The effect of passive smoking on the risk of otitis media in Aboriginal and non-Aboriginal children in the Kalgoorlie–Boulder region of Western Australia

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ABSTRACT

Objectives: To determine the risk of otitis media (OM) associated with passive smoking in young children, and any competing effect between passive smoking and childcare attendance.

Design, participants and setting: Prospective cohort study of 100 Aboriginal and 180 non-Aboriginal children born in Kalgoorlie Regional Hospital between 1 April 1999 and 31 January 2003. These children underwent routine clinical examinations by an ear, nose and throat specialist up to three times before the age of 2 years, and tympanometry at routine field follow-up visits from the age of 4 months. Childrens' mothers were interviewed at 1–3 weeks postpartum to provide sociodemographic data.

Main outcome measures: Associations between OM and exposure to environmental tobacco smoke (ETS) and childcare attendance.

Results: 82 Aboriginal and 157 non-Aboriginal children attended for routine clinical examinations. OM was diagnosed at least once in 74% of Aboriginal children and 45% of non-Aboriginal children; 64% of Aboriginal children and 40% of non-Aboriginal children were exposed to ETS. Exposure to ETS increased the risk of specialist-diagnosed OM in Aboriginal children (OR, 3.54; 95% CI, 1.68–7.47); few attended childcare. Non-Aboriginal children exposed to ETS but not attending childcare were at increased risk of OM (OR, 1.91; 95% CI, 1.07–3.42) while those attending childcare had no increased smoking-related risk. Tympanometry was performed on 87 Aboriginal and 168 non-Aboriginal children; a type B tympanogram (suggesting fluid in the middle ear) was also associated with passive smoking in Aboriginal children.

Conclusions: Reducing the exposure of children to ETS is a public health priority, especially for the Aboriginal population. A smoke-free environment will help reduce the burden of OM.

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Otitis media (OM) is the most common paediatric illness for which medical advice is sought;[1] in the general population,10%–20% of children have more than three episodes of OM before the age of 1 year.[2] The resultant hearing loss can affect educational outcomes and social circumstances in adulthood. In Aboriginal children, OM typically starts at a younger age, is much more common and is more likely to result in hearing loss than in non-Aboriginal children.[3-6]

Passive smoking can increase the adherence of bacteria to the respiratory epithelium, depress local immune function and decrease mucociliary action, and thus may be a risk factor for the development of OM.[7,8] While there are conflicting reports on the link between environmental tobacco smoke (ETS) exposure and the development of OM,[2,9-13] several meta-analyses indicate that passive smoking leads to an increased risk of OM in children of 50%–80%.[13-16] Despite high smoking rates in indigenous populations,[10,17-20] no studies investigating an association between smoking and OM in a cohort of Australian Aboriginal people have been undertaken.

Childcare attendance has also been reported to be a risk factor for OM in young children.[9,11,21,22] However, a competing effect may exist between ETS exposure and attendance at childcare. Children who are exposed to ETS in the home environment and who also attend a childcare facility will have less exposure to ETS than children who do not attend childcare and who live with smokers; therefore childcare may be beneficial for some children. A comparable competing effect between ETS exposure and childcare attendance has recently been reported in a study on passive smoking and pneumococcal carriage, which is associated with increased risk of OM.[23,24]

The Kalgoorlie Otitis Media Research Project (KOMRP) in Western Australia collected social, demographic, environmental and biological data to investigate causal pathways to OM in Aboriginal and non-Aboriginal children.[25] To establish priorities for intervention, we looked at the relationship between passive smoking and development of OM before the age of 2 years among participants in the KOMRP, and investigated any competing effect between passive smoking and attendance at childcare.

METHODS

We undertook this research in the Kalgoorlie–Boulder area of WA, a semi-arid region about 600 kilometres east of Perth. We enrolled 100 Aboriginal and 180 non-Aboriginal children born in Kalgoorlie Regional Hospital between 1 April 1999 and 31 January 2003 into a prospective cohort study. Children were followed regularly to the age of 2 years. Multiple births, children with severe congenital abnormalities and those whose birthweight was less than 2000 g were not eligible. Details of the methods used during the study are reported elsewhere.[25]

Aboriginal and non-Aboriginal research assistants interviewed mothers or guardians of the children enrolled in the study 1–3 weeks postpartum in their homes. They collected information on smoking behaviour of parents and other household members. Mothers or guardians were asked: "Do you smoke now?" and "Do you smoke inside the house, outside only or both?" If the baby's father was part of the family unit, we asked the same questions about the baby's father. Lastly, we asked the mother or guardian whether anybody else smoked inside the house. An answer of "yes" to any of these questions was taken to indicate ETS exposure. Information was also collected about feeding practices, parental education and employment, family history of allergy or atopy, and number, age, and childcare or school attendance of other children in the household. The number of adults and chil-

1 Otitis media outcome and demographic, social and environmental predictor variables for 82 Aboriginal and 157 non-Aboriginal children*

Predictor variable		Aboriginal	Non-Aboriginal
Otitis media at least once	Yes	61 (74.4%)	70 (44.6%)
	No	21 (25.6%)	87 (55.4%)
Exposure to environmental tobacco smoke (indoors and outdoors)	Yes	51 (63.8%)	63 (40.1%)
	No	29 (36.2%)	94 (59.9%)
Other children in house	Yes	65 (79.3%)	79 (50.3%)
	No	17 (20.7%)	78 (49.7%)
Mother worked recently	Yes	24 (29.6%)	113 (72.0%)
	No	57 (70.4%)	44 (28.0%)
Mother has post-school education	Yes	33 (41.3%)	121 (68.0%)
	No	47 (58.7%)	36 (32.0%)
Sex	Male	49 (59.8%)	80 (51.0%)
	Female	33 (40.2%)	77 (49.0%)
Exclusive breastfeeding 6–8 weeks postpartum	Yes	34 (46.6%)	98 (64.9%)
	No	39 (53.4%)	53 (35.1%)
Maternal age (years) at delivery	< 25	44 (53.7%)	23 (14.6%)
	25–29	20 (24.4%)	44 (28.0%)
	⩾ 30	18 (21.9%)	90 (57.4%)
Crowding (> 1 person/room)	Yes	59 (72.0%)	21 (13.4%)
	No	23 (28.0%)	136 (86.6%)
Family history of allergy or atopy	Yes	52 (64.2%)	122 (77.7%)
	No	29 (35.8%)	35 (22.3%)
Gestational smoking	Yes	37 (45.7%)	31 (19.7%)
	No	44 (54.3%)	126 (80.3%)
Childcare ever attended	Yes	8 (11.1%)	52 (34.4%)
	No	74 (88.9%)	99 (65.6%)

* Discrepancies between total numbers of children for different variables are the result of missing data. ◆

dren living in the house as well as the number of rooms in the house were documented to obtain a measure of crowding (number of people/room). Research assistants performed further routine follow-up on study participants at ages 6–8 weeks, and 4, 6, 12, 18 and 24 months, when they again enquired about feeding practices and whether the study participant was attending childcare. At these follow-up visits, we also asked the child's mother or guardian whether they smoked, and whether anybody smoked inside the house. However, as preliminary analysis indicated little change from responses at first interview and, in view of potential bias towards children remaining in the study, analyses were based on the first interview.

We scheduled three routine clinical examinations by an ear, nose and throat (ENT) specialist: before the age of 6 months, at 6–11 months and at 12–24 months. A clinical diagnosis was established by otoscopy, pneumatic otoscopy and tympanometry, and was based on national clinical guidelines.[26] Presence of acute otitis media (AOM), otitis media with effusion (OME) or perforation of the tympanic membrane (TM) with or without purulent discharge was recorded.

We performed tympanometry during routine ENT clinics from the age of 4 months onwards. From 1 May 2000, after training by an audiologist (SAW), research assistants also performed tympanometry during their routine field follow-up of children aged 4 months or older; this became routine practice once a dedicated tympanometer became available in July 2002. Interobserver variation was assessed regularly.

The final diagnosis was based on the child's most severely affected ear. If a diagnosis could not be made for one ear (eg, because of wax), then the child's final diagnosis was based on the diagnosis for the other ear.

Ethical approval to conduct this study was given by the WA Aboriginal Health Information and Ethics Committee, Northern Goldfields Health Service and Nursing Education Ethics Committee in Kalgoorlie, Princess Margaret Hospital Ethics Committee and the Confidentiality of Health Information Committee of the Health Department of WA.

Data analysis

Two different outcomes indicative of OM were considered: diagnosis of OM (ie, AOM, OME, TM perforation with or without discharge) at a routine ENT clinic visit, and a type B tympanogram reading (indicative of fluid in the middle ear or a TM perforation) at routine ENT clinic or at field follow-up visits.

Presence or absence of OM diagnosed by an ENT specialist and tympanometry outcomes were analysed by longitudinal logistic growth modelling on a complete case basis with an underlying quadratic age dependence of disease risk assumed. Generalised estimating equations (GEE) with robust standard errors were used in these models to account for the within-subject dependencies in the data. Missing data assumptions inherent in the modelling approach were examined using Mann–Whitney U tests to compare numbers of clinical examinations and log-rank tests for time-to-study-entry (first examination) and loss-to-follow-up times.

The logistic models give rise to odds ratios (ORs) for disease risk which were assumed to be constant over time. Relative risk for a given OR was estimated on the basis of the modelled disease prevalence at 12 months of age[27] and population-attributable risk was estimated from relative risk and observed exposure prevalence.[28]

In all analyses, the primary independent variable was the presence or absence of exposure to ETS, assessed from interview responses. Indoor and outdoor ETS exposures were initially considered separately, but disease risk was not found to be affected by the reported location of the exposure, so these two categories were combined. Potential confounding factors shown in Box 1 were first assessed in univariate models and were included as covariates in final multivariate models if the univariate effect was significant ($P < 0.10$).

Separate analyses were performed for Aboriginal and non-Aboriginal children. SAS, version 9 (SAS Institute Inc, Cary, NC, USA) was used to perform all the longitudinal modelling while SPSS, version 12 (SPSS Inc, Chicago, Ill, USA) was used to perform the cross-tabulations.

2 Results of univariate and multivariate analyses* of risk factors for otitis media diagnosed in 82 Aboriginal and 157 non-Aboriginal children at routine ear, nose and throat clinics

	Aboriginal				Non-Aboriginal			
	Univariate		Multivariate†		Univariate		Multivariate†	
Predictor variable	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
Environmental tobacco smoke exposure	**4.24 (2.03–8.83)**	**< 0.001**	**3.54 (1.68–7.47)**	**< 0.001**	1.28 (0.76–2.15)	0.35	1.32 (0.78–2.23)	0.30
Sex (male v female)	0.76 (0.36–1.61)	0.47			**1.72 (1.03–2.89)**	**0.039**	**1.78 (1.07–2.97)**	**0.027**
Season (winter v summer)	0.66 (0.34–1.29)	0.22			1.20 (0.73–1.96)	0.47		
Mother's age								
25–29 v < 25 years	1.01 (0.44–2.33)	0.98			1.15 (0.51–2.58)	0.73		
⩾ 30 v < 25 years	1.45 (0.62–3.41)	0.39			1.20 (0.58–2.45)	0.62		
Mother with post-school education	0.62 (0.30–1.29)	0.20			1.20 (0.61–2.36)	0.61		
Exclusive breastfeeding at 6–8 weeks	1.30 (0.63–2.69)	0.48			1.22 (0.70–2.14)	0.48		
Other children in house	**3.06 (1.24–7.58)**	**0.016**	**2.75 (1.09–6.96)**	**0.033**	**1.80 (1.05–3.07)**	**0.032**	**1.98 (1.14–3.43)**	**0.016**
Mother employed	0.46 (0.21–1.02)	0.05	0.68 (0.30–1.54)	0.35	0.74 (0.44–1.24)	0.25		
Crowding (> 1 person/room)	**2.67 (1.16–6.18)**	**0.021**	1.77 (0.74–4.24)	0.20	1.20 (0.57–2.52)	0.63		
Family history of allergy or atopy	0.86 (0.39–1.88)	0.70			0.64 (0.35–1.15)	0.14		
Gestational smoking	1.78 (0.86–3.67)	0.12			0.87 (0.45–1.67)	0.68		
Recent childcare attendance	Not calculated‡				1.49 (0.80–2.78)	0.21		

OR = odds ratio. Comparison for each variable was yes v no unless otherwise indicated. Bold type indicates a statistically significant result ($P < 0.05$).
* All models adjusted for age and age^2. † Covariates were included in final multivariate models where the univariate effect was significant ($P < 0.10$).
‡ Very few Aboriginal children attended childcare. ◆

RESULTS

Diagnosis of OM at routine clinic

There were 167 successful routine clinic examinations by an ENT specialist on 82 of the 100 enrolled Aboriginal children. Of these examinations, 101 (61%) resulted in a diagnosis of OM (82 OME, 13 AOM and six TM perforations with purulent discharge). OM was diagnosed at least once in 74% of the Aboriginal children; 64% of Aboriginal children were exposed to ETS (Box 1). Of the 180 enrolled non-Aboriginal children, 157 children (87%) had a total of 364 clinic examinations; 102 examinations (28%) resulted in a diagnosis of OM (91 OME and 11 AOM). OM was diagnosed at least once in 45% of non-Aboriginal children, and 40% of non-Aboriginal children were exposed to ETS (Box 1).

Box 2 shows that exposure to ETS was a significant predictor of OM in Aboriginal children (OR, 3.54; 95% CI, 1.68–7.47, after adjustment for confounders), but was not significant in non-Aboriginal children (OR, 1.32; 95% CI, 0.78–2.23). Assuming an OM prevalence of 49% in Aboriginal children who were not exposed to ETS (this is the fitted mean value for age 12 months), the estimated OR equated to a relative risk for ETS of 1.58. The estimated population-attributable risk of OM resulting from passive smoking among Aboriginal children was thus 27%, assuming the prevalence of ETS exposure found in our study. Other significant (at $P < 0.05$) predictors of OM from multivariate modelling were the presence of other children in the household for both Aboriginal and non-Aboriginal children, and being male for non-Aboriginal children only.

We found a significant interaction between exposure to ETS and childcare attendance in non-Aboriginal children, with childcare attendance reducing the impact of ETS exposure in the home on OM. For children who had not attended childcare in the period before a clinic examination (as assessed by the interview response immediately before the examination), ETS was a significant predictor of OM (OR, 1.91; 95% CI, 1.07–3.42, after adjustment for confounders). This equated to a relative risk of 1.47, and assuming an OM prevalence of 33% among children who were not exposed to ETS, a population-attributable risk of 16%. There was no significant ETS effect for non-Aboriginal children who recently attended childcare (OR, 0.39; 95% CI, 0.12–1.27). This interaction was not investigated among Aboriginal children because they rarely attended childcare (Box 1). Interactions between ETS exposure and crowding (number of persons/room) were not significant.

We investigated associations between the key model predictor, ETS exposure, and both the number of ENT examinations for each child and the age at the first and last examination to determine whether relevant missing-completely-at-random model assumptions were violated. No significant associations were found.

Tympanometry

Tympanometry was performed on 249 occasions on 87 Aboriginal children, either by

research assistants in the field or by audiologists at routine ENT clinics; 148 (59%) were type B tympanograms. Of the Aboriginal children, 73 (84%) had a type B tympanogram on at least one occasion. Tympanometry was performed on 659 occasions on 168 non-Aboriginal children, and 161 (24%) were type B tympanograms; 89 (53%) had a type B tympanogram at least once.

ORs from models of documented type B tympanograms were broadly similar to those for clinical diagnosis of OM. Exposure to ETS was a significant predictor of having a type B tympanogram in Aboriginal children (OR, 2.38; 95% CI, 1.14–4.97, after adjustment for confounders), but was not significant in non-Aboriginal children (OR, 1.12; 95% CI, 0.70–1.79).

DISCUSSION

We found that children exposed to passive smoking in the home, and who were not attending organised childcare were at increased risk of OM. The relative risk of OM related to passive smoking in our study was consistent with that found in previous studies.[13-15] Our results highlight the importance of reducing childrens' exposure to passive smoking, and that this is particularly important among Aboriginal people in whom rates of both smoking and OM are high. While Australia has been a world leader in reducing ETS exposure in public places and in reducing smoking rates in the general population, public health campaigns have not been as successful in Indigenous populations. European colonisation resulting in marginalisation and social deprivation, the lack of sustained appropriate programs, and smoking being seen as the norm in many communities are some of the reasons for limited success in reducing smoking rates among Indigenous Australians.[29-31] However, with an estimated population-attributable risk of OM due to passive smoking of 27%, such campaigns have the potential to reduce the burden of OM substantially. There is a need for more input by Indigenous Australians in the development of intervention programs, and a need for appropriate levels of funding to ensure programs are sustainable and to improve access to nicotine replacement therapy.[29]

We found that attending childcare reduced the risk of OM among those exposed to passive smoking, presumably because children who attend childcare spend less time among people smoking in the home environment. This is in keeping with studies elsewhere indicating that the greatest exposure of children to tobacco smoke is in the home and in cars, and that interventions aimed at banning smoking in homes can reduce ETS exposure.[32,33] Contrary to findings of other studies,[9,11,21,22] we did not find childcare attendance to be an independent risk factor for OM. Childcare attendance was rare among Aboriginal children in our study, in line with nationwide patterns.[34] A previous study has concluded that appropriate childcare facilities for Aboriginal children are urgently needed to improve early development and educational outcomes,[35] and our findings suggest that, given the high rates of smoking in and around homes, such facilities may offer the additional benefit of reducing the burden of OM in Aboriginal children.

A major strength of our study was its inclusion of both clinic-based and field-based outcome measures for OM. Also, the longitudinal nature of the study ensured that the progress of the study population was closely monitored to the age of 2 years, and all routine clinical assessments for OM were included in the analyses. Our findings appear to conflict with those of a recent study involving indigenous North American children, which found no association between passive smoking and any documented episode of OM in the first 6 months of life.[10] However, when we analysed our data to the age of 6 months, we found that the association did not reach significance and that it was necessary to include all OM diagnoses up to age 2 years to achieve sufficient power to detect the association between passive smoking and OM.

A potential limitation of our study was that exposure to ETS was assessed by interview response, rather than by measurement of urine cotinine levels which can detect the presence and the amount of tobacco smoke exposure. Many Aboriginal children have multiple carers, and it is possible that the person who was interviewed was not always familiar with the smoking habits of all carers and others visiting or living with these other carers. Furthermore, indoor and outdoor exposure may not have been documented reliably. Despite these limitations, the relative risk of OM associated with passive smoking found in our study was consistent with results of a study which used cotinine levels.[36]

In our study, there may have been incomplete assessment of socioeconomic status, as the household income of study participants was not recorded. Instead we relied on indicators such as maternal education, employment and crowding to capture the variation in socioeconomic status which may confound the association between smoking and disease. Indeed, it has been argued elsewhere that smoking may be merely a surrogate marker of socioeconomic disadvantage.[12]

The multidisciplinary nature of the KOMRP provides an ideal opportunity to examine the relative contributions of upper respiratory tract bacterial carriage, mucosal immune function and socioeconomic risk factors to the causal pathways leading to OM. We have shown an association between passive smoking and OM, and a beneficial effect of childcare in this context. We conclude that interventions to reduce the exposure of young children to ETS, especially in the home environment, should be urgently applied and evaluated.

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COMPETING INTERESTS

None identified.

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