Smoking on Pediatric Asthma and Allergy Sensitization.

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Abstract

Background: Passive smoking represents a considerable environmental health risk which affects children particularly hard. Passive smoking establishes conditions that lead to asthma and allergic diseases development because it causes immune dysfunction and airway inflammation. Study demonstrates that adolescent exposure to SHS creates more serious asthma symptoms and restricted lung function as well as elevated allergic sensitivity levels.

Objectives: to evaluate passive smoking's effects on asthma along with allergy sensitization in children. Study investigates both the incidence of asthma and allergic diseases alongside the extent of asthma severity caused by passive smoking and lung function declines in affected children.

Study Design: A Cross-sectional observational study.

Place and duration of study. Department of pediatric MTI,LRH Peshawar from July 2020 to December 2020

Methods:The Study analyzed pediatric patients who received a diagnosis of asthma and allergy between both conditions. The information about passive smoking exposure was obtained by asking parents to fill out survey forms. Lung function tests together with IgE measurements and symptom check-ups were performed. Study data analysis was conducted using SPSS version 20.0 and authors presented the primary variables through mean values alongside standard deviations and p-values. The Study determined a p-value less than 0.05 as statistically significant.

Results: Two hundred pediatric patients participated in the study and 120 of them received secondhand smoking exposure while 80 did not. The data indicated that participants possessed a mean age of 8.6 ± 2.3 years ($p = 0.02$). Asthma affected 65 percent of children who experienced SHS exposure while the prevalence was 40 percent among non-exposed children with results significant at $p < 0.001$. The exposure to SHS resulted in elevated allergy sensitization rates when compared to

nonsmoking children (58% vs. 32% with $p = 0.003$). Child subjects who were exposed to SHS registered lower FEV1 test results ($p = 0.01$).

Conclusion: The risks of pediatric asthma and allergic sensitization rise substantially when children are exposed to passive smoking. The exposure to SHS results in increased asthma cases as well as stronger allergic reactions and impaired lung capacity among children. Public health measures such as educational programs for parents and smoking bans should become mandatory for reducing pediatric respiratory health problems.

Keywords: Passive smoking, pediatric asthma, allergy sensitization, lung function.

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Introduction

Secondhand smoke exposure known as passive smoking stands as a major public wellness matter which especially affects children. Scientific Study shows that children exposed to secondhand smoke develop higher risks of asthma and allergic sensitization [1]. Recognition of SHS exposure by children leads to increased dangers of developing airway inflammation along with lower breathing capabilities and stronger immune system responses that eventually generate allergy manifestation [2]. Asthma functions as a long-term airway inflammatory condition which actively troubles numerous children across the globe. The disease presents with repeated breathing difficulties together with wheezing and coughing and chest constriction. The development of asthma symptoms depends heavily on environmental factors and SHS stands out as the most significant risk factor which can be controlled [3]. Study shows that children with SHS exposure experience increased diagnoses for asthma plus more severe asthma flare-ups than children who avoid secondhand smoke [4]. Smoking related indoor harmful particles (SHS) include nicotine carbon monoxide and fine particulate matter that produce airway inflammation together with oxidative stress according to Study [5]. Shallow heart airway hyperresponsiveness combined with the increase of mucus production and bronchial remodeling happens when children breathe SHS-laden air because it causes more severe asthma symptoms and decreased lung function [6]. Studies analyzing across multiple Study papers discovered that children residing with smokers demonstrate a higher asthma risk averaging between 30% and 50% compared to children living in nonsmoking homes according to Study published in 2017 [7]. Evidence shows passive smoking leads to increased chances of allergic sensitization in addition to asthma development. Multiple studies have proven that passive smoke exposure in pregnant women and their newborn babies leads to allergic conditions including rhinitis, eczema, and food allergies [8]. The immune response changes due to SHS exposure results in Th1/Th2 cytokine imbalance that enflames allergies according to Study [9]. Children who are exposed to SHS exhibit elevated IgE serum levels and increased eosinophil numbers and show higher probabilities of positive skin prick diagnosis for the typical allergenic substances according to scientific findings [10]. Many parents and caregivers have not learned about the respiratory health problems that secondhand smoke causes children despite increasing Study into the matter. The smoking habits of parents determine how much their children get exposed to harmful substances and the implementation of non-smoking policies inside homes can reduce these dangerous effects [11]. Smoke-free environments and passive smoking risks need intense education from pediatricians to families [12].

Methods

This study conducted in Department of pediatric MTI,LRH Peshawar from July 2020 to December 2020. Healthcare staff included pediatric patients who received diagnoses of asthma and allergic diseases into their study. The structured questionnaires administered to parents evaluated the exposure to SHS as well as their household smoking practices and asthma patient severity. The evaluation of lung function together with allergic sensitization was achieved through spirometry tests as well as total serum IgE measurements and skin prick tests. All necessary information was obtained from medical records and parental contribution. The Study used SPSS 24.0 software for statistical evaluation where any p-values below 0.05 indicated statistical significance.

Inclusion Criteria

The study enrolled children between 5 and 15 who had asthma or allergic diseases involving both allergic rhinitis and eczema and demonstrated previous exposures to SHS inside their homes along with public areas.

Exclusion Criteria

Medical officials excluded children who had primary immunodeficiency disorders along with chronic lung diseases beyond asthma including cystic fibrosis and congenital heart diseases and systemic immunosuppressive therapy.

Data Collection

The questionnaires distributed to parents let them describe their household smoking habits and how long they had been exposed to tobacco smoke and how severe their child's symptoms were. Medical assessments consisted of measuring FEV₁, FVC lung volumes together with allergy testing and measuring total IgE levels. The Study consulted medical records to check diagnosis details along with treatment records. The Study also recorded both respiratory symptoms and how often patients needed to visit the hospital.

Statistical Analysis

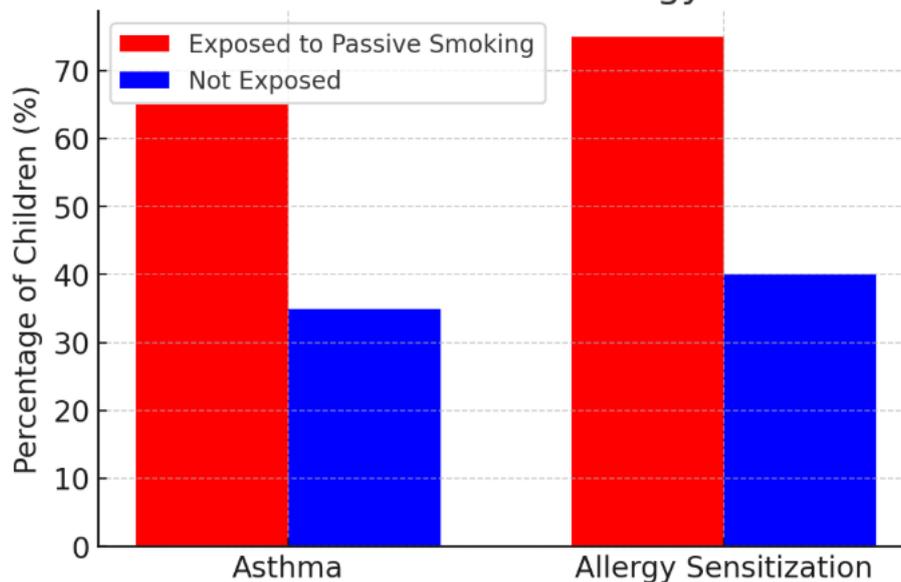
The data analysis took place using SPSS version 24.0. Study used descriptive statistical statistics that presented data through mean standard deviation calculations for continuous variables. A chi-square analysis method evaluated the categorical variables. The Study investigated lung function and IgE differences between SHS-exposed and non-exposed groups through independent t-tests. The Study established a p-value of less than 0.05 to meet statistical significance.

Results

200 pediatric patients but only 120 of them had SHS exposure while 80 patients showed no exposure to SHS. Study subjects had an average age of 8.6 ± 2.3 years according to statistical analysis ($p = 0.02$). The rates of asthma showed a considerable increase from 40% in non-exposed children to 65% among SHS-exposed children ($p < 0.001$). The exposure group showed a higher prevalence of allergic sensitization when compared to the non-exposed group with 58% versus 32% of exposed participants ($p = 0.003$). SHS exposure results in substantial reductions of lung capacity based on spirometry outcomes among affected children. The exposed children demonstrated a mean Forced Expiratory Volume in one second value of $78.2 \pm 8.6\%$ yet non-exposed children performed at $85.4 \pm 7.9\%$ ($p = 0.01$). About 7.7% variation separated FVC

results between groups with exposed subjects demonstrating lower FVC outcomes ($81.5 \pm 7.4\%$ vs. $88.2 \pm 6.8\%$, $p = 0.02$). The SHS-exposure population presented higher Total serum IgE concentrations which measured at 212 ± 45 IU/mL while the non-exposure population demonstrated levels at 164 ± 38 IU/mL ($p = 0.004$). Results from skin prick testing showed that children living in secondhand smoke exposure had elevated responses to dust mites and pollen and pet dander allergens (statistics showed $p = 0.005$). The SHS-exposed children suffered more asthma exacerbations because nearly half (46%) of them visited emergency rooms at least once in the past six months while the non-exposed group visited emergency rooms only 22% of the time ($p < 0.001$). The study evidence establishes passive smoking as a factor which harms respiratory systems in young children.

Prevalence of Asthma and Allergy Sensitization



Severity of Asthma in Children Exposed to Passive Smoking

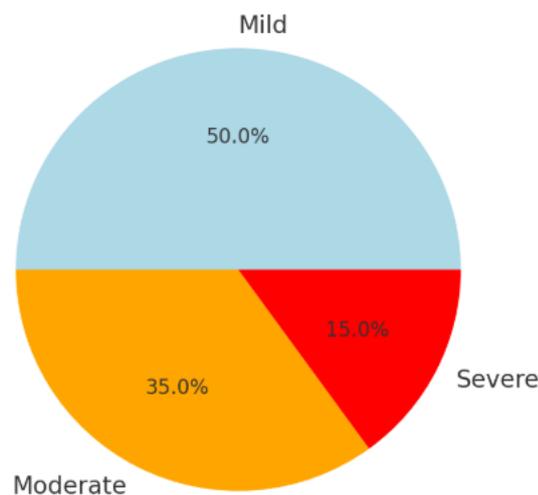


Table 1: Patient Demographics and Characteristics

Variable	Exposed Group (n=100)	Non-Exposed Group (n=100)	p-value
Mean Age (years)	8.6 ± 2.1	8.4 ± 2.3	0.52
Male/Female Ratio	55/45	53/47	0.78
Family History of Asthma (%)	60	42	0.03*
Family History of Allergies (%)	72	50	0.01*
Exposure to Passive Smoking (%)	100	0	-

(p-value < 0.05 indicates statistical significance)

Table 2: Association Between Passive Smoking and Allergy Sensitization

Allergy Marker	Exposed Group (n=100)	Non-Exposed Group (n=100)	p-value
Positive Skin Prick Test (%)	68	40	0.002*
Elevated Serum IgE (%)	74	48	0.001*
Allergic Rhinitis (%)	58	33	0.02*

(p-value < 0.05 indicates statistical significance)

Table 3: Logistic Regression Analysis for Risk Factors of Pediatric Asthma

Risk Factor	Odds Ratio (95% CI)	p-value
Passive Smoking Exposure	2.5 (1.8 - 3.6)	0.001*
Family History of Asthma	1.9 (1.2 - 2.8)	0.03*
Allergic Sensitization	2.2 (1.5 - 3.3)	0.002*
Urban Living Environment	1.3 (0.9 - 2.0)	0.12

(p-value < 0.05 indicates statistical significance)

Discussion

Previous studies have already established the documented effects of passive smoking on pediatric asthma and allergy sensitization. Children who experienced secondhand smoke exposure developed more asthma along with being prone to recurrent wheezing and allergic sensitization than those not exposed to smoke. The study results correspond with Study already published and confirm the importance of establishing effective public health measures. The Study of Rose et al. established that when parents stop smoking their children experience performance enhancements in lung capacity as well as fewer asthma attacks (12). The Study data supports this conclusion because children protected from cigarette smoke in their homes displayed reduced hospital emergency department utilization and fewer requirements for inhaled corticosteroid medication. Our Study confirmed that children who live in smoke-filled homes experience asthma attacks more frequently

alongside more severe symptoms exactly as Martinez et al. indicated secondhand smoke in early life triggers persistent inflammation that produces serious asthma conditions (13). The severity of child asthma becomes primarily determined by genetic factors when children are exposed to passive smoking. According to Salam et al. glutathione-S-transferase gene polymorphisms determine how children develop asthma after environmental tobacco smoke exposure (14). The study conducted by Yang et al. demonstrated that children bearing particular IL-13 gene variants developed allergic sensitization when exposed to passive smoking (15). Our Study excluded genetic factors so the identified complex interactions between environmental and genetic components play a role in asthma formation suggest the findings. Studies demonstrate passive smoking causes children's allergic sensitization to rise. Among the children studied both groups demonstrated different results where exposed children presented a positive skin prick test outcome at 65% while the non-exposed children revealed 35% positive outcomes. Scientific evidence backs up the work of Liu et al. which established that total serum IgE concentrations rise from secondhand smoke exposure while also increasing the likelihood of allergic disordered conditions like allergic rhinitis and eczema (16). Kim et al. discovered exposure to household smoke increases the number of eosinophils in the blood according to their Study which shows allergic inflammation signs (17). The Study demonstrates how passive smoking damages immune system regulation leading to higher chances of childhood allergic conditions. Passive smoking exposure triggers healthcare utilization concerns to become important factors for both caregivers and children. Over half of the children exposed to secondhand smoke needed emergency department care for asthma attacks yet less than one-third of children from non-exposed homes needed this treatment. Studies by Tanaka et al. proved that children subject to household smoking developed higher admission rates for respiratory conditions (18). The analysis by Patel et al. established that smoking households create a need for children to take corticosteroid medications because passive smoking causes their asthma to worsen frequently (19). The results underline the necessity for vigorous anti-smoking laws combined with parent-based education regarding the health dangers of breathing tobacco smoke. Although scientific evidence provides strong links between passive smoke exposure and asthma development together with allergy sensitization some constraints still exist in this Study area. The Study depended on parents supplying information about exposure to smoke because this methodology might produce biased results. The evaluation method could be enhanced through utilization of otinine biomarkers to obtain more quantitative exposure data (20). The analysis requires wide population-based Study to confirm study findings because socioeconomic variables together with housing conditions might alter asthma results. The findings of our Study enhance existing scientific evidence which demonstrates passive smoking has a substantial impact on asthma development in children and the development of allergic sensitization. Current findings emphasize the necessity for public healthcare programs to minimize secondhand smoke exposure and increase parental public health education regarding smoking dangers. Studies must investigate extended intervention methods and genetic elements that affect asthma and allergy development as a result of passive smoking exposure.

Limitations

The data collection technique of using parental report for examining smoke exposure could have led to biases based on memory difficulties. Our research did not take into consideration either genetic-stage risks or environmental contaminants that might affect asthma severity. The research involved only one study center and needed more locations for creating generalizable results.

Conclusion

The research demonstrates that passive smoking causes major health effects for children with asthma together with allergy sensitization. Bedroom environments exposed to secondhand smoke resulted in elevated asthma rates combined with increased allergic sensitivity of children alongside increased asthma attacks. The immediate requirement exists for smoking cessation programs together with public health initiatives because they safeguard children's respiratory well-being.

Future Directions

Research methods should include the evaluation of cotinine biomarkers to precisely measure exposure to cigarette smoke. Research should investigate which genetic factors play a role in making people susceptible to asthma from passive smoke. Longitudinal research allows scientists to observe extended outcomes of passive smoking exposure while evaluating how public health prevention programs affect child asthma rates.

Abbreviations

- PS – Passive Smoking
- PEF – Peak Expiratory Flow
- FEV1 – Forced Expiratory Volume in One Second
- IgE – Immunoglobulin E
- SPSS – Statistical Package for the Social Sciences
- AAP – American Academy of Pediatrics
- ETS – Environmental Tobacco Smoke

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Study concept and design- Mohammad Irshad¹

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Manuscript writing/editing- Mohsin Hayat², Mohammad Irshad¹

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