

Review

Effects of passive smoking on children's health: a review

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SUMMARY Since the mid-1980s there has been increasing interest in the effects of passive smoking on the health of children. It has been estimated that the total nicotine dose received by children whose parents smoke is equivalent to their actively smoking between 60 and 150 cigarettes per year. This review article considers the evidence for a relationship between passive smoking and disorders such as: prenatal damage to the fetus; poor growth indicators; respiratory illness; atopy and asthma; coronary heart disease; and sudden infant death syndrome. We conclude that paediatricians should not be complacent about the hazards of passive smoking for children and that public health education efforts should be continued.

Introduction

Since the mid-1980s there has been increasing interest in the effects of passive smoking on the health of children [1,2]. As children spend much of their early life in the presence of their parents, children who have parents that smoke will have a prolonged and close exposure to environmental tobacco smoke.

More than 4000 substances have been identified in cigarette smoke. Of these, nicotine is a highly toxic alkaloid that is both a ganglionic stimulant and depressant. Carbon monoxide is known to interfere with oxygen transport and utilization [3].

The local context

The estimated overall rate of smoking among the Jordanian population is 31.7% [4]. Of concern is the estimated rate of smoking among Jordanian doctors (63%), a figure considerably higher than that of the general population. Moreover, in one study 27% of doctors in Jordan did not explain smoking hazards to patients and 36% even smoked in front of patients [5]. In Al-

Khobar, Saudi Arabia, the overall smoking rate among the parents of schoolboys [6] is 18% (32% of fathers and 4% of mothers). This is in spite of the fact that Saudi Arabia has no contribution to the world's production of tobacco [7].

Measuring exposure to smoke

Considerable work has been undertaken to identify ways of measuring the extent of tobacco smoke exposure in non-smokers. Of these nicotine and cotinine levels in the body have received the most attention. Cotinine has become increasingly accepted as a short-term marker because of its relatively long half-life (approximately 20 hours, compared with approximately 2 hours for nicotine). It also is less susceptible to fluctuations during exposure to tobacco smoke and can be conveniently measured in blood, urine and saliva [8,9]. Cotinine measurements can provide an assessment of recent exposure to environmental tobacco smoke, but they do not indicate the duration of exposure nor do

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they indicate the intake of other components of tobacco smoke that may be more important.

It has been estimated from cotinine measurements that the total nicotine dose received by children whose parents are smokers is equivalent to their actively smoking between 60 and 150 cigarettes per year [1]. Salivary cotinine concentrations measured in schoolchildren have been found to correlate strongly with the smoking habits of their parents, particularly their mothers. The measured cotinine levels correlate closely with atmospheric nicotine levels measured within the home and with the results of questionnaires about household smoking [1]. However, Strachan et al. [10] also found significant levels of cotinine in children from non-smoking households. This indicates that children are exposed to tobacco smoke from sources other than their parents and that simply inquiring about parental smoking will underestimate a child's exposure. So-called 'unexposed' persons have been found to have on average 8.5 ng of cotinine per mL of urine [11]. Since the only source of cotinine or nicotine in body fluids is tobacco products, primarily through exposure to smoke, it follows that 'unexposed' persons are also exposed to environmental tobacco smoke.

Active smoking during pregnancy

Smoking during pregnancy is associated with a 10% increase in prenatal mortality and an increase in the incidence of premature labour and placental abruption [12]. Smoking may also cause congenital damage to the developing respiratory system of the fetus, either to the bronchial tree or to the developing lung vasculature, as has been shown in the umbilical vessels [13].

Another possible effect on the respiratory system caused by smoking in pregnancy is interference with the immune system that predisposes the infant to respiratory infections. Cigarette smoking is immunosuppressive both in vivo and in vitro [14]. Abnormalities of leucocyte function have been measured in the children of parents who smoke and in immune function in cord blood from mothers who smoke [15]. More recently, smoking during pregnancy has been associated with mild neurodevelopmental handicaps [16].

Poor growth

Infants born to mothers who have smoked during pregnancy are lighter and shorter than infants of non-smoking mothers [8,9]. Berkey et al. assessed the association between passive smoking and attained height and growth rate [17]. Children of mothers who smoked were found to be shorter than those of non-smoking mothers. There was also a negative correlation between the attained height and the number of cigarettes the mother smoked daily.

Rona et al. studied children enrolled at birth into the National Study of Health and Growth [18]. Children whose parents smoked more than 10 cigarettes a day were on average 0.6 cm shorter than those of non-smoking parents. Whether the effect of passive smoking on height is indirect, e.g. by increasing respiratory infections in early childhood or direct in its toxic effect, was not investigated.

Respiratory illness

Children are particularly vulnerable to the detrimental effects of passive smoking, as their respiratory systems are structurally and immunologically immature and are

developing rapidly. The mechanism by which passive smoking increases respiratory symptoms and decreases lung function in children is not known. Parental smoking has been shown to enhance allergic sensitization in infants and school-children with a close family history of atopic disease [19,20]. In adult smokers, tobacco smoke damages the number and the function of alveolar macrophages and polymorphs and causes goblet cell hyperplasia and increased mucus secretion. Changes in antibody and cell-mediated immune responses have also been documented.

In a prospective study of 850 infants, the number of episodes of wheezing and non-wheezing lower respiratory illness were significantly increased if the mother smoked [21]. The overall odds ratio was 1.5 if the mother smoked at all 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. Infants who did not attend day-care nursery were at increased risk from maternal smoking, perhaps because they have more prolonged exposure to maternal smoke. Taylor and Wadsworth [22] and Evans and Golding [23] in retrospective analyses of data from the British National Child Health and Education Study confirmed these findings.

The increase in lower respiratory disease in infancy caused by passive smoking may have important implications for the child in later life. Barker and Osmond [24], in an important series of studies of the relationship between infant and adult disease, have found strong evidence of a direct causal link between acute lower respiratory infection in early childhood and chronic bronchitis in adults. Children exposed to environmental tobacco smoke have more lower respiratory illness, more

middle ear effusion and more viral respiratory illness than unexposed children [25].

The Harlap and Davies study of 10 672 infants showed a definite dose-response relationship between maternal smoking and hospital admissions for bronchitis and pneumonia [26]. Infants of mothers who smoked had an admission rate that was 28% higher than that of infants of non-smokers. Colley et al. [27] in a study of 2200 children found that the incidence of pneumonia and bronchitis was significantly associated with the parents' smoking habit: if both parents were non-smokers, the annual incidence was 7.8%; if one parent smoked, it was 11.4%; and if both parents smoked, it was 17.6%. As in the Harlap and Davies study the effect of smoking was independent of birth weight, socioeconomic class and family size.

An innovative French study [28] used the prevalence of tonsillectomy and adenoidectomy as indices of repeated respiratory infection in early childhood; 42% of children with one smoking parent and 51% of those with two smoking parents had tonsillectomy, adenoidectomy or both as compared with 28% of the children of non-smokers.

Persistent middle ear effusion (glue ear) is the most common cause of deafness in children and an important cause of delayed language development. Persistent middle ear effusion is the most common indication for surgery among children in the UK. In a population based study of 736 school-children aged 7 years, one third of cases were statistically attributable to exposure to tobacco smoke [29].

Many factors influence the development of persistent middle ear effusion, but Iversen et al. [30] found that parental smoking was the only home environmental factor that influenced the prevalence of

middle ear effusion. By calculating the attributable risk, they estimated that parental smoking accounted for 15% of effusions in children aged 1 to 7 years and 36% of cases in children aged 6 to 7 years.

The mechanism by which passive smoking causes middle ear disease is unclear. Tobacco smoke may have a direct effect on the mucosa of the middle ear causing oedema, abnormal mucociliary clearance, blockage of the Eustachian tube and impaired ventilation of the middle ear. There may be an indirect effect by adenoidal enlargement or by increasing susceptibility to infection.

Atopy and asthma

It has long been known that atopy runs in families and that this is likely to be due to a genetic susceptibility. More recent studies have implied that atopy has a dominant inheritance pattern and that the gene locus is in chromosome 11 [31]. Airway growth is adversely affected and wheezing illnesses are more prevalent in households where an adult smokes. Salivary cotinine levels, which are a reasonable marker for passive smoking, correlate inversely with small airway function [32].

Passive smoking increases the frequency and the severity of symptoms in children with asthma. Weitzman et al. [33] in an analysis of data from 4000 children aged 0–5 years found that maternal smoking of more than 10 cigarettes a day was associated with higher rates of asthma (odds ratio 2.1), an increased likelihood of using asthma medication (odds ratio 4.6) and an earlier onset of asthma (odds ratio 2.6).

A study of asthmatic children showed that asthma-related visits to the emergency department were significantly (63%) higher in smoking households than households without smokers [34]. Controls for

other household irritants and the subjects' own smoking did not affect the result.

When compared with asthmatic children whose mothers did not smoke, the children of smoking mothers had lung function indices that were 13%–23% lower and had a fourfold greater degree of responsiveness to an inhaled histamine challenge, indicating airway narrowing and bronchial hyperactivity [35]. In these studies, children with atopic dermatitis were particularly susceptible to the adverse effects of maternal smoking, which increased with the duration of exposure to environmental tobacco smoke and the age of the child [36,37].

Coronary heart disease

Cardiovascular disease, the leading cause of mortality in the USA and most other developed countries, is strongly associated with several risk factors including smoking [38].

A study by Neufeld et al. [39] showed that children of smokers had an 11.2% (4.9 mg/dL) reduction in high-density lipoprotein (HDL) cholesterol levels relative to children of non-smokers. Adjustment for confounding variables reduced this difference to 8.5% (3.7 mg/dL). This reduction was larger than that reported in other studies of environmental tobacco smoke exposure in children [40,41]. However, the effect was comparable to the effects of active smoking among adolescents.

Sudden infant death syndrome

Sudden infant death syndrome (SIDS) is currently defined as the sudden death of an infant that remains unexplained by clinical or autopsy evidence. It is likely that the immediate cause of death in SIDS is a

functional one acting through the cardio-respiratory system. A number of risk factors have been identified by epidemiological studies [42]. Prone sleeping position, overheating and parental smoking have been targeted as the most important modifiable factors for public health action. The earliest study to examine the association between maternal prenatal smoking and SIDS was carried out in Canada in the early 1960s [43].

Haglund and Cnattingius examined maternal smoking as a risk factor for SIDS in a prospective study of 99% of all births in Sweden over a three-year period [44]. There were 190 cases of SIDS among 260 000 live births (0.7 per 1000), representing 27% of all infant deaths between 1 week and 1 year of age. Maternal smoking was the most important preventable risk factor for SIDS. The authors estimated that if maternal smoking could be eliminated, the number of cases of SIDS would be reduced by 27%.

Maternal smoking is an important and potentially avoidable risk for death in early life. It may be that smoking is associated with abnormalities in brain development and that one manifestation of this might be a tendency to central apnoea.

Conclusion

Paediatricians should not be complacent about the hazards of passive smoking. The smoking habits of parents may be the most important influence on whether the children will themselves become active smokers in adolescence. Paediatricians and everyone responsible for the health of children should routinely ask parents about smoking in the home and inform them of the risk to their children that smoking represents. Given the available evidence, parents can be told that avoiding smoking during pregnancy and after their child is born offers an opportunity to reduce significantly the risk of illness in their children.

Parents who already smoke but wish to give up need to be offered informed guidance and continuing support. This will require liaison between paediatricians, general practitioners and local community services, such as smoking cessation clinics.

Despite evidence of satisfactory knowledge about smoking among doctors and parents, efforts in public health information should continue.

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