Morbidity attributable to second hand cigarette smoke in New Zealand

Report to the Ministry of Health

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Summary

Second hand smoke (SHS) increases the risk of many diseases: children are especially susceptible. Many New Zealanders are still exposed to SHS despite the progress that has been made in the last ten years in reducing tobacco use. For example, approximately a third of secondary school students live in households with smokers, and 39% of indoor workers are exposed to smoke during working hours (including tea and lunch breaks).

The aim of this report is to estimate the amount of non-fatal illness in New Zealand that is caused by SHS. To do this we have combined measures of the prevalence of exposure with estimates of the increase in relative risk of illness due to SHS and measures of the total burden of illness in New Zealand.

We conclude that each year SHS causes
- More than 500 hospital admissions of children under 2 years suffering from chest infections
- Almost 15,000 episodes of childhood asthma
- More than 27,000 GP consultations for asthma and other respiratory problems in childhood
- Fifteen hundred hospital operations to treat glue ear
- Approximately 50 cases of meningococcal disease
- Approximately 1200 admissions to hospital for ischaemic heart disease
- Almost 500 admissions for persons suffering from strokes

There are effects on adults of exposures both at home and at work: each year, for instance, admissions to hospital following heart attacks include about 190 events that would not have occurred if all work places had been totally smoke-free.

Maori are more severely affected than non-Maori, since they are more commonly exposed to SHS and background rates of disease are higher than in the non-Maori population.

These figures should not be treated as precise measures because there are many uncertainties involved in calculations of this kind. But they provide a robust indication of the magnitude of the burden of illness due to SHS.

1. objectives of this report

This report aims to estimate the burden of non-fatal illness in New Zealand that may be attributed to second hand cigarette smoke (SHS). It follows a report on mortality attributable to SHS.[1]

We have not attempted to carry out a systematic review of the very large amount of scientific literature on the topic of SHS and health. Others have done so recently and we have drawn on their work, giving priority to the reviews that in our judgement are most comprehensive and up to date. Our sources include reports of government working groups such as [2] [3] and individual scientists [4] [5].

2. conditions affected by SHS

Tables 1-3 list conditions that were considered for inclusion in this report. Judgements were made on the strength and consistency of the evidence relating diseases to SHS. We conclude that the scientific evidence for conditions listed in Table 1 is sufficiently strong to include them all in an estimate of attributable morbidity. Those graded as "+++" have been linked with SHS in many countries, in many studies, extending over more than twenty years and the argument for cause and effect is very strong, in our view. The literature on stroke and meningococcal disease is less substantial, but it is consistent, plausible on the basis of what we know about the other effects of SHS and therefore warrants inclusion (graded as "++").

Table 2 lists conditions that have been linked with SHS, but in these instances the data are limited and do not justify inclusion, in our judgement. Table 3 shows health problems that are caused by parental smoking, but are not due to inhalation of SHS. These are beyond the scope of our report. However, interventions to reduce exposure to SHS may lead to savings in these areas (i.e. fewer fires and less active smoking modelled on parental habits).

3. methods of estimating attributable risk

The counter-factual in our calculation of attributable risk is "no exposure to SHS". What constitutes "exposure" to SHS varies from one condition to another: we have applied to each outcome the definition of exposure that was used to calculate the estimates of relative risk. Exposures for lung cancer and vascular disease were lagged by 15 years and 7 years respectively. The reasoning for this is outlined in the mortality report. In brief, the major effect of tobacco smoke on carcinogenesis occurs at an early stage in the process, so that there is a delay between exposure and diagnosis of the resulting cancers. On the basis of observations of active smokers, we estimate this delay is about 15 years on average. A similar argument is applied to vascular diseases caused by tobacco, but the picture is less clear. The effect of SHS on cardiovascular risk is due to a combination of acute (increased clotting) and chronic (vessel wall narrowing) processes and it is not certain what is the balance of these effects. We have chosen to factor in a lag of 7 years, influenced strongly by the evidence of early arterial wall thickening caused by SHS{Howard G, Burke GL, et al. 1994 #38360}. However we acknowledge that this is contentious - others would argue for a stronger weighting of acute effects, which would mean reducing the lag substantially. (Note that the effect of incorporating a shorter lag for stroke and ischemic heart disease would be to reduce the number of attributable events due to past exposures, as levels of SHS have generally declined in the last decade. The number of future avoidable cases

of disease would not change, but the benefits of eliminating exposure to SHS would be experienced earlier than forecast in this paper.)

We applied the following standard formula for population attributable risk:

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

where P_e is prevalence of exposure and RR is relative risk of disease, and PAR is the proportion of the cases of disease (or its rate) that is caused by the exposure.

This formula was re-arranged to calculate the excess number of cases (or deaths) attributable to exposure:

$$\text{Attributable number} = N\, p_e\, (I_e\, I_u)$$
$$= N\, I_p\, PAF$$
$$= N\, I_u\, p_e\, (RR-1)$$

where N is the total number of people in the population, I_p is incidence in the total population and I_u is incidence amongst unexposed.

We estimated the current burden of illness using available statistics on case load, hospitalisations, surgical procedures, numbers of notified cases and GP visits. The PAR was then applied to these statistics to estimate the amount of illness in New Zealand that may be attributed to SHS.

We have presented the results in two tables. Table 6 shows the current levels of illness that would not have occurred if there had been no exposure to SHS in the past. Table 7 estimates the burden of illness in the future that would be avoided if exposures to SHS ceased altogether. This provides a guide to the extent of morbidity that may be avoided if actions were taken now to control SHS. The effect on acute respiratory illnesses would be apparent very rapidly; it would be 2007 approximately before the "savings" in heart disease were realised, and the impact on lung cancer would be further delayed to about 2015.

Table 1. Diseases included in the calculation of morbidity attributable to SHS

Disease	Comments	Key references	Grading
Lower respiratory illness in children	Includes bronchiolitis, bronchitis, pneumonia, croup. Effect most marked at ages 0 - 2 years	Cook & Strachan[4], NH&MRC[2]	+++
Asthma in children	Up to age 15 years but effect greater in pre-school children, evidence strongest for exacerbations of established asthma, often mediated by LRI. Likely there are effects of smoke exposure both in utero (impeding airway growth) and postnatally (causing irritation of airways)	Gold[6], Cook & Strachan[4], NH&MRC[2]	+++
Middle ear disease	Outcome defined in various ways (recurrent OM, acute OM, OM with effusion etc) but association with SHS consistent	Strachan & Cook [7]	+++
Lung cancer	Most studies report association of SHS and death from lung cancer: due to almost invariably low survival, the effect on incidence will be similar	Hackshaw[8] Taylor[9]	+++
Coronary heart disease	Outcome in epidemiological studies is mostly admission to hospital for AMI. RR estimates for SHS are similar for fatal and non-fatal episodes.	Thun[10]Law[11]	+++
Stroke	Relatively few studies have reported on this association, but these include a substantial study from NZ. The findings are consistent with mechanistic studies of vessel narrowing and altered platelet function[12].	Bonita[13] You[14]	++
Meningococcal disease	Major public health issue in New Zealand. SHS a risk factor in large NZ case-control study and in studies overseas	Baker et al[15] Kriz [16]Moodley[17]	++

Table 2. Conditions associated with SHS but not included in these estimates of attributable morbidity

Disease	Comments	Key references
Chronic obstructive lung disease	Entirely plausible that an irritant such as SHS should aggravate this condition, as indicated by case reports. But no systematic studies to quantify the size of the effect population-wide	[18]
Menstrual disorders	Evidence of effect of active smoking, recent literature suggesting a link also with SHS	[19, 20]
Other respiratory tract cancers than lung cancer	To be expected that SHS will have effects, by analogy with active smoking. Limited epidemiological evidence.	[21]
Breast cancer	Early literature suggesting a possible link, with the possibility of a genetically susceptible sub-group	[22, 23]
Invasive pneumococcal disease	Limited number of studies, may be particularly important with immuno-compromised groups	[24]
Atopy	Limited data to suggest that children of smokers are more likely to exhibit atopic tendency	[2, 3]
Low birth weight	Developing literature to indicate that exposure of the mother to SHS in pregnancy increases the risk of LBW, and the health effects for the infant that follow. Not sufficient evidence at this stage to quantify the likely effects on variables such as hospital admissions	[25]
Respiratory symptoms in adults due to exposure to SHS at work	Case histories and epidemiological studies from overseas, but no NZ information	[26]
Asthma in adults	Case histories of acute exacerbations in adults with asthma, consistent with effects in children, but no systematic studies	[27]

Table 3. Health problems of children associated with parental smoking, but not mediated by SHS and not included in this report

Condition	References	Comment
Effects of active smoking	28	Children who grow up in a home with a parent who smokes are roughly twice as likely to become a smoker themselves
Injuries due to house fires	29 30	The presence of one or more smokers in a household is a strong risk factor for household fires and injury due to these fires, particularly in the presence of poor quality housing and furnishings

Table 4. Definitions of exposure and relative risk for conditions attributed to SHS

Outcome	Exposure	Relative risk (95% CI)	Comments
Lower respiratory illness in children under 2 years			
• episode of LRI	Mother smokes	2.08 (1.59 - 2.71)	Ref: [4] [31]
• hospital admission	Mother smokes	1.53 (1.25-1.86)	
• GP visit	Mother smokes	1.5	Christchurch Longitudinal Study reported doubling of consultation rate in first year, much reduced effect in second year
Asthma incidence			
Under age 6	Mother smokes	1.31 (1.22-1.41)	Pooled estimates for incidence from Cook & Strachan. [4]SHS also exacerbates established asthma [2] [3] [4]
Age 6-15	Mother smokes	1.13 (1.04-1.22)	
Middle ear disease			
• acute otitis media	Either parent smokes	1.0 - 1.6	Depending on "recurrent" or "with effusion", detection or referral[7]
• chronic OM	Either parent smokes	1.21 - 1.48	
Lung cancer			
• mortality	Live with a smoker	1.24 (1.13-1.36)	Numerous meta-analyses give similar results [8] [9]
• mortality	Exposed to SHS at work	1.24 (1.13-1.36)	
Coronary heart disease			
• hospital admissions of first MI	Spouse who smokes	1.30 (1.22-1.38)	Meta-analyses: no significant difference between fatal and nonfatal MI [11] [32]
• hospital admission	Exposure to SHS at work	1.21 (1.04-1.41)	
Stroke			
• first ever stroke	Exposed to SHS at home or work	1.82 (1.34-2.49)	Fatal and non-fatal strokes combined[14] [33]
• hospitalised cases	Living with a spouse who smokes	2.0 (1.3-3.1)	
Meningococcal disease	One or more smokers in the household	1.4 (1.0-1.8)	Auckland children aged less than 8 yrs 1997-1999 [15]. Note that higher RRs reported elsewhere [16] [17] [34]

Table 5. Exposure to Second Hand Smoke in New Zealand. Key references

Date	Population	Definition of exposure	Prevalence of exposure to SHS	Reference
1997	Infants visited by Plunket nurses at 3 months	Mother smoked during pregnancy	26% (all mothers), 49% (Maori mothers)	Tuohy P, Griffiths M, Brooke M et al, 1997. SIDS risk factor final report. Unpublished report to the Ministry of Health.
1997	Secondary school children aged 14-15	Exposed to smoke at home	35% (all students), 54% (Maori)	ASH survey of secondary school students
1987-8	Adults, 35-64 yrs, never-smokers	Exposed to SHS at work	33.6% (men) 23.4% (women)	Kawachi [35]
1991	Indoor workers	Exposed to SHS during working hours Exposed during tea and lunch breaks	19% 39%	Public Health Commission[36]
1996	Indoor workers	Exposed to SHS during working hours Exposed during tea and lunch breaks	14% 36%	Ministry of Health[37]
1991-3	Infants, at 2 month visit	Mother smokes Father smokes	22.8% 27.7%	Mitchell et al[38]
1986	Adult non-smokers 35-64 years	Exposed to SHS in their homes	12.7% (men) 16.1% (women)	Kawachi[35]
1996	Adult non-smokers	Live with someone who regularly smokes around them inside the home	14.1%	Ministry of Health [37]
1996	Adults exposed to SHS	Mean number of hours per day exposed to others smoke	3.7 hrs (at work) 3.4 hrs (away from work) 3.7/3.1 (European/other) 3.7/4.4 (Maori)	Ministry of Health [37]

Notes on the estimates of attributable and avoidable cases

1. Lower Respiratory Illness (LRI)

There is a tendency for larger RR estimates to be reported when the outcome is number of cases than when the study endpoint is hospitalisations (suggesting that the effect of SHS is more on incidence than severity). In this table we have used the lower (hospitalisation) relative risks to estimate attributable morbidity.

The exposure prevalence is an estimate based on published surveys of the proportion of mothers who smoke postnatally.

Hospitalisations per year - we have estimated that three quarters of admissions in the 0-4 age group are aged less than 2 years. (Source: Selected Morbidity Data for publicly funded hospitals 1995-96. NZ Health Information Service, Ministry of Health 1998)

Cases of LRI per year: there are no routinely reported statistics. Workings in the table are based on an Australian longitudinal study which reported an incidence of 30 episodes of LRI per 100 children in the first 24 months in the homes of non-smokers [39]. There are approximately 140,000 NZ children in the 0-2 age group.

Our estimates of GP visits due to LRI are based on the WaiMedCa# survey which found that respiratory problems were identified in about 35% of GP encounters with pre-school children in the Waikato in 1991-1992, and about one in ten childhood respiratory problems were labelled as "bronchitis". It is likely that other lower respiratory illnesses were included in the headings of "viral infection" and "asthma", and so for this exercise we have assumed that 5% of GP encounters with children under 2 are due to a lower respiratory illness. New Zealand children under 5 make between 6 and 9 visits to a GP each year (Crampton P, personal communication). We have used an average of 7.5 encounters per year to estimate the total number of visits occurring in the country each year that result from LRI.

2. Asthma

Exposure means "mother smokes". It is estimated that 23% of New Zealand children live with a mother who is a current smoker (on the basis of Mitchell's report on smoking by parents of infants[40]). This is consistent with surveys of secondary school students that indicate approximately a third of New Zealand teenagers are exposed to SHS at home, and with smoking prevalence data (28% of women aged 25-44 reported that they were current smokers in the 1996/97 New Zealand Health Survey[41]).

The prevalence of wheeze in childhood was estimated from the ISAAC studies to be about 27% (30.2% age 6-7; 24.5% age 13-14 [42]). The total numbers of NZ children were estimated to be 279,603 (0-4 years) and 832,083 aged 6-15. (Statistics New Zealand)

Admissions to public hospitals due to asthma in 1997/8 were 9738 (including 1246 day patients), with an average inpatient stay of 3.1 days. [43] Over half these were persons under 18 years.

Asthma made up 22% of all childhood respiratory problems reported in the WaiMedCa study. We estimate from the WaiMedCa data that 7.7% of GP encounters for children are due to asthma, and assuming GP visit rates of 7.5 per year (under 6) and 5 per year (6-15 years), and populations of 350,000 children under 6 and 558,000 6-15 years, this amounts to 202,125 GP visits each year for asthma in the younger age group and 214,830 visits in the 6-15 years group.

3. Chronic otitis media

Exposure in these calculations means "either parent smokes". We estimate this proportion for children aged 0-9 (since older children are rarely admitted to hospital for this condition) is 0.3. Relative risk is taken as the midpoint of the range of quoted estimates (1.21-1.48) - 1.35.

Ear infections were the most frequently reported childhood problem in the WaiMedCa study, occurring in slightly more than 20% of GP encounters in the 1-14 age group. There was no distinction made in this survey between acute and chronic infections. We have assumed a 50/50 distribution, and on this basis estimate that 10% of GP encounters in the 0-9 age gr0up result from chronic otitis media. We have used a figure of 5 GP encounters per year and a national population of 570,000 children aged less than 10 years to estimate the total number of GP visits due to chronic OM.

4. Myringotomy for treatment of glue ear (with or without insertion of tubes)

There were 8584 operations for insertion of grommets reported in the public sector purchasing data (1995-6) - 89% were day cases (presumably the remaining cases were inpatients). Total day patient admissions in the private hospital sector in 1995 for myringotomy were 7290.

5. Coronary heart disease

Exposure in the epidemiological studies was defined as "spouse who smokes". We assume this is equivalent to "living with someone who regularly smokes around them inside the house", and note that there appears to have been little change in the proportion exposed by this definition during the 1990s[1]. However exposure at work was more common seven years ago (the estimate for men and women combined is 23.5%)

[#] McAvoy B//Davis P//Raymont A//Gribben B, The Waikato Medical Care (WaiMedCa) Survey 1991-1992, NZMJ 1994; 107: 388-433

The susceptible group includes never-smokers and ex-smokers: we assume that this constitutes half the total of male cases and 64% of female cases (refer to mortality report). Overall we estimate that 55% of the admissions due to IHD (ICD 410-414) and operations for ischaemic heart disease involved never- or ex-smokers. Admissions data refer to 1997/98[43]; surgery figures are based on 1995/96 returns[44].

6. Stroke

In 1997/98 there were 4956 admissions for stroke in 35-74 age group (public hospitals only). We assume that 57% of these admissions involved never- or ex-smokers (50% of men, 64% of women), based on the findings of the Auckland Heart Study (R Jackson, personal communication)

7. Meningococcal disease

Exposure was defined in the relevant epidemiological studies as "living in a home with one or more smokers". Current prevalence of exposure in children 0-8 years is estimated to be 35%, based on surveys of younger age groups and findings from Auckland study of meninogoccal disease.[15] Since the onset of the serogroup B meningococcal disease epidemic in 1990 the total number of cases in New Zealand has varied from year to year, with a peak of 613 cases in 1997. Children under 8 make up about two thirds of cases. For the purpose of this exercise, we have assumed 600 cases per year, of which 400 occur amongst children 0-8 years.

8. Lung cancer

We have assumed that 8% of lung cancers in New Zealand occur among never-smokers (men and women combined). This is based on an estimate made in 1989 [35].

<u>Discussion</u>

The major findings are:

- the overall burden of illness due to SHS is substantial (more than 500 hospital admissions per year for children under 2 with chest illnesses, for example)

- the major impact is on childhood illnesses: asthma, meningococcal disease, glue ear and respiratory infections

- there are effects on adults of exposures both at home and at work: each year, for instance, admissions to hospital following heart attack include about 190 events that would not have occurred if all work places had been totally smoke-free

- Maori are likely to be more severely affected than non-Maori, since they are more commonly exposed to SHS and background rates of diseases are higher than in the non-Maori population

These figures are intended to be a guide to the size of the health problem caused by SHS in New Zealand. They are not precise measures, since there are many assumptions and unknowns in the calculations. Some of this uncertainty is inescapable when attributable risk calculations are carried out, and some of the uncertainty results from deficiencies in this particular data set.

The first point to bear in mind about attributable risk in general is that the arithmetic assumes no changes in other causes. In reality, all the conditions that are influenced by SHS are affected by other factors as well - for any disease there are multiple causes. We have assumed here that SHS is the first causal factor to be removed. In practice, other causes of disease are likely to alter before there are changes in levels of SHS. For example, the factors (many unknown) which have sustained the epidemic of meningococcal disease in New Zealand may recede in the next few years. If this was so, the total number of cases of disease and the number of cases attributable to SHS would both fall. (Another, more subtle consideration is whether the effect of SHS on the risk of disease would change. It is plausible, for example, that the effect of SHS on the risk of meningococcal disease depends on the extent of crowding in households. If crowding were reduced, the total number of cases of meningococcal disease would be expected to fall, <u>and</u> the RR associated with SHS might also be reduced even if the proportion of parents who smoked was unchanged.)

The most important uncertainties in the data available on SHS are to do with the relative risk estimates and the prevalence of exposure. The risk estimates for conditions graded '+++' in Table 1 are relatively robust, in our judgement. But the estimates for stroke and meningococcal disease are based on a small number of studies and need to be interpreted with caution. To include these conditions may lead to an over-estimate of the attributable morbidity. On the other hand it is likely that as evidence accrues at least some of the conditions listed in Table 2 (and not included in this report) will be accepted as SHS-related.

The estimates of prevalence of exposure are conservative. For example, the estimates of exposure to SHS at work exclude exposures occurring in lunch and tea breaks. Estimates of childhood exposures to SHS do not include exposures due to smoking by visitors to the home, or exposures occurring outside the home. Our calculations of attributable risk for LRI and asthma are based on the proportion of mothers who smoke, since the relevant relative risk estimates are based on this measure of exposure. However this excludes any effect of smoking by fathers, which is likely to be less than that resulting from mothers' smoking, but will not be negligible. In general, exposure to SHS is considerably under-reported. In a national survey in the United States, for example, cotinine was detected in saliva of 88% of the participants, but only 37% reported that they were aware of being exposed to SHS. [45] For all these reasons we believe the overall effect of errors relating to exposure will be to under-estimate the true attributable morbidity.

Where there are no data, we have assumed that the effect of SHS on rates of health service use is the same as the effect on disease incidence. For example, epidemiological studies report that new cases of asthma are 31% more common in children under 6 who are exposed to SHS (Table 4); we have assumed that hospital admissions for asthma in this age group are also increased by 31% when children are exposed to SHS. This assumption leads to error if SHS is related not just to incidence, but also to disease severity. If SHS causes asthma to be more severe than usual, for example, then the effect on hospital admissions will be greater than a 31% increase. There is some evidence to suggest that the error is not a large one, for respiratory illnesses at least. In a two year study of children admitted to Wellington hospitals with acute lower respiratory illness, there was no consistent association between severity of illness and exposure to SHS. (Al-Delaimy W, unpublished PhD thesis 1999)

The period between exposure and occurrence of disease is likely to be very short for the childhood conditions included in these calculations. However the exposures responsible for occurrence of cancer, heart disease and stroke may have taken place months to decades earlier. We don't know exactly what these intervals might be. Almost certainly there is a mix of early- and late-stage effects, as with active smoking. As approximations, we have used in this report figures of 15 years (lung cancer) and 7 years (heart disease and stroke), as with the mortality report[1]. This means that number of cases of lung cancer occurring in 2001 that are attributable to SHS depends on the frequency and intensity of exposures to SHS in the late 1980s; for heart disease and stroke the critical exposure period is the early 1990s.

Susceptibility is not a great issue for childhood illnesses, apart from possible modifying factors such as breast feeding (see below). However the calculation of attributable effects on adults varies considerably depending on whether ex-smokers are treated as susceptible to the effects of SHS. In our view, ex-smokers should be included in the population at risk of SHS-induced cardiovascular diseases. The reasons for this are laid out in the accompanying report on attributable mortality. However we have restricted the estimates of SHS-induced lung cancer to never-smokers.

Our calculations assume that the effect of SHS on disease risk is uniform across population sub-groups. This is not always the case. For instance, the effect of SHS on chest illnesses in infancy is much less if babies are breast fed rather than bottle fed [46], high intakes of vitamin C may reduce the damage done to blood vessels by tobacco smoke [47] and the increased risk of heart disease is greater in people who already have raised blood cholesterol levels[48]. But on the whole, not enough is known about the effects of SHS to take account of

variations in susceptibility in calculations of attributable risk. This is not a major problem if the burden of illness estimates are applied, as intended, to the national level. Extrapolations to sub-groups might be subject to error if the distribution of effect modifying factors differed from that in the national population.

We have assumed the effects of exposures at home are independent of those at work, and we have added together the number of cases of illness attributed to SHS at home and at work. If these effects are not independent of one another then our numbers will either over- or under-estimate the true burden of disease. Unfortunately there is no information on joint effects but any error resulting from this assumption is likely to be small. In the most recent NZ population survey it was estimated that only 12.5% of those exposed to SHS are exposed both at home and work[37].

The amount of illness that could be prevented if absolutely no-one was exposed to SHS in the future is slightly less than the attributable fraction of illness currently experienced. The reason is that some of the conditions included in our calculations have long latent periods, and current levels of exposure to SHS are lower than those that applied in the past.

The number of potentially preventable hospitalisations is around 3,200 per year: this is approximately 0.6% of the total publicly funded inpatient episodes per year. A more detailed assessment of the direct costs is included as an Appendix to this report. Martin Tobias and Roberth Lynn (Ministry of Health) have taken the population attributable fractions from Tables 6 and 7 and applied these to disease-specific estimates of hospital costs. (Note that this excludes the costs of primary health care.) Total attributable hospital costs are estimated to be about $8.7 million per year

In conclusion, these estimates are subject to error and uncertainty, but we believe they provide a reasonably firm indication of the magnitude and nature of the burden of morbidity due to SHS. We have not attempted a formal economic costing of these effects, but the impacts clearly are expensive.

Hospitalisations attributable to exposure to second hand smoke

Martin Tobias

	Total discharges	Total daycases	ALOS	Total inpatient bed days	Average cost per case $	Attributable fraction	Total attributable hospital costs $
LRTI	6846	905	3.2	19110	2066	0.11	155 582
Asthma (0-5)	2647	609	1.8	3764	993	0.07	183 993
Asthma (6-14)	1447	245	2.2	2606	1018	0.04	58 922
Chronic O.M.	6103	5821	1.6	439	793	0.10	483 968
Myringotomy	2898	2366	2.3	1213	1046	0.10	303 131
IHD	26233	3345	5.1	116572	4243	0.04	4 452 265
Stroke (35-74)	4846	372	6.3	28206	4639	0.11	2 472 865
Lung cancer	1835	306	6.6	10113	3810	0.03	209 741
Meningo dis	524	26	4.9	2434	5652	0.12	355 398
Total							8 675 865

Notes

1. I have used the most recent data available, since the estimates are future orientated: 12 months to 30 June 2000
2. Data source is NMDS public hospital data (NZHIS) filtered to exclude duplicates, transfers, certain other categories (eg well babies)
3. Inpatients are defined as having stayed at least one night in hospital. Patients admitted but with length of stay of 0 nights are regarded as daypatients
4. Note that some admissions for meningococcal disease and stroke are recorded as daypatients, although this is unlikely – these may represent deaths in hospital in the first 12 hours post admission
5. Attributable fractions are those calculated by Woodward and Laugeson, applied to the most recent data. The fractions have been rounded to the nearest 2 decimal points. For IHD and stroke, the average of home and work exposures has been used. For stroke, the average of male and female has been used. IHD is an inclusive category that includes AMI. It has been assumed that admissions for Chronic otitis media and myringotomy are non-overlapping. Note that for meningococcal disease, hospitalisations and notifications approximately coincide
6. Average costs per case are based on the cost weights used by the HFA for deriving 1999/00 contracted prices for medical and surgical services. These weights incorporate both daypatient and inpatient services.
7. I am grateful to Robert Lynn for providing the cost weights and hospital throughput data.

8. The total direct cost attributable to SHS is estimated to be $8.7 million per year at present (0.3% of current annual public expenditure on hospital services). Approximately 75% of the total direct hospital cost attributable to SHS is accounted for by IHD and stroke-related hospitalisations.

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