Published in final edited form as:

Acta Paediatr. 2010 January ; 99(1): 99–105. doi:10.1111/j.1651-2227.2009.01506.x.

Pre- and Postnatal Parental Smoking and Acute Otitis Media in Early Childhood

Siri E. Håberg, MD, PhD¹, Yngvild E. Bentdal, MD^{2,3}, Stephanie J. London, MD, PhD⁴, Kari J. Kværner, MD, PhD^{2,5}, Wenche Nystad, PhD¹, and Per Nafstad, MD, PhD^{1,6}

¹ Division of Epidemiology, Norwegian Institute of Public Health, Oslo, Norway

² Faculty Division of Akershus University Hospital, University of Oslo, Norway

³ Department of Otorhinolaryngology, Akershus University Hospital, Norway

⁴ Epidemiology branch and Laboratory of Respiratory Biology, National Institute of Environmental Health Sciences, National Institutes of Health, Department of Health and Human Services, Research Triangle Park, NC, USA

⁵ Department of Special Needs Education, University of Oslo, Norway

⁶ Department of General Practice and Community Medicine, Medical Faculty, University of Oslo, Norway

Abstract

Aim—To explore associations between acute otitis media in early childhood and prenatal and postnatal tobacco smoke exposure.

Methods—Subjects were 32,077 children born 2000 – 2005 in the Norwegian Mother and Child Study with questionnaire data on tobacco smoke exposure and acute otitis media up to 18 months of age. Multivariate regression models were used to obtain adjusted relative risks for acute otitis media.

Results—Acute otitis media was slightly more common in children exposed to parental smoking. The incidence from 0–6 months was 4.7% in unexposed children, and 6.0% in children exposed both pre- and postnatally. After adjusting for postnatal exposure and covariates, the relative risk for acute otitis media 0–6 months when exposed to maternal smoking in pregnancy was 1.34, 95% confidence interval: 1.06–1.69. Maternal smoking in pregnancy was associated with acute otitis media up to 12 months of age. Compared to non-exposed children, there was a slightly increased risk of recurrent acute otitis media for children exposed both pre- and postnatally with a relative risk of 1.24, 95% confidence interval: 1.01–1.52.

Conclusion—Even in a cohort with relatively low exposure levels of parental smoking, maternal smoking in pregnancy was associated with an increased risk of acute otitis media in early childhood.

Keywords

Acute otitis media; Smoking; Pregnancy; Norway; Cohort studies

Correspondence: Siri Eldevik Håberg, Division of Epidemiology, Norwegian Institute of Public Health, P.O. Box 4404, Nydalen, NO-0403 Oslo, Norway, Telephone: +4721078332/+4793426990, Fax: +4721078260, siri.haberg@fhi.no.

There are no financial or other issues that might lead to conflict of interest.

INTRODUCTION

Numerous studies have addressed adverse effects of tobacco smoke exposure on respiratory health in early childhood (1–3), including associations between tobacco smoke exposure and early life middle ear infections (4–7). In 2006, the US Surgeon General reviewed available literature on the relationship between parental smoking and otitis media in children (8), and concluded that there was sufficient evidence “to infer a causal relationship between parental smoking and middle ear disease in children, including acute and recurrent otitis media and chronic middle ear effusion” (8). However, only three studies were eligible for pooled analyses of parental smoking and risk of acute otitis media (AOM) (8–11), each of these studies included less than 750 children. The pooled analyses did not find parental smoking to increase the risk of acute middle ear infections in children (8).

Increased awareness on adverse health effects of involuntary tobacco smoke exposure has probably reduced secondhand smoking in many westernized societies. Data from the US and from Norway support the view that exposure levels have declined during the last decades (12–14). Much of the existing knowledge on health effects of secondhand smoking is based on studies conducted in populations with higher exposure levels than are common in the same societies today. Few studies have considered different types of exposures to parental smoking in relation to otitis media in children, for example prenatal versus postnatal exposure (6), and possible diverging effects of maternal and paternal smoking (15–17).

Questionnaire data from 32,077 children was used to assess associations between parental smoking and early life middle ear infections in a population with low smoking exposure, accounting for a variety of potential confounding factors.

SUBJECTS AND METHODS

Subjects

This study is based in the Norwegian Mother and Child Cohort Study (MoBa) conducted by the Norwegian Institute of Public Health. The MoBa study is described elsewhere.(18). In brief, MoBa is a pregnancy cohort started in 1999, including 100,000 pregnant women by summer 2008. Around 44% of the invited women agreed to participate. The current study included the first 32,077 children born between 2000 and 2005, for which questionnaires up to 18 months of age were obtained. Children included in this study had information available from four questionnaires: from week 13 – 17 of pregnancy, from week 30 of pregnancy, and from age 6 months and 18 months. The questionnaires can be viewed at the MoBa website (19). In addition, information from the Medical Birth Registry of Norway (MBRN) was available (20).

Written informed consent was obtained from each participant before inclusion in MoBa, and the study has been approved by the Regional Committee for Ethics in Medical Research, the Norwegian Data Inspectorate and the Institution Review Board of the National Institute of Environment Health Sciences, USA.

Methods

The main outcome measures were maternal report of acute otitis media (AOM) in the child up to ages 6, 12 and 18 months as reported in questionnaires at 6 and 18 months of age. Mothers were asked if their child ever experienced ear infections, and AOM was defined as any episode reported in the questionnaire at 6 months, and in the 18 months questionnaire in age intervals from 6 – 11 months, and 12 – 18 months. Recurrent episodes of AOM were defined according to the suggested definition of four or more episodes in a twelve month period (21), in this study from age 6 months to 18 months. Outcomes were dichotomous,

and reference children were those with negative answers on AOM-questions in respective questionnaires.

Smoking data were obtained from three questionnaires; pregnancy weeks 17 and 30, and the 6 month post-partum questionnaire which covered the last three months of pregnancy and maternal and paternal smoking after birth. We used the mother's responses regarding both her smoking and smoking by the child's father. Children were defined as exposed to maternal smoking in pregnancy if their mother smoked daily (more than zero cigarettes per day) or occasionally at any point in pregnancy. Early pregnancy exposure was maternal smoking reported in week 17, and exposure later in pregnancy was any report of maternal smoking in the subsequent questionnaires covering the pregnancy. Average number of cigarettes per day smoked in pregnancy was calculated from the reported numbers on the three questionnaires covering the pregnancy. Occasional smokers were assigned a value of 0.5 cigarettes per day. Children with fathers who smoked either daily or occasionally at any point during their partner's pregnancy were regarded as exposed to prenatal paternal smoking. Postnatal exposure was defined as any smoking by mother or father in the first three months post-partum.

In the regression models we included covariates that presumptively were associated with parental smoking either in pregnancy or after birth, and at the same time possibly affected the risk of AOM. In addition to the child's sex, several potential confounders were included as covariates: maternal atopy, maternal educational level, maternal age, season of birth, parity, birth weight, preterm birth, and breastfeeding at 6 months of age. Although birth weight and preterm birth (born before 37 completed weeks of gestation) may be considered to be on the causal pathway of effects related to prenatal smoking exposure, these birth characteristics may influence parental smoking after birth and were therefore included in the full model. However, analyses were also done without birth weight and preterm birth in the models. Daycare attendance in the first year of life is uncommon in Norway and was not included in the final model, but analyses were also done with daycare up to 18 months of age as a covariate (in three categories: home with parent, nanny/private home, kindergarden).

Data were analyzed using Stata 9.2 (Stata Corporation, College Station, Texas). For regression analyses, we used the binreg command with the relative risk option. This is a generalized linear model with a log-link for binary data.

Fathers' smoking during pregnancy and after the birth was highly correlated (tetrachoric correlation = 0.99). Hence, we did not expect to be able to disentangle effects of pre- and postnatal contributions of fathers' smoking, and modeled only fathers' postnatal smoking. We created a postnatal exposure variable with four mutually exclusive categories – neither parent, mother only, father only, and both parents smoked after the birth. To attempt to examine independent effects of maternal smoking in pregnancy versus postnatal smoke exposure, we ran models with maternal smoking in pregnancy and this postnatal exposure variable. Results are presented with pre- and postnatal smoking exposures not adjusted to each other and mutually adjusted.

As an alternative to adjustment, we also created an exposure variable of four mutually exclusive categories of maternal smoking in pregnancy and parental smoking (either parent) first three months after birth: not exposed in pregnancy or after birth, exposed in pregnancy but not after birth, exposed after birth but not in pregnancy, and exposed both in pregnancy and after birth.

Dose-response relationships were investigated by exploring effects in relation to number of cigarettes smoked in average by the mother during pregnancy. We stratified by maternal

atopy to explore if effects of smoking differed in children of atopic and non-atopic mothers. We also investigated effects of maternal smoking at different times in pregnancy (early only, late only, and continuously) by comparing children exposed at different times in pregnancy to children not exposed to maternal smoking in pregnancy. When obtaining adjusted relative risks the nine covariates listed in Table 1 were included in the models. Children without information on AOM were not included in analyses, amounting to 4.2% of respondents to the 6 months questionnaire and 1.8% of respondents to the 18 months questionnaire. Children with missing information on exposure to maternal smoking in any of the questionnaires covering the pregnancy amounted to 6.5%. For postnatal exposure 7.0% of the children had missing information. Children with missing information were not included in the respective variables for prenatal and postnatal exposure to smoking.

RESULTS

Table 1 presents the smoking prevalence in different categories of covariates, and the percentage of children exposed to maternal smoking in pregnancy and parental smoking (smoking by either parent) after birth. Smoking was unevenly distributed according to categories of several covariates, including breastfeeding at 6 months of age, maternal education, and maternal age. In the first three months post partum, 9.0 percent of children had mothers who smoked. Only 1.6% of the children were exposed to maternal smoking postnatally but not prenatally. Mothers who smoked during pregnancy smoked an average of 4.1 cigarettes per day. Only 83 women reported smoking an average of 15 or more cigarettes per day in pregnancy. After birth, smoking mothers smoked an average of 5.0 cigarettes per day. Fathers smoked an average of 9.1 cigarettes per day after birth.

Table 2 presents crude and adjusted effects of prenatal exposure to maternal smoking and postnatal exposure in four categories: no postnatal exposure, maternal only, paternal only or both parents smoking after birth. AOM before 12 months of age and recurrent AOM were more common among children exposed to smoking, and the incidence proportions were higher in children exposed to maternal smoking in pregnancy. After controlling for postnatal exposure, effects of maternal smoking in pregnancy (presented in table 2) were greatest for AOM before 6 months of age, weaker in children 6 to 11 months, and for AOM between 12 and 18 months the effect was weakest in magnitude and did not reach statistical significance. Crude effects of postnatal exposure weakened after adjustment for covariates.

After adjusting for prenatal exposure, postnatal exposure to parental smoking was not independently associated with significant increased risks of AOM at any age. For recurrent AOM we did not obtain significant effects for any of the smoking exposures when maternal smoking in pregnancy and postnatal exposure (smoking by either parent) were mutually adjusted for each other as shown in table 2.

Table 3 shows incidence proportions of AOM and recurrent AOM and relative risks in mutually exclusive smoking categories based on prenatal (maternal) and postnatal (parental) timing of exposure. Children who neither were exposed to maternal smoking in pregnancy nor to parental smoking after birth had the lowest incidence of AOM before 12 months of age, and the lowest incidence of recurrent AOM. The risk of AOM up to 6 months of age, and recurrent AOM from 6 to 18 months of age was increased in children who were exposed both before and after birth. Postnatal exposure alone did not significantly increase the risk of AOM or recurrent AOM.

Of the 3260 children exposed to maternal smoking in pregnancy, only 515 children were exposed in early pregnancy only (mothers stopped smoking during pregnancy), and 430 children were exposed only after week 17 (mothers started smoking during pregnancy). We

found no evidence of different effects between maternal smoking early versus late in pregnancy. Also, there was no evidence of dose response effects related to number of cigarettes smoked (data not shown). Further, effects on AOM of tobacco smoke exposure in pregnancy or after birth did not materially differ between children with atopic and non atopic mothers. We repeated analyses without birth weight and preterm birth in the models, and also with models including daycare attendance up to 18 months as a covariate, and the findings remained essentially unchanged.

DISCUSSION

The incidence proportions of AOM up to 18 months of age were highest in children exposed to maternal smoking in pregnancy. After adjusting for potential confounders and postnatal tobacco smoke exposure, maternal smoking in pregnancy was associated with AOM up to 12 months of age with greatest impact in the first 6 months of life. Independent effects of postnatal exposure were not found. However, children who were exposed both in utero and after birth had increased risk of recurrent AOM from 6 to 18 months of age.

Few studies have addressed effects of pre- and postnatal exposure to smoking on acute otitis media in childhood, and only one study from 1999 focused on prenatal smoking exposure and offspring risk of middle ear disease (6). This study found strong dose-response effects with amount smoked by the mother during pregnancy. They also found that the association between acute ear infections and maternal smoking during pregnancy was independent of postnatal smoking exposure. Even so, there seems to be a general agreement that early life exposure to tobacco smoke products increases the risk of developing middle ear infections (8). The mechanism linking in utero cigarette exposure with the development of later middle ear infections is unknown. Maternal smoking during pregnancy has been reported to cause histological changes in fetal alveolar and bronchial epithelium (22). This also may apply to the epithelium of the middle ear and the Eustachian tube. An alternative explanation is that in utero smoke exposure may interfere with the immune system, predisposing the child to otitis media (6).

Many mechanisms have been proposed as to why passive smoking after birth may lead to AOM, including ciliostasis, goblet cell hyperplasia, and mucus hypersecretion, which could cause accumulation of mucus and bacteria in the middle ear (23). In the western world, increased awareness of adverse effects of tobacco smoke exposure have probably reduced parents' smoking and involuntary exposure to tobacco smoke in children. In Norway, increased parental awareness and legislation against public smoking may have contributed in reducing the number of children exposed to parental smoking and lowered the level of exposure in children who are still exposed. Around 11% of mothers participating in this study reported smoking in pregnancy, which was a lower prevalence than for pregnant women in general in Norway (24). The exposure levels found in MoBa may represent future exposure levels in Norway and in other countries with decline in parental smoking, thus, the MoBa study enables us to explore impacts on childhood respiratory health in a population with low exposure levels and high awareness.

The participation rate was around 44%, and when comparing women participating in MoBa with the birth registry which includes all births in Norway, there were indications of some differences between MoBa participants and the general population. For example, women in MoBa were slightly older and smoked less. Thus, prevalence estimates based on MoBa may not be representative for the Norwegian population in general. However, lower smoking prevalence and smoking intensity in the study populations would not necessarily bias associations, but could make it more difficult to detect effects and dose-response relationships.

The number of smoking parents and number of cigarettes consumed by those who smoked was quite low compared to some earlier studies (6,10). Even so, we found increased occurrence of AOM and recurrent AOM in children exposed to tobacco smoke, and our findings support the view that maternal smoking in pregnancy is of most importance. After controlling for several potential confounders and after mutually adjusting the different exposure indicators to each other, the effects were rather weak, suggesting, as expected, that low exposure levels are associated with weak effects.

AOM and smoking habits were assessed by self administered questionnaires. This is a common method of addressing health effects of tobacco smoke exposure in large studies, but may lead to some inaccuracy in outcomes and exposures. Random misclassification would be expected to dilute associations, and any systematic misclassification would most likely also lead to underestimation of associations in this study. If differential misclassification were to occur, it would most likely occur because of underreporting of outcomes among smoking parents. This would lead to underestimations of effects. However, the validity of self reported maternal smoking in pregnancy is supported by several studies (25,26). Postnatal exposure levels obtained through questionnaires are probably less accurate than prenatal exposure. Levels of tobacco smoke exposure postnatally may be influenced by conditions difficult to account for, such as differences in time and space between the child and the smoker, room size and ventilation, number of smokers in the room, and so on. Hence, associations with postnatal exposures may have been attenuated due to these sources of misclassification. Children who are exposed both in pregnancy and after birth may have mothers who smoked more heavily in pregnancy and after birth than mothers who stopped smoking at either time point. It may also be that continuous smokers have partners who smoked more heavily than partners of non-smoking mothers. This would give higher exposure levels in utero and also a higher postnatal exposure compared to children exposed only in utero or only after birth. The effects found for children exposed both in utero and after birth are difficult to disentangle into pre- and postnatal contributions. Also, it may be that mothers who smoke continuously in and after pregnancy differ in several ways from mothers who stop smoking at some point. In analyses, estimates were obtained by adjusting for relevant characteristics that may differ between these groups of mothers, however confounding not unaccounted for may have influenced the results. Postnatal smoking exposure included maternal and paternal smoking. More fathers than mothers smoked after birth, and few mothers who did not smoke in pregnancy started smoking after giving birth. Usually the mothers spend more time with their children during the first year, and exposure levels related to paternal smoking may be low, which combined with little contrast in maternal smoking behavior, may make postnatal effects difficult to capture.

Reports of AOM were based on maternal reports, and some children with AOM could be missed while others may be wrongly classified as AOM. Nevertheless, misclassification of AOM is likely to dilute the true associations. Parents have been found to both over- and under report episodes of otitis media in children in questionnaires when comparing with medical records (27). However, a recent study found that parental reports of children's recent AOM history correlated well with medical records (28), thus in the current study where mothers were asked relatively shortly after disease episodes, reports of AOM would probably be trustworthy. Although the AOM incidence is lower than in some studies from other westernized countries, in Norway there is a high rate of breast feeding and low daycare attendance in the first year of life, and these factors may contribute to a lower incidence. The incidence of AOM in the first year of life in this study is similar to what has been found in another Norwegian study population (29). It could be speculated that AOM could be present, but undiagnosed in young children. If this were to happen more commonly among children of smokers, it would most likely lead to underestimation of effects. Misclassification of

smoking and AOM may bias results in positive directions (for example if smokers reported more AOM), however we believe this is less likely.

Few studies have investigated dose-response relations between parental smoking and AOM, and results are not consistent (7,9,11,30). Lack of dose dependent effects may reflect the methodological challenges in obtaining correct levels of exposures. Even if this study used information from a cohort of more than 30,000 children, there were few heavy smokers, limiting the power to detect dose response relationships and to explore effects related to timing of exposure. Disentangling of effects of pre- and postnatal parental smoking were challenging, as smoking exposures were correlated and even in this large cohort relatively few children had contrasts of high and low exposure levels.

This study supports earlier findings of increased occurrence of AOM in children exposed to tobacco smoke in early life. Even in a society with rather low levels of exposure and high parental awareness of adverse effects of passive smoking, maternal smoking in pregnancy was associated with a modestly increased risk of AOM in early childhood.

Acknowledgments

We gratefully acknowledge Hein Stigum at the Norwegian Institute of Public Health in Oslo, Norway, for the advice in statistical analyses and comments given in the methods section. The donations of questionnaire data from MoBa participants are gratefully acknowledged. The Norwegian Mother and Child Cohort Study is supported by the Norwegian Ministry of Health, NIH/NIEHS (grant no N01-ES-85433), NIH/NINDS (grant no.1 U01 NS 047537-01), and the Norwegian Research Council/FUGE (grant no. 151918/S10). This study was supported by the Norwegian Association of Heart and Lung patients with EXTRA funds from the Norwegian Foundation for Health and Rehabilitation, and by the Intramural Research Program of the NIH, National Institute of Environmental Health Sciences.

Abbreviations

AOM	Acute Otitis Media
MoBa	The Norwegian Mother and Child Cohort Study
RR	Relative Risk
CI	Confidence interval

References

1. Cook DG, Strachan DP. Health effects of passive smoking-10: Summary of effects of parental smoking on the respiratory health of children and implications for research. *Thorax* 1999;54:357–66. [PubMed: 10092699]
2. Lux AL, Henderson AJ, Pocock SJ. Wheeze associated with prenatal tobacco smoke exposure: a prospective, longitudinal study. ALSPAC Study Team. *Arch Dis Child* 2000;83:307–12. [PubMed: 10999864]
3. Pattenden S, Antova T, Neuberger M, Nikiforov B, De Sario M, Grize L, et al. Parental smoking and children's respiratory health: independent effects of prenatal and postnatal exposure. *Tob Control* 2006;15:294–301. [PubMed: 16885578]
4. DiFranza JR, Aligne CA, Weitzman M. Prenatal and postnatal environmental tobacco smoke exposure and children's health. *Pediatrics* 2004;113:1007–15. [PubMed: 15060193]
5. Stahlberg MR, Ruuskanen O, Virolainen E. Risk factors for recurrent otitis media. *Pediatr Infect Dis* 1986;5:30–2. [PubMed: 3945573]
6. Stathis SL, O'Callaghan DM, Williams GM, Najman JM, Andersen MJ, Bor W. Maternal cigarette smoking during pregnancy is an independent predictor for symptoms of middle ear disease at five years' postdelivery. *Pediatrics* 1999;104:e16. [PubMed: 10429134]

7. Stenstrom R, Bernard PA, Ben Simhon H. Exposure to environmental tobacco smoke as a risk factor for recurrent acute otitis media in children under the age of five years. *Int J Pediatr Otorhinolaryngol* 1993;27:127–36. [PubMed: 8258480]
8. US Dept. of Health and Human Services. Atlanta: 2006. [homepage on the internet] The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. [cited 2009 March 10]. Available from: URL: www.surgeongeneral.gov/library/secondhandsmoke/report/chapter6.pdf
9. Daly KA, Brown JE, Lindgren BR, Meland MH, Le CT, Giebink GS. Epidemiology of otitis media onset by six months of age. *Pediatrics* 1999;103:1158–66. [PubMed: 10353923]
10. Homoe P, Christensen RB, Bretlau P. Acute otitis media and sociomedical risk factors among unselected children in Greenland. *Int J Pediatr Otorhinolaryngol* 1999;49:37–52. [PubMed: 10428404]
11. Lubianca Neto JF, Burns AG, Lu L, Mombach R, Saffer M. Passive smoking and nonrecurrent acute otitis media in children. *Otolaryngol Head Neck Surg* 1999;121:805–8. [PubMed: 10580242]
12. Pirkle JL, Bernert JT, Caudill SP, Sosnoff CS, Pechacek TF. Trends in the exposure of nonsmokers in the U.S. population to secondhand smoke: 1988–2002. *Environ Health Perspect* 2006;114:853–8. [PubMed: 16759984]
13. US Dept. of Health and Human Services. Atlanta: 2006. [homepage on the internet] The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. [cited 2009 March 10]. Available from: URL: www.surgeongeneral.gov/library/secondhandsmoke/report/chapter4.pdf
14. Statistics Norway. [homepage on the internet]. Smoking in Norway. 2007. [cited 2009 March 10] Available from: URL: www.ssb.no/english/subjects/03/01/royk_en/
15. Adair-Bischoff CE, Sauve RS. Environmental tobacco smoke and middle ear disease in preschool-age children. *Arch Pediatr Adolesc Med* 1998;152:127–33. [PubMed: 9491037]
16. Daigler GE, Markello SJ, Cummings KM. The effect of indoor air pollutants on otitis media and asthma in children. *Laryngoscope* 1991;101:293–6. [PubMed: 2000018]
17. Stenstrom C, Ingvarsson L. Otitis-prone children and controls: a study of possible predisposing factors. 1. Heredity, family background and perinatal period. *Acta Otolaryngol* 1997;117:87–93. [PubMed: 9039487]
18. Magnus P, Irgens LM, Haug K, Nystad W, Skjaerven R, Stoltenberg C. Cohort profile: the Norwegian Mother and Child Cohort Study (MoBa). *Int J Epidemiol* 2006;35:1146–50. [PubMed: 16926217]
19. The Norwegian Institute of Public Health. [homepage on the internet] The Mother and Child Cohort Study: Questionnaires. [cited 2009 March 10] Available from: URL: www.fhi.no/eway/default.aspx?pid=238&trg=MainArea_5811&MainArea_5811=5903:0:15,3138:1:0:0::0:0
20. Irgens LM. The Medical Birth Registry of Norway. Epidemiological research and surveillance throughout 30 years. *Acta Obstet Gynecol Scand* 2000;79:435–9. [PubMed: 10857866]
21. Dowell SF, Marcy SM, Phillips WR, Gerber MA, Schwartz B. Otitis Media---Principles of Judicious Use of Antimicrobial Agents. *Pediatrics* 1998;101:165–71.
22. Chen MF, Kimizuka G, Wang NS. Human fetal lung changes associated with maternal smoking during pregnancy. *Pediatr Pulmonol* 1987;3(1):51–8. [PubMed: 3588051]
23. Etzel RA, Pattishall EN, Haley NJ, Fletcher RH, Henderson FW. Passive smoking and middle ear effusion among children in day care. *Pediatrics* 1992;90 (2 Pt 1):228–32. [PubMed: 1641287]
24. The Medical Birth Registry of Norway. Annual Report 2003–2004. Bergen: 2006.
25. Nafstad P, Kongerud J, Botten G, Urdal P, Silsand T, Pedersen BS, et al. Fetal exposure to tobacco smoke products: a comparison between self-reported maternal smoking and concentrations of cotinine and thiocyanate in cord serum. *Acta Obstet Gynecol Scand* 1996;75:902–7. [PubMed: 9003090]
26. George L, Granath F, Johansson AL, Cnattingius S. Self-reported nicotine exposure and plasma levels of cotinine in early and late pregnancy. *Acta Obstet Gynecol Scand* 2006;85:1331–7. [PubMed: 17091413]

27. Alho OP. The validity of questionnaire reports of a history of acute otitis media. *Am J Epidemiol* 1990;132:1164–70. [PubMed: 2260548]
28. Vernacchio L, Vezina RM, Ozonoff A, Mitchell AA. Validity of parental reporting of recent episodes of acute otitis media: a Slone Center Office-Based Research (SCOR) Network study. *J Am Board Fam Med* 2007;20:160–3. [PubMed: 17341752]
29. Bentdal YE, Karevold G, Nafstad P, Kverner KJ. Early acute otitis media: Predictor for AOM and respiratory infections in schoolchildren? *Int J Pediatr Otorhinolaryngol* 2007;71:1251–59. [PubMed: 17559950]
30. Collet JP, Larson CP, Boivin JF, Suissa S, Pless IB. Parental smoking and risk of otitis media in pre-school children. *Can J Public Health* 1995;86:269–73. [PubMed: 7497415]

TABLE 1

Maternal smoking in pregnancy and parental smoking in the first three months after birth in strata of mother and child characteristics, for children born 2000 – 2005 in the Norwegian Mother and Child Study.

Characteristics	n	Maternal smoking in pregnancy %	Parental smoking after birth %
Overall	32 077	10.9	26.5
Sex			
Male	16 305	10.8	26.4
Female	15 733	11.0	26.6
Maternal education (years)			
≤12	12 481	19.3	38.3
> 12 and ≤16	13 188	6.4	20.9
>16	5697	3.5	14.5
Birth weight (grams)			
< 2500	1223	14.9	28.6
2500 – 4500	29 243	10.9	26.4
> 4500	1537	7.5	26.4
Born preterm (<37 weeks)			
No	29 982	10.9	26.3
Yes	1906	11.5	28.9
Maternal history of atopy			
No	22 665	11.1	26.9
Yes	9412	10.4	25.6
Season born			
Winter	7534	11.4	26.5
Spring	9038	9.8	25.6
Summer	7971	11.2	27.4
Fall	7496	11.3	26.6
Breastfeeding, months			
< 6	6457	21.8	39.9
≥ 6	25 620	8.2	23.3
Maternal age (years)			
< 25	4001	17.4	37.7
25 – 30	14 638	9.5	25.2
> 30	13 438	10.5	24.6
Parity			
0	14 448	10.6	26.1
1	11 329	10.9	27.5
> 1	6300	11.4	25.6

TABLE 2

Incidence proportions (%), crude and adjusted (a) relative risks (RR) with 95% confidence intervals (CI) for acute otitis media at different ages, according to maternal smoking in pregnancy and parental smoking after birth, for 32,077 children born 2000 – 2005 in the Norwegian Mother and Child Study.

		Acute Otitis Media 0 – 6 months				Acute Otitis Media 6 – 11 months				Acute Otitis Media 12 – 18 months				Recurrent Otitis Media 6 – 18 months			
Smoking exposure	n	%	cRR	aRR [‡]	aRR [‡] (95% CI)	%	cRR	aRR [‡]	aRR [‡] (95% CI)	%	cRR	aRR [‡]	aRR [‡] (95% CI)	%	cRR	aRR [‡]	aRR [‡] (95% CI)
Prenatal (maternal)																	
No	26,745	4.8	1	1		17.1	1	1		23.6	1	1		4.4	1	1	
Yes	3260	6.0	1.27*	1.19*	(1.06 to 1.68)	19.4	1.13*	1.09*	1.13*	(1.00 to 1.27)	24.5	1.04	1.03	6.0	1.35*	1.26*	(0.86 to 1.51)
Postnatal (parental)																	
Mother																	
No	21,932	4.7	1	1		17.0	1	1		23.7	1	1		4.4	1	1	
Yes	808	5.4	1.13	1.06	(0.57 to 1.17)	18.8	1.11	1.04	0.97	(0.81 to 1.15)	23.2	0.98	1.00	6.1	1.39*	1.28	(0.76 to 1.69)
Father																	
No	4861	5.0	1.06	1.05	(0.90 to 1.20)	17.8	1.05	1.04	1.04	(0.97 to 1.12)	23.9	1.01	1.01	4.5	1.02	1.02	(0.85 to 1.20)
Yes	1627	5.7	1.21	1.07	(0.62 to 1.12)	18.0	1.06	1.00	0.90	(0.77 to 1.04)	22.7	0.96	0.95	5.6	1.27	1.15	(0.76 to 1.49)

[‡] Adjusted for the child's sex, maternal age, maternal atopy, parity, maternal educational level, season born, birth weight, preterm birth, and breastfeeding at six months.

[‡] Models with maternal smoking in pregnancy and parental postnatal smoking mutually adjusted in addition to adjustment by other covariates.

* P-value < 0.05.

TABLE 3

Incidence proportions (%), crude (c) and adjusted (a) relative risks (RR) with 95% confidence intervals (CI) for acute otitis media at different ages, according to maternal prenatal and parental postnatal smoking, for 32,077 children born 2000 – 2005 in the Norwegian Mother and Child Study.

Smoking exposure		Acute Otitis Media				Acute Otitis Media				Recurrent Otitis Media			
		Age 0 – 6 months				Age 6 – 11 months				Age 12 – 18 months			
Pre	Post	n	%	cRR	aRR [†]	(95% CI)	%	cRR	aRR [†]	(95% CI)	%	cRR	aRR [†]
No	No	21,117	4.7	1	1		16.9	1	1		23.7	1	1
No	Yes	4879	4.8	1.03	1.01	(0.88 to 1.17)	17.8	1.05	1.04	(0.97 to 1.12)	23.6	1.01	1.00
Yes	No	531	5.8	1.23	1.24	(0.86 to 1.77)	19.0	1.12	1.13	(0.94 to 1.35)	24.0	1.03	0.99
Yes	Yes	2591	6.0	1.28*	1.19*	(1.00 to 1.42)	19.1	1.13*	1.08	(0.99 to 1.18)	24.4	1.04	1.03
												1.34*	1.24*

[†] Adjusted for the child's sex, maternal age, birth weight, preterm birth, maternal atopy, parity, maternal educational level, season born and breastfeeding at 6 months.

* P-value < 0.05