

Appendix 2: Methods for attributing risk

In this section we describe our methods for assessing the contribution of 14 health risks to the total burden of disease and injury in Australia. For most risks, our analyses are based on methods developed by the WHO CRA project and described in detail elsewhere (Ezzati et al. 2004a). Briefly, the main inputs are the prevalence of exposure to a health risk in a population and information on the risk of disease, injury or death (referred to here as relative risk or hazard) from this exposure, which is typically derive from systematic reviews of the international literature. Our analyses are not comprehensive since choices had to be made about which risks to include on the basis of certain criteria, as outlined at the beginning of Chapter 4. We begin by describing the methodological basis of our analyses, the population attributable fraction.

Estimating population attributable fractions

The population attributable fraction (PAF) is a subtype of a more general measure – the ‘potential impact fraction’ (PIF). The PIF measures the proportional reduction in disease or injury burden experienced by a population that would occur if the population were subjected to an alternative or ‘counterfactual’ distribution of exposure to a particular health risk. If the alternative exposure scenario is set to a level such that it represents the lowest possible risk in a population (no exposure, for example), the PIF represents the total amount of burden that is attributable to that risk; in this instance it is called the ‘population attributable fraction’ (Eide & Heuch 2001; Miettinen 1974). For health risks that are measured on a continuous scale, the PIF can be defined thus:

$$PIF = \frac{\int_{x=0}^m RR(x)P(x) dx - \int_{x=0}^m RR(x)P'(x) dx}{\int_{x=0}^m RR(x)P(x) dx}$$

Where $RR(x)$ = relative risk at exposure level, $P(x)$ = population distribution of exposure, $P'(x)$ = counterfactual distribution of exposure, and m = maximum exposure level

(Equation 1)

When a risk is measured on a categorical scale, the discrete version of the PIF formula is (Eide & Heuch 2001; Walter 1980):

$$PIF = \frac{\sum_c P_c RR_c - \sum_c P_c^* RR_c}{\sum_c P_c RR_c}$$

Where c = an index for category, P = prevalence, and P^* = prevalence after a change, and RR = relative risk

(Equation 2)

The difference between Equation 1 and Equation 2 in practical terms is that the latter can easily be resolved in a spreadsheet environment, whereas the former requires more advanced mathematical techniques. Equation 2 is mathematically the same as the PAF formula for risk factors with multiple categories given by English and colleagues (Equation 3), if the counterfactual is set as the hypothetical minimum distribution (English et al. 1995).

$$PAF = \frac{\sum_c P_c (RR_c - 1)}{\sum_c P_c (RR_c - 1) + 1}$$

(Equation 3)

Choice of theoretical minimum

Calculating a PAF requires the explicit characterisation of an exposure distribution that represents the lowest possible level of risk in a population. This has been termed the ‘theoretical minimum exposure distribution’ and corresponds to zero exposure for some risks (for example smoking). For other risks, however, zero exposure is inappropriate because it is physiologically impossible (for example systolic blood pressure, BMI and cholesterol). In this case the lowest levels observed in specific populations and epidemiological studies described in the literature are used instead. For example, a theoretical minima of 115 mmHg for systolic blood pressure and 3.8 mmol/L for total cholesterol (each with a small standard deviation) are the lowest levels at which the dose-response relationships have been characterised (Chen et al. 1991; Eastern Stroke and Coronary Heart Disease Collaborative Research Group 1998; Law et al. 1994). For factors with protective effects (fruit and vegetable consumption and physical activity), the theoretical minimum exposure distribution is based on information from high exposure populations about the level to which the benefits continue to accrue given current scientific evidence.

Estimating attributable burden

Age- and sex-specific PAFs are calculated for each health risk and health outcome pair using the relationships in Equations 1 and 2. Where a relative risk of disease or injury is different to the relative risk for death, two PAFs are calculated, one for non-fatal burden and the other for fatal burden. PAFs are then multiplied with the relevant burden estimates for that health outcome and the sum of the burden across all outcomes affected by a health risk constitutes the total attributable burden for that risk. For example, if there are 1,000 deaths from ischaemic heart disease and 500 from stroke in a particular age and sex category, and the PAFs for cholesterol leading to ischaemic heart disease and stroke are 0.5 and 0.3 respectively, the mortality attributable to high cholesterol equals $1,000 \times 0.5 + 500 \times 0.3 = 650$. In other words, if the population had been exposed to the hypothetical minimum cholesterol distribution instead of the current distribution, 650 fewer deaths would have occurred.

Table A2.3 summarises the exposure levels, theoretical minima, health outcomes and sources of relative risks for each of the 14 health risks analysed in this report. Table A2.4 summarises

our estimates of exposure in the Australian population to each of these risks. A brief description the specific methods we used for each risk is provided below.

Tobacco

Given the long lag time between exposure to tobacco smoke and the occurrence of cancers and COPD, the attributable burden cannot be estimated from the current prevalence of smoking. Even with good historical information on smoking prevalence, it is not straightforward to determine the current amount of illness that is due to smoking because the lag time between the relevant exposure and disease is variable. Therefore, we used the method of Peto and colleagues, who proposed an artificial compound prevalence measure of the relevant past exposure to tobacco (Peto et al. 1992). This 'smoking impact ratio' is derived from a comparison of lung cancer mortality rates in the population of interest and lung cancer mortality rates among non-smokers and smokers observed in a large long-term follow-up study in the United States. We used this smoking impact ratio instead of the current prevalence in the standard calculation of attributable fractions for the other cancers and COPD. Compared with cancers and COPD, the mean time between exposure to tobacco and all other adverse health outcomes is considerably shorter. We therefore used prevalence estimates of smoking for adults aged 18 years and over in 2001, two years before our baseline year of 2003 (ABS 2001c).

Our previous calculations of attributable mortality burden included only those diseases for which English and colleagues report strong evidence of an association (English et al. 1995). For this report, we added other conditions for which reasonable evidence of an association with tobacco exists (AIHW: Ridolfo & Stevenson 2001): cancer of the stomach, endometrial cancer, peripheral vascular disease, pneumonia, inflammatory bowel disease, injuries from fires and Parkinson's disease. Tobacco has a small protective effect against Parkinson's disease and endometrial cancer. We omitted peptic ulcer disease, given evidence of its largely infectious aetiology. We also added the burden attributable to smoking from macular degeneration (Mitchell et al. 1999; Tomany et al. 2004).

In addition, we calculated the burden from passive smoking using attributable fractions for lung cancer, ischaemic heart disease, and asthma in children (NHMRC 1997a). For lower respiratory tract infection, sudden infant death syndrome and otitis media in children due to passive smoking, we used the prevalence of maternal smoking (Turrell et al. 2002) and relative risks from the US Surgeon General's Report (US Department of Health and Human Services 2006) and NHMRC (NHMRC 1997a). We also estimated the burden of low birth weight due to smoking during pregnancy using the relative risk from Ridolfo and Stevenson (2001) and estimates of smoking during pregnancy from Laws and Sullivan (2005).

High blood pressure

We used the AusDiab study (Dunstan et al. 2002) to estimate distributions of high blood pressure by age and sex in the Australian population. Despite a low response rate AusDiab is the only recent and representative study that has measured this risk in Australia. Relative risks came from Lawes and colleagues (2004a). We used the CRA theoretical minimum distribution for blood pressure (mean 115, SD 6 mmHg) as the counterfactual in this analysis.

High body mass

We used the AusDiab study (Dunstan et al. 2002) to estimate distributions of body mass index (BMI) by age and sex in the Australian population. Relative risk of type 2 diabetes came from the Asia Pacific Cohort Collaboration (2006); the relative risk of all remaining conditions associated with high body mass came from James and colleagues (2004). We used the CRA theoretical minimum distribution for BMI (mean 21, SD 1 kg/m²) as the counterfactual in this analysis.

Physical inactivity

Recent developments have led to the treatment of physical inactivity as a four-level categorical variable by subdividing the exposure group labelled as 'sufficiently active' in the CRA project into those 'meeting current recommendations' and 'highly active'. While physical activity levels equivalent to 2.5 hours per week of moderate-intensity activity (approximately 4000kJ/week) are considered an important target for population health benefits, the protective effects are expected to continue to higher levels. Therefore, the theoretical minimum exposure distribution was chosen to be the whole population in the 'high active' category to increase consistency with the counterfactual exposure distribution of other risk factors (Bull 2003; Murray et al. 2003; Powles & Day 2002) (Table A2.1). The required prevalence data were derived from the NHS 2001 (ABS 2001c). The exercise related questions in this survey relate to physical exercise undertaken for recreation, sport, health or fitness purposes, conceptually excluding physical activity undertaken as a part of work or for other purposes. This may underestimate the amount of physical activity undertaken, and therefore our analyses may overestimate the burden of disease attributable to physical inactivity.

The associated hazards were modified to correspond to the new referent category of 'highly active'. Given no available quantitative meta-analysis with comparable categories, risk estimates were derived from a synthesis of recent reviews (Kelley & Goodpaster 2001; Kesaniemi et al. 2001; Kohl 2001; Oguma et al. 2002; Thune & Furberg 2001; Williams 2001) and findings from several recent studies in which the results were reported separately by intensity of activity as well as total volume of activity (Manson et al. 2002; Sesso et al. 2000). The relative risk of ischaemic heart disease for the inactive group compared to 'high active' was set at 2.0, based on reviews of studies with both physical activity and fitness measures as well as a recent study's differential results for moderate versus vigorous activity. The likely linear dose-response relationship (Kesaniemi et al. 2001) was represented by the arithmetic midpoints for those classified as 'meeting current recommendations' and 'insufficiently active'. For stroke, the mean of nine studies summarised in the systematic review and meta-analysis by Blair and colleagues (2001) was used (relative risk of 2.0). The findings from the review by Thune and Furberg (2001) were used to derive the risk estimate for colon and breast cancer. There has been no quantitative review of diabetes and physical activity; therefore the relative risks from the CRA project were adjusted by the same magnitude as for ischaemic heart disease. It is recognised that these estimates of risk are derived from a synthesis of the available scientific evidence and alternative interpretations are possible.

Table A2.1 Physical activity exposure categories

Physical activity level	Definition
High	3 sessions x at least average 40 minutes vigorous AND total of at least 1500 METmins/week ^(a)
Recommended	3 sessions x at least average 20 minutes vigorous OR 5 x 30 minutes moderate OR 600 METmins/week
Insufficient	Some activity but not meeting recommendation
Inactive	No activity

(a) The standard metabolic equivalent, or MET, level. This unit is used to estimate the amount of oxygen used by the body during physical activity. One MET = the energy (oxygen) used by the body sitting quietly, perhaps while talking on the phone or reading a book. The harder the body works during the activity, the higher the MET.

High blood cholesterol

We used the AusDiab study (Dunstan et al. 2002) to estimate distributions of high blood cholesterol by age and sex in the Australian population. Relative risks came from Lawes and colleagues (2004b). We used the CRA theoretical minimum distribution for serum cholesterol (mean 3.8, SD 0.5 mmol/L) as the counterfactual in this analysis.

Alcohol

There are a number of recent data sources on the prevalence of alcohol consumption in the Australian population, including the 2004 National Drug Strategy Household Survey (NDSHS 2004) (AIHW & DoHA 2005) and the 2001 National Health Survey (NHS 2001) (ABS 2001c). The NHS 2001 focuses on the quantity of alcohol consumption on the three most recent days on which alcohol was consumed in the week prior to interview, while the NDSHS 2004 explicitly quantifies the amount of alcohol drunk on the day prior to interview. Of these, only the NHS 2001 collected information on the type and brand of alcoholic drinks consumed as well as the number. Also, the NHS 2001 gives average daily alcohol consumption over the previous week in millilitres. For this reason, we used the NHS 2001 to estimate the prevalence of alcohol consumption for adults aged 18 years or over.

We categorised the prevalence of alcohol consumption into the four levels used in English and colleagues' analysis of the risks of alcohol consumption (English et al. 1995), and with the NHMRC's recommendations on alcohol consumption (NHMRC 1992) (Table A2.2). The prevalence of each level of alcohol intake was estimated by age and sex from the average weekly consumption of alcohol after conversion to standard drinks per day. Data for people interviewed on each day of the week were reweighted to obtain prevalence of alcohol consumption based on equal samples for each day of the week. Those that last drank alcohol more than 1 week ago were classified as abstainers.

Table A2.2 Classification and prevalence of alcohol intake levels used in this report

Alcohol intake	Average number of standard drinks (= 10 g alcohol) per day	
	Males	Females
Abstinence	0–0.25	0–0.25
Low	0.26–4.00	0.26–2.00
Hazardous	4.01–6.00	2.01–4.00
Harmful	>6	>4

Source: English et al. 1995

We used relative risks and population attributable fractions from Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001) for conditions for which there is evidence of causation by alcohol consumption. English and colleagues (1995) estimated that 44% of fire injuries are attributable to alcohol; this was not updated by Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001). We revised these estimates with the addition of more recent studies, and produced a separate PAF for fire injuries and scalds or other burns for both YLD and YLL. We also updated the drowning PAF of 0.34 from Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001) with age-specific estimates from Driscoll and colleagues (AIHW: Driscoll et al. 2004) who found that 17% of unintentional drownings were attributed to alcohol (blood alcohol content of at least 0.10 g/100 ml). English and colleagues (1995) derived a PAF of 0.07 for alcohol, and occupational and machine injuries. We applied this to all machinery accidents, and to the occupational YLD PAFs for injury codes not already covered elsewhere in alcohol. For YLL we applied a PAF of 0.051 (Driscoll et al. 2001) to the occupational YLL PAFs for injury codes not already covered elsewhere in alcohol.

Low fruit and vegetable consumption

We used the National Health Survey (ABS 2001c) to estimate distributions of fruit and vegetable consumption by age and sex in the Australian population. Relative risks came from Lock and colleagues (2004). We used the CRA theoretical minimum risk distribution for fruit and vegetable consumption (mean 600, SD 50g/day) as the counterfactual in this analysis.

Illicit drugs

In addition to being a direct cause of death, illicit drugs are also risk factors for conditions such as HIV/AIDS, hepatitis, low birth weight, inflammatory heart disease, poisoning, and suicide & self-inflicted injuries. By definition, heroin, benzodiazepine, cannabis and other drug dependence and harmful use are due to illicit drug use; therefore the entire burden due to these conditions was attributed to this risk factor category. For infective endocarditis and suicide we used the attributable fractions for illicit drugs developed by English and colleagues (1995). The proportion of inflammatory heart disease that was due to infective endocarditis was derived from hospital data. The infective endocarditis PAF was then applied to this proportion only.

The proportion of HIV due to injecting drug use was based on diagnosed HIV from the Australian HIV Public Access Dataset (National Centre in HIV Epidemiology and Clinical Research 2005b). We use diagnosed rather than newly acquired HIV, which is in keeping

with YLD estimates, and due to the apparent stabilisation of HIV incidence over recent years. AIDS cases and deaths attributable to injecting drug use were from the Australian AIDS Public Access Dataset (National Centre in HIV Epidemiology and Clinical Research 2005a). Time to death (year of death minus year of AIDS diagnosis) was added to the midpoint of the age at diagnosis range to approximate age range at death. For those age-at-death ranges available in the AIDS Public Access Dataset, we used the age-specific proportion attributable to injecting drug use. For all other ages we applied the all-age proportion. For cases of HIV and AIDS, and AIDS deaths, we assumed that all cases with exposure category 'male homosexual contact and injecting drug use' were attributable to male homosexual contact.

The proportion of newly acquired hepatitis B and C cases due to injecting drug use was from the HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia Annual Surveillance Report 2004 (National Centre in HIV Epidemiology and Clinical Research 2004).

The proportion of road traffic accidents due to illicit drug use was derived from Drummer and colleagues (2004). We applied the methodology used by Ridolfo and Stevenson on earlier data from Drummer (1994).

For low birth weight we used prevalence of cannabis and opioid diagnosis during pregnancy in New South Wales and relative risks from Burns and colleagues (2006). The relative risk for antepartum haemorrhage attributable to illicit drug use was from English and colleagues (1995), with the prevalence being heroin or cocaine use in past 12 months for females aged 15–49 years from the NDSHS 2004.

The odds ratio for schizophrenia attributable to cannabis use was from Semple and colleagues (2005). This odds ratio is the result of a meta-analysis of seven studies with different classifications of psychosis and cannabis use. Despite these differences, there was consistency in the unadjusted odds ratios. We used prevalence of daily cannabis use over the last 12 months from the 2004 NDSHS (AIHW & DoHA 2005) to calculate the PAF to be applied to schizophrenia.

Occupational exposures and hazards

The attributable burden of occupational exposures and hazards was based on the following methods. Work-related fatal injuries were derived from the National Worker's Compensation Statistics Database accessed online via National Occupational Health and Safety Commission (NOHSC) Online Statistics Interactive (NOSI) <www.nosi2.nohsc.gov.au/>. Since compensation statistics do not cover all occurrences of occupational injury deaths, we inflated these figures according to a study carried out by Driscoll and colleagues (AIHW: Driscoll et al. 2004), that investigated the coverage of work-related traumatic deaths by official occupational health and safety and compensation agencies in Australia. Work-related deaths by mechanism, nature and industry from the NOSI database were inflated according to the proportion of all work-related deaths in 1989–92 covered by compensation agencies by industry (Table 3 in AIHW: Driscoll et al. 2004).

In the absence of more reliable information, the attributable fractions for non-fatal injuries were derived from an analysis of the National Hospital Morbidity Database 2002–03. For each age–sex–injury group, the attributable fraction for occupational injuries was estimated as the ratio of hospital episodes where 'workplace' was specified as the place where the injury occurred to the total hospital episodes where a place of occurrence was specified.

Where possible we derived non-injury attributable fractions by following the CRA methods (Concha-Barrientos et al. 2004). This produced age- and sex-specific attributable fractions for

lung cancer, leukaemia, COPD, asthma, adult onset hearing loss, and chronic back pain (which we also applied to slipped disc). For each of the remaining cancer categories, we derived attributable fractions from a study carried out for the National Institute of Occupational Health and Safety (Kerr et al. 1996). This study also provided attributable fractions for a number of other chronic diseases, including neurological disorders, cardiovascular diseases, chronic respiratory diseases and renal disease. Attributable fractions for osteoarthritis were derived separately, based on relative risks of self-reported arthritis for blue collar workers compared to managers, administrators and professionals (AIHW: Turrell et al. 2006).

Child sexual abuse and intimate partner violence

Girls that experience child sexual abuse are more likely to experience intimate partner violence than non-abused girls (Mouzos & Makkai 2004). Women that experience multiple types of abuse, including child sexual abuse and intimate partner violence, have a higher risk of depression than those subject to only one form of abuse (Arias 2004; Messman-Moore et al. 2000; Nicolaidis et al. 2004). The 2001 Victorian Burden of Disease Study produced estimates of the burden attributable to intimate partner violence but did not calculate the burden attributable to child sexual abuse (DHS 2005; Vos et al. in press). Conversely the CRA project (Andrews et al. 2004) produced estimates of the burden attributable to child sexual abuse but not intimate partner violence. In this study we estimated the burden attributable to child sexual abuse and intimate partner violence. Further, to avoid over-estimating the burden when both of these risk factors are present we estimated an adjusted relative risk to account for the combined exposure state of having experienced both child sexual abuse and intimate partner violence.

We estimated the prevalence of 'intimate partner violence without child sexual abuse' and 'child sexual abuse and intimate partner violence combined' from the Women's Safety Survey (ABS 1996). We used two categories of exposure to intimate partner violence, namely physical or sexual violence by a partner in the last 12 months, and physical or sexual violence by a partner more than 12 months ago. Given that the Women's Safety Survey asks only one question regarding child sexual abuse ('Whether experienced sexual abuse when a child') we used the CRA project priors for Australia for the prevalence of child sexual abuse (based upon epidemiological studies) and assumed no trend in prevalence of child sexual abuse. We subtracted the prevalence of 'child sexual abuse and intimate partner violence combined' from the child sexual abuse priors to estimate the prevalence of child sexual abuse without intimate partner violence.

Messman-Moore and colleagues (2000) looked at the mean psychological functioning indices for women who had experienced (a) both contact child sexual abuse and adult victimisation (revictimisation); (b) adult victimisation only (multiple or once only); (c) contact child sexual abuse only; and (d) no abuse history. From these group means and standard errors we calculated an effect size using Hedges' adjusted *g* for standardised mean difference (Egger et al. 2001). We then converted the effect sizes into odds ratios for risk of depression, anxiety and post-traumatic stress disorder by exposure group using the methods described by Hasselblad and Hedges (1995).

These odds ratios, along with relative risks for contact child sexual abuse from the CRA project (Andrews et al. 2004), and relative risks for intimate partner violence from the Women's health Australia study (see DHS 2005: page 29) were then used to derive relative

risks for 'contact child sexual abuse only', 'intimate partner violence only', and 'child sexual abuse and intimate partner violence combined'.

Since Messman-Moore and colleagues (2000) define child sexual abuse as contact only, for non-contact child sexual abuse we used the CRA project relative risks and prevalence for that category unadjusted. In our main results we combined anxiety and depression together into one category. We therefore found the mean relative risk from the derived relative risks for depression, anxiety, and post-traumatic stress disorder symptoms. We applied the same relativities from the anxiety and depression relative risks for child sexual abuse only, intimate partner violence only, and combined child sexual abuse and intimate partner violence, to the intimate partner violence and child sexual abuse relative risks for other conditions (alcohol use disorders, other drug use disorders, and self-inflicted injuries).

For ease of reporting, the population attributable fraction calculated for the 'combined child sexual abuse and intimate partner violence category' was proportionately redistributed to either child sexual abuse or intimate partner violence. To calculate the population attributable fractions for those disease categories that only apply to intimate partner violence (smoking, cervical cancer, sexually transmitted diseases, eating disorders and physical injuries) we used the relative risks for intimate partner violence from the Women's health Australia study, and the prevalence of intimate partner violence (including those women who may have also experience child sexual abuse) from the Women's Safety Survey. The proportion of homicide due to intimate partner violence (52%) was from the 2003–2004 National Homicide Monitoring Program Annual Report (Mouzos 2005). Violence YLD was based on the proportion of hospitalisations for assaults where the relationship of the victim of assault to the perpetrator was recorded as spouse or domestic partner (including ex-spouse and ex-partner). The assaults where this relationship was unspecified were proportionately redistributed.

Due to a lack of data on the prevalence of intimate partner violence among males, and on the related health outcomes, for males we only estimated the burden due to child sexual abuse. Analyses were based on methods developed for the CRA project described elsewhere (Andrews et al. 2004). We used the CRA priors for Australia for the prevalence of male child sexual abuse (based upon epidemiological studies).

Urban air pollution

Numerous studies have documented that urban air pollution has a range of effects on health, from irritated eyes to death. The effects of short-term exposure are generally demonstrated through time-series studies on daily events (for example mortality, hospitalisations, emergency department attendance) (Cohen et al. 2004; Simpson et al. 2005a, 2005b). The effects of long-term exposure have been demonstrated in large cohort and cross-sectional studies, mainly in the US and Europe (Cohen et al. 2004; Pope et al. 2002). We estimated the burden due to both long- and short term exposure to urban air pollution, and present the results for long-term exposure only as a minimum estimate, and the combination of long- and short-term exposure as a more inclusive but less certain higher estimate.

Long-term exposure

For chronic exposure to urban air pollution, our analyses are based on methods developed for the CRA project (Cohen et al. 2004). The main data inputs were: (a) annual 24-hour average particulate matter concentrations (particulate matter with an aerodynamic diameter

of less than 10 and 2.5 micrometres, PM₁₀ and PM_{2.5}) as an indicator of exposure to pollution from combustion sources; and (b) information on the relative risk of mortality. In the CRA method, the population attributable fraction was calculated from these inputs as the difference in disease experience in a population and the hypothetical disease experience if the population were exposed to the hypothetical minimum of particulate matter (PM_{2.5} 7.5µg/m³; PM₁₀ 15µg/m³). However, there is evidence that there may be no safe level of exposure to particulate matter (WHO Europe 2004). We therefore set the theoretical minimum to zero in our analyses.

Our estimates for long-term exposure are based on the contributions of two health outcomes: cardiopulmonary disease and lung cancer in adults aged 30 years and older. Attributable burden was estimated using risk coefficients from a large cohort study of adults in the United States (Pope et al. 2002). We did not use the CRA method of attributing acute respiratory infection in children aged 0–4 years as this method applies a relative risk based on daily exposure to annual exposure levels. Given the availability of daily urban air pollution data in Australia, and more appropriate relative risk estimates from Australian pollution concentration and mortality data, we used the estimates generated using the short-term effects methods described below.

We based exposure on annual mean levels for 2002 in the following urban areas: Sydney, Newcastle, Wollongong, Melbourne, Geelong, Brisbane, Perth, Adelaide, Canberra (including Queanbeyan), and Hobart. Annual concentrations were derived from data supplied by the state and territory environmental protection authorities, except for Adelaide and Hobart where we used published estimates (DPIWE 2004; Gooding & Riordan 2004). PM_{2.5} concentration was not available for Geelong, Hobart or Canberra. For Geelong, we estimated the concentration from Melbourne's PM₁₀:PM_{2.5} ratio. For Hobart and Canberra, we based our estimates on the average PM₁₀:PM_{2.5} ratio for those cities with original data (that is, Brisbane, Melbourne, Perth, Sydney, Adelaide, Newcastle and Wollongong). Due to temporal trends in particulate matter concentration, the linking of current exposure to chronic outcomes may underestimate the attributable burden if exposure levels were higher in the past. However, the use of recent exposure data is in keeping with the CRA methods.

Short-term exposure

Short-term exposure to urban air pollution has been associated with day-to-day variations in hospital admissions and mortality (Simpson et al. 2005a, 2005b). However, translating these findings into burden of disease estimates is not straightforward. The difficulty with estimating attributable morbidity is that published risks are established for the impact on hospitalisations only. An increase in hospitalisations for causes related to urban air pollution is likely to largely reflect exacerbation of existing disease rather than new disease events. Our YLD estimates are based on incident cases and their average duration at a particular level of severity. Thus the impact of urban air pollution on morbidity needs to be estimated as either a proportion of new cases of disease or a worsening of the condition for an undefined period of time. Until these methodological issues can be resolved we consider only a mortality component of the short-term health consequences of urban air pollution.

The problem with attributing mortality to the short-term impact of urban air pollution is that there is equivocal evidence regarding the extent of 'harvesting', that is, imminent deaths brought forward by only a short period of time (less than a month) that were imminent anyway, or 'new deaths' that would not have occurred in the absence of urban air pollution. This has a major bearing on our estimates of YLL: if harvesting occurs, YLL will be only a

fraction of that normally calculated for each death. There is much debate in the literature on this topic. There are some arguments that harvesting does not play a role in the effects of urban air pollution. For instance there is an increase in deaths when longer lags between exposure and outcomes (up to 4 months) of urban air pollution are estimated, rather than a decrease (Schwartz 2001; Zeger et al. 1999). (The need to control for seasonal variation in these analyses makes it difficult to extend these analyses over the longer term as longer lags become strongly correlated with seasonal changes). This finding has been interpreted to indicate that harvesting is not an important issue. However, it could also be the case that urban air pollution exposure leads to chronic rather than acute effects on mortality. A further argument put forward by the same authors is that the largest increase in deaths was seen in people dying outside a hospital, while one would have expected a greater increase in hospital deaths if harvesting were bringing deaths forward in people who were already ill. The authors do not comment, however, on whether this may be due to the protective effect of the hospital environment. We concluded that there is no consensus on the relative contribution of deaths brought forward by urban air pollution nor on the size of the true acute impact on mortality. We therefore present the chronic impact as a lower estimate of the burden due to urban air pollution and add an alternative estimate of the combined short-term and long-term effects, ignoring any harvesting, as an upper bound.

Recent Australian research has provided the most applicable risk coefficients describing the effect of short-term exposure to urban air pollution on mortality (Simpson et al. 2005b). We applied these to daily urban air pollution data to estimate the attributable mortality burden of this risk. Following expert advice, our estimates were based on an averaged 0–1 day lag (that is, exposure to urban air pollution on the day of death and the day before death) of the contributions of two pollutants to two causes of death: all cause mortality (excluding accidental and other external causes of death) due to particle exposure (in units of light scattering by nephelometry, bsp), and respiratory deaths due to exposure to ozone. The choice of including these two pollutants and excluding others was made after discussion with the researchers (Simpson, Williams and Barnett) and justified by the finding that the impact on mortality of NO₂, CO and particles largely overlaps and hence including all three would lead to overestimation. The impact of SO₂ is considered small in Australia but ozone has a significant impact on respiratory mortality independent of that of other pollutants.

Estimates were calculated with a theoretical minimum exposure level of zero. This is based on evidence that at the population level there appears to be no safe level of exposure to particles or ozone (WHO Europe 2004).

A decision was made to work with exposure data from 2002 rather than 2003 (the reference year for our study) because 2003 is considered an outlier year by the environmental protection authorities for pollutant readings. Daily urban air pollution data were supplied by the Victorian, New South Wales, Australian Capital Territory, Queensland and Western Australian environmental protection authorities. We calculated a PAF for each day by urban area, pollutant, and underlying cause of death, with the assumption that the entire population of that urban area was exposed. This was applied to daily 2002 mortality data, and aggregated to age- and sex-specific annual PAFs. We aggregated the number of deaths and YLL attributable to urban air pollution in specific areas (Sydney, Newcastle, Wollongong, Melbourne, Geelong, Brisbane, Perth, Adelaide, Canberra including Queanbeyan, and Hobart), calculated this as a proportion of all deaths or YLL in Australia and, finally, applied this proportion to 2003 mortality estimates.

We did not gain access to daily Tasmanian or South Australian urban air pollution data. Particle levels for Adelaide and Hobart were therefore extrapolated from published annual mean PM₁₀ levels (Air Monitoring Unit, EPA SA 2003; DPIWE 2004), and the average ratio of bsp:PM₁₀ for Brisbane, Sydney and Melbourne from Simpson and colleagues (2005b). The ratio of the extrapolated mean bsp for Adelaide and Hobart to the annual mean bsp level for the cities for which we had detailed exposure data was then applied to the annualised PAF for these cities to extrapolate the PAFs for the two cities with missing exposure data. Ozone levels for Adelaide were based on the published average for 2002 (Gooding & Riordan 2004). Ozone is not routinely monitored in Hobart (DPIWE 2006); we therefore did not include this region in our analysis of respiratory deaths due to ozone exposure.

Unsafe sex

All sexually transmitted diseases were attributed to unsafe sex. The PAFs for HIV/AIDS and hepatitis B and C due to unsafe sex were derived as described in the section on illicit drugs. Previous Australian and Victorian burden of disease studies have used a PAF of 0.90 for cervical cancer. In this study we attributed all cervical cancer to sexual transmission of the human papilloma virus. Munoz and colleagues (2003) found that 90.7% cases had HPV DNA detected. Similarly, in a meta-analysis Clifford and colleagues (2003) found that HPV DNA was present in 80–89% of cases. However, research by Walboomers and colleagues (1999), in which they revisited a previous study, suggests that nearly all cases that were negative for HPV DNA were false negatives. They revised up the estimates of cases testing positive for HPV DNA from 93% to 99.7%. Bosch and Munoz (2002) suggest that in most studies where 5–15% of cases are negative for HPV these are false negatives.

Osteoporosis

Osteoporosis causes no disability or death per se; it does, however, increase the risk of fracture. Therefore we treated osteoporosis as a risk factor in this study rather than as a disease in its own right, as was done in the previous Australian burden study. The WHO Task-Force for Osteoporosis recommends that the condition be defined by level of bone mineral density (BMD). We therefore based our PAF calculations on the population distribution of BMD, and relative risks associated with decreasing BMD.

In Australia there are two large studies that have measured population BMD, one based in Geelong and the other in Dubbo. Both the Geelong Osteoporosis Study and the Dubbo Osteoporosis Epidemiology Study state that the population they cover is representative of the Australian population (Nguyen et al. 2001; Sanders et al. 1998). Mean BMD and standard deviations (SDs) for the Geelong and Dubbo studies were supplied by the study custodians. We used Geelong data for ages <60 and combined Geelong and Dubbo data for 60 years or over by fitting a Weibull distribution. From this distribution we plotted BMD by age for ages 25 years or over and fitted a polynomial distribution ($R^2=0.998$). We then predicted mean BMD from this equation for 5-year age groups from 60 years.

For males, we assumed that the difference between the Dubbo and Geelong BMD means for women would also apply to males if Geelong data were available. We therefore increased Dubbo means by the ratio of female Dubbo sampled mean to the combined mean. We assumed deviations from the line were sampling error, and predicted mean BMD by age group from the fitted quadratic equation ($R^2=0.925$). We assumed the SD for Dubbo applied.

The WHO Task-Force for Osteoporosis recommends that the condition be defined in Caucasian women as a BMD 2.5 SDs or more below the young female reference mean (Genant et al. 1999). The Australia and New Zealand Bone and Mineral Society and Osteoporosis Australia recommended that data from the Geelong Osteoporosis Study be used to establish a standardised reference range for Australia (Henry et al. 2004). We therefore used the mean BMD and SD for young women aged 20–29 from this study as the theoretical minimum, and also used this population for the osteoporosis cut-off (Henry et al. 2004).

There is currently no Australian reference mean BMD and SD for young adult men. We estimated these values by multiplying the Australian young female mean and SD (Henry et al. 2004) by the ratio of male to female mean and SD from the USA's National Health and Nutrition Examination Survey (NHANES) (Looker et al. 1998). The NHANES used Hologic densitometers while both the Dubbo and Geelong studies used Lunar densitometers. These machines do not give standardised results. We therefore converted the Hologic estimates to Lunar by applying the formula available at <www.courses.washington.edu/bonephys/opBMDs.html>.

Relative risks and odds ratios from a number of studies were pooled to estimate the relative risk of low impact fracture per 0.1g/cm² decrease in BMD measured at the femoral neck (EPOS Group 2002; Fujiwara et al. 2003; Kroger et al. 1995; Nguyen et al. 2005a, 2005b; Papaioannou et al. 2005; Schott et al. 2005; Schuit et al. 2004; Stone et al. 2003). Where a study used Hologic or Norland densitometers, and the relative risk was per SD change in BMD, we converted the study's SD estimates to Lunar.

We derived PAFs for a number of fracture sites. Where possible these sites were linked directly to a single nature of injury category. In some cases (for example hip) we applied the PAF to a proportion of a category based on the distribution of fracture sites in the National Hospital Morbidity Database 2002–03. Since most studies that we included in the calculation of relative risks excluded fractures resulting from high impact causes, we applied the PAFs to fractures resulting from falls, striking and crushing accidents, and other unintentional injuries.

For attributable YLL, we applied the site-specific fracture YLD PAFs to the site-specific mortality distribution for vertebral, pelvis and femur fracture to derive a site-specific YLL PAF. This was applied to deaths with an underlying cause of falls, striking and crushing accidents, other unintentional injuries, ill-defined falls or osteoporosis, where a fractured spine, pelvis or femur was mentioned. If more than one fracture was mentioned we applied the larger PAF, that is, for fractured pelvis and femur we applied the PAF for femur. We assumed that all deaths with an underlying cause of osteoporosis but no mention of vertebral, pelvis, or femur fracture, were attributable to osteoporosis. To determine the burden of disease code-specific YLL PAF for osteoporosis we calculated the proportion of burden of disease code-specific deaths attributable to osteoporosis. Osteoporosis and ill-defined fall deaths were redistributed to falls. If we were to limit the deaths attributable to osteoporosis to only those that were coded to osteoporosis, the overall number of deaths would have been considerably smaller.

Table A2.3: Definitions, theoretical minima, health outcomes and data sources for 14 selected health risks

Health risk	Exposure variable	Theoretical minimum	Outcomes	Sources for exposure estimates	Sources for hazard estimates
High blood pressure	Level of usual systolic blood pressure	115 (SD 6) mmHg	Ischemic heart disease, stroke, hypertensive heart disease	AusDiab study (Dunstan et al. 2002)	Meta-analysis of 61 cohort studies with 1,000,000 North American and European participants (Prospective Studies Collaboration (Lawes et al. 2003))
High blood cholesterol	Level of usual total blood cholesterol	3.8 (SD 0.6) mmol/L (147 (SD 23) mg/dL)	Ischemic heart disease, ischemic stroke	AusDiab study (Dunstan et al. 2002)	Meta-analysis of 10 cohorts with 490,000 North American and European participants, and 29 cohorts with 350,000 participants from the Asia-Pacific region
High body mass index (BMI)	Body mass index, BMI (weight over height squared)	21 (SD 1) kg/m ²	Ischemic heart disease, stroke, hypertensive heart disease, diabetes, osteoarthritis, endometrial cancer, kidney cancer, colon cancer, post-menopausal breast cancer	AusDiab study (Dunstan et al. 2002)	Meta-analysis of 33 cohorts with 310,000 participants for cardiovascular disease risks, 27 cohorts for cancer risks, and systematic review of cohort studies for diabetes risk
Low fruit and vegetable consumption	Fruit and vegetable intake per day	600 (SD 50) g intake per day for adults	Ischemic heart disease, stroke, colorectal cancer, gastric cancer, lung cancer, oesophageal cancer	National Health Survey 2001 (ABS 2001c)	Systematic review and new meta-analysis of published cohort studies
Osteoporosis	Bone mineral density of the femoral neck	Males 1.107 (SD 0.140) g/cm ² , Females 1.018 (SD 0.127) g/cm ² With osteoporosis defined 2.5 or more SD below this mean	Fractured hip, femur, humerus, clavicle, forearm/wrist, elbow, spine, rib, pelvis, lower leg, patella, foot, heel, toe, hand, finger from falls, striking and crushing accidents, other unintentional injuries	Dubbo Osteoporosis Epidemiology Study (Nguyen 2005) and Geelong Osteoporosis Study (Kotowicz 2005)	Pooled analysis of 10 studies (EPOS Group 2002; Fujiwara et al. 2003; Kroger et al. 1995; Nguyen et al. 2005a, 2005b; Papaioannou et al. 2005; Schott et al. 2005; Schuit et al. 2004; Stone et al. 2003)

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Table A2.3 (continued): Definitions, theoretical minima, health outcomes and data sources for 14 selected health risks

Health risk	Exposure variable	Theoretical minimum	Outcomes	Sources for exposure estimates	Sources for hazard estimates
Physical inactivity	Four categories: inactive, insufficient, recommended level and highly active	All in 'highly active' group	Ischemic heart disease, stroke, breast cancer, colon cancer, diabetes	National Health Survey 2001 (ABS 2001c)	Systematic review of published literature and new meta-analysis of cohort studies
Tobacco	Past smoking	No smoking	COPD, cancers of mouth, oesophagus, lung, pancreas, larynx, bladder, kidney, stomach and uterus	Peto-Lopez method	Systematic reviews by English and colleagues (1995) and Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001)
	Current daily smokers	No smoking	Ischemic heart disease, stroke, peripheral vascular disease, Parkinson's disease, pneumonia (adults), fire injuries, macular degeneration	National Health Survey 2001 (ABS 2001c)	Systematic reviews by English and colleagues (1995) and Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001); Tomy and colleagues (2004) for age related macular degeneration
	Passive smoking	No smoking	Ischemic heart disease, stroke	National Health Survey 1995 (ABS 1995)	Systematic reviews by English and colleagues (1995) and Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001)
	Maternal smoking; smoking while pregnant	No smoking	Asthma, pneumonia (children), sudden infant death syndrome, otitis media, low birth weight	National Health Survey 2001 (ABS 2001c), <i>Australia's Mothers and Babies 2003</i> (AIHW: Laws & Sullivan 2005)	Systematic reviews by English and colleagues (1995) and Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001); US Surgeon General's Report on Involuntary exposure to tobacco smoke (US Department of Health and Human Services 2006); systematic review by Anderson and Cook (1997).

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Table A2.3 (continued): Definitions, theoretical minima, health outcomes and data sources for 14 selected health risks

Health risk	Exposure variable	Theoretical minimum	Outcomes	Sources for exposure estimates	Sources for hazard estimates
Alcohol	Average number of standard drinks per day	Low level of drinking	Cancers of the mouth and oropharynx, oesophagus, liver, larynx and breast; inflammatory heart disease, hypertensive heart disease, ischemic heart disease, stroke, alcohol dependence and harmful use, gallbladder and bile duct disease, pancreatitis, road traffic accidents, falls, fires/burns/scalds, drowning, machinery accidents, suffocation and foreign bodies, suicide and self-inflicted injuries, homicide and violence, occupational injuries	National Health Survey 2001 (ABS 2001c)	Systematic reviews by English and colleagues (1995) and Ridolfo & Stevenson (AIHW: Ridolfo & Stevenson 2001); the National Coroners Information System (Driscoll et al. 2001, 2004) for alcohol-related drownings and occupational YLL; fire injuries and fatalities pooled results from published studies; scalds and burns from Levy and colleagues (2004)
Illicit drug use	Use of illicit drugs	Abstinence	Heroin or polydrug, benzodiazepine, cannabis, and other drug dependence and harmful use	AusBoD drug use and dependence models	PAF = 1 by definition
	Use of illicit drugs	Abstinence	HIV/AIDS, hepatitis B, hepatitis C, inflammatory heart disease, suicide and self-inflicted injuries, road traffic accidents	Population attributable fraction direct from the literature	Incorporated findings from a multi-centre case-control study on 3,398 fatally injured drivers over Victoria, NSW and Queensland (examining psychoactive drugs); viral hepatitis and sexually transmissible infections from <i>Australia Annual Surveillance Report 2004</i> ; and systematic literature reviews by English and colleagues (1995) and Ridolfo and Stevenson (AIHW: Ridolfo & Stevenson 2001)
	Daily cannabis use	No cannabis use, or use less often than daily	Schizophrenia	National Drug Strategy Household Survey 2004	Meta-analysis of 7 published case-control or cohort studies (examining link between psychosis and cannabis use).

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Table A2.3 (continued): Definitions, theoretical minima, health outcomes and data sources for 14 selected health risks

Health risk	Exposure variable	Theoretical minimum	Outcomes	Sources for exposure estimates	Sources for hazard estimates
Unsafe sex	Unprotected sex	Abstinence/protection	Sexually transmissible diseases, abortion, cervical cancer, HIV/AIDS, hepatitis B & C	AusBoD sexually transmissible diseases, abortion, and cervical cancer models; PAF direct from the literature	PAF=1 (sexually transmissible diseases, abortion, cervical cancer); HIV/AIDS proportion from the Australian HIV and AIDS Public Access Datasets (National Centre in HIV Epidemiology and Clinical Research 2005a, 2005b); hepatitis B & C fraction from the National Centre in HIV Epidemiology and Clinical Research (National Centre in HIV Epidemiology and Clinical Research 2004)
Child sexual abuse	Non-contact only, contact intercourse	No abuse	Anxiety & depression, alcohol dependence & harmful use, heroin or polydrug use & dependence, benzodiazepine dependence & harmful use, cannabis dependence & harmful use, other drug dependence & harmful use, suicide and self-inflicted injuries	CRA priors for Australia	Systematic review and new meta-analysis of published studies (Andrews et al. 2004)
Intimate partner violence	Physical or sexual violence by current or previous partner	No history of sexual or physical violence by an intimate partner	Anxiety & depression, alcohol dependence & harmful use, heroin or polydrug use & dependence, benzodiazepine dependence & harmful use, cannabis dependence & harmful use, other drug dependence & harmful use, suicide and self-inflicted injuries, tobacco smoking, cervical cancer, syphilis, chlamydia, gonorrhoea, other sexually transmissible diseases, anorexia nervosa, bulimia nervosa, other eating disorders, falls, other unintentional injuries, homicide & violence	Women's Safety Survey 1996 (ABS 1996)	Australian Longitudinal Study on Women's Health (Brown et al. 1999); 2003–2004 National Homicide Monitoring Program (NHMP) Annual Report (Mouzos 2005); National Hospital Morbidity Database 2002–03 (AIHW 2003a)

(continued)

Table A2.3 (continued): Definitions, theoretical minima, health outcomes and data sources for 14 selected health risks

Health risk	Exposure variable	Theoretical minimum	Outcomes	Sources for exposure estimates	Sources for hazard estimates
Occupational exposures and hazards	Exposure in the workplace to disease-causing agents such as carbon monoxide, dyes, inorganic and organic dusts, pesticides, metals, metal fumes, petrochemicals, plastics, solvents, isocyanate and nitroglycerine or nitroglycerol	No exposure	All accidents, intentional and unintentional injuries, cancers, heart disease, neurological disorders, chronic respiratory disorders, renal disease, osteoarthritis, slipped disc, occupational overuse syndrome	National Worker's Compensation Statistics Database 2003, National Coroners Information system 2003, and <i>Best estimates of the magnitude of health effects of occupational exposure to hazardous substances</i> (Kerr et al. 1996)	Systematic review of published literature, hospital inpatient data, mortality datasets, National Health Survey results, workers compensation data, notified industrial accident reports and special disease registry datasets
Urban air pollution	Exposure to particulate matter and/or oxygen (i.e. total population of cities of interest)	No exposure	Short-term exposure: cardiovascular, respiratory, and other deaths Long-term exposure: lung cancer, ischemic heart disease, stroke, inflammatory heart disease, hypertensive heart disease, COPD	Assume all residing in relevant geographical areas exposed; particulate and ozone levels from state environmental protection agencies	Time series analysis of short-term effects of urban air pollution in four Australian cities (Simpson et al. 2005a); long-term exposure effects from Pope and colleagues (2002)

Table A2.4: Prevalence of health risks by age and sex

Health risk	Category	Males							Females								
		0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+
Blood pressure (mmHg)	mean	124	131	140	148	154	115	126	138	146	150
	SD	11	16	17	19	19	12	17	19	22	21
Blood cholesterol (mmol/L)	mean	5.5	5.8	5.6	5.6	5.3	5.2	5.8	6.0	6.1	5.9
	SD	1.0	1.1	0.9	0.9	1.0	1.0	1.1	0.9	1.0	1.0
BMI (kg/m ²)	mean	26.8	27.5	27.2	27.1	25.8	25.4	27.2	28.5	27.0	24.9
	SD	4.1	4.0	3.7	3.8	3.5	5.4	5.7	5.8	5.2	4.5
Fruit and vegetable consumption (g/day)	mean	445	452	496	538	538	538	484	506	569	602	577	577
	SD	241	235	245	230	219	219	237	228	240	234	217	217
Bone mineral density (BMD) (g/cm ³)	Mean	0.93	0.87	0.77	0.85	0.78	0.66
	SD	0.15	0.14	0.16	0.13	0.12	0.12
Physical activity (% population in categories)	High	10%	3%	3%	1%	1%	0%	4%	2%	1%	1%	0%	0%
	Recommended	47%	37%	37%	41%	44%	30%	37%	32%	35%	38%	27%	17%
Tobacco (% population in categories)	Insufficient	23%	29%	29%	26%	22%	21%	35%	38%	33%	28%	24%	24%
	Inactive	20%	31%	32%	33%	33%	49%	25%	28%	30%	33%	45%	59%
Tobacco (% population in categories)	Current smoker	30%	31%	23%	16%	9%	7%	25%	25%	18%	12%	9%	2%
	Prenatal exposure	16%	16%
Alcohol (% population in categories)	Maternal smoking	27%	27%
	Abstainer	37%	35%	33%	43%	49%	56%	57%	59%	58%	66%	73%	76%
Alcohol (% population in categories)	Low	48%	51%	52%	43%	45%	40%	35%	32%	32%	25%	20%	22%
	Hazardous	7%	7%	8%	8%	4%	2%	7%	7%	7%	7%	6%	2%
Alcohol (% population in categories)	Harmful	7%	7%	7%	6%	2%	2%	1%	2%	3%	2%	1%	0%

(continued)

Table A2.4 (continued): Prevalence of health risks by age and sex

Health risk	Category	Males										Females									
		0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+				
Illicit drugs (% population in categories)	Daily cannabis use	4%	4%	1%	0%	0%	0%	2%	2%	0%	0%	0%	0%				
	Prenatal exposure – opioids	0%	0%				
Prenatal exposure – cannabis	Prenatal exposure – cannabis	1%	1%				
	Maternal use – heroin				
Maternal use – cocaine	Maternal use – cocaine				
	No abuse	100%	96%	96%	94%	94%	94%	94%	98%	79%	79%	71%	71%	71%	71%	71%	71%				
Child sexual abuse (% population in categories)	Non-contact only	0%	1%	1%	2%	2%	2%	2%	1%	6%	6%	9%	9%	9%	9%	9%	9%				
	CSA	0%	2%	2%	3%	3%	3%	3%	1%	11%	12%	16%	16%	16%	16%	16%	16%				
Intimate partner violence (% population in categories)	Intercourse CSA	0%	1%	1%	1%	1%	1%	1%	0%	3%	3%	5%	5%	5%	5%	5%	5%				
	Sexual or physical violence				
Occupational exposure to ergonomic stressors (% population in categories)	Low	14%	9%	8%	3%	1%	15%	22%	21%	10%	10%	10%	10%				
	Moderate	44%	41%	35%	13%	3%	18%	17%	18%	4%	1%	1%	1%				
Occupational exposure to ergonomic stressors (increases risk of osteoarthritis) (% population in categories)	High	2%	3%	4%	4%	2%	0%	0%	0%	0%	0%	0%	0%				
	Blue collar workers	40%	40%	34%	12%	3%	8%	9%	10%	3%	1%	1%	1%				

(continued)

Table A2.4 (continued): Prevalence of health risks by age and sex

Health risk	Category	Males										Females									
		0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+	0-4	5-14	15-29	30-44	45-59	60-69	70-79	80+				
Occupational exposure to noise (% population in categories)	85-90 dBA	5%	5%	5%	2%	1%	1%	4%	3%	3%	1%	0%	0%				
	>90 dBA	4%	4%	3%	1%	0%	0%	1%	1%	1%	0%	0%	0%				
Occupational exposure to leukaemogens (% population in categories)	Low	3%	4%	4%	1%	0%	0%	3%	4%	4%	1%	0%	0%				
	High	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%				
Occupational exposure to lung carcinogens (% population in categories)	Low	21%	28%	23%	8%	2%	2%	6%	9%	7%	2%	0%	0%				
	High	2%	3%	3%	1%	0%	0%	1%	1%	1%	0%	0%	0%				
Occupational exposure to agents causing COPD (% population in categories)	Low	18%	27%	25%	11%	4%	4%	7%	11%	10%	3%	1%	1%				
	High	12%	14%	11%	3%	1%	1%	1%	2%	2%	0%	0%	0%				
Occupational exposure to agents causing asthma (% population in categories)	Background	23%	10%	17%	67%	90%	90%	31%	28%	33%	84%	97%	97%				
	Administration	6%	12%	13%	4%	1%	1%	16%	21%	18%	4%	1%	1%				
Technical	Technical	16%	30%	28%	10%	3%	3%	16%	27%	24%	5%	1%	1%				
	Sales	10%	4%	3%	2%	0%	0%	19%	6%	5%	1%	0%	0%				
Agriculture	Agriculture	2%	3%	4%	4%	2%	2%	0%	1%	1%	1%	0%	0%				
	Mining	1%	1%	1%	0%	0%	0%	0%	0%	0%	0%	0%	0%				
Transport	Transport	6%	8%	9%	3%	1%	1%	1%	1%	1%	0%	0%	0%				
	Manufacturing	31%	28%	21%	7%	1%	1%	6%	6%	6%	1%	0%	0%				
Services	Manufacturing	6%	4%	4%	2%	1%	1%	11%	10%	11%	3%	1%	1%				
	Services	6%	4%	4%	2%	1%	1%	11%	10%	11%	3%	1%	1%				