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REVIEW ARTICLE

Association Between Maternal Smoking During Pregnancy and Risk of Autism Spectrum Disorder in Offspring: A Systematic Review

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ABSTRACT

Autism Spectrum Disorder (ASD) is a developmental condition that affects how individuals communicate, interact socially, and behave. One of the factor that is gaining attention is maternal smoking during pregnancy. Nicotine and other substances in tobacco smoke can cross the placenta and may interfere with fetal brain development. Despite these concerns, research findings on whether smoking during pregnancy increases the risk of ASD have been mixed. This review examines current evidence on the relationship between maternal smoking during pregnancy and the risk of ASD in offspring. It aims to assess the consistency and strength of reported associations, identify key confounding variables and evaluate the methodological quality of included studies. A structured search was carried out using PRISMA guidelines to identify observational studies published between January 1st 2013 and April 30th 2025. The review focused on studies with cohort, case-control, or cross-sectional designs. Study quality were assessed using tools from the Joanna Briggs Institute (JBI). From an initial pool of 6,915 records, 11 studies met the inclusion criteria. Most were conducted in high-income settings, with sample sizes ranging from fewer than 200 to over two million participants. Five studies reported statistically significant links between maternal smoking and ASD or its subtypes, though the observed effects were small. The remaining six studies, including the only one

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that used serum cotinine to verify smoking status, did not find a significant association. Differences in results appeared to relate to how smoking exposure and ASD were defined and measured, as well as differences between study populations. There is no clear or consistent evidence that maternal smoking during pregnancy is an independent risk factor for ASD. While some research points to a possible connection, results vary and are often limited by how smoking and ASD were assessed. Further research using more objective exposure data, larger and more diverse populations, and detailed subgroup analyses is needed to clarify whether any meaningful association exists.

INTRODUCTION

Autism spectrum disorder (ASD) is a neurodevelopmental condition that usually emerges in early childhood and is marked by difficulties in social communication together with restricted or repetitive patterns of behavior. The ICD 11 now groups childhood autism, atypical autism, and Asperger's syndrome under a single diagnosis, acknowledging their shared features and the wide range of severity across individuals. According to the DSM 5, ASD is also characterized by problems in social interaction, restricted or repetitive interests, and, for some individuals, delays in language. Beyond these core features, the condition can present quite differently from one child to another. Certain children experience further challenges such as anxiety, inattention, or heightened sensory sensitivity, whereas others show few of these, reflecting the variability that defines the spectrum.

The prevalence of autism spectrum disorder (ASD) has been steadily increasing, though reported figures differ widely across regions. In the United States, the proportion of children aged 3 to 17 with a diagnosis rose from 2.5% in 2016 to 3.6% in 2022 (Li & He, 2024). Global prevalence figures remain inconsistent. Salari et al. (2022) estimated an overall rate of 0.6%, with regional values ranging from 0.4% in Asia to about 1% in the Americas and Africa, and up to 1.7% in Australia. Talantseva et al. (2023) reported a similar pooled estimate of 0.72%, with higher prevalence in high income regions such as North America. These differences are likely shaped by diagnostic access, health infrastructure, and cultural perceptions than by true differences in underlying rates.

While ASD is less common than many other psychiatric or developmental conditions, its burden is considerable. The disorder carries lifelong costs linked to early intervention programs, healthcare, education, and therapies. Added to this are indirect economic effects, including reduced workforce participation and lost productivity among both affected individuals and caregivers (Rosanoff et al., 2015). Families also report persistent emotional and psychological pressures, and in more severe cases, care requirements may extend into adulthood, drawing further on social and healthcare systems
(Rosanoff et al., 2015).

ASD has been linked to multiple interacting biological and environmental factors. Prenatal factors reported in the literature include maternal infections, certain medications, chemical exposures, advanced maternal age, and complications such as gestational diabetes or pre eclampsia (Ravi & Mendonca, 2023; Ou et al., 2019; Gialloreti et al., 2019; Vui et al., 2023). Complications around birth such as caesarean section, breech delivery, the need for resuscitation, jaundice, seizures, or respiratory problems have been described more frequently among children later diagnosed with ASD (Myat et al., 2025; Vui et al., 2023). After birth, some studies suggest that early infections or signs of atypical development may play a part in increased vulnerability (Myat et al., 2025). Other factors including air pollution, low income, and minority cultural or language status may further shape vulnerability (Ng et al., 2017; Antonopoulou, 2023).

Among these possible influences, maternal smoking during pregnancy has gained particular attention. Tobacco smoke contains nicotine and other compounds capable of crossing the placenta and interrupting key processes of fetal brain development, such as neuronal growth and communication (Ahmad et al., 2025). Yet, despite repeated study, findings remain mixed, and the strength of the association between smoking and ASD has not been clearly established. Since smoking is a modifiable factor, clarifying this association is of high public health importance, particularly for efforts to prevent prenatal exposure to neurotoxicants (Hertz Picciotto et al., 2022). In this review, we bring together current evidence to assess how consistent the findings are, what potential confounders might be involved, and how the quality of existing studies shapes interpretation.

MATERIALS AND METHODS

A Search Strategy

Four electronic databases namely PubMed, Scopus, ScienceDirect, and Semantic Scholar were searched for studies published between January 1, 2013, and April 1, 2025. A standardized, pilot-tested data extraction form based on the PICOT framework was used (Hosseini et al., 2024). The search focused on observational studies assessing the link between maternal smoking during pregnancy and ASD in children, using the Boolean terms: (maternal smoking) OR (smoking during pregnancy) OR (prenatal smoking) AND (autism) OR (autistic) OR (ASD). The review followed PRISMA 2020 guidelines.

Eligibility Criteria

This review focused on observational studies, including cohort, case-control, and cross-sectional designs, that explored the relationship between maternal active smoking during pregnancy and the diagnosis of ASD in children. The eligibility criteria includes studies that involves human participants, clearly identify active maternal smoking during any stage of pregnancy as the exposure, and assess ASD or autism-related traits as the outcome. Only peer-reviewed journal articles published in English between January 1, 2015, and April 1, 2025 were considered. Studies were excluded if they were systematic reviews, meta-analyses, editorials, conference abstracts, or if they did not specifically examine maternal smoking in relation to ASD outcomes.

Selection Process

A total of 6,915 records were identified from PubMed, Scopus, ScienceDirect, and Semantic Scholar. After excluding 852 records for reasons such as language, access, and publication date, 6,063 remained. Duplicate removal left 5,637 unique records for screening. Title and abstract review excluded 5,593 studies that did not meet inclusion criteria, resulting in 44 articles for full-text assessment. Of these, 33 were excluded due to ineligible ASD criteria, unrelated exposures, or inaccessible full texts. Finally, 11 studies were included in the review. Throughout, two independent reviewers screened records, resolving any disagreements through discussion to ensure objectivity and rigor.

Data Collection Procedure

Data were extracted using a form designed for this review, in line with established methodology. Two reviewers worked independantly to collect information such as study type, location, participant numbers, and basic demographics (Stoll et al., 2019). Methods used to assess smoking exposure and the approaches to statistical analysis were noted, with particular attention given to confounders and reported results. Main results were recorded in a uniform format to allow comparison. Reviewer disagreements were settled through discussion, or by involving a third reviewer if needed. Authors of included studies were contacted when data were incomplete or unclear.

Quality Appraisal

Study quality and risk of bias were evaluated

using the Joanna Briggs Institute (JBI) Critical Appraisal Tools, with the checklist matched to each study design—cohort, case-control, or cross-sectional (Checklist for Systematic Reviews and Research Syntheses JBI, 2017). Items on the checklist were scored 1 for "Yes" and 0 otherwise. Each study's total score was calculated as a percentage, and studies were categorized as high (>70%), moderate (50–70%), or low (<50%) quality based on this result.

Synthesis of Results

After completing data extraction and quality appraisal, findings from all included studies were summarized using a narrative synthesis. This approach was chosen due to variation in study designs, exposure measures, and outcome definitions, which limited the possibility of meta-analysis. The synthesis followed established guidelines and began with a theoretical framework for understanding how maternal smoking might influence ASD risk. Studies were compared based on design, exposure assessment methods, and reported statistical associations. Attention was given to study populations, contextual factors, and how methodological choices affected results. Differences in findings were explored in relation to variations in population, exposure definitions, confounder adjustment, and outcome measurement.

RESULTS

From the initial pool of 6,915 records identified through PubMed, Scopus, ScienceDirect, and Semantic Scholar, removal of 6,063 duplicates left 852 articles for screening. After excluding non-English publications, those outside the ten-year window, and non-open access studies, 48 articles underwent title review and 44 full-texts were evaluated. Ultimately, 11 studies met the inclusion criteria for this systematic review. A summary of the selection process is presented in the PRISMA flow diagram below (Figure 1)

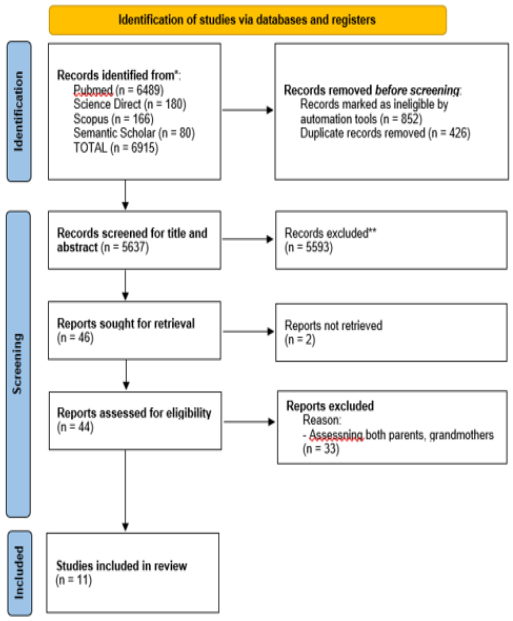

Figure 1: Selection process according to PRISMA flow diagram healthcare in Asia

The studies represented a geographically diverse set of populations, with most conducted in high-income countries such as Turkey, Canada, the US, UK, Sweden, Saudi Arabia, South Korea, and Finland and one from Nepal. Designs included retrospective and prospective case-control, cohort, and cross-sectional methodologies, with sample sizes ranging from fewer than 200 to over two million participants. Using design-specific JBI tools for studies accordingly, none of the studies falling into the moderate. All eleven studies in the distribution table were classified as high quality, suggesting a generally low risk of bias across the evidence base. Summary of distributions is in Table 1.

Table 1: Distributions of the studies included in the review.

No	Study (Author, Year)	Location	Income Group	Quality
1	Bozkurt et al., 2025	Turkey	Upper-middle	High
2	Bhattarai et al., 2018	Nepal	Low	High
3	Saunders et al., 2019	Canada	High	High

4	Berger et al., 2023	USA (California)	High	High
5	Caramaschi et al., 2018	UK	High	High
6	Lebena et al., 2024	Sweden	High	High
7	von Ehrenstein et al., 2020	USA (California)	High	High
8	Abdelkader et al., 2024	Saudi Arabia	High	High
9	Hertz-Picciotto et al., 2022	Multicohort (mainly US, Europe)	High	High
10	Kim et al., 2021	South Korea	High	High
11	Tran et al., 2013	Finland	High	High

Assessment of maternal smoking status during pregnancy relied mainly on self-report methods or parental recall, except for Berger et al. (2023), which combined self-report with serum cotinine measurement, and Caramaschi et al. (2018), which included epigenetic scoring. ASD diagnoses were largely based on clinical or registry sources, though some studies utilized parental report or educational records. Details on exposure and outcome assessment approaches are outlined in Table 2.

Table 2: Distributions of the studies included in the review.

No	Study (Author, Year)	Smoking Diagnosis Method	ASD Diagnosis Method
1	Bozkurt et al., 2025	Self-declared (parental recall)	Clinical diagnosis
2	Bhattarai et al., 2018	Self-declared (parental recall)	Clinical diagnosis
3	Saunders et al., 2019	Self-declared (maternal recall)	Clinical diagnosis
4	Berger et al., 2023	Self-reported & biomarker (cotinine)	Clinical diagnosis (severe ASD)
5	Caramaschi et al., 2018	Self-declared & epigenetic score	Mixed (clinical/ parental/ education)
6	Lebena et al., 2024	Self-declared (harmonized data)	Mixed (not all clinical)
7	von Ehrenstein et al., 2020	Self-declared (parental report)	Clinical diagnosis
8	Abdelkader et al., 2024	Self-declared (maternal report)	Clinical diagnosis (registry)
9	Hertz-Picciotto et al., 2022	Self-declared (maternal report)	Clinical diagnosis
10	Kim et al., 2021	Self-declared (maternal/paternal report)	Parental report (ASSQ score)
11	Tran et al., 2013	Self-declared (maternal report)	Clinical diagnosis (registry)

Propotions of maternal smoking among ASD cases varied considerably from 1.24% in the UK Avon Longitudinal Study to 20% in a Canadian case-control cohort. In South Korea, very low maternal smoking prevalence meant researchers instead analyzed paternal smoking. Such variability reflects differences in population smoking behaviours as well as study methodology.

Of the 11 included studies, 5 reported a significant positive association, while 6 found no significant link between maternal smoking during pregnancy and increased ASD risk or specific ASD subtypes. Saunders et al. (2019) reported a greater than twofold increase in ASD risk in a Canadian cohort (OR 2.56, 95% CI 1.19–5.49, p=0.016). Hertz-Picciotto et al. (2022) found a modest but significant association in pooled US cohorts (RR 1.44, 95% CI 1.02–2.03, p=0.03). Von Ehrenstein et al. (2020) observed elevated ASD risk in California linked to any maternal smoking before or during pregnancy (OR 1.15, 95% CI 1.04–1.26, p<0.05), with heavier exposure increasing risk further (OR 1.55, 95% CI 1.21–1.98, p<0.05). In Finland, Tran et al. (2013) identified a significant association for the pervasive developmental

disorder (PDD) subtype (OR 1.2, 95% CI 1.04–1.5, $p<0.05$). The South Korean study by Kim et al. (2021) reported a notable relationship for paternal smoking (RR 2.74, 95% CI 1.71–4.4, $p<0.001$); maternal smoking was too rare for robust analysis.

In contrast, six studies, including those from Turkey (Bozkurt et al., 2025), Nepal (Bhattarai et al., 2018), Saudi Arabia (Abdelkader et al., 2024), Sweden (Lebena et al., 2024), the UK (Caramaschi et al., 2018), and California (Berger et al., 2023) did not find a significant association. Berger et al. (2023) stands out for its use of objective biomarker data, finding no link between serum cotinine levels in pregnancy and ASD outcome. Additional prevalence and findings for each study are summarized in Table 3.

DISCUSSION

Reviewing the available studies reveals signs of a possible association between maternal smoking during pregnancy and ASD risk, though findings are far from uniform. The proportion of mothers who smoked among ASD cases stretches from as low as 0.25% to as high as 32.4% (Saunders et al., 2019; Hertz-Picciotto et al., 2022; von Ehrenstein et al., 2021; Kim et al., 2021). These differences stem from variations in population backgrounds, smoking habits, and research methods. This observed relationship appears in both case-control and cohort studies, but inconsistencies remain and the evidence is not definitive.

Biologically, nicotine crosses the placenta, accumulating in the fetus and possibly distorting early brain development. It affects nicotinic acetylcholine receptors before neural networks are fully formed, and may change gene expression, possibly through DNA methylation, as well as increase oxidative stress and inflammation in developing tissue (Zhuang et al., 2024). Beyond cholinergic pathways, prenatal nicotine impacts dopamine and glutamate signaling, impairs neuronal growth, and animal data show brain structure changes that mirror certain ASD-related impairments (Alkam et al., 2017; Polli et al., 2020; Mamiya et al., 2016; Zhu et al., 2012).

While biological pathways suggest tobacco exposure could influence brain development, strong statistical links with ASD were not found in most well-conducted studies. This may partly reflect the major influence of genetics, which tends to overshadow modest environmental effects like maternal smoking. For example, large cohort data show that ASD risk is much more strongly associated with family history than with prenatal tobacco exposure (Lebeña et al., 2024). Some studies have noted possible associations in specific ASD subtypes or particular groups, rather than across all cases (Larsson et al., 2009). The landscape is made more complex by shared genetic and epigenetic factors seen in other neurodevelopmental conditions, including schizophrenia and ADHD (Havdahl et al., 2021).

The studies reviewed also present several methodological limitations. Details regarding intensity, timing, and duration of smoking were often incomplete, limiting assessment of dose-response effects (Abdelkader et al., 2024). In places where maternal smoking is rare, associations are more difficult to detect and confidence intervals widen, as seen in studies from Nepal and Turkey (Bhattarai et al., 2018; Bozkurt et al., 2025). Social and cultural factors shape both smoking prevalence and ASD risk, adding further variability. Reliance on self-reported smoking, subject to recall bias and stigma, likely leads to underestimation of true exposure.

Much of the available research has been conducted in high-income regions, limiting the generalizability of the results to settings with different social and environmental conditions, particularly in low- and middle-income countries. Additionally, the number of cases where mothers smoked

during pregnancy is often modest, frequently below 10% which limits the ability to detect subtle or dose-related risks. When studies do report an association, the effect is typically small and can disappear after accounting for confounding factors.

To summarize, current evidence does not show a consistent or strong association between maternal smoking in pregnancy and ASD, with small observed effects often disappearing after adjustment for confounders. Future work should focus on more robust exposure measurements, such as biomarkers, and make an effort to include wider and more varied populations. Research in more diverse, especially low- and middle-income, populations and coordinated, population-based, clinical and biological studies using standardized methods are needed to clarify whether specific subgroups face increased risk. Combining evidence from clinical, biological, and epidemiological approaches will be important to addressing these uncertainties. Public health efforts should continue to support smoking cessation during pregnancy. More research involving larger and more varied populations, along with better ways of measuring exposure, is needed to clarify any potential role maternal smoking might play in ASD.

Table 3: Prevalence and association of maternal smoking and ASD in included studies

Study (Author, Year)	Study type	Smoking Mothers Among ASD Cases	Significant Association	Key Result / Effect Size (if significant)
Saunders et al., 2019	Case-Control	20.2%	Yes	OR = 2.56 (1.19–5.49), p = 0.016
Hertz-Picciotto et al., 2022	Cohort	Range 4.4%–32.4% (across cohorts)	Yes (after exclusions)	RR = 1.44 (1.02–2.03), p = 0.03
von Ehrenstein et al., 2020	Case-Control	2.6%	Yes	Any smoking: OR = 1.15 1.04–1.26), p < 0.05; Heavy: OR = 1.55 (1.21–1.98), p < 0.05
Kim et al., 2021	Cohort	0.25-0.3%	Yes	RR 2.74 (1.71-4.4), p<0.001
Tran et al., 2013	Case-Control	19%	Yes (PPD)	OR 1.2 (1.04-1.5), p<0.05
Bozkurt et al., 2025	Retrospective Case-Control	Mild-moderate 5% Severe 4.2%	No	p = 0.315
Bhattarai et al., 2018	Case-Control	8.6%	No	OR = 7.28 (0.81–65.9)
Berger et al., 2023	Case-Control	2.4%	No	OR = 1.64 (self-report) OR = 0.73 (cotinine)
Caramaschi et al., 2018	Prospective Cohort	1.24%	No	RR = 0.67 (0.4–1.12)
Lebena et al., 2024	Prospective Cohort	14.4%	No	p = 0.210
Abdelkader et al., 2024	Cross-sectional	10.2%	No	p = 0.828

CONCLUSION

There is no consistent evidence that maternal smoking during pregnancy is an independent risk factor for ASD. A few studies have found weak associations, but the results vary and are likely affected by genetic predispositions, social and demographic influences, how exposure was measured and differences across study populations. If a link does exist, it is likely to be small, vary by context, or apply only to certain subgroups. Even though a direct connection to ASD remains unclear, the harmful effects of prenatal smoking on maternal and child

health are well documented. For that reason, public health efforts should continue to support smoking cessation during pregnancy. This systematic review tackles a clinically relevant public health issue with transparency, but its conclusions are limited by incomplete literature coverage, residual confounding in primary studies, and variation in study designs and ASD assessment methods. More research involving larger and more varied populations, along with better ways of measuring exposure, is needed to clarify any potential role maternal smoking might play in ASD.

CONFLICT OF INTEREST

The authors declare no conflict of interest

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