Australian Government

Australian Safety and Compensation Council

OCCUPATIONAL CANCER IN AUSTRALIA

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FOREWORD

The Australian Safety and Compensation Council (ASCC) leads and coordinates national efforts to prevent workplace death, injury and disease in Australia and aims to improve national workers' compensation arrangements and return to work of injured employees.

Through the quality and relevance of the information it provides, the ASCC seeks to influence the awareness and activities of every person and organisation with a role in improving Australia's occupational health and safety (OHS) performance.

The *National OHS Strategy 2002-2012, (*the National Strategy) which was endorsed by the Workplace Relations Ministers' Council on 24 May 2002, records a commitment by all Australian, State and Territory governments, the Australian Chamber of Commerce and Industry and the Australian Council of Trade Unions, to share the responsibility of ensuring that Australia's performance in work-related health and safety is continuously improved.

The National Strategy sets out five 'national priorities' to achieve short-term and long-term improvements.

The priorities are to:

- reduce high incidence and high severity risks
- improve the capacity of business operators and worker to manage OHS effectively
- prevent occupational disease more effectively
- eliminate hazards a the design stage, and
- strengthen the capacity of government to influence OHS outcomes.

In March 2004 it was agreed by the then National Occupational Health and Safety Commission (NOHSC) that, under the national priority to prevent occupational disease more effectively, eight disease categories would be considered for particular focus under any national action plan. These are work-related musculoskeletal disorders; mental disorders, noise-induced hearing loss; respiratory diseases; occupational cancers; contact dermatitis; infectious and parasitic diseases, and cardiovascular disease.

To assist the setting of national action priorities to prevent these diseases, reports were prepared for members on each disease category. The following report is an extract of the information provided to members on the causes and risk factors for cardiovascular disease, the available data on the magnitude and severity for the disease category within Australia, approaches to prevention and evidence for their effectiveness.

ACKNOWLEDGMENTS

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EXECUTIVE SUMMARY

"Cancer" is a term for diseases in which abnormal cells divide without control. The term cancer is used to encompass a range of different diseases (eg adenocarcinoma of lung, melanoma, hepatocellular carcinoma). There are a number of well-known sets of circumstances which have demonstrated without doubt the link between some occupational exposures and some types of cancer. The problem arises when we examine more subtle effects on common cancers. The question then arises: "Is this cancer one which would have occurred anyway, or is it is a direct result of the person's occupation?"

Magnitude of the problem

There is no available data on the real magnitude of occupational cancer. Because of difficulties in proving causation, and the long lag time between occupational exposure and cancer, very few cancers are reported to compensation authorities. To estimate the magnitude of the problem of occupational cancer, we need to use more indirect methods to determine what proportion of cancers are due to occupational causes.

In this paper, the proportion of cancers due to occupation has been estimated using the Finnish estimates (which are the most recent and the most evidenced based) and applying them to Australian cancer data. Using this method, it was estimated that about 11% of incident cancers in males and 2% in females may be caused by occupation. This equates to about 5000 cancers a year.

The paper also provides an estimate of the number of workers potentially exposed to carcinogens at work. A recent estimate from the European Union (Kogevinas et al., 1998) on percentage of workers exposed to carcinogens in particular industries was applied to the Australian industry profile. Using this approach it was estimated that approximately 1.5 million workers in Australia are potentially[1] exposed to carcinogens at work. However, this does not consider the adequacy of workplace controls which may eliminate or reduce the exposure.

Occupations and Industries

The international agency for research in cancer (IARC) lists carcinogenic risks posed to humans by a variety of agents and circumstances. While it is known that the carcinogens and similar occupations and industries occur in Australia further work is required to characterise exposure in Australian workplaces in order to focus efforts on prevention in risk areas.

Prevention activities

The task of reviewing prevention activities is complicated because there are many different substances which do or may cause different types of cancer and these are used in many different occupations. In addition, there is no easily accessible information on prevention activities which have been successful (or have failed).

[1] This calculation does not take into account control measures, thus they are "potentially" exposed.

The most effective means of preventing exposure is to ban the carcinogen. This is used for a small number of occupational carcinogens, with asbestos being the best-known example. Alternatively, a carcinogenic substance may be substituted with one that is not carcinogenic, or is less hazardous. A potentially expensive, but effective means of preventing exposure to carcinogens is to introduce engineering controls, for example, ventilation, enclosure or partial enclosure. Other methods include isolation in which the carcinogen is separated from workers by distance or using robotics. Safe work procedures can also be introduced in order to reduce exposure to carcinogens. Examples include limiting the times at which outdoor work is performed, or ensuring that dust is dampened down.

Use of personal protective equipment (PPE) is the least efficient way of controlling hazardous exposures. It means that the emphasis is entirely on the worker to comply, regular maintenance and fitting checks are required, and the PPE may be uncomfortable or hot, or may make tasks difficult or dangerous. Unfortunately, PPE such as respirators, gloves or overalls are often used as the first step to decrease worker exposure to carcinogens.

As occupational cancers can be difficult to identify and control, their prevention is likely to be more effective by implementing complementary actions such as education towards employers, workers, and physicians, research in order to identify new hazards and their impact on workers' health and review of workplace safety and standards on an ongoing basis. Ideally, this process is best informed by a comprehensive national surveillance scheme to assist in effective targeting of preventive measures.

Conclusions

Very little co-ordinated information is available in Australia on how many people are potentially exposed to known or suspected carcinogens, how aware workers are of carcinogenic substances, whether regulations about carcinogenic substances are being followed, how many cancers are caused by occupational exposure to carcinogens, and what preventive activities including workplace controls are being undertaken.

DEFINITIONS

Cancer

"Cancer" is a term for diseases in which abnormal cells divide without control. Cancer cells can invade nearby tissues and can spread through the bloodstream and lymphatic system to other parts of the body.

Cancer is really a term of convenience for a group of diseases. There are two main ways to group cancers: according to the site of origin, and according to the type of cell. The main subgroups for type of cell are carcinomas, leukaemias, lymphomas and sarcomas. The site of origin can be any organ of the body, such as the lung or the ovary.

Cancers of different cell types or different sites of origin are quite different in their causes, their behaviour, their response to treatment and their prognosis. So when discussing cancer as an entity, it is important to realize that the overall picture of "cancer" depends on which cancers are most common in that population.

Carcinogen

A carcinogen is a substance or agent that causes the development of or increases the incidence of cancer. (*Mosby's medical, nursing & allied health dictionary*. 6th ed. 2002)

Carcinogenic risk

"Carcinogenic risk" is the probability that exposure to an agent will lead to cancer in humans. (IARC)

Carcinogenesis

The process of initiating and promoting cancer

Histology

The science dealing with the microscopic identification of cells and tissues

1. OCCUPATIONAL CANCER: OVERVIEW AND CONTEXT

1.1 Cancer overview

In 2000, there were over 85 000 cases of cancer in Australia (not including non melanoma skin cancer) and over 35 000 deaths (Australian Institute of Health and Welfare (AIHW) & Australasian Association of Cancer Registries (AACR), 2003). In addition, a recent study estimated that in 2002, there had been 118 000 treated squamous cell carcinomas and 256 000 treated basal cell carcinomas, meaning there were about 374 000 treated non-melanoma skin cancers each year (National Cancer Control Initiative, 2003).

The most common cancer sites in males in Australia are prostate, colorectal, lung, and melanoma. In females the most common sites of cancers are breast, colorectal, melanoma and lung (Australian Institute of Health and Welfare (AIHW) & Australasian Association of Cancer Registries (AACR), 2003).

Because of different prognoses with different cancers, the cancers that cause the most death are somewhat different to those that occur most commonly. In males, the most common causes of cancer death are lung, prostate, colorectal and unknown primary site. In females they are breast, lung, colorectal and unknown primary site. Cancers of unknown primary site are ones that are diagnosed late after they have spread through the body.

1.2 Occupational cancer overview

A number of well-known sets of circumstances have demonstrated without doubt the link between some occupational exposures and cancer. Most lay people are aware of the strong link between asbestos work and mesothelioma. Other well-established causal links have been made between vinyl chloride manufacture and angiosarcoma of the liver, and between cadmium and prostate cancer. For known occupational carcinogens, the usual occupational hygiene hierarchy of controls should be followed (elimination, substitution, engineering controls, safe work procedures, personal protective equipment).

An occupational cause is relatively easy to prove if the cancer is a very unusual type, or the carcinogenic effect of the exposure is so strong that the number of cancers occurring in an occupation is clear. The problem arises when we examine more subtle effects on more common cancers. The question then arises: “Is this cancer one which would have occurred anyway, or is it is a direct result of the person’s occupation?”

Current carcinogenesis theory inclines to the view that cancer is a multi-step process which arises from a combination of multiple genetic predispositions and multiple environmental insults. This process may take many years to result in cancer. For example, not everyone who is exposed to asbestos has the same risk of getting lung cancer. Possible

reasons for this include: differences in dose of asbestos; other factors which might also contribute to the risk of lung cancer (eg smoking, or other chemicals at work); the long time between exposure and cancer (so the person may die of something else first); and genes which increase or decrease the risk of lung cancer (perhaps ones which code for genetic repair mechanisms etc). Teasing out these different influences is not easy, especially when the relevant occupational exposures may have occurred many years earlier and been poorly documented. In addition, there is no way to differentiate histologically between cancers caused by different factors.

There are many thousands of chemicals which are in use in industry today, some of these are known to cause cancer, and some are known not to be associated with cancer. There are also many chemicals for which the evidence is not yet strong enough to determine whether or not they are carcinogenic. Prevention of occupational cancer in Australia therefore consists of two major challenges: to ensure that workers are not exposed to known carcinogens: and to extend our knowledge regarding the carcinogenicity of chemicals used in industry today, and of those chemicals which may be introduced in the future.

2. MAGNITUDE OF THE OCCUPATIONAL CANCER BURDEN

In order to estimate the magnitude of the problem of occupational cancer, we need to determine what proportion of cancers are due to occupational causes. In order to do this four different items of data are required:

1. A list of carcinogens
2. An estimate of the relative risk of each cancer type for each carcinogen
3. The proportion of people exposed to each carcinogen at work
4. The number of cancers in the country

Except for the last point, obtaining these data is quite difficult because:

1. While we have a list of confirmed occupational carcinogens (category 1 carcinogens on the *List of Designated Hazardous Substances*, as classified using the *Approved Criteria for Classifying Hazardous Substances),* there may be other carcinogens that do not appear on the List. The List is not an exhaustive compilation of all hazardous substances used in Australia, however, suppliers of substances not included on the List are still obliged to classify them according to the Approved Criteria. The process of confirming that an agent is a definite human carcinogen is extremely difficult and time consuming. Several international organisations do have programs of classifying carcinogens , the best of which is the program co-ordinated by the International Agency for Research in Cancer (IARC).

2. We have poor evidence on the potency of even confirmed carcinogens
3. There is little information on prevalence of exposure to occupational chemicals in the workplace.

Due to factors discussed previously[2], it is difficult to ascribe individual cases of cancer to a person's work. It is thus not possible to simply count the number of cases of cancer caused by work or contributed to by work. For this reason, we must rely on estimates of the proportion of cancers due to occupational factors. Appendix 1 discusses in full previous attempts to estimate the proportion of cancers due to occupational factors in Australia and overseas.

3. BURDEN OF DISEASE FROM OCCUPATIONAL CANCER IN AUSTRALIA

As discussed above, in order to estimate the burden of disease the important pieces of information are: the definitive list of carcinogenic substances; an estimate of the relative risk of cancer for each carcinogen; the number of people exposed at work; and the number of cancers. In Appendix 2, the issues for each of these items are outlined, as relates to developing estimates for the burden of occupational cancer in Australia.

3.1 Determining the number of occupationally-caused cancers in Australia

The most recent and most evidence-based estimate of the percent of cancers caused by occupational exposures is that of Nurminen and Karjalainen in 2001. Their estimates of the percent of cancers, which are caused by occupation, can be applied to Australian numbers of incident cancers. To do this, the number of incident cancers in 2000 (excluding non-melanoma skin cancers) was obtained from the Australian Institute of Health and Welfare (Australian Institute of Health and Welfare (AIHW) & Australasian Association of Cancer Registries (AACR), 2003). The Finnish estimate of the percent of each site of cancer, which was occupationally caused, was then multiplied by the number of cancers at that site in Australia. This calculation suggests that about 5 000 cancers a year may be occupationally caused (Appendix 2, Table A2.1 and Table A2.2). This is about 11% of incident cancers in males and 2% in females.

Australian cancer registries do not routinely collect data on non-melanoma skin cancer (NMSC). In 2002, a population survey was carried out to determine the number of treated NMSCs in Australia (National Cancer Control Initiative, 2003). The Finnish review suggested that 13.1% of NMSCs in males and 3.8% of NMSCs in females were caused by occupational exposure (Nurminen and Karjalainen, 2001). If these proportions are applied to the number of NMSCs in Australia (National

[2] Long latency between exposure and manifestation of disease and the multi-factorial nature of cancer

Cancer Control Initiative, 2003), about 34 000 NMSCs in Australia may be caused by occupation.

4. NUMBER OF AUSTRALIAN WORKERS POTENTIALLY EXPOSED TO CARCINOGENS

An alternative to estimating the number of cancers is to use the number of workers exposed to carcinogens as a measure of the problem. Unfortunately, this information is almost impossible to collect at a national level in Australia even for prohibited and notifiable carcinogens on the NOHSC lists. Notifications of use go to individual state and territory authorities, that do not have databases which are easily accessible.

In 2001, NOHSC attempted to estimate the number of Australian workers potentially exposed to just one well-regulated substance for the "Regulatory Impact Statement of the Proposed Phase Out of Chrysotile Asbestos". NOHSC found that chrysotile asbestos was used by 3 manufacturers and 7 857 processing and end use companies. Of these, 7 856 of the processing and end use companies covered some 33 000 separate work sites (repair shops). They estimated that 300 people were exposed to chrysotile in manufacturing, and from 10 000 to 22 000 were exposed in processing and end use. This takes into account an estimate from Victorian WorkCover that 2 in 5 employees in the automotive repair industry would be exposed to asbestos.

It would be possible to repeat this exercise for either the 23 prohibited and notifiable substances on the NOHSC lists or the 57 different occupations or substances mentioned by Nurminen and Karjalainen (Nurminen and Karjalainen, 2001). To do this, for each carcinogen in turn, it would be necessary to determine all jobs in which exposure to the carcinogen could occur. The lack of concordance between exposures and job classifications would make this task quite difficult. Once the list of jobs had been decided upon, the proportion of workers in that job with exposure to each carcinogen would be estimated. The number of workers in each job could then be obtained from ABS statistics.

An alternative is to use overseas estimates of the proportion of workers exposed and apply them to Australian statistics on the number of workers in each industry. The difficulty with this approach is the potential differences in the profile of industries in different countries. In the European Union, an international group of experts was asked to provide documented estimates of the number of workers exposed to carcinogens .(Kogevinas et al., 1998, Kauppinen et al., 2000) They undertook the following steps:

- defined carcinogens as those classified by IARC as Group 1, Group 2A and some 2B.
- collected labour force data for 55 industrial classes
- collected the prevalence of workers exposed to each substance in each industry from Finland and the United States

- entered this information into a specially designed exposure information system called CAREX which calculated the number of workers exposed to each substance
- earmarked industries with low level exposures
- estimated the number of workers exposed to multiple substances and converted the number of exposures to the number of exposed workers
- generated final estimates of number of workers exposed.

From this process the finding was that 32 million workers (of a total 139 million employed workers) in the EU in 1990-3 were potentially occupationally exposed to carcinogens. This is 23% of the workforce. If we consider that 23% of the Australian workforce might be exposed to occupational carcinogens, then it is possible that approximately 1 691 400 workers in Australia may be occupationally exposed. However, it should be noted that this does not consider the adequacy of workplace controls which may eliminate or reduce the potentia; exposure.

However, the overall proportion calculated by the EU study, has within it a weighting for different industries depending on how many people in the EU are working in that particular industry. The profile of industry in Australia differs from that in the EU in several important respects. So a more accurate estimation may be to use the individual estimates for each industry and apply them to Australian data. The occupational groupings from the EU study (Kogevinas et al., 1998) were converted to the Australian and New Zealand Standard Industrial Classification groups (Australian Bureau of Statistics, 1993). The number of workers in each occupational group was obtained (Australian Bureau of Statistics, 2001). The number of workers in each group was then multiplied by the EU estimate of the proportion of workers in that group exposed to carcinogens. This calculation demonstrates that approximately 1 494 090 workers in Australia may be potentially occupationally exposed to carcinogens (Appendix 4).

Developing these estimates has involved a number of assumptions since Australian data are not available for a number of critical values. These estimates should therefore be considered only a rough estimate of the true number of Australian workers exposed to occupational carcinogens. Again, these estimates do not take into account what measures may be in place to control occupational exposure.

In spite of this, these estimates and the estimates on the proportion of cancers due to work in Australia are a useful indicator of the importance of occupational cancer in Australia. The following section will consider some known occupational carcinogens and deal with some of these in an Australian context.

5. OCCUPATIONAL CARCINOGENS

5.1 Occupational carcinogens

Appendix 3 of this report provides an overview of the IARC system for classification of carcinogens. Based on IARC evaluations, supplemented by other data, Siemiatycki et al. (2004) summarised current knowledge on occupational carcinogens, the occupations and industries in which they are found, and their target organs. The authors examined agents and exposure circumstances as categorised by IARC (see Appendix 3B) as Group 1; Group 2A and Group 2B.

They then reviewed each one and identified those that are occupational carcinogens based on whether there are, or have been, significant numbers of workers exposed to the substance at significant levels. They excluded from their list some classes of agents (such as hormones and pharmaceuticals) on the grounds that occupational exposures are rare or very infrequent or at very low doses. Also excluded were viruses, such as human immunodeficiency virus (HIV), hepatitis B and C although health care workers may be at risk.

With these criteria they derived the following lists of occupational carcinogens:

- 28 definite human occupational carcinogens (IARC group 1);
- 27 probable human occupational carcinogens (IARC group 2A);
- 113 possible human occupational carcinogens (IARC group 2B); and
- 18 occupations and industries that possibly, probably or definitely entail excess risk of cancer (IARC groups 1, 2A and 2B).

Their complete tables can be found in their paper, but for our purposes we will deal with some of the definite human occupational carcinogens and occupations and industries at dot points 1 and 4 above and their tables for these are shown in Table 1 (Section 5.2).

5.2 Industries, occupations and exposures

As mentioned elsewhere in this report, the nature and extent of exposure to and use of human carcinogens in Australian workplaces is not well characterised. The industries and occupations listed in Table1 below are known to exist in Australia. However, a substantial body of work is required to give the Australian workplace profile, that is:

- identify the work processes involved that give rise to exposures;
- what measures are in place to control worker exposure;
- estimates of worker population size;
- what is the progress of technological advancement in identified industries to eliminate carcinogenic substances or implement work processes that do not give rise to exposure to carcinogens; and
- evaluation of Australian cancer data for work-relatedness.

These profiles can then better inform prevention strategies that focus on carcinogens in the workplace in risk areas. Substances would need to be looked at in priority order.

A sample profile on wood workers and wood dust exposure is under development in the NOHSC Office and is shown at Appendix 5.

Table 1.1 (Reproduced from Siemiatycki et al. (2004)

Table 3. Substances and mixtures that have been evaluated by IARC as definite (group 1) human carcinogens and that are occupational exposures.

Substance or mixture	Occupation or industry in which the substance is found[a]	*IARC Monograph* volume (year)[b]	Human evidence[c]	Animal evidence[c]	Site(s)
Physical agents					
Ionizing radiation and sources thereof, including, notably, X rays, γ rays, neutrons, and radon gas	Radiologists; technologists; nuclear workers; radium-dial painters; underground miners; plutonium workers; cleanup workers following nuclear accidents; aircraft crew	Vol. 75 (2000a) Vol. 78 (2001a)	Sufficient	Sufficient	Bone[d] Leukemia[d] Lung[d] Liver[d] Thyroid[d] Others[d]
Solar radiation	Outdoor workers	Vol. 55 (1992b)	Sufficient	Sufficient	Melanoma[d] Skin[d]
Respirable dusts and fibers					
Asbestos	Mining and milling; by-product manufacture; insulating; shipyard workers; sheet-metal workers; asbestos cement industry	Suppl. 7 (1987)	Sufficient	Sufficient	Lung[d] Mesothelioma[d] Larynx[e] GI tract[e]
Erionite	Waste treatment; sewage; agricultural waste; air pollution control systems; cement aggregates; building materials	Suppl. 7 (1987)	Sufficient	Sufficient	Mesothelioma[d]
Silica, crystalline	Granite and stone industries; ceramics, glass, and related industries; foundries and metallurgical industries; abrasives; construction; farming	Vol. 68 (1997b)	Sufficient	Sufficient	Lung[d]
Talc containing asbestiform fibers	Manufacture of pottery, paper, paint, and cosmetics	Suppl. 7 (1987)	Sufficient	Inadequate	Lung[d] Mesothelioma[d]
Wood dust	Logging and sawmill workers; pulp and paper and paperboard industry; woodworking trades (e.g., furniture industries, cabinetmaking, carpentry and construction); used as filler in plastic and linoleum production	Vol. 62 (1995b)	Sufficient	Inadequate	Nasal cavities and paranasal sinuses[d]
Metals and metal compounds					
Arsenic and arsenic compounds	Nonferrous metal smelting; production, packaging, and use of arsenic-containing pesticides; sheep dip manufacture; wool fiber production; mining of ores containing arsenic	Suppl. 7 (1987)	Sufficient	Limited	Skin[d] Lung[d] Liver (angiosarcoma)[e]
Beryllium	Beryllium extraction and processing; aircraft and aerospace industries; electronics and nuclear industries; jewelers	Vol. 58 (1993a)	Sufficient	Sufficient	Lung[d]
Cadmium and cadmium compounds	Cadmium-smelter workers; battery production workers; cadmium-copper alloy workers; dyes and pigments production; electroplating processes	Vol. 58 (1993a)	Sufficient	Sufficient	Lung[d]
Chromium compounds, hexavalent	Chromate production plants; dyes and pigments; plating and engraving; chromium ferro-alloy production; stainless-steel welding; in wood preservatives; leather tanning; water treatment; inks; photography; lithography; drilling muds; synthetic perfumes; pyrotechnics; corrosion resistance	Vol. 49 (1990a)	Sufficient	Sufficient	Lung[d] Nasal sinuses[e]
Selected nickel compounds, including combinations of nickel oxides and sulfides in the nickel refining industry	Nickel refining and smelting; welding	Vol. 49 (1990a)	Sufficient	Sufficient	Lung[d] Nasal cavity and sinuses[d]
Wood and fossil fuels and their by-products					
Benzene	Production; solvents in the shoe production industry; chemical, pharmaceutical, and rubber industries; printing industry (rotogravure plants, bindery departments); gasoline additive	Suppl. 7 (1987)	Sufficient	Limited	Leukemia[d]
Coal tars and pitches	Production of refined chemicals and coal tar products (patent-fuel); coke production; coal gasification; aluminum production; foundries; road paving and construction (roofers and slaters)	Suppl. 7 (1987)	Sufficient	Sufficient	Skin[d] Lung[e] Bladder[e]

Continued next page

Table 1.2 (Reproduced from Siemiatycki et al. (2004)

Table 3. Continued

Substance or mixture	Occupation or industry in which the substance is found[a]	*IARC Monograph* volume (year)[b]	Human evidence[c]	Animal evidence[c]	Site(s)
Mineral oils, untreated and mildly treated	Production; used as lubricant by metal workers, machinists, engineers; printing industry (ink formulation); used in cosmetics, medicinal and pharmaceutical preparations	Suppl. 7 (1987)	Sufficient	Inadequate	Skin[d] Bladder[e] Lung[e] Nasal sinuses[e]
Shale oils or shale-derived lubricants	Mining and processing; used as fuels or chemical-plant feedstocks; lubricant in cotton textile industry	Suppl. 7 (1987)	Sufficient	Sufficient	Skin[d]
Soots	Chimney sweeps; heating-unit service personnel; brick masons and helpers; building demolition workers; insulators; firefighters; metallurgical workers; work involving burning of organic materials	Vol. 35 (1985)	Sufficient	Inadequate	Skin[d] Lung[d] Esophagus[e]
Monomers					
Vinyl chloride	Production; production of polyvinyl chloride and co-polymers; refrigerant before 1974; extraction solvent; in aerosol propellants	Suppl. 7 (1987)	Sufficient	Sufficient	Liver (angiosarcoma)[d] Liver (hepato-cellular)[e]
Intermediates in plastics and rubber manufacturing					
Bis(chloromethyl) ether and chloromethyl methyl ether (technical grade)	Production; chemical intermediate; alkylating agent; laboratory reagent; plastic manufacturing; ion-exchange resins and polymers	Suppl. 7 (1987)	Sufficient	Sufficient	Lung (oat cell)[d]
Aromatic amine dyes					
4-Aminobiphenyl	Production; dyestuffs and pigment manufacture	Suppl. 7 (1987)	Sufficient	Sufficient	Bladder[d]
Benzidine	Production; dyestuffs and pigment manufacture	Suppl. 7 (1987)	Sufficient	Sufficient	Bladder[d]
2-Naphthylamine	Production; dyestuffs and pigment manufacture	Suppl. 7 (1987)	Sufficient	Sufficient	Bladder[d]
Pesticides					
Ethylene oxide	Production; chemical industry; sterilizing agent (hospitals, spice fumigation)	Vol. 60 (1994)	Limited	Sufficient	Leukemia[d]
2,3,7,8-Tetrachlorodibenzo-*para*-dioxin (TCDD)	Production; use of chlorophenols and chlorophenoxy herbicides; waste incineration; PCB production; pulp and paper bleaching	Vol. 69 (1997a)	Limited	Sufficient	All sites combined[d] Lung[e] Non-Hodgkin lymphoma[e] Sarcoma[e]
Others					
Aflatoxin	Feed production industry; workers loading and unloading cargo; rice and maize processing	Vol. 82 (2002b)	Sufficient	Sufficient	Liver[d]
Involuntary (passive) smoking	Workers in bars and restaurants; office workers	Vol. 83 (2004)	Sufficient	Sufficient	Lung[d]
Mustard gas	Production; used in research laboratories; military personnel	Suppl. 7 (1987)	Sufficient	Limited	Larynx[d] Lung[e] Pharynx[e]
Strong inorganic-acid mists containing sulfuric acid	Pickling operations; steel industry; petrochemical industry; phosphate acid fertilizer manufacturing	Vol. 54 (1992a)	Sufficient	Not available	Larynx[d] Lung[e]

[a]Not necessarily an exhaustive list of occupations/industries in which this agent is found; not all workers in these occupations/industries are exposed. The term "production" is used to indicate that this substance is man-made and that workers may be exposed in the production process. [b]Most recent IARC evaluation; for those referenced to Supplement 7 (IARC 1987), it is possible that the 1987 review was quite perfunctory and that the essential evidence was cumulated at an earlier date. [c]As judged by the IARC working group; we added the notation "not available" to signify those substances for which there was no evidence at all. [d]We judged that evidence for an association with this site was strong. [e]We judged that evidence was suggestive.

Table 1.3 (Reproduced from Siemiatycki et al. (2004)

Table 6. Occupations or industries that have been evaluated by IARC as definitely (group 1), probably (group 2A), or possibly (group 2B) entailing excess risk of cancer among workers.

Occupation or industry	Suspected substance	*IARC Monograph* volume (year)[a]	Group	Site(s)
Aluminum production	Pitch volatiles; aromatic amines	Suppl. 7 (1987)	1	Lung,[b] bladder[b]
Auramine manufacture	2-Naphthylamine; auramine; other chemicals; pigments	Suppl. 7 (1987)	1	Bladder[b]
Boot and shoe manufacture and repair	Leather dust; benzene and other solvents	Suppl. 7 (1987)	1	Leukemia,[b] nose,[b] paranasal sinuses,[b] bladder[c]
Carpentry and joinery	Wood dust	Suppl. 7 (1987)	2B	
Coal gasification	Coal tar; coal-tar fumes; PAHs	Vol. 34 (1984)	1	Skin (including scrotum),[b] bladder,[b] lung[b]
Coke production	Coal-tar fumes	Suppl. 7 (1987)	1	Skin (scrotum),[b] lung,[b] bladder,[c] kidney[c]
Dry cleaning	Solvents and chemicals used in "spotting"	Vol. 63 (1995a)	2B	
Furniture and cabinet making	Wood dust	Suppl. 7 (1987)	1	Nose and sinonasal cavities[b]
Hairdressers and barbers	Dyes (aromatic amines, amino-phenols with hydrogen peroxide); solvents; propellants; aerosols	Vol. 57 (1993b)	2A	Bladder,[c] lung,[c] non-Hodgkin lymphoma,[c] ovary[c]
Hematite mining, underground, with radon exposure	Radon daughters; silica	Suppl. 7 (1987)	1	Lung[b]
Iron and steel founding	PAHs; silica; metal fumes; formaldehyde	Suppl. 7 (1987)	1	Lung[b]
Isopropanol manufacture, strong-acid process	Diisopropyl sulfate; isopropyl oils; sulfuric acid	Suppl. 7 (1987)	1	Paranasal sinuses,[b] larynx,[b] lung[c]
Magenta manufacture	Magenta; *ortho*-toluidine; 4,4´-methylene bis(2-methylaniline); *ortho*-nitrotoluene	Vol. 57 (1993b)	1	Bladder[b]
Painters		Vol. 47 (1989c)	1	Lung,[b] bladder,[c] stomach[c]
Petroleum refining	PAHs	Vol. 45 (1989b)	2A	Bladder,[c] brain,[c] leukemia[c]
Printing processes	Solvents; inks	Vol. 65 (1996)	2B	
Production of art glass, glass containers, and pressed ware	Lead; arsenic; antimony oxides; silica; asbestos; other metal oxides; PAHs	Vol. 58 (1993a)	2A	Lung[c]
Rubber industry	Aromatic amines; solvents	Suppl. 7 (1987)	1	Bladder,[b] stomach,[c] larynx,[c] leukemia,[c] lung[c]
Textile manufacturing industry	Textile dust in manufacturing process; dyes and solvents in dyeing and printing operations	Vol. 48 (1990b)	2B	

[a]Most recent IARC evaluation; for those referenced as Supplement 7 (IARC 1987), it is possible that the 1987 review was quite perfunctory and that the essential evidence was cumulated at an earlier date. [b]We judged that the evidence for an association with this site was strong. [c]We judged that the evidence was suggestive.

6. PREVENTION ACTIVITIES

The task of reviewing prevention activities is complicated because there are many different substances which do or may cause cancer and a full review of all activities addressing each possible substance would be prohibitive. In addition, there is no easily accessible information on prevention activities which have worked (or not). In this section we will summarize the types of activity aimed at preventing cancer and give international and national examples of each type of activity categorized according to the hierarchy of control.

6.1 Elimination

Elimination of the substance is the most effective means of preventing exposure. The mechanisms may include banning of production (or mining), import, sales or use. Asbestos is the most obvious example of where prohibition is being used to control the effects. All types of asbestos will be banned throughout the EU by 2005, and Argentina, Chile, Croatia and Saudi Arabia have also banned asbestos. In the United States the EPA published the Asbestos: Manufacture, Importation, Processing, and Distribution in Commerce Prohibitions; Final Rule (40 CFR Part 763, Subpart I). The rule will eventually ban about 94 percent of the asbestos used in the U.S. (based on 1985 estimates). Asbestos is still mined, imported and used in many developing countries.

In Australia, all forms of asbestos are subject to prohibition. For amosite, crocidolite, actinolite, anthophyllite, and tremolite asbestos, the ban extends to all use except for removal and disposal and in situations where

the asbestos occurs naturally and is not used for any new application. For chrysotile asbestos, there is a limited list of exempted circumstances in which it may still be used. These are: bona fide research or analysis; when handled for storage awaiting disposal; for removal or disposal; or where encountered during non-asbestos mining.

6.2 Substitution

Substitution involves replacing a carcinogenic substance with one that is not carcinogenic, or is less carcinogenic. Methylene chloride is a probable carcinogen (classified as 2B by IARC) which is used mainly as a solvent, in paint removers, degreasers and aerosol products, and in the manufacture of foam polymers. In the US, a survey of some previously large users of this chemical found that many of the companies had moved to substitute methylene chloride with aqueous methods of cleaning (Roelofs and Ellenbecker, 2003). The overall impact of this substitution was deemed beneficial from the standpoint of both worker health and production.

6.3 Engineering controls

Engineering controls may be introduced in order to reduce exposure of workers to carcinogens, for example, ventilation, enclosure or partial enclosure. Other methods include isolation in which the carcinogen is separated from workers by distance or by using robotics to undertake the tasks involving high exposure.

For example, in a foundry in the US, an inspection by the regulating body disclosed high levels of crystalline silica (Irwin, 2003). The foundry made significant changes to the engineering controls in order to comply with the requirements of the regulators. This necessitated several weeks' closure, but the improved workplace was likely to have delivered significant long-term savings.

In Australia, monitoring of chrysotile asbestos exposure of car mechanics found very low levels of exposure (Yeung et al., 1999). The authors attributed this to the wet cleaning or aerosol spray methods used to replace the traditional compressed air jet cleaning.

6.4 Safe work procedures

Safe working procedures refers to establishing procedures in the workplaces in order to reduce exposure to carcinogens. Examples include limiting the times at which outdoor work is performed, or ensuring that dust is dampened down.

For example, in the US construction industry, silica dust exposures were reduced about 3-fold if wet dust suppression was used, and about 6-fold if ventilated cabs were used (Rappaport et al., 2003).

6.5 Personal protective equipment

Use of personal protective equipment (PPE) is the least efficient way of controlling hazardous exposures. It means that the emphasis is entirely on the worker to comply with the use of PPE, regular maintenance and fitting checks of PPE with each worker are required, and the PPE may be uncomfortable or hot, or may make tasks difficult or dangerous. However, PPE such as respirators, gloves or overalls are used in many industries to decrease personal exposure to carcinogens. Similarly, the use of hats, long sleeved shirts and sunscreens is advocated as a way to avoid occupational sun exposure (Woolley et al., 2002).

6.6 Education

As occupational cancers can be difficult to identify and control, their prevention is likely to be more effective by also implementing complementary actions such as education towards employers, workers, and physicians, research in order to identify new hazards and their impact on workers' health and review of workplace safety and standards on an ongoing basis. Ideally, this process is best informed by a comprehensive national surveillance scheme to assist in effective targeting of preventive measures.

7. NATIONAL PREVENTION ACTIVITY

National actions which will ultimately contribute to the reduction of exposure to occupational cancer risk factors are occurring. These include the creating or updating hazardous substances and dangerous goods regulations, standards and codes of practice, improving . Examples of ongoing or recent actions include:

- Work to contribute to the globally harmonised system of classification and labelling of chemicals (GHS), and its implementation in Australia and Overseas.
- Work under the National Dangerous Goods Framework which is designed to enable a nationally consistent regulatory approach to the control of workplace dangerous goods.
- Implementation of the NOHSC National Standard for the Storage and Handling of Workplace Dangerous Goods [NOHSC:1015(2001)].
- Recent amendments to the Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(1999)].
- New regulations have been or are being developed in most States and Territories to prohibit smoking in licensed premises and other public places.
- Recent amendments to the exposure standards for crystalline silica.
- Declaration of a revised Code of Practice for the Safe Removal of Asbestos 2nd Edition [NOHSC: 2002 (2005)].

- Declaration of a new Code of Practice for the Management and Control of Asbestos in Workplaces [NOHSC:2018 (2005)].
- Release of the Hazardous Substances Information System and internet database that allows people to find information on hazardous substances that have been classified in accordance with the *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(2004] 3rd Edition and/or have National Exposure Standards declared under the NOHSC *Adopted National Exposure Standards for Atmospheric Contaminants in the Occupational Environment* [NOHSC:1003(1995)] or subsequent updates. http://www.nohsc.gov.au/applications/hsis/
- Proposed amendments to the Adopted National Exposure Standards for Atmospheric Contaminants in Occupational Environment [NOHSC:1003(1995)]

For information about actions being undertaken by agencies within Australia, readers are directed to the following websites for details about current programs.

- Australian Department of Health and Ageing. http://www.health.gov.au/
- NSW WorkCover Authority http://www.workcover.nsw.gov.au/default.htm
- Victorian WorkCover Authority http://www.workcover.vic.gov.au/dir090/vwa/home.nsf
- WorkSafe Western Australia http://www.safetyline.wa.gov.au/
- South Australian WorkCover Authority http://www.workcover.com/ or Workplace Services South Australia http://www.eric.sa.gov.au/home.jsp
- Queensland Division of Workplace Health and Safety http://www.whs.qld.gov.au/
- Workplace Standards Tasmania http://www.wst.tas.gov.au/node/WST.htm
- Northern Territory WorkSafe http://www.nt.gov.au/deet/worksafe/
- ACT WorkCover http://www.workcover.act.gov.au/
- Comcare http://www.comcare.gov.au/
- Australian Council of Trade Unions http://www.actu.asn.au/
- Australian Chamber of Commerce and Industry. http://www.acci.asn.au/
- Australasian Faculty of Occupational Medicine (AFOM) http://www.racp.edu.au/afom/

8. CONCLUSIONS

This report has provided an estimate of the size of the problem of occupational cancers in Australia. Accurate estimates are complicated as currently there are no routinely collected Australian statistics which give information on the real number of occupationally caused cancers. Previous calculations of the number of cases of cancer caused by occupation have been obvious underestimates because of gaps in our knowledge of which substances cause cancer and what the risks of each type of cancer are from each of these substances. And a fundamental problem is the long time delay between exposure and development of cancer which means it is not possible to use the number of cancers as a measure of how successful any prevention initiative has been.

This review has found that approximately 13.8% of cancer deaths in males and 2.2% of cancer deaths in females are caused by occupational exposures. This means that, every year, about 5000 invasive cancers and about 34 000 non-melanoma skin cancers are being caused by occupational exposures in Australia. This estimate was based on the most recent and most accurate evidence available but still required a number of assumptions, so should be considered to be approximate until better data are available.

Approximately 1.5 million Australian workers may currently be exposed to occupational carcinogens. Again this estimate has involved a number of estimations and assumptions and should be considered to be approximate.

The number of workers exposed and the number of cancers are not necessarily similar. Because cancer takes a long time to develop, although many substances are no longer used, a large number of workers will have been exposed to these substances in the past, and many current and future cancers will be due to these exposures.

When we consider the prevention of occupational cancer there are two main themes: we need to prevent or decrease the exposure of workers to established carcinogens; and we need to develop systems such that new carcinogens are identified and classified.

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APPENDIX 1. INTERNATIONAL AND AUSTRALIAN ESTIMATES OF OCCUPATIONAL CANCER

1. International estimates of occupational cancer

The most often quoted statistic concerning the number of cancers due to occupational factors is one which was estimated by Doll and Peto in 1981 (Doll and Peto, 1991). They concluded that 4% of cancer deaths were due to occupational factors. However, although many of the assumptions underlying the methods would have been reasonable in 1981, they are somewhat doubtful now.

Exposures and working conditions used for the calculations were those in the 1940s to 1970s and are quite different to those encountered today. In addition, data on cancer deaths in the US in 1978 was used to weight the results. The mix of cancers in Australia today (or in fact in the US today) is quite different to that of 25 years ago, with more melanomas and fewer stomach cancers and lung cancers. Most importantly, Doll and Peto only included cancers occurring before the age of 65. Because cancer has such a long latency, this will exclude a large number of cancers which may be occupationally caused.

The method of categorizing cancer sites according to likelihood of being occupationally caused was also quite unusual. First, 15 sites of cancer were stated as "not known to be produced by occupational hazards" and no cases were attributed to occupational causes. These sites included melanoma and lip cancer, as Doll and Peto felt that "exposure to UV light associated with work in the open air" should be ignored. In addition, they included breast cancer, which has been linked with a number of occupations, although the association with occupational physical activity is probably the strongest.

For a further 10 sites of cancer 1% of male cancers and 0.5% of female cancers were considered to be occupationally attributable based on knowledge in 1981. This group includes cancers for which there is now reasonable evidence of an occupational link such as colorectal cancer (for which occupational physical activity is thought to be preventive), non-Hodgkin lymphoma (which has been consistently linked with pesticides and solvents), kidney cancer (which has been associated with solvents and metals), and pancreatic cancer (which has been associated with pesticides, solvents and rubber chemicals).

Doll and Peto then assumed proportions of the remaining 11 sites based on "the crude and unreliable basis of our interpretation of the literature and clinical impression". This included 1% of prostate cancers thought to be due to cadmium exposure, and 2% of laryngeal cancers in mustard gas manufacturers and perhaps in nickel refiners. Perhaps the biggest surprise is in the categorization of mesotheliomas: occupational causes were assumed for only 15% of male and 5% of female peritoneal cancers, and only 25% of male and 5% of female pleural cancers. The biggest number of occupational cancers came from lung cancer, of which 15% of

male cancers and 5% of female cancers were considered to be occupationally induced.

Doll and Peto state in their review that is “odd that despite the passionate debates that have taken place about the likely magnitude of the number of US cancer deaths that are or will be attributable to occupation, no routine system has been adopted in the US for generating reliable information.” Unfortunately, this statement remains as true today as it was 23 years ago. They suggested that a large case-control study should be instigated to examine the effect of occupations on cancer risk.

Such a study was the Montreal Case-Control Study which ran from 1979 to 1985 (Siemiatycki, 1995). Cases were subjects with one of any of 19 different sites of cancer (n = 3 730) and controls were randomly selected from the population (n = 533). A second set of controls was chosen from subjects in the study with cancer other than the one being studied. Extremely detailed occupational assessment was performed examining occupational exposure to nearly 300 different substances, including known carcinogens and chemicals for which there was no evidence as to carcinogenicity. In 1995 (Siemiatycki, 1995), the investigators used their data to estimate the population attributable risk percent due to occupational exposures for 9 different sites of cancer under two different assumptions: first if only substances with strong evidence of carcinogenicity were used, and second if any statistically significant exposures in the Montreal study were accepted (Table A1.1). Even if we consider these second set of proportions to be at the high end of plausible estimates, the gap between the results from the two methods is startling. This suggests that previous estimates have been vastly underestimated because of our limited knowledge of the number of chemicals which are carcinogenic, and the proportions of the population exposed to these chemicals at work.

Table A1.1 Proportion of cancers of major sites occupationally caused using two different assumptions (from (Siemiatycki, 1995))

Cancer site	Recognized carcinogens	Significant findings
Oesophagus	3.5	20.4
Stomach	4.0	14.1
Colon	0.4	3.4
Rectum	0	21.8
Pancreas	0	20.6
Lung	8.0	20.3
Prostate	0.2	9.9
Bladder	1.2	10.8
Kidney	0	20.8

In Canada, Kraut reviewed the routine statistics available on occupationally caused diseases (such as compensation statistics) and concluded that they were underestimated by several orders of magnitude(Kraut, 1994). He felt that Cullen's summary of literature estimates of between 4 and 10% of cancers was closer to the truth (Cullen et al., 1990).

In the US, Leigh and colleagues assumed all mesotheliomas and 6% to 10% of other cancer deaths were due to occupational causes (Leigh et al., 1997). This was based on an examination of the attributable risk of lung and bladder cancers from case-control studies which produced an estimate of 3-4% of all cancer deaths, as well as a qualitative synthesis of other evidence suggesting that the estimate should be higher. They assumed the same proportion of new cancer cases were occupationally caused as deaths, and concluded that 6-10% of new cancers were occupationally caused.

Another issue in calculating the proportion of cancers attributable to occupation is the fact that many blue-collar workers, who have high occupational exposures, also smoke. Axelson developed a method of adjusting for differing smoking rates in different groups. His method did not rely on identifying carcinogens, but assumed that any cancers not due to smoking were due to occupation (Axelson, 2002). Using this method, he calculated that 24% of lung cancers in economically active men were due to occupational exposures.

In the US, Steenland and co-workers used relative risks for carcinogens classified by the International Agency for Research in Cancer as carcinogenic (see Appendix 3B) and national occupational surveys from the 1980s to determine the percent of people exposed to these carcinogens (Steenland et al., 2003). For lung cancer and bladder cancer, they also used carcinogens classified as Probably Carcinogenic by IARC. They did not include UV radiation. Their final estimate was that 2.4-4.8%

of cancer deaths were occupationally related, 0.8-1% among females and 3.3-7.3% among males.

Another attempt to estimate the total burden of fatalities from occupational cancer was undertaken in Finland in 2001 by Nurminen and Karjalainen (Nurminen and Karjalainen, 2001). The authors used census data on occupation and a database of occupational hygiene measurements to determine numbers of workers exposed to 74 chemical physical and microbiological agents. They then calculated the attributable fraction using risk ratios for these 74 agents from methodologically strong studies. Points to note in these estimations were that they did not include any estimates of risk from sedentary occupations and that their estimate of the proportion of melanomas and non-melanoma skin cancers was based on the assumption that only occupations with similar sun exposure to Finnish seafarers had enough sun exposure to cause melanoma. Their study suggests that 13.8% of cancer deaths in males and 2.2% of cancer deaths in females were occupationally caused. Their site-specific estimates are outlined in Table A1.2.

Table A1.2 Percent of cancers of different sites attributed to occupation (from (Nurminen and Karjalainen, 2001))

	% of cancers attributed to occupation	
Cancer site	**Male**	**Female**
Oral cavity	1.0	0.3
Pharynx	2.0	0.5
Oesophagus	6.4	0.2
Stomach	10.3	5.4
Colon	5.6	0.0
Rectum	3.1	0.1
Liver	3.5	5.3
Gallbladder	0.2	0.4
Pancreas	13.4	3.5
Nose and nasal sinuses	24.0	6.7
Larynx	9.3	0.5
Bronchus and lung	29.0	5.3
Bone	0.6	0.6
Melanoma	4.3	0.4
Non melanoma skin cancer	13.1	3.8
Mesothelioma	90.0	25.0
Breast	-	1.7
Cervix	-	5.9
Corpus uteri	-	1.1
Ovary	-	2.1
Prostate	6.0	-
Kidney	4.7	0.8
Bladder	14.2	0.7
Brain	10.6	1.3
Hodgkin's disease	3.9	0
Non-Hodgkin lymphoma	13.5	3.1
Leukaemia	18.5	2.5

In New Zealand, Pearce and colleagues reviewed the literature thoroughly and determined that the Nurminen estimates were the most appropriate

to use to calculate the burden of occupational cancer (Pearce et al., 2004a). They estimated that 5-9% of cancer deaths in men over 30 years old and 0.5-2% in women over 30 years old were occupationally related. The proportions of new cancer cases were similar.

Recently, the World Health Organization has updated their international estimates of the burden of disease due to different causes (Concha-Barrientos et al., 2004). The methods were very complex, but for occupational cancers, only lung cancer, leukaemia and mesothelioma were examined. They found 9% of lung cancer and 2% of leukaemia worldwide was caused by occupational factors (Driscoll et al., In press). For mesothelioma, they used absolute risk estimates obtained from the literature, and applied these to estimates of asbestos exposure. This study was limited by having to use data from all countries, so if data were missing from, say, developing countries, that cause could not be used. The authors felt their estimates were a considerable underestimate.

A different approach to all the above was taken by Pearce and colleagues in New Zealand (Pearce et al., 2004b). They established a panel of experts who reviewed all male cases of three cancers (non-Hodgkin lymphoma, bladder cancer and leukaemia). A full occupational history was taken from each case and then the panel reviewed the history in the light of internationally published literature to see if it was likely that the cancer had been caused by occupational exposures. Using this method, they found that 23% of bladder cancers were likely to be occupationally related. In comparison, there had been no notifications of bladder cancer to the compensation scheme run by the occupational health and safety authorities.

1B Australian estimates of occupational cancer

Given the considerable effort and expense involved in estimating the burden of occupational cancer, it is not surprising that few groups have attempted to calculate estimates for Australia.

Winder and Lewis applied the Doll and Peto estimates for individual cancers to the Australian cancer rates (Winder and Lewis, 1991). Not surprisingly, they concluded that a similar proportion (3.8%) of Australian cancer deaths were due to occupational exposures. The authors felt this was an underestimate particularly for lung cancer, mesothelioma, melanoma and cancers caused by ionizing radiation.

Kerr and colleagues produced the most comprehensive estimate to date of morbidity and mortality in Australia resulting from work related cancers (Kerr et al., 1996). They used the 1981 estimates from the Doll and Peto paper with some minor changes in that the proportions of mesothelioma deaths attributable to work were increased substantially. Their estimate of 3.2% of all cancer deaths attributable to occupation was similar to the attributable fraction estimates of Doll and Peto and Winder and Lewis (Doll and Peto, 1991, Winder and Lewis, 1991) .

The Australian Institute of Health and Welfare also used the Kerr report estimates to determine that occupational causes could be responsible for 2.1% of cancer deaths (Mathers et al., 1999).

1C Routinely collected statistics in Australia

Some estimates are also available from routinely collected statistics in Australia. These are thought to be considerable underestimates due to the previously discussed problems with attributing cancer to an occupational exposure which may have occurred many years or decades previously.

The 2001 National Health Survey asked about long term medical conditions which were work-related. They estimated that there were 6 500 prevalent cancers in 2001 which is about 2% of all cancers. There is considerable uncertainty about these estimates (Australian Bureau of Statistics, 2002) as people in hospitals and hospices were excluded, and prevalence is not usually a useful measure for cancer due to the relatively high case-fatality.

The National Data Set for Compensation-based Statistics (NDS) collects data for injuries or diseases for workers' compensation claims lodged with Commonwealth, State and Territory workers' compensation schemes. Over three years (2001-2003) the NDS recorded 403 claims for neoplasms, including 151 for mesotheliomas. The most common causes of compensated claims during this period were sun exposure (22%) and asbestos (21%). 41% of claims on the database did not have any known agency.

APPENDIX 2. DETERMINING THE RELATIVE RISK, NUMBER OF PEOPLE EXPOSED AND NUMBERS OF CASES

2A. Determining the relative risk of cancer

The current theory of carcinogenesis suggests that cancer is due to a combination of factors. The effect of a specific factor can be large or can be small and can differ by cancer site. For example, occupational exposure to asbestos increases the risk of mesothelioma much more than it does colon cancer.

So an accurate determination of the relative risk of cancer due to one or more occupational causes would need to consider each chemical and each type of cancer. This is a huge amount of work. Most previous studies have avoided doing this in a comprehensive way. For example, Doll and Peto did not use relative risk estimations, but instead estimated the proportion of deaths due to each type of cancer deaths caused by occupational causes based on previous estimates from the 1970s and their own opinions (Doll and Peto, 1991). Morrell (Morrell et al., 1998) used three previous studies: in Israel in 1992; in the US in 1989; and the Doll and Peto study from 1981. Steenland used similar methods to Doll and Peto, but included a greater range of substances and cancers, and extracted the data in a more systematic way (Steenland et al., 2003). However the authors only examined 10 sites of cancer.

The Finnish review is the only recent review in which original studies were reviewed to provide relative risks for each cancer/substance combination (Nurminen and Karjalainen, 2001). The methodological quality of the studies was assessed and then the best of the studies were used to identify the most plausible relative risk.

2B. Determining the number of people exposed at work

Studies tend to look at the issue of occupation and cancer in one of two ways: either by determining the risk of cancer for workers in a particular occupation; or by determining the risk of cancer for workers exposed to particular chemicals. The risk of cancer is obviously related to the chemical exposure, not the occupational group

One important issue to be considered is that not all people within an occupational group have the same chemical exposures. Therefore when doing studies of an occupation, the result is diluted by having a combined exposed and unexposed group. This results in lower risk estimates than when a pure exposed group is used.

Another issue is that by using occupational groupings, the number of people exposed to a chemical is split across a number of different groups. For example, some people in each of the occupations of shipbuilder, wharf worker and mechanic will have exposure to asbestos. If we look at each occupation separately we may only have a few cases of cancer and the

effects of asbestos exposure will be difficult to tease out. However, if we combine the groups, we may have different proportions of the workers exposed in each occupation so again we may dilute the effect.

With these two disadvantages it would seem that it would be more scientifically valid to determine risks of cancer for workers exposed to particular chemicals. However, this route also poses difficulties as it is much more difficult to obtain information on the chemicals to which individuals are exposed, compared to information on occupational grouping.

Another layer of complication is introduced because of the way occupational data are coded. Some codes include subjects in a wide variety of occupations and it is difficult to know whether it is more appropriate to include all occupations within that code as exposed or unexposed. For example, among doctors, ionizing radiation is a common exposure only for radiologists, some surgeons and some gastroenterologists. If we wanted to determine how many people in Australia were exposed to occupational ionizing radiation, would we include all doctors as exposed, when many aren't exposed at all? Or would we exclude all doctors, thus also excluding those who have significant potential for exposure?

2C. Determining the number of cancers

Cancer registries are databases on which are recorded cases of cancer as they are diagnosed. Cancer registration is an important and fundamental tool in cancer monitoring. In Australia, each of the eight states and territories maintain a cancer registry of new cases of malignant cancer. Any health professional who diagnoses cancer (usually the pathologist) is required by law to notify their diagnosis to the state cancer registry. Within the registry, the data are cross-checked to determine if the cancer is new, and details including identifying information on the person as well as the type and site of cancer and date of diagnosis are recorded. Except for Tasmania, Australian cancer registries do not collect data on non-melanoma skin cancer.

In order to co-ordinate cancer statistics on a national basis, the National Cancer Statistics Clearing House (NCSCH) was established in 1986 at the Australian Institute of Health and Welfare. The NCSCH receives data from individual State and Territory cancer registries on cancer diagnosed in residents of Australia. The aim of the NCSCH is to foster the development and dissemination of national cancer statistics for Australia and specifically to:

- enable computation and publication of national statistics on cancer;
- allow tracking of interstate movement of cancer cases via record linkage;

- facilitate exchange of scientific and technical information between cancer registries and promote standardisation in the collection and classification of cancer data; and
- facilitate cancer research both nationally and internationally.

Cancer registries in Australia do not contain information on occupation. The recommended Clinical Cancer Core Data Set and Data Dictionary does not even mention occupation (National Cancer Control Initiative, 2004). Any information on occupation in other routine data sets such as hospital morbidity data is incomplete, unsystematically collected and coded, and includes large proportions of people classified as retired or on illness benefits because of their disease. So while the number of cancers is relatively easy to find, obtaining information on the occupations of those with cancer is less straightforward.

An important issue in calculating the burden of occupational cancer is whether the appropriate measure of this burden is the number of new cases of cancer or the number of deaths from cancer. While death is the most serious consequence of a disease, and thus contributes the most "impact" to the burden of disease, the diagnosis of a cancer from which death does not occur also has significant consequences for the individual concerned as well as to society. A diagnosis of cancer results in the person undergoing various treatments which are often complex, time-consuming and may have significant side effects. In addition, the whole process involves considerable worry and stress for the individual and the family. Financial costs to society include not only the cost of medical treatments, but also the time off work for the individual and their carers. In some cases, where the cancer is usually rapidly fatal (such as mesothelioma) the number of deaths is very similar to the number of new cancers. In cancers which are rarely fatal, such as non-melanoma skin cancer, the numbers are very different. All previous studies of the burden of occupational cancer have used deaths from cancer rather than new cases.

Table A2.1: Calculations of number of cases of cancer caused by occupation.

MALES

Cancer site	% of cancers attributable to occupation	No. of cancers 2000	No. of cancers occupationally caused
Oral cavity	1	936	9.36
Larynx	2	286	5.72
Oesophagus	6.4	711	45.504
Stomach	10.3	1267	130.501
Colon	5.6	4739	265.384
Rectum	3.1	2020	62.62
Liver	3.5	526	18.41
Gallbladder	0.2	243	0.486
Pancreas	13.4	912	122.208
Nose and nasal sinuses	24	88	21.12
Larynx	9.3	481	44.733
Bronchus and lung	29	5278	1530.62
Bone	0.6	93	0.558
Melanoma	4.3	4470	192.21
Mesothelioma	90	391	351.9
Prostate	6	10512	630.72
Kidney	4.7	1470	69.09
Bladder	14.2	2139	303.738
Brain	10.6	807	85.542
Hodgkin's disease	3.9	232	9.048
Non-Hodgkin lymphoma	13.5	1864	251.64
Leukemia	18.5	1428	264.18
TOTAL		**40893**	**4415.3**

Table A2.2: Calculations of number of cases of cancer caused by occupation.

FEMALES

Cancer site	% of cancers attributable to occupation	No. of cancers 2000	No. of cancers occupationally caused
Oral cavity	0.3	498	1.494
Pharynx	0.5	86	0.43
Oesophagus	0.2	354	0.708
Stomach	5.4	713	38.502
Colon	0	4229	0
Rectum	0.1	1197	1.197
Liver	5.3	201	10.653
Gallbladder	0.4	337	1.348
Pancreas	3.5	896	31.36
Nose and nasal sinuses	6.7	52	3.484
Larynx	0.5	69	0.345
Bronchus and lung	5.3	2782	147.446
Bone	0.6	69	0.414
Melanoma	0.4	3761	15.044
Mesothelioma	25	75	18.75
Breast	1.7	11314	192.338
Cervix	5.9	745	43.955
Corpus uteri	1.1	1564	17.204
Ovary	2.1	1201	25.221
Kidney	0.8	935	7.48
Bladder	0.7	747	5.229
Brain	1.3	608	7.904
Hodgkin's disease	0	189	0
Non-Hodgkin lymphoma	3.1	1593	49.383
Leukemia	2.5	942	23.55
TOTAL		**35157**	**643.4**

APPENDIX 3. DETERMINING WHICH SUBSTANCES ARE CARCINOGENIC

3A. Determining which substances are carcinogenic

A number of different organisations have established systems whereby they categorize chemicals according to their carcinogenicity. These use evidence from toxicological studies, from experiments on animals, and from epidemiological studies on humans. Studies on animals are not considered as useful as those on humans because of differences between species such as in the way that animals metabolise chemicals. In addition there are obvious differences in the way laboratory animals and free-living humans live their lives.

For obvious ethical reasons, epidemiological studies rely on observations in which people exposed and unexposed to the chemical of interest are compared. The problem inherent in these designs is that there may be important differences between those who are exposed and who are not. For example, in smoking, in education, or in ethnic group. These differences may contribute to differences in cancer rates, which make it difficult to attribute the cancers to the chemical being studied. Also, studies of cancer require a large number of subjects in order to give statistically reliable results. Many studies are too small to produce definitive answers to the questions of carcinogenicity. In addition, every study to investigate one chemical, or one occupation costs a great deal in time and in money. Thus only a small proportion of all chemicals have been intensively examined.

However, it is obviously important to make attempts to combine all the information available on each chemical to determine whether it is carcinogenic or not. Several organisations have developed systems to do this and below we summarize the most well-known of these systems.

3B. International Agency for Research in Cancer (IARC)

The IARC Monographs series publishes independent assessments by panels of international experts of the carcinogenic risks posed to humans by a variety of agents, mixtures and exposures. Since its inception in 1972, the series has reviewed more than 895 agents.

The expert panels consider the evidence on whether the agent causes cancer in animals, as well as epidemiological data on whether the agent causes cancer in humans. The body of evidence is considered as a whole, in order to reach an overall evaluation of the carcinogenicity to humans of an agent, mixture or circumstance of exposure. Four categories are possible:

Group 1: The agent is carcinogenic to humans. This category is used when there is sufficient evidence of carcinogenicity in humans.

Group 2A: The agent is probably carcinogenic to humans. This category is used when there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals.

Group 2B: The agent is possibly carcinogenic to humans. This category is used for agents, mixtures and exposure circumstances for which there is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals.

Group 3: The agent is not classifiable as to its carcinogenicity to humans. This category is used most commonly for agents, mixtures and exposure circumstances for which the evidence of carcinogenicity is inadequate in humans and inadequate or limited in experimental animals.

Group 4: The agent is probably not carcinogenic to humans. This category is used for agents or mixtures for which there is evidence suggesting lack of carcinogenicity in humans and in experimental animals.

Currently IARC has classified about 63 agents, 13 mixtures and 15 exposure circumstances as Group 1. This includes a number of occupations (eg painter, cabinet maker) as well as different chemicals used primarily in occupational circumstances. A further 31 agents and 3 exposure circumstances are classified in group 2A. Not all these agents are occupational carcinogens and a review of the IARC classifications found that 28 agents were definite occupational carcinogens, and a further 27 were probable occupational carcinogens (Siemiatycki et al., 2004).

3C. United States Department of Health and Human Services Report on Carcinogens (RoC)

The RoC identifies and discusses substances which may pose a carcinogenic hazard to human health and to which a significant number of people in the US may be exposed. Substances on the RoC are classified into one of two groups:

- Known to be human carcinogen.
- Reasonably anticipated to be human carcinogenic.

The 10th RoC classifies 52 substances into the known carcinogens group, and a further 176 substances into the reasonably anticipated carcinogens group.

3D. United States Environmental Protection Authority (EPA)

Various departments within the EPA assess hazards for their human carcinogenic potential. Agents can be classified as:

- Carcinogenic to humans: when there is convincing epidemiologic evidence demonstrating causality between human exposure and cancer.

- Likely to be carcinogenic to humans: when the available data are adequate to demonstrate carcinogenic potential to humans.
- Suggestive evidence of carcinogenic potential: when the evidence from human or animal data is suggestive of carcinogenicity but is not sufficient for a stronger conclusion.
- Inadequate information to assess carcinogenic potential.
- Not likely to be carcinogenic to humans.

These classifications have changed over time, but it is clear that the EPA are much more conservative than any other organisation. They have only classified three substances (arsenic, benzene, chromium IV) as carcinogenic to humans, and a further 102 as likely or probable carcinogens. They have not evaluated any form of asbestos.

3E. National Occupational Health and Safety Commission (NOHSC)

Under NOHSC's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)]1 (National Model Regulations) and the Australian, State and Territory government regulations introduced in accordance with the National Model Regulations, *manufacturers* and *importers* of substances supplied for use at work are required to determine whether they are hazardous to health before supply.

To determine whether a substance is a hazardous substance, manufacturers and importers should first refer to the *List of Designated Hazardous Substances* (the List), published by NOHSC. The List comprises the more common hazardous substances that meet NOHSC's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(2004)] (the Approved Criteria). Under the National Model Regulations if a substance is on the List then it is a hazardous substance. The List identifies a range of hazardous properties of chemicals including whether the substance is carcinogenic. The List is therefore an aid to classifying hazardous substances, however, it is not necessarily an exhaustive list of all hazardous substances. If a substance (or mixture of substances) is not on the List, then importers and manufacturers must use the Approved Criteria to determine whether it is hazardous and derive the appropriate classification information.

For the purpose of classification and labelling, and having regard to the current state of knowledge, the Approved Criteria defines the following three classification categories for substances that pose a carcinogenic hazard:

Category 1: Substances known to be carcinogenic to man. There is sufficient evidence to establish a causal association between human exposure to a substance and the development of cancer.

Category 2: Substances that should be regarded as if they are carcinogenic to man. There is sufficient evidence to provide a strong presumption that human exposure to a substance may result in the development of cancer, generally on the basis of:
• appropriate long-term animal studies,
• other relevant information.

Category 3: Substances that cause concern for man owing to possible carcinogenic effects but in respect of which the available information is not adequate for making a satisfactory assessment. There is some evidence from appropriate animal studies, but this is insufficient to place the substance in Category 2.

Under this system 60 individual substances on the List have been classified as category 1, over 700 as category 2 and 123 as category 3. These numbers are not easily comparable to the IARC numbers as the NOHSC list includes each individual substance identified by its separate CAS number, while the IARC classifications group compounds. For example, IARC classifies coal tars and pitches as group 1 carcinogens, while NOHSC lists each eight chemically distinct coal tar pitch compounds as category 1 carcinogens. The NOHSC list is also confined to chemical substances and does not include physical agents such as ionizing or solar radiation.

In addition to the *Approved Criteria for Classifying Hazardous Substances* and the *List of Designated Hazardous Substances*, NOHSC published the *National Model Regulations for the Control of Scheduled Carcinogenic Substances [NOHSC:1011 (1995)]* and the *National Code of Practice for the Control of Scheduled Carcinogenic Substances [NOHSC:2014(1995)].* Substances are scheduled according to a set of general and specific criteria, including that they shall have been classified as Category 1 or Category 2 carcinogens in accordance with the Approved Criteria and with consideration to the nature and extent of exposure in Australian workplaces. In addition Schedule 1 (prohibited) carcinogens are those where a reasonably practicable substitute is available and Schedule 2 (notifiable) carcinogens are those where: no reasonably practicable substitute is available; and there is an absence of other regulatory controls to prevent or minimise exposure to the required standard; and it is practicable and purposeful to notify.

Eleven substances have been classified as prohibited and 12 as notifiable carcinogens:

Prohibited Carcinogenic Substances

2-Acetylaminofluorene

Aflatoxins

4-Aminodiphenyl

Amosite (brown asbestos)

Benzidine and its salts

Bis(Chloromethyl) ether

Chloromethyl methyl ether

Crocidolite (blue asbestos)

4-dimethylaminoazobenzene

2-Naphthylamine and its salts

4-Nitrodiphenyl

Notifiable carcinogenic substances (employers must notify the relevant public authority of the intention to use one of these substances).

Acrylonitrile

Benzene (feedstock containing more than 50% of benzene by volume)

Chrysotile (white asbestos)

Cyclophosphamide

3,3'-Dichlorobenzidine and its salts

Diethyl sulfate

Dimethyl sulfate

Ethylene dibromide

4,4'-Methylene bis(2-chloroaniline)

2-Propiolactone

o-Toluidine and o-Toluidine hydrochloride

Vinyl chloride monomer

NOHSC Current Review Activities

The NOHSC Office is currently reviewing the national framework for workplace chemicals. It has been agreed through the NOHSC consultative process that the new framework will be based around the Globally Harmonised System for Classification and Labelling of Chemicals (the GHS).

The GHS is intended to develop a single, globally harmonized system to address classification of chemicals, labels, and safety data sheets. It is a hazard-based classification system that considers the health, physicochemical and environmental properties of chemicals and provides guidance on hazard communication, including product labels and safety data sheets.

The United Nations Economic and Social Council's Sub-Committee of Experts on the Globally Harmonized System of Classification (UNSCEGHS) has developed the GHS since the end of 2001. The Office of the National Occupational Health and Safety Commission (NOHSC Office) provides the Australian expert to the UNSCEGHS.

The first GHS document was approved in December 2002 and is intended to serve as the initial basis for global implementation of the GHS. The target date for global implementation of the GHS is 2008.

Australia is committed to the implementation of the GHS across the chemical sector and the review of the NOHSC workplace chemicals framework provides a timely opportunity to align the national workplace chemicals instruments with the GHS.

Adoption of the GHS classification criteria would alter the names of the carcinogen categories that are assigned, although the categories align fairly well with the categories from the Approved Criteria.

The GHS criteria define the following hazard categories for carcinogens:

Category 1: Known or presumed human carcinogen

- Category 1A: Known to have carcinogenic potential for humans; the placing of a chemical is largely based on human evidence.
- Category 1B: Presumed to have carcinogenic potential for humans; the placing of a chemical is largely based on animal evidence.

Category 2: Suspected human carcinogens

APPENDIX 4. CALCULATIONS OF NUMBER OF WORKERS POTENTIALLY EXPOSED TO CARCINOGENS AT WORK.

Numbers of employed workers, exposures, and exposed workers (in thousands) in the European Union by industry in 1990-3 converted to Australian workers. From (Kauppinen et al., 2000)

ISIC-2 code	Industry	Employed workers (n)	Exposed workers (n)	% workers occupationally exposed	No. of Australian workers	No. of Australian workers occupationally exposed
11	Agriculture and hunting	7900	3000	37.97	305572	116040
12	Forestry and logging	410	350	85.37	10968	9363
13	Fishing	230	150	65.22	11795	7692
21	Coal mining	370	1	0.27	18910	51
22	Crude petroleum and natural gas production	130	43	33.08	5038	1666
23	Metal ore mining	62	29	46.77	30027	14045
29	Other mining	270	190	70.37	7002	4927
311-2	Food manufacturing	2700	310	11.48	129608	14880
313	Beverage industries	410	59	14.39	25573	3680
314	Tobacco manufacture	88	4	4.55	2144	97
321	Manufacture of textiles	1300	220	16.92	22030	3728
322	Manufacture of wearing apparel	1500	340	22.67	30966	7019
323	Manufacture of leather and products of leather	180	40	22.22	3279	729
324	Manufacture of footwear	460	88	19.13	3684	705
331	Manufacture of wood and wood and	770	500	64.94	46218	30012

	cork products					
332	Manufacture of furniture and fixtures	790	600	75.95	48471	36813
341	Manufacture of paper and paper products	730	140	19.18	16320	3130
342	Printing, publishing, and allied industries	1700	440	25.88	99327	25708
351	Manufacture of industrial chemicals	1000	350	35.00	11773	4121
352	Manufacture of other chemical products	950	340	35.79	36256	12976
353	Petroleum refineries	130	74	56.92	6245	3555
354	Manufacture of petroleum and coal products	26	18	69.23	1339	927
355	Manufacture of rubber products	380	140	36.84	6477	2386
356	Manufacture of plastic products	840	330	39.29	29074	11422
361	Manufacture of pottery, china, and earthware	260	170	65.38	7645	4999
362	Manufacture of glass and glass products	300	130	43.33	8245	3573
369	Manufacture of other non-metallic mineral products	640	430	67.19	7206	4842
371	Iron and steel basic industries	850	380	44.71	41234	18434
372	Non-ferrous metal basic industries	360	160	44.44	26429	11746
381	Manufacture of fabricated metal products	2800	810	28.93	30641	8864
382	Manufacture of machinery except electrical	3800	830	21.84	192589	42065
383	Manufacture of electrical machinery	3000	440	14.67	25891	3797
384	Manufacture of transport equipment	3000	970	32.33	90034	29111
385	Manufacture of instruments, etc	540	190	35.19	14507	5104

39	Other manufacturing industries	400	110	27.50	14647	4028
41	Electricity, gas, and steam	1200	430	35.83	40423	14485
42	Water works and supply	220	84	38.18	19937	7612
5	Construction	11000	6100	55.45	558565	309750
6	Wholesale and retail trade and restaurants	24000	3500	14.58	2058658	300221
711	Land transport	4200	1700	40.48	199679	80822
712	Water transport	350	180	51.43	10467	5383
713	Air transport	450	290	64.44	43296	27902
719	Services allied to transport	1400	580	41.43	62468	25880
72	Communication	2600	590	22.69	148418	33679
8	Financing, insurance, real estate, business services	13000	1100	8.46	1232696	104305
91	Public administration and defence	11000	1600	14.55	369913	53806
92	Sanitary and similar services	1400	360	25.71	-	0
931	Education services	9000	330	3.67	595403	21831
932	Research and scientific institutes	490	100	20.41	25374	5178
933	Medical, dental, other health services	8200	730	8.90	111374	9915
934	Welfare institutions	4000	210	5.25	-	0
935-9	Business, professional, and other organisations	1500	230	15.33	-	0
94	Recreational and cultural services	2100	270	12.86	202589	26047
95	Personal and household services	32000	1600	5.00	300732	15037
96	International organisations	160	1	0.63	-	0
	Total	**139 000**	**32 000**	**23.02**	**7 347 156**	**1 494 090**

APPENDIX 5. WORKPLACE PROFILE FOR WOOD WORKERS AND WOOD DUST EXPOSURE

5A. Wood workers and wood dust exposure

Both IARC (vol 62, 1995) and the US Tenth Report on Carcinogens classify wood dust as a known human carcinogen. Wood dust has been shown to be associated with cancer of the nasal cavities and paranasal sinuses in many epidemiological studies. The association between adenocarcinoma of the nasal cavities and paranasal sinuses and exposure to hardwood dust is particularly strong. There were too few studies to evaluate cancer risks attributable to softwood dust alone. In their review of studies, which included a pooled analysis of 12 separate case control studies, IARC concluded that these studies consistently found that occupational exposure to wood dust is causally related to adenocarcinoma of the nasal cavities and paranasal sinuses. (IARC. Wood dust. Summary of data reported and evaluation, 1995)

IARC also reviewed studies of the association between nasopharyngeal cancer and occupational exposure to wood dust. IARC concluded that overall these studies provide suggestive but inconclusive evidence for a causal role of occupational exposure to wood dust in cancers of the nasopharynx. (IARC 1995). Subsequent to the publication of the IARC monograph on wood dust (1995), IARC conducted analyses of the two largest data sets ever assembled to examine the relationship between cancer and wood dust. In the pooled re-analysis of cohort studies, a large excess of nasopharyngeal cancer was found in furniture workers in jobs with the greatest likelihood of wood dust exposure (Demers & Boffetta 1998, p. 90). This study also suggested that the risk of multiple myeloma may be elevated amongst wood workers. The authors conclude that the overall pattern suggests that chemical agents that may occur within wood-related industries (such as paints, solvents and pesticides) as well as wood dust may play a role in the development of multiple myeloma (Demers and Boffetta 1998, p. 90).

Nurminen and Karjalainen (2001) estimated that 15.7 per cent of cancers of the nose and nasal sinuses in men and 2.4 per cent in women could be attributed to exposure to wood dust. Two separate Australian studies published of patients with adenocarcinoma of the nose and paranasal sinuses, one in Victoria (Ironside and Mathews 1975) and one in Tasmania (Franklin 1982) found that there were a significantly higher proportion of woodworkers than in the general population (7 of 19 cases in Victoria and 9 of 13 cases in Tasmania).

5B. Australian cancer incidence

Incidence of these cancers is rare. In Australia in 2001 there were 101 new cases of nasopharyngeal cancer (73 in men & 28 in women) and 128 new cases of cancer of the nasal cavity (92 in men and 36 in women) (AIHW 2004). There were also 48 deaths (40 in men and 8 in women) due to nasopharyngeal cancer and 45 (33 in men and 12 in women) due to cancer of the nasal cavities.

5C. Occupational exposure to wood dust

Demers and Boffetta (1998, p. 9) found that the highest wood dust exposures occurred amongst woodworking machine operators, cabinet makers, furniture finishers, carpenters and related wood workers employed in the manufacture of wood products. In most cases these jobs involved sanding, milling or machining dry wood resulting in the generation of large amounts of finer dust, which would be more likely to remain airborne.

Moderately exposed workers included cabinet makers, carpenters and related wood workers not employed in the mass production of wood products, as well as loggers, sawmill operators, wood chippers and grinders employed in the pulp and paper industry. Some of these processes involved work with fresh wood and/or processes that would generate wood with larger particle sizes that would not necessarily be inhaled into the sino-nasal passages.

Australian workers potentially exposed to wood dust:

Cabinet makers	21 786
Carpentry & joinery tradesperson	73 526
Wood machinists and turners	3 598
Wood processing machine operators	4 909
Wood products factory hands	8 668
Forestry & logging workers	4 007

Data from 2001 census data coded according to the Australian Classification of Occupations.

Based on the work of Demers and Boffetta (1998) above, it is likely that wood machinists and turners and wood processing machine operators would fall into the high exposure category; cabinet makers, carpentry and joinery tradespersons and wood products factory hands may fall into the high or moderate exposure category depending on the nature of their workplace and forestry and logging workers are more likely to fall into the moderate exposure category.

5D. Control of wood dust

The Australian exposure standard for hardwood dust is 1 mg/m3 (TWA) and 5 mg.m3 (TWA) for softwood exposure. A study of personal inhalable wood dust exposures in New South Wales logging sites (two), sawmills (four), one major wood chipping operation and five joineries, found that overall 62% of the exposures exceeded the current standards (Alwis et al. 1999). Among joineries, 95% of the hardwood exposures and 35% of the softwood exposures were above the relevant standards. In two of the sawmills, poorly maintained ventilation or ineffectively ventilated machines resulted in exposures as high as 51 mg/m3 (for a molder operator) and 67 mg/m3 (for a picket machine operator). In the joineries, handheld sanding operations nearly always resulted in exposures above the standard, with exposure measured as high as 35 mg/m3 (copy lathes) and 49 mg/m3 (horizontal band sander).
An earlier study of wood dust exposure during furniture manufacture in Adelaide (Pisaniello et al. 1991) found that 78% of personal hardwood dust exposures exceeded the standard, while only 16% of personal softwood/reconstituted wood dust exposures exceeded the standard for softwood dust exposure. Both studies found respiratory protection was almost never worn.

The authors of the NSW study concluded that the elevated exposures were due to a combination of factors including:

- lack of awareness of potential health effects of wood dust exposure among both management and workers;
- ageing equipment;
- inadequate and ineffective dust extraction systems (or none, especially for handheld tools);
- poor maintenance of the ventilation system in some;
- non-segregation of dusty processes; and
- dry sweeping and the use of compressed air jets to clean surfaces and machinery.

The Adelaide study found that local exhaust systems were used widely with fixed woodworking machinery but were generally lacking for hand tools. This study also found that the dry sweeping and the use of compressed air for cleaning were common.