Review Article

Passive Smoking and Lung Disease in Children, Systematic Review

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ABSTRACT

Background: Children's morbidity and mortality are greatly increased by passive cigarette smoke exposure. Preterm delivery, intrauterine growth retardation, perinatal death, respiratory illnesses, neurobehavioral issues, and lower academic performance have all been linked to environmental tobacco smoke (ETS) as having negative effects on children's health. This systematic review aims to comprehensively assess recent literature on prevalence of passive smoking in association with lung diseases in children.

Methods: Comprehensive searches were conducted across PubMed, Web of Science, Science Direct, Cochrane Library, and Google Scholar to include pertinent literature. The Rayyan QRCI tool facilitated this systematic approach.

Results: A total of 490 study articles resulted from the systematic search, and 53 duplicates were deleted. Title and abstract screening were conducted on 437 studies, and 390 studies were excluded. Forty-seven reports were sought for retrieval, and only 7 articles were not retrieved. Finally, 40 studies were screened for full-text assessment; 17 were excluded for wrong study outcomes, and 6 for the wrong population type. Seventeen eligible study articles were included in this systematic review.

Conclusion: ETS exposure has a considerable negative impact on children's morbidity and death. Children appear to be the population group most vulnerable to the negative impacts of ETS. There should be a consolidation of more efficient governmental policies and initiatives to safeguard preschoolers from ETS.

Keyword: Passive smoking, SHS, ETS, respiratory disease.

Introduction

Tobacco was once viewed as an ornamental plant, and it was not until much later that it started to be considered as an herb with unique medicinal qualities. It was eventually discovered that tobacco possessed potent insecticidal and addictive qualities. Additionally, there have been reports regarding the detrimental effects of tobacco on human health [1].

Smoking is classified as a chronic disease as a result of the World Health association (WHO). Progressive disease is also "contagious." It is regarded as a neurobiotic addiction as well. Although a smoker's behavior or ability to operate is unaffected by nicotine addiction, both their overall health and the health of others around them are negatively impacted [1].

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Tobacco smoke includes at least fifty cancer-causing agents, 4,000 toxic chemicals, including oxidizing agents, and toxic metals. Tobacco use is a leading reason of cancer and the greatest avoidable cause of death. There are presently 1.3 billion smoker individuals or tobacco users, and each year, over 5 million people globally pass away from diseases caused by cigarette smoke. Because environmental tobacco smoke (ETS) includes far smaller fragments than regular smoking, it can enter a child's lungs and airways more easily [2]. Passive cigarette smoke exposure considerably increases mortality and morbidity in youngsters. Youngsters appear the demographic group extremely sensitive to detrimental consequences of ETS [3]. Not just at home, but also in eateries, daycare centers, cars, buses, and other public places, children are exposed to passive smoke. Children's primary source of ETS is their household. The health of the fetus may be jeopardized as a result of fetal and maternal contact to ETS created by fathers smoking in the home [4]. ETS, also known as tobacco smoke produced by a regular smoker that comes from smoked tobacco exhale and the burning portion of the cigarette, is inhaled by nonsmokers [5]. ETS exposure in children's households has been observed to range from 27.6% in Africa to 0% in Europe, 34.3% of the population in the Southeast Asian region, 50.6% in the western Pacific region, and as high as 77.8% in Europe). Premature delivery, intrauterine growth delay, newborn death, respirational illnesses, and neurobehavioral disorders are only a few of the detrimental effects of ETS that have been connected to pediatric health [6]. Passive smoking has been proven to cause incorrect interferon production in children, increasing the chance of recurring respiratory infections. Furthermore, exposed children are more likely to develop allergic rhinitis than non-exposed children [7, 8].

Methods

PRISMA is an abbreviation for Preferred Reporting Items for Systematic Reviews and Meta-Analyses. This systematic review adhered to the criteria.

Study Design and Duration: this systematic review was completed in August of 2023. Strategy for searching: To discover the relevant studies, a thorough search of PubMed was conducted. We restricted our search to English and took into consideration each database's particular quirks. To discover pertinent research, the following keywords were translated into PubMed Mesh terms: "ETC," "Passive smoking," "SHS," and "respiratory disease." Publications in full English, studies that were available for free, and human trials were all included in the search results.

Criteria of Inclusion:

• Any study that investigates passive smoking and lung disease in children.

•The articles are free to read.

• Only children studies are included.

Exclusion criteria:

We excluded studies done among adults.

We excluded studies with no age criteria.

Case studies, letters to the editor, and dispute answers were not authorized. A language other than English.

Extraction of information: Rayyan (QCRI) was utilized to detect duplicates in the output of the search method. The relevance of the titles and abstracts in the combined search results was determined by the researchers using an assortment of inclusion/exclusion criterion. Each manuscript that complied with the requirements for inclusion was given a comprehensive evaluation by the reviewers. After giving it some thought, the writers suggested some different approaches to resolving disputes. The writers were able to gather data on the investigations' names, authors, study year, nation, participants, gender, diagnostic tool, main findings, and conclusions.

Strategy for data synthesis: To provide a qualitative evaluation of the results and research components, summary tables were made utilizing data from pertinent studies. The most effective method for using data from the included study articles was selected after data extraction for the systematic review.

Assessment of the risk of bias

The ROBINS-I risk of bias estimation strategy for non-randomized trials of interventions was used to evaluate the quality of the research that was included [24]. Confounding, research subject selection, action classification, deviations from intended actions, missing data, assessment of outcomes, and selection of the provided result were the seven issues covered.

Results

Search outcomes: The systematic search returned 490 study papers, 53 of which were deleted as duplicates. 390 articles were rejected after being assessed for title and abstract. Only seven things were not retrieved out of the 47 that were looked for. Finally, 40 papers were chosen for full-text review. 17 were removed because the research results were erroneous, and 6 were removed because the population type was inappropriate. This systematic review included seventeen study papers that were suitable for inclusion. In this section, a summary of the research selection approach is offered (Figure 1).

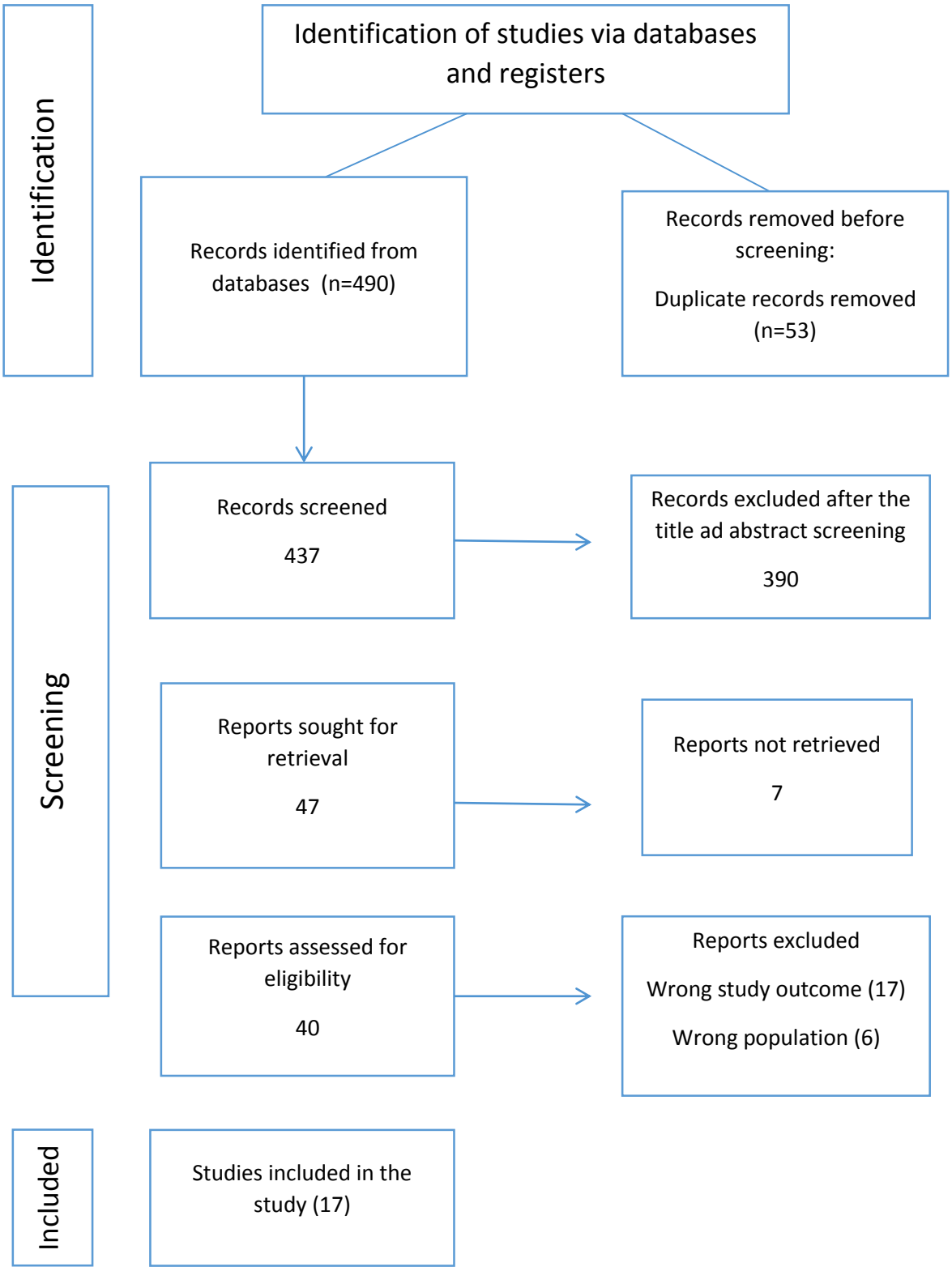

Figure (1): PRISMA flowchart summarizes study selection process.

Characteristics of the research included

(Table 1) The sociodemographic characteristics of the chosen studies are provided. Our findings included 11792 individuals from 17 studies. Eight investigations were undertaken in the United States [9, 13, 14, 15, 18, 19, 24], and one in the United Kingdom [11], one each in Vietnam [12], the Netherlands [16], Italy [20], and Egypt [23]. Four studies [9, 20, 21, 23] were observational in nature, one was cross-sectional [12], and one was a pilot study [13]. Six studies [10, 16, 18, 19, 22, 24] were randomized controlled trials, and two were clinical trials [11, 17].

(Table 2) highlights the clinical aspects of the trials covered. Our first study looked at the demographic and psychosocial differences between homes with and without resident smokers [9], two studies looked at the efficacy of combining urine cotinine level feedback with motivational interviewing (MI) to prevent passive smoking in children at high risk of developing asthma [10, 15], and one looked at passive smoking in children with cystic fibrosis [11]. Two studies were conducted to better understand the factors that influence children's health problems and exposure to secondhand smoke [12,18], another to evaluate the effectiveness of financial incentives for reducing passive smoking in children [13], and another to determine the cause and sequence of consistently elevated cotinine concentrations [14]. A study was conducted to assess the effectiveness of typical preventative children's visits between the ages of one and four years, during which experts systematically assessed environmental tobacco smoke exposure and symptoms similar to asthma [16], in order to explain how ETS influences leukotriene synthesis, gastric reflux, and respiratory infections in children with poor asthma control [17]. A research looked at the effectiveness of an emergency department and home-based environmental management approach in urban children who had frequent emergency room visits owing to asthma [19]. Another research was conducted to identify possibly modifiable risk factors associated with pollution that were the cause of babies being hospitalized with acute bronchiolitis [20], to look at the relationship between tobacco smoke exposure and the risk of re-hospitalization in asthmatic children [21]. The National Heart, Lung, and Blood Institute-sponsored Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents were the subject of a research examining approaches for adoption [22]. A research was conducted to investigate the effect of secondhand smoking on lung functioning in school-aged athletes and non-athletes [23]. The final study looked at how feedback from secondhand smoke exposure encourages quitting in parents of asthmatic children versus parents of normal children, and whether the higher intervention intensity of the enhanced-precaution adoption model (PAM) leads to more cessation than a previously studied intervention [24].

Discussion

Worldwide awareness of the negative consequences of environmental tobacco smoke is growing. On this significant problem, an increasing number of original studies and meta-analyses are being published. In the current study, seventeen studies were included. Children's asthma, middle ear illness, allergy disorders, and lower respiratory infections are only a few of the conditions that seem to be made more likely by passive smoking, especially bacterial infections. Several studies have been conducted revealed that newborns exposed to parental smoking had decreased forced expiratory flows, according to a recent study by Stocks and Dezateux [25]. It is challenging to separate the impact of perinatal and postnatal exposure on pulmonary function throughout infancy, despite several studies showing reduced lung function in neonates subjected passively to tobacco smoke throughout pregnancy. The forced expiratory volume in 1 second and the maximum expiratory flow are now reduced by 0.5% and 2%, respectively, by passive smoking. Smoke exposure during pregnancy can have effects that extend at least until adolescence. In the United States, Mannino et al. discovered a substantial correlation between lower lung function in kids aged 8 to 16 years and increased blood cotinine levels [26, 27]. After adjusting for demographic, parental, and participant factors, A population-based study done in six US cities between 2000 and 2006 found that childhood ETS exposure from two or more smokers, as equated to nothing, is related with early emphysema in old age [28]. Numerous research have shown that parental smoking has a significant influence on newborns' health, as well as children's asthma and wheezing illnesses [29, 30]. ETS exposure has been linked to an increase in wheezing problems and asthmatic symptoms. In comparison to children born to nonsmokers, a higher prevalence of wheeze was reported in a study of 3-year-old infants who were exposed to ETS both prenatally and postnatally (odds ratio [OR], 1.14) [31]. Corresponding to the mothers' smoking rates, motherly smoking enhanced the risk of asthma in a dose-dependent way throughout the first seven years of life [32]. According to one study that looked at the relationship between maternal smoking and respiratory symptoms in school-aged children, smoking by either parent had an OR of 1.21 for asthma, 1.40 for wheeze, and 1.35 for cough [33, 34].

Table (1): Sociodemographic characteristics of the included participants.

Study	Study design	Country	Participants	Age range (months)
Wu et al., 2019 [9]	Observational	USA	157	5 yrs: 12yrs
Hutchinson et al., 2017 [10]	Randomized controlled trial		58 families	0-13 yrs
Smyth et al., 1994 [11]	Clinical trial	UK	108	5-16 yrs
Ngo et al., 2020 [12]	cross-sectional	Vietnam	435	0-6 yrs
Jassal et al., 2021 [13]	pilot trial	USA	135	2-12 yrs
Butz et al., 2016 [14]		USA	116	3-12 yrs
Blaakman et al., 2013 [15]		USA	140	3-10 yrs
Hafkamp-de Groen et al., 2014 [16]	Randomized Trial	Netherlands	7775	1-4 yrs
Lang et al., 2013 [17]	Clinical trial		306	6-17 yrs
Butz et al., 2019 [18]	Randomized controlled trial	USA	222	SD 2.7 yrs
Butz et al., 2020 [19]	Randomized controlled trial	USA	222	
Nenna et al., 2017 [20]	observational	Italy	426	0-3 yrs
Howrylak et al., 2014 [21]	observational		774	1-16 yrs
LaBresh et al., 2014 [22]	Randomized controlled trial			
Elshazly et al., 2020 [23]	observational	Egypt	46	8-15 yrs
Borrelli et al., 2016 [24]	Randomized Trial	USA	560	3-17 yrs

Table (2): Clinical characteristics and outcomes of the included studies.

Study	Outcomes	Duration	Conclusion
Wu et al., 2019 [9]	Urban environments continue to have disturbingly high levels of indoor SHS exposure. But a sizable percentage of this exposure seems to come exclusively from outside sources that penetrate the house. Due to a lack of functional support and physical isolation, carers in these households had a stronger desire but fewer agencies to prevent SHSe.		Public measures that focus on these elements might aid in reducing exposure in this population's particular vulnerability.
Hutchinson et al., 2017 [10]	After 6 and 9 months of the trial, the intervention group's percentage of children who were exposed to PS at home was much lower than that of the control group. After 3 and 12 months of study, there were no discernible differences between the groups.	6 months	These findings imply that an effective technique to reduce PS in children may involve MI in conjunction with feedback regarding the child's urine cotinine levels. For a longer-lasting intervention impact, a programme longer than six months may be required. The study's sample size, dropout rate, and amount of missing values were all limitations.
Smyth et al., 1994 [11]	Cotinine was markedly lesser in the cystic fibrosis group than in the control group. Furthermore, for every 10 cigarettes smoked each day in the home by the parents in this group, the child's forced expiratory volume in one second and forced vital capacity declined by 4% and 3%, respectively.		In our study, urine cotinine did not indicate lung health. In children with cystic fibrosis, reduced FEV1 and FVC were associated with rising household smoking. It should be strongly discouraged for parents of children with cystic fibrosis to smoke.
Ngo et al., 2020 [12]	In comparison to rural areas, urban youngsters were more regularly exposed to it at home and in public. Children of non-government workers were more likely to have come into contact with passive smoking over the previous 7 days compared to kids whose parents were farmers. Additionally, children who lived in a household with strict smoking regulations were less likely to be exposed to passive smoking in the previous 7 days than those who did not.	7 days	According to the results of our study, establishing a no-smoking policy and a smoke-free home can help lessen children's exposure to passive smoking. There are a lot of carers out there who don't know much about passive smoking, especially in rural regions. Urban children are most frequently exposed to passive smoking at home, followed by public places.
Jassal et al., 2021 [13]	The primary end measure was mean change values for monthly pediatric cotinine levels, which were not significantly different in the intervention cohort across a 6-month follow-up period and were assessed on an intention-to-treat basis.	6 months	Financial rewards given to adults who contributed to pediatric TSE did not lower cotinine levels in children.

Butz et al., 2016 [14]	According to our findings, children with a positive 6-month cotinine level had a mean of one PCP visit within the previous 3 months. Our findings also indicate that depending on self-report of SHS exposure will lead to the incorrect classification of about one-third of kids as not having been exposed.	12 months	According to the study, consecutive salivary cotinine measurement lowers the misclassification of asthmatic children who were allegedly not exposed to SHS.
Blaakman et al., 2013 [15]	79% of PCGs completed the entire intervention, compared to only 17% of other smoking carers. Nearly all (98%) PCGs felt the programme may be useful to others and were pleased with the care the study nurses provided.		Delivery of the intervention was enabled through community collaborations, meticulous tracking, perseverance, flexibility, and acceptance. High engagement rates and satisfaction ratings show those nurses' nonjudgmental demeanour helped carers feel comfortable discussing their opinions and thinking about changing their behavior.
Hafkamp-de Groen et al., 2014 [16]	In an exploratory per-protocol analysis, children whose parents were interviewed using the brief assessment form at the intervention well-child centers had a lower risk of being exposed to ETS at home than children who visited the control well-child centers.		The prevalence of physician-diagnosed asthma and wheezing was not decreased by systematic assessment and counselling of asthma-like symptoms and ETS exposure in early childhood by well-child care professionals using a brief assessment form, and FeNO, Rint, or HRQOL were not improved at age 6 years.
Lang et al., 2013 [17]	ETS from any context was related with an increase in symptomatic respiratory infections. Children with domestic indoor exposure had worse asthma control and a trend for increased mean urine leukotriene E4.	6 months	Exposure to domestic smoking was linked to greater rates of symptomatic respiratory illness and worse asthma control. Children with asthma who have poor asthma control frequently experience ETS exposure, which may worsen asthma control by encouraging respiratory infections.
Butz et al., 2019 [18]	SHS exposure had no effect on the frequency of the symptoms. Children with asthma may be more likely to experience worsened daytime and nocturnal symptoms in the autumn due to high carer daily life stress	12 months	In children with poorly managed asthma, careful monitoring of symptoms and medication use during the autumn months, as well as interventions to reduce carer stress, may reduce asthma morbidity.
Butz et al., 2020 [19]	Children who tested positive for allergic sensitizations, were younger, used controller medications more frequently, were randomly assigned to the CON group, and had no exposure to SHS had a higher risk of returning to the ED within a year.	12 months	Children with poorly managed asthma and SHS exposure did not have a reduction in asthma ED revisits after receiving home-based EC treatments. Young age, increasing controller drug use, and allergic sensitization were significant predictors of asthma ED visits.

Nenna et al., 2017 [20]	The questionnaire indicated the following risk factors for acute bronchiolitis: nursing for less than three months, the presence of older siblings, having more than four roommates, and cooking with seed oil. Renovations made to the home within the last year and concurrent daily smoking exposure involving more than 11 cigarettes and two or more smoking roommates were more prevalent factors in cases than in controls.		Inhaling cooking oil vapors is one of the risk factors for acute bronchiolitis linked to both indoor and outdoor pollution.
Howrylak et al., 2014 [21]	Having measurable serum or salivary cotinine was related with a higher risk of readmission, whereas caregiver reports of any cigarette exposure were not. 39.1% of kids whose parents said they had never used tobacco had detectable serum cotinine, and 69.9% had detectable salivary cotinine. 87.6% of the kids with reported exposure had serum cotinine that could be detected, and 97.7% of them had salivary cotinine that could be detected.	1 year	Having measurable serum or salivary cotinine was related with a higher risk of readmission, whereas caregiver reports of any cigarette exposure were not. 39.1% of kids whose parents said they had never used tobacco had detectable serum cotinine, and 69.9% had detectable salivary cotinine. 87.6% of the kids with reported exposure had serum cotinine that could be detected, and 97.7% of them had salivary cotinine that could be detected.
LaBresh et al., 2014 [22]	The study includes a number of Care patterns within a practice may differ if MOC participation determines clinician degrees of participation in the intervention. Third, only particular components of the recommendation were the emphasis.	1 year	The NHLBI Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents will be used in 32 practices as part of the cluster-randomized Young Hearts Strong Starts pediatric practice-based quality improvement and guideline adoption intervention, which will evaluate the effectiveness of a multifaceted approach to changing care systems.
Elshazly et al., 2020 [23]	The results showed that the study groups' forced vital capacity, forced expiratory volume in 1 second, and peak expiratory flow increased significantly, but only in the non-exposed children. However, there were no statistically significant differences between football players and bikers or between youngsters exposed to passive smoking and those who were not ($p>0.05$).	3 months	The findings of this study point to the effects of exercise on lung health without distinguishing between the effects of football players and bikers.
Borrelli et al., 2016 [24]	Aim 1: The 30-day and 7-day ppa at 2 months (the primary endpoint) were more than twice as likely to be achieved by parents of asthmatic children, and they also had non-detectable levels of SHSe compared to HCs. Stronger TM effects were produced by higher treatment intensity. Aim 2: In comparison to PAM, enhanced-PAM was more likely to accomplish the primary goal of 30-day ppa, 4-months, and improved asthma outcomes.	12 months	Parents of asthmatic children seem to be more motivated to stop smoking than parents of healthy children by the smoking cessation intervention (Motivational Interviewing plus biomarker feedback). Parents of children with asthma who receive more intensive assistance are more likely to give up smoking and experience better asthma outcomes.

Strachan and Cook found a relationship between maternal smoking and an increase in lower respiratory infection (LRI) in a review. ETS exposure increased the chance of bronchiolitis hospitalization by up to thrice. LRI had an OR of 1.69 when compared to neither parent smoking [35]. It is known that exposure to ETS is linked to a higher incidence of upper respiratory tract infections. Additionally, a few of studies have linked parental smoking to an uptick in infections of the lower and upper respiratory tracts (LRTI) [36, 37]. Gürkan et al. observed that when either the mother or both parents smoked, children with respiratory syncytial virus bronchiolitis had greater cotinine levels than children with non-smoking parents [38]. This evidence shows that extended cigarette smoke exposure may raise the risk of acute respiratory disease. Passive smoking can aggravate airway irritation and lead to the development of respiratory diseases in children who already have LRTI [36]. According to a meta-analysis, home ETS exposure considerably raises the incidence of LRI, particularly bronchiolitis, in neonates. Smoking by either parent or other family members significantly increased the risk of LRI [39]. The precise mechanism by which ETS may contribute to these infections is unknown, but it may involve immune system regulation or suppression, increased bacterial adhesion factors, or a combination of these factors, disruption of the respiratory tract's mucociliary system, or even increased toxicity of some low-level toxins that are difficult to detect using standard techniques [37].

Conclusion

ETS exposure has a significant deleterious influence on the mortality and morbidity of youngsters. Adequate data has demonstrated a significant relationship involving ETS and pediatric health difficulties such as infections, respiratory diseases, and neurobehavioral abnormalities. These deleterious consequences of ETS exposure should concern doctors. Children are exposed to secondhand smoke in places other than their homes, such as public places. To protect kids from ETS, more efficient government regulations and programs should be consolidated.

Conflict of Interest

None

Funding

None

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