

Tobacco Free Initiative

**International Consultation on Environmental
Tobacco Smoke (ETS) and Child Health**

**11-14 January 1999
Geneva, Switzerland**

Consultation Report

World Health Organization
Division of Noncommunicable Diseases
Tobacco Free Initiative
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I. Executive Summary

Responding to the 1997 Declaration on Children's Environmental Health of the Environment Leaders of the Eight (G8)*, WHO convened an International Consultation on Environmental Tobacco Smoke (ETS) and Child Health in Geneva, Switzerland from 11 to 14 January 1999. The Consultation brought together experts from developed and developing countries to examine the effects of ETS on child health and to recommend interventions to reduce these harmful effects and eliminate children's exposure.

The Consultation concluded that ETS is a real and substantial threat to child health, causing death and suffering throughout the world. ETS exposure causes a wide variety of adverse health effects in children, including lower respiratory tract infections such as pneumonia and bronchitis, coughing and wheezing, worsening of asthma, and middle ear disease. Children's exposure to environmental tobacco smoke may also contribute to cardiovascular disease in adulthood and to neurobehavioural impairment.

The Consultation also concluded that maternal smoking during pregnancy is a major cause of sudden infant death syndrome (SIDS) and other well-documented health effects, including reduced birth weight and decreased lung function. In addition, the Consultation noted that ETS exposure among nonsmoking pregnant women can cause a decrease in birth weight and that infant exposure to ETS may contribute to the risk of SIDS.

The scope of these health effects on children is broad, given that almost half of the world's children are regularly exposed to ETS. Most have no choice in the matter. Preventing children's exposure to tobacco smoke will lead to improved child, adolescent, and ultimately adult health, resulting in reduced mortality and substantial savings in health care and other direct costs.

Swift action to highlight the need for strong public policies to protect children from exposure to tobacco smoke is essential. These policies should aim to ensure the right of every child to grow up in an environment free of tobacco smoke. This can be achieved by two complementary strategies: eliminating children's contact with tobacco smoke *in utero* and in childhood, and reducing overall consumption of tobacco products. Effectively implementing these strategies requires combining educational programmes and legislative interventions aimed particularly at eliminating tobacco use in settings frequented by children.

* Canada, France, Germany, Italy, Japan, Russian Federation, United Kingdom of Great Britain and Northern Ireland, United States of America

II. Introduction

In response to the Declaration on Children's Environmental Health, adopted by the Environment Leaders of the Eight (G8) in May 1997, the World Health Organization (WHO) convened an International Consultation on Environmental Tobacco Smoke (ETS) and Child Health in Geneva from 11 to 14 January 1999. Experts from developing and developed countries gathered to examine the effects of exposure to tobacco smoke on child health and to develop recommendations for action to eliminate this exposure.

The adverse health effects of tobacco use among smokers are well described. Tobacco use generally begins during adolescence and continues through adulthood, sustained by addiction to nicotine. Smoking causes about four million deaths annually, mainly attributable to cardiovascular disease, lung cancer and other cancers, and chronic lung disease. Tobacco smoke is also an important source of indoor air pollution, contributing to a noxious environment, eye irritation, and unpleasant odour. Recent evidence demonstrates that ETS exposure increases risks of lung cancer and ischaemic heart disease among non-smoking adults.

The vast majority of children exposed to tobacco smoke do not choose to be exposed. Children's exposure is involuntary, arising from smoking, mainly by adults, in the places where children live, work and play. Given that more than a thousand million adults smoke worldwide, WHO estimates that around 700 million, or almost half of the world's children, breathe air polluted by tobacco smoke, particularly at home. The large number of exposed children, coupled with the evidence that ETS causes illness in children, constitutes a substantial public health threat.

This report concludes that the evidence of this harm to children is consistent and robust. Even if certain questions still require further research, there is more than sufficient evidence of harm to demand action to reduce children's involuntary exposure to tobacco smoke. Furthermore, this involuntary and harmful exposure can also be seen as a human rights violation, given the provisions of Articles 6 and 24 of the 1989 United Nations Convention on the Rights of the Child. These articles create obligations for signatory governments to guarantee children's right to life, to create an environment that maximizes the survival and development of children, and to implement measures ensuring and recognizing children's right to the highest attainable standard of health. In addition, the Convention's reporting guidelines include the requirement to identify risks to children from environmental pollution and report measures taken to reduce these risks.

The report addresses the threat to child health in three sections. The first summarizes the health effects of children's exposure to tobacco smoke. The second addresses measurement issues central to action: measuring health impacts, children's exposure and the economic burden of that exposure. The third turns to interventions and includes evidence of effective policies and programmes to reduce children's exposure to tobacco smoke along with a framework for designing cost-effective responses to this threat. It is intended that this report will serve as an impetus for taking strong and immediate action to ensure that children around the world can grow up in an environment where their health is not compromised by being forced to breathe other people's tobacco smoke.

III. Health effects of children's exposure to tobacco smoke

This section presents evidence of the harmful impact of ETS exposure on children's health. Table 1 summarizes previous conclusions as to that impact. The present report specifically addresses respiratory and middle ear disease, reduced foetal growth, sudden infant death syndrome (SIDS), neurodevelopmental/behavioural outcomes, cardiovascular effects, and childhood cancer.

Several adverse consequences of involuntary tobacco smoke exposure of children are well understood and beyond the scope of this report. Specifically, parental and peer smoking are critical and detrimental influences on future regular tobacco use. In addition, maternal smoking during pregnancy causes well-established, demonstrable harm by reducing birth weight and increasing infant mortality.

For young children, the major source of exposure to tobacco smoke is smoking by parents and other household members. Maternal smoking is typically the largest source of a child's exposure because of the cumulative effects of exposure *in utero* and close proximity to the mother during early life. As children grow older, the relative contribution of other exposure sources, including smoking in public places, increases. Distinguishing between the effects of maternal smoking during pregnancy and the child's exposure to both maternal and paternal tobacco smoke after birth can be difficult because children exposed to the first are usually exposed to the second as well. While identifying the specific source of tobacco smoke exposure may improve intervention targeting, cumulative tobacco exposure over a lifetime, whether *in utero*, from ETS exposure, or as an active smoker, causes both acute illness and chronic disease.

A. Respiratory health and middle ear disease

The effects of parental smoking on children's lungs have been extensively described in the reports listed in Table 1. This section focuses on lower respiratory tract illness during infancy, asthma, respiratory symptoms, lung function, and middle ear disease, updating previously published quantitative syntheses of the evidence on parental smoking and respiratory health of children. Studying children whose fathers smoke and mothers do not can help separate postnatal from prenatal effects of tobacco smoke.

Lower respiratory tract illness

Parental smoking is an important cause of lower respiratory tract illnesses (e.g. croup, bronchitis, bronchiolitis, and pneumonia) during the first years of life. Of over 40 studies, all but one reported increased risks among children whose parents smoke. Pooling the studies' results, children whose mothers smoke are estimated to have a 1.7-fold (95% CI = 1.6-1.9) higher risk of these illnesses than children of nonsmoking mothers. Paternal smoking alone causes a 1.3-fold (95% CI = 1.2-1.4) increase in risk. This result is strong evidence for a causal role of ETS exposure, since it is uncomplicated by maternal smoking during pregnancy. Similar effects were seen for both wheezing and non-wheezing illnesses, and across studies done in communities and those done in hospitals. Furthermore, adjustment for confounding did not change the estimates, and evidence of exposure-response relationships (i.e. increasing risks of illness with increasing exposure to tobacco smoke) was frequently observed. Because lower respiratory tract illness is a common cause of childhood morbidity, small increases in average individual risk coupled with widespread exposure imply large population impacts.

Asthma and respiratory symptoms

Asthma is the most common chronic disease of childhood, and environmental factors play an important role in determining both onset and severity. Both asthma and respiratory symptoms (wheeze, cough, breathlessness and phlegm) are increased among children whose parents smoke, on the basis of over 60 studies of school-aged children . The pooled relative risks for either parent smoking range from 1.2 to 1.4. Evidence supporting a causal role for ETS exposure comes from the small but significant effects of paternal smoking when the mother does not smoke. As with lower respiratory tract illness, adjustment for confounding did not significantly change the results and exposure-response relationships have been described. Smaller relative risks in school-aged children compared to infants are consistent with reduced exposure.

Whereas there is clear evidence that tobacco smoke exposure causes non-allergic wheezing in early life, it probably does not cause the underlying asthmatic trait. In addition, parental smoking perinatally is not associated with allergic sensitisation. Nevertheless, ETS exposure causes exacerbations of symptoms in children with asthma. In many countries, this has led to the standard clinical practice of recommending avoidance of tobacco smoke for children with asthma.

Lung function

During childhood, the lung grows as height increases. Damage to the lung during its development may have lasting effects and reduce the lung's reserve capacity. Substantial evidence demonstrates that maternal smoking during pregnancy causes sizeable adverse effects on neonatal lung mechanics. Maternal smoking is also associated with small deficits in lung function in school-aged children. However, distinguishing between residual effects of maternal smoking during pregnancy and childhood ETS exposure to explain these deficits is difficult.

Middle ear disease

Early childhood exposure to ETS is also causally associated with a major child health burden: acute and chronic middle ear disease. Over 40 studies with different designs have investigated effects of parental smoking across a range of outcomes from acute otitis media to surgery for glue ear. Pooled relative risks for these outcomes range from 1.2 to 1.4 and are statistically significant. No single study simultaneously addresses selection bias, information bias and confounding; where these have been investigated, however, the associations with parental smoking persist virtually unchanged. Few studies have compared the effects of maternal and paternal smoking and none have compared the effects of prenatal and postnatal exposure. Moreover, prognostic studies, showing improvement when the child has a smoke-free environment, and consideration of biologic mechanisms strongly suggest that postnatal ETS causes increased risk of middle ear disease in children.

Summary

Substantial benefits to children would arise if parents stopped smoking. While an important message must be for the mother to stop smoking before pregnancy, additional important benefits would result from postnatal cessation. Smoking by the father or other adults may have adverse effects during gestation and has definite effects after birth including increased relative risks of lower respiratory tract infection during infancy, and respiratory symptoms, middle ear disease among older children. Although these increased risks are modest, these are common health problems around the

world. Thus small increases in risk translate into a substantial burden of disease for children arising from exposure to ETS.

Research directions

Further studies might be helpful to distinguish between the effects of *in utero* exposure to tobacco smoke and postnatal exposure. Studies in settings where women rarely smoke but tobacco smoke exposure from paternal and other sources is high would be particularly valuable. Where longitudinal studies are underway, analyses to investigate how changes in exposure are related to changes in outcome would be a valuable contribution. In addition, studies demonstrating the reversibility of adverse effects with reduced exposure to tobacco smoke, particularly in high-risk groups such as children with asthma and other health conditions exacerbated by tobacco exposure, would be useful.

B. Foetal Growth

The adverse effects of maternal smoking during pregnancy on foetal growth have been known for some time. Low birth weight (LBW, defined generally as less than 2500 grams) and intrauterine growth retardation (IUGR) are important risk factors for childhood morbidity and mortality. Over 30 studies have examined the effects of nonsmoking mothers' exposure to ETS during pregnancy on foetal growth (e.g. by studying mean birth weight or LBW/IUGR) or preterm birth. Using mean birth weight as the outcome, studies from different countries and with different study designs have consistently found birth weights reduced among the offspring of nonsmoking women exposed to tobacco smoke during pregnancy. Many of the studies attempted to control for potential confounding factors. Pooling the results in a meta-analysis yields a mean reduction in birth weight ranging from 25 to 40 grams, depending on the subset of studies included. In addition, pooling estimates for LBW or IUGR yields a relative risk ranging from 1.2 to 1.4. Furthermore, relative risks and mean birth weight reductions were generally higher in higher exposure groups and in studies using a biomarker to measure exposure, and thus likely increasing the accuracy of exposure measurement. These results are supported by animal studies of sidestream smoke exposure. Thus, the weight of evidence indicates that ETS exposure in pregnant nonsmokers causes a small reduction in foetal growth.

At the population level, a small change in average birth weight could affect large numbers of infants because of the frequency of exposure. Furthermore, a shift in the birth weight distribution could have profound impacts on infants already at risk, moving them to a critically low birth weight.

Research directions

Additional studies of mean birth weight are unlikely to change the pooled measure of effect to any extent, but studies using a biomarker of exposure to increase the accuracy of exposure measurement and examine dose levels may still be warranted. Only a small number of studies have examined the role of ETS in increasing the risk of preterm birth, and additional studies would be valuable. In addition, research is needed to examine potentially more susceptible groups, such as those defined by maternal age or ethnicity.

C. Sudden infant death syndrome (SIDS)

There is sufficient evidence to conclude that maternal smoking causes a marked increase in SIDS. Almost 50 studies have examined this relationship and all indicate an increased risk. Since

reductions in the prevalence of prone sleeping position, eight studies have examined maternal smoking and SIDS. The pooled unadjusted relative risk from these studies is approximately 5, indicating that infants of mothers who smoke have almost five times the risk of SIDS compared with infants of mothers who do not smoke. Adjustment for potential confounders lowers the risk estimate; however, many studies over-adjust, e.g. by controlling for birth weight, resulting in an inappropriately low estimate of the risk. Pooling the adjusted results still yields a significantly increased risk of SIDS from maternal smoking.

With the available data, it is difficult to distinguish the effect of active maternal smoking during pregnancy from that of postnatal ETS exposure of the infant from smoking by the mother. While the mechanism for SIDS is unknown, the predominant effect from maternal smoking is generally attributed to *in utero* exposure of the foetus.

However, clear evidence for an ETS effect arises from six studies examining SIDS and paternal smoking where the mother is a nonsmoker. The pooled unadjusted relative risk from these studies is 1.4 which, though smaller than the effect seen for maternal smoking (RR = 4.7), still indicates a significant risk.

Research directions

Additional studies are unlikely to influence the pooled estimate of risk from maternal smoking substantially. Studies of nonsmoking mothers may clarify to what extent postnatal tobacco smoke exposure contributes to the risk of SIDS. Given the overwhelming evidence of maternal smoking's effects in increasing the risk of SIDS, interventions to assist pregnant women to stop smoking should be given high priority.

D. Neurodevelopmental effects

When compared to children of nonsmokers, children of smokers perform more poorly in school. They also have lower scores in cognitive functioning tests - in particular, language and auditory processing - and have more behavioural problems, including conduct disorders, hyperactivity, and decreased attention spans. Cognitive and behavioural deficits in children have lifelong consequences and result in increased costs for education and social services.

Seventeen studies have addressed the effects of ETS exposure on child development and behaviour. Most have controlled for sociodemographic characteristics and some have demonstrated dose-response relationships, with greater deficits among children with higher exposures. In some studies, children's postnatal ETS exposure and ETS exposure of nonsmoking mothers during pregnancy have been independently associated with subtle changes, albeit statistically significant, in child development and behaviour. Adverse effects resulting from children's postnatal exposure to tobacco smoke are biologically plausible in light of evidence of altered brain development in animal models. Taken as whole, however, these studies are difficult to interpret, in part due to the possible influence of uncontrolled confounding factors. Thus, the effects of prenatal and postnatal ETS exposure on cognition and behaviour remain unclear.

Research directions

Future studies need to focus on specific effects or outcomes associated with different periods of exposure. Particular attention should be given to controlling for essential explanatory variables such as parental intelligence, social class, and home environment.

E. Cardiovascular effects

In adults, active and passive smoking cause cardiovascular disease. In children, adolescents, and young adults, there is some evidence that ETS exposure may accelerate the evolution of cardiovascular disease. These studies document deleterious effects on oxygen transport, high-density lipoprotein (HDL) cholesterol, and possibly endothelial function.

In addition, the adverse effects of tobacco smoke exposure in adults in thrombosis, endothelial function, and low-density lipoprotein (LDL) oxidation can be plausibly inferred to occur in children. Studies of atherosclerosis in young adults indicate that exposure to tobacco smoke during childhood may also accelerate the progressive development of atherosclerosis. Evidence from animal models supports this initiation. However, the magnitude and clinical significance of these changes with respect to disease during adulthood are at present unknown. Neither the relationship of tobacco smoke exposure to vascular events during childhood nor the reversibility of these adverse effects has been assessed.

Research directions

Due to the limited number of studies in this area, further work on the evolution of atherosclerosis during youth and into young adulthood, with particular attention to tobacco smoke exposure, would be valuable. In addition, measurable cardiovascular endpoints of tobacco smoke exposure occurring in children and youth need to be developed. Such endpoints would also aid in evaluating reversibility of effects when exposure is discontinued. Focusing on studies of children, adolescents, and young adults with other major cardiovascular risk factors to examine whether tobacco smoke exposure enhances the effect of other risk factors would also add substantially to current knowledge.

F. Childhood cancers

Tobacco smoke, whether voluntarily or involuntarily inhaled, includes numerous carcinogens. Among adults, active smoking has been causally linked to cancers at a number of sites and exposure to ETS is an established cause of lung cancer among nonsmokers.

On the issue of childhood neoplasms, the pooled estimate of the relative risk of any childhood neoplasm from exposure to maternal smoking is 1.11 (95% CI = 1.00-1.23, based on 11 studies), and that for leukaemia is 1.14 (95% CI = 0.97-1.33, based on 8 studies). While fewer studies are available on paternal smoke exposure, associations are suggested for brain tumours and lymphomas. No consistent pattern of dose-response relationship has been found with either maternal or paternal smoking in the limited set of studies addressing this issue. Although known risk factors for childhood cancer do not appear to confound the observed associations, the small increases in risk might be the result of confounding by unknown factors. In most available studies, no distinction is made between preconceptional, *in utero* and postnatal exposure to parental smoke, making it difficult to assess the separate contribution of these periods of exposure. In conclusion, there is suggestive evidence linking exposure to tobacco smoke and childhood cancer.

Research directions

Future studies on exposure to tobacco smoke and childhood cancer should be based on large series of cases and should address the contribution of exposure preconceptionally, *in utero*, and postnatally.

G. Summary

- ETS exposure is causally associated with increased risks of lower respiratory tract illnesses, including bronchitis and pneumonia, in the first years of life.
- ETS exposure is a cause of chronic respiratory symptoms in school-aged children.
- ETS exposure increases the severity and frequency of symptoms in children with asthma.
- ETS exposure is causally associated with increased risk of acute and chronic middle ear disease.
- Maternal smoking is a cause of small reductions in lung function. The predominant effect may be from smoking during pregnancy.
- ETS exposure of nonsmoking women during pregnancy is a cause of small reductions in average birth weight.
- Maternal smoking is a major cause of SIDS. The predominant effect is believed to be from *in utero* exposure. There is also some evidence that postnatal ETS exposure contributes to the risk of SIDS.
- Parental smoking is associated with learning difficulties, behavioural problems, and language impairment in their children. There is some evidence that both ETS exposure to nonsmoking women during pregnancy and children's postnatal ETS exposure may contribute to small impairments.
- ETS exposure is associated with physiological changes in children that may increase the risk of cardiovascular disease.
- There is suggestive evidence that parental smoking may increase the risk of some childhood cancers. However, the potential roles of preconceptional, *in utero*, and postnatal exposures are unknown.

Because many of these adverse health effects are common ailments and ETS exposure is very widespread, even small increases in average individual risk result in large population risks. In other words, a large number of cases of these illnesses can be attributed to ETS exposure. Furthermore, there may be susceptible subgroups of children that are at even higher individual risk, although this issue has not been well studied. Finally, while the effects may be difficult to measure, it is plausible that children's exposure to ETS and consequent health effects may increase risks of further adverse health effects in adulthood.

IV. Measurement: health impact, exposure and economic impact

Estimating the public health impact of ETS exposure in children, the extent of that exposure, and the economic costs associated with it are essential to provide the information necessary for action to reduce the public health consequences of ETS on child health.

A. Measuring health impact

Health impact can be measured using attributable risk methods. These combine information on the extent to which disease outcomes are increased among children exposed to tobacco smoke with data on the number of children who are exposed in order to estimate the proportion of disease caused by tobacco smoke. For many of tobacco smoke's effects on children, epidemiological evidence is sufficiently strong to estimate the attributable risk of disease. From a public health perspective, even if individual risks are only slightly increased, exposure to tobacco smoke nevertheless has a major health impact because almost half of the world's children are exposed.

Measures of impact are needed to motivate and guide policy development and interventions to protect children from tobacco smoke exposure. Given limited resources, intervention priorities would ideally be established on the basis of measures of impact. Some tools are already available for this purpose but further development is encouraged, particularly of generally applicable models. At the most basic level, surveys of tobacco use including questions to assess children's household exposure to tobacco smoke are needed.

B. Measuring exposure

Children's involuntary exposure to tobacco smoke can be measured in several ways: air sampling, uses of biomarkers, and application of survey instruments. Air sampling involves measuring concentrations of such markers as respirable suspended particulates or nicotine. Biomarkers involve measuring concentrations of smoke components in biological materials, most commonly cotinine in saliva or urine. Both cotinine measurement and air sampling are limited to describing current exposure. Biomarkers may also be valuable to validate questionnaire responses.

Numerous studies have established that young children's exposure to tobacco smoke comes mainly from smoking within the home, especially by parents. Maternal smoking has a greater impact on children's measured cotinine levels than paternal smoking. Smoking by other household members and child care personnel accounts for a smaller proportion of children's total exposure.

From a survey design perspective, this point is critical, because children's exposure can be measured with a few simple questions about parental smoking. Three kinds of surveys are generally used for this purpose: household, school-based and general surveys. In household surveys, both parental smoking and the presence of children can be directly measured. In school-based surveys, children can accurately report whether or not their parents smoke but are less able to give valid reports of parental cigarette consumption. Surveys of smoking in the general adult population may also be useful, as smoking prevalence among all adults between the ages of 20 and 50 years is similar to that among parents with children.

In most countries, particularly those where people are generally not aware of tobacco smoke's harmful effects on children's health, parental smoking status will reflect children's exposure, as smoking parents are unlikely to minimize their children's exposure by not smoking in their

presence. However, in countries where concerns about children's exposure are growing, more parents, including smokers, are initiating smoking policies in the home and reporting that they generally restrict smoking to outdoors. In the future, therefore, parental smoking status by itself may become a less valid indicator and information regarding self-reported behaviours to protect children will be needed. Moreover, these self-reports will need to be validated by biomarkers of children's actual exposure to offset socially desirable but inaccurate questionnaire responses.

Estimating the public health burden of children's exposure to tobacco smoke requires accurate data on the number of children exposed, on a country-by-country basis. Based on present estimates of global cigarette smoking prevalence, WHO estimates that almost half of all children worldwide live in a home with at least one smoker. However, accurate country-level estimates are lacking, and few countries have data tracking the proportion of children living in smoking households over time.

WHO has already developed standardized questionnaires to measure adult smoking prevalence. Modifying them to include items enquiring specifically about household smoking and any rules adopted to protect children would be a potentially cost-effective way of gathering this crucial information. In addition to questionnaire-based surveys, cross-national surveys, incorporating a biomarker such as cotinine, in as many cultures, climates and regions as possible would quantify the extent of exposure worldwide and determine the significance of parental smoking as a predictor of dose across countries and cultures. If repeated over time, such a series would establish trends within and across countries in children's actual exposure to tobacco smoke, taking into account both changes in cigarette smoking prevalence and the effects of measures adopted to protect children from exposure.

C. Measuring economic impact

Estimating costs of children's exposure to tobacco smoke is important in evaluating tax and fiscal policy, managing public and private health care costs, and evaluating programmes to induce smokers to stop smoking or reduce the amount they smoke. Governments can use economic impact estimates as they consider policy and programme choices, since cost-benefit analyses may contribute to decision-making. Economic impact estimates are also useful from a health services perspective, because they highlight the savings if adults stop smoking or otherwise reduce exposure of children to tobacco smoke.

Costs arise from the established adverse health effects of children's exposure to tobacco smoke. Tobacco smoke exposure contributes to morbidity in children, increasing short-term direct health costs. These costs are largely preventable, because the behaviour of adults who are exposing infants and children to tobacco smoke can be altered, yielding direct savings for those who pay for health care services, whether government, insurers, or individuals.

Morbidity costs accrue from direct outlays for health care for children experiencing middle ear infections, lower respiratory tract illness and asthma, all of which are increased among children exposed to tobacco smoke. Children with these conditions will require more services from physicians, more prescription drugs and more hospitalizations. Their families may well suffer indirect costs in the form of time spent seeking care.

Indirect costs also arise from the additional mortality of children exposed to tobacco smoke. Economists typically estimate these costs in terms of the value of life lost. Specifically, maternal

smoking during pregnancy or exposure of the infant to smokers causes additional deaths from sudden infant death syndrome (SIDS). Although the predominant tobacco exposure of an infant dying of SIDS occurs *in utero* as a result of maternal smoking during pregnancy, risk of death appears to be elevated by exposure to tobacco smoke after birth, and death as an infant entails a substantial loss of years of life.

Using mortality and morbidity rates, together with estimates of the increased rates attributable to tobacco smoke, researchers have measured the costs of children's exposure to tobacco smoke, mostly in developed countries. These studies have generally estimated the extra costs associated with health care services related to the childhood conditions discussed in section III of this report. While these studies are limited in number and the cost estimates vary, annual health care costs attributable to children's involuntary exposure to tobacco smoke in the United States alone are estimated at approximately US\$ 1 000 million (1997 dollars). If the effects of active maternal smoking in reducing birth weight are included, this figure more than doubles. Furthermore, the long-term costs of special educational or other services for those with developmental impairments secondary to low birth weight are not included in this estimate. Considering indirect costs would further increase the estimated costs.

D. Extrapolating to developing countries

The majority of studies measuring costs of children's exposure to tobacco smoke have been in developed countries. For developing countries, these costs, while likely to be significant, may be affected by country-specific conditions. The magnitude of costs in any country depends not only on the levels of children's exposure and rates of disease, but also on the monetary values placed on health care and other resources. If a country lacks a well developed health care system, costs will appear artificially low as individuals cannot have access to care. As less developed countries develop and their health systems evolve, costs will become more real and are likely to grow from their current levels as smoking prevalence rises in these countries, more children are affected, and services become available to offer them treatment for the illnesses in which tobacco smoke is implicated.

E. Summary

The epidemiological evidence indicates that the effects of children's exposure to tobacco smoke are significant. Measuring these exposures in developing countries and monitoring the health effects of tobacco smoke in all countries continues to be a priority. While developing countries may not at first seek to precisely estimate the costs of children's exposure to tobacco smoke, they will need to monitor levels of children's exposure over time. In this manner they will have an indication of the direction of change in adverse health effects and health care costs borne by their citizens.

V. Interventions to eliminate children's exposure to environmental tobacco smoke

A. The need for action

In view of the significant health risks posed to children by ETS, public health policies are needed to protect this vulnerable population. The aim of such policies is to ensure the right of every child to grow up in an environment free of tobacco smoke, in accordance with the United Nations Convention on the Rights of the Child.

The tobacco industry has deliberately misrepresented the extent of the harms of passive smoking, and has undertaken a campaign to discredit the scientific findings and confuse the public. Yet the evidence brought together in this report demonstrates an overwhelming scientific consensus about the health risks to children from involuntary exposure to tobacco smoke. These risks are avoidable and can often be addressed simply and without great expense. This section recommends a range of interventions and outlines the policy tools needed to eliminate childhood exposure to tobacco smoke.

B. Strategies for Reducing Children's Exposure to Tobacco Smoke

Strategies to prevent children's involuntary exposure to tobacco smoke fall into two general groups: a primary focus on eliminating children's contact with the tobacco smoke of others, and secondary benefits from reducing the prevalence and consumption of tobacco products. Protecting children from tobacco smoke is essential to comprehensive tobacco control, which includes preventing the initiation of smoking, eliminating involuntary exposure to tobacco smoke, and supporting smoking cessation. The following key points should be considered in any intervention programme:

- Successfully eliminating children's involuntary exposure to tobacco smoke requires a comprehensive health promotion effort with two main thrusts: legislation and education.
- Legislation encompasses all regulatory approaches to controlling where and when people can smoke. Education includes public information, debate, and advocacy, all designed to encourage behaviour change. These two approaches are complementary, and the appropriate mix of education and legislation will depend on the local and cultural conditions of the particular country or region.
- Governments have a responsibility to legislate to control exposure to tobacco smoke in public spaces. Legislation to ban smoking in places children frequent is an important first step. These include schools, child care and health care facilities, and places where young people gather, including sports clubs, restaurants (especially fast food outlets), shopping centres and public transport. Smoking restrictions in the workplace are important to protect pregnant women and minors.
- Legislation is of limited value in reducing exposure in private homes. Educational strategies, including education about the risks to children from ETS exposure and steps to eliminate exposure, are likely to be more effective in these settings. Educational programmes must strategically target household decision-makers, either directly or indirectly, such as through other family members. Similar strategies are needed to protect children from ETS exposure in cars.
- Effective education may ease implementation of legislation but does not do away with the need for enforcement of legislation.
- Health warnings on cigarette packages advising smokers that their tobacco smoke is injurious to children and others should be included in the range of required messages.
- Health care workers are an important component of any education strategy as they can counsel children about avoiding smoke and adults about the importance of smoke-free air for children. This role is particularly critical when young patients are already suffering from an ETS-induced

illness. Training for physicians and other health professionals should include an adequate attention to health impacts of ETS.

- Skilful use of mass media, based on principles of communication science, coupled with advocacy that links government public health policy with efforts by health professionals and grassroots groups, is essential to the success of any educational campaign.
- Particular attention should be given to interventions to assist pregnant women to stop smoking. Preventing them from relapsing and inducing their partners to stop smoking will increase the likelihood of a smoke-free environment for children. Training for health professionals who work with pregnant women is crucial.
- A secondary benefit of reduced tobacco consumption is reduced exposure of children to tobacco smoke. WHO and other international and intergovernmental bodies recommend measures such as: 1) banning all advertising and promotion of tobacco products, 2) increasing prices beyond inflation, 3) regulating tobacco products, and 4) prohibiting sales to minors as steps towards reducing overall consumption.
- Interventions should be monitored and evaluated from the perspective of both efficacy and cost-effectiveness.

VI. Conclusions and recommendations

- ⇒ Almost half of the world's children are involuntarily exposed to tobacco smoke.
- ⇒ Exposure to environmental tobacco smoke causes increased risks of several illnesses in children and may increase the risk of death from sudden infant death syndrome (SIDS). Exposure of nonsmoking women to environmental tobacco smoke during pregnancy also causes reductions in fetal growth.
- ⇒ Children do not choose this exposure. Their right to grow up in an environment free from tobacco smoke must be safeguarded through actions by national and local governments, voluntary bodies, community leaders, health workers, educators and parents.
- ⇒ Reducing children's exposure to tobacco smoke requires a two-pronged strategy: reducing smoking in spaces where children live, play, and learn, and reducing overall tobacco consumption.
- ⇒ Effective public policy is important to protect this vulnerable group.
- ⇒ To maximize impact, policies to protect children from tobacco smoke exposure should be implemented as part of comprehensive tobacco control programmes.
- ⇒ Legislated restrictions on smoking in public places and the workplace will protect nonsmokers in general and vulnerable groups such as children and pregnant women in particular.

- ⇒ Young children's greatest exposure to tobacco smoke occurs at home. Increasing the percentage of tobacco-free homes is generally not amenable to legislation but can be achieved by a combination of mass media campaigns and smoking restrictions in public places and the workplace.
- ⇒ Programmes to raise awareness and motivate behaviour change among pregnant women and their partners are needed to reduce the harmful effects of prenatal and postnatal exposure to tobacco smoke.
- ⇒ Interventions through legislation and education need to be culturally specific.
- ⇒ Surveys, using biomarkers where possible, will be necessary to plot changes in children's involuntary exposure and monitor the effectiveness of interventions

Table 1. Summary of conclusions from earlier health agency reports on environmental tobacco smoke and child health

Report ¹	Lower respiratory tract infections ²	Middle ear disease	Chronic respiratory symptoms	Asthma	Lung function	Sudden infant death syndrome (SIDS)
United States Surgeon General (1986)	More frequent in children whose parents smoke	Suggestive evidence that middle ear effusion ³ is more common in children whose parents smoke	More frequent in children whose parents smoke	Not reviewed	Small decrements in children whose parents smoke	Not reviewed
United States Environmental Protection Agency (1992)	ETS is causally associated with increased risk	ETS is causally associated with increased prevalence of middle ear effusion	ETS is causally associated with increased prevalence	ETS is causally associated with additional episodes and increased severity of symptoms in asthmatic children; suggestive evidence that ETS causes new cases of asthma	ETS is causally associated with small reductions	Strong evidence that maternal smoking increases the risk of SIDS. Data inadequate to assess specific role of ETS
California Environmental Protection Agency (1997) ⁴	ETS is causally associated	ETS is causally associated	ETS is causally associated	ETS is causally associated with asthma exacerbation and induction	Suggestive evidence of causal association with ETS	ETS is causally associated
Australian National Health and Medical Research Council (1997) ⁵	ETS has cause-and-effect relationship	Causal link between ETS and middle ear effusion	Not reviewed	Causal relationship between ETS and asthma	Association with ETS exposure	Causal association with ETS
United Kingdom Scientific Committee on Tobacco and Health (1998)	ETS is a cause	Parental smoking causes acute and chronic middle ear disease	Convincing evidence that parental smoking increases risk	ETS is a cause of asthma attacks	Not reviewed	ETS has cause-and-effect association

¹ See bibliography for details of source.

² In infants and very young children.

³ i.e., fluid in the middle ear, or “glue ear”.

⁴ The report also concluded that exposure of pregnant nonsmokers to ETS is causally associated with reduced foetal growth and that there is suggestive evidence that ETS is causally associated with adverse impacts on cognition and behaviour.

⁵ The report also concluded that there is suggestive evidence that exposure of pregnant nonsmokers to ETS causes reduced foetal growth.

VII. Background Reading & Additional Sources

Australian National Health and Medical Research Council. *The Health Effects of Passive Smoking* (1997) ISBN 0-642-27270-0

Further details and full text available at:

<http://www.health.gov.au/nhmrc/advice/nhmrc/foreword.htm>

California Environmental Protection Agency Office of Environmental Health Hazard Assessment. *Health Effects of Exposure to Environmental Tobacco Smoke*. (1997)

Further details and full text available at:

http://www.oehha.org/archive/tobacco_smoke.html

Report of the Scientific Committee on Tobacco and Health. Department of Health, UK (1998)

Further details at: <http://www.official-documents.co.uk/document/doh/tobacco/contents.htm>

United States Environmental Protection Agency. EPA Office of Research and Development, "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders", EPA/600/6-90/006F, December 1992.

Further details of this and other EPA work on ETS at:

<http://www.epa.gov/ncea/smoking.html>

<http://www.epa.gov/iaq/ets.html>

US Department of Health and Human Services. *The health consequences of involuntary smoking: a report of the Surgeon General*. Washington DC: US Government Printing Office, 1986. (DHSS Pub No. (PHS) 87-8398).

Background Papers

The background papers prepared for the consultation are available as a WHO Technical Document WHO/NCD/TFI/99.11 or can be found on WHO's Tobacco Free Initiative website:

<http://www.who.int/toh>

Adverse Health Effects

Synthesis: The Health Effects of Tobacco Smoke Exposure on Children. J. Samet, Department of Epidemiology School of Hygiene and Public Health Johns Hopkins University, Baltimore, MD, USA

Association of *in utero* or Postnatal Environmental Tobacco Smoke Exposure and Neurodevelopmental and Behavioral Problems in Children. B. Eskenazi, School of Public Health, University of California, Berkeley; R. Castorina, School of Public Health, University of California, Berkeley.

Effects of Maternal and Paternal Smoking on Children's Respiratory Health. D. Cook, Department of Public Health Sciences St. Georges Hospital Medical School, London and D. Strachan.

Effects of Passive Smoking on the Cardiovascular System in Children and Adolescents. S.S. Gidding, Pediatrics and Preventive Medicine, Northwestern University Medical School; Attending Cardiologist, Children's Memorial Hospital, Chicago, IL.

Parental Tobacco Smoke and Childhood Cancer. P. Boffetta, International Agency for Research, Lyon, France; J. Trédaniel, Saint Louis Hospital, Paris, France; A. Greco, University of Lyon, France.

Prenatal Exposure to Environmental Tobacco Smoke and Fetal Health. G.C. Windham, Reproductive Epidemiology Section, Division of Environmental and Occupational Disease Control, Department of Health Services, Oakland, CA.

Smoking and Sudden Infant Death Syndrome. E.A Mitchell, University of Auckland, Auckland, New Zealand; Milerad, Department of Women and Child Health, A. Lindgren, Children Hospital at Karolinska Institute, Stockholm, Sweden

Exposure to ETS and Public Health Impact

Children's Exposure to Passive Smoking: Survey Methodology and Monitoring Trends. M.J Jarvis, Health Behaviour Unit, Department of Epidemiology and Public Health, University College, London.

Magnitude of Smoking Attributable Costs. K. Adams, Rollins School of Public Health, Emory University, Atlanta, GA.

Properties of Environmental Tobacco Smoke. W. Rickert, President, Labstat Inc., Kitchener, Ontario, Canada.

Intervention Strategies

Environmental Tobacco Smoke Public Policies and Interventions: Using Communication and Outreach to Reduce Childhood Exposure to ETS. W. Long, Environmental Protection Specialists, Office of Air and Radiation, US Environmental Protection Agency, Washington, D.C.

Risk Communication, Children's Health, and Environmental Tobacco Smoke. V.T. Covello, Director, Center for Risk Communication, New York, New York

Risk Perception and Communication: Environmental Tobacco Smoke and Child Health. W. Leiss, Eco-Research Chair in Environmental Policy, School of Policy Studies, Queen's University, Kingston, Ontario, Canada.

Theories of Behavior Change in Relation to Environmental Tobacco Smoke Control to Protect Children. R. Borland, Centre for Behavioural Research in Cancer, Anti Cancer Council of Victoria, Victoria, Australia.

VIII. Annex 1

Extracts of the 1997 Declaration of the Environment Leaders of the Eight on Children's Environmental Health

Miami, May 1997

“Environmental Tobacco Smoke: Children exposed to environmental tobacco smoke are more likely to suffer from reduced lung function, lower respiratory tract infections and respiratory irritations. Asthmatic children are especially at risk. Many of these symptoms lead to increased hospitalizations of children.

We affirm that environmental tobacco smoke is a significant public health risk to young children and that parents need to know about the risks of smoking in the home around their young children. We agree to cooperate on education and public awareness efforts aimed at reducing children’s exposure to environmental tobacco smoke.”

Implementation Actions on Protecting Children’s Health and Environment which the Environment Leaders of the Eight have agreed to promote within their governments and countries:

“Environmental Tobacco Smoke: Convene a scientific conference, through WHO or another appropriate scientific organization, to synthesize and share the latest scientific information on risks to infants and children from environmental tobacco smoke and compile information on the most effective education strategies concerning exposure to children.”

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