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# Global estimate of the burden of disease from second-hand smoke



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## **List of abbreviations**

CDC	Centres for Disease Control and Prevention
CI	Confidence interval
Cal-EPA	Californian Environmental Protection Agency
DALY	Disability-adjusted life years
ECRHS	European Community Respiratory Health Survey
GYTS	Global Youth Tobacco Survey
GBD	Global burden of disease
IARC	International Agency for Research on Cancer
IHD	Ischaemic heart disease
LRI	Lower respiratory infection
OR	Odds ratio
PAF	Population attributable fraction
RR	Relative risk
SHS	Second-hand smoke
TFI	Tobacco Free Initiative
WHO	World Health Organization



## **Executive summary**

This document presents an estimation of the global burden of disease from exposure to second-hand smoke (SHS). The evidence on exposure-risk relationships is mainly drawn from recent reviews and meta-analyses. Exposure is estimated from major surveys and other published reports on both children and adults. Missing exposure information has been modelled on the basis of active smoking prevalence. Estimates of the burden of disease are calculated by age group, gender and country population, and results are also presented for 14 regions, children and adults, and by gender. Uncertainties are discussed and a sensitivity analysis has been performed.

Second-hand smoke is estimated to have caused about 603 000 premature deaths in 2004. These include 166 000 deaths from lower respiratory infections and 1100 from asthma in children, and 35 800 deaths from asthma, 21 000 deaths from lung cancer and 379 000 deaths from ischaemic heart disease (IHD) in adults. This disease burden amounts in total to about 10.9 million disability-adjusted life years (DALYs). Of all deaths attributable to SHS, 28% occur in children, and 47% in women.

The main sources of uncertainty include the estimation of exposure in the presence of incomplete data, the global use of a common indicator for received dose, the exposure-risk relationship – in particular its application to different parts of the world with different smoking, behavioural and housing patterns, and the burden of disease in non-smokers. Additional information that has been considered in this study includes biomarker studies, daily number of cigarettes smoked per smoker in each region, number of persons smoking per household, and the findings of epidemiological studies performed in developing countries. We have examined the possible impacts of variations in the critical parameters in the global burden of disease (GBD) analysis, and conclude that the presented estimate is reasonably robust.



## **1 Introduction**

The relationship between exposure to second-hand smoke (SHS) and a variety of health endpoints in children and adults has been examined extensively in the epidemiological and experimental literature. Several authoritative reviews and reports have been published by national and international organizations as well as researchers within the field (WHO 1999; Jaakkola and Jaakkola 2002a and 2002b; IARC 2004; Cal-EPA 2005; US Surgeon General 2006). The general method for estimations of the burden of disease attributable to SHS is outlined in a previous report (Öberg et al. 2010), summarizing the most recent evidence base for linking SHS with a variety of health effects among both adults and children. The report also includes a list of health effects with a clear causal relationship to SHS exposure and recommended that these be used for burden of disease assessments. The present report compiles available data on exposure to SHS and applies the previously suggested method to estimate the global burden of disease (GBD) attributable to SHS by disease, age, sex and region (for 14 WHO subregions: see Annex, Table A2 country grouping into WHO subregions).

### **1.2 Defining second-hand smoke**

Second-hand smoke, for which the term environmental tobacco smoke has also been used in the literature, is formed from the sidestream smoke emitted into the environment from the smoldering of cigarettes and other tobacco products between puffs and from the mainstream smoke exhaled by the smoker. The terms “passive smoking” or “involuntary smoking” are also often used to describe the exposure to SHS. However, these suggest that while involuntary or passive smoking is not acceptable, voluntary or active smoking is acceptable. In this document we use the term recommended by the Tobacco Free Initiative, i.e. SHS.

### **1.3 Properties of second-hand smoke**

Smoke that is emitted into the environment from the smouldering cigarette between puffs (sidestream smoke) is the principal contributor to SHS. Other components of SHS are exhaled mainstream smoke, and (mainstream) smoke emitted at the mouthpiece during puff drawing.

Cigarette smoking is the main source for SHS exposure because it is the most prevalent form of tobacco smoking although specific patterns differ between countries. Tobacco smoke contains thousands of chemicals that are released during burning as gases, vapours and particles. The US National Toxicology Program estimates that at least 250 chemicals in SHS are known to be toxic or carcinogenic (US Surgeon General 2006). Several individual compounds found in tobacco smoke have also been listed as developmental or reproductive toxicants under California’s Proposition 65 (e.g. carbon monoxide, lead and nicotine). However, the health effects of exposure to SHS cannot be estimated from any individual constituents, as SHS is a complex mixture and exposure to this mixture may be more relevant to health effects than any of the individual substances alone. Thus, the exposure-response relationships are generally assessed by epidemiological studies using exposure to SHS as a whole.

### **1.4 Current and ex-smokers**

Numerous studies have concluded that exposure to SHS is harmful to never-smokers but effects on ex-smokers and current smokers have not been studied extensively. Current and ex-smokers are excluded from most studies on the health effects of SHS, not because they are considered as not at risk from exposure to SHS, but to avoid confusion with active smoking in assessing the effects of SHS. In fact, smokers may be especially susceptible to the adverse effects of SHS, because their exposure levels are high due to their close

proximity to the source of SHS exposure (i.e. their cigarette), or because they already have smoking-caused diseases, either clinically or pre-clinically. A few studies have documented associations between SHS and adverse health effects in active smokers (e.g. Lam et al. 2005)

The question of whether current and ex-smokers should be included in environmental burden of disease (EBD) calculations has previously been examined (Öberg et al. 2010). In this report, we exclude current and ex-smokers from the sensitive population at risk, emphasize the paucity of relevant data on the effect of SHS in smokers, and include only non-smokers in the burden of disease estimates. This is a similar approach to that taken by most national assessors and will most likely lead to an underestimation of the health impact. As described below, we recommend that the calculation be carried out by an indirect method, i.e. by subtracting the burden of disease related to active smoking from the total burden of disease, and then excluding current smokers from the population at risk.

## 2 Methods

### 2.1 Summary of the method used to estimate the environmental burden of disease at global level

The method proposed in the previous report (Öberg et al. 2010) is summarized by the main following steps.

- **Study population.** The study population for global assessment includes all non-smokers worldwide, grouped into 14 subregions. The selected outcomes are specific to three age groups and vary between young children ( $\leq 4$  years), children ( $\leq 14$  years) and adults ( $\geq 15$  years).
- **Selection of diseases.** In this estimation, we only include outcomes with sufficient evidence of a causal relation to SHS exposure and for which effects may be quantified with available health statistics. We have restricted this report to outcomes for which there is a broad consensus on a causal relation to SHS, which are extensively studied in many populations using a variety of valid study methods, for which positive findings have been reported quite consistently and for which there are plausible mechanisms. The relative risk (RR) is used as recommended in the previous report (Öberg et al. 2010). The following six outcomes are included:
  - acute lower respiratory infections for children  $\leq 4$  years
  - acute otitis media for children  $\leq 3$  years
  - asthma (induction and exacerbation) for children  $\leq 14$  years
  - asthma (induction) for adults  $\geq 15$  years
  - lung cancer for adults  $\geq 15$  years
  - ischaemic heart disease for adults  $\geq 15$  years.
- **Health statistics.** Health statistics data for the outcomes of interest are extracted from the WHO global database on burden of disease by country (WHO 2007). The health statistics that will be used include deaths and disability-adjusted life years (DALYs). Disability-adjusted life years are the sum of the years of life lost (YLL) due to premature mortality and the years of healthy life lost due to disability (YLD) from the incident cases of the health condition (Murray CJL et al. 2002).

$$\text{DALY} = \text{YLL} + \text{YLD}$$

- **Assessing prevalence of exposure.** The estimated prevalence of exposure to SHS in the early years of the 21st century for each country was based on published data, or modelled mainly according to the proportion of children with at least one smoking parent. Published data of SHS included national and multinational surveys such as the Global Youth Tobacco Survey (GYTS). For countries with no available national data on parental smoking, male and female smoking prevalence reported in the WHO Mpower report (WHO 2008a), the WHO European Database on Tobacco Control (WHO 2009a), WHO Global Infobase (WHO 2009b), and the American Cancer Society (Shafey et al. 2003) were used to model exposure to SHS.

The percentage of men and women reporting regular exposure (most days or nights) to SHS at any site was obtained from national and multinational surveys such as the European Community Respiratory Health Survey (ECRHS). For countries with no available data on exposure to SHS among adults, a model based on available data and male smoking prevalence from the WHO Global Infobase was used to estimate exposure to SHS. When calculating the attributable fraction for lung cancer, which has a long latency period between exposure and detectable disease, exposure data from around 1990 were used. Otherwise, burden of disease calculations were based on the most recent smoking data available.

- **Calculating the population attributable fraction.** The following formula was used for estimating the population attributable fraction (PAF) for each health outcome:

$$PAF = \frac{p(RR - 1)}{p(RR + 1) + 1}$$

where p is the proportion of the population in the specified age group exposed to SHS and RR is relative risk for outcome and population group (Levin 1953).

- **Calculating attributable burden.** The attributable burden for every outcome and country was calculated by multiplying the population attributable fraction with the disease statistics (deaths or DALYs) for the total burden among the study population. Adult asthma, lung cancer and ischaemic heart disease are strongly related to active smoking. Therefore, the burden of disease among non-smokers (ns) was calculated as:

$$B_{(ns)} = (B - (B \times PAF_{sm})) \times (1 - p_s)$$

where B is the total burden of disease among both smokers and non-smokers,  $PAF_{sm}$  is the population attributable fraction related to active smoking, and  $p_s$  is the smoking prevalence.

The burden attributable to SHS exposure among non-smokers was estimated as:

$$\text{Attributable burden} = PAF_{SHS} \times B_{ns}$$

Estimates were developed by country, and separately for children, men and women. The regional burden of disease was calculated by summing up all national attributable deaths and DALYs in the specified region.

## **2.2 Exposure assessment**

Exposure assessment is essential to perform any estimation of health impacts. Several kinds of assessment techniques have been used for assessing SHS exposure, including personal questionnaires, measurement of indoor air concentrations of SHS constituents, or personal monitors and biomarkers in saliva, urine, blood and hair (Jaakkola and Jaakkola 1997; Jaakkola and Samet 1999). Epidemiological studies generally rely on questionnaire measures.

### ***Time period of exposure***

Depending on the outcome, it may be important to estimate both the past and present prevalence of SHS exposure. For outcomes that undergo several phases of development, there may be a considerable lag between exposure and effect (Jaakkola and Samet 1999). Based on what is known for active smoking, it has been estimated that the latency period for lung cancer is between 10 and 20 years. The interval between exposure to SHS and cardiovascular effects is much shorter, with some changes occurring more or less immediately (e.g. platelet activity) while other, longer term effects may not be evident for one to five years. In this report, we use data from 1980 to 1995 (1992 as average) on SHS and smoking prevalence for the assessment related to lung cancer. For the remaining health outcomes, we assumed a constant exposure to SHS over time and used estimates of recent exposure prevalence, i.e. since the year 2000.

### ***Data sources and search strategy***

Data on SHS exposure are available from two main types of sources: (a) the GYTS; and (b) various national and multinational surveys. For the identification of national SHS data, the following search strategy was applied: a combination of key words including “second hand smoke”, “environmental tobacco smoke” and “passive smoking” combined with names of Member States or regions (e.g. “Africa”) were searched in PubMed. This was complemented with similar search terms in Google’s search engine. The results are summarized in the Annex.

### ***Countries not covered by surveys***

For those countries with no identified national data on SHS exposure, we developed models for exposure estimations, mainly based on active smoking rates (see below for model details). For the remaining countries without any data related to active smoking, we used the regional average to estimate exposure.

### ***SHS exposure among children***

#### ***Available exposure surveys***

The WHO and the US Centers for Disease Control and Prevention (CDC) developed the GYTS (CDC and WHO 2008) to measure tobacco use among young people (13–15 years) across countries using a common methodology and core questionnaire. Between 1999 and 2007, a total of 146 countries representing all WHO regions participated in the GYTS, constituting the main SHS data source for children and presenting an important coverage globally. The survey asked about children’s exposure to SHS in their home or in other places during the last seven days, and if they had one or more parent who smoked. In this study, exposure is defined as answering yes to the second question. In most countries, there is a similar or higher prevalence of exposure from the first question regarding any exposure to SHS. The definition used here (i.e. parental smoking) corresponds relatively well to the ones usually used in studies on health effects. In addition to the GYTS database, national exposure data compiled according to the described search strategy were also used.

In 140 of the 192 countries, representing 90% of all children globally, children were assessed by survey data on parental smoking (Table 1). Of these, data from 13 countries (mainly from western Europe) were from sources other than GYTS.

**Table 1 Proportion of countries and children in countries covered by surveys on parental smoking, by region**

Region	Coverage	
	Countries (no./total)	Children (%)
Africa	27/46	75
Americas	33/35	99
Eastern Mediterranean	21/21	100
Europe	36/52	59
South-East Asia	9/11	99
Western Pacific	14/27	92
Global	140/192	90

In countries with several sub-national GYTS surveys, the mean value was calculated. Where repeated surveys have been performed, the most current was used for further calculations. For a few countries, only one sub-national survey was reported, which was, however, considered to be nationally representative in the absence of other data. Countries with no data on parental smoking were excluded from the GYTS-dataset.

#### *Modelling SHS exposure for countries without SHS survey data*

While data on children’s SHS exposure were available for 140 countries, exposure in 52 other countries was poorly documented. However, where data were missing, it was possible to estimate values using the correlation between adult smoking, parental smoking and possibly other country characteristics. The most comprehensive and comparative information on active smoking prevalence among adults is summarized in the WHO Mpower report (WHO 2008a). It provides national data on male and female smoking prevalence for the vast majority of countries.

Several approaches were explored to model children’s exposure. By region (and sub-region, if different patterns were observed), correlations were explored with (a) maternal smoking; (b) paternal smoking; and (c) parental smoking. Any “parental smoking” was calculated by subtracting the likely proportion of smoking population living together according to the following equation:

$$\text{Any parental smoking} = Ms + Fs - (Ms \times Fs)$$

where Ms and Fs are the male and female smoking prevalence, respectively. This model has also recently been suggested and evaluated for three European countries (Patja et al. 2009).

Parental smoking generally correlated better with children’s exposure to SHS than maternal or paternal smoking, and linear correlation was most often better than power or logarithmic correlation.

For each subregion requiring modelling of missing data of children's SHS exposure, the model with the best correlation was developed. In addition to maternal, paternal and parental smoking, the percentage of urban population of a country (UN population statistics), and its gross national income (GNI, World Bank) were inserted into the models, and the parameter was maintained in the model if the correlation improved. The model only improved in Africa when using the percentage of the urban population in the regression model. The percentage of urban population in particular, combined with parental smoking, correlated relatively well in the African regions. The resulting models used are summarized in Table 2. Available data and models are represented in Figure 1.

**Table 2 Models used for estimating children's SHS exposure**

Region	Survey data	Model <sup>a</sup>
<i>Africa (Afr)</i>	<i>27/46 countries covered by survey data.</i>	<i>% exposed to SHS = 0.816 + 0.786 × parental smoking + 0.200 × % urban population; R2=0.63.</i>
<i>Americas (Amr)</i>	<i>A: entirely covered by survey data; B: 31/33 countries covered by survey data.</i>	<i>A: no model needed; B, D: the only two countries not covered also lack active smoking data in the coherent dataset used<sup>b</sup>, therefore, the sub-regional average is applied instead of modelling.</i>
<i>Eastern Mediterranean (Emr)</i>	<i>All countries covered by survey data</i>	<i>No model needed</i>
<i>Europe (Eur)</i>	<i>A: 15/27 countries covered by survey data; B: 12/16 countries covered by survey data; C: 9/9 countries covered by survey data.</i>	<i>A<sup>b</sup>: % exposed to SHS = 25.3 + 0.547 × parental smoking; R2=0.54; Eur B: % exposed to SHS = 10.4 + 1.28 × parental smoking; R2=0.60; Eur C: No model needed.</i>
<i>South-East Asia (Sear)</i>	<i>9/11 countries covered by survey data.</i>	<i>The only two countries not covered also lack active smoking data in the coherent dataset used<sup>c</sup>, therefore the sub-regional average is applied instead of modelling.</i>
<i>Western Pacific (Wpr)</i>	<i>A: no countries covered by survey data; B: 14/27 countries covered by survey data.</i>	<i>A: model from all A-country data (Americas A, Europe A) used; % exposed to SHS = - 120.0 + 43.66 × Ln (parental smoking); R2=0.50; B: no model with correlation above R2=0.40 was found, therefore the subregional average is applied.</i>

<sup>a</sup> Models are only used for countries without available survey data; for countries without data on active smoking the subregional average of exposure to SHS is being used instead of modelling.

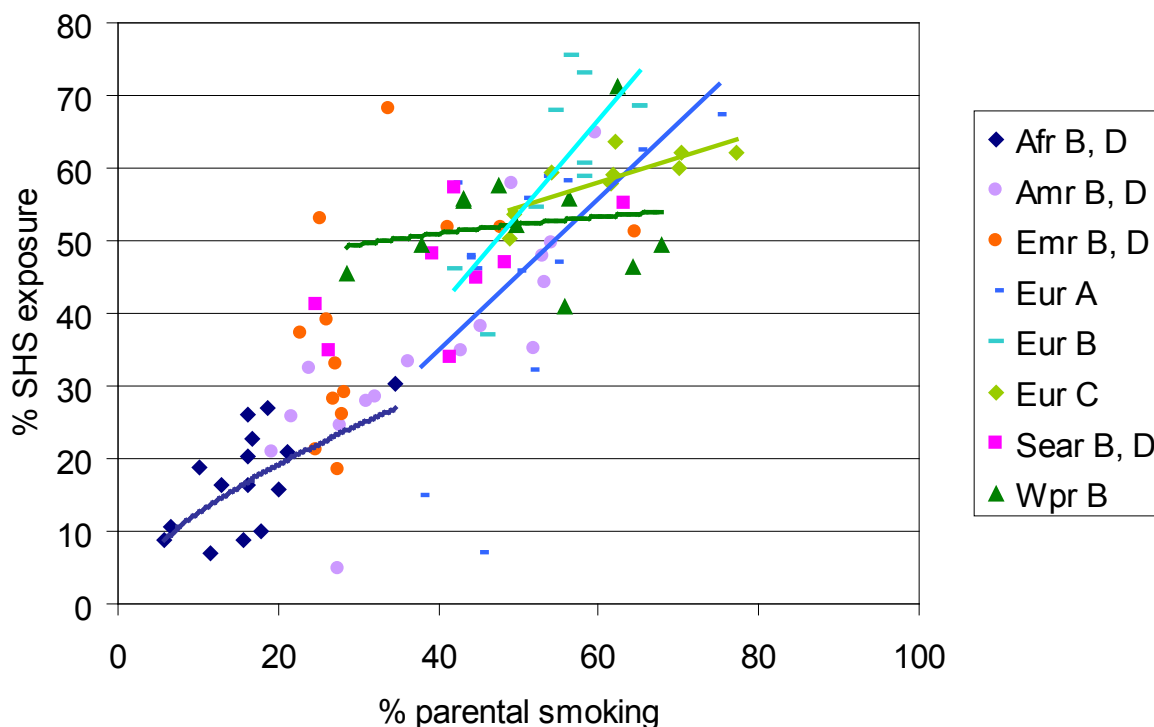
<sup>b</sup> Without outliers Finland, Norway, Sweden.

<sup>c</sup> WHO Report on the Global Tobacco Epidemic, 2008 (WHO 2008a).



When no model with a correlation higher than  $R^2=0.50$  could be found, or no active smoking data were available from the coherent database used (WHO 2008a), the sub-regional average values were used. However, this concerned either very few countries within one region (e.g. in the Americas, South-East Asia), or relatively small states (e.g. in the Western Pacific).

Figure 1 Children's SHS exposure and parental smoking, models used and survey data



Lines represent models used; points represent survey data from GYTS.

Afr, Africa; Amr, Americas; Emr, Eastern Mediterranean; Eur, Europe; Sear, South-East Asia; Wpr, Western Pacific.

#### Estimated SHS exposure among children

The global average of children with at least one smoking parent (according to the definition from GYTS) is estimated to be 41% (Table 3). The highest level, almost 68%, is found in the Western Pacific Region. The lowest exposure is estimated in Africa, with about 13% of children living in families with at least one smoking parent. The estimated exposure among children by country is shown in Annex, Table A2 with sources of data given.

**Table 3 Proportion of children under 15 years with one or more parent who smokes (based on survey data and modelling)**

Subregion	SHS exposure (%)
<i>AfrD</i>	13
<i>AfrE</i>	13
<i>AmrA</i>	25
<i>AmrB</i>	29
<i>AmrD</i>	22
<i>EmrB</i>	37
<i>EmrD</i>	34
<i>EurA</i>	51
<i>EurB</i>	61
<i>EurC</i>	61
<i>SearB</i>	53
<i>SearD</i>	36
<i>WprA</i>	51
<i>WprB</i>	68
<i>Global</i>	41

WHO subregional country grouping in Annex, Table A2.

### **Exposure among adults**

#### *Available exposure surveys*

In contrast to children, we found no international databases that document SHS exposure among adults. However, several national reports have been published. In addition, the European Community Respiratory Health Survey covers 14 countries (Janson et al. 2001; Janson et al. 2006). The first survey (ECRHS I) was conducted in 1990–1994 and included young adults (aged 20–44 years). The investigated subjects were followed up in a second survey approximately nine years later (ECRHS II). The survey collected data on exposure to SHS among population cohorts from a large number of countries. The design has been described in detail (Burney et al. 1994; ECRHS 2002). Participants were asked if they had regularly (most days or nights) been exposed to tobacco smoke in the last 12 months. If so, they were classified as being exposed to SHS. In addition, SHS exposure during pregnancy was estimated in a survey of women in nine developing countries, who were asked how often they were indoors and around people who were smoking (Bloch et al. 2008). Women who responded “frequently” or “always” were classified as exposed.

In total, we were able to identify some national data on SHS exposure from 21 and 29 of 192 countries, for men and women, respectively (see Annex, Table A1 for detailed information). The coverage of adults’ SHS exposure by survey data reaches 41% and 63%, for men and women respectively, on a global scale (Table 4). Active smoking data of around 1990 were used to estimate the number of lung cancers attributable to SHS.

Table 4 Proportion of adults in countries covered by surveys on exposure to SHS, by region

Region	Countries no./ total women	Coverage adult women (%)	Countries no./ total men	Coverage adult men (%)
Africa	2/46	9	0/46	0
Americas	7/35	69	4/35	66
Eastern Mediterranean	1/21	28	0/21	0
Europe	13/52	41	13/52	42
South-East Asia	2/11	79	1/11	14
Western Pacific	4/27	86	3/27	78
<b>Global</b>	<b>29/192</b>	<b>63</b>	<b>21/192</b>	<b>41</b>

#### Modelling SHS exposure in countries without SHS survey data

As for children, it is likely that SHS exposure is related to the prevalence of active smoking (for which data are available in most countries). In many countries, male smoking is the largest source of SHS exposure. Especially in developing countries, smoking is quite uncommon among females. Therefore, male smoking is likely to be a relatively good proxy of SHS exposure. In countries with relatively low smoking prevalence, there is also generally a lower exposure to SHS exposure.

Female, male and combined smoking were compared in linear, power and exponential models, with data points representing country data. The best correlation for countries with available data on active smoking and adult SHS data was shown for power models ( $y = a \times x^b$ ) in both developed and developing countries, and male smoking. Scandinavian countries were again considered as outliers, and were not considered in model development. The models with the best correlation were the following:

$$\text{SHS}_{(\text{women, developing countries})} = 0.612 \times (\text{male smoking prevalence})^{1.10}$$

( $R^2 = 0.69$ )

$$\text{SHS}_{(\text{men developing countries})} = \text{data not sufficient for modelling}$$

$$\text{SHS}_{(\text{men, developed countries})} = 0.0275 \times (\text{male smoking prevalence})^{2.00}$$

( $R^2 = 0.50$ )

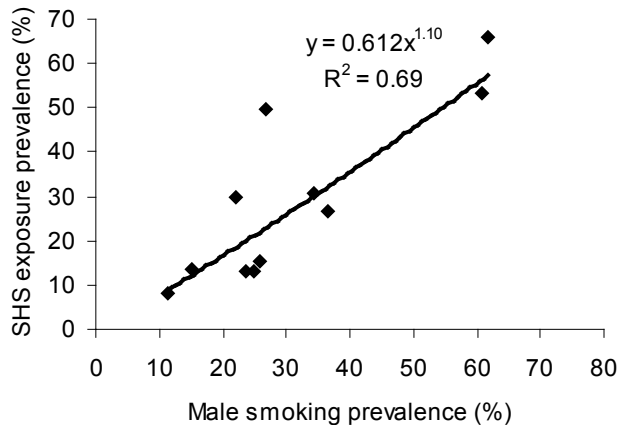
$$\text{SHS}_{(\text{women, developed countries})} = 0.0019 \times (\text{male smoking prevalence})^{2.72}$$

( $R^2 = 0.68$ )

The models were capped at the value of 68% of adults in a country exposed to SHS, as that had been the highest exposure observed.

In the absence of sufficient data for modelling men's exposure to SHS in developing countries, the men's model for developed countries was chosen. This approach results in lower estimates of exposure, as compared to using the women's model for developing countries.

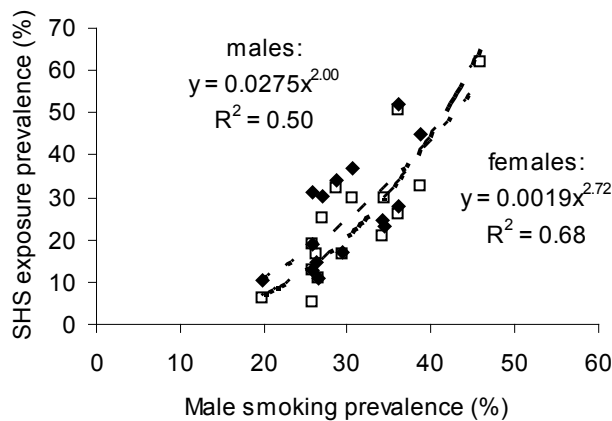
Figure 2 Women's SHS exposure and male active smoking in developing countries



Diamonds: individual country data.

—, model.

Figure 3 SHS exposure and male active smoking in developed countries



Diamonds, individual country data for males; squares: individual country data for females.

- - -, model for males; —, model for females.

The correlation between existing survey data for SHS adult exposure and the modelled data is moderately strong ( $R^2 = 0.50, 0.68$  and  $0.69$ ).

Overall, the model seems to predict adult SHS exposure relatively well, and survey data cover half of the world's adult population. However, the African, Eastern Mediterranean, and South-East Asian regions are quite poorly covered by survey data, especially male SHS exposure (Table 5).

*Estimated SHS exposure among adults*

Globally, it was estimated that about one third of adults are exposed to SHS, in terms of having regularly been exposed to tobacco smoke. There were relatively small differences globally between men and women (Table 5), but certain regions showed discrepancies. The highest exposure was estimated in European region C with 66% the population exposed. The lowest regional exposure was estimated in the African region (4–11%). The estimated exposure among men and women by country is shown in Table A2 of the Annex, which indicates sources of data.

**Table 5** *The proportion of non-smoking adults exposed regularly to SHS, by WHO region, based on survey data and modelling*

Subregion	SHS exposure men (%)	SHS exposure women (%)
<i>AfrD</i>	7	11
<i>AfrE</i>	4	9
<i>AmrA</i>	16	15
<i>AmrB</i>	14	22
<i>AmrD</i>	15	19
<i>EmrB</i>	24	25
<i>EmrD</i>	21	35
<i>EurA</i>	35	32
<i>EurB</i>	52	54
<i>EurC</i>	66	66
<i>SearB</i>	32	56
<i>SearD</i>	23	19
<i>WprA</i>	50	54
<i>WprB</i>	53	51
<b>Global</b>	<b>33</b>	<b>35</b>

See Annex, Table A2 for country grouping into WHO subregions.

### 2.3 Health effects selected for the global estimate of burden of disease

Exposure to SHS has been shown to be associated with many adverse health effects among children and adults. Evidence on various health outcomes related to SHS is constantly being updated. In this report, we used the previously suggested core set of outcomes selected based on there being “sufficient” evidence of a causal relation between SHS exposure and the health condition, and sufficient data for quantification of health impacts (Öberg et al. 2010). It includes outcomes for which there is a broad consensus on the existence of a causal relationship with SHS exposure, which are extensively studied in many populations using a variety of valid study methods. In general, these studies report consistently positive findings, for which there are plausible mechanisms, and quantitative information (in terms of relative risk).

The strongest evidence exists in adults for lung cancer, ischaemic heart disease, and asthma (new cases), and in children for low birth weight, childhood chronic respiratory symptoms, lower respiratory illness in young children, asthma (new cases and exacerbation), middle-ear effusion and infection in young children, reduced pulmonary function among children and sudden infant death syndrome (SIDS) (Öberg et al. 2010). In addition, health statistics on the selected outcomes are needed to calculate the attributable burden. Based on this criterion and the recommendations of the previous methods guide, we decided to estimate the burden of disease for six outcomes: lower respiratory infections and acute otitis media in young children; child asthma; adult asthma; lung cancer; and ischaemic heart disease. Disease statistics on SIDS were not available globally, and estimates for this outcome are, therefore, not included.

A summary of outcomes for quantification with their respective evidence levels is provided in tables 6 (children) and 7 (adults). A thorough description of the available evidence underlying each outcome and the selection of relevant risk ratios is described in more detail in the following sections and in the guide describing the applied methods in more detail (Öberg et al. 2010).

**Table 6 Recommended risk estimates and summary of conclusions of recent reviews of health effects of exposure to SHS in children<sup>a</sup>**

Outcomes in children	Description	Age (years)	Recommended		Reference	Level	Agency <sup>b</sup>		
			RR/OR (95% CI)	Exposure			A	B	C
<b>Developmental effects</b>									
<i>Low birth weight</i>	<i>Prevalence of LBW (&lt;2500 g) at term</i>	0	1.38 (1.13–1.69)	<i>Non-smoking mother; any exposure at work or at home</i>	<i>Windham et al. 1999</i>	1	***	***	***
<i>Pre-term delivery</i>	<i>Incidence of births before gestation week 37</i>	0	1.57 (1.35–1.84)	<i>Non-smoking mother; any exposure at work or at home</i>	<i>Cal-EPA 2005</i>	2	<i>n</i>	***	**
<i>Sudden infant death syndrome</i>	<i>Incidence of SIDS</i>	<1	1.94 (1.55–2.43)	<i>Smoking mother<sup>c</sup></i>	<i>Anderson &amp; Cook 1997</i>	1	**	***	***
<i>Spontaneous abortion / perinatal death</i>	–	–	–	–	–	3	<i>n</i>	**	*
<i>Congenital malformation</i>	–	–	–	–	–	3	<i>n</i>	*	*
<i>Neuropsychological development</i>	–	–	–	–	–	3	**	**	<i>n</i>
<i>Physical development</i>	–	–	–	–	–	3	<i>n</i>	*	*

## Respiratory effects

<i>Lower respiratory infection (LRI)</i>	<i>Incidence of acute lower respiratory infection (ALRI) and hospitalizations</i>	<2	1.55 (1.42–1.69)	<i>Either parent</i>	<i>United States Surgeon General 2006</i>	1	***	***	***
<i>Decreased pulmonary function</i>	–	<14	–	–	–	1	<i>n</i>	**	***
<i>Chronic respiratory symptoms:</i>									
<i>- wheeze</i>	<i>Prevalence of chronic wheeze</i>	<14	1.26 (1.20–1.33)	<i>Either parent</i>	<i>United States Surgeon General 2006</i>	1	***	***	***
<i>- cough</i>	<i>Prevalence of chronic cough</i>	<14	1.35 (1.27–1.43)	<i>Either parent</i>	<i>United States Surgeon General 2006</i>	1	***	***	***
<i>Otitis media:</i>									
<i>- acute</i>	<i>Incidence of acute otitis media (excluding studies on specifically recurrent otitis)</i>	<8	<i>No reliable summary estimate available</i>	<i>Either parent</i>	<i>United States Surgeon General 2006</i>	1	–	–	–
<i>- acute and recurrent</i>	<i>Incidence of any acute otitis media</i>	<4	1.66 (1.33–2.06)	<i>Either parent</i>	<i>Uhari et al. 1996</i>	1	***	***	***
<i>- recurrent</i>	<i>Incidence of recurrent otitis media</i>	<4	1.32 (1.14–1.52)	<i>Either parent</i>	<i>United States Surgeon General 2006</i>	1	–	–	***
<i>- serous</i>	<i>Incidence of middle ear effusions</i>	<4	1.33 (1.12–1.58)	<i>Either parent</i>	<i>United States Surgeon General 2006</i>	1	–	***	–
<i>Asthma:</i>									
<i>- onset</i>	<i>Incidence of new cases of asthma</i>	<14	1.32 (1.24–1.41)	<i>Either parent</i>	<i>Cal-EPA 2005</i>	1	<i>n</i>	***	**
<i>- exacerbation</i>	<i>Asthma symptoms, hospitalizations, etc.</i>	–	–	–	–	1	***	***	***
<i>Cancer</i>									
<i>Childhood cancers<sup>e</sup></i>	–	–	–	–	–	3	**	**	**

–, not available or not relevant in view of quantification; CI, confidence interval; OR, odds ratio; RR, relative risk.

<sup>a</sup> Shaded fields represent disease outcomes proposed for quantification.

<sup>b</sup> A = WHO (1999); B = Cal-EPA (2005); C = United States Surgeon General (2006). The asterisks represent the conclusion in the report regarding level of evidence, where \* = the evidence of causality is concluded to be “inconclusive”, “little”, “unclear” or “inadequate”; \*\* = the evidence of causality is concluded to be “suggestive”, “some” or “may contribute”; \*\*\* = the evidence of causality is concluded to be “sufficient” or “supportive”; and n = not evaluated in the report.

<sup>c</sup> Value is based on maternal smoking.

<sup>d</sup> Based on the Cal-EPA (1997; 2005) reviews and diagnostic difficulties of acute otitis; the risk estimate of Etzel et al. (1992) of 1.38 (1.21–1.56) can be used for an interim estimate, which corresponds to the mid-point of other reviews (see discussion in relevant section below).

<sup>e</sup> IARC (2004) makes a distinction between all sites (\*\*) and individual sites (\*).



Table 7 Recommended risk estimates and summary of conclusions of recent reviews of health effects of exposure to SHS in adult non-smokers<sup>a</sup>

Outcomes in adults	Description	Age (years)	Recommended RR/OR (95% CI)	Exposure	Reference	Level	Agency <sup>b</sup>		
							B	C	D
<b>Reproductive effects</b>									
Female fertility	-	-	-	-	-	3	**	*	n
Other female reproductive toxicity (e.g. menopausal age)	-	-	-	-	-	3	**	*	n
Male reproductive toxicity	-	-	-	-	-	3	*	*	n
<b>Respiratory effects</b>									
<b>Asthma:</b>									
- induction	Adult-onset incident asthma	>20	1.97 (1.19–3.25)	At home and/or at work	Jaakkola et al. 2003	1	***	**	n
- exacerbation	Asthma symptoms, hospitalizations, etc.	-	-	-	-	2	***	**	n
Chronic obstructive pulmonary disease (COPD)	Prevalence of COPD	-	1.55 (1.09–2.21)	At home	Eisner et al. 2005	2	*	**	n
Acute irritant symptoms and effects	-	-	-	-	-	1	***	***	n
Chronic respiratory symptoms:	-	-	-	-	-	-	**	**	n
- wheeze	Prevalence	>18	1.99 (1.41–2.82)	At home and/or at work	Leuenberger et al. 1994	2	-	**	n
- phlegm	Prevalence	>18	1.69 (1.23–2.31)	At home and/or at work	Leuenberger et al. 1994	2	-	-	n
- dyspnoea	Prevalence	>18	1.44 (1.18–1.75)	At home and/or at work	Leuenberger et al. 1994	2	-	-	n
Pulmonary function	-	-	-	-	-	3	**	**	n
<b>Cancer</b>									
Cancer (all cancer):	-	-	-	-	-	3	**	n	*
- lung	Incidence	>15	1.21 (1.13–1.30)	At home	United States Surgeon General 2006	1	***	***	***
			1.22 (1.13–1.33)	At work					
- breast	Incidence of premenopausal cases	15–50	1.68 (1.31–2.15)	At home and/or at work	Johnson 2005	2	***	**	*
	Incidence of any breast cancer	>15	1.25 (1.08–1.44)	At home and/or at work	Cal-EPA 2005	3	*	**	*
- nasal sinus cavity	-	-	-	-	-	2	***	**	*
- nasopharyngeal	-	-	-	-	-	3	**	*	*
- cervical	-	-	-	-	-	3	**	*	*
- urinary tract/bladder	-	-	-	-	-	3	*	n	*
- stomach	-	-	-	-	-	3	*	n	*
- brain	-	-	-	-	-	3	*	n	*
- leukaemia	-	-	-	-	-	3	*	n	n
- lymphoma	-	-	-	-	-	3	*	n	n

Cardiovascular diseases									
<i>Ischaemic heart disease (IHD)</i>	<i>Incidence of any &gt;15 IHD</i>		1.27 (1.19–1.36)	<i>Non-smokers at home or at work</i>	<i>United States Surgeon General 2006</i>	1	***	***	<i>n</i>
<i>Stroke</i>	–	–	1.82 (1.34–2.49)	<i>Non-smokers at home or at work</i>	<i>Bonita et al. 1999</i>	2	**	**	<i>n</i>

–, not available or not relevant in view of quantification; CI, confidence interval; OR, odds ratio; RR, relative risk.

<sup>a</sup> Shaded fields represent disease outcomes proposed for quantification.

<sup>b</sup> B = Cal-EPA (2005); C = United States Surgeon General (2006); D = IARC (2004). The asterisks represent the conclusion in the report regarding level of evidence, where \* = the evidence of causality is concluded to be “inconclusive”, “little”, “unclear” or “inadequate”; \*\* = the evidence of causality is concluded to be “suggestive”, “some” or “may contribute”; \*\*\* = the evidence of causality is concluded to be “sufficient” or “supportive”; and n = not evaluated in the report.

## Effects on children

### Lower respiratory infection

Given the high incidence, lower respiratory infections (LRIs) have the potential to significantly contribute to the total burden of disease from SHS exposure, especially among children in developing countries. This group of conditions includes respiratory infections such as acute bronchitis, bronchiolitis, respiratory syncytial virus infections and pneumonia (Jaakkola and Jaakkola 2002a). These are all most common in very young children (<2 years), and for this age group, there is widespread agreement on the evidence of a causal relationship between SHS exposure and LRI. In this report, an odds ratio (OR) of 1.55 (1.42–1.69) obtained from the US Surgeon General’s Report (US Surgeon General 2006) was used to calculate the attributable burden due to smoking by either parent for the first two years of life (Öberg et al. 2010). An excess risk of about one third (OR=1.18) (Li et al. 1999) was used for the age group between two and five years.

Considering the uncertainties in the transferability of the indicator of exposure and corresponding risk ratios, alternative risk ratios are being applied in the sensitivity analysis.

### Otitis media

Acute otitis media occurs when there is an infection, typically bacterial, in the middle ear leading to pain, fever, and a potential for tympanic membrane perforation. Secondary complications may lead to sustained hearing loss, communication difficulties and educational impairment in children. The epidemiological data strongly support a causal relationship between SHS exposure in the home and middle-ear disease with acute otitis media, recurrent otitis media with effusion and serous otitis media (middle-ear effusion without acute infection), among children under two years of age.

While there is consensus that the evidence supports a causal relationship between SHS exposure and acute otitis media, there seems to be less consensus about the most likely risk value to use.

As most of the available disease statistics include otitis (without separating acute and recurrent cases), separate assessment of acute and recurrent otitis is not included in the estimation of disease burden. As previously suggested (Öberg et al. 2010), the risk rate of 1.38 (1.21–1.56) could be used as the estimate based on a high-quality study, which included prospective assessment and use of biomarkers for assessing SHS exposure (Etzet et al. 1992; Cal-EPA 2005). Alternatively, the OR of 1.66 (1.33–2.06) reported by Uhari

and colleagues for increased risk of acute or recurrent otitis media associated with one or more parents smoking could be used as the higher estimate (Uhari et al. 1996). The US Surgeon General's report (2006) presents a lower pooled OR of 0.99 (0.70–1.40) based on only three studies covering different age ranges, but nevertheless concludes that there is a causal relationship between SHS exposure and acute otitis media.

### *Asthma*

Asthma is a chronic inflammatory disease of lower airways that is characterized by reversible airways obstruction (i.e. narrowing), periodic attacks of wheezing, shortness of breath, and a feeling of tightness in the chest. Asthma is the most common chronic respiratory condition of childhood. It has been suggested that the onset of new cases as well as the exacerbation (additional episodes and increased severity in those who already have an underlying disease) of childhood asthma have increased substantially in recent years in many parts of the world.

There is a high level of agreement among scientists that SHS causes new cases of asthma in children who were previously free from this disease, as well as exacerbates the existing cases. Disease statistics use incident cases combined with severity of asthma to calculate DALYs. Thus, it has been recommended that the OR of 1.32 (1.24–1.41) for the onset of asthma estimated in the meta-analysis based on the most updated references (Cal-EPA 2005) be used for further calculations of the health burden (Öberg et al. 2010).

### ***Effects on adults***

#### *Asthma*

Asthma symptoms and obstructive changes in lung function typically occur in episodes in asthma, but the inflammation underlying asthma is chronic in nature. Asthmatic airway narrowing is a result of swelling of the lining of airways, tightening of the smooth muscle, and increased secretion of mucus in the airways, often resulting in difficulty in breathing.

There is consistent evidence that SHS exposure has a causal link with new cases of asthma in adults (Cal-EPA 2005; Gilmour et al. 2006; Jaakkola and Jaakkola 2006). Based on a limited number of references, the US Surgeon General concluded that the evidence is suggestive but not sufficient (2006), while the California Environmental Protection Agency (2005) included updated references covering studies published since 2000, and concluded that the evidence supports a causal relationship between SHS and the onset of adult asthma.

The strongest evidence on SHS exposure and onset of adult asthma comes from the Finnish Environment and Asthma Study, which was a population-based incident case-control study that corresponded to a follow-up of approximately 100 000 adults for 5.8 years in a geographically defined area in southern Finland. It based the diagnosis of asthma on extensive clinical and lung function investigations, ascertained detailed SHS exposure history both at home and at work in the past 12 months and over a lifetime, and adjusted for a large set of potential confounders (Jaakkola et al. 2003). The relative risk estimate suggested for the disease burden calculation is 1.97 (95% CI 1.19–3.25) for SHS exposure at work and/or at home based on the Finnish study (Jaakkola et al. 2003). This is in the middle zone of estimates from other studies on adult asthma, their ORs ranging from 1.14 to 4.7.

### *Lung cancer*

Deaths from lung cancer outnumber deaths from any other cancer, worldwide (World Cancer Report, 2003) and the major cause is active tobacco smoking. Since the earliest reviews in the mid-1980s (IARC 1986; NRC 1986; US Surgeon General 1986), lung cancer has been firmly established as a health effect causally related to SHS exposure. Many meta-analyses summarizing the evidence on SHS exposure and the risk of lung cancer have been reported (Law 1996; Dockery and Trichopoulos 1997; Hackshaw et al. 1997; Boffetta et al. 1998; Hackshaw 1998; Wells 1998a; Zhong et al. 2000; Taylor et al. 2001; US Surgeon General 2006; Taylor, Najafi and Dobson 2007).

In summary, lung cancer among non-smokers is causally related to SHS both from spousal exposure and from exposure at work, and the suggested ORs are 1.21(1.13–1.30) and 1.22 (1.13–1.33), respectively (US Surgeon General 2006). In this report, we have not specified the exposure by site and have used the OR from spousal smoking as a proxy for any regular exposure.

### *Ischaemic heart disease*

Ischaemic heart disease is the most frequent cause of death in many parts of the world, therefore, any increase in risk due to SHS may have a substantial impact on the environmental burden of disease. Pooled estimates from meta-analyses consistently show a statistically significant increase in risk of approximately 30% (Wells 1994; Law et al. 1997; Wells 1998b; He et al. 1999; Thun, Henley and Apicella 1999).

The recent review carried out by the US Surgeon General (2006) concluded that SHS exposure is associated with increased risk for IHD mortality (fatal events), morbidity (nonfatal events), and symptoms. The overall pooled estimate related to any SHS exposure among non-smokers was 1.27 (1.19–1.36). The effects of home and workplace exposures are expected to be additive and no stratified meta-analyses were performed by the US Surgeon General (2006) due to limitations in the precision of the estimates for workplace exposure. Nonetheless, point estimates are similar for men and women and by exposure source (i.e. work or home).

A recent study (Whincup et al. 2004) based on the more accurate assessment of short-term exposure using biomarkers has shown higher risks of coronary heart disease (CHD) for SHS exposure (RR=1.45–1.57 according to exposure category). This type of study is, however, not the best for estimating the burden of disease at global level as the measure of exposure needs to match exposure prevalence data that are internationally available. Such studies based on biomarkers indicate, however, that misclassification of exposure may lead to an underestimation of effect when using exposure measures based on self-reporting (Whincup et al. 2004).

## **2.4 Equivalence of exposure definition in surveys and in epidemiological studies**

In order to estimate the burden of disease attributable to SHS, it is important to obtain and apply exposure information that matches as closely as possible the data used to derive the exposure-response relationship. In most instances, the etiological studies are questionnaire-based. Continuous data on exposure levels is rarely available either for describing exposure-response relationships or for assessing exposure in large groups. Therefore, we utilized a binary classification of exposure to separate the study population into those exposed to SHS and those not exposed.

The definition used in many epidemiological studies and surveys for “exposed to SHS” is “having a parent who smokes” for children, and “being exposed to others’ smoke”, or “living with a smoker” and/or “working in the same space with a smoker” in adults. The main surveys used as sources for exposure data for children (the GYTS survey) indeed report “having a parent who smokes” as the exposure indicator. The available multinational data on adults asked non-smokers whether the respondent was being exposed to tobacco smoke “most days or nights” (ECRHS studies) or “How often are you indoors and around people who are smoking cigarettes or other types of tobacco products?” (Bloch et al. 2008).

#### *Applicability of risk estimates to developing countries*

Additional concerns need to be analysed when considering the applicability of risk estimates to parental smoking in developing countries. As mentioned in the guide summarizing the methods for estimating disease burden from SHS (Öberg et al. 2010), the risk estimates for the various health outcomes as listed in tables 7 and 8 mainly come from studies conducted in developed countries. Yet the exposure situation in developing countries using the same exposure variable (i.e. having a parent who is a smoker) may differ significantly from the bulk of the health effects studies, due to the following conditions:

- the ratio of mothers compared to fathers smoking may differ – lower mother to father smoking ratios may reduce the risk estimate;
- the mean daily number of cigarettes smoked by smokers differs – lower numbers of cigarettes smoked at home may reduce the SHS concentration, provided other parameters remain the same;
- ventilation may be increased – in warmer climates, ventilation may be increased, and consequently SHS concentrations may be lower;
- ventilation may be increased or decreased – in modern buildings common in developed regions forced ventilation may lower the SHS concentrations, but extensive insulation may increase the SHS concentrations;
- the number of adults living in the same home as children may be higher – the prevalence of SHS exposure compared to the parental smoking rate may be increased;
- the number of people by room may be higher – people may spend more time, or may sleep more frequently, in rooms where people smoke.

In order to use the exposure indicator corresponding best to the exposure situation used in the health effect studies, those factors are analysed here in more detail.

#### (a) Number of cigarettes smoked by smokers

The number of cigarettes smoked by smokers varies across regions, and is often higher in developed countries than in developing countries. Table 8 provides an overview of the mean numbers of cigarettes smoked by smokers.

Table 8 Mean number of daily cigarettes consumed by smoker in each region<sup>a</sup>

Region	Mean number of cigarettes per current smoker per day	Mean number of cigarettes per daily smoker per day
<i>Africa</i>		
<i>total</i>	8	10
<i>without Algeria and South Africa<sup>b</sup></i>	6	8
<i>North America</i>	14	17
<i>Latin America and the Caribbean</i>	12	13
<i>Eastern Mediterranean</i>	14	19
<i>Europe (Eur A)</i>	14	15
<i>Europe (Eur B, C)</i>	14	16
<i>South-East Asia</i>		
- including bidis	16	20
- not including bidis	9	10
<i>Western Pacific (Wpr A)</i>	23	27
<i>Western Pacific (Wpr B)</i>	14	15

<sup>a</sup> Occasional smokers, i.e. those who do not smoke daily, are assumed to consume no more than 3.5 cigarettes per week, i.e. an average of one cigarette every other day; in Latin America and the Caribbean, occasional smokers are assumed to smoke seven cigarettes per week.

<sup>b</sup> Countries with higher national incomes and smoking rates than most other countries in the region.

Sources: Mackay et al. 2002; CDC 2005; European Commission 2006; Monteiro et al. 2007; WHO 2008b; Wipfli et al. 2008. According to the sources, the definition of «current smoker» may vary; this may explain the differences between «current» and «daily» smokers according to the region.

See the Annex, Table A2 for country grouping into WHO subregions.

The number of cigarettes consumed by a smoker is indeed lower in certain developing regions, in particular in Africa, and Latin America and the Caribbean. However, additional factors (as outlined above) have an influence on the biologically effective dose of SHS.

#### (b) Number of adults smoking at home and crowding

The parents are not always the only smokers in the home. The GYTS (CDC and WHO 2008) assesses both the exposure of children to SHS at home (during the last week) and percentage of children with at least one parent who smokes. In Africa, the percentage of children with SHS exposure in the home is almost twice as high as the percentage of parental smoking (Table 9), while in the other developing regions the percent of children exposed to SHS showed little difference to the percentage of children with parental smoking. This indicates that in Africa in particular, more adults tend to smoke in the home than just the parents, adding in this way additional exposure to the one usually accounted for by the indicator “parental smoking”.

**Table 9 Smoking at home in the presence of children compared to parental smoking**

Region	Ratio of smoke at home to parental smoking [-]
<i>Africa</i>	1.9
<i>Americas (Amr B, D)</i>	1.0
<i>Eastern Mediterranean</i>	1.1
<i>Europe (Eur B, C)</i>	1.3
<i>South-East Asia</i>	0.9
<i>Western Pacific (Wpr B)</i>	0.9

Source: GYTS (CDC and WHO 2008).

See Annex, Table A2 for country grouping into WHO subregions.

Also, an analysis of hair nicotine showed a strong gradient for an increasing number of smokers sharing children's bedrooms (Wipfli et al. 2008).

#### (c) Paternal versus maternal exposure

The odds ratio of children's health effects when exposed to SHS have been assessed for maternal, paternal and any parental exposure separately. It appears that the effect of maternal smoking on LRI (OR = 1.61(1.47–1.75)) is somewhat higher than the effect of paternal smoking (OR = 1.31(1.16–1.48)). Also, the smoking rate of women is generally higher in the countries of studies providing the effect estimate, than in developing countries. Using an effect estimate for parental exposure as opposed to estimating health effects separately in relation to maternal and paternal smoking could, therefore, lead to an overestimate of the effect in developing countries where women smoke less.

#### (d) Awareness about possible harm of SHS

In developed countries, awareness of the possible harm of SHS, in particular to children, may be higher than in developing countries. The indoor smoking behaviour of an adult smoker aware of SHS risks may, therefore, tend to cause less exposure, for example, by their smoking outdoors instead of indoors.

#### (e) Epidemiological studies

Epidemiological studies conducted in developing countries reflect, to some extent, the different exposure conditions, when using the same exposure indicators as used in developed countries. They could, therefore, provide some indication of the similarity or difference in exposure conditions. Unfortunately, they are often not of sufficient quantity or quality to constitute an independent body of evidence in themselves.

A review of studies on LRI in children exposed to SHS (Öberg et al. 2010) has shown a similar (although somewhat lower) effect on health of 1.37 (1.25–1.50) for studies from developing countries for the age group

0–5 years as compared to 1.55 (1.42–1.69) for all studies for the age group 0–2 years. A somewhat lower risk estimate is expected for the age group 0–5 years compared to the first two years of life only, as studies have shown that the strongest effect is detected in the youngest children (Li et al. 1996).

The Surgeon General's report (2006) shows consistently higher risk estimates for lung cancer and SHS in Asian studies (the only developing region with such studies) as compared to North American and European studies. All Asian studies were from the People's Republic of China. Another review comparing study results across three continents showed no sign of important heterogeneity across Asia, Europe and North America (Taylor et al. 2007). However Asian studies, primarily from China, showed higher risk estimates. Explanations offered for these higher rates include the fact that, in China, people live in smaller residences shared with more people (so potentially more smokers).

For ischaemic heart disease, the Surgeon General's report (2006) again provides (unsignificantly) higher risk estimates for the two studies from developing countries (Argentina and China). While more scattered, the study results from developing countries on asthma did not differ significantly from those conducted in developed countries for children, while for adults the respective reviews did not report any studies from developing countries. The only study from developing countries of acute otitis media showed a negative association.

In summary, studies from developing countries did not tend to show consistently lower effects than in developed countries.

(f) Smoking products other than manufactured cigarettes

In certain regions, products other than manufactured cigarettes are widely spread, and their consumption can even reach that of cigarettes. Examples include the consumption of bidis in India (Table 9), or Kretek cigarettes in Indonesia. While the effects of second-hand smoke have not been definitely documented, they are considered as "equally likely to cause harm to the health of non-smokers" in a recent monograph on Bidi smoking and public health (Gupta and Asma, 2008). New scientific studies on the content and emissions of bidis will, however, be needed to gather additional evidence on possible harm.

(g) Direct measure of exposure through hair nicotine

Level of nicotine in hair probably provides a relatively good indication of the exposure level to SHS. In fact, rather than speculating on the aggravating or mitigating effects of higher ventilation, more crowding, more male versus female smoking or lower numbers of cigarettes smoked per day in relation to studies providing effect estimates for the health outcomes, a direct measure of personal exposure may be more relevant. A recent multi-country study on SHS exposure, covering 31 countries, compared hair nicotine of children and women across countries (Wipfli et al. 2008). The results of the study are summarized in Table 10. This is the largest multi-country study of hair nicotine concentrations. It provided evidence that the levels of SHS exposure in Asia are slightly higher than in Europe, while the levels are slightly lower in Latin America. Comparability to hair nicotine assessed by other studies is limited because of varying laboratory methodologies (Al-Delaimy 2002).



**Table 10 Hair nicotine in children and women**

Region	Hair nicotine in children [ng/mg]	Hair nicotine in women [ng/mg]	Mean number of cigarettes smoked by smoker [-]
Asia <sup>1</sup>	0.87	0.50	14
Europe and Middle East <sup>2</sup>	0.72	0.37	18
Latin America <sup>3</sup>	0.50	0.26	12

Source: Wipfli et al. 2008.

<sup>1</sup> Corresponds to the regions Sear and Wpr (used in this report) combined.

<sup>2</sup> Corresponds mainly to Eur B and C; the two countries from the Middle East (Syria and Egypt) are amongst the countries with the highest hair-nicotine levels in the region.

<sup>3</sup> Corresponds to Amr B and C.

The European countries assessed in the study by Wipfli (Wipfli et al. 2008) are from central/eastern Europe, rather than western Europe where many health effect studies were originated. However, these values are probably closest to the exposure situation as found in the health effect study countries.

The composition of the cigarettes, and the way they are smoked are additional factors determining the effective dose of SHS. For example, people in poorer communities take more puffs per cigarette, and leave shorter stubs, than smokers in affluent communities. Studies conducted in the USA illustrate that cotinine levels in black Americans tend to be higher than in white Americans, although the average number of cigarettes smoked per day tends to be higher among whites (Mannino et al. 2001). The reasons for this are likely to be differences in smoking patterns and living conditions.

#### (h) Selected approach

One might be tempted to use a dose-response relationship of SHS effect with the number of cigarettes smoked by smoker to account for differences in active smoking intensity (under the assumption that active smoking intensity is associated with SHS exposure). Another way to account for active smoking intensity would be to use the excess lung cancer death rates as a proxy for cumulative exposure from active tobacco smoking (Peto et al. 2003) (and assume the same ratio of impact in developing to developed countries for SHS as for active smoking). However, factors other than active smoking intensity need to be considered when trying to assess harmful levels of exposure to SHS. As discussed in this section, the relevant exposure and biologically effective dose of SHS is, in addition to the number of cigarettes smoked, influenced by many factors, such as proximity to smokers, the volume of indoor space, duration of contact, ventilation systems, number of people per house, etc. The smoking rate is just one determinant or indicator of the level of exposure and has, therefore, not been retained as the basis for estimation of SHS health impacts.

In summary, and given the various factors acting on exposure and available evidence, the following approaches are proposed for each region.

- Africa (Afr D, E). Smoking intensity by smoker is lower, and women's smoking is very low in many countries as compared to men's smoking. However, smoking at home is nearly twice as high as parental smoking prevalence, and crowding in homes is more important. All seven studies performed in Africa

(of limited quality) have shown important positive associations between SHS and LRI. Occasional smokers (i.e. smokers who do not smoke daily) were removed from exposure assessment in order to adjust to some extent to the low SHS intensity they may produce (although these were taken into account in the developed country studies), which corresponded to a reduction in SHS exposure prevalence of 27%.

- North America (Amr A). Conditions are similar to those assessed in most of the health effect studies, therefore no adjustment is necessary, and relative risks are directly applicable.
  - Latin America and the Caribbean (Amr B, D). Occasional smokers were removed in order to adjust to some extent to the low SHS intensity they may produce, corresponding to a reduction in SHS exposure prevalence of 25%
  - Eastern Mediterranean (Emr B, D). Smoking intensity and hair nicotine levels are comparable to European levels (Table 9; Wipfli et al. 2008). The risk estimates of developed countries are therefore directly applied.
  - Europe (Eur A), North America (Amr A). Conditions are similar to those assessed in most of the health effects studies, therefore no adjustment is necessary, and relative risks are directly applicable.
- Europe (Eur B, C). Parental smoking is unlikely to cause different exposure situations than in developed countries, given relative similarities in smoking intensity, housing conditions and climate. The risk estimates of developed countries are, therefore, applied.
- South-East Asia (Sear B, D). Smoking intensity is higher when including exposure to bidi smoke and lower when counting only manufactured cigarettes. Health impacts of bidis have not been definitely documented, however, hair nicotine in Indian children and women is at least as high as in the countries where the health effect studies have been conducted (Table 9; Wipfli et al. 2008). The risk estimates of developed countries are, therefore, applied.
- Western Pacific (Wpr A, B). Number of cigarettes smoked are higher in Wpr A than in the countries where the health effect studies have been conducted, and health effect studies in Asia (mainly China) indicate higher effects for lung cancer; the risk estimates of developed countries are, nevertheless, applied.

In summary, evidence seems to indicate that the health effect varies little across studied continents, even though certain variables, such as numbers of cigarettes smoked, etc., vary. However, as in Africa and Latin America, the mean numbers of daily cigarettes smoked by smokers are considerably lower, we only counted daily smokers in those regions as smokers (rather than the “current” smokers, which include smokers consuming less than one cigarette per day). The number of non-smokers exposed to SHS have been reduced accordingly in these regions.

However, given the uncertainties outlined in this section, in particular linked to the transferability of risk ratios from developed to developing countries, we applied a number of alternative approaches in the sensitivity analysis (Section 3.3). These alternative approaches include the application of a pooled risk ratio for lower respiratory infection from developing country studies only (RR=1.34), and the separate estimation of health impacts on children caused by paternal and maternal smoking (as opposed to parental smoking, given that the paternal-to-maternal smoking ratio is much lower in developing countries).

## 2.5 Available health data for the selected outcomes

### Lower respiratory infection

Lower respiratory infections are a major cause of deaths, with an estimated 3.9 million deaths worldwide in the year 2004 (WHO 2009c). Among the youngest children (0–4 years), 1.8 million die each year due to LRI, with these deaths occurring mainly in children of the developing world (Table 11). The DALYs related to LRI in the age group 0–4 years are about 63 million, of these 53% occur in the African regions (Afr D and Afr E) and 24% in the south-eastern region of Asia (Sear B, Sear D).

*Table 11 Burden of lower respiratory infections (deaths and DALYs<sup>a</sup>) in children 0–4 years by WHO subregion, 2004*

Subregion	Males 0–4 years		Females 0–4 years		Total 0–4 years	
	Deaths	DALYs	Deaths	DALYs	Deaths	DALYs
<i>Afr D</i>	245 000	8 688 000	231 000	8 070 000	476 000	16 758 000
<i>Afr E</i>	250 000	8 787 000	223 000	7 718 000	474 000	16 505 000
<i>Amr A</i>	340	12 500	260	9 800	600	22 200
<i>Amr B</i>	18 700	819 000	14 800	672 000	33 500	1 491 000
<i>Amr D</i>	8 500	314 000	8 000	297 000	16 500	611 000
<i>Emr B</i>	5 500	212 000	5 400	210 000	10 900	421 000
<i>Emr D</i>	122 000	4 344 000	117 000	4 147 000	239 000	8 491 000
<i>Eur A</i>	170	6 300	140	5 300	310	11 600
<i>Eur B</i>	16 800	578 000	13 700	475 000	30 600	1 054 000
<i>Eur C</i>	2 100	72 000	1 500	52 400	3 600	124 000
<i>Sear B</i>	10 500	404 000	9 800	387 000	20 300	791 000
<i>Sear D</i>	207 000	7 393 000	193 000	6 888 000	401 000	14 281 000
<i>Wpr A</i>	130	4 800	89	3 300	220	8 100
<i>Wpr B</i>	36 800	1 385 000	36 900	1 380 000	73 700	2 765 000
<i>Global</i>	925 000	33 019 000	855 000	30 314 000	1 780 000	63 333 000

<sup>a</sup> DALYs, disability-adjusted life years.

Source: WHO 2009c.

See Annex, Table A2 for country grouping into WHO subregions.

### Otitis media

Otitis media, i.e. middle-ear infections, is one of the most commonly diagnosed problems in outpatient pediatrics and general practice. An average toddler may have several painful episodes every year. Without antibiotic treatment, acute otitis media can progress to cause perforation of the eardrum, mastoiditis, or even meningitis, a

potentially fatal disease. Very few children die as a consequence of middle-ear infections but otitis media causes considerable morbidity.

The numbers of deaths and DALYs from otitis media were extracted from the WHO database on burden of disease (WHO 2009c) (Table 12). The data included any kind of acute otitis and there is no separation for recurrent otitis, which is also causally related to SHS exposure. Serous otitis is not included in the data. The fatal cases are too few to be included in a detailed analysis. The rate of DALYs per capita does not differ much across developing regions (about 0.46 DALYs/1000 children 0–4 years), and is about one third lower in developed regions.

**Table 12 Burden of acute otitis media (deaths and DALY<sup>a</sup>) in children 0–4 years, by WHO subregion, 2004**

Subregion	Total deaths	DALYs		DALYs
	(0–4 yrs)	Males 0–4 yrs	Females 0–4 yrs	Total 0–4 yrs
<i>Afr D</i>	41	12 100	11 900	24 100
<i>Afr E</i>	92	14 800	14 500	29 300
<i>Amr A</i>	11	3 500	3 400	6 900
<i>Amr B</i>	130	12 200	11 400	23 700
<i>Amr D</i>	10	2 200	2 100	4 300
<i>Emr B</i>	0	3 100	3 000	6 200
<i>Emr D</i>	130	12 800	12 300	25 100
<i>Eur A</i>	7	3 400	3 100	6 600
<i>Eur B</i>	4	2 900	2 800	5 600
<i>Eur C</i>	10	1 900	1 700	3 600
<i>Sear B</i>	0	5 900	5 700	11 700
<i>Sear D</i>	210	37 800	34 900	72 700
<i>Wpr A</i>	2	1 200	1 100	2 300
<i>Wpr B</i>	89	27 400	24 200	51 600
<i>Global</i>	740	141 000	132 000	274 000

<sup>a</sup> DALYs, disability-adjusted life years.

Source: WHO 2009c.

See Annex, Table A2 for country grouping into WHO subregions.

### **Asthma among children 0–14 years**

Asthma is the most common chronic respiratory condition in childhood. Asthma is reported to be a fatal disease for about 12 000 children worldwide and causes approximately 6.4 million DALYs in 2004 (Table 13). About 26% of these deaths and 20% of the DALYs occur in Sear D. The death rate from asthma is one or lower per 10 000 children in all regions. However, the highest rates of DALYs are found in the Americas and Wpr A with about 5–6 DALYs per 1000 children <14 years. In contrast to LRI and otitis, the DALY rates are also relatively high in the developed regions.

Table 13 Burden of asthma (deaths and DALYs<sup>a</sup>) in children 0–14 years, by WHO subregion, 2004

Subregion	Males <14 years		Females <14 years		Total <14 years	
	Deaths	DALYs	Deaths	DALYs	Deaths	DALYs
<i>Afr D</i>	820	351 000	840	245 000	1 700	596 000
<i>Afr E</i>	770	361 000	720	366 000	1 500	727 000
<i>Amr A</i>	98	177 000	56	203 000	150	381 000
<i>Amr B</i>	390	372 000	380	436 000	770	808 000
<i>Amr D</i>	100	75 100	61	84 100	160	159 000
<i>Emr B</i>	59	58 900	52	64 400	110	123 000
<i>Emr D</i>	620	210 000	430	206 000	1 000	416 000
<i>Eur A</i>	40	120 000	26	134 000	66	254 000
<i>Eur B</i>	330	71 200	260	78 300	590	149 000
<i>Eur C</i>	12	23 600	7	27 500	19	51 100
<i>Sear B</i>	490	85 600	440	93 800	920	179 000
<i>Sear D</i>	1 400	602 000	1 700	656 000	3 100	1 258 000
<i>WprA</i>	24	60 500	11	68 900	35	129 000
<i>WprB</i>	790	554 000	790	598 000	1 600	1 152 000
<i>Global</i>	6 000	3 122 000	5 800	3 262 000	11 700	6 383 000

<sup>a</sup> DALYs, disability-adjusted life years.

Source: WHO 2009c.

See Annex, Table A2 for country grouping into WHO subregions.

### Asthma among adults (>15 years)

Asthma among people older than 15 years is estimated to have caused approximately 275 000 deaths and almost 10 million DALYs globally in 2004 (Table 14). The fatality rate is about 0.1 or less deaths per 10 000 in all regions. The mean DALY rate is about 2.2 DALYs per 1000 adults.

Table 14 Burden of asthma (deaths and DALYs<sup>a</sup>), by WHO subregion, in the population >15 years, 2004

Subregion	Men >15 years		Women >15 years		Total >15 years	
	Deaths	DALYs	Deaths	DALYs	Deaths	DALYs
<i>Afr D</i>	14 500	425 000	12 600	300 000	27 000	725 000
<i>Afr E</i>	16 700	475 000	15 700	462 000	32 500	937 000
<i>Amr A</i>	1 600	236 000	2 800	167 000	4 400	403 000
<i>Amr B</i>	3 600	451 000	5 100	304 000	8 700	754 000
<i>Amr D</i>	840	73 400	680	47 000	1 500	120 000
<i>Emr B</i>	3 500	114 000	2 500	76 700	6 000	191 000
<i>Emr D</i>	9 800	432 000	5 700	248 000	15 500	680 000
<i>Eur A</i>	3 400	240 000	5 100	183 000	8 500	423 000
<i>Eur B</i>	4 300	129 000	3 500	89 600	7 800	218 000
<i>Eur C</i>	11 600	143 000	7 700	82 800	19 300	226 000
<i>Sear B</i>	10 500	227 000	10 600	201 000	21 100	427 000
<i>Sear D</i>	38 800	1 428 000	37 000	1 178 000	75 800	2 606 000
<i>Wpr A</i>	1 700	121 000	2 100	97 500	3 800	218 000
<i>Wpr B</i>	24 100	1 196 000	19 000	751 000	43 100	1 946 000
<i>Global</i>	145 000	5 689 000	130 000	4 185 000	275 000	9 875 000

<sup>a</sup> DALYs, disability-adjusted life years.

Source: WHO 2009c,

See Annex, Table A2 for country grouping into WHO subregions.

### Lung cancer

Lung cancer caused annually approximately 1.3 million deaths and 11 million DALYs in the population >15 years in 2004 (Table 15). In contrast to many other SHS-related outcomes that show a greater burden in developing countries, about 46% of this disease burden is found in the developed regions (Amr A, Eur, Wpr A), reflecting the history of heavy tobacco use 10–20 years ago. The average rates of fatal lung cancers among men and women are 0.2 and 0.1 deaths per 1000 persons >15 years. The mean death rate is of about 0.6 per 1000 in developed regions, and 0.2 per 1000 in developing regions.

Most cases of lung cancer occur in the age group 60–80 years, due to the long latency period of the carcinogenic process of this cancer. Lung cancer is also more common among men than women, reflecting the higher smoking prevalence in the past among men.

**Table 15 Burden of lung cancer (deaths and DALYs<sup>a</sup>), by WHO subregion, in the population >15 years, 2002**

Subregion	Men >15 years		Women >15 years		Total >15 years	
	Deaths	DALYs	Deaths	DALYs	Deaths	DALYs
<i>Afr D</i>	9 100	88 700	1 900	20 000	11 000	106 000
<i>Afr E</i>	10 000	107 000	3 700	42 100	13 700	147 000
<i>Amr A</i>	106 000	797 000	79 800	625 000	186 000	1 372 000
<i>Amr B</i>	38 400	350 000	15 100	148 000	53 500	486 000
<i>Amr D</i>	3 000	24 600	1 500	14 700	4 400	38 200
<i>Emr B</i>	6 300	57 600	1 200	13 600	7 400	69 700
<i>Emr D</i>	20 800	217 000	4 100	53 000	25 000	265 000
<i>Eur A</i>	159 000	1 239 000	55 900	462 000	215 000	1 640 000
<i>Eur B</i>	56 900	566 000	12 600	130 000	69 500	688 000
<i>Eur C</i>	69 700	713 000	16 300	151 000	86 100	857 000
<i>Sear B</i>	36 400	336 000	14 700	156 000	51 100	484 000
<i>Sear D</i>	82 000	782 000	21 000	219 000	103 000	979 000
<i>Wpr A</i>	50 100	321 000	20 000	125 000	70 000	418 000
<i>Wpr B</i>	293 000	2 695 000	132 000	1 280 000	425 000	3 908 000
<i>Global</i>	941 000	8 292 000	380 000	3 440 000	1 321 000	11 457 000

<sup>a</sup> DALYs, disability-adjusted life years.

Source: WHO 2009c.

See Annex, Table A2 for country grouping into WHO subregions.

### Ischaemic heart disease

Each year, ischaemic heart disease causes approximately 7.2 million deaths and the loss of 60 million DALYs (Table 16), and is the most frequent cause of death in the world. The largest numbers of deaths and DALYs lost are estimated to occur in Sear D and Eur C. The average rate per 1000 capita is estimated to be 1.6 deaths and 13.2 DALYs for persons >15 years, with the highest rates in Eur C (5.9 deaths and 47 DALYs per 1000 persons >15 years). In contrast, 0.6 deaths and 5.2 DALYs per 1000 persons are reported from Amr D.

**Table 16 Burden of ischaemic heart disease (deaths and DALYs<sup>a</sup>), by WHO subregion, in the population >15 years, 2004**

Subregion	Men >15 years		Women >15 years		Total >15 years	
	Deaths	DALYs	Deaths	DALYs	Deaths	DALYs
<i>Afr D</i>	96 700	1 020 000	73 500	706 000	170 000	1 693 000
<i>Afr E</i>	97 900	1 032 000	77 100	732 000	175 000	1 735 000
<i>Amr A</i>	283 000	1 959 000	265 000	1 204 000	548 000	2 942 000
<i>Amr B</i>	194 000	1 871 000	151 000	1 216 000	346 000	2 990 000
<i>Amr D</i>	17 800	158 000	13 200	105 000	30 900	253 000
<i>Emr B</i>	89 200	974 000	55 600	496 000	145 000	1 435 000
<i>Emr D</i>	251 000	2 883 000	181 000	1 729 000	432 000	4 528 000
<i>Eur A</i>	342 000	2 330 000	317 000	1 293 000	660 000	3 364 000
<i>Eur B</i>	237 000	2 235 000	211 000	1 459 000	449 000	3 596 000
<i>Eur C</i>	541 000	5 605 000	646 000	3 903 000	1 187 000	9 343 000
<i>Sear B</i>	149 000	1 658 000	127 000	1 264 000	277 000	2 874 000
<i>Sear D</i>	976 000	10 629 000	752 000	7 805 000	1 728 000	18 127 000
<i>Wpr A</i>	71 300	539 000	59 800	267 000	131 000	758 000
<i>Wpr B</i>	467 000	4 102 000	430 000	2 950 000	897 000	6 793 000
<i>Global</i>	3 814 000	36 994 000	3 361 000	25 130 000	7 175 000	60 431 000

<sup>a</sup> DALYs, disability-adjusted life years.

Source: WHO 2007.

See Annex, Table A2 for country grouping into WHO subregions.

## 3 Results

### 3.1 Burden of disease attributable to second-hand smoke

#### Lower respiratory infection

The burden of LRI was estimated on the basis of an OR of 1.55 (1.42–1.69) (US Surgeon General 2006) for the first two years of life, and a reduced OR of 1.18 for ages 2–4 years (Öberg et al. 2010). In 2004, the global population attributable fraction of lower respiratory infections to SHS exposure was 9% within the age group 0–4 year (and 6% of all LRI), with a range between 5% (Africa) and 20% or above (in central and eastern



Europe, and the Western Pacific region). This represents about 165 000 deaths from LRI among children up to the age of four years that could be avoided if children's exposure to SHS were eliminated (Table 17).

The regional results show a substantial burden of mortality and morbidity among very young children in all regions. The main disease burden of LRI from SHS is, however, found in regions with a high mortality of LRI and high SHS exposure rates. The highest burden of disease related to SHS is found in Sear D, where 56 000 children are estimated to have died from LRI caused by SHS exposure in 2004. Several regions reach death rates per 1000 children under five years of 0.3 and above (Africa, eastern Mediterranean region D, eastern European region B, and South-East Asian region D).

**Table 17 Burden of lower respiratory infections attributable to SHS exposure, in children 0–4 years, 2004**

Subregion	Population attributable fraction <sup>a</sup>	DALYs <sup>b</sup>		Deaths	
		Total	Per 1000 children	Total	Per 1000 children
<i>Afr D</i>	0.05	816 000	14.4	23 200	0.41
<i>Afr E</i>	0.04	699 000	10.7	20 000	0.31
<i>Amr A</i>	0.11	2 400	0.1	65	0.00
<i>Amr B</i>	0.12	185 000	4.1	4 200	0.09
<i>Amr D</i>	0.09	57 400	6.1	1 600	0.16
<i>Emr B</i>	0.16	68 500	4.7	1 800	0.12
<i>Emr D</i>	0.13	1 083 000	21.9	30 500	0.62
<i>Eur A</i>	0.19	2 300	0.1	60	0.00
<i>Eur B</i>	0.18	185 000	10.9	5 400	0.32
<i>Eur C</i>	0.23	28 200	2.4	820	0.07
<i>Sear B</i>	0.22	174 000	6.3	4 500	0.16
<i>Sear D</i>	0.14	1 996 000	12.8	56 000	0.36
<i>Wpr A</i>	0.18	1 400	0.2	39	0.01
<i>Wpr B</i>	0.23	641 000	5.6	17 100	0.15
<b>Global</b>	<b>0.09</b>	<b>5 939 000</b>	<b>9.6</b>	<b>165 000</b>	<b>0.27</b>

<sup>a</sup> Within age group.

<sup>b</sup> DALYs, disability-adjusted life years.

See Annex, Table A2 for country grouping into WHO subregions.

### Otitis media

The burden of disease due to otitis media was calculated based on the OR of 1.38 (1.21–1.56) for acute otitis (Etzel et al. 1992; Cal EPA 2005), for the first three years of life. The value of 1.66 (1.33–2.06) was used in the sensitivity analysis (Uhari et al. 1996). The global population attributable fraction due to SHS was estimated as 9% within the age group 0–4 year (and 1.7% of all LRI), with a range between 3% for Africa and 15% for Western Pacific region (Wpr) B.

The global number of deaths attributable to SHS is estimated at less than 100 annually. However, the disability caused by otitis attributable to SHS is quite substantial (Table 18), with most DALYs estimated for Wpr B, which also carries the highest disease burden of SHS-related otitis per capita (0.07 DALYs per 1000 children <5 years attributable to SHS).

**Table 18 Burden of acute otitis media attributable to SHS exposure, in children 0–4 years old, 2004**

Subregion	Population	DALYs <sup>b</sup>	
	attributable fraction <sup>a</sup>	Total	Per 1000 children
<i>Afr D</i>	0.03	820	0.01
<i>Afr E</i>	0.03	970	0.01
<i>Amr A</i>	0.06	440	0.02
<i>Amr B</i>	0.07	1 700	0.04
<i>Amr D</i>	0.06	240	0.03
<i>Emr B</i>	0.09	570	0.04
<i>Emr D</i>	0.08	2 100	0.04
<i>Eur A</i>	0.12	800	0.04
<i>Eur B</i>	0.12	700	0.04
<i>Eur C</i>	0.14	500	0.04
<i>Sear B</i>	0.13	1 500	0.05
<i>Sear D</i>	0.09	6 600	0.04
<i>Wpr A</i>	0.12	270	0.04
<i>Wpr B</i>	0.15	7 800	0.07
<b>Global</b>	<b>0.09</b>	<b>24 900</b>	<b>0.04</b>

<sup>a</sup> Within age group.

<sup>b</sup> DALYs, disability-adjusted life years.

See Annex, Table A2 for country grouping into WHO subregions.

**Asthma among children 0–14 years**

The disease burden due to asthma was calculated based on the OR of 1.32 (1.24–1.41) from the most updated meta-analysis by the California Environmental Protection Agency (2005). The average population attributable fraction in children due to SHS was 10%, with a range between 4% for Africa and 18% for Wpr B. The mortality from childhood asthma is relatively low with around 1000 deaths of children below five years attributable to SHS in 2004. However, the number of DALYs related to SHS is substantial, amounting to about 660 000 DALYs in 2004 (Table 19). The highest number of DALYs in absolute numbers is estimated for Wpr B (202 000) and the lowest in Eur C (8300). The per capita DALY rate of asthma per 1000 children 0–14 years ranges from 0.15 in Afr D to 0.76 in Wpr A.

*Table 19 Burden of asthma attributable to SHS exposure, for children 0–14 years old, 2004*

Subregion	Population attributable fraction <sup>a</sup>	DALYs <sup>b</sup>	
		Total	Per 1000 children
<i>Afr D</i>	0.04	22 000	0.15
<i>Afr E</i>	0.04	29 400	0.17
<i>Amr A</i>	0.07	27 500	0.39
<i>Amr B</i>	0.08	66 600	0.49
<i>Amr D</i>	0.07	10 700	0.39
<i>Emr B</i>	0.11	13 300	0.29
<i>Emr D</i>	0.09	39 200	0.27
<i>Eur A</i>	0.14	35 800	0.52
<i>Eur B</i>	0.15	22 600	0.41
<i>Eur C</i>	0.16	8 300	0.22
<i>Sear B</i>	0.14	25 700	0.31
<i>Sear D</i>	0.10	130 000	0.28
<i>Wpr A</i>	0.14	18 100	0.76
<i>Wpr B</i>	0.18	202 000	0.54
<i>Global</i>	0.10	651 000	0.35

<sup>a</sup> Within age group.

<sup>b</sup> DALYs, disability-adjusted life years.

See Annex, Table A2 for country grouping into WHO subregions.

**Asthma among adults (≥15 years)**

The burden of asthma in adults was calculated with the OR of 1.97 (1.19–3.25) from the Finnish Environment and Asthma Study (Jaakkola et al. 2003). Only the burden among non-smokers was considered for estimating the burden of disease due to SHS exposure, based on the method outlined in Section 2.1 and the burden attributable to active smoking (C. Mathers, personal communication, 2009; Ezzati and Lopez 2004a).

Globally, the population attributable fraction due to SHS is estimated to have reached 9% and 22% for men and women (Table 20), respectively, in 2004, and 13% for the total adult population. The resulting disease burden of adult asthma attributable to SHS amounts to 36 000 deaths annually (Table 20). The highest absolute number is estimated to occur in Sear D (9800). Asthma is usually an outcome causing disability rather than death. The number of DALYs attributable to SHS is estimated at approximately 1.2 million worldwide, with most DALYs estimated for Wpr B (360 000). The highest disease burden per 1000 adults is estimated for Emr D (0.42 DALYs per 1000 adults), and the lowest in Amr A (0.11 DALYs per 1000 adults).

*Table 20 Burden of asthma attributable to SHS exposure, population ≥15 years, 2004*

Subregion	Population attributable fraction <sup>a</sup>		DALYs <sup>b</sup>		Deaths	
	Men	Women	Total	Per 1000		
				Adults	Total	Per 1000
<i>Afr D</i>	0.04	0.08	39 200	0.20	1 600	0.008
<i>Afr E</i>	0.03	0.08	52 100	0.23	1 800	0.008
<i>Amr A</i>	0.07	0.07	28 700	0.11	290	0.001
<i>Amr B</i>	0.07	0.14	73 400	0.23	930	0.003
<i>Amr D</i>	0.09	0.13	12 600	0.26	140	0.003
<i>Emr B</i>	0.11	0.18	26 100	0.26	730	0.007
<i>Emr D</i>	0.09	0.23	97 200	0.42	2 200	0.010
<i>Eur A</i>	0.11	0.13	51 200	0.15	1 100	0.003
<i>Eur B</i>	0.10	0.22	33 300	0.20	1 300	0.008
<i>Eur C</i>	0.08	0.29	35 400	0.18	3 300	0.016
<i>Sear B</i>	0.09	0.26	71 700	0.32	3 700	0.017
<i>Sear D</i>	0.11	0.14	324 000	0.36	9 800	0.011
<i>Wpr A</i>	0.15	0.27	44 100	0.33	700	0.005
<i>Wpr B</i>	0.12	0.29	357 000	0.30	8 100	0.007
<i>Global</i>	0.09	0.22	1 246 000	0.27	35 800	0.008

a Within age group.

b DALYs, disability-adjusted life years.

See Annex, Table A2 for country grouping into WHO subregions.

## Lung cancer

The disease burden due to lung cancer incidence was calculated based on the OR of 1.21 (1.13–1.30) from the meta-analysis by the US Surgeon General (2006). Lung cancer has been known for two decades to be causally related to SHS. However, active smoking remains by far the most important known risk factor for lung cancer globally. The burden of active smoking (C. Mathers, personal communication, 2009; Ezzati and Lopez 2004a) was deducted before estimating the burden of disease from lung cancer due to SHS exposure in non-smokers. Globally, about 75% of the lung cancer burden among men is related to active smoking, as compared to 48% among women. Of the burden in non-smokers, SHS is estimated to cause about 21 000 deaths and about 216 000 DALYs worldwide (Table 21). The vast majority is estimated Wpr B, with 56% of the global burden of lung cancer from SHS exposure. The DALY rate per 1000 persons >15 years is estimated to amount to 0.010 DALYs for lung cancers attributable to SHS in Wpr B, compared to 0.05 as a global mean.

**Table 21 Burden of lung cancer attributable to SHS exposure, population ≥15 year olds, 2004<sup>a</sup>**

Subregion	Population attributable fraction <sup>b</sup>		DALYs <sup>c</sup>		Deaths	
	Men	Women	Total	Per 1000	Total	Per 1000
				Adults		Adults
<i>Afr D</i>	0.01	0.03	1 900	0.01	180	0.001
<i>Afr E</i>	0.02	0.04	3 200	0.01	280	0.001
<i>Amr A</i>	0.00	0.00	5 400	0.02	600	0.002
<i>Amr B</i>	0.01	0.02	7 200	0.02	680	0.002
<i>Amr D</i>	0.02	0.03	1 000	0.02	90	0.002
<i>Emr B</i>	0.02	0.05	1 600	0.02	140	0.001
<i>Emr D</i>	0.01	0.03	3 800	0.02	320	0.001
<i>Eur A</i>	0.01	0.02	16 800	0.05	2 000	0.006
<i>Eur B</i>	0.00	0.04	7 600	0.05	750	0.004
<i>Eur C</i>	0.00	0.05	10 300	0.05	1 100	0.005
<i>Sear B</i>	0.01	0.02	7 100	0.03	630	0.003
<i>Sear D</i>	0.02	0.04	20 500	0.02	1 900	0.002
<i>Wpr A</i>	0.01	0.04	7 600	0.06	940	0.007
<i>Wpr B</i>	0.02	0.05	121 000	0.10	11 800	0.010
<i>Global</i>	0.01	0.03	216 000	0.05	21 400	0.005

<sup>a</sup> Based on estimates of exposure in the early 1990s.

<sup>b</sup> Within age group.

<sup>c</sup> DALYs, disability-adjusted life years.

See Annex, Table A2 for country grouping into WHO subregions.

### Ischaemic heart disease

As for lung cancer, active smoking is a major risk factor for ischaemic heart disease and the burden related to active smoking was subtracted from the total burden before estimating the fraction attributable to SHS exposure among the non-smoking population. Globally, approximately 25% of IHD deaths among men and 7% among women are estimated to be attributable to active smoking.

The most suitable risk estimate of IHD related to any SHS exposure among non-smokers is a RR of 1.27 (1.19–1.36) according to the US Surgeon General's report (2006). On this basis, we estimated that 379 000 deaths from IHD were attributable to exposure to SHS (Table 22). The highest number (94 000) of these deaths is found in the Eur C and the lowest number in Amr D (980). The total DALYs from IHD attributable to SHS exposure in the world is estimated to reach 2.8 million. Most DALYs attributable to SHS from IHD are estimated Sear D (683 000). The highest death rate by 1000 people is also Eur C (0.47 per 1000 persons), and the lowest rates are found in the African and American regions.

**Table 22 Burden of ischaemic heart disease attributable to SHS exposure, population ≥15 years, 2004**

Subregion	Population attributable fraction <sup>a</sup>		DALYs <sup>b</sup>		Deaths	
	Men	Women	Total	Per 1000 Adults	Total	Per 1000 Adults
<i>Afr D</i>	0.01	0.03	29 300	0.15	3 100	0.02
<i>Afr E</i>	0.01	0.02	24 300	0.11	2 600	0.01
<i>Amr A</i>	0.02	0.02	61 900	0.23	12 600	0.05
<i>Amr B</i>	0.02	0.04	92 100	0.29	11 400	0.04
<i>Amr D</i>	0.02	0.04	7 800	0.16	980	0.02
<i>Emr B</i>	0.03	0.06	60 100	0.60	6 200	0.06
<i>Emr D</i>	0.03	0.08	214 000	0.93	22 000	0.10
<i>Eur A</i>	0.04	0.05	152 000	0.43	32 300	0.09
<i>Eur B</i>	0.04	0.09	208 000	1.23	30 000	0.18
<i>Eur C</i>	0.03	0.11	588 000	2.94	94 100	0.47
<i>Sear B</i>	0.02	0.11	176 000	0.79	18 400	0.08
<i>Sear D</i>	0.03	0.04	683 000	0.75	67 100	0.07
<i>Wpr A</i>	0.05	0.09	48 000	0.37	8 800	0.07
<i>Wpr B</i>	0.04	0.11	490 000	0.41	68 700	0.06
<i>Global</i>	0.03	0.07	2 836 000	0.62	379 000	0.08

<sup>a</sup> Within age group.

<sup>b</sup> DALYs, disability-adjusted life years.

See Annex, Table A2 for country grouping into WHO subregions.

**Total burden of disease attributable to SHS**

Approximately 603 000 premature deaths worldwide from the selected five outcomes are attributable to SHS (tables 23–25). Most of these deaths are caused by IHD in adults (63%), and LRI among young children (27%). Adult asthma and lung cancer contribute to a comparable amount of deaths (6% and 4%, respectively), while only a few children die from otitis and asthma related to SHS (Figure 4).

In addition to deaths, the disease burden attributable to SHS exposure is also expressed in DALYs that measure both the years of life lost and years lived with disability due to SHS-related diseases. The global amount of DALYs attributable to SHS is estimated at approximately 10.9 million (Table 24). For DALYs, the LRI among young children accounts for the largest burden (54%), followed by IHD (26%), adult asthma (11%), and child asthma (6%). Lung cancer and otitis media contributed to approximately 2.0% and 0.2% of DALYs caused by SHS.

**Table 23 Total deaths and DALYs<sup>a</sup> from second-hand smoke by outcome, 2004**

Health outcome	Deaths		DALYs	
	Children	Adults	Children	Adults
<i>LRI</i>	165 000	NA	5 939 000	NA
<i>Otitis</i>	<100	NA	24 900	NA
<i>Asthma</i>	1 100	35 800	651 000	1 246 000
<i>Lung cancer</i>	NA	21 400	NA	216 000
<i>IHD</i>	NA	379 000	NA	2 836 000
<b>Total</b>	<b>166 000</b>	<b>436 000</b>	<b>6 616 000</b>	<b>4 297 000</b>

<sup>a</sup> DALYs, disability-adjusted life years.

NA, not applicable.

**Table 24 Total deaths and DALYs<sup>a</sup> from second-hand smoke by subregion, 2004**

Subregion	DALYs		Deaths	
	Total	Per 1000 people	Total	Per 1000 people
<i>Afr D</i>	910 000	2.6	28 200	0.08
<i>Afr E</i>	809 000	2.1	24 700	0.06
<i>Amr A</i>	126 000	0.4	13 600	0.04
<i>Amr B</i>	427 000	0.9	17 300	0.04
<i>Amr D</i>	89 800	1.2	2 800	0.04
<i>Emr B</i>	170 000	1.2	8 900	0.06
<i>Emr D</i>	1 439 000	3.8	55 200	0.15
<i>Eur A</i>	259 000	0.6	35 500	0.08

<i>Eur B</i>	456 000	2.0	37 500	0.17
<i>Eur C</i>	670 000	2.8	99 300	0.42
<i>Sear B</i>	456 000	1.5	27 300	0.09
<i>Sear D</i>	3 160 000	2.3	135 000	0.10
<i>Wpr A</i>	120 000	0.8	10 400	0.07
<i>Wpr B</i>	1 820 000	1.2	107 000	0.07
<b>Global</b>	<b>10 913 000</b>	<b>1.7</b>	<b>603 000</b>	<b>0.09</b>

<sup>a</sup> DALYs, disability-adjusted life years.

See Annex, Table A2 for country grouping into WHO subregions.

**Table 25 Total deaths and DALYs<sup>a</sup> from second-hand smoke, by population group and subregion, 2004**

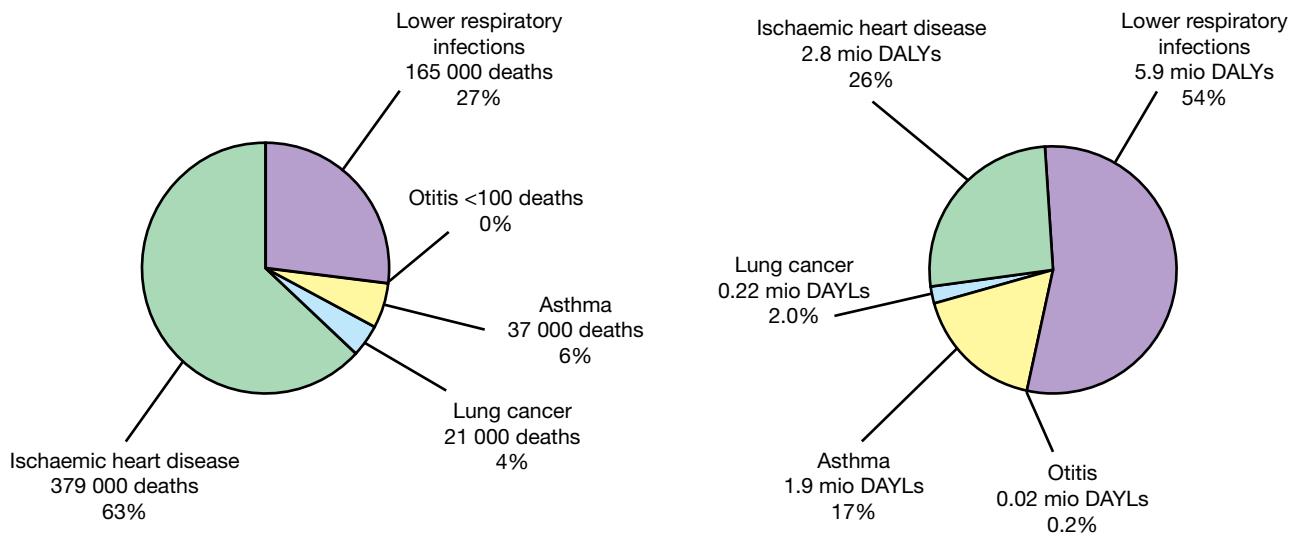
Subregion	DALYs			Deaths		
	Children	Men	Women	Children	Men	Women
<i>Afr D</i>	839 000	28 000	42 500	23 300	1 800	3 100
<i>Afr E</i>	729 000	25 100	54 600	20 100	1500	3 100
<i>Amr A</i>	30 400	58 200	37 800	76	7 100	6 300
<i>Amr B</i>	254 000	73 900	98 800	4 200	5 300	7 700
<i>Amr D</i>	68 400	10 900	10 400	1 600	560	650
<i>Emr B</i>	82 300	45 200	42 600	1 800	3 400	3 700
<i>Emr D</i>	1 124 000	118 000	197 000	30 600	8 700	15 900
<i>Eur A</i>	38 800	120 000	100 000	70	16 300	19 100
<i>Eur B</i>	208 000	96 300	152 000	5 500	11 800	20 200
<i>Eur C</i>	37 000	163 000	470 000	820	20 600	77 900
<i>Sear B</i>	201 000	58 900	196 000	4 600	5 000	17 800
<i>Sear D</i>	2 132 000	498 000	529 000	56 300	38 300	40 500
<i>Wpr A</i>	19 800	46 200	54 400	44	4 200	6 200
<i>Wpr B</i>	851 000	371 000	598 000	17 400	31 100	58 500
<b>Global</b>	<b>6 616 000</b>	<b>1 713 000</b>	<b>2 584 000</b>	<b>166 000</b>	<b>156 000</b>	<b>281 000</b>

<sup>a</sup> DALYs, disability-adjusted life years.

See Annex, Table A2 for country grouping into WHO subregions.



Figure 4 Distribution of total deaths and DALYs attributable to SHS, 2004



Number of deaths/DALYs attributable to exposure to SHS, 2004; percentage of total deaths/DALYs attributable to SHS; total = 100%.

In comparison, active smoking was estimated to cause 5.1 million deaths and 56.9 million DALYs worldwide in 2004 (WHO 2009c). Thus, the deaths and DALYs attributable to SHS represent approximately 12% of deaths and 19% of DALYs attributable to active smoking. The comparison with active smoking should be interpreted with caution, since the estimates relied on different causes of deaths and disability, datasets and methods. One important difference compared to the active smoking burden is the impact of LRI among young children, which is not caused by active smoking.

Of all deaths attributable to SHS, 28% occur among children, mostly young children in developing regions (Table 25). In terms of DALYs, children carry 61% of the burden attributable to SHS.

There are also pronounced gender differences. About 64% of adult deaths attributable to SHS occur in women. This is partly explained by the higher proportion of non-smokers among women in most regions, and so a greater proportion of the female population is considered to be “at risk” of the effects of SHS (as the disease burden from SHS has, in this study, only been estimated in non-smokers). Another consideration would be differences in SHS exposure patterns between the two genders.

There are pronounced differences in the pattern of diseases caused by SHS in the most developed regions (Amr A, Eur A and Wpr A), as compared to the other regions. The most significant difference relates to LRI that contributes to the burden in the developing regions, while this outcome has a very low impact on the the most developed regions. Asthma among children and adults, and ischaemic heart disease, in contrast, seem to have a higher impact in the developed regions.

### 3.2 Estimates of uncertainty

The sources of uncertainty for this estimate may come from various components of the estimate.

#### Exposure assessment

- **Definition of exposure**  
As highlighted in the section on exposure assessment, a number of points may add to uncertainty in estimating exposure. In this report, exposure definition needs to match the exposure definition used in health effects studies as closely as possible, and needs to be a commonly assessed variable. Exposure to SHS in childhood is, therefore, defined as having one or more parents who smoke. This is an imprecise measure, since children may be exposed to SHS outside the home, and parents who smoke may avoid exposing their children to tobacco smoke, or other adults than the parents may smoke around children. Similarly, self-reported exposure to SHS (by adults) is known to be a relatively insensitive measure – biomarker studies show that exposure to SHS is more prevalent than surveys indicate. This means the estimates of exposure to SHS that have been derived for this report are subject to error. However, by and large, these definitions of exposure match those used in the studies that established the relationships between SHS and disease outcomes, and therefore provide internal consistency to the burden of disease calculations.
- **Missing exposure data**  
Estimates of exposure may carry a large number of uncertainties due to missing data. Estimates for countries without survey data or without recent surveys, and where exposures are likely to have undergone important recent modification, are therefore, prone to greater uncertainties. Modelling of missing exposure data is addressed in more detail in Section 2.
- **Lack of exposure assessment of active smoking in children**  
Active smoking was not assessed in children. As active smoking may affect asthma in children, omission of assessment of active smoking in children may have led to an overestimate of the effect of SHS.

#### Choice of study population

- **Consideration of effects in non-smokers**  
Most effect studies cover never-smokers or non-smokers, and no effect is therefore assumed in smokers. This does not necessarily mean that there is no effect in smokers, but that the effect is difficult to study in smokers given the strong effect of active smoking. As mentioned in Section 2, isolated studies seem to indicate that the effect of SHS in smokers is similar. The effect in ex-smokers have not always been assessed, but due to the lack of information on the number of never-smokers, it was assumed that health effects due to SHS occur also in ex-smokers.

#### Disease burden of the study population

- **Uncertainties inherent to the disease burden estimate**  
The total disease burden estimate by disease also carries a number of uncertainties, which vary according to disease and region.

- **Estimation of burden in non-smokers**  
In this study, only the burden of active smoking was deduced from the total burden before estimating the burden from SHS. In addition, the non-active smoking burden was multiplied by the proportion of non-smokers. The remaining burden was considered as a proxy for the burden in non-smokers. The uncertainties introduced by doing so include all the uncertainties in the estimates of disease burden from active smoking (derived from Ezzati and Lopez, 2004b; WHO 2009c), and the assumption that the non-smoking burden is distributed equally in smokers and non-smokers.
- **Lack of disease statistics**  
No global disease statistics data were available for some outcomes that are currently established as causally related to SHS exposure, e.g. sudden infant death syndrome. Therefore, those health impacts could not be quantified at global level.

#### Effect size

- **Measures of effect size**  
The relative risks used in the population attributable fraction calculations are the best estimates of the relationship between exposure to SHS and the incidence of disease available in the current evidence base, but may be subject to random and systematic errors, only some of which can be quantified.
- **Effect of multiple exposures**  
The effects in case of multiple exposures, for example, exposure to indoor smoke from solid fuel use, have not been studied extensively. Second-hand smoke and indoor smoke from solid fuel smoke may have synergistic effects. Given that studies from China indicate similar effect sizes for SHS in populations also exposed to indoor smoke from solid fuel use, no adjustment for any co-exposures has been carried out (see discussion in Öberg et al. 2010).
- **Application of effect to other populations**  
Most of the effect studies have been conducted in Europe and the USA, and have been applied to other populations, which may have other vulnerabilities and other co-exposures, in particular in developing countries. This may have introduced additional uncertainties. However, studies performed in other parts of the world have shown similar effect sizes for certain health outcomes (Section 2.3).
- **Application of effect size to alternative exposure patterns**  
The health effects have been estimated for exposure patterns encountered in the study populations, i.e. in the USA and Europe. When applied to other populations with different exposure patterns, for example, different numbers of cigarettes smoked per day, different numbers of people per room, smokers sharing a bedroom with children, the effect size may differ. In Africa and Latin America, the mean number of cigarettes smoked daily is generally lower and we, therefore, used only the number of daily smokers rather than current smokers to estimate exposure to SHS. Also the paternal to maternal smoking ratio is much lower in certain regions when compared to most of the effect size studies, which could indicate a lower exposure intensity (and a lower effect size). However, studies of hair nicotine have shown similar means of hair nicotine across continents (see Section 2.4).

As it is difficult to quantify the major sources of uncertainty, in particular related to exposure, it is not possible to perform a proper uncertainty analysis (e.g. by Monte Carlo modelling). It would be possible to provide lower and upper estimates on the basis of statistical confidence intervals around the relative risks, but these reflect only one kind of uncertainty. We selected the estimates for relative risk from the most recent meta-analyses based on the most updated references or from the best quality individual studies identified in the literature search in case no meta-analysis was available. Many of these studies have addressed potential sources of bias, for example, by adjusting for potential confounders.

These estimates may be adjusted as additional evidence on health effects becomes available. The current absence of data on causality for some health effects excluded from this assessment does not mean that the burden of these outcomes is negligible or absent.

### 3.3 Sensitivity analysis

We have performed a sensitivity analysis to estimate the effects of certain quantifiable uncertainties. Table 26 lists some assumptions underlying the estimates (column 1) and alternative conditions (column 2), and the numbers of deaths attributable to SHS have been recalculated accordingly (column 3).

**Table 26 Effects of varying assumptions on estimates of mortality caused by second-hand smoke, 2004<sup>a</sup>**

Assumption in best estimate	Alternative condition	Effect on number of deaths in 2004 (%) (resulting alternative number)
1. Best estimate relative risk (RR)/ odds ratio (OR) from meta-analyses or original papers.	Upper 95% confidence limit of all RRs/ORs.	Increased by 29% (778 000 deaths)
	Lower 95% confidence limit of all RRs/ORs.	Decreased by 28% (433 000 deaths)
2. Current smokers are not susceptible to SHS.	Regard all non-smoking burden in smokers as influenced by SHS.	Increased by 30% (784 000 deaths)
3. The non-smoking burden in ex-smokers is influenced by SHS.	Ex-smokers are not susceptible to the effect of SHS, and 25% of non-smokers are ex-smokers.	Decreased by 17% (497 000 deaths)
4. The RR for lower respiratory infection from SHS in children is the same in developing countries as in the pooled estimate from developed countries.	The RR for LRI from SHS in developing countries is lower, and is of 1.34 (instead of 1.55), based on developing country studies only.	Decreased by 10% (545 000 deaths)
5. Parental smoking (coupled with available risk ratios) are adequate indicators of exposure for studied population.	Father to mother ratio is lower in parts of the population, and exposure intensity is not compensated for by other factors such as additional adult smokers in the household, smaller housing etc. The impacts of paternal and maternal smoking are estimated separately for the total population.	Decreased by 10% (544 000 deaths)

<sup>a</sup> Baseline number of deaths for comparison: 603 000 deaths in 2004 (Table 23).

Particular uncertainties govern the estimate of disease burden of otitis attributable to SHS. This is due to the risk estimate, given that the separate documentation of acute otitis and recurrent otitis has been difficult. In addition, no disease burden estimates for otitis were available for the age group 0–3 years (but only 0–5 years), so some extrapolations had to be made as well at that level. Using, for example, the pooled estimate of OR=1.66 by Uhari (Uhari et al. 1996) instead of the risk estimate of 1.38 used in this study would increase the number of DALYs from 25 000 to 39 000 (56%).

### **3.4 Comparisons with other studies**

#### **Comparison of results with previous national studies**

Several national studies have estimated the mortality and morbidity attributable to SHS and demonstrate variations in the estimated numbers depending on underlying assumptions (Table 27).

Certain studies have, however, used alternative methods presenting important differences with the methods proposed in this study. For example, the studies presenting results for the USA (Table 27) do not deduce the burden of active smoking and regard all deaths from the relevant diseases, even the active-smoking burden in smokers, as influenced by SHS. Such studies tend to provide significantly higher estimates of SHS burden than in this study.

Table 27 National estimates of deaths per year attributable to SHS

Country	Outcome	Attributable disease burden (deaths per year)	Reference	Year of previous study
Australia <sup>a</sup>	LCA <sup>b</sup>	10	National Health and Medical Research Council	1997
	IHD <sup>c</sup>	100	National Health and Medical Research Council	1997
Austria	LCA	35	Tredaniel et al. 1997	1990
Belgium	LCA	36	Tredaniel et al. 1997	1990
Canada	CHD <sup>d</sup>	803	de Groh and Morrison 2002	1997
Canada	LCA	347	Makomaski Illing and Kaiserman 1999	1996
	LCA + IHD	1 107	Makomaski Illing and Kaiserman 2004	1998
China	IHD	33 800	Gan et al. 2007	2002
	LCA	22 000	Gan et al. 2007	2002
Denmark	LCA	23	Tredaniel et al. 1997	1990
Finland	LCA	13	Tredaniel et al. 1997	1990
France	LCA	105	Tredaniel et al. 1997	1990
	LCA	70	Alipour et al. 2006	ND
Germany	IHD	ND	ND	ND
	CHD	8 970	Heidrich et al. 2007	2003
	CHD	2 140	Keil et al. 2005	2005
	LCA	260	Keil et al. 2005	2005
	LCA	254	Tredaniel et al. 1997	1990
Greece	LCA	42	Tredaniel et al. 1997	1990
Ireland	LCA	10	Tredaniel et al. 1997	1990
Italy	LCA	200	Tredaniel et al. 1997	1990
Luxembourg	LCA	1	Tredaniel et al. 1997	1990
Netherlands (the)	LCA	37	Tredaniel et al. 1997	1990
New Zealand	CHD	195	Woodward and Laugesen 2001	1999
	LCA	9	Woodward and Laugesen 2001	1999
Portugal	LCA	21	Tredaniel et al. 1997	1990

Country	Outcome	Attributable disease burden (deaths per year)	Reference	Year of previous study
Spain	IHD	1 017–2 947	Lopez et al. 2007	2002
	LCA	109–290	Lopez et al. 2007	2002
	LCA	89	Tredaniel et al. 1997	1990
Sweden	CHD	520	Swedish National Board of Health and Welfare 2001	1999
	LCA	21	Tredaniel et al. 1997	1990
	LCA	45	Swedish National Board of Health and Welfare 2001	1999
United Kingdom (the)	IHD	5 513	Jamrozik 2005	2003
	LCA	278	Tredaniel et al. 1997	1990
	LCA	1 532	Jamrozik 2005	2003
USA	IHD	22 669–69 553	Cal-EPA 2005	2000
	LCA	3 423–8 866	Cal-EPA 2005	2000

ND, not determined.

<sup>a</sup> Adult never-smokers exposed to partner smoking at home only.

<sup>b</sup> Lung cancer.

<sup>c</sup> Ischaemic heart disease.

<sup>d</sup> Coronary heart disease.

### Comparison of results from this report with recent reports of the effects of smoke-free legislation

Several studies have recently reported on the health impacts of laws that prohibit smoking in public places (Samet 2006). In Italy, for example, the smoke-free legislation introduced in January 2005 was followed by a reduction in hospital admissions for acute coronary events. There were reductions of 11.2% (CI 6.9–15.3%) and 7.9% (CI 3.4–12.2%) in acute coronary events in 35–64-year olds and 65–74-year olds, respectively, and no evidence of effect in the very elderly (Cesaroni et al. 2008). It is not clear how much of this change may be due to reduced smoking rates, and how much to reduction in exposure to SHS. In the USA, a study highlighted a reduction of 27% in hospitalizations for acute myocardial infarctions in a city after the introduction of a smoke-free policy in public places (Bartecchi et al. 2006).

Overall, the recent studies on reductions in hospital admissions for heart disease immediately following implementation of smoke-free legislation provide further weight to the burden of disease calculations for IHD included in this report.

### 3.5 Conclusions

We report the first systematic, internally consistent and comprehensive global estimates of the health impacts of SHS. The estimates indicate that SHS worldwide contributes to about 0.7% of the total disease burden, and represents about 12% of deaths (and 19% of DALYs) of the disease burden caused by active smoking.

Despite the uncertainties inherent in such estimates, they provide information relevant to policy-makers on the magnitude of the present problem and indicates the size of the health gain that may be achieved by reducing exposures to SHS.

Additional data collection is necessary if refinement of estimates of disease burden is required, in particular in those countries having not yet assessed the proportion of people exposed to SHS. Initiatives such as the Global Adult Tobacco Survey (GATS, joint initiative between CDC Foundation, WHO, Centers for Disease Control and Prevention and Johns Hopkins Bloomberg School of Public Health) are starting to gather such information in terms of exposure. Additional studies performed in developing countries could improve the precision of estimates under the different exposure conditions experienced in those regions. We expect that further research on other disease outcomes related to SHS will expand the evidence base, and may justify adding new diseases to the list of conditions included in the disease burden calculations.

Country-specific estimates can be developed according to the methods used for the global estimate. Full details of the method and guidance for developing country-level estimates using local or other data are available in the specific guide accompanying this global estimate (Öberg et al. 2010). Such a national estimate may be useful for defining national policies aiming at limiting SHS exposure.

Based on the current results, burden of disease from SHS contributes a significant amount of deaths due to ischaemic heart disease, adult asthma and lung cancer and also deaths due to LRI among children, especially in low-income countries, which have less resources to treat such conditions. When considering the disease burden in DALYs, taking into account also the non-fatal conditions, the estimates show the significant impact on chronic diseases, such as childhood and adult asthma, in addition to IHD in adults and LRI in children. The results suggest that the global potential to improve health by reducing SHS exposure of both children and adults is substantial.



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## 5 Annex

**Table A1 Available national data on occurrence of second-hand smoke exposure among children and adults**

Region/ country	Type of study	Year of sample	Children (%)	Adult males (%)	Adult females (%)	Total adults (%)	Reference	Source
<b>Africa (Afr D, E)</b>								
South Africa	Cross-sectional pregnant woman N=394	1992	–	–	70	–	Steyn et al. 1997	IARC 2004, Table 1.7
Democratic Republic of the Congo (the)	N=847 Pregnant women	2004–2005	–	–	8.3	–	Bloch et al. 2008	–
Democratic Republic of the Congo (the)	N=847 Respondents young child	2004–2005	6.5	–	–	–	Bloch et al. 2008	–
Zambia	N=909 Pregnant women	2004–2005	–	–	13.7	–	Bloch et al. 2008	–
Zambia	N=847 Respondents young child	2004–2005	13.1	–	–	–	Bloch et al. 2008	–
<b>North America (Amr A)</b>								
North America (24 cities study)	N=14845 8–11 yrs Smoker in household	1988–1990	50.4	–	–	–	Pattenden et al. 2006	–
Canada	Exposure in last month Non-smokers 12–14 yrs	2000–2001	33.3	–	–	–	Canadian Community Health Survey	<a href="http://www.statcan.ca">http://www. statcan.ca</a>
Canada	Exposure in last month Non-smokers >12 yrs N=131535	2000–2001	–	30.2	25.3	27.6	Canadian Community Health Survey	<a href="http://www.statcan.ca">http://www. statcan.ca</a>
Canada	Exposure in last month Non-smokers >12 yrs N=135573	2003	–	higher	lower	33	Canadian Community Health Survey	Perez 2004
USA	Cross-sectional Non-smoking women >40 years N=2326	1996–1997	–	–	14 at home, 19 at work	–	Stamatakis et al. 2002	IARC 2004, Table 1.7



Region/ country	Type of study	Year of sample	Children (%)	Adult males (%)	Adult females (%)	Total adults (%)	Reference	Source
USA	Cross-sectional Serum cotinine >17 years N=9744	1988–1991	–	43.5	32.9	–	Pirkle et al. 1996	IARC 2004, Table 1.7
USA	Cross-sectional employees N=20801	–	–	–	–	52.4	Thompson et al. 1995	IARC 2004, Table 1.7
USA, CA	Cross-sectional workers N=7301	–	–	35.8	22.9	31.3	Borland et al. 1992	IARC 2004, Table 1.7
USA, CA	Cross-sectional telephone interviews N=1579	1987–1988	–	–	–	43	Jenkins et al. 1992	IARC 2004, Table 1.7
USA, CA	Survey non-smokers ≥18 yrs Exposure at work and/or at home during the two weeks preceding the survey N=14700	1999	–	–	–	12.75	Gilpin et al. 2001	Cal-EPA 2005
USA, Portland	ECRHS 1	1990–1994	–	25.00	11.7	17.1	Personal communication	C. Janson
USA, Portland	ECRHS 2	2000	–	18.75	5.32	10.8	Personal communication	C. Janson
USA	<3 yrs At least one smoker at home	1999–2002	11.1	–	–	–	NHANES study	Surgeon General's (SG's) report
USA	3–19 yrs At least one smoker at home	1999–2002	22.6	–	–	–	–	SG's report
USA	>4 yrs >0.05 ng/ml	2001–2002	–	–	–	43	Pirkle et al. 2006	SG's report
USA	>4 years >0.05 ng/ml	1999–2000	–	–	–	51	–	SG's report
USA	>4 years >0.05 ng/ml	1991–1994	–	–	–	80	–	SG's report
USA	>4 years >0.05 ng/ml	1988–1991	–	–	–	88	–	SG's report
USA	>20 years At least one smoker at home	–	–	–	–	6.56	–	SG's report
USA	Non-smokers Exposure at home	1988–1991	–	–	–	17.4	Pirkle et al. 1996	SG's report

Region/ country	Type of study	Year of sample	Children (%)	Adult males (%)	Adult females (%)	Total adults (%)	Reference	Source
Latin America and Caribbean (Amr B, D)								
Argentina	N=796 Pregnant women	2004–2005	–	–	30.7	–	Bloch et al. 2008	–
Argentina	N=796 Respondents young child	2004–2005	13.6	–	30.7	–	Bloch et al. 2008	–
Argentina	N=50,000 households	2005	–	45.3	40.4	42.6	National Institute of Statistics and Census (INDEC) 2008	–
Uruguay	N=716 Pregnant women	2004–2005	–	–	26.5	–	Bloch et al. 2008	–
Uruguay	N=716 Respondents young child	2004–2005	18.2	–	26.5	–	Bloch et al. 2008	–
Ecuador	N=746 Pregnant women	2004–2005	–	–	12.9	–	Bloch et al. 2008	–
Ecuador	N=746 Respondents young child	2004–2005	5.2	–	12.9	–	Bloch et al. 2008	–
Brazil	N=749 Pregnant women	2004–2005	–	–	29.6	–	Bloch et al. 2008	–
Brazil	N = 749 Respondents young child	2004–2005	20.9	–	29.6	–	Bloch et al. 2008	–
Brazil	N = 23 000 Adults in the 16 Brazilian regional capitals <sup>a</sup>	2002–2003	–	–	–	21.1	Instituto Nacional de Cancer 2003	–
Guatemala	N=752 Pregnant women	2004–2005	–	–	13.2	–	Bloch et al. 2008	–
Guatemala	N=752 Respondents young child	2004–2005	5.9	–	13.2	–	Bloch et al. 2008	–
<b>Europe (Eur)</b>								
Europe (seven countries)	Case-control study N=1542	1988–1994	–	71 at work	46 at work	53 at home	Boffetta et al. 1998	IARC 2004, Table 1.7
Austria	N=3776 6–8 yrs Smoker in household	1996–1998	62.5	–	–	–	Pattenden et al. 2006	–

Region/ country	Type of study	Year of sample	Children (%)	Adult males (%)	Adult females (%)	Total adults (%)	Reference	Source
Belgium	Cohort study	1990–1994	–	58.57	48.29	53.25	ECRHS 1	C. Janson (personal communication)
Belgium	Cohort study	2000	–	33.82	32.19	32.6	ECRHS 2	C. Janson (personal communication)
Bulgaria	N=2973 7–11 yrs Smoker in household	1996	70.9	–	–	–	Pattenden et al. 2006	–
Czech	N=2962 7–11 yrs Smoker in household	1996	57.4	–	–	–	Pattenden et al. 2006	–
Denmark	N=805 3–4 yrs Exposed weekly	1996	47	–	–	–	Lund et al. 1998	–
Estonia	Cohort study	1990–1994	–	47.50	33.33	37.8	ECRHS 1	C. Janson (personal communication)
Estonia	Cohort study	2000	–	27.50	27.59	27.6	ECRHS 2	C. Janson (personal communication)
Finland	N=662 3–4 yrs Exposed weekly	1996	7	–	–	–	Lund et al. 1998	–
Finland	Cohort study N=58721 15–64 years	2000	–	14.3	13.0	–	Jousilahti et al. 2002	IARC 2004, Table 1.7
France	Cohort study	1990–1994	–	37.86	45.48	41.58	ECRHS 1	C. Janson (personal communication)
France	Cohort study	2000	–	23.14	29.94	26.45	ECRHS 2	C. Janson (personal communication)
Germany	N=1972 6–12 yrs Smoker in household	1992–1993	45.8	–	–	–	Pattenden et al. 2006	–
Germany	Cohort study	1990–1994	–	47.85	42.11	44.2	ECRHS 1	C. Janson (personal communication)
Germany	Cohort study	2000	–	28.04	26.15	27.1	ECRHS 2	C. Janson (personal communication)

Region/ country	Type of study	Year of sample	Children (%)	Adult males (%)	Adult females (%)	Total adults (%)	Reference	Source
Germany	N=6945	1998	–	60.4	51	55.2	–	Schulze & Lampert 2006
Germany	Health survey	1998	–	36.7	37.8	–	Bundes-Gesundheits survey [Federal Health Survey]	Gesundheitsberichterstattung des Bundes [Federal Health Monitoring System]
Hungary	N=3031 7–11 yrs Smoker in household	1996	55.9	–	–	–	Pattenden et al. 2006	–
Iceland	N=675 3–4 yrs Exposed weekly	1996	46	–	–	–	Lund et al. 1998	–
Iceland	Cohort study	1990–1994	–	49.57	47.55	48.4	ECRHS 1	C. Janson (personal communication)
Iceland	Cohort study	2000	–	31.30	19.01	24.5	ECRHS 2	–
Italy	N=9073 6–10 yrs Smoker in household	1995	58.2	–	–	–	Pattenden et al. 2006	–
Italy	Cohort study	1990–1994	–	62.12	48.63	54.53	ECRHS 1	C. Janson (personal communication)
Italy	Cohort study	2000	–	36.82	29.96	33.5	ECRHS 2	–
Netherlands	N=1913 7–12 yrs Smoker in household	1997–1998	58.1	–	–	–	Pattenden et al. 2006	–
Norway	N=583 3–4 yrs Exposed weekly	1996	32	–	–	–	Lund et al. 1998	–
Norway	Cohort study	1990–1994	–	37.58	30.63	34.1	ECRHS 1	C. Janson (personal communication)
Norway	Cohort study	2000	–	19.23	11.25	15.2	ECRHS 2	–
Poland	N=2643 7–11 yrs Smoker in household	1996	64.9	–	–	–	Pattenden et al. 2006	–
Russia	N=5412 8–12 yrs Smoker in household	1999	46.1	–	–	–	Pattenden et al. 2006	–

Region/ country	Type of study	Year of sample	Children (%)	Adult males (%)	Adult females (%)	Total adults (%)	Reference	Source
Slovakia	N=2531 7–11 yrs Smoker in household	1996	48.4	–	–	–	Pattenden et al. 2006	–
Spain	Cohort study	1990–1994		68.48	62.96	65.12	ECRHS 1	C. Janson (personal communication)
Spain	Cohort study	2000	–	52.17	50.60	51.24	ECRHS 2	–
Sweden	N=683 3–4 yrs Exposed weekly	1996	15	–	–	–	Lund et al. 1998	–
Sweden	Cohort study	1990–1994	–	32.4	24.49	28.57	ECRHS 1	Janson et al. 2006; C. Janson (personal communication)
Sweden	Cohort study	2000	–	10.35	5.92	8.17	ECRHS 2	–
Sweden	Cross-sectional N=30 000 <12 yrs Any parental	2003	15	–	–	–	Miljöhälsora- pport 2005 (Swedish National Board)	–
Sweden	Any exposure daily to ETS	1999	–	–	–	12	Miljöhälso- rapport 2001 (Swedish National Board)	–
Switzerland	N=2748 6–12 yrs Smoker in household	1992–1993	48.2	–	–	–	Pattenden et al. 2006	–
Switzerland	Cohort study	1990–1994	–	24.44	23.31	23.9	ECRHS 1	Janson et al. 2006; C. Janson (personal communication)
Switzerland	Cohort study	2000	–	17.04	16.54	16.8	ECRHS 2	–
The Netherlands	Cohort study	1990–1994	–	67.57	66.67	67	ECRHS 1	Janson et al. 2006; C. Janson (personal communication)
The Netherlands	Cohort study	2000	–	44.74	32.73	38.6	ECRHS 2	–
United Kingdom	Cohort study	1990–1994	–	43.41	37.78	39.83	ECRHS 1	Janson et al. 2006; C. Janson (personal communication)
United Kingdom	Cohort study	2000	–	24.62	20.94	22.2	ECRHS 2	–

Region/ country	Type of study	Year of sample	Children (%)	Adult males (%)	Adult females (%)	Total adults (%)	Reference	Source
United Kingdom	Estimated	2003	–	–	–	37 (home) 11 (work)	–	Jamrozik 2005
<b>South-East Asia (Sear)</b>								
India (Orissa)	N=886 Pregnant women	2004–2005	–	–	10.8	–	Bloch et al. 2008	–
India (Orissa)	N=886 Respondents young child	2004–2005	10.7	–	–	–	Bloch et al. 2008	–
India (Karnataka)	N=736 Pregnant women	2004–2005	–	–	19.9	–	Bloch et al. 2008	–
India (Karnataka)	N=736 Respondents young child	2004–2005	27.8	–	–	–	Bloch et al. 2008	–
Indonesia	N=60,000 households	2001	70	31.8	66.0	48.9	Central Bureau of Statistics, Jakarta, Indonesia, 2001	National Socio-Economic Survey 2001
Pakistan	N=824 Pregnant women	2004–2005	–	–	49.9	–	Bloch et al. 2008	–
Pakistan	N=824 Respondents young child	2004–2005	51.4	–	–	–	Bloch et al. 2008	–
<b>Western Pacific (Wpr)</b>								
Australia	ECRHS1	1990–1994	–	20.65	18.97	19.7	Personal communication	Janson et al. 2006; C. Janson (personal communication)
Australia	ECRHS2	2000	–	10.97	10.77	10.8	Personal communication	Janson et al. 2006; C. Janson (personal communication)
Japan	Cross-sectional Pregnant women N=16396	2002	–	–	62.1	–	Ohida et al. 2007	–
Japan	Cross-sectional Pregnant women N=19386	2006	–	–	52.7	–	Ohida et al. 2007	–

Region/ country	Type of study	Year of sample	Children (%)	Adult males (%)	Adult females (%)	Total adults (%)	Reference	Source
Japan	Cross-sectional Non-smoking women Spousal smoking N=28414	1990–1994	–	–	49.1	–	Kurahashi et al. 2008	–
China	Cross-sectional Age15–69 N=120298	1996	–	–	–	53.5	Yang et al. 1999	IARC 2004, Table 1.7
New Zealand	Exposure at home Last 7 days	2003–2004	–	–	–	20	Gillespie 2005	–
New Zealand	Exposure at home	1996	–	14.7	16.5		Woodward et al. 2001	–

–, not determined.

GYTS data are provided in Table A2.

<sup>a</sup> Survey not retained, as urban sample may overestimate national exposure.

Table A2 Estimated recent exposure to second-hand smoke for children and adult men and women

WHO subregion	Country	Children		Men		Women	
		(%)	Source	(%)	Source	(%)	Source
Afr D	Algeria	25	M1a	20	M3Ab	21	M3A
Afr D	Angola	14	Ac	9	M3Bd	14	M3B
Afr D	Benin	11	S1e	11	M3A	16	M3A
Afr D	Burkina Faso	12	S1	3	M3A	9	M3A
Afr D	Cameroon	14	A	9	M3B	14	M3B
Afr D	Cape Verde	14	A	2	M3A	7	M3A
Afr D	Chad	10	M1	2	M3A	7	M3A
Afr D	Comoros	19	S1	6	M3A	12	M3A
Afr D	Equatorial Guinea	14	A	9	M3B	14	M3B
Afr D	Gabon	14	A	9	M3B	14	M3B
Afr D	Gambia (the)	14	A	17	M3A	20	M3A
Afr D	Ghana	8	S1	0	M3A	4	M3A
Afr D	Guinea	14	A	51	M3A	33	M3A
Afr D	Guinea-Bissau	14	A	9	M3B	14	M3B
Afr D	Liberia	14	A	9	M3B	14	M3B
Afr D	Madagascar	14	A	9	M3B	14	M3B
Afr D	Mali	15	S1	3	M3A	9	M3A
Afr D	Mauritania	20	S1	5	M3A	11	M3A
Afr D	Mauritius	31	S1	23	M3A	23	M3A
Afr D	Niger	8	S1	9	M3B	14	M3B
Afr D	Nigeria	11	S1	1	M3A	6	M3A
Afr D	Sao Tome and Principe	14	A	12	M3A	17	M3A
Afr D	Senegal	15	S1	4	M3A	10	M3A
Afr D	Seychelles	23	S1	16	M3A	19	M3A
Afr D	Sierra Leone	14	A	21	M3A	22	M3A



WHO subregion	Country	Children		Men		Women	
		(%)	Source	(%)	Source	(%)	Source
Afr D	Togo	9	S1	9	M3B	14	M3B
Afr E	Botswana	22	S1	4	M3B	10	M3B
Afr E	Burundi	12	A	4	M3B	10	M3B
Afr E	Central African Republic (the)	12	A	4	M3B	10	M3B
Afr E	Congo (the)	14	S1	1	M3A	6	M3A
Afr E	Côte d'Ivoire	13	M1	2	M3A	8	M3A
Afr E	Democratic Republic of the Congo (the)	5	S2f	2	M3A	6	S2
Afr E	Eritrea	6	S1	3	M3A	9	M3A
Afr E	Ethiopia	6	S1	0	M3A	4	M3A
Afr E	Kenya	14	S1	8	M3A	14	M3A
Afr E	Lesotho	24	S1	21	M3A	22	M3A
Afr E	Malawi	7	S1	4	M3A	10	M3A
Afr E	Mozambique	15	S1	6	M3A	12	M3A
Afr E	Namibia	22	S1	14	M3A	18	M3A
Afr E	Rwanda	12	A	1	M3A	5	M3A
Afr E	South Africa	26	M1	10	M3A	15	M3A
Afr E	Swaziland	12	S1	1	M3A	6	M3A
Afr E	Uganda	17	S1	4	M3A	10	M3A
Afr E	United Republic of Tanzania (the)	12	S1	5	M3A	11	M3A
Afr E	Zambia	19	S1	3	M3A	10	S2
Afr E	Zimbabwe	24	S1	4	M3A	10	M3A
Amr E	Canada	33	S3g	30	S3	25	S3
Amr E	Cuba	50	S1	47	M3A	47	M3A
Amr E	United States of America (the)	23	S4h	13	S8i	13	S8
Amr B	Antigua and Barbuda	15	S1	13	M3B	17	M3B

WHO subregion	Country	Children		Men		Women	
		(%)	Source	(%)	Source	(%)	Source
Amr B	Argentina	44	S1	45	S9j	35	S2, S9
Amr B	Bahamas	16	S1	6	M3A	12	M3A
Amr B	Barbados	16	S1	6	M3A	29	M3A
Amr B	Belize	25	A	13	M3B	17	M3B
Amr B	Brazil	26	S1	7	M3A	22	S2
Amr B	Chile	49	S1	42	M3A	30	M3A
Amr B	Colombia	32	S1	21	M3A	22	M3A
Amr B	Costa Rica	21	S1	3	M3A	9	M3A
Amr B	Dominica	19	S1	13	M3B	17	M3B
Amr B	Dominican Republic (the)	19	S1	4	M3A	10	M3A
Amr B	El Salvador	16	S1	7	M3A	13	M3A
Amr B	Grenada	22	S1	13	M3B	17	M3B
Amr B	Guyana	26	S1	13	M3B	17	M3B
Amr B	Honduras	18	S1	0	M3A	4	M3A
Amr B	Jamaica	24	S1	7	M3A	13	M3A
Amr B	Mexico	29	S1	13	M3A	18	M3A
Amr B	Panama	19	S1	48	M3A	32	M3A
Amr B	Paraguay	26	S1	12	M3A	17	M3A
Amr B	Saint Kitts and Nevis	12	S1	13	M3B	17	M3B
Amr B	Saint Lucia	21	S1	15	M3A	19	M3A
Amr B	Saint Vincent and the Grenadines	23	S1	7	M3A	13	M3A
Amr B	Suriname	37	S1	13	M3B	17	M3B
Amr B	Trinidad and Tobago	25	S1	28	M3A	25	M3A
Amr B	Uruguay	36	S1	32	M3A	20	S2
Amr B	Venezuela (Bolivarian Republic of)	26	S1	18	M3A	20	M3A
Amr D	Bolivia (Plurinational State of)	33	S1	28	M3A	25	M3A

WHO subregion	Country	Children		Men		Women	
		(%)	Source	(%)	Source	(%)	Source
Amr D	Ecuador	4	S1	1	M3A	10	M3A
Amr D	Guatemala	18	S1	2	M3A	10	S2
Amr D	Haiti	17	S1	3	M3A	9	M3A
Amr D	Nicaragua	20	A	9	M3B	14	M3B
Amr D	Peru	30	S1	28	M3A	25	M3A
Emr B	Bahrain	33	S1	18	M3A	20	M3A
Emr B	Iran (Islamic Republic of)	37	S1	12	M3A	17	M3A
Emr B	Jordan	51	S1	68	M3Ck,j	47	M3A
Emr B	Kuwait	37	S1	24	M3A	23	M3A
Emr B	Lebanon	68	S1	23	M3A	23	M3A
Emr B	Libyan Arab Jamahiriya (the)	32	S1	30	M3B	26	M3B
Emr B	Oman	21	S1	16	M3A	19	M3A
Emr B	Qatar	27	S1	37	M3A	29	M3A
Emr B	Saudi Arabia	19	S1	17	M3A	20	M3A
Emr B	Syrian Arab Republic (the)	52	S1	46	M3A	32	M3A
Emr B	Tunisia	52	S1	61	M3A	36	M3A
Emr B	United Arab Emirates (the)	26	S1	19	M3A	21	M3A
Emr D	Afghanistan	26	S1	36	M3B	37	M3B
Emr D	Djibouti	40	S1	68	M3C	44	M3A
Emr D	Egypt	53	S1	14	M3A	18	M3A
Emr D	Iraq	39	S1	17	M3A	20	M3A
Emr D	Morocco	28	S1	19	M3A	21	M3A
Emr D	Pakistan	29	S1	19	M3A	50	S2
Emr D	Somalia	43	S1	33	M3B	27	M3B
Emr D	Sudan	17	S1	16	M3A	19	M3A
Emr D	Yemen	41	S1	68	M3C	46	M3A

WHO subregion	Country	Children		Men		Women	
		(%)	Source	(%)	Source	(%)	Source
Eur A	Andorra	53	M2l	35	M3A	35	M3A
Eur A	Austria	63	S5m	56	M3A	56	M3A
Eur A	Belgium	49	M2	34	S10n	32	S10
Eur A	Croatia	59	S1	38	M3A	38	M3A
Eur A	Cyprus	56	S1	35	M3A	35	M3A
Eur A	Czech Republic (the)	50	S1	35	M3A	35	M3A
Eur A	Denmark	47	S6o	35	M3A	35	M3A
Eur A	Finland	7	S6	14	S11p	13	S11
Eur A	France	52	M2	23	S10	20	S10
Eur A	Germany	46	S5	37	S12q	38	S12
Eur A	Greece	67	S1	68	M3C	68	M3C
Eur A	Iceland	46	S6	31	S10	19	S10
Eur A	Ireland	49	M2	17	M3A	17	M3A
Eur A	Israel	49	M2	26	M3A	26	M3A
Eur A	Italy	58	S5	37	S10	30	S10
Eur A	Luxembourg	55	M2	37	M3A	37	M3A
Eur A	Malta	51	M2	28	M3A	28	M3A
Eur A	Monaco	47	A	28	M3B	24	M3B
Eur A	Netherlands (the)	58	S5	45	S10	32	S10
Eur A	Norway	32	S6	19	S10	11	S10
Eur A	Portugal	55	M2	40	M3A	40	M3A
Eur A	San Marino	47	A	21	M3A	21	M3A
Eur A	Slovenia	48	S1	24	M3A	24	M3A
Eur A	Spain	55	M2	52	S10	51	S10
Eur A	Sweden	15	S7r	10	S10	6	S10
Eur A	Switzerland	48	S5	17	S10	17	S10

WHO subregion	Country	Children		Men		Women	
		(%)	Source	(%)	Source	(%)	Source
Eur A	United Kingdom (the)	55	M2	25	S10	21	S10
Eur B	Albania	46	S1	42	M3A	42	M3A
Eur B	Armenia	68	S1	68	M3C	68	M3C
Eur B	Azerbaijan	59	A	25	M3C	25	M3C
Eur B	Bosnia and Herzegovina	69	S1	64	M3A	64	M3A
Eur B	Bulgaria	76	S1	54	M3A	54	M3A
Eur B	Georgia	73	S1	68	M3C	68	M3C
Eur B	Kyrgyzstan	37	S1	55	M3A	55	M3A
Eur B	Poland	59	S1	52	M3A	52	M3A
Eur B	Romania	61	S1	55	M3A	55	M3A
Eur B	Serbia and Montenegro	69	S1	49	M3A	57	M3A
Eur B	Slovakia	55	S1	46	M3A	46	M3A
Eur B	Tajikistan	31	S1	48	M3B	48	M3B
Eur B	The Former Yugoslav Republic of Macedonia	64	S1	43	M3A	43	M3A
Eur B	Turkey	70	M2	68	M3C	68	M3C
Eur B	Turkmenistan	59	A	20	M3A	20	M3A
Eur B	Uzbekistan	46	M2	16	M3A	16	M3A
Eur C	Belarus	60	S1	68	M3C	68	M3C
Eur C	Estonia	59	S1	28	S10	28	S10
Eur C	Hungary	58	S1	54	M3A	54	M3A
Eur C	Kazakhstan	54	S1	52	M3A	52	M3A
Eur C	Latvia	64	S1	68	M3A	68	M3A
Eur C	Lithuania	59	S1	53	M3A	53	M3A
Eur C	Republic of Moldova	50	S1	57	M3A	57	M3A
Eur C	Russian Federation (the)	62	S1	68	M3C	68	M3C

WHO subregion	Country	Children		Men		Women	
		(%)	Source	(%)	Source	(%)	Source
Eur C	Ukraine	62	S1	68	M3C	68	M3C
Sear B	Indonesia	55	S1	32	S13	66	S13
Sear B	Sri Lanka	41	S1	16	M3A	19	M3A
Sear B	Thailand	48	S1	38	M3A	29	M3A
Sear D	Bangladesh	34	S1	45	M3A	32	M3A
Sear D	Bhutan	19	S1	41	M3B	30	M3B
Sear D	Democratic People's Republic of Korea (the)	46	A	68	M3C	45	M3A
Sear D	India	35	S1	18	M3A	15	S2
Sear D	Maldives	45	S1	45	M3A	31	M3A
Sear D	Myanmar	47	S1	49	M3A	33	M3A
Sear D	Nepal	57	S1	17	M3A	20	M3A
Sear D	Timor-Leste	46	A	41	M3B	30	M3B
Wpr A	Australia	43	M2	11	S10	11	S10
Wpr A	Brunei Darussalam	47	A	43	M3A	43	M3C
Wpr A	Japan	54	M2	57	M3A	62	S14t
Wpr A	New Zealand	45	M2	15	S15u	17	S15
Wpr A	Singapore	47	A	20	M3A	20	M3A
Wpr B	Cambodia	50	S1	25	M3A	24	M3A
Wpr B	China	71	S1	54	S11	54	S11
Wpr B	Cook Islands	52	S1	36	M3A	28	M3A
Wpr B	Fiji	46	S1	17	M3A	19	M3A
Wpr B	Kiribati	52	A	68	M3C	43	M3A
Wpr B	Lao People's Democratic Republic	46	S1	68	M3C	45	M3A
Wpr B	Malaysia	52	A	68	M3C	40	M3A
Wpr B	Marshall Islands	52	A	57	M3B	35	M3B

WHO		Children		Men		Women	
subregion	Country	(%)	Source	(%)	Source	(%)	Source
Wpr B	Micronesia (Federated States of)	48	S1	57	M3B	35	M3B
Wpr B	Mongolia	58	S1	54	M3A	34	M3A
Wpr B	Nauru	52	A	60	M3A	36	M3A
Wpr B	Niue	52	A	68	M3C	44	M3A
Wpr B	Palau	52	A	41	M3A	30	M3A
Wpr B	Papua New Guinea	38	S1	57	M3A	35	M3A
Wpr B	Philippines (the)	56	S1	39	M3A	29	M3A
Wpr B	Republic of Korea	56	S1	68	M3C	41	M3A
Wpr B	Samoa	49	S1	68	M3C	44	M3A
Wpr B	Solomon Islands	52	A	57	M3B	35	M3B
Wpr B	Tonga	52	A	68	M3C	46	M3A
Wpr B	Tuvalu	65	S1	68	M3C	39	M3A
Wpr B	Vanuatu	41	S1	68	M3C	40	M3A
Wpr B	Viet Nam	55	S1	48	M3A	32	M3A

<sup>a</sup> M1: modelled exposure (see Table 3 for model details), based on parental active smoking, adjusted for removal of occasional smokers (27% for Africa and 25% for Latin America and the Caribbean (Amr B, D)).

<sup>b</sup> M3A: modelled exposure based on men's smoking; SHS (men) = 0.0275 \* (male smoking prevalence) 2.00 ; SHS (women, developing countries) = 0.612 \* (male smoking prevalence) 1.10 ; SHS (women, developed countries) = 0.0019 \* (male smoking prevalence) 2.72 ; men's active smoking rate from Mpower (WHO 2008a), WHO infobase (WHO 2008c) or ACS country profiles (ACS 2008).

<sup>c</sup> A: subregional GYTS average.

<sup>d</sup> M3B: modelled exposure based on men's smoking (as above); men's active smoking rate origin from regional average.

<sup>e</sup> S1: from Global Youth Tobacco Survey.

<sup>f</sup> S2: from Bloch et al. 2008.

<sup>g</sup> S3: from Canadian community health survey.

<sup>h</sup> S4: from Surgeon General report, 2006.

<sup>i</sup> S8: from Cal-EPA 2005.

<sup>j</sup> S9: from Argentinian national survey on risk factors, 2008.

<sup>k</sup> M3C: model data capped at 68%.

<sup>l</sup> M2: modelled exposure (see Table 3 for applied model details), based on parental active smoking.

<sup>m</sup> S5: from Pattenden et al. 2006.

<sup>n</sup> S10: from ECRHS (Janson et al. 2006).

<sup>o</sup> S6: from Lund et al. 1998.

<sup>p</sup> S11: from IARC Table 1:7 in IARC 2004.

<sup>q</sup> S12: from German national health survey 1998.

<sup>r</sup> S7: from Swedish environmental health report 2001.

<sup>s</sup> S13: from Indonesian national socio-economic survey 2001.

<sup>t</sup> S14: from Ohida et al. 2007.

<sup>u</sup> S15: from Woodward and Laugesen 2001.

Figures have been computed by WHO to ensure comparability; thus they are not necessarily the official statistics of Member States, which may use alternative rigorous methods. Data based on modelling (M) should be considered as indicative only.





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