ABSTRACT. Results of epidemiologic studies provide strong evidence that exposure of children to environmental tobacco smoke is associated with increased rates of lower respiratory illness and increased rates of middle ear effusion, asthma, and sudden infant death syndrome. Exposure during childhood may also be associated with development of cancer during adulthood. This statement reviews the health effects of environmental tobacco smoke on children and offers pediatricians a strategy for promoting a smoke-free environment.

Effects of Tobacco Smoke on Children

In 1992, 48 million American adults (26.5%) currently smoked cigarettes. [1] A recent national survey indicated that 43% of children 2 months to 11 years of age live in homes with at least one smoker. [2] Because many young children spend a large proportion of their time indoors, [3] they may have significant exposure to environmental tobacco smoke.

Environmental tobacco smoke from cigarettes, cigars, and pipes is composed of more than 3800 different chemical compounds. [4] Concentrations of respirable suspended particulate matter (particulates of <2.5 µm) can be two to three times higher in homes with smokers than in homes with no smokers. [5] Cigarette smoking is the most important factor determining the level of suspended particulate matter and respirable sulfates and particles in indoor air. [6,7]

Passive smoking has a harmful effect on the respiratory health of children. [4,8,9] This statement reviews the evidence that children exposed to environmental tobacco smoke have higher rates of lower respiratory illness during their first year of life, higher rates of middle ear effusion, and higher rates of sudden infant death syndrome. In addition, children with asthma whose parents smoke have more severe symptoms and more frequent exacerbations.

Passive Smoking and Lower Respiratory Illness

The first effect of passive smoking to be documented in children was an increased rate of illnesses affecting the lower respiratory tract. Cameron [10] reported a positive correlation between the presence of a smoker in the home and the incidence of perceived disease in children.
Harlap and Davies [11] interviewed pregnant women to determine their smoking habits and then studied hospital admissions for infants younger than 1 year. The infants whose mothers smoked were 38% more likely to be admitted to the hospital for bronchitis and pneumonia than were those whose mothers did not smoke. This increased likelihood was mainly among infants 6 to 9 months of age; admissions increased with the number of cigarettes smoked by the infants’ mothers.

Rantakallio [12] showed that, among children younger than 1 year, those with mothers who smoked cigarettes were almost four times as likely to be hospitalized as were the infants of nonsmoking mothers, and the number of hospitalizations increased with the number of cigarettes the mother smoked per day. During the first 5 years of life, pneumonia and bronchitis were about twice as likely and acute nasopharyngitis and sinusitis in the upper respiratory tract were about 1.5 times as likely to develop in children whose mothers smoke.

Colley et al [13] found a consistent gradient in the incidence of pneumonia and bronchitis in the child's first year of life in relation to the parents’ smoking habits. Infants with two parents who smoked were more than twice as likely to have had pneumonia and bronchitis as were infants with parents who did not smoke.

Fergusson et al [14] showed that pneumonia and bronchitis in an infant's first year of life increased with increasing maternal smoking in an approximately linear manner: increases of five cigarettes a day resulted in an increase of 2.5 to 3.5 incidents of lower respiratory illness per 100 children at risk.

Passive Smoking and Serious Infectious Illnesses

Berg and colleagues [15] determined that among children 3 to 59 months of age, passive smoking was associated with an almost fourfold risk of a serious infectious illness requiring hospitalization.

Passive Smoking and Middle Ear Effusions

After a case-control study of risk factors for persistent middle ear effusions in Seattle, Kraemer and colleagues [16] reported that children who lived in households where more than three packs of cigarettes were smoked per day were more than four times as likely to be admitted to the hospital for tympanostomy tube placement than were children whose parents did not smoke.

Iversen and colleagues [17] studied children up to 7 years of age in Danish day care centers and demonstrated that middle ear effusion as measured by tympanometry was about 60% more likely to develop in children whose parents smoked. They estimated the overall fraction of middle ear effusion attributable to passive smoking to be 15%.

To determine risk factors for glue ear (serous otitis media), Black [18] performed a case-control study of 150 children 4 to 9 years old undergoing myringotomy in Oxford, England. Children undergoing myringotomy were about 50% more likely to have lived in households where someone smoked than were control children.

Hinton [19] studied 115 children undergoing ear tube insertion for otitis media with effusion and a control group of 36 children from an orthoptic clinic. Children admitted for ear operations were more likely to have at least one parental smoker at home than the children in the control group.

Etzel et al [20] studied 132 children in a day care center to determine whether passive smoking was associated with an increased risk of middle ear effusion during the 18-month period between 6 and 24 months of age. In this study, the children were classified as exposed or not exposed to cigarette smoke on the basis of serum cotinine concentrations at 1 year of age. Middle ear
effusion was diagnosed with the use of pneumatic otoscopy. The 45 children exposed to environmental tobacco smoke had an average of 7.1 episodes of middle ear effusion between 6 and 24 months of age, whereas the 87 children unexposed to environmental tobacco smoke had 5.8 episodes during that period. The average duration of middle ear effusion was 28 days among those in the exposed group and 19 days among those in the unexposed group. An estimated 8% of the middle ear effusions were attributed to exposure to environmental tobacco smoke.

Strachan et al [21] studied the relationship between passive smoking and middle ear effusion in 736 7-year-old school children in Edinburgh. In this study, investigators used objective measures of passive smoking and middle ear effusion, salivary cotinine concentrations, and impedance tympanometry. Children with type B tympanograms in one or both ears were categorized as having middle ear effusions. The results of this study indicated that detectable salivary cotinine was associated with type B tympanograms, even after adjustment for sex and the type of housing in which the children lived (rented versus owned). The authors estimated that at least one third of the cases of middle ear effusion among children in this age group may have been attributable to passive smoking.

Owen and colleagues [22] monitored 435 healthy children by tympanometry in the home every 2 to 4 weeks until 2 years of age. Of the children, 41% were exposed to household cigarette smoke. The authors found a significant association between the number of cigarettes smoked by household members and the frequency of otitis media with effusion during the second year of life.

Ey and colleagues [23] found that heavy maternal smoking (20 or more cigarettes per day) was a significant risk factor for recurrent otitis media during the first year of life. No association was found with paternal smoking.

**Passive Smoking and Asthma**

Children with asthma whose parents smoke may have more frequent exacerbations and more severe symptoms. [24-35] In one of the few interventions reported in the literature, Murray and Morrison [30] demonstrated that if parents expose their children with asthma to less cigarette smoke, the asthmatic symptoms the children have will be less severe.

**Passive Smoking and Sudden Infant Death Syndrome**

A growing body of evidence links exposure to environmental tobacco smoke to sudden infant death syndrome. [36-48] This relationship seems to be independent of birth weight and gestational age.

**Passive Smoking and Lipid Profiles**

Passive smoking has also been reported to alter lipid profiles in adolescents. Feldman et al [49] studied 391 nonsmoking adolescent students and found that those with elevated plasma cotinine concentrations had an 8.9% greater ratio of total cholesterol to high-density lipoprotein cholesterol and 6.8% lower high-density lipoprotein cholesterol than those with lower plasma cotinine concentrations. This may shed light on the mechanism of increased risk of coronary heart disease in passive smokers.

**Passive Smoking and Cancer**

Many studies link passive smoking to lung cancer in nonsmoking adults living with spouses who smoke. [50-57] The US Environmental Protection Agency [9] reviewed this subject and concluded that environmental tobacco smoke is a group A human carcinogen, the classification used when sufficient evidence from epidemiologic studies exists to support a causal association between exposure and cancer. A small number of studies have examined the relationship between exposure to environmental tobacco smoke during childhood and cancer risk. Sandler and
colleagues [58] found that the overall cancer risk was greater for individuals with exposures to environmental tobacco smoke during both childhood and adulthood than for individuals with exposure during only one period. When specific cancer sites or types were considered, Sandler et al [58] found that leukemia and lymphoma among adults were significantly related to exposure to maternal passive smoke before 10 years of age. [59]

Conclusion

Results of epidemiologic studies provide evidence that exposure of children to environmental tobacco smoke is associated with increased rates of lower respiratory illness and increased rates of middle ear effusion, asthma, and sudden infant death syndrome. Exposure during childhood to environmental tobacco smoke may also be associated with development of cancer during adulthood.