

# Deaths in New Zealand attributable to second hand cigarette smoke

A report to the New Zealand Ministry of Health  
September 2000

Alistair Woodward  
Department of Public Health  
Wellington School of Medicine

Murray Laugesen  
Health New Zealand  
Auckland

Acknowledgements: We are grateful for comments on earlier drafts by Ichiro Kawachi, Martin Tobias and Rod Jackson.

## Summary

There are about 388 deaths caused by SHS in New Zealand each year. This represents an additional 8% over and above deaths due to direct smoking and about three quarters the number of people killed each year on the roads in New Zealand in recent years. However there are many uncertainties associated with this calculation. We have explored the effect of varying the assumptions on which the estimate is based, and on this basis suggest a plausible range of 180 - 621 deaths per year. Extrapolations from other national estimates of deaths attributable to SHS overlap this range.

*Summary of estimated deaths attributable to second hand smoke (includes stroke deaths before age 75, otherwise deaths at any age)*

	<i>Work</i>	<i>Home</i>	<i>total</i>
Heart disease	98.4	144.8	243.3
Stroke	43	44.7	87.7
SIDS	-	-	50
Lung cancer	3.5	3.7	7.2
All causes			388.2

## Introduction

Second hand smoke is released into the environment by people smoking cigarettes, cigars and pipes. It is known also as environmental tobacco smoke, and includes smoke emitted from the glowing end of cigarettes ('sidestream smoke'), 'mainstream' smoke exhaled by active smokers and small quantities of smoke that diffuse through cigarette paper or mouthpiece.<sup>1</sup>

Numerous reviews have concluded that second hand smoke (SHS) causes disease.<sup>1 2 3 4</sup> Since the first reports in the early 1980s<sup>5</sup> the number of conditions associated with SHS has increased, and the evidence that the associations are likely to be cause and effect has strengthened. Attention has been paid mostly to the increase in the risk of disease for individuals exposed to SHS (the 'relative risk'). Less well-documented is the impact of SHS on populations (the 'attributable risk' or the burden of illness due to SHS). From a policy maker's point of view, the attributable risk is important because it provides a guide to the size of the problem.<sup>6 1</sup>

In New Zealand the only detailed assessment of attributable mortality is that carried out in 1989 by Kawachi et al<sup>7</sup>. They estimated the number of heart disease and lung cancer deaths in New Zealand that were caused by SHS in the mid-1980s. Exposures at work and in the home were treated separately. A range of numbers was calculated based on the 95% confidence intervals around the relative risk estimates. The total number of deaths due to passive smoking from lung cancer and coronary heart disease was estimated to be 273 per year (range 112-442).

We repeated the calculations of Kawachi et al using recent information on exposures to SHS and disease risks. The findings are compared with estimates of attributable risk obtained using other approaches. We explore the assumptions that underlie these calculations and propose a range within which the true burden of mortality due to second hand smoke is likely to fall.

## Methods

Information on disease risks due to SHS was sought by searching the scientific literature using key words "tobacco smoke pollution", "second hand smoke", "environmental tobacco smoke" and "passive smoking". Exposure data for SHS were obtained from a national survey conducted in 1996 for the Ministry of Health<sup>8</sup>. The New Zealand Health Information Service provided rates and numbers of deaths in New Zealand by cause.<sup>9</sup>

We calculated Population Attributable Risk (PAR) using the standard formula<sup>10</sup>

$$PAR = P_e (RR - 1) / P_e (RR - 1) + 1$$

where  $P_e$  is prevalence of exposure and RR is relative risk of disease.

## Results

Estimates of lung cancer and coronary heart disease deaths attributable to SHS are shown in Table 1. The numbers of attributed deaths (7.2 and 243.3) are slightly less than those reported by Kawachi et al<sup>7</sup> (30 and 249).

Unlike studies of SHS in the home, where living with a current smoker has generally been the criterion for exposure, there is no common or standard definition of exposure to SHS at work. Studies of disease risks associated with SHS at work have used a wide variety of measures.<sup>11</sup> In the face of this ambiguity, we have chosen a relatively restrictive definition: never-smokers who report that in their workplace one or more persons smoke cigarettes around them during actual work hours. This shows a drop in the prevalence of exposure to SHS at work from 33.6% for men and 23.4% for women in the 1980s<sup>7</sup> to a 1996 estimate for men and women combined of 13.5%<sup>8</sup>. Such a reduction is expected since the Smoke-free Environments Act 1990 banned smoking in many workplaces.<sup>12</sup> Exposure to SHS during tea and lunch breaks (but not during work hours) was reported by a further 14% of men and women in the 1996 survey. Later in the paper we examine the consequences of enlarging the definition of "exposed at work" to include those exposed only during breaks. The prevalence of exposure to SHS at home appears to have changed little between the 1980s and 1990s: about 14.1% of non-smokers in New Zealand in 1996 reported that they lived with one or more people who regularly smoked cigarettes inside the house while they were at home – Kawachi et al used estimates of 12.7% (men) and 16.1% (women).

We estimate the RR of lung cancer associated with exposure to SHS at home to be 1.24 for both women and men, based on two recent meta-analyses<sup>13 14</sup> compared with a RR of 1.3 used by Kawachi et al. But we have applied the same RR to exposures in the workplace, based on a review of epidemiological studies<sup>15</sup>, while Kawachi et al postulated higher risks (2.2) in the absence, at that time, of direct evidence.

The estimated RR for coronary heart disease is based on a meta-analysis by Law et al<sup>16</sup>. Law et al summarized 19 studies of passive smoking and heart disease and calculated an increased risk due to exposure to smoking by a spouse of 30% (RR = 1.30) compared with a RR of 1.2 in the earlier paper. The estimate of 1.30 was similar for men and women and did not vary between studies that counted nonfatal infarcts and those in which the outcome was death due to coronary heart disease. For the workplace, Kawachi et al estimated risk ratios of 2.3 for men and 1.9 for women on the basis of imputed exposures. We have used a RR of 1.2 for both men and women obtained from meta-analyses of eight workplace studies published since 1989<sup>17 18</sup>.

We have assumed that ex-smokers are at increased risk of heart disease due to SHS, whereas Kawachi et al calculated attributable deaths only among never-smokers. Relatively little is known about ex-smokers and SHS because in general they have been excluded from studies of tobacco smoke and heart disease. This is not because there is any evidence that ex-smokers are immune to SHS, but it has been assumed that it would be more difficult to demonstrate an effect in this group due to uncontrolled confounding factors. We think that the default position in an assessment of attributable mortality should be to include ex-smokers as susceptibles, and we offer two pieces of evidence to back up this position. One of the largest cohort studies of SHS and heart disease, the Nurses Health Study, reported findings for ex-smokers as well as never smokers. Compared with ex-smokers not exposed to SHS, the adjusted RR of heart attack was 1.82 (95% CI 0.76 - 4.34) among those occasionally exposed and 2.38 (0.98 - 5.79) among those regularly exposed to SHS<sup>19</sup>. Although these estimates are imprecise and are not statistically significant, the size of the risk increase and the

exposure-response pattern suggest that ex-smokers are indeed susceptible to the effects of SHS. There is supporting evidence from an investigation of pre-clinical effects. Howard et al <sup>20</sup> measured the thickness of the wall of the carotid artery using ultrasound, and found amongst 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 confounders).

The proportions of deaths due to heart disease that occur among never smokers and exsmokers will change over time as a result of variations in prevalence and intensity of tobacco use and changing disease incidence. We have assumed that 50% of males and 64% of females dying from coronary heart disease are never- or ex-smokers based on the findings of the Auckland Heart Health Study in 1986-8 (Jackson R, personal communication).

The calculations in Table 1 assume that exposure to SHS in the workplace increases risk of death from coronary heart disease at all ages. Kawachi et al restricted the attributable deaths to the under 65 years age group, on the grounds 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 disease risk past the age of retirement. The scientific literature on mechanisms of action of SHS on the cardiovascular system has expanded enormously in the last decade, and "experimental studies of ETS and heart disease demonstrate that acute exposure of humans and other species to [SHS] affects platelet function, vascular endothelium, and myocardial exercise tolerance at exposure concentrations widely prevalent in the workplace"<sup>21</sup>. This means that, as with active smoking, there are likely to be both thrombogenic and atherogenic components of the increased risk due to SHS <sup>22</sup>. 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. For this reason we think the default assumption in the estimation of attributable mortality should be that exposures in the workplace affect death rates at all ages. Moreover, we see no reason why an age cut-off should be applied to deaths related to exposures in the home, where the issue of latency is irrelevant. The estimate of a 30% increase in risk of disease due to exposure in the home is based on studies which had no age bounds (in fact the *average* age of individuals included in the meta-analysis was 65<sup>16</sup>).

Other things being equal, the number of deaths attributed to SHS will vary with the total number of deaths, which is in turn a function of mortality rates, population size and age structure. Since 1989 the number of heart disease deaths in New Zealand has fallen due to the substantial drop in the coronary death rate, despite increases in the size of the population and the proportion in older age groups. The number of lung cancer deaths has fallen in men, but risen amongst women. We have assumed that the proportion of lung cancer cases occurring among never smokers has remained the same as in 1989 (8%).

### *Stroke*

We have included stroke in the calculation of attributable mortality on the basis of two recent studies that have investigated associations with SHS. One of these, by Bonita et al<sup>23</sup>, is particularly relevant as it examined causes of stroke among adults aged 35-74 in Auckland in the early 1990s. It is one of the first case-control studies of SHS to be carried out after national smoke-free legislation so one would expect incidental exposures to SHS to be less common,

and the risk estimates are likely to be less affected by measurement error than previously. (Mis-classification of exposure typically causes risks to be under-estimated.)

Bonita et al found that the odds of a first ever stroke (fatal and non-fatal combined) were increased among nonsmokers and long-term ex-smokers who reported that they were exposed to SHS compared with those not so exposed at home or at work (OR = 1.82, 95% confidence interval 1.34 - 2.49). This result was present after adjusting for important potential confounders and is consistent with findings from a similar study in Melbourne. In the Australian study, 452 hospitalized cases of first-episode ischemic stroke were compared with 452 age- and sex-matched neighbourhood controls. The risk of stroke was twice as high for participants whose spouses smoked as for those whose spouses did not smoke (95% confidence interval 1.3 - 3.1), after adjustment for the subject's own smoking, heart disease, hypertension, diabetes, and education level.<sup>24</sup>

In Table 1 we have calculated attributable stroke mortality using the RR estimate published by Bonita et al, and estimates of prevalence of exposure to SHS at home and work that were cited previously – 0.14 (at home) and 0.13 (at work). We have assumed that ex-smokers experience the increased relative risk of stroke mortality that applies to never smokers, for the reasons given above, and that the PAR% for stroke deaths is the same as that for first ever, non-fatal strokes.

In New Zealand in 1997 there were 626 deaths due to cerebrovascular disease (ICD9 430-8) in adults aged 35-74.<sup>9</sup> (Deaths at age 75 and over are excluded to match the age restrictions in the study by Bonita et al.)

#### *Sudden Infant Death Syndrome*

The rate of deaths due to SIDS has been high in New Zealand by international standards, although there has been a marked decline (among non-Maori largely) in recent years.<sup>25</sup> 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 of SIDS in New Zealand<sup>26</sup> to estimate the impact of SHS (defined in this case by whether or not the mother smoked after birth). (Table 1) This was a case-cohort study based on a survey of all infants born in New Zealand between October 1991 and September 1993. There were 232 SIDS cases and these were compared with 1200 randomly selected control subjects. Maternal smoking was found to be the major risk factor, and its effect was modified strongly by bed-sharing. Using the exposure prevalence and RR figures from the paper by Mitchell et al, 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.

It is difficult to distinguish an effect of maternal smoking during pregnancy on risk of SIDS from an effect of SHS either antenatally or postnatally. However there is reason to conclude that SHS acts independently of trans-placental exposure to smoke products from the mother's (active) smoking.<sup>27</sup> Moreover, from a policy viewpoint, it could be argued that all tobacco-related cases of SIDS should be counted as SHS deaths. Whether the smoke reaches the child via the placenta or direct into the lungs after birth, the policy interventions are likely to be much the same: media campaigns and legislated smoking restrictions.

### *Mortality among Maori*

It is not possible to quantify precisely the burden of mortality for Maori, but for several reasons we expect that Maori will be disproportionately affected by SHS.

- ?? A Maori nonsmoking adult is likely to be surrounded by twice as many smokers per household on average, because nearly half of Maori smoke and nearly one quarter of non-Maori smoke<sup>28</sup>
- ?? Maori children can be expected to have twice the SHS exposure of non-Maori children<sup>29</sup>
- ?? A Maori worker is more likely to be exposed to smoking during actual working hours<sup>8</sup>
- ?? The actual number of cigarettes smoked per Maori smoker is almost the same as for non-Maori smokers, so adjusting for consumption does not diminish the risk to Maori nonsmokers
- ?? Rates of lung cancer and heart disease are higher among Maori than non-Maori<sup>30</sup>, so for these conditions the death rate attributable to SHS will also be higher.

### **Discussion**

Estimates of attributable mortality inevitably contain many uncertainties, some of which are quantifiable. In Table 2 we have listed what we see as the most important assumptions underlying the estimate of deaths due to SHS. The values in the first column include our best estimates of key inputs to the attributable mortality equations. In the next column we have laid out plausible, alternative conditions, and have then recalculated the numbers of deaths due to SHS.

The most contentious question is whether some of the deaths that occur amongst current smokers should be attributed to SHS. If current smokers were treated as equally susceptible to the harmful effects of SHS, and if it was assumed that their exposure to SHS was the same as ex- and never-smokers (a conservative assumption), the total attributed deaths would be 710 per year. In other words, according to these assumptions about 322 smokers die each year in New Zealand as a result of SHS. Clearly current smokers are exposed to SHS from both their own cigarettes and those of others around them. What is not known is whether SHS has the same effect on risk of disease in the presence of mainstream smoke as it does when mainstream smoke is absent. As with exsmokers, current smokers have been excluded from SHS risk assessments in the past to reduce the number of complicating factors and uncertainties. This is the cleanest course of action from a scientific perspective, but policy-makers might consider what the implications would be if a fraction of direct smoking deaths were re-assigned to passive smoking. The fraction may be relatively small (about 5% according to our estimates), but significant nevertheless in terms of policy choices since interventions to reduce SHS exposures may have different costs and benefits to those aimed at reducing active smoking. For example, it is important that politicians and the public are correctly informed about the potential gains from bans on smoking in workplaces, bars and restaurants, where many of those exposed to SHS are current smokers.

During the 1990s smoking bans were introduced in many workplaces, so one would expect an increasing proportion of workers to be exposed to SHS only during lunch and tea breaks. There is no information available on the intensity of these exposures. In some instances, exposure during breaks may be slight and of little significance to disease risk. However in other circumstances crowded and smoky break rooms may cause substantial exposures. As part of the sensitivity analysis we explored the effect of using a more inclusive definition of exposure to SHS in the workplace. If workers exposed to SHS only during breaks are included, estimate of attributable deaths rises to 534 deaths per year.

Given the importance of coronary heart disease as a contributor to the total of attributable deaths, we have repeated the calculations using the results of the largest and arguably the best conducted study of SHS and heart disease to date, the US Nurses Health Study. This study reported RRs of 1.70 for women exposed at home and 1.92 for women exposed at work.<sup>19</sup>

Obviously this is not a comprehensive list of all the assumptions that are included in a calculation of attributable risk, and we have taken the simplest possible approach by varying only one parameter at a time. But we suggest the results in Table 2 do indicate a plausible range of attributable deaths. 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 (180 - 621) 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 2 (the effect of including active smokers is the only exception), and overlaps the estimates provided from overseas (see below). It should be noted that this is a snap-shot picture of attributable risk. We have not made any allowance for the present effects of past exposures to SHS, and have not attempted to model future impacts although it is almost certain that smoking patterns and disease rates will change. However as an indication of the returns that might be achieved by effective interventions, we have calculated the reduction in mortality attributable to SHS that would occur if prevalence of exposure to SHS was halved in the home and reduced by 90% at work.

There are other sources of uncertainty that are difficult to put into numbers. Our calculations assume that there are no interactions between SHS and other exposure factors. In fact there are well-known situations where the effect of tobacco smoke in the presence of a joint exposure is different to the effect of smoke alone. For instance, SHS is more strongly associated with acute respiratory illness when an infant is bottle fed than when a child in similar circumstances is breast fed<sup>31</sup>. The effects of active smoking on vessel wall thickening are modified by ethnicity (higher in Black Americans) and by the presence of other risk factors (hypertension or diabetes)<sup>20</sup>. It would seem likely that interactions such as these influence the occurrence of SHS-attributable deaths, but too little is known to estimate the impact in numerical terms. One exception may be bed-sharing and SIDS. In the national study by Mitchell et al<sup>26</sup>, it was estimated that the combination of bed-sharing and maternal smoking explained about 26% of SIDS cases in New Zealand, over and above the 46% of cases that could be attributed to maternal smoking alone. This suggests that if the prevalence of bed-sharing did not change, eliminating maternal smoking altogether would prevent a total of 72% of SIDS cases, saving 28 deaths a year in addition to those counted in Table 1.

The list of diseases in Table 1 excludes a number of important conditions that are believed to occur more commonly after exposure to SHS, and may be fatal. Asthma and acute respiratory illnesses in childhood are two examples: there is strong evidence that SHS exacerbates asthma and increases rates of chest infections.<sup>1</sup> However in New Zealand few children die from these diseases (in 1997 there were no deaths under 10 years of age coded as asthma, and only 11 due

to pneumonia and influenza). Consequently it makes no appreciable difference to include them in the attributable mortality calculations, important as they may be as causes of attributable morbidity. SHS has also been linked with several neoplasms other than lung cancer, including sinonasal cancers and cancer of the cervix<sup>4</sup>. However, in our judgement the evidence for a causal effect of SHS is not sufficiently strong to warrant adding these causes of death to Table 1.

There has been a marked fall in New Zealand in the number of cigarettes consumed per smoker since 1987, and this means that there is likely to have been 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.<sup>32</sup>) 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 RR estimates derived from an earlier, heavier-smoking period may over-estimate the impact of SHS. This is important for conditions such as coronary heart disease where there may be a relatively short interval between exposure and death. We have not attempted to quantify the magnitude of the possible over-estimate since neither the timing nor the shape of the exposure-response relation are certain. In the case of lung cancer, where the interval between exposure and incidence of disease is much longer, deaths occurring in the 2000s are likely to be linked to exposures in the 1980s or earlier.

How do these numbers compare with estimates of the burden of mortality calculated elsewhere? In Table 3 we summarize recent reports from Australia and the US and show how the results might be extrapolated to New Zealand. These are offered as a rough check that the PAR calculations shown in Table 1 are of the right order of magnitude. (The many variations between countries in smoking rates, patterns of exposure to SHS and the incidence of disease mean that the extrapolations are rough approximations only.) The estimates of attributable deaths appear to pass this test - the Australian figures give a rather smaller number of deaths, while the US figures suggest a larger burden of mortality, but the differences are no more than 2-4 fold.

It has been suggested that deaths due to SHS could be estimated as a fraction of deaths due to active smoking, since the factors underlying the two are likely to be similar, and estimates currently exist for active smoking mortality.<sup>33</sup> The ratio of 1:8 is based on estimates in the United States of about 420,000 deaths due to active smoking per year and 53,000 deaths due to passive smoking.<sup>34</sup> The number of attributable direct smoking deaths in New Zealand in 1996 was 4700<sup>32</sup>. On this basis, the number attributable to SHS exposure would be about 590. This assumes that the US estimates are accurate, that the NZ active smoking burden is comparable with the US figure (although in fact the number of cigarettes per smoker is fewer here) and that the relative impacts of active and passive smoking are the same in the two countries. (Higher levels of SIDS in New Zealand suggest that the latter assumption may not be borne out.)

#### *For further analysis*

The extrapolations from US and Australian mortality figures could be refined by accounting for the differences in background disease rates in New Zealand. Estimates could also be calculated using varying assumptions about the degree of SHS exposure in this country. For example, differences in smoking rates can be quantified - in 1996 the consumption of cigarettes per US adult (as a summary measure of both prevalence and cigarettes per smoker) was 2499 compared to 1477 cigarettes or equivalent in New Zealand.

Many of the deaths amongst ex-smokers would have been counted as direct smoking deaths, and all or some of these ex-smoker deaths should perhaps be subtracted from the direct smoking total. For example, the smoking attributable deaths calculated by Peto and Lopez<sup>35</sup> include deaths among those who have quit smoking. As noted already, the question of whether deaths should be attributed to active or passive smoking has more than theoretical importance, because we pinpoint the policy solution by classifying the cause of death more correctly. In fact SHS smoking deaths in smokers are likely to be at a higher rate than for nonsmokers, since their exposures are much higher. Subtracting these re-assigned deaths from the direct smoking deaths may lead to a radical reappraisal of what the policy solution should be.

Years of life lost would give a more complete picture of premature mortality due to SHS. It would be possible also to estimate morbidity due to this exposure, represented by a combined measure such as the DALY, and/or by estimates of attributable hospitalisations and GP visits.

## **Conclusion**

There are about 388 deaths caused by SHS in New Zealand each year. This is equal to an additional 8% over and above deaths due to direct smoking, or about three quarters the number of people killed each year on the roads in New Zealand in recent years (509 deaths in 1999<sup>9</sup>). There are many uncertainties associated with this calculation. We have explored the possible impact of varying the assumptions on which the estimate is based, and on the basis of these calculations suggest a plausible range of 180 - 621 deaths per year. Extrapolations from other national estimates of deaths attributable to SHS overlap this range.

## References

1. National Health and Medical Research Council. The health effects of passive smoking. A scientific information paper. Canberra, Australia: Commonwealth of Australia, 1997.
2. Surgeon General. The health consequences of involuntary smoking. Rockville, Md: US Department of Health and Human Services, 1986.
3. Environmental Protection Agency (EPA). Respiratory health effects of passive smoking: lung cancer and other disorders. Washington, DC: EPA Office of Research and Development, 1992.
4. Office of Environmental Health Hazard Assessment. Health effects of exposure to environmental tobacco smoke. Final report September 1997. Sacramento: California Environmental Protection Agency, 1997.
5. Hirayama T. Cancer mortality in nonsmoking women with smoking husbands based on a large-scale cohort study in Japan. *Prev Med* 1984; 13:680-90.
6. Wells AJ. An estimate of adult mortality in the United States from passive smoking. *Environ Internat* 1988; 14:249-65.
7. Kawachi I, Pearce NE, Jackson RT. Deaths from lung cancer and ischaemic heart disease due to passive smoking in New Zealand. *NZ Med J* 1989; 102:337-40.
8. Ministry of Health. Environmental tobacco smoke study 1996. Wellington: Ministry of Health, 1996.
9. Ministry of Health. Mortality and demographic data 1997. Wellington: Ministry of Health, 2000.
10. Hennekens CH, Buring JE. *Epidemiology in Medicine*. Boston: Little, Brown and Co, 1987.
11. Steenland K. Risk assessment for heart disease and workplace ETS exposure among nonsmokers. *Environ Health Perspect* 1999; 107 Suppl 6:859-63.
12. Woodward A, Fraser T. Passive smoking in New Zealand: health risks and control measures. *NZ Health Report* 1997; 4:35-6.
13. Hackshaw AK, Law MR, Wald NJ. The accumulated evidence on lung cancer and environmental tobacco smoke. *BMJ* 1997; 315:980-8.
14. Taylor R, Cumming R, Woodward A, Black M. Passive smoking and lung cancer: a cumulative meta-analysis. (Submitted) .
15. Samet JM. Workshop summary: assessing exposure to environmental tobacco smoke in the workplace. *Environ Health Perspect* 1999; 107(Suppl 2):309-12.
16. Law MR, Morris JK, Wald NJ. Environmental tobacco smoke exposure and ischaemic heart disease: an evaluation of the evidence. *BMJ* 1997; 315:973-80.
17. Steenland K. Risk assessment for heart disease and workplace ETS exposure among nonsmokers. *Environ Health Perspect* 1999; 107(Suppl 6):859-63.
18. Wells AJ. *J Am Coll Cardiol* 1998; 31:1-9.
19. Kawachi I, Colditz GA, Speizer FE et al. A prospective study of passive smoking and coronary heart disease. *Circulation* 1997; 95:2374-9.
20. Howard G, Wagenknecht LE, Burke GL *et al*. Cigarette smoking and progression of atherosclerosis: The Atherosclerosis Risk in Communities (ARIC) Study [see comments]. *JAMA* 1998; 279(2):119-24.
21. Howard G, Thun MJ. Why is environmental tobacco smoke more strongly associated with coronary heart disease than expected? A review of potential biases and experimental data. *Environ Health Perspect* 1999; 107 Suppl 6:853-8.

22. Howard G, Burke GL, Szklo M, Tell GS, Eckfeldt J, Evans G et al. Active and passive smoking are associated with increased carotid wall thickness. *Arch Int Med* 1994; 154:1277-82.
23. Bonita R, Duncan J, Truelsen T, Jackson RT, Beaglehole R. Passive smoking as well as active smoking increases the risk of acute stroke [see comments]. *Tob Control* 1999; 8(2):156-60.
24. You RX, Thrift AG, McNeil JJ, Davis SM, Donnan GA. Ischemic stroke risk and passive exposure to spouses' cigarette smoking. Melbourne Stroke Risk Factor Study (MERFS) Group. *Am J Public Health* 1999; 89(4):572-5.
25. Ministry of Health. Mortality and demographic data 1996. Wellington: Ministry of Health, 1998.
26. Mitchell EA, Tuohy PG, Brunt JM *et al.* Risk factors for Sudden Infant Death Syndrome following the Prevention Campaign in New Zealand. *Pediatrics* 1997; 100:835-40.
27. Klonoff-Cohen HS, Edelstein SL, Lefkowitz ES *et al.* The effect of passive smoking and tobacco exposure through breast milk on sudden infant death syndrome [see comments]. *JAMA* 1995; 273(10):795-8.
28. Crampton P, Salmond C, Woodward A, Reid P. Socioeconomic deprivation and ethnicity are both important for anti-tobacco health promotion. *Health Education & Behavior* 2000; 27:317-27.
29. Te Puni Kokiri. Maori smoking patterns . Wellington: Te Puni Kokiri, 1999.
30. Pomare E, Keefe-Ormsby V, Ormsby C et al. Haoura. Maori standards of health III. Wellington: Eru Pomare Maori Health Research Centre, 1995.
31. Woodward A, Douglas RM, Graham NMH, Miles H. Acute respiratory illness in Adelaide children: breast feeding modifies the effect of passive smoking. *J Epidemiol Comm Health* 1990; 44(3):224-30.
32. Ministry of Health. Tobacco Facts. Wellington: Ministry of Health, 1999.
33. Glantz SA, Parmley WW. Passive smoking and heart disease. Mechanisms and risk. [Review]. *JAMA* 1995; 273(13):1047-53.
34. Glantz SA, Parmley WW. Passive smoking and heart disease: epidemiology, physiology and biochemistry. *Circulation* 1991; 83(1):1-11.
35. Peto R, Lopez AD, Boreham J et al. Mortality from smoking in developed countries 1950 - 2000. Oxford: Oxford University Press, 1994.

Table 1. Mortality attributable to Second Hand Smoke, New Zealand

	Prevalence of exposure to SHS	Relative Risk (95% CI)	Population Attributable Risk %	Total deaths per year (1997)	Deaths among never-smokers and ex-smokers	Deaths attributed to SHS
<b>1. LUNG CANCER</b>						
At home						
- men	14.1%	1.24 (1.13 - 1.36)	3.3%	882	70.6*	2.3
- women	14.1%	1.24	3.3%	530	42.4*	1.4
At work						
- men	13.5%	1.24 (1.13 - 1.36)	3.1%	882	70.6*	2.2
- women	13.5%	1.24	3.1%	530	42.4*	1.3
<b>2. CORONARY HEART DISEASE</b>						
At home						
- men	14.1%	1.30 (1.22 - 1.38)	4.1%	3614	1807.0	73.3
- women	14.1%	1.30	4.1%	2755	1763.2	71.6
At work						
- men	13.5%	1.21 (1.04 - 1.41)	2.8%	3614	1807.0	49.8
- women	13.5%	1.21	2.8%	2755	1377.5	48.6
<b>3. STROKE</b>						
At home						
- men	14.1%	2.10 (1.33 - 3.32)	13.4%	325**	209.6	28.2
- women	14.1%	1.66 (1.07 - 2.57)	8.5%	301**	194.2	16.5
At work						
- men	13.5%	2.10 (1.33 - 3.32)	12.9%	325**	209.6	27.1
- women	13.5%	1.66 (1.07 - 2.57)	8.2%	301**	194.2	15.9
<b>4. SUDDEN INFANT DEATH</b>						
Males and females combined	19.7%#	5.3 (2.71 - 10.28)	46%	109		50.0
<b>TOTAL</b>						<b>388.2</b>

Notes:

\*never-smokers only \*\*deaths before age 75

#exposure defined as maternal smoking 2 months after birth, no bed-sharing

Table 2. Effects of varying key assumptions underlying estimates of mortality attributed to Second Hand Smoke

Assumption	Alternative conditions	Effect on attributed deaths
------------	------------------------	-----------------------------

1. 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 60% (n=620.8) Decreased by 54% (n=180.0)
2. Ex-smokers susceptible to effects of SHS	Ex-smokers not susceptible to effects of SHS: proportion of deaths among never-smokers - heart disease 0.32 (males), 0.42 (females) <sup>7</sup> ; stroke 0.31 <sup>23</sup>	Reduced by 29% (n=275.6)
3. Current smokers not susceptible to SHS	Regard all deaths from lung cancer, heart disease and stroke as influenced by SHS	Increased by 83% (n=710.5)
4. Ex-smokers as likely to be exposed to SHS as never-smokers	Exposure more common for ex-smokers: $p_e = 0.161$ work, $0.188$ home <sup>8</sup>	Increased by 11.2% (n=434.8)
5. Workplace exposure to SHS affects risk of heart disease beyond age 65	Workplace exposure to SHS does not affect risk beyond age 65 - number of heart disease deaths under 65 years: 818 males, 215 females	Reduced by 21% (n=304.9)
6. RR heart disease due to home exposure = 1.30 <sup>16</sup>	For women, RR heart disease due to home exposure = 1.70 <sup>19</sup>	Increased by 22.4% (n=475.1)
7. RR heart disease due to work exposure = 1.2 <sup>11</sup>	For women, RR heart disease due to work exposure = 1.92 <sup>19</sup>	Increased by 37.7% (n=534.4)
8. 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) <sup>8</sup>	Increased by 41.6% (n=549.6)
9. No change in future exposures to SHS	Prevalence of SHS exposure reduced by ?? 50% in the home ?? 90% at work	Reduced by 62% (n=148.0)
10. No adjustment for possible double counting of attributed deaths due to individuals being exposed at both home and work	Of all adults exposed to SHS, estimated that 12.5% exposed at both home and work. <sup>8</sup>	Reduced by 5% (n=368.8)

---

Table 3. Extrapolation to New Zealand from other national estimates of deaths attributed to SHS

Reference	Findings (deaths per year due to SHS)	Extrapolation	Number of deaths in New Zealand
Australia - National Health & Medical Research Council <sup>1</sup>	Heart disease - 80	Assume	Heart disease - 48.6
	Lung cancer - 11	?? RR ex-smokers = never smokers	Lung cancer - 6.6
	(excludes ex-smokers and exposures outside the home)	?? Prevalence exposure at work = exposure at home ?? Population ratio 3.8:18.8 (millions)	
United States - California EPA <sup>4</sup>	Heart disease - 35,000-62,000	Assume	Heart disease - 472-871
	Lung cancer - 7,000	?? Population ratio 3.8:270.3	Lung cancer - 42
	SIDS - 1,900-2,700		SIDS - 27-38
	Infant pneumonia - 130-200		Infant pneumonia - 2-3 All causes - 563-954
United States - Steenland <sup>11</sup>	Heart disease - 1,710 (deaths due to work exposures only, among never-smokers aged 35-69)	Assume ?? Population ratio 3.8:270.3	Heart disease - 24 (never-smokers, age 35-69, resulting from exposures in workplace)