Mortality and morbidity from coronary heart disease attributable to passive smoking

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Aims Passive smoking is associated with increased risk of coronary heart disease (CHD). This study estimates CHD mortality and morbidity attributable to passive smoking in Germany and demonstrates variations in the number of estimated deaths depending on underlying assumptions.

Methods and results Prevalence of passive smoking from the German National Health Survey, CHD deaths from national mortality statistics, number of incident CHD cases, and relative risks from meta-analyses were used to estimate mortality and morbidity from passive smoking applying the concept of population attributable risk. Sensitivity analyses were carried out to investigate the impact of different assumptions in terms of exposure definition, relative risk, and population at risk on estimated mortality. Exposure to environmental tobacco smoke (ETS) at home accounts for 2148 [approximate 95% confidence interval (CI) 1471–2736] deaths from CHD and 3776 (95% CI 2588–4800) incident CHD cases among non-smokers every year in Germany. In sensitivity analyses, consideration of exposure to ETS at work and at any location yielded 2597 (95% CI 1784–3295) and 8970 (95% CI 6252–11 243) attributable CHD deaths, respectively. Applying different populations at risk showed a range of 1174 (95% CI 803–1494) to 13 792 (95% CI 9655–17 225) attributable deaths from CHD.

Conclusion The estimated burden of passive smoking heavily depends on the definition of underlying parameters. Using an evidence-based approach reveals a substantial burden of passive smoking in terms of CHD mortality and morbidity reflected by six CHD deaths and 10 incident CHD cases every day in Germany.

KEYWORDS Coronary heart disease; Passive smoking; Population attributable risk; Mortality; Morbidity

Introduction Over the past two decades, compelling evidence emerged that exposure to environmental tobacco smoke (ETS), also referred to as passive smoking, is associated with an increase in risk of coronary heart disease (CHD) by 25% among non-smokers.1–4 Both acute (via platelet aggregation and endothelial dysfunction) and long-term (via atherosclerosis) effects of ETS exposure have been shown to contribute to the increase in CHD risk.5,6 Although the magnitude of this association is rather small, the impact of passive smoking on population health, however, may be substantial as passive smoking is a common exposure and CHD is the most common cause of death in industrialized countries.7 The burden of passive smoking in terms of attributable mortality and morbidity has rarely been estimated and showed great variations. For example, in UK passive smoking might account for about 5500 annual deaths from CHD.8 In the United States, 35 000–62 000 deaths due to CHD may be attributable to passive smoking.9 A number of assumptions and judgements lie behind these figures. Estimating the number of deaths attributable to passive smoking is complex since there are multiple settings of exposure, different reported relative risks, and different definitions of the population at risk (never smokers, former smokers, current smokers), which substantially influence resulting figures.

This study aimed to estimate CHD mortality and morbidity associated with passive smoking in Germany using data from the most recent representative German National Health Survey. We carried out sensitivity analyses to investigate the range of estimated mortality figures using different assumptions in terms of exposure definition, relative risk, and population at risk.

Methods CHD mortality and morbidity attributable to ETS were calculated using (i) the prevalence of passive smoking in non-smokers, (ii) relative risk (RR) of CHD caused by passive smoking among non-smokers, and (iii) the number of CHD deaths and incident CHD events in Germany.

Exposure to passive smoking and risk of coronary heart disease associated with passive smoking

Data on the prevalence of active and passive smoking were derived from the German National Health Survey 1998, a representative survey among 3450 men and 3674 women aged 18–79 years.10 The general question 'Did you smoke in the past or are you currently...
smoking?” with diverse subcategories allowed to differentiate between never smokers, ex-smokers, and current smokers. For main analyses, ex-smokers and never smokers were combined to non-smokers since disease risk falls quickly after quitting smoking and the risk for ex-smokers is closer to the risk for never smokers than for current smokers. Passive smoking was assessed by the following question: ‘In the daytime or at night, do you frequently stay in rooms where people smoke?’ Participants were asked to give multiple answers to this question with respect to exposure at home, at workplace, and at any other locations. For the age group ≥80 years, we used prevalences from age group 75–79 years. A relative risk of CHD (ICD 10 I20–I25) of 1.25 associated with passive smoking was adopted from meta-analyses.

Mortality and incidence of coronary heart disease

Mortality from CHD was derived separately for men and women in 5-year age groups from the official German mortality statistics for the year 2003.

The number of incident CHD cases in Germany was estimated using the WHO DisMod II software (http://www.who.int/evidence/dismod/). This programme is based on a mathematical model for a multi-state life table that describes a single disease. The model is based on population size and all-cause mortality and requires three disease input variables to calculate epidemiological measures of the specific disease. We used CHD mortality as the first input variable. These data were obtained from official German mortality statistics 2003 for 5-year age groups and both sexes. The second input variable was prevalence of CHD, which was estimated as the proportion of participants of the German National Health Survey 1998 who gave a positive answer to the question ‘Did you ever have a CHD event?’ As the German National Health Survey included only participants younger than 80 years, prevalence for older subjects was extrapolated using estimates from the Rotterdam Study. For our purpose, individual data from the German National Health Survey and the published aggregated data from the Rotterdam Study were analysed jointly by a weighted logistic regression. Prevalence of CHD was modelled as a function of age and study region (Germany vs. Rotterdam), which allowed to forecast CHD prevalence in older ages in Germany in 5-year age groups for both sexes. Since the prevalent state of CHD as defined above does not allow for remission, we used a remission rate equal to zero as the third input variable. Thus, incidence as estimated with the DisMod II approach, refers to first-in-a-lifetime events only, whereas, on the other hand, mortality figures do not differentiate between first and recurrent events. Consequently, due to this inevitable methodological difference, all following analyses and estimations of attributable mortality refer to first and recurrent events, whereas incidence figures refer to first events only. Case-fatality cannot be calculated from these analyses.

Sensitivity analyses of passive smoking and coronary heart disease

In sensitivity analyses, exposure to ETS was defined as (i) ETS exposure at home; (ii) ETS exposure at home and/or at work; and (iii) ETS exposure at any location (home, workplace, other). We further explored the effect of considering different populations at risk from passive smoking, i.e. (i) never smokers only, (ii) never and former smokers, and (iii) never, former, and current smokers.

Statistical methods

The estimated number of attributable CHD deaths and incident CHD events was derived in three steps:

(i) We calculated CHD specific population attributable risks (PAR) for passive smoking using the standard formula \( \text{PAR} = \frac{p \times (RR - 1)}{1 + p \times (RR - 1)} \) where \( p \) is the prevalence of exposure to ETS and \( RR \) is the relative risk of CHD from passive smoking. PAR was computed separately for men and women in 5-year age groups using an RR of 1.25 for CHD due to passive smoking across all age groups.

(ii) We computed the total number of CHD deaths and incident CHD cases among non-smokers in Germany as a basis for calculating attributable mortality and incidence due to passive smoking. Because official mortality statistics do not differentiate between smokers and non-smokers, these figures were estimated using the formula \( n = (1 - p) \times m \), where \( m \) is the number of all CHD deaths from the official German mortality statistics or estimated number of incident CHD cases from the DisMod II approach described above. \( RR \), the relative risk of CHD due to active smoking of 2.0 was used across both sexes and all age groups.

(iii) The estimated number of attributable CHD deaths and incident CHD events was computed applying the PAR as specified above to age- and sex-specific CHD mortality and incidence among non-smokers. These figures were added up to the total number of deaths and incident cases attributable to passive smoking.

To account for the uncertainty inherent in estimated figures we used two approaches. The uncertainty inherent in our estimates comes from different sources: (i) random error of the estimates of relative risks; (ii) random error of the estimates of the prevalence of passive smoking; (iii) uncertainty in the estimation of number of CHD deaths and CHD incidence among non-smokers; (iv) uncertainty from the DisMod II approach. First, we performed sensitivity analyses to quantify the impact of different assumptions in terms of exposure and population at risk as specified above on the estimated burden of passive smoking. Secondly, assuming that the random error of the estimates of the relative risk is the major contribution to the uncertainties of our estimates and that Items (ii)–(iv) above are negligible, we calculated approximate 95% CI using the lower (1.17) and upper (1.32) confidence limits of the RR of 1.25 for CHD due to passive smoking reported from meta-analysis.

All computations and data management were carried out with the SAS software package (version 9.1), except for the estimation of CHD incidence, which was carried out with the WHO DisMod II software.

Results

Estimates for mortality and morbidity from passive smoking in Germany

Overall, 2148 (95% CI 1471–2736) CHD deaths every year in Germany are attributable to passive smoking (Figure 1). Sixty-six percent of deaths (1423; 95% CI 975–1812) occur among women. Eighty-eight percent of deaths (1883; 95% CI 1287–2396) occur in people aged ≥65 years. With regard to incidence, 3776 (95% CI 2588–4800) new CHD cases per year in Germany are attributable to passive smoking (Figure 2). Fifty-nine percent of incident cases occur among women (2220; 95% CI 1522–2820), and 64% (2432; 95% CI 1665–3094) in people aged ≥65 years.

Sensitivity analyses for mortality from coronary heart disease

Prevalence of ETS exposure among never smokers and current non-smokers is about 12%, considering exposure at home only and almost doubles each with incorporating also
ETS exposure at work and ETS exposure at any location (Table 1). Depending on relative risk and exposure definition, the estimated number of deaths ranges from 803 to 5870 among never smokers, and from 1471 to 11 243 among current non-smokers, respectively. If the total population including smokers is considered to be at risk, prevalence of exposure to ETS ranges from 27 to 61% depending on exposure definition; and the estimated number of deaths amounts to 4032–17 225.

Discussion

This study indicates that about 2100 deaths from CHD and about 3800 incident CHD cases per year in Germany may be attributed to exposure to passive smoking. In our point of view, this figure is based on the most valid approach, namely considering only exposure to ETS at home and using relative risks that were derived from published meta-analyses. Sensitivity analyses revealed a wide range of estimated mortality from CHD that may differ up to 20-fold in extreme cases underlining the importance of underlying assumptions. Therefore, the estimated figures should not be interpreted as precise estimates but rather as order of magnitude or range.

Our results show that, overall, almost two-thirds of attributable CHD mortality and morbidity occur among women, particularly in higher age. Non-smoking women were more often exposed to ETS than men (14 vs. 10%) and thus more women are at risk from ETS. Because women have a higher life expectancy than men and develop CHD, on average, 10 years later than men, most of the cases in age > 75 years are to be found in women. Conversely, attributable mortality under the age of 75 years occurs predominantly among males.

The reported 2100 CHD deaths and 3800 incident CHD cases related to passive smoking in Germany are likely to be conservative estimates that underestimate the real burden of passive smoking in the population. For these estimates we did not consider exposure to ETS at work since most of the evidence comes from studies that used spousal smoking or home exposure. However, workplace exposure to ETS has been shown to increase the risk of heart disease. Including exposure to ETS at work in our study would result in 21% increase in CHD deaths per year (2600 cases) attributable to passive smoking in Germany. Furthermore, these numbers were estimated without considering exposure to ETS at locations other than home or work, which is likely to have an effect on health. Though the reported figures are likely to be conservative they are not the most conservative estimates possible. Particularly considering never-smokers only, rather than never- and ex-smokers combined, would result in lower estimates. But as pointed above, there are good reasons to assume that ex-smokers are similar to never-smokers with respect to CHD risk from ETS.

A rough comparison of our results with figures from the United States and the UK can be carried out assuming that mortality rates and age distributions do not differ substantially between these countries and Germany. After adjustment for the different population size it can be seen that the population attributable mortality risks related to passive smoking in our study are lower than those reported from the United States and the UK. The US figures of 35 000–62 000 CHD deaths were estimated for the year 1985. Considering declining mortality rates for CHD, 47 000 attributable CHD deaths were estimated for the year 1994. As CHD mortality rates decreased further since that time, it is likely that the number of attributable CHD deaths is lower today. A main characteristic explaining much of the difference to our study is the high prevalence of passive smoking in the US studies which amounted to up to 61% in men and 76% in women including spousal, home, and workplace exposure. It has been shown that the prevalence of passive smoking in the US sharply declined in recent years. The US estimates further considered a so-called background environmental exposure from sources other than spousal smoking that accounted for 60% of attributable deaths from CHD. Our sensitivity analyses showed that the estimated attributable mortality is very sensitive to exposure definition and prevalence. Considering
any exposure to passive smoking in our analyses that would correspond best to background environmental exposure resulting in a four-fold increase of estimated number of deaths. In the UK, 5500 deaths from CHD have been reported to be attributable to passive smoking.6 Only home and workplace exposure to ETS were considered in these analyses. Prevalence of passive smoking, unlike to our study, had to be estimated indirectly, since no representative data on ETS exposure were available for the UK. The estimated prevalence of passive smoking at home, however, was very similar to our study (13 vs. 12%). The main difference to our estimations is that in the UK study only non-smokers but also active smokers were considered as population at risk although there is no evidence that active smokers are at additional risk from passive smoking. The evidence of the detrimental effects of passive smoking comes from studies that included only non-smokers. The mechanisms by which active and passive smoking damage health are very similar.26,27 There is a non-linear dose–response relationship between risk of CHD and smoking. The risk of CHD rapidly increases by 50% with smoking five cigarettes per day, and the risk increases much slower with higher daily cigarette consumption.27 This suggests that the pathway leading to disease may be rapidly saturated after exposure to active smoking, and passive smoking may not add substantially to the effect of active smoking. Our sensitivity analyses showed that including active smokers leads to an almost three-fold increase in the estimated mortality from passive smoking as compared with considering only non-smokers. In our view, considering active smokers at risk from passive smoking is not meaningful and will produce inflated figures that overestimate the impact of passive smoking on health.

Our study is subject to several limitations. As described above, there is some inevitable uncertainty inherent in estimated figures. To address uncertainty, we performed sensitivity analyses and provide approximate 95% CI. We used prevalence data from the German National Health Survey 1998; the most recent survey that provided representative, population-based data on passive smoking. However, these data are already some years old and exposure to ETS may have changed over the last years. Therefore, we explored changes in smoking habits between 1998 and 2003, the relevant year for our estimations. A further German National Health Survey from 2003 which assessed active but not passive smoking revealed virtually no changes in smoking habits28 indicating that passive smoking did not change substantially either. Other published results on passive smoking support this conclusion.29 Exposure data for the age group ≥80 years were extrapolated from the age group of 75–79 years. However, as changes in smoking habits are less likely in these age groups, this procedure is unlikely to lead to misclassification. Since national mortality statistics do not differentiate between smokers and non-smokers, we had to calculate mortality rates among non-smokers indirectly. We used a single relative risk across all ages although relative risks may differ depending on age.30 Currently, there are no age-specific relative risks for coronary heart disease and passive smoking available.

### Conclusions

We demonstrated that a reasonable, evidence-based choice of relative risk, exposure location and thereby exposure prevalence, and population at risk are key factors for the estimation of attributable CHD-mortality and morbidity due to passive smoking. Depending on these choices, the range of estimated number of deaths may vary up to 20-fold in extreme cases. These extreme figures cannot be seen as realistic estimates. Therefore, estimates for CHD mortality attributable to passive smoking reported in the literature should be interpreted very cautiously.

Applying the best available evidence resulted in about 2100 CHD deaths and 3800 incident CHD cases due to...
passive smoking every year in Germany. This is likely to be a conservative estimate that may underestimate the real burden of passive smoking. The morbidity and mortality burden of passive smoking reflected by about six CHD deaths and 10 incident CHD cases every day in Germany is substantial and action to protect non-smokers from passive smoking is overdue.

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**Conflict of interest:** none declared.

**Appendix**

The number of CHD deaths and incident CHD cases among non-smokers were estimated from the number of all CHD deaths or CHD cases, respectively, and further information. Let \( I \), \( I_0 \), and \( I_1 \) denote the incidence rates for CHD (fatal or fatal plus non-fatal) in a certain population of size \( n \) as a whole, and in smokers and non-smokers within this group, respectively. If \( p_s \) denotes prevalence of smoking, we may write \( I = (1 - p_s)I_0 + p_sI_1 \). With \( RR_s = I_1 / I_0 \), the RR for CHD due to smoking, we get \( I = (1 - p_s)I_0 + p_sRR_sI_0 \). Multiplying by \( n \) yields \( m = nI = nI_0 \left[ 1 - p_s + p_sRR_s \right] \). Rearranging this equation yields the number of CHD deaths or CHD events in a population of size \( n \) that would occur if the incidence rate for everyone would be that of non-smokers, i.e. \( nI_0 = m / \left[ 1 - p_s + p_sRR_s \right] \). For the subgroup of non-smokers, which has the size \( (1 - p_s)n \) rather than \( n \), this is \( m_0 = (1 - p_s)m / \left[ 1 + p_s(RR_s - 1) \right] \).

**References**