Health effects of passive smoking · 4

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Parental smoking, middle ear disease and adenotonsillectomy in children

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Abstract

***Background* – A systematic quantitative review was conducted of evidence relating parental smoking to acute otitis media, recurrent otitis media, middle ear effusion, and adenoidectomy and/or tonsillectomy.**

***Methods* – Forty five relevant publications were identified after consideration of 692 articles selected by electronic search of the Embase and Medline databases using keywords relevant to passive smoking in children. The search was completed in April 1997 and identified 13 studies of acute otitis media, nine of recurrent otitis media, five of middle ear effusion, nine of glue ear surgery, and four of adenotonsillectomy. A quantitative meta-analysis was possible for all outcomes except acute otitis media, using random effects modelling where appropriate to pool odds ratios from each study.**

***Results* – Evidence for middle ear disease is remarkably consistent, with pooled odds ratios if either parent smoked of 1.48 (95% CI 1.08 to 2.04) for recurrent otitis media, 1.38 (1.23 to 1.55) for middle ear effusion, and 1.21 (0.95 to 1.53) for outpatient or inpatient referral for glue ear. Odds ratios for acute otitis media are in the range 1.0 to 1.6. No single study simultaneously addresses selection bias, information bias and confounding, but where these have been investigated or excluded in the design or analysis, the associations with parental smoking persist virtually unchanged. Large French and British studies are inconsistent with regard to the association of parental smoking and tonsillectomy.**

***Conclusions* – There is likely to be a causal relationship between parental smoking and both acute and chronic middle ear disease in children.**

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Keywords: parental smoking, middle ear disease, adenotonsillectomy, children, passive smoking.

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A possible link between parental smoking and the risk of otitis media with effusion in children was first suggested in 1983.[1] A number of subsequent epidemiological studies have investigated the association of exposure to environmental tobacco smoke and diseases of the ear, nose and throat (ENT), and the evidence has been the subject of periodic narrative reviews.[2–4] Only two studies have attempted a quantitative meta-analysis.[5 6] In one,[5] based on papers published up to 1992 inclusive, the authors did not distinguish clearly between studies of different types of ENT disease. The other,[6] based on literature published up to 1994, reviewed parental smoking briefly along with other risk factors for acute otitis media.

This paper systematically reviews the evidence relating parental smoking to acute otitis media, recurrent otitis media, "glue ear" (otitis media with effusion), and ENT surgery in children. Each of these outcomes is considered separately, including a quantitative meta-analysis where appropriate.

Methods

Published papers, letters, and review articles were selected by an electronic search of the Embase and Medline databases using the search strategy described in detail elsewhere.[7] Briefly, all passive smoking references were selected by the MESH heading *tobacco smoke pollution* and/or textword combinations ({*passive*, *second-hand*, *second hand*, *involuntary*, *parent**, *maternal*, *mother**, *paternal*, *father** or *household*} **and** {*smok**, *tobacco** or *cigarette**}). Papers were then restricted to children by relevant textwords or by the age group as specified in the title or abstract. This search, completed in April 1997, yielded 3625 references of which 1593 contained keywords relevant to respiratory or allergic disease. These 1593 abstracts were reviewed and 99 papers relevant to ENT disease were selected by the textwords *tympanom**, *otitis*, *middle ear*, *glue ear* or *tonsil**.

Among these 99 papers, 37 publications presented quantitative information relevant to this review and a further six were identified by citations in previous overviews or individual studies. Two cross sectional surveys of schoolchildren[8 9] were identified in the course of another systematic review in this series. These 45 papers related to 42 studies: 12 cross sectional surveys, 14 case-control studies, 15 longitudinal studies, and one controlled trial of surgical intervention for middle ear effusion.

Table 1 Design, sample size and recruitment criteria: studies excluded from meta-analyses

Reference no.	*Year*	*Country*	*Age*	*Design*	*Outcome*	*Sample size*	*Case definition*	*Source of controls or cohort*
Acute otitis media in pre-school children								
19	79	Denmark	3	Cohort	AOM	494	AOM episodes	Random sample of children
16	82	Finland	0–4	C-C	AOM	200	AOM in past year	Health centre controls
15	84	Denmark	3–4	Cohort	AOM, OME	681	History of AOM	Random sample of birth cohort
18	84	Belgium	2–6	Survey	AOM, OME	2065	AOM, tympanogram	"Healthy" kindergarten pupils
17	87	USA (GA)	0–4	Survey	AOM	609	AOM past 2 weeks	Random sample of households
20	88	Finland	0–3	Cohort	AOM	1294	AOM episodes	Random sample of urban area
14	90	Sweden	0–3	Cohort	Acute RTI	113	AOM/OME/URTI/LRTI	Population-based birth cohort
21	96	Finland	0–2	Cohort	AOM	825	AOM episodes	Population-based birth cohort
Ear infections in schoolchildren								
8	86	Israel	?	Survey	Infection	1449	Ear infection ever	2nd & 5th grade schoolchildren
13	92	Italy	6–14	Survey	"Otitis"	2304	Ever had otitis	Random sample of schoolchildren
9	95	Israel	?	Survey	Infection	6302	Ear infection ever	2nd & 5th grade schoolchildren
22	96	Thailand	6–10	Survey	AOM or OME	2384	History & examination	Three primary schools
Middle ear effusion: prevalence								
34	88	Holland	3	Cohort	OME	1439	Flat tympanogram	Population-based birth cohort
36	88	USA (UT)	?	Survey	OME	45	Flat tympanogram	Outpatients, half with AOM
35	90	Japan	4–5	C-C	OME	201	Tympanometry & exam	Population screening survey
Middle ear effusion: natural history								
45	93	UK	2–11	Trial	Resolution	66	No effusion	Untreated ears with OME
46	94	UK	3–9	Trial	Resolution	133	No effusion	Trial participants with OME
Hearing loss								
47	92	Ireland	10 months	Survey	Impairment	87	Distraction test	Routine postnatal screening

AOM = acute otitis media; ROM = recurrent otitis media; OME = otitis media with effusion (glue ear); OP = outpatients; PD = physician-diagnosed; C-C = case-control study.
Reference 34 reports the same study as reference 30 and in less detail. Reference 30 appears in table 2.

Studies were grouped according to the outcome measure as follows: acute otitis media (11), recurrent otitis media (9), population surveys of effusion (5), population studies of deafness (1), clinic-based studies of referrals for glue ear surgery (9), adenoidectomy or tonsillectomy (4), and postoperative natural history studies (1). Three publications[10–12] contributed information on more than one outcome measure (tables 1 and 2).

Odds ratios relating parental smoking to each health outcome were pooled using weights inversely proportional to their variance (the "fixed effect" assumption). In addition, "random effects" models were used as described in detail elsewhere[7] if there was evidence of statistically significant heterogeneity of the passive smoking effect between studies.

Results

ACUTE OTITIS MEDIA

Episodes of acute middle ear infection are common in young children and various methods have been used to report the incidence of the condition. Some have reported lifetime prevalence, with or without a physician diagnosis, and at various ages.[12–15] Others present period prevalence over a year[16] or the recent past,[17 18] or prospective data counting episodes of acute otitis media over the period of the study.[10 14 19–21] Few studies present quantitative information in relation to parental smoking habits.

Three early studies[15 16 19] report no significant association ($p>0.05$) of acute otitis media with parental smoking without details of the supporting data. Acute otitis media in the previous two weeks was not associated with exposure to environmental tobacco smoke in a household survey in Atlanta, Georgia (odds ratio 1.1, $p=0.83$).[18] A Belgian cohort study[19] found no effect of postnatal smoking by either parent, but an increased incidence of acute otitis media in the offspring of mothers who smoked during the first trimester of pregnancy (odds ratio 1.38, $p<0.05$; no confidence interval published). An increased risk of acute otitis media episodes in the offspring of smokers also emerged in a Finnish cohort[20] but was not tested for significance (odds ratio 1.6). The monthly incidence of acute otitis media among 2512 children in northern Finland[10 21] was significantly higher if parents smoked (odds ratio 1.17, 95% CI 1.06 to 1.30). Among a clinic-based birth cohort in Boston, Massachusetts[12] the cumulative incidence of acute otitis media in the first year of life was also significantly more common if the parents smoked (odds ratio 1.38, 95% CI 1.03 to 1.80).

Three cross sectional studies of schoolchildren have related a history of "otitis"[13] or "ear infection"[8 9] to parental smoking. In two Israeli studies the odds ratios for ear infection among children whose parents smoked were 1.30 (1.04 to 1.64)[8] and 1.12 (1.01 to 1.25).[9] Among Italian children[13] otitis was more common in homes where parents smoked >20 cigarettes daily (odds ratio 1.38, 95% CI 1.00 to 1.89). A fourth cross sectional study from southern Thailand[22] combined history and otological examination to assess the presence of "any otitis media". This composite outcome was positively but non-significantly associated with smoking by the father (odds ratio 1.58, 95% CI 0.89 to 2.83). Maternal smoking was rare and inversely associated with otitis media (odds ratio 0.41, 95% CI 0.06 to 3.00).

Although these studies are too varied to permit a formal meta-analysis, they would be consistent with a weak adverse effect of parental smoking on the incidence of acute otitis media in children, with an odds ratio in the range 1.0–1.6. None adequately addressed dose-response or confounding effects.

Table 2 Design, sample size and recruitment criteria: studies included in meta-analyses

Reference no.	*Year*	*Country*	*Age*	*Design*	*Outcome*	*Sample size*	*Case definition*	*Source of controls or cohort*
Recurrent otitis media								
23	85	Finland	2–3	C-C	ROM	395	>3 PD AOM (OP clinic)	Same health centre as cases
11	86	Finland	<4	Survey	ROM	321	>3 PD AOM recorded	<3 AOM (population sample)
24	88	Finland	<2	Cohort	ROM	108	>5 PD AOM by age 2	No PD AOM, same physician
12	89	USA (MA)	<1	Cohort	ROM	877	>3 PD AOM by age 1	Clinic-based birth cohort
12	89	USA (MA)	<3	Cohort	ROM	698	>3 PD AOM by age 3	Clinic-based birth cohort
12	89	USA (MA)	<7	Cohort	ROM	498	>3 PD AOM by age 7	Clinic-based birth cohort
25	91	USA (NY)	(~4)	C-C	ROM	246	>2 PD AOM in 8 months	Private clinic health check
26	93	Canada	<5	C-C	ROM	170	>4 PD AOM in 12 months	Ophthalmology clinic
10	93	Finland	<2	Cohort	ROM	2512	>3 PD AOM by age 2	Population-based birth cohort
27	95	USA (AZ)	<1	Cohort	ROM	1013	>3 PD AOM in 6 months	Population-based birth cohort
28	95	Canada	<4	Cohort	ROM	918	>4 AOM recalled	Population-based birth cohort
Middle ear effusion: prevalence								
29	85	Denmark	3–6	Cohort	OME	337	Flat tympanogram	Day care centre (6 tests)
30	89	Holland	2–4	Cohort	OME	435	Flat tympanogram	Population sample (9 tests)
31	90	UK	7	Survey	OME	864	Flat tympanogram	Population sample (1 test)
32	92	USA (NC)	<3	Cohort	OME	132	Otoscopy and symptoms	Day care centre attenders
Middle ear effusion: referral for surgery								
1	83	USA (WA)	?	C-C	OME (OP)	152	Operation for OME	General surgical clinic
37	85	UK	4–9	C-C	OME (OP)	442	Operation for OME	Clinic and community controls
38	88	UK	(~6)	C-C	OME (OP)	70	ENT OP referrals	Orthoptic clinic
39	89	UK	1–12	C-C	OME (OP)	151	Grommet insertion	Orthoptic clinic
40	91	UK	1–11	C-C	OME (OP)	230	Grommet insertion	Orthopaedic and eye clinics
41	91	Germany	1–8	C-C	OME (OP)	328	Otalgia and deafness	Various paediatric clinics
42	92	UK	2–12	C-C	OME (OP)	163	Bilateral OME >3 months	Orthopaedic & surgery clinics
43	93	Sweden	<7	Cohort	OME (OP)	1022	Grommet insertion	Population-based birth cohort
44	95	USA (AL)	<3	C-C	OME (OP)	350	Grommet insertion	General paediatric clinic
Tonsillectomy and/or adenoidectomy								
48	78	France	10–20	Survey	Ad/tons	3920	Recall of surgery	General population sample
49	86	UK	2–15	Survey	Tonsils	154	Tonsillectomy	Children of hospital visitors
11	86	Finland	<4	C-C	Adenoids	425	Adenoidectomy and ROM	General population sample
50	93	UK	(~6)	C-C	Tonsils	120	Tonsillectomy	Orthoptic clinic

AOM = acute otitis media; ROM = recurrent otitis media; OME = otitis media with effusion (glue ear); OP = outpatients; PD = physician-diagnosed; C-C = case-control study.
Reference 11 appears twice but with mutually exclusive comparisons.
Reference 12 appears three times with potentially overlapping comparisons (but sample attrition).

RECURRENT OTITIS MEDIA

The quality of epidemiological evidence improves considerably for recurrent otitis media,[10–12 23–28] usually defined as more than a specified number of episodes of physician-diagnosed acute otitis media in a defined study period (table 2). Most of these studies report on the relationship of recurrent otitis media to smoking by either parent, although two[25 27] present data only for smoking by the mother or the father separately (table 3). The more recent studies have adjusted for multiple confounders and derive similar odds ratios before and after adjustment (table 4), suggesting that uncontrolled confounding is unlikely to be a major issue in the interpretation of the crude odds ratios. All three studies which tested for the presence of a dose-response relationship found it to be significant at the 5% level (table 3).

One birth cohort study[12] reports on the relationship of parental smoking to recurrent otitis media at three ages (1, 3 and 7 years). The size of the cohort is different at each age due to sample attrition, but the case group increases because of accumulation of children with at least three episodes of otitis media. For the purposes of meta-analysis the results from the three year follow up are used, as this age corresponds most closely to that used in other similar studies.

Figure 1 summarises the results of a comparison of children of smoking and non-smoking parents. There is weak evidence of heterogeneity among the seven odds ratios for either parent smoking ($\chi^2 = 13.5$, df = 6, p = 0.036). Some variation is to be expected given the different age ranges and case definitions in the various studies. Under the fixed effects assumption the pooled odds ratio for recurrent otitis media if either parent smokes is 1.41 (95% CI 1.19 to 1.66). Using the random effects model the pooled estimate is 1.48 (95% CI 1.08 to 2.04). This may be a slight overestimate as it is greater than the odds ratios for maternal smoking in the two studies that reported only this measure and not the effect of either parent smoking (table 3).

MIDDLE EAR EFFUSION

Population surveys

Four cross sectional or longitudinal studies of general population samples have objectively measured the presence of middle ear effusion by tympanometry[29–31] or otoscopy.[32] All found an increase in prevalence of "glue ear" in children exposed to parental smoking (table 3), despite some differences in the age of the subjects. In one of the tympanometric studies[31] a significant dose-response relationship was found with the salivary level of cotinine, thus offering an entirely objective assessment of the association of environmental tobacco smoke and middle ear effusion.[33] In the longitudinal study of otoscopic abnormalities among day-care attenders[32] serum cotinine levels were used to define exposed and unexposed children.

Figure 1 summarises the results of these studies. There is no evidence of heterogeneity of odds ratios in a pooled analysis of these four studies ($\chi^2 = 0.7$, df = 3, p = 0.87) and the fixed effects assumption is therefore appropriate. The pooled odds ratio for glue ear if either

Table 3 Unadjusted relative risks associated with parental smoking in each study

Reference no.	Outcome	Cases	Controls	Odds ratios (95% CI) for smoking by: Either parent	Mother	Father	Dose-response present?	
Recurrent otitis media								
23	ROM	188	207	1.96 (1.28 to 3.00)			–	
11	ROM	100	221	1.54 (0.93 to 2.56)			–	
24	ROM	28	80	2.40 (0.91 to 6.33)			–	
12	ROM <1y	129	748	1.42 (0.96 to 2.11)			–	
12	ROM <3y	303	395	1.04 (0.76 to 1.43)			–	
12	ROM <7y	368	130	1.18 (0.77 to 1.80)			–	
25	ROM	125	246	–	0.90 (0.54 to 1.50)	0.83 (0.50 to 1.39)	–	
26	ROM	85	85	2.54 (1.23 to 5.41) m			Yes	Total cigs/day
10	ROM	960	1552	1.00 (0.68 to 1.48)			–	
27	ROM	169	844	–	1.33 (0.90 to 1.95)		Yes	Mother >20/day
28	ROM	164	754	1.69 (1.19 to 2.43)			Yes	Total cigs/day
Middle ear effusion: prevalence								
29	OME	183	154	1.55 (0.98 to 2.45)			–	
30	OME	128	307	1.11 (0.59 to 2.09)			No	Total cigs/day
31	OME	82	782	1.41 (0.87 to 2.28)			Yes	No. of smokers*
32	OME	(total = 132)		1.38 (1.21 to 1.56) r			–	
Middle ear effusion: referral for surgery								
1	OME (OP)	76	76	1.45 (0.72 to 2.94)			Yes	No. of smokers
37	OME (OP)	150	292	–			Yes	Cigs × years
38	OME (OP)	26	44	1.10 (0.37 to 3.23)			No	Total cigs/day
39	OME (OP)	115	36	2.04 (0.89 to 4.71)			–	
40	OME (OP)	115	115	0.72 (0.41 to 1.27) m	1.23 (0.70 to 2.15) m		No	Total cigs/day
41	OME (OP)	164	164	–	1.92 (1.20 to 3.06)	1.37 (0.87 to 2.17)	No	Total cigs/day
42	OME (OP)	100	63	1.21 (0.61 to 2.39)			–	
43	OME (OP)	176	846	0.87 (0.49 to 1.55)			–	
44	OME (OP)	175	175	1.65 (1.05 to 2.59) p	1.28 (0.65 to 2.54) p	1.54 (0.89 to 2.66) p	No	No. of smokers
Tonsillectomy and/or adenoidectomy								
48	Ad/tons	1490	2430	2.07 (1.80 to 2.38)	1.68 (1.44 to 1.95)	1.89 (1.64 to 2.17)	Yes	Cigs by each
49	Tonsils	93	61	2.06 (1.06 to 4.00)			–	
11	Adenoids	114	321	2.06 (1.30 to 3.26)			–	
50	Tonsils	60	60	2.10 (1.01 to 4.35)	2.29 (1.02 to 5.13)	1.26 (0.55 to 2.90)	Yes	Estimated ETS

AOM = acute otitis media; ROM = recurrent otitis media; OME = otitis media with effusion (glue ear); OP = outpatients; cigs = cigarettes; ETS = environmental tobacco smoke exposure; r = incidence density ratio (published 95%CI based on episodes rather than individuals may be inappropriately narrow); m = matched analysis; p = 95% CI derived from p value.
* Dose response assessed by salivary cotinine levels appears in a separate paper.[33]

Table 4 Effect of adjustment for potential confounders in each study

Reference no.	Outcome	Exposure	Unadjusted odds ratio	Adjusted odds ratio	Factors adjusted for or addressed in text
Recurrent otitis media					
23	ROM				None
11	ROM				None
24	ROM				SES similar in cases & controls
12	ROM <1y				None
12	ROM <3y				None
12	ROM <5y				None
25	ROM				None
26	ROM	Either parent	2.54	2.68	Age, sex, FH OM, atopy, SES, daycare, breastfeeding
10	ROM	Either parent	1.00	0.99	Sex, sibs, atopy, daycare, breastfeeding
27	ROM	Mother >20/day	2.10	1.78	Sex, sibs, daycare, breastfeeding, FH hayfever
28	ROM	Both parents	2.08	1.80	Sex, FH OM, daycare, SES
Middle ear effusion: prevalence					
29	OME	Either parent	1.55	1.60	Age
30	OME				None
31	OME	Both parents	1.89	1.80	SES, crowding, cooking fuel, dampness
32	OME				Sex. race, infection, atopy, breastfeeding, heating
Middle ear effusion: referral for surgery					
1	OME (OP)	Both parents	2.81	2.80	Age, sex
37	OME (OP)				None
38	OME (OP)				None
39	OME (OP)				None
40	OME (OP)				Age, sex, race, SES (by matching)
41	OME (OP)				Age, sex (by matching), SES (all armed forces)
42	OME (OP)				Area, SES similar in cases & controls
43	OME (OP)				None
44	OME (OP)				Age, area, SES similar in cases & controls
Tonsillectomy or adenoidectomy					
48	Ad/tons				Sex, sibs (separate stratified tabulations)
49	Tonsils				None
11	Adenoids				None
50	Tonsils				Age, sex, SES similar in cases & controls

AOM = acute otitis media; ROM = recurrent otitis media; OME = otitis media with effusion (glue ear); OP = outpatients; PD = physician-diagnosed; C-C = case-control study; FH = family history; SES = socioeconomic status.

parent smokes is 1.38 (95% CI 1.23 to 1.55). The result is almost identical (1.39, 95% CI 1.03 to 1.87) if the analysis is restricted to the three tympanometric studies.

Three other papers have reported briefly upon the association of tympanometric abnormalities with parental smoking (table 1). One[34] reports in lesser detail upon one of the cohorts analysed above.[30] Another is based on a case-control sample drawn from a larger population screening survey [35] and comments only that there were no significant differences

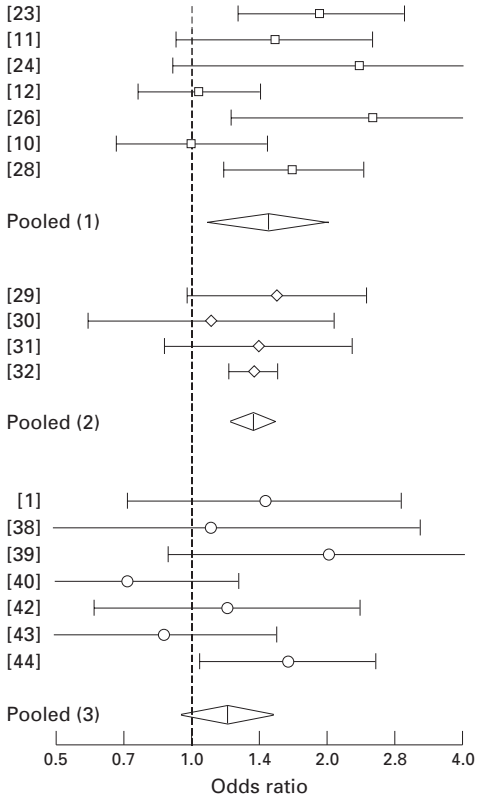

Figure 1 Odds ratios and 95% confidence intervals for the effect of either parent smoking on middle ear disease in children. Open squares = recurrent otitis media (pooled odds ratio 1, random effects); open diamonds = middle ear effusion (pooled odds ratio 2, fixed effect); open circles = outpatient referral for glue ear (pooled odds ratio 3, fixed effect). See table 3 for data points. Studies are displayed in the order in which they appear in table 3. Note that the x axis is logarithmic. The pooled odds ratios are: pooled (1) 1.48 (95% CI 1.08 to 2.04), pooled (2) 1.38 (95% CI 1.23 to 1.55), pooled (3) 1.21 (95% CI 0.95 to 1.53).

between 67 cases and 134 controls in exposure to two or more household smokers. The third study[36] relates to tympanometric findings among a sample of 45 outpatients, half of whom were attending for acute otitis media. Of those exposed to smoking in the home, 65% had abnormal tympanograms compared with 29% of those unexposed (odds ratio 4.9, 95% CI 1.4 to 17.2). This study is difficult to interpret because of the inclusion of a high proportion of subjects with acute ear infection.

Clinic referrals

Nine studies[1 37–44] address the relationship between exposure to environmental tobacco smoke and outpatient referral or operative intervention for glue ear (table 2). These are mostly of case-control design, with controls selected from non-ENT clinics, although one birth cohort study is included.[43] The age range in many of these studies is wide, although most of the subjects are under eight years of age. No studies under this heading were excluded from the quantitative overview, although two were excluded from the meta-analysis. One of these reported a significant dose-response relationship to cumulative passive tobacco exposure but no odds ratio for current parental smoking,[37] and the other[41] presented odds ratios for maternal and paternal smoking separately but not in combination (table 3).

Figure 1 summarises the results for the remaining seven studies. The odds ratios do not display significant heterogeneity ($\chi^2 = 8.1$, df = 6, $p = 0.23$) so the fixed effects assumption applies. The pooled odds ratio for either parent smoking is 1.21 (95% CI 0.95 to 1.53). The odds ratios reported separately for maternal and paternal smoking in one of the excluded studies[41] are higher than this (table 3), but where the effects of mother's smoking or father's smoking can be compared within a study with the odds ratio for either parent smoking,[40 44] the latter is greater (table 3).

Six of the eight studies assessed dose-response and in only two of these was it significant at the 5% level (table 3). Only one study[36] compared odds ratios before and after adjustment, and then only for age and sex. However, several of the case-control studies were matched for age, sex, and socioeconomic status or comment that these variables were similarly distributed in cases and controls (table 4). The extent of residual confounding by unmeasured factors related to disease incidence, prognosis, or referral thus remains uncertain.

Natural history

Middle ear effusion commonly resolves spontaneously and about one third of cases may remit between outpatient referral and operative treatment. One of the case-control studies discussed above[39] found a similar rate of spontaneous resolution in children of at least one smoking parent (31.5% of 130 ears) as in those of non-smoking parents (31% of 100 ears).

Insight into the long term natural history of untreated effusions emerges from controlled trials of operative intervention for glue ear.[45 46] Among 133 children followed for five years after adenoidectomy or adenotonsillectomy, persistence of fluid at the end of the study was three times more likely if either parent smoked (odds ratio 3.32, 95% CI 1.17 to 9.41).[46] A similar finding emerged by survival analysis in a trial of unilateral grommet insertion for middle ear effusion.[45] Among 66 untreated ears followed over five or more years spontaneous resolution of fluid was less common (hazard ratio 0.44, 95% CI 0.22 to 0.87). This corresponds approximately to a twofold or threefold difference in rates of resolution.

HEARING LOSS

Only one study relating parental smoking directly to hearing impairment was found.[47] This was based on a sample of 87 Irish children attending for routine developmental screening at 10 months of age. A persistently abnormal distraction test was five times more common in infants passively exposed to cigarette smoke ($p<0.05$) and the authors calculated that 75% of the cases of hearing loss were statistically

attributable to exposure to environmental tobacco smoke. This small study deserves to be repeated on a larger scale.

ADENOIDECTOMY AND TONSILLECTOMY

Four studies[11 48–50] relating to adenoidectomy, tonsillectomy or adenotonsillectomy without specific reference to glue ear as an indication were identified (table 2). These were remarkably consistent in the odds ratio relating to either parent smoking (table 3). There is no heterogeneity of odds ratios ($\chi^2 = 0.00$, df = 3, p = 0.99) and the pooled odds ratio is 2.07 (95% CI 1.82 to 2.35).

The pooled analysis is dominated by the large population survey of French secondary schoolchildren.[48] This study also reported in some detail on patterns of smoking by each parent, and on the exposure-response relationship which was significant ($p<0.05$) whether assessed as the number of smoking parents or as the daily cigarette consumption by each parent separately. The major omission from this study is information on socioeconomic status.

We have previously analysed longitudinal data from the British 1958 birth cohort relating both parental smoking and tonsillectomy to the incidence of childhood asthma.[51] We therefore investigated whether the strong association between household smoke exposure and tonsillectomy could be replicated in this large nationwide cohort. Among 10 931 children whose parents were interviewed at age 16 (in 1974) there were 7864 from homes where one parent smoked. The lifetime prevalence of tonsillectomy as reported by parents was almost the same among children from non-smoking households (20.5%, 630/3067) as among those exposed to parental smoking (20.5%, 1613/7864). The odds ratio is 1.00 with a tight 95% confidence interval (0.90 to 1.11) that does not overlap those of the French study.[48]

Discussion

Evidence from different study designs and for different disease outcomes relating to middle ear disease in young children is remarkably consistent in suggesting a modest increase in the risk associated with parental smoking. A pooled odds ratio of the order of 1.2–1.5 reasonably summarises the effect of either parent smoking on the incidence and recurrence of acute otitis media and the prevalence of middle ear effusion, determined objectively by tympanometry or otoscopy. There is also some evidence that parental smoking adversely affects the long term prognosis of untreated glue ear.

No single study addresses all methodological concerns about selection (referral) bias, information (reporting) bias, or confounding. However, where these have been investigated or excluded by objective measurement, matched design or multivariate analysis, the association of exposure to environmental tobacco smoke and middle ear disease persists with little alteration in the magnitude of effect. It is therefore reasonable to conclude, as have recent overviews,[2 4–6] that a causal relationship between parental smoking and both acute and chronic middle ear disease in young children is likely.

Only one study addressed the effects of parental smoking on ear disease at different ages,[12] but this suggests a slightly stronger association in the first year of life than subsequently, as is evident in studies of lower respiratory illness.[7] Few studies have compared the effect of smoking by the mother and father, and none have compared the effects of prenatal and postnatal exposure to maternal smoking. These are important issues to be addressed by further research.

The published evidence relating parental smoking to adenoidectomy and/or tonsillectomy is sparse and heavily influenced by a single cross sectional study in which both surgical history and parental smoking habits were reported by teenage children.[48] Our previously unpublished findings from the British 1958 birth cohort cast doubt on the generalisability of the French results.[48] The discrepancy may relate to different referral patterns and clinical indications for tonsillectomy in the two countries. In the light of the British findings it would be premature to conclude that a causal association exists between parental smoking and the wide range of disease conditions which may predispose to tonsillectomy.

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