

THORAX

Editorials

Passive smoking and the health of children

There is increasing interest in the effects of adult smoking on the health of children.¹⁻³ Tobacco smoke is the commonest indoor environmental pollutant to which children are exposed. In Britain over 3.5 million young children, or half of those aged under 10 years, live with at least one adult who smokes.⁴ In 20% of homes both parents smoke, in 11% only the mother smokes, and in a further 19% only the father smokes.⁵ As they spend much of their early years with their parents, these children have prolonged exposure to environmental tobacco smoke. The respiratory tract of children is vulnerable as it is structurally and immunologically immature. Because the lungs develop rapidly in early life, any insult during this sensitive period may affect growth and future lung function. So what do we know of the hazards of passive smoking in childhood?

Assessing the evidence

There are no standardised methods for testing the association between passive smoking and child health.^{6,7} Prospective, case-control, and cross sectional study designs have been used, and different – often poorly defined – outcome measures have been employed. Many studies have been flawed by bias. The degree to which confounding factors such as socioeconomic status, family size, atopy, and infection have been allowed for has varied. As 90% of mothers who smoke in pregnancy are still smoking five years later,^{8,9} it is often impossible to separate the effects of intrauterine exposure from postnatal exposure.

It is also difficult to quantify exposure to environmental tobacco smoke. Early studies simply reported whether one or both parents smoked, and the average number of cigarettes they consumed each day. Recent studies have quantified exposure by measuring levels of the marker cotinine.^{5,10,11} Cotinine is the main metabolite of nicotine and a sensitive (97%) indicator of exposure to tobacco smoke. It can be measured in serum, saliva, or urine by chromatography or radioimmunoassay. Levels in children correlate closely with the number of smokers in the home and their daily cigarette consumption.^{5,10} Maternal smoking results in higher cotinine levels than paternal smoking,⁵ and social class, season, and the day of the week all influence levels.⁵ Repeated cotinine assays over a prolonged period show an acceptable degree of stability.¹¹ Using cotinine levels, Jarvis has estimated that the dose of nicotine inhaled by children whose parents smoke is equivalent to the children themselves actively smoking 60–150 cigarettes a year.¹¹

Cotinine can be detected in many children from non-smoking homes,^{5,12} indicating that children inhale environmental tobacco smoke from sources other than their parents, and that questions about parental smoking may underestimate exposure. Cotinine measurements provide

an objective assessment of recent exposure to environmental tobacco smoke, but do not tell us about the duration of exposure or about the intake of other components of environmental tobacco smoke which may be more important than nicotine.

Effects of passive smoking on the health of children

Although passive smoking is important in many childhood disorders,^{1-3,13} most attention has been paid to the effects on the respiratory tract and on infant mortality. Not only are these effects the most clinically important, but it is in these areas that the evidence for a causal effect is most convincing.

PASSIVE SMOKING AND DEATH IN INFANCY

For 30 years we have known that maternal smoking in pregnancy increases fetal morbidity and mortality.¹⁴ Maternal smoking increases the risk of miscarriage, stillbirth, and neonatal death by 25–35%:^{1,2,14,15} this represents an annual loss of almost 5000 fetuses and newborn infants in England and Wales.¹ Smoking increases the incidence of prematurity and of low birth weight as a result of impaired intrauterine growth – two important causes of morbidity.^{1,2,14,16}

Many studies have examined the relation between maternal smoking and death in infancy.^{1-3,17} There has been particular interest in the association with the sudden infant death syndrome, the commonest cause of death in children aged one week to one year in developed countries. (The sudden infant death syndrome (SIDS) is defined as the sudden and unexpected death of an apparently healthy infant in whom a thorough postmortem examination reveals no explanation for the death.)

In an American study of 2720 infant deaths¹⁸ the infant mortality rate was 12.1 deaths per 1000 births among infants of mothers who had smoked during pregnancy compared with 7.6 among infants of non-smokers (mean odds ratio 1.6). After allowing for maternal age, parity, education, and marital status, and for birth weight, the odds ratios for smoking were particularly high for deaths from respiratory disease (3.4) and SIDS (1.9). The authors estimated that, if no mother had smoked, infant mortality would have been reduced by 10% and deaths from SIDS and respiratory illness by 28% and 46% respectively. In a national prospective study in Sweden maternal smoking was examined as a risk factor for the 190 cases of SIDS which occurred in the 260 000 births.¹⁹ Maternal smoking was the most important preventable risk factor and showed a clear dose effect: the infants of women who smoked 1–9 cigarettes a day had a relative risk of developing SIDS of 1.8, and in those with mothers who smoked 10 or more a day the risk was 2.7 times higher than infants of non-

smoking mothers. The authors calculated that 27% of cases of SIDS were the result of maternal smoking, an attributable risk remarkably similar to that from the American study.¹⁸ A case-control study of 128 infant deaths in New Zealand found that smoking in pregnancy increased the risk of SIDS by 2.7 after correction for confounding factors.²⁰ Again there was a strong dose-response effect, with a fivefold increase in risk to infants whose mothers smoked more than 20 cigarettes a day. The attributable risk associated with smoking was 40%.

In these studies it was impossible to separate the effects of intrauterine and postnatal tobacco exposure. However, Schoendorf and Kiely showed that the risk of SIDS was higher in those infants whose mothers smoked both prenatally and postnatally than in infants whose mothers smoked only postnatally.²¹ From this study, and others that have shown that paternal smoking has an independent effect on the risk of SIDS, it appears that intrauterine exposure is the more important risk, but that this is increased further by postnatal passive smoking.¹⁷ Advice to parents to avoid smoking both before and after the birth has been an important part of successful national campaigns to reduce the toll of SIDS.¹⁷

RESPIRATORY ILLNESS IN INFANCY

Twenty years ago Harlap and Davies showed a dose-response relation between maternal smoking and hospital admissions for bronchitis or pneumonia in infancy.²² Infants of mothers who smoked had an admission rate 28% higher than that of infants of non-smokers. Colley *et al* showed that the incidence of pneumonia and bronchitis in the first year of life was significantly associated with the parents' smoking habits: if neither parent smoked the annual incidence was 7.8%, if one smoked it was 11.4%, and if both smoked it was 17.6%.²³ As in the study by Harlap and Davies the effects of smoking were independent of birth weight, socioeconomic class, and family size. In a cohort of infants followed from birth Fergusson *et al* showed a dose-related effect of maternal smoking on the incidence of pneumonia, bronchitis, and bronchiolitis.²⁴ Infants of smoking mothers were twice as likely to see a doctor for respiratory illness as those of non-smokers. Infants admitted to hospital with respiratory syncytial viral bronchiolitis are more likely to have mothers who smoke than control subjects.^{25,26}

In a prospective study of 850 infants the numbers of episodes of wheezing and non-wheezing lower respiratory illness were greater if the mother smoked.²⁷ The overall odds ratio was 1.5 if the mother smoked, and 1.8 if she smoked more than 20 cigarettes a day. Infants of smoking mothers developed respiratory illness earlier than those of non-smokers. Paternal smoking had no detectable effect.

Two analyses of data from the British National Child Health and Education Study have confirmed that smoking increases infant chest diseases but conclude that smoking during pregnancy is more important than smoking postnatally.²⁸ In Shanghai, however, where women do not smoke during pregnancy, admission rates for respiratory illness in infancy correlate with the cigarette consumption of other family members, indicating that postnatal passive smoking has an influence that is not explained by maternal smoking during pregnancy.²⁸

The increase in respiratory disease in infancy caused by passive smoking has implications for adult life. Barker, in studies of the relation between infant and adult disease, has found strong evidence of a direct causal link between acute lower respiratory infection in early childhood and chronic bronchitis in adults.²⁹ He concludes that lung infection during the sensitive period of rapid growth in infancy has

deleterious effects on lung function which persist into adult life, and that reducing respiratory infection in infancy may reduce the incidence of chronic bronchitis in the next generation.

RESPIRATORY ILLNESS IN THE OLDER CHILD

The relation between parental smoking and respiratory symptoms in children over the age of two is less clear, and some studies that have shown a harmful effect in infancy have found no effect in older children.²³ In a prospective study of 4800 children aged 5–11 years there was a significant association between the frequency of wheeze, cough, and episodes of bronchitis, and the number of cigarettes smoked by the parents.³⁰ The relative risk of having frequent wheeze rose from 1.0 if neither parent smoked to 1.3 if they smoked 10 cigarettes a day, and to 1.6 if they smoked 20 per day. Data from the Harvard longitudinal study revealed a highly significant dose-response relation between cigarette consumption and the frequency of eight defined respiratory illnesses or symptoms in children aged 6–9 years.³¹ Current maternal smoking increased the frequency of these illnesses by 20–35%. Paternal smoking had a smaller effect.

PASSIVE SMOKING, WHEEZE, AND ASTHMA

Parental smoking is important in childhood asthma. In children aged 0–5 years maternal smoking was associated with a higher prevalence of asthma (odds ratio 2.1), increased use of asthma medication (odds ratio 4.6), and an earlier onset of asthma (odds ratio 2.6) than was observed in the children of non-smoking mothers.³² In a prospective birth cohort of 9670 British children, 18% of children had recurrent wheeze by the age of 10.³³ The incidence of wheeze increased by 14% when mothers smoked over four cigarettes a day, and by 49% when mothers smoked 15 or more a day. Murray and Morrison^{34–36} have shown that maternal, but not paternal, smoking increases the frequency and severity of asthma symptoms. When compared with asthmatic children whose mothers did not smoke, the children of smoking mothers had spirometric indices that were 13–23% lower, and a fourfold greater degree of responsiveness to histamine challenge indicating airway narrowing and hyperreactivity. Boys and children with atopic dermatitis were particularly susceptible to the effects of maternal smoking, which increased with the duration of exposure to environmental tobacco smoke. Chilmonczyk *et al* found that higher urinary levels of cotinine in children with asthma were associated with a higher incidence of acute exacerbations and poorer expiratory flow rates.³⁷

Surprisingly, parental smoking does not increase the risk of hospital admission for acute asthma.^{26,32,38} Reese *et al* found no association between urinary cotinine levels and asthma admissions, but they reported a positive correlation with admissions for bronchiolitis.²⁶ Ehrlich *et al* showed a correlation between recent passive smoking (as assessed by questionnaire and urinary levels of cotinine) and a diagnosis of asthma, but no association with acute exacerbations.³⁸ They suggested that passive smoking increases hyperreactivity rather than producing bronchospasm.

As well as worsening symptoms and lung function in children with established asthma, both epidemiological and physiological studies have shown that tobacco exposure in early life is an important factor in the development of wheeze and asthma in children.³⁹ In a prospective trial of allergen avoidance in 120 neonates at high risk of atopic disease, parental smoking was an important risk factor for the development of recurrent wheeze in infancy.⁴⁰ In another prospective study of allergic disease in children

Cogswell and colleagues found that 62% of the children of smokers developed wheeze by the age of five, compared with 37% of children of non-smokers.⁴¹ However, Burr *et al* found no association between parental smoking and allergic disease at the age of seven, even though there was a strong association between wheeze and passive smoking in the first year of life in this longitudinal cohort.⁴²

PASSIVE SMOKING AND INFANT LUNG FUNCTION

Studies of infant lung function support the theory that early exposure to environmental tobacco smoke has a prolonged effect on respiratory function and growth.³⁹ Martinez *et al* have shown that diminished respiratory conductance and lower maximal expiratory flows in early infancy, measured before any respiratory infection, predispose to the development of recurrent wheeze during the first three years of life.^{43,44} There were insufficient mothers who smoked to assess the role of smoking in these studies. Hanrahan and Tager have shown that maternal smoking in pregnancy, as assessed by questionnaire and prenatal cotinine measurements, results in significantly reduced forced expiratory flow rates and an increased incidence of wheezing illness in infancy.^{45,46} They suggest that smoking in pregnancy may impair fetal airway development and alter the elastic qualities of the lung. (This hypothesis is supported by a small histological study of the effects of smoking on fetal lung growth in experimental rats.⁴⁷) In another physiological study parental smoking contributed to the development of bronchial hyperresponsiveness in the first months of life.⁴⁸

PASSIVE SMOKING AND LUNG FUNCTION IN OLDER CHILDREN

There are conflicting results from studies of lung function in older children.^{7,13} Most, but not all, studies have shown a small but significant dose related reduction in airway function. Maternal smoking is more important than paternal smoking. Younger children, boys, and atopic children are particularly affected.

Evidence that passive smoking has a significant effect on lung function has come from the Harvard longitudinal study of childhood risk factors for the development of adult chronic obstructive airway disease. In a seven-year prospective study of 1156 children from this cohort there was a significant association between maternal smoking and a lower forced expiratory volume in one second (FEV₁) and forced mid expiratory flow rate.⁴⁹ Maternal smoking lowered the expected growth in FEV₁ by 7–10% over this period, even after correction for confounding influences. Cross sectional surveys of British children have shown that small airway function is inversely related to cotinine levels.^{12,50} The differences in spirometric indices between those children with the lowest cotinine levels and those with the highest were small (6–7%).

In one of these studies passive smoking did not increase bronchial hyperreactivity to a free running exercise test.¹² However, parental smoking did increase bronchial responsiveness to inhaled carbachol in nine year old Italian boys, but not in girls.⁵¹ Frischer *et al* also studied the relation between maternal smoking and the response to exercise in 1800 children.⁹ Current exposure to environmental tobacco smoke was not associated with abnormal bronchial responsiveness, but the odds of being hyperresponsive to exercise were higher in children exposed to maternal smoking in their first year of life, particularly if they had asthma or positive skin prick tests.

Conclusions

There is now convincing evidence that, if parents smoke, they increase the risk of illness and, in some cases, of death

in their children. Although the most important effects of adult smoking are on fetal and infant mortality and on respiratory illness, passive smoking has many other effects on children.^{1-3,13} For example, it increases the incidence of persistent middle ear effusion ("glue ear") – the commonest cause of deafness and the commonest reason for surgery in children – by one third.¹ Passive smoking is responsible for over 17 000 admissions to hospital of preschool children each year.¹ The annual cost of the additional health care for the 3.5 million children who live with smokers has been estimated to be £143 million for England and Wales.⁵² One sixth of cases of lung cancer in non-smoking adults can be attributed to exposure to cigarette smoke in childhood and adolescence.⁵³ If parents smoke their children are more likely to become smokers themselves during adolescence.¹

In the past it was suggested that maternal smoking was more important than paternal smoking because children spent more time with their mothers. There is now a growing body of evidence, however, that it is smoking during pregnancy and its effects on fetal development that is the major reason for this finding. Combined with all the epidemiological data on respiratory illness in the children of smokers, the studies of infant lung function support the hypothesis that early exposure to tobacco smoke, either in utero or in infancy, decreases lung function and increases bronchial responsiveness, particularly if there is a family history of atopic disease.

Recognising the effects of adult smoking on the health of children is important, but we have yet to reduce this hazard to the health of children. Despite health education campaigns, the prevalence of smoking in teenage girls has not fallen in the last decade.¹ Other strategies have been suggested by the Royal College of Physicians.¹ It is up to all of us – whether we care for children or their parents – to consider how we may best reduce this avoidable burden on the health of children.

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