How many deaths are caused by second hand cigarette smoke?

Alistair Woodward, Murray Laugesen

Abstract

***Objectives*—To estimate the number of deaths attributable to second hand smoke (SHS), to distinguish attributable and potentially avoidable burdens of mortality, and to identify the most important sources of uncertainty in these estimates.**

***Method*—A case study approach, using exposure and mortality data for New Zealand.**

***Results*—In New Zealand, deaths caused by past exposures to second hand smoke currently number about 347 per year. On the basis of present exposures, we estimate there will be about 325 potentially avoidable deaths caused by SHS in New Zealand each year in the future. We have explored the effect of varying certain assumptions on which the calculations are based, and suggest a plausible range (174–490 avoidable deaths per year).**

***Conclusion*—Attributable risk estimates provide an indication for policy makers and health educators of the magnitude of a health problem; they are not precise predictions. As a cause of death in New Zealand, we estimate that second hand smoke lies between melanoma of the skin (200 deaths per year) and road crashes (about 500 deaths per year).**

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Keywords: second hand smoke; attributable risk; mortality; lung cancer; heart disease; stroke; sudden infant death syndrome

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Much has been written about disease risks and second hand smoke (SHS)[1–4] but less well-documented is the effect of SHS on populations (the attributable risk or the burden of illness caused by SHS). Measures such as population attributable risk provide a guide to the size of the problem caused by a particular environmental exposure.[5] The number of attributable deaths or cases of disease is not necessarily the same as the numbers that are avoidable.[6] But these calculations give a picture of the likely magnitude of the burden of illness, and in this way assist decisions on the allocation of resources to improve public health.

In New Zealand the only previous assessment of mortality attributable to SHS was carried out in 1989 by Kawachi and colleagues.[7] They estimated 273 deaths per year from heart disease and lung cancer (range 112–442 based on the 95% confidence intervals (CI) around the relative risk estimates). We repeated these calculations including more recent information on exposures to SHS and disease risks. We have attempted to account for changing patterns of exposure over time, and report separately on present deaths caused by past exposures (attributable mortality) and future (potentially avoidable) deaths caused by current exposures to SHS. We explore the assumptions that underlie these calculations and propose a range within which the true burden of mortality caused by second hand smoke is likely to fall.

Methods

Information on disease risks from SHS was sought by searching the scientific literature using key words "tobacco smoke pollution", "second hand smoke", "environmental tobacco smoke", and "passive smoking". We used Medline, bibliographies of major reviews in the field, and discussions with colleagues. Smoking rates in the New Zealand population were obtained from reports on New Zealand censuses and other historical survey data.[8] Estimates of current exposures to SHS were based on a national survey conducted in 1996 for the Ministry of Health.[9] The New Zealand Health Information Service provided rates and numbers of deaths in New Zealand by cause.[10]

We used "no exposure to SHS" as the counterfactual and calculated population attributable risk (PAR) using the standard formula[11]: $PAR = P_e (RR - 1) / P_e (RR - 1) + 1$ where P_e is prevalence of exposure and RR is relative risk of disease.

We estimated that 55% of males and 53% of females dying from coronary heart disease are never- or ex-smokers, based on results from the Auckland Heart Health Study conducted between 1986 and 1993.[12] Similar proportions for stroke were obtained from the study by Bonita and colleagues,[13] and we have assumed the proportion of lung cancer deaths among never smokers is 8%, as reported by Kawachi and associates.[7] The proportion of adults (15–64 year olds) in paid employment in New Zealand has been close to 70% for the last decade, with rather higher proportions among men compared to women. Consequently we have made the conservative assumption that 80% of male deaths and 60% of female deaths occur among individuals who were during their lifetime at risk of exposure to SHS in the workplace.

Results

We do not know how total mortality varies with changing levels of exposure to SHS. Therefore calculations of attributable risk must begin

Table 1 Potentially fatal conditions associated with second hand smoke (SHS)

Condition	*Strength of evidence of cause and effect*	*Comments*	*Estimate of time from exposure to death*
Lung cancer	+++++	Numerous aetiological studies and weight of evidence reviews reporting increased mortality following exposure to SHS	10–20 years
Coronary heart disease	+++++		5–10 years
Sudden infant death syndrome	++++	High quality research reports increased death rates with SHS (this includes studies from New Zealand)	< 1 year
Stroke	+++		5–10 years
Asthma	++	Many studies report increased morbidity with exposure to SHS, particularly in vulnerable groups, but little evidence of effects on mortality	
Lower respiratory infections	++		
Low birth weight	++		
Meningococcal disease	+	Literature is limited: association with SHS has not been demonstrated consistently in a range of settings	
Breast cancer	+		
Other respiratory tract cancers	+		
Cancer of cervix	+		

with the question of which fatal diseases are influenced by SHS. Table 1 lists conditions which are potentially life threatening and have been associated with SHS. The first group includes conditions that have been closely studied in many populations using a variety of study methods. The second group includes two conditions that have been less extensively researched, but large well conducted studies in New Zealand populations report associations between SHS and fatal outcomes.[13 14] The evidence of an association with sudden infant death syndrome (SIDS) is stronger than that for stroke, but in both instances the New Zealand studies are consistent with reports from other countries.[15–17] Non-causal explanations are, in our view, unlikely, but estimates of the strength of the effect of SHS must be treated with caution. Third, we have listed conditions that are well known as causes of morbidity caused by exposure to SHS, but there is scant evidence of an effect on deaths. In the final group are conditions that have been linked with SHS, but the literature in these instances is limited.

The first two groups in table 1 were included in the calculation of deaths caused by SHS. We provided for each condition our best estimate of the increased risk of death caused by SHS (the source of these figures is shown in the table) and an estimate of the average time from exposure to death, based on what is known about the effects of active smoking.

In table 2 we report what is known about historical exposures to SHS in New Zealand, and these data are combined with the relative risk estimates to calculate numbers of deaths (table 3). The first figure in the final column refers to current deaths related to exposures to SHS in the past. We have also calculated the deaths that will occur in the future, associated with present exposures to SHS. This latter figure, which represents potentially avoidable deaths, assumes that the lag time between exposure and mortality has elapsed for all conditions. In other words, the number we have calculated refers to the deaths avoided, from about 2015 onwards, if exposure to SHS ceased immediately.

Discussion

Attributable risk estimates should be regarded as a guide to policy decisions rather than a precise prediction of the number of lives that will be saved by public health interventions. For one thing, the number of attributed deaths depends on the order in which the component causes of different diseases are removed.[18] We have assumed that control of SHS is the first intervention in each instance although in practice this may not be the case. For example, parents may change the position in which babies are put to sleep sooner than change their smoking patterns. If this happens the total number of sudden infant deaths will fall, as will the number attributable to SHS, although these latter deaths will make up a greater fraction of the total (that is, the PAR% will rise).

Estimates of attributable mortality contain other uncertainties, some of which are quantifiable. In table 4 we have listed what we see as the most important assumptions underlying our estimate of deaths caused by SHS. The values in the first column include key inputs to the attributable and avoidable mortality equations. In the next column we have laid out plausible, alternative conditions, and have then recalculated the numbers of deaths caused by SHS.

We have treated ex-smokers as susceptible to the effects of SHS on heart disease and stroke. There is no a priori reason why this group should be immune, and there are some data in support. The US Nurses Health Study reported an 82% increase in risk of heart attack (95% CI 0.76 to 4.34) among ex-smokers occasionally exposed to SHS and a relative risk of 2.38 (95% CI 0.98 to 5.79) among those regularly exposed to SHS.[19] Howard and colleagues[20] measured the thickness of the wall of the carotid artery using ultrasound, and found among never smokers that the increase in vessel wall thickness over three years was 22% greater in those reporting exposure to SHS: among past smokers there was a similar effect (an 18% greater increase associated with SHS exposure, after adjustment for potential confounding factors). We know of no empirical studies and no theoretical reasons indicating that time since quitting is likely to affect the relation between passive smoking and risk of heart disease, although the absolute risk of heart disease will

tend to be higher in recent ex-smokers. For this reason we have treated all ex-smokers as susceptible to the effects of SHS, but acknowledge the uncertainty that surrounds this assumption because of paucity of data.

What age groups are at risk of disease caused by SHS? Kawachi and colleagues[7] argued that most of the effects of SHS on heart disease are likely to be acute and transitory and therefore work exposures would have little impact on

Table 2 Exposure to second hand smoke (SHS) in New Zealand

Date	*Population*	*Definition of exposure*	*Prevalence of exposure to SHS*	*Reference*
1986	Adult non-smokers 35–64 years	Exposed to SHS in their homes	12.7% (men) 16.1% (women)	Kawachi et al[7]
1987-8	Adults, 35–64 years, never smokers	Exposed to SHS at work	33.6% (men) 23.4% (women)	Kawachi et al [7]
1991	Indoor workers	Exposed to SHS during working hours Exposed during tea and lunch breaks	19% 39%	Public Health Commission[31]
1991-3	Infants, at 2 month visit	Mother smokes Father smokes	22.8% 27.7%	Mitchell et al[14]
1993	Adult non-smokers, sample of multi-industry workforce	Regular exposure to SHS at work or at home	49% (men) 33% (women) 43% (European) 60% (Maori) 56% (Pacific Islands)	Whitlock et al[27]
1996	Adults exposed to SHS	Mean number of hours per day exposed to others smoke	3.7 hours (at work) 3.4 horrs (away from work) 3.7/3.1 (European or other ethnicity) 3.7/4.4 (Maori)	Ministry of Health [9]
1996	Adult non-smokers	Live with someone who regularly smokes around them inside the home	14.7% (men) 16.5% (women)	Ministry of Health [9]
1996	Indoor workers (non-smokers)	Exposed to SHS during working hours Exposed only during tea and lunch breaks	19.1% (men) 6.2% (women) 24.0% (men and women)	Ministry of Health[9]
1997	Secondary school children aged 14–15	Exposed to smoke at home	35% (all students) 54% (Maori)	ASH survey of secondary school students. Unpublished report
1997	Infants visited by child health nurses at 3 months	Mother smoked during pregnancy	26% (all mothers) 49% (Maori mothers)	Tuohy P, *et al*, 1997. SIDS risk factor final report.*

*Unpublished report to the Ministry of Health.
ASH, Action on Smoking and Health; SIDS, sudden infant death syndrome.

Table 3 Deaths caused by second hand smoke (SHS), New Zealand

	Prevalence of exposure to SHS (%): past/current	*Relative risk (95% CI)*	*Population attributable risk (%): present/future*	*Total deaths per year*	*Deaths among never smokers and ex-smokers**	*Deaths caused SHS: current/future*
1. Lung cancer (ICD-9 162)						
At home						
Men	12.7/14.7	1.24 (1.13 to 1.36)[32]	0.029/0.033	882	70.6†	2.1/2.4
Women	16.1/16.5	1.24	0.037/0.033	530	42.4†	1.6/1.6
At work						
Men	33.6/19.1	1.24 (1.13 to 1.36)[33]	0.075/0.056	882	70.6†	4.2/2.5
Women	23.4/6.2	1.24	0.053/0.014	530	42.4†	1.4/0.4
2. Coronary heart disease (ICD-9 410–414)						
At home						
Men	13.7/14.7	1.30 (1.22 to 1.38)[23]	0.039/0.041	3614	1987.7	78.5/84.0
Women	16.3/16.5	1.30	0.043/0.041	2755	1460.2	68.1/68.9
At work						
Men	26.3/19.1	1.21 (1.04 to 1.41)[25]	0.057/0.049	1823‡	1002.7	42.0/30.9
Women	13.8/6.2	1.21	0.037/0.012	657‡	348.2	5.9/2.7
3. Stroke (ICD-9 430–438)						
At home						
Men	13.7/14.7	2.10 (1.33 to 3.32)[13]	0.128/0.134	325‡	209.6	27.5/29.2
Women	16.3/16.5	1.66 (1.07 to 2.57)	0.091/0.085	301‡	194.2	18.9/19.1
At work						
Men	26.3/19.1	2.10 (1.33 to 3.32)[13]	0.239/0.213	325‡	209.6	37.6/29.1
Women	13.8/6.2	1.66 (1.07 to 2.57)	0.109/0.036	301‡	194.2	9.7/4.6
4. Sudden infant death (ICD-9 798)						
Males and females combined	19.7§/19.7	5.3 (2.71 to 10.28)[14]	0.459/0.459	109		50.0/50.0
Total						347.3/325.2

Past prevalence of exposure to SHS refers to circa 1985 (lung cancer), 1993 (heart disease and stroke), and 1999 (sudden infant death syndrome—SIDS). Future deaths refer to circa 2015 (lung cancer), 2007 (heart disease and stroke), and 2001 (SIDS).
Deaths per year based on 1997 data, with the exception of SIDS (1991-93).
*It is assumed that 80% of adult males and 60% of adult females are in paid employment, and therefore at risk of exposure to SHS at work. These fractions have been applied to the estimated numbers of deaths among non-smokers to calculate the number potentially susceptible to the effects of SHS in the workplace.
†Never-smokers only; ‡deaths before age 75; §exposure defined as maternal smoking two months after birth, no bed-sharing.
ICD-9, International Classification of Disease, 9th revision.

Table 4 Effects of varying key assumptions on estimates of future mortality caused by second hand smoke (SHS)

Assumption	*Alternative conditions*	*Effect on number of future deaths per year*
1. SHS raises risk of stroke death (RR for men 2.1, for women 1.7)	SHS does not affect the risk of dying from stroke	Decreased by 26% (new n=243.2)
2. Best estimate RR from meta-analyses or original papers	Upper 95% confidence limit of all RRs Lower 95% confidence limit of all RRs	Increased by 52% (n=490.4) Decreased by 46% (n=174.6)
3. Ex-smokers susceptible to effects of SHS	Ex-smokers not susceptible to effects of SHS: proportion of deaths among never smokers: heart disease 0.22 (males), 0.28 (females)[12]; stroke 0.31[13]	Decreased by 45% (n=180.0)
4. Current smokers not susceptible to SHS	Regard all deaths from lung cancer, heart disease, and stroke as influenced by SHS	Increased by 87% (n=606.8)
5. Workplace exposure to SHS affects risk of heart disease beyond age 65	Workplace exposure to SHS does not affect risk beyond age 65	Decreased by 6% (n=306.4)
6. Exposure to SHS at work excludes exposures at tea and lunch breaks, $p_e = 0.135$	Exposure at work defined as "more than 1 cigarette per day smoked around you at work, including lunch and tea breaks", p_e=0.356 (males), 0.246 (females)[9]	Increased by 16% (n=390.5)
7. No change in future exposures to SHS	Prevalence of SHS exposure reduced by: 50% in the home; 90% at work	Decreased by 55% (n=146.0)
8. Effect of exposure to SHS at home is independent of exposure at work	Exposure to SHS at work does not add to risk of disease in those exposed also at home (effectively, prevalence of exposure at work reduced by 20%)	Decreased by 4% (n=312.3)

RR, relative risk.

disease risk past the age of retirement. However, there is evidence of effects of SHS on platelet function, vascular endothelium, and myocardial exercise tolerance, at levels commonly found in the workplace.[21] This means that, as with active smoking, there are likely to be both thrombogenic and atherogenic components of the increased risk caused by SHS[22]. The relative importance of the different routes of action is not known, but it is implausible that exposures before age 65 should have no effect on risk of disease over age 65. However, we have included an upper age limit of 75 for the workplace exposures, on the grounds that the time from exposure to heart death is unlikely to be greater than 10 years. In the home, the estimate of a 30% increase in risk of disease is based on studies which had no age bounds (in fact, the average age in this pooled analysis was 65 years[23]). There are no data on stroke risk from SHS by age, but we have excluded from our analysis deaths at age 75 and over to match the age restrictions in the study by Bonita and colleagues.[13]

Depending on how "exposure" is defined, in the recent New Zealand surveys the proportion of all those exposed to SHS who are exposed at both home and work is between 10–30%.[24] This is consistent with recent data from elsewhere. For example, in the NHANES III study in the USA 20% of those exposed at work reported that they were also exposed at home.[25] Should those who are exposed at home and work be treated as susceptible to the effects of SHS in both settings? We think so, since the relative risk estimates in the epidemiological literature were calculated on the basis of exposure at home or at work (none, as far as we know, were calculated on the basis of exposure in one setting but not the other). Should the effects at home and at work be regarded as independent of one another? There are no data to directly answer this question, so we conclude the most appropriate course of action is to add the numbers of cases attributed to SHS at home and at work. A priori, there are probably stronger grounds for suspecting a negative interaction than a positive one: for coronary heart disease at least the dose–response relation suggests that the pathways leading to disease may be rapidly saturated following exposure to SHS. Therefore, as part of the sensitivity analysis we repeated the calculation of avoidable deaths under the extreme assumption that those exposed at home would not be affected by exposures at work. The effect is small (a reduction of 5% in the attributed deaths).

The rate of deaths caused by SIDS has been high in New Zealand by international standards, although there has been a notable decline (among non-Maori largely) in recent years.[26] It appears that this decline is associated with changes in infant sleeping position, and possibly also a reduction in smoking by parents. We have used the results of the most comprehensive study in New Zealand[14] to estimate the prevalence of SIDS and the impact of SHS (defined in this case by whether or not the mother smoked after birth). A case–cohort study was conducted including all infants born in New Zealand between October 1991 and September 1993. There were 232 SIDS cases in the cohort and these were compared with 1200 randomly selected control subjects. Maternal smoking was found to be the strongest risk factor, and its effect was modified by bed sharing. Using the exposure prevalence and relative risk figures from the paper by Mitchell and colleagues,[14] and including only the effect of SHS independent of bed sharing, we estimate that there are approximately 50 SIDS cases a year attributable to SHS. This does not take account of maternal smoking during pregnancy, but epidemiologic and mechanistic data suggest that SHS does act independently of transplacental exposure to

smoke products from the mother's (active) smoking.[16]

Population attributable mortality is an average and it is important to note that the effect of tobacco on health is unevenly shared across the population. For instance, exposure to SHS is known to vary by socioeconomic status.[27] Ethnicity is also a marker of susceptibility: in New Zealand, disease rates are generally higher among the indigenous Maori who make up approximately 15% of the national population, and smoking rates are also higher among Maori. A Maori non-smoking adult or child is likely to be surrounded by twice as many smokers per household, on average.[28]

The list of assumptions in table 4 is not comprehensive, and we have taken the simplest possible approach by varying only one parameter at a time. But we suggest the results do indicate a plausible range of future deaths caused by current exposures to SHS. The total number was most sensitive to varying the relative risk estimates between the upper and lower bounds of the 95% confidence interval. The spread of attributed deaths (174–490) obtained in this way is not a statistical confidence interval, but it might be viewed as a policy confidence interval. It includes almost all the other estimates shown in table 4 (the effect of including active smokers is the only exception). We repeat that this is a snapshot picture of the impact of SHS. We have not attempted to model future trends although it is almost certain that smoking patterns and disease rates will change. However, as an indication of the magnitude of the gains that might be achieved by effective interventions, we have calculated the reduction in mortality caused by SHS that would occur if prevalence of exposure to SHS was halved in the home and reduced by 90% at work. We have not included in this estimate effects on the prevalence of active smoking—social policies that restrict smoking at work and in other public places have important secondary effects on smoking rates.[29]

There has been a pronounced fall in New Zealand in the number of cigarettes consumed per smoker since 1987, which may have led to a reduction in the intensity of exposure to SHS. (Estimates of mean cigarettes per smoker, based on tax paid consumption, have fallen from 23 per day in 1987 to 14 per day in 1999.[24]) Workplaces are also likely to be less smoky, on average, as a result of the changes that have occurred since the Smoke-free Environments Act. As a consequence, extrapolation of relative risk estimates derived from an earlier, heavier smoking period may overestimate the impact of SHS, but in our view there is insufficient information available to attempt to quantify the possible overestimate.

In conclusion, we estimate that past exposures to SHS are responsible for about 347 deaths per year in New Zealand. Some of these deaths result from exposures that occurred 10–20 years ago, and since that time prevalence of smoking has fallen, and so have exposures to SHS at work and in public places. This means that the number of "avoidable" deaths in the future is slightly less: we estimate that there would be approximately 325 fewer deaths in New Zealand each year if present exposures to SHS ceased altogether. This number is about 7% of the deaths in New Zealand estimated to be caused by direct smoking. There are many uncertainties associated with this calculation, but an indication of the magnitude of the problem may be obtained by comparisons with other causes of death. Our calculations suggest that the number of deaths caused by SHS lies between the number of people who die from melanoma of the skin (about 200 per year in New Zealand) and the number killed in road crashes (509 deaths in 1997).[30]

What this paper adds

Second hand smoke (SHS) has been linked with many health problems, some of them fatal. But less has been written about questions of attribution that concern policy makers. How big is the problem—that is, how many deaths are caused by second hand smoke? How does this compare with other, preventable causes of mortality? How confident can we be in such estimates? And how many deaths are avoidable?

If SHS was eliminated today, we estimate there would be about 325 fewer deaths per year in New Zealand in the future. Many approximations and judgements lie behind this estimate, the most important being the choice of relative risks and the question of whether ex-smokers are affected by SHS (we think they are). The true number is likely to lie between the number of deaths caused by melanoma and those resulting from road crashes.

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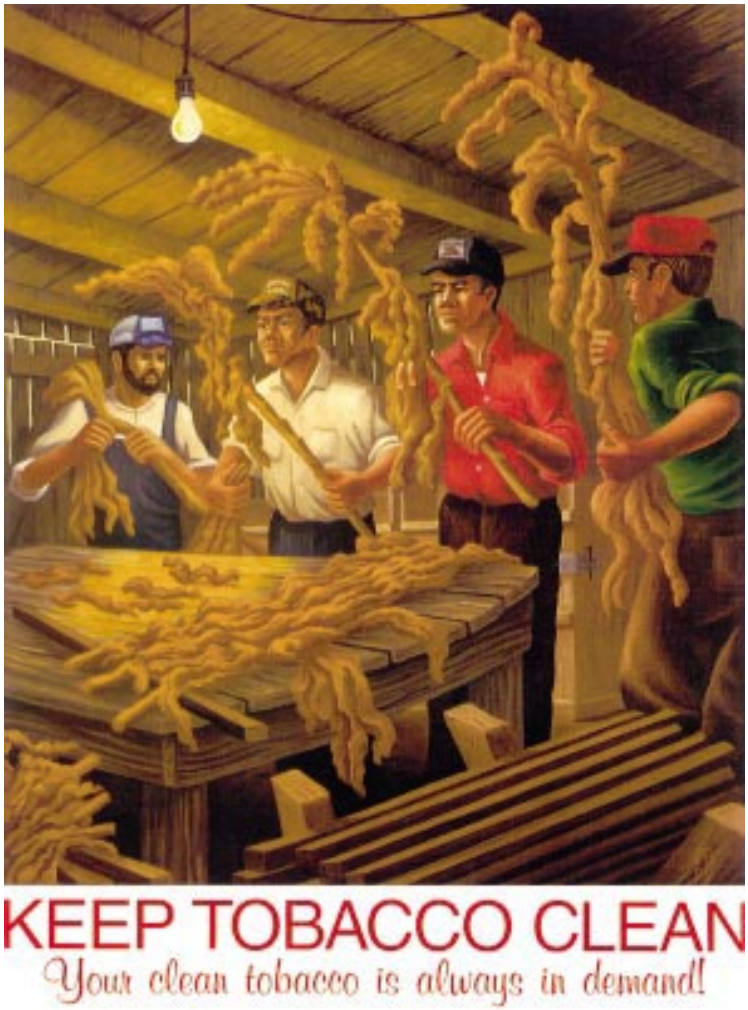

A poster urging clean stripping and preparation of tobacco—we wouldn't want any nasty dirt in here would we? Found at the Carrolton Tobacco Warehouse in Kentucky, USA. Contributed by Melanie Wakefield.