

# WORK-RELATED CARDIO-VASCULAR DISEASE AUSTRALIA

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Australian Government

Australian Safety and Compensation Council

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## **ACKNOWLEDGMENTS**

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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 at 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.

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## **EXECUTIVE SUMMARY**

### ***Background***

The project is a literature-based review of epidemiological studies on work-related cardiovascular disease (CVD), including coverage of the magnitude and severity, causes, affected occupational groups and possible prevention approaches, with specific focus on Australia.

### ***Definitions and a consideration of relevant concepts and issues***

The term 'cardiovascular disease' is used to include many forms of circulatory disease. For the purposes of this report, 'cardiovascular disease' is synonymous with 'ischaemic heart disease' because this is the definition used in most relevant studies. Disorders of the arteries of the brain (cerebrovascular disease), the limbs (peripheral vascular disease) or other blood vessels are not considered in this report.

Work-related cardiovascular disease is cardiovascular disease caused by occupational factors that increase the oxygen requirements of the heart or decrease the capacity of the heart to use oxygen.

It can be very difficult to link a specific work-related exposure to the development of cardiovascular disease in an individual person. This is because of issues of latency, multiple possible risk factors, lack of specific work-related features, and various factors that influence diagnosis.

### ***Methods***

Most of the information presented in this report is based on published, peer-reviewed literature. Other relevant information from online sources has been included where appropriate. No new investigations were undertaken to obtain general information on exposure or risk.

Statistical information on the extent of work-related cardiovascular disease in Australia was obtained from several sources. Information included national workers' compensation accepted claims and presentations to general practitioners.

### ***Main occupational risk factors for cardiovascular disease***

There are many work-related factors that have been implicated as potentially increasing the risk of a cardiovascular event. The evidence is strongest with exposure to four particular chemicals, namely carbon disulphide and, in terms of acute exposure, carbon monoxide, methylene chloride and nitroglycerin. There is also good evidence for the role of environmental tobacco smoke and psychosocial factors, particularly low job control, and considerable evidence for noise and shiftwork. Other exposures, for which the evidence is less strong, include chronic low-level exposure to carbon monoxide, methylene chloride and nitroglycerin, other

chemicals, long working hours, electromagnetic fields, temperature extremes, diesel exhaust and other particulates, organic combustion products, manual work or strenuous occupations, sedentary work, and certain specific occupations.

### ***Prevalence of cardiovascular disease risk factors in the Australian workforce***

Exposure to carbon monoxide comes mainly from vehicle exhausts, but can occur in other situations. Levels of carboxyhaemoglobin high enough to cause cardiac problems in persons with pre-existing coronary artery disease occur in exposed smokers.

Exposure to environmental tobacco smoke is highest in workers in the service and hospitality industries, such as restaurant and bar workers. Exposure is much less common in office workers following the adoption of non-smoking policies in many offices and other workplaces..

There is no direct measure of the prevalence of exposure to hazardous noise levels in Australian workplaces. However, estimates for 1986 and 2002 both suggested about 12% of the workforce (about one million workers in 2002) are occupationally exposed to hazardous levels of noise. Workers' compensation data suggest that the occupation groups with the highest exposure to hazardous levels of noise are tradespersons, intermediate production and transport workers, and labourers.

An Australian Bureau of Statistics (ABS) working arrangements survey conducted in November 2003 found that 14% of workers reported having performed shiftwork in the previous four weeks. Nearly half (46%) of these workers reported working on a 'rotating' shift roster. Intermediate production and transport workers was the occupation group with the highest prevalence of shiftwork (24%). Industries with the highest prevalence of shiftwork were mining (44%), health and community services (32%), accommodation, cafes and restaurants (31%), transport and storage (27%) and personal and other services (25%).

There is little or no exposure to carbon disulphide or nitroglycerin in Australia.

The recent ABS working arrangements survey found that 33% of workers reported performing overtime on a regular basis.

The incidence of exposure to other risk factors that occur in a range of occupations is not known.

### ***Overall estimates of work-related cardiovascular disease***

There have been several studies in different countries that have estimated the extent of work-related cardiovascular disease. The resulting population-related risks have been based on a variety of exposures, most

particularly environmental tobacco smoke, psychosocial factors such as low job control, noise and shiftwork. These risks ranged from 1% to 19%, with the highest estimates being 6% to 18% in the United States, and 19% (males) and 9% (females) in Finland.

### ***Estimates of work-related cardiovascular disease in Australia***

There is limited information on the extent of work-related cardiovascular disease in Australia. Information that is available comes from a variety of sources, including published studies, workers' compensation and general practitioner data sources.

The only estimate of work-related cardiovascular disease population attributable risk in Australia was 1%, or a total of 800 deaths due to cardiovascular disease arising from occupational exposure to hazardous substances. This estimate excluded non-chemical exposures such as environmental tobacco smoke, low job control and noise. The estimate of the number of work-related cardiovascular deaths in Australia is almost certainly a major underestimate of the number of work-related cardiovascular deaths in Australia, based on the most recent attributable risk estimates used elsewhere.

Workers' compensation information cannot be considered a reliable or valid indicator of the extent of work-related cardiovascular disease in Australia, for a number of reasons. Some limited information on work-related cardiovascular disease is available from these data sources, but the results are almost certainly gross underestimates.

There is no useful information on work-related cardiovascular disease available for hospital admissions.

### ***Approaches to prevention of work-related cardiovascular disease***

CVD prevention activities derived from the literature and analysis of the data suggest that the largest gains are likely to be made by targeting the following areas:

- Implementation and enforcement of non-smoking policies. This appears to be an effective way to decrease occupational exposure to environmental tobacco smoke, as the use of designated non-smoking areas are ineffective controls, as they only ever have a limited effect in the concentration of ETS (environmental tobacco smoke) in non-smoking sections.
- Prevention of exposure to carbon monoxide. This type of exposure comes largely from vehicle exhausts, but can occur in other situations. Appropriate prevention should be achievable through the application of standard control procedures using the hierarchy of control. In areas where carbon monoxide exposure may occur atmospheric monitoring is essential.



- Prevention of exposure to noise. Approaches to controlling noise in the workplace are well known. All aspects of the hierarchy of control are relevant, particularly eliminating noise through engineering improvements, isolating noise through enclosure of noisy processes, and, where the other approaches are inadequate, the use of effective personal protective equipment as part of a comprehensive hearing protection program. More specific prevention suggestions have been outlined in the Noise Induced Hearing Loss (NIHL) profile report.
- Prevention of exposure to psychosocial risk factors in the workplace. However, identifying areas that need to be changed, and making the necessary changes to workplace structure and culture, can be very difficult. More specific prevention suggestions will be outlined in the Mental Disorders profile report.
- Optimising shift design. While shiftwork has been associated with increased CVDs, without knowing the mechanism(s) underlying this relationship, it is difficult to plan appropriate interventions. A recent review argued that, in the absence of definitive information, it is appropriate to advise shiftworkers and night workers to eat regularly and to maintain healthy lifestyles (particularly in regard to smoking), and to consider the optimum design and transition for the shift cycle
- Health promotion programs. The worksite has been promoted as a very important place for basing health promotion activities, because certain occupations are known to have a higher level of CVD risk factors and to have a higher risk of cardiovascular disease. Despite apparent support for the concept, low participation rates have hampered many programs.

In general, most of the CVD risk prevention actions outlined will occur as part of national programs to prevent exposure to ETS, hazardous chemicals, and to psychosocial risk factors (as part of the mental disorders) and NIHL more generally.

### ***Conclusions***

Work-related cardiovascular disease is an important cause of work-related morbidity and mortality. However, the extent of the problem is difficult to identify accurately, and currently can only be studied through the use of population methods. Effective prevention activities exist for some exposures, but for others there is insufficient information on prevention activities and their effectiveness.

## **1. INTRODUCTION**

### **1.1 Background and report aims**

This report is a literature-based review of epidemiological studies on work-related cardiovascular disease in Australia, including coverage of the magnitude and severity, causes, affected occupational groups and possible prevention approaches.

### **1.2 Outline of the structure of the report**

The report has seven main sections. These present, in order:

- definitions and a consideration of relevant concepts and issues;
- an outline of the methods used in obtaining information for the report;
- information on the main occupational risk factors for cardiovascular disease;
- information on the prevalence of these risk factors in the Australian workforce;
- overall estimates of work-related cardiovascular disease;
- estimates of work-related cardiovascular disease in Australia; and
- a summary of what is known about approaches to preventing work-related cardiovascular disease.

## **2. DEFINITIONS AND RELEVANT ISSUES AND CONCEPTS**

### **2.1 Definitions**

#### ***Cardiovascular disease***

The heart functions as a pump, circulating the blood around the body. To perform this function, it requires oxygen to be supplied to the muscle of the heart. This oxygen comes from the blood, which is delivered to the heart muscle by small blood vessels called coronary arteries. Problems may occur if the heart requires more oxygen than is delivered by the blood. The main causes of this include not enough blood being delivered to the heart muscle (because the coronary arteries are partially or completely blocked); and the heart requiring more oxygen because it is pumping harder or faster than normal (as occurs with exercise or heat stress).

There are various definitions for 'cardiovascular disease' depending on the context in which it is used. Although the term is used to include many or all forms of circulatory disease, for the purposes of this report, 'cardiovascular disease' is synonymous with 'ischaemic heart disease', a term used interchangeably with 'coronary artery disease' and one used in most relevant studies. It describes abnormality of the heart arising from disease of the coronary arteries. Disorders of the arteries of the brain (cerebrovascular disease), the limbs (peripheral vascular disease) or other blood vessels are not included. However, these conditions are often associated with cardiovascular disease because they have many of the same causes or risk factors. Hypertension is also not included, but is considered for certain exposures in relation to the increase in risk of cardiovascular disease associated with hypertension.

The most common form of cardiovascular disease is atherosclerosis, in which fatty plaques form inside the arteries, blocking the arteries and causing decreased blood flow. If blood flow is decreased enough, it can result in part of the heart muscle getting insufficient blood. This usually causes chest pain, known as 'angina'. If the blood flow is cut off completely, part of the heart muscle may die. This is what occurs in a heart attack (also known as a 'myocardial infarct'). Other outcomes that can occur include heart failure, where the heart doesn't pump strongly enough; abnormally fast, slow or irregular beating of the heart (known as "arrhythmia"); and sudden death.

Cardiovascular disease has two aspects – a chronic process in which the coronary arteries develop atheromatous plaques over many years, and an acute process in which there is rupture of an atheromatous plaque in, or thrombus of, a diseased coronary artery.

### ***Work-related cardiovascular disease***

Work-related cardiovascular disease is CVD that is caused or exacerbated by occupational factors. The term also usually includes disease resulting from occupational exposures or factors that increase the oxygen requirements of the heart or decrease the capacity of the heart to use the oxygen. Various disease inclusions and exclusions have been used in studies of work-related CVD, and in many studies these inclusions and exclusions have not been made explicit.

## **2.2 Relevant issues**

For several reasons, it can be very difficult to link a specific work-related exposure to the development of cardiovascular disease in an individual person. These include latency, multiple possible risk factors, lack of specific work-related features, and various factors that influence diagnosis. These are considered in this section.

### ***Latency***

For many causative factors, there is likely to be a long latency between exposure and the development of clinically significant CVD. This means that the disease usually develops slowly, over many years, making the connection between exposure and disease difficult to identify. The risk of CVD may decrease fairly quickly once exposure ceases (Steenland et al. 2003), although the extent and rate of this decrease is not certain, and it may be that the increased risk persists for many years (Leigh and Schnall 2000).

### ***Multiple underlying risk factors***

There are many known general risk factors for cardiovascular disease. The main ones are smoking, hypertension, hyperlipidaemia, diabetes, family history, obesity and lack of exercise (Wilson et al. 1998). Many workers will have one or more of these risk factors in addition to other risk factors associated with their occupation. In addition, some of these general risk factors (e.g. lack of exercise) may be directly related to their occupation.

### ***Lack of specific work-related features***

There are no anatomical or pathological differences between CVD arising from work exposures and CVD arising from non-work exposures.

### ***Acute coronary events versus underlying cardiovascular disease***

It can be very difficult to link an acute coronary event (such as angina, a heart attack or an arrhythmia) to one or more work-related factors. Cardiovascular disease is usually identified because the person experiences an acute coronary event, but there are many factors that can influence when a person experiences such an event. This means that occupational exposures can make an important contribution to a person developing CVD but the person might have a heart attack whilst not at work. Conversely, a person might have a heart attack at work because of CVD that developed due to non-work exposures. Or strenuous work activity might precipitate a heart attack in a person who has underlying CVD that is not related to work. Often there is no clear-cut cause for a particular cardiac event. Both occupational and non-occupational factors might contribute to the development of underlying CVD, and one or both might contribute to the occurrence of a particular acute coronary event. This means that, in the vast majority of individual cases, it is not possible to say with confidence whether occupational factors did, or did not, contribute to the development of CVD or the occurrence of a particular acute coronary event.

## **2.3 Population attributable risk**

The above issues mean that it is usually necessary to take a population-based approach when assessing the role of occupational factors in the development of cardiovascular disease. Two main approaches are used. One involves the conduct of epidemiological studies to estimate risk (or relative risk) arising from particular exposures. The second approach is to produce an estimation of the proportion or percentage of CVD in the community that is due to occupational exposures, known as the Population Attributable Risk (PAR) or the Population Attributable Fraction.

The PAR is the proportion (often expressed as percentage) of all cases of a particular condition that is due to a particular exposure (or group of exposures). In this instance, the PAR of interest is the proportion (or percentage) of all CVD in the community that is due to occupational exposures. The PAR is dependent on the relative risk of developing the condition due to the exposure, and the proportion of the population that has that exposure. The relative risk is likely to be similar in different populations and different countries, provided the circumstances of exposure are similar. However, the proportion of the population exposed to a particular factor might vary considerably between different regions, different countries and different time periods. Therefore, the PAR for a particular exposure may also vary considerably between different regions, countries and time periods.

There is sometimes very limited information on relative risk and/or exposure prevalence, leading to estimates of PAR being developed through the so-called Delphi method. Essentially, this involves a group of experts deciding between them what the likely PAR is, based on available research and their experience. Early estimates of the role of work in the development of CVD tended to use the Delphi method because of a lack of appropriate data. More recent estimates have been entirely based on data, although this has often meant that only a limited number of exposures could be taken into account.

## **2.4 Age**

The rate of ischaemic heart disease events increases considerably with age. This has important implications for the estimated burden of work-related cardiovascular disease. If an upper age limit is used (such as persons less than 70 years), as has been the approach in many studies, the number of work-related CVD cases estimated using a certain PAR will be much less than if older persons are included. Whether an upper age limit should be used, and what that limit should be, depends on the pathological mechanism(s) associated with the exposures of interest, the age at which exposure ceases, the latency between exposure and disease manifestation, and the extent and rate of risk reversal once exposure ceases.

### **3. METHODS**

#### **3.1 Introduction**

Most of the information presented in this report is based on published, peer-reviewed literature. Other relevant information from online sources has been included where appropriate. No new investigations were undertaken to obtain general information on exposure or risk.

#### **3.2 Identifying and reviewing relevant literature**

English language literature published up to August 2004 was searched for relevant articles. Initial key words used were "heart disease", "employ\*", "job", "occupation", "cardiovascular", "blood", and "work".

The searches were conducted through Medline (via PubMed) and OSHROM, which incorporates HSELINE, RILOSH, CISDOC, NIOSHTIC, and MEDLINE. Secondary follow-up of sources cited in reference lists was also undertaken.

The relevance of papers was determined by considering the abstracts or the full text of each article identified through the literature search. For major articles, the full text was read, but only the abstracts were used for some less important studies. A final decision on inclusion was reached after the methodology, results and conclusions for each relevant paper were critically appraised.

#### **3.3 Data sources**

Statistical information on the extent of work-related CVD in Australia was obtained from several sources. National workers' compensation information for accepted new claims registered from 1998/1999 to 2002/2003 inclusive came from National Data Set for Workers' Compensation-based Statistics data and was supplied by NOHSC. Information on presentations to general practitioners came from a previous NOHSC study of data for April 1998 to March 2000 (Hendrie and Driscoll 2003; National Occupational Health and Safety Commission 2001b), based on data from the Bettering the Evaluation and Care of Health (BEACH) study (Britt et al. 1999). Combined data for April 1999 to March 2002 was supplied by NOHSC, but results were not available at the same level of detail as from the earlier study.

#### **3.4 Ethical issues**

No important ethical issues were relevant to the project. All the literature used was in the public domain, and the workers' compensation and BEACH data were obtained and used under the terms of confidentiality agreements with the original data providers.

## **4. MAIN OCCUPATIONAL RISK FACTORS**

### **4.1 Introduction**

There are many work-related factors that have been connected with increasing the risk of sustaining a cardiovascular event in relation to work and the subject has been reviewed several times over the last two decades (for example, see (Kristensen 1989a, 1989b, 1999; Kurppa et al. 1984; Rosenman 1984; Steenland 1996; Steenland et al. 2000)). The evidence is strongest for four chemical exposures - carbon disulphide and, in terms of acute exposures, carbon monoxide, methylene chloride and nitroglycerin.

There is also evidence for the role of environmental tobacco smoke and psychosocial factors, particularly low job control, and evidence for noise and shiftwork. Other exposures for which there is evidence include chronic low-level exposure to carbon monoxide, methylene chloride and nitroglycerin, other chemicals, long working hours, electromagnetic fields, temperature extremes, diesel exhaust and other particulates, organic combustion products, manual work or strenuous occupations, sedentary work, and certain specific occupations (Table 1 next page).

**Table 1 Suggested risk factors for work-related cardiovascular disease**

<b>Probability</b>	<b>Factor</b>
Definite	Carbon disulphide Carbon monoxide (acute exposures) Methylene chloride (acute exposures) Nitroglycerin (acute exposures)
Probable	Environmental tobacco smoke Low job control Noise Shiftwork
Possible	Carbon monoxide, methylene chloride and nitroglycerin (chronic low-level exposure) Effort reward imbalance (and/or other psychosocial factors) Long working hours Electromagnetic fields Exhaust (diesel) Lead Manual work / strenuous occupations Sedentary work Other chemicals (arsenic, cobalt, dioxin) Organic combustion products Temperature extremes Specific occupations

#### 4.2 Carbon monoxide

Carbon monoxide exposure decreases the oxygen-carrying capacity of the blood by converting haemoglobin to carboxyhaemoglobin (COHb). It increases the risk of experiencing an acute ischaemic heart disease event, probably by decreasing the oxygen supply to the heart, and possibly by increasing the underlying level of ischaemic heart disease (Herbert et al.



2000; Kristensen 1989a; Nurminen and Karjalainen 2001; Steenland et al. 2000; Stern et al. 1988). Levels of COHb seen in prolonged lower level exposures are not likely to be high enough to cause myocardial ischaemia in persons who do not smoke (smoking increases the concentration of COHb in the blood). However, in smokers with pre-existing coronary artery disease, prolonged exposure to carbon monoxide may lead to blood levels of COHb high enough to precipitate an acute ischaemic event (Steenland et al. 2000; Wickramatillake et al. 1998).

### **4.3 Environmental tobacco smoke**

Environmental tobacco smoke is a mixture of smoke exhaled by another person and sidestream smoke from another person's cigarette (Mitchell and Sanders 2002; Samet 1999). There is good, but not overwhelming, evidence that exposure to environmental tobacco smoke increases the risk of developing cardiovascular disease, probably through the same mechanisms that increase ischaemic heart disease risk related to smoking (Jaakkola and Samet 1999; Jousilahti et al. 2002; Kawachi and Colditz 1999; Kawachi et al. 1989; National Health and Medical Research Council 1997b; Steenland 1999; Steenland et al. 2003).

### **4.4 Low job control and other psychosocial factors**

Psychosocial factors related to work have been proposed for more than decade as a potential cause of CVD. Study of this area has been complicated by difficulties separating occupational influences from non-occupational influences, and identifying which psychosocial factors may be important. Early studies concentrated on job 'stress', usually measured by the demands placed on the worker. However, it seems more likely that it is the combination of high demands and low control, or perhaps particularly low control, that are the important factors. 'High demand' covers factors such as excessive workload, confronting leadership and management style, lack of job security, high emotional demands in the job and conflict with colleagues. 'Job control' refers to the extent to which the worker has some involvement in decisions relating to his/her responsibilities and work practices. In addition, there is evidence of an independent effect from an imbalance between effort and reward, and high demands on their own may not be a cardiovascular risk factor. The postulated mechanism for development of cardiovascular disease related to these factors is through effects on neuroendocrine and autonomic systems, and possibly through changes to blood pressure (Bosma et al. 1997; Bosma et al. 1998; Eaker et al. 2004; Franke et al. 2002; Holmes 2001; Malinauskienė et al. 2004; Marmot et al. 1997; Peter and Siegrist 2000; Steenland 1996; Steenland et al. 2003; Steenland et al. 2000; Theorell et al. 1998). A recent Australian National Heart Foundation position statement concluded that there is poor evidence of support of a causal relationship between work-related 'stressors' and coronary heart disease (Bunker et al. 2003), based on conflicting evidence from three

relevant reviews (Kuper et al. 2002; Rozanski et al. 1999; Schnall et al. 1994). However, a more recent review concluded that 'job strain' is an important CVD risk factor (Belkic et al. 2004).

An Australian study looked at rates of ischaemic heart disease mortality in New South Wales, and at changes in these rates over time, in relation to various factors, including occupation. Standardised rates were the highest in labourers and the lowest in clerical and sales workers. Rates decreased over time in all occupations, but the differences in rates between occupation groups with the highest rates and those with the lowest rates increased over time, meaning that occupationally-related inequalities became greater over time. There was no control of known general risk factors for cardiovascular disease (such as smoking, obesity and hypertension), but the author suggested that at least some of the differences may be due to the "social and physical work environment", such as jobs that are boring and characterised by low levels of autonomy (Burnley 1998, 1999).

Other Australian studies have found similar results – higher rates of ischaemic heart disease among males tradespersons, labourers and/or plant and machine operators and drivers compared with other occupations (Bennett 1996; Dobson et al. 1991; McMichael and Hartshorne 1982).

#### **4.5 Noise**

There is no conclusive evidence for a direct connection between exposure to high noise levels and the development of CVD. is suggestive but not conclusive. However, there is good evidence that exposure to high noise levels increases mean blood pressure, probably via effects on neurohormonal substances such as cortisol, adrenaline and noradrenaline. Increases in mean blood pressure are strongly related to increases in the risk of developing CVD, so an indirect connection between noise and CVD is plausible (Babisch 2003; Melamed et al. 1999; Passchier-Vermeer and Passchier 2000; Stansfield and Matheson 2003; Van Kempen et al. 2002).

#### **4.6 Shiftwork**

The connection between shiftwork and CVD is also suggestive but not conclusive. Changes in circadian rhythm, social disruption and behavioural changes have been suggested as possible pathways linking shiftwork to increased risk of CVD. These pathways are separate but probably inter-related. The connection may be direct, through neurohormonal effects; or indirect, related to increased chance of developing other risk factors such as diabetes, hypertension and smoking, or worsening of their control (Boggild and Knutsson 1999; Costa 2000; Steenland et al. 2000; van Amelsvoort et al. 2004; Van Cauter and Spiegel 1999).

## 4.7 Other exposures

Carbon disulphide (Nurminen and Hernberg 1985) and nitroglycerin (Hogstedt and Andersson 1979) have a direct toxic effect on the heart and acute exposures increase the risk of sustaining an ischaemic heart disease outcome. Chronic low-level exposure to carbon disulphide appears to also increase risk, but there is equivocal information about the effect of chronic, low-level exposure to nitroglycerin. Methylene chloride, an organic solvent, is metabolised to carbon monoxide, which is the origin of its toxic effects. Like carbon monoxide, acute exposures to methylene chloride clearly increase the risk of sustaining an ischaemic heart disease outcome, but information about the effect of chronic, low-level exposure is equivocal (Amsel et al. 2001; International Programme on Chemical Safety 1996; Steenland et al. 2000). Other occupational chemical exposures that may increase the risk of ischaemic heart disease include antimony, arsenic, cobalt, dioxin and lead, but the mechanisms are unclear. In the case of lead, exposure causes hypertension, and this may result in an increased risk of ischaemic heart disease (Flesch-Janys et al. 1995; Kristensen 1989a; Moller and Kristensen 1992; Olsen and Kristensen 1991).

The relationship between long working hours (as opposed to shiftwork) and various cardiovascular and related outcomes has been recently reviewed (van der Hulst 2003). Two studies have examined the relationship between long working hours and the risk of CVD outcomes. These found an increased risk with 'long' hours of work (greater than 8.5 hours in one study and greater than 11 hours in the other), and one also found an increased risk with 'short' hours of work (less than 7.5 hours) (Emdad et al. 1998; Sokejima and Kagamimori 1998). An association has also been found between longer working hours and both hypertension and several cardiovascular indices such as heart rate, but the association is not strong or consistent (Steenland et al. 2000; van der Hulst 2003).

Electromagnetic fields have been proposed as a possible risk factor for CVD because of the suspected potential for precipitating arrhythmia or reducing heart rate variability. Although evidence of increased risk in exposed workforces has been found, several recent studies have not found such evidence, and overall the evidence must be considered equivocal (Ahlbom et al. 2004; Hakansson et al. 2003; Johansen 2004; Savitz et al. 1999).

There is some evidence that exposure to fine particulates might increase inflammation in the lungs, with resulting release of acute phase reactants, particularly fibrinogen, leading to increased coagulability. This in turn could increase the likelihood of coronary artery blockage in persons with pre-existing ischaemic heart disease (Pope et al. 2004; Sjogren 1997). Exposure to diesel exhaust, implicated as a possible risk factor for work-related CVD, might operate via this mechanism (Finkelstein et al. 2004).

Occupational exposure to organic combustion products has been implicated as an increased risk of myocardial infarction, an effect for which several possible pathogenic pathways have been proposed (Gustavsson et al. 2001).

Paradoxically, there is some evidence for both sedentary (Kristensen 1989b; Steenland et al. 2000) and strenuous occupations (Mukerji et al. 1999) increasing the risk of ischaemic heart disease.

Temperature extremes and high physical demands may increase the risk of an acute coronary event by increasing the workload on the heart, but the evidence is not strong and studies have been conflicting (Steenland et al. 2000).

A range of specific occupations have been associated with an increased risk of ischaemic heart disease (for example, see (Aronson et al. 1999; Calvert et al. 1999; Eliopoulos et al. 1984; Franke et al. 2002)) without identifying a clear causative exposure, although one or more of the above exposures could be expected to be relevant. Occupations identified in more than one study as having an increased risk of myocardial infarction and/or ischaemic heart disease morbidity or mortality are shown in Table 2 next page (taken from (Steenland et al. 2000)).

**Table 2 Occupations identified<sup>1</sup> as having an increased risk of myocardial infarction and/or ischaemic heart disease morbidity or mortality<sup>2</sup>**

<b>Men</b>	<b>Women</b>
Air traffic controllers	Bus drivers
Bakers	Cleaners
Bus drivers	Home help
Butchers	Rubber and plastics workers
Cannery workers	Self-employed in hotel and catering
Cooks	Taxi drivers
Firefighters	Unskilled workers in tube, sheet and steel construction
Fishermen	Waitresses
Foundry workers	
Hairdressers	
Lorry drivers	
Paper industry workers	
Police officers	
Prison wardens	
Rubber and plastics workers	
Ship's deck officers and sea pilots	
Storekeepers	
Taxi drivers	
Waiters	
Warehousemen	

1: Only includes studies in which the lower limit of the 95% confidence interval was greater than 1.0.

2: Taken from (Steenland et al. 2000), Table 10, page 59.

## **5. PREVALENCE OF RISK FACTORS IN THE AUSTRALIAN WORKFORCE**

### **5.1 Introduction**

This chapter summarises information on prevalence of occupational exposure to probable work-related cardiovascular risk factors in Australian workplaces. Unfortunately, there is limited information available.

### **5.2 Carbon monoxide**

Exposure to carbon monoxide comes largely from vehicle exhausts, but can occur in other situations. Australian studies have found that prolonged high level exposures can occur if combustion engines are operated in enclosed or confined spaces (e.g. forklifts operated in-doors, or pumps operated in a confined space); in foundries; if the atmosphere in a mine is not properly ventilated after explosives are detonated; in indoor car parks; and in compressed air diving if the exhaust of the compressor is near the air intake (Gorman et al. 1992; Lewis et al. 1992; Wickramatillake et al. 1998). However, there is no comprehensive information on levels of exposure to carbon monoxide in the Australian workforce.

One study investigating levels of carbon monoxide and COHb in Australian workplaces focussed on indoor work areas where carbon monoxide exposure was likely to be significant (such as a foundry, automobile assembly plant, underground mine, automobile plant and indoor car park). The study found carbon monoxide levels of 12 ppm or less and that levels of COHb were found to be related to carbon monoxide exposure. Levels of COHb high enough to cause cardiac problems in persons with pre-existing coronary artery disease occurred in exposed smokers (Wickramatillake et al. 1998).

Another study of blast furnace workers found some readings of COHb in non-smokers of close to 5% (Lewis et al. 1992).

### **5.3 Environmental tobacco smoke**

Most Australian workplaces ban smoking. The main exceptions are service and hospitality workplaces and outdoor workplaces. Based on Victorian data, 70% of indoor workplaces had smoking bans by 2000, and most of these bans were in place by the early 1990s (VicHealth Centre for Tobacco Control 2001). Bans were much less common in outdoor workplaces, with only 20% reporting a total smoking ban and only 45% reporting some form of smoking restriction ((Letcher and Borland 2000) cited in (VicHealth Centre for Tobacco Control 2001)). The extent to which bans on smoking are enforced and actually prevent exposure of workers is not clear, but it appears that they are effective(Chapman et al. 2001).

Exposure to environmental tobacco smoke is highest in workers in service and hospitality occupations such as restaurant workers and bar workers. A 1999 survey of managers in registered clubs in New South Wales found that 59% reported that workers in their establishment were exposed to environmental tobacco smoke at work (Mitchell and Sanders 2002). Following the adoption of non-smoking policies in many offices and other workplaces, exposure is probably much less common in office workers (VicHealth Centre for Tobacco Control 2001).

#### **5.4 Low job control and other psychosocial factors**

A wide range of the Australian workforce is potentially exposed to situations of low job control, sometimes coupled with high job demands. However, the extent of this exposure is not known.

#### **5.5 Noise**

There is no direct measure of the prevalence of exposure to hazardous noise levels in Australian workplaces. However, estimates for 1986 ((Waugh 1986) cited in (National Occupational Health and Safety Commission 2004a)) and 2002 (National Occupational Health and Safety Commission 2004a) (the latter estimate based on exposure data from the early 1990s) both suggested about 12% of the workforce (about one million workers in 2002) are occupationally exposed to hazardous levels of noise.

Workers' compensation data suggest that the occupation groups with the highest exposure (prevalence of exposure and/or intensity of exposure) to hazardous levels of noise are tradespersons, intermediate production and transport workers, and labourers (National Occupational Health and Safety Commission 2004a).

#### **5.6 Shiftwork**

An Australian Bureau of Statistics (ABS) working arrangements survey conducted in November 2003 provides information on the prevalence of shiftwork in Australia. The survey covered all employed persons aged 15 years or over, excluding school students aged 15 to 19 years. Fourteen per cent of the surveyed people reported having performed shiftwork in the previous four weeks. Nearly half (46%) of these workers reported working on a 'rotating' shift roster, with other roster types classified as 'regular morning/afternoon' (13%), 'regular evening, night or graveyard shift' (20%), or 'other' (which included split shift, on-call, irregular shifts and other types of shifts) (21%). Intermediate production and transport workers was the occupation group with the highest prevalence of shiftwork (24%). Industries with the highest incidence of shiftwork were mining (44%), health and community services (32%), accommodation, cafes and restaurants (31%), transport and storage (27%) and personal and other services (25%). Shiftwork prevalence ranged with age from

11% to 16% for workers less than 70 years, but this variation may have been related to differences in occupation or industry. There was little variation with sex (Australian Bureau of Statistics 2004).

### **5.7 Other exposures**

Exposure to methylene chloride occurs in a wide range of occupations, but particularly in furniture stripping. Uses for methylene chloride include as a degreaser, paint remover, aerosol propellant, and in plastics manufacturing (Estill and Spencer 1996). The prevalence and intensity of exposure in the Australian workforce is not known, although there have been at least three fatalities (probably not from an ischaemic heart disease event) since 2000 arising from high level acute exposure to methylene chloride (Driscoll et al. 2004; NICNAS 2004). There is little or no exposure to carbon disulphide or nitroglycerin in Australia.

In terms of longer work hours, the ABS working arrangements survey mentioned above for shiftwork also provided information on overtime work. The survey found that 33% of workers reported performing overtime on a regular basis. Overtime was more common in male workers (44%) compared to female (29%), and full-time workers (46%) compared to part-time (13%). Overtime work prevalence ranged with age from 11% to 40% for workers less than 70 years, with the highest proportions being for workers aged between 25 and 54 years, but this variation may have been related to differences in occupation or industry. Occupations with the highest prevalence of overtime were managers and administrators (63%), professionals (51%), associate professionals (46%), tradespersons and related workers (41%) and intermediate production and transport workers (41%) (Australian Bureau of Statistics 2004).

Exposure to other risk factors occurs in a range of occupations, but the prevalence is not known.

### **5.8 General risk factors**

Several studies have investigated the relationship between general risk factors for CVD and occupation. A study of physical activity found that less-skilled workers were less likely to report both leisure-time physical activity and vigorous leisure-time physical activity, but that combined vigorous activity at work and home was not strongly related to occupation (Salmon et al. 2000). Another study surveyed a rural area and found smoking was associated with any employment (compared to unemployment) and with working in a job that was not at professional or managerial level (compared to persons with a professional or managerial job) (Peach and Bath 1999). Other population based-studies in Australia have produced similar findings of a worse cardiovascular risk profile in lower-skilled workers (Bennett 1996; Opit et al. 1984; Steele et al. 1991; Veitch et al. 1999b).



## **6. OVERALL ESTIMATES OF THE BURDEN OF WORK-RELATED CARDIOVASCULAR DISEASE**

Several studies in different countries have estimated the total burden of work-related CVD. These estimates have been made as part of larger studies examining the role of many occupational exposures in work-related morbidity and/or mortality, or in studies specifically addressing cardiovascular disease. The resulting population-attributable related risks have been based on a variety of exposures, most particularly environmental tobacco smoke, psychosocial factors such as low job control, noise and shiftwork.

The population-based attributable risks ranged from 1% to 22%, but the estimates come from recent studies in the United States and Scandinavia. These studies estimated that work-related exposures resulted in 6% to 18% of cardiovascular disease in the United States (Steenland et al. 2003) and 19% (males) and 9% (females) of CVD in Finland (Nurminen and Karjalainen 2001) (Table 3).

**Table 3** Published estimates of overall population attributable risk for cardiovascular disease related to work

Country	Year	PAR (%)	Exposures included	Conditions included	Age range	Reference
United States	1997	6.3 – 18.0	Noise, low job control, shiftwork, environmental tobacco smoke	"Coronary heart disease"	< 70 years	(Steenland et al. 2003)
Finland	1996	16.9 (all) 18.9 (males) 9.1 (females)	Noise, shiftwork (involved job strain), environmental tobacco smoke, engine exhausts (including carbon monoxide)	Ischaemic heart disease	24 – 74 years (assumed risk decreased from age 60 years)	(Nurminen and Karjalainen 2001)
Australia	1992	1	"Hazardous substances" – excluded environmental tobacco smoke	"Cardiovascular disease"	Not stated	(Morrell et al. 1998)
United States	1992	5 - 10	"Common chemical exposures such as lead, carbon monoxide and solvents"; job strain; psychosocial stress	"Cardiovascular" and "cerebrovascular" disease	< 65 years	(Leigh et al. 1997)
United States	1992	5 - 20	Not stated, but implicitly covered all potential exposures	Two analyses: a "long" list which included all circulatory disease; and a "short" list, which included only ischaemic heart disease; hypertension; atherosclerosis; cerebrovascular disease	Various: < 70 years, < 75 years, and no upper age limit	(Leigh and Schnall 2000)

Table 3 continued

Country	Year	PAR (%)	Exposures included	Conditions included	Age range	Reference
Canada	1989	1 - 3	Ischaemic heart disease; hypertension; atherosclerosis; cerebrovascular disease	Ischaemic heart disease	< 75 years	(Kraut 1994)
Denmark	1980s	16 (male) 22 (female)	Monotonous, high-paced work; shiftwork; noise; "chemical exposures"; environmental tobacco smoke	"Cardiovascular disease"	< 70 years	(Olsen and Kristensen 1991)
New York	1987	5 - 10	Not stated, but implicitly covered all potential exposures	Ischaemic heart disease; hypertension; atherosclerosis; cerebrovascular disease	25 – 64 years	(Markowitz et al. 1989)

\*:

## **7. THE BURDEN OF WORK-RELATED CARDIOVASCULAR DISEASE IN AUSTRALIA**

### **7.1 Introduction**

There is limited information on the extent of work-related CVD in Australia. Information that is available comes from a variety of sources, including published studies, workers' compensation claims data and general practitioner data sources. The available information is presented in this section.

### **7.2 Population-based estimates**

The only estimate of work-related CVD PAR in Australia comes from a study of morbidity and mortality arising from occupational exposure to hazardous substances. This study (conducted by a team lead by Professor Charles Kerr at the University of Sydney, and widely known as "The Kerr Report") did not develop its own PAR estimates, adopting what were believed to be conservative estimates from international literature available at the time of the study (1995). Non-chemical exposures such as environmental tobacco smoke, job control and noise were excluded from consideration because the focus of the study was workplace hazardous substances. Using a PAR of 1%, the study estimated that each year in Australia there were about 800 deaths due to cardiovascular disease arising from occupational exposure to hazardous substances (Morrell et al. 1998). An attribution rate of 3% (presented as part of a sensitivity analysis) resulted in an estimate of 2,400 deaths each year. These estimates caused a lot of controversy when they were released, primarily because the number of deaths was perceived to be too high (Christophers and Zammit 1997). Although some of the criticism was constructive, much of it appeared ill-informed, and there has been no published attempt to provide better estimates (Driscoll 1997).

The Kerr study estimates appear to be based on all persons 15 years or older. It is not clear whether or not there should be a maximum age used. In the recent United States study by Steenland and co-workers (Steenland et al. 2003), an upper age limit of 69 years was used for cardiovascular deaths, the authors arguing that the risk resulting from occupational exposure probably dropped quickly after exposure ceased, and this was presumed to have ceased by about the age of 70 years. The authors commented that, because the number of coronary heart disease deaths increases sharply after the age of 69, their estimates would be conservative if their assumption was wrong. The study by Nurminen excluded people aged more than 74 years, and progressively decreased the risk used for people between aged 60 and 74 years (Nurminen and Karjalainen 2001).

The estimate of an attributable fraction of 1% in the Kerr study clearly results in a major underestimate of the number of work-related cardiovascular deaths in Australia, based on the most recent attributable risk estimates used elsewhere, as shown in Table 1. The most appropriate attributable risk estimate to use is not clear because, as mentioned earlier, it is dependent on both the relative risk and the prevalence of exposure, and the main exposures can be expected to vary considerably between and within countries. However, based on the estimates in other studies, it seems likely the appropriate estimate would be at least 10%, and closer to 15% if only persons less than 70 are considered.

### **7.3 Workers' compensation-based estimates**

Workers' compensation data systems are very unlikely to be a good source of information on cases of work-related cardiovascular disease, for reasons discussed earlier. There are very few situations in which an acute coronary event, or underlying heart disease, in an individual can be confidently connected to occupational exposures. Exceptions might be heart attacks that occur after acute high exposure to carbon monoxide or heavy manual work in very hot conditions, but even in these instances the major cause of the underlying disease might well be non-occupational. Similarly, occupational factors could have been important in the development of ischaemic heart disease that results in someone having a heart attack in a situation that has no connection to work.

Therefore, it is difficult for a claim for work-related ischaemic heart disease to be confidently assessed as work-related, and likely that many instances of work-related heart disease are never recognised by the worker or the treating doctor as being related to work. Conversely, people who sustain an acute coronary event whilst working may make a successful claim for compensation, even though the fact that the event occurred while working was coincidental.

Also, a large minority of workers are not represented in workers' compensation statistics (Macaskill and Driscoll 1998).

These factors mean that workers' compensation data cannot be considered a reliable or valid indicator of the extent of work-related CVD in Australia. Notwithstanding this, national workers' compensation data provide an indication as to whether any such claims are being accepted, and the number of such claims. Over the period 1998/1999 to 2002/2003, there were between 70 and 94 claims each year for ischaemic heart disease (at a rate of between 9 and 12 per million employees), and between 11 and 49 claims for other heart disease (at a rate of between 1.4 and 6.7 cases per million employees). (There were an additional eight to twelve claims for hypertension.) These are gross underestimates, based on the number of deaths (which would be expected to be much less than the number of non-fatal cases) estimated in the Kerr study, which

data from other countries suggest is itself a significant underestimate (Table 4).

#### **7.4 General practitioner-based estimates**

As with workers' compensation data systems, general practitioner data systems can be expected to provide little useful information on work-related cardiovascular disease. Most of the same issues apply to both data sources. In addition, it is more likely that a person with an acute coronary event will go to a hospital emergency department than to a general practitioner.

The BEACH study is the only reliable source of information on work-related consultations with general practitioners in Australia. The study involved a cluster random sample of all general practitioner consultations in Australia. A comprehensive analysis of work-related BEACH consultations covering the years April 1998 to March 2000 identified only 52 cases involving cardiovascular problems, and only nine of these were identified as new problems. Thirty-five per cent of the consultations at which these problems were managed were covered by workers' compensation payments (Hendrie and Driscoll 2003; National Occupational Health and Safety Commission 2001b).

The 52 cases represent only the work-related cardiovascular cases in the study sample. These extrapolate to each year Australian general practitioners managing about 15,000 problems (2,500 new problems) identified as work-related CVD. However, given the small numbers in the sample, the uncertainties that must be associated with diagnosis of any cardiovascular condition as being related to work, and the many cases of work-related CVD that certainly would not be identified through general practice consultations, these should be considered significant underestimates.

**Table 4 Accepted workers' compensation claims<sup>1</sup> for cardiovascular disease and hypertension. Australia, 1998/1999 to 2002/2003**

	1998/1999	1999/2000	2000/2001	2001/2002	2002/2003
<b>Ischaemic heart disease<sup>2</sup></b>					
Number	88	73	99	70	94
Rate <sup>3</sup>	12.1	9.3	12.4	8.6	11.2
95% CI	10.7 – 14.0	9.0 – 9.6	10.8 – 14.4	7.7 – 9.7	9.9 – 12.9
<b>Other heart disease<sup>4</sup></b>					
Number	49	39	38	31	11
Rate	6.7	4.9	4.7	3.8	1.4
95% CI	5.9 – 7.7	4.8 – 5.1	4.2 – 5.5	3.4 – 4.3	1.2 – 1.6
<b>Total heart disease<sup>5</sup></b>					
Number	137	112	137	101	105
Rate	18.8	14.2	17.1	12.4	12.6
95% CI	18.3 – 19.4	14.1 – 14.3	16.5 – 17.8	12.0 – 12.8	12.1 – 13.0

1: Data supplied by NOHSC, from NOSI.

2: TOOCS code 710

3: Cases per million employees

4: TOOCS code 720

5: TOOCS codes 710 and 720

## 8. APPROACHES TO PREVENTION OF WORK-RELATED CARDIOVASCULAR DISEASE

### 8.1 Introduction

The limited specific information about effective prevention activities related to the main work-related cardiovascular risk factors is reviewed in this section.

## **8.2 Carbon monoxide**

Exposure to carbon monoxide comes largely from vehicle exhausts, but can occur in other situations. Prolonged high level exposures can occur if combustion engines are operated in enclosed or confined spaces (e.g. forklifts operated in-doors or pumps operated in a confined space); if the atmosphere in a mine is not properly ventilated after explosives are detonated; and in compressed air diving if the exhaust of the compressor is near the air intake (Gorman et al. 1992; Wickramatillake et al. 1998). Appropriate prevention in such situations should be achievable through the application of standard control procedures using the hierarchy of control. In areas where carbon monoxide exposure may occur, atmospheric monitoring is essential.

## **8.3 Environmental tobacco smoke**

The occupational health and safety acts in place in all Australian states and territories require employers to provide a safe and healthy workplace for all workers. This, and possibly relevant legal cases, have probably been an important stimulus to the adoption of non-smoking policies in most Australian workplaces (VicHealth Centre for Tobacco Control 2001). However, exposure remains in many public hospitality workplaces such as restaurants, clubs and hotels; and in entertainment areas, factories, warehouses and some outdoor workplaces (VicHealth Centre for Tobacco Control 2001; Wiggers et al. 2000). It has been argued that reliance on self-regulation and duty of care in hospitality workplaces will not result in the adoption of non-smoking policies, and that legislation banning smoking in public areas would be a more effective method of protecting the health of employees (Shiell and Chapman 2000). The National Tobacco Strategy supports a combination of education, encouragement and enforcement for workplaces (VicHealth Centre for Tobacco Control 2001). The legislative approach in Australia has been recently reviewed (National Occupational Health and Safety Commission 2001a).

Non-smoking policies appear to be an effective way to decrease occupational exposure to environmental tobacco smoke. A recent New Zealand study of serum cotinine levels (which are directly related to exposure to tobacco smoke) in office workers and hospitality workers found the levels were much higher in hospitality workers who worked in workplaces that did not have smoke-free policies for customers compared to workers in those workplaces that did not allow customers to smoke (Bates et al. 2002; Bates et al. 2001). Another New Zealand study of nicotine levels in hair found very similar results (Al-Delaimy et al. 2001).

The creation of smoke-free indoor areas separated from designated smoking areas has only a limited effect on the concentration of environmental tobacco smoke in non-smoking sections and is therefore an ineffective control. Implementation and enforcement of non-smoking policies appears to be an effective way to decrease occupational exposure



to environmental tobacco smoke, as the use of designated non-smoking areas (Hammond 1999; Mitchell and Sanders 2002; National Occupational Health and Safety Commission 2003; Replace and Lowrey 1980). A recent New South Wales study of indoor air quality in registered clubs found a reduction of about 50% in nicotine levels and 66% in levels of particulate matter (10 µm) (PM10) in the ambient air of designated non-smoking areas, compared to the levels in the smoking areas. However, the level of particulates was still considerably higher than in outdoor areas (Cains et al. 2004). A similar study in South Australia found similar results (Cenko et al. 2004). The New Zealand study of serum cotinine levels found workers in workplaces with designated smoking areas had lower levels than workers in workplaces that did not have designated areas, but the levels were still much higher than in workers employed in workplaces where smoking was not allowed at all (Bates et al. 2002; Bates et al. 2001). In the *Guidance Note on the Elimination of Environmental Tobacco Smoke in the Workplace* {NOHSC:3019(2003)}, NOHSC recommends that exposure to environmental tobacco smoke should be excluded in all Australian workplaces, and that this exclusion should be implemented as soon as possible (National Occupational Health and Safety Commission 2003).

Several studies suggest a high level of public support for banning of smoking in all workplaces, including hospitality workplaces, but concern remains about the potential economic impact of such a change (Borland et al. 1990b; Chapman et al. 2001; Tzelepis et al. 2003; Walsh and Tzelepis 2003; Walsh et al. 2002; Wiggers et al. 2001).

There have been several interventions aimed at encouraging workplaces to implement no-smoking policies. However, the only one to be evaluated found a similar level of improvement in intervention and control worksites (VicHealth Centre for Tobacco Control 2001).

Workplace bans on smoking are designed to eliminate exposure to environmental tobacco smoke, but there is good evidence that they also decrease the number of cigarettes smoked by workers, and possibly also the prevalence of smoking in workers (Borland et al. 1990a; Borland et al. 1991; Chapman et al. 1999). Some of the decrease in smoking may be reversed over time (Owen and Borland 1997).

#### **8.4 Low job control and other psychosocial factors**

Many approaches have been suggested to control the potential impact of psychosocial factors in the workplace. These are examined in detail in the separate profile report on mental disorders (National Occupational Health and Safety Commission 2004b).

## **8.5 Noise**

Approaches to controlling noise in the workplace are well known (National Occupational Health and Safety Commission 2004a). All aspects of the hierarchy of control are relevant, particularly eliminating the noise through engineering improvements, isolating the noise through enclosure of noisy processes, and, where the other approaches are not sufficient, the use of effective personal protective equipment as part of a comprehensive hearing protection program. These approaches are examined in detail in the separate profile report on occupational, noise-induced hearing loss (National Occupational Health and Safety Commission 2004a).

## **8.6 Shiftwork**

Without knowing the mechanism(s) underlying the relationship between shiftwork and cardiovascular risk, it is difficult to plan appropriate interventions. There has been research undertaken concerning optimum shift design from a health and safety perspective. A Swedish study of policemen found lower levels of several CVD risk factors (triglyceride, blood pressure and blood sugar levels) in workers on forward rotation shifts compared to those on backward rotation (Kristensen 1999, 2000). A recent review argued that, in the absence of definitive information, it is appropriate to advise shiftworkers and night workers to eat regularly and to maintain healthy lifestyles (particularly in regard to smoking), and to consider the optimum design and transition for the shift cycle. The organisational aspect was seen as potentially the more important because it could minimise the underlying problem exposures (Boggild and Knutsson 1999).

## **8.7 Health promotion activities**

The worksite has been promoted as potentially a very important place for basing health promotion activities, because certain occupations are known to have a higher level of CVD risk factors and to have a higher risk of CVD (Commonwealth Department of Human Services and Health 1994; National Health and Medical Research Council 1997a; Veitch et al. 1999b). Despite apparent support for the concept, low participation rates have hampered many programs (Barratt et al. 1994; Heaney and Goetzel 1997). Barriers to participation seem to include costs of the program, lack of awareness of potential benefit, reluctance by the workers to be involved in physical activity, and concerns regarding injury risk and loss of productivity (Veitch et al. 1999a). A recent paper suggests that a fundamental component of a successful workplace health promotion program is to include it as part of a comprehensive approach to occupational health and safety prevention (Sorensen et al. 2003).

Where participation has been high, Australian programs of various types, including behavioural counselling and multi-faceted programs, have been shown to result in improvements in cardiovascular risk profiles (Gomel

and Oldenburg 1990; Gomel et al. 1997; Gomel et al. 1993), but this has not been universal (Edye et al. 1989).

In general, however, most of the CVD risk prevention actions outlined will occur as part of national programs to prevent exposure to ETS, hazardous chemicals, and to psychosocial risk factors (as part of the mental disorders) and NIHL more generally.

## 9. NATIONAL PREVENTION ACTIVITY

All NOHSC members are undertaking a range of general prevention initiatives which ultimately impact on the risk of CVDs. NOHSC's recent activities to reduce the risks of cardiovascular disease include the launch of Hazardous Substances Information System in 2005. This online database, which is available via NOHSC's website, allows users to retrieve hazard classification information and exposure standards for over 3 500 hazardous substances, including those that are risk factors for cardiovascular disease. In 2004, the *National Code of Practice for Noise Management and Protection of Hearing at Work* [NOHSC: 2009 (2004)] was released in order to reduce noise exposure at work.

Some national actions are occurring in relation to updating and revising regulations, standards and codes of practice in relation to the control of hazardous substances generally or to specific hazardous substances. New regulations have been or are being developed in most states and territories to prohibit smoking in licensed premises and other public places. For specific information on risk factors such as noise, smoking, hazardous substances, please read other disease profile reports such as those on noise-induced hearing loss, occupational cancer, and occupational respiratory diseases.

For more information on national prevention activities, readers are directed to the following websites:

Department of Health and Ageing [www.health.gov.au](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/>

The Australian Council of Trade Unions (ACTU) <http://www.actu.asn.au/>

The Australian Chamber of Commerce and Industry (ACCI)  
<http://www.acci.asn.au/>

Australasian Faculty of Occupational Medicine (AFOM)  
<http://www.racp.edu.au/afom/>

The National Heart Foundation <http://www.heartfoundation.com.au/>

Baker Heart Research Institute <http://www.baker.edu.au/external/>

## **10. SUMMARY**

Work-related cardiovascular disease is likely to be an important cause of work-related morbidity and mortality in Australia. However, the extent of the problem is difficult to assess accurately, and currently can only be studied through the use of population approaches. Effective prevention activities exist for some exposures, but for others there is insufficient information on prevention activities and their effectiveness.

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## **GLOSSARY**

ABS	Australian Bureau of Statistics
ICD-10	International Classification of Disease
NDS	National Data Set for Compensation-based Statistics
NIOSH	National Institute for Occupational Safety and Health
NOHSC	National Occupational Health and Safety Commission
OHS	Occupational Health and Safety
TOOCS	Type of Occurrence Classification System