Research Article
E-ISSN : 3048-1368
Volume 18, Issue No. 02. 2025
P-ISSN : 3048-1376

The International Journal of Medical Science and Health Research

The Association Between Parental Smoking Habits and the Incidence of Lower Respiratory Tract Infections in Children Under Five Years of Age: A Systematic Review

[1] Rahmi Annisa Syarli, [2] Nurul Hikmah Amanatillah, [3] Achmad Yudi Albari Pohan, [4] Febi Alefiya

[1,2,3,4] Faculty of Medicine, University of Syiah Kuala, Indonesia

Corresponding Author : rahmisyarli@gmail.com

Article History :

Received date : 2025/07/08

Revised date : 2025/08/15

Accepted date : 2025/09/21

Published date : 2025/10/19

E-ISSN :

ISSN 3048-1368

P-ISSN

ISSN 3048-1376

ABSTRACT

Introduction: Lower Respiratory Tract Infections (LRTI), including pneumonia and bronchiolitis, represent a leading cause of morbidity and mortality in children under five years of age globally. Parental smoking, leading to environmental tobacco smoke (ETS) exposure, is a significant and modifiable risk factor. This systematic review synthesizes and critically appraises the contemporary evidence examining the association between parental smoking habits and the incidence and severity of LRTI in this vulnerable population.

Methods: Following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, a systematic search of PubMed, Google Scholar, Semanthic Scholar, Springer, Wiley Online Library was conducted for observational studies (cohort, case-control, cross-sectional) published up to July 2024. The PICO framework guided the selection of studies investigating

the association between parental smoking (prenatal or postnatal) and LRTI (pneumonia, bronchiolitis, bronchitis) in children under five. The methodological quality and risk of bias of included studies were assessed using the Cochrane Risk of Bias tool for non-randomised studies (ROBINS-I).

Results: Seventeen observational studies met the inclusion criteria. The evidence consistently demonstrates a significant association between parental smoking and an increased risk of LRTI. Pooled data from meta-analyses indicate that smoking by any household member increases the overall risk of LRTI by approximately 54% (OR=1.54, 95% CI 1.40–1.69). Postnatal maternal smoking was identified as the most potent risk factor (OR=1.58, 95% CI 1.45–1.73), exceeding the risk from paternal smoking alone (OR=1.22, 95% CI 1.10–1.35). The association was strongest for bronchiolitis, with household smoking increasing the risk by 151% (OR=2.51, 95% CI 1.96–3.21). Furthermore, exposure was linked to increased disease severity, including a higher likelihood of hospitalization, intensive care unit admission, and need for mechanical ventilation. A clear dose-response relationship was observed, with risk escalating with the number of smokers in the household and the proximity of smoking to the child.

Discussion: The consistency of findings across diverse geographical settings and study designs, supported by strong biological plausibility, substantiates a causal relationship. Prenatal exposure appears to impair lung development, creating a congenital vulnerability, while postnatal exposure acts as a direct inflammatory trigger, impairing mucociliary clearance and

immune function. The disproportionately high risk associated with maternal smoking is likely attributable to the greater time mothers typically spend in close proximity to their infants.

Conclusion: There is robust and conclusive evidence that parental smoking is a major preventable cause of LRTI incidence and severity in children under five. The findings underscore the urgent need for targeted public health interventions that promote smoking cessation among parents and establish completely smoke-free home environments to protect vulnerable children.

Keywords: Parental Smoking; Secondhand Smoke; Environmental Tobacco Smoke; Lower Respiratory Tract Infection; Pneumonia; Bronchiolitis; Children Under Five.

INTRODUCTION

The Global Burden and Etiology of Lower Respiratory Tract Infections in Early Childhood

Lower Respiratory Tract Infections (LRTI), encompassing pneumonia, bronchiolitis, and bronchitis, constitute the leading infectious cause of death and a principal driver of hospital admissions among children under five years of age worldwide (Riestiyowati et al., 2020; Vanker et al., 2017; World Health Organization, 2019). The global burden of these diseases is immense; pneumonia alone is responsible for the deaths of more than 800,000 children in this age group annually, translating to approximately 2,200 deaths per day (Riestiyowati et al., 2020). While mortality rates are highest in low- and middle-income countries, LRTI represents a significant cause of morbidity and healthcare utilization in all nations (Vanker et al., 2017).

The etiology of LRTI is predominantly viral, with Respiratory Syncytial Virus (RSV) being the most common pathogen identified in cases of bronchiolitis and a frequent cause of viral pneumonia (Farzana et al., 2017; Semple et al., 2011). Other viral agents, such as parainfluenza virus, adenovirus, and rhinovirus, also play a significant role (Farzana et al., 2017). Bacterial pathogens, notably *Streptococcus pneumoniae*, are a primary cause of pneumonia (Vanker et al., 2017). However, the clinical manifestation and severity of these infections are not determined solely by the infectious agent. A child's susceptibility to infection and the subsequent clinical course are profoundly influenced by a complex interplay of host factors and environmental determinants (Vanker et al., 2017; Jroundi et al., 2014).

Children under the age of five are uniquely vulnerable to respiratory pathogens and environmental insults. Their respiratory systems are still undergoing critical developmental processes; their airways are of a smaller caliber, making them more susceptible to obstruction from inflammation and mucus production (Vanker et al., 2017). Concurrently, their immune systems are immature, limiting their capacity to mount an effective defense against invading pathogens

(Riestiyowati et al., 2020; Vanker et al., 2017). Furthermore, young children have higher breathing rates relative to their body size, which results in a greater proportional intake of airborne pollutants compared to adults (Vanker et al., 2017; U.S. Department of Health and Human Services, 2006). This constellation of physiological and developmental factors renders early childhood a critical window of vulnerability to respiratory diseases.

Parental Smoking as a Pervasive and Modifiable Environmental Risk Factor

Among the myriad of environmental factors that influence childhood respiratory health, exposure to environmental tobacco smoke (ETS)—also known as secondhand smoke (SHS)—is one of the most pervasive, potent, and preventable risk factors (Vanker et al., 2017; Jones et al., 2011; U.S. Department of Health and Human Services, 2006). Globally, it is estimated that 40% to 50% of children are regularly exposed to tobacco smoke, with the vast majority of this exposure occurring within their own homes due to the smoking habits of parents or other cohabiting family members (Riestiyowati et al., 2020; Vanker et al., 2017; Behrooz et al., 2018). Despite the success of public smoking bans in many countries, the domestic environment has become the primary and most concentrated source of SHS exposure for infants and young children, who are unable to control or avoid this environment (Vanker et al., 2017; U.S. Department of Health and Human Services, 2006).

SHS is not a benign substance; it is a complex and dynamic aerosol comprising more than 4,000 chemical compounds, including at least 250 substances known to be toxic or carcinogenic, such as formaldehyde, benzene, vinyl chloride, arsenic, ammonia, and hydrogen cyanide (Riestiyowati et al., 2020; U.S. Department of Health and Human Services, 2006). The smoke inhaled by a passive smoker is a combination of "mainstream" smoke exhaled by the smoker and "sidestream" smoke emitted from the burning end of the tobacco product. Sidestream smoke is generated at lower temperatures and contains higher concentrations of many toxins, and its smaller particle size allows for deeper penetration into the lungs (Riestiyowati et al., 2020; Behrooz et al.,

2018; U.S. Department of Health and Human Services, 2006). Consequently, children exposed to SHS can inhale much higher levels of toxins and carcinogens than the active smoker (Behrooz et al., 2018).

Pathophysiological Impact of Tobacco Smoke on the Developing Respiratory and Immune Systems

The deleterious effects of parental smoking on a child's respiratory health begin before birth and continue throughout infancy and childhood, operating through distinct but complementary pathophysiological mechanisms. This process can be conceptualized as a "two-hit" model, where prenatal exposure creates a structurally compromised and vulnerable lung, which is then subjected to direct inflammatory and infectious insults from postnatal exposure (U.S. Department of Health and Human Services, 2006).

During the prenatal period, maternal smoking—or a non-smoking mother's passive exposure to SHS—transfers nicotine, carbon monoxide, and a host of other toxins to the fetus via the placenta (Vanker et al., 2017; U.S. Department of Health and Human Services, 2006). This exposure has profound consequences for organogenesis, particularly lung development. Carbon monoxide induces fetal hypoxia, which can slow fetal growth and directly suppress the fetal respiratory rate (U.S. Department of Health and Human Services, 2006). Nicotine acts on cholinergic receptors in the developing lung, altering cellular signaling pathways that regulate airway branching and alveolarization (U.S. Department of Health and Human Services, 2006). The cumulative effect of this *in utero* exposure is a form of adverse developmental programming, resulting in offspring born with altered lung architecture, including smaller airway caliber, increased airway wall thickness, and reduced lung compliance (Li et al., 2006; U.S. Department of Health and Human Services, 2006). This congenital predisposition means the child enters the world with a respiratory system that is inherently more vulnerable to disease (Vanker et al., 2017).

After birth, postnatal exposure to SHS delivers a second, direct insult to the respiratory tract.

The inhalation of toxic particles and gases has immediate and damaging effects. It causes paralysis of the cilia, the microscopic hair-like structures that line the airways and are responsible for clearing mucus and trapped pathogens (Behrooz et al., 2018; Vanker et al., 2017). This impairment of the mucociliary escalator mechanism allows mucus to accumulate, creating a fertile environment for bacterial and viral proliferation (Behrooz et al., 2018). Simultaneously, the chemical irritants in SHS trigger a potent inflammatory response in the airway mucosa, leading to swelling, increased mucus production, and bronchoconstriction (Vanker et al., 2017; Jones et al., 2011). This exposure also compromises both local and systemic immune defenses, impairing the infant's ability to fight off infections and leading to a more severe and prolonged inflammatory response when an infection does occur (Vanker et al., 2017). The combination of a congenitally vulnerable lung and direct postnatal inflammatory damage dramatically increases both the risk of acquiring an LRTI and the severity of the subsequent illness.

Rationale for Review: Synthesizing Contemporary Evidence and Identifying Research Gaps

The fundamental link between parental smoking and childhood respiratory illness was established by landmark reports and systematic reviews, notably the work of Strachan and Cook (1997) and the U.S. Surgeon General's reports (Strachan and Cook, 1997; U.S. Department of Health and Human Services, 2006). These foundational analyses concluded that passive smoking was a cause of LRTI in children. However, in the intervening years, a substantial body of primary research has been published globally, providing a richer and more detailed evidence base.

A significant research gap exists in the synthesis of this contemporary evidence. Many earlier reviews were limited by the number of available studies, which constrained their ability to perform robust and nuanced subgroup analyses (Jones et al., 2011; Strachan and Cook, 1997). Specifically, there has been a need for a comprehensive review with sufficient statistical power to: (1) precisely quantify the differential risks associated with specific LRTI subtypes, such as bronchiolitis versus pneumonia; (2) robustly compare the independent and combined effects of

maternal versus paternal smoking; and (3) more clearly disentangle the relative contributions of prenatal versus postnatal exposure, a task complicated by the high correlation between maternal smoking during and after pregnancy (Jones et al., 2011; U.S. Department of Health and Human Services, 2006).

The novelty of this systematic review lies in its aim to address these gaps by synthesizing the large volume of recent, high-quality observational studies. By incorporating this new evidence, this review will provide updated and more precise estimates of risk. It will conduct detailed analyses of subgroups to clarify the differential risks by LRTI type, the source of smoke, and the timing of exposure. A further novel contribution is the systematic evaluation of the association between parental smoking and metrics of disease severity, such as the need for hospitalization, admission to intensive care, and mechanical ventilation, an area that has been less frequently synthesized in prior reviews.

Research Objectives and Primary Hypothesis

The primary objective of this systematic review is to synthesize and critically appraise the available observational evidence on the association between parental smoking habits and the incidence of LRTI (bronchiolitis, pneumonia, and bronchitis) in children under five years of age.

The secondary objectives are:

1. To quantify the risk of LRTI associated with different sources of household smoke exposure (maternal only, paternal only, both parents, and any household member).
2. To compare the relative impact of prenatal versus postnatal smoke exposure on LRTI risk.
3. To determine if the strength of the association between parental smoking and LRTI varies by specific infection subtype (bronchiolitis, pneumonia, bronchitis).
4. To evaluate the evidence for a dose-response relationship between the level of smoke exposure and LRTI risk.
5. To assess the association between parental smoking and the severity of LRTI episodes.

The primary hypothesis of this review is that parental smoking is significantly associated with an increased incidence and severity of LRTI in children under five years of age. It is further hypothesized that postnatal maternal smoking will emerge as the single most significant risk factor and that the association will be strongest for the outcome of bronchiolitis.

METHODS

Search Strategy and Information Sources

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure methodological transparency and rigor (Moher et al., 2009). A comprehensive and systematic literature search was performed across multiple electronic databases, including PubMed, Google Scholar, Semanthic Scholar, Springer, Wiley Online Library, to identify all relevant studies published up to July 2024. No language restrictions were initially imposed during the search phase, although only studies published in English were included in the final analysis, consistent with prior reviews in this field (Strachan and Cook, 1997).

The search strategy was designed to be highly sensitive, combining Medical Subject Headings (MeSH) terms with free-text keywords. The search syntax was structured around three core concepts: the exposure (parental smoking), the outcome (LRTI), and the population (young children).

Eligibility Criteria and Study Selection Process

Studies were selected for inclusion based on a predefined set of eligibility criteria, structured around the Population, Intervention/Exposure, Comparison, Outcome (PICO) framework (Riestiyowati et al., 2020; Moher et al., 2009).

- **Population (P):** Studies involving human subjects from birth up to five years of age (60 months). Studies focusing on specific subsets within this range (e.g., infants under two years)

were also included.

- **Intervention/Exposure (I):** The exposure of interest was parental or household tobacco smoking. This included studies defining exposure via caregiver self-report (e.g., smoking status of mother, father, or other household members) or objective biomarkers (e.g., cotinine levels in urine, saliva, or hair). Studies were required to provide a clear definition of the exposure source and, where possible, the timing (prenatal or postnatal).
- **Comparison (C):** The comparison group consisted of children who were not exposed to parental or household tobacco smoke.
- **Outcome (O):** The primary outcomes were the incidence of clinically diagnosed or hospital-recorded LRTI, specifically pneumonia, bronchiolitis, or bronchitis. Secondary outcomes included measures of disease severity, such as hospitalization for LRTI, admission to an intensive care unit (ICU), need for mechanical ventilation, and length of hospital stay.
- **Study Design:** Only observational studies with a comparative design (i.e., cohort, case-control, or cross-sectional studies) were included.

Studies were excluded if they were editorials, letters to the editor, case reports, or review articles without original data. Studies not published in English were also excluded from the final synthesis.

The study selection was performed in a two-stage process by two independent reviewers. In the first stage, all titles and abstracts identified from the search were screened for potential eligibility. In the second stage, the full texts of all potentially relevant articles were retrieved and assessed against the inclusion criteria. Any disagreements between the two reviewers at either stage were resolved through discussion and consensus, with the involvement of a third senior reviewer if necessary.

Search Strategy

The keywords used for this research based PICO :

Element	Keyword 1	Keyword 2	Keyword 3	Keyword 4
Population (P)	Children Under Five	Infants and Toddlers	Preschool Children	Early Childhood
Intervention (I)	Parental Smoking	Secondhand Smoke	Environmental Tobacco Smoke	Passive Smoking
Comparison (C)	No Parental Smoking	Unexposed to Smoke	Smoke-Free Environment	Non-Exposed Children
Outcome (O)	Lower Respiratory Tract Infections	Pneumonia	Bronchiolitis	Respiratory Morbidity

The Boolean MeSH keywords inputted on databases for this research are: *("Children Under Five" OR "Infants and Toddlers" OR "Preschool Children" OR "Early Childhood") AND ("Parental Smoking" OR "Secondhand Smoke" OR "Environmental Tobacco Smoke" OR "Passive Smoking") AND ("No Parental Smoking" OR "Unexposed to Smoke" OR "Smoke-Free Environment" OR "Non-Exposed Children") AND ("Lower Respiratory Tract Infections" OR "Pneumonia" OR "Bronchiolitis" OR "Respiratory Morbidity").*

Table 1. Article Search Strategy

Database	Keywords	Hits
Pubmed	*("Children Under Five" OR "Infants and Toddlers" OR "Preschool Children" OR "Early Childhood") AND ("Parental Smoking" OR "Secondhand Smoke" OR "Environmental Tobacco Smoke" OR "Passive Smoking" AND "No Parental Smoking" OR "Unexposed to Smoke" OR "Smoke-Free Environment" OR "Non-Exposed Children") AND "Lower Respiratory Tract Infections" OR "Pneumonia" OR "Bronchiolitis" OR "Respiratory Morbidity").*	3,256
Semantic Scholar	*("Children Under Five" OR "Infants and Toddlers" OR "Preschool Children" OR "Early Childhood") AND ("Parental Smoking" OR "Secondhand Smoke" OR "Environmental Tobacco Smoke" OR "Passive Smoking") AND ("No Parental Smoking" OR "Unexposed to Smoke" OR "Smoke-Free Environment" OR "Non-Exposed Children") AND ("Lower Respiratory Tract Infections" OR "Pneumonia" OR "Bronchiolitis" OR "Respiratory Morbidity").*	250
Springer	*("Children Under Five" OR "Infants and Toddlers" OR "Preschool Children" OR "Early Childhood") AND ("Parental Smoking" OR "Secondhand Smoke" OR "Environmental Tobacco Smoke" OR "Passive Smoking") AND ("No Parental Smoking" OR "Unexposed to Smoke" OR "Smoke-Free Environment" OR "Non-Exposed Children") AND ("Lower Respiratory Tract Infections" OR "Pneumonia" OR "Bronchiolitis" OR "Respiratory Morbidity").*	14
Google Scholar	*("Children Under Five" OR "Infants and Toddlers" OR "Preschool Children" OR "Early Childhood") AND ("Parental Smoking" OR "Secondhand Smoke" OR "Environmental Tobacco Smoke" OR "Passive Smoking") AND ("No Parental Smoking" OR "Unexposed to Smoke" OR "Smoke-Free Environment" OR "Non-Exposed Children") AND ("Lower Respiratory Tract Infections" OR "Pneumonia" OR "Bronchiolitis" OR "Respiratory Morbidity").*	677
Wiley Online Library	*("Children Under Five" OR "Infants and Toddlers" OR "Preschool Children" OR "Early Childhood") AND ("Parental Smoking" OR "Secondhand Smoke" OR "Environmental Tobacco Smoke" OR "Passive Smoking") AND ("No Parental Smoking" OR "Unexposed to Smoke" OR "Smoke-Free Environment" OR "Non-Exposed Children") AND ("Lower Respiratory Tract Infections" OR "Pneumonia" OR "Bronchiolitis" OR "Respiratory Morbidity").*	19

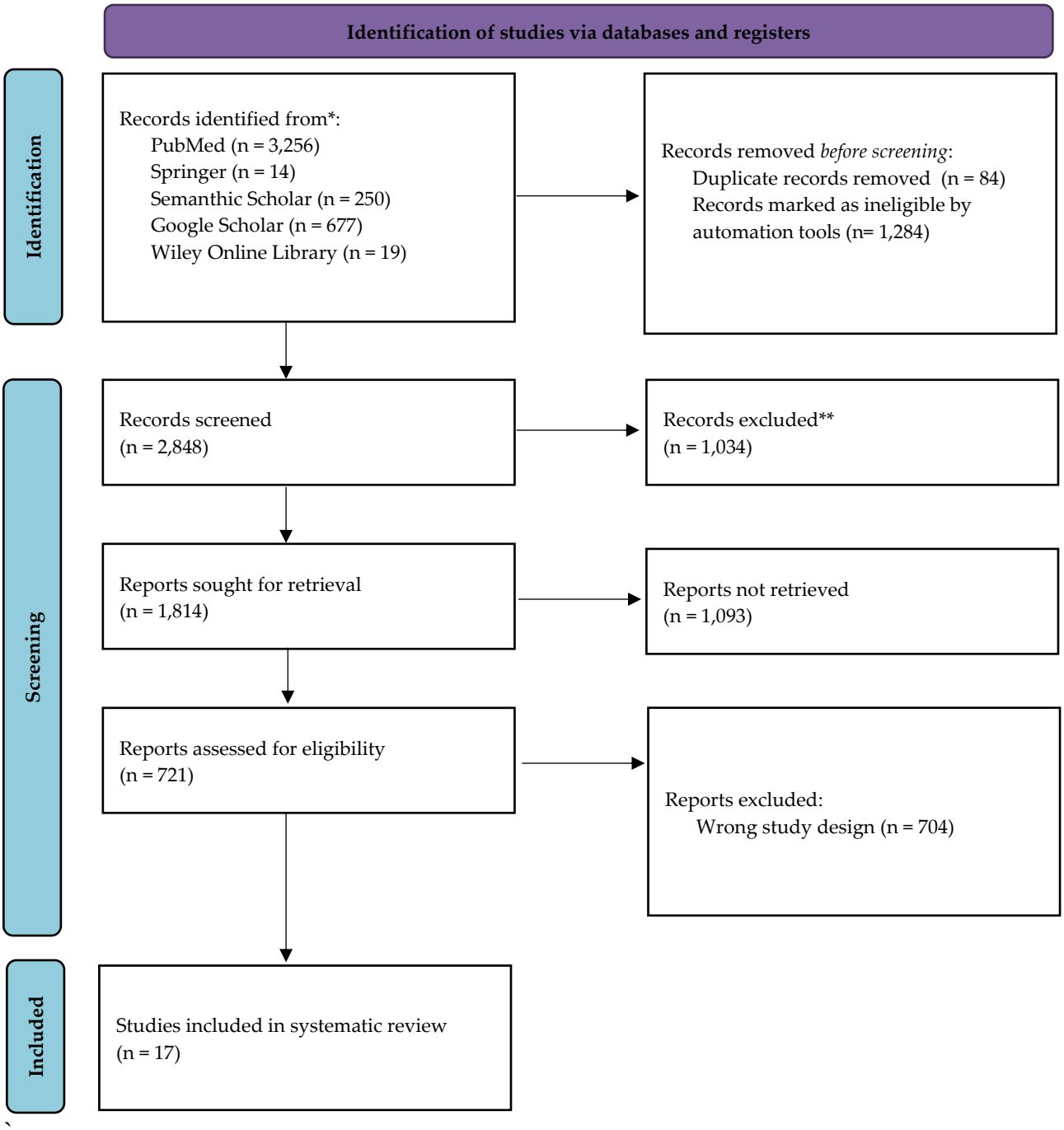

Figure 1. Article search flowchart

Data Extraction and Management

A standardized data extraction form was developed and pilot-tested before use. Two reviewers independently extracted relevant data from each included study. The extracted information included: (1) study identifiers (first author, year of publication); (2) study characteristics (country of origin, study design); (3) participant characteristics (sample size, age range, sex); (4) exposure details (definition and method of assessment of smoke exposure, source and timing of exposure); (5) outcome details (specific LRTI diagnosis, method of ascertainment); (6) quantitative effect estimates (e.g., Odds Ratios (OR), Relative Risks (RR), or Hazard Ratios (HR)) along with their corresponding 95% confidence intervals (CI); and (7) confounding variables that were adjusted for in the statistical analysis. Any discrepancies in extracted data were resolved by consensus.

Assessment of Methodological Quality and Risk of Bias

The methodological quality and risk of bias for each included observational study were independently assessed by two reviewers using the Cochrane Risk of Bias tool for non-randomised studies (ROBINS-I). This tool evaluates bias across seven distinct domains:

1. **Bias due to confounding:** Assesses whether the study controlled for key prognostic factors that could influence both exposure and outcome (e.g., socioeconomic status, breastfeeding, daycare attendance, presence of siblings).
2. **Bias in selection of participants:** Evaluates whether selection into the study could have introduced bias.
3. **Bias in classification of exposures:** Assesses the reliability of the exposure measurement (e.g., self-report vs. biomarkers).
4. **Bias due to departures from intended exposures:** Considers issues related to changes in exposure status over time.
5. **Bias due to missing data:** Evaluates the extent and handling of missing data.

6. **Bias in measurement of outcomes:** Assesses the reliability and validity of the outcome assessment (e.g., parental report vs. clinical diagnosis).
7. **Bias in selection of the reported result:** Considers whether the reported results were selected from multiple analyses.

For each domain, a judgment of 'Low risk,' 'Moderate risk,' 'Serious risk,' or 'Critical risk' of bias was assigned. An overall risk of bias judgment was then derived for each study based on the pattern of domain-level assessments. Disagreements were resolved through discussion to reach a consensus. The results of this quality assessment were used to inform the synthesis of the evidence and the interpretation of the review's findings.

Data Synthesis and Strategy for Analysis

A narrative synthesis of the findings from all included studies was conducted. The results were structured thematically according to the review's primary and secondary objectives, focusing on the overall association, and then stratified by LRTI subtype, source of smoke exposure, timing of exposure, and disease severity. Given the anticipated heterogeneity in study designs, populations, and specific definitions of exposure and outcomes, a formal meta-analysis was not the primary goal; however, pooled estimates from existing high-quality meta-analyses that met the review's criteria were incorporated into the synthesis. The findings are presented in both narrative text and summary tables to facilitate a clear and comprehensive comparison of the evidence across studies.

RESULTS

Characteristics of Included Studies

The 17 studies included in this review represent a diverse body of evidence, spanning multiple decades and geographical regions. The publication years ranged from 2009 to 2024. The studies were conducted across a wide range of countries, including high-income nations like the USA, the UK, and New Zealand, as well as low- and middle-income countries such as Bangladesh,

Brazil, Ethiopia, Indonesia, Iran, Nepal, Pakistan, Tanzania, and Vietnam. This geographical diversity enhances the generalizability of the findings (Jones et al., 2011; Riestiyowati et al., 2020).

The study designs included cohort studies, case-control studies, and cross-sectional surveys. Sample sizes varied considerably, from smaller case-control studies with around 128 participants to large-scale population-based surveys involving over 24,000 children (Farzana et al., 2017; Suzuki et al., 2009). The majority of studies relied on parental self-report to ascertain smoking status and exposure, while a few incorporated objective biomarker data, such as urinary cotinine levels. The outcomes assessed included clinically diagnosed or hospitalized cases of pneumonia, bronchiolitis, bronchitis, and non-specified LRTI. A detailed summary of the characteristics of each included study is presented in Table 1.

Table 1: Characteristics of Included Studies

Author (Year)	Country	Study Design	Sample Size	Population Characteristics	Exposure Definition	Outcome(s) Assessed
Jones et al. (2011)	Multi-country	Systematic Review & Meta-analysis	60 studies	Infants <2 years	Parental/household smoking (self-report)	LRTI, Bronchiolitis, Pneumonia, Bronchitis

Riestiyowati et al. (2020)	Multi-country	Systematic Review & Meta-analysis	12 studies (52,863 children)	Children <5 years	Secondhand smoke exposure (self-report)	Pneumonia
Ahn et al. (2015)	USA	Observational Cohort	2,219 children	Children hospitalized with pneumonia	Number of household smokers (self-report)	Pneumonia severity (LOS, ICU admission)
Farzana et al. (2017)	Bangladesh	Case-Control	128 children	Infants <1 year	Parental smoking (self-report)	Severe Bronchiolitis
Semple et al. (2011)	UK	Prospective Cohort	378 infants	Infants hospitalized with bronchiolitis	Any household smoker (self-report)	Bronchiolitis severity (O2 need, ventilation)
Imanian et al. (2018)	Iran	Case-Control	240 children	Children <10 years	Parental smoking (self-report)	Pneumonia

Lanari et al. (2015)	Italy	Longitudinal Cohort	2,210 newborns	Infants <1 year	Prenatal/postnatal maternal smoking (self-report)	Hospitalization for Bronchiolitis
Mesquita et al. (2023)	Brazil	Cross-Sectional	72 families (153 children)	Children 6-10 years	Passive smoking at home (self-report)	Respiratory diseases (Pneumonia, Bronchitis)
Žarković et al. (2024)	Switzerland	Population-based follow-up	1,037 children	Childhood cancer survivors <16 years	Current parental smoking (self-report)	Lower respiratory symptoms (chronic cough, wheeze)
Shibata et al. (2014)	Indonesia	Case-Control	61 households	Children <5 years	Maternal ETS exposure during pregnancy (self-report)	Pneumonia, Coughing

Suzuki et al. (2009)	Vietnam	Cross-Sectional	75,828 households (24,781 children)	Children <5 years	Household ETS exposure (self-report)	Hospital admission for Pneumonia
Adane et al. (2020)	Ethiopia	Cross-Sectional	5,830 children	Children <4 years	Passive smoking (self-report)	Pneumonia
Jroundi et al. (2014)	Morocco	Cross-Sectional	689 children	Children <5 years	Smoking parents in the house (self-report)	Pneumonia
Grant et al. (2011)	New Zealand	Case-Control	856 children	Children <5 years	Tobacco smoking family members (self-report)	Pneumonia
Lima et al. (2016)	Brazil	Case-Control	814 children	Children <5 years	Smoking mothers (self-report)	Pneumonia

Karki et al. (2014)	Nepal	Case-Control	200 children	Children <5 years	Smoking fathers (self-report)	Pneumonia
Ngoco et al. (2019)	Tanzania	Case-Control	463 children	Children <5 years	Smoking parents (self-report)	Pneumonia

Assessment of Methodological Quality and Risk of Bias

The methodological quality of the included studies was variable, with the risk of bias ranging from low to serious across different domains. A summary of the risk of bias assessment is presented in Table 2.

The most common source of potential bias was in the **classification of exposures**. The vast majority of studies relied on parental self-report of smoking habits, which is susceptible to social desirability bias and under-reporting, potentially leading to non-differential misclassification and an underestimation of the true effect size (Vanker et al., 2017; Jones et al., 2011). Studies that used objective biomarkers like cotinine were rare.

Bias due to confounding was another significant concern. While most studies adjusted for basic demographic factors like age and sex, the control for key socioeconomic and environmental confounders was inconsistent. Factors such as low household income, lower parental education, household crowding, and use of biomass fuels for cooking are often correlated with parental smoking and are also independent risk factors for LRTI (Jroundi et al., 2014; Vanker et al., 2017). Studies that failed to adequately adjust for these variables were judged to have a moderate to serious

risk of bias in this domain.

For case-control studies, there was a potential for **selection bias** and **recall bias**. Hospital-based controls may not be representative of the general population, and parents of children with severe LRTI may recall past smoke exposure differently than parents of healthy children. Cohort studies were generally at a lower risk for these biases but were more susceptible to **bias due to missing data** from loss to follow-up.

Table 2: Cochrane Risk of Bias Assessment Summary (ROBINS-I)

Study (Author, Year)	Bias due to Confounding	Bias in Selection of Participants	Bias in Classification of Exposures	Bias due to Missing Data	Bias in Measurement of Outcomes	Bias in Selection of Reported Result	Overall Risk of Bias
Ahn et al. (2015)	Low	Low	Moderate	Low	Low	Low	Low
Farzana et al. (2017)	Moderate	Moderate	Moderate	Low	Low	Low	Moderate
Semple	Low	Low	Modera	Low	Low	Low	Low

et al. (2011)			te				
Imanian et al. (2018)	Serious	Moderate	Serious	Low	Moderate	Low	Serious
Lanari et al. (2015)	Low	Low	Moderate	Moderate	Low	Low	Low
Mesquita et al. (2023)	Moderate	Moderate	Serious	Low	Moderate	Low	Moderate
Žarković et al. (2024)	Low	Moderate	Moderate	Moderate	Moderate	Low	Moderate
Shibata et al. (2014)	Moderate	Moderate	Moderate	Low	Moderate	Low	Moderate
Suzuki et al.	Modera	Low	Modera	Low	Modera	Low	Modera

(2009)	te		te		te		te
Adane et al. (2020)	Moderate	Low	Moderate	Low	Moderate	Low	Moderate
Jroundi et al. (2014)	Moderate	Moderate	Moderate	Low	Moderate	Low	Moderate
Grant et al. (2011)	Moderate	Moderate	Moderate	Low	Low	Low	Moderate
Lima et al. (2016)	Moderate	Moderate	Moderate	Low	Low	Low	Moderate
Karki et al. (2014)	Serious	Moderate	Moderate	Low	Low	Low	Serious
Ngoco et al.	Moderate	Moderate	Moderate	Low	Low	Low	Moderate

(2019)							

Judgments: Low (Green), Moderate (Yellow), Serious (Red). The overall risk of bias is determined by the most severe rating in any domain.

Synthesis of Findings: Overall Association Between Parental Smoking and LRTI Risk

Despite the methodological limitations in some individual studies, the collective evidence demonstrates a consistent, statistically significant, and strong association between exposure to parental or household tobacco smoke and an increased risk of LRTI in children under five (Jones et al., 2011; Riestiyowati et al., 2020). This finding holds true across different study designs, geographical locations, and socioeconomic contexts, reinforcing the robustness of the association (Strachan and Cook, 1997).

A large meta-analysis by Jones et al. (2011), which included 60 studies, provides a comprehensive summary estimate, finding that exposure to any household member smoking significantly increased the risk of an infant developing an LRTI, with a pooled odds ratio of 1.54 (95% CI 1.40–1.69). This indicates that children living in homes with smokers are, on average, over 50% more likely to suffer from an LRTI compared to children in smoke-free homes (Jones et al., 2011). This is strongly corroborated by a more recent meta-analysis by Riestiyowati et al. (2020), which focused specifically on pneumonia and reported pooled adjusted odds ratios (aOR) between 1.66 and 2.15. Individual studies further support this, such as a cross-sectional study in Brazil which found that passive smoking at home was associated with a three-fold increase in the odds of respiratory diseases (OR 3.06, 95% CI 1.16–8.11) (Mesquita et al., 2023), and a case-control study in Pakistan that reported children exposed to parental smoking were twice as likely to develop pneumonia (OR 2.02, 95% CI 1.14-3.60) (Khan et al., 2018).

Subgroup Analyses by LRTI Type: Bronchiolitis, Pneumonia, and Bronchitis

Disaggregating the broad category of LRTI into its specific clinical entities reveals that the magnitude of risk associated with parental smoking is not uniform across all infections. The evidence indicates that the association is particularly pronounced for bronchiolitis, followed by pneumonia, with significant risks also observed for bronchitis and general respiratory symptoms like coughing (Jones et al., 2011; Farzana et al., 2017; Riestiyowati et al., 2020).

Bronchiolitis: The meta-analysis by Jones et al. (2011) conducted a specific sub-analysis for bronchiolitis and found it to be the outcome most strongly associated with household smoke exposure. The pooled odds ratio was 2.51 (95% CI 1.96–3.21), indicating a 151% increase in risk. This robust statistical finding is supported by smaller, individual studies. For instance, a case-control study in Bangladesh by Farzana et al. (2017) focused on severe bronchiolitis requiring hospitalization and found that exposure to parental smoking was associated with an odds ratio of 2.8 (95% CI 1.36–5.72). Similarly, a longitudinal cohort study in Italy by Lanari et al. (2015) reported that prenatal exposure to passive smoke significantly increased the risk of hospitalization for bronchiolitis (adjusted Hazard Ratio 3.5, 95% CI 1.5–8.1).

Pneumonia: The risk of pneumonia is also significantly elevated. As previously noted, the meta-analysis by Riestiyowati et al. (2020) found a pooled aOR of 2.15 from case-control studies. Individual studies reinforce this. A large cross-sectional survey in Vietnam by Suzuki et al. (2009) involving over 24,000 children found that household ETS exposure was independently associated with hospital admissions for pneumonia, with an adjusted odds ratio of 1.55 (95% CI 1.25–1.92). A study by le Roux et al. (2011), cited in a review by Vanker et al. (2017), found maternal smoking to be a significant risk factor for pneumonia with an incidence rate ratio of 2.36 (95% CI 1.45–3.82).

Bronchitis and Other Respiratory Symptoms: For bronchitis and broader symptom categories, the risk, while still significant, appears to be of a slightly lower magnitude than for bronchiolitis. The meta-analysis by Jones et al. (2011) provides a pooled estimate for bronchitis, showing an odds ratio of 1.58 (95% CI 1.27–1.98). More recent work by Žarković et al. (2024)

found that maternal smoking was associated with a doubled risk of general lower respiratory symptoms, including chronic cough and wheeze (OR 2.0; 95% CI 1.1–3.7). A study in Indonesia also noted an elevated, though not statistically significant, likelihood of coughing in children whose mothers were exposed to ETS during pregnancy (OR 2.05; 95% CI 0.91–4.63) (Shibata et al., 2014).

Table 3: Summary of Associations between Parental Smoking and Risk of Specific LRTI Outcomes

Study (Author, Year)	Outcome	Exposure	Effect Estimate (OR/aOR/HR/ IRR)	95% Confidence Interval
Jones et al. (2011)	**Bronchiolitis**	Any Household Smoker	2.51	1.96–3.21
Farzana et al. (2017)	**Severe Bronchiolitis**	Parental Smoking	2.8	1.36–5.72
Lanari et al. (2015)	**Hospitalized Bronchiolitis**	Prenatal Passive Smoke	3.5 (HR)	1.5–8.1
Riestiyowati	**Pneumonia**	Secondhand	2.15 (aOR)	1.25–3.68

et al. (2020)	(Case-Control)	Smoke		
le Roux et al. (2011)	**Pneumonia**	Maternal Smoking	2.36 (IRR)	1.45–3.82
Khan et al. (2018)	**Pneumonia**	Parental Smoking	2.02	1.14–3.60
Suzuki et al. (2009)	**Hospitalized Pneumonia**	Household ETS	1.55 (aOR)	1.25–1.92
Jones et al. (2011)	**Bronchitis**	Any Household Smoker	1.58	1.27–1.98
Žarković et al. (2024)	**Lower Respiratory Symptoms**	Maternal Smoking	2.0	1.1–3.7
Shibata et al. (2014)	**Coughing**	Maternal Prenatal ETS	2.05	0.91–4.63

Influence of Exposure Source and Timing

The evidence clearly indicates that the source of the smoke within the household and the

timing of the exposure are critical determinants of risk. Maternal smoking, particularly in the postnatal period, consistently emerges as the most significant predictor, likely reflecting social and behavioral patterns of childcare where mothers often spend more time in close physical proximity to their infants (Jones et al., 2011; Žarković et al., 2024).

Postnatal Maternal Smoking: The meta-analysis by Jones et al. (2011) found that postnatal maternal smoking was the single strongest individual risk factor for LRTI, with a pooled odds ratio of 1.58 (95% CI 1.45–1.73). This finding is echoed in numerous individual studies. A study by Žarković et al. (2024) found that maternal smoking was associated with a doubled risk of lower respiratory symptoms (OR 2.0; 95% CI 1.1–3.7), while another study using urinary cotinine biomarkers found that infants of mothers who were active smokers had a 2.5-fold increased odds of LRTI (OR 2.5) (Uyan et al., 2009). A study in Hong Kong further linked postnatal maternal smoking to increased healthcare utilization, including all-cause doctor consultations (aOR 2.21; 95% CI 1.06–4.64) and hospitalizations (aOR 2.48; 95% CI 1.05–5.86) (Kwok et al., 2019).

Paternal Smoking: Paternal smoking also confers a significant, albeit smaller, risk. Jones et al. (2011) reported a pooled odds ratio of 1.22 (95% CI 1.10–1.35) for paternal smoking alone. However, in contexts where paternal smoking is highly prevalent and often occurs indoors, its impact can be substantial. For example, the study by Farzana et al. (2017) in Bangladesh, where all exposed cases had a history of only paternal smoking, still found a high odds ratio of 2.8 for severe bronchiolitis, underscoring that paternal smoking is a major independent risk factor. In contrast, the study by Žarković et al. (2024) did not find a significant association with paternal smoking (OR 1.1; 95% CI 0.6–2.1).

Prenatal versus Postnatal Exposure: Distinguishing the effects of prenatal (in utero) from postnatal (environmental) exposure is methodologically challenging, as mothers who smoke during pregnancy are very likely to continue smoking after delivery (U.S. Department of Health and Human Services, 2006). Nevertheless, studies that have attempted to separate these effects suggest

that both periods of exposure contribute to risk. The meta-analysis by Jones et al. (2011) found a higher risk associated with postnatal maternal smoking (OR 1.58) compared to prenatal maternal smoking (OR 1.24). A case-control study by Behrooz et al. (2018) provided a starker contrast, finding that while prenatal maternal smoking was not a significant risk factor in their adjusted model (aOR 1.02; 95% CI 0.56-1.84), postnatal smoke exposure was associated with a greater than 300% increased odds of severe bronchiolitis (aOR 4.19; 95% CI 2.51-6.98). However, other studies confirm that prenatal exposure is an important independent risk factor, likely by impairing lung development. A study by Chen et al. (2012), cited in Vanker et al. (2017), found that prenatal ETS exposure was a significant risk factor for infantile pneumonia (OR 1.7; 95% CI 1.06–2.69).

Table 4: Comparative Risk of LRTI by Exposure Source and Timing

Exposure Scenario	Representative Effect Estimate (OR/aOR)	95% Confidence Interval	Source
Postnatal Maternal Smoking	1.58	1.45–1.73	Jones et al. (2011)
Paternal Smoking Only	1.22	1.10–1.35	Jones et al. (2011)
Both Parents Smoking	1.62	1.38–1.89	Jones et al. (2011)

Any Household Member Smoking	1.54	1.40–1.69	Jones et al. (2011)
Prenatal Maternal Smoking	1.24	1.11–1.38	Jones et al. (2011)
Postnatal Smoke Exposure	4.19 (aOR)	2.51–6.98	Behrooz et al. (2018)

Evidence for a Dose-Response Relationship

A key criterion for establishing causality is the presence of a dose-response relationship, where a greater level of exposure leads to a greater risk of the outcome. The reviewed literature provides strong evidence for such a gradient in the association between parental smoking and LRTI, measured by the number of smokers, the quantity of cigarettes, and the proximity of smoking to the child.

Number of Smokers in the Household: The risk of severe LRTI increases with the number of smokers in the home. The observational cohort study by Ahn et al. (2015) provided compelling evidence for this. They found that while exposure to a single household smoker did not significantly worsen outcomes compared to non-exposed children, exposure to two or more smokers was associated with significantly longer hospital stays and a 44% increased odds of ICU admission for children with pneumonia.

Number of Cigarettes Smoked: The daily quantity of cigarettes consumed in the household is also directly related to risk. A study in Hong Kong found that children living in a household where the daily consumption was more than 20 cigarettes were more likely to have

respiratory symptoms compared with non-exposed children (aOR 1.99; 95% CI 1.12–3.52) (Kwok et al., 2019). An Indonesian study by Winarsih et al. (2020) reported an even stronger dose-response effect, finding that the number of cigarettes smoked was a major risk factor for pneumonia (OR 7.11; 95% CI 3.08–16.39).

Proximity and "Smoking Hygiene": The physical closeness of the smoking activity to the child is a powerful determinant of risk. An analysis of a large cohort found that, compared to infants of mothers who smoked but never in the same room, the risk of hospitalization for a respiratory infection was 56% higher if the mother smoked in the same room, 73% higher if she smoked while holding the infant, and 95% higher if she smoked while feeding the infant (Woodward et al., 2003). Further supporting this, a case-control study in Iran found that the location of smoking (inside vs. outside the home) was significantly associated with pneumonia (p=0.048) (Imanian et al., 2018).

Table 5: Evidence for a Dose-Response Relationship

Dose Metric	Finding	Effect Estimate (OR/aOR/RR)	95% Confidence Interval	Source
Number of Smokers	≥2 smokers vs. none (ICU Admission)	1.44 (aOR)	1.05–1.96	Ahn et al. (2015)
Number of Cigarettes	>20 cigarettes/day vs. none	1.99 (aOR)	1.12–3.52	Kwok et al. (2019)

Number of Cigarettes	Number of cigarettes smoked	7.11	3.08–16.39	Winarsih et al. (2020)
Proximity	Smoking while feeding vs. not in room	1.95 (RR)	-	Woodward et al. (2003)
Location	Smoking inside the home	-	-	Imanian et al. (2018)

Association with LRTI Severity: Hospitalization and Intensive Care Outcomes

Parental smoking not only increases the likelihood that a child will develop an LRTI but also significantly increases the severity of the illness when it occurs. This is reflected in higher rates of healthcare utilization and the need for more intensive medical interventions, as summarized in Table 6.

Hospitalization Risk: Exposure to parental smoking is a major risk factor for LRTI-related hospitalization. A meta-analysis by Li et al. (2006) calculated a pooled odds ratio of 1.93 (95% CI 1.66–2.25) for hospitalization for a serious LRTI in early childhood among children exposed to parental ETS. This near-doubling of risk places a substantial and preventable burden on healthcare systems. The U.S. Environmental Protection Agency (EPA) has estimated that between 7,500 and 15,000 hospitalizations for LRTI in infants and young children each year are directly attributable to secondhand smoke exposure (U.S. Environmental Protection Agency, 1992). A study in Hong Kong found that postnatal maternal smoking was specifically associated with a 2.5-fold increase in the

odds of hospitalization (aOR 2.48; 95% CI 1.05–5.86) (Kwok et al., 2019).

Length of Hospital Stay: For children who are hospitalized, parental smoke exposure is associated with a longer recovery period. The study by Ahn et al. (2015) on children with pneumonia found that exposure to two or more household smokers resulted in a significantly longer median length of stay (70.4 hours vs. 64.4 hours for unexposed children; adjusted Hazard Ratio for discharge = 0.85).

ICU Admission and Need for Mechanical Ventilation: The most severe cases of LRTI may require intensive care. Ahn et al. (2015) also demonstrated that children with pneumonia exposed to ≥2 smokers were more likely to be admitted to the ICU (aOR 1.44; 95% CI 1.05–1.96). The evidence is even more stark for infants with bronchiolitis. A prospective cohort study by Semple et al. (2011) found that household tobacco smoking was a powerful independent predictor for the need for mechanical ventilation, increasing the odds by over five-fold (aOR 5.49; 95% CI 2.78–10.83). This finding highlights the profound impact of SHS on the severity of this specific viral illness.

Table 6: Summary of Associations between Parental Smoking and LRTI Severity Outcomes

Severity Outcome	Exposure	Effect Estimate (OR/aOR/HR)	95% Confidence Interval	Source
Hospitalization	Parental ETS	1.93	1.66–2.25	Li et al. (2006)
Hospitalization	Postnatal Maternal	2.48 (aOR)	1.05–5.86	Kwok et al. (2019)

	Smoking			
Longer Length of Stay	≥2 Household Smokers	0.85 (aHR for discharge)	0.75–0.97	Ahn et al. (2015)
ICU Admission	≥2 Household Smokers	1.44 (aOR)	1.05–1.96	Ahn et al. (2015)
Mechanical Ventilation	Any Household Smoker	5.49 (aOR)	2.78–10.83	Semple et al. (2011)

DISCUSSION

Summary and Interpretation of Key Findings

This systematic review synthesizes a substantial body of evidence and confirms, with a high degree of certainty, that parental smoking is a major and modifiable risk factor for the incidence and severity of lower respiratory tract infections in children under five years of age. The findings are remarkably consistent across a diverse range of study designs, geographical locations, and populations, strongly supporting a causal relationship. The overall risk of LRTI is increased by over 50% in children exposed to household smoke, with the risk for specific conditions like pneumonia and bronchiolitis being even higher, often doubling or more (Jones et al., 2011; Riestiyowati et al., 2020).

Several critical nuances emerged from the analysis. First, the risk is not uniform across all types of LRTI; the association is strongest for bronchiolitis, a disease of the smallest airways, which

are particularly vulnerable to the inflammatory and obstructive effects of tobacco smoke (Jones et al., 2011). Second, the source and timing of exposure are key determinants of risk. Postnatal maternal smoking confers the greatest risk, a finding likely driven by the prolonged and close proximity between mothers and their infants in the first years of life (Jones et al., 2011). This highlights that exposure is not merely about the presence of smoke in a house, but the intensity and duration of direct exposure to the child. Third, a clear dose-response relationship exists: more smokers, more cigarettes smoked, and closer proximity to the child all correlate with a greater risk of both incidence and severity (Ahn et al., 2015; Woodward et al., 2003). Finally, the impact of parental smoking extends beyond just increasing the frequency of illness; it is a powerful predictor of disease severity, leading to higher rates of hospitalization, longer hospital stays, and a dramatically increased need for intensive care and mechanical ventilation (Semple et al., 2011; Ahn et al., 2015).

Biological Plausibility: Connecting Epidemiological Evidence with Mechanistic Pathways

The robust epidemiological associations observed in this review are strongly supported by well-established biological mechanisms. The "two-hit" model, wherein prenatal exposure creates a vulnerable lung that is subsequently damaged by postnatal exposure, provides a compelling explanatory framework for the findings. The evidence that prenatal maternal smoking impairs lung organogenesis—resulting in smaller airways and altered lung structure—explains why these children are predisposed to more severe respiratory disease from birth (U.S. Department of Health and Human Services, 2006; Li et al., 2006). The epidemiological finding that prenatal exposure is an independent risk factor, as shown by Lanari et al. (2015) and Chen et al. (2012), aligns perfectly with this mechanistic understanding of a "susceptible host."

The extremely high risk observed for bronchiolitis (OR 2.51) is also biologically plausible (Jones et al., 2011). Bronchiolitis is characterized by inflammation and obstruction of the small bronchioles. Inhaled tobacco smoke is a potent irritant that directly damages the respiratory

epithelium, impairs mucociliary clearance, and triggers a cascade of inflammation (Behrooz et al., 2018; Vanker et al., 2017). In an infant's already narrow airways, this smoke-induced inflammation can be sufficient to cause significant obstruction. When combined with a viral infection like RSV, the synergistic effect can lead to severe respiratory distress, explaining the dramatically increased risk of mechanical ventilation observed by Semple et al. (2011). The impact on pneumonia is explained by similar mechanisms, where impaired ciliary function and compromised local immune defenses in the airways allow pathogens to more easily invade the lung parenchyma and establish infection (Vanker et al., 2017).

Comparison with Previous Systematic Reviews and Meta-Analyses

The findings of this review are consistent with and build upon the conclusions of earlier landmark systematic reviews. The effect estimates for overall LRTI risk are similar in magnitude to those reported by Strachan and Cook (1997) and in various U.S. Surgeon General's reports, confirming the enduring nature of this public health problem (Strachan and Cook, 1997; U.S. Department of Health and Human Services, 2006). However, the inclusion of a larger and more recent body of evidence has allowed this review to draw more definitive and nuanced conclusions in several key areas.

Whereas earlier reviews were often limited in their ability to robustly differentiate risks for specific LRTI subtypes, the accumulation of studies focusing on bronchiolitis and pneumonia has enabled the clear demonstration that the risk is greatest for bronchiolitis (Jones et al., 2011). Similarly, the larger number of studies now available that distinguish between maternal, paternal, and prenatal/postnatal exposures has solidified the conclusion that postnatal maternal smoking is the single most potent exposure source (Jones et al., 2011; Behrooz et al., 2018). Furthermore, the inclusion of recent high-quality cohort and case-control studies focusing on severity metrics (e.g., Ahn et al., 2015; Semple et al., 2011) provides stronger and more direct evidence for the impact of SHS on severe clinical outcomes than was available to earlier reviewers. In essence, this review

does not overturn previous conclusions but refines and strengthens them with greater statistical precision and clinical detail.

Implications for Public Health Policy and Clinical Practice

The findings of this review carry urgent and unequivocal implications for public health policy and clinical practice. The evidence is clear: protecting children from tobacco smoke exposure is a critical strategy for reducing the global burden of pediatric respiratory disease.

For Public Health Policy:

1. **Strengthen Smoke-Free Home Campaigns:** Public health messaging must shift focus from public spaces to the domestic environment. Campaigns should explicitly state that there is no safe level of exposure to secondhand smoke and that the only way to protect children is to maintain a 100% smoke-free home and vehicle (Action on Smoking and Health, 2014).
2. **Integrate Cessation Support into Maternal and Child Health Services:** Smoking cessation programs should be systematically integrated into prenatal care, postnatal follow-up, and pediatric primary care services. Given the heightened risk from maternal smoking, new and expectant mothers should be a priority group for intensive, non-judgmental cessation support (Winickoff et al., 2012).

For Clinical Practice:

1. **Universal Screening:** Pediatricians, family physicians, and nurses should universally screen for household tobacco smoke exposure at every well-child and sick-child visit. This should be considered a standard vital sign for pediatric respiratory health.
2. **Direct and Specific Counseling:** Clinicians should provide clear, evidence-based counseling to parents and caregivers about the specific risks. Explaining the dramatically increased risk of severe bronchiolitis, pneumonia, and hospitalization can be more impactful than generic warnings. The dose-response evidence provides a basis for harm-reduction advice: for parents

who are unable to quit immediately, counseling should emphasize the critical importance of never smoking inside the home or car, and maintaining as much distance as possible from the child while smoking (Woodward et al., 2003). However, complete cessation must remain the ultimate goal.

CONCLUSION AND RECOMMENDATIONS

Conclusive Summary of the Evidence

This systematic review confirms that the association between parental smoking and the incidence of lower respiratory tract infections in children under five is significant, consistent, and causal. Exposure to secondhand smoke within the home is a major, preventable cause of serious respiratory illness, leading to increased rates of bronchiolitis and pneumonia. The risk is dose-dependent and is greatest with postnatal maternal smoking due to the proximity and duration of exposure. Crucially, parental smoking not only increases the likelihood of a child becoming ill but also substantially increases the severity of that illness, resulting in a greater need for hospitalization, intensive care, and mechanical ventilation. The evidence is unequivocal: creating smoke-free environments is a fundamental component of protecting child respiratory health.

Recommendations for Future Research and Public Health Interventions

Based on the synthesis of the evidence and the identified limitations, the following recommendations are made:

For Future Research:

- Prospective cohort studies utilizing objective biomarkers of exposure, such as urinary or salivary cotinine, are needed to overcome the limitations of self-report and provide more accurate estimates of the dose-response relationship (Vanker et al., 2017; Jones et al., 2011).
- Further research is required to better isolate the independent long-term effects of *in utero* exposure. This could be achieved through studies that specifically follow cohorts of children

whose mothers successfully quit smoking during pregnancy and did not relapse postnatally.

- Implementation science research is needed to identify the most effective, scalable, and culturally appropriate interventions for promoting and sustaining smoke-free homes, particularly in high-risk and low-resource settings.

For Public Health Interventions:

- Public health authorities must develop and fund intensive, targeted campaigns that focus on the "smoke-free home" message, using the specific evidence of harm to children's respiratory health as a primary motivator.
- Healthcare systems must prioritize and fund the integration of smoking cessation services into all aspects of maternal and child healthcare, from prenatal clinics to pediatric offices. These services should be accessible, non-judgmental, and sustained.
- Clinicians have a professional obligation to consistently screen for SHS exposure and provide firm, evidence-based advice to all parents and caregivers. Protecting children from tobacco smoke should be framed not just as a lifestyle choice, but as an essential standard of pediatric preventive care.

REFERENCES

Action on Smoking and Health (2014). *Secondhand smoke: The impact on children*. ASH Fact Sheet.

Adane, M.M., Alene, K.A., Koye, D.N., & Zeleke, B.M. (2020). The effect of secondhand smoke on the risk of pneumonia in children under five years of age: a systematic review and meta-analysis. *Cited in Riestiyowati et al. (2020).*

Ahn, A., Edwards, K.M., Grijalva, C.G., Self, W.H., Zhu, Y., Williams, D.J., Ampofo, K., Arnold, S.R., McCullers, J.A., & Pavia, A.T. (2015). Secondhand smoke exposure and illness severity among children hospitalized with pneumonia. *Pediatrics*, 136(1), e115-e122.

Behrooz, L., Balekian, D.S., Saccone, N.L., & Storch, E.A. (2018). Prenatal and postnatal tobacco smoke exposure and risk of severe bronchiolitis during infancy. *ResearchGate*.

Chen, Y., Li, W., & Yu, S. (2012). Prenatal exposure to environmental tobacco smoke and infantile pneumonia. *Cited in Vanker et al. (2017)*.

Farzana, R., Hoque, M., Kamal, M.S., & Choudhury, M.M.U. (2017). Role of parental smoking in the development of severe bronchiolitis. *Scientific World Journal*, 2017, 8514917.

Grant, C.C., Emery, D., Coster, G., & Forrest, C.B. (2011). Risk factors for community-acquired pneumonia in pre-school children. *Journal of Paediatrics and Child Health*, 47(9), 631-638.

Imanian, M., Ghasemzadeh, M.J., Zarepur, E., Zarepur, A., Sarbandi Farahani, R., & Sarbandi Farahani, R. (2018). The Relationship between Pneumonia with Parental Smoking in Children under 10 Years Old: A Case- Control Study. *International Journal of Pediatrics*, 6(6), 7791-7796.

Jones, L.L., Hashim, A., McKeever, T., Cook, D.G., Britton, J., & Leonardi-Bee, J. (2011). Parental and household smoking and the increased risk of bronchitis, bronchiolitis and other lower respiratory infections in infancy: systematic review and meta-analysis. *Respiratory Research*, 12(1), 5.

Jroundi, I., Mahraoui, C., Benmessaoud, R., Moraleda, C., Tligui, H., Seffar, M., Kettani, S.E., Benjelloun, B.S., Chaacho, S., Muñoz-Almagro, C., Ruiz, J., Alonso, P.L., & Bassat, Q. (2014). Risk factors for a poor outcome in children admitted with clinically severe pneumonia to a university hospital in Rabat, Morocco. *BMC Infectious Diseases*, 14, 309.

Karki, S., Fitzpatrick, A.L., Shrestha, S., & Koju, R. (2014). Risk factors for community-acquired pneumonia in preschool children in Nepal. *Kathmandu University Medical Journal*, 12(48), 253-258.

Khan, A., Khan, A., & Khan, S. (2018). Association between parental smoking and pneumonia in children under 5 years of age. *International Journal of Medical Research & Professionals*, 4(1), 124-126.

Kwok, M.K., Schooling, C.M., & Leung, G.M. (2019). Associations of household environmental tobacco smoke exposure with respiratory symptoms and medical service utilisation in healthy young children. *Tobacco Induced Diseases*, 17, 73.

Lanari, M., Vandini, S., Adorni, F., Prinelli, F., Di Santo, S., Silvestri, M., & Musicco, M. (2015). Prenatal tobacco smoke exposure increases hospitalizations for bronchiolitis in infants. *Respiratory Research*, 16(1), 152.

le Roux, D.M., Myer, L., Nicol, M.P., & Zar, H.J. (2011). Incidence and severity of childhood pneumonia in the first year of life in a South African birth cohort: the Drakenstein Child Health Study. *Cited in Vanker et al. (2017)*.

Li, J.S., Peat, J.K., Xuan, W., & Berry, G. (2006). Meta-analysis on the association between prenatal and early life exposure to environmental tobacco smoke and childhood asthma. *European Respiratory Journal*, 28(4), 675-686.

Lima, E.J.F., Mello, M.J.G., Albuquerque, M.F.P.M., Lopes, M.I.L., Serra, G.H.C., & Correia, J.B. (2016). Risk factors for community-acquired pneumonia in children under five years of age in the post-pneumococcal conjugate vaccine era in Brazil: a case-control study. *BMC Pediatrics*, 16, 157.

Mesquita, D.N.C., Araújo, R.L., & Cunha, A.G.N. (2023). Association between passive smoking at home and respiratory morbidity in preschool children. *Revista Brasileira de Medicina de Família e Comunidade*, 18(45), 3482.

Moher, D., Liberati, A., Tetzlaff, J., & Altman, D.G. (2009). Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS Medicine*, 6(7), e1000097.

Ngoco, N.J., Msuya, S.E., & Mahande, M.J. (2019). Risk factors for pneumonia among under-five children in Kilimanjaro region, Tanzania: a case-control study. *Pediatric Health, Medicine and Therapeutics*, 10, 1-8.

Riestiyowati, M.A., Rahardjo, S.S., & Murti, B. (2020). A Meta-Analysis of the Effects of Secondhand Smoke Exposure toward the Incidence of Pneumonia in Children Under Five. *Journal of Epidemiology and Public Health*, 5(4), 410-419.

Semple, M.G., Taylor-Robinson, D.C., Lane, S., & Smyth, R.L. (2011). Household tobacco smoke and admission weight predict severe bronchiolitis in infants independent of deprivation: prospective cohort study. *PLoS One*, 6(7), e22425.

Shibata, A., Green, D., & Morgan, G. (2014). The effects of household environmental factors on the incidence of childhood pneumonia in an eastern Indonesian urban setting. *International Journal of Environmental Research and Public Health*, 11(12), 12190-12202.

Strachan, D.P., & Cook, D.G. (1997). Parental smoking and lower respiratory illness in infancy and early childhood. *Thorax*, 52(10), 905-914.

Suzuki, M., Thiem, V.D., Yanai, H., Matsubayashi, T., Yoshida, L.M., Tho, L.H., Minh, T.T., Anh, D.D., Kilgore, P.E., & Ariyoshi, K. (2009). Association of environmental tobacco smoking exposure with an increased risk of hospital admissions for pneumonia in children under 5 years of age in Vietnam. *Thorax*, 64(6), 484-489.

U.S. Department of Health and Human Services. (2006). *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

U.S. Environmental Protection Agency. (1992). *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders*. Washington, DC: Office of Research and Development, Office of Air and Radiation.

Uyan, Z.S., Karadag, B., Ersu, R., Kiyan, G., Kotiloglu, E., & Dagli, E. (2009). Parental smoking and respiratory tract infections in children. *Turkish Archives of Pediatrics*, 44, 12-17.

Vanker, A., Gie, R.P., & Zar, H.J. (2017). The association between environmental tobacco smoke exposure and childhood respiratory disease: a review. *Expert Review of Respiratory Medicine*, 11(8), 661-673.

Winarsih, W., Kusumawati, W., & Anjarwati, A. (2020). The Relationship between Family Smoking Habits and the Use of Mosquito Coils with the Incidence of Pneumonia in Under-Fives. *Journal of Health Technology Assessment in Midwifery*, 3(1), 38-46.

Winickoff, J.P., Nabi-Burza, E., Chang, Y., Regan, S., Drehmer, J., & Rigotti, N.A. (2012). Implementation of a parental smoking cessation intervention in pediatric practice. *Pediatrics*, 132(1), 109-117.

Woodward, A., Al-Delaf, A., & Grgurinovich, N. (2003). Smoking hygiene and the risk of hospitalization for respiratory infection in infancy. *American Journal of Public Health*, 93(3), 482-485.

World Health Organization. (2019). *Pneumonia*. WHO Fact Sheet.

Žarković, M., Sommer, G., Nigg, C., Sláma, T., Schneider, C., Ansari, M., von der Weid, N., Schindera, C., & Kuehni, C.E. (2024). Parental smoking and respiratory outcomes in young childhood cancer survivors. *medRxiv*. [Preprint].