

Relation Between Passive Tobacco Smoke Exposure And The Development Of Bacterial Meningitis In Children

Raymond C. Bredfeldt, MD, Stephen R. Cain, Gordon E. Schutze, MD,
Talmage M. Holmes, PhD, and Linda A. McGhee, MD

Background: The adverse effects of passive smoking have received wide attention in recent years. Although a number of childhood illnesses are known to be affected by exposure to tobacco smoke, to date the medical literature is silent about the effect of tobacco smoke on the development of bacterial meningitis in children. The purpose of this study was to learn whether any such association exists.

Methods: Parents of 93 children admitted for bacterial meningitis were surveyed to ascertain which of the children were exposed to passive tobacco smoke. A similar survey was conducted of an age- and sex-matched control group.

Results: Children admitted for bacterial meningitis were significantly more likely to have been exposed to tobacco smoke than was a control group of children admitted for abdominal surgery. ($P=0.017$; odds ratio 2.63, 95 percent confidence interval 1.15, 4.87).

Conclusions: The results of this study indicate that bacterial meningitis in children should be added to the growing list of illnesses associated with passive exposure to cigarette smoke. (J Am Board Fam Pract 1995; 8:95-8.)

Tobacco use is a well-accepted risk factor for a number of illnesses. In recent years concern has been raised about the effect of the exposure to environmental cigarette smoke (passive smoke) as a risk factor in the development of a variety of diseases as well. Children exposed to household tobacco smoke appear to have an increased rate of acute exacerbations of asthma and less efficient pulmonary function.¹⁻⁸ Sudden infant death syndrome might be more common in children exposed to passive cigarette smoke.^{9,10} Children exposed to household tobacco smoke are also more prone to develop otitis media and have prolonged episodes of middle ear effusion following these infections.¹¹⁻¹³ Similarly, exposure to cigarette smoke is a risk factor for hearing deficits in children.¹⁴ Other problems that have been associated with passive tobacco smoke in children include more frequent upper and lower respiratory tract infections, inflammatory bowel disease, and decreased cognitive performance.¹⁵⁻¹⁹

Despite the growing list of childhood illnesses associated with passive cigarette smoke exposure, the medical literature is silent about the effect, if any, cigarette smoke might have on the development of bacterial meningitis in children. Only one report from Europe in 1982 raised the question of a possible link between passive tobacco smoke exposure and meningitis. Haneberg and associates²⁰ noted that passive smoke exposure was more common among 115 children with symptomatic meningococcal disease than in a control population. The purpose of this study was to investigate further whether a link exists between passive exposure to cigarette smoke and the development of bacterial meningitis in children.

Methods

From 1989 to 1992, there were 93 children older than 2 months admitted to Arkansas Children's Hospital with a condition that after appropriate evaluation proved to be bacterial meningitis. Causative organisms included meningococcus (26 percent), *Haemophilus influenzae* (47 percent), and pneumococcus (18 percent). Eight cases (9 percent) were partially treated on admission; as a result, specimens of the cerebral spinal fluid grew no organisms when culture was attempted. Nevertheless, the cerebrospinal fluid had the

Submitted, revised, 12 July 1994.

From the Fayetteville Family Practice Residency, Fayetteville (RCB, LAM), Department of Family and Community Medicine, University of Arkansas for Medical Sciences, Little Rock (SRC, TMH), and Arkansas Children's Hospital, Little Rock (GES). Address reprint requests to Raymond C. Bredfeldt, MD, Fayetteville Family Practice Residency, 2907 East Joyce, Fayetteville, AR 72703.

same characteristics as that found with bacterial meningitis. For the purposes of this study, children less than 2 months of age were excluded, because meningitis in this age group could represent prenatal and neonatal factors.

The caregivers of these children were contacted through a telephone survey. The respondents were questioned whether the child was exposed to passive tobacco smoke in the home or in a day-care setting at the time of admission to Arkansas Children's Hospital. If a smoker resided in the child's household, but did not smoke in the house itself, the child was considered nonexposed.

A similar survey was undertaken of an age- and sex-matched control group of children admitted to the same hospital for abdominal surgery. Surgical diagnoses included hernia repair, appendicitis, intussusception, volvulus, bowel obstruction, and Meckel diverticulum. This group was used as a control, because these surgical problems were unlikely to be related to tobacco exposure.

Telephone respondents were first asked whether a household member smoked at the time of the child's hospitalization. In addition, the respondents were asked whether the child was exposed to passive cigarette smoke in a regular day-care or baby-sitting setting (at least once weekly). Although an attempt was made to quantitate the amount of household cigarette consumption, the relatively small number of study participants necessitated an analysis based on exposure or no exposure.

Finally, each study participant was categorized according to hospital payment method to determine indirectly whether any meaningful difference existed in the socioeconomic status between the study group and the control group. These categories included Medicaid, third-party payer, and self or no pay.

Results

Telephone contact was made after persistent efforts for a total of 73 meningitis cases and 74 control cases. Response rates were 78.6 percent and 79.6 percent, respectively. Forty-five children who had bacterial meningitis had a history of exposure to passive cigarette smoke either in the home or in a day-care setting. In comparison, 30 children similarly exposed were admitted for abdominal surgery ($P=0.017$) (Table 1). This increased exposure to cigarette smoke among chil-

Table 1. Passive Cigarette Smoke Exposure at Home or in Day Care of Children Older Than 2 Months Hospitalized for Meningitis and Abdominal Surgery (Control).*

Status	Meningitis n=73	Control n=74
Exposed	45	30
Unexposed	28	44

* $P=0.017$, odds ratio 2.36, confidence interval 1.15, 4.87.

dren admitted for bacterial meningitis was also seen when household exposure only was analyzed ($P=0.039$) (Table 2). Both study groups had markedly similar patterns in payment methods (Table 3).

Discussion

The results of this investigation indicate that passive exposure to cigarette smoke is associated with increased risk of bacterial meningitis in children. The mechanism by which passive cigarette exposure might increase this risk is one of conjecture. Further studies could be undertaken both to confirm these results and to clarify the mechanism of this phenomenon. Several studies have suggested that cigarette smokers tend to have depressed immune systems.²¹⁻²³ One could theorize that those exposed to passive cigarette smoke could be similarly affected. Cigarette smoke might also have an adverse effect on local defense mechanisms in the nasopharynx. Smokers have been shown to carry *Neisseria meningitidis* longer in the nasopharynx than nonsmokers.²⁴ The results of our study might, therefore, simply reflect a longer exposure time to the infecting organism in the nasopharynx of children exposed to passive tobacco smoke.

Perhaps the greatest criticism of this work is that the study relied on caregiver recall, sometimes of events several years past. Obviously this

Table 2. Passive Cigarette Smoke Exposure at Home of Children Older Than 2 Months Hospitalized for Meningitis and Abdominal Surgery (Control).*

Status	Meningitis n=73	Control n=74
Exposed	40	27
Unexposed	33	47

* $P=0.039$, odds ratio 2.11, confidence interval 1.03, 4.35.

Table 3. Payment Method of Study Participants.*

Payment Method	Meningitis n=73	Control n=74
Medicaid	29	29
Third party	30	28
Self or no pay	14	17

*Chi-square=0.3525; P=0.8384.

bias should have affected both the control and study group similarly. Thus, this bias should not necessarily have had a major effect on the results.

Another potential criticism could be that the control group was matched by age and sex, but not specifically by socioeconomic status. It is conceivable that children of a lesser socioeconomic status, for example, might be more prone to the development of bacterial meningitis and might independently be more likely to be exposed to passive cigarette smoke. That essentially no difference existed between the study group and the control group in hospital reimbursement methods gives indirect evidence that there was no significant difference in the socioeconomic status of these groups.

Despite these potential criticisms, the results of this study indicate that bacterial meningitis is more likely to develop in children exposed to passive cigarette smoke than in those who are not so exposed. Considering the potentially devastating complications of this disease, this information needs to be confirmed and shared with childhood caregivers.

References

- Chilmonczyk BA, Salmun LM, Megathlin KN, Neveux LM, Palomaki GE, Knight GJ, et al. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. *N Engl J Med* 1993; 328:1665-9.
- Schmitzberger R, Rhomberg K, Buchele H, Puchegger R, Schmitzberger-Natzmer D, Kemmler G, et al. Effects of air pollution on the respiratory tracts of children. *Pediatr Pulmonol* 1993; 15:68-74.
- Murray AB, Morrison BJ. The decrease in severity of asthma in children of parents who smoke since the parents have been exposing them to less cigarette smoke. *J Allergy Clin Immunol* 1993; 91:102-10.
- Lebowitz MD, Sherrill D, Holberg CJ. Effects of passive smoking on lung growth in children. *Pediatr Pulmonol* 1992; 12:37-42.
- Ehrlich R, Kattan M, Godbold J, Saltzberg DS, Grimm KT, Landrigan PJ, et al. Childhood asthma and passive smoking. Urinary cotinine as a biomarker of exposure. *Am Rev Respir Dis* 1992; 145:594-9.
- Dekker C, Dales R, Bartlett S, Brunekreef B, Zwanenburg H. Childhood asthma and the indoor environment. *Chest* 1991; 100:922-6.
- Murray AB, Morrison BJ. Passive smoking by asthmatics: its greater effect on boys than on girls and on older than younger children. *Pediatrics* 1989; 84:451-9.
- Duff AL, Pomeranz ES, Gelber LE, Price GW, Faris H, Hayden FG, et al. Risk factors for acute wheezing in infants and children: viruses, passive smoke and IgE antibodies to inhalant allergens. *Pediatrics* 1993; 92:535-40.
- Mitchell EA, Ford RP, Steward AW, Taylor BJ, Becroft DM, Thompson JM, et al. Smoking and the sudden infant death syndrome. *Pediatrics* 1993; 91:893-6.
- Schoendorf KC, Kiely JL. Relationship of sudden infant death syndrome to maternal smoking during and after pregnancy. *Pediatrics* 1992; 90:905-8.
- Etzel RA, Pattishall EN, Haley NJ, Fletcher RH, Henderson FW. Passive smoking and middle ear effusion among children in day care. *Pediatrics* 1992; 90:228-32.
- Strachan DP, Jarvis MJ, Feyerabend C. Passive smoking, salivary cotinine concentrations and middle ear effusions in 7-year-old children. *BMJ* 1989; 298:549-52.
- Hinton AE. Surgery for otitis media with effusion in children and its relationship to parental smoking. *J Laryngol Otol* 1989; 103:559-61.
- Lyons RA. Passive smoking and hearing loss in infants. *Ir Med J* 1992; 85:111-2.
- Strachan DP, Jarvis MJ, Feyerabend C. The relationship of salivary cotinine to respiratory symptoms, spirometry, and exercise-induced bronchospasm in seven-year-old children. *Am Rev Respir Dis* 1990; 142:147-51.
- Barr MB, Weiss ST, Segal MR, Tager IB, Speizer FE. The relationship of nasal disorders to lower respiratory tract symptoms and illness in a random sample of children. *Pediatr Pulmonol* 1992; 14:91-4.
- Wright AL, Holberg C, Martinez FD, Taussig LM. Relationship of parental smoking to wheezing and nonwheezing lower respiratory tract illnesses in infancy. *J Pediatr* 1991; 118:207-14.
- Lashner BA, Shaheen NJ, Hanauer SB, Kirschner BS. Passive smoking is associated with an increased risk of developing inflammatory bowel disease in children. *Am J Gastroenterol* 1993; 88:356-9.
- Bauman KE, Flewelling RL, LaPrelle J. Parental cigarette smoking and cognitive performance of children. *Health Psychol* 1991; 10:282-8.
- Haneberg B, Tonjum T, Rodahl K, Gedde-Dahl TW. Factors preceding the onset of meningococcal disease, with special emphasis on passive smoking, symptoms of ill health. *NIPH Ann* 1993; 6:169-73.

21. Johnson JD, Houchens DP, Kluwe WM, Craig DK, Fisher GL. Effects of mainstream and environmental tobacco smoke on the immune system in animals and humans: a review. *Crit Rev Toxicol* 1990; 20:369-95.
22. Lannan S, McLean A, Drost E, Gillooly M, Donaldson K, Lamb D, et al. Changes in neutrophil morphology and morphometry following exposure to cigarette smoke. *Int J Exp Pathol* 1992; 73:183-91.
23. Tardif J, Borgeat P, Laviolette M. Inhibition of human alveolar macrophage production of leukotriene B₄ by acute in vitro and in vivo exposure to tobacco smoke. *Am J Respir Cell Mol Biol* 1991; 2:155-61.
24. Stuart JM, Cartwright KA, Robinson PM, Nothmann ND. Effect of smoking in meningococcal carriage. *Lancet* 1989; 2:723-5.