Review Article Pol J Public Health, Vol. 135 (2025): 25-29

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The impact of passive tobacco smoke exposure on the disease course and frequency of exacerbations in pediatric inflammatory bowel disease

Abstract

Inflammatory bowel diseases (IBD), including ulcerative colitis and Crohn's disease, are an increasing health problem in the pediatric population, with rising incidence globally. Environmental factors are crucial in the etiology and progression of these disorders. A key modifiable risk factor is exposure to tobacco smoke, both active and passive. This study aimed to analyse current knowledge on the impact of passive smoking on the risk and course of IBD in children.

The literature review shows that although a direct link between passive smoking and pediatric IBD etiology is not conclusively proven, there is strong evidence suggesting its negative effect on disease activity and prognosis. Prenatal and early childhood exposure, in particular, may influence immune mechanisms and predispose children to disease development and exacerbations. Children with IBD exposed to secondhand smoke have more frequent flare-ups, require more hospitalizations, and often need intensified immunosuppressive treatment.

The importance of preventive actions is also emphasised, especially education of parents and caregivers to limit children's exposure to tobacco smoke. Effective strategies to reduce passive smoking at home and at the legislative level are necessary. A comprehensive interdisciplinary approach covering medical, social, and legal aspects is essential to improve the quality of life among children with IBD and reduce related health and economic burdens.

This analysis highlights the need for further research and preventive measures to minimise the harmful impact of passive smoking on the course of IBD in the pediatric population.

Keywords: nflammatory bowel disease, pediatric, children, Crohn's disease, ulcerative colitis, passive smoking, public health, prevention.

DOI: 10.12923/2083-4829/2025-0006

INTRODUCTION

Inflammatory bowel diseases (IBD), which include Crohn's disease (CD) and ulcerative colitis (UC), are chronic autoimmune conditions characterised by alternating periods of remission and exacerbation [1-7]. In recent years, an increasing incidence of these disorders has been observed in the pediatric population [2,6,7]. It is estimated that children and adolescents currently account for approximately 1.4% of all IBD cases [2].

Global incidence trends indicate a rising prevalence of IBD, with the highest increases reported in Northern Europe and in North America, and the lowest in Southern Europe, Asia, and the Middle East [6,8]. The current pediatric incidence rate is estimated from 0.2 to 23 cases per 100,000 children annually in Europe, and 1.1 to 15.2 per 100,000 in North America [9]. Some sources predict that pediatric IBD could affect up to 1% of the entire population by 2030 [6].

The rapid increase in the incidence of IBD among very young children, estimated at 7.2% annualy, is a particular cause for concern. Approximately 4% of IBD cases are diagnosed before the age of 5, and 18% before the age of 10. The peak incidence, however, occurs during adolescence [6,10].

The pathophysiology of IBD is complex and involves multiple contributing factors. Among the most significant are genetic susceptibility and environmental exposures [1-6]. It is generally accepted that environmental factors play a key role in IBD susceptibility, while genetic factors modulate the disease course and clinical manifestations [4].

Relevant environmental factors include exposure to stress, dietary habits, frequent antibiotic use in childhood (which alters the gut microbiota), the hygiene hypothesis (suggesting that reduced early-life exposure to microbes impairs immune regulation), and air pollution, including secondhand tobacco smoke exposure [2,6,10].

Children's exposure to tobacco smoke is a significant public health concern. Globally, it is estimated that over 40% of children are regularly exposed to secondhand smoke. In Poland, WHO data indicate that this figure exceeds 30% [11]. The health consequences of such exposure extend beyond respiratory illnesses and include gastrointestinal diseases such as IBD [11,12].

This issue also imposes substantial social and economic burdens, including increased school absenteeism, higher hospitalisation rates, reduced quality of life, and elevated healthcare costs [12].

While exposure to tobacco smoke is a recognised environmental factor in IBD pathogenesis, the specific impact of passive smoking on pediatric-onset IBD remains insufficiently

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understood. This issue requires systemic public health interventions, including legislative measures, education, and family support. A better understanding of this relationship could contribute to the prevention of chronic diseases in children.

AIM

The aim of this study is to review the available scientific literature regarding the impact of passive exposure to tobacco smoke on disease course and frequency of exacerbations in children with inflammatory bowel disease (IBD), with particular emphasis on the public health relevance of this issue and the need to implement effective preventive strategies.

MATERIAL AND METHODS

This study is a non-systematic literature review. The databases PubMed, Google Scholar, and Scopus were searched for articles published between the 1st of January 2020, and the 31 st May 31 2025, using the following keywords: "inflammatory bowel disease", "pediatric", "children", "Crohn's disease", "ulcerative colitis", "passive smoking", "environmental factors IBD", and "environmental tobacco smoke", "prevention". Only articles published in English, that focused on children and adolescents up to 18 years of age diagnosed with inflammatory bowel disease (Crohn's disease and ulcerative colitis), were included. Eligible studies evaluated the impact of passive tobacco smoke exposure on disease courses or the frequency of flare-ups. The review included original research articles, review papers, and meta-analyses published within the last five years, including observational studies (cohort and cross-sectional) and clinical studies.

Studies were excluded if they focused exclusively on active smoking without reference to passive exposure, involved only adult populations, were case reports, commentaries, or letters to the editor without original data, or were not available in full text. Publications unrelated to inflammatory bowel disease or written in languages other than English without the possibility of reliable translation were also excluded.

As this was a non-systematic review, no formal assessment of the quality of the studies or risk of bias was performed. Some of the included studies had methodological limitations, such as small sample sizes or a lack of standardised definitions of tobacco smoke exposure.

State of the Current Knowledge

Mechanism of Tobacco Smoke Action on Intestinal Mucosa

It is clear that smoking can influence the course of inflammatory bowel diseases (IBD) through the effects of tobacco smoke on the intestinal mucosa [13]. Several mechanisms have been proposed by which components of tobacco smoke may affect the intestinal mucosa, one of them involves nicotinic acetylcholine receptors present in intestinal epithelial cells and T lymphocytes [13-15].

Richardson et al. demonstrated in their study that the alpha-3 subunit was detected in many cells of the healthy colon, including the intestinal epithelium. Smoking and nicotine did not alter its quantity in either healthy individuals or patients with ulcerative colitis (UC). However, its levels were higher in healthy subjects compared to UC patients [14]. In contrast, Razani-Boroujerdi S et al. identified the alpha-7-nAChR receptor in T lymphocytes, which is very similar to the neuronal receptor and regulates intracellular calcium levels through cooperation with the TCR/CD3 complex. Unlike neurons, this receptor in T lymphocytes does not form a classical calcium channel. Since T lymphocytes play a key role in the intestinal immune response, their activation by nicotine can affect inflammation and the integrity of the intestinal epithelium [15]. Clinical studies on nicotine administration in patients with ulcerative colitis yielded only modest and inconclusive effects, which suggests that nicotine alone is likely not the sole tobacco smoke component influencing disease course [13,16]. Other mechanisms indicate that substances contained in tobacco smoke modulate cellular responses and affect the levels of inflammatory cytokines. Nicotine and other toxins in tobacco smoke can induce oxidative stress and inflammation, as well as lead to epigenetic modifications that regulate genes related to immune response and inflammation induction [13,17-21].The impact of smoking on altered mucus composition and production of mucus in the intestine has also been documented. Furthermore, tobacco smoke influences the risk of microthrombosis formation in the capillaries of the intestinal mucosa, impairing blood flow [13,16].

The impact of Passive Smoking on the Risk and Course of Inflammatory Bowel Diseases in Children

Studies from the last decade have not demonstrated a significant association between passive exposure to tobacco smoke and the risk of developing IBD in children. Nevertheless, active smokinghas been recognised as a significant factor, whic increases this risk [22,23]. Han D.Y. et al. found no evidence that passive smoking influenced the course, exacerbation, or incidence of Crohn's disease (CD) in children, including those born to mothers who smoked during pregnancy [22]. No significant correlation was observed between passive smoking and IBD incidence among preschoolers, older children, or adolescents [22]. In contrast, active smoking markedly increased the risk individuals who had smoked more than one cigarette in their lifetime had a 1.94 fold higher risk of developing CD [22]. Similar findings were reported by Basson A. et al., who also found no influence of passive smoking in age groups 0-5, 6-10, and 11-18 years on CD incidence [23]. Bernstein C.N. et al. confirmed the absence of a link between passive smoking and IBD risk in children, while emphasising the role of active smoking [24]. It is important to note that these studies had limitations, including small sample sizes, challenges in assessing tobacco smoke exposure, and varied observation periods. However, recent meta-analyses have provided new insights, identifying passive smoking as a potential risk factor for IBD alongside others such as family infection history, antibiotic use, non-white race, and residence in high-income countries [2]. A meta-analysis carried out in 2021 by Agrawal et al. confirmed no significant association between childhood passive smoking and IBD risk but revealed a significant effect of maternal smoking during pregnancy, increasing the risk of IBD diagnosis in children by 49% [1,25,26]. This meta-analysis also suggested that passive smoking, especially during prenatal and early childhood periods, may increase the risk of developing ulcerative colitis (UC) in children, whereas the focus previously more on CD risk [1].

Fetal exposure to tobacco smoke has also been linked with low birth weight, obesity, behavioral disorders, and immunological, epigenetic, and microbiota changes. Recent studies indicate that exposure to passive tobacco smoke may be

TABLE 1. Review of studies on the impact of passive smoking on the course and risk of inflammatory bowel disease in children .

Author (Year)	Study Type	Population/Age Group	Main Findings	Conclusion
Bernstein et al. 2006 [24]	Population-based retrospective case-control study	Children and adolescents, 16-50 years, Canada	No link between passive smoking and IBD	Active smoking linked to increased CD risk
Han et al. (2010) [22]	Retrospective case-control study	Children, 0-18 years, New Zealand, (315 CD patients, 536 controls)	No significant association between passive smoking and IBD	Active smoking increased CD risk; passive exposure not significant
Basson et al. (2014) [23]	Retrospective cohort study	Children, 0-18 years, South Africa, (194 CD patients, 213 controls)	No correlation with passive smoking	Passive smoking in adolescence (11-18 years) associated with increased CD risk (OR = 1.93)
Smith et al. (2019) [27]	Prospective cohort study	Children, 5-18 years, USA, (209 CD patients, 164 controls)	Higher relapse rate and more hospitalizations in exposed children	Suggests passive smoking may worsen disease course
García et al. (2020) [29]	Prospective cohort study	Children, 6-18 years, Spain, (145 children IBD-38 exposed to ETS)	No significant correlation with relapse frequency	Limitations in exposure measurement noted
Agrawal et al. (2021) [1]	Meta-analysis	13 studies, global data, children 0-18 years (data from over several thousand pediatric IBD patients)	No link with childhood passive exposure; 49% higher IBD risk if mother smoked during pregnancy	Prenatal exposure is a significant risk factor
Lee et al. (2021) [28]	Prospective cohort study	Children, 0-15 years, Japan	Higher disease activity and CRP levels in exposed children	Possible association with increased inflammation
Thacker et al. (2024) [2]	Meta-analysis	36 studies, global data, children 0-18 years (data from over several thousand pediatric IBD patients)	Passive smoking among risk factors for pediatric IBD	Suggests environmental influence of passive exposure
Sigvardsson et al. (2024) [35]	Prospective cohort study	Children, 0-18 years, Scandinavian	Early exposure to tobacco smoke increases risk of developing IBD	Confirms the impact of passive smoking in early childhood on IBD development

associated with more severe disease courses and more frequent relapses of IBD in children [27,28]. Some research shows that children exposed to passive smoking experience more frequent disease exacerbations, require hospitalisation, and need steroid treatment more often (Smith et al. [27]). Similarly, Lee et al. reported higher disease activity and elevated inflammatory markers in children exposed to tobacco smoke [28].

However, not all data are consistent García et al. found no significant correlation between passive smoking and IBD exacerbation frequency in children, assigning discrepancies to limitations in exposure assessment methods [29].Table 1 presents an overview of studies on the impact of passive smoking on the risk and course of inflammatory bowel diseases in children.

Epidemiological and Population Data on Children's Exposure to Tobacco Smoke

Passive exposure to tobacco smoke (Environmental Tobacco Smoke, ETS) remains a serious public health issue, particularly among children. According to data from the World Health Organisation (WHO), over 40% of children worldwide are regularly exposed to tobacco smoke in domestic or public environments [11]. Frequently, this is due to parental unawareness of the harmful effects of tobacco smoke and its constituents on their children's health [11,30].

European data indicate that in some EU countries, up to one in three children live in households where at least one adult smokes tobacco in the presence of children [30]. The Global Youth Tobacco Survey (GYTS) reports that between 30% and 50% of school-aged children are regularly exposed to tobacco smoke both at home and in public places, despite existing smoking bans [30-32].

Data on the population of children with inflammatory bowel disease (IBD) are more limited; however, available studies suggest that the proportion of passive exposure may be as high or even higher than in the general pediatric population [33]. One study showed that approximately 35% of children with IBD were regularly exposed to tobacco smoke at home [1,33-35].

Similar observations confirmed that children with IBD are more likely to be raised in environments where at least one person smokes tobacco at home [34]. Furthermore, some analyses indicate that passive smoking exposure is more frequent among children with Crohn's disease than those with ulcerative colitis [34]. Considering that even short-term exposure to ETS can exacerbate inflammation and worsen the course of chronic diseases, the scale of this phenomenon represents a significant clinical and systemic challenge.

Significance of Passive Smoking for Public Health in Children with IBD

Exposure of children to tobacco smoke poses a significant public health threat, especially in the context of chronic diseases such as inflammatory bowel disease (IBD). Passive smoking at home and vehicle environments contributes to increased risk of disease onset and exacerbations in young patients, with negative impacton their immunological development and overall health, which also includes mental health and social functioning [27,28]. In response to these threats, effective preventive measures are essential, such as the implementation and enforcement of smoking bans in places frequented by children, particularly homes and vehicles [2,36]. Equally important is the introduction of supporting programs for parents and caregivers in smoking cessation, which have the potential to significantly reduce children's exposure to the toxic substances found in tobacco smoke [1,37-39]. Moreover, education for both parents and healthcare professionals should include information on the impact of passive smoking on IBD progression, enabling improved prevention and therapeutic support for patients [25,29]. An integrated public health approach is crucial to reducing disease burden and improving quality of life for children affected by IBD [37-39]. The importance of preventive strategies and health policy measures such as anti-nicotine campaigns, smoking bans in homes, and child protection education – should be emphasized more strongly [40].

However, there is a lack of data on how to effectively limit exposure in practice, making smoking cessation support programs for parents and strengthening legal regulations vital [37-39,41]. The long-term health consequences of passive smoking include more severe disease courses, poorer quality of life, higher treatment costs due to more frequent hospitalisations,

increased school absenteeism linked to social withdrawal, and disrupted psychological development in children. These factors impose additional burdens on healthcare systems and society [11,42].

CONCLUSIONS

The conducted literature review indicates that although current scientific evidence does not unequivocally confirm an association between passive exposure to tobacco smoke and the risk of developing inflammatory bowel disease (IBD) in children, increasing data suggest a significant impact of this environmental factor on the clinical course and frequency of disease exacerbations, particularly in ulcerative colitis. Exposure of children with IBD to tobacco smoke, especially in the home environment, canbe associated with greater symptom severity, higher inflammatory activity, more frequent hospitalisations, and intensified immunosuppressive therapy. Particularly concerning are data on prenatal exposure, including maternal smoking during pregnancy, which significantly increases the risk of IBD development in offspring and may modulate immunological and epigenetic mechanisms of the inflammatory response.

Available epidemiological data confirm that a considerable proportion of children, including those diagnosed with IBD, remain exposed to tobacco smoke, which constitutes a serious public health problem. Passive smoking therefore remains an important, modifiable environmental factor whose elimination could yield measurable health benefits in the pediatric IBD population. Considering the rising incidence of IBD in children and adolescents and the potential influence of environmental risk factors on disease course, it is imperative to implement effective preventive strategies at both individual and systemic levels.

In the context of clinical practice and public health, it is recommended to intensify educational efforts targeting parents, caregivers, and healthcare professionals, emphasising the negative impact of tobacco smoke exposure on the course of pediatric IBD. Implementing effective interventions to eliminate passive smoking in home environments such as smoking bans in the presence of children and smoking cessation support is also crucial. Legislative measures to limit children's exposure to tobacco smoke in private and public spaces, along with the development of prevention programs addressing environmental aspects in chronic disease management in children, are of key importance.

An integrated interdisciplinary approach encompassing education, legislation, and psychological and social support may contribute to the improvment of the quality of life for pediatric patients with IBD and to the reduction of the long-term health and economic burdens associated with this group of diseases.

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