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Parental Smoking and the Risk of Middle Ear Disease in Children

A Systematic Review and Meta-analysis

Laura L. Jones, PhD; Amal Hassanien, MSc; Derek G. Cook, PhD; John Britton, FRCP; Jo Leonardi-Bee, PhD

Objective: A systematic review and meta-analysis of studies of the association between secondhand tobacco smoke (SHTS) and middle ear disease (MED) in children.

Data Sources: MEDLINE, EMBASE, and CAB abstracts (through December 2010) and reference lists.

Study Selection: Sixty-one epidemiological studies of children assessing the effect of SHTS on outcomes of MED. Articles were reviewed, and the data were extracted and synthesized by 2 researchers.

Main Outcome Exposures: Children's SHTS exposure.

Main Outcome Measures: Middle ear disease in children.

Results: Living with a smoker was associated with an increased risk of MED in children by an odds ratio (OR) of 1.62 (95% CI, 1.33-1.97) for maternal postnatal smoking and by 1.37 (95% CI, 1.25-1.50) for any household member smoking. Prenatal maternal smoking (OR, 1.11; 95% CI, 0.93-1.31) and paternal smoking (OR, 1.24; 95% CI, 0.98-1.57) were associated with a nonsignificant increase in the risk of MED. The strongest effect was on the risk of surgery for MED, where maternal postnatal smoking increased the risk by an OR of 1.86 (95% CI, 1.31-2.63) and paternal smoking by 1.83 (95% CI, 1.61-2.07).

Conclusions: Exposure to SHTS, particularly to smoking by the mother, significantly increases the risk of MED in childhood; this risk is particularly strong for MED requiring surgery. We have shown that per year 130 200 of child MED episodes in the United Kingdom and 292 950 of child frequent ear infections in the United States are directly attributable to SHTS exposure in the home.

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Author Affiliations: UK Centre for Tobacco Control Studies, Division of Epidemiology and Public Health, University of Nottingham, Nottingham, England (Drs Jones, Britton, and Leonardi-Bee and Ms Hassanien) and Division of Population Health Sciences and Education, St George's University of London, London, England (Dr Cook).

MIDDLE EAR DISEASE (MED) is a common illness among children that accounts for a large number of physician visits and that, if untreated, can cause considerable disability through hearing impairment.[1] It is estimated that around 10% of children have 3 episodes of acute otitis media (AOM) before their first birthday,[2] whereas middle ear effusion is the most common reason for admission of young children to hospitals for surgery, putting a heavy financial burden on health care services.[3] Furthermore, adenoidectomy and particularly adenotonsillectomy, which are surgical treatments for otitis media with effusion (OME), have been associated with significant morbidity and mortality, including that arising from surgery.[4] Middle ear effusion is associated with hearing loss in children, which may lead to delayed linguistic and cognitive development.[3] The prevalence of MED is higher among children with learning impairment.[5]

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In 1998, a systematic review by Strachan and Cook of articles published through to 1996 found a significant association between parental smoking and MED.[6] However, the authors concluded that few studies had compared the effect of smoking by the mother and father and none had compared the effect of prenatal and postnatal tobacco smoke exposure to MED. This original review was commissioned for a UK government Scientific Committee on Tobacco and Health[7] and was subsequently updated as part of the 2006 US Surgeon General's report on the effects of involuntary exposure to tobacco smoke, which concluded that there was sufficient evidence to infer a causal relationship between parental smoking and otitis media in childhood.[8] Since these early reviews of articles published through 2001, the evidence base on the association between parental smoking and MED in child-

hood has significantly increased. To date, however, these new studies have not been subject to meta-analysis. We have therefore performed a systematic review and meta-analysis of the epidemiological data to provide contemporary estimates of the effects of smoking by parents and other household members on the risk of MED in childhood. This work was performed as part of a more extensive review of the effects of passive smoking in children, for the Royal College of Physicians.[9]

METHODS

SYSTEMATIC REVIEW METHODS

Any epidemiological study assessing the effect of secondhand tobacco smoke (SHTS) exposure (including household exposure, defined as ≥1 smoker living in the household but not specifying where, if anywhere, that person smokes in the home) smoking, paternal smoking, maternal smoking during and after pregnancy) were included in the review. (It should be noted that it was not possible in the current [or previous] review to identify studies that measured paternal smoking independently of maternal smoking, ie, the father smokes but the mother does not.) Outcomes of interest were MED, subdivided into middle ear infections (including AOM, OME, recurrent otitis media, chronic otitis media); hearing impairment (including hearing loss, deafness, glue ear [a condition in which the middle ear fills with fluid, leading to hearing impairment]), and surgery related to MED (including adenotonsillectomy, tonsillectomy, adenoidectomy, and grommet/pressure equalization tube insertion).

We searched MEDLINE, EMBASE, and CAB abstracts (from January 1997 through December 2010), using the keywords *tobacco smoke, cigarette smoking, passive smoking, parental smoking, maternal smoking, environmental tobacco smoking, secondhand smoke, children, infants, adolescents, pediatric, otitis media with effusion, deafness, adenoidectomy, middle ear disease, adenotonsillectomy, acute otitis media, recurrent otitis media, middle ear effusion, glue ear, otitis, tympanum, tonsil, otitis interna*. Hand searching of reference lists was also performed. No language restrictions were imposed during the searches; however, to be consistent with the original review,[6] we report only those studies published in English.

Titles, abstracts, and full texts from the identified studies were reviewed independently by 2 of us (by A.H. and J.L.B. or by L.L.J. and J.L.-B.) to identify eligible studies. Data were independently extracted by 2 authors (by A.H. and J.L.-B. or by J.L.-B. and L.L.J.) using a data extraction form, and methodological quality was assessed using the Newcastle-Ottawa Quality Assessment Scale.[10] A score of 6 or higher was chosen a priori to indicate higher methodological quality. In addition, all studies included in the previous review[6] were assessed for methodological quality using the same methods. Disagreements were resolved through discussion.

STATISTICAL ANALYSIS

Data were extracted as unadjusted odds ratios (ORs), or in preference, OR adjusted for potential confounding variables, with standard errors of 95% CIs. Pooled ORs and 95% CIs were estimated using random effect meta-analyses. Heterogeneity was assessed using recognized methods (I^2).[11] Random effect meta-regression analyses were conducted to investigate reasons for heterogeneity based on definition of MED (middle ear infection, surgery, and hearing impairment), methodological quality (higher vs lower), study design (cohort, cross-sectional, and case-control), ascertainment of SHTS exposure (biochemical vs self-report), and by date of publication. Exposure was defined as household, paternal, and maternal; maternal was split into prenatal and postnatal. When high levels (I^2>75%) were detected between the studies, we performed sensitivity analyses excluding outlier results. Data were analyzed using free, downloadable Review Manager software (version 5.0.23; The Cochrane Collaboration; http://ims.cochrane.org/revman/download) and STATA MP/11.0 for Windows (StataCorp LP, College Station, Texas). $P<.05$ was considered statistically significant. The analysis was performed in accordance with the Meta-analysis of Observational Studies in Epidemiology (MOOSE) guidelines.[12]

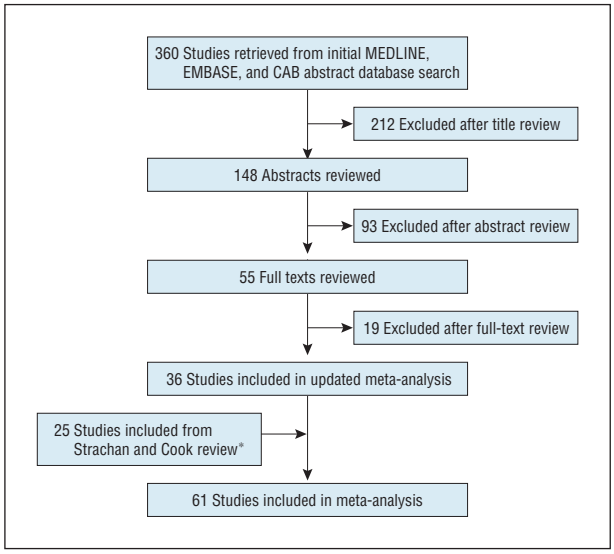

Figure 1. Flow diagram of included and excluded studies. *See study by Strachan and Cook.[6]

POPULATION-ATTRIBUTABLE FRACTION ESTIMATION

We estimated the proportion of children in England who live in a household in which at least 1 person smokes using data from the Health Survey for England[9] and used the formula $p(OR-1)/[p(OR-1)+1]$, in which p is the proportion of the cohort exposed to SHTS, and OR the odds ratio for MED in children with a member of the household who smokes, to estimate the proportion of children whose MED is attributable to household smoking exposure. We then used national MED prevalence[9] data for England and Wales to estimate the number of disease episodes generated as a result of household SHTS exposure. In addition, we estimated the number of MED episodes generated as a result of household SHTS exposure for the US population using similar methods with relevant data taken of the proportion of children in the United States who live with someone who smokes inside the home[13] and national MED prevalence data for children in the United States.[14]

RESULTS

From 360 titles published since 1997 identified in the literature search, 55 abstracts were deemed potentially eligible, and of these, 36 were included following the full-text review (**Figure 1**). The reasons for exclusion were not having a comparative group without the outcome,[15-17] not assessing SHTS as an exposure,[18] not assessing MED as an outcome,[19,20] being published in a lan-

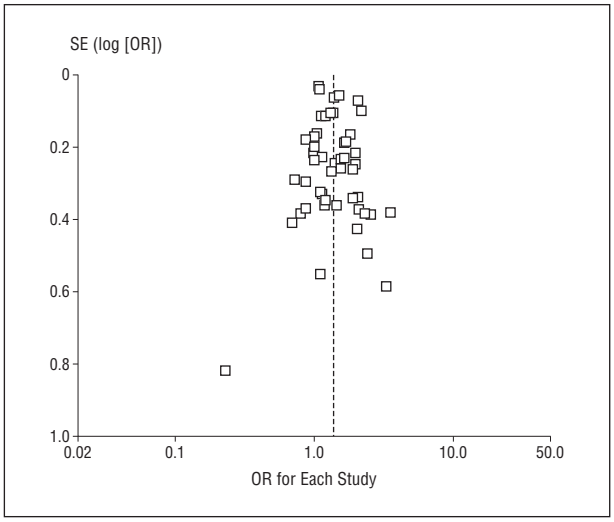

Figure 2. Funnel plot for household secondhand smoke exposure against middle ear disease. Plot shows the standard error (SE) of the odds ratio (OR) vs the OR for each study (random effects model). The vertical dotted line indicates the pooled effect estimate; and the squares, individual studies.

guage other than English,[21-27] only reporting statistical significance (*P* value) of the result without data,[28-31] or only having exposure to SHTS data as a confounder in the analysis.[32] Combining the results from this updated search with the previous review (25 studies) resulted in 61 epidemiological studies (eTable and eReferences(http://www.archpediatrics.com); Figure 1).

Of the 61 studies included, 15[33-47] were cross-sectional surveys, 22[48-69] were case-control studies, and 24[70-93] were cohort studies. Seventeen disease outcomes were reported within these studies: acute infection and serious otitis media,[84] AOM,* chronic suppurative otitis media,[62] earache,[83] glue ear,[54] hearing loss,[71] MED,[48,72] otitis media,[38,41,45,50,75,82] OME,† otitis prone,[67] recurrent otitis media,[51,63,68,74,78,80,89,90] suppurative otitis media,[35] surgery (adenoids/tonsils),[43] surgery (OME),[55,60,61,64,86] surgery (otitis media),[39,58,59] surgery (recurrent otitis media),[66] and surgery (tonsils).[57,69]

METHODOLOGICAL QUALITY OF STUDIES AND PUBLICATION BIAS

The overall median score for methodological quality was 5.5 (range, 2-8) (eTable and eReferences); with 34 studies judged to be of high quality; the remaining 27 were deemed to be of lower quality primarily owing to a combination of a lack of biochemical validation of SHTS exposure, lack of representativeness of the study sample, and/or lack of adjusted analyses. There was no evidence of publication bias identified from funnel plots. The funnel plot for household exposure and the risk of MED is presented in **Figure 2**.

EFFECTS OF MATERNAL POSTNATAL SMOKING

Meta-analysis of the 20 studies of postnatal maternal smoking showed a statistically significant increase in the risk of MED in childhood by 1.62 (95% CI, 1.33-1.97). High levels of heterogeneity were present in this analysis (I^2=93%), which was predominately related to 1 study[53]; excluding this study reduced the pooled estimate to 1.39 (95% CI, 1.30-1.61; I^2=85%). Pooled estimates for each of the outcome categories showed that the increase in risk of MED was driven predominantly by an increase in the risk of surgery for MED (OR, 1.86; 95% CI, 1.31-2.63; 5 studies; **Figure 3**) and to a lesser extent by hearing impairment (OR, 1.74; 95% CI, 1.08-2.81; 1 study) and middle ear infection (OR, 1.53; 95% CI, 1.22-1.92; 14 studies). In a meta-regression based on method of ascertainment of SHTS exposure, studies that used self-reported data showed a higher increase in disease risk (OR, 1.70; 95% CI, 1.29-2.25; 17 studies) than studies that used biochemical validation (OR, 1.29; 95% CI, 0.86-1.94; 3 studies). In a subgroup analysis based on study design, case-control studies showed a statistically significant increase in the risk of MED in children (OR, 2.09; 95% 1.19-3.66; 10 studies), unlike cohort (OR, 1.19; 95% CI, 0.94-1.49; 6 studies) and cross-sectional (OR, 1.28; 95% CI, 0.88-1.86; 4 studies) study designs that were not statistically significantly associated with an increase in disease risk. Similar pooled estimates were also shown for the meta-regression analysis based on methodological quality and date of publication (**Table 1**). In a multiple meta-regression adjusting for study design, publication date, ascertainment, and methodological quality, none of the factors independently predicted the OR for maternal postnatal smoking.

EFFECTS OF MATERNAL PRENATAL SMOKING

All of the 6 studies of prenatal maternal smoking were identified from the updated search because they were published after 1996. Prenatal maternal smoking was not associated with a statistically significant increase in the risk of MED (OR, 1.11; 95% CI, 0.93-1.31; 6 studies); however, high levels of heterogeneity were seen between the studies (I^2=79%). Excluding the study[88] with outlier results had marginal effects on the pooled estimate (OR, 1.06; 95% CI, 0.94-1.19; I^2=62%). Similarly, no statistically significant pooled estimates were seen for meta-regression analyses stratified by study design, ascertainment of smoking status, and methodological quality (Table 1).

EFFECTS OF PATERNAL SMOKING

Exposure to paternal smoking was associated with a nonsignificant (*P*=.07) increase in the odds of MED in childhood by 1.24 (95% CI, 0.98-1.57; 12 studies). Very high levels of heterogeneity were seen in the analysis (I^2=87%), which was predominately related to 1 study[43]; excluding this study explained most of the heterogeneity and had marginal effects on the pooled estimate (OR. 1.13; 95% CI. 0.97-1.32; I^2=39%). Subgroup analysis based on the definition of outcome showed that the increased risk of disease was due to a strong association between paternal SHTS exposure and the risk of surgery for MED (OR, 1.83; 95% CI, 1.61-2.07; 4 studies) (**Figure 4**). The association between paternal smoking and middle ear infection was not statistically significant (OR, 1.06; 95% CI, 0.91-1.24; 8 studies; *P*=.47). Similar pooled estimates were

*References 34, 37, 40, 70, 73, 87, 88, 92, 93.
†References 33, 36, 42, 44, 49, 52, 53, 56, 65, 76, 77, 79, 81, 85.

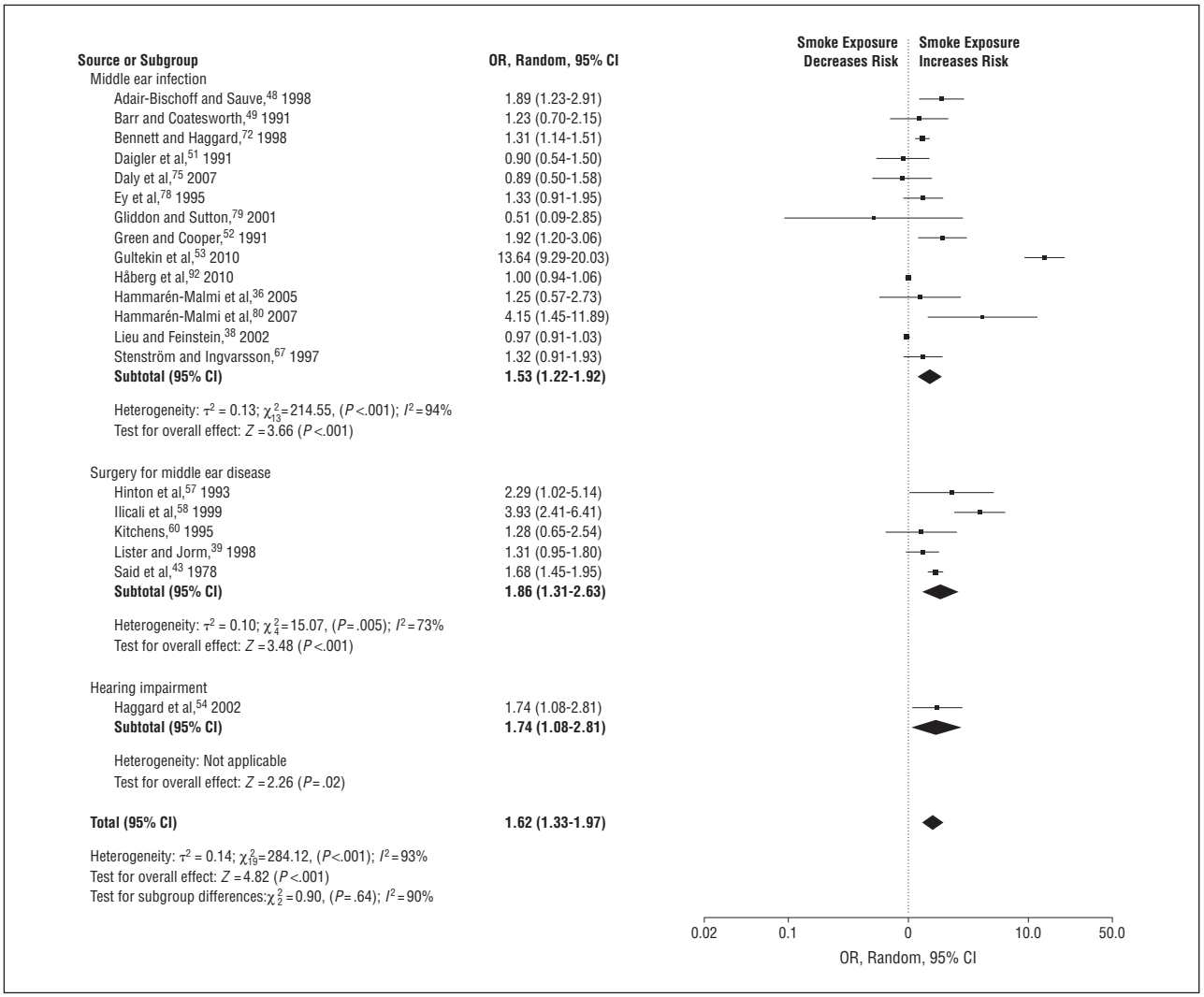

Figure 3. Relationship between secondhand tobacco smoke exposure by maternal smoking after birth and the risk of middle ear disease using a meta-analysis of comparative epidemiologic studies. Data are presented as odds ratios (ORs) subgrouped by the definition of middle ear disease outcome. Each small square denotes the OR for a single study, with horizontal lines denoting 95% CIs. The center of each diamond denotes the pooled OR and the corners the 95% CIs. An OR greater than 1 indicates a higher risk of the outcome in those exposed to secondhand tobacco smoke.

also seen for meta-regression analyses stratified by study design, ascertainment of smoking status, date of publication, and methodological quality (**Table 2**). In a multiple meta-regression adjusting for study design, publication date, ascertainment, and methodological quality, none of the factors independently predicted the OR for paternal smoking.

EFFECTS OF HOUSEHOLD SMOKING

A pooled estimate derived from the 49 studies that defined exposure as household smoking (the study by Jacoby et al[82] is shown in **Figure 5** as 2 separate entries given the differing estimates reported for the 2 samples: aboriginal vs nonaboriginal) demonstrated a statistically significant increase in the risk of MED by an OR of 1.37 (95% CI, 1.25-1.50; 49 studies). High levels of heterogeneity were seen between the studies (I^2=76%); excluding the studies[43,44] with outlier results had marginal effects on the pooled estimate (OR, 1.36; 95% CI, 1.24-1.48; I^2=69%). Subgroup analysis based on the definition of outcome showed that the increase in risk was mainly attributable to a increase in risk of surgery for MED (OR, 1.62; 95% CI, 1.32-1.98; 11 studies; Figure 5) and to a lesser extent middle ear infection (OR, 1.32; 95% CI, 1.20-1.45; 38 studies). Meta-regression analysis based on study design showed varied pooled estimates, with case-control studies showing the highest increase in disease risk (OR, 1.55; 95% CI, 1.35-1.77; 18 studies), followed by cross-sectional studies (OR, 1.33; 95% CI, 1.10-1.60; 13 studies) and cohort studies (OR, 1.27; 95% CI, 1.13-1.43; 18 studies). Similar pooled estimates were also seen for analyses stratified by ascertainment of smoking status, date of publication, and methodological quality (Table 2). In a multiple meta-regression adjusting for study design, publication date, ascertainment, and methodological quality, none of the factors independently predicted the OR for household smoking.

POPULATION-ATTRIBUTABLE FRACTION

Data from the Health Survey for England[9] indicated that

Table 1. Summary of Overall Effect and Meta-regression Analysis of Maternal Prenatal and Postnatal Passive Smoke Exposure on the Risk of Middle Ear Disease in Childhood

	Maternal Prenatal				Maternal Postnatal			
	OR (95% CI)	Studies, No.	I^2	*P* Value[a]	OR (95% CI)	Studies, No.	I^2	*P* Value[a]
Overall effect	1.11 (0.93-1.31)	6	79	NA	1.62 (1.33-1.97)	20	93	NA
Outcome								
Middle ear infection	1.15 (0.98-1.35)	5	79	.36	1.53 (1.22-1.92)	16	94	.63
Surgery for middle ear disease	NA	0	N/A		1.86 (1.31-2.63)	5	73	
Hearing loss	0.61 (0.35-1.08)	1	NA		NA	0	NA	
Hearing impairment	NA	0	NA		1.74 (1.08-2.81)	1	NA	
Study design								
Cohort	1.14 (0.84-1.53)	5	82	.98	1.19 (0.94-1.49)	6	76	.14
Cross-sectional	1.07 (0.97-1.18)	1	NA		1.28 (0.88-1.86)	4	94	
Case-control	NA	0	NA		2.09 (1.19-3.66)	10	92	
Methodological quality								
High	1.17 (0.95-1.44)	4	85	.49	1.83 (1.21-2.76)	10	96	.47
Low	0.74 (0.24-2.27)	2	41		1.47 (1.12-1.94)	10	88	
Publication date								
Through to 1996	NA	0	NA	NA	1.48 (1.22-1.80)	7	31	.58
Post 1996	1.11 (0.93-1.31)	6	79		1.72 (1.35-2.21)	13	95	
Ascertainment								
Self-report	1.17 (0.87-1.57)	4	85	.56	1.70 (1.29-2.25)	17	94	.56
Biochemical	0.75 (0.22-2.61)	2	47		1.29 (0.86-1.94)	3	82	

Abbreviations: I^2, percentage of heterogeneity; NA, not applicable; OR, odds ratio.
[a] *P* value from random effect meta-regression analysis.

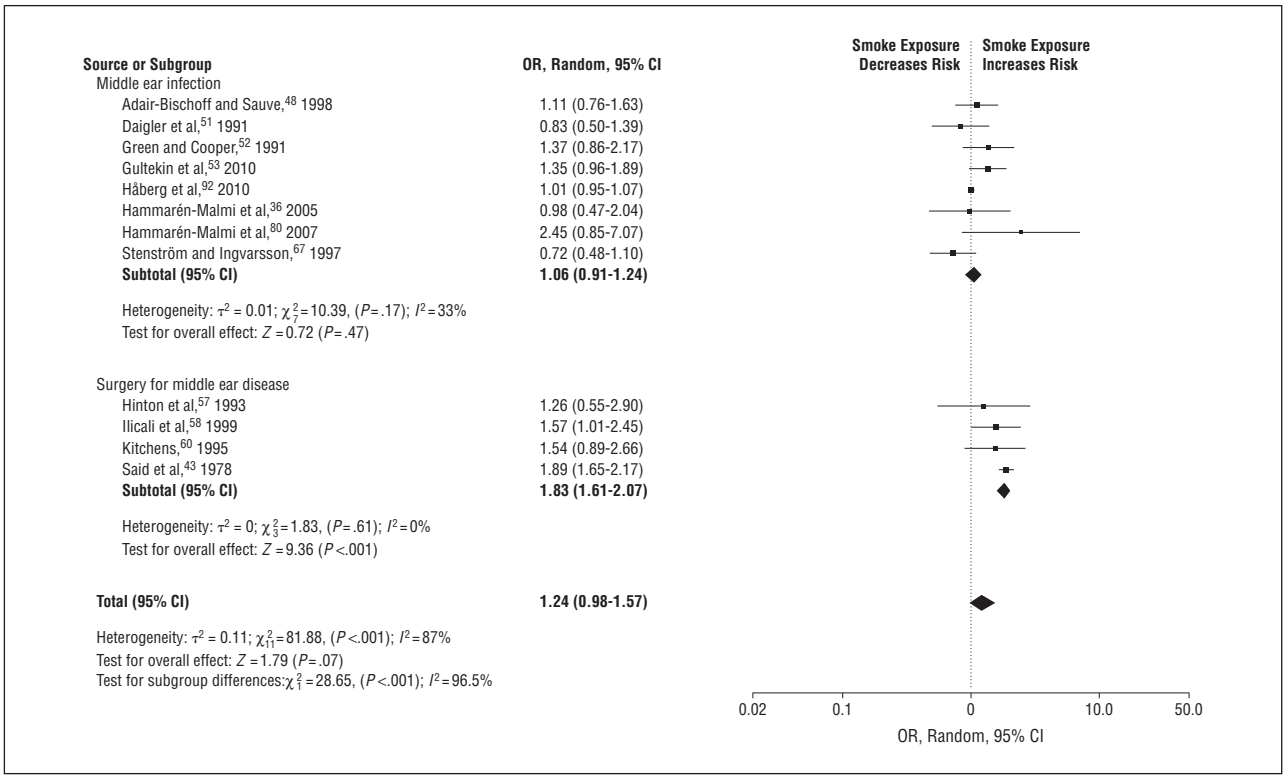

Figure 4. Relationship between paternal secondhand tobacco smoke exposure and the risk of middle ear disease using a meta-analysis of comparative epidemiologic studies. Data are presented as odds ratios (ORs) subgrouped by the definition of middle ear disease outcome. Each small square denotes the OR for a single study, with horizontal lines denoting 95% CIs. The center of each diamond denotes the pooled OR and the corners the 95% CIs. An OR greater than 1 indicates a higher risk of the outcome in those exposed to secondhand tobacco smoke.

in 2007, around 22% of children up to 15 years old lived in a household in which someone smokes. Using the OR for household smoking (1.37) as the estimated relative risk of developing MED, the proportion of children developing MED likely to be attributable to exposure to smoking in the home is estimated at 7.5%. In 2008 there were about

Table 2. Summary of Overall Effect and Meta-regression Analysis of Household and Paternal Passive Smoke Exposure on the Risk of Middle Ear Disease in Childhood

	Household				Paternal			
	OR (95% CI)	Studies, No.	I^2	*P* Value[a]	OR (95% CI)	Studies, No.	I^2	*P* Value[a]
Overall effect	1.37 (1.25-1.50)	49	76	NA	1.24 (0.98-1.57)	12	87	NA
Outcome								
Middle ear infection	1.32 (1.20-1.45)	38	73	.11	1.06 (0.91-1.24)	8	33	<.001
Surgery for middle ear disease	1.62 (1.32-1.98)	11	41		1.83 (1.61-2.0)	4	0	
Hearing loss	NA	0	NA		NA	0	NA	
Hearing impairment	NA	0	NA		NA	0	NA	
Study design								
Cohort	1.27 (1.13-1.43)	18	46	.25	2.45 (0.85-7.07)	1		.28
Cross-sectional	1.33 (1.10-1.60)	13	90		1.51 (0.82-2.78)	2	66	
Case-control	1.55 (1.35-1.77)	18	25		1.14 (0.90-1.44)	7	39	
Methodological quality								
High	1.30 (1.19-1.43)	28	67	.40	1.19 (0.94-1.50)	5	8	.82
Low	1.41 (1.18-1.68)	21	71		1.25 (0.91-1.74)	7	92	
Publication date								
Through to 1996	1.46 (1.26-1.70)	21	60	.27	1.40 (1.01-1.94)	5	64	.22
Post 1996	1.31 (1.17-1.46)	28	77		1.11 (0.92-1.35)	7	49	
Ascertainment								
Self-report	1.40 (1.25-1.56)	40	74	.32	1.25 (0.97-1.62)	11	88	.74
Biochemical	1.22 (1.03-1.45)	9	66		1.11 (0.76-1.63)	1	NA	

Abbreviations: I^2, percentage of heterogeneity; NA, not applicable; OR, odds ratio.
[a] *P* value from random effect meta-regression analysis.

1 735 710 episodes of MED in children younger than 16 years in the United Kingdom.[9] A 7.5% attributable fraction translates into approximately 130 200 new episodes of MED arising from exposure to smoking in the home in the United Kingdom. Data from the National Health and Nutrition Examination survey[13] indicated that between 2007 and 2008, 18.2% US children aged 3 to 11 years lived with someone who smoked inside the home. Annually, there were 4.65 million children reported to have frequent ear infection (≥3 episodes in the previous 12 months).[14] A 6.3% attributable fraction corresponds to approximately 292 950 children aged 3 to 11 years, with frequent ear infections arising from exposure to SHTS in the home in the United States.

COMMENT

Middle ear disease is a significant cause of morbidities in children and has been shown to be associated with parental SHTS exposure.[6] This relationship has been further explored in the current systematic review and meta-analysis, providing novel findings that suggest that maternal postnatal smoking, rather than maternal prenatal or paternal smoking, has the strongest influence on disease risk. This may suggest that the effect is due to ambient smoke pollution from the child's close proximity to the primary caregiver, not to development effects. However, it important to consider that only 6 prenatal studies met the inclusion criteria in the current study and hence may be underpowered to detect an association. Therefore, further well-conducted research studies are needed. In addition, only 2 of the studies[88,92] adjusted for the effect of postnatal exposure in their analyses. Because there is high concordance of prenatal and postnatal smoking by the mother it is difficult to assess the independent effect of smoking during pregnancy on the risk of MED.

We additionally found that smoking by any household member was statistically significantly associated with an increased risk of disease in children, which translates to an additional 130 200 episodes of MED per year in the United Kingdom, and an additional 292 950 frequent ear infections (≥3 episodes in the previous 12 months) per year in US children aged 3 to 11 years, that are directly attributable to exposure to SHTS.

From meta-regression analysis exploring the different MED outcomes (middle ear infection, surgery for middle ear infection, hearing impairment, or hearing loss), we found that the effect of SHTS exposure was strongest for surgery for MED, with an increased risk of 1.86 for maternal postnatal, 1.83 for paternal, and 1.62 for household smoking. In addition, maternal postnatal smoking was shown to increase the risk of hearing impairment by an OR of 1.74 (95% CI, 1.08-2.81), although this estimate is based on only 1 study of high methodological quality.[54]

Our findings are likely to be representative estimates of the true effects of exposure to SHTS on the risk of MED in children because they are based on results of a comprehensive search, including data identified through hand searching of reference lists and previous reviews. However, there are limitations to this review. We elected to keep methods consistent with the original strategy[6] and included only studies written in English in the meta-analyses. In addition, the definition of household smoking did not allow us to clearly differentiate between children who lived with smokers who smoked inside the house and children who lived with smokers who smoked outside of the house. We were inevitably limited in the range of confounding factors that could be adjusted for

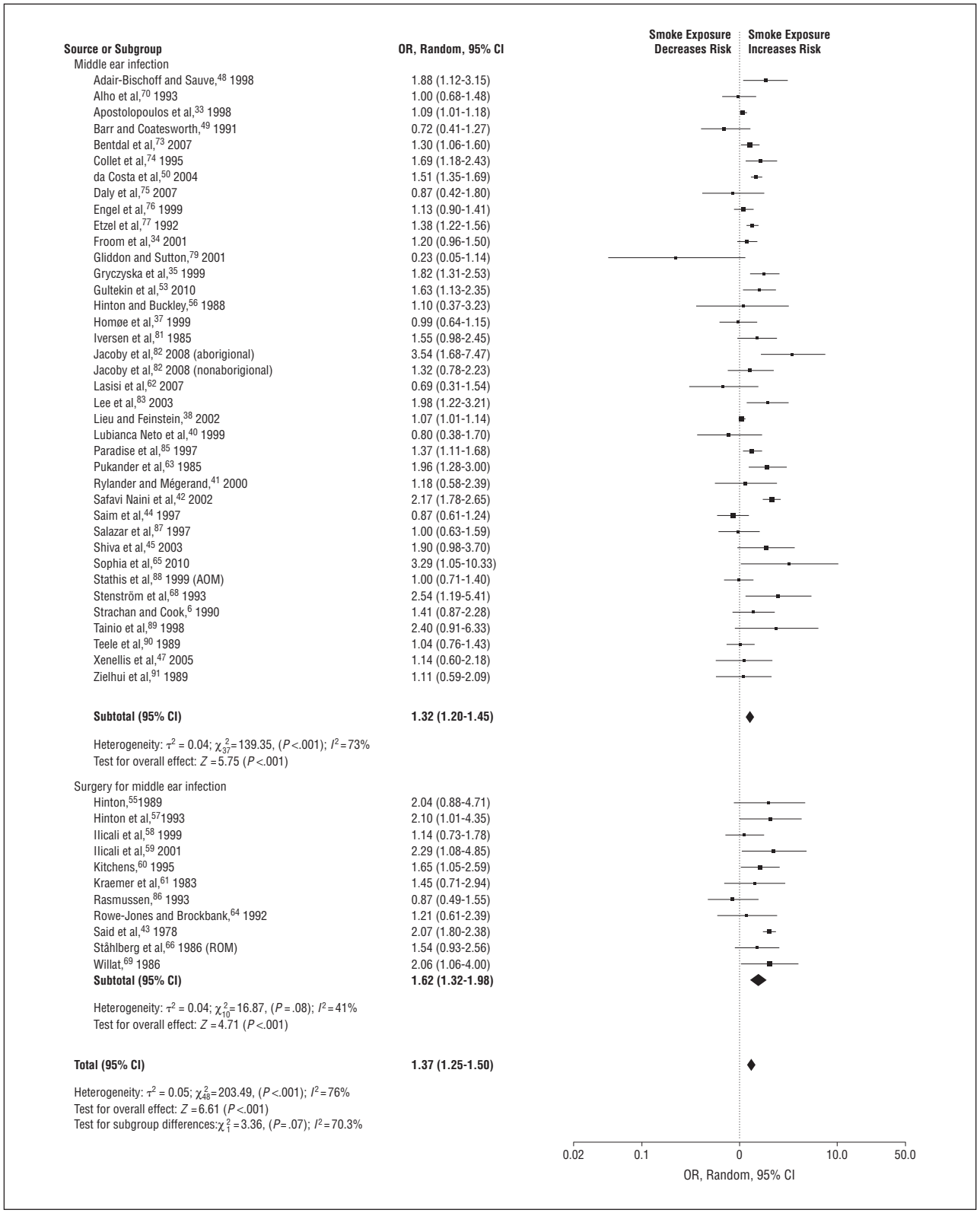

Figure 5. Relationship between secondhand tobacco smoke exposure by any household member and the risk of middle ear disease using a meta-analysis of comparative epidemiologic studies. Data are presented as odds ratios (ORs) subgrouped by the definition of middle ear disease outcome. Each square denotes the OR for a single study, with horizontal lines denoting 95% CIs. The center of each diamond denotes the pooled OR and the corners the 95% CIs. An OR greater than1 indicates a higher risk of the outcome in those exposed to secondhand tobacco smoke). AOM indicates acute otitis media; ROM, recurrent otitis media.

in our analyses. Although the high-quality studies generally adjusted for maternal age and socioeconomic status, other potential confounders, such as smoking by other individuals in the household and location of smoking either inside or outside of the house, were not consistently adjusted for in the analyses. A further limitation

was that high levels of heterogeneity were observed in some comparisons; however, this seemed to be related to the results from 1 or 2 studies within each analysis; therefore, most of the studies were consistent. We investigated reasons for heterogeneity by performing meta-regression analyses; however, these analyses revealed relatively consistent findings. Generally, the pooled results did not differ appreciably among studies of different methodological quality, publication date, or study design.

In conclusion, this study confirms that household smoking, in particular, maternal postnatal smoking, causes a statistically significant increase in the risk of MED in childhood, and identifies that 1 of the main consequences of children's exposure to SHTS is the significant increase in the risk of having to have surgery for chronic MED. Surgical treatments for otitis media, such as grommet–pressure equalization tube insertion, have been shown to be questionable in their effectiveness, associated with high risk, and resource and cost intensive.[94] Therefore, primary prevention through the reduction of risk factors, such as exposure to SHTS, is key to reducing the burden of MED in childhood. Although evidence is emerging to suggest that the incidence of MED has been declining in recent years in England,[95] perhaps as a reflection of a reduction in the number of parents who smoke, MED is still a major public and child health concern, with a total of 1.74 million episodes estimated in the United Kingdom[9] and 4.65 million episodes of frequent ear infection (≥3 episodes in the previous 12 months) in the United States[14] each year. We have shown that 7.5% (130 200) of the episodes in the United Kingdom and 6.3% (292 500) of the episodes in the United States are directly attributable to SHTS exposure in the home, respectively, all of which are avoidable. The findings from this study should encourage renewed efforts to promote smoke-free environments for children.

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Correspondence: Laura L. Jones, PhD, Division of Epidemiology and Public Health, University of Nottingham, City Hospital Campus, Hucknall Road, Nottingham NG5 1PB, England (laura.jones@nottingham.ac.uk)
Author Contributions: Dr Leonardi-Bee had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. *Study concept and design:* Cook, Britton, and Leonardi-Bee. *Acquisition of data:* Jones, Hassanien, and Leonardi-Bee. *Analysis and interpretation of data:* Jones, Hassanien, and Leonardi-Bee. *Drafting of the manuscript:* Jones and Hassanien. *Critical revision of the manuscript for important intellectual content:* Jones, Hassanien, Cook, Britton, and Leonardi-Bee. *Statistical analysis:* Cook and Leonardi-Bee. *Obtained funding:* Britton. *Administrative, technical, and material support:* Jones and Britton. *Study supervision:* Jones, Britton, and Leonardi-Bee.
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Online-Only Material: The eTable and eReferences are available at http://www.archpediatrics.com.

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