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Background Paper

Synthesis: The Health Effects of Tobacco Smoke Exposure on Children

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INTRODUCTION

Involuntary and avoidable exposure of children to tobacco smoke can begin before birth and continue throughout childhood. Active smoking by the mother exposes the developing fetus to agents in tobacco smoke that cross the placental barrier, as may involuntary exposure of non-smoking pregnant women to tobacco smoke. For the unborn child of a smoking mother, oxygen delivery may be compromised by carbon monoxide in cigarette smoke which avidly binds to hemoglobin, reducing its oxygen-carrying capacity. After birth, the child may be exposed to tobacco smoke at home from the smoking of parents and other household members or in childcare facilities or public places. Each of these routes of exposure of children to tobacco smoke is potentially avoidable, although differing prevention strategies may be needed for each. A finding that these exposures had adverse effects on the health of children would provide a clear impetus for the implementation of prevention programs.

An extensive body of scientific evidence addresses the health consequences of exposure of children to tobacco smoke. This review, prepared for the World Health Organization's Consultation on Environmental Tobacco Smoke (ETS), offers a synthesis of the literature on adverse health effects of tobacco smoke exposure on children. It draws on a set of review papers prepared for the Consultation, each addressing a specific set of health outcomes: fetal growth (1), neurodevelopment and behavior (2), sudden infant death syndrome (SIDS) (3), childhood cancer (4), cardiovascular disease (5), and respiratory effects (6). This review covers general methodological issues involved in investigating health effects of smoking on children, principles for interpreting the evidence on adverse effects of exposure, and the current status of the evidence.

Concern about the effects of tobacco smoke exposure on children is not new, and clinicians have long noted the adverse effects of exposure to tobacco smoke on the health of children. More formal observational research using epidemiological methods began decades ago with studies of the effects of maternal smoking during pregnancy (7) and surveys on the respiratory health of children in relation to household smoking (8-10). By the mid-1960s, effects of maternal smoking during pregnancy had been well-documented, with a number of studies showing reduced birth weight for children born to smoking mothers (7). Across the 1970s, mounting evidence linked parental smoking, particularly maternal smoking, to increased risk for more severe lower respiratory illnesses during the first years of life and to increased prevalence of respiratory symptoms in schoolchildren. In the late 1970s, Tager and colleagues (11) reported that smoking in the home was associated with reduced lung function; over the next decade this finding was confirmed as cohort studies showed that exposure to smoking at home reduced the growth of lung function during childhood (12). Additional adverse health effects have now been linked to involuntary exposure of children to tobacco smoke including causation and exacerbation of asthma, otitis media, and developmental consequences.

The evidence on passive smoking and children has been reviewed periodically, beginning with the 1984 U.S. Surgeon General's report (13). These systematic reviews have identified an enlarging list of adverse effects causally associated with exposure of children to tobacco smoke (Table 1 and Appendix A). This synthesis builds from the foundation afforded by these prior reviews, updating conclusions based on the background papers prepared for this Consultation.

METHODOLOGICAL CONCERNS

Overview

This Consultation focuses on the consequences of exposure of children to cigarette smoking. This exposure may begin before birth as the fetus is exposed either by active smoking by the mother or as a consequence of passive exposure of the mother to ETS. After birth, the child's exposure to parental smoking has been the principal source of concern;

maternal smoking and paternal smoking have been separately characterized because of potentially differing contributions to the exposures of children to ETS.

To an extent, researchers have attempted to isolate "independent" effects of these sources of ETS exposure to children. The exposures take place during differing windows of potential biologic susceptibility, e.g., fetal development and postnatal growth, and the exposures may differ qualitatively and quantitatively. Characterizing the consequences of the various exposures of children to cigarette smoke is complicated by patterns of cigarette smoking which tightly link prenatal and postnatal exposures. Mothers who smoke actively during pregnancy are unlikely to stop smoking after pregnancy and passive exposure of the mother to ETS in the home during pregnancy will also inevitably be followed by postnatal exposure of the infant to ETS at home. Thus, all potential possibilities in a matrix defined by the sources of prenatal and postnatal exposure cannot be investigated readily and researchers have been frustrated in attempting to characterize consequences of less frequent, but biologically relevant exposure patterns. For example, prenatal ETS exposure of the mother without any other exposures to the child is infrequent. Nonetheless, the observational evidence addresses intrinsically the exposure profiles of public health concern.

The possibility of bias has been raised repetitively as an explanation for the diverse associations of involuntary exposure to tobacco smoke with adverse health effects, whether in children or in adults. Much of the evidence on passive smoking comes from observational studies, which are inherently subject to bias, and the biological mechanisms underlying effects of passive smoking have been less certain than those considered relevant to active smoking. Critics of the evidence on passive smoking, many supported by the tobacco industry, have relentlessly argued that uncontrolled bias can explain fully the observed associations. Postulated biases include forms of differential information bias that inflate risks; uncontrolled confounding by various factors that are assumed to be associated with exposure to tobacco smoke and are also independent risk factors for the health outcomes of concern. These criticisms have been voiced in published literature and also as commentary in public hearings: for example, the Science Advisory Board's peer review of the risk assessment prepared by the U.S. Environmental Protection Agency and the hearings on proposed new indoor air quality regulations by the U.S. Occupational Safety and Health Administration.

The synthesis papers prepared for this consultation echo some of these concerns, citing information bias and confounding as potential points of weakness in some epidemiological studies on passive smoking and children. Misclassification of exposure is raised in several of the reviews and emphasis is placed on biomarkers of ETS exposure as a "gold standard." In this section, I consider these specific methodological concerns, addressing information bias and confounding specifically.

Information Bias

Information bias may be either non-differential, or random, or differential in relationship to either exposure or outcome (14). While information bias may affect either exposure or outcome, the potential consequences of information bias have received the greatest attention in studies of ETS with regard to exposure classification. In studies of cigarette smoke and children, exposure classification has been based primarily on questionnaires that assess the smoking of the parents and other household members. The resulting exposure classification of children should reflect accurately the sources of tobacco smoke in key environments, while leaving the potential for misclassification of the level of exposure to ETS generally or to specific components that may be considered biologically relevant.

Differential information bias in the assignment of exposure to children in epidemiological studies might arise in the context of case-control studies involving interviews with parents to characterize their smoking. Parents of cases might report smoking differentially in comparison with parents of controls; both under-reporting and over-reporting can be reasonably postulated, if case parents tend to minimize an exposure that they may consider harmful or if they report information more meticulously than control parents.

Differential information bias might also occur in cross-sectional studies for the same reasons but should not affect the results of cohort studies.

Non-differential information bias may affect the findings of any of the observational designs, particularly if there is an attempt to apply quantitative or semi-quantitative exposure measures, such as duration or intensity of exposure. Source description, i.e., the persons smoking, should generally be reported accurately, but the relationship between source and actual exposures of children is complex and determined by characteristics of the rooms in which smoking is taking place and the proximity of the child to the smokers. In general, non-differential information bias tends to reduce estimates of effect towards the null, reducing the estimated level of effect and widening confidence intervals. Risk estimates affected by non-differential information bias can be assumed generally to be moved away from the actual value towards the null value, i.e., no effect.

Information bias in the classification of exposure to ETS can be addressed by using direct monitoring techniques, including using diffusion badges for nicotine, or by using biomarkers such as level of cotinine in body fluids. Monitoring and biomarkers have been used to assess the validity of questionnaires for exposure classification, and biomarkers have also been used in several studies as the primary basis for exposure classification ((15), for example). The reliance on questionnaires in most studies as the primary basis for exposure classification should not be construed as a general weakness of the epidemiologic evidence. Biomarkers, while useful for validation studies, have intrinsic variability and measure only a single component of cigarette smoke, e.g., nicotine. For example, Coultas and colleagues (16) showed substantial temporal variation in nicotine levels in homes and in urinary cotinine levels in children living in these homes, which had reportedly constant smoking. Additionally, any particular biomarker may have uncertain relevance as an indicator of the biologically relevant exposure or dose for a specific health effect. Nicotine, for example, is present in ETS as a gas-phase constituent and cotinine, a nicotine metabolite, may not be an appropriate index of exposure to cigarette smoke particles.

Confounding

Confounding occurs if the effect of the exposure of interest, cigarette smoke, is mingled with the effects of one or more other factors, referred to as confounding factors. Textbook definitions of confounding indicate that confounding occurs if a risk factor for a disease is associated with the exposure of interest in the data at hand ((17, 18), although confounding may arise under less straightforward circumstances ((14). To meet these conditions, the potential confounding factor needs to be associated independently with the outcome of interest. Long lists of potential confounding factors have been offered for health outcomes considered in this Consultation, but many do not meet the criterion of independent association with these outcomes. Furthermore, these factors are not necessarily associated with the measure of cigarette smoke exposure, the second condition for confounding.

The review papers consider uncontrolled confounding as a potential limitation of many studies on children's exposure to tobacco smoke. While this concern is appropriate, the potential consequences of confounding appear overstated. First, some of the potential confounding factors may not meet the criteria for introducing confounding. Second, bias from confounding is not unidirectional and may increase or decrease risk estimates. Third, some factors offered as potential confounding factors, e.g., socioeconomic status, may in fact lie in the causal pathway for exposure to tobacco smoke. Adjustment for such factors as potential confounders may represent a mis-specification of the underlying causal pathways. Fourth, consistency of findings across diverse populations, likely to have differing distributions of confounding factors, also weighs against confounding as the explanation for associations of cigarette smoke exposure with adverse health effects.

EVALUATING CAUSALITY OF ASSOCIATIONS

Overview: Criteria for Causality

Evidence on active smoking and disease has been amassed periodically and evaluated to identify associations of smoking with disease and to assess the causality of the associations. The U.S. Surgeon General's Reports (for example,(7, 13, 19)), the reports of the Royal College of Physicians in the United Kingdom, and the 1986 report of the International Agency for Research on Cancer(20) offered such comprehensive assessments. Criteria have been offered for assessing the causality of associations; their application to smoking is a model example(7, 14). These criteria represent guidelines for interpretation, providing a framework for bringing together observational and experimental data and more fundamental understanding of biological mechanisms. While deficiencies of these criteria have been identified(14), they have not been replaced and they remain as the accepted framework for interpreting epidemiologic evidence. The 1986 report of the U.S. Surgeon General(19) specifically addressed the applicability of the criteria to the evaluation of evidence on involuntary smoking.

The criteria have been applied to the evidence on smoking and disease because of the policy implications of a finding that exposure to tobacco smoke, whether active or passive, *causes* disease. The judgment that an association is causal indicates that the evidence has crossed a threshold for certainty and inherently signals that prevention may be warranted. The criteria are inherently conservative in calling for replication and biologic understanding. A single study would not provide a sufficient basis for identifying a causal association between a risk factor and a disease.

Application of the criteria to the evidence on exposure of children to tobacco smoke, as in several of the reviews, is appropriate and provides a gauge of the current extent of the evidence. For some of the outcomes, e.g., neurodevelopmental effects and SIDS, understanding of potential mechanisms and biologic plausibility is of necessity limited. Direct experiments with children are not possible and animal models are either limited or unavailable, and of uncertain relevance in any case. For public health purposes, judgments can be reasonably taken, recognizing that the full suite of evidence needed to meet the criteria may never be available.

Meta-Analysis

Several of the review papers use meta-analysis to summarize the findings of published studies. Meta-analysis offers a quantitative synthesis of the available studies and also provides an indication of the heterogeneity of findings across studies. While its application to the literature on ETS has been controversial(21, 22), meta-analysis is a widely applied method for summarizing the results of studies in the biomedical literature. Meta-analysis may be particularly informative if individual studies have inadequate statistical power and provide effect estimates with broad confidence intervals, sometimes spanning a range that may extend from protection to upper values of public health concern.

The point estimate of effect coming from a meta-analysis should not by itself serve as the indicator of the causality of an association. Similarly, statistical significance of a meta-analysis is not interpreted properly as a measure of causality.

Causal Models

The pathways potentially linking cigarette smoke exposure to adverse health effects in children are complex, involving both indirect and direct mechanisms of action. Factors considered to be potentially confounding may, in fact, be linked to cigarette smoke exposure within multidimensional causal webs. Socioeconomic status, for example, is now tightly linked to exposure patterns in a number of countries and adverse effects of socioeconomic status on health may be mediated through its environmental correlates, such as exposure to cigarette smoke or housing quality. Pathways for effects of smoking on children may be indirect. For example, Eskenazi and Castorina(2) comment that effects of ETS exposure on

cognitive development and behavior might reflect consequences of lower birth weight or otitis media with hearing loss, rather than representing a direct effect of smoke exposure.

In general, analyses of data within specific studies are based on relatively simplistic, linear models of the effects of tobacco smoke exposure and other risk factors on risk for the adverse outcomes of interest. These models do not reflect the potentially more complex and possibly non-linear relationships of tobacco smoke exposure with adverse health effects in children. We should acknowledge the possibility that these models are mis-specified in interpreting their results.

SYNTHESIS

Fetal Growth

Windham (1) summarizes the evidence on prenatal exposure to ETS and fetal growth, evaluating the literature on three different measures: mean birth weight, intra-uterine growth retardation (IIUGR) or low birth weight, and gestational age and preterm delivery. An extensive literature already exists on birth weight and active smoking by the mother, along with well-supported conclusion that maternal smoking is causally associated with reduced birthweight averaging around 200 grams (Table 1). Windham concludes, consistent with the California Environmental Protection Agency report (23), that ETS exposure is associated with reduced birth weight. The pooled estimate from a meta-analysis of 19 studies is a reduction of 31 grams. The evidence on IUGR indicates an increased risk associated with ETS exposure, suggesting that the birth weight effect comes from IUGR, as with active maternal smoking. The overall effect estimate from the meta-analysis, approximately 10% of the effect for active maternal smoking, fits within a plausible range and appears robust to control for potential confounding factors.

Windham's review notes appropriately that the overall estimate of effect refers to a population. While the mean estimate, approximately 1% of birth weight, may appear small, a shift of the birth weight distribution would move some infants towards a higher-risk birth weight, particularly if there are some infants at greater risk because of level of ETS exposure or other factors. As a population-level effect, this reduction should accordingly not be dismissed as trivial and without clinical implications. Windham offers a research agenda, which is justified given the high levels of ETS exposure of pregnant women in many countries. However, the evidence warrants cautioning pregnant women against both active smoking and involuntary exposure to ETS.

Sudden Infant Death Syndrome

Sudden Infant Death Syndrome, a tragic event for parents, has been extensively investigated, primarily using the case-control approach. Mitchell and Milerad (3) have identified nearly 50 studies that provide findings on tobacco smoke exposure and risk for SIDS. This literature is limited potentially by uncertainty as to the underlying mechanisms by which ETS might cause SIDS and the relevant confounding factors. Nonetheless, clear evidence exists that maternal smoking is associated with increased risk for SIDS, although the comparative contributions to the risk of prenatal and postnatal exposure cannot be readily separated. The evidence does not indicate a strong effect of smoking by the father or of other persons in the household.

Mitchell and Milerad apply causal criteria to the evidence, noting that the criteria are generally met. They offer a series of hypotheses about possible mechanisms, while noting that data in support of specific hypotheses are limited. There are a number of plausible hypotheses, but whether they can be tested adequately in either human populations or animal models is uncertain. While Mitchell and Milerad do not conclude directly that maternal smoking is a cause of SIDS that conclusion is tenable and has been made in the recent report of the United Kingdom's Committee on Smoking and Health (Table 1) (24). While uncertainty remains regarding the role of prenatal and postnatal maternal smoking, the causal conclusion with regard to maternal smoking and SIDS is sufficient to warrant preventive measures.

Neurodevelopmental and Behavioral Problems

Maternal smoking during pregnancy has been assessed as a risk factor for a variety of behavioral and neurodevelopmental problems in children: reduced general intellectual ability, skills in language and auditory tasks, academic achievement, and behavioral problems such as hyperactivity and decreased attention spans. Eskenazi and Castorina(2) focus on ETS exposure to the pregnant woman and child, attempting to exclude the effects of maternal active smoking during pregnancy. They review 17 studies that provide relevant data, although smoking patterns, which so tightly link prenatal and postnatal exposure, complicate the assessment of ETS effects. Further complicating interpretation of the literature is the diversity of outcome measures and of analytic approaches followed in attempting to "adjust" for the effects of potential confounding factors. Sample sizes also appear to limit the statistical power of a number of the studies. Eskenazi and Castorina comment thoughtfully on the complex causal pathways that may relate ETS exposure to neurodevelopmental and behavioral effects.

Their guarded conclusion with regard to effects of ETS exposure on neurodevelopment and behavior seems appropriate. Effects are plausible but not yet established.

Respiratory Health

Respiratory effects of ETS exposure have been investigated extensively and conclusions have been reached by a number of groups on the causality of associations of various outcomes with ETS exposure (Table 1). There is substantial understanding of mechanisms by which tobacco smoke and other inhaled pollutants affect the lung, and biologic plausibility for the observed effects can be supported readily. Cook and Strachan(6) offer an updating of a previously published set of quantitative syntheses of the evidence on ETS exposure and respiratory health of children. The outcomes considered include lower respiratory illness in infancy, asthma, respiratory symptoms, lung function, and acute and chronic middle ear disease.

The conclusions offered by Cook and Strachan parallel those offered in other recent reviews (Table 1). ETS exposure can be considered as a cause of lower respiratory illness (croup, bronchitis, bronchiolitis, pneumonia) in children, onset of asthma and worsening of asthma, respiratory symptoms, reduced lung growth, and middle ear disease. The meta-analyses for these effects carried out by Cook and Strachan indicate increased and statistically significant risks. While overall estimates indicate relatively modest effects, i.e., summary estimates generally below 1.5, the health outcomes are generally frequent events and the public health impact from ETS exposure is consequently substantial.

For lower respiratory illnesses, the evidence links ETS exposure to more severe episodes, particularly during the first several years of life. There is a possibility that some of the risk reflects lasting consequences of prenatal exposure, although the finding that paternal smoking alone increases risk suggests that postnatal ETS exposure by itself can increase risk. Asthma, the most common chronic disease of childhood, has a genetic basis but environmental factors appear to have a strong role in determining the onset of the disease and the clinical status of children with asthma. The evidence on ETS and exacerbation of asthma has long shown that ETS exposure can exacerbate asthma in children and standard clinical practice is to recommend avoidance of ETS by children with asthma. As the evidence has mounted, its has become clear that ETS exposure is also a cause of childhood asthma. Early childhood exposure to ETS has also been causally associated with another frequent set of problems: acute and chronic middle ear disease.

During childhood, the lung completes its development as formation of the alveoli is completed during the first few years of life and lung function grows, in parallel to the increase in height. Thus, damage to the lung during childhood may have lasting effects and compromise the lung's reserve capacity. Exposure to ETS during the school years has now been shown to reduce the rate of lung function growth. The reduction, estimated to be several percentage points of predicted lung function on average, would not be expected to have

implications for the function status of specific individuals. Rather, it is another indicator of an adverse effect at the population level.

Childhood Cancer

Cigarette smoke, whether actively or passively inhaled, includes numerous carcinogens. In adults, cigarette smoking has been causally linked to a number of cancers and smoking is the dominant cause of lung cancer. Active maternal smoking and ETS exposure of pregnant women and children could thus plausibly increase childhood cancer risk. Boffetta and colleagues (4) offer a quantitative summary of the evidence on maternal smoking and ETS exposure and childhood cancer risk. They consider the evidence on the principal childhood cancers. A consistent pattern of association was not found, although the data are limited. Their call for further research seems warranted, although large, carefully designed studies will be needed.

Cardiovascular Disease

Active cigarette smoking is a well established cause of coronary heart disease, stroke, and atherosclerosis, and passive smoking has now been linked to coronary heart disease as well (23-25). These disorders have lengthy natural histories that may be affected by childhood risk factors. Gidding(5) reviews evidence on ETS exposure and cardiovascular effects in children. The research findings address initiation of atherosclerosis, oxygen transport, lipid profile, vascular function, and thrombosis. The evidence is still fragmentary but provides some indications of adverse effects of ETS exposure on the cardiovascular health of children. Research to link ETS exposure in childhood to cardiovascular disease risk in adulthood will need to be longitudinal in design and may not be feasible. Further investigations of ETS exposure and cardiovascular outcomes in children are warranted, however, to follow the leads summarized by Gidding.

CONCLUSIONS

The evidence summarized in the review papers prepared for this Consultation offers a convincing picture of tobacco smoke exposure as a cause of death, disease, and ill-health in children. While there are gaps in our understanding of the biological mechanisms for some of the effects, the evidence warrants a causal interpretation for maternal smoking and SIDS, ETS exposure and birth weight, and ETS exposure and respiratory health. Findings on ETS exposure and other health effects considered in the review papers provide a basis for concern and a rationale for further research.

The review papers and the scientific literature focus generally on the effects of tobacco smoke exposure on specific diseases and conditions. The studies do not provide a full picture of the impact of tobacco smoke exposure on the health of children; that picture can be synthesized from the evidence. The possibility of adverse effects begins before birth as maternal smoking or passive exposure of the mother reduces birth weight. Effects of exposure during gestation may be carried over as an increased risk for SIDS, effects on lung function, and neurodevelopmental effects. During childhood, exposure to smoking by the mother and father may compromise respiratory health, increasing risk for respiratory infections and asthma and impairing lung growth. There may be other risks, such as increased risk for childhood or adult cancers and adverse effects on the cardiovascular system. Some children are certain to sustain multiple adverse consequences of tobacco smoke exposure, and undoubtedly some children are particularly vulnerable. While we lack markers for identifying these children, exposure of all children to tobacco smoke should be reduced.

Table 1. Adverse Effects from Exposure of Children to Tobacco Smoke

Health Effect	SG 1984	SG 1986	EPA 1992	UK 1998
Increased prevalence of respiratory illnesses	Yes/a	Yes/a	Yes/c	Yes/c
Decrement in pulmonary function	Yes/a	Yes/a	Yes/a	
Increased frequency of bronchitis, pneumonia	Yes/a	Yes/a	Yes/a	
Increase in chronic cough, phlegm		Yes/a		
Increased frequency of middle ear effusion		Yes/a	Yes/c	Yes/c
Increased severity of asthma episodes and symptoms			Yes/c	Yes/c
Risk factor for new asthma			Yes/a	
Risk factor for SIDS				Yes/a

Yes/a = association
Yes/c = cause

SG = Reports of the Surgeon General of the United States(13),(19)

EPA = Environmental Protection Agency(26)

UK = Report of the Scientific Committee on Tobacco and Health(24)

Appendix A. Conclusions related to the health effects of children's exposure to environmental tobacco smoke, as they appear in cited reviews.

1984 SG (13):

1. The children of smoking parents have an increased prevalence of reported respiratory symptoms, and have an increased frequency of bronchitis and pneumonia early in life.
2. The children of smoking parents appear to have measurable but small differences in tests of pulmonary function when compared with children of nonsmoking parents. The significance of this finding to the future development of lung disease is unknown.

1986 SG (19):

1. The children of parents who smoke have an increased frequency of hospitalization for bronchitis and pneumonia during the first year of life when compared to the children of nonsmoking parents.
2. The children of parents who smoke have an increased frequency of a variety of acute respiratory illnesses and infections, including chest illnesses before 2 years of age and physician-diagnosed bronchitis, tracheitis and laryngitis, when compared with the children of nonsmokers.
3. Chronic cough and phlegm are more frequent in children whose parents smoke compared with children of nonsmokers. The implications of chronic respiratory symptoms for respiratory health as an adult are unknown and deserve further study.
4. The children of parents who smoke have small differences in tests of pulmonary function when compared with the children of nonsmokers. Although this decrement is insufficient to cause symptoms, the possibility that it may increase susceptibility to chronic obstructive pulmonary disease with exposure to other agents in adult life, e.g., active smoking or occupational exposure, needs investigation.
5. A number of studies report that chronic middle ear effusions are more common in young children whose parents smoke than in children of nonsmoking parents.

1992 EPA (26):

1. ETS exposure is causally associated with an increased risk of lower respiratory tract infections (LRIs) such as bronchitis and pneumonia. This report estimates that 150,000 to 300,000 cases annually in infants and young children up to 18 months of age are attributable to ETS.
2. ETS exposure is causally associated with increased prevalence of fluid in the middle ear, symptoms of upper respiratory tract irritation, and a small but significant reduction in lung function.
3. ETS exposure is causally associated with additional episodes and increased severity of symptoms in children with asthma. This report estimates that 200,000 to 1,000,000 asthmatic children have their condition worsened by exposure to ETS.
4. ETS exposure is a risk factor for new cases of asthma in children who have not previously displayed symptoms.

UK 1998 (24):

1. Smoking in the presence of infants and children is a cause of serious respiratory illness and asthmatic attacks.
2. Sudden infant death syndrome, the main cause of post-neonatal death in the first year of life, is associated with exposure to environmental tobacco smoke. The association is judged to be one of cause and effect.

3. Middle ear disease in children is linked with parental smoking and this association is likely to be causal.

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