# The Future Excess Fraction of Occupational Cancer Among Those Exposed to Carcinogens at Work in Australia in 2012

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## Abstract

**Background:** Studies in other countries have generally found approximately 4% of current cancers to be attributable to past occupational exposures. This study aimed to estimate the future burden of cancer resulting from current occupational exposures in Australia.

**Methods:** The future excess fraction method was used to estimate the future burden of occupational cancer (2012-2094) among the proportion of the Australian working population who were exposed to occupational carcinogens in 2012. Calculations were conducted for 19 cancer types and 53 cancer-exposure pairings.

**Results:** The cohort of 14.6 million Australians of working age in 2012 will develop an estimated 5.2 million cancers during their lifetime, of which 90,000 (1.7%) are attributable to occupational exposure in those exposed in 2012. The majority of these will be lung cancers (n=32,000), malignant mesotheliomas (n=17,000), and leukaemias (n=8,500).

**Conclusions:** A significant proportion of future cancers will result from occupational exposures. This estimate is lower than previous estimates in the literature; however, our estimate is not directly comparable to past estimates of the occupational cancer burden because they describe different quantities – future cancers in currently exposed versus current cancers due to past exposures. The results of this study allow us to determine which current occupational exposures are most important, and where to target exposure prevention.

**Keywords:** Cancer, Occupations, Prevention, Workplace

# The Future Excess Fraction of Occupational Cancer Among Those Exposed to Carcinogens at Work in Australia in 2012

## 1.1 Introduction

Cancer represents a significant public health concern, accounting for approximately 19% of the total disease burden in Australia in 2012 [1]. Fortunately, cancer is largely preventable, with the majority of causal factors being environmental [2]. An important subset of preventable cancers is occupational cancers, in which the exposures are encountered involuntarily and often unknowingly, and in most cases there are obvious means of exposure reduction or prevention. Cancers commonly linked with occupation include cancers of the lung, bladder, and sinonasal cavity.

Approximately 3.6 million Australian workers were estimated to be exposed to occupational carcinogens in their current job in 2012 [3]. This represented nearly 40% of the Australian workforce. Estimating the burden of cancer resulting from these occupational exposures is a useful tool for policy planning and the prioritisation of potential intervention and control measures to prevent or reduce exposure at work.

The most common approach to calculating the burden of occupational cancer is the attributable risk approach, which estimates the proportion of cancer cases in a single year which are attributable to exposures occurring in the past [4]. The most widely cited paper to use this approach estimated that 4% of all cancer deaths in the United States occurring in 1978 were attributable to occupation [5]. Other attributable risk estimates of occupationally-related cancers have included a New Zealand study (1.8-4.2% of cases annually) [6], a more comprehensive study in the United Kingdom (4% of cases in 2005) [7, 8], and mortality studies in the United States (2.4-4.8% of 1997 cancer deaths) [9] and Finland (8% of cancer deaths in 1996) [10]. In Australia, a 2006 estimate suggested that approximately 5,000 cancers per year (11% in males and 2% in females) were attributed to occupational exposures

[11]. However, the prevalence of exposure used in the latter study was based on Finnish exposure data from the 1950s and was criticised as being inappropriate for Australian conditions [12].

An alternative approach to calculating the burden of occupational cancer is the future excess fraction (FEF) method, which estimates the excess number of exposure-related cancers occurring over a number of years in the future among the proportion of the population exposed in a specific year [13]. This approach is useful when current exposure prevalence data are available, because data do not need to be extrapolated over time and no assumptions about cancer latency need be made.

The decision to use the attributable risk or FEF method in a particular study is dependent upon the aim of that study and the type of data available. The attributable risk method is used to answer the question of how many occupational cancers now and in the future result from past exposures, whereas the FEF method is appropriate for the question of how many people who are exposed now will develop cancer in the future. The FEF method may therefore be particularly useful for policy planning as it can show how many cancers will occur in the future in a currently exposed population under various exposure scenarios.

The aim of the current study was to use the FEF method to estimate the future burden of occupational cancer in Australia among those occupationally exposed to carcinogens in 2012.

### 1.2 Methods

The FEF method was used to estimate the future burden of occupational cancer among the Australian working age population who were exposed to carcinogens at work in 2012. We included 38 carcinogens prioritised on the basis of their evidence of carcinogenicity to humans (Group 1 or 2A) according to the International Agency for Research on Cancer (IARC) as at July 2011 and which were used in Australian workplaces [14]. The list of

carcinogens by cancer site published on the IARC website [15] was consulted to identify the cancer sites associated with each of these 38 carcinogens. All cancer-carcinogen combinations listed with sufficient or limited evidence were included in the calculations. In total, calculations were conducted for 19 different cancer types and 53 cancer-carcinogen combinations.

Non-melanoma skin cancers were excluded from these calculations as comprehensive incidence data by year and sex are not available for non-melanoma skin cancer in Australia [16].

### *1.2.1 Data sources*

The cohort for this study was defined as those aged between 18 and 65 in 2012 (i.e. the working age population in 2012). A matrix showing the proportionate survival of an individual at each future age (until the year 2094) was first calculated using a double decrement life-table to adjust for competing causes of mortality, whereby the two endpoints were death and first diagnosis of the cancer of interest (such that an individual no longer contributed person-years after they either died or were diagnosed with the cancer of interest, whichever occurred first). This matrix was then multiplied by the 2012 mid-year population statistics obtained from the Australian Bureau of Statistics (ABS) [17] to obtain the future person-years-at-risk for the cohort until 2094. Matrices were calculated separately by cancer type and sex, such that 38 such matrices were created (19 cancer types for male and female).

Information concerning the prevalence of exposure to carcinogens at work in 2012 was obtained from our previous Australian Work Exposures Study [3], supplemented by the Australian Work Exposures Study-Western Australia. These data provided an estimate of exposure prevalence for each of the 38 carcinogens, generated separately by sex and occupational group. Information about the qualitative level of exposure was included, enabling the exposed population to be divided into a ‘low’ exposure group (those assessed as

having a ‘low’ or ‘medium’ level of exposure to the carcinogen of interest) and a ‘high’ exposure group (those assessed as having a ‘high’ level of exposure to the carcinogen of interest).

Relative risk estimates for high and low exposures for each of the 53 cancer-carcinogen combinations were sought from the literature (see Supplementary Table S1). In the majority of cases, the relative risks selected by Rushton and colleagues [7] were considered appropriate for our study as they were derived from meta-analyses and/or were based on relevant exposures. Where these were unsuitable for Australian circumstances (e.g. melanoma and solar radiation exposure), a literature review was conducted. Preference was given to risk estimates derived from meta-analyses, followed by key studies in the area. Where no relative risk estimate for low exposure was available, and where it seemed the effect would not be strong, no excess risk was assumed (i.e. relative risk of 1.0).

To estimate the number of cancers occurring in the future to 2094, we applied the most recent available (2012) age- and sex-specific cancer incidence rates obtained from the Australian Institute of Health and Welfare [18] to population projections by sex and single year of age from 2013 to 2094 obtained from the ABS [19]. This demographic-only projection assumes that cancer incidence rates will remain constant in the future. As the accuracy of this approach is unknown, we also conducted a sensitivity analysis using cancer incidence rates projected using the R-based software ‘Canproj’ [20], which uses a decision tree to determine and conduct the most appropriate projection model based on past (observed) cancer registrations (see Supplementary Methods).

Projected cancer incidence was estimated to 2094 as this was the year in which the youngest members of the cohort (those aged 18 in 2012) would turn 100. Incidence was projected for each of the 19 cancer types separately by sex.

### *1.2.2 Statistical analysis*

We used the FEF method to estimate the proportion of future occupational cancers which will occur among Australian workers who were exposed to carcinogens at work in 2012, as a result of their exposure. This method has been described in detail previously [13].

As a first step, the lifetime risk ($LR_P$) of each of the 19 cancers in the 2012 Australian working age population (the cohort), irrespective of exposure, was calculated by multiplying the estimated person-years-at-risk and age- and sex-specific incidence rates and then dividing by the number of people in the cohort ($N_P$). All calculations were conducted separately by sex and cancer type.

#### *1.2.2.1 For each cancer-exposure pairing:*

The excess lifetime risk ($LR_x$) of each cancer in the exposed subjects in the cohort was calculated taking into account the $LR_P$, the excess risk associated with exposure to the carcinogen, and the number of people in the cohort. The $LR_x$ was then multiplied by the number exposed to the carcinogen in order to estimate the number of cancers attributable to exposure to that carcinogen (the future excess number, or FEN). These calculations were conducted separately for high and low exposure (where available), and then combined to give an overall FEN for that cancer-exposure pairing.

Future excess fractions (FEFs) for each carcinogen were calculated by dividing the FEN by the number of cancers that would have occurred in the population regardless of exposure ($LR_P$ multiplied by $N_P$).

#### *1.2.2.2 For each cancer type:*

In order to obtain a combined FEF ($FEF_{combined}$) for each cancer, taking into account multiple or overlapping exposures, the complement of the product of complements [4] was calculated, as follows:

$$FEF_{combined} = 1 - \prod_k (1 - FEF_k)$$

That is, for each exposure *k*, the complement of the FEF was taken (i.e. $1 - FEF_k$). The resulting fractions were then multiplied together, and the complement of these taken as the $FEF_{combined}$. The $FEF_{combined}$ was multiplied by the number of cases of the relevant cancer that would have occurred in the population regardless of exposure to obtain the combined FEN ($FEN_{combined}$).

#### *1.2.2.3 Across all cancers:*

To determine the overall future excess fraction of occupational cancer, the $FEN_{combined}$'s for each cancer were summed to obtain the overall number of attributable cancers. This number was then divided by the sum of all cancers that would have occurred in the population to obtain the overall FEF. This procedure was followed to also obtain an FEF for males and females separately.

### 1.3 Results

Our cohort of the Australian working age population in 2012 was estimated to be 14,594,000 in total (7,299,000 males and 7,295,000 females). An estimated 5,171,500 cancers were predicted to occur over their lifetime (2,576,500 cases in males and 2,595,000 in females).

The FEFs and FENs for each of the 19 cancers, adjusted for multiple exposures, are summarised in Table 1. In total, for the cohort of working age males in 2012, 3.1% (n=79,500) of future cancer cases were found to be attributable to occupational exposures. For the cohort of working age females in 2012, 0.4% (n=10,500) of future cancer cases were attributable to occupational exposures. Overall, we estimated that for the 2012 working age population, 1.7% (n=90,000) of future cancer registrations will occur in those who were exposed to occupational carcinogens in that year, as a result of their exposure.

**Table 1.** Estimated occupational future excess fractions (%) and future excess numbers (n) arising among the cohort of working age Australians in 2012, by cancer site

| | Future excess fraction (%) | | | Future excess number (n) [a] | | |
|---|---|---|---|---|---|---|
| Cancer site | Male | Female | Total | Male | Female | Total |
| Bladder | 2.0 | 0.3 | 1.6 | 3,500 | <500 | 3,500 |
| Breast | 1.1 | 0.7 | 0.8 | <100 | 6,000 | 6,000 |
| Colorectal | 0.1 | 0.0 | 0.1 | 1,000 | 0 | 1,000 |
| Kidney | 0.1 | <0.1 | 0.1 | <500 | <100 | <500 |
| Larynx | 10.9 | 1.2 | 9.9 | 4,000 | <100 | 4,000 |
| Leukaemia | 5.1 | 2.0 | 3.9 | 7,000 | 1,500 | 8,500 |
| Lip | 10.5 | 1.9 | 7.9 | 4,500 | 500 | 5,000 |
| Liver | 0.1 | 0.0 | 0.1 | <100 | <100 | <100 |
| Lung | 6.1 | 0.3 | 3.8 | 31,000 | 1,000 | 32,000 |
| Melanoma of the skin | 1.4 | 0.2 | 0.9 | 6,000 | 500 | 7,000 |
| Mesothelioma | 31.7 | 0.2 | 26.7 | 16,500 | <100 | 17,000 |
| Nasal | 23.8 | 1.0 | 15.4 | 1,000 | <100 | 1,000 |
| Nasopharynx | 2.9 | 0.1 | 2.2 | <500 | <100 | <500 |
| non-Hodgkin Lymphoma | 0.0 | 0.1 | 0.1 | <100 | <100 | <500 |
| Ocular melanoma | 8.2 | 1.4 | 5.3 | 1,000 | <500 | 1,000 |
| Ovary | - | 0.0 | 0.0 | - | 0 | 0 |
| Pharynx | 7.9 | 0.8 | 6.3 | 500 | <100 | 500 |
| Stomach | 2.0 | 0.1 | 1.3 | 2,000 | <100 | 2,000 |
| Thyroid | 0.0 | <0.1 | <0.1 | <100 | <100 | <100 |
| **Overall** | **3.1** | **0.4** | **1.7** | **79,500** | **10,500** | **90,000** |

[a] All numbers rounded to the nearest 500 to avoid a false sense of precision.

When restricted to Group 1 (definite) carcinogens, we estimated that 3.0% (n=78,500) of future cancer cases in males and 0.2% (n=6,000) of future cancer cases in females, or 1.6% (n=84,000) of future cancer cases overall, were attributable to occupational exposures among the 2012 working age population. Further, when restricted to those cancer-carcinogen combinations with sufficient evidence only [15], we estimated that 2.3% (n=59,000) of future cancer cases in males and 0.2% (n=5,000) of future cancer cases in females, or 1.2% (n=64,000) of future cancer cases overall, were attributable to occupational exposures for the cohort of working age Australians in 2012.

The highest FEFs for males were for mesothelioma (32%), nasal cancer (24%) and laryngeal cancer (11%) (Table 1). For females, the highest FEFs were for leukaemia (2%), lip cancer (2%), and ocular melanoma (1%).

The cancer sites with the highest FENs were lung, mesothelioma, and leukaemia for males, and breast, leukaemia, and lung for females (Table 1).

Table 2 presents the number of registrations for each cancer with more than 50 cases attributable to occupational exposure. Results by sex are presented in Supplementary Tables S2 and S3. Overall, asbestos exposure contributes the largest number of cancer registrations, followed by solar ultraviolet radiation and benzene.

Lung cancer was the largest contributor to the overall FEN of occupational cancers. Thirteen occupational exposures were considered as causing lung cancer (Table 2), with the exposures contributing the most cases differing by sex (see Supplementary Tables S2 and S3). Silica was the largest contributor to the overall number in males, followed by diesel engine exhaust and asbestos. In females, the largest contributor was polycyclic aromatic hydrocarbons other than vehicle exhausts, followed by environmental tobacco smoke and silica.

**Table 2.** Cancer registrations attributable to occupational exposure in cohort of working age Australians in 2012, by carcinogen and cancer site (including only carcinogens and sites with at least 50 attributable cases)

| | **Cancer site (ICD-10 code/s)** [a] | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Carcinogen** | **Lung (C33-C34)** | **Mesothelioma (C45)** | **Leukaemia (C91-C95)** | **Melanoma of the skin (C43)** | **Breast (C50)** | **Lip (C00)** | **Larynx (C32)** | **Bladder (C67)** | **Stomach (C16)** | **Nasal (C30)** | **Ocular melanoma (C69)** | **Colorectal (C18-C20)** | **Pharynx (C10, C13)** | **Kidney (C64)** | **NHL (C82-C86)** | **Nasopharynx (C11)** | **Liver (C22)** | **Total attributable registrations** |
| Asbestos | 4,500 | 17,000 | - | - | - | - | <500 | - | 1,000 | - | - | 1,000 | <100 | - | - | - | - | **23,500** |
| Solar UVR | - | - | - | 7,000 | - | 5,000 | - | - | - | - | 1,000 | - | - | - | - | - | - | **13,000** |
| Benzene | - | - | 8,500 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | **8,500** |
| ETS | 4,000 | - | - | - | - | - | 4,000 | - | - | - | - | - | 500 | - | - | - | - | **8,500** |
| DEE | 5,500 | - | - | - | - | - | - | 2,000 | - | - | - | - | - | - | - | - | - | **7,500** |

| | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Silica | 7,000 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | **7,000** |
| PAHs | 4,000 | - | - | - | - | - | - | 1,500 | - | - | - | - | - | - | - | - | - | **5,500** |
| Shiftwork | - | - | - | - | 4,500 | - | - | - | - | - | - | - | - | - | - | - | - | **4,500** |
| Nickel | 4,000 | - | - | - | - | - | - | - | - | 500 | - | - | - | - | - | - | - | **4,500** |
| Arsenic | 2,500 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | **2,500** |
| Chromium VI | 1,500 | - | - | - | - | - | - | - | - | 500 | - | - | - | - | - | - | - | **2,000** |
| Ethylene oxide | - | - | - | - | 1,000 | - | - | - | - | - | - | - | - | - | <500 | - | - | **1,500** |
| Lead | - | - | - | - | - | - | - | - | 1,000 | - | - | - | - | - | - | - | - | **1,000** |
| Ionising radiation | <100 | - | <100 | - | 500 | - | - | - | - | - | - | - | - | - | - | - | <100 | **500** |
| Wood dust | - | - | - | - | - | - | - | - | - | 500 | - | - | - | - | - | <500 | - | **500** |
| Cadmium | 500 | - | - | - | - | - | - | - | - | - | - | - | - | <500 | - | - | - | **500** |
| Trichloroethylene | - | - | - | - | - | - | - | - | - | - | - | - | - | <100 | <100 | - | <100 | **<500** |

| | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1,3-Butadiene | - | - | <500 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | **<500** |
| Formaldehyde | - | - | <100 | - | - | - | - | - | - | <100 | - | - | - | - | - | <100 | - | **<100** |
| **Total** | **32,000** | **17,000** | **8,500** | **7,000** | **6,000** | **5,000** | **4,000** | **4,000** | **3,500** | **2,000** | **1,000** | **1,000** | **500** | **<500** | **<500** | **<500** | **<100** | |

[a] All numbers rounded to the nearest 500 to avoid a false sense of precision.

DEE: Diesel Engine Exhaust; ETS: Environmental Tobacco Smoke; NHL: Non-Hodgkin lymphoma; PAHs: Polycyclic Aromatic Hydrocarbons other than vehicle exhausts; UVR: Ultraviolet Radiation

Mesothelioma had the next highest number of registrations due to occupational exposure, with asbestos being the only exposure considered. Significant differences by sex were found in the occupational FEF for mesothelioma, with occupational asbestos exposure contributing 32% to the overall number of cancers in males and <1% in females (Table 1). This is likely to be because few females are occupationally exposed to asbestos [3].

**1.4 Discussion**

Occupational cancer is to a large extent preventable, and regulation and control of workplace exposures is feasible in the majority of instances. This means that there are clear opportunities for policy action to reduce the number of cancers occurring in the community. The results of the current study allow us to determine which cancers contribute most to the overall future burden of occupational disease and, perhaps more importantly, which occupational exposures are the most important contributors. These findings help determine where best to target preventive measures.

Our estimate of 90,000 future cancers (1.7% of the future total) due to occupational exposure is lower than previous estimates. However, our estimate is not directly comparable with previous studies of burden of disease, primarily because we used an innovative methodology, the FEF method, which estimates future rather than current cases of cancer. We chose this method because we thought it more useful in policy setting to predict future cases of cancer based on the current industrial environment, rather than current cases resulting from past exposures. Our national survey of exposure to carcinogens enabled use of the FEF method by providing information on current workplace exposures. Disparities in results between this and other studies may be due to a number of methodological differences. Most importantly, past studies using the attributable risk approach have based their burden estimates on past exposures, whereas we have estimated here using current (2012) exposures, which will often be lower in both magnitude and prevalence than exposures in the past. In

addition, there may be differences in the carcinogens and carcinogen/cancer combinations examined, the levels of exposure included, and the incidence rates of different cancers, as well as differences in the industrial environment between countries (for example, in 2012 Australia had a larger mining sector than many other countries).

The FEF method used here provides an estimate of the proportion of future cancers only in those members of the cohort who were occupationally exposed in 2012. Whilst we have used the best-available estimate of exposure prevalence in this calculation, our current estimate necessarily excludes cancer registrations from those in the cohort who were unexposed in 2012 but had been exposed in the past or would be exposed in the future. The current estimate therefore likely to be an underestimate of the total burden of cancer arising from occupational exposures.

As with all burden of disease studies, a number of assumptions were made for the FEF approach. By using current prevalence of exposure to denote the proportion of people exposed, we assumed a normal distribution around this prevalence with regards to level and length of exposure. We also assumed that the relative risks used in our calculations were relevant to the exposure levels observed in the prevalence estimates. We used relative risks for 'ever exposed' in an attempt to ensure consistency between risk and prevalence estimates; however, these relative risks were based on studies of past exposures and so may not be wholly relevant to currently exposed workers. In addition, we did not explicitly include a latency period in our estimates, as we assumed that some of those exposed in the index year had been exposed for some time in the past and therefore may have developed cancer soon after the index year. Furthermore, a small overestimation is likely because we used the incidence rates for all registrations of the cancer of interest, not just first cancer.

We used current cancer incidence rates, with future cancer numbers projected forward on the basis of demographic change only, an approach used in previous Australian [2] and

international work [21]. As with all estimates of future cancer incidence, the accuracy of this approach is unknown and there is thus a degree of uncertainty around these projections. Incidence rates may change in the future as a result of the changing prevalence of risk factors (e.g. falling smoking rates) as well as screening patterns [2]. To that end, we conducted a sensitivity analysis using projected cancer incidence rates. This analysis produced similar results (albeit a slightly lower FEN and FEF) to that presented here (Supplementary Table S4).

We have presented the results by cancer and agent in order to allow a variety of approaches to reducing the burden of occupational cancer. The first approach would be to attempt to reduce the prevalence of a particular cancer by reducing the level of exposure to one or more associated carcinogens. For example, reducing exposure to carcinogens associated with lung cancer, the largest contributor to the overall burden, may have the biggest effect on the number of future occupational cancers. Possible examples of this include the improvement of exhaust treatment to control diesel engine exhaust exposure [22, 23].

A different approach may be to reduce the numbers exposed to a particular carcinogen such as solar radiation, which would reduce the number of future melanomas of the skin and eye as well as lip cancers. A third approach may be to focus on a particular occupation or industry, and through a combination of education and enforcement reduce exposure and hence risk. Reductions in exposure could be made by substitution of safer alternatives (e.g. lead-free solder) or use of personal protective equipment (e.g. long-sleeved shirts to protect against solar radiation).

The results of this study have the potential to lead to clear policy advice and recommendations around occupational exposure prevention.

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**Conflicts of interest**

None.

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**Supplementary Table S1.** Relative risks used to calculate the future excess fraction of occupational cancer in Australia

| Cancer | Exposure | IARC group | RR-High (95% CI) | RR-Low (95% CI) [a] | Study reference |
|---|---|---|---|---|---|
| Bladder | *ortho*-toluidine | 1 | 3.90 (2.57-5.68) | 2.47 (0.67-6.33) | 1 |
| | DEE | 2A | 1.24 (1.10-1.41) | 1.03 (0.84-1.26) | 2 |
| | PAHs | 2A | 1.44 (1.20-1.74) | — | 2 |
| | Perchloroethylene | 2A | 1.08 (0.82-1.42) | — | 3 |
| Breast | Ionising radiation | 1 | 1.44 (1.26-1.65) | — | 4 |
| | Ethylene oxide | 2A | 1.87 (1.12-3.10) | — | 5 |
| | Shiftwork | 2A | 1.20 (1.08-1.33) | — | 6 |
| Colorectal | Asbestos | 2A | 1.15 (1.01-1.31) | — | 7 |
| Kidney | Trichloroethylene | 1 | 1.32 (1.17-1.50) | — | 8 |
| | Cadmium | 2A | 1.4 (1.1-1.8) (males); 2.5 (1.2-5.3) (females) | — | 9 |
| Larynx | Acid mists | 1 | 4.28 (2.13-8.58) | 1.91 (0.97-3.78) | 10 |
| | Asbestos | 1 | 1.38 (1.17-1.60) | — | 11 |
| | ETS | 2A | 5.45 (1.69-17.52) | — | 12 |
| Leukaemia | Benzene | 1 | 2.62 (1.57-4.39) | 1.64 (1.10-2.39) | 13 |
| | 1,3-butadiene | 1 | 2.3 (0.6-8.3) | 1.3 (0.4-4.3) | 14 |
| | Formaldehyde | 1 | 1.54 (1.18-2.00) | — | 15 |
| | Ionising radiation | 1 | 1.027 [b] | — | 16; 17 |
| Lip | Solar UVR | 2A | 1.95 (1.82-2.09) | — | 18 |

| | | | | | |
|---|---|---|---|---|---|
| Liver | Vinyl chloride | 1 | 2.86 (1.83-4.25) | — | 19 |
| | Ionising radiation | 2A | 1.01 [b] | — | 16; 20 |
| | Trichloroethylene | 2A | 1.30 (1.09-1.55) | — | 21 |
| Lung | Arsenic | 1 | 2.05 (1.43-2.85) | 1.74 (0.75-3.43) | 11 |
| | Asbestos | 1 | 1.48 (1.44-1.52) | 1.18 (1.13-1.23) | 22 |
| | Beryllium | 1 | 2.34 (1.45-3.76) | 1.53 (1.01-2.29) | 23 |
| | Cadmium | 1 | 1.19 (1.09-1.29) | — | 11 |
| | Chromium VI | 1 | 1.18 (1.12-1.25) | — | 24 |
| | DEE | 1 | 1.47 (1.29-1.67) | — | 25 |
| | ETS | 1 | 1.24 (1.18-1.29) | — | 26 |
| | Ionising radiation | 1 | 1.005 (males); 1.021 (females) [b] | — | 11; 16 |
| | Nickel | 1 | 1.4 (0.6-3.4) | — | 27 |
| | PAHs | 1 | 1.31 (1.16-1.48) | — | 28 |
| | Silica | 1 | 1.32 (1.23-1.41) | — | 29 |
| | Acid mists | 2A | 1.36 (0.97-1.84) | — | 30 |
| | Cobalt | 2A | 1.93 (1.03-3.62) | 1.30 (1.00-1.66) | 31 |
| Melanoma | Solar UVR | 1 | 1.1 (0.8-1.5) | — | 32 |
| Mesothelioma | Asbestos | 1 | 41.6 (12.3-140.0) | 6.40 (2.52-16.30) | 33 |
| Nasal/paranasal | Leather dust | 1 | 11.70 (5.34-22.20) | — | 34 |
| | Nickel | 1 | 8.70 (1.05-31.41) | — | 35 |
| | Wood dust | 1 | 5.8 (4.2-8.0) (males); 1.5 (0.7- | — | 36 |

| | | | | | |
|---|---|---|---|---|---|
| | | | 3.2) (females) | | |
| | Chromium VI | 2A | 5.18 (2.37-11.30) | — | 37 |
| | Formaldehyde | 2A | 1.66 (1.27-2.17) | — | 38 |
| Nasopharynx | Formaldehyde | 1 | 1.84 (0.84-3.49) | — | 39 |
| | Wood dust | 1 | 2.40 (1.10-4.50) | — | 40 |
| NHL | Ethylene oxide | 2A | 1.34 (0.96-1.89) | — | 41 |
| | Trichloroethylene | 2A | 1.29 (1.00-1.66) | — | 42 |
| Ocular melanoma | Artificial UVR | 1 | 2.05 (1.20-3.51) | — | 43 |
| | Solar UVR | 2A | 1.7 (1.1-2.7) | — | 44 |
| Ovary | Asbestos | 1 | 1.77 (1.37-2.28) | — | 45 |
| Pharynx | Asbestos | 2A | 1.44 (1.04-2.00) | — | 7 |
| | ETS | 2A | 3.99 (1.06-15.08) | — | 12 |
| Stomach | Asbestos | 2A | 1.66 (1.49-1.86) | 1.21 (1.06-1.38) | 20; 46 |
| | Inorganic lead | 2A | 1.33 (1.18-1.49) | — | 47 |
| Thyroid | Ionising radiation | 1 | 1.09 [b] | — | 16; 48 |

[a] Where no relative risk estimate for low exposure was available, an arbitrary estimate of 1 (no excess risk) was used.

[b] Confidence intervals not available for the relative risk model used (16)

DEE: Diesel Engine Exhaust; ETS: Environmental Tobacco Smoke; NHL: non-Hodgkin Lymphoma; PAHs: Polycyclic Aromatic Hydrocarbons other than vehicle exhausts; UVR: Ultraviolet Radiation

**Supplementary Table 1 References**


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**Supplementary Table S2.** Cancer registrations attributable to occupational exposure in cohort of working age Australians in 2012, by carcinogen and cancer site (including only carcinogens and sites with at least 50 attributable cases in whole cohort), males

| Carcinogen | Cancer site (ICD-10 code/s) [a] | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Lung (C33-C34) | Mesothelioma (C45) | Leukaemia (C91-C95) | Melanoma of the skin (C43) | Lip (C00) | Larynx (C32) | Bladder (C67) | Stomach (C16) | Nasal (C30) | Ocular melanoma (C69) | Colorectal (C18-C20) | Pharynx (C10, C13) | Kidney (C64) | Nasopharynx (C11) | Breast (C50) | NHL (C82-C86) | Liver (C22) | Total attributable registrations |
| Asbestos | 4,500 | 17,000 | — | — | — | <500 | — | 1,000 | — | — | 1,000 | <100 | — | — | — | — | — | **23,500** |
| Solar UVR | — | — | — | 6,000 | 4,500 | — | — | — | — | 1,000 | — | — | — | — | — | — | — | **11,500** |
| ETS | 3,500 | — | — | — | — | 4,000 | — | — | — | — | — | 500 | — | — | — | — | — | **8,000** |
| DEE | 5,500 | — | — | — | — | — | 1,500 | — | — | — | — | — | — | — | — | — | — | **7,000** |
| Silica | 7,000 | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | **7,000** |
| Benzene | — | — | 7,000 | — | — | — | — | — | — | — | — | — | — | — | — | — | — | **7,000** |
| PAHs | 3,500 | — | — | — | — | — | 1,500 | — | — | — | — | — | — | — | — | — | — | **5,000** |

| | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Nickel | 3,500 | — | — | — | — | — | — | — | 500 | — | — | — | — | — | — | — | — | **4,000** |
| Arsenic | 2,500 | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | **2,500** |
| Chromium VI | 1,500 | — | — | — | — | — | — | — | 500 | — | — | — | — | — | — | — | — | **2,000** |
| Lead | — | — | — | — | — | — | — | 1,000 | — | — | — | — | — | — | — | — | — | **1,000** |
| Wood dust | — | — | — | — | — | — | — | — | 500 | — | — | — | — | <500 | — | — | — | **500** |
| Cadmium | <500 | — | — | — | — | — | — | — | — | — | — | — | <500 | — | — | — | — | **500** |
| Trichloroethylene | — | — | — | — | — | — | — | — | — | — | — | — | <100 | — | — | <100 | <100 | **<500** |
| 1,3-Butadiene | — | — | <500 | — | — | — | — | — | — | — | — | — | — | — | — | — | — | **<500** |
| Shiftwork | — | — | — | — | — | — | — | — | — | — | — | — | — | — | <100 | — | — | **<100** |
| Formaldehyde | — | — | <100 | — | — | — | — | — | <1 | — | — | — | — | <100 | — | — | — | **<100** |
| Ethylene oxide | — | — | — | — | — | — | — | — | — | — | — | — | — | — | <100 | <100 | — | **<100** |
| Ionising radiation | <1 | — | <100 | — | — | — | — | — | — | — | — | — | — | — | <100 | — | <1 | **<100** |

[a] All numbers rounded to the nearest 500 to avoid a false sense of precision

DEE: Diesel Engine Exhaust; ETS: Environmental Tobacco Smoke; NHL: non-Hodgkin Lymphoma; PAHs: Polycyclic Aromatic Hydrocarbons other than vehicle exhausts; UVR: Ultraviolet Radiation

**Supplementary Table S3.** Cancer registrations attributable to occupational exposure in cohort of working age Australians in 2012, by carcinogen and cancer site (including only carcinogens and sites with at least 50 attributable cases in whole cohort), females

| Carcinogen | Cancer site (ICD-10 code/s) [a] | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **Breast (C50)** | **Leukaemia (C91-C95)** | **Lung (C33-C34)** | **Melanoma of the skin (C43)** | **Lip (C00)** | **Bladder (C67)** | **Ocular melanoma (C69)** | **NHL (C82-C86)** | **Larynx (C32)** | **Stomach (C16)** | **Nasal (C30)** | **Kidney (C64)** | **Pharynx (C10, C13)** | **Mesothelioma (C45)** | **Nasopharynx (C11)** | **Liver (C22)** | **Colorectal (C18-C20)** | **Total attributable registrations** |
| Shiftwork | 4,500 | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | **4,500** |
| Benzene | — | 1,500 | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | **1,500** |
| Ethylene oxide | 1,000 | — | — | — | — | — | — | <100 | — | — | — | — | — | — | — | — | — | **1,500** |
| Solar UVR | — | — | — | 500 | 500 | — | <500 | — | — | — | — | — | — | — | — | — | — | **1,000** |
| Ionising radiation | 500 | <100 | <100 | — | — | — | — | — | — | — | — | — | — | — | — | <1 | — | **500** |
| PAHs | — | — | 500 | — | — | <100 | — | — | — | — | — | — | — | — | — | — | — | **500** |
| ETS | — | — | <500 | — | — | — | — | — | <100 | — | — | — | <100 | — | — | — | — | **500** |

| | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| DEE | — | — | <500 | — | — | <100 | — | — | — | — | — | — | — | — | — | — | — | **<500** |
| Silica | — | — | <500 | — | — | — | — | — | — | — | — | — | — | — | — | — | — | **<500** |
| Nickel | — | — | <500 | — | — | — | — | — | — | — | <100 | — | — | — | — | — | — | **<500** |
| Formaldehyde | — | <100 | — | — | — | — | — | — | — | — | <100 | — | — | — | <100 | — | — | **<100** |
| Asbestos | — | — | <100 | — | — | — | — | — | 0 | <100 | — | — | 0 | <100 | — | — | 0 | **<100** |
| Chromium VI | — | — | <100 | — | — | — | — | — | — | — | <100 | — | — | — | — | — | — | **<100** |
| Cadmium | — | — | <100 | — | — | — | — | — | — | — | — | <100 | — | — | — | — | — | **<100** |
| Lead | — | — | — | — | — | — | — | — | — | <100 | — | — | — | — | — | — | — | **<100** |
| Wood dust | — | — | — | — | — | — | — | — | — | — | <1 | — | — | — | <1 | — | — | **<1** |
| Arsenic | — | — | 0 | — | — | — | — | — | — | — | — | — | — | — | — | — | — | **0** |
| 1,3-Butadiene | — | 0 | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | **0** |
| Trichloroethylene | — | — | — | — | — | — | — | 0 | — | — | — | 0 | — | — | — | 0 | — | **0** |

[a] All numbers rounded to the nearest 500 to avoid a false sense of precision.

DEE: Diesel Engine Exhaust; ETS: Environmental Tobacco Smoke; NHL: non-Hodgkin Lymphoma; PAHs: Polycyclic Aromatic Hydrocarbons other than vehicle exhausts; UVR: Ultraviolet Radiation

**Supplementary Table S4.** Results of sensitivity analysis showing estimated occupational future excess fractions (%) and future excess numbers (n) arising among the cohort of working age Australians in 2012 using 'Canproj' projected incidence rates, by cancer site

| | Future excess fraction (%) | | | Future excess number (n) [a] | | |
|---|---|---|---|---|---|---|
| Cancer site | Male | Female | Total | Male | Female | Total |
| Bladder | 2.0 | 0.3 | 1.6 | 2,000 | <500 | 2,500 |
| Breast | 1.1 | 0.7 | 0.8 | <500 | 6,000 | 6,000 |
| Colorectal | 0.1 | 0.0 | 0.1 | 1,000 | 0 | 1,000 |
| Kidney | 0.1 | 0.0 | 0.1 | <500 | <100 | <500 |
| Larynx | 10.9 | 1.2 | 9.7 | 3,000 | <100 | 3,000 |
| Leukaemia | 5.9 | 4.6 | 5.6 | 6,500 | 1,500 | 8,000 |
| Lip | 10.5 | 1.9 | 7.3 | 3,000 | 500 | 3,500 |
| Liver | 0.1 | 0.0 | 0.1 | <100 | <100 | <100 |
| Lung | 6.1 | 0.3 | 3.6 | 25,000 | 1,000 | 26,000 |
| Melanoma of the skin | 1.4 | 0.2 | 0.9 | 5,000 | 500 | 5,500 |
| Mesothelioma | 31.7 | 0.2 | 21.9 | 7,500 | <100 | 7,500 |
| Nasal | 23.9 | 1.0 | 15.9 | 1,500 | <100 | 1,500 |
| Nasopharynx | 2.9 | 0.1 | 2.1 | <500 | <100 | <500 |
| Non-Hodgkin Lymphoma | <0.1 | 0.1 | 0.1 | <100 | <100 | <500 |
| Ocular melanoma | 8.2 | 1.4 | 5.4 | 1,000 | <500 | 1,000 |
| Ovary | - | 0.0 | 0.0 | - | 0 | 0 |
| Pharynx | 7.9 | 0.8 | 6.4 | 500 | <100 | 500 |
| Stomach | 2.1 | 0.1 | 1.3 | 2,000 | <100 | 2,000 |
| Thyroid | 0.0 | <0.1 | <0.1 | <100 | <100 | <100 |
| **Overall** | **2.5** | **0.4** | **1.4** | **58,500** | **10,000** | **68,500** |

[a] All numbers rounded to the nearest 500 to avoid a false sense of precision.

**Supplementary Method**

**Click here to download e-component: Supplementary Method.docx**