Occupational cancers
National Cancer Control Policy
1 Occupational cancers

1.1 About this chapter

This chapter of the National Cancer Prevention Policy was developed by Cancer Council Australia's expert Occupational and Environmental Cancer Committee and endorsed by its principal Public Health Committee. The chapter was reviewed by Professor Lin Fritschi, Professor of Epidemiology, School of Public Health, Curtin University, Professor Bernard Stewart, Head of Cancer Control Program, South East Sydney Public Health Unit, and Professor Tim Driscoll, Professor of Epidemiology at the University of Sydney's School of Public Health. It was published in February 2016.

Contact: Deshanie Rawlings

1.1.1 Contents

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2 Overview

Workers in a significant number of industries and workplaces may be exposed to chemical and physical compounds at higher concentrations and for longer periods than people in the general population. It was estimated that in Australia in 2012, 3.6 million current workers, or 40% of the working population, were potentially exposed to carcinogens in the course of their work\textsuperscript{[1]}.

Reducing the burden of occupational cancer requires the implementation of policy based on evidence from two areas of research – epidemiological studies that identify occupational carcinogens and occupational hygiene studies that identify effective interventions to eliminate or reduce the exposure of workers to them.

Due to the time lag typically seen between exposure and cancer incidence, epidemiological studies generally have a historical context. That is, they provide evidence of the consequences of exposure to carcinogens at least five years, and possibly up to decades, previously. This body of evidence is described in the links section of this chapter.

Occupational hygiene research concerns current circumstances of exposure to identified occupational carcinogens. Eliminating or reducing exposure to known carcinogens in occupational settings can be achieved through a range of interventions, described in the effective interventions section of this chapter.

This chapter of the National Cancer Prevention Policy also covers the impact of occupational cancers in Australia, describes the relevant policy context and outlines Cancer Council Australia’s policy priorities to reduce the burden of occupational cancer.

References

3 Impact: the burden of occupational cancer

3.1 Australia

The Australian Work Exposures Study (AWES) estimated that in 2012, 3.6 million current workers, or 40% of the working population, were potentially exposed to carcinogens in the workplace\(^1\). The study found that men were more likely to be exposed than women with approximately 2,727,000 men (58% of the working population) and 877,000 women (21%) potentially exposed to at least one carcinogen at work\(^1\).

A number of occupational groups were estimated to have 100% of workers exposed to at least one occupational carcinogen. These included farmers, drivers, transport workers and various tradespersons. Common exposures were diesel engine exhaust, solar ultraviolet radiation, silica and benzene. See Table 1 and Table 2 for occupational groups for men and women most likely to be exposed to at least one carcinogen in an occupational setting.

National exposure surveillance is not routinely carried out in Australia, meaning that population-based data concerning the prevalence and extent of exposure over time is not available\(^2\).

Table 1. Top occupational groups in Australia potentially exposed to carcinogens - males\(^1\)

<table>
<thead>
<tr>
<th>Occupational group</th>
<th>Most common exposures</th>
<th>% exposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heavy vehicle drivers</td>
<td>DEE, solar UVR, ETS</td>
<td>100</td>
</tr>
<tr>
<td>Farmers</td>
<td>Solar UVR, DEE, PAHs</td>
<td>100</td>
</tr>
<tr>
<td>Plumbers</td>
<td>Solar UVR, ETS, lead</td>
<td>100</td>
</tr>
<tr>
<td>Painters</td>
<td>Solar UVR, lead, wood dust</td>
<td>100</td>
</tr>
<tr>
<td>Miners</td>
<td>Silica, DEE, solar UVR</td>
<td>100</td>
</tr>
<tr>
<td>Carpenters</td>
<td>Wood dust, solar UVR, formaldehyde</td>
<td>98.8</td>
</tr>
<tr>
<td>Animal and horticultural</td>
<td>Solar UVR, benzene, DEE</td>
<td>98.5</td>
</tr>
<tr>
<td>Vehicle trades</td>
<td>DEE, asbestos, lead</td>
<td>97.4</td>
</tr>
<tr>
<td>Handypersons</td>
<td>Solar UVR, wood dust, silica</td>
<td>97.0</td>
</tr>
<tr>
<td>Passenger transport</td>
<td>DEE, shiftwork, solar UVR</td>
<td>96.8</td>
</tr>
<tr>
<td>Automobile drivers</td>
<td>DEE, solar UVR, ETS</td>
<td>95.4</td>
</tr>
<tr>
<td>Engineers</td>
<td>Solar UVR, DEE, silica</td>
<td>92.9</td>
</tr>
</tbody>
</table>
Occupational group | Most common exposures | % exposed
---|---|---
Outdoor workers | Solar UVR, DEE, benzene | 92.3
Construction workers | Silica, solar UVR, ETS | 90.7
Electrical workers | Solar UVR, ETS, lead | 89.4
Emergency workers | DEE, PAHs, lead | 89.1
Metal workers | Artificial UVR, chromium VI, nickel | 87.1
Warehousing | Solar UVR, DEE, ETS | 86.2
Scientists | Solar UVR, DEE, benzene | 78.4
Other health professionals | Ionising radiation, PAHs, solar UVR | 73.6

DEE - diesel engine exhaust, UVR - ultraviolet radiation, ETS - environmental tobacco smoke, PAHs - polycyclic aromatic hydrocarbons

Table 2. Top occupational groups in Australia potentially exposed to carcinogens - females

Occupational group | Most common exposures | % exposed
---|---|---
Farmers | Solar UVR, DEE, benzene | 100
Automobile drivers | DEE, Solar UVR, benzene | 100
Passenger transport | Shiftwork, DEE, ETS | 100
Heavy vehicle drivers | DEE, solar UVR, ETS | 100
Handypersons | Solar UVR, ETS, DEE | 100
Electrical workers | Solar UVR, ETS, DEE | 100
Vehicle trades | DEE, asbestos, ETS | 100
Metal workers | Ionising radiation, DEE, formaldehyde | 100
Construction workers | Silica, ETS | 100
Miners | Silica, lead, nickel | 100
Animal and horticultural | Solar UVR, benzene, DEE | 91.7
Engineers | Solar UVR, DEE, benzene | 85.7
Emergency workers | Solar UVR, shiftwork, PAHs | 83.3
Scientists | Ionising radiation, DEE, solar UVR | 69.1
Carpenters | Wood dust, DEE | 66.7
Hospitality | ETS, solar UVR, PAHs | 59.0
Nurses | Shiftwork, ionising radiation, PAHs | 52.1
Machine operators | Solar UVR, DEE, ETS | 46.2
Food service | ETS, PAHs, shiftwork | 44.4
Food factory | Shiftwork | 33.3

DEE - diesel engine exhaust, UVR - ultraviolet radiation, ETS - environmental tobacco smoke, PAHs - polycyclic aromatic hydrocarbons

Estimating cancer burden associated with occupation is challenging for a number of reasons, including: limited data on exposure; uncertainty about the strength of evidence on association and causation; and uncertainty about latency (time lag between exposure and diagnosis). These challenges may explain much of the variation in findings from different studies.
Due to the long latency between exposure and the occurrence of most cancers, occupational cancers identified today will reflect the effect of exposures several decades previously. Similarly, the burden of cancers resulting from current exposures will not be seen for several decades. Long latency and the fact that cancer incidence is more common means that occupational cancers mainly occur in older persons.

3.2 Global impact

The World Health Organization assessed worldwide mortality and morbidity resulting from exposures to selected occupational hazards in 2000. The selected occupational risk factors were responsible for 9% of lung cancer cases, 2% of leukaemia cases, and close to 100% of mesothelioma cases worldwide\(^3\). In 2004, WHO estimated that 177,000 deaths from lung cancer, leukaemia and mesothelioma worldwide were attributed to occupational carcinogens, with the majority (137,000) occurring in males\(^4\).

Various estimates of global burden have been made (WHO, 2009; Global Burden of Disease, 2013). The most recent estimates come from the Global Burden of Disease Study in 2013 which estimated that there were 314,000 occupational cancer deaths worldwide\(^5\). Lung cancer, mesothelioma, laryngeal cancer and leukaemia were the most common cancer types.

There are also a range of country-specific estimates such as a UK study which found that workplace exposures accounted for 4% of cancer cases (5.7% in men and 2.2% in women) and 5.3% of cancer deaths (8.2% in men, 2.3% in women) in Great Britain in 2005\(^6\).

Estimating the cancer burden is challenging for a number of reasons including the lack of good quality data on exposure, uncertainty about the nature or strength of the association between the exposure and cancer incidence and uncertainty about latency. This explains much of the variation between the estimates arising from different studies.

3.3 References


4 Occupational and environmental carcinogen position statements

Pesticides and cancer
Position statement - Pesticide and cancer

5.1 Key messages

- The term ‘pesticides’ describes hundreds of synthetic and naturally occurring chemicals (i.e. those produced by plants) designed or naturally produced to deter insects and other agricultural pests, including weeds. The broad term of pesticides includes both herbicides and insecticides. The wide-ranging variety of synthetic and naturally occurring chemical pesticides makes it difficult to accurately assess their potential to cause cancer in humans.

- Australian evidence examining the link between pesticide exposure and cancer is very limited. This is because a) there have been very few studies conducted, b) studies examining cancer risk from exposure to specific pesticides have been limited by the small numbers of people in the studies, c) the wide range of chemicals used in pesticides, and d) exposure to other possible carcinogens in workers who may also use pesticides. Theses factors make it impossible to currently establish direct links between pesticides used in Australia and cancer.

- Three chemicals used as pesticides – arsenic, ethylene oxide and lindane – are among agents rated as Group 1 carcinogens, or conclusive causes of cancer, by the International Agency for Research on Cancer (IARC), as is the chemical 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), which may occur as a contaminant in certain pesticides. Arsenic and arsenic compounds are no longer used in pesticides in Australia; ethylene oxide is used in three pesticides licensed for use in Australia until July 2012.

- Six specific pesticides - captafol, ethylene dibromide, glyphosate, malathion, diazinon and dichlorophenyltrichloroethane (DDT) - are classed as a probable cause of cancer (Group 2A).
5.1 Key messages

- Seven pesticides: tetrachlorvinphos, parathion, metolachlor, pendimethalin, permethrin, trifluralin and 2,4-dichlorophenoxyacetic acid (2,4-D) have been classified as possible causes of cancer (Group 2B). Inadequate evidence was available to determine whether these chemicals caused cancer and further research is recommended.
- The use of household pesticides, such as insecticides, can expose people to pesticide residues in the home. However, there is no clear evidence linking household pesticide use in Australia with cancer.
- Meta-analyses (i.e. analyses of multiple international studies) have shown an association between women exposed to insecticides in pregnancy and an increased risk of leukaemia in their children. These studies were limited; causation could not be confirmed. More and better quality research is required into a possible link between antenatal exposure to insecticides and childhood leukaemia.
- There is no evidence that pesticide residues on food consumed in Australia cause cancer. (In fact, consumption of foods most commonly associated with pesticide use – fresh vegetables and fruit – can help to prevent cancer.) The level of pesticide residue on foods sold in Australia is regularly monitored by government agencies to help ensure levels stay well within agreed safety limits.
- Where specific pesticides are demonstrated to increase cancer risk in humans, the people most likely to be adversely effected are those who have the highest level of exposure. This is most likely to be people who work with those pesticides as a routine part of their job.
- Overall, the evidence on whether pesticides cause cancer is limited due to the reasons outlined above, so it is impossible to conclude whether or not there is a link between pesticide exposure and cancer.

5.2 Overview

Pesticides are widely used in agriculture, other workplaces and households. Some chemicals used in pesticides have been linked to cancer through laboratory and epidemiological research. However, there is no conclusive evidence linking pesticide use in general with cancer.

The wide range of chemicals used in pesticides, and possible co-factors leading to cancer in people exposed to pesticides, make it impossible to establish direct links between pesticides used in Australia and cancer. However, there is also insufficient evidence to conclusively show there is no link between pesticide exposure, either through direct chemical or residual contact, and cancer.

This position statement provides an overview of the evidence on the carcinogenic potential of occupational, dietary and residual/environmental exposure to pesticides.

5.3 Specific pesticide components

The term “pesticides” includes hundreds of individual chemicals; exposure therefore describes contact with a wide range of products.
Determining which particular pesticide chemicals account for a specific health effect is difficult. Finding evidence of carcinogenicity in humans is difficult as studies need very large numbers of people followed for decades, with detailed information about specific pesticide exposure including how much pesticide and length of time of exposure. Animal experiments can provide some indication of potential carcinogenicity of pesticides, but their results are not always applicable to humans. Mechanistic evidence is also important to consider, to ensure that the mechanism by which an agent works to cause cancer in cells, so as to explain how the agent (e.g. the chemical) is likely to operate in humans. For example, the IARC originally classified the herbicide atrazine as a possible human carcinogen (Group 2B) on the basis of rat experiments. However, the mechanisms turned out to be irrelevant to humans, the chemical was downgraded to Group 3 (unclassifiable)\(^1\) and later epidemiological studies showed no link between atrazine and cancer\(^2\)\(^3\). A 2015 IARC evaluation upgraded the herbicide glyphosate from a possible (Group 2B) to probable human carcinogen (Group 2A) based on strong mechanistic evidence\(^4\).

Arsenic compounds are a known cause of lung cancer and have been classed as Group 1 carcinogens\(^5\), meaning that they have conclusively been shown to cause cancer in humans, by the IARC (see Appendix 1).

Ethylene oxide is classed within Group 1\(^5\) and is licensed as an ingredient in five fumigant products, at least until July 2013\(^6\). In 2015 IARC classified the insecticide lindane as Group 1 due to epidemiological studies which reported significant increases in non-Hodgkin lymphoma risk with increasing occupational exposure to lindane\(^7\). Aside from these clear exceptions, no specific pesticide has been conclusively linked to a specific cancer, and suggested links do not group by class or type of pesticide.

The IARC has also classified the “spraying and application of non-arsenical insecticides” as a probable cause of cancer\(^8\). However, only six specific pesticides – captafol, ethylene dibromide, glyphosate, malathion, diazinon and dichlorodiphenyltrichloroethane (DDT) – are classed within this category. While there was limited evidence of carcinogenicity in humans found, there was strong mechanistic evidence for the carcinogenicity of glyphosate, malathion and diazinon with all three agents inducing DNA and/or chromosomal damage in human and animal cells in vitro. Several pesticides have been classed as possible causes of cancer (Group 2B).

Phenoxy herbicides, chlorothalonil, dichlorvos, and sodium ortho-phenylphenate are licensed for agricultural use, although some are under review. Para-dichlorobenzene is not used as an agricultural pesticide, but is used in mothballs and urinal cakes. In March 2015, IARC classified the insecticides tetrachlorvinphos and parathion as possibly carcinogenic to humans (Group 2B) based on convincing evidence that these pesticides cause cancer in laboratory animals \(^4\)(see Appendix 1). In June 2015, the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) was classified as Group 2B based on limited evidence in experimental animals and strong mechanistic evidence that 2,4-D induces oxidative stress, a mechanism that can operate in humans\(^7\).
The US Agricultural Health Study is the largest prospective study to assess the link between pesticide exposure and cancer. It recruited more than 57,000 pesticide applicators (mostly male) and 32,000 spouses of applicators (mostly female). In 2010, a review of the study’s 28 publications found that 19 out of 32 pesticides were associated with at least one type of cancer, including lung, pancreatic, bowel (colon and rectal), prostate, brain and bladder cancer, melanoma, leukaemia, non-Hodgkin lymphoma and multiple myeloma\(^9\). However, for most of these pesticides the “highest exposure” categories included fewer than 12 cases, meaning little could be concluded regarding the causal nature of these associations on the available evidence\(^9\). Further research is required.

Of the 19 pesticides associated with cancer in the Agricultural Healthy Study, six were singled out for future investigation based on corresponding animal toxicity data. Of these six, alachlor is not permitted for use in Australia; carbaryl is under review, and metolachlor, pendimethalin, permethrin and trifluralin are in use. The IARC has classified permethrin and trifluralin as Group 3 (inadequate evidence)\(^8\), but has not evaluated metolachlor and pendimethalin. The US Environmental Protection Agency describes permethrin as a “likely” carcinogen and the other five as “possible” carcinogens\(^10\).

Outside the Agricultural Health Study, a small number of studies have assessed cancer risk according to exposure of individual pesticides. Some results are reviewed in Weichenthal et al\(^9\), however a full systematic analysis is beyond the scope of this position statement. Many of these studies suffer from similar weaknesses\(^11\) – exposure to pesticides is generally measured through self-reports, which makes studies vulnerable to recall bias (that is the accuracy of people's memory of what pesticides and how much they were exposed to). Validation analyses show that self-reporting provides a reasonable measure of the highest and lowest exposure levels, but is less effective at quantifying moderate exposures. Furthermore, pesticide exposure varies significantly between occupations. It can also be intense during certain tasks but cumulatively low, since those tasks are performed only on a few days a year and often vary over the years as pesticide types and application methods change. Farmers and family members may have additional exposure from inadvertent contact, but “bystander exposure” (that is exposure to people who were in the vicinity at the time pesticides are applied but no involved in applying the pesticide) is very difficult to measure.

### 5.4 Occupational pesticide exposure

A number of international studies have found higher incidence and mortality rates from specific cancers among people occupationally exposed to pesticides, including farmers and pesticide applicators\(^9\), pesticide manufacturing workers\(^12\)\(^13\), golf course superintendents\(^14\) and market gardeners or orchardists\(^15\). There is, however, no increase found in the incidence or mortality of these cancers among pest control workers (e.g. exterminators)\(^16\).
Meta-analyses (compilations of multiple studies) have reported higher than average levels of various cancers among farmers and pesticide applicators (see Blair and Freeman for a review\(^\text{[11]}\)). These include non-Hodgkin lymphoma\(^\text{[17][18][19][20]}, \text{leukaemia}\(^\text{[12][13][21]}, \text{multiple myeloma}\(^\text{[22]}, \text{brain cancer}\(^\text{[23]}, \text{prostate cancer}\(^\text{[24][25][26]}, \text{lip cancer}\(^\text{[11]}\) and skin cancer\(^\text{[11]}\). However, most of the associations were relatively weak, with occupational exposures attributable for a 10-40% increase in risk, depending on cancer type. Exceptions include: two meta-analyses which found a two-fold higher risk of lip cancer among farmers\(^\text{[11]}\); and a meta-analysis that found a two-fold higher risk of leukaemia among pesticide applicators (employees applying pesticides) and a six-fold higher risk among pesticide manufacturing workers\(^\text{[18]}\).

It is not clear if pesticides are attributable for these elevated incidence rates, because workers in these sectors are also exposed to a range of other potential carcinogens. For example, agricultural workers are regularly exposed to diesel exhaust, solvents, metals, grain dusts, zoonotic (transmissible from animals to humans) viruses and ultraviolet radiation, all of which could influence or "confound" the relationship between pesticides and cancer\(^\text{[27]}\).

In addition, a study in Western Australia found that 78% of farm jobs have “no likelihood of pesticide exposure”\(^\text{[28]}\). The study noted “classification of all farm jobs as pesticide-exposed is likely to substantially over-estimate the number of individuals exposed”\(^\text{[28]}\).

The long time lag between environmental exposures and the development of some cancers may make it difficult to draw conclusions about current workplace exposures. This time lag also means it is difficult to study new pesticides, as associated cancers may occur many years after their introduction. For example, agricultural workers could develop cancers through exposure to arsenic and arsenic compounds used in pesticides many years ago but no longer permitted in Australia.

### 5.5 Exposure to pesticides in the home

Exposure to pesticides in the home includes professional applications (e.g. professional fumigation and other pest control services), the use of household sprays and other retail pesticides, and chemicals brought into the home from workplaces\(^\text{[29]}\).

Pesticides can persist indoors from carpets, where they are protected from environmental degradation; such residues can be measured in samples of carpet dust\(^\text{[30][31]}\). Children may experience greater exposure and adverse reactions to such pesticide residues, because their concentration is higher closer to the floor\(^\text{[29]}\) and a child’s metabolism builds up different levels of toxic metabolites to that of adults\(^\text{[32]}\).

A number of studies have assessed the risk of various cancers among both adults and children following residential pesticide exposure. There have been positive results from isolated, small studies for prostate cancer\(^\text{[33]}\), neuroblastoma\(^\text{[34]}\) and childhood brain tumours\(^\text{[35]}\); inconsistent evidence for breast cancer\(^\text{[36][37][38][39]}\) and non-Hodgkin lymphoma\(^\text{[40][41][42]}\); and no strong evidence for Wilms’ tumour\(^\text{[43][44]}\) or germ cell tumours\(^\text{[45]}\).
5.6 Exposure to pesticides through diet

Pesticides are sprayed on crops and thus may end up in the human body through diet. Food Standards Australia New Zealand and the Australian Pesticides and Veterinary Medicines Authority monitor levels of pesticide residue in Australian foods to ensure they remain within approved food safety levels. These agencies determine an Acceptable Daily Intake (ADI) for each chemical, which reflects the amount “that can be ingested daily over a lifetime without appreciable risk to health”.

The 20th Australian Total Diet Survey, conducted in 2003 (the most recent survey), screened 65 types of food for pesticide residues, including chlorinated organic pesticides, organophosphorus pesticides, synthetic pyrethroids, carbamates and fungicides. The survey report concluded that “the levels of pesticide residues... in our food are very low, and in all cases they are within acceptable safety limits”.[46]

For most pesticides of concern, Australians are exposed to less than 0.2% of the ADI through their diet.[46] The report recommended that pesticide residue monitoring should be undertaken less frequently, although it should also be expanded to focus on chemicals beyond those registered for use in Australia (given the importation of certain foodstuffs).[46]

Analysis shows the effect of dietary synthetic pesticides on cancer would be minimal, given the tiny proportion of synthetic pesticides ingested compared with those naturally produced by plants to deter insects and other animals.[47] It is estimated that more than 99% of the pesticides we eat are naturally occurring, yet around 60% of both synthetic and natural pesticides have been shown to cause cancer in rodent tests.[47]

There is also no evidence that eating foods most likely to contain pesticide residues.[46] i.e. fruit, vegetables and cereals – increases cancer risk. On the contrary, evidence shows that eating such foods can reduce cancer risk.[47][48]

5.7 Environmental pesticide exposure

People can be exposed to pesticides that seep into the water supply or food chain, persisting for a long time in the environment. The persistent, residual nature of such compounds enables them to be measured in the human body in blood and breast milk.

Some of these chemicals, such as organochlorine pesticides, have been shown to be endocrine disruptors with the ability to mimic or block natural hormones like oestrogen and testosterone. These properties have been hypothesised to increase the risk of hormonal cancers such as breast or prostate cancers, although there is not enough evidence to support a causal link.[49][50]

As a case study, the organochlorine DDT has been extensively studied as a risk factor for breast cancer. It is now banned in Australia and other parts of the world, but in the 1940s and 1950, it was heavily used as an insecticide. The IARC classifies DDT in Group 2B (possible carcinogen) after three rounds of evaluation, in 1974, 1987 and 1991.[8][51][52]. The most recent IARC evaluation in 2015 upgraded DDT to a Group 2A (probable
carcinogen\[7\]. Most epidemiological studies\[53\], including the Long Island nested case-control study\[54\], do not support a conclusive link between DDT and cancer although there is some evidence that exposure in early life or adolescence could increase the longer-term risk of breast cancer\[55\]. Epidemiological studies have similarly not supported a link between environmental exposure to organochlorine pesticides in general and breast cancer \[56\]. However, studies on non-Hodgkin lymphoma, liver cancer and testicular cancer provided limited evidence for the carcinogenicity of DDT\[7\].

### 5.8 Childhood cancer and parental exposure to pesticides

There is some evidence that parental exposure to pesticides could increase the risk of cancer in the next generation. A 2011 meta-analysis considered 40 studies and concluded that maternal occupational pesticide exposure before birth is associated with a 48% increased risk of childhood leukaemia and a 53% increased risk of lymphoma, while paternal exposure before or after birth was associated with a 49% higher risk of brain cancer \[57\].

Two other meta-analyses found that maternal occupational pesticide exposure before birth was associated with a 62% and 109% higher risk of childhood leukaemia risk respectively \[58\][59\]. Neither study found an association between paternal exposure and childhood leukaemia \[58\][59\].

One meta-analysis of 15 case-control studies concluded that residential exposure to pesticides during pregnancy increased the risk of childhood leukaemia by 54% \[60\]. The association was especially strong for insecticides – a doubling of risk – and it remained significant after stratifying for high-quality studies with the most accurate exposure measurements and adjustment to confounding factors. Another meta-analysis of 13 studies concluded that residential pesticide exposure was linked to a 74% higher risk of childhood leukaemia, with the strongest risk for exposure during pregnancy (2.2-fold) and insecticide exposure (73%) \[59\].

Findings from the Childhood Leukemia International Consortium published in 2014 suggest it may be important to investigate occupational pesticide exposure by sub-type of leukaemia. This study pooled data from 13 case-control studies and findings for acute lymphoblastic leukaemia (ALL) were different from those for acute myeloid leukaemia (AML). For maternal occupational pesticide exposure during pregnancy a significantly increased risk was found for AML but not ALL. For paternal occupational pesticide exposure at the time of conception a significantly increased risk was found for ALL but not AML \[61\].

Recent studies have suggested that parental exposure to pesticides may also be associated with brain cancer. In 2013, a meta-analysis of 20 studies from 1974 to 2010 supported an association between parental occupational exposure to pesticides and brain tumours in children and young adults \[62\].

A 2011 meta-analysis has suggested that paternal exposure to pesticides either before or after birth increases the risk of brain cancer in children by 50-65\% \[57\]. The study found no evidence to suggest that maternal exposure to pesticides before or after birth was associated with an increased risk of brain cancer \[57\].

An Australian case-control study also published in 2013 suggested that preconception pesticide exposure, and possibly exposure during pregnancy, is associated with an increased risk of childhood brain tumour \[63\].
All studies analysed to 2010 were susceptible to various forms of reporting bias\textsuperscript{[64]}. For example, the studies were case-control – i.e. based on data on exposure levels provided by individuals with a specific cancer compared to individuals without that cancer. Self-reported data on previous exposures is often unreliable, particularly when derived from people with a cancer that they think may be linked to a possible cause (this is known as “recall bias”).

Most studies used small sample sizes and were unable to single out any specific pesticide of concern. Van Maele-Fabry et al. concluded that “data were too scarce” to assert a causal link between residential pesticide exposure and leukaemia\textsuperscript{[59]}. They called for more studies on interactions between genetic predisposition in individuals and environmental exposures, while suggesting “it may be opportune to consider preventive actions including educational measures to increase the awareness of the public and particularly of pregnant women about the potential adverse influence of pesticides on children’s health”\textsuperscript{[59]}.

Moreover, variations in the size, quality and consistency (e.g. of data sets) of studies made it difficult to draw conclusions. However, while the limitations and flaws of these studies weakened the overall evidence, there was still an association between residential pesticides and leukaemia\textsuperscript{[59],[60]}.

It should be noted that association indicates a possible link and is not conclusive evidence of causation.
5.9 Appendix 1. Overview of pesticide carcinogenicity classifications

The IARC has classified various pesticides according to their carcinogenic potential (see Siemiatycki et al. for a full list). The full list of agents classified by the IARC Monographs is available on the IARC website.

Table 1. Pesticide carcinogenicity classifications and licensing in Australia

<table>
<thead>
<tr>
<th>IARC group</th>
<th>Pesticide</th>
<th>Human evidence</th>
<th>Animal evidence</th>
<th>Use in Australia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>Arsenic and arsenic compounds</td>
<td>Sufficient</td>
<td>-</td>
<td>No longer used as crop insecticide. Use of arsenic trioxide as a wood preservative is allowed if treated wood is covered after application; use of copper chrome arsenic for the same purpose has been heavily restricted since 2005</td>
</tr>
<tr>
<td></td>
<td>Ethylene oxide</td>
<td>Limited (leukaemia)</td>
<td>Sufficient</td>
<td>Licensed as an ingredient in five fumigant products, until July 2013</td>
</tr>
<tr>
<td></td>
<td>TCDD</td>
<td>Limited (cancer overall)</td>
<td>Sufficient</td>
<td>Not specifically used as a pesticide, but sometimes found as a contaminant in chlorophenoxy herbicides</td>
</tr>
<tr>
<td></td>
<td>Lindane</td>
<td>Sufficient (non-Hodgkin's lymphoma)</td>
<td>Sufficient</td>
<td>Used in insect control in agriculture and for treatment of human ectoparasites (scabies and lice)</td>
</tr>
<tr>
<td></td>
<td>Spraying and application of nonarsenical insecticides</td>
<td>Limited (strongest evidence for lung cancer; weaker for brain, leukaemia, non-Hodgkin lymphoma, multiple</td>
<td>Not available</td>
<td>-</td>
</tr>
</tbody>
</table>
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6 Policy context

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In Australia there are a number of government agencies and structures in place for development and implementation of policy addressing occupational cancer risk, including:

- Safe Work Australia;
- National Industrial Chemical Notification and Assessment Scheme, within the Federal Department of Health;
- Cancer Australia, which has links with the International Agency for Research on Cancer;
- Australian Pesticides and Veterinary Medicines Authority; and
- jurisdictional work safety agencies.

Greater coordination across sectors is pivotal to reducing occupational cancer risk through evidence-based policy and regulation.

6.1 Australian Work Health and Safety Strategy 2012-2022

Overarching national occupational health and safety policy in Australia is contained in the Australian Work Health and Safety Strategy 2012-2022\(^1\). The strategy provides a 10-year national framework to drive improvements in work health and safety in Australia. It is aimed at all aspects of work health and safety including regulators, industry, unions, other organisations and governments.

The strategy sets out four objectives\(^1\):

- reduced incidence of work-related death, injury & illness
- reduced exposure to hazards & risks;
- improved hazard controls; and
- improved work health and safety infrastructure.

Cancer, and specifically skin cancer, is identified in the strategy as one of six national priority work-related disorders for the first five years of the strategy.
The strategy was developed by Safe Work Australia which operates as an independent government agency. Safe Work Australia is made up of representatives from each state and territory government and the Australian Government, as well as members representing the interests of workers and employers.

6.2 Legislation/regulation

Historically, the regulatory framework surrounding carcinogens has been complex and fragmented, partly because carcinogenic substances are variously regulated as workplace hazards, consumer products and environmental pollutants.

In 2009, Safe Work Australia began the development and evaluation of the model work health and safety laws for national implementation\(^2\). The model work health and safety laws are the basis for harmonised laws across Australia. The Commonwealth, states and territories are responsible for regulating and enforcing the laws in their jurisdictions. The Model Work Health and Safety Regulations and first stage Model Codes of Practice developed by Safe Work Australia have been implemented by the Federal Government and all State and Territory Governments with the exception of Victoria and Western Australia.

The federal Work Health and Safety Act 2011 commenced on January 1, 2012, with the aim to provide a balanced and nationally consistent framework to secure the health and safety of workers and workplaces. The Act aims to do this through the elimination or minimisation of risks arising from work, promoting the provision of workplace health and safety education and training and strengthening the national harmonisation of laws relating to work health and safety to facilitate a consistent national approach.

National codes of practice are in place to underpin the prohibition, control and management of scheduled carcinogenic substances in the workplace\(^3\)[4]. The extent to which Australian workers are protected against occupational carcinogens is determined by the rigour of enabling legislation and regulation, and its enforcement.

Specific carcinogens are further regulated by exposure standards (e.g. when a hazardous chemical contains a specific amount of a carcinogen) and codes of practice. For example, the Radiation Protection Standard for Occupational Exposure to Ultraviolet Radiation aims to limit occupational exposure to ultraviolet radiation from artificial sources and provides guidance on minimising occupational exposure to uncontrollable sources of ultraviolet radiation, such as the sun\(^5\).

There is no clear and comprehensive data on the implementation and effectiveness of regulations designed to reduce occupational exposures to carcinogens across jurisdictions, and no national agency with a remit to collect and publish this important information.

6.3 National Strategic Plan for Asbestos Awareness and Management in Australia

In June 2012 the Australian Government established the Asbestos Management Review to make recommendations for a national strategic plan to improve asbestos awareness and management.
The National Strategic Plan for Asbestos Awareness and Management 2013 – 2018 was released in July 2013. The plan sets out a national approach to asbestos eradication, handling and awareness in Australia with the aim of preventing harmful exposure to asbestos.

The Asbestos Management Review had noted that the management of asbestos is regulated by all levels of government in Australia. Local, state, territory and Commonwealth agencies operate under an array of legislative instruments to cover workplace, environmental and public health contexts. The division of responsibility had resulted in a variety of approaches to dealing with the management of harmful asbestos. The national strategic plan recommended a framework which jurisdictions work both cooperatively and independently in order to prevent exposure to asbestos. The Asbestos Safety and Eradication Agency was established by the Australian Government in 2012 to promote the national strategic plan.

6.3.1 James Hardie

James Hardie was Australia’s largest manufacturer of asbestos-containing materials until the mid-1980s. In 2001, James Hardie established the Medical Research and Compensation Foundation (MRCF) to address all future asbestos claims. In 2004, the NSW Government commissioned a judicial inquiry into the MRCF and found funds for asbestos victims to be inadequate\(^6\). Following the inquiry, James Hardie agreed to pay compensation through a voluntary compensation fund.

6.3.2 ACT - Mr Fluffy

During the 1960s and 70s, a Canberra based company known as ‘Mr Fluffy’ installed loose-fill asbestos insulation in approximately 1000 houses in the ACT, Queanbeyan and surrounding areas. In December 2014, the ACT Government announced a buyback scheme for all houses affected by Mr Fluffy loose-fill asbestos insulation in the ACT. The ACT Government has offered to purchase all affected houses for site remediation by eradicating exposure risks to loose-fill asbestos and the demolition of these houses has commenced. The NSW Government has announced a “Make Safe” assistance package for NSW residents who have loose-fill asbestos in their homes to assist with safely managing asbestos.

6.4 Data and registries

The current approach to managing occupational cancer in Australia is limited and fragmented, creating a significant obstacle to collecting and reporting on data on carcinogen exposure in occupational settings.

To date, data collection on occupational exposures has primarily been limited to mesothelioma and asbestos exposure. In April 2010, Safe Work Australia announced that the new Australian Mesothelioma Registry would be managed by a consortium led by the Cancer Institute NSW and include some of Australia’s leading experts in asbestos related disease. The registry replaced the Australian Mesothelioma Register (which had operated since 1985) and collect notifications of all new mesothelioma cases from state and territory cancer registries and information on mesothelioma patients’ past exposures to asbestos\(^7\).
6.5 References


7 Link between occupation and cancer

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7.1 International data

The association between occupation and cancer has been known for centuries and in some instances this link has lead to the identification of carcinogens.

The International Agency for Research on Cancer (IARC) has for decades maintained a rigorous program evaluating and ranking substances according to their capacity to cause cancer in humans. A number of the agents classified by IARC as carcinogens have primarily occupational exposures\[1\]\[2\]\[3\]. The exposures ranked as Group 1 carcinogens (carcinogenic to humans) and Group 2A carcinogens (probably carcinogenic to humans) and that occur in an occupational setting are outlined in Table 1 below. Many other widely used occupational agents remain under review by the IARC as Group 2B (possible human carcinogens).

In addition, IARC have evaluated a number of work processes and occupations for their capacity to cause cancer in humans. While risk is higher (or probably higher) for people working in these settings or occupations, specific carcinogens have not been identified\[1\], Table 2 outlines the work processes and occupations ranked by IARC as carcinogenic or probably carcinogenic to humans. The level of evidence about the connection between an exposure and a particular cancer varies for different exposure-cancer pairs, even for those exposures defined as human carcinogens.
IARC acknowledges the complexities of identifying lists of potential occupational carcinogens including:

- information on industrial processes and exposures is often poor;
- exposures to well-known carcinogens occur at different intensities in different occupations; and
- levels of exposure can change over time in a given occupation as industrial processes or materials change.

Therefore any list of occupational exposures can only refer to the relatively small number of exposures that have been investigated for carcinogenic risk. The same factors complicate the estimates of the burden of cancer attributable to occupation.

Table 1. Group 1 and Group 2A carcinogens with primarily occupational exposure\(^2\)[4][3]

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Cancer type</th>
<th>Main industry or use</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group 1 carcinogens</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1,3-Butadiene</td>
<td>Leukaemia</td>
<td>Plastic, rubber</td>
</tr>
<tr>
<td>2,3,7,8-tetrachlorodibenzo-p-dioxin, 2,3,4,7,8-pentachlorodibenzofuran, and 3,3’,4,4’,5-pentachlorobiphenyl</td>
<td>All cancers (soft-tissue sarcoma, non-Hodgkin lymphoma, lung)</td>
<td>Metal, electrical, combustion by-products</td>
</tr>
<tr>
<td>2-Naphthylamine*</td>
<td>Bladder</td>
<td>Pigment, rubber</td>
</tr>
<tr>
<td>4,4’-Methylenebis(2-chlorobenzenamine) (MOCA)</td>
<td>NA **</td>
<td>Polyurethane industry</td>
</tr>
<tr>
<td>4-Aminobiphenyl</td>
<td>Bladder</td>
<td>Rubber, pigment</td>
</tr>
<tr>
<td>Aflatoxins</td>
<td>Liver</td>
<td>Agriculture</td>
</tr>
<tr>
<td>Arsenic and arsenic compounds</td>
<td>Lung, skin</td>
<td>Glass, metals, pesticides</td>
</tr>
<tr>
<td>Asbestos</td>
<td>Mesothelioma, lung, larynx, ovary (pharynx, stomach, bowel)</td>
<td>Insulation, construction</td>
</tr>
<tr>
<td>Benzene</td>
<td>Leukaemia (lymphoma, myeloma)</td>
<td>Solvent, fuel</td>
</tr>
<tr>
<td>Benzidine</td>
<td>Bladder</td>
<td>Pigment</td>
</tr>
<tr>
<td>Benzo[a]pyrene</td>
<td>NA **</td>
<td>Coal industry, aluminium production</td>
</tr>
<tr>
<td>Beryllium and beryllium compounds</td>
<td>Lung</td>
<td>Aerospace, metals</td>
</tr>
<tr>
<td>Bis(chloromethyl) ether (BCME) and chloromethyl methyl ether (CMME)*</td>
<td>Lung</td>
<td>Plastic</td>
</tr>
<tr>
<td>Cadmium and cadmium compounds</td>
<td>Lung</td>
<td>Pigment, battery</td>
</tr>
<tr>
<td>Chloromethyl methyl ether*</td>
<td>Lung</td>
<td>Chemical</td>
</tr>
<tr>
<td>Chromium[VI] compounds</td>
<td>Nasal cavity, lung</td>
<td>Metal plating, pigment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Construction,</td>
</tr>
<tr>
<td>Exposure</td>
<td>Cancer type</td>
<td>Main industry or use</td>
</tr>
<tr>
<td>------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>Coal-tar pitch</td>
<td>Lung (bladder)</td>
<td>electrodes</td>
</tr>
<tr>
<td>Coal-tars</td>
<td>Skin, lung</td>
<td>Fuel</td>
</tr>
<tr>
<td>Diesel engine exhaust</td>
<td>Lung</td>
<td>Transport, mining</td>
</tr>
<tr>
<td>Dyes metabolized to benzidine</td>
<td>NA ** (breast, non-Hodgkin lymphoma, multiple myeloma, chronic lymphocytic</td>
<td>Pigment</td>
</tr>
<tr>
<td>Ethylene oxide</td>
<td>leukaemia)</td>
<td></td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>Nasopharynx, leukaemia (sinonasal)</td>
<td>Plastic, textile</td>
</tr>
<tr>
<td>Gallium arsenide</td>
<td>NA **</td>
<td>Semiconductors</td>
</tr>
<tr>
<td>Mineral oils, untreated and mildly treated</td>
<td>Skin</td>
<td>Lubricant</td>
</tr>
<tr>
<td>Mists from strong inorganic acids</td>
<td>Larynx (lung)</td>
<td>Chemical</td>
</tr>
<tr>
<td>Nickel compounds</td>
<td>Nasal cavity, lung</td>
<td>Metal, alloy</td>
</tr>
<tr>
<td>ortho-Toluidine</td>
<td>Bladder</td>
<td>Pigment, rubber</td>
</tr>
<tr>
<td>Radon-222 and its decay products</td>
<td>Lung</td>
<td>Mining</td>
</tr>
<tr>
<td>Shale oils</td>
<td>Skin</td>
<td>Lubricant, fuel</td>
</tr>
<tr>
<td>Silica, crystalline</td>
<td>Lung</td>
<td>Construction, mining</td>
</tr>
<tr>
<td>Soot, as found in occupational exposure of chimney sweeps</td>
<td>Skin, lung</td>
<td>Combustion by-product</td>
</tr>
<tr>
<td>Sulfur mustard (mustard gas)*</td>
<td>Lung (larynx)</td>
<td>Chemical weapon</td>
</tr>
<tr>
<td>Talc containing asbestiform fibres</td>
<td>Lung</td>
<td>Paper, paint</td>
</tr>
<tr>
<td>Welding arc (UV radiation)</td>
<td>Ocular melanoma</td>
<td>Welding</td>
</tr>
<tr>
<td>Vinyl chloride</td>
<td>Liver</td>
<td>Plastic</td>
</tr>
<tr>
<td>Welding fumes</td>
<td>Lung</td>
<td>Welding</td>
</tr>
<tr>
<td>Wood dust</td>
<td>Nasal cavity</td>
<td>Wood</td>
</tr>
<tr>
<td><strong>Group 2A carcinogens</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acrylamide</td>
<td></td>
<td>Plastic</td>
</tr>
<tr>
<td>Benzidine-based dyes</td>
<td>(Bladder)</td>
<td>Pigment, leather</td>
</tr>
<tr>
<td>Captafol</td>
<td></td>
<td>Pesticide</td>
</tr>
<tr>
<td>α-Chlorinated toluenes (benzal chloride, benzo-trichloride, benzyl chloride, benzoyl chloride)</td>
<td></td>
<td>Pigment, chemical</td>
</tr>
<tr>
<td>4-Chloro-o-toluidine</td>
<td>(Bladder)</td>
<td>Pigment, textile</td>
</tr>
<tr>
<td>Cobalt metal with tungsten carbide</td>
<td>(Lung)</td>
<td>Hard metal production</td>
</tr>
<tr>
<td>Creosotes</td>
<td>(Skin)</td>
<td>Wood</td>
</tr>
<tr>
<td>Diazinon</td>
<td>(non-Hodgkin lymphoma, leukaemia, lung)</td>
<td>Agriculture, insecticide</td>
</tr>
<tr>
<td>Diethyl sulfate</td>
<td></td>
<td>Chemical</td>
</tr>
</tbody>
</table>
### Table 2. Industrial processes and occupations listed as Group 1 and Group 2A carcinogens

<table>
<thead>
<tr>
<th>Work process or occupation</th>
<th>Cancer type</th>
<th>Main industry or use</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group 1 carcinogens</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aluminium production</td>
<td>Lung, bladder</td>
<td>Chemical</td>
</tr>
<tr>
<td>Auramine production</td>
<td>Bladder</td>
<td>Chemical</td>
</tr>
<tr>
<td>Boot and shoe manufacture and repair</td>
<td>Nasal cavity, leukaemia</td>
<td>Plastic, textile</td>
</tr>
<tr>
<td>Chimney sweeping</td>
<td>Skin, lung</td>
<td>Plastic, textile</td>
</tr>
</tbody>
</table>

Note: Suspected target organs are given in parentheses

* Agent mainly of historical interest
** Not applicable (agent classified on the basis of mechanistic evidence)
<table>
<thead>
<tr>
<th>Work process or occupation</th>
<th>Cancer type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coal gasification</td>
<td>Lung</td>
</tr>
<tr>
<td>Coal-tar distillation</td>
<td>Skin</td>
</tr>
<tr>
<td>Coke production</td>
<td>Lung</td>
</tr>
<tr>
<td>Furniture and cabinet making</td>
<td>Nasal cavity</td>
</tr>
<tr>
<td>Haematite mining (underground) with exposure to radon</td>
<td>Lung</td>
</tr>
<tr>
<td>Iron and steel founding</td>
<td>Lung</td>
</tr>
<tr>
<td>Isopropanol manufacture by the strong-acid process</td>
<td>Nasal cavity</td>
</tr>
<tr>
<td>Magenta production</td>
<td>Bladder</td>
</tr>
<tr>
<td>Painter</td>
<td>Lung, bladder (leukaemia in offspring with maternal exposure)</td>
</tr>
<tr>
<td>Paving and roofing with coal tar pitch</td>
<td>Lung</td>
</tr>
<tr>
<td>Rubber manufacturing</td>
<td>Leukaemia, lymphoma, bladder, lung, and stomach (prostate, oesophagus and larynx)</td>
</tr>
</tbody>
</table>

**Group 2A carcinogens**

- Manufacture of art glass, glass containers and pressed ware (Lung, stomach)
- Carbon electrode manufacture (Lung)
- Hairdresser or barber (Bladder, lung)
- Petroleum refining (Leukaemia, skin)
- Shift work (Breast)

Note: Suspected associated cancers are shown in parentheses

### 7.2 Australian setting

In Australia, mesothelioma caused by asbestos exposure is probably the best known occupational cancer. However, some carcinogens to which the general public can be exposed, such as ultraviolet (UV) radiation from sunlight and second-hand tobacco smoke, are also important carcinogens in an occupational context.

A 2012 study prioritised occupational carcinogens in Australia based primarily on the potential for occupational exposure and evidence of use in Australian industry, as there was limited exposure information at the time the study was conducted. Carcinogens were prioritised through an assessment framework based on evidence of carcinogenicity using IARC criteria, use in occupational circumstances and use in Australian industry. The priority list (see Table 3) comprises 38 established or probable carcinogenic agents present in Australian workplaces and is designed to guide priorities for preventive action in the Australian setting.

In terms of occupational exposure, the most common carcinogens are estimated to be solar UV radiation, diesel engine exhaust, second-hand tobacco smoke, benzene, lead and silica. See the Impact section for more information.
Table 3. Priority carcinogens for Australia\[5\]

<table>
<thead>
<tr>
<th>Group</th>
<th>Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Combustion products</td>
<td>Diesel engine exhaust, polycyclic aromatic hydrocarbons (PAHs), second-hand tobacco smoke</td>
</tr>
<tr>
<td>Inorganic dusts</td>
<td>Asbestos, crystalline silica dust in the form of quartz or cristobalite</td>
</tr>
<tr>
<td>Organic dusts</td>
<td>Leather dust, wood dust</td>
</tr>
<tr>
<td>Metals</td>
<td>Arsenic and inorganic arsenic compounds, beryllium and beryllium compounds, cadmium and cadmium compounds, chromium (VI) compounds, cobalt metal and tungsten carbide, inorganic lead compounds, nickel compounds</td>
</tr>
<tr>
<td>Radiation</td>
<td>Artificial ultraviolet radiation (UVA, UVB, UVC), ionising radiation, radon-222 and it’s decay products, solar radiation</td>
</tr>
<tr>
<td>Other industrial chemicals</td>
<td>Formaldehyde, glycidol, 4, 4´-methylenebis(2-chloroaniline) (MOCA), nitrosamines, ortho-toluidine (2-aminotoluene), polychlorinated biphenyls (PCBs), styrene-7, 8-oxide, tetrachloroethylene (perchloroethylene), trichloroethylene, vinyl chloride</td>
</tr>
<tr>
<td>Non-chemical agents</td>
<td>Shiftwork that involves circadian disruption</td>
</tr>
</tbody>
</table>

7.3 Occupational carcinogens

7.3.1 Ultraviolet radiation

UV radiation in the form of sunlight is Australia’s most prevalent occupational carcinogen and is particularly important for outdoor workers\[6\]. Over 2 million Australian workers (37% of the Australian male working population and 8% of the female working population) were estimated to be significantly exposed to solar radiation in 2011-2012 in the course of their work\[6\].

Among people exposed to UV radiation in occupational settings, exposure is more likely among males and those in lower socioeconomic and regional areas\[7\]. Although sun protection is used by the majority (95%) of those exposed in occupational settings, only 9% are fully protected\[7\]. Solar UVR is strongly linked to the occurrence of melanoma and the risk of melanoma has been found to be significantly increased for career full-time firefighters, particularly among those who were employed for more than 10 years\[8\].

For information on the impact of UV radiation on cancer and recommendations for reducing workplace UV exposure, see the UV radiation chapter of the National Cancer Prevention Policy and the Sun protection in the workplace position statement.
7.3.2 Asbestos

All forms of asbestos are ranked by IARC as Group 1 carcinogens\(^4\). Asbestos exposure is responsible for high levels of mesothelioma and lung cancer incidence and mortality worldwide and also causes laryngeal and ovarian cancer\(^4\). Around 90-100% of mesothelioma cases are linked with asbestos exposure\(^9\)\(^{10}\).

Australia has one of the highest rates of mesothelioma incidence in the world\(^{11}\)\(^{12}\)\(^{13}\). Mesothelioma incidence rates have steadily increased in Australia over the last 30 years\(^{14}\). Total cases are expected to reach around 18,000 by 2020\(^{15}\).

Mesothelioma, resulting from occupational asbestos exposure, is one of the most comprehensively studied occupational cancers in Australia. The most extensively documented exposure to asbestos by area relates to the mining town of Wittenoom in Western Australia, where mining operations caused disease not only in the workforce, but also among residents\(^{16}\)\(^{17}\)\(^{18}\).

A retrospective analysis of mesothelioma in Australia between 1945 and 2000 found Australia's high incidence was linked to a history of prevalent asbestos use, of all fibre types, across varied occupational and environmental settings\(^{11}\). The Australian Mesothelioma Registry (AMR) provides estimates of asbestos exposure data based on the exposure profiles of those diagnosed with mesothelioma\(^{19}\). Based on cases diagnosed since July 1\(^{st}\), 2010 over 60% of people diagnosed were identified by the AMR as having ‘possible’ or ‘probable’ asbestos exposure in an occupational setting\(^{20}\).

While commercial production and use of asbestos have been banned in Australia for many years, inadvertent exposure remains a problem because of its widespread presence in building materials such as fibro, which is in place in thousands of established structures. Mesothelioma cases related to renovation are projected to continue to increase due to the number of homes still containing asbestos building products\(^{21}\). Exposure to asbestos during home renovation is common\(^{22}\). From 2005-2008 home renovators accounted for 8.4% of all men and 35.7% of all women diagnosed with mesothelioma\(^{21}\).

Unless occupational health and safety standards are improved Australia-wide, opportunities to minimise future disease burden associated with the high prevalence of asbestos-containing materials already in the community will not be realised. "Lessons must be learned to help revive the currently waning societal commitment to occupational health and safety in Australia and elsewhere", to prevent unnecessary asbestos-caused disease\(^{23}\). See Effective interventions for more information.

7.3.3 Second-hand tobacco smoke

Second-hand tobacco smoke causes lung cancer and has been classified as a Group 1 carcinogen\(^{24}\).

Despite workplace bans on smoking, second-hand tobacco smoke remains one of the most common occupational carcinogens in Australia, with over 1.1 million working men and 240,000 working women estimated to be exposed\(^{6}\).
Given the time lag between exposure and diagnosis, the full impact of tobacco smoking in the workplace has not yet been evident in cancer incidence and mortality data.

For more information, see the Tobacco control chapter of the National Cancer Prevention Policy.

### 7.3.4 Diesel engine exhaust

Diesel engine exhaust is classified by IARC as a Group 1 carcinogen. Diesel engine exhaust is one of the most common occupational carcinogens in Australia, with over 1.3 million working men and 250,000 working women estimated to be exposed\(^6\). Occupational exposure to diesel exhaust increases lung risk and probably also increases bladder cancer risk\(^{25-26}\).

Professional drivers potentially exposed to diesel exhaust are at increased risk of lung cancer\(^{27}\) and probably of bladder cancer\(^{26}\). There is good evidence that lung cancer mortality is associated with occupational exposure to diesel exhaust in miners\(^{28}\).

Combined data from three occupational cohort studies suggest that diesel engine exhaust at levels common in the workplace and in outdoor air appear to pose substantial excess lifetime risks of lung cancer\(^{29}\). Two independent studies of trucking industry workers\(^{30,31}\) and a study of non-metal miners\(^{32}\) were included in this meta-regression. Past occupational and environmental exposure to diesel engine exhaust was estimated to be responsible for 1.3% and 4.8% of annual lung cancer deaths in the United States and the United Kingdom respectively\(^{29}\).

### 7.3.5 Pesticides and herbicides

Worldwide, occupational exposure to pesticides and herbicides have been clearly implicated as a carcinogenic hazard, although studies have been unable to consistently identify specific agents or classes of agents.

In March 2015, the International Agency for Research on Cancer (IARC) assessed the carcinogenicity of five organophosphate pesticides\(^3\). Glyphosate (a herbicide), malathion and diazinon (insecticides) were classified as probably carcinogenic to humans (Group 2A). There was limited evidence of carcinogenicity of glyphosate to humans based on non-Hodgkin lymphoma associated with occupational exposure studies in the US\(^{33}\), Canada\(^{34}\) and Sweden\(^{35}\) as well as evidence of cancer in laboratory animals. For the insecticides malathion and diazinon, there was limited evidence of carcinogenicity for non-Hodgkin lymphoma, prostate cancer (malathion only) and lung cancer (diazinon only). The epidemiological evidence for malathion comes from studies of agricultural exposures in the US\(^{36}\), Canada\(^{34}\) and Sweden\(^{35}\). Malathion also caused tumours in rodents. The evidence for carcinogenicity of diazinon comes from studies of agricultural exposures in the US\(^{36}\) and Canada\(^{34}\). While there was limited evidence of carcinogenicity in humans found, there was strong mechanistic evidence of the carcinogenicity of glyphosate, malathion and diazinon with all three agents inducing DNA and
chromosomal damage in human and animal cells in vitro. The insecticides tetrachlorvinphos and parathion were classified as possibly carcinogenic to humans (Group 2B) based on sufficient evidence that these pesticides cause cancer in laboratory animals. Given the recent IARC findings, the Cancer Council recommends the Australian Pesticides and Veterinary Medicines Authority considers altering current usage and/or handling requirements for these particular herbicides.

Pesticides are a heterogeneous group of chemicals, with different substances and mixtures in use having changed over time. Retrospective exposure assessment identifying the type and extent of individuals' exposure to pesticides is likely to result in misclassification. Inconsistency in the evidence on pesticides and cancer underscores the need for a cautious approach to working with these potentially carcinogenic substances and for more research on the potential harms associated with specific agents.

Investigation of a cohort of Australian workers exposed to pesticides did not find evidence of a relationship between occupational pesticide exposure and cancer, nor other non-injury-related mortality. However, a separate case-control study found substantial exposure to any pesticide was associated with a trebling of the risk of non-Hodgkin lymphoma. Subjects with substantial exposure to organochlorines, organophosphates, and "other pesticides" (all other pesticides excluding organochlorines, organophosphates and herbicides) and herbicides other than phenoxy herbicides had similarly increased risks, although the increase was statistically significant only for “other pesticides”.

See the Cancer Council position statement on Pesticides and cancer for more information.

### 7.3.6 Ionising radiation

Ionising radiation is classified as a Group 1 carcinogen to humans by the IARC. A retrospective cohort study examined cancer incidence among Australian nuclear industry workers and found no evidence of a significant increase in cancer risk. Exposure to ionising radiation is carefully controlled under strict Occupational Health and Safety regulations and workers who have potential for exposure carry personal monitors.

### 7.3.7 Welding

Welding activities produce many hazards through the production of contaminants in welding fume and ultraviolet (UV) radiation in the welding arc, both of which have been classified as Group 1 carcinogens (carcinogenic to humans) by IARC. Exposure to welding fumes or welding arc can increase your risk of developing lung cancer and ocular melanoma respectively. There is limited evidence for increased risk of kidney and other cancers.
Welding fume is made when a metal is heated above its boiling point. The metal cools and then condenses into fume which produces fine particles that can be inhaled. Welding fumes contain potential carcinogens including metallic oxides, silicates and fluorides\textsuperscript{[42][4]}. A positive exposure-response association was found between welding fume exposure and lung cancer\textsuperscript{[45][46][47]}. The increase in lung cancer was unable to be explained by asbestos exposure and/or tobacco smoking\textsuperscript{[44][45][46][47][48]}.

Electric arc and laser welding give off UV radiation. UV exposure was found to increase the risk of developing an ocular melanoma generally between two and ten times\textsuperscript{[51][50]}. These results remained consistent even in studies that adjusted for sun exposure and/or sunbed use. Furthermore, a relationship was found between ocular melanoma and both duration of employment as welder\textsuperscript{[51][50]} and eye burns\textsuperscript{[53]}.

### 7.4 Other occupational risk factors

#### 7.4.1 Sedentary work

There is mixed evidence for a positive association between occupational sitting and cancer\textsuperscript{[54][55]}. However, physical inactivity is an important cancer risk factor and should therefore be addressed in the workplace.

For more information see the Overweight and obesity, physical activity and nutrition chapter of the National Cancer Prevention Policy, and the Policy context section of this chapter.

#### 7.4.2 Shift work

Shift work involving circadian disruption has been classified by IARC as a probable human carcinogen (Group 2A)\textsuperscript{[56]}. Shift work is common in a number of industries, including manufacturing, mining, healthcare, hospitality, communication and transport. Night work causes most disruption to the circadian clock, the 24-hour biological cycle that governs sleeping and waking in humans\textsuperscript{[57]}. The strongest evidence for carcinogenicity of shift work is for breast cancer\textsuperscript{[56]}. Women working shift work involving nights appear to have an increased risk of developing breast cancer\textsuperscript{[58][59][60][61][62]}. There are however, some inconsistencies with a dose-response relationship between shift work and breast cancer\textsuperscript{[58][63]}.
7.5 Parental exposures and cancer risk in children

The impact of occupational exposures may go beyond the individual worker and contribute to increased cancer in family members. Research has suggested that parental exposure to certain carcinogens or work environments may increase cancer risk in their children - implicated exposures include vehicle exhaust, painting, wood trades, pesticides and other agricultural exposures, electronics, textiles and second-hand smoke during pregnancy.[64][65][66][67][68][69] There are, however, no definitive findings that enable quantification of the risk.

See the Pesticides and cancer position statement for more information on the link between parental exposure to pesticides and cancer in offspring.

7.6 Health inequality

Cancer incidence and mortality in Australia is higher among less socio-economically advantaged people as measured by factors such as income, educational attainment, employment and skill levels in specific occupations.[70][71] While much of this disparity in cancer incidence can be attributed to risk factors such as smoking, obesity/overweight, poor diet and sedentary behaviour – which are more prevalent among people with socioeconomic disadvantage[72] - the risk of occupational cancer appears to be higher among socially disadvantaged people, who are more likely to be employed in relatively unskilled jobs where exposure to carcinogens is higher.[9][70].

Industrial and workplace exposures to carcinogens are more common among manual workers, or employees with lower levels of job skills, education and training.[9][73][74]. In industrialised countries, occupational exposures may be responsible for about one third of the excess of all cancers occurring in people in lower socioeconomic groups.[74].

Eliminating or reducing exposure to occupational carcinogens is therefore an important part of redressing health disparities linked to socioeconomic disadvantage in Australia.

7.7 References


8 Effective interventions

8.1 Monitoring and surveillance

Comprehensive monitoring of exposure to carcinogens in the workplace and health monitoring of workers potentially exposed to carcinogens are both important strategies in reducing the burden of occupational cancers [1].

Recent research has provided comprehensive data on the extent of potential carcinogen exposure in occupational settings in Australia [2]. However, there are no systems in place for routine, systematic assessment of exposure, awareness, compliance with regulations, cancer incidence or prevention activities with respect to occupational cancers [3]. Improved data on the circumstances of occupational exposure to carcinogens and their effect over lengthy time periods is essential to reduce the burden of occupational cancers [4].
Occupational exposure matrices such as the CAREX (CARcinogen EXposure) database provide exposure data and estimates of exposed workers by carcinogen and industry for Group 1 (carcinogenic to humans), Group 2A (probably carcinogenic) and selected Group 2B (possibly carcinogenic) carcinogens for 19 countries in the European Union \(^5\) and Canada \(^6\).

Documentation of all carcinogens and carcinogenic processes used in Australian workplaces by statutory authorities such as Safe Work Australia and the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) would facilitate research on occupational exposures. However, this would require considerable resources, and the most effective way to accomplish such monitoring is unclear.

Surveillance strategies in addition to monitoring of exposures in the workplace include biological monitoring of the uptake of potential carcinogens in the body, monitoring of conditions related to carcinogen exposure, testing DNA for interactions with carcinogens and screening for pre-clinical or early cancer \(^7\).

Improved health monitoring of workers who may have been exposed to occupational carcinogens should assist both in the collection of data and in increasing opportunities for earlier detection which should lead to improved survival. State-based agencies, such as the Dust Diseases Board of NSW, run occupational respiratory screening services. Currently, no systematic collection of exposure data occurs in Australia however, several possible sources of data exist:

- **NICNAS** are responsible for assessing (but not regulating) health and environmental impacts of industrial chemicals.
- **The National Pollutant Inventory** publishes data on annual emissions of 93 specific chemicals identified as important due to their possible effect on human health and the environment.
- **Safe Work Australia** publishes some research relating to workplace health, but does not provide ongoing estimates of the number of workers exposed to particular carcinogens.

There are limitations to undertaking carcinogen exposure and cancer surveillance. A number of cancers linked to occupation, such as lung cancer and mesothelioma, cannot be detected early through the screening of asymptomatic people. Moreover, current diagnostic technology cannot distinguish between cancers potentially caused by occupational exposures and those that would have occurred irrespective of workplace carcinogens. Examples include cancers in nurses who have worked with cytotoxic drugs and smelter workers who are diagnosed with lung cancer.

### 8.2 Inter-sectoral partnerships

Effective cancer control policy, particularly where exposures are widespread, requires cross-sectoral partnerships across a range of sectors such as health, industry, trade, and environment \(^8\). Building on this type of partnership approach is integral to reducing the incidence of occupational cancers.
Occupational health and safety in Australia is the remit of a range of organisations, offering abundant opportunities for joint approaches to policy and industry compliance. Overall occupational safety is in the remit of every state and territory government (through occupational health and safety agencies and workers’ compensation authorities) and several federal regulatory authorities, including Safe Work Australia, NICNAS and specialist agencies concerned with the maritime, petroleum and transport industries. Recent examples of collaboration include:

- the establishment of the Nanotechnology Work Health and Safety Advisory Group involving Safe work Australia, NICNAS, the Australian Council of Trade Unions (ACTU) and Australian Chamber of Commerce and Industry (ACCI), among others;
- the ACTU joining the International Metalworkers’ Federation and other international union bodies in 2008 to support the Occupational Cancer/Zero Cancer campaign[^9]; and
- the ACTU and Cancer Council Australia’s joint national forum, kNOw Cancer in the Workplace held in December 2009[^10].

Greater collaboration across these inter-sectoral groups should be sought. More broadly, peak business organisations such as ACCI and the Australian Industry Group, along with professional groups like the Faculty of Occupational and Environmental Medicine (within the Australasian College of Physicians) and the Australian Institute of Occupational Hygienists are potential partners.

8.3 Integrated health protection and health promotion programs

The workplace provides a structured framework for delivering health messages, whether relating to hazardous exposures and harmful work practices or to individual health behaviours, such as smoking, nutrition and physical activity.

Recent approaches to health promotion in the workplace have been based on the premise that home and work life both contribute to individual health and that best results are achieved when behaviours in both settings are addressed. Integrated programs that target both workplace safety and individual health have the potential to increase the effectiveness of separate programs in each setting[^11]. Employees who see that management is making a genuine attempt to improve workplace safety may also be more likely to engage in safe work practices and other wellness programs, particularly blue collar workers[^11][^12].

The World Health Organisation’s Global Plan of Action on Workers’ Health 2008-2017[^13] endorses integrated programs for occupational safety and health promotion initiatives for individual health, including support for smoking cessation, healthy diet and physical activity, and promoting mental and family health. In the US, the national WorkLife Initiative (an alliance between the US Department of Health and Human Services, the Centers for Disease Control and the National Institute for Occupational Health and Safety) calls for the same approach[^14]. In Australia, the Government’s National Preventive Health Taskforce identified an opportunity for introducing and evaluating integrated workplace health improvement programs along the lines of the WorkLife Initiative[^15]. However, the evidence base supporting the effectiveness of such programs is required.
8.4 Evaluating potential risks

While evidence is clear that a number of industries and materials cause occupational cancer (see the Links section of this chapter), there may be other widespread current work practices that have not yet been identified as increasing cancer risk. The potential time lag between carcinogen exposure and cancer diagnosis underscores this potential problem, particularly in relation to practices and technologies that have been introduced in recent years. It is therefore critical to evaluate the potential health risks of new work practices, materials and technologies as they are introduced, while recognising that evaluation is difficult to perform comprehensively.

For example, little is known about the long-term health risks for workers using nanotechnology (the manipulation of particles so small that they can penetrate human cellular structures). Under the Nanotechnology Work Health and Safety Program, Safe Work Australia conduct nanotechnology research and provide guidance on the potential work safety and health implications of nanotechnology applications\textsuperscript{[16]}. Ongoing monitoring and evaluation will be key to building the evidence base around nanotechnology, to determine if there is an increase in cancer risk associated with the industry.

8.5 Legislation to improve policy

Regulation has an important role to play in preventing occupational cancer. Currently, the scope and design of relevant regulations in Australia is inherently complex because of Federal and State Government relationships, and does not comprehensively addressing all recognised hazards.

Both legislative and common law impose a duty of care on employers to provide a safe workplace. Regulations in a range of state-based industrial and other legislative frameworks underpin employers’ responsibilities for preventing work-related injury and limiting exposure to workplace carcinogens.

Legislation changes in 2012 have provided a platform for harmonisation of health and safety laws between Australian jurisdictions, as well as with international standards. The Model work health and safety laws were introduced under the Intergovernmental Agreement for Regulatory and Operational Reform in Occupational Health and Safety. All jurisdictions have committed to adopting the legislation. The model work health and safety laws include implementation of the Globally Harmonized System of Classification and Labelling of Chemicals (GHS) for occupational and environmental hazardous substances. GHS is an internationally agreed system which harmonises classification systems and hazard communication globally.

See Policy context for more information on legislation.

8.6 Compensation to improve policy

Compensation can be payable to individuals deemed to have developed cancer as the result of occupational exposures, with cancers related to the use of asbestos the most prevalent example. Authorities such as the Dust Diseases Board of NSW have been involved in such compensation cases.
As well as providing some financial compensation to affected individuals, a potential benefit of litigation is that the disincentive of paying damages may prompt employers to improve occupational health and safety practices and contribute to tighter laws. The recent work on deemed diseases by Safe Work Australia should streamline legitimate claims for occupational cancer and raise awareness of the risk as well as increase the incentives to adequately work-related exposure to carcinogens\textsuperscript{17}.

8.7 Primary prevention

Primary prevention of cancers through removal of carcinogens or reductions in exposure has proven effective in a range of occupational settings\textsuperscript{18}\textsuperscript{19}. Despite this, there is a tendency for primary prevention to be under-resourced and uncoordinated, and to not make full use of existing knowledge\textsuperscript{8}.

Primary prevention of occupational cancers requires explicit legislation across health, social security and labour sectors\textsuperscript{8}. See the section on legislation to improve policy for more information.

Banning carcinogenic substances or practices and replacing them with less hazardous alternatives is potentially the most effective way to eliminate occupational cancers. However, this may not always be feasible\textsuperscript{7}. Where the use of carcinogens cannot be avoided, there is an established hierarchy of preventive measures for limiting exposures. Engineering controls may be introduced to isolate substances or reduce exposure through the use of robotics, enclosed working systems or ventilation. Work practices may also be altered – for example by limiting outdoor work when UV radiation is at its peak or introducing procedures to dampen down dusty environments.

Personal protective equipment (PPE) in the form of respirators, gloves and other special clothing also have an important place, but must be regularly checked, properly maintained and correctly used. Barriers to the effectiveness of PPE include that it can be uncomfortable to wear and make tasks more difficult or dangerous. PPE also places the onus for compliance on the employee. For these reasons, PPE should not be relied upon as the only form of exposure protection. Unfortunately, it is often the first line of defence used in the workplace against hazardous substances, including occupational carcinogens\textsuperscript{3}.

8.8 Research

Comprehensive research is essential to develop a strong evidence base to underpin policy across the cancer control spectrum. This is a critical priority in relation to occupational cancers, where there are significant gaps in the evidence base on the nature and extent of exposure to carcinogens in Australian workplaces, and on their impact on cancer occurrence. This significantly restricts the development of evidence-based occupational health and safety policy. Building the evidence base is therefore a major public health policy priority in itself.

Some data on occupational cancer in Australia has been collected for more than 50 years, preceding the establishment of population-based cancer registries\textsuperscript{3}. Studies have typically involved job descriptions, or carcinogenic exposures identified elsewhere, and attempts to apply the findings to Australian workplaces.
Compared with occupational cancer studies in more populous North American and Western European settings, numbers of Australians in particular industries tend to be smaller. Despite this limitation, results of local research provide clear justification for further studies.

8.9 References


9 Version information

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