Section report

Health Effects of “Passive Smoking” in Children

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ETS = environmental tobacco smoke; ms = mainstream smoke; ss = sidestream smoke; RSV = respiratory syncytial virus

Although the prevalence of cigarette smoking has been steadily decreasing in the United States over recent years, 29 percent of adults and 30 percent of adolescents still currently smoke. As a consequence, the opportunity for infants and children to be exposed to environmental tobacco smoke (ETS) remains substantial, and the observed health effects attributed to such exposure will continue to be an issue for the foreseeable future. This review will summarize the current state of knowledge about the health effects of exposure to ETS in infants and children. Comprehensive reviews of the entire subject of involuntary smoking can be found in the recent reports of the Surgeon General of the United States and the report of the National Research Council. The known effects of direct maternal smoking on the outcomes of pregnancy and subsequent development of the infant are beyond the scope of this review and are well summarized elsewhere.

Environmental tobacco smoke is a combination of mainstream smoke (ms), the smoke inhaled by the smoker, and sidestream smoke (ss), the smoke that comes from the burning end of the cigarette. Sidestream smoke represents the major component of ETS. Although there are qualitative and quantitative differences between ms and ss, their overall composition is similar. The central issue for health effects relates to quantitative differences in exposure between an active and an involuntary smoker, such as an infant or child. Factors such as the number of active smokers in a given environment and the size and ventilation characteristics of that environment further modify the exposure potential for the involuntary smoker.

Quantitative assessment of involuntary exposure of infants and children to ETS has been very imprecise and probably inaccurate. Most studies estimate exposure by questionnaire report of parental smoking without consideration of factors such as: (1) the accuracy of the parental smoking history; (2) the actual amount of time that a parent (or other individual) smokes in the presence of the infant/child; (3) the intensity (number of cigarettes and smokers) of smoking at any given time; and (4) the physical characteristics of the environment in which the smoking is occurring. Exposure outside the home environment is usually not considered.

The use of biologic markers, particularly of cotinine (a unique metabolic product of nicotine), has provided objective data that questionnaire reports of parental smoking and infant/child exposure to such smoking are accurate in terms of the dichotomy, “exposed”-“unexposed,” and, in some cases, in terms of magnitude of current exposure (eg, the number of cigarettes smoked by a mother). Unfortunately, this marker cannot provide objective verification of past exposure or cumulative exposure over the life of an infant/child.

Despite the limitations of the exposure data, the overall consistency in the findings of the studies of the health effects of exposure to ETS has made it possible to identify a series of potentially important health effects in infants and children that should be communicated by physicians to their patients.

Prenatal Exposure to Environmental Tobacco Smoke

Relatively few studies have been reported that relate the effects of involuntary exposure to ETS on the developing fetus. However, at least three studies have indicated that pregnant women who are exposed to ETS are more likely to give birth to infants of lower birth weight than women not so exposed. In one large study of women whose nonsmoking status was verified...
by serum cotinine measurements, there was a statistically significant inverse relationship between levels of serum cotinine measured in the second trimester and the birth weight of the infant. The estimated magnitude of the passive exposure effect has been in the range of about −25 g for a full-term pregnancy vs about −200 g attributed to the effect of direct maternal smoking during pregnancy. An effect as large as −100 g has been suggested by one of these studies.

**Acute Respiratory Illness Morbidity**

Community-based, longitudinal, epidemiologic studies that have used a variety of different end points generally have found an increased risk of acute respiratory illness morbidity. Among infants and children aged two years or less, increased occurrence of episodes of bronchitis/pneumonia (with or without hospitalization, excess risk, 1.4 to 2.6) and increased occurrence of tracheitis and laryngitis have been reported. This effect has been strongest for episodes in the first year of life and more consistently associated with maternal smoking. However, some studies have suggested that the increased risk for hospitalization may extend up to age five years and that paternal smoking may contribute an additional risk. The excess risk appears to be independent of socioeconomic factors, type of feeding (breast vs non-breast), birth weight, and sex of the infant. In the case of older children, the data are less easily interpreted. Only when morbidity is defined in terms of an acute "respiratory illness" (not further defined) in the year prior to the survey does there appear to be an increase in morbidity. In several of these studies, paternal smoking, in addition to maternal smoking, contributed to the excess risk.

Of particular interest for the clinician have been several studies that have related the occurrence of specific clinical syndromes and maternal smoking. The occurrence of maternal cigarette smoking has been found to be increased among children hospitalized for respiratory syncytial virus (RSV) infection in the first year of life, in the absence of any other underlying pulmonary disease. A similar observation has been made for infants with bronchopulmonary dysplasia who were hospitalized with RSV infection. Increased frequency of tonsillectomy, adenoidectomy and chronic middle ear effusions also have been attributed to parental smoking. For these latter morbidities, the combined smoking of both parents appeared to contribute to the excess risk.

Taken in aggregate, the data with regard to acute respiratory morbidity show the most consistent increase in risk for children under the age of two years. The specific elements of exposure and biologic susceptibility that define this risk remain unknown. Moreover, the long-term health consequences of this increased risk remain speculative.

**Chronic Respiratory Symptoms**

A large number of epidemiologic studies have reported an increased occurrence of chronic respiratory symptoms (exclusive of asthma) in children between the ages of 5 and 20 years. In the case of chronic cough, studies have been uniform in their observation of increasing prevalence with an increasing number of parents who smoke. In the case of chronic phlegm production and the occurrence of wheezing (in the absence of a specific diagnosis of asthma), most, but not all, studies demonstrate an increased prevalence with increasing parental smoking. For all of these chronic symptoms, the excess risk associated with two vs 0 smoking parents has been less than two.

In contrast to the studies of acute respiratory morbidity, studies of chronic respiratory symptoms involve age ranges where active smoking by the children themselves could confound the observed results. Although the results of some of the studies may have been influenced by misclassification of smoking adolescents as nonsmokers, the overall conclusions most likely are valid, since similar results have been observed in studies that have controlled for the active smoking status of the children.

**Asthma, Bronchial Responsiveness, and Atopy**

Several lines of evidence suggest that exposure of infants and children to ETS, especially that generated by mothers, plays a role in the occurrence and severity of asthma.

Some epidemiologic studies have demonstrated an increased occurrence of asthma and persistent wheezing in children of smoking mothers. In one such study, 18 to 34 percent of asthma was attributed to maternal smoking. These data have been supported by clinical studies on the role of RSV infection in the occurrence of asthma, which also have demonstrated an increased occurrence of maternal smoking in RSV-infected children, but the extent to which this exposure contributed to the subsequent increased airways reactivity and decreased peak expiratory flow rates observed in the RSV-infected children was not addressed directly.

Epidemiologic studies have demonstrated that bronchial responsiveness to carbachol or cold air is increased in children with smoking parents (mothers only in one study) and that this observation is due largely to a greater degree of responsiveness in asthmatic patients with smoking mothers vs those with nonsmoking mothers. In one of these studies, virtually no maternal smoking occurred during pregnancy, which suggests that postpartum exposure to ETS has a direct role in the observed effects of ETS on bronchial responsiveness. Responsiveness to isoproterenol also has been associated with parental smoking.

A clinical study has observed that bronchial respon-
siveness is greater and levels of FEV₁ and FEF25-75 are lower in asthmatic subjects with smoking mothers vs those without. In that same study, these differences were shown to be most striking during the times of the year when homes were least likely to be well ventilated and when exposure to ETS might be expected to be greatest. A large study of inner-city asthmatic children showed that asthmatic subjects with smoking parents appear to have more emergency room visits for asthma, even after adjustment for asthma severity and asthma self-management practices.

Some insight into the possible mechanisms by which ETS might influence the occurrence and/or severity of asthma comes from recent data on the possible effects of cigarette smoke on the immune system. Cigarette smoking has been shown to increase levels of IgE and to enhance the occurrence of a specific IgE response to aeroallergens. Increased frequency of response to skin tests has been observed in the children of smoking parents/mothers, particularly in male children in one study. Postnatal exposure to ETS has been associated with raised levels of total IgE, and cord blood IgE levels have been found to be increased in infants whose mothers smoked during pregnancy. Infants of these latter mothers were found to have a four-fold excess risk of developing a clinically manifest allergic disease state. Even the study that failed to document an increased allergic sensitization in the children of smoking mothers did find that maternal smoking was significantly more common in children with clinically recognizable allergic disease. Thus, exposure to ETS may function as an environmental agent that enhances biologic mechanisms that relate to bronchial responsiveness and asthma. The association of ETS with acute respiratory illness morbidity, as discussed previously, and the observed association of increased early life respiratory illness morbidity in skin test positive children that has been observed in at least one study also suggests that ETS may play an important role in the hypothesized relationship between infant lower respiratory illness (especially RVS-associated) and the subsequent occurrence of asthma.

**PULMONARY FUNCTION**

A large number of studies have investigated the relationship between involuntary exposure to ETS and pulmonary function in children. While not all of these studies have been uniform in their demonstration of an effect, the vast majority of both longitudinal and cross-sectional epidemiologic studies have observed an effect of such exposure. The most consistent effects have been observed for maternal smoking, but a number of studies have observed a supplemental effect of paternal smoking or an effect confined to paternal smoking.

Deficits in level have been observed for FEV₁, FEF25-75, Vmax25, Vmax50, and Vmax75 as have deficits in the rate of growth and change in level of FEV₁ and FEF25-75 in adolescents and children. Estimates of the cumulative effect on FEV₁ of childhood exposure to maternally generated ETS have been in the range of about 3 to 5 percent by age 20 years.

The sex of the child appears to relate to the observed effect of maternal ETS. Overall, most studies have found that female children are more likely to be affected than male children. The middle portion of the maximum expiratory flow-volume or time-volume curves are most consistently affected in females, although deficits in the proximal portions of these curves have been reported. When an effect has been observed for males, the portion of the curves affected has been less consistent.

The explanation for the predominance of the maternal smoking effect remains speculative. The explanation most often given relates to the time mothers spend with their children relative to that of fathers. Another explanation for which there is some supporting data is the possibility that maternal smoking during pregnancy produces effects on the lung that parallel the general growth-retarding effects of smoking on the fetus. Several studies have produced results that are compatible with this possibility. A similar interpretation has been given to the effect of maternal smoking on reduced height in their exposed children. Data from experimental animal systems also are compatible with this explanation.

The major unanswered question relates to the long-term consequences of the observed small deficits in pulmonary function that have been attributed to ETS exposure. Most studies of the effect of involuntary exposure to ETS on the pulmonary function of adults offer little insight, since they generally have not evaluated childhood exposures. One investigation that sought specifically to evaluate a semiquantitative measure of lifetime exposure to ETS on the pulmonary function of subjects aged 15 to 35 years did find that exposure to maternal smoking and exposure to ETS during childhood had a significant effect on FEF25-75 in males. Effects in females in this study only related to exposure in the work environment. Thus, the matter remains unresolved.

**Conclusions**

The aggregate of the data across all of the reported health effects leads to the inescapable conclusion that ETS poses a health risk to infants and children. Unfortunately for the physician who has to advise his/her patients, current estimates of the magnitude of risk of the various health effects noted above are imprecise at best and unknown in some cases. None-
theless, certain recommendations to the parents of infants and children seem justified:

1. Use of cigarettes in the home, especially by the mother of the child, will increase the possibility of that child's experiencing a respiratory illness episode that requires the attention of a physician or hospitalization. This risk is greatest over the first two years of life, especially over the first 12 months. Parents should be counseled not to smoke or to permit anyone else to smoke when the infant is present and should make very effort to quit smoking and to prohibit visitors from smoking in the home.

2. Across the entire range of infancy, childhood, and adolescence, the symptoms and physiologic consequences of asthma may be worsened by exposure to ETS, especially that generated by the mother. Every effort should be made to eliminate ETS from the home of an asthmatic child. This issue is of particular importance in those environments with a cold weather season that leads to decreased ventilation of the home environment.

3. Use of cigarettes in the home may lead to an increased chance of the child's having chronic respiratory symptoms such as cough, phlegm, and wheeze in the absence of a diagnosis of asthma and chronic middle-ear effusions in a child so predisposed. The long-term health consequences of the occurrence of such symptoms are still unknown. Efforts to create a smoke-free home environment in relation to the risk of chronic symptoms should be stressed in families with a familial history of chronic respiratory diseases, especially obstructive airways diseases.

4. Parental smoking, especially maternal smoking, may lead to small reductions in the growth of lung function in children. Again, while the long-term health consequences of these reductions are unknown, families with a history of chronic obstructive airways diseases should be urged to create a smoke-free home environment for their children. This recommendation needs to be tempered by the possibility that the effects observed in childhood may be "carryover" effects related to smoking of the mother during pregnancy.

The report of the National Research Council5 has addressed several areas in which data are needed for a better understanding of the health effects of exposure to ETS in childhood. These recommendations can serve as a useful guide for physicians in terms of appropriately qualifying the above advise to parents:

1. Research is needed to evaluate the role of ETS in the development of airway hyperresponsiveness.

2. More information is required from long-term longitudinal studies of the effect of exposure to ETS on lung function.

3. The pathophysiologic mechanism of increased susceptibility to viral infections in very young children exposed to ETS needs to be clarified.

4. Studies of patients with obstructive lung diseases (eg, cystic fibrosis) need to be carried out to identify effects of ETS.

REFERENCES

1 US Surgeon General. The health consequences of involuntary smoking. Rockville, MD: US Dept of Health and Human Services (CDC), DHHS (CDC) publication no. 87-8398, 1987


