Environmental tobacco smoke as a risk factor for respiratory disease in children

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Abstract

Respiratory diseases are a frequent reason for using health care. In 1995–1996, diseases of the respiratory tract (ICD 460–519) contributed seven of the top 15 reasons for visits to physician offices among children under 15 years of age in the United States. Environmental tobacco smoke (ETS) is a wide-spread environmental pollutant that has been long linked with respiratory problems. This paper will review the available literature on the role ETS plays in respiratory diseases, including asthma. This review focuses not only on the respiratory problems caused by ETS, but also examines the influence of age at exposure on the consequences of ETS and the importance of the differing sources of ETS exposure. As ETS is a completely preventable form of environmental pollution, the success or failure of various types of interventions will also be reviewed. © 2001 Elsevier Science B.V. All rights reserved.

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1. Introduction

Respiratory diseases are a major health burden in children. Among children under 15 years of age respiratory diseases (ICD 460–519) account for seven of the top 15 diagnosis assigned to visits to physicians offices in the United States during 1995–1996 (Schappert and Nelson, 1999). In addition, respiratory diseases account for approximately one-third of the hospitalizations of children and adolescents less than 15 years of age in the United States, with asthma (ICD 493) being one of the leading causes of hospitalization for children (Owings and Lawrence, 1999). A wide variety of factors have been identified as risks for respiratory disease. Prominent among these factors is environmental tobacco smoke (ETS). The importance of ETS in childhood respiratory morbidity can be seen in a report from the 1987 National Medical Expenditure Survey (NMES), a survey of the US civilian population, which estimated that maternal smoking increased health...
care expenditures for respiratory conditions among children under 5 years of age by US$120 per year (in 1995 dollars) (Stoddard and Gray, 1997).

It is important to understand the mechanisms through which ETS influences respiratory diseases in childhood to more effectively evaluate and implement interventions to reduce this burden. This review will first discuss the sources of ETS exposure, the impact on symptoms, pulmonary functions, disease development, and the implications of time of exposure. Finally, interventions will be reviewed with regards to their effectiveness. This review will focus primarily on impact of ETS on children up to the age of 5 years. This period of life is felt to be critical for the development of the respiratory system and is the period of onset for most asthma and allergy.

2. Exposure

One of the two methods, self-report of exposure to cigarette smoking or measured cotinine, a metabolite of nicotine usually estimates exposure to ETS. Because of the different areas of focus—self-report usually looking at exposures in the household and measured cotinine levels, which reflect any exposure to ETS—the two methods can give very different results with respect to the proportion of the population exposed to ETS. Based on self-report, the National Health Interview Survey (NHIS), a survey of the US population, reported approximately 50% of US children aged 5 years and less are exposed to ETS in their homes (Overpeck and Moss, 1991). Using the presence of detectable cotinine as the criteria for exposure a different picture emerges. Detectable levels of serum cotinine were found in 88% of the US population who claimed to be nonsmokers in the third National Health and Nutrition Examination Survey (NHANES), 1988–1991 (Pirkle et al., 1996). Further work directly comparing the two methods has shown when using urinary cotinine, a cutoff of 30 ng/ml of cotinine/creatinine or more accurately reflects smoking exposure in the home (Henderson et al., 1989).

The literature does not give clear guidance as to which measure (self-report or cotinine) is the best for documenting ETS exposure. While cross-sectional studies looking at risk factors have found self-reported ETS exposure and measured cotinine levels are highly correlated (Chilmonczyk et al., 1990; Henderson et al., 1989), other types of studies have not always found the same equivalence. For example, during a year-long follow-up after an intervention to reduce ETS exposure in the home, the control group self-reported less ETS exposure while the measured cotinine value of their children increased (Hovell et al., 2000). In contrast, in a year-long study to document the impact of ETS exposure on lower respiratory infections self-reported ETS exposure was statistically significantly correlated with LRIs while measured cotinine was not (Margolis et al., 1997). Therefore, it appears clear that neither measurement will be the best in all circumstances.

The most important source of ETS exposure is within the home. Not surprisingly smoking by the mother is consistently associated with higher ETS exposure than paternal smoking (Chilmonczyk et al., 1990; Irvine et al., 1997). Presumably this reflects the increased amount of time the child spends with the mother. ETS exposure among children of smoking mothers can be influenced by a number of factors such as the mother’s avoidance of direct exposure of the child—the child of a smoking parent who reports only smoking outside of the house has a lower urinary/creatinine ratio as compared with a child whose parent is reported smoking in the house (21 vs. 51 ng/ml, $P < 0.05$) (Winkelstein et al., 1997). In contrast, breastfeeding by a smoking mother can lead to higher cotinine levels than non-breastfeeding by a smoking mother, although the importance of ETS exposure through breastfeeding on the development of respiratory disease is unclear (Chilmonczyk et al., 1990).

Other influences both inside and outside of the home can affect the level of ETS exposure. Within a home the number of smokers is associated with an increased level of exposure (Chilmonczyk et al., 1990; Irvine et al., 1997). ETS exposure tends to decrease as the child grows older (Irvine et al., 1997). This may reflect the increasing ability of the child to avoid being around the mother or...
others when they are smoking. Cotinine levels tend to be lower in the summer than in the winter (Chilmonczyk et al., 1990; Ronchetti et al., 1994). Finally, community levels of smoking are correlated to cotinine levels of children from non-smoking households within that community (Jarvis et al., 2000).

Although in-house exposure remains a primary source of exposure for children outside of the home exposures should not be forgotten. Recurrent ETS exposure outside the home may occur at the home of relatives, e.g. grandparents (Hopper and Craig, 2000) or by a childcare provider in a residential daycare (Holberg et al., 1993).

3. Morbidity

During the first year of life exposure to ETS has been consistently found to have an impact on the respiratory system regardless if the impact is defined by symptoms such as wheeze (Bisgaard et al., 1987) or cough (Charlton, 1984) specific diseases such as bronchitis/tracheitis (Pedreira et al., 1985) or bronchiolitis (McConnachie and Roghmann, 1986) or healthcare utilization such as medical visits (Fergusson et al., 1981) or hospital admissions for respiratory problems (Harlap and Davies, 1974) or pulmonary functions (Stick et al., 1996; Tager et al., 1983). Other studies, which have evaluated the impact of smoking across the first 5 years of life, have found the impact on respiratory symptoms to be the strongest in the first 2 years of life (Fergusson et al., 1981; Gergen et al., 1998; Stein et al., 1999). However, in children of all ages ETS exposure has been found to be associated with increased respiratory symptoms such as wheeze and cough (Forastiere et al., 1992; Henderson et al., 1995; Mannino et al., 2001; Somerville et al., 1988).

Many debates have centered on whether the damage caused by ETS exposure occurs through pre-natal ETS exposure, post-natal ETS exposure or whether each has an independent effect. It has been very difficult to separate these effects because of the high degree of correlation between pre- and post-natal smoking. Most women who smoke continue to smoke during their pregnancy, only about 40–50% of smoking women quit during pregnancy and of those who quit about 70% restart after delivery—most within first 3 months (Ebrahim et al., 2000; Fingerhut et al., 1990).

It is clear that pre-natal exposure affects the respiratory system. Studies looking at pulmonary function tests (PFTs) done within 3 days of birth (Stick et al., 1996) and within the first 6 months of life (Tager et al., 1993) have found reduced pulmonary functions in children exposed to ETS in utero. These abnormalities predispose the infant to an increased frequency of lower respiratory tract infections during the first year of life (Martinez et al., 1988). Follow-up of infants with reduced PFTs at birth to 6 years of age found that by the age of 6 years they still had reduced PFTs but were no longer experiencing wheezing (Martinez et al., 1995). Reduced PFTs are also found in older children exposed to ETS (Chen and Li, 1986; Mannino et al., 2001; Rona and Chinn, 1993). Potentially more important is the report from a 7-year follow-up of 5–9-year old children, which reported reduced growth of lung functions in children exposed to ETS (Tager et al., 1983). The long-term consequences of this loss of lung capacity remain to be determined.

The most direct evidence that post-natal ETS exposure has an independent influence on respiratory health comes from reports out of China where few women smoke. Paternal smoking was found to increase hospital admissions for respiratory illnesses in the first 18 months of life (Chen et al., 1986) and to be associated with decrease PFTs among older children of 8–16 years of age (Chen and Li, 1986). Thus it appears both pre- and post-natal ETS exposures play a role in respiratory health.

4. Atopy and asthma

The role ETS plays in the development of atopy is of great interest, as atopy is closely related to the development of childhood asthma. While exposure to ETS has been associated with an increase in sensitization to food allergens in the first few years of life, (Kulig et al., 1999), exposure to ETS does not appear to increase the sensitivity to
inhalant allergens which are more closely associated with asthma (Kulig et al., 1999; Strachan and Cook, 1998a). Likewise exposure to ETS is not associated with an increase in IgE in children (Oryszczyn et al., 1991; Strachan and Cook, 1998a,b). Moreover, atopy (defined by either allergen skin tests or self report) or more specifically the absence of the atopy appears to identify children most affected by ETS. When cohorts of children are divided on the basis of their atopic status ETS exposure has been reported to be associated with wheeze and asthma only in the non-atopics (Burr et al., 1993; Chen et al., 1996).

With regard to the role of ETS in the development of asthma there has been considerable controversy. One longitudinal study found late onset (after age 3 years) wheezing was associated with maternal smoking and allergy (Martinez et al., 1995). Another study reported that only children of women with less than a high school education developed asthma upon exposure to ETS (Martinez et al., 1992). Still another study reported that only children with atopic dermatitis developed asthma on exposure to maternal smoking (Murray and Morrison, 1990). Other longitudinal studies of infants over the first 6–10 years of life found ETS was not associated with development of asthma (Horwood et al., 1985; Lilljeqvist et al., 1997; Neuspiel et al., 1989; Sherman et al., 1990).

Childhood asthma is considered to be an allergic disease. Thus if ETS caused the development of asthma in a person who would not otherwise become asthmatic, ETS should be associated with a change in that person’s atopic status. However, from the various studies cited above one is led to the conclusion that ETS increases asthma but does not change the atopic status of an individual. This seemingly contradictory conclusion is not an artifact of comparing across studies but can also be seen within the same study. For example, among a cohort of children followed closely through age of 6 years, maternal smoking increased the risk of doctor diagnosed asthma (RR = 1.27; 95% confidence interval (CI), 1.04–1.55) while having no influence or being slightly protective against the development of allergen skin tests (OR = 0.83; 95% CI, 0.67–1.03) (Oddy et al., 1999).

Population data offer still another perspective on this problem. Over the last several decades asthma has been increasing in USA (Mannino et al., 1998). During this same time, the amount of cigarette smoking has decreased—in 1965, 33.7% of females were current cigarette smokers while by 1998 only 22.1% reported current smoking (MacKay et al., 2000). Thus the documented increase in asthma in USA has occurred during a period of decreasing cigarette smoking.

If ETS is not related to the development of asthma what role does it play? Numerous reports have found that ETS exposure is associated with an increased prevalence (Gortmaker et al., 1982) and severity of asthma (Chilmonczyk et al., 1993; Weitzman et al., 1990). Yet other work attempting to document the onset of asthma attacks with changes in ETS exposure found no association (Ehrlich et al., 1992; Ogborn et al., 1994). These apparent conflicting findings can be reconciled if one looks at ETS exposure as sensitizing pre-existing asthmatics to other triggers of asthma attacks such as viral infections (Johnston et al., 1995) rather than causing the development of asthma in individuals (Strachan and Cook, 1998a). This ‘sensitizing’ effect of ETS would cause more wheezing among asthmatics, both diagnosed and undiagnosed. This increased frequency of wheezing would heighten the chances that an undiagnosed asthmatic would receive the diagnosis of asthma. As much asthma is undiagnosed in the population (Speight et al., 1983), the potential for a significant rise in prevalence is very real.

5. Intervention

The data are quite clear that ETS exposure is detrimental to the respiratory health of children. ETS exposure is a completely avoidable risk factor for respiratory disease. The decrease in cigarette smoking in USA and other countries around the world over the last 20 years has helped reducing the exposure to ETS. Data from 1988 to 1998 from England have shown that as
the level of active smoking has decreased in England, salivary cotinine levels have decreased in children of non-smoking households (Jarvis et al., 2000). However, smoking during pregnancy, an important exposure for respiratory health, still remains a big problem. It is estimated that between 40 and 50% of women continue to smoke after they learn that they are pregnant (Ebrahim et al., 2000; Fingerhut et al., 1990). A recent report looking at smoking habits of US women between 1987 and 1996 found that the decline in smoking among pregnant women was not greater than but rather paralleled the decline seen among the general US female population. The parallel decline in smoking between pregnant and non-pregnant women appears to indicate that decrease among pregnant women is due to a decline in smoking initiation among women of childbearing age rather than the effect of specific interventions aimed at reducing smoking during pregnancy (Ebrahim et al., 2000). Thus the public health message to avoid smoking during pregnancy is still not being communicated to women in ways to cause them to change their behavior.

Attempts at reducing post-natal exposure have met with mixed results (Chilmonczyk et al., 1992; Eriksen et al., 1996; Greenberg et al., 1994; Hovell et al., 2000; Woodward et al., 1987). A variety of strategies have been tried. The level of intensity of the intervention appears to be an important determinant in whether or not the intervention is successful. Low level interventions such as a brief contact with a health visitor at a well child visit (Eriksen et al., 1996) or measuring the infant’s cotinine followed by a phone call and a letter from the physician did not reduce ETS exposure (Chilmonczyk et al., 1992). Whereas a more intense program, which included seven sessions over 3 months and set the goal of avoiding ETS exposure rather than quitting smoking was found to have reduced both reported ETS exposure and urine cotinine levels after 12 months (Hovell et al., 2000).

A major concern about the timing of an intervention remains even with the success of this program. As has been noted previously, the effects of ETS occur both pre- and post-natal. Thus while reducing post-natal exposure is helpful, it does not eliminate the measurable lung damage which has already occurred from pre-natal exposure.

In addition to the smokers themselves efforts must also be focused on healthcare providers to ensure they are actively counseling their patients about the dangers of ETS. A study of the patient counseling practices of pediatricians with regards to smoking found that pediatricians spent more time counseling on personal smoking in youths than counseling parents to stop smoking (Zapka et al., 1999). Another study reported that while approximately 54% of smoking caretakers wanted advice from their pediatrician about quitting smoking, only 12.5% reported that they received such advice (Hopper and Craig, 2000).

6. Conclusions

ETS exposure is an important and avoidable risk factor for respiratory diseases among children. Much work has gone into documenting when and how ETS causes its damage. The general decline in smoking in the United States and other countries has lead to a decrease in exposure to ETS but far too many infants still suffer from needless exposure. Smoking during pregnancy requires increased attention. Redoubled efforts must be directed towards pregnant women to induce them to stop smoking and remain smokeless after the birth of their child. Ideally the effort should emphasize stopping before the women becomes pregnant as there is no evidence to suggest there is a period during gestation when ETS is harmless. Unfortunately, practical, cost-effective programs to eliminate ETS exposure during pregnancy and/or childhood are lacking. Further research needs to develop and evaluate methodologies to more effectively modify behavior with regards to smoking. Efforts should not be solely focused on the individual but must include the health care provider. During each contact with the medical system, providers must inquire about ETS exposure and assist in reducing it. Much more work needs to be done before this preventable burden of disease is eliminated.


