Environmental tobacco smoke exposure and children's health

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Abstract

Almost half of the child population is involuntarily exposed to environmental tobacco smoke (ETS). The ETS exposure gives rise to an excessive risk of several diseases in infancy and childhood, including sudden infant death syndrome, upper and lower respiratory infections, asthma and middle ear diseases. It is also linked to cancer, and behavioural problems and neurocognitive deficits in children. Thus protecting children from ETS exposure is a complex and important issue. The best improvement for children's health is to be gained when parents will quit smoking or when that is not possible for them to stop smoking in their children's environment. Paediatricians, because of their

authority, and their frequent and regular contact with parents, play a leadership role in protecting children from ETS exposure. An ideal approach to help parents to quit smoking seems to be initial minimal-contact advice provided from their paediatrician with feedback and supplemental printed materials, loading to greater intensity and duration in follow-up home visits.

Introduction

Environmental Tobacco Smoke (ETS), also known as Second Hand Tobacco Smoke (SHTS), refers to the mixture of side stream smoke coming from the burning tip of the cigarette and exhaled mainstream smoke. Passive smoke includes a range of tobacco combustion by-products, including carcinogens (1).

Children can be exposed to ETS during prenatal and postnatal period. Prenatal exposure refers to the maternal smoking during pregnancy or maternal passive exposure to ETS during pregnancy. Postnatal exposure refers to the child's exposure to ETS from tobacco products smoked in their surroundings. Young children's exposure to ETS occurs mainly at home, but also in other indoor environments (e.g., cars, schools or public places).

Based on the data from epidemiological studies, we can assume that about 20-30% of women actively smoke during pregnancy. In addition, about half of the non-smokers, so 35 to 40% in total are exposed to passive smoking (2). Many studies dealing with smoking cessation interventions for pregnant women indicate that even minimal assistance can increase the cessation rate (3). Unfortunately, up to 60% of these women return to smoking within the first six months postpartum and 80-90% experience a smoking relapse 12 months after delivery (4). Almost half of the child population is involuntarily exposed to tobacco smoke at home (5).

There is a great need to develop studies assessing and monitoring children's exposure to ETS and the risk of developing ETS-related diseases. It is particularly important to find effective programs to protect children from ETS exposure during the prenatal and postnatal period.

Measures of exposure to ETS

It is usually difficult to separate the effects of prenatal or postnatal exposure because if the mother exposes her child to nicotine during pregnancy, she probably does so also in the newborn period.

Passive smoking of the pregnant or lactating woman or of the child can produce a nicotine level similar to a low level of active smoking.

Self-reported smoking behaviour, indoor air monitoring (air nicotine and fine particulate levels), and biomonitoring are useful tools for estimating ETS exposure. In homes where smoking takes place, ETS is the main source of indoor fine particulate matter, with 24-hour average $PM_{2.5}$ levels increasing by about $1\mu g/m^3$ per cigarette smoked per day. Cotinine, the major metabolite of nicotine, is a very specific and sensitive biomarker of recent ETS exposures and has an elimination half-life of about a day, or longer in children. Carbon monoxide and thiocyanate measures are nonspecific because the first can result from multiple exposure and the second can originate from dietary sources. The most useful biomarker of foetal exposure during pregnancy is cord serum cotinine level, which distinguishes between mothers who smoked, mothers who were passively exposed to ETS, and mothers who had neither exposure. Maternal smoking is the major determinant of urinary and salivary cotinine and hair nicotine levels among young children (6).

Children's vulnerability

Children are more likely than adults to suffer from health effects from ETS exposure and smoking in the home environment is their most important source of exposure (7, 8). They spend most of the time in homes, day-care centres, schools, cars and other indoor environments. They are suspected to be more susceptible to a range of environmental exposures. The nervous, respiratory and reproductive systems of young children are not yet fully developed and growing fast making these systems vulnerable. Also children are in some periods less able to excrete certain toxins or to make toxic metabolites. Compared with adults, children have higher relative ventilation rates leading to higher internal exposure. Furthermore, young children are often not able to remove themselves from exposure, or they are not aware of certain dangers of which adults are aware.

Impact of ETS exposure on pregnancy

Smoking in preconceptual time or during pregnancy is a significant and preventable factor increasing the risk of ectopic pregnancy, placental abruption, placenta praevia and preterm premature rupture of the membranes (9).

Foetal exposure to tobacco smoke constituents due to maternal smoking or maternal exposure to ETS can negatively influence the pregnancy outcome. Active smoking during pregnancy reduces birth weight by 10-15 g per cigarette smoked daily (average 250g) (10). The overall evidence suggests that ETS exposure of pregnant women reduces slightly the mean birth weight of the foetus. It was concluded that the best studies on ETS show weight decrement from 25 to 100 grams and pooled weight decrement was -24.9g (-33.7g to - 16.1g) (11) and – 82g (-126g to -37g) in three studies that were based on cotinine measurements (saliva, serum) and adjusted for at least one confounder (12, 13, 14). It means that the birth weight distribution shifts to lower weights with exposure to tobacco smoke. At the population level this shift would lead to an increase in the number of low birth weight infants. It may also put infants who are already compromised at even higher risk. Low birth weight (LBW) is associated with several health effects in adulthood, such as an increase in the incidence of coronary heart disease, stroke, hypertension, type 2 diabetes mellitus, insulin resistance, serum lipids and premature pubarche (15).

ETS exposure adversely affects foetal growth as was documented based on meta-analysis of studies examining the risk of Intra Uterine Growth Restriction (IUGR) or LBW at term. Based on 11 studies 20% increase of risk was observed (16).

It is well known, that maternal active smoking during pregnancy has been associated with preterm delivery (<37 weeks of gestation). In a similar way a higher risk has been associated with greater number of hours of ETS exposure (17). In a study in which ETS exposure assessment was based on nicotine concentration in maternal hair sampled after delivery, the risk of preterm delivery (<37 weeks) was increased in the high- and medium-exposure categories compared with the low one (18).

The longer the gestation for a given birth weight, the lower the infant mortality rate. Infants born preterm are also subject to a number of complications associated with physiological immaturity, including cerebral palsy, hyaline membrane disease, sepsis, and seizure disorders (19). Maternal smoking increases the likelihood for a child to be born with a small head circumference (20). Children who are born to smoking mothers experience catch-up growth in weight and partial catch-up growth in length, but the differences in head circumference persist to at least 5 years of age (21).

Health effects after birth

Sudden Infant Death Syndrome

Exposure to ETS during prenatal and postnatal period increases the risk of Sudden Infant Death Syndrome (SIDS). SIDS is defined as a sudden, unexpected death of an infant, without evidence of any fatal illness at autopsy. In most developed countries SIDS is the most common single cause of death among infants between 1 month and 1 year old (22).

Prenatal smoking is almost invariably associated with postnatal smoking. Therefore it is difficult to judge whether the effect is due to prenatal or postnatal exposure. Based on a systematic review conducted by Anderson and Cook (22) the risk for SIDS was increased almost twice in children with postnatal parental exposure after controlling for prenatal maternal smoking. The fact that infants who died from SIDS had a higher nicotine concentration in their lung tissue compared with non-SIDS cases supports the statement that postnatal exposure is related to SIDS (23, 22). Dose-response relationship between SIDS and both prenatal and postnatal maternal smoking was found in most of the studies (22).

Possible reasons for the association between passive smoking and SIDS are abnormalities in brain development with a tendency to central apnoea (24). The nicotine may affect the ventilatory response to hypoxia (24). There is also some evidence that maternal smoking is associated with abnormal pulmonary development in neonates independent of postnatal effect (25). Postnatal smoking might affect the risk of SIDS due to direct irritation of the airways or the promotion of respiratory infection.

Lower respiratory tract infections in early childhood

Based on meta-analysis of epidemiological studies, 60% increased risk of lower respiratory infections in early childhood was associated with children's ETS exposure due to either one of the parents smoking (26). Similar risk estimates were found for maternal smoking and smoking of other household members. In smoking households, children are at greater risk of hospitalisation for respiratory illness (27, 28). Risk of lower respiratory tract infections is highest in the first year of life, and remains elevated until about the age of 3 years (26).

Chronic respiratory symptoms

Chronic respiratory symptoms like wheeze, chronic cough and chronic phlegm are about 30% more prevalent in populations of children exposed to ETS due to parental smoking (meta-analysis of 60 studies) (29). The prevalence of all symptoms increases with the number of parents who smoke.

Reduced pulmonary functions

Several reviews have indicated that parental smoking has adverse effects on pulmonary function in children. In the Harvard Six Cities study 8706 schoolchildren were followed annually between the age of 6 –18 years (30). Small reductions in FEV1 through adolescence were associated with both current and pre-school exposures to maternal smoking, with an annual decrement of 3.8ml (-6.4ml to -1.2ml). In a pooled analysis of school age children, exposure to ETS was associated with reduction of 1.4% in FEV1, the mid and end expiratory flow rates show a decrease of 5% and 4.3% respectively (31). It is likely that part of this effect is attributable to in utero exposure as smoking during pregnancy has adverse effects on the pulmonary function measured in the neonatal period (32).

Passive smoking due to maternal smoking is a stronger determinant of lung function than the smoking of father or other household members, probably due to prenatal effects and closer contact of the child with the mother (31).

Increased sensitisation rate to allergens

The systematic review of the effects of parental smoking on immunoglobulin (IgE) levels, skin prick positivity, and allergic rhinitis or eczema in children was conducted by Strachan and Cook (33). Based on this meta-analysis they indicated that parental smoking, either before or immediately after birth was unlikely to increase the risk of allergic sensitisation in children. In the study by Kulig et al. children who were prenatally and postnatally exposed to tobacco smoke had significantly higher risk of sensitization to food allergens than unexposed children. No significant association between tobacco smoke exposure and specific sensitization to inhalant allergens was found (34). Another study indicated that parental smoking was associated with a significant enhancement of expression of the most important markers of allergic sensitization in the children of smoking parents which was particularly evident for boys (35).

Asthma and exacerbation of asthma symptoms

Epidemiological data indicate that ETS exposure increases both the prevalence and severity of asthma (36). Clinically diagnosed asthma was found to be associated with parental smoking in a meta-analysis of 37 studies (16). Similar results were provided by a meta-analysis of 25 studies on the relationship between asthma and either parent smoking (29). Additional evidence has come from a meta-analysis of 8 longitudinal studies, which concluded that incidence of asthma or wheezing was related to maternal smoking (29). While maternal smoking had a greater effect than paternal smoking, the effect of paternal smoking alone was clearly significant suggesting that the postnatal effect is important (29). Parental smoking increases the frequency of attacks, the number of emergency department visits and the risk of intubation (37). The severity of asthma decreases in children when exposure was reduced (19).

In the causation of respiratory disease, particulate matter as well as vapour phase components of ETS, such as carbon monoxide, may be important. Mechanisms by which maternal smoking during pregnancy and children's ETS exposure might enhance asthma, include an irritant effect, increased bronchial hyperreactivity, altered variations in pulmonary function, and increased sensitivity to allergens (38, 39, 40).

Middle ear disease

Evidence consistently indicates an increased risk for both acute and chronic middle ear diseases in children exposed to parental smoking (41, 42).

The possible explanation for the relation between ETS exposure and middle ear disease is a direct effect of cigarette smoke on host defences (43).

Cancer

Tobacco smoke contains over 40 known carcinogens. ETS originates mainly from side stream smoke, in which, because of the lower temperatures of idling cigarettes causing less complete combustion, there are higher concentrations of most carcinogens compared to mainstream smoke. ETS is a known human carcinogen, based on epidemiological studies indicating a causal relationship between ETS exposure and increased risk of lung cancer. (1).

Many epidemiological studies have evaluated the association of childhood cancer risk with exposure to parental smoking (1, 44, 45, 46). It is difficult to distinguish whether the exposure to tobacco

smoke from parents was in preconception, prenatal or postnatal period. Mothers and fathers who smoke during pregnancy have usually smoked before the pregnancy and continue to smoke after delivery.

A ten percent increased risk for all cancers combined was found for maternal smoking during pregnancy based on meta-analysis of epidemiological studies conducted by Boffetta et al. (1). There were no discrepancies among the results of the cohort studies and those of the case-control studies and no evidence of publication bias. The results on exposure to maternal tobacco smoke before pregnancy and after delivery are inconsistent.

In most studies no significant association between maternal smoking and brain tumours or all tumours of the central nervous system (CNS) were found. On the other hand the results on the exposure to paternal tobacco smoke suggest an association with the brain tumour and CNS tumours. The evidence for maternal smoke points to a possible weak effect on lymphatic and hematopoietic organs. The higher risk for lymphomas was found for paternal smoking exposure. The higher risk of specific neoplasms estimated for paternal than for maternal smoking can be related to different mechanism of action of carcinogens. In maternal smoke it is direct transplacental effect and in paternal smoke mainly via preconceptional alterations.

Several other types of childhood cancer have been studied in relation to parental smoking including kidney cancer, eye tumours, endocrine tumours, Ewing's sarcoma and lung cancer but the results are too sparse to allow a conclusion (1).

Biomarker-based studies have provided clear evidence of the potential of ETS exposure of children to cause genotoxic damage, which may be associated with carcinogenesis. For example, increased levels of carcinogen-DNA adducts (some of which may reflect increased cancer risk) have been found in blood leukocytes of children of smoking mothers (47), while metabolites of tobacco-specific carcinogens have been detected in the urine of ETS-exposed children (48, 49).

ETS is a complex mixture of thousands of chemicals, so the role of specific components in the induction of health effects and the associated mechanisms are not known with certainty. For carcinogenesis, it is thought that polycyclic aromatic hydrocarbons (PAHs) and nitrosamines may play major roles through mechanisms involving genotoxicity (induction of DNA damage) as well as pathways connected with the production of reactive oxygen species.

The levels of carcinogen-DNA adducts found in the cord blood DNA of newborns of smoking mothers, as well as in blood leukocyte DNA of children exposed to ETS, tended to be higher than those of their mothers, suggesting that the foetus and children may have increased susceptibility to the genotoxic effects of airborne PAHs (48).

Neurodevelopmental and behavioural problems

There is increasing evidence that ETS exposure leads to negative behavioural and neurocognitive effects in children. Maternal smoking during pregnancy has been associated with 10% deficits in general intellectual ability, language/auditory-related tasks, academic achievement and behavioural problems such as hyperactivity and decreased attention spans (19, 50). The studies reported poorer academic performance measured by school progress or by achievement test scores in relation to paternal, maternal or household smoking. Some researchers have found decrements in performance on a range of cognitive, perceptual, central auditory and linguistic abilities associated with postnatal ETS exposure after controlling for prenatal maternal smoking. Postnatal ETS exposure has also been associated with behavioural problems in children such as attention disorder with hyperactivity (50).

The commonly accepted mechanism by which ETS is associated with behavioural or neurodevelopmental difficulties is altered brain development resulting from foetal hypoxia. Nicotine may also target specific neurotransmitter receptors in the brain causing abnormalities in cell proliferation and differentiation (50).

Potential for interventions

Environmental tobacco smoke is a significant public health problem. The protection of children from ETS exposure because of health consequences should start in prenatal or even in preconceptional period. Many studies dealing with smoking cessation interventions for pregnant women indicate that even minimal assistance can increase the cessation rate (3). In pregnancy, usual care providers, especially physicians and midwives, are well placed to provide smoking cessation interventions because of their knowledge, authority and regular contact with pregnant women (51).

Regulatory restrictions or legal interventions to protect children such as eliminating smoking in day care settings, schools and public places do not address homes as the main source of children exposure

to ETS. It is difficult to monitor and regulate behaviour in private residential settings. Thus protecting children from ETS exposure in home environments is a complex and sensitive issue.

Paediatricians have a unique and important role to play in the prevention and treatment of childhood and adolescents tobacco use, the protection of patients from the harmful effects of environmental tobacco smoke, and the encouragement of smoking cessation among parents (52). First, paediatricians have frequent and regular contact with parents of young children. Because of their authority, the physicians may provide the greatest motivation to quit smoking comparing to friends, relatives, legal restrictions, higher tasks and antismoking advertising (53). Several studies have demonstrated physician effectiveness in smoking cessation interventions during the scope of regular practice (54, 55). Finally there are many guidelines, which support this type of interventions.

There are several components of effective smoking cessation intervention given by paediatricians (52). It is important to identify the ETS sources in children's environment (parental, grandparents and other caretakers smoking, smoking in cars, and tobacco smoke in day care and schools). Parents should get information on the adverse effects of ETS and benefits of quitting smoking. This information has the best impact when it is delivered on a person-to-person level to the parents. Paediatricians should discuss the way in which parents can quit smoking or at least reduce tobacco smoke in their child's environment. During the follow-up visits all changes in smoking habits should be evaluated. The National Cancer Institute developed an approach to brief office-based smoking cessation treatment for adults which is referred to as the 4-A model (ask, advice, assist, arrange follow-up) (56).

The best improvement for children's health is to be gained when parents will quit smoking or when that is not possible for them to stop smoking in their children's environment. Parents can alter their smoking behaviour in ways that might reduce their children's exposure to ETS such as: smoking outdoors, improving ventilation, or smoking in another room or away from the child.

A review that summarised evidence for some methods to reduce ETS exposure effectively was conducted by Gehrman et al. (53). Out of 19 reviewed studies, 11 studies found significant reduction in reported ETS exposure. The evidence from this review suggests that interventions can be effective in reducing children's exposure. The bests results were found for the more rigorous study designs, the interventions of greater intensity and duration and those based on sound behaviour change.

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