
Article:
Peixinho, Jessica, Toseeb, Umar orcid.org/0000-0002-7536-2722, Mountford, Hayley S et al. (2 more authors) (2022) The effects of prenatal smoke exposure on language development- a systematic review. Infant and child development. e2331. ISSN 1522-7227

https://doi.org/10.1002/icd.2331

Received: 25 May 2021 | Revised: 4 March 2022 | Accepted: 19 April 2022

DOI: 10.1002/icd.2331

REVIEW ARTICLE

WILEY

The effects of prenatal smoke exposure on language development - a systematic review

Jessica Peixinho[1] | Umar Toseeb[2] | Hayley S. Mountford[1] | Isabel Bermudez[1] | Dianne F. Newbury[1]

[1]Faculty of Health and Life Sciences, Oxford Brookes University Headington Campus, Oxford, UK

[2]Department of Education, University of York, York, UK

Correspondence
Dianne F. Newbury, Faculty of Health and Life Sciences, Headington Campus, Oxford Brookes University, Gipsy Lane, Oxford OX3 0BP, UK.
Email: diannenewbury@brookes.ac.uk

Funding information
Leverhulme Trust, Grant/Award Number: RPG-2017-381

Handling Editor: Emily Farran

Abstract

The negative health effects of cigarette smoking during pregnancy (SDP) on the foetus are well known. Despite previous reports of poor cognitive performance in offspring exposed to SDP, few studies specifically consider language outcomes according to maternal smoking. In this study, we systematically review the literature to assess the relationships between SDP and child language. Of the 14 studies reviewed, 13 (93%) reported significant associations between maternal smoking or exposure and language outcomes. Despite this consistent association, only 8 of the 13 studies reporting associations (62%) concluded direct relationships between exposure and outcome. The remaining studies suggested that the relationship between smoking and language could be explained by factors such as maternal IQ, socioeconomic status (SES) and parental age. Future studies should apply careful study designs allowing for confounding factors across child, parental, environmental and genetic influences. Our review suggests that smoking cessation is likely to positively affect child language outcomes.

Highlights

- Does maternal smoking during or exposure to smoking during pregnancy affect the language outcomes in exposed offspring?

- A systematic review of the literature highlighted consistent negative effects of smoking or smoke exposure during pregnancy on language outcomes.
- Exposure to SDP is associated with language. Mothers must be educated regarding the effects of tobacco smoking on language outcomes.

KEYWORDS
exposure, language, maternal, prenatal, smoke

1 | INTRODUCTION

A variety of evidence shows that smoking while pregnant can lead to adverse effects on the mother and foetus (Nuffield Trust, 2019). This evidence has led to widespread medical and societal sanctions against tobacco smoking during pregnancy (SDP) that correlate with a reduction in women who smoke at the time of delivery; in England in 2019–2020, 10.4% of pregnant women smoked at the point of delivery compared to 14.2% ten years earlier (2009–1010) (Nuffield Trust, 2019). More recently, the emergence of new tobacco products, including electronic cigarettes and hookah, has become common among youth and women of reproductive age women with the potential to increase rates of infants born exposed to nicotine or tobacco (Bowker et al., 2021).

A recent study predicted that the prevalence of smoking in England is projected to decrease to 10.8% by 2022, down from 14.4% in 2018 (Song, Elwell-Sutton, & Naughton, 2020). However, significant differences have been reported across socioeconomic groups (Song, Elwell-Sutton, Naughton, & Gentry, 2020) whereby individuals from lower socioeconomic backgrounds are more likely to smoke.

Nicotine is the major reinforcing component of tobacco smoke. Nicotine is an alkaloid naturally found in the nightshade family of plants, including the tobacco plant and primary exposure to this chemical is through active or passive smoking (Fagerström, 2014). In the brain, nicotine binds nicotinic acetylcholine receptors (nAChR) activating the reward system and exerts its action in the brain through $\alpha4\beta2^*$ nAChR (*denotes possible assembly with other nicotinic subunits) (Tapper et al., 2004).

The various negative effects of smoking have been long established and well reported. Smoking not only impacts the individual but also others in proximity. Maternal smoking and exposure to cigarette smoke during pregnancy remain a substantial health concern. It is estimated that 1.7% of pregnant women worldwide and 8.1% of pregnant women in Europe smoke (Lange et al., 2018). The effects of maternal smoking on new-born children are also of concern as studies have shown that infants nursed by smoking mothers have detectable amounts of nicotine and cotinine (the primary metabolite of nicotine) in their serum and urine (Luck & Nau, 1985). In addition, longer breastfeeding duration has been linked to more favourable outcomes on cognitive development (Kim & Choi, 2020) which may hence encourage more mothers, despite their smoking status, to breastfeed.

Maternal SDP has well-established and direct negative effects on birth outcomes including low birth weight and preterm birth (Salihu & Wilson, 2007), both of which are markers of foetal health and are associated with neurological and psychiatric outcomes (Hack et al., 2005). Prenatal and early nicotine exposure is further associated with negative perinatal health including respiratory and ear infections, asthma, reduced cognitive function and behavioural difficulties (DiFranza et al., 2004) which may have serious health implications. In terms of neurodevelopmental disorders, in utero smoke exposure is primarily associated with an increased risk of Attention Deficit Hyperactivity Disorder (ADHD) (odds ratio [OR] = 2.39) (Langley et al., 2005), (pooled risk ratio [RR] = 1.58) (He et al., 2020) and Conduct Disorder (OR = 2.06) (Ruisch et al., 2018) and has been reported to increase the risk of schizophrenia by

29% (Hunter et al., 2020). Smoking is associated with some subtypes of Autistic Spectrum Disorders (ASD) such as Pervasive Developmental Disorder (PDD) (Tran et al., 2013) and ASD-not otherwise specified, (Kalkbrenner et al., 2012), although these findings were not supported by meta-analyses (Jung et al., 2017; Rosen et al., 2015; Tang et al., 2015). Interestingly, a single study has reported that the genetic risk of dyslexia modulates the performance of memory in interaction with maternal SDP, although this was only true for variation within a single candidate gene of five studied by this group, *DYX1C1* (Mascheretti et al., 2013; Mascheretti et al., 2015). Furthermore, smoking was not identified as an independent risk factor in another study of environmental contributors to dyslexia by the same group (Mascheretti et al., 2013) or by systematic reviews of risk factors in dyslexia (Becker et al., 2017; Mascheretti et al., 2018).

There are many mechanisms by which exposure to tobacco, containing many thousands of chemicals, may influence cognitive development. Smoking reduces foetal blood flow and oxygen levels and nicotine has been found to be a teratogen in animals in whom it crosses the placenta and stimulates foetal cholinergic neurons affecting neuronal migration, synaptogenesis and apoptosis (Dwyer et al., 2008). Additionally, nicotine and other chemicals present in tobacco smoke can affect critical cellular processes such as protein synthesis and enzyme activity (Dempsey & Benowitz, 2001; Jauniaux et al., 2001). Nonetheless, it has been highlighted that the ascertainment of a direct causal relationship between SDP and offspring outcomes, such as ADHD or conduct problems requires careful study design (Rice et al., 2018) in terms of phenotype (Clifford et al., 2012) and exposure (Jung et al., 2017) measurement as well as the avoidance of confounding factors that are associated with both exposure and outcome. In particular, inherited factors, maternal IQ and socioeconomic status have all been shown to increase the likelihood of starting to smoke (Agrawal et al., 2008; Hiscock et al., 2012; Reid et al., 2010) and may act as confounders as these factors are also associated with an increased risk of neurodevelopmental disorder (Batty et al., 2006; Özmert et al., 2005; Thapar et al., 2009). Fifty years ago, the smoking prevalence for all education groups was consistent, with nearly 40% of degree level educated individuals and approximately 45% of individuals in all other education groups smoking. Recently, this has decreased to 6.5% of degree level and 23.1% of individuals with a high school education (secondary school) or less in the US (Drope et al., 2018). The latter pinpoints the importance of education and SES as a confounder when considering smoking but many other confounders exist and, importantly, can have bidirectional effects. As such studies that do not adjust for confounder factors can overestimate the association between smoking and cognition (Batty et al., 2006). In particular, shared genes, environments and behaviours can all influence language and SDP.

There are well-established ages by which most linguistic developmental milestones are expected to be achieved. Active vocabulary begins to develop in the second year. Indeed, after the first year of life, word comprehension increases rapidly and a child's ability to understand language largely surpasses their ability to produce it (Fenson et al., 2000). The time when children begin school, at around 6 years, is considered vital for their cognitive development. Introduction to teaching alters the linguistic input to which a child is exposed (Riva et al., 2000) and by the age of 6 children have a well-developed vocabulary that is vast and have complete phonological production ability (Hoff, 2009). In addition from early childhood (6 years) to puberty (around 12 years), strategies for generating and integrating information emerge, including more sophisticated use of language through the use of more complex sentences and grammar (Rosselli et al., 2014) Considering the above, the ages of 2 to 12 years appears particularly relevant regarding language trajectories.

In the current study, we use a systematic review design to investigate the relationship between maternal smoking or smoke exposure and childhood language development. In a previous study of 1102 children, Tomblin and colleagues reported that maternal and paternal SDP were associated with an increased risk of developmental language disorder (DLD) (Tomblin et al., 1997; Tomblin et al., 1998). However, in line with the studies described above, this association disappeared when parental education was included in their model. This leads to the conclusion that parental smoking is not independently associated with DLD (Tomblin et al., 1998). Eicher and colleagues found that children exposed to prenatal nicotine performed 4.8%–5.4% worse on language tasks (Nonword repetition and verbal comprehension) at age 8 than children without smoke exposure (Eicher et al., 2013). They further reported that language performance was dosage-sensitive with regard to the level of prenatal exposure, as was the risk of language

impairment (LI) (exposure ≤17 mg/day nicotine LI OR = 1.25, exposure >17 mg/day, LI OR = 3.84). However they did not compare the number of cigarettes smoked or tar: nicotine ratio (Eicher et al., 2013). Social class and sex were included as covariates in these analyses but neither maternal education or IQ were directly controlled for (Eicher et al., 2013). Another study considering overall risk factors for LI has highlighted the importance of various risk factors such as very low birth weight (OR = 2.2), low 5 min Apgar score (OR = 2.0), lower maternal education (OR = 1.3–1.6), being an unmarried mother (OR = 1.4), and later stage of commencement of prenatal care (OR = 1.2–1.3) in the risk of the development of LI (Stanton-Chapman et al., 2002) This study, however, did not conclude tobacco use to be a major risk factor for development of LI (OR = 1.0) (Stanton-Chapman et al., 2002).

Although other studies may include language and communication as part of their consideration of cognition or neurodevelopment, there are few which focus primarily on language development or disorder in relation to smoking. Studies have demonstrated discrepancies in language development compared to other cognitive abilities and indeed other communication abilities. A study of early communication development in toddlers highlighted differing developmental patterns in their levels of social, speech, and symbolic skills (Maatta et al., 2012). Considering smoking's links to cognition and the fact that smoking has been shown to impair synaptic maturation in the auditory brainstem which in turn may affect auditory processing (Baumann & Koch, 2017) it is important to consider language in a smoking context. In addition, the critical role of language in the overall development of the child highlights it is vital to robustly examine the association between smoking and language development. In this systematic review, we aim to examine published studies, which consider language outcomes after prenatal exposure to nicotine.

2 | METHODS

2.1 | Sources

A systematic review of journal articles published between the years 2000 and 2020 was conducted. Web Of Science (https://clarivate.com/webofsciencegroup/solutions/web-of-science/) and Pubmed (https://pubmed.ncbi.nlm.nih.gov/) were searched using comprehensive search strategies as detailed below. The reference lists of identified articles were also searched to identify additional relevant references. Data collection was completed between February and March 2021.

2.2 | Search strategy

(((((([Child*]) AND ((((((((((((((([Develop*]) OR ([Language])) OR ([languages*])) OR ([Language*])) OR ([Language[MeSH Terms]])) OR ([Neuro*])) OR ([Vocab*])) OR ([Grammar])) OR ([Attention deficit hyperactivity disorder])) OR ([Attention deficit hyperactivity disorder[MeSH Terms]])) OR ([Autis*])) OR ([Dyslex*])) OR ([Dysprax*])) OR ([Speech])) OR ([Speech[MeSH Terms]]))))) AND ((((((((([Nicotin*]) OR ([Cigar*])) OR ([tobacco products])) OR ([Tobacco products[MeSH Terms]])) OR ([tobacco])) OR ([tobacco[MeSH Terms]])))))) AND ((((((((((([Parent*]) AND ([Smok*]))) OR ((([Matern*]) AND ([Smok*])))) OR ((([Passive]) AND ([Smok*])))) OR ((([secondhand]) AND ([Smok*])))) OR (((secondhand) AND (smok*)))) OR ((([household]) AND ([Smok*])))) OR ((([household*]) AND ([Smok*]))))))) AND ((((((((((([prenatal*]) OR ([prenatal])) OR ([pregnan*])) OR ([uterus])) OR ([utero])) OR ([uterus[MeSH Terms]])) OR ([Mother])) OR ([mothers])) OR ([mum])) OR ([Mothers[MeSH Terms]])))) AND ([Expos*]).

The same search terms were used in the Web Of Science, with the exception of the inclusion of MeSH terms as this is not available on this platform. Filters applied to both were that these studies must have been conducted in the last 20 years, the study must be in English, and outcomes should be articles or letters. In Web Of Science, no measures were included at the search stage to exclude animal studies as there was no clear option in its search engine but any animal studies were excluded at further stages.

The search was limited to studies conducted from the year 2000 to 2020 to make the search more manageable. The search terms included neurodevelopmental disorders such as ADHD and ASD due to the fact that language development is usually relevant to these conditions. In order to draw conclusions and make comparisons between papers, only studies that specifically discussed language outcomes were included in the final analyses.

Papers yielded from these searches were examined in two stages. The first considered only information in the title and abstract and acted as a broad screen to exclude non-relevant results. A second stage considered more detailed information from the full text and screened for in-depth details of the study design. The same inclusion and exclusion criteria were employed across both stages, as detailed below.

Inclusion criteria:

1. Human study population.
2. Examined prenatal exposure to smoking or nicotine.
3. Study includes an assessment of nicotine measures obtained during pregnancy and up to 6 months of age (to help ensure better memory of events).
4. Study considered specific measures of language as an outcome (this did not need to be the primary focus of the study).

Exclusion criteria:

1. Paper not in English.
2. Paper was a review, systematic review, opinion piece or meta-analysis.
3. Language outcomes were tested before 2 or after 12 years.
4. Study considered only an neurodevelopmental disorder or broad cognition (no specific measures of language were considered).
5. Paper focused upon prenatal drug use or factors other than nicotine exposure.
6. Study participants were selected to have a certain disorder.
7. Paper could not be accessed.

2.3 | Study selection and data extraction

Two authors independently screened titles and abstracts for all search results. Discordant decisions were resolved by further assessment of paper content and discussion between the authors. All papers which met the inclusion criteria above were catalogued in detail noting the size of the study population, ascertainment criteria, how nicotine was measured, age of children considered in the analyses, outcome measurements, and the confounders identified. Quality assessments of the studies were conducted according to the Kuyper, (1991) checklist (Table S1). At this stage, further studies were excluded if the language outcome was not verbal, if the age of the child's language assessment did not fall into the range above, if the exposure did not specifically consider maternal SDP or exposure or if they did not meet the quality criteria. Where multiple studies in the final list used the same cohort, one study was selected on the basis of the relevance of the outcomes studied and the sample size.

3 | RESULTS

Initial literature searches yielded 1376 studies from the Web Of Science and 911 articles from PubMed (Figure 1). After title and abstract review, 420 studies were taken forward for a full review. Of these, 134 were found to be

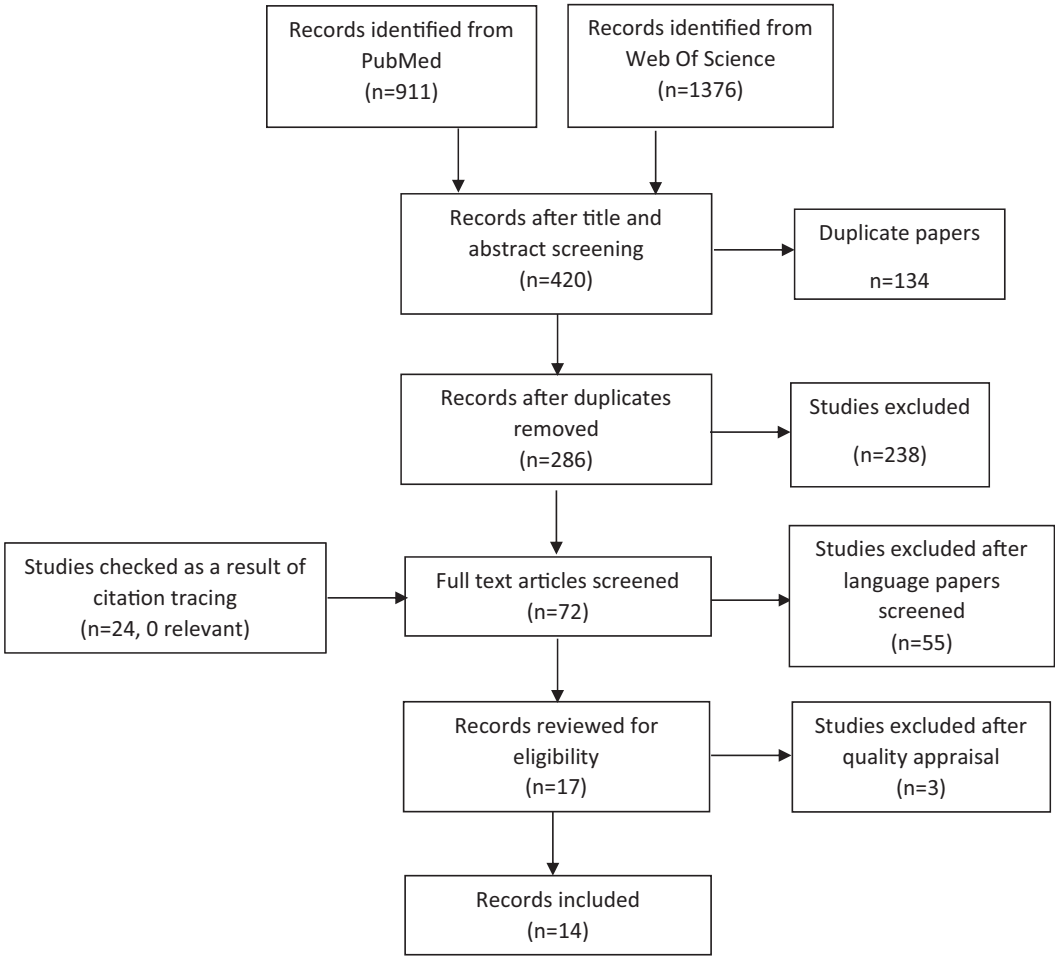

FIGURE 1 Flow chart of the study screening process. See text for details of inclusion and exclusion criteria at each stage.

duplicate studies that were deposited in both PubMed and Web Of Science. After removing duplicate studies, 286 papers were examined in greater detail. The study exposure and outcome measurements were evaluated for their relevance to the aims of this systematic review (consideration of child language and nicotine exposure during pregnancy). This screening led to the exclusion of a further 238 studies and the addition of 24 further papers identified from citation tracing, leaving 72 papers that were then taken forward for an in-depth full-text review. At this stage, full study design and outcomes were recorded and studies, which met our full inclusionary criteria (as detailed in methods; primarily a measurement of a verbal language outcome between the ages of 2 and 12 years, relation of this outcome to maternal tobacco SDP or exposure) were retained leaving 17 articles. Additional reviews were performed to ensure that the studies met high-quality research (as defined by Kuyper, 1991) and to confirm that no one cohort was represented twice. These additional screens led to the exclusion of three further studies, leaving 14 papers, which were then taken forward to the systematic review (Tables 1 and S1). All papers were independently screened by two authors at each screening stage. Classification concordance was 85% across all stages. A summary of study findings is shown in Figure 2.

TABLE 1 Fourteen studies are included in the systematic review (method details).

Author (year)	Title (PMID)	Sample ascertainment and size	How was smoking assessed (classification groups)?	Outcome variables
MacArthur et al. (2001)	Effects at age 9 of maternal smoking in pregnancy: experimental & observational findings (11213007).	$n = 1218$. American population cohort ascertained for RCT of smoking. Recruited from antenatal clinics.	Maternal questionnaire post-delivery. (non-smokers, stopped by 6 weeks, stopped 7–16 weeks, stopped >17 weeks, persistent smokers).	Clinical assessment of VIQ* at 9–11 years (mean = 9.4 years) - British Ability Scales (BAS).
Huijbregts et al. (2006)	Interrelations between maternal smoking during pregnancy, birth weight & sociodemographic factors in the prediction of early cognitive abilities (28360824).	$n = 1544$. Canadian prospective birth cohort (Québec Longitudinal Study of Children's Development).	Maternal questionnaire, 5 months after birth (non-smokers, 1–9, ≥10 cigarettes/day).	Clinical assessment of vocabulary at 3.5 years - Peabody Picture Vocabulary Test (PPVT).
Julvez et al. (2007)	Maternal smoking habits & cognitive development of children at age 4 years in a population-based cohort (17550944).	$n = 420$. Spanish prospective birth cohort. Recruited all women presenting for antenatal care over a 12-month period.	The maternal repeated questionnaire, 3rd trimester to 4 years. (non-smoker, pregnancy smoker (>1 cigarette in pregnancy), post-natal (but not pregnancy) smoker).	Clinical assessment of VIQ at 4 years - McCarthy Scales of Children's Abilities (MCSA, Spanish-adaptation).
Alati et al. (2008)	Intrauterine exposure to alcohol & tobacco use and childhood IQ: Findings from a parental-offspring comparisonwithin ALSPAC (18670372).	$n = 4332$. UK prospective birth cohort (Avon Longitudinal Study of Parents & Children - ALSPAC).	Mother & partner repeated questionnaire, 1st trimester (non-smoker, 1–9 cigarettes/day, 10–19 cigarettes/day, 20+ cigarettes/day)	Clinical assessment of VIQ at 8 years - Wechsler Intelligence Scale for Children (WISC), abbreviated.
Gilman et al. (2008)	Maternal smoking during pregnancy & children's cognitive & physical development: a causal risk factor? (18653646).	$n = 35{,}566$. American prospective birth cohort study (the Collaborative Perinatal Project - CPP).	Maternal repeated questionnaire, 1st trimester to birth (non-smoker, 1–9 cigarettes/day, 10–19 cigarettes/day, 20+ cigarettes/day).	Clinical assessment of VIQ at 7 years - Wechsler Intelligence Scale for Children (WISC), abbreviated.
Hsieh et al. (2008)	*CYP1A1* Ile462Val & *GSTT1* modify the effect of cord blood cotinine on neurodevelopment at 2 years of age (18577398).	$n = 145$. Taiwanese prospective birth cohort (Taiwan Birth Panel Study).	Cord blood cotinine levels at delivery. (unexposed (<0.16 ng/mL), exposed (0.16–14 ng/mL).	Clinical assessment of developmental indices at 2 years - Comprehensive Developmental Inventory for Infants & Toddlers (CDIIT), language scale.
Heinonen et al. (2011)	Longitudinal study of smoking cessation before pregnancy & children's cognitive abilities at 56 months of age (21397413).	$n = 973$. Finnish prospective birth cohort (Arvo Ylppö Longitudinal Study - AYLS).	Mother & partner questionnaire at birth (non-smokers, pre-pregnancy (1–10 cigarettes/day), pre-pregnancy (>10 cigarettes/day), persistent smoker).	Clinical assessment of vocabulary & receptive comprehension at 56 months - Verbal competence test (Finnish) & Logopädischer Sprachverständnis Test (LSVT).

(Continues)

TABLE 1 (Continued)

Author (year)	Title (PMID)	Sample ascertainment and size	How was smoking assessed (classification groups)?	Outcome variables
Eriksen et al. (2012)	Effects of tobacco smoking in pregnancy on offspring intelligence at the Age of 5 (23316364)	$n = 1782$. Danish prospective birth cohort (Lifestyle During Pregnancy Study, subset of Danish National Birth Cohort).	Maternal questionnaire, 17 gestational weeks (non-smokers, 1–9 cigarettes/day, ≥10 cigarettes/day).	Clinical assessment of VIQ at 5-years - Danish abbreviated version of the Wechsler Primary & Preschool Scales of Intelligence Revised (WPPSI-R).
Hernandez-Martinez et al. (2017)	Effects of prenatal nicotine exposure on infant language development: A cohort follow up study (27465062).	$n = 92$. Prenatal sample recruited for a single-site study of the effects of prenatal smoke exposure on cognition at Sant Joan University Hospital in Reus (Spain). Women recruited at <11 weeks of singleton pregnancy with no complications.	Maternal repeated questionnaire, trimesters 1,2,3 (non-smokers, smokers, exposed to second-hand smoke).	Clinical assessment of language & vocabulary at 30 months - Bayley Scales of Infant Development (BSID-II), MacArthur Bates Communicative Development Inventory & Peabody Picture Vocabulary Test (Spanish adaptation, PPVT-III).
Mohamed et al. (2018)	Early life second-hand smoke exposure assessed by hair nicotine biomarker may reduce children's neurodevelopment at 2 years of age (28803192).	$n = 107$. Malaysian prenatal sample from a single hospital site for a study on maternal–infant adiposity. Women were recruited in the second trimester with no complications.	Direct measure of nicotine levels in maternal hair samples 1–5 days after delivery (quantitative).	Parental questionnaire of language at 2 years - communication scale of Ages & Stages Questionnaire (ASQ-3, translated Malay version).
Polanska et al. (2017)	Environmental tobacco smoke exposure duringpregnancy & child neurodevelopment (28714930).	$n = 292$. Polish prospective birth cohort (REPRO-PL).	Direct measure of maternal cotinine from saliva, 1st, 2nd & 3rd trimesters. The maternal questionnaire of home smoke exposure.	Clinical assessment of language at 2 years - Bayley Scales of Infant & Toddler Development (BSD-III, language scale).
Lee et al. (2019)	Exposure to prenatal second-hand smoke and early neurodevelopment: MOCEH study (30894196)	$n = 352$. South Korean prospective cohort (Korean multicentre birth cohort study, Mothers & Children Environmental Health, MOCEH).	Direct measure of maternal cotinine from urine 12–20 gestational weeks (cotinine levels below the median (≤1.9 ng/mL) or above median (>1.9 ng/mL)).	Clinical assessment of language at 2 years - Mental Development Index (MDI) of Korean version of Bayley Scale of Infant Development II (K-BSID-II).
Neumann et al. (2019)	A longitudinal study of antenatal & perinatal risk factors in early childhood cognition: Evidence from Growing Up in New Zealand (30974313).	$n = 4587$. New Zealand prospective birth cohort (Growing Up in New Zealand Study).	Maternal questionnaire, before pregnancy & 3rd trimester (smoker, non-smoker pre-pregnancy).	Clinical assessment of vocabulary at age 4.5 years - Peabody Picture Vocabulary Test (PPVT-III).
Moore et al. (2020)	Prenatal exposure to tobacco & offspring neurocognitive development in the healthy start study (31759580)	$n = 246$. American prenatal sample from a single hospital site for a study on neonatal adiposity (Healthy Start Study). Women were recruited at <24 weeks of pregnancy with no complications.	Direct measure of maternal cotinine from urine, 27 gestational weeks (non-smoker (below limit of detection), smoker (cotinine detected)).	Parental questionnaire of language at 4.5-years - communication scale of Ages & Stages (ASQ-3). Clinical assessment of receptive vocabulary at 4.5 years - picture vocabulary (NIH Toolbox).

Author (Year)	PMID	Sample size 92-999 1000-9999 >10000	Smoking measurement questionnaire direct measurement	Outcome measure	Significance P<0.05 P<0.001 P<0.0001 NS	Effect direct indirect no effect
MacArthur et al (2001)	11213007			VIQ 9-11yrs		
Huijbregts et al (2006)	28360824			Vocab 3-5yrs		
Julvez et al (2007)	17550944			VIQ 4yrs		
Alati et al (2008)	18670372			VIQ 8yrs		
Gilman et al (2008)	18653646			VIQ 7yrs		
Hsieh et al (2008)	18577398			Language 2yrs		
Heinonen et al (2011)	21397413			Vocab & comprehension 4-5yrs		
Eriksen et al (2012)	23316364			VIQ 5yrs		
Hernandez-Martinez et al (2016)	27465062			Language & vocab 2-3yrs		
Mohamed et al (2017)	28803192			Language 2 yrs		
Polanska et al (2017)	28714930			Language 2 yrs		
Lee et al (2019)	30894196			Language 2 yrs		
Neumann et al (2019)	30974313			Vocab 4-5yrs		
Moore et al (2020)	31759580			Language & vocab 4-5yrs		

FIGURE 2 Summary of broad study findings.

3.1 | Study findings

The 14 studies included in this review considered 11 different population nationalities: American, Spanish, Taiwanese, UK, Finnish, Canadian, Polish, Malaysian, South Korean, New Zealand, and Danish populations (Table 1). The combined number of participants across the 14 studies was 51,656, with the smallest study comprising 92 participants and the largest comprising 35,566.

Of the 14 studies included in this review, 13 (93%) reported negative associations between maternal prepregnancy smoking, smoking during pregnancy or exposure to smoke and childhood language outcomes (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Huijbregts et al., 2006; Julvez et al., 2007; Lee et al., 2019; MacArthur et al., 2001; Mohamed et al., 2018; Neumann et al., 2019; Polanska et al., 2017) (Figure 2, Table 2). Differences in study design and reporting methods make it difficult to directly compare effects between studies but six of the fourteen studies reviewed (43%) report highly significant effects (Alati et al., 2008; Gilman et al., 2008; Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Huijbregts et al., 2006; MacArthur et al., 2001) ($p \leq 0.001$) and seven found marginal effects ($0.001 < p < 0.05$) (Eriksen et al., 2012; Heinonen et al., 2011; Julvez et al., 2007; Lee et al., 2019; Mohamed et al., 2018; Neumann et al., 2019; Polanska et al., 2017) (Figure 2, Table 2). Although most studies found consistent associations, their conclusions differed; five concluded that the effects of smoking on child language could be explained by indirect effects, primarily socioeconomic in nature (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Huijbregts et al., 2006; MacArthur et al., 2001) (Figure 2, Table 2). The other eight studies reported a direct effect of nicotine levels on child language (Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Hsieh

TABLE 2 Fourteen studies were included in a systematic review (results summary).

Author (year)	Title (PMID)	*p*-value (effect size, where given)[a]	Study conclusion	Confounders and effects
MacArthur et al. (2001)	Effects at age 9 of maternal smoking in pregnancy: experimental & observational findings (11213007).	$p < 0.001$ (Max VIQ change $= -3.7$)	Significant association between maternal smoking and VIQ (persistent vs. stopped-smokers). The effect was indirect.	Parental factors (Mother's educational level and age), birth factors (birthweight, birth-length, head circumference, breastfeeding) and family/home factors (parity, home location, maternal employment) were independent predictors of VIQ. Association to smoking was accounted for by these variables.
Huijbregts et al. (2006)	Interrelations between maternal smoking during pregnancy, birth weight & sociodemographic factors in the prediction of early cognitive abilities (28360824).	$p < 0.001$ ($\beta \pm SE = -0.17 \pm 0.034$)	Significant association between maternal smoking and vocabulary. Effect was indirect	Parental factors (maternal education), birth factors (birthweight, gestation, sex) and family/home factors (family income) were independent predictors of vocabulary. Association to smoking was accounted for by maternal education and birth weight.
Julvez et al. (2007)	Maternal smoking habits & cognitive development of children at age 4 years in a population-based cohort (17550944).	$p = 0.03$ ($\beta = -0.59$, 95% CI $= -1.11$ to -0.07)	Marginal association between maternal smoking and VIQ. The effect was direct.	Parental factors (maternal education) and family/home factors (social class) were independent predictors of VIQ. Association to smoking remained after adjusting for these effects.
Alati et al. (2008)	Intrauterine exposure to alcohol & tobacco use and childhood IQ: Findings from a parental-offspring comparison within ALSPAC (18670372).	$p < 0.001$ (Mean VIQ change $= -2.63$, 95% CI $= -3.42$ to -1.84)	Significant association between maternal and paternal smoking and VIQ Effect was indirect.	Parental factors (Parental education), child factors (sex) and family/home factors (social class, parity, home ownership and house crowding) were independent predictors of VIQ. Association to smoking was accounted for by parental education.
Gilman et al. (2008)	Maternal smoking during pregnancy & children's cognitive & physical development: a causal risk factor? (18653646).	$p < 0.001$ (Max VIQ change $= -0.77$, 95% CI $= -1.12$ to -0.41, adjusted model)	Significant association between maternal smoking and VIQ Effect was indirect.	Parental factors (Mother's educational level, parental age, marital status, parental mental health) and family/home factors (social class, parity, maternal employment) were independent predictors of VIQ. Association to smoking remained after adjusting for these variables.

TABLE 2 (Continued)

Author (year)	Title (PMID)	*p*-value (effect size, where given)[a]	Study conclusion	Confounders and effects
Hsieh et al. (2008)	*CYP1A1* Ile462Val & *GSTT1* modify the effect of cord blood cotinine on neurodevelopment at 2 years of age (18577398).	$p < 0.0001$ ($\beta \pm SE = -10.15 \pm 2.24$)	Significant association between maternal cotinine levels and language. Effect was direct	Parental factors (maternal education and ethnicity) and family/home factors (income) were independent predictors of language. Association to smoking remained after adjusting for these effects.
Heinonen et al. (2011)	Longitudinal study of smoking cessation before pregnancy & children's cognitive abilities at 56 months of age (21397413).	$p < 0.05$ ($\beta = -12.83$, 95% CI $= -21.30$ to -4.35, pre-pregnancy smoking)	Marginal association between smoking >10 cigarettes/day before pregnancy and language comprehension. Effect was direct.	Parental factors (Parental education), birth factors (sex) and family/home factors (social class, parity, home ownership and house crowding) were independent predictors of comprehension. Association to smoking remained after accounting for these variables.
Eriksen et al. (2012)	Effects of tobacco smoking in pregnancy on offspring intelligence at the age of 5 (23316364)	$p < 0.05$ (max VIQ change $= -2.5$, 95% CI $= -4.7$ to -0.4)	Significant association between smoking >10 cigarettes/day and VIQ Effect was indirect.	Parental factors (parental education, maternal IQ, maternal age, maternal BMI), family factors (parity, smoke in house, parental marital status, home environment) were associated with child outcomes. Association to smoking was accounted for by these variables.
Hernandez-Martinez et al. (2017)	Effects of prenatal nicotine exposure on infant languagedevelopment: A cohort follow up study (27465062).	$p = 0.001$ (mean Language Development Age change $= -1.24$)	Significant association between smoking and language development. Effect was direct	Parental factors (maternal age) and family/home factors (social class) were independent predictors of language. Association to smoking remained after accounting for these variables.
Mohamed et al. (2018)	Early life second-hand smoke exposure assessed by hair nicotine biomarker may reduce children's neurodevelopment at 2 years of age (28803192).	$p = 0.025$ ($\beta = -1.920$)	Marginal association between hair cotinine level and communication. Effect was direct.	Parental factors (parental education), child factors (sex) and family factors (household income) were independently associated with the communication. Association to smoking remained after adjusting for these variables.
Polanska et al. (2017)	Environmental tobacco smoke exposure duringpregnancy & child neurodevelopment (28714930).	$p = 0.009$ ($\beta = -5.19$, adjusted model)	Marginal association between maternal cotinine levels in 1st and 2nd trimester and language development. Effect was direct.	Models were adjusted for parental factors (maternal IQ, maternal age, alcohol consumption), family factors (SES, parental marital status and parity) and birth factors (gestation, pregnancy complications, breastfeeding). Association to smoking remained after accounting for these variables.

(Continues)

TABLE 2 (Continued)

Author (year)	Title (PMID)	*p*-value (effect size, where given)[a]	Study conclusion	Confounders and effects
Lee et al. (2019)	Exposure to prenatal second-hand smoke and early neurodevelopment: MOCEH study (30894196)	$p = 0.04$ ($\beta = -2.73$, 95% CI $= -5.32$ to -0.15, adjusted model)	Association between urinary cotinine and language development. Effect was direct.	Parental factors (maternal education, maternal age), birth factors (birthweight, breastfeeding), family factors (home location) and genetic factors (polymorphisms in *GSTM1/GSTT1* genes) were associated with development. Association to smoking remained even after accounting for these variables.
Neumann et al. (2019)	A longitudinal study of antenatal & perinatal risk factors in early childhood cognition: Evidence from Growing Up in New Zealand (30974313).	$p < 0.05$ (OR language below expected $= 1.28$ (95% CI $= 1.04$–1.57, adjusted model, pre-pregnancy smoking).	Marginal association between smoking pre-pregnancy and receptive language. Effect was direct.	Parental factors (maternal anxiety/depression and maternal diet) were independently associated with vocabulary outcomes. Association to smoking remained even after accounting for these variables.
Moore et al. (2020)	Prenatal exposure to tobacco & offspring neurocognitive development in the healthy start study (31759580)	$p = 0.83$, OR $= 1.8$ (95% CI $= -3.0$ to 6.6, adjusted model)	No association between smoking and receptive vocabulary or communication difficulties.	Parental factors (maternal education, maternal age, maternal ethnicity), birth factors (birthweight and breastfeeding) and family factors (family income) were associated with language outcomes.

[a]Effect sizes are reported with non-smokers as the baseline. In many papers, multiple comparison groups (e.g., different smoking levels) and different outcomes were considered. In these studies, the maximum effect is reported. Effect sizes will not be comparable across studies. All effects reported are for unadjusted baseline models unless stated. See each individual paper for details of measures, models and effects.

et al., 2008; Julvez et al., 2007; Lee et al., 2019; Mohamed et al., 2018; Neumann et al., 2019; Polanska et al., 2017) which remained significant even after correcting for possible socioeconomic confounders (Figure 2, Table 2).

Seven (50%) investigations (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Heinonen et al., 2011; Lee et al., 2019; Mohamed et al., 2018; Polanska et al., 2017) explored the relationship between nicotine dosage and language and six of these reported stronger effects in groups who smoked heavily during pregnancy (Alati et al., 2008; Eriksen et al., 2012; Heinonen et al., 2011; Lee et al., 2019; Mohamed et al., 2018; Polanska et al., 2017). Similarly, six studies (43%) categorized language outcomes in relation to the point of nicotine exposure (Heinonen et al., 2011; Huijbregts et al., 2006; Julvez et al., 2007; MacArthur et al., 2001; Mohamed et al., 2018; Polanska et al., 2017) and four of these (67%) found that smoking before or during early pregnancy had the biggest effects on the outcome (Heinonen et al., 2011; Julvez et al., 2007; Mohamed et al., 2018; Polanska et al., 2017).

Only one study (7.1%) failed to find significant association between prenatal smoke exposure and language (Moore et al., 2020). This investigation included 246 individuals and considered prenatal cotinine levels (no exposure, $n = 181$ vs. exposure $n = 65$) in relation to dichotomised communication scores and a continuous measure of receptive vocabulary. Analyses were adjusted for possible confounders including maternal age, sex, race, annual household income, non-specified maternal psychiatric disorder and maternal daily caloric intake during pregnancy. They reported that children who were exposed to nicotine prenatally had a decreased inhibitory control and poor fine motor skills, however, no significant differences were found in terms of the language-specific outcomes mentioned above.

3.2 | Study design

The majority of the studies included in this review (11 of 14, 79%) were prospective birth cohort investigations, where mothers were recruited during pregnancy from multiple sites (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Heinonen et al., 2011; Hsieh et al., 2008; Huijbregts et al., 2006; Julvez et al., 2007; Lee et al., 2019; MacArthur et al., 2001; Neumann et al., 2019; Polanska et al., 2017) (Table 1). These population studies did not apply ascertainment criteria regarding maternal smoking and, instead, these data were collected as part of a broad investigative battery. Only one sample set was specifically ascertained to investigate the effects of smoking on cognition (Hernandez-Martinez et al., 2017). Two additional studies (Mohamed et al., 2018; Moore et al., 2020) also ascertained targeted sample sets, focussing on the effects of prenatal smoking on infant adiposity, although they also collected information regarding language development. These three targeted studies tended to have smaller sample sizes (mean $n = 148$, range 92–246) than the population-based studies (mean $n = 4655$, range = 145–35,566) but did not differ in their analytical approaches, which primarily relied upon regression modelling and included covariates for possible confounder effects.

3.3 | Nicotine exposure

The selection criteria applied within this systematic review specified that information regarding nicotine exposure had to be collected from mothers within 6 months of birth (see methods). However, the exact time-point of data acquisition differed between studies (Figure 2, Table 1). Five studies (36%) collected exposure data at a single time-point during pregnancy, three (21%) in the second trimester (14–26 weeks gestation) (Alati et al., 2008; Eriksen et al., 2012; Lee et al., 2019) and two (14%) in the third trimester (27–40 weeks gestation) (Moore et al., 2020; Neumann et al., 2019). Five further studies (36%) collected this information post-delivery; four within a week of delivery (Heinonen et al., 2011; Hsieh et al., 2008; MacArthur et al., 2001; Mohamed et al., 2018) and one study five months after birth (Huijbregts et al., 2006). The remaining four studies (29%) took repeated measures throughout

pregnancy in the first, second, and third trimesters (Gilman et al., 2008; Hernandez-Martinez et al., 2017; Polanska et al., 2017) and every year up to 4 years postnatally (Julvez et al., 2007).

Nine of the 14 studies (64%) used parental questionnaires to assess nicotine exposure (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Huijbregts et al., 2006; Julvez et al., 2007; MacArthur et al., 2001; Neumann et al., 2019) while 5 (36%) used direct measurement of cotinine; a metabolite of nicotine (Hsieh et al., 2008; Lee et al., 2019; Mohamed et al., 2018; Moore et al., 2020; Polanska et al., 2017) (Figure 2, Table 1). Direct measures can provide a more accurate measurement of exposure and allow exposure to be treated as a continuous variable enabling the investigation of possible dosage effects. Although in reality, only three studies (21%) performed a continuous regression (Lee et al., 2019; Mohamed et al., 2018; Polanska et al., 2017). Direct measurements were made using urine (Lee et al., 2019; Moore et al., 2020) or saliva samples at prenatal visits (Polanska et al., 2017), cord blood (Hsieh et al., 2008) or hair samples (Mohamed et al., 2018). Direct measurement is more expensive and time-consuming and this is therefore reflected in the sample sizes; studies which employed questionnaires tended to be larger than those with cotinine measurement (mean $n = 5612$, range = 92–35,566, compared to mean $n = 228$, range = 107–352 respectively). With the exception of Moore et al. (2020), all studies that employed cotinine measurements concluded that there was a direct effect between nicotine exposure and language outcomes, although these conclusions were always based upon results of marginal significance ($p > 0.001$) (Hsieh et al., 2008; Lee et al., 2019; Mohamed et al., 2018; Polanska et al., 2017). In contrast, studies that employed questionnaires reported both significant and marginal results with direct and indirect effects, regardless of the time point collected.

Six studies (43%) sub-categorized smokers in terms of the number of cigarettes smoked daily (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Heinonen et al., 2011) or by quantitative cotinine levels (Lee et al., 2019; Polanska et al., 2017). Five studies (36%) also considered the time point of exposure (prenatal, postnatal, or persistent) (Heinonen et al., 2011; Julvez et al., 2007; MacArthur et al., 2001; Mohamed et al., 2018; Polanska et al., 2017). The remaining five studies (36%) employed a binary consideration (smokers vs. non-smokers) (Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Huijbregts et al., 2006; Moore et al., 2020; Neumann et al., 2019). No obvious differences were observed in the findings across these studies in terms of the direction of effects or significance levels.

One difficulty in considering nicotine exposure is the challenge of distinguishing between direct exposure and environmental passive exposure (Jung et al., 2017). The use of maternal questionnaires considers only self-declared cigarette consumption, that is, active exposure. Whilst the direct measurement of cotinine quantifies both active and passive exposure levels, questionnaires were used to assess nicotine exposure in ten of the fourteen studies (71%) included in this review (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Huijbregts et al., 2006; Julvez et al., 2007; MacArthur et al., 2001; Neumann et al., 2019; Polanska et al., 2017). Six studies which employed questionnaires did attempt to address passive exposure through the use of paternal or home environment data (Alati et al., 2008; Eriksen et al., 2012; Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Huijbregts et al., 2006; Julvez et al., 2007; Polanska et al., 2017). One study (Hernandez-Martinez et al., 2017) reported non-significant effects of these environmental exposures while three studies reported significant effects (Alati et al., 2008; Eriksen et al., 2012; Huijbregts et al., 2006). The other two studies included these covariates in their models but did not report their significance.

3.4 | Language outcomes

Although all the studies in this review were screened and selected to consider child language development, the methods of ascertaining language ability varied between studies, as did the age of child assessment (Table 1). Five of the fourteen studies (36%) included in this review considered verbal IQ (VIQ) as a measure of language ability (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Julvez et al., 2007; MacArthur et al., 2001). The exact IQ test

varied between studies but each has overlapping subtests and represents direct clinical measures of language ability across a range of developmental domains. MacArthur et al. (2001) employed the British Ability Scales (Elliot et al., 1983) which includes subtests of word definitions and verbal similarities when children were 9–11 years (mean age = 9.4 years), Julvez et al. (2007) used McCarthy's Scales of Children's Abilities which includes the assessment of vocabulary, verbal memory, verbal fluency, and verbal similarities when children were 4 years (McCarthy, 1972). The remaining three studies used an abbreviated version of the Wechsler Intelligence Scales for Children (Wechsler, 1992, 2006), which considers verbal comprehension verbal reasoning, verbal memory, verbal fluency, vocabulary, and verbal similarities. These measurements were taken at 5 years (Eriksen et al., 2012), 7 years (Gilman et al., 2008) or 8 years of age (Alati et al., 2008).

Six other studies (43%) used broad assessment measures of language development at early ages, between 2 and 5 years of age (Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Lee et al., 2019; Mohamed et al., 2018; Moore et al., 2020; Polanska et al., 2017). Mohamed et al., (2018), and Moore et al., (2020) used the Ages and Stages Questionnaire (ASQ-3) which combines direct testing with parental questionnaires to assess language development and considers both language production and understanding. Mohamed et al., (2018) applied this test to assess early communication at 2 years of age, while Moore et al. (2020) used it to assess later communication at 4–5 years of age. Polanska et al. (2017), Hernandez-Martinez et al., (2017), and Lee et al. (2019) used the Bayley Scales of Infant Development (BSID) (Bayley, 1993; Park & Cho, 2006), a clinical assessment that can be used to capture development across mental and motor scales in young children (0–42 months). The Mental Development Index (MDI) of the Bayley Scales includes a specific scale of language development. While Hernandez-Martinez et al. (2017) and Polanska et al. (2017) employed the more focused language scale at the age of 1 and 2 years (of which we used the latter information). Lee et al. (2019) used the broader Mental Development Index at 2 years of age. Finally, Hsieh et al. (2008), used the Comprehensive Developmental Inventory for Infants and Toddlers (CDIIT) (Wang et al., 1998) at 2 years of age. This is a broad developmental battery, which consists of direct assessment across cognitive, emotional and motor domains and includes a language subscale.

Vocabulary forms a subtest of many of the batteries used above and has long been considered as a proxy for early language development. Five studies (36%) included in this review considered specific tasks of receptive vocabulary as an outcome measure (Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Huijbregts et al., 2006; Moore et al., 2020; Neumann et al., 2019). In two studies (Huijbregts et al., 2006; Neumann et al., 2019) vocabulary was the sole language outcome and was assessed with the Peabody Picture Vocabulary Test (PPVT) (Dunn et al., 1997; Dunn & Dunn, 1981) at 42 months (Huijbregts et al., 2006) or 54 months (Neumann et al., 2019). Three further studies (23%) considered receptive vocabulary alongside additional language measures. Hernandez-Martinez et al., (2017) combined the PPVT (Campbell et al., 2001) with the MacArthur-Bates Communicative Development Inventor (López Ornat et al., 2005), which focuses on vocabulary production and comprehension as well as a gesture. Alongside these two vocabulary tests, they also completed the BSID-II as described above. Each of these tasks was completed at different times across the ages of 6–30 months. Heinonen et al. (2011) included an alternative picture naming test verbal competence test alongside a language comprehension task (following instructions) at 56 months. Moore et al. (2020) used the picture vocabulary task from the NIH toolbox and combined this with the ASQ-3 index described above at 48, 54 and 60 months of age.

Studies that employed VIQ as a language outcome measure (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Julvez et al., 2007; MacArthur et al., 2001) generally considered an older age group (mean age 79 months, range 4 years to 11 years). In addition, these were more likely to report p-values ≤0.001 (Alati et al., 2008; Gilman et al., 2008; MacArthur et al., 2001) and indirect effects (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; MacArthur et al., 2001) than studies of developmental language indices (Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Lee et al., 2019; Mohamed et al., 2018; Moore et al., 2020; Polanska et al., 2017) or vocabulary (Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Huijbregts et al., 2006; Moore et al., 2020; Neumann et al., 2019); these generally involved testing at younger ages (mean age 34 months, range 2 to 5 years) and were more likely to report marginal p-values ($0.001 \geq p \leq 0.05$) (Heinonen et al., 2011; Hsieh et al., 2008; Lee

et al., 2019; Mohamed et al., 2018; Neumann et al., 2019; Polanska et al., 2017) and direct effects (Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Lee et al., 2019; Mohamed et al., 2018; Neumann et al., 2019; Polanska et al., 2017).

3.5 | Confounding effects

As outlined in the introduction, it has previously been argued that confounder effects, particularly maternal education/IQ may lead to the inflation of association between smoking and child development (Batty et al., 2006; Tomblin et al., 1998). Indeed, Stanton-Chapman (Stanton-Chapman et al., 2002) and colleagues have identified maternal education as a significant risk factor for LI. All of the 14 studies in this systematic review included some consideration of confounder effects by the inclusion of covariates within their models (Table 2). Some included covariates in their baseline model, other tested specifically for the effects of possible confounders. Common confounder effects can be split into child factors (including sex, ethnicity, health), birth factors (including prenatal and perinatal effects), family factors (such as SES, diet and parity), parental factors (such as education, age, alcohol consumption and environmental smoke exposure) and test factors (such as assessment point or evaluator).

In line with previous research, the most commonly identified significant confounder effects were SES and maternal education/IQ. Twelve studies (86%) included maternal education/IQ in their analyses (Alati et al., 2008; Eriksen et al., 2012; Heinonen et al., 2011; Hsieh et al., 2008; Huijbregts et al., 2006; Julvez et al., 2007; Lee et al., 2019; MacArthur et al., 2001; Mohamed et al., 2018; Moore et al., 2020; Neumann et al., 2019; Polanska et al., 2017) and eight of these (67%) (Alati et al., 2008; Eriksen et al., 2012; Hsieh et al., 2008; Huijbregts et al., 2006; Julvez et al., 2007; Lee et al., 2019; MacArthur et al., 2001; Moore et al., 2020) explicitly reported this to be a significant confounder, although it did not explain all of the variance in all of these studies. Two studies (14%) (Heinonen et al., 2011; Mohamed et al., 2018) reported this factor to be non-significant in their models. Twelve studies (86%) included indicators of SES such as home location, ownership, income and employment, in their analyses (Alati et al., 2008; Gilman et al., 2008; Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Huijbregts et al., 2006; Julvez et al., 2007; Lee et al., 2019; MacArthur et al., 2001; Mohamed et al., 2018; Moore et al., 2020; Neumann et al., 2019; Polanska et al., 2017) and nine of these (75%) reported it to be a significant confounder effect (Alati et al., 2008; Gilman et al., 2008; Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Julvez et al., 2007; Lee et al., 2019; MacArthur et al., 2001; Mohamed et al., 2018; Moore et al., 2020). Only one study (8%) (Huijbregts et al., 2006) reported SES to have no effect.

Other commonly identified confounders included parental age (maternal and/or paternal) birth weight, breastfeeding and environmental smoke exposure. Each of these factors was investigated in at least 8 of the 14 (57%) studies reviewed and was found to be significant by the majority.

Child sex is often considered as a confounding factor in studies of language development but was not reported to act as such in this instance. Thirteen studies (93%) included sex as a covariate and only two of these reported it as a significant confounder (Alati et al., 2008; Mohamed et al., 2018).

Other factors which were largely reported as non-significant were pregnancy complications (such as preeclampsia and gestational diabetes), maternal alcohol consumption, maternal body mass index (BMI) and study-related factors. These factors were consistently reported as non-significant in terms of confounder effects, although most were only included across a few of the studies reviewed (5 or less).

4 | CONCLUSIONS

Despite the vast literature regarding the effects of nicotine exposure on foetal health and child cognition, there is little research regarding direct effects on language development. In this systematic review, we screened over 1000 papers focused on 14 papers that specifically considered language outcomes in relation to *in-utero* nicotine exposure.

Thirteen of the 14 papers examined (93%) reported a negative association between maternal smoking or exposure and language outcomes (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Huijbregts et al., 2006; Julvez et al., 2007; Lee et al., 2019; MacArthur et al., 2001; Mohamed et al., 2018; Neumann et al., 2019; Polanska et al., 2017).

As with previous reports, there was some inconsistency regarding the nature of the relationship between maternal smoking or exposure and language development; eight studies concluded that smoking directly impaired early language (Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Julvez et al., 2007; Lee et al., 2019; Mohamed et al., 2018; Neumann et al., 2019; Polanska et al., 2017) while five concluded that the observed effects could be explained by confounding factors (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Huijbregts et al., 2006; MacArthur et al., 2001). These confounding factors varied between study designs making it hard to make a conclusion about the direction of effects.

Various studies have highlighted a strong correlation between IQ and education (Barber, 2005; Matarazzo & Herman, 1984; Ritchie et al., 2013). Commonly identified confounders included maternal IQ/education (significant confounder in eight of twelve studies that considered this factor; (Alati et al., 2008; Eriksen et al., 2012; Hsieh et al., 2008; Huijbregts et al., 2006; Julvez et al., 2007; Lee et al., 2019; MacArthur et al., 2001; Moore et al., 2020), SES (significant confounder in nine of twelve studies that considered this factor; (Alati et al., 2008; Gilman et al., 2008; Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Julvez et al., 2007; Lee et al., 2019; MacArthur et al., 2001; Mohamed et al., 2018; Moore et al., 2020) and parental age (significant confounder in six of ten studies that considered this factor; (Eriksen et al., 2012; Gilman et al., 2008; Hernandez-Martinez et al., 2017; Lee et al., 2019; MacArthur et al., 2001; Moore et al., 2020). Educational differences in smoking, with less-educated individuals being more likely to smoke, have been well documented in the literature (Cutler & Lleras-Muney, 2010; de Walque, 2007; Drope et al., 2018; Jürges et al., 2011; Kenkel et al., 2006; Maralani, 2013). Education is widely regarded as a driver of social progression and SES is often used as a proxy for education (Reilly et al., 2010). It is widely established that education and IQ, whilst different, are highly correlated at the behavioural level. An early study found that those who completed 16 years or more of education had a higher mean IQ (FSIQ = 115.3) than those who completed 12 years (FSIQ = 100.1) (Matarazzo & Herman, 1984). Another study reported that IQ was higher in countries, which extended education as indexed by secondary school enrolment and conversely that IQ was lower in countries with high levels of illiteracy (Barber, 2005). A more recent review has found that years of education were positively associated with IQ and that these associations continued into later life (Ritchie et al., 2013). Nonetheless, as previously discussed, it can be difficult to disentangle cause and effect within models that consider directly measured behaviours (as happened to be the case for all studies included in our systematic review). Correlations do not indicate causation and questions remain as to the direction of any causal effects, especially when those effects are transgenerational in nature. The recent application of Mendelian randomisation methods in large population cohorts has shown that the effects of cognitive ability upon smoking behaviour attenuate when educational attainment is introduced into the model. This finding indicates that the effects of educational attainment drive the relationship between cognition and smoking (Sanderson et al., 2019; Wells & Ostberg, 2021). However, an important limitation is noted for these findings in as much as they do not allow for transgenerational effects where parental education may have an effect on child smoking status that is not explained by the education level of the child (Sanderson et al., 2019). Importantly, the same dynastic effects could be applied to language and smoking where individual genetics directly affects parental language ability which then has an effect upon child language irrespective of smoking. Such complexities underline the need for careful study designs and well-powered cohorts when considering these effects (D'Onofrio et al., 2014).

Individuals living in low SES areas often have a higher level of tobacco use (Laveist et al., 2007; Reid et al., 2010; Zhang et al., 2013). SES, in turn, has been linked to reduced cognition (Özmert et al., 2005; Sarsour et al., 2011; Turkheimer et al., 2003) and lower academic achievement (Crosnoe et al., 2010; Marks, 2006). Similarly, maternal education level is associated with the academic and language abilities of children (Hanscombe et al., 2012; Reilly et al., 2010). None of these effects are linear and each involves many interacting factors making the complex relationships difficult to disentangle at the behavioural level (Batty et al., 2006; Puglisi et al., 2017).

Birthweight and breastfeeding were also commonly identified as confounders across the studies in this review. These two factors have also been previously related to child language and cognition (Hack et al., 1995; Kim & Choi, 2020). Extremely low birth weight has negative impacts that span both childhood and adulthood and has been described as a marker of the child's later neurological and psychiatric outcomes (Hack et al., 2005). Although smoking during breast-feeding has not been directly linked to cognition (Gibson & Porter, 2020), nicotine has been shown to transfer through breastmilk to the baby and also changes the composition and taste of milk (Napierala et al., 2016) which can lead to earlier weaning and lower weight (Horta et al., 2001) both of which, in turn, are associated with reductions in cognitive outcomes. These findings again highlight the complexity of these interacting effects and suggest that further studies will be required to disentangle these relationships at the behavioural level.

Existing studies have consistently suggested a small effect of biological sex on early language in favour of girls but this is reported to be dependent on age as well as the language component assessed (Bouchard et al., 2009; Simonsen et al., 2014; Thal et al., 2004). Conversely, it has been argued that there are more similarities than differences between genders regarding their language ability (Rhoda Kesler Unger, 2001). In this review, the biological sex of the child was not found to be a significant confounder by the majority of the 13 studies that included it in their adjustments. Our review identified a clear consensus that there is a dose-response effect of smoking on general health. All seven studies that considered differing doses of smoking found a negative dose–response relationship between prenatal smoking and language outcomes (Alati et al., 2008; Eriksen et al., 2012; Gilman et al., 2008; Heinonen et al., 2011; Lee et al., 2019; Mohamed et al., 2018; Polanska et al., 2017). These findings reflect those in the overall literature and are in line with those from animal and epidemiological studies (Hellstrom-Lindahl et al., 1998; Huizink & Mulder, 2006; Levin & Simon, 1998; Linnet et al., 2003; Weitzman et al., 2002). Animal studies similarly show that the neuronal effects of smoking are more pronounced at earlier gestational periods (Slotkin et al., 2015). The latter is reflected in the studies used in this systematic review in which four studies reported that smoking before or during early pregnancy had the biggest effects on the outcome (Heinonen et al., 2011; Julvez et al., 2007; Mohamed et al., 2018; Polanska et al., 2017).

Study design and sample size did not seem to affect the trends observed; one of the smallest studies in this review was the only one that failed to find an association (Moore et al., 2020). It should be noted however, that sample sizes can affect the relative effect sizes associated with any given *p*-value. Where reported, we include both effect size and *p*-value in Table 2. Perhaps unexpectedly, studies that employed direct measures of cotinine as a proxy of nicotine exposure (Hsieh et al., 2008; Lee et al., 2019; Mohamed et al., 2018; Polanska et al., 2017) generally had less significant findings than those which relied upon questionnaires. Direct measurement of nicotine by measurement of its major metabolite, cotinine, present in saliva, urine, or hair is often considered the "gold standard" for smoking detection as inconsistencies have been reported between self-report and cotinine concentrations (Britton et al., 2006). However, direct measurement methods also have limitations as cotinine only has a half-life of approximately 19–24 hours (Benowitz et al., 1983), and can be produced by nicotine replacement therapies such as nicotine patches, leading to false positives as the nicotine present in these is metabolized the same way. The reduced association in studies that employed direct measurements may reflect shared confounder factors between questionnaire data, smoking and language which would act to conflate the association between the two latter factors falsely increasing the association signal in studies, which rely upon questionnaire data. Conversely, it should also be noted that we restricted our review to include only studies that assessed smoking within 6 months of birth. This restriction was applied to maximize the reliability of smoking measures and hence the validity of our conclusions. Nonetheless, it is not necessarily true that retrospective reports are less reliable than contemporaneous measurements. In particular, since many women try to give up smoking during pregnancy, their memory of smoking habits during this period may show increased accuracy (Pickett et al., 2005). Studies show that the correlation between cotinine and contemporaneous reports is 70% (Petitti et al., 1981) and that retrospective reports are usually within 1%–3% of contemporaneous reports (Kenkel et al., 2003).

Just as exposure measurement may affect results, so may the choice of outcome measurement. In this review, we observed that studies, which employ measures of verbal IQ at later developmental stages (Alati et al., 2008;

Eriksen et al., 2012; Gilman et al., 2008; Julvez et al., 2007; MacArthur et al., 2001) reported stronger associations than investigations that employed early language indices or vocabulary measures (Heinonen et al., 2011; Hernandez-Martinez et al., 2017; Hsieh et al., 2008; Huijbregts et al., 2006; Lee et al., 2019; Mohamed et al., 2018; Moore et al., 2020; Neumann et al., 2019; Polanska et al., 2017). These studies also tended to report indirect associations that could be explained by confounder effects. There is debate in the literature as to the exact construct measured by each of the tests employed. Tests of VIQ assess the ability to access and apply acquired knowledge of words, including verbal concept formation, reasoning and expression rather than a specific construct of language itself (Lange et al., 2018). The age at which these tests are performed will also affect performance with different strategies typically applied to different age groups. Studies show that the heritability of intelligence increases over the life span reflecting a "genetic amplification" by which children select differential environments which act to compound genetic propensities (Plomin & Deary, 2015). This effect is also described in relation to language, where environmental factors account for a greater proportion of language variability earlier in development (Hayiou-Thomas et al., 2012; Tosto et al., 2017). Thus it could be argued that the strengthened association in older children again represents a falsely inflated association due to shared genetic confounders. This hypothesis is supported by research on ADHD where it has been suggested that genetically sensitive study designs, such as Mendelian randomisation, should be employed in the testing of causal hypotheses about prenatal exposure and offspring outcome (Rice et al., 2018; Thapar et al., 2009).

Finally, it should be noted that any systematic review is limited by its choice of search terms and papers included in the final review stages. Whilst our search terms were optimized to return relevant papers, they do not reflect the entire field. For example, we note that none of the 14 studies included in the final review stage employed a quasi-experimental design. This point is of particular relevance when considering confounder effects, which were noted as a primary influencing factor in our findings. All of the studies explored here employed a *post-hoc* adjustment to allow for specific measured confounder effects. In contrast, quasi-experimental methods allow for unmeasured confounders. Such studies indicate that associations between smoking during pregnancy and child cognition and behaviour may be explained by confounding factors rather than the direct effects of smoking (D'Onofrio et al., 2013). In particular, sibling comparison studies have shown that the association between smoking and reading outcomes can be explained by shared genetic and environmental factors (Ellingson et al., 2014; Micalizzi et al., 2021).

In conclusion, our systematic review finds consistent evidence for an association between maternal SDP or exposure and reduced language performance at early ages. However, the review also highlighted the complexities of the relationships within this process. Potential confounder factors include maternal IQ/education, SES, parental age, birth weight and breastfeeding and future studies should be carefully designed to account for these confounder effects. We observed strengthened relationships between smoking and language at points, which suggest inflation by study design rather than a true increase in association, again highlighting the need for careful study design supporting previous conclusions in this area (Thapar et al., 2009) and the findings of more sensitive approaches (D'Onofrio et al., 2013).

Despite systematic reviews upholding more robustly than other reviews, there are still limitations to be considered. Only studies in English and those with full text available were included meaning that potentially relevant studies may have been omitted. Additionally, despite the fact that efforts were made to carry out a broad and complete search, the possibility remains that some may have been overlooked. Only two databases were searched in this review and more could have been searched.

All of the studies included in our review used language measurements in population cohorts. Many of the studies we included looked at language as a corollary of cognition rather than focusing upon language itself meaning that outcomes differed between studies and none included clinical cohorts of language disorder. While it is possible that the findings here may be relevant to language disorder, it is also possible that risk effects differ between typical language development and language disorder. Previous studies (Eicher et al., 2013; Tomblin et al., 1997; Tomblin

et al., 1998) have suggested a link between smoking and language disorder but our review does not allow us to comment on the findings from this literature.

Future studies should aim to address weaknesses by considering careful study design which allows for confounding factors across child, parental, environmental and genetic influences. The network of effects underlying the associations identified here is so complex that more detailed studies of interactions between factors will be required. Such studies should extend beyond behavioural measurements and, if possible, include consideration of inherited effects (Thapar et al., 2009). Genetic and epigenetic effects were not considered in any of the papers we reviewed but, nonetheless can confer considerable risk for smoking, cognition and language and may interact with environmental factors to mediate outcomes (Agrawal et al., 2008; Newbury et al., 2009).

To conclude, this systematic review suggests a specific association between exposure to SDP pregnancy and language development. This may be used for the education of expectant mothers regarding the little-understood effects of tobacco smoking, including nicotine exposure specifically on language outcomes. Smoking cessation may help to optimize child outcomes in terms of language and would have positive effects on other aspects of child development bearing in mind that the most nicotine replacement drug strategies are nicotine mimetics.

AUTHOR CONTRIBUTIONS

Jessica Peixinho: Formal analysis; methodology; writing – original draft; writing – review and editing. **Umar Toseeb:** Conceptualization; methodology; supervision; writing – review and editing. **Hayley S. Mountford:** Methodology; supervision; writing – review and editing. **Isabel Bermudez:** Methodology; supervision; writing – review and editing. **Dianne F. Newbury:** Conceptualization; formal analysis; funding acquisition; investigation; methodology; project administration; supervision; validation; writing – original draft; writing – review and editing.

ACKNOWLEDGEMENTS

We are grateful to Nigel Groome and Oxford Brookes University for funding JPs studentship and to the Leverhulme Trust for funding research in the Newbury lab. We would like to thank all members of the Newbury lab for their feedback and advice on this research.

FUNDING INFORMATION

This research was funded by a Nigel Groome Studentship from Oxford University and funding from the Leverhulme Trust [RPG-2017-381] awarded to Dianne Newbury.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1002/icd.2331.

DATA AVAILABILITY STATEMENT

NA

ORCID

Jessica Peixinho https://orcid.org/0000-0002-5982-9986

REFERENCES

Agrawal, A., Pergadia, M. L., Waldron, M., Bucholz, K. K., Heath, A. C., Madden, P. A. F., Knopik, V. S., & Martin, N. G. (2008). Correlates of cigarette smoking during pregnancy and its genetic and environmental overlap with nicotine dependence. *Nicotine & Tobacco Research*, *10*(4), 567–578. https://doi.org/10.1080/14622200801978672

Alati, R., Macleod, J., Hickman, M., Sayal, K., May, M., Smith, G. D., & Lawlor, D. A. (2008). Intrauterine exposure to alcohol and tobacco use and childhood IQ: Findings from a parental-offspring comparison within the Avon longitudinal study of parents and children. *Pediatric Research*, *64*(6), 659–666. https://doi.org/10.1203/PDR.0b013e318187cc31

Barber, N. (2005). Educational and ecological correlates of IQ: A cross-national investigation. *Intelligence*, *33*(3), 273–284. https://doi.org/10.1016/j.intell.2005.01.001

Batty, G. D., Der, G., & Deary, I. J. (2006). Effect of maternal smoking during pregnancy on offspring's cognitive ability: Empirical evidence for complete confounding in the US national longitudinal survey of youth. *Pediatrics*, *118*(3), 943–950. https://doi.org/10.1542/peds.2006-0168

Baumann, V. J., & Koch, U. (2017). Perinatal nicotine exposure impairs the maturation of glutamatergic inputs in the auditory brainstem. *The Journal of Physiology*, *595*(11), 3573–3590. https://doi.org/10.1113/JP274059

Bayley, N. (1993). *Manual for the Bayley Scales for Infant Development* (2nd ed.). Psychological Corporation.

Becker, N., Vasconcelos, M., Oliveira, V., Santos, F. C. D., Bizarro, L., Almeida, R. M. M., Salles, J. F., & Carvalho, M. R. S. (2017). Genetic and environmental risk factors for developmental dyslexia in children: Systematic review of the last decade. *Developmental Neuropsychology*, *42*(7–8), 423–445. https://doi.org/10.1080/87565641.2017.1374960

Benowitz, N. L., Kuyt, F., Jacob, P., Jones, R. T., & Osman, A.-L. (1983). Cotinine disposition and effects. *Clinical Pharmacology & Therapeutics*, *34*(5), 604–611. https://doi.org/10.1067/mcp.2000.107086

Bouchard, C., Trudeau, N., Sutton, A. N. N., Boudreault, M.-C., & Deneault, J. (2009). Gender differences in language development in French Canadian children between 8 and 30 months of age. *Applied PsychoLinguistics*, *30*(4), 685–707. https://doi.org/10.1017/S0142716409990075

Bowker, K., Lewis, S., Ussher, M., Naughton, F., Phillips, L., Coleman, T., Orton, S., McRobbie, H., Bauld, L., & Cooper, S. (2021). Smoking and vaping patterns during pregnancy and the postpartum: A longitudinal UKcohort survey. *Addictive Behaviors*, *123*, 107050. https://doi.org/10.1016/j.addbeh.2021.107050

Britton, G. R. A., Brinthaupt, J., Stehle, J. M., & James, G. D. (2006). The effectiveness of a nurse-managed perinatal smoking cessation program implemented in a Rural County. *Nicotine & Tobacco Research*, *8*(1), 13–28. https://doi.org/10.1080/14622200500431536

Campbell, J. M., Bell, S. K., & Keith, L. K. (2001). Concurrent validity of the Peabody picture vocabulary test-third edition as an intelligence and achievement screener for low SES African American children. *Assessment*, *8*(1), 85–94. https://doi.org/10.1177/107319110100800108

Clifford, A., Lang, L., & Chen, R. (2012). Effects of maternal cigarette smoking during pregnancy on cognitive parameters of children and young adults: A literature review. *Neurotoxicology and Teratology*, *34*(6), 560–570. https://doi.org/10.1016/j.ntt.2012.09.004

Crosnoe, R., Leventhal, T., Wirth, R. J., Pierce, K. M., Pianta, R. C., & Nichd Early Child Care Research Network. (2010). Family socioeconomic status and consistent environmental stimulation in early childhood. *Child Development*, *81*(3), 972–987. https://doi.org/10.1111/j.1467-8624.2010.01446.x

Cutler, D. M., & Lleras-Muney, A. (2010). Understanding differences in health behaviors by education. *Journal of Health Economics*, *29*(1), 1–28. https://doi.org/10.1016/j.jhealeco.2009.10.003

D'Onofrio, B. M., Class, Q. A., Lahey, B. B., & Larsson, H. (2014). Testing the developmental origins of health and disease hypothesis for psychopathology using family-based quasi-experimental designs. *Child Development Perspectives*, *8*(3), 151–157. https://doi.org/10.1111/cdep.12078

D'Onofrio, B. M., Lahey, B. B., Turkheimer, E., & Lichtenstein, P. (2013). Critical need for family-based, quasi-experimental designs in integrating genetic and social science research. *American Journal of Public Health*, *103*(Suppl 1), S46–S55. https://doi.org/10.2105/AJPH.2013.301252

de Walque, D. (2007). Does education affect smoking behaviors? Evidence using the Vietnam draft as an instrument for college education. *Journal of Health Economics*, *26*(5), 877–895. https://doi.org/10.1016/j.jhealeco.2006.12.005

Dempsey, D. A., & Benowitz, N. L. (2001). Risks and benefits of nicotine to aid smoking cessation in pregnancy. *Drug Safety*, *24*(4), 277–322. https://doi.org/10.2165/00002018-200124040-00005

DiFranza, J. R., Aligne, C. A., & Weitzman, M. (2004). Prenatal and postnatal environmental tobacco smoke exposure and children's health. *Pediatrics*, *113*(4 Suppl), 1007–1015. https://doi.org/10.1542/peds.113.4.S1.1007

Drope, J., Liber, A. C., Cahn, Z., Stoklosa, M., Kennedy, R., Douglas, C. E., Henson, R., & Drope, J. (2018). Who's still smoking? Disparities in adult cigarette smoking prevalence in the United States. *CA: a Cancer Journal for Clinicians*, *68*(2), 106–115. https://doi.org/10.3322/caac.21444

Dunn, L., & Dunn, L. (1981). *Peabody Picture Vocabulary Test-Revised: Manual for Forms L and M*. American Guidance Service.

Dunn, L., Williams, K., Wang, J., & Booklets, N. (1997). *Peabody Picture Vocabulary Test, (PPVT-III): Form IIA*. American Guidance Service.

Dwyer, J. B., Broide, R. S., & Leslie, F. M. (2008). Nicotine and brain development. *Embryo Today: Reviews*, *84*(1), 30–44. https://doi.org/10.1002/bdrc.20118

Eicher, J. D., Powers, N. R., Cho, K., Miller, L. L., Mueller, K. L., Ring, S. M., Tomblin, J. B., & Gruen, J. R. (2013). Associations of prenatal nicotine exposure and the dopamine related genes ANKK1 and DRD2 to verbal language. *PLoS ONE*, *8*(5), e63762. https://doi.org/10.1371/journal.pone.0063762

Ellingson, J. M., Goodnight, J. A., Van Hulle, C. A., Waldman, I. D., & D'Onofrio, B. M. (2014). A sibling-comparison study of smoking during pregnancy and childhood psychological traits. *Behavior Genetics*, *44*(1), 25–35. https://doi.org/10.1007/s10519-013-9618-6

Elliot, C. D., Murray, D. J., & Pearson, L. S. (1983). *British Ability Scales–R*. NFER-NELSON Publishing Company.

Eriksen, H. L. F., Kesmodel, U. S., Wimberley, T., Underbjerg, M., Kilburn, T. R., & Mortensen, E. L. (2012). Effects of tobacco smoking in pregnancy on offspring intelligence at the age of 5. *Journal of Pregnancy*, *2012*, 945196. https://doi.org/10.1155/2012/945196

Fagerström, K. (2014). Journal of smoking cessation nicotine: Pharmacology, toxicity and therapeutic use. *Journal of Smoking Cessation*, *9*, 53–59. https://doi.org/10.1017/jsc.2014.27

Fenson, L., Bates, E., Dale, P., Goodman, J., Reznick, J. S., & Thal, D. (2000). Measuring variability in early child language: don't shoot the messenger. *Child Development*, *71*(2), 323–328. https://doi.org/10.1111/1467-8624.00147

Gibson, L., & Porter, M. (2020). Drinking or smoking while breastfeeding and later academic outcomes in children. *Nutrients*, *12*(3), 829. https://doi.org/10.3390/nu12030829

Gilman, S. E., Gardener, H., & Buka, S. L. (2008). Maternal smoking during pregnancy and children's cognitive and physical development: A causal risk factor? *American Journal of Epidemiology*, *168*(5), 522–531. https://doi.org/10.1093/aje/kwn175

Hack, M., Klein, N. K., & Taylor, H. G. (1995). Long-term developmental outcomes of low birth weight infants. *The Future of Children*, *5*(1), 176–196. https://doi.org/10.2307/1602514

Hack, M., Taylor, H. G., Drotar, D., Schluchter, M., Cartar, L., Wilson-Costello, D., Klein, N., Friedman, H., Mercuri-Minich, N., & Morrow, M. (2005). Poor predictive validity of the Bayley scales of infant development for cognitive function of extremely low birth weight children at school age. *Pediatrics*, *116*(2), 333–341. https://doi.org/10.1542/peds.2005-0173

Hanscombe, K. B., Trzaskowski, M., Haworth, C. M., Davis, O. S., Dale, P. S., & Plomin, R. (2012). Socioeconomic status (SES) and children's intelligence (IQ): In a UK-representative sample SES moderates the environmental, not genetic, effect on IQ. *PLoS ONE*, *7*(2), e30320. https://doi.org/10.1371/journal.pone.0030320

Hayiou-Thomas, M. E., Dale, P. S., & Plomin, R. (2012). The etiology of variation in language skills changes with development: A longitudinal twin study of language from 2 to 12 years. *Developmental Science*, *15*(2), 233–249. https://doi.org/10.1111/j.1467-7687.2011.01119.x

He, Y., Chen, J., Zhu, L. H., Hua, L. L., & Ke, F. F. (2020). Maternal smoking during pregnancy and ADHD: Results from a systematic review and meta-analysis of prospective cohort studies. *Journal of Attention Disorders*, *24*(12), 1637–1647. https://doi.org/10.1177/1087054717696766

Heinonen, K., Raikkonen, K., Pesonen, A. K., Andersson, S., Kajantie, E., Eriksson, J. G., Wolke, D., & Lano, A. (2011). Longitudinal study of smoking cessation before pregnancy and children's cognitive abilities at 56 months of age. *Early Human Development*, *87*(5), 353–359. https://doi.org/10.1016/j.earlhumdev.2011.02.002

Hellstrom-Lindahl, E., Gorbounova, O., Seiger, A., Mousavi, M., & Nordberg, A. (1998). Regional distribution of nicotinic receptors during prenatal development of human brain and spinal cord. *Developmental Brain Research*, *108*(1–2), 147–160. https://doi.org/10.1016/s0165-3806(98)00046-7

Hernandez-Martinez, C., Voltas Moreso, N., Ribot Serra, B., Arija Val, V., Escribano Macias, J., & Canals Sans, J. (2017). Effects of prenatal nicotine exposure on infant language development: A cohort follow up study. *Maternal and Child Health Journal*, *21*(4), 734–744. https://doi.org/10.1007/s10995-016-2158-y

Hiscock, R., Bauld, L., Amos, A., & Platt, S. (2012). Smoking and socioeconomic status in England: The rise of the never smoker and the disadvantaged smoker. *Journal of Public Health (Oxford, England)*, *34*(3), 390–396. https://doi.org/10.1093/pubmed/fds012

Hoff, E. (2009). *Language Development*. Wadsworth/Cengage Learning.

Horta, B. L., Kramer, M. S., & Platt, R. W. (2001). Maternal smoking and the risk of early weaning: A meta-analysis. *American Journal of Public Health*, *91*(2), 304–307. https://doi.org/10.2105/ajph.91.2.304

Hsieh, C. J., Liao, H. F., Wu, K. Y., Hsieh, W. S., Su, Y. N., Jeng, S. F., Yu, S. N., & Chen, P. C. (2008). CYP1A1 Ile462Val and GSTT1 modify the effect of cord blood cotinine on neurodevelopment at 2 years of age. *Neurotoxicology*, *29*(5), 839–845. https://doi.org/10.1016/j.neuro.2008.05.006

Huijbregts, S. C., Seguin, J. R., Zelazo, P. D., Parent, S., Japel, C., & Tremblay, R. E. (2006). Interrelations between maternal smoking during pregnancy, birth weight and sociodemographic factors in the prediction of early cognitive abilities. *Infant and Child Development*, *15*(6), 593–606. https://doi.org/10.1002/icd.480

Huizink, A. C., & Mulder, E. J. (2006). Maternal smoking, drinking or cannabis use during pregnancy and neurobehavioral and cognitive functioning in human offspring. *Neuroscience & Biobehavioural Reviews*, *30*(1), 24–41. https://doi.org/10.1016/j.neubiorev.2005.04.005

Hunter, A., Murray, R., Asher, L., & Leonardi-Bee, J. (2020). The effects of tobacco smoking, and prenatal tobacco smoke exposure, on risk of schizophrenia: A systematic review and meta-analysis. *Nicotine & Tobacco Research*, *22*(1), 3–10. https://doi.org/10.1093/ntr/nty160

Jauniaux, E., Biernaux, V., Gerlo, E., & Gulbis, B. (2001). Chronic maternal smoking and cord blood amino acid and enzyme levels at term. *Obstetrics & Gynecology*, *97*(1), 57–61. https://doi.org/10.1016/s0029-7844(00)01108-x

Julvez, J., Ribas-Fito, N., Torrent, M., Forns, M., Garcia-Esteban, R., & Sunyer, J. (2007). Maternal smoking habits and cognitive development of children at age 4 years in a population-based birth cohort. *International Journal of Epidemiology*, *36*(4), 825–832. https://doi.org/10.1093/ije/dym107

Jung, Y., Lee, A. M., McKee, S. A., & Picciotto, M. R. (2017). Maternal smoking and autism spectrum disorder: Meta-analysis with population smoking metrics as moderators. *Scientific Reports*, *7*(1), 4315. https://doi.org/10.1038/s41598-017-04413-1

Jürges, H., Reinhold, S., & Salm, M. (2011). Does schooling affect health behavior? Evidence from the educational expansion in Western Germany. *Economics of Education Review*, *30*(5), 862–872. https://doi.org/10.1016/j.econedurev.2011.04.002

Kalkbrenner, A. E., Braun, J. M., Durkin, M. S., Maenner, M. J., Cunniff, C., Lee, L. C., Pettygrove, S., Nicholas, J. S., & Daniels, J. L. (2012). Maternal smoking during pregnancy and the prevalence of autism spectrum disorders, using data from the autism and developmental disabilities monitoring network. *Environmental Health Perspectives*, *120*(7), 1042–1048. https://doi.org/10.1289/ehp.1104556

Kenkel, D., Lillard, D., & Mathios, A. (2003). Smoke or fog? The usefulness of retrospectively reported information about smoking. *Addiction*, *98*(9), 1307–1313. https://doi.org/10.1046/j.1360-0443.2003.00445.x

Kenkel, D., Lillard, D., & Mathios, A. (2006). The roles of high school completion and GED receipt in smoking and obesity. *Journal of Labor Economics*, *24*(3), 635–660. https://doi.org/10.1086/504277

Kim, K. M., & Choi, J. W. (2020). Associations between breastfeeding and cognitive function in children from early childhood to school age: A prospective birth cohort study. *International Breastfeeding Journal*, *15*(1), 83. https://doi.org/10.1186/s13006-020-00326-4

Kuyper, B. J. (1991). Bringing up scientists in the art of critiquing research. *Bioscience*, *41*(4), 248–250. https://doi.org/10.2307/1311414

Lange, S., Probst, C., Rehm, J., & Popova, S. (2018). National, regional, and global prevalence of smoking during pregnancy in the general population: A systematic review and meta-analysis. *The Lancet Global Health*, *6*(7), e769–e776. https://doi.org/10.1016/S2214-109X(18)30223-7

Langley, K., Rice, F., van den Bree, M. B., & Thapar, A. (2005). Maternal smoking during pregnancy as an environmental risk factor for attention deficit hyperactivity disorder behaviour. A review. *Minerva Pediatrica*, *57*(6), 359–371.

Laveist, T. A., Thorpe, R. J., Jr., Mance, G. A., & Jackson, J. (2007). Overcoming confounding of race with socio-economic status and segregation to explore race disparities in smoking. *Addiction*, *102*(Suppl 2), 65–70. https://doi.org/10.1111/j.1360-0443.2007.01956.x

Lee, M., Ha, M., Hong, Y.-C., Park, H., Kim, Y., Kim, E.-J., Kim, Y., & Ha, E. (2019). Exposure to prenatal secondhand smoke and early neurodevelopment: Mothers and Children's environmental health (MOCEH) study. *Environmental Health*, *18*(1), 22. https://doi.org/10.1186/s12940-019-0463-9

Levin, E. D., & Simon, B. B. (1998). Nicotinic acetylcholine involvement in cognitive function in animals. *Psychopharmacology*, *138*(3–4), 217–230. https://doi.org/10.1007/s002130050667

Linnet, K. M., Dalsgaard, S., Obel, C., Wisborg, K., Henriksen, T. B., Rodriguez, A., Kotimaa, A., Moilanen, I., Thomsen, P. H., Olsen, J., & Jarvelin, M. R. (2003). Maternal lifestyle factors in pregnancy risk of attention deficit hyperactivity disorder and associated behaviors: Review of the current evidence. *The American Journal of Psychiatry*, *160*(6), 1028–1040. https://doi.org/10.1176/appi.ajp.160.6.1028

López Ornat, S., Gallego, C., Gallo, P., Karousou, A., Mariscal, S., & Martínez, M. (2005). *Inventorios de Desarrollo Comunicativo MacArthur*. TEA Ediciones.

Luck, W., & Nau, H. (1985). Nicotine and cotinine concentrations in serum and urine of infants exposed via passive smoking or milk from smoking mothers. *The Journal of Pediatrics*, *107*(5), 816–820. https://doi.org/10.1016/s0022-3476(85)80427-3

Maatta, S., Laakso, M. L., Tolvanen, A., Ahonen, T., & Aro, T. (2012). Developmental trajectories of early communication skills. *Journal of Speech, Language, and Hearing Research*, *55*(4), 1083–1096. https://doi.org/10.1044/1092-4388(2011/10-0305)

MacArthur, C., Knox, E. G., & Lancashire, R. J. (2001). Effects at age nine of maternal smoking in pregnancy: Experimental and observational findings. *BJOG: An International Journal of Obstetrics & Gynaecology*, *108*(1), 67–73. https://doi.org/10.1111/j.1471-0528.2001.00006.x

Maralani, V. (2013). Educational inequalities in smoking: The role of initiation versus quitting. *Societal Science & Medicine*, *84*, 129–137. https://doi.org/10.1016/j.socscimed.2013.01.007

Marks, G. N. (2006). Family size, family type and student achievement: Cross-national differences and the role of socioeconomic and school factors. *Journal of Comparative Family Studies*, *37*(1), 1–24. https://doi.org/10.3138/jcfs.37.1.1

Mascheretti, S., Andreola, C., Scaini, S., & Sulpizio, S. (2018). Beyond genes: A systematic review of environmental risk factors in specific reading disorder. *Research in Developmental Disabilities*, *82*, 147–152. https://doi.org/10.1016/j.ridd.2018.03.005

Mascheretti, S., Bureau, A., Battaglia, M., Simone, D., Quadrelli, E., Croteau, J., Cellino, M. R., Giorda, R., Beri, S., Maziade, M., & Marino, C. (2013). An assessment of gene-by-environment interactions in developmental dyslexia-related phenotypes. *Genes, Brain and Behaviour*, *12*(1), 47–55. https://doi.org/10.1111/gbb.12000

Mascheretti, S., Facoetti, A., Giorda, R., Beri, S., Riva, V., Trezzi, V., Cellino, M. R., & Marino, C. (2015). GRIN2B mediates susceptibility to intelligence quotient and cognitive impairments in developmental dyslexia. *Psychiatric Genetics*, *25*(1), 9–20. https://doi.org/10.1097/YPG.0000000000000068

Matarazzo, J., & Herman, D. (1984). Relationship of education and IQ in the WAIS-R standardization sample. *Journal of Consulting and Clinical Psychology*, *52*(4), 631–634. https://doi.org/10.1037/0022-006X.52.4.631

McCarthy, D. (1972). *Manual for the McCarthy Scales of Children's Abilities* (M. TEA Ediciones, Spain, PA, Trans.). Psychological corporation.

Micalizzi, L., Marceau, K., Evans, A. S., Brick, L. A., Palmer, R. H. C., Heath, A. C., & Knopik, V. S. (2021). A sibling-comparison study of smoking during pregnancy and risk for reading-related problems. *Neurotoxicology and Teratology*, *84*, 106961. https://doi.org/10.1016/j.ntt.2021.106961

Mohamed, N. N., Loy, S. L., Lim, P. Y., Al Mamun, A., & Jan Mohamed, H. J. (2018). Early life secondhand smoke exposure assessed by hair nicotine biomarker may reduce children's neurodevelopment at 2years of age. *Science of the Total Environment*, *610-611*, 147–153. https://doi.org/10.1016/j.scitotenv.2017.08.030

Moore, B. F., Shapiro, A. L., Wilkening, G., Magzamen, S., Starling, A. P., Allshouse, W. B., Adgate, J. L., & Dabelea, D. (2020). Prenatal exposure to tobacco and offspring neurocognitive development in the healthy start study. *The Journal of Pediatrics*, *218*, 28–34. https://doi.org/10.1016/j.jpeds.2019.10.056

Napierala, M., Mazela, J., Merritt, T. A., & Florek, E. (2016). Tobacco smoking and breastfeeding: Effect on the lactation process, breast milk composition and infant development. A critical review. *Environmental Research*, *151*, 321–338. https://doi.org/10.1016/j.envres.2016.08.002

Neumann, D., Herbert, S. E., Peterson, E. R., Underwood, L., Morton, S. M. B., & Waldie, K. E. (2019). A longitudinal study of antenatal and perinatal risk factors in early childhood cognition: Evidence from growing up in New Zealand. *Early Human Development*, *132*, 45–51. https://doi.org/10.1016/j.earlhumdev.2019.04.001

Newbury, D. F., Winchester, L., Addis, L., Paracchini, S., Buckingham, L.-L., Clark, A., Cohen, W., Cowie, H., Dworzynski, K., Everitt, A., Goodyer, I. M., Hennessy, E., Kindley, A. D., Miller, L. L., Nasir, J., O'Hare, A., Shaw, D., Simkin, Z., Simonoff, E., Slonims, V., Watson, J., Ragoussis, J., Fisher, S. E., Seckl, J. R., Helms, P. J., Bolton, P. F., Pickles, A., Conti-Ramsden, G., Baird, G., Bishop, D. V. M., & Monaco, A. P. (2009). CMIP and ATP2C2 modulate phonological short-term memory in language impairment. *The American Journal of Human Genetics*, *85*(2), 264–272. https://doi.org/10.1016/j.ajhg.2009.07.004

Nuffield Trust. (2019). Smoking in pregnancy. How has the percentage of women who smoke during pregnancy changed over time? Retrieved from https://www.nuffieldtrust.org.uk/resource/smoking-in-pregnancy

Özmert, E. N., Yurdakök, K., Soysal, Ş., Kulak-Kayıkçı, M. E., Belgin, E., Özmert, E., Laleli, Y., & Saraçbaşi, O. (2005). Relationship between physical, environmental and sociodemographic factors and school performance in primary schoolchildren. *Journal of Tropical Pediatrics*, *51*(1), 25–32. https://doi.org/10.1093/tropej/fmh070

Park, H., & Cho, B. (2006). *Korean Bayley Scales of Infant Development* (2nd ed.). Kids Pop Publishing Corporation.

Petitti, D. B., Friedman, G. D., & Kahn, W. (1981). Accuracy of information on smoking habits provided on self-administered research questionnaires. *American Journal of Public Health*, *71*(3), 308–311. https://doi.org/10.2105/ajph.71.3.308

Pickett, K. E., Rathouz, P. J., Kasza, K., Wakschlag, L. S., & Wright, R. (2005). Self-reported smoking, cotinine levels, and patterns of smoking in pregnancy. *Paediatric and Perinatal Epidemiology*, *19*(5), 368–376. https://doi.org/10.1111/j.1365-3016.2005.00660.x

Plomin, R., & Deary, I. J. (2015). Genetics and intelligence differences: Five special findings. *Molecular Psychiatry*, *20*(1), 98–108. https://doi.org/10.1038/mp.2014.105

Polanska, K., Krol, A., Merecz-Kot, D., Ligocka, D., Mikolajewska, K., Mirabella, F., Chiarotti, F., Calamandrei, G., & Hanke, W. (2017). Environmental tobacco smoke exposure during pregnancy and child neurodevelopment. *International Jounral of Environmental Research and Public Health*, *14*(7), 796. https://doi.org/10.3390/ijerph14070796

Puglisi, M. L., Hulme, C., Hamilton, L. G., & Snowling, M. J. (2017). The home literacy environment is a correlate, but perhaps not a cause, of variations in Children's language and literacy development. *Scientific Studies of Reading*, *21*(6), 498–514. https://doi.org/10.1080/10888438.2017.1346660

Reid, J. L., Hammond, D., Boudreau, C., Fong, G. T., Siahpush, M., & Collaboration, I. T. C. (2010). Socioeconomic disparities in quit intentions, quit attempts, and smoking abstinence among smokers in four western countries: Findings from the

international tobacco control four country survey. *Nicotine & Tobacco Research*, 12(Suppl 1), S20–S33. https://doi.org/10.1093/ntr/ntq051

Reilly, S., Wake, M., Ukoumunne, O. C., Bavin, E., Prior, M., Cini, E., Conway, L., Eadie, P., & Bretherton, L. (2010). Predicting language outcomes at 4 years of age: Findings from early language in Victoria study. *Pediatrics*, *126*(6), 1530–1537. https://doi.org/10.1542/peds.2010-0254

Rhoda Kesler Unger. (2001). *Handbook of the Psychology of Women and Gender*. John Wiley & Sons, Inc.

Rice, F., Langley, K., Woodford, C., Davey Smith, G., & Thapar, A. (2018). Identifying the contribution of prenatal risk factors to offspring development and psychopathology: What designs to use and a critique of literature on maternal smoking and stress in pregnancy. *Development and Psychopathology*, *30*(3), 1107–1128. https://doi.org/10.1017/S0954579418000421

Ritchie, S. J., Bates, T. C., Der, G., Starr, J. M., & Deary, I. J. (2013). Education is associated with higher later life IQ scores, but not with faster cognitive processing speed. *Psychology and Aging*, *28*(2), 515–521. https://doi.org/10.1037/a0030820

Riva, D., Nichelli, F., & Devoti, M. (2000). Developmental aspects of verbal fluency and confrontation naming in children. *Brain and Language*, *71*(2), 267–284. https://doi.org/10.1006/brln.1999.2166

Rosen, B. N., Lee, B. K., Lee, N. L., Yang, Y., & Burstyn, I. (2015). Maternal smoking and autism Spectrum disorder: A meta-analysis. *Journal of Autism and Developmental Disorders*, *45*(6), 1689–1698. https://doi.org/10.1007/s10803-014-2327-z

Rosselli, M., Ardila, A., Matute, E., & Velez-Uribe, I. (2014). Language development across the life span: A neuropsychological/neuroimaging perspective. *Neuroscience Journal*, *21*, 585237. https://doi.org/10.1155/2014/585237

Ruisch, I. H., Dietrich, A., Glennon, J. C., Buitelaar, J. K., & Hoekstra, P. J. (2018). Maternal substance use during pregnancy and offspring conduct problems: A meta-analysis. *Neuroscience & Biobehavioural Reviews*, *84*, 325–336. https://doi.org/10.1016/j.neubiorev.2017.08.014

Salihu, H. M., & Wilson, R. E. (2007). Epidemiology of prenatal smoking and perinatal outcomes. *Early Human Development*, *83*(11), 713–720. https://doi.org/10.1016/j.earlhumdev.2007.08.002

Sanderson, E., Davey Smith, G., Bowden, J., & Munafo, M. R. (2019). Mendelian randomisation analysis of the effect of educational attainment and cognitive ability on smoking behaviour. *Nature Communications*, *10*(1), 2949. https://doi.org/10.1038/s41467-019-10679-y

Sarsour, K., Sheridan, M., Jutte, D., Nuru-Jeter, A., Hinshaw, S., & Boyce, W. T. (2011). Family socioeconomic status and child executive functions: The roles of language, home environment, and single parenthood. *Journal of the International Neuropsychological Society*, *17*(1), 120–132. https://doi.org/10.1017/S1355617710001335

Simonsen, H. G., Kristoffersen, K. E., Bleses, D., Wehberg, S., & Jørgensen, R. N. (2014). The Norwegian communicative development inventories: Reliability, main developmental trends and gender differences. *First Language*, *34*(1), 3–23. https://doi.org/10.1177/0142723713510997

Slotkin, T. A., Skavicus, S., Card, J., Stadler, A., Levin, E. D., & Seidler, F. J. (2015). Developmental neurotoxicity of tobacco smoke directed toward cholinergic and serotonergic systems: More than just nicotine. *Toxicological Sciences*, *147*(1), 178–189. https://doi.org/10.1093/toxsci/kfv123

Song, F., Elwell-Sutton, T., & Naughton, F. (2020). Impact of the NHS stop smoking services on smoking prevalence in England: A simulation modelling evaluation. *Tobacco Control*, *29*(2), 200–206. https://doi.org/10.1136/tobaccocontrol-2018-054879

Song, F., Elwell-Sutton, T., Naughton, F., & Gentry, S. (2021). Future smoking prevalence by socioeconomic status in England: A computational modelling study. *Tobacco Control*, *30*, 380–385. https://doi.org/10.1136/tobaccocontrol-2019-055490

Stanton-Chapman, T. L., Chapman, D. A., Bainbridge, N. L., & Scott, K. G. (2002). Identification of early risk factors for language impairment. *Research in Developmental Disabilities*, *23*(6), 390–405. https://doi.org/10.1016/s0891-4222(02)00141-5

Tang, S., Wang, Y., Gong, X., & Wang, G. (2015). A meta-analysis of maternal smoking during pregnancy and autism Spectrum disorder risk in offspring. *International Journal of Environmental Research and Public Health*, *12*(9), 10418–10431. https://doi.org/10.3390/ijerph120910418

Tapper, A. R., McKinney, S. L., Nashmi, R., Schwarz, J., Deshpande, P., Labarca, C., Whiteaker, P., Marks, M. J., Collins, A. C., & Lester, H. A. (2004). Nicotine activation of alpha4* receptors: Sufficient for reward, tolerance, and sensitization. *Science*, *306*(5698), 1029–1032. https://doi.org/10.1126/science.1099420

Thal, D. J., Reilly, J., Seibert, L., Jeffries, R., & Fenson, J. (2004). Language development in children at risk for language impairment: Cross-population comparisons. *Brain and Language*, *88*(2), 167–179. https://doi.org/10.1016/S0093-934X(03)00096-8

Thapar, A., Rice, F., Hay, D., Boivin, J., Langley, K., van den Bree, M., Rutter, M., & Harold, G. (2009). Prenatal smoking might not cause attention-deficit/hyperactivity disorder: Evidence from a novel design. *Biological Psychiatry*, *66*(8), 722–727. https://doi.org/10.1016/j.biopsych.2009.05.032

Tomblin, J. B., Hammer, C. S., & Zhang, X. (1998). The association of parental tobacco use and SLI. *International Journal of Language and Communication Disorders*, *33*(4), 357–368. https://doi.org/10.1080/136828298247686

Tomblin, J. B., Smith, E., & Zhang, X. (1997). Epidemiology of specific language impairment: Prenatal and perinatal risk factors. *Journal of Communication Disorders*, *30*(4), 325–343. https://doi.org/10.1016/s0021-9924(97)00015-4

Tosto, M. G., Hayiou-Thomas, M. E., Harlaar, N., Prom-Wormley, E., Dale, P. S., & Plomin, R. (2017). The genetic architecture of oral language, reading fluency, and reading comprehension: A twin study from 7 to 16 years. *Developmental Psychology*, *53*(6), 1115–1129. https://doi.org/10.1037/dev0000297

Tran, P. L., Lehti, V., Lampi, K. M., Helenius, H., Suominen, A., Gissler, M., Brown, A. S., & Sourander, A. (2013). Smoking during pregnancy and risk of autism spectrum disorder in a Finnish National Birth Cohort. *Paediatric and Perinatal Epidemiology*, *27*(3), 266–274. https://doi.org/10.1111/ppe.12043

Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., & Gottesman, I. I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*, *14*(6), 623–628. https://doi.org/10.1046/j.0956-7976.2003.psci_1475.x

Wang, T. M., Su, C. W., Liao, H. F., Lin, L. Y., Chou, K. S., & Lin, S. H. (1998). The standardization of the comprehensive developmental inventory for infants and toddlers. *Psychological Test*, *45*, 19–46.

Wechsler, D. (1992). *Wechsler Intelligence Scale for Children - Third* (UK ed.). Psychological Corporation.

Wechsler, D. (2006). *Manual for the Wechsler Preschool and Primary Scale of Intelligence- Revised* (Danish version ed.). Dansk Psykologisk Forlag.

Weitzman, M., Byrd, R. S., Aligne, C. A., & Moss, M. (2002). The effects of tobacco exposure on children's behavioral and cognitive functioning: Implications for clinical and public health policy and future research. *Neurotoxicology and Teratology*, *24*(3), 397–406. https://doi.org/10.1016/s0892-0362(02)00201-5

Wells, L., & Ostberg, V. (2021). How do educational disparities in smoking develop during early life? A Swedish longitudinal study. *SSM Population Health*, *15*, 100859. https://doi.org/10.1016/j.ssmph.2021.100859

Zhang, Y., Tardif, T., Shu, H., Li, H., Liu, H., McBride-Chang, C., Liang, W., & Zhang, Z. (2013). Phonological skills and vocabulary knowledge mediate socioeconomic status effects in predicting reading outcomes for Chinese children. *Developmental Psychology*, *49*(4), 665–671. https://doi.org/10.1037/a0028612

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

How to cite this article: Peixinho, J., Toseeb, U., Mountford, H. S., Bermudez, I., & Newbury, D. F. (2022). The effects of prenatal smoke exposure on language development - a systematic review. *Infant and Child Development*, e2331. https://doi.org/10.1002/icd.2331