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Short Article

Environmental tobacco smoking and its consequences for allergy and asthma in children - a review

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ABSTRACT

Background. Asthma is one of the most common chronic respiratory diseases in children. Among important non-modifiable environmental risk factors—independent of genetic predisposition—is exposure to environmental tobacco smoke (ETS). Children are particularly vulnerable due to the immaturity of their respiratory and immune systems.

Aim. This study reviews the impact of passive smoking on asthma and allergy outcomes in children.

Material and methods. Studies were selected from PubMed, including publications from 2020 to 2026. The search terms included "passive smoking," "asthma," and "children." Non-English articles were excluded.

Results. The literature demonstrates a strong association between ETS exposure and increased risk of asthma and allergic diseases in children. Both prenatal and postnatal exposure were associated to higher incidence of wheezing, bronchial hyperreactivity, and physician-diagnosed asthma. ETS exposure was also associated with elevated immunoglobulin E (IgE) levels and increased sensitization to allergens, as well as a higher prevalence of atopic dermatitis and allergic rhinitis. A dose-response relationship was observed, with greater exposure linked to more severe symptoms. Mechanistically, ETS impairs mucociliary clearance,, disrupts epithelial barriers, and promotes airway inflammation. Early-life exposure may have long-term effects on lung development and immune function.

Conclusion. ETS is a significant and preventable risk factor for asthma and allergic diseases in children. Reducing prenatal and postnatal exposure is essential to protect respiratory health and improve long-term outcomes.

Key words: passive smoking, environmental tobacco smoking, allergy, asthma, children.

MATERIALS AND METHODS

This study was conducted as a narrative literature review aimed at synthesizing current scientific evidence regarding environmental tobacco smoking and its consequences for allergy and asthma in children.

Literature was searched for studies published between 2020 and 2026 using using PubMed, an online biomedical database, with the following keywords:: passive smoking, asthma, and children. The review included various study designs, such as randomized controlled trials (RCTs), observational studies, clinical trials, systematic reviews, and meta-analyses.

Inclusion criteria were as follows:

Studies addressed current developments and clinical practice.

Studies presented novel insights and in-depth analysis.

Twenty-one studies were eventually selected from the articles found for review. Articles were excluded if they did not meet predefined inclusion criteria, or were unavailable in full text or English.

INTRODUCTION

Tobacco smoke contains over 4,500 toxic chemicals, including particulate matter, oxidative gases, heavy metals, and at least 50 carcinogenic substances [1].

Exposure to tobacco smoke affects individuals who inhale it from the surrounding environment, including shared households and workplaces (so-called environmental tobacco smoke) [1,2]. Recent studies demonstrate that pyrolysis and combustion from indoor cigarette smoking are key contributors to increased levels of particulate matter and toxic chemicals in the home environment [1].

Environmental tobacco smoke (ETS) significantly impacts respiratory health and represents an important risk factor for respiratory diseases in children, particularly allergies and asthma [3–5].

DISCUSSION

Asthma as a common disease in the pediatric population

Asthma is among the most common chronic respiratory diseases in children, with global prevalence varying by region (3.4–29.2% of the pediatric population) [3,6]. The disease presents with respiratory symptoms of varying severity, and with acute exacerbations occasionally fatal. Asthma severity is influenced by multiple factors, including house dust mites, mold, air pollution, and other allergens [6].

Important non-modifiable environmental risk factors for asthma, independent of genetic predisposition, include environmental tobacco smoke (ETS), including secondhand smoke (SHS). Secondary smoke (SHS), also called passive or involuntary smoking, differs from active (firsthand) smoking [7]. Mainstream smoke contains approximately twice the nicotine, five times more carbon monoxide, and up to 50 times more ammonia compared to smoke directly inhaled by smokers. Children are particularly vulnerable to passive ETS exposure due to both the exposure route and immaturity of their respiratory and immune systems. School-aged children in smoking households experience higher rates of bronchitis, bronchiolitis, and pneumonia, with reduced lung function. [2].

Asthma may recur in adulthood, associated with atopy and continued smoking, especially in non-atopic individuals,. Recurrent adult asthma often has a more severe course than adult-onset asthma, with more frequent exacerbations and greater airflow limitation [8].

Smoking and its impact on respiratory health

The mechanisms by which secondhand smoke affects the respiratory system resemble those of active smoking; however, the effects are typically less pronounced due to lower exposure levels. Nevertheless, the subtle effects of secondhand smoke on pediatric respiratory health may have widespread consequences in children’s daily environments [7,9].

The World Health Organization estimates that over 700 million children worldwide are exposed to passive smoking. Children are particularly susceptible due to narrower airways, higher ventilation rates relative to body weight, and immature immune systems. Asthmatic children are especially susceptible. Secondhand smoke exposure in asthmatic children is associated with more frequent exacerbations and increased bronchial hyperresponsiveness [6,9,10].

Numerous studies have examined the pathophysiological and clinical interactions between tobacco smoking and asthma,, leading to the concept of the “smoker’s asthma phenotype.” Asthmatic smokers exhibit different airway inflammation patterns compared to non-smoking asthmatics. Tobacco smoke induces significant changes in asthma endotypes, with predominance of activated macrophages and neutrophils in sputum, airways, and lung tissue,, similar to early chronic obstructive pulmonary disease (COPD). Long-term tobacco exposure leads to synergistic inflammatory responses and airway remodeling, including chronic mucus hypersecretion,, explaining accelerated lung function decline and greater airflow obstruction severity in asthmatic patients [5].

Genetics, asthma and environmental tobacco smoking

Genetic factors influence asthma risk associated with secondhand smoke (SHS) exposure, as demonstrated in the Cincinnati Childhood Allergy and Air Pollution Study. It was demonstrated that variants in the N-acetyltransferase 1 (NAT1) gene modified children's susceptibility to developing asthma in the context of passive smoking exposure [1]. Prenatal smoking's adverse health effects are largely mediated by epigenetic mechanisms, including DNA methylation changes at loci associated with fetal tobacco exposure. Studies suggest transgenerational effects: maternal grandmother's smoking during pregnancy increases grandchild asthma risk, independent of maternal smoking. [1,4]. Paternal prenatal smoking exposure is associated with asthma in 6-year-old children,, likely via IgE-independent mechanisms. Paternal prenatal smoking is associated with epigenetic changes in LMO2 and IL-10 genes, linked to later asthma development [4].

Tobacco smoke affects the ubiquitin-proteasome pathway,, which maintains cellular protein quality control. Disruption via secondhand smoke exposure may contribute to chronic lung disease development in children [7].

Tobacco smoking and the respiratory microbiome

Like the gastrointestinal tract, the respiratory tract is colonized by diverse bacteria, viruses, and fungi. Tobacco smoke exposure may alter microbiota composition, potentially causing persistent dysbiosis. One study showed that pharyngeal microbiota of tobacco smoke-exposed individuals had higher abundances of *Porphyromonas*, *Neisseria*, and *Gemella* compared to non-exposed individuals [1].

Tobacco smoke components may directly influence the respiratory microbiota by enhancing *Staphylococcus aureus* biofilm formation. Additionally, tobacco products may contain bacteria and fungi, including potential pathogens. Tobacco smoke increases mucus production, impairs mucociliary clearance, and induces chronic pulmonary inflammation [1,9,11].

Chronic respiratory diseases, including asthma and bronchiectasis, are associated with altered respiratory microbiota composition. Asthmatic airways, characterized by chronic inflammation and excessive mucus secretion, create different ecological niches than healthy airways. However, whether dysbiosis causes disease or results from it remains unclear [1,5,9].

The respiratory microbiota composition correlates with pro-inflammatory cytokine levels in murine lungs,, suggesting microbiota changes may modulate the host inflammatory response [1].

Different studies on the impact of passive smoking and asthma in children

Passive smoking exposure, during prenatal and early childhood periods, is associated with increased asthma risk, poorer disease control, more frequent and severe exacerbations, and other chronic conditions. Prenatal tobacco exposure increases carboxyhemoglobin levels and causes placental vasoconstriction, raising vascular resistance, reducing fetal blood flow, and causing chronic fetal hypoxia. This impairs cellular development and respiratory system formation [1,4,8,12]. Postnatal ETS exposure increases asthma risk by 21–85%, with strongest effects in children exposed during the first two years of life [1,4,12].

A five-cohort European study showed maternal smoking during infancy increased asthma risk by 15%. Children's increased susceptibility results from immune and respiratory immaturity, plus higher relative ventilation per unit body weight [1].

A 131-country study showed substantial adolescent (aged 13–15) exposure to secondhand smoke at home (43.9%) and in public spaces (55.8%), with 46.5% having smoking parents. These exposures may have persistent effects into adulthood due to developing organisms' vulnerability to tobacco toxins [1,13].

A study revealed high current smoking prevalence among low-income urban adolescents (24% in past 30 days), with similar rates between girls and boys. While developing countries show higher smoking rates among boys, global prevalence is higher among girls [3,13].

Approximately 15% of young smokers initiated smoking at age ≤10 years,, consistent with other developing regions. Over half of children initiating smoking at age ≤12 years became regular smokers within 2–3 years, indicating high addiction risk. High smoking prevalence among girls is concerning, as many continue smoking during reproductive years and pregnancy, resulting in offspring respiratory morbidity (pneumonia, recurrent wheezing). A South American study found 34.4% of reproductive-age women smoked, with 24.8% continuing during pregnancy. Female smokers have higher airway obstruction risk than male smokers [1,3,9,13].

Early smoking initiation among disadvantaged children is largely influenced by peer acceptance, easy access, and household smokers [1,9,13].

Biomarkers of exposure to passive smoking

Cotinine, the primary metabolite of nicotine, is widely recognized as the most reliable indicator of exposure to tobacco smoke. Compared to nicotine, it has a longer half-life in the body, which facilitates its detection and the assessment of exposure. In the context of forensic medicine, the measurement of cotinine levels in urine serves not only as a clinical marker of exposure to environmental tobacco smoke (ETS), but also as objective evidence in legal proceedings, such as child custody cases, investigations of neglect, or public health–related litigation where the harmful effects of tobacco smoke are considered [6,14,15].

Additionally, the impact of passive smoking on the composition of the nasal microbiota and its relationship with cotinine levels and mNF% values has been investigated. The results indicate that exposure to passive tobacco smoke (PSE) leads to reduced biodiversity and alterations in microbiota composition, as confirmed by both α- and β-diversity analyses. Dysbiosis promotes the emergence of new, specific bacterial taxa as well as the overgrowth of environmentally favored species [6,14].

These changes may result from tobacco smoke's suppression of Th1 and Th17 responses while having limited effects on Th2 responses,, thereby facilitating selected bacterial species colonization and persistence. Correlation analysis showed *Moraxella nonliquefaciens* was negatively associated with passive smoking exposure (assessed by cotinine levels),, and positively associated with nasal airflow, suggesting it is part of a healthy nasal microbiota in children [6,11,14].

Higher cotinine levels were positively correlated with *Staphylococcus epidermidis* and *S. quinivorans* presence,, indicating these bacteria's greater capacity to adapt to environmental changes from tobacco smoke exposure. Passive smoking may disrupt nasal microbial homeostasis and exacerbate allergic rhinitis symptoms [11,14,15].

Other consequences of passive smoking on children's health

Environmental tobacco smoke (ETS) induces acute cardiovascular dysfunction in children. Tobacco smoke exposure during childhood promotes cardiovascular risk factor accumulation, including obesity, dyslipidemia, and hypertension. ETS toxins, such as benzo[a]pyrene, accelerate atherosclerotic development. Children exposed to home ETS are more likely to develop hypertension and show increased cardiovascular reactivity [2].

Cigarette smoking contributes to dental caries development in adults. ETS exposure is associated with decreased vitamin C levels, which promotes *Streptococcus mutans* growth. In vitro studies demonstrate increased *S. mutans* and *Streptococcus sanguinis* growth under various atmospheric conditions (air, CO_2, cigarette smoke), suggesting nicotine may stimulate cariogenic bacteria proliferation. ETS weakens immunity by reducing serum immunoglobulin G (IgG) levels, inhibiting T-helper lymphocyte activity, and limiting phagocytosis [16].

A meta-analysis of 32 studies ($n \approx 190{,}000$) found parental smoking was not a strong risk factor for eczema, food allergy, or food hypersensitivity in children ≤3 years. Other environmental factors, such as early microorganism exposure, may more significantly shape immune function and modulate allergy risk. Subgroup analyses examined active and passive parental smoking across developmental periods; however, methodological limitations (self-reported exposure and symptoms, lack of tobacco product type data) may introduce recall and misclassification bias [17].

Protection of children from tobacco [15,18–21].

- The FDA should regulate all tobacco and nicotine products to protect public health.
- Prevention of tobacco use, screening, and treatment should be adequately funded and specifically targeted toward the pediatric population.
- Tobacco control research should be prioritized and funded by both governmental and private sources.
- Prices of tobacco and nicotine products should be increased to reduce smoking initiation among children and adolescents.
- A minimum legal sales age of at least 21 years for tobacco products should be enforced.
- All flavorings, including menthol, should be banned in all tobacco and nicotine products.
- Comprehensive legislation should be enacted to prohibit the use of all tobacco and nicotine products in all places where children live, learn, play, work, and visit.
- All forms of advertising and promotion of tobacco and nicotine products accessible to children and adolescents should be prohibited.
- Advertising and point-of-sale displays of tobacco and nicotine products visible to children and adolescents should be banned.
- The depiction of tobacco and nicotine products in films and other media, including streaming platforms accessible to children and adolescents, should be restricted.
- Tobacco industry–sponsored mass media campaigns and school-based tobacco control education programs should be prohibited.
- Tobacco control programs for children and adolescents should include anti-tobacco messaging addressing health effects and industry manipulation.
- All children and adolescents should be legally prohibited from working on tobacco farms and in tobacco production, including refugees and migrants.
- All tobacco or nicotine products legally sold to young adults (≥21 years) should meet minimum standards that limit addiction or initiation, appropriate for children, adolescents, and young adults.
- Research and action priorities in tobacco control should be based on “tobacco endgame” strategies aimed at preventing tobacco use disorders and ending the tobacco epidemic.

SUMMARY

Environmental tobacco smoke (ETS) remains a major, preventable risk factor affecting pediatric respiratory and immune health. This review highlights ETS’s significant role in asthma and allergic disease development and exacerbation. Tobacco exposure during prenatal and early childhood periods contributes to impaired lung development, increased airway inflammation, and heightened allergen sensitization.

Children are uniquely vulnerable due to respiratory and immune immaturity, combined with greater relative indoor pollutant exposure. ETS increases asthma onset risk while worsening disease control, increasing symptom frequency, exacerbations, and healthcare use. Associations with allergic rhinitis and atopic dermatitis further emphasize tobacco smoke’s broad immunomodulatory effects.

Given the consistent evidence linking ETS exposure with adverse health outcomes, reducing children's exposure to tobacco smoke should be a public health priority. Smoking cessation programs, parental education, and comprehensive smoke-free policies are essential. Limiting ETS exposure significantly improves respiratory outcomes and pediatric quality of life.

DISCLOSURE

Author's Contribution Statement:

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In preparing this work, the authors used ChatGPT for the purpose of checking grammar, punctuation and improving the readability of the article. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

REFERENCES:

1. Eguiluz-Gracia I, Mathioudakis AG, Bartel S, et al. The need for clean air: The way air pollution and climate change affect allergic rhinitis and asthma. *Allergy*. 2020;75(9):2170-2184.https://doi.org/10.1111/all.14177

2. Mehdi Jafri SF, Hussain SF, Zehra W. Passive Smoking And The Health Of Children: A Primary Concern In Pakistan. *J Pak Med Assoc*.2023;73(11):2315-2316. https://doi.org/10.47391/JPMA.9421

3. Mukharesh L, Phipatanakul W, Gaffin JM. Air pollution and childhood asthma. *Curr Opin Allergy Clin Immunol*. 2023;23(2):100-110. https://doi.org/10.1097/ACI.0000000000000881

4. Tiotiu AI, Novakova P, Nedeva D, et al. Impact of Air Pollution on Asthma Outcomes. *Int J Environ Res Public Health*. 2020;17(17):6212. https://doi.org/10.3390/ijerph17176212

5. Riesco JA, Rábade C, Signes-Costa J, Cabrera E, Jimenez CA. Frequency and characteristics of asthma in smokers attending smoking cessation units in Spain. *PLoS One*. 2023;18(12):e0294587. https://doi.org/10.1371/journal.pone.0294587

6. Agarwal D, Choudhary R, Narayan S, Yadav PK, Singh KK. Assessment of Passive Smoking in Children With Asthma Using Urinary Cotinine Levels and Its Association With Asthma Severity. *Cureus*. 2025;17(7):e88583. https://doi.org/10.7759/cureus.88583

7. He Z, Wu H, Zhang S, et al. The association between secondhand smoke and childhood asthma: A systematic review and meta-analysis. *Pediatr Pulmonol*. 2020;55(10):2518-2531.https://doi.org/10.1002/ppul.24961

8. Miligkos M, Oh J, Kwon R, et al. Epidemiology of asthma across the ages. *Ann Allergy Asthma Immunol*. 2025;134(4):376-384.e13. https://doi.org/10.1016/j.anai.2024.12.004

9. Pijnenburg MW, Frey U, De Jongste JC, Saglani S. Childhood asthma: pathogenesis and phenotypes. *Eur Respir J*. 2022;59(6):2100731. https://doi.org/10.1183/13993003.00731-2021

10. Załęski A, Szwejkowska M, Rykowska D, Nitsch-Osuch A. Impact of parental smoking on influenza and others respiratory infections in children. *Przegl Epidemiol*. 2020;74(3):475-482. https://doi.org/10.32394/pe.74.41

11. Elwany S, Gamea MA, Talaat I. Passive smoking induces nasal biofilms in children. *Int J Pediatr Otorhinolaryngol*. 2021;146:110755. https://doi.org/10.1016/j.ijporl.2021.110755

12. Yamada M, Tanaka K, Nagata C, Arakawa M, Miyake Y. Sex differences in the association between smoking exposure and prevalence of wheeze and asthma in 3-year-old children. *J Asthma*. 2023;60(7):1369-1376. https://doi.org/10.1080/02770903.2022.2147081

13. Mallol J, Urrutia-Pereira M, Mallol-Simmonds MJ, Calderón-Rodríguez L, Osses-Vergara F, Matamala-Bezmalinovic A. Prevalence and Determinants of Tobacco Smoking Among Low-Income Urban Adolescents. *Pediatr Allergy Immunol Pulmonol*. 2021;34(2):60-67. https://doi.org/10.1089/ped.2021.0018

14. Brindisi G, Marazzato M, Brunetti F, et al. Allergic rhinitis, microbiota and passive smoke in children: A pilot study. *Pediatr Allergy Immunol*. 2022;33 Suppl 27(Suppl 27):22-26. https://doi.org/10.1111/pai.13621

15. Akar-Ghibril N, Phipatanakul W. The Indoor Environment and Childhood Asthma. *Curr Allergy Asthma Rep*. 2020;20(9):43. https://doi.org/10.1007/s11882-020-00941-5

16. Dantham P, Nuvvula S, Ismail AF, Akkilagunta S, Mallineni SK. Association between passive smoking and dental caries status in children: A cross-sectional analytical study. *Dent Med Probl*. 2024;61(2):209-216. https://doi.org/10.17219/dmp/156655

17. Lau HX, Lee JW, Yap QV, Chan YH, Samuel M, Loo EXL. Smoke exposure and childhood atopic eczema and food allergy: A systematic review and meta-analysis. *Pediatr Allergy Immunol*. 2023;34(8):e14010. https://doi.org/10.1111/pai.14010

18. Hageman JR, Alcocer Alkureishi L, Montalto GJ. Protection of Children and Adolescents from Tobacco and Nicotine. *Pediatr Ann*. 2023;52(8):e277-e278. https://doi.org/10.3928/19382359-20230711-01

19. Umutlu S, Kocataş S. The effects of planned education given to parents on smoking in the home environment and passive exposure of children aged 0-5: A quasi-experimental pre-

post intervention study from Turkey. *J Pediatr Nurs*. 2023;72:e228-e237. doi:10.1016/j.pedn.2023.07.023 https://doi.org/10.1016/j.pedn.2023.07.023

20. Radó MK, Mölenberg FJM, Westenberg LEH, et al. Effect of smoke-free policies in outdoor areas and private places on children’s tobacco smoke exposure and respiratory health: a systematic review and meta-analysis. *Lancet Public Health*. 2021;6(8):e566-e578. https://doi.org/10.1016/S2468-2667(21)00097-9

21. Hashemi-Aghdam MR, Shafiee G, Ebrahimi M, et al. Trend of passive smoking and associated factors in Iranian children and adolescents: the CASPIAN studies. *BMC Public Health*. 2022;22(1):603. https://doi.org/10.1186/s12889-022-13045-8