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REVIEW ARTICLE

Association between postnatal second-hand smoke exposure and ADHD in children: a systematic review and meta-analysis

Anyan Huang[1] · Kusheng Wu[2] · Zemin Cai[2] · Yuhang Lin[1] · Xuanzhi Zhang[1] · Yanhong Huang[1]

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Abstract

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder which is caused by the interplay of genetic and environmental risk factors such as second-hand smoke (SHS). The association between postnatal exposure to SHS and ADHD risk in children was still inconclusive. We performed a systematic review and meta-analysis to explore the definite association. We searched for relevant studies from PubMed, Embase, Ovid, and Web of Science databases up to January 2020. We used random effect models to calculate pooled odds ratio (OR) with 95% confidence interval (CI). Subgroup analyses and sensitive analyses were also performed to solve the heterogeneity. According to our inclusion criteria, 9 studies including 6 cross-sectional studies, 2 cohort studies, and 1 case-control study were included in the final analysis. Postnatal exposure to SHS increased the risk of ADHD in children (OR: 1.60; 95% CI: 1.37–1.87). Children who exposed to SHS were found a slight risk for conduct problems (OR: 1.33, 95% CI: 1.00–1.77). Among the studies which used cotinine as a biomarker for SHS exposure, a lower pooled OR (OR = 1.16, 95%CI = 1.01, 1.33) was observed between cotinine and ADHD in children. Our meta-analysis results suggested that SHS exposure may be a risk factor for ADHD. We also found that SHS exposure may be associated with some adverse behavioral outcomes. More prospective studies should be conducted to confirm the relationship between SHS exposure and ADHD in children.

Keywords Attention deficit hyperactivity disorder · Second-hand smoke · Postnatal · Behavioral outcomes · Children · Meta-analysis

Introduction

Attention deficit hyperactivity disorder (ADHD) is one of the most commonly diagnosed neurodevelopmental disorders which are characterized by developmentally inappropriate hyperactivity, inattention and impulsiveness, with an estimated worldwide prevalence of 5% in school-age children (Thapar et al. 2012; Wong et al. 2019). Although the etiology of ADHD is still not fully understood, there is consistent evidence that genetic factors contribute to the etiology of ADHD and that susceptibility genes interact with environmental risk factors in complex ways (Thapar et al. 2007). ADHD often co-occurs with other psychiatric diseases, such as conduct disorder and oppositional defiant disorder, which could be explained by both shared genetic and environmental influences (Tistarelli et al. 2020). In addition, some prospective studies have found that ADHD symptoms can persist into adulthood which may lead to poor quality of life and continuous financial loss (Klein et al. 2012). De Ridder and De Graeve (2006) pointed that ADHD in children had led to a significant increase in the use of healthcare and a negative impact on school performance and parental productivity. Given the social burden of ADHD and its impact on patients' lives, attention should be paid to the prevention of ADHD.

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✉ Kusheng Wu
kswu@stu.edu.cn

✉ Yanhong Huang
12yhhuang@stu.edu.cn

[1] Mental Health Center, Shantou University Medical College, North Taishan Road, Shantou 515065, Guangdong Province, China

[2] Department of Preventive Medicine, Shantou University Medical College, Shantou 515041, Guangdong Province, China

Second-hand smoke (SHS) has long been a public concern. It refers to tobacco smoke inhaled by both the active first-hand smoker and the people around. Tobacco smoke contains at least 250 toxic chemicals, and some toxic metals ingredients have been shown to cause serious harm to humans (Pagani 2014). In addition, tobacco smoke and its byproducts are among the most common indoor pollutants globally, with about 40% of children, 35% of women, and 33% of men regularly exposed to SHS indoors (Oberg et al. 2011). Among them, infants and children may be more susceptible to SHS because of their immature respiratory, circulatory, and neurological systems (DiFranza et al. 2004). To date, most of studies have focused on the effects of maternal active smoking during pregnancy on children's behavioral outcomes (Minatoya et al. 2019; Obel et al. 2016; Sourander et al. 2019). A meta-analysis found that prenatal exposure to maternal smoking during pregnancy was significantly associated with childhood ADHD after adjusting for parental psychiatric history and social socioeconomic status (Dong et al. 2018). Although most studies addressing tobacco smoke and behavioral outcome are more abundant with regard to prenatal exposure, there is rationality to think that postnatal SHS exposure evokes comparable adverse outcome. Herrmann et al. (2008) founded that children with either prenatal or postnatal tobacco exposure have more adverse behavioral outcomes such as oppositional defiant disorder, conduct disorder, delinquency, and ADHD. A longitudinal study which included over 2000 children age 4 to 11 years old also found that postnatal SHS exposure was associated with behavior problems in children, even after controlling for multiple potential confounders (Weitzman et al. 1992). Animal studies in adult rats indicated that exposure to SHS during adolescence causes long-term cognitive deficits (Counotte et al. 2009). However, some studies on the relationship between postnatal exposed to SHS and ADHD in children still have inconsistent results. Rückinger et al. (2010) argued that their studies did not show an association between exposure to SHS after birth and ADHD in children. Two recent studies using NHANES data also failed to find an increased risk of ADHD in children who exposed to SHS (Braun et al. 2006; Froehlich et al. 2009).

Therefore, in order to better clarify the relationship between SHS exposure and the risk of ADHD in children, we conducted a systematic review using meta-analysis. Given the adverse effects of ADHD, it makes sense to determine whether SHS exposure is related to ADHD in children. Moreover, the significance of the results will prompt people to quit smoking and create a healthy living environment for children.

Methods

Search strategy

We used the search terms related to postnatal, ADHD, and second-hand smoke to seek relevant studies in 4 major databases: PubMed, Embase, Ovid, and Web of Science. We retrieved studies started from database building to January 15, 2020. We not only searched the published literatures but also manually retrieved the references of obtained articles. The details of the search terms in each database were reported in Online Resource 1.

Selection criteria

We included population-based observational studies (cross-sectional studies, case-control studies, or cohort studies, including prospective and retrospective cohort studies) which reported the strength of association, expressed as an odds ratio (OR) or relative ratio (RR) with 95% confidence intervals (CIs), between SHS exposure and ADHD. All the included studies were published in English. The age limit for children is under 18 and there are no area restrictions. If multiple studies use the same population, we included newer studies.

Children with ADHD are identified through (1) the diagnosis of ADHD is by the Diagnostic and Statistical Manual of Mental Disorders (DSM) or the International Classification of Diseases (ICD); (2) any medical records of ADHD; (3) using medication for ADHD, such as stimulants; (4) scores were above the symptom threshold using other valid assessment scales for ADHD; and (5) parent or guardian reported.

The measurements of SHS exposure are based on (1) using questionnaires to ask parents or guardians whether their children are exposed to SHS after birth; (2) information about children exposure to SHS in medical or other records.

Quality assessment

Study and outcome quality of case-control and cohort studies were assessed using the Newcastle–Ottawa Scale (NOS) (Stang 2010) which is a valid assessment tool. The NOS consists of 8 items with a maximum score of 9. If the score of the study is greater than 6, we define it as a high-quality study. The methodological quality of cross-sectional studies was assessed using an 11-item checklist which was recommended by the Agency for Healthcare Research and Quality (AHRQ) (Rostom and Cranney 2004). We classified the quality of the cross-sectional study into 3 categories: high quality (scored 8–11), moderate quality (scored 4–7), and low quality (scored 0–3) (Online Resource 1).

Data extraction

Two reviewers (Huang and Cai) independently extracted information on study characteristics based on the recommendations of the Meta-analysis of Observational Studies in Epidemiology (MOOSE) guidelines (Stroup et al. 2000) and the Preferred Reporting Items for Systematic Reviews and Meta Analyses (PRISM) statement (Liberati et al. 2009). Characteristics of the selected studies included the first author, publication year, study design, study area, age of children in ADHD, gender, number of case groups and total population, measurement of SHS smoke exposure, different diagnosis methods of ADHD, various adjusted confounders, and risk estimates (OR or RR with 95% CI). We selected the adjusted OR of the model with the most adjustment factors, if there were multiple models in the study. If there was any dispute in the process of data extraction and analysis, the third reviewer (Wu) will discuss with the first two reviewers and re-evaluate the original study, finally reaching a consensus.

Publication Bias

The Begg's test (Begg and Mazumdar 1994) and Egger's test (Egger et al. 1997) ($p < 0.05$) were used to evaluate publication bias in selected studies. We also visualized publication bias using funnel plots. If Begg's test or Egger's test indicates that the study has publication bias, we will implement trim-and-fill method (Duval and Tweedie 2000) to adjust publication bias.

Statistical analysis

We calculated the pooled OR which represents the association between SHS exposure and the risk of ADHD in children. In this analysis, we directly considered the RR in the study results as the OR. We also used the Cochrane Q test and calculated the I^2 statistic (Higgins et al. 2003) to indicate the degree of heterogeneity. If heterogeneity exists ($I^2 > 30\%$) among the selected studies, we will use the random effects model to calculate the pooled OR. Additionally, we used subgroup analysis to explore the heterogeneity sources. Sensitivity analysis was used to estimate the impact of a single study on pooled effect size by excluding one study at a time. All of the statistical analyses were performed using STATA 12.0 (Stata Corp., College Station, TX).

Results

Study selection process

The flow chart of study selection process is shown in Fig. 1. According to our search strategy, we searched from PubMed, Embase, Ovid, and Web of Science databases and found a total of 634 potentially related literatures. After studies which were duplicated being removed, 384 articles were left. Then, based on the title and abstract of the articles, we excluded 361 literatures, including 16 systematic reviews, 8 animal studies, and 6 meta-analyses. We further examined the full-text of remaining 23 literatures according to the previously proposed inclusion criteria, 8 studies were included. Among them, Braun's study (Braun et al. 2006) which used the same population data as Max's (Max et al. 2013) was excluded because it was published earlier and included fewer people. Moreover, we included 2 other articles through reference retrieval manually. Finally, we selected 9 studies (Bauer et al. 2015; Cho et al. 2018; Joo et al. 2017; Kabir et al. 2011; Max et al. 2013; Padrón et al. 2016; Rückinger et al. 2010; Tiesler et al. 2011; Twardella et al. 2010) for the systematic review and meta-analysis.

Study characteristics

The characteristics of the selected studies are shown in Table 1. We included 6 cross-sectional studies (Bauer et al. 2015; Cho et al. 2018; Kabir et al. 2011; Max et al. 2013; Padrón et al. 2016; Twardella et al. 2010), 2 cohort studies (Rückinger et al. 2010; Tiesler et al. 2011), and 1 case-control study (Joo et al. 2017) for our systematic review and meta-analysis. All of the studies were published between 2010 and 2018 including 6663 ADHD patients and 93,825 controls (Table S1). Among the included studies, 3 were from Germany (Rückinger et al. 2010; Tiesler et al. 2011; Twardella et al. 2010), 2 from Korea (Cho et al. 2018; Joo et al. 2017), 3 from the USA (Bauer et al. 2015; Kabir et al. 2011; Max et al. 2013), and 1 from Spain (Padrón et al. 2016). Most studies (Cho et al. 2018; Kabir et al. 2011; Max et al. 2013; Padrón et al. 2016; Rückinger et al. 2010) collected information about SHS exposure in children based on interviews from parents or guardians. A few studies (Bauer et al. 2015; Joo et al. 2017; Tiesler et al. 2011; Twardella et al. 2010) used questionnaires filled out by parents or guardians to obtain details of children's SHS exposure. Both cohort studies (Rückinger et al. 2010; Tiesler et al. 2011) as well as two cross-sectional studies (Padrón et al. 2016; Twardella et al. 2010) assessed ADHD symptoms in children at age 10 using the Strengths and Difficulties Questionnaire (SDQ) (Goodman 1997). Some studies (Cho et al. 2018; Kabir et al. 2011; Max et al. 2013) collected information directly from parents or guardians on whether their children were diagnosed with ADHD. In addition, Max et al. (2013) also considers children who used stimulant drugs to be ADHD patients. Only two selected studies (Bauer et al. 2015; Joo et al. 2017) adopted the diagnostic criteria for ADHD from the Diagnostic and Statistical Manual of Mental Disorders, four Edition (DSM-IV) (Association 1994) and the

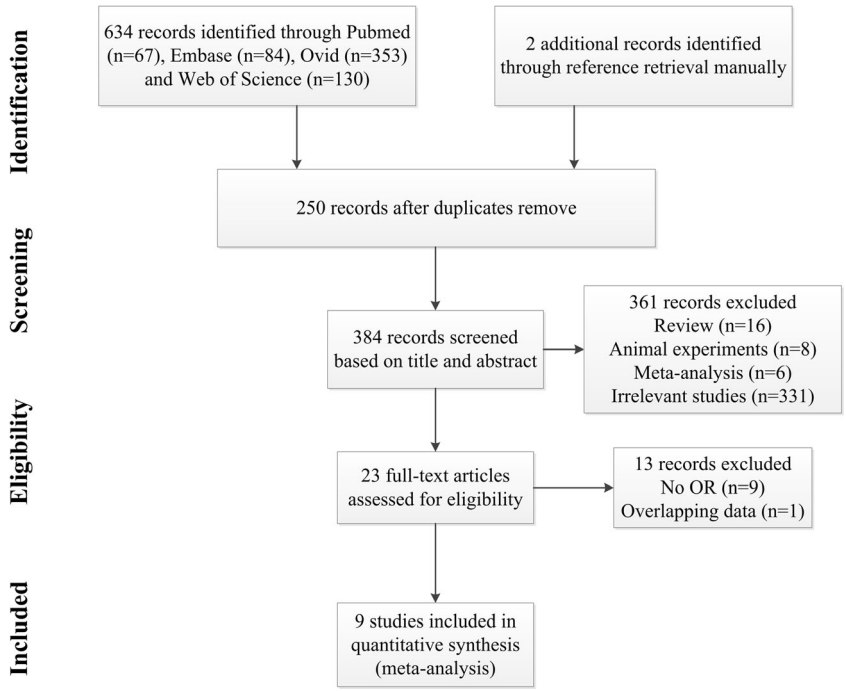

Fig. 1 Flow chart of the study selection process

International Classification of Diseases, 9th revision (ICD–9) (Preedy and Watson 2010). In addition, we evaluated the methodological quality of all selected studies. All of the included cohort and case-control studies (Joo et al. 2017; Rückinger et al. 2010; Tiesler et al. 2011) were high quality (score > 6). Two cross-sectional studies (Padrón et al. 2016; Twardella et al. 2010) were rated as high quality and the remaining (Bauer et al. 2015; Cho et al. 2018; Kabir et al. 2011; Max et al. 2013) were rated as medium quality (Table S1).

Postnatal SHS exposure and the risk of ADHD in children

We conducted a meta-analysis of all selected studies using random effect model. The pooled adjusted OR was 1.61 (95% CI: 1.37, 1.88). It is indicated that there is a positive association between postnatal SHS exposure and ADHD in children (Fig. 2). Heterogeneity of medium degree among studies was found ($I^2 = 42.5\%$, $p = 0.084$). Of the included studies, four studies used the SDQ scale to assess children behavior problems. We extracted the adjusted OR of two dimensions (conduct problems and peer problems) in the SDQ scale for meta-analysis. No association was found between postnatal SHS exposure and peer-relationship problems in children, the pooled OR was 1.14 (95% CI: 0.96, 1.37; $I^2 = 0\%$, $p = 0.413$) (Fig. S1). However, children who exposed to SHS were found a slight risk for conduct problems (OR = 1.33, 95% CI: 1.00, 1.77; $I^2 = 61.5\%$, $p = 0.051$) (Fig. 3). In the results of these studies, we did not combine the adjusted OR of other dimensions of the SDQ scale (emotional symptoms, prosocial behavior), because data were lacking in some studies.

Publication bias

The funnel plot did not show significant asymmetry (Fig. S2). The results of Begg's test ($Z = 1.77$, $p = 0.076 > 0.05$) and Egger's test ($t = 1.96$, $p = 0.091 > 0.05$) indicated that there was no significant publication bias in the selected studies. We did not use trim-and-fill method here because no significant publication bias was found.

Sensitivity analysis

We excluded one study at a time from the included studies and recalculated the pooled OR with 95% CI. The significant change was not found in the comparison between the new pooled OR and the total pooled OR (Fig. S3).

Subgroup analysis

We also conducted subgroup analysis based on different study design, regions, ADHD diagnosis methods, and adjustment factors. The studies which observed a lower pooled OR were conducted in Korea (OR = 1.55, 95% CI: 1.11, 2.17; $I^2 = 0.2\%$, $p = 0.317$) and the USA (OR = 1.49, 95% CI: 1.29, 1.72; $I^2 = 0\%$, $p = 0.561$). A higher pooled OR (OR = 1.81, 95% CI: 1.23, 2.65) was observed in the studies from Europe, but there was a high heterogeneity ($I^2 = 73.2\%$, $p = 0.011$) among these studies (Fig. S4). The pooled OR of cross-sectional studies was 1.71 (95% CI: 1.38, 2.12) and a

Table 1 Characteristics of Included Studies of Second-hand Smoke Exposure and ADHD in children

Author	Year	Study design	Location	Age (y)	Identification of SHS exposure	Diagnosis of ADHD	Adjustment variables
Cho et al	2018	Cross-sectional	Korea	< 18	A health care interview to an adult	Doctor diagnosis	Child's age, sex and any adult with depression in the household
Padron et al	2016	Cross-sectional	Spain	5–14	A face to face interview to parents or legal guardian for how long was the child exposed to tobacco smoke in the home	SDQ	Child's sex, age, country of birth, physical activity, exposure to SHS outside the home, child's weight, household structure, parental characteristics, the mental health of the parents, and five neighborhood environmental risk factors
Bauer et al	2015	Cross-sectional	US	0–6	Any positive response to question asking families whether any household member smoked	ICD-9	Child gender, race/ethnicity, insurance status, and parental depressive symptoms
Max et al	2013	Cross-sectional	US	4–15	Household respondent reported home exposure by a household interviews	Doctor diagnosis or stimulant medication use	Maternal smoking during pregnancy, gender, age, race/ethnicity, preschool attendance, health insurance coverage, exposure to lead
Kabair et al	2011	Cross-sectional	US	< 12	Interviews parents and asked of parents "Does anyone smoke inside child's home?"	Doctor diagnosis	Household member smoking status, child's age group, gender, race/ethnicity, household composition, residence, primary language spoken, household poverty status, mother's education, foreign status of mother, legal marital status of mother, total number of children in the household, total number of adults in the household and low birth weight
Twardella et al	2010	Cross-sectional	German	5–7	Data were collected via a questionnaire, which had to be filled out by a parent	SDQ	Gender, area, foreign nationality, single parent, parental education, relative poverty, low birth weight, smoking of the mother before and during pregnancy
Joo et al	2017	Case-control	Korea	6–10	Obtained from the children's parents or guardians by means of a self-administered questionnaire	DSM-IV	Mother's education level, family history of ADHD, parental marital status, teenage mother, and blood lead concentration
Ruckinger et al	2010	Cohort	German	10	Collected smoking status from parents at follow-up interviews when the child was 1, 2, 3, 4, 6, and 10 years of age	SDQ	Sex, study center, intervention group, parental education, father's employment, age of mother at birth, child's time in front of screen, and being single father/mother
Tiesler et al	2011	Cohort	German	10	Questionnaire data collected between the follow-ups at ages 6, 12, 18 and 24 months, 4 and 6 years	SDQ	Sex, study center, parental educational level, the age of mother at birth, being single mother/father and time spent in front of a screen

SHS, second-hand smoke; *ADHD*, attention deficit hyperactivity disorder; *SDQ*, Strengths and Difficulties Questionnaire; *ICD-9*, International Classification of Diseases, 9th revision; *DSM-IV*, Diagnostic and Statistical Manual of Mental Disorders, fourth Edition

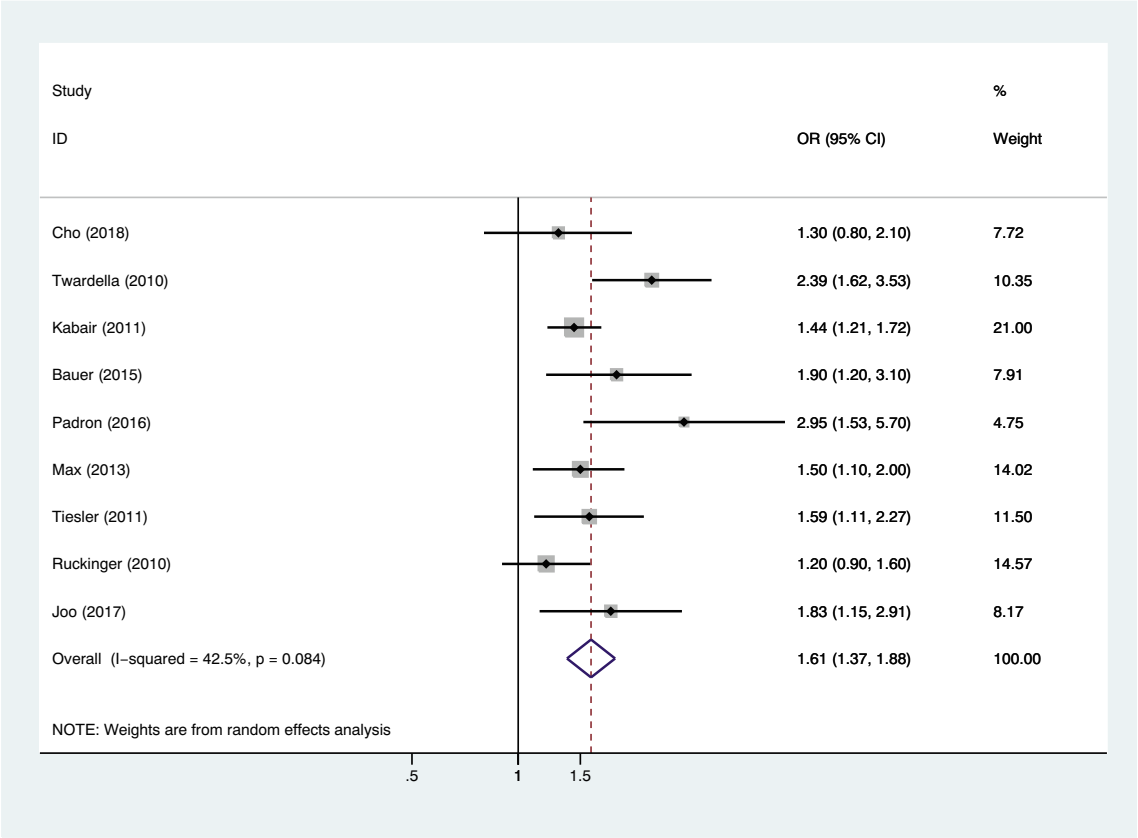

Fig. 2 The forest plot of adjusted ORs of included studies

moderated heterogeneity was found ($I^2 = 50.9\%$, $p = 0.07$). The pooled OR of cohort studies was 1.35 (95% CI: 1.03, 1.78; $I^2 = 50.9\%$, $p = 0.07$) (Fig. S5). A strong association was found in the studies using SDQ to assess the ADHD in children (OR = 1.81, 95% CI: 1.23, 2.65; $I^2 = 73.2\%$, $p = 0.011$). The studies which adjusted parental mental health have a higher pooled OR (OR = 1.82, 95% CI: 1.36, 2.45) than the studies not adjusted (OR = 1.53, 95% CI: 1.27, 1.84). A lower pooled OR was observed in the studies after adjusted maternal prenatal smoke (OR = 1.53, 95% CI: 1.30, 1.80). Since the number of included studies was less than 10, meta-regression was not performed. The results of subgroup analyses are summarized in Table 2.

Discussion

We used adjusted ORs for meta-analysis of included studies to uncover the association between SHS exposure and ADHD in children. SHS exposure after birth may be associated with ADHD in children. Children who exposed to SHS have a 1.6 times higher risk of developing ADHD than children not exposed. In addition, we also selected studies that used the SDQ scale to evaluate children's behavioral problems under SHS exposure and combined some of the results of these studies. We found that children exposed to postnatal SHS had a greater risk of conduct problems, consistent with the findings of (Wang et al. 2019). However, we did not find a significant association between SHS and peer relationship problems in children.

Due to the moderate heterogeneity among the selected studies, we conducted a subgroup analysis based on some common characteristics to explore the sources of heterogeneity. When performing subgroup analyses based on different study designs, we found that there was still moderate heterogeneity in cross-sectional and cohort studies. A higher pooled OR was observed in Europe than in the USA and Korea. However, studies in Europe are highly heterogeneous due to the large differences in study populations. We also found that all of the selected Europe studies used the SDQ scale to assess ADHD symptoms. Therefore, we continued to perform another subgroup analysis based on the diagnosis method of ADHD. Except for the European study, the rest of the studies ascertained ADHD in children by parent-reported clinical diagnosis records. Heterogeneity was not found in these studies, whereas the lower

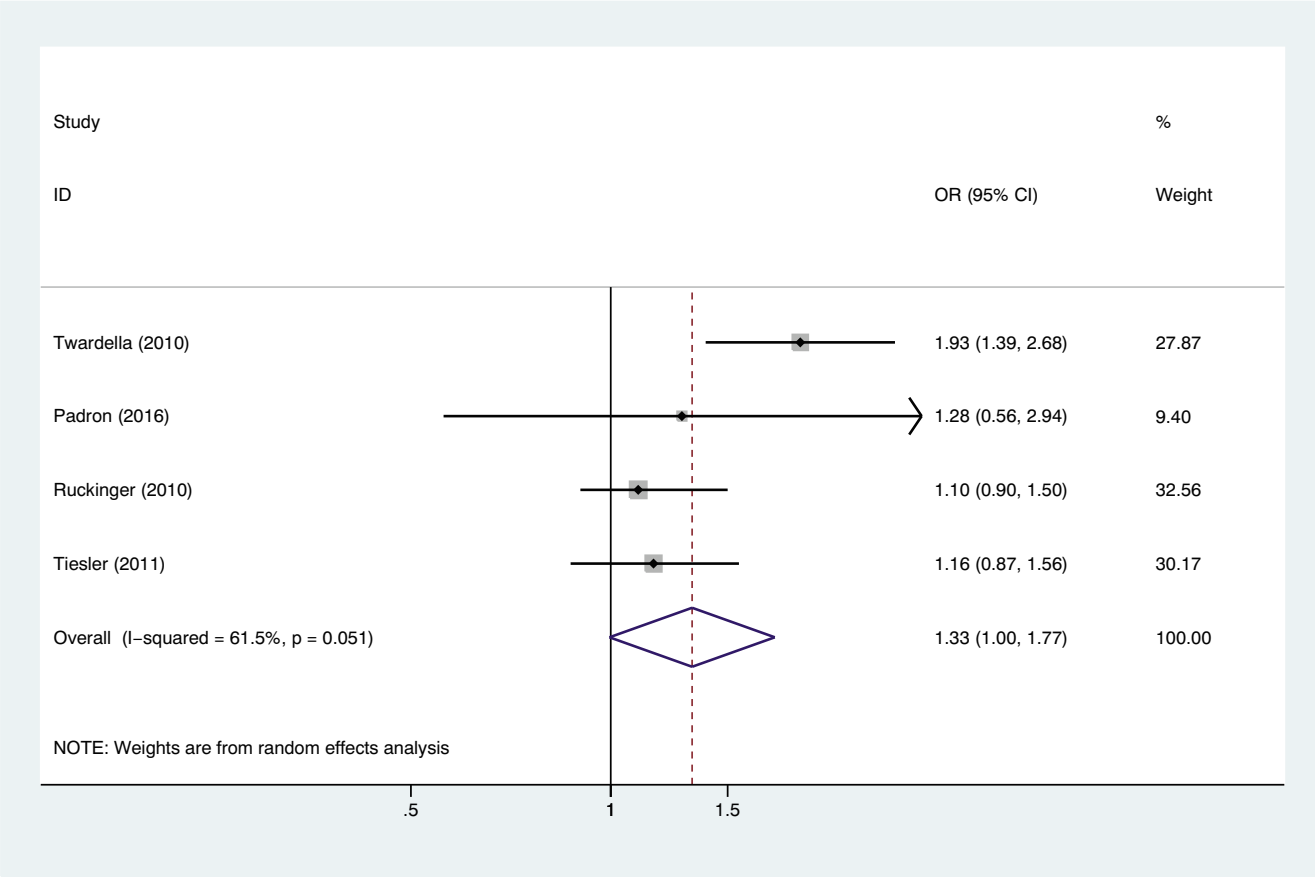

Fig. 3 The pooled OR of included studies of the association between SHS exposure and conduct problems in children

pooled OR was observed. So, it is reasonable to believe that the heterogeneity of the included studies is due to the different methods and criteria of diagnosing ADHD.

Notably, many studies focused on maternal smoking during pregnancy and concluded that prenatal maternal smoking is a risk factor for ADHD in offspring (Langley et al. 2005; Schmitz et al. 2006). Some meta-analyses also reported the similar results (Dong et al. 2018; Huang et al. 2018). We combined the ORs of included studies that adjusted for maternal prenatal smoking and still found a strong association between postnatal SHS exposure and ADHD in children. Heath and Picciotto (2009) suggest that this may be caused by nicotine-mediated neurobiological disorders. Nicotine exposure in the environment may cause a variety of changes at the molecular and cellular levels of neurons. Fergusson et al. (1993) also found that after controlling for maternal prenatal smoking and other confounding factors, children exposed to SHS after birth showed a significant increase in ADHD symptoms compared with children not exposed to SHS.

The heritability of ADHD has been confirmed by many studies. Twin studies show that identical twins are more likely to develop ADHD than fraternal twins (Thapar et al. 1999). Moreover, ADHD also seems to share a genetic predisposition to other psychiatric problems (Cole et al. 2009; Lichtenstein et al. 2010; Thapar et al. 2001). However, the inherited factor is not the only explanation of ADHD. Environmental risk factors also play an important role (Thapar et al. 2012). We found a significant association between SHS exposure and ADHD in children from the studies that adjusted for parental psychiatric history. It further suggested that exposed to SHS may be a risk factor for ADHD in children. In addition, ADHD may be associated with indicators of social and economic disadvantage, including shorter parental education, lower family income, and younger parents (Cho et al. 2018). Animal studies also revealed a link between postnatal SHS exposure and ADHD. Slotkin et al. (2006) observed that changes in brain cell development in rhesus monkeys exposed to environmental SHS during perinatal or early life were similar to those seen in rodents exposed to nicotine before birth. However, studies on SHS exposure and ADHD in children are still controversial. A small number of studies found no association between SHS exposure and ADHD (Braun et al. 2006; Froehlich et al. 2009; Rückinger et al. 2010). Bandiera et al. (2011) pointed that SHS after birth was positively correlated with ADHD symptoms but could not be directly diagnosed as ADHD.

Table 2 Subgroup analysis of association between exposure to SHS and ADHD in children

	No. Studies	OR (95% CI)	I^2 (%)	p for Heterogeneity
Study design				
Cross-sectional	6	1.71(1.38, 2.12)	50.9	0.07
Cohort	2	1.35(1.03, 1.78)	30.7	0.23
Region				
USA	3	1.49(1.29, 1.72)	0	0.561
Korea	2	1.55(1.11, 2.17)	0.2	0.317
Europe	4	1.81(1.23, 2.65)	73.2	0.011
ADHD diagnosis				
Clinical diagnosis	5	1.50(1.31, 1.71)	0	0.698
SDQ	4	1.81(1.23, 2.65)	73.2	0.011
Adjusted by age and gender				
Yes	4	1.53(1.24, 1.88)	34.8	0.204
No	5	1.69(1.31, 2.18)	54.1	0.069
Maternal prenatal smoke				
Yes	7	1.53(1.30, 1.80)	30.7	0.194
No	2	1.86(1.18, 2.93)	71.1	0.063
Parental educational level				
Yes	4	1.50(1.29, 1.74)	0	0.531
No	5	1.71(1.29, 2.26)	65	0.022
Parental mental health				
Yes	4	1.82(1.36, 2.45)	24.4	0.265
No	5	1.53(1.27, 1.84)	50.6	0.088
Total	9	1.61(1.37, 1.88)	42.5	0.084

Most of the studies used parent-reported tobacco use to determine SHS exposure of children. It often causes the misclassification from recall bias and response bias. To reduce such bias, some studies used cotinine as an objective indicator of tobacco exposure to assess the association between SHS exposure and ADHD in children (Max et al. 2013). As the main plasma metabolite of nicotine, cotinine is currently considered to be the best biomarker for tobacco smoke exposure (Seccareccia et al. 2003). However, some studies shown that cotinine has a short half-life (less than 24 h). Therefore cotinine cannot be used as an appropriate biomarker for the long-term effects of SHS (Jarvis et al. 1988). In order to explore the relationship between cotinine level in children and ADHD, we selected related studies from the same database and conducted a meta-analysis (Braun et al. 2006; Joo et al. 2017; Kim et al. 2018; Max et al. 2013). We observed a lower pooled OR (OR = 1.16, 95%CI = 1.01, 1.33) which indicated that there was a low association between cotinine and ADHD. High heterogeneity was observed across selected studies (I^2 = 89.6%, $p < 0.01$) (Fig. S6). This may be due to the lack of original studies and the difference in measurement accuracy among different periods (Max et al. 2013). Moreover, there are differences in the concentration of cotinine in blood, urine, and saliva (Torres et al. 2018). Inconsistencies in cotinine sources can lead to reduced comparability among various studies. Nevertheless, other studies (Gehring et al. 2006) suggested that the agreement between cotinine concentrations and questionnaire reports of smoking was good. Despite some misclassification, the questionnaire report remains a valid measure for estimating SHS exposure in preschool and school children.

Our study has several advantages. To our knowledge, this is the first meta-analysis assessing the relationship between postnatal SHS exposure and ADHD in children. Although there was moderate heterogeneity among our included studies, we found a positive association between postnatal exposure to SHS and ADHD in children in our subgroup analysis. It is indicated that our findings are highly reliable. Moreover, we also found that there may be a link between SHS exposure and some adverse behavioral problems in children. Tistarelli et al. (2020) reported that some behavioral problems often co-occur with ADHD. Therefore, the results of our study can provide a clue and basis for the mechanism research of ADHD in the future.

The limitations of our study should also be considered. First of all, most of the studies included were cross-sectional studies, with relatively low levels of evidence quality. Second, the language of included studies was limited to English and probably could not reflect all the results. Third, the diagnosis of ADHD is a comprehensive process that requires a detailed

assessment of current and previous symptoms. It is insufficient to diagnose ADHD using a single assessment scale. Different assessment methods for ADHD are also prone to bias. Fourth, measurements of SHS exposure are largely based on subjective reports, which may lead to a misclassification of SHS exposure. Due to the large differences in the classification of SHS exposure in selected studies, we were unable to combine these studies to explore the dose-response relationship between SHS exposure and ADHD in children.

Conclusions

The results of our meta-analysis suggest that postnatal exposure to SHS may be associated with ADHD in children. Exposure to SHS can also lead to a variety of adverse behavioral outcomes in children. Therefore, parents should stop smoking to create a good growing environment for their children. Further prospective studies should fully adjust for potential confounding factors to determine whether there is a causal relationship between SHS and ADHD. In addition, the mechanisms by which passive smoking increases the risk of ADHD in children also need to be elucidated by more high-quality researches.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest

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