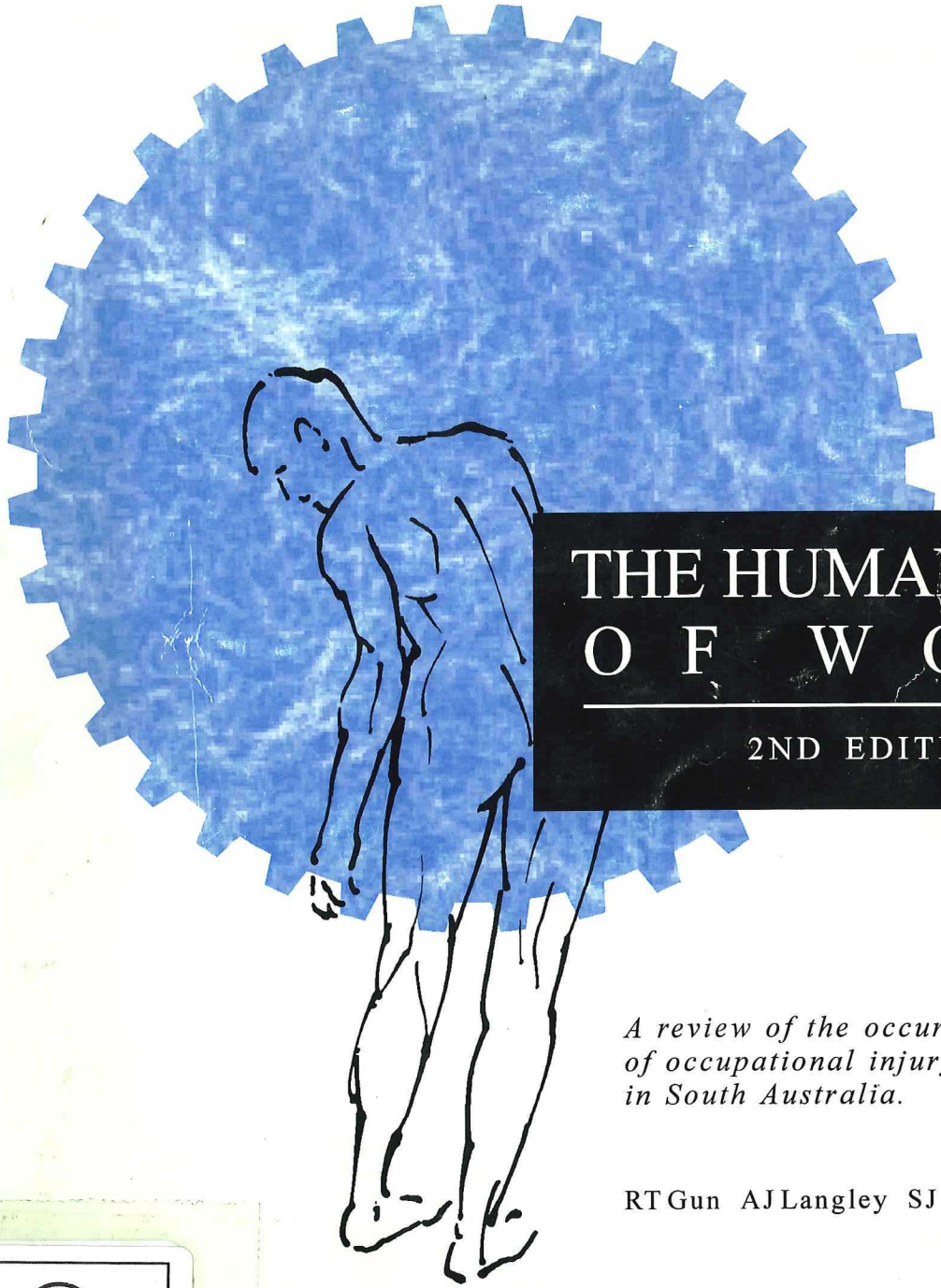

THE HUMAN COST OF WORK

2ND EDITION

A review of the occurrence and causes of occupational injury and disease in South Australia.

RT Gun AJ Langley SJ Dundas K McCaul

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SOUTH AUSTRALIAN HEALTH COMMISSION

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The objectives of this publication are to:

- detail the incidence and the prevalence of occupational injuries and diseases in South Australia
- highlight the public health importance of occupational injury and disease in South Australia
- provide sufficient information to rank, in general terms, the principal causes of occupational morbidity and mortality in South Australia both in total and for the major industry classifications
- identify deficiencies in current statistical databases.

We hope that the publication will enable:

- priorities to be established for occupational health and safety interventions at a State-wide and industry level; and
- improvements in statistical databases directed to the collection of occupational morbidity and mortality data.

Preface

This volume is an update and a revision of *The Human Cost of Work*, published by the South Australian Health Commission in 1987. It represents the first opportunity to revisit the available occupational injury and disease data following the introduction of the Occupational Health Safety and Welfare Act of 1987. Unfortunately a comparison with previous statistics has been complicated by the introduction of the WorkCover system in the same year as the new health and safety act. Since the data are based on successful compensation claims, which are in turn heavily influenced by the entitlements to compensation, the trends in reported injury are very sensitive to changes in the compensation legislation and its manner of administration. This renders the task of evaluating underlying injury trends quite difficult. However injury rates have been obtained which have been adequate to obtain a valid comparison.

This project has been undertaken under the auspices of, and with financial assistance from the Epidemiology Branch of the Public and Environmental Health Service.

The project has been supported by a grant from the Research and Education Committee of the WorkCover Corporation. Thanks are due to Jan Powning, Chairperson, and Louisa Bowes, Senior Research Officer, for the assistance. A grant was also provided by the Occupational Health Division of the Department for Industrial Affairs. Thanks are due to Milton Lewis, Director, for the assistance of the Division.

Thanks are due also to the Research and Analysis Unit of the WorkCover Corporation for providing extensive data and many *ad hoc* analyses; to the staff of the Adelaide office of the Australian Bureau of Statistics (ABS), and Brian Richings of the Canberra office of the ABS for information from the 1989-90 National Health Survey; to David Wilson, Sally Brinkman, Kevin Priest, Lesley Adlam and Anne Taylor of the Public & Environmental Health Service; the staff of the South Australian Health Commission Library; Anne Halford from the Australian Bureau of Statistics; Rhonda Pfeiffer from the South Australian Health Commission's ISIS database; Elizabeth King from the Injury Prevention Forum; Chris Cobb from Births, Deaths & Marriages; John Mobberly from Comcare for the supply of Commonwealth injury data; Jenny Cassidy from the Department for Industrial Affairs for State Government data; Tim Dale from the Australian Bureau of Statistics (Canberra) for providing specific data from the 1989-90 Australian Health Survey; and David Roder, Director, Epidemiology Branch, Public & Environmental Health Service for his support and assistance.

The text represents the personal views of the authors which are not necessarily the views of their organisations or the South Australian Health Commission.

Contents

Methods & data sources

Injury data

The basic injury data were obtained from the Research and Analysis Unit of the WorkCover Corporation. The unit also undertook specific analyses on back injuries and RSI according to our definitions, so as to provide data which were comparable to pre-WorkCover data from the ABS.

Injury statistics for State Government employees were obtained from the Government Workers Rehabilitation and Compensation Office in the SA Department of Industrial Affairs. This agency does not insure the SA Health Commission, the State Transport Authority (TransAdelaide) or the Electricity Trust of South Australia. SA Health Commission and local government injury data were obtained from WorkCover. Injury data for Commonwealth Government employees in South Australia were obtained from Comcare.

The workforce injury data were obtained from the ABS annual series *Industrial Accidents - South Australia, Catalogue No 6301.4*, now discontinued.

For deriving accident rates, the accident numbers were divided by the number of employed persons in the labour force, excluding Commonwealth Government employees and the self-employed. The labour force data were obtained from the 1971 to 1991 Censuses. For estimates in inter-censal years, the numbers were interpolated from the preceding and succeeding Censuses, assuming a linear increase in number of persons employed. The numbers employed in the State Government Sector were obtained from the Office of the SA Commissioner for Public Employment. Employment numbers in the SA Health Commission were obtained from the Health Commission itself, and in local government from the 1991 Census.

The number of Commonwealth employees in South Australia was estimated from the 1991 Census. For deriving rates in Commonwealth employees insured through Comcare, an adjustment had to be made for the estimated number of employees in Australia Post and Telecom, which insure separately.

Comparisons were also made between injury rates of government employees - State, local and Commonwealth - and the overall workforce. However the rates are not strictly comparable, because of the varying proportion of employees in manual occupations. For example, the State and Commonwealth Government workforces have relatively few manual workers, and this factor would be expected to produce a lower lost-time injury rate for that reason alone. Local government on the other hand has a relatively high proportion of manual workers. Accordingly, the data for the overall workforce were standardised by applying occupation-specific rates to the numbers employed in the corresponding occupational categories in the public sector. In this way an estimate was made of the expected injury rate in the overall workforce if its occupational categories were distributed in the same proportions as in the government workforce. This estimate was approximate only, since the numbers employed in each occupation category in the public sector were not obtainable directly, and had to be estimated from 1991 Census data.

For information on the prevalence of back disorders in the population, data were extracted from the 1989 Australian Health Survey, and an analysis of prevalence according to industry and occupation, stratified by age and sex, was supplied by the Australian Bureau of Statistics.[1]

Cancer analyses

International trends in cancer rates and the role of occupation were reviewed from a range of sources in the scientific literature, but two especially important sources were the monographs of the

International Agency for Research on Cancer[2], and Volumes 19/20 of the series Cancer Surveys (Cold Spring Harbor Laboratory Press) - Trends in Cancer Incidence and Mortality.[3]

Comparison of mortality rates between blue collar workers and white collar workers were undertaken for lung cancer, bladder cancer and leukaemia. Rates could not be computed directly from population statistics, since the census data only give the respondent's current occupation, which in the age groups of most cancer patients, is "retired" or "pensioner". Accordingly proportionate rates were estimated from the method of Miettinen and Wang,[4] in which the numbers of cancer deaths in the occupational group of interest (eg blue collar workers) are expressed in proportion to the number of "ancillary" diseases unrelated to the factor of interest, which in this case is social class (ie blue collar/white collar). The "ancillary" diseases were cancers of the rectum and rectosigmoid colon, pancreas, kidney and brain, none of which are significantly related to social class as assessed by broad category of occupation.

To estimated the contribution of smoking to the difference between the cancer rates of white collar and blue collar workers, smoking prevalence data for these strata were obtained from the SA Health Omnibus Survey for the years 1990-95.[5]

Use was also made of the annual report of the South Australian Cancer Registry. The estimate of the proportion of cancers attributable to occupation was in some instances based on comparisons of the male and female cancer incidence rates in different age strata. Use was also made of the 1991 report of the Cancer Registry, which included an analysis of observed to expected cancer rates of different cancers between 10 broad categories of occupation.

Using the above data sources, a variety of methods were opportunistically used to derive the proportion of current incident cancers attributable to occupation.

To estimate the number of workers *currently* exposed to carcinogens, a telephone survey of possible users was undertaken by the Occupational Health Division of the Department of Industrial Affairs. Employment numbers in the relevant industry categories were obtained from Census data and from WorkCover.

To derive the estimates of the number of cancers generated from current exposures to carcinogens, the mean of the age-specific cancer incidence rates for each of the 5-year age strata from 55-59 years to 75-79 years was multiplied by the attributable risk ([RR-1]/RR) for the cancer for those exposed, and multiplied by the number of exposed workers. The age-specific rates were obtained from the 1994 SA Cancer Registry, and the relative risks (RR) were obtained from an approximate mean of relative risk estimates cited in the relevant monographs of the IARC. The current workforce was assumed to represent the number of person-years of a cohort employed for an average period of 10 years, and to contribute cancers at the calculated rate each year from 10th to the 40th year from the closure of the cohort. Thus the workforce is assumed to have "turned over" every 10 years, and three successive 10-year cohorts will contribute to the excess cancers in any future year after the 10-year lag time; accordingly the estimated proportion of cancers attributable to occupation in any one year will be 3 times the estimated contribution from the 1985-95 cohort.

Effects of physical hazards

The prevalence of noise-induced hearing loss was estimated from SA Health Omnibus Survey for 1994, which was analysed for hearing loss attributed to occupational noise, according to occupation. The occurrence of hearing loss was recorded if the respondent answered in the affirmative to the question "Do you usually have difficulty hearing what people say to you in a quiet room (either) when they speak loudly to you, if they speak normally to you, (or) if they whisper to you?" Information was also obtained about severe hearing loss, ie requiring the use of a hearing aid.

WorkCover data on claims for noise-induced hearing loss were obtained. To succeed, a claimant must have at least a 5.1% noise-induced hearing loss, weighted in the manner defined by the National Acoustic Laboratories (Publication No. 188 of 1988), after allowance for presbyacusis. The data do not include claims paid by exempt employers.

Data on heat strain, effects of cold and pressure were supplied by WorkCover. The data included dates of heat strain effects, and ambient temperature for those days was obtained from the Bureau of Meteorology.

Respiratory disease

WorkCover data on respiratory disease were used. SA death records for 1989 to 1993 were scanned for deaths from lobar pneumonia in welders, and for deaths from pneumoconioses.

Comparative data on pneumoconioses were supplied by the NSW Dust Diseases Board.

A questionnaire on the occurrence of respiratory diseases was sent to registered specialists in respiratory medicine practising in South Australia, and a copy of the questionnaire is reproduced in the Appendix.

Skin diseases

WorkCover data on compensable skin diseases and Australian Health Survey data were used for this analysis.

Infective & parasitic disease

Data on claims for skin conditions were supplied by WorkCover.

Psychological disorders

WorkCover data were obtained for claims for compensable mental disorders, and corresponding data for State and Commonwealth employees in SA were obtained from the GWRCO and Comcare.

National Death Index data have only started to incorporate occupational data from 1993, and only data for that year were available. Accordingly an analysis of suicide rates for Australia according to occupation was undertaken, using occupational data from the 1991 Census as denominators. The South Australian death registry was used to calculated suicide rates by occupation for the period 1990-93.

Miscellaneous effects of chemical exposures

To estimate the occurrence of systemic diseases according to occupation, a questionnaire was circulated to haematologists, nephrologists and neurologists seeking information on conditions they have seen which might have been work-related. The diseases and occupations selected were based on the Sentinel Health Events (Occupational) developed by the US National Occupational Institute for Occupational Health and Safety, and the questionnaires are shown in the Appendix.[6]

WorkCover data for injuries caused by chemicals were analysed according to the types of chemical.

SA hospital separation data for chemical poisonings were obtained, and those occurring in a factory, a mine or a farm were analysed according to the chemical agent.

SA death registry data for chemical deaths were examined, but they do not indicate which deaths were work-related.

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1. Injuries

R T GUN

Trends in accidents and injuries, 1970-71 to 1990-91 - trends in occupational fatalities - injury rates by occupation.-.some occupations of interest - injury rates by industry - some industry groups of interest - State Government employees - Local Government - Commonwealth Government.

Summary

The first injury data since the introduction of the SA Occupational Health, Safety and Welfare Act 1986 show an increase in the number of reported injuries. This is due to an increase in the proportion of all injuries resulting in more than 5 days of lost time, which are the only injuries included in the published statistics. However the total number of injuries in the 3-year period 1988-89 to 1990-9 is unchanged. Data on deaths from industrial injury are available up to 1993-94, and have shown a sustained fall over the 3-year period 1991-92 to 1993-94.

As expected, the occupational injury rate is highest in the manual occupations. The highest rates were in the category male labourers and related workers, in which the injury rate was 25 times the rate in male professional workers. After allowing for differences in hours worked, the injury rate was 75% higher in men than in women.

Industries with the highest injury rates in men were manufacturing, especially the metal and food industries, construction, road transport and storage. In women the highest rates were in miscellaneous manufacturing and manufacturing of transport equipment.

The highest rate of severe injuries (100% permanent disability) occurred in road transport.

The average number of compensated lost days per injury increased 3-fold for the year 1988-89 compared with the previous published statistics in 1986-87. In 1990-91 the average number had fallen but was still double that of 1986-87. However the increase was due to the increased duration of entitlement to benefits in the new workers' compensation system, so that there is no reason to suspect any increase in the number of severe injuries.

After allowing for different occupational profiles, the lost-time injury rate for male State Government employees was approximately equal to the rate of the overall male workforce. For females the rate was approximately one third higher than that of the overall female workforce. There was an especially high rate in female employees of the South Australian Health Commission. In local government on the other hand the rate for males was approximately 50% higher than that of the overall male workforce but the female rate was approximately equal to the rate of the overall female workforce. For Commonwealth Government employees in SA (excluding Telecom and Australia Post), male and female rates were greater than those of other employed workers in South Australia (approximately 60% and 100% respectively).

Trends in accidents & injuries, 1970-71 to 1990-91

This analysis covers a 20-year period punctuated by 3 significant changes, all implemented in October 1987.

(i) The introduction of the WorkCover scheme, which transferred the workers compensation insurance for most employers to the WorkCover Corporation. Some large employers, including State and Local Governments, retained the right to self-insure, although they were still bound by the requirements of the WorkCover Act. Only Commonwealth Authorities remained outside the scheme.

(ii) With the introduction of the WorkCover scheme, the responsibility for collecting data for compensable injuries was transferred from the ABS to the WorkCover Corporation.

(iii) The introduction of the Occupational Health, Safety and Welfare Act of South Australia. This is based on the same principles as the UK Health and Safety at Work Act. Its main features are reliance on employers to identify and control hazards in their own work places rather than on detailed requirements spelt out in regulations, and establishment of consultative structures in the workplace, including management and employee representatives, to formulate safety policies and practices.

All three measures profoundly affect the injury statistics. The last statistical bulletin produced by the ABS was for the year 1986-87. WorkCover published no data for 1987-88, and by the time the first WorkCover statistics appeared in 1994 (for the years 1988-89 to 1990-91) the ABS industrial injury reporting unit had been disbanded. This created difficulties in ensuring that pre- and post-WorkCover data were comparable.

The average number of lost days per claim is of critical importance to the published statistics, because they only relate to claims resulting in more than 5 days or more lost time, which are only about one fifth of all claims. Thus an increase in the proportion of claims which lead to more than 5 days of lost time will show an increase in the injury statistics, even though the accident frequency is unchanged. Since this proportion is likely to vary according to the level of benefits and the efficiency of the rehabilitation process, the injury statistics are heavily influenced by the WorkCover system itself.

These influences on the injury statistics complicate the task of determining trends in injury numbers following the introduction of the new occupational health and safety legislation (the Occupational Health, Safety and Welfare Act of 1986).

The cumulative injury incidence rates for the period 1970-71 to 1990-91 are shown in Table 1.1. The data are compiled from ABS data up to 1986-87 and from WorkCover data thereafter. Both data sets are for occupational injuries only, ie they exclude journey accidents and occupational diseases, and cover only accidents which resulted in a successful compensation claim where there were more than 5 days of lost working time.

The main features of the data are a steady increase in injury rates from the early 1970s, a reduction in rates in the late 1970s which was sustained through the 1980s, and a sharp increase from the late 1980s with the commencement of WorkCover and the new occupational health and safety legislation.

The increase in the number and rates of claims in the early 1970s may be due to significant increases in the level of benefits enacted at that time - since when injured workers have received virtually the same level of benefits as they would have received in wages if they had remained at work.

The decline in the injury rate in 1982-83 may be partly due to the effects of the economy, which went into recession in the early 1980s. An economic downturn can influence the number of injuries and the number of claims in a number of ways:

- workers are concerned about their job security and may be reluctant to make claims
- less experienced workers are laid off
- overtime may decrease.

However this is unlikely to be the full explanation, since the low rate was sustained into the mid-1980s, by which time the economy had recovered. An ABS bulletin in the early 1980s attributed the fall in reported accident numbers to amendments to the workers' compensation legislation.[1] However there was no amendment at that time which was likely to affect the accident data in a significant way.

Table 1.1. *Injury numbers (with more than 5 days of lost time) and rates, South Australia, 1970-71 to 1990-91. Source: ABS to 1986-87, WorkCover 1988-89 to 1990-91.*

Year	*Number of injuries (persons)*	*Rate (injuries/1000 person/years)*
1970-71	9500	23.1
1971-72	12000	28.7
1972-73	15000	35.4
1973-74	17000	39.5
1974-75	20000	45.9
1975-76	18625	42.1
1976-77	15040	33.8
1977-78	13420	30.1
1978-79	12253	27.3
1979-80	12613	28.0
1980-81	14013	31.0
1981-82	14015	30.8
1982-83	10882	23.7
1983-84	10604	23.0
1984-85	11551	24.9
1985-86	11779	25.3
1986-87	10878	22.9
1988-89	14213	29.0
1989-90	16196	32.5
1990-91	14068	27.8

(Injury estimates were revised in years following original publication. However the estimates for 1986-87, the last year of ABS data, were not revised and may therefore be too low.)

The increase in injury numbers and rates from 1988-89 cannot be explained in the level of benefits, since these did not significantly change with the introduction of WorkCover. The most likely explanation is an increase in the proportion of claims resulting in more than 5 days of lost time.

As mentioned above, the official statistics relate only to claims resulting in more than 5 days in lost time. If for any reason the proportion of such claims to all claims increases, the statistics may show an increase. To test whether this explains the increase in accident numbers and rates, an indirect comparison was made of the pre-WorkCover proportion of claims of more than 5 days to all claims can be made with the proportion post-WorkCover. The former were computed from ABS data on all effective claims, which were published as an Appendix in the ABS bulletins. Comparable post-WorkCover were available, although only from companies insured through WorkCover, not from exempt (ie self-insuring) employers. These two sets of figures are compared in Table 1.2.

Table 1.2. *Claims resulting in more than 5 days of lost time, as a percentage of all effective claims, 1970-71. Source: ABS to 1986-87, WorkCover 1988-89 to 1990-91. (These data include journey and recess claims.)*

Injury year	*Claims >5 days lost time*	*Total claims*	*% Proportion*
Pre-WorkCover			
1970-71	9500	56600	16.8
1971-72	12000	61000	19.7
1972-73	15000	75000	20.0
1973-74	17000	87000	19.6
1974-75	20000	84000	23.8
1975-76	18625	78000	23.9
1976-77	15040	75100	20.0
1977-78	13420	66500	20.2
1978-79	12253	64900	18.9
1979-80	12613	69700	18.1
1980-81	14013	72400	19.4
1981-82	14015	73400	19.1
1982-83	10882	60800	17.9
1983-84	10604	58200	18.2
1984-85	11551	63800	18.1
1985-86	11779	67200	17.3
1986-87	10878	64100	17.0
WorkCover (non-exempt employers only)			
1988-89	12056	51110	23.6
1989-90	14033	56520	24.8
1990-91	11795	49345	23.9
1991-92	9309	40470	23.0
1992-93	9225	39210	23.5
1993-94	9670	40550	23.8

The Table shows that with the advent of WorkCover there was an immediate and sustained increase in the number of accidents leading to more than 5 days of lost time in proportion to all accidents, from 18% to 23%. *

This demonstrates that the increase in the number of claims with more than 5 days of lost time since the introduction of WorkCover, as shown in the official statistics, is the result of a higher proportion of such claims as a proportion of all claims, rather than an increase in the total number of claims.

The amount of lost time per claim has also risen steeply for claims of more than 5 lost days. This is also seen in Table 1.3, which shows that the total lost time and the average lost time per injury trebled for the first full year post-WorkCover. The figures fell in subsequent years, but were still more than double the pre-WorkCover figures in 1990-91.

This increase following the introduction of WorkCover has been so dramatic that a close examination has been made as to whether the figures are comparable. However there is no obvious difference in the way the WorkCover and the ABS compiled the statistics to account for the difference.

* *The post-WorkCover figures only cover non-exempt employers, but this is unlikely to be a significant source of bias, since since over 80% of the claims with more than 5 days of lost time were in non-exempt employers. Thus for example if the proportion of 23% leading to more than 5 days of lost time applies to all employers, the actual total number of claims for all employers in 1990-91 would be about 59000, which is comparable to the number of claims in the mid-1980s.*

The increase in the average lost time per claim is likely to be due to legislative changes in the duration of benefits. Under the previous legislation, weekly benefits ceased when the total payment reached a certain level (although a lump sum redemption might be payable at the same time), whereas under the WorkCover legislation benefits continue until the worker returns to work or reaches retiring age, whichever occurs earlier. Thus since the advent of WorkCover there have appeared a large number of workers who would have been discharged from the system under the old scheme but who are now eligible to receive continued benefits.

Table 1.3. *Time lost in industrial accidents, and average lost time per accident, 1970-71 to 1990-91 (for accidents resulting in more than 5 days of lost time).*

Year	*Total days lost*	*Average days lost per injury (in excess of 5)*
1970-71	36245	3.81
1971-72	44267	3.68
1972-73	57590	3.83
1973-74	67202	3.95
1975-76	96407	5.17
1976-77	83694	5.56
1977-78	74778	5.57
1978-79	77448	6.32
1979-80	75990	6.02
1980-81	78849	5.62
1981-82	84246	6.01
1982-83	64374	5.91
1983-84	66902	6.30
1984-85	82080	7.10
1985-86	90587	7.69
1988-89	303507	21.3
1989-90	282720	17.4
1990-91	197890	14.0

Table 1.4 gives a comparison of the distribution of injuries according to time lost (males only) for 1985-86 and 1990-91, ie before and after the introduction of WorkCover.

The table shows that there has been a large comparative increase in the numbers of long-term disabilities. Thus there has been a 50% increase in the number of claims with 13-25 weeks of lost time, almost a 75% increase with 26-51 weeks, almost a trebling for 52-103 weeks, and 435 claims with more than 2 years compared with 8 in 1985-86. Comparable increases have occurred in female workers.

Table 1.4. Distribution of time lost in accidents in 1985-86 and 1990-91 - males.

Time lost (weeks)	*Total number of accidents*	
	1985-86	*1990-91*
<2	3166	4067
2-3	2398	1867
4-5	919	1003
6-7	557	603
8-12	590	783
13-25	425	679
26-51	263	453
52-103	133	376
104-155	7	351
156+	1	84
Total	8459	10266

A comparison of the distribution of lost time per injury pre- and post-WorkCover (Figures 1.1 and 1.2) show the *proportions* of claims which resulted in long periods off work also escalated after WorkCover was introduced.

The data suggest that, contrary to expectations, the abolition of lump sum redemption payments did not succeed in shortening return-to-work rates.

Probably the most important issue is whether there has been any change in the number and severity of injuries. As discussed above, the increase in the number of injuries in the published statistics indicate nothing more than that the changes in the compensation scheme are causing more claims of more than 5 days of lost time than previously, and do not mean that there is a true increase in the number of accidents occurring. Similarly, the increase in the average lost time per injury is probably not due to any increase in the physical severity of the average injury, but is also due to changes in the duration of entitlement to weekly benefits.

However, although the number of injuries has not risen, it has not fallen either. There is thus no evidence in the injury statistics that the Occupational Health, Safety and Welfare Act resulted in any improvement in health and safety performance in its first 4 years of operation. However, bearing in mind that the subordinate legislation (regulations and codes of practice) and the administrative structures take a considerable time to be put in place, it is probably premature to make a judgement on this legislation.

Fatal injuries

Fatality rates are also taken from the compensation statistics. These statistics under-enumerate the true rate of fatal accidents, since they exclude most self-employed persons, including many in high-risk industries such as farming or fishing. Worksafe Australia was undertaking a nationwide occupational fatality study, based on coroners' reports, while this report was being prepared; however the results were not available in time for publication. Although the compensation statistics under-enumerate the occupational fatality rate, they are a useful index of trends over time in employed persons, since unlike lost-time injuries they are not affected by changes in the compensation system.

The number and rate of fatal accidents from 1975-76 to 1993-94 are shown in Table 1.5.

Fatality numbers and rates, being much lower than total injuries, are more prone to fluctuate from year to year . The death rates have in fact varied by a factor of 2.5 over the 20-year period, ranging from 11 in 1977-78 ($2.46/10^5$ person-years) to 31 in 1988-89 ($6.33/10^5$ person-years).

The most salient feature of the statistics is that the fatality rate has shown a sustained decrease in the past 3 years, so that the average rate is the lowest of any 3-year period since 1975-76, when the comparative data begin.

Table 1.5 *Work-related fatalities and fatality rates, 1975-76 to 1993-94 (excludes deaths from diseases).*

Year	*Number of fatalities*			*Fatality rate*
	Male	*Female*	*Total*	*(deaths/100,000py)*
1975-76	*	*	18	4.1
1976-77	*	*	16	3.6
1977-78	*	*	11	2.5
1978-79	*	*	20	4.5
1979-80	*	*	21	4.7
1980-81	*	*	20	4.4
1981-82	*	*	21	4.6
1982-83	*	*	26	5.0
1983-84	*	*	16	3.5
1984-85	*	*	16	3.5
1985-86	*	*	14	3.0
1986-87	*	*	11	2.5
1987-88**	9	0	9	1.9
1988-89	28	1	29	5.9
1989-90	20	0	20	4.0
1990-91	20	0	20	4.0
1991-92	10	1	11	2.2
1992-93	12	3	15	2.9
1993-94	10	3	13	2.5

* *Number of fatalities by gender unavailable before 1984-85*

** *1987-88 data are for 9 months only*

Thus unlike the total injury rates, the fatality rates have gone down, but only since 1991-92, that is, over the most recent period, for which total injury rates are not yet analysed. Thus the fatality data for 1991-92 to 1993-94 are suggestive of some improvement following introduction of the Occupational Health, Safety and Welfare Act. However it is not necessarily the new legislation which is responsible for the improvement. It is possible that the low figures for this period are a function of random variation, since a comparable rate occurred previously, in 1977-78. It is also possible that the improvement is a continuation of a long-term trend, which as seen from Figure 1.3, has been downwards over the 20-year period. However this is only a weak trend ($r = -0.31$, not statistically significant). It is thus premature to conclude that the new legislation has resulted in an improvement, but this conclusion is consistent with the fatality statistics. It will be of interest to see the total injury rates for the period 1991-92 to 1993-94 when they become available.

The trend in fatality rates is also shown graphically, in Figure 1.3.

The fatality numbers for the years immediately preceding WorkCover (1984-85 to 1986-87) are probably an underestimate. It was the practice of the ABS to revise annual fatality statistics upwards in the years following their original publication, probably to add fatalities in which the coroner's findings were not available in the year of occurrence. From 1984-85 the ABS discontinued this revision.

Injury rates by occupation

Table 1.6 shows mean injury rates by occupational category for the first 3 years of the WorkCover statistics.

Figure 1.1: Non-Fatal Accidents: 1985-86/1990-91
Males

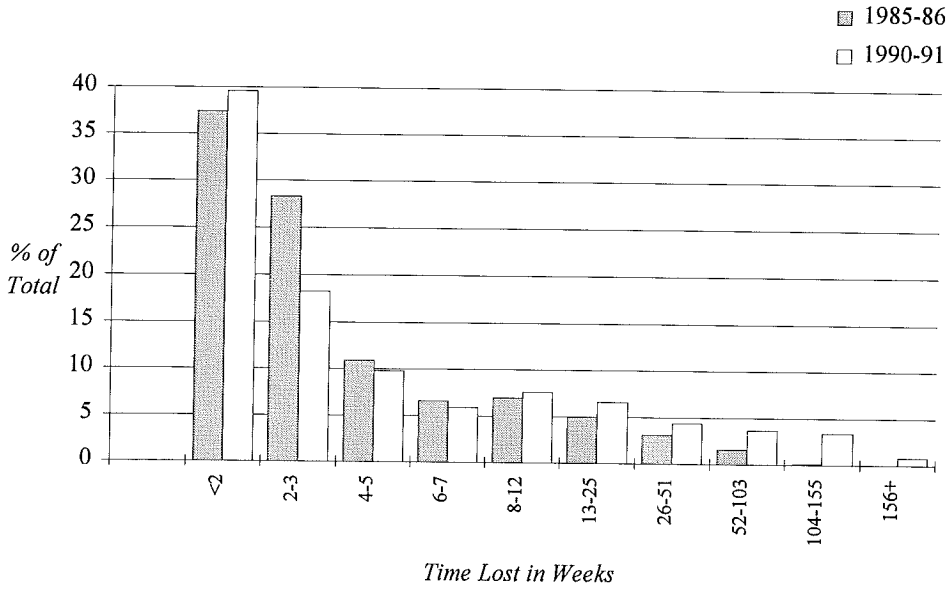


Figure 1.2: Non-Fatal Accidents: 1985-86/1990-91
Females

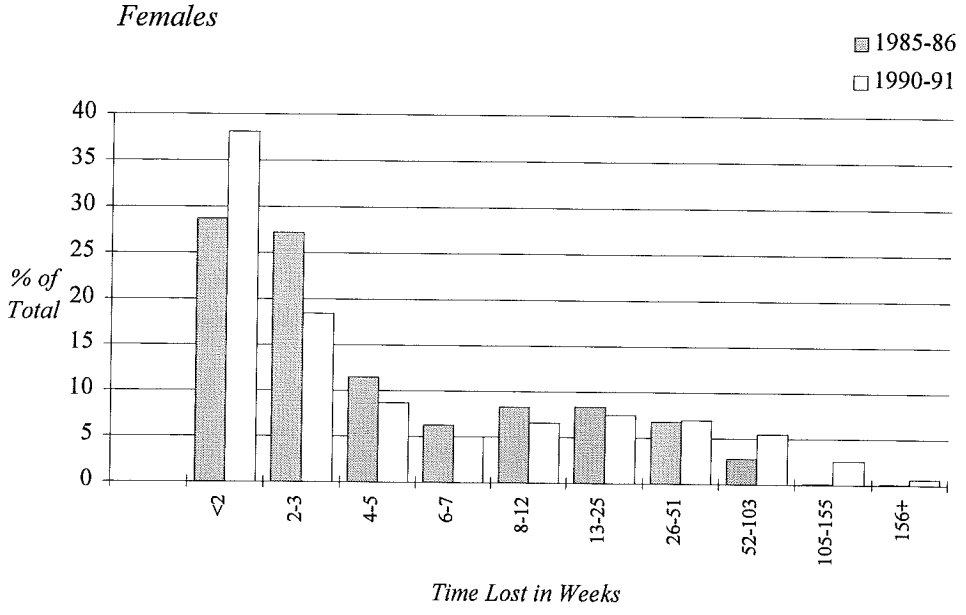


Table 1.6. *Industrial injury rates (number of injuries/1000 person-years) by occupational category by gender, 1988-91.*

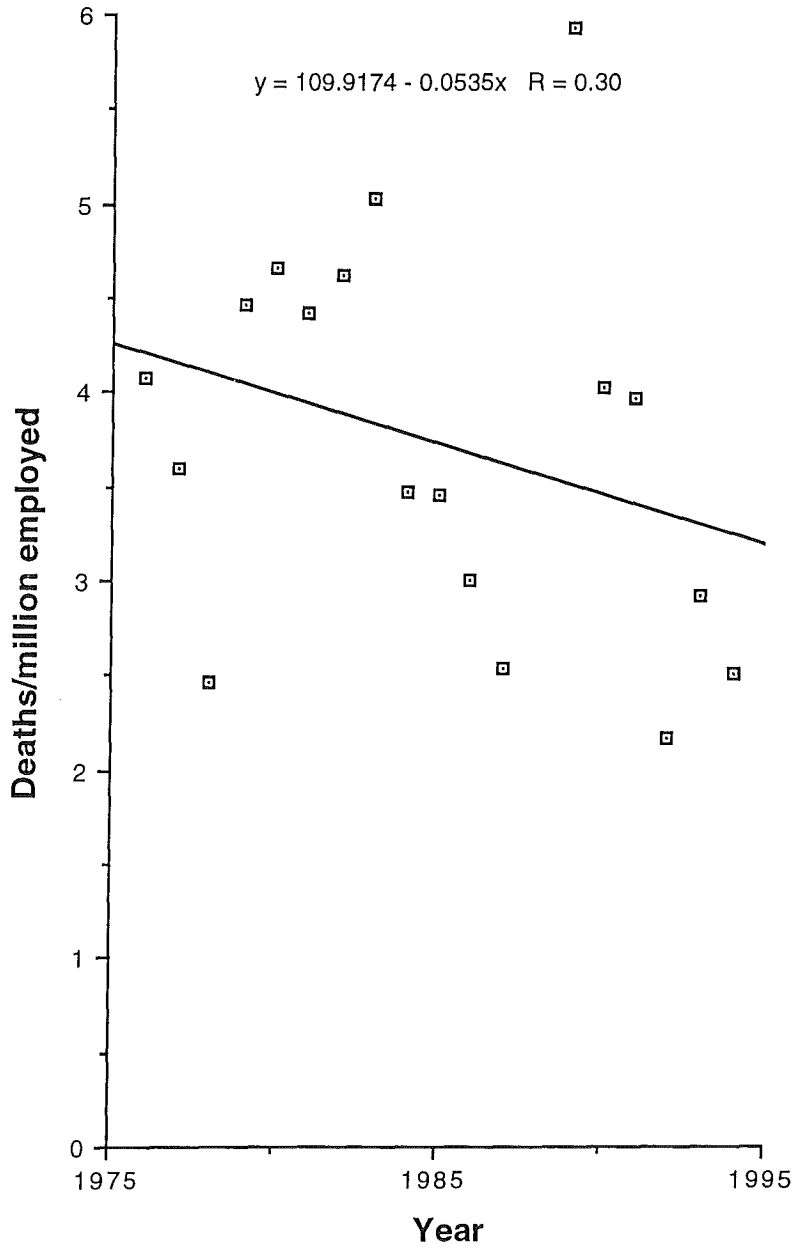
Occupation	*Males*	*Females*
Managers & administrators	6.2	4.6
Professionals	3.9	6.5
Para-professionals	24.3	19.9
Tradespersons	64.7	22.8
Clerks	8.5	5.6
Salespersons & personal service workers	11.7	14.3
Plant & machine operators & drivers	77.1	43.8
Labourers & related workers	102.8	52.4
Total	44.0	18.3

There is a large variation - more than 25-fold - between the category with the lowest rate (professionals) to the highest (labourers and related workers). By far the highest rates of injury occur in blue-collar workforce. This finding is expected, not only because manual work is likely to be more physically hazardous than non-manual work, but also because manual workers, because of their job demands, are less likely to be fit to return to work within 5 days following injury and are thus more likely to appear in these statistics.

Amongst individual occupational categories, the following showed the greatest accident numbers:

	Mean number of injuries/year
Males	
Other trades assistants & factory hands	1141
Truck drivers	763
Structural steel, boilermaking & welding tradesmen	633
Metal fitters and machinists	631
Storemen	413
Farm hands and assistants	338
Other construction & mining labourers	331
Carpenters & joiners	298
Vehicle mechanics	271
Assemblers	206
Females	
Cleaners	483
Registered nurses	305
Ward helpers	305
Other trades assistants & factory hands	220

Figure 1.3: Occupational fatality rates in SA, 1975-1994.



Male rates vs female rates

As shown in Table 1.6, the injury rate for females is only about 40% of that of males. The lower female rate is probably a reflection of differing types of tasks performed rather than any biological differences in accident proneness. Thus in the sales category, where male and female workers are likely to perform similar tasks, the accident rates are similar for the two sexes, whereas in the category of unskilled labourers, where there is likely to be little similarity in the tasks performed, males have double the accident rates of females.

However, part of the difference in rates between men and women is due to different exposure times. Since a higher proportion of female workers are employed part-time, women will, on average, be less likely to incur an occupational injury.

The extent to which this factor accounts for the difference can be quantified by measuring accident rates in terms of number of accidents per million hours worked (ie the frequency rate) rather than as number of accidents per thousand persons employed as in Table 1.6.

The method of doing this calculation was as follows:

(i) The ABS Bulletin *Labour Force, South Australia*, was used to derive the average number of hours worked per week for male workers and female workers for each quarterly period 1988-1991. It was calculated that males worked 39.95 hours per week and females 28.18 hours per week on average.

(ii) The male and female workforce numbers were taken from the 1991 Census.

(iii) It was then calculated that the average frequency rate for the 3-year period was 21.4 injuries/million hours worked for men, and 12.3/million hours worked for women.

The frequency rate ratio (males/females) was estimated at 1.75, compared with a ratio of 2.48 if no allowance is made for the difference in average number of hours worked.

Thus it is concluded that male work, on average, carries about a 75% higher risk of a lost time injury compared with female work, after allowing for the reduced mean exposure time of females.

Injury rates by industry

Table 1.7 shows mean injury rates by occupational category for the first 3 years of the WorkCover statistics.

In male workers, manufacturing industry stands out as the most important source of injuries. 40% of the injuries occurred in the manufacturing sector, although it employs only 25% of the male workforce. Thus manufacturing industry has a relative risk of injury of 1.6, ie a 60% greater risk of injury than the average for the total male workforce.

Table 1.7. *Industrial injury rates (number of injuries/1000 person-years) by occupational category by gender, 1988-91.*

Industry	*Males*	*Females*
Agriculture, forestry, fishing & hunting	49.2	15.0
Mining	59.2	14.3
Manufacturing		
Food, beverages and beverages	86.1	41.4
Textiles, clothing and footwear	48.3	30.9
Wood,wood products & furniture	77.6	32.6
Paper, paper products, printing & publishing	29.1	20.7
Chemicals, petroleum & coal products	31.4	6.8
Non-metallic mineral products	91.8	8.6
Basic metal products	106.1	40.9
Fabricated metal products	108.1	38.5
Transport equipment	92.4	74.1
Other machinery & equipment	54.1	33.4
Miscellaneous manufacturing	73.1	96.8
Total manufacturing	73.4	40.1
Electricity, gas & water	49.9	15.4
Construction	84.4	6.1
Wholesale & retail trade		
Wholesale trade	28.6	9.3
Retail trade	21.1	10.9
Total wholesale & retail trade	28.9	10.6
Transport & storage		
Road transport	92.4	24.6
Rail transport	2.1	0
Water transport	22.9	38.9
Air transport	35.7	15.0
Other transport	2.7	0
Services to transport	46.4	3.2
Storage	86.7	16.1
Total transport & storage	62.2	12.5
Communications	8.5	6.3
Finance, property & business services	9.9	8.1
Public administration & defence	43.8	10.1
Community services		
Health	26.3	30.8
Education, museum, library services	12.1	10.9
Welfare & religious institutions	25.1	24.2
Other community services	58.9	14.1
Total community services	26.7	22.2
Recreation, personal & other services	21.5	14.8
Total	44.6	18.1

Within the manufacturing sector, the highest rates are to be found in the metal trades, transport equipment manufacturing, and non-metallic mineral products.

Outside the manufacturing sector, the most significant sources of injury are construction and road transport. The construction sector contributed 14% of all injuries, with a relative risk of 1.86.

As discussed in the preceding section, the female injury rate is 75% less than that of male workers, even after allowing for the differing number of hours worked. The only industries with injury rates above the mean for all male workers are miscellaneous manufacturing and manufacturing of transport equipment.

Three industries of special interest

Miscellaneous manufacturing

This sector had by far the highest injury rate of all sectors in the female workforce. It includes leather tanning and leather products, rubber products, plastics, ophthalmic articles, jewellery and brooms and brushes.

Road transport

Road accidents tend to lead to severe injury, because of the high degree of kinetic energy imparted from sudden deceleration. Accordingly workers in the road transport industry may be expected to experience a high proportion of injuries due to motor vehicle accidents, and the statistics bear this out. Of the 1739 reported injuries in males in the road transport sector, the agency of accident was *lifting equipment and means of transport* in 654, or 37.6%, compared with 12.9% of injuries in the male workforce as a whole. In females in the road transport industry the excess risk of injuries from this source was even higher - 39.3%, compared with only 7.1% for the overall female workforce.

Also as expected, this industry has a relatively high proportion of severe injuries. Altogether there were 63 injuries in males resulting in 100% disability, of which 18 occurred in the road transport industry, that is, 28.6%, although only 9.6% of the male workforce was employed in this industry (ie relative risk 3.0). Of the 13 injuries in female workers leading to 100% disability two, or 15.4%, occurred in road transport, although only 6.3% of the female workforce worked in that industry (relative risk 2.4).

Manufacturing of transport equipment

Much of the transport equipment manufacturing industry is, of course, the manufacture of motor vehicles. As South Australia is an important centre of motor vehicle manufacturing, a comparison has been made between the injury rate in this sector with those of the same industry category in Victoria, which is Australia's other principal car-manufacturing centre. As shown in Table 1.8, the rates are very similar for the two States.

Table 1.8. Injury rates, manufacturing of transport equipment, South Australia and Victoria, 1988-91.

	Rate (injuries/1000 person-years)	
	South Australia	*Victoria*
Males	92.5	92.9
Females	74.1	81.4
Persons	89.6	90.6

State Government employees

As a self-insuring employer, the State Government maintains its own workers' compensation system, administered by the Government Workers' Rehabilitation and Compensation Office (GWRCO). Some State authorities, including the South Australian Health Commission, the Electricity Trust of SA, and the State Transport Authority (TransAdelaide) insure separately.

Statistics about injury numbers and lost time are from the GWRCO. Employee numbers were obtained from the annual report of the Commissioner for Public Employment, and used to derive injury rates.

Table 1.9. *Injury rates for State Government employees insured through the GWRCO, and for the total South Australian workforce covered by the WorkCover legislation, 1988-1991. All rates expressed as number of claims, of 5 days or more of lost time, per 1000 person years.*

	SA government employees	*Total workforce SA*	*Total workforce SA adjusted**	*Rate ratio***
Males	34.7	44.0	32.3	1.07
Females	18.2	18.3	13.5	1.35

**For method of adjustment see text.*

*** Rate ratio: observed rate as a proportion of expected rate after adjustment*

As shown in Table 1.9, the government has a lower claims rate than the overall workforce in males, and the rate for females in the government is very similar to that of the overall workforce. However the two sets of rates are not strictly comparable, as the Government has a lower proportion of employees in manual occupations. Accordingly an adjustment has been made to derive an expected injury rate in the overall workforce if its occupational categories were distributed in the same proportions as in the State Government workforce. (The method of adjustment is described in the Methods section).

As shown in Table 1.9, after adjustment for the relatively low number of manual workers in the public sector, the rate in the government sector was similar to the overall male rate.

When adjustment was made to allow for the differences in the occupational profiles in females, the rate in the government sector was significantly higher than the overall female rate (18.3/1000py compared with 13.5/1000py, $p<0.05$).

It is concluded that, after adjusting for the fact that State Government workers are in general engaged in more sedentary occupations than the overall workforce, male government workers have approximately the same lost time injury rate as other male workers. However female government workers have a higher rate than other female workers by more than one third.

South Australian Health Commission

Unlike other SA Government Departments, the Health Commission does not insure through the GWRCO and insured through the SGIC for the period under study. Injury data were obtained from WorkCover, and an estimate of the number of employees insured was obtained directly from the Health Commission itself. The mean reported injury rates for male employees for the period 1988-89 to 1990-91 was 35.1 per 1000 person-years - similar to the rate for other State Government employees. However for female Health Commission employees the rate was almost double that of other female State Government employees. SA Health Commission statistics are compared with the overall workforce in Table 1.10.

Table 1.10. *Injury rates for SA Health Commission employees, and for the total South Australian workforce covered by the WorkCover legislation, 1988-1991. Rates expressed as number of claims, of 5 days or more of lost time, per 1000 person years.*

	SAHC employees	*Other SA govt employees*	*Total workforce SA*
Males	35.1	34.7	44.0
Females	32.5	18.2	18.3

Local government employees

Local government injury data were obtained from WorkCover, and employment numbers obtained from the 1991 Census.

Table 1.11. *Injury rates for local government employees, and for the total South Australian workforce covered by the WorkCover legislation, 1988-1991. All rates expressed as number of claims, of 5 days or more of lost time, per 1000 person years.*

	Local government employees	*Total-workforce SA*	*Total work-force SA adjusted**	*Rate ratio***
Males	85.1	44.0	56.0	1.52
Females	17.3	18.3	14.7	1.18

* *Method of adjustment as described under State Government employees*

** *Rate ratio: observed rate as a proportion of expected rate after adjustment*

As shown in Table 1.11, the reported injury rate for males was 85.1 per 1000 person-years, almost double the rate for the overall male workforce. Local government has a higher than average proportion of manual workers, so that for the purpose of comparison with the overall workforce, the latter was adjusted, using the same method as for the State Government workforce. Despite this adjustment there was still a relatively high injury rate (relative risk 1.52). Thus the high lost-time injury rate in local government is in part due to the relatively high proportion of local government workers in manual occupations, but even after adjusting for this difference the rate in local government was higher than expected by a factor of over 50%.

On the other hand the rate for female employees was 17.3/1000 person-years - similar to the rate for the overall female workforce. After adjustment for the occupational make-up of the local government workforce, there was a small excess reported injury rate in female employees of local government (relative risk 1.18).

Comparisons of lost time rate

The estimates for the average lost time per injury are shown in Table 1.11. The average lost time per injury for State Government employees is somewhat higher than for the workforce as a whole, both for males and females. However the figures need to be interpreted with caution, as the WorkCover estimate for State Government employees is only approximate. The average lost time per injury for female employees of the SA Health Commission is about one-half of the average for the State Government, and the rate for local government is about one-half of the average for the State for both males and females.

Table 1.11. Average number of lost days per injury, 1988-89 to 1990-91, South Australia.

	Males	*Females*
Total employed workforce[1]	16.2	22.0
State Government employees[2]	20.0	24.5
SA Health Commission	15.5	11.9
Local government	8.5	10.5

1 excludes Commonwealth Government employees
2 excluding SA Health Commission, ETSA and STA.

Commonwealth Government employees

Employees of the Commonwealth, except those employed by Telecom Australia and Australia Post, are insured under a separate scheme, Comcare. Injury numbers were obtained from Comcare, and employment numbers taken from the 1991 census. Injury rates for 1988-89 are less than half the rates for the two following years; they thus appear to be incomplete, and figures for this year have therefore been excluded from the analysis.

The injury rates, and comparisons with rates for the overall SA workforce, are shown in Table 1.12.

The injury rate for male employees of the Commonwealth was 37.1/1000 person-years compared with 44.0/1000 person-years for all male workers in SA. There is a relatively low proportion of manual workers in the Commonwealth Government workforce, so that for the purpose of comparison with the overall workforce, the latter was adjusted, using the same method as for the State Government workforce. After the adjustment, the comparative data indicate that the lost-time injury rate for Commonwealth employees was more than 50% greater than that of the overall SA workforce after allowing for the relatively low numbers of Commonwealth employees in non-manual occupations.

Table 1.12. Injury rates for Commonwealth Government employees (1989-90 to 1990-91), and for the total South Australian workforce (1988-89 to 1990-91). All rates expressed as number of claims, of 5 days or more of lost time, per 1000 person years.

	C'wealth Govt employees	*Total workforce SA*	*Total work-force SA adjusted**	*Rate ratio***
Males	37.1	44.0	23.6	1.57
Females	23.1	18.3	11.7	1.97

* *Method of adjustment as described under State Government employees*
** *Rate ratio: observed rate as a proportion of expected rate after adjustment*

For female employees of the Commonwealth the rate was 23.1/1000 person-years compared with 18.3/1000 person-years for all female workers in SA. After adjustment, the lost-time injury rate was approximately double the rate expected from the occupation-specific rates of the overall female workforce.

There were a number of uncertainties in this analysis:

(i) uncertainty over the number of employees of Telecom and Australia Post, which had to be subtracted from the total number of Commonwealth employees. Surprisingly, these authorities appeared unable to provide information on how many employees they had in SA

(ii) in estimating expected rates assumptions had to be made over how much allowance should be made for the occupational profile of Australia Post and Telecom

(iii) the low injury numbers in 1988-89, which led to the arbitrary decision to exclude that year from the analysis.

Accordingly the above conclusions on the injury rates in Commonwealth employees should be interpreted with caution.

Discussion

The Occupational Safety, Health and Welfare Act of 1986 marked a philosophical change in the administration and enforcement of workplace health and safety. This legislation is modelled on the UK Health and Safety at Work Act of 1974, and there is still little objective evidence that it actually reduces the rate of injury. It is therefore of great importance that the efficacy of the SA legislation be monitored from trends in injury, death and disease.

In the above analysis, efforts have been made to identify trends in the reported injury rates which are due to changes in the compensation and reporting systems, so that the underlying trends in injury rates can be seen.

The increase in the rate of reported injuries since the introduction of the WorkCover scheme does not appear to be due to an increase in the actual number of injuries. Rather it is the result of a sharp increase in the proportion of injuries leading to more than 5 days of lost time. After allowing for this factor it is clear that the actual injury rate has remained unchanged.

The introduction of the WorkCover scheme was undoubtedly accompanied by a very sharp increase in the average lost time per injury: not only the proportion of injuries leading to more than 5 days of lost time increased, but the average lost time per injury overall increased suddenly and steeply. It is unlikely that these findings mean that the average physical severity of injuries increased; it is almost certain that they are the changes in the workers' compensation law and its administration. It is important to recall that the statistics are based on the number of lost days *for which compensation is payable.* Prior to the WorkCover scheme, the period for which weekly benefits were payable was limited, whereas under the WorkCover scheme the entitlement, at least for total incapacity, continues until the worker returns to work or reached retiring age. Accordingly, since the advent of WorkCover, significant numbers of workers who have received benefits for more than two years have appeared in the statistics for the first time, adding significantly to the average number of lost days for which benefits were paid.

Although there has been no actual increase in the injury rate, there has not been a decline either, so that the injury statistics suggest that if an improvement in safety performance is the expected result of the Occupational Safety, Health and Welfare Act of 1986, it has not yet become manifest. However, these data only go as far as June 1991, less than four years after the Act was promulgated. Furthermore, much of the infrastructure for the new legislation was not in place by this time, so that it is premature to draw any conclusion on the basis of the trends in injury rates.

In contrast, the fatality statistics (Table 1.5) do suggest an improvement. Fatality statistics have the advantage of being less likely to be influenced by the compensation system. They also extend to 1993-94, a further 3 years beyond the period covered by the injury statistics, and it is this 3-year period in which the apparent improvement has occurred. Again, however, it would be premature to conclude that the legislation has had a demonstrable benefit. There were comparably low fatality rates in 2 successive years previously - in 1985-86 and 1986-87 - so that the data of the last 3 years may not necessarily

represent a trend. Furthermore fatality data usually require upward revision in years following first publication, so that the rates for these most recent years may be too low. Another reason for caution is the mortality trend in Victoria, where similar legislation was introduced in 1985, and where no improvement has occurred. The annual number of work-related fatalities in Victoria, which has similar legislation to that in South Australia, has been as follows:[2]

1985-86	28
1986-87	46
1987-88	34
1988-89	51
1989-90	34
1990-91	46
1991-92	34
1992-93	51

A regression line of the fatality rates in South Australia actually shows a steady downward trend in fatality rates since 1975-76 (although the trend is not statistically significant at the 95% level of confidence). This suggests that there may be more important factors in occupational safety than what is written in the statutes. One factor may be changes in the profile of industry, with some of the more hazardous processes disappearing. Other factors may be improved management practices and increased attention to safety matters by the trade union movement. Starting with the establishment of the establishment of the Trade Union Training Authority in the 1970s, the union movement has undertaken extensive training in safety matters for its members.

The lost time injury rates by occupation and industry have shown little change since the analysis of early 1980s data in *The Human Cost of Work*. The injury rates are still much commoner in manual workers than in white-collar workers, injuries and fatalities are still much commoner in men than in women, and "miscellaneous manufacturing" is still a high injury category for women.

The lost-time injury rate in male employees of the State Government is not significantly different from that of other male workers, after adjusting for non-comparability of occupational categories, but there is a 37% increase in the rate for female State Government employees. In local government the reverse was found, with male workers showing a 50% higher rate than for the overall male workforce.

Commonwealth employees showed increased reported injury rates - for males more than 50%, and for females nearly 100%, above the rates for the State as a whole. Although there were significant difficulties in obtaining statistics comparable with those of the overall workforce, the magnitude of the difference suggests a reported injury rate which could not be explained by the degree of physical hazards in Commonwealth workplaces.

The tendency for Government to have a higher than average rate of reported injuries might be construed as over-reporting. However it might equally mean that employees in the private sector tend to under-report. Either way it suggests that public servants are more likely to make a claim leading to more than 5 days off following injury.

References

1. Australian Bureau of Statistics. Industrial Accidents - South Australia, 1984-85. Catalogue No. 6301.4

2. Occupational Health and Safety Authority. A Statistical Profile of Occupational Health and Safety in Victoria, 1993. Vic stat bulletin

2. Occupational cancer

R T GUN

Occurrence of occupational cancer in South Australia at the present time - current occupational exposures which may increase the risk of future cancers.

Summary

Mesotheliomas are occurring at a rate of about 25 per year in South Australia as a result of past asbestos exposures. This is lower than the rate in the early 1990s. Mesothelioma rates are especially high in the Whyalla, Port Augusta and Western Metropolitan regions. Lung cancer related to asbestos exposure is more difficult to recognise, although there are probably fewer of them than mesothelioma cases.

There is a significantly higher proportionate rate of lung cancer in blue collar workers compared with white collar workers, and up to 25% of all male lung cancers are unexplained even after allowing for differences in smoking rates. Some of this, possibly 8%, of all male lung cancers, could be related to occupational exposures.

There is an excess of bladder cancers in males compared with females which is incompletely explained by the difference in smoking prevalence. The cause of the excess in males is difficult to explain, but occupational factors are not an obvious explanation since they are equally common in white collar and blue collar workers. An excess incidence in fitters and turners may be due to use of untreated or mildly treated mineral oils, which are now almost obsolete.

Leukaemias, specifically acute myeloid and chronic lymphatic leukaemias, occur in excess in men and in excess in blue collar workers compared with white collar workers. The excess accounts for 29% of male leukaemias, and smoking is an unlikely explanation. Some occupational risk factors may be responsible.

There is a significant excess of cancers of the lip in outdoor workers, due to exposure to solar radiation.

There is an unexplained excess of kidney cancers in males, but occupational factors are unlikely to be a major contributor to the excess.

The proportion of the workforce currently exposed to recognised chemical carcinogens is low, and the consequent risk of future cancers from these factors is estimated to be about 2.5 of the current number of cancers in males. Even this estimate may be too high, since it assumes that relative risks are just as high as in the epidemiological studies on which the categorisations of carcinogenicity were based, whereas exposures in most of the industries and chemicals discussed are likely to be much lower.

Occupational cancers can also arise from other than chemical exposure, for example from occupational exposure to solar radiation and from occupational acquisition of Hepatitis C infection. There is also some epidemiological evidence suggesting a cancer hazard from psychological stress at work, although this subject has not been extensively studied.

Particular vigilance is needed in the asbestos removal industry, which is a new cohort of workers at risk.

Introduction

Occupational cancer is not readily identified, for 3 reasons:

1. Many cancers can be caused by different combinations of causal factors. Thus a different constellation of component causes can be responsible for the same cancer type in different individuals.

2. Cancers of occupational origin are no different in clinical presentation nor in histopathological appearance from cancers of non-occupational origin. (An exception is lung cancer from asbestos exposure, which commonly shows a higher number of asbestos fibres and asbestos bodies in lung tissue than lung cancers which are not asbestos-related).
3. The long induction latency period of most cancer-causing exposures - several years or even several decades - means that most incident cancers occur years after relevant occupational exposures, by which time recollections of exposures may be faint, and exposure records non-existent.

Consequently it is not usually possible to make a judgement whether there is an occupational contribution in an individual cancer case. Thus there is no readily identifiable list of lung cancer cases, similar to the WorkCover record of occupational skin diseases. The South Australian Health Commission maintains a record of all invasive cancers occurring in the State, a record which for practical purposes is complete, except that its occupational data are insufficient to make an assessment of whether any individual cancer is even possibly work-related, although there are exceptions in the case of cancers which are usually related to occupation, such as mesothelioma from asbestos exposure.

Estimates of the occurrence and distribution of occupation-related cancer must therefore rely on population estimates based on epidemiological studies, rather than on registers of particular cases. A number of such estimates have been made, and the most widely-cited is a study by Doll and Peto published in 1981, in which an estimate of the fraction of each cancer type attributable to occupation was applied to the number of deaths from the corresponding cancer in the US in the year 1978.[1] The resulting overall estimate was that between 2% and 8% of all cancers in the US were attributable to occupation, with a best estimate of 4% - 7% in males and 1% in females.

In the earlier SA Health Commission publication, *The Human Cost of Work*, we made an estimate using this borrowed methodology: the attributable fractions in the US study were applied to the total number of cancers recorded in the SA Central Cancer Registry from its inception in 1977 until 1984. Since then a separate estimate of the occupational cancer burden in Australia, using the same technique, was published by Winder and Lewis,[2] and the method was again used, with minor modifications, in a report for Worksafe Australia in January 1994.[3]

Although this method was used in our previous publication, there are difficulties in using it to estimate the occupational cancer risk in Australia. The incidence of various cancers varies widely between countries due to the difference in the distribution of risk factors, such as diet and living standards, as well as occupational factors. The proportion of a cancer type, eg bladder cancer related to occupation can vary greatly between countries, according to the number of people exposed to carcinogens, the intensity of the exposure, and the presence of concurrent risk factors. It is therefore meaningless to estimate the proportion of cancers due to occupation in one country from a proportion derived elsewhere, especially when the total incidence of the cancer is different. For example, a lower incidence of lung cancer in Australia than in the US might actually be due to the occurrence of fewer occupational cancers in Australia, so that applying the fraction attributable to occupation used in the US to derive the number of lung cancers in Australia would give an overestimate. If on the other hand Australia had a lower rate of lung cancer *because* there were fewer non-occupational cancers, the method would yield an underestimate. In this chapter estimates of the burden of occupational cancer will be made using local data as far as possible.

This chapter examines the question of occupational cancer from 2 perspectives: (i) a review of the likely contribution of occupation to the present incidence of cancer and cancer mortality in South Australia, and (ii) an examination of the extent of present exposure to recognised carcinogens to estimate the possible extent of cancer being generated from current workplace exposures in South Australia.

Current cancer incidence in South Australia

To estimate the contribution of occupation to cancer incidence and mortality, comparisons have been made of age-stratified incidence and mortality between males and females, and between white collar workers and blue collar workers. Where appropriate, allowance is made for differences in smoking prevalence. Thus the analyses are based on an assumption that female workers are not exposed to carcinogens, and that amongst males only blue collar workers are exposed.

Mesotheliomas. There were 25 incident cases of pleural mesothelioma in South Australia in 1994, 22 in men and 3 in women. On the basis of local and national data, it is estimated that about 80% of the male cases have a history of probable exposure to asbestos. However even the cases with no known asbestos exposure occur more commonly in males, so that it is probable that nearly all other cases are work-related as well.[4] (Mesothelioma is not related to smoking, so some unknown occupational exposure is possibly responsible, acting either as an independent cause or as a co-factor with the background exposure to which most people are exposed.) There is a "spontaneous" incidence of mesothelioma of about 1-2/million person-years in both men and women. After allowing for these cases, about 21 of the pleural mesotheliomas, 20 in men and 1 in women are likely to be work-related, although they may not all be caused by asbestos.

Pleural mesothelioma cases recorded by the Cancer Registry since 1977 have shown a heavy excess in the regions of the State which include Port Augusta and Whyalla, cities which were reliant on industries which involved heavy asbestos exposure. In the Port Augusta region, where the Commonwealth Railways and the power stations were the main employers, there have been 18 male mesothelioma cases, with a standardised incidence ratio of 3.8, ie nearly a 4-fold excess risk, which is statistically significant. There was only one female case. In Whyalla, where a large shipyard was located, there were also 18 cases, with a relative risk of 3.9, which was statistically significant.* There was also a significantly high relative risk (1.4) in the Western Metropolitan Region, which includes Port Adelaide, formerly a centre of shipbuilding and the location of an asbestos building products manufacturer, and the site of Adelaide's main power station.

Peritoneal mesotheliomas are much less common. There were 2 cases in SA in 1994, and that has been the average number each year since 1989. Occupational information is scant, but none of the job titles on the death certificates is of a job associated with asbestos exposure Moreover the association of asbestos with peritoneal mesothelioma is believed to be less than with pleural mesothelioma. However all cases since 1989 except one have been in males, suggesting some occupational association.

Lung cancer. Although lung cancer has been causally linked to several occupational exposures, the strength of the association has been difficult to measure because of the very strong association with smoking. For the purpose of this exercise a number of methods were used to estimate the contribution of occupational exposures.

(i) *Lung cancers in SA attributable to asbestos.* Data were extracted from a pilot occupational surveillance study on occupational cancer in South Australia.[5] Case-control studies were carried out on incident male lung cancer cases and population-based controls, and odds ratios generated for a number of exposures including asbestos. All 62 lung cancer cases were smokers, and for the estimates they were compared with the 85 controls who had ever smoked.

The estimated relative risk (odds ratio) for probable or possible asbestos exposure was 1.05, an elevated risk of only 5%, which was not statistically significant (95% confidence interval 0.5-2.1). The estimate was based on 24 exposed cases. With possible exposures excluded, the relative risk was 1.00 (95% confidence interval 0.5-2.2), ie there was no excess risk; this was based on 15 exposed cases. For those with prolonged and intense exposure there were only 2 cases, with a

* *A number of mesothelioma cases in Whyalla probably incurred their asbestos exposures in overseas shipyards before emigrating to work in the Whyalla shipyards.*

relative risk of 2.8; this elevation was not statistically significant, as would be expected from so few cases.

On this basis the estimated proportion of lung cancers attributable to asbestos exposure is nil.

(ii) *Lung cancers in SA attributable to exposure to fumes, mists or dusts.* The estimate of occupation-related lung cancer in the US by Doll and Peto was partly based on a large prospective cancer study by the American Cancer Society, in which the lung cancers were assumed to have occurred in workers occupationally exposed to fumes, mists or dusts (including asbestos). In the SA pilot study, a similar estimate was obtained for workers with these exposures, which were equated to exposure to asbestos and/or products of combustion and/or welding fumes. The estimated relative risk for these exposures was 1.1 (95% confidence interval 0.5-2.5) based on 51 exposed cases for probable or possible exposure, and 1.02 (95% confidence interval 0.4-2.4), based on 45 exposed cases for probable exposure only; neither estimate was statistically significant. For prolonged and intense exposure the relative risk was 0.97, based on 4 exposed cases.

The estimated attributable fraction again was nil.

(iii) *Analysis of SA Cancer Registry data.* Although notifications to the SA Cancer Registry contain information on current occupation, the occupational title in most cases is "pensioner" or "retired", since cancer is so often diagnosed after retirement from work. However an occupation other than these appears in about 20% of cases, mostly from those under retiring age, and the accumulated data of those cases with a pre-retirement occupation were analysed. A three-stage process was used to estimate the fraction of lung cancers attributable to occupation. First, the lung cancer rates of white collar and blue collar workers were compared. Comparable population (ie denominator) data on numbers employed in different occupational groups do not exist, so the relative rates of lung cancer in blue collar workers to white collar workers were computed indirectly using the method of Miettinen as an index of relative risk.[6] This method involves comparing the number of deaths from lung cancer with the number of deaths from causes unrelated to the factor of interest, in this case from diseases unrelated to social class, (defined according to blue-collar or white collar occupational category). The diseases chosen for comparison were cancers of the rectum and rectosigmoid colon, kidney, pancreas and brain, which SA Cancer Registry analyses have shown to be independent of social class. Secondly, an estimate was made of the contribution to the relative risk from the different smoking prevalences of white-collar and blue-collar workers. The prevalences of smoking in white-collar and blue-collar workers were obtained from the SA Health Omnibus Surveys for 1990 to 1994. The contribution from smoking was estimated using the method of Axelson and Steenland, (which assumes that the relative risk of smoking on the two groups is the same).[7] The assumed relative risk of smoking was obtained from a recent published estimate: 10 for smokers and 6.5 for ex-smokers.[8] Thirdly, the total excess risk in blue-collar workers was discounted by the estimated contribution from their higher smoking prevalence. The residual excess risk then represents an estimate of the risk attributable to occupational exposures. (Of course, this is an overestimate to the extent that factors other than smoking and occupation contribute to the excess, and an underestimate to the extent that occupational exposures cause lung cancers in white-collar workers.

The relative risk of lung cancer in blue-collar workers (relative to white-collar workers) was 1.6 (Table 2.1).

The prevalence of smoking was: for blue collar workers 35% smokers, 31% ex-smokers, and for white collar workers 21% smokers, 33% ex-smokers. On this basis the relative risk from the difference in smoking prevalence was 1.24, which would have resulted had there been 874 lung cancers in blue-collar workers (instead of the 1128 as shown in Table 2.1).

Table 2.1 *Computation of relative risk for incident lung cancers in males in SA, 1977-94. The other cancers used for comparison were cancers of the rectum and rectosigmoid colon, kidney, pancreas and brain.*

	Lung cancers	*Other cancers*
Blue-collar occupations	1128	799
White-collar occupations	615	697

Relative risk = 1.6

The attributable fraction caused by occupation was calculated using the method of Miettinen.[9] The estimate of the fraction of lung cancers attributable to influences other than smoking was:

(Fraction attributable to being in a blue-collar occupation) - (fraction attributable to being in a blue-collar occupation from smoking effect alone)

= (1128/[1128+615])(0.6/1.6) - (874/[874+615])(0.24/1.24)

= 0.11

On this basis the estimate of the fraction of lung cancers attributable to occupation is 11%.*

(iv) *Analysis of SA Death Registry data.* Death certificates now state the usual occupation of the deceased during his or her working life. This was exploited in a death registry analysis, in which lung cancer deaths and other deaths for comparison were extracted from the ABS file from the SA Death Registry from 1989 to 1993. Most occupational data had been coded, and where it was not the original death certificates were retrieved and the occupational title coded. The occupational groups were then categorised into white collar or blue collar occupations. The lung cancer rate in white-collar workers was compared with that for blue-collar workers, using the same method as with the Cancer Registry data. Although only 5 years of data could be used, nearly all were useable because occupational data were available on people who died after reaching the usual retiring age.

The overall relative risk of lung cancer in blue-collar workers (compared with white-collar workers) was 2.18, as shown in Table 2.2

Table 2.2 *Computation of relative risk (mortality odds ratio) for deaths from lung cancer in males aged 40 years or more in SA, 1989-93. The other cancers used for comparison were cancers of the rectum and rectosigmoid colon, kidney, pancreas and brain.*

	Lung cancers	*Cancers unrelated to social class*
Blue-collar occupations	1168	300
White-collar occupations	580	326

Relative risk = 2.18

As computed in the previous analysis, the relative risk from the difference in smoking prevalence was 1.24, which would have resulted had there been 782 lung cancers in blue-collar workers (instead of the 1168 as shown in Table 2.2).

The estimate of the fraction of lung cancers attributable to influences other than smoking was:

** This estimate is based on the assumption that the relative risk of smoking is the same in blue-collar and white-collar workers. However if, for example, the average smoking blue-collar worker smokes more than the average smoking white-collar worker, the relative risk, assumed in the foregoing analysis to be 10 for both groups, might be higher in the former group. Again, if the blue-collar workers on average started smoking at an earlier age than white-collar workers, the excess lung cancer prevalence in the blue-collar workers would be greater and the fraction attributable to other factors such as occupation would be correspondingly less.**

(Fraction attributable to being in a blue-collar occupation) - (fraction attributable to being in a blue-collar occupation from smoking effect alone)

$= (1168/[1168+580])(1.18/2.18) - (782/[782+580])(0.24/1.24)$

$= 0.25$

Thus using the same method as with the cancer registry data, the estimated fraction of cancers attributable to occupation is 25% (subject to the same qualifications as footnote to the previous analysis).

(v) *A questionnaire* was sent to respiratory physicians requesting estimates of the number of lung cancer cases seen in the previous 5 years in people with asbestosis, and of lung cancer cases in people who had worked in industries associated with asbestos exposure. (The industries selected were those which account for 84% of the cases in the National Mesothelioma Surveillance Program with known asbestos exposure.) Not all respiratory physicians replied to the questionnaire, but responses were received from the 3 major teaching hospitals. In most cases no formal records are maintained, so that respondents emphasised that they were able to provide estimates only.

Two teaching hospitals reported on the number of cancers of asbestosis complicated by lung cancer, the number being 5 in both cases. One other respiratory physician reported one case of lung cancer in a person with pre-existing asbestosis.

One teaching hospital reported 75 lung cancers in the 5-year period from the industries associated with asbestos exposure. The other respondents lacked adequate occupational information to make an estimate.

As discussed in Chapter 5, 54 cases of asbestosis were reported by respiratory physicians over a 5-year period.

Whether asbestos itself causes lung cancer or whether asbestosis is a necessary precursor to asbestos-related lung cancer is in dispute. A recently-published case-control study of lung cancer in which chest x-rays were examined blind for asbestosis supports the former. There was an odds ratio of 1.5 for asbestos exposure in the absence of asbestosis, and 2 in the presence of asbestosis.[10] There is no reason to assume a priori that asbestosis is a necessary precursor to lung cancer (it is not in the case of mesothelioma), and for the purpose of this assessment it is assumed that it is not. However there is a higher risk of lung cancer following asbestos exposure if asbestosis is present, and the reporting of 54 cases suggests that there is a significant lung cancer risk from this source. Assuming that this represents some under-reporting, there could be about 10 lung cancer cases per year in people with known asbestosis. There are inadequate data to estimate the fraction of lung cancer cases associated with undetected asbestosis or with asbestos exposure with no asbestosis.

Evaluation

Lung cancer. The five estimates of occupation-related lung cancer are thus 0% (asbestos only), 0% (fumes, mists or dusts), 13% maximum, 26% maximum, and an unknown figure exceeding 10 per year.

It is difficult to explain these estimates in a coherent way. The case-control data (the first two estimates) would suggest that past asbestos exposures sufficient to cause lung cancer have been infrequent in the South Australian workforce. The estimates are based on small numbers, but they are unlikely to be a gross underestimate; it is unlikely that the true odds ratio for asbestos exceeds 2.[5] If the true odds ratio is 1.5, and is applied to the proportion of cases in the case-control study with more than slight exposure to asbestos (5/62),[5] the resulting estimate is 2.6% of lung cancers attributable to asbestos. To this figure other occupation-related lung cancers would have to be added.

The estimates based on cancer registry and death registry data are 13% and 26% respectively. The latter is more reliable, since it contains more cases in people beyond retiring age, which is when most lung cancers occur. However the figure of 26% is possibly an overestimate, as it includes all causes other

than smoking, so that some of the 26% could be due to lifestyle factors such as diet. Furthermore the estimate assumes that the relative risk of lung cancer is the same in white collar smokers as in blue collar smokers. This would only be so if the average number of cigarettes smoked by smokers in the two categories were the same. The relative risk of smoking is very sensitive to the amount smoked (hence the large difference in the lung cancer rates between men and women). For example, if the relative risk of smoking were 8 in white collar workers and 12 in blue collar workers due to differential cigarette use, the estimate of lung cancer attributable to occupation would be only 8.5%.

Thus the total contribution of occupation to lung cancer probably lies between the estimate for asbestos of 2.6% and a maximum of 26%. The estimate of no lung cancers at all due to fumes, mists or dusts suggests that the contribution of airborne contaminants, at least those other than asbestos, is small, so that the true level is probably towards the lower end of this range.

Bladder cancer. Many different occupational factors have been identified as possible causes of bladder cancer. The fraction of bladder cancers attributable to occupation have been estimated to be as low as 1% and as high as 20%, and the variation may reflect different occupational exposures in different countries. Textile dyeing, leather work, the rubber industry, motor vehicle driving, exposure to aromatic amines and cutting oils have been among the commonly-mentioned risk factors, but the findings have been inconsistent. Most of the findings have been based on case-control studies based on occupational titles, which are crude means of assessing exposures to individual chemicals. A recent study of male cases of bladder cancer in Montreal, in which detailed assessments were made of individual chemical exposures, has found little evidence to support an association with most exposures previously identified as causing bladder cancer. The authors found only one substance - aromatic amines, two occupations - motor vehicle drivers, and textile dyers) and one industry - motor transport, to have a probable association with bladder cancer. On this basis they estimated that 6.5% of bladder cancers in that occupation were attributable to occupation.[11] The authors made no attempt to estimate the corresponding figure in females.

Smoking is a strong risk factor for bladder cancer, the majority of cases occurring in smokers. The increase in risk of bladder cancer smoking has been variously estimated to be 3- to 4-fold in males, but some what lower in females. Urinary tract infection is also a likely risk factor, more so in females than in males.

In 1992 the South Australian Cancer Registry undertook an analysis of occupational features of bladder cancers, based on those cancers registered between 1977 and mid-1991 for which there were adequate occupation data.[12] Male and female data were combined and observed numbers of cancers compared with expected numbers for 10 broad occupational categories and there was no significant difference between observed and expected numbers. The only individual occupational groups with an excess were fitters and turners (12 observed/6.8 expected) and electricians (11 observed/4.8 expected). The former could be due to the use of mineral oils, although most cutting fluids used in recent years contain treated oils, in which there is little or no cancer risk. The excess in electricians is more difficult to explain, although some contact with PCBs is a possible explanation.

We undertook a separate analysis of bladder cancer cases in the same manner as described above for lung cancer, comparing the bladder cancer incidence in blue collar and white collar workers from the SA Cancer Registry. The result of the analysis is shown in Table 2.3.

Table 2.3 *Computation of relative risk for incident bladder cancers in males in SA, 1977-94. The other cancers used for comparison were cancers of the rectum and rectosigmoid colon, kidney, pancreas and brain.*

	Bladder cancers	*Other cancers*
Blue-collar occupations	169	799
White-collar occupations	171	697

Relative risk = 0.86

We also analysed SA bladder cancer mortality data in males from 1989 to 1993 from SA Death Registry data. The results are shown in Table 2.4.

As with the Cancer Registry analysis, there was no excess in blue collar workers.

These findings of no excess in blue collar workers in either analysis are difficult to interpret in the light of the well-established association with smoking. However the strength of association between bladder cancer and smoking is less than with lung cancer (about 3-4 fold) so that the effect of different smoking prevalence is much less. One possible interpretation is that white collar workers are exposed to occupational bladder carcinogens and blue collar workers are not.

Table 2.4 *Computation of relative risk for deaths from bladder cancer in males aged 40 years or more in SA, 1989-93. The other cancers used for comparison were cancers of the rectum and rectosigmoid colon, kidney, pancreas and brain.*

	Bladder cancers	*Cancers unrelated to social class*
Blue-collar occupations	65	300
White-collar occupations	73	326

Relative risk (blue collar/white collar) = (65/73)/(300/326) = 0.97

The Cancer Registry data nevertheless show about a 4- to 5-fold excess of bladder cancer in males. If the relative risk of smoking in males is 4, the different smoking prevalence is unlikely to account for more than about one third of the difference, so that the remaining excess in males is related to occupation, lifestyle and other factors. However, given the absence of any excess in blue collar workers in both of the above analyses, the fraction attributable to work exposures is likely to be small. The incidence of bladder cancer is highest in developing countries - about 15/100000 person-years in Europe and North America, compared with 3-4/100000 in Brazil and Israel. This suggests that there may be an inverse social class gradient. Possibly there are more lifestyle risk factors in higher socioeconomic groups, which mask the effect of smoking and occupation in lower socioeconomic groups. In South Australia the rate in males has been declining from 16 to 11/100000 person-years over the last two decades, suggesting a declining influence of smoking and occupational factors.

In summary, a coherent explanation of the data is difficult to find. There is an excess in males incompletely explained by smoking, but the remaining excess is more likely to be attributable to lifestyle factors than to occupational carcinogens.

In females, most bladder cancer cases occurred other than in paid employment, ie, the occupation was given as home duties or otherwise not in the labour force. This may be due to most women being out of the labour force by the age of 50.

Laryngeal cancer. The most important risk factors for this cancer are tobacco and alcohol. Asbestos has been cited as a possible risk factor, but the evidence for such an association is not consistent. Strong inorganic acids containing sulphuric acid have been associated with laryngeal cancer, and have been

categorised as a human carcinogen for that reason. The 1992 Cancer Registry analysis showed an excess in blue collar workers. Some individual occupational categories showed an excess, but none were in occupations likely to be exposed to strong acid mists. Occupational factors are unlikely to contribute significantly to the incidence of laryngeal cancer in South Australia, where the incidence is low by world standards.

Liver cancer. Risk factors for this cancer include Hepatitis B and Hepatitis C infection, alcoholic cirrhosis and aflatoxin. Liver cancer rates are low in comparison with rates in other countries, possibly reflecting a low prevalence of Hepatitis B. The Cancer registry data showed an excess in blue-collar occupations, but no occupational association appeared likely from the analysis.

Vinyl chloride monomer is a powerful cause of angiosarcoma of the liver, but not of the commoner hepatocellular cancer. Vinyl chloride is not manufactured or used in South Australia.

Occupations associated with the production or selling of alcoholic beverages and those at risk of occupational contact with Hepatitis B such as health care workers or child care centre workers could be at increased risk of liver cancer, but no such association is apparent at present.

Leukaemia. Leukaemia has been attributed to a number of occupational exposures, including ionising radiation and benzene. However in a review of world-wide trends in leukaemia, it has been concluded that no trends between different populations are attributable to occupation, as neither the proportion of the population exposed nor the magnitude of the risk are large enough.[13] Whilst a significant proportion of the population in Australia is exposed to benzene, the exposure is unlikely to be great enough to make a measurable contribution to the burden of leukaemia, and the same is true for ionising radiation.

An association between leukaemia and work in electrical occupations has been hypothesised on the basis of surveillance of death certificates in Washington State, USA.[14] Electromagnetic radiation has been hypothesised as a possible cause, but the question in dispute.

The SA Cancer Registry analysis found no significant variation in the observed/expected frequencies of leukaemia in the different broad occupational categories.

The age-sex distribution of the different leukaemia types vary, and this may reflect different causal factors. Acute myeloid leukaemia (AML) is about 30% commoner in males than in females, and chronic lymphatic leukaemia (CLL) is nearly twice as common in males, whereas the incidences of chronic myeloid leukaemia and acute lymphatic leukaemia are approximately equal in males and females. The male excess of AML first appears at age 60, which would be consistent with some occupational causation, as would all CLL which is a disease of the middle-aged and the aged. These combined excesses of AML and CLL from middle age onwards make up about 29% of leukaemia in males.

We undertook an analysis of AML and CLL in males from SA Death Registry records from 1989 to 1993, similar to those described above for lung and bladder cancer. The results are set out in Tables 2.5 and 2.6.

demonstrate an association. Nothing in the South Australian Cancer Registry data suggests an increased risk in occupational group likely to be exposed to herbicides. The condition occurs in equal frequency in males and females.

Non-Hodgkin's lymphoma (NHL). This cancer is showing a sharp increase world-wide. The 1994 report of the SA Cancer Registry reports an increase an increase of 40% in the standardised incidence rate of diffuse NHL in males since 1977-80. In females the rate has increased 22%. It is associated with decreased immunity, and is thus common in AIDS and in people who have been treated with immunosuppressive therapy. However not all of the increase can be accounted for by these two factors. A number of studies have suggested an association with agricultural use of phenoxyacetic acid herbicides such as 2,4-D, but the SA Cancer Registry occupational analysis showed no excess in farmers. There is a small excess in males, but this extends through all ages. Overall the evidence for a significant contribution to NHL from occupation is not persuasive, at least in South Australia.

Cancer of the kidney. There has been some evidence of an increase in renal cell cancer in coke oven workers and in workers in the aluminium reduction industry. In South Australia there are few workers in the former and none in the latter. There is a higher incidence in males from the age of about 30 years onwards. This would be consistent with some relationship with smoking and occupational factors, which are likely to have higher prevalences in males. However if these were significant influences in the occurrence of renal cell carcinoma, a higher SMR would be expected in blue collar workers compared with white collar workers, whereas Cancer Registry data suggest that the mortality rate for kidney cancer is independent of social class. Thus it is likely that occupational factors are only responsible, at most, for occasional cases of renal cell cancer.

Cancer of the brain. There has been some indication of excess brain cancer in the petroleum industry, and a possible association with nonionising radiation. However there is only a slight excess in males, and there is nothing suggestive of occupational exposure in the Cancer registry analysis.

Thyroid cancers. The study by the Cancer Registry noted an excess of thyroid cancers in medical practitioners and nurses, suggesting a possible excess from past radiation exposures.[12]

Cancer of the scrotum. This cancer is caused by contact with polycyclic aromatic hydrocarbons. There have been 8 cases in South Australia since 1977.

Melanoma. Melanoma incidence is almost equal in men and women, although the anatomical distribution is different. Whereas men have a higher incidence of melanoma of the trunk, women have a higher incidence on the lower limbs. Although solar radiation is the most important known risk factor, the incidence does not appear to be related to occupational exposure to sunlight. SA Cancer Registry data show an excess in white collar workers compared with blue collar workers (relative risk 1.7).

Nonmelanotic skin cancer. These cancers are not notifiable to the Cancer Registry. However they are strongly related to exposure to sunlight, and are commoner in outdoor workers.

Other cancers. It is likely that occupational factors do not contribute to other cancers, such as cancer of the stomach, breast, female reproductive system, male reproductive system except for the scrotum, the eye, myeloma and Hodgkin's Disease. A possible exception is stomach cancer, which has been reported in excess in the rubber industry.

Overall evaluation

With a few exceptions, it is not possible to make an estimate of the fraction of cancer types attributable to occupation, but an estimate can be made of the range within which the true fraction lies. The maximum estimate is derived by assuming that the excess incidence in blue collar workers compared with white collar workers, after allowing for differences in smoking prevalence, is due to occupation. This overestimates the occupational contribution to the extent that lifestyle factors, differences between blue collar and white collar smokers in the average number of cigarettes smoked per day, and any other factors contribute to cancer incidence. There is also an underestimate to the extent that occupation contributes to cancer in white collar workers. A summary of the estimates for cancer in males is given in Table 2.7.

Table 2.7. Estimates of contribution of occupation to cancer types in males.

Cancer type	*Number SA 1994*	*% attrib to occupation*	*Comments*
Mesothelioma	24	100	Negligible peritoneal
Lung	451	2.6-26	Probably <10%
Leukaemia	111	0-29	AML and CLL
Lip	105	30	

In addition to the cancers in Table 2.7, the following cancers are likely to be work-related in some cases, but the numbers in any one year are unlikely to be high enough to influence population statistics:

Larynx
Liver
Nasal cavities and paranasal sinuses
Bone
Scrotum
Bladder
Kidney

The following cancers are possibly occupation-related in a small number of cases:

Rectum
Non-Hodgkin's lymphoma
Connective tissue
Pancreas
Thyroid
Stomach
Brain

The following cancers are unlikely to be work-related:

Breast
Female reproductive system
Male reproductive system
Eye
Myeloma
Hodgkin's disease
Mouth and pharynx
Colon
Melanoma

The impact of current carcinogenic exposures in South Australia

An alternative means of evaluating the risk of occupational cancer is to examine the current exposures to occupational carcinogens, and the likely implications for the future occurrence of cancer. In this survey only substances or occupations categorised by the International Agency for Research on Cancer (IARC)

cells, and this is possibly the reason for these agents being associated with cancers of the haematopoietic and lymphopoietic systems. This raises the possibility of cancer risk to pharmacy and nursing personnel who dispense and administer these drugs to cancer patients.

Approximately 120 pharmacists and nurses are regularly involved in the dispensing and administering of these drugs to cancer patients in SA. The drugs are prepared under controlled conditions, with the use of vertical laminar flow cabinets and stringent controls over materials handling. There is also likely to be a small number of health care workers involved in the disposal of contaminated waste from these agents. This is also subject to control procedures, although it is not known how effective these are in controlling exposures. It is not certain whether there is any risk to these health care workers, whose exposure is clearly much less than that of cancer patients to whom significant quantities are administered intentionally. One unpublished study on pharmacy workers in SA measured the occurrence of a marker of genetic damage in pharmacy personnel compared with cancer patients being treated with cytotoxic drugs and with unexposed controls. The proportion in pharmacy personnel was similar to that of the controls, and in both of these groups the proportion was significantly less than in the cancer patients.

Given the low exposure to pharmacy workers and nurses, and their small numbers, it is unlikely that the use of these agents is contributing to the burden of occupational cancer.

Arsenic. Arsenic is widely distributed in the environment. Industries where significant exposure might occur include timber treatment, pesticide application, treatment of lead-containing ores, leather manufacturing and in anti-fouling paints. It is estimated that about 300 workers are exposed or potentially exposed to arsenic.

Occupational arsenic exposure is associated with skin cancer and respiratory cancer. Non-melanotic skin cancer is not recorded by the SA Cancer Registry, and it is not possible to estimate the contribution from arsenic exposure (nor from any other occupational exposure). The relative risk respiratory cancer is about 3 in smelting and about 2 in other industrial use. On this basis it is estimated that about 1.3 cancers are generated annually from arsenic exposure.

Chromium. Hexavalent chromium is categorised as a human carcinogen, but trivalent chromium is not. The slightly soluble chromium salts are considered to be a greater cancer risk than those which are highly soluble or insoluble. The evidence for human carcinogenicity comes mainly from studies on workers in chromate production, pigment production and chrome-plating.

Local industries where significant chromium exposure could occur include stainless steel welding, hard-chrome plating, leather manufacturing, timber treatment, and paint manufacturing and manufacture of PVC piping. The estimated number potentially exposed in these industries is about 750.

Chromium production and use of chromate pigment has been associated with a large increase in risk of lung cancer, and a smaller relative risk - between 1.4 and 1.8 - in chromium-plating. Assuming a relative risk of 5 for the former and 1.4 in the latter (allowing for some contribution from smoking), these exposures would be generating 2.2 lung cancers per year.

Benzene. Benzene is especially toxic to bone marrow, and exposures can lead to aplastic anaemia and granulocytopaenia. Not surprisingly, leukaemia is a further consequence of prolonged benzene exposure. As discussed in Chapter 10, current exposures are unlikely to be sufficient to produce acute toxic effects on bone marrow. However it is also possible that a leukaemia risk may still exist at exposure levels insufficient to produce nonmalignant disease.

Low level exposures (up to 1ppm) are usual in workers in the petroleum industry, and somewhat higher levels occur in sections of the chemical industry. The Australian Petroleum industry maintains its own surveillance program for its own employees, and the 1992 report shows an Standardised Incidence Rate of 2.8 for leukaemias, an increase which is statistically significant. There is also a trend to increasing risk with increased benzene exposure. There is evidence that the risk is greatest for workers first

employed before 1954, when benzene exposures were probably higher than at present. The industry workforce is predominantly male.[15]

For the purposes of this estimate it is assumed that about 600 males are employed in the petroleum industry in SA, and a further 400 with comparable benzene exposure. It is also assumed that the excess leukaemias are caused by benzene. On this basis it is estimated that benzene exposures are generating 1 leukaemia case per year.

Nickel. The IARC has categorised the following nickel exposures as carcinogenic to humans: nickel sulphate, and nickel sulphide and nickel oxide in nickel refining. Excess lung cancers and nasal cancers have occurred in workers in the electrolysis process of nickel refining, but in other jobs with nickel exposure such as electroplating, and manufacturing and welding of stainless steel, it has not been possible to demonstrate a carcinogenic effect of nickel independent of concurrent exposures to other carcinogens such as chromium.

The exposures of interest are sinonasal cancer and lung cancer, with relative risks of 26 and 2.25 respectively. As a worst-case scenario it is assumed that nickel exposures are carcinogenic in electroplating and anodising, and in the welding of stainless steel. On this basis about 1150 workers are exposed, leading to 1.3 excess sinonasal cancers and 4.1 excess lung cancers per annum. However, because all these workers are concurrently exposed to chromium, there may be some element of double counting in these cancers.

Coal tar & coal tar pitches. These exposures have been associated with cancers of the scrotum, and excess cancers of the larynx and lung. Some studies have also shown excesses cancers of the oral cavity, stomach, kidney, renal pelvis and bladder. Assuming that the laryngeal and lung cancers are causally related to these exposures, the relative risks are assumed to be 2.7 and 1.6 respectively.

Exposures are likely in employees of local government, the State Government and bitumen-paving contractors, a total of about 3000 workers. Accordingly the estimated excess laryngeal and lung cancers from these occupations are 1.2 and 5.2 per annum respectively.

All scrotal cancers are likely to be related to exposure to these agents, but this cancer is uncommon - there have been 8 in South Australia since 1977.

Cadmium. Studies carried out 20-30 years ago suggested that cadmium is a cause of prostatic cancer, but more recent studies indicate that it is not. The IARC has categorised cadmium as a human carcinogen on the basis of a US study showing excess lung cancers. This study has been criticised by some UK researchers who are currently re-analysing the US data.[16]

Exposures to cadmium can occur from silver soldering in the plumbing and radiator repair industries, and the manufacture and recycling of plastics containing cadmium pigments.

Estimates of the likely occurrence of cancer from current exposures is made difficult by the uncertainty of the association between cadmium and lung cancer. A recent review of the subject has focussed on the combined data from 17 plants in the UK, with a conclusion that the excess cancers may not be due to cadmium. A stratified analysis was undertaken according to whether workers had or had not incurred concurrent exposure to arsenic as well as to cadmium. For those exposed to both arsenic and cadmium the Standardised Mortality Rate (SMR) from lung cancer was 147%, and for those exposed to cadmium only the SMR was 82%.[17]

Strong inorganic acid mists containing sulphuric acid. Although the findings of epidemiological studies have not been entirely consistent in finding excess cancers from this exposure, most have demonstrated an excess of laryngeal and lung cancer, with relative risks of 2-3 and 1.6 respectively. Exposures to this chemical are widespread, with the more significant exposures in sulphuric acid manufacturing, electrolytic metal refining, electroplating, fertiliser manufacturing, leather tanning, battery making, bottling, electric power generation, textile manufacturing, bottling and auto servicing. However most

workers in these would not be highly exposed, and it is probable that the number of workers subject to frequent or intense exposure would approximate 1000. On this assumption the current exposures will be responsible for 0.4 excess laryngeal cancers and 1.7 lung cancers per annum.

Ethylene oxide. This agent is used for sterilisation of medical equipment which cannot be autoclaved. Animal studies have suggested an association with leukaemia. Some human studies have also linked ethylene oxide with leukaemias or other haematopoietic cancers, but the findings have not been consistent. In recent years the use of this agent has been centralised in South Australia, with items for re-sterilisation being freighted to 2 or 3 central facilities. Engineering controls and controlled operating procedures have reduced exposures to levels which have been, in most cases, too low to measure. The centralised facilities have also meant that only a small number of workers (about 30) are working with this agent, so that it is highly unlikely that it is now making any contribution to the population cancer burden under current conditions of use.

Mineral oils, untreated & mildly treated. The occurrence of scrotal cancer is recognised as causally related to exposure to mineral oils. Bladder cancer has been associated with these exposures, but the findings have been inconsistent. In the recent comprehensive study from Quebec, no significant association was found with bladder cancer. However the SA cancer registry analysis of cancer by occupation showed an excess of bladder cancer in fitters and turners, suggestive of an association with mineral oils. Several studies have found excess cancers from exposure to mineral oils, but the cancer types have varied. This suggests that mineral oils do not cause any specific cause of cancer. It is a matter of conjecture whether a true carcinogen can produce one cancer type in one group of workers and a different type in another group.

It is only untreated or mildly-treated mineral oils which are likely to be carcinogenic. Current refining processes extract all of the polycyclic aromatic hydrocarbons which were the probable cause of the scrotal cancers. Whilst the entire workforce of metal fitting and metal machinists are potentially exposed to mineral oils, not all would be using untreated or mildly-treated oils. The association has tended to be less with the more refined mineral oils used currently. The SA total workforce in these occupations is about 10000, and it is unlikely that more than 20% would be using untreated or mildly-treated oils. The near-absence of scrotal cancers suggests that this is not a significant risk from current usage. Although the occurrence of other cancer types from these oils is unproven, it is quite possible that the observed excess of bladder cancer in fitters and turners in the occupational study conducted by the SA Cancer Registry (12 cases observed/6.8 expected) was related to mineral oil exposure. On the foregoing assumptions it is estimated that 1 bladder cancer per annum is being generated from this source.

Soots. The cancer of concern here is cancer of the scrotum, associated with chimney sweeping. About 10-15 people are employed as chimney-sweeps, but their current activities due not involve the same intimate scrotal contact with carcinogenic substances as when this occupational cancer was first described in the 18th century.

Beryllium. It has proven impossible to obtain an estimate of the number of workers exposed to this metal. Beryllium has been classified as a carcinogen on the basis of epidemiological studies showing an excess of lung cancer. A recent critique of the epidemiological literature has suggested that the findings of excess cancer may have been due to a statistical anomaly consequent upon the "healthy worker effect".[18] In any case, the probable infrequent use of beryllium and the absence of any known cases of beryllium disease indicates that the incidence of beryllium-related lung cancer is insignificant.

Radon & its decay products. This exposure is associated with risk of lung cancer. Probably some of the lung cancer burden of the whole population is caused by environmental radon daughter exposure. About 300 workers are occupationally exposed, and are likely to be incurring greater than the ambient exposures, although individual exposures are well below the levels occurring in the epidemiological studies where the cancer risk was demonstrated. It is not established that radon daughter exposures at

these levels will produce excess lung cancers, but if so the relative is likely to be less than 2, since the lowest estimate of the doubling dose is 44 working level months (cumulative) - a level unlikely to be reached under present working conditions. A relative risk of 1.05 may be a realistic maximum estimate, which would lead to 0.05 lung cancers per annum being generated.

Laboratories & chemical suppliers. In the previous analysis of occupational cancer in *The Human Cost of Work* the results of a survey of the use of carcinogenic substances was presented. The survey asked whether any of the following carcinogenic or possibly carcinogenic substances were used or distributed:

β-naphthylamine
4 aminobiphenyl
4 nitrobiphenyl
N,N-bis (2 chloroethyl)-2-naphthylamine
Bis (2-chloroethyl) sulphide
Bischloromethyl ether
2-acetylaminofluorene
3,3'-dimethoxybenzidine
3,3-dichlorobenzidine
4-dimethylaminobenzene
alpha-naphthylamine
Ethyleneimine
Methylchloromethyl ether
β-propiolactone
N-nitrosamine
Methylnitrosourea
4,4 methylene bis(2-chloroaniline) or MOCA
auramine
magenta
o-aminoazotoluene
aflatoxins
methylazomethanol acetate
benzo(a)pyrene
dibenz(a)anthracene
benzidine
toxaphene
polychlorinated biphenyls
benzene
acrylonitrile
1,2-dibromo-3-chloropropane
vinyl chloride monomer

Of those who responded, the number reported to be using or distributed one or more of the above list is shown in Table 2.8.

Table 2.8. Result of the 1987 Survey of Carcinogenic Substances (SA Health Commission).

Premises	*Use*	*Non-use*
Chemical suppliers	1	18
Agricultural chemical suppliers	1	15
Industrial manufacturers & wholesalers	0	29
Medical laboratories	17	5
Research laboratories	6	9
Inspection and testing services	1	8
Tertiary education institutions	11	4
Analytical chemical suppliers	1	0
Chemical process industry	0	1
Analytical laboratories	8	2

It is likely that laboratories and similar institutions use these chemicals in very small quantities. The number of workers exposed is likely to be small except for tertiary institutions, where successive student intakes may be exposed for short periods. Whilst the volume of chemicals used and the number of workers using them are low, the safe use of these chemicals cannot be assumed, and it is quite possible that some cancers could be generated from these workplaces

Estimate of the total number of cancers being generated from current exposures. The foregoing estimates add up to 30-31 cancers per year from current exposures. To project from this estimate to the proportion of cancers occurring in any one year attributable to occupational exposure requires several more assumptions. The estimates are based on the cumulative exposure of the current workforce. Some further assumptions are (i) members of the workforce have been employed at their present jobs for an average of 10 years, so that they are equivalent to a "cohort" employed for 10 years, with the cohort being replaced every 10 years, (ii) each cohort contributes excess cancers at the estimated rate each year for 30 years after a 10-year latency period. (For example the 1985-1995 employment cohort contributes excess cancers from 2005 to 2035). Thus in any one year, 3 "cohorts" are contributing to the cancer burden, so that if all past and future exposures were at 1995 levels, there would be 92 occupation-caused cancers in males in any one year. If the total incident cancers in males were the same as in 1995, this figure would represent about 2.5% of all male cancers.

Discussion

This estimate may be too high, since it assumes that relative risks are just as high as in the epidemiological studies on which the categorisations of carcinogenicity were based, whereas exposures in most of the industries and chemicals discussed are likely to be much lower. For example, the estimate for nickel includes workers who work in electroplating and anodising, whereas their lung cancer risk is unlikely to be as great as in the nickel-exposed occupations on which the relevant epidemiological studies are based.

On the other hand the estimate of the number of cancers being generated from current exposures is not comprehensive, as it is based mainly upon chemical exposures categorised by the IARC as carcinogenic. A number of other cancers would need to be added to these:

(i) *Non-melanotic skin cancer from exposure to solar radiation.* This condition is not notifiable to the cancer registry but is extremely common. It is curable in the great majority of cases.

(ii) *Lip cancer due to solar radiation in outdoor workers.* At the present rate of occurrence, about 10 cases would be generated each year from this exposure. Increasing ultraviolet radiation from depletion of stratospheric ozone would tend to increase this risk, but current health promotion campaigns on skin care would tend to offset this.

(iii) *Cancer of the cervix in prostitutes.*

(iv) *Liver cancer and colon cancer in occupations involving the production and sale of alcoholic beverages.* It is not certain that this is truly an occupational cancer, as people accustomed to high alcohol consumption may gravitate to these industries. In any case the brewing industry now bans alcohol consumption on site, for safety reasons.

(v) *Liver cancer from chronic liver disease from occupationally-acquired Hepatitis B and C.* Although health care workers can now be protected against the former by vaccination, this is not possible for Hepatitis C. Unless and until a vaccine is available for Hepatitis C, liver cancer as a complication of this infection appears certain to occur.

(vi) The contribution of *nonionising radiation* to cancer is no clearer than it was 10 years ago.

(vii) There is a possible significant contribution to cancer from *occupational stress* (discussed in Chapter 8).

These other causes may well be more significant causes of cancer than carcinogenic chemicals. It is of course quite likely that there are other cancer-causing chemicals in use which are cancer-causing, but the above estimates of risk suggest that overall the problem of occupational carcinogens are small in relation to other factors. This is not because chemical carcinogens do not exist, but because exposures to agents such as asbestos are increasingly coming under control, and there are too few exposed workers to make a significant contribution to cancer incidence in the population.

Probably the greatest cause for concern is the extent to which current cancers are unexplained, and which may be occupation-related. Of principal concern are (i) leukaemias (specifically AML and CLL) which occur in excess in males from the age of 50, are not explicable by smoking and are more common in blue collar workers, but cannot be accounted for by known carcinogens, and (ii) bladder cancers, which are equally common in blue collar and white collar workers, and much commoner in males than females although the excess in men appears to be incompletely accounted for by the difference in smoking prevalence

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3. *Musculoskeletal injury*

R T GUN

Back injuries - trends in back injury rates - back injuries according to industry & occupation - rates in public sector employees - prevalence of back disorders in the workforce according to age, sex, industry & occupation - upper limb disorders - trends in incidence rates - rates according to industry & occupation - rates in public sector employees.

Summary

The rate of back injuries leading to more than 5 days of lost working time has increased more than 50% since the mid-1980s. Part of the increase is due to a higher proportion of claims leading to more than 5 days of lost time and hence appearing in the reported statistics, but there is also a probable increase in the absolute number of back injury claims. The highest rates in males occurred in the manufacturing and construction industries and in blue-collar occupations. Rates were about 70% higher in males than in females after allowing for the difference in the number of part-time workers (more females work part-time). State and Commonwealth Government employees had a low back injury rate, except in the South Australian Health Commission. The back injury rate of 12.5/1000 person-years in female SA Health Commission employees represents a 60% increase over the rate reported in the 1980s. An exceptionally high rate of back injuries was found in males in the local government sector.

The prevalence of back disorders in the community is higher in men than in women in all age groups. No industry or occupation groups have significantly elevated prevalence of back disorders. This is probably due to a selection effect, ie people with back problems which make working difficult change to more comfortable jobs, or if unable to do that, they move out of the workforce altogether. Exceptions were in males in agriculture and male managers and administrators; the former have a high frequency of back problems, probably because farmers are less likely to change their occupation, for family, social or financial reasons, and the latter because they can continue to work despite having back problems.

Although many physically demanding tasks have been mechanised out of existence in recent years, no benefit is seen in the rate of back injuries. The prevalence data also show that later birth cohorts have more back problems than do earlier birth cohorts, so that the increasingly sedentary nature of work in the Australian economy is apparently causing more back problems, not less. The Code of Practice for Manual Handling has not been in operation for many years, so that it is premature to pronounce it a failure. Nevertheless the principles underlying the Code of Practice have been recognised for some time - for example in the health care industry, where back injury rates have nevertheless increased. This suggests that a broader strategy may be required to reduce the incidence of back injury and the prevalence of back disorders.

The occurrence of upper limb disorders such as RSI has increased since the 1980s. The epidemic of this disorder in keyboard operators in the 1980s has gone, and the condition is now overwhelmingly preponderant in blue-collar workers. The incidence of conditions diagnosed as carpal tunnel syndrome has increased. This may be due to increased frequency of diagnosis from the use of nerve-conduction testing rather than a true increase in incidence.

Almost certainly, the true incidence of upper limb disorders is substantially underestimated by these statistics based on workers' compensation claims. Apart from the fact that the statistics only include

claims resulting in more than 5 days lost time, it is probable that a significant number of workers with upper limb problems choose not to claim compensation.

Introduction

Musculoskeletal injury forms an important subset of the injury statistics examined in Chapter 1, and deserves special attention because of some unique characteristics - in relation to causation, prevention and management.

These injuries are caused primarily from the actions of voluntary muscle - either from overexertion, repeated exertion, or both. Injuries in general result from the effects of the uncontrolled release of energy, and usually the energy source is external to the body of the injured person - from machinery, a falling ladder, a hot surface, etc. However in a substantial minority of cases, mainly musculoskeletal injuries, the energy source is internal - ie the contraction of voluntary muscle of the person who is injured. In most such cases the injury is confined to the muscle concerned or its tendinous or bony attachments, or to structures on which the muscle exerts leverage, such as the spine. On the other hand, injuries resulting from external energy sources commonly involve other tissues. For example a strained forearm muscle from lifting is unlikely to affect tissues other than the muscle or its attachments, whereas a sprained wrist from a fall may involve other tissues from direct trauma (e.g. bruising) or from passive stretching (e.g. torn ligaments).

The second important distinction of these types of injury is in relation to prevention. Prevention of musculoskeletal injuries from overexertion or repeated exertion is concerned mainly with reducing the demands on voluntary muscle in manual handling procedures. Since 1991 all Australian States have had in place regulations and a Code of Practice for Manual Handling as a means of preventing such injuries.

Finally these injuries are important because of their disproportionate contribution to lost time. Whereas injuries where tissue damage is obvious such as cuts, fractures, burns and amputation usually recover spontaneously with medical treatment with the worker returning to work relatively quickly, injuries where no pathology is obvious frequently are less responsive to a medical model of treatment and result in extended time off work, and even failure to return to work at all.

Accordingly, separate consideration is given here to two categories of musculoskeletal disorders - those of the back and those of the upper limb.

Back injuries

Back injuries may be identified by accident type - overexertion injuries of the trunk - or by injury type - sprains and strains of the trunk. However the "overlap" between injuries categorised in these two ways is not perfect. In the previous analysis of data from the 1980s (*The Human Cost of Work*) the latter classification was used, and for purposes of comparison it has been used again.

From 21 May 1991 WorkCover changed its coding system to that of the National Data Set, proposed by Worksafe Australia and adopted by the States as a minimum standard. Comparable data are not available after this date, but fortunately this affects only the last 5 weeks of the 3-year period of study. The injury rates have been computed by dividing the injury numbers for the period 30 June 1988 to 20 May 1991 by the person-time covered. To compute the person-time for 1990-91 the workforce numbers were multiplied by 47/52.

As in Chapter 1, the reported injuries are those resulting in more than 5 days of lost time.

Overall back injury rates in comparison with rates reported in the 1980s

Sprains and strains of the trunk accounted for approximately 30% of all injuries in both males and females.

Rates for both male and female workers for the 3-year period 1988-89 to 1990-91 were substantially higher than those previously reported for the period 1982-83 to 1984-85. The comparisons are shown in Table 3.1.

Table 3.1. Back injury rates for the periods 1982-83 to 1984-85 and 1988-89 to 1990-91, South Australia (no. back injuries/1000 person-years).

	1982-83 to 1984-85	*1988-89 to 1990-91*	*% increase*
Males	8.8	13.4	52
Females	3.4	5.7	68

In males the lost-time injury rate for this injury category was more than 50% higher than the rate previously reported for the 3-year period 1982-83 to 1984-85. Part of this increase is probably due to an increase in the proportion of injuries which lead to more than 5 days of lost time, and hence are reported in the statistics. As shown in Chapter 1, the rate for all reported injuries increased over the same period by about 25%, due to more injuries leading to more than 5 days of lost time rather than an absolute increase in the number of injuries. However in the case of back injuries this still leaves about one-half of the increase unexplained. This suggests that there was probably an absolute increase in the number of back injuries since the early 1980s. A real increase in the number of back injuries is suggested by the increase as a proportion of all injuries - from 26% in the period 1982-83 to 1984-85 to 30% in the period 1988-89 to 1990-91.

In females the findings were similar: the rate was 5.7/1000 person-years (31% of all injuries) in 1988-89 to 1990-91 compared with 3.4/1000 person-years (28% of all injuries) in the period 1982-83 to 1984-85.

Back injury rates by occupation

The rates of back injury by broad occupational category are shown in Table 3.2.

There was almost a 50-fold variation in back injury rates in males, from 0.68/1000 person-years in professionals to 31.18/1000 person-years in labourers and related workers.

In females there was a 13-fold variation in injury rates in males, from 1.28/1000 person-years in professional occupations to 16.46/1000 person-years in the category labourers and related workers.

Table 3.2. *Rates of sprains and strains of the trunk (number of injuries/1000 person-years) by occupational category by gender, 1988-89 to 1990-91.*

Occupation	*Males*	*Females*
Managers & administrators	1.8	1.3
Professionals	0.7	1.9
Para-Professionals	4.9	7.9
Tradespersons	17.9	5.0
Clerks	2.4	1.3
Salespersons & personal service workers	3.8	4.8
Plant & machine operators & drivers	23.1	9.9
Labourers & related workers	31.2	16.5
Total	12.7	5.5

In interpreting these data it is important to note that the information relates to injuries with more than 5 days of lost time. This has two important implications. First, as with other injuries, it may mean that most back claims are not reported in the statistics. Secondly, these data overstate the difference in back injury rates between manual and white-collar occupations, since the tasks performed by the former make it more difficult for them to continue working with their back pain.

Of individual male occupations, those with high injury numbers included assemblers, metal finishers and machinists, "other trades assistants and factory hands", storemen and truck drivers. Occupations with the highest number of injuries in females included enrolled nurses, registered nurses, ward helpers, cleaners, and "other trades assistants and factory hands".

Back injury rates by industry

Industry-specific rates of back injury are shown in Table 3.3. In males the rates varied by a factor of 10 - from 38.6/1000 person-years in the manufacturing of non-metallic mineral products to 3.70/1000 person-years in finance, property and business services.

In the male workforce rates tended to be higher in industries associated with manual occupations, ie mining, manufacturing, construction, electricity, gas and water and transport and storage. However the category Public Administration and Defence had a surprisingly high rate - 14.6/1000 person-years, slightly higher than the State average. Other manufacturing categories with high rates (greater than 30/1000 person years) were basic metal products and fabricated metal products. Outside the manufacturing sector the highest rate was in construction - 27.3/1000 person-years.

Table 3.3. *Rates of sprains and strains of the trunk (number of injuries/1000 person-years) by industry category by gender, 1988-89 to 1990-91.*

Industry	*Males*	*Females*
Agriculture, forestry, fishing & hunting	11.0	3.2
Mining	17.6	4.8
Manufacturing		
Food, beverages, tobacco	22.5	11.1
Textiles, clothing footwear	14.9	7.2
Wood, wood products, furniture	23.3	7.7
Paper, paper products, printing & publishing	8.3	4.5
Chemicals, petroleum & coal products	10.5	0.9
Non-metallic mineral products	38.7	4.2
Basic metal products	31.6	16.7
Fabricated metal products	31.7	11.4
Transport equipment	24.1	16.4
Other machinery & equipment	16.9	8.0
Miscellaneous manufacturing	26.4	20.1
Total manufacturing	21.5	9.7
Electricity, gas and water	19.3	3.3
Construction	27.3	1.2
Wholesale & retail trade	7.6	3.3
Transport & storage	18.6	4.2
Finance, property & business services	3.7	2.2
Public administration & defence	14.6	2.8
Community Services		
Health	10.7	12.8
Education, museum & library services	3.2	2.7
Welfare & religious institutions	8.1	8.5
Other community services	13.0	2.1
Total community services	7.7	8.3
Recreation, personal & other services	5.8	3.7
Total	13.4	5.7

Similarly the highest rates in the female workforce were in the manufacturing sector, the highest rate being in miscellaneous manufacturing - 20.1/1000 person-years. This includes industries such as leather and leather products, rubber and rubber products, plastic and plastic products, ophthalmic articles, jewellery and brooms and brushes.

The rate for males was higher than for females in all industry categories except community services, where the rates were higher in the health and welfare sub-categories.

Part of the difference between male and female rates is due to the different exposure time, that is, more men work fulltime. Indeed the method of calculation, in person-years, affects the ratio of male rates to female rates. Expressed in this way the male back injury rate was 2.4 times that of females. When allowance is made for hours worked, ie the rates are expressed in terms of injuries per million hours worked, the male:female rate ratio for back injury reduces to 1.7.

(The overall rates for males and females are slightly higher than in the analysis by occupation, due to updating of the data in the period between the analyses.)

State Government employees

Table 3.4 shows the back injury rates for employees of State, local and Commonwealth Governments and the South Australian Health Commission, with the comparable figures for the total workforce covered by the WorkCover legislation. (Thus the Overall SA workforce includes all Government sectors except the Commonwealth).

Table 3.4. *Rates of sprains and strains of the trunk (number of injuries/1000 person-years), for the SA workforce overall and the various tiers of government, 1988-89 to 1990-91.*

Sector	*Males*	*Females*
Overall SA workforce*	12.7	5.5
State Government (exc SA Health Comm)	8.8	3.4
SA Health Commission	13.7	12.5
Local government	25.5	4.9
Commonwealth Government**	5.2	2.4

* *Covers all categories, including private sector, but excludes Commonwealth employees*

** *excludes Australia Post and Telecom*

Back injury rates in the State Government sector were low. This is probably an effect of the relatively low proportion of the State Government workforce employed in manual jobs. The 1991 Census data show that tradespersons, plant and machine operators and drivers make up only 30% of the State Government workforce compared with 45% of the overall male workforce.

The rate in females employed by the South Australian Health Commission was more than twice the overall female rate. This no doubt reflects the high proportion employed as nurses. The back injury rate of 12.5/1000 person-years in female SA Health Commission employees represents a 60% increase over the rate reported in the 1980s in *The Human Cost of Work.* The back injury rate in males employed by the SA Health Commission is similar to that of female employees.

A remarkably high rate of back injuries was found in males in the local government sector - double that of the State average. This is in part due to the proportion on manual workers, which is higher than in the overall SA workforce (64%, compared with 45%).

Commonwealth employees, both male and female, have a substantially lower back injury rate than workers covered under the WorkCover scheme. As discussed in Chapter 1, this is likely to be partly due to the relatively low proportion engaged in manual jobs.

Prevalence of back disorders in the workforce

The Australian Health Survey is a periodic survey conducted by the Australian Bureau of Statistics, in which a sample of Australian householders are interviewed about their health, with simultaneous demographic information being obtained such as age, occupation and industry of employment. Information from the 1983 survey was reported previously in *The Human Cost of Work.* The most recent survey was conducted in 1989-90, and the ABS has provided information on the occurrence of back conditions in the labour force.[1] Information was obtained from respondents on the occurrence of back problems - sciatica, disorder of the intervertebral disc or unspecified back disorder - either as chronic problems or problems which required treatment in the 2-week period prior to interview. Chronic disorders accounted for nearly all of the occurrences.

The prevalence of chronic back disorder by age and sex is shown in Table 3.5.

Table 3.5. Percentage prevalence of chronic back disorder, by age and sex, Australia, 1989-90.

Age	*Males*	*Females*
15-44 years	15.2	13.3
45-64 years	22.8	18.4
65 years and over	15.7	12.1
Total (unadjusted)	17.2	14.3

Men have a higher prevalence of back disorder, by about 20% in all age categories. Bearing in mind the higher proportion of men than women in paid employment, and the larger proportion who are working who are in fulltime work, this difference seems surprisingly low. This suggests that paid employment may only be responsible for a small proportion of the prevalence of back disorders; this possibility is discussed below. It also suggests that there are certain specifically female factors specific to women responsible for some of the prevalence of back disorders in women, possibly including child-bearing, child-rearing, simultaneous responsibilities for paid and unpaid employment, and biological factors.

The ratio of 1.2:1 for men to women reporting back disorders in this household survey is of course much less than the equivalent ratio (2.3:1) obtained from the compensation data. This is probably partly due to the tasks performed by male workers, which are on the whole more physically demanding, and therefore less likely to be within the worker's capability when he has a back injury.

The finding that the prevalence of back disorder was lower in the oldest age group than in the 45 to 64-year group is unexpected. This may have a simple explanation, ie that back problems improve when the worker has retired. On the other hand there may be a cohort effect. The 65 and over age group represents the pre-1925 birth cohort. They started work before 1940 and mostly stopped work by 1990. The 45-64 age group represents the 1925-1940 birth cohort, who started work between 1940 and 1960, and most were still working in 1990 when the survey was undertaken. During this time important changes occurred in working conditions. Many of the heaviest jobs disappeared or were mechanised or automated. Yet in spite of this the younger age cohort has a higher prevalence of back disorders. A possible explanation is that the earlier age cohorts had to maintain a higher level of physical fitness than those now in the workforce, or that the heavier work demands produced a higher degree of physical fitness. If this were so, it may mean that the disappearance of heavy jobs has resulted in more back disorders, not less.

Table 3.6 shows the relative prevalences of chronic back disorders in different industry categories. To derive this table, an expected rate was derived for each industry category, which is the prevalence of back disorder expected if there were no relationship between industry and the probability of back disorder, after adjusting for age differences.

Table 3.6. *Age-standardised prevalence ratio of chronic back disorders by industry - males. (Source: Australian Health Survey, 1989-90.)*

Industry	*Observed prevalence*	*Expected prevalence*	*Obs/Exp (%)*
Agriculture	64.7	48.5	133
Mining	18.0	17.3	104
Manufacturing	135.7	156.8	86
Electricity etc	18.8	17.7	106
Construction	107.1	95	113
Wholesale	139.5	139.2	100
Transport	64.7	56.8	114
Communication	19.8	18.6	106
Finance	82.6	78.3	105
Public Admin	42.1	47.6	88
Community services	80.3	82.3	98
Recreation	44	42	104

Most of the industries show a prevalence of back disorder close to the expected level. This is possibly a selection effect, ie people who develop back problems which interfere with their work will tend to move to another job. If this caused enough people with back problems in, say, the construction industry, to move to wholesale trade, this would tend to even up the prevalences in the two industries. All the same, the ability to change industries is not likely to be great. A more probable explanation is that people with severe back disorders will leave the workforce altogether.

The only industry group with a prevalence substantially above the expected rate is agriculture. This is no doubt a reflection of the high rate of Australian farms which are owner-operated (even in the age of agri-business), so that farmers cannot leave the farm (for financial reasons) or do not wish to do so (for family or personal reasons).

Table 3.7 shows the relative prevalence of back disorders in different industry categories in females.

Table 3.7. *Age-standardised prevalence ratio of chronic back disorders by industry - females. (Source: Australian Health Survey, 1989-90.)*

Industry prevalence	*Observed prevalence*	*Expected (%)*	*Obs/Exp*
Agriculture	18.7	17.5	107
Mining	2.0	2.2	91
Manufacturing	47.5	50.7	94
Electricity	2.3	1.7	135
Construction	8.3	9.4	89
Wholesale	98.6	97.5	101
Transport	8.4	11.8	71
Communication	5.8	4.7	123
Finance	61.5	64.3	96
Public admin	23.3	23	101
Community services	146	138	106
Recreation	52	50	104

Again, the prevalence rates are close to the expected values - even in the community services sector, which includes nurses. The relatively high rates in communications and electricity, gas and water are difficult to explain except on the basis of sampling error.

Table 3.8 shows the relative prevalence of back disorders by occupational category in both males and females.

Almost all occupational categories have a prevalence of back disorders close to their expected value, even closer than in the analysis of prevalence by industry. This probably reflects the effects of people with back disorders changing occupations or migrating out of the workforce altogether.

Table 3.8. *Age-standardised percentage prevalence ratio of chronic back disorders by occupation. (Source: Australian Health Survey, 1989-90.)*

Occupation	*Males*	*Females*
Managers & administrators	127	108
Professionals	96	104
Para-professionals	96	109
Tradespersons	103	108
Clerks	100	97
Salespersons & personal service workers	103	100
Plant & machine operators & drivers	100	97
Labourers & related workers	90	100

Discussion

The main legislative weapon to combat the occurrence of occupational back injury is the *Worksafe Australia Regulations and Code of Practice on Manual Handling*, which were adopted in all Australian States in the early 1990s. Close monitoring of the trends in back injury rates is important to evaluate the effectiveness of the regulatory strategy.

Comparison of back injury rates reported in the early 1980s fails to show any improvement. Indeed the reported rate of back injury has increased. Although this is partly due to the increase in the proportion of injuries which lead to more than 5 days of lost time which has occurred since the introduction of the WorkCover scheme, there has probably been an absolute increase in the total number of back injury claims also, since the proportion of all claims contributed by back injuries (in males) has risen from about 26% in the period 1982-83 to 1984-5 to 30% in the period 1988-89 to 1990-91.

Since the Manual Handling Regulations and Code of Practice was not introduced until well into the triennial period under study it would be unrealistic to expect it to result in a reduction in the reported rate of back injuries so soon. Nevertheless the type of measures required in the Code of Practice have been practised to some extent even before the Code of Practice became law. Up to the early 1980s the only effective regulation was a prohibition on lifting weights above certain limits by women and minors. This provision was abolished because its effect was to deny certain jobs to women. Instead it was expected that employers would use other means of reducing the number of manual handling injuries, such as undertaking task analyses and modifying tasks so as to minimise overexertion and inappropriate methods of manual handling.

Irrespective of what the law has prescribed, it is probable that many heavy tasks have been mechanised out of existence, yet the back injury rate has not fallen and has probably risen. The advent of the Code of Practice may change this for the better by ensuring wider compliance, but trends since the 1980s suggest that this cannot be taken for granted. Thus the available evidence suggests that in spite of the disappearance of the more physically arduous tasks and in spite of increasing awareness of the need to assess the risks of manual handling tasks, the problem of musculoskeletal injury is no less than it was 10 years ago. This suggests that there are other important factors in the causation of such injuries apart from physical workload. One possibility is that whilst heavy work may be a risk factor for back injury, lack of physical activity may also be a risk factor. It appears plausible that people who are not accustomed to

physical work may be more vulnerable to injury from the occasional heavy task that most people must perform occasionally.

A possible contributing factor in the increased back injury rate since the mid-1980s is an increased reporting rate, ie an awareness effect following the introduction of the Manual Handling Code of Practice.

There is cause for similar concern from the 1989-90 Australian Health Survey data on chronic back disorders. The above analysis of these data show that later birth cohorts have more back problems than do earlier birth cohorts, so that the increasingly sedentary nature of work in the Australian economy is apparently causing more back problems, not less. Again this suggests that there are other important factors apart from physical workload. The finding that the prevalence of back disorders in men is not greatly above that in women provides support for this view, as does the weak relationship between occurrence of back disorders and occupation (manual versus non-manual), and other findings suggesting that the contribution of having a manual occupation to the likelihood of back injury is only about 15%.[2]

It is thus probable that much more than the Code of Practice on Manual Handling will be required not only to reduce the incidence of compensable back injury, but also of chronic back disorders in the community generally.

This has some implications for the workers' compensation system. Although occupation is only one contributing factor to the occurrence of back injury, the attending doctor has a difficult problem. It is not always possible to tell by clinical or other diagnostic means which cases are work-caused and which are not, and the understandable reaction of attending doctors is to attribute the injury to work wherever there is doubt. Whilst this ensures justice to those whose injuries are work-caused, it means that industry is internalising what is a community-wide problem, and is discriminatory against others with non-occupational injuries or spinal diseases, who are not so entitled, and whose only entitlement may be the Commonwealth disability pension.

The high rate of back disorders in nurses was noted in the previous report, and is a continuing cause of concern. Despite the fact that many hospitals have invested a great deal in training programs for nursing staff, and lifting devices have been standard in most health care institutions for several years, the back injury rate in the health industry has risen sharply since the early 1980s - from 6.7 to 10.7/1000 person-years in males, and from 7.8 to 12.8/1000 person-years in females.

The high rate of back injuries in male workers in the local government sector is partly explicable by the high proportion of workers engaged in manual occupations. Nevertheless this workforce is not engaged in any of the really high-risk industry sectors, so there may be other factors. An aging workforce may be a contributory cause, but age-stratified data are not available. The impression is that local government has an uncontrolled problem.

It is of considerable concern that it will not be possible to compare future statistics on the occurrence of back injuries with these data. It is with considerable difficulty that comparability of the present data with pre-WorkCover data could be assured. This will not happen in future due to the adoption of the Worksafe Australia National Data Set.

Upper limb disorders (including RSI)

The previous report was compiled at the time of what was known as the RSI (repetitive strain injury or occupational overuse syndrome) epidemic and in that report incidence rates were presented for the period 1980-81 to 1985-86. The data were subsequently updated to 1986-87 in a separate paper,[2] which showed that the occurrence of the syndrome known as RSI was endemic, particularly in blue-collar workers, with a superimposed epidemic which was more prevalent in clerical workers, especially keyboard operators.

The epidemic was a matter of controversy in several respects, including whether the condition exists at all. Unlike other upper limb disorders such as tennis elbow, medial epicondylitis or deQuervain's disease, "RSI" is not to be found in the International Classification of Disease (ICD-9). Nevertheless, arm pain, often without any accompanying clinical or radiological signs is not uncommon in manual workers, and numerous such cases occurred in the 1980s of such severity that they resulted in prolonged physical and even psychological incapacity. Thus despite the absence of a mention in the ICD-9, numerous such cases appeared in the compensation statistics.

In *The Human Cost of Work* RSI cases were classified differently according to whether they had been assigned to the category of a disease or an injury.

Diseases were placed in one of the following ICD-9 categories:

354 mononeuritis of the upper limb and mononeuritis multiplex (this category includes carpal tunnel syndrome)

726 peripheral enthesopathies and allied syndromes (enthesopathies are disorders of peripheral ligaments or muscular attachments)

727 other disorders of synovium, tendon and bursa

728 disorders of muscle, ligament and fascia

729 other disorders of soft tissue

Accidents which were caused by repetitive movement were assigned a special type of accident code, code 44.

In the previous analysis all the above disease categories were included, together with accidents whose type of accident code was code 44 and which affected the upper extremity.

Trends in RSI since 1980-81

Data for these 3 categories and the totals for the period 1980-81 to 1990-91 for males and females are presented in Tables 3.9 and 3.10 respectively.

Perhaps contrary to expectations, the data do not suggest the end of an epidemic - rather the total number of cases in males has risen in recent years, and in females the recent rates are comparable to those occurring in the 1980s.

Table 3.9. Repetitive strain injuries and related disorders, males, 1980-81 to 1990-91.

Year	*CTS*	*RSI*	*Accident 44*	*Total*
1980-81	22	172	90	284
1981-82	33	209	97	339
1982-83	27	214	79	320
1983-84	18	241	98	357
1984-85	23	226	140	389
1985-86	17	196	157	370
1986-87	19	191	135	345
1987-88	-	-	-	-*
1988-89	43	367	79	489
1989-90	80	443	95	618
1990-91	64	374	47	485

CTS: carpal tunnel syndrome (ICD-9 354)
RSI: ICD-9 codes 726-729
Accident 44: Accidents, caused by repetitive motion, affecting the upper limb
* *no statistics available for 1987-88*

The data also show some changes between categories of upper limb disorders. Carpal tunnel syndrome (CTS) makes up 11.7% of the claims since WorkCover was introduced, compared with 6.6% for the years 1980-81 to 1986-87. CTS has become increasingly recognised as an occupational disorder. Extensive use of nerve-conduction testing on people presenting with impaired nerve function of the hand has led to the identification of median nerve compression in the carpal tunnel, with the probable result that some cases which would have been classified as "RSI" (ICD 726-729) or "injury due to repetitive movement" (accident code 44) in the 1980s are classified as CTS in the 1990s.

Table 3.10. Repetitive strain injuries and related disorders, females, 1980-81 to 1990-91.

Year	*CTS*	*RSI*	*Accident 44*	*Total*
1980-81	22	137	77	236
1981-82	34	150	92	276
1982-83	32	182	95	309
1983-84	19	240	137	396
1984-85	22	245	345	612
1985-86	27	189	292	508
1986-87	20	97	217	334
1987-88	-	-	-	-*
1988-89	43	235	129	407
1989-90	49	324	94	467
1990-91	35	256	79	370

CTS: carpal tunnel syndrome (ICD-9 354)
RSI: ICD-9 codes 726-729
Accident 44: Accidents, caused by repetitive motion, affecting the upper limb
* *no statistics available for 1987-88*

Incidence rates

Table 3.11 shows a comparison of incidence rates for upper limb disorders between the year 1983-84 and the period 1988-89 to 1990-91. They show that the female and male rates have converged - the female rate declining and the male rate increasing since the early 1980s.

Table 3.11. Incidence rates for RSI and related disorders for 1983-84 and the period 1988-89 to 1990-91, South Australia (no. cases/10^4 person-years).

	1982-83	*1988-89 to 1990-91*	*% increase*
Males	14	22	52
Females	26	21	68

Upper limb disorders by occupation

Table 3.12 shows the incidence of these conditions by occupation. The categories with the highest incidence are the blue-collar manual occupations, ie tradespersons, plant and machine operators and drivers, and labourers.

Table 3.12. Incidence of (number of injuries/10^4 person-years) by occupational category by gender, 1988-91.

Occupation	*Males*	*Females*
Managers and administrators	2.5	3.3
Professionals	1.0	2.7
Para-Professionals	3.9	3.2
Tradespersons	51.2	21.7
Clerks	4.4	9.1
Salespersons & personal service	3.3	7.9
Plant & machine optrs, drivers	27.8	97.5
Labourers & related workers	57.6	76.9
Total	21.9	20.6

There has been a sharp decline in the incidence of this condition in clerical workers since the RSI epidemic of the 1980s. Table 3.13 shows a comparison of the incidence of RSI and related disorders in clerical and manual occupations between the year 1982-83 and the period 1988-89 to 1990-91. These figures should be interpreted with caution, as the occupational classification system has changed since the mid-1980s data were analysed. Nevertheless the occupations classified as "clerical" are likely to be similar for both periods. The manual occupations in the 1985-86 data were those in the category "tradespersons, process workers and labourers", whereas for the later period, the categories were "tradespersons", "plant and machine operators and drivers", and "labourers and related workers".

Table 3.13. Incidence rates for RSI and related disorders for 1985-86 and the period 1988-89 to 1990-91, selected occupational categories (no. cases/10^4 person-years).

	1985-86	*1988-89 to 1990-91*	*% increase*
Female clerical workers	25	9	-64
Female tradespersons, labourers etc	106	80	-25
Male tradespersons, labourers etc	23	48	109

In the 1980s RSI was considered to be an epidemic affecting primarily female clerical workers. In fact the greatest numbers and the highest incidence rates in females were in the manual blue-collar occupations. Nevertheless there was a relatively high number of cases in female clerical workers, although the incidence was much lower than in the blue-collar occupations (ie a high proportion of the female workforce is employed in clerical occupations). More recently there has been a very steep decline in the incidence of these disorders in female clerical workers. The rate in female blue-collar workers has declined slightly, while the rate in blue-collar manual workers has doubled since the middle 1980s. However female workers in manual occupations are still the most-affected category.

Upper limb disorders by industry

The distribution of RSI cases by industry is shown in Table 3.14.

By far the highest rate was in females in the category miscellaneous manufacturing: the rate in this sector has in fact more than doubled since 1985-86.

Table 3.14. Incidence rates of RSI and related disorders (number of injuries/10^4 person-years) by industry category by gender, 1988-89 to 1990-91.

Industry	*Males*	*Females*
Agriculture, forestry, fishing & hunting	61.9	81.4
Mining	15.5	11.1
Manufacturing		
Food, beverages & tobacco	75.2	105.6
Textiles, clothing & footwear	36.1	95.0
Wood, wood products, printing & publishing	40.1	63.0
Paper, paper products, printing & publishing	13.2	63.4
Chemicals, petroleum & coal products	12.5	9.3
Non-metallic mineral products	55.3	0
Basic metal products	56.6	63.9
Fabricated metal products	55.4	85.8
Transport equipment	51.2	139.9
Other machinery & equipment	30.8	113.9
Miscellaneous manufacturing	45.1	231.2
Total manufacturing	44.4	101.2
Electricity, gas & water	10.1	46.7
Construction	47.2	4.4
Wholesale & retail trade	10.6	13.2
Transport & storage	20.0	3.9
Communication	0	0
Finance, property & business services	4.6	11.7
Public administration & defence	9.9	9.2
Community Services		
Health	5.8	13.8
Education, museum & library services	2.1	5.9
Welfare & religious institutions	3.9	11.4
Other community services	11.9	5.2
Total community services	5.3	10.3
Recreation, personal & other services	7.9	11.7
Total	22.8	20.9

Government employees

Table 3.15 shows the rates for employees of State, local and Commonwealth Governments and the South Australian Health Commission, with the comparable figures for the total workforce covered by the WorkCover legislation. (Thus the overall figures include all Government sectors except the Commonwealth).

RSI appears almost to have disappeared in the State Government sector (outside the SA Health Commission). This may reflect the general decline in the occurrence of this condition in female clerical workers, who were relatively heavily affected in the 1980s. As was seen for back injuries, there was a high rate of upper limb disorders in men employed in local government.

Table 3.15. Incidence rates of RSI and related disorders (number of injuries/10^4 person-years) government and overall workforce, 1988-89 to 1990-91.

Sector	*Males*	*Females*
Overall SA workforce[1]	21.9	20.6
State Government [2]	0.3	0.7
SA Health Commission	6.9	14.0
Local government	27.6	12.0
Commonwealth Government[3]	5.9	19.1

1 Covers all categories, including private sector, but excludes Commonwealth employees
2 excludes SA Health Commission, ETSA and STA
3 excludes Australia Post and Telecom

The relatively low rate in the Commonwealth sector on males is probably due to the relatively low number of manual workers. The rate in female Commonwealth employees is about equal to that of the overall female workforce, which is higher than would be expected given that few of these female workers would be employed in blue collar manual occupations.

Discussion

The "RSI epidemic" of the 1980s primarily affected female clerical workers. The incidence of these conditions has declined sharply in this category since then. Although this decline coincided with vigorous intervention programs (the provision of "ergonomic" furniture, job rotation, limits on keystroke rates and rest breaks are now commonplace), it is not clear what features of the intervention programs, if any, are responsible. Elsewhere it has been suggested that possible explanations for the reduction in RSI incidence in keyboard operators, were (i) a Hawthorne effect, ie a non-specific behavioural or psychological effect induced by the intervention irrespective of its content, (ii) employees not reporting the condition for fear of discrimination, including job loss and future unemployability from being stigmatised as an RSI case, or (iii) a selection effect, ie that all those at risk have either developed the condition already or have transferred to occupations with less risk.[3] However it is still quite possible that the workplace interventions have been responsible for the decline in the incidence of these conditions in keyboard operators.

The incidence of these disorders has not abated in blue collar occupations, and has actually increased in males, but by far the highest incidence is in females in the category miscellaneous manufacturing. In an analysis of data from the 1980s it was argued that this finding is attributable to many women having to work in repetitive, unfulfilling tasks which generate feelings of boredom, alienation and powerlessness.[3] However more recent sociological research suggests that women in general, even those in low-paid, low-status jobs experience more job satisfaction than men. Although women experience more constraints on their employment opportunities than men, such constraints are more likely to result in loss of work satisfaction in men than in women.[4] One explanation proposed is that paid work is accorded less importance than "the marriage career" in women's expectations and aspirations, so that they invest less of themselves in their paid jobs than men, whereas men seek more substantial rewards from the work domain which is their main life activity.[5] Indeed, an Australian study has concluded that for working mothers, paid work, although not "leisure", is often seen as a welcome break from the relentless demands of housekeeping and child-raising.[6] These findings call into question the belief that the high rate of claims for RSI and related disorders in unskilled blue-collar women workers derives from lack of job satisfaction or lack of autonomy in their jobs or in their employment options. Rather, they favour the more simple proposition that the best prospects for preventing such disorders lies in more mechanistic measures, such as reduction in the repetitive activities of particular muscle groups, and making work postures more comfortable.

It should be emphasised that the true incidence of upper limb disorders is probably underestimated by these statistics based on workers' compensation claims. Apart from the fact that the statistics only include claims resulting in more than 5 days lost time, it is probable that a significant number of workers with upper limb problems choose not to make any compensation claim at all. A recent report from Connecticut gives an indication of the extent of such underestimation. Whereas compensation data for 1992 show 1571 cases of injury from repeated trauma (including carpal tunnel syndrome), a survey of a probability sample of the State workforce showed an equivalent estimate for the same year at 3454.[7]

These seemingly disparate findings in fact point to a coherent conclusion. Many women working in repetitive tasks suffer from upper limb problems. Some of these women make compensation claims but many do not. Regardless of what factors - pain, disability, social factors, psychological factors, - motivate workers to claim or not to claim compensation, there remains an underlying high prevalence of upper limb disorders which requires attention, principally by addressing specific workplace issues, such as repetitive tasks, postures, mechanical workload and physical fitness.

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4. *Effects of exposure to physical agents*

R T GUN

Noise and noise-induced hearing loss - heat strain - effects of exposure to cold - barotrauma - vibration - radiation.

Community prevalence data from the SA Health Omnibus Survey of 1994 showed a prevalence of self-reported hearing loss of 15.7% in adults. The prevalence in males was 18.7% and in females 13.9%. The prevalence of hearing loss was higher in workers whose main lifetime occupations were in the "blue-collar" than in the "white-collar" groups in all age groups and in both sexes. If the excess prevalence in males represents the burden of noise-induced hearing loss, 26% of the hearing loss in adult males is due to noise exposure at work.

However there is now evidence of an emerging problem in the female workforce, as seen by the reported occurrence of hearing loss in 22 of the 74 blue-collar female workers sampled in the workforce, and the lodgement of 24 successful claims for noise-induced hearing loss by female employees of non-exempt employers (compared with 555 males) in the 3-year period under study.

The proportion of cases attributable to noise could be underestimated, as it may be higher in the elderly male population who are no longer in the labour force; this group was excluded from this analysis as their past occupation was not recorded in the survey.

120 subjects had obtained a professional opinion that they had a hearing loss caused by occupational exposure to noise (although this included 51 subjects who had given affirmative answers to the specific question on difficulty in hearing). 3 of these 120 subjects reported a severe hearing loss, and 36 had had a hearing aid prescribed.

There is a very high prevalence of hearing loss - 35% - in farmers aged 45 years and over.

The rate of compensation claims for noise-induced hearing loss in different industries correlated moderately well with a priori expectations of noise exposure ($r = 0.52$).

The frequency of claims for noise-induced hearing loss was in the non metallic minerals manufacturing industry; this includes this includes manufacturing of glass and glassware, clay, bricks, cement, concrete, glass wool plaster etc. Other industries with apparently high claims rates are manufacturing of basic metal and fabricated metal products.

In the 3-year period 1988-89 to 1990-91 there were 8 recorded cases of heat strain resulting in more than 3 days of lost time. Maximum ambient temperatures on the days of these occurrences ranged from 27.5 to 42.9^{o}C.

Since 1990-91 there have been several serious cases of barotrauma (from decompression), affecting divers in the tuna farming industry, which has been established in South Australia after the period covered by these data.

Noise

A number of adverse health effects have been examined for a possible association with noise exposure, for example, raised blood pressure and psychological effects. However the health effect most clearly associated with noise exposure is hearing loss, and since this is the only noise-related health effect which is sufficiently well documented to quantify, it will be the only effect considered here.

Acute noise exposure can cause hearing loss, from damage to the acoustic nerve, or from damage to structures associated with sound conduction (tympanic membrane or the foramen rotundum). However

noise-induced hearing loss is mainly an insidious condition, resulting from repeated noise exposure which damages the cochlear nerve endings. A threshold shift occurs, ie there is an elevation in the minimum sound pressure level required for the sound to be heard; initially the threshold shift is reversible, but repeated exposure to high-level noise leads to permanent hearing loss. Characteristically the frequencies affected first are in the region of 4000Hz, which is above the speech range, so that mild degrees of hearing loss may not be noticed by the subject. Comparisons of a hearing loss survey based on interviews (the US National Health Interview Survey) with a survey based on audiometry (the US National Health and Nutrition Examination Survey) suggest that the individual is likely to become aware of hearing difficulty when there is a 20 decibel or more hearing loss.

Prevalence and distribution of hearing loss

Data are available on hearing loss from the Health Omnibus Survey, an annual survey of a sample of South Australian households, conducted by the South Australian Health Commission. Data from the 1994 survey have been analysed according to respondents' main lifetime occupation. (This survey uses a probability sampling strategy, so that the numbers given in the tables are obtained from sample numbers adjusted to represent the true population proportions.)

Of 2884 adult persons interviewed, 472 (15.7%) reported a hearing loss. The prevalence in males was 18.7% and in females 13.9%.

The distribution of hearing loss in males by main lifetime occupation and age is shown in Table 4.1. The prevalence of hearing loss was 12% in the 15-45 year age group and 28% in the 45-years-and-over age group.

Table 4.1. Hearing loss by occupation and age, males. (Source: Health Omnibus Survey)

	15-44 yrs		*>45 yrs*	
	HL	*Persons*	*HL*	*Persons*
Higher professionals	7	114	15	67
Managers and administrators	5	26	6	33
Higher clerical	3	20	1	14
Higher sales	5	38	15	39
Lower professional	7	52	17	42
Farmers	0	8	13	35
Higher production	24	166	36	119
Higher service	7	24	2	12
Middle production	24	164	38	147
Lower clerical	3	48	8	29
Lower sales	3	31	1	16
Farm workers	2	28	4	14
Lower service	2	37	2	9
Lower production	9	66	11	28
Total	100	821	168	604

Table 4.2 shows the comparable data for female workers.

Table 4.2. Hearing loss by occupation and age, females. (Source: Health Omnibus Survey, 1994)

	15-44 yrs		*>45 yrs*	
	HL	*Persons*	*HL*	*Persons*
Higher professionals	4	88	4	22
Managers and administrators	8	76	31	148
Higher clerical	7	97	12	78
Higher sales	1	13	3	15
Lower professional	12	103	13	82
Farmers	0	1	4	10
Higher production	1	8	4	22
Higher service	3	48	5	26
Middle production	5	36	12	35
Lower clerical	11	107	16	86
Lower sales	14	108	6	53
Farm workers	2	8	2	11
Lower service	4	77	17	68
Lower production	0	15	4	17
Total	72	784	132	674

The prevalence of hearing loss was lower in females than in males in both age strata - 9% in the 15-45 year age group, and 20% in the 45-years-and-over age group.

One means of assessing the contribution of employment to hearing loss is to compare the prevalence in occupational groups which *a priori* may be expected to have a higher noise exposure with groups which are not. Accordingly it is hypothesised that the manual blue-collar occupations will have a greater prevalence of hearing than other occupational groups due to greater occupational exposure to noise. In Table 4.3 the prevalence of hearing loss and NIHL in these occupational categories are compared. The manual blue-collar occupations in this survey are taken to be the higher, middle and lower production worker categories, which correspond to the categories tradespersons, plant and machine operators, and labourers and service workers used elsewhere in this volume.

Table 4.3. Prevalence of hearing loss and diagnosed noise-induced hearing loss (NIHL) according to occupational category.

	15-44 years		*>45 years*	
	Proportion	*Percent*	*Proportion*	*Percent*
Males				
White collar	42/390	11	66/261	25
Blue collar	57/396	14	85/294	29
Farmers	2/36	6	17/49	35
All males	101/822	12	168/604	28
Females				
White collar	62/716	9	107/579	18
Blue collar	6/59	10	22/74	30
Farmers	2/9	22	6/21	29
All females	70/784	9	132/674	20

White-collar occupations: all other than blue collar and farmers and farm labourers.
Blue-collar occupations: higher, middle and lower production workers.

Blue collar occupations are seen to have a higher prevalence of hearing loss in both sexes and both age categories. The difference in prevalence between blue-collar and white collar occupations in males is only 3% in the 15-44 year age group and 4% in the 45+ category. The smallness of this difference may be due to occupational noise experienced by people who spent some time in manual jobs before entering white-collar occupations.

The 30% prevalence of hearing loss in blue-collar female workers aged 45 years and over is higher than expected, being greater than the corresponding figure for males; however the estimate for females is based on relatively small numbers.

If it is assumed that the entire excess prevalence of hearing loss in males is caused by noise exposure, the estimate of hearing loss due to noise exposure in the adult male population caused by noise is a little over 5%.

As shown in Table 4.3, there is a very high prevalence of hearing loss - 35% - in farmers aged 45 years and over.

360 subjects in the survey had had the cause of their hearing loss diagnosed by a doctor or other professional person. In 120 of these - 112 males and 8 females - the diagnosed cause was exposure to loud noise at work. However 51 of these subjects had not reported difficulty in hearing.

The data suggest that the occurrence of occupational hearing loss is very low in females, although it is greater than zero.

Severe occupational hearing loss

The Health Omnibus Survey data were also analysed for the severity of hearing loss in those cases which were attributed to noise exposure. This was assessed in two ways: (i) from respondents whose hearing loss was work-related and was so severe that they reported difficulty in hearing even if spoken to loudly, and (ii) people whose hearing loss was work-related and who need to wear a hearing aid.

Of 120 cases of hearing loss caused by occupational exposure to noise, 3 reported severe hearing loss, and 36 had had a hearing aid prescribed.

Compensable hearing loss

Data on compensable cases of noise-induced hearing loss are incomplete, as the WorkCover Corporation only has data on claims made by employees of companies who insured through WorkCover. Data on self-insurers were missing. Over the three-year period 1988-89 to 1990-91 there were 579 successful claims for noise-induced hearing loss paid by WorkCover - 555 in males and 24 in females.

The distribution of claims by industries is shown in Table 4.4. The industry sector with the greatest number of claims was transport and storage. Other categories with high claim numbers were in the manufacturing sector - basic metal, fabricated metal, and non-metallic mineral products.

Table 4.4. Noise-induced hearing loss by industry group (non-exempt employers only), 1988-89 to 1990-91.

	Males	*Females*	*Employed males**
Agriculture, forestry, fishing	4	0	10764
Mining	35	0	2984
Manufacturing			
Food, beverages, tobacco	14	1	8860
Textiles, clothing, footwear	14	2	2218
Wood, wood products, furniture	19	1	4738
Paper and printing	6	1	4992
Chemical, petroleum products	5	0	1839
Non-metallic minerals	53	1	2277
Basic metal products	65	2	4591
Fabricated metal products	64	1	4747
Transport equipment	23	0	10408
Other machinery and equipment	43	4	7554
Miscellaneous manufacturing	14	6	3615
Total manufacturing	320	19	17274
Electricity, gas and water	7	1	6209
Construction	36	0	17274
Wholesale and retail trade	43	0	51142
Transport and storage	74	0	11630
Finance, property & business services	9	0	22318
Public administration	2	0	10760
Community services	21	2	34310
Recreation, personal & other services	7	1	6209
Not classifiable	1	0	
Total	555	24	239724

* *Total includes employees of exempt organisations.*

The number of males employed in each industry by non-exempt employers is not available, hence it is not possible to calculate rates. However an indication of comparative rates between industries may be obtained from the total number employed in each industry category, and these numbers (from the 1991 Census) are shown in Table 4.4. Because some industries are predominantly covered by exempt employers, the apparent claims rate will appear low; examples of such industry categories are electricity, gas and water, and community health services (both largely employed by the State Government and thus exempt), and basic metal manufacturing (of which at least one-half are employed by an exempt employer). Nevertheless some indication can be obtained of the relative severity of the industrial noise problems in various categories. Non-metallic mineral manufacturing stands out as having the highest rate of hearing loss; this includes manufacturing of glass and glassware, clay, bricks, cement, concrete, glass wool plaster etc. Other industries with apparently high claims rates are manufacturing of basic metal and fabricated metal products.

Noise exposure

Data on noise exposure in local industry are not available. Table 4.5 shows an analysis of noise exposures based on the US National Occupational Hazard Survey of 1972-74. Noise exposure in this survey was not quantified, but was based on a measurement of 85dB(A) or more for at least 30 minutes

Discussion

The analysis of the community prevalence data shows a prevalence of hearing loss of 18.7% in adult males compared with 13.9% in females. If the excess prevalence in males represents the burden of noise-induced hearing loss, 26% of the hearing loss in adult males is due to noise exposure at work.

However there is now evidence of an emerging problem in the female workforce, as seen by the reported occurrence of hearing loss in 22 of the 74 blue-collar female workers sampled in the workforce, and the lodgement of 24 successful claims for noise-induced hearing loss by female employees of non-exempt employers (compared with 555 males) in the 3-year period under study.

The proportion of cases attributable to noise in males could be underestimated, as it may be higher in the elderly male population who are no longer in the labour force; this group was excluded from this analysis as their past occupation was not recorded in the survey.

Of the 120 subjects in this study who had obtained a professional opinion that the cause of their hearing loss was occupational noise, 51 had not reported difficulty in hearing. The cause of this anomaly is unclear. Possibly some subjects had had some hearing loss diagnosed audiometrically, despite the absence of any subjective awareness of difficulty in hearing. This is quite possible since some impairment as shown by audiometry is compatible with no perception of hearing loss, especially if the impairment is in the frequency range above 2000Hz.

The high rate claims for noise-induced hearing loss in the manufacturing of non-metallic mineral products suggests that this industry has an uncontrolled noise problem. On the other hand, the traditionally noisy timber industry has had a relatively low rate of claims. This is not likely to be attributable to regulatory control, as there has never been a prosecution for breach of the noise regulations, although they have been in force in South Australia since 1972.

Heat strain

In the 3-year period 1988-89 to 1990-91 there were 8 recorded cases of heat strain resulting in more than 5 days of lost time. All cases were males. The occupations of the cases were:

2 furnace operators
a farm hand
a construction worker
a garbage collector
a library clerk
a designer
other trades assistant

The task performed by the library clerk was described as shifting core trays, which suggests foundry work. The employment classification "library clerk" may therefore be an error. The "other trades assistant" was a man who had been clearing a road of leaves in 42-degree heat.

It would be expected that individuals affected by heat strain would recover fairly quickly, so that the proportion of cases resulting in more than 5 days of lost time would be low. Accordingly it is likely that the total number of cases of heat strain is considerable higher than the number of reported cases shown in the statistics.

Information was obtained from the Bureau of Meteorology on the maximum temperature in Adelaide on each of the days in which these cases occurred. (However it is not known whether these cases occurred in the Adelaide metropolitan area.) The maximum temperatures were: 33.5°C, 38.9°C, 27.5°C, 36.2°C, 35.5°C, 31.1°C and 42.9°C.

All of these cases occurred in the summer months. Not all of these maximum temperatures were extreme, which indicates that other factors probably contributed to the total heat burden, such as a high physical workload, hot industrial processes, heavy clothing impairing the evaporation of sweat, or failed airconditioning. Nevertheless, the fact that all cases but one occurred when the temperature was above

30°C shows the need to control all heat stress risk factors in the summer months, despite the low humidity which usually accompanies hot weather in South Australia.

Effects of cold

There were four cases of effects of cold. One was a worker who was overcome by cold when working in a freezer. One other case occurred in a freezer, the worker suffering frost bite and synovitis in both feet. The other two cases were of muscular strain aggravated by cold.

Barotrauma

These are the effects of high ambient pressure or from decompression. This would include divers affected by bends.

The number of such cases in the period under study is not easy to determine, but it is likely that there were at least six cases. Three of these were cases of pressure effects on the ear in people travelling in aircraft, and the other three were divers.

In the years since 1990-91 there have been several serious cases of barotrauma in divers in the tuna farming industry, which has only been established in South Australia after the period covered by these data.

Hand-arm vibration

Prolonged use of vibrating hand tools (chain saws, jackhammers, angle-grinders) can lead to upper limb disorders. The effects may be neurological, with sensory impairment of the fingers, or episodes of circulatory insufficiency of the fingers, as in Raynaud's phenomenon. The latter condition occurring in association with vibration is known as vibration white finger. The injury classification system does not readily allow vibration-related conditions to be identified.

Other risk factors for Raynaud's phenomenon include smoking and cold weather, and it is likely that vibration white finger results from a combination of occupational use of vibrating hand tools and the other risk factors. Although the workforce would include a significant number of people using vibrating hand tools, the absence of cases of vibration white finger is probably explicable on the same basis as the low number of cases of cold injury, ie the South Australian climate.

Whole-body vibration

Drivers of heavy (eg earth-moving) equipment, trucks and buses are subject to whole-body vibration. The effects of whole-body vibration are not well defined, although there is some epidemiological evidence that it can contribute to spinal disorders.[4] There is no means of identifying the role of whole-body vibration in any individual with a back problem, so that the available data cannot give any information in the extent to which vibration contributes to the prevalence in the population.

Radiation

Barring a major accident, ionising radiation exposures are not sufficient to cause any acute effects on exposed workers.

Various types of cancer may result from exposure to ionising radiation. In South Australia the greatest potential risk arises from underground uranium miners exposed to radon daughters, and health care workers using irradiating apparatus or radioisotopes. This subject is addressed in Chapter 2.

Nonionising radiation, by definition, does not have the energy to cause ionisation of living tissue with the resultant genetic damage. Nevertheless there is some epidemiological evidence to suggest that nonionising radiation at certain frequencies (eg radiofrequency) has some association with excess risk of brain cancer and leukaemia. The significance of these associations for persons occupationally exposed to nonionising radiation is the subject of much current research.[5,6]

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5. *Occupational respiratory disease*

R T GUN

Effects of environmental contaminants on the respiratory system - acute chemical bronchitis - asthma - infection - silicosis - asbestosis - extrinsic allergic alveolitis - pneumothorax - other chest conditions.

Summary

There were 148 cases of compensable respiratory disease in the period 1998-89 to 1990-91, the most common cause being asthma (35 cases in males, 16 in females). Only 9 of these cases were attributed to isocyanates, vegetable dusts or colophony, which are the more common exposures associated with occupational asthma.

An examination of the SA death registry data for 1989-1993 failed to confirm the recent UK finding of excess mortality from lobar pneumonia in welders.

There were 4 deaths from silicosis and 11 deaths from asbestosis in SA from 1989-1993.

A survey of respiratory physicians found that 19 cases of silicosis and 54 cases of asbestosis have been seen in South Australia in the last 5 years. This suggests that workers' compensation data understate the occurrence of these dust diseases. Industries associated with silicosis included mining and sandblasting; and industries associated with asbestosis included asbestos mining, milling and manufacturing, insulation work, construction and demolition, shipping, boiler fabrication, railways, and power station construction and maintenance.

There were 5 cases of pneumothorax. Two cases were from penetrating injuries, and the other 3 appear to have been caused by overexertion.

There were 4 claims for vocal cord nodules in teachers. There were also several cases of non-specific respiratory disease in teachers, suggesting that some teachers are successfully claiming that upper respiratory infections are work-caused in circumstances where other workers cannot or do not.

Introduction

Inhalation of airborne contaminants can give rise to a variety of respiratory disorders. The nature of the adverse response varies with the contaminant, and the characteristic responses to various contaminants is set out in table 5.1.

Table 5.1. Respiratory effects of some atmospheric contaminants (reproduced from The Human Cost of Work).

Effect	*Causal agents*
Acute bronchitis or pneumonitis	Irritants, eg chlorine
Increased susceptibility to infection	NO_2 (natural gas cooking, tobacco smoke, welding), ozone
Increased mucus production (chronic bronchitis)	Tobacco smoke, coal mining, cotton dust (byssinosis)
Reversible bronchospasm (asthma)	Numerous agents, including isocyanates, grain dust and flour, colophony, Western Red Cedar and other wood dusts, cotton dust (byssinosis)
Extrinsic allergic alveolitis	Fungal spores
Fibrosis (pneumoconiosis)	Silica, asbestos
Granuloma formation	Beryllium
Emphysema	Tobacco smoke, cadmium
Cancer	Tobacco smoke, other combustion products, asbestos, radon daughters, arsenic, nickel.

Reported cases of compensable respiratory disorders

The cases of compensable respiratory disorders in South Australia for the 3-year period 1988-89 to 1990-91 are shown in Table 5.2. Only cases which resulted in more than 5 days of lost time are recorded in the statistics. The statistics may include some recurrences, as the data do not include any individual identifiers which would enable recognition of any people who appear more than once for the same condition.

Table 5.2. Respiratory disorders, South Australia, 1988-89 to 1990-91.

	Males	*Females*
Acute chemical bronchitis	2	3
Asthma	35	16
Pneumonia	4	6
Silicosis	1	0
Asbestosis	2	0
Extrinsic allergic alveolitis	1	0
Pneumothorax	4	1
Allergic rhinitis	8	2
Other conditions	41	22
Total	98	50

Over the 3-year period there were 148 such cases of respiratory disorders, 98 in males and 50 in females.

Acute chemical bronchitis or pneumonitis

There were 4 cases of bronchitis or pneumonitis from acute exposure, from exposure of a teacher to chlorine from a swimming pool, from exposure of an enrolled nurse to floor stripper, (presumably a solvent such as methylene chloride), exposure of a labourer to dust from a building site, and 2 of workers exposed to unspecified fumes.

Asthma

Occupational asthma is defined in various ways. It includes asthma arising *de novo* from an occupational cause in a person not previously known to be asthmatic. A broader definition would include asthma aggravated by an occupational exposure in a person already known to be asthmatic. The following data will include both categories, since the data cannot distinguish between the two (and in any case both the management and the entitlement to compensation is no different).

WorkCover statistics show that in 1988-89 to 1990-91 there were 35 asthma cases in males and 16 in females. The distribution by occupation is shown in Table 5.3

Male tradespersons and labourers were the most commonly affected occupational groups. No particular occupation occurred with high frequency. However there were 5 cases in spray painters, or panel beaters.

Table 5.3. Cases of asthma by occupation.

	Males	*Females*
Managers and administrators	0	0
Professionals	1	2
Paraprofessionals	3	1
Tradespersons	13	1
Clerks	1	5
Sales and personal services	0	2
Plant and machine operators	5	0
Labourers	12	5
Total	35	16

Most of the records fail to state the causal exposure. A few give enough information to make some attempt to determine whether the asthma was precipitated by any of the generally-recognised causes of asthma.

Isocyanates

Only in 2 cases was isocyanates specified as the cause. Two other cases occurred in vehicle spray painters and may therefore have been caused by isocyanates. Intriguingly, one case of asthma was attributed to "cyanates" (in a jewellery worker).

Vegetable dusts

There was only one case attributed to grain dust and one in a cabinetmaker caused by "dust", presumably wood dust. One case occurred in a bakery employee.

Solder

Colophony, a flux in lead solder, is a well-recognised cause of asthma. One case was attributed to soldering fumes, although colophony was not cited.

Animal allergy

One case was attributed to being near a cat, and one case was attributed to allergy to lobster.

Cold

Two cases were attributed to exposure to cold, and another to a temperature change.

Cigarette smoke

Two cases were attributed to cigarette smoke, and one other to "smoke".

Other causes

Other cases of interest included:

- one case in a hairdresser
- one in a shoe repairer
- one case, described as severe, from exposure to floor stripper
- one case from exposure to welding fumes. The stated cause was "welding fumes (aluminium)". If aluminium was being welded, ozone may have been the cause.
- one case in a scientific technician from exposure to an enzyme.

Infection

Certain atmospheric contaminants are believed to increase susceptibility to respiratory infections. Oxides of nitrogen are respiratory irritants, and are suspected of causing an increased rate of respiratory infections. This hypothesis has arisen from studies comparing towns which have converted to natural gas with others which have not.[1] Natural gas burns at a higher temperature than coal gas, and produces oxides of nitrogen, and higher rates of respiratory infections have been reported in communities where natural gas is used.

This could have some significance in occupational environments where oxides of nitrogen are evolved. Certain types of welding, for example, produce nitrogen dioxide as well as ozone. A number of studies have suggested that welding carries an increased risk of respiratory disease. Another occupation considered to be at risk is working in grain terminals, where nitric oxide has been associated with a pneumonitis and known as silo-fillers' disease.

More recently the British Office of Population, Census and Statistics has reported a disproportionate mortality from lobar (pneumococcal) pneumonia in welders.[2] This finding has been so consistent that the author has claimed that lobar pneumonia should be recognised as an occupation-related disease in welders.

In the WorkCover data over the 3-year period there were 8 recorded cases of pneumonia (other than chemical pneumonitis) - 3 in males and 5 in females. They included one attributed to exposure to cold, one in a ward helper who caught the infection from a patient, one who developed infection after exposure to fumes, and a teacher who apparently developed an infection in the normal course of duty.

None were in welders. This may be due to lack of recognition of a work-related problem by doctors, since lobar pneumonia caused by exposure is unlikely to have any unique features. Even a cluster of cases at a workplace is likely to go unrecognised unless all welders visited the same doctor.

To examine this issue further, South Australian mortality data for 5 years - 1989-93 - were examined for any association between lobar pneumonia and welding. Death registry data are difficult to analyse by occupation, since the occupational classifications are very broad. Nevertheless there was only one death record, in a female, where occupation was associated in any way with welding. Of course it is possible that no association was found because lobar pneumonia cases caused by welding fumes may be treatable and do not therefore appear in the death records; however the hypothesis from the UK was based on death records. On the basis of the available data, therefore, there is no evidence to support the hypothesis that welding carries a risk of lobar pneumonia.

Silicosis

Three methods were employed to measure the occurrence of silicosis:

(i) Information from WorkCover on compensable claims.

(ii) Analysis of SA mortality data, 1989-93, from the Australian Bureau of Statistics.

(iii) A questionnaire, addressed to respiratory physicians practising in SA, on the number of cases of silicosis seen since 1989, and the industries responsible.

- There was one case of silicosis compensated in the 3-year period. There was insufficient detail to identify the process which gave rise to the exposure.
- There were 4 deaths certified as caused by silicosis in the period 1989-93. In 3 cases the occupational category was "Managers and administrators" and the other case was a pensioner. All cases were male.
- Not all respiratory physicians replied to the questionnaire, but responses were received from the 3 major teaching hospitals. In most cases no formal records are maintained, so that respondents emphasised that they were able to provide estimates only. With these qualifications it was estimated that about 19 cases had been seen since 1989. Industries identified included mining and sandblasting, but in most cases no occupational information was available.

Asbestosis

The same three methods used to assess the occurrence of asbestosis were also used for asbestosis.

(i) There were 2 cases compensated for asbestosis. However both cases were also described as mesothelioma. It is not clear whether these cases had both conditions, or whether the coder was unaware of the distinction between the two.

(ii) There were 11 deaths due to asbestosis in the period 1989 to 1993, all in men. The occupational categories were listed as follows:

managers and administrators	2
tradespersons	2
public servant	1
foreman/supervisor	1
plant and machine operators & drivers	1
labourers and related workers	2
pensioners	2

(iii) As with silicosis, most physicians responding to the questionnaire stressed that they were providing estimates only. The total of the estimated numbers of cases of asbestosis from 1989 to the present (July 1995) was 54.

Again, occupational information was incomplete. However where industries were mentioned, they were those usually associated with asbestos-related disease, ie asbestos mining, milling and manufacturing, insulation work, construction and demolition, shipping, boiler fabrication, railways, and power station construction and maintenance.

Respondents were asked to exclude cases of pleural plaques without lung disease.

Extrinsic allergic alveolitis

One case of this condition was reported. It was attributed to exposure to spray paint, in the industry category "manufacturing of non-metallic mineral products".

Pneumothorax

There were 5 reported case of pneumothorax. The available details are as follows:

(i) A plumber lifting a hot water unit into position

(ii) A truck driver sustaining a haemopneumothorax when a crowbar being used to dig a hole struck him in the chest

(iii) A worker lifting a roll of surfacing material

(iv) A nurse performing lifting duties

(v) A panel beater suffered a burn from a welding rod from a fellow worker, the rod penetrating his chest.

Other chest conditions

There were 10 cases of allergic rhinitis, 8 in males and 2 in females.

The other cases of respiratory disease were mostly classified as either "acute laryngitis and tracheitis" or "other disease of the respiratory tract". Of these 53 cases, 19 were cases of teachers. Some of these teachers attributed their illness to the occupational hazard of speaking. 4 of these cases were stated to have nodules on the vocal cords, but it is not known if any of these were recurrent claims. Some other cases in teachers were of laryingitis, presumably aggravated by speaking. In other cases involving teachers the diagnosis was less specific (eg other disease of the respiratory tract) and the link between respiratory disease and work was less clear.

Discussion

The most salient feature of the data on asthma is that they are of little or no value for the surveillance of serious respiratory disease, such as asthma. There are 2 possible means by which this might be corrected, and either way the information must be obtained direct from the doctor who makes the diagnosis. One way would be for the information obtained by WorkCover to be subject to quality control by a person with nosological training, and who can check any information by making direct contact with the doctor, as occurs with the SA Cancer Registry. The alternative would be to emulate the SHIELD program of the United Kingdom in which periodic questionnaires are sent to respiratory physicians and occupational physicians, seeking details of occupational respiratory conditions and their cause.[3]

There is a marked difference between the exposure information on the causes of asthma in the South Australian data compared with the SHIELD program, in which a high proportion of cases are attributable to well-recognised causes of occupational asthma. As suggested above, the absence of any recognised cause in most cases in the South Australian data may be due to the nature of the compensation system: any medical condition to which work made any contribution at all was compensable. Thus although the asthma may not have been initiated by any of the known

occupational asthmagens, it may have been judged to have been aggravated in a non-specific (ie non-immunologically mediated) way by an occupational exposure to an airborne irritant.

The gender distribution of adult-onset asthma suggests that occupation is not a large contributor to the incidence of asthma. If a condition is commonly associated with occupation (or with smoking), it is usual to find a preponderance in men. Adult-onset asthma, on the other hand, is commoner in women, according to the most recent Australian Health Survey.[4] Although data are not available on the incidence of asthma, the Survey included questions both on whether the respondent had experienced asthma during the 2-week period prior to interview and whether he/she was a chronic asthma sufferer. The proportion of affirmative answers to both questions was consistently higher in women than in men.

The occurrence of 5 cases of pneumothorax is surprising, since this is not generally regarded as an occupation-related condition. Two of these cases were caused by a penetrating injury, but the other 3 were cases of closed pneumothorax associated with lifting. Presumably the workers strained against a closed glottis, raising their intrathoracic pressure, and rupturing the pleural surface of the lung. This is a surprising outcome, and should prompt some enquiry as to whether these workers had some pre-existing lung condition such as asthma or emphysema, which predispose to formation of bullae on the lung surface which may rupture leading to pneumothorax. If this was the mechanism, some note should be taken of the risk of pneumothorax to workers with asthma or emphysema who have to lift weights.

There were 4 cases of vocal cord nodules in teachers. This problem is known to occur in occupations requiring frequent use of the voice such as singers, as well as in teachers.[5] At the same time, the frequency of claims for non-specific respiratory disease in teachers, in circumstances which appear to arise simply from the performance of their normal duties, suggests that they are successfully claiming compensation for conditions for which other workers must use their sick leave. Of course, it is plausible, indeed probable, that many upper respiratory infections are caused by person-to-person transmission at work, thus theoretically conferring an entitlement to compensation. However it appears that the Education Department is less critical of such claims from its own workforce than are other employers in this State.

The questionnaire to respiratory physicians indicated an occurrence of about 20 cases of silicosis over a period of about 5 years, compared with only one case in three years compensated under WorkCover. The cases seen by the respiratory physicians were not necessarily new cases, so that some may have already received compensation payments under the pre-WorkCover arrangements. The low number of deaths from silicosis is as expected, since the condition is not always severely disabling and death often occurs eventually from an unrelated cause. For this reason mortality data are an inadequate source for surveillance of silicosis.

Despite the informal nature of this estimate, it does have some have some plausibility when compared with the data from the NSW Dust Diseases Board. In 1994 there were 22 successful new claims for silicosis in NSW, and in 1993-94 there were 258 workers receiving compensation payments for silicosis. After allowing for the differences in population and workforce numbers, there is probably a higher incidence of silicosis in NSW, which is attributable to Sydney being built on sandstone.

The findings of this survey show that there is a need to establish a formal system of continuing surveillance for silicosis. It is important to identify the industries and exposures responsible for this condition, and particularly whether any people in the current workforce are still being exposed to levels of silica likely to result in this preventable disease.

As with silicosis, the asbestosis cases were not necessarily incident cases. In NSW the total number of workers receiving compensation for asbestosis in 1993-94 was 329, ie about 50% higher than the number of silicosis cases, whereas in South Australia the estimated number of asbestosis cases was

(allergens) are usually of low molecular weight and are relatively simple compounds which combine with skin proteins to form complete antigens which stimulate the immune system to produce an allergic reaction. The first allergic response will take about five days to develop after a sensitizing exposure. Once sensitized the time between exposure and an allergic dermatitis may be as short as 6 hours but usually 12-24 hours. Sensitization may occur on the first exposure or after many years of exposure.

Only some of a population are likely to be affected by sensitizers whereas irritants will affect virtually the entire population if there is a sufficient duration of exposure. The word "contact" is used to describe either irritant or allergic dermatitis.

NIOSH[3] considers that the reason for substantial under-estimates of occupational skin diseases are under-recognition, under-reporting and misclassification.

Occupational dermatitis has been investigated by Drs Freeman and Rosen[4] in Sydney and Drs Wall and Gebauer[5] in Western Australia. Major industries and occupations identified as risk factors for allergic or irritant contact dermatitis were:

- hairdressing, food, construction, medical, and maintenance[4];
- food handlers/chefs/butchers;
- maintenance fitters/plant operators;
- medical/dental/nurses/nurses/veterinarians;
- construction;
- and hairdressers/beauticians [5].

Smit et al[6] found the prevalence of hand dermatitis in the general population in the Netherlands was 5.2% for men and 10.6% for women. Amongst various occupational groups hand dermatitis ranged in prevalence from 2.9% in office workers to approximately 30% in nurses. Smit et al suggested that key issues related to the prevalence of dermatitis were heavy exposures (eg. in nurses from frequent washing of the hands) and low to moderate exposures to irritants in combination with mechanical stress (such as are found with manual workers). Smit et al found that sick leave was required in only 3-9% and medical attention in 15-30% of cases.

In a study of "over 230 workers" in the Melbourne construction industry, Nixon[7] found 24 workers with allergic reactions to epoxy chemicals, 22 of whom demonstrated allergic contact dermatitis with patch testing with two others demonstrating contact urticaria identified by prick or scratch testing. Six of the workers showed immediate and delayed hypersensitivity reactions. The majority did not take time off work and "almost 30% of the workers with allergic reactions did not even consult a doctor". In this study the rate of reactions to epoxy resin hardeners (which are involved in floor coatings, painting, adhesives, concrete repair and bonding, and laminated carbon fibre) was higher than in overseas studies and it was suggested that this may be due to "the increased volatility of the chemicals in the warmer Australian working conditions".

The commonest allergens seen in the Freeman and Rosen [4] study were potassium dichromate; thiuram mix; and epoxy resin. The study was of 570 patients with acute occupational dermatitis. 36% were between 15 and 24 years of age and "most of these were women apprentices in hairdressing and the food industry". 24% developed dermatitis in their first year of an occupation but the average period before the development of contact dermatitis was 7.3 years. The peak incidence period in the construction industry was the 35 to 44 year age group. Irritant dermatitis was more common in the hairdressing and medical/dental group but in the construction workers 67% had allergic dermatitis. Overall 40% had allergic dermatitis and 60% had irritant dermatitis and there was no statistically significant difference in outcome between the two groups. The rate of improvement was greater in people who did not have a past history of hayfever, eczema or asthma than those who did.

Slovak [8] notes that people with an atopic disposition make-up "just over one third of the working population" and that more than a quarter of current occupations "involve exposure to a potential sensitizer". In some industries (eg. the platinum and proteolytic enzyme industries) people with an atopic disposition are screened out from employment but this will not exclude problems entirely. There are differing definitions of atopy which result in descriptions of markedly different populations. There can also be considerable variations over time for people considering themselves to have a positive family or past personal history (Slovak, 1993). A difference of 40% was noted in histories taken 5 to 7 years apart. Slovak considers the use of atopy as a screening tool is "scientifically unsubstantiated... (and) ...unacceptable".

Information about toxicity to the skin is often limited and difficult to obtain or to interpret. Much toxicological data relates to single applications at full strength [3].

NIOSH[3] suggests that the major causes of irritant dermatitis are:

- soaps, detergents, miscellaneous cleaning agents;
- solvents;
- hard particulate dusts (eg. fibrous glass);
- food products;
- miscellaneous plastics or other resins (p70).

Risk factors

Risk factors for irritant dermatitis include[3]:

- an atopic constitution;
- increased hydration of the skin's outermost protective layer, the stratum corneum (such as by wearing plastic gloves for prolonged periods);
- chemical contact with more permeable areas of skin (eg. eyelids, face and genital skin);
- pre-existing skin diseases or injuries (eg. abrasions or dermatitis may significantly increase the absorption of chemicals when the physical protective barrier is disrupted).

Risk factors for allergic dermatitis are similar to those for irritant dermatitis except for atopy which is not considered to increase the risk for allergic dermatitis. Pre-existing irritant dermatitis may be a predisposing factor the prognosis (outlook) for irritant or allergic dermatitis is often not good particularly with delayed diagnosis and treatment. Even with changes of occupation the dermatitis often persists[3].

Sunlight as a risk factor for skin cancer, is being more widely appreciated and preventative measures are being developed by industry.

Nature of the conditions

Infectious diseases comprise 40-50% of the skin conditions. They are allocated to skin diseases (rather than infectious disease) if they affect only the skin. As such cellulitis is included as are carbuncles (boils) and furuncles (collection of boils).

Compared to the data in *The Human Cost Of Work* [9] which covered the years 1982-1985, the rate of claims approximately doubled in the period 1988-1991 to about 220 reported cases each year. The percentage of claims in the last year of the survey (1990-1991) was markedly less for both males and females comprising 23% and 26% respectively instead of the expected 33%.

Contact dermatitis (and other eczema) was the most common condition for both males and females (See Table 1).

Table 6.1. Reported skin conditions, 1988-91

Skin conditions	*Males*	*Females*
Contact dermatitis & other eczema	57%	77%
Infective conditions [a]	32%	14%
Other conditions	11%	9%
Total	100%	100%

a Infective conditions include:

Other cellulitis and abscess
Other local infections of the skin and sub-cutaneous tissue
Cellulitis and abscess of finger & toe
Carbuncles & furuncles
Impetigo

b Other conditions include:

Corns & callosities
Pilonidal cysts
Other disorders of the skin and sub-cutaneous tissue
Other hypertrophic & atrophic conditions of the skin
Disorder of the sebaceous glands
Diseases of the nail
Erythematosquamous dermatosis
Chronic ulcer of the skin
Psoriasis & similar disorders
Urticaria
Disorder of sweat glands
Dermatitis due to substances taken internally

Incidence data

Industries

Incidence varies between 0 (communications) and 12.4 (manufacturing) per 10,000 person years. Rates for industries above the mean (5) were: manufacturing (12.4),mining (9.5); construction (8.6), and recreation, personal and other services (7.4).

For males the rates varied between 0 (communications) and 13.5 (manufacturing). Industries above the mean rate (6.4) were manufacturing (13.5), agriculture, forestry, fishing and hunting (10.8), mining (10.1), and construction (9.8).

For females the rates varied between 0 (communications) and 9.1 (manufacturing). Rates for industries above the mean (3.3) were manufacturing (9.1), recreational, personal and other services (8.4), mining (5.6), and agriculture, forestry, fishing and hunting (3.6).

Occupations

For combined males and females the rate per 10,000 person years ranged from 0.2 (clerks) to 13.2 (tradespersons). Besides tradespersons other occupations with greater than the average rate of 5 were labourers and related workers (13.1) and plant and machine operators (7.8). (Check data for labourers and related workers).

For males the rates per 10,000 person years varied from 0.2 (clerks) to 11.9 (tradespersons). Besides tradespersons (11.9), the other occupational classification above the average rate (6.4) was plant and machine operators and drivers (7.5).

For females the rates ranged from 0.2 (managers and administrators, and clerks) to 22 (tradespersons). The occupational category 'tradespersons', includes hairdressers which have been identified in many studies as being of high risk. Besides tradespersons (22), other occupations above the average rate of 3.3 were plant and machine operators and drivers (9.3) and labourers and related workers (9.7).

Table 6.2. Skin diseases by industry

Industry	*Total Cases (3years)*	*Workforce Total*	*Rate per 10,000 person years Males & Females*	*Rate per 10,000 person year Males*	*Rate per 10,000 person years Females*
Agriculture, forestry, fishing & hunting	40	15,188	8.8	10.8	3.6
Mining	10	3,523	9.5	10.1	5.6
Manufacturing	294	79,244	12.4	13.5	9.1
Electricity, gas & water	5	6,916	2.3	2.6	0
Construction	52	20,262	8.6	9.8	1.0
Wholesale & retail trade	74	93,420	2.6	3.5	1.7
Transport & storage	18	14,949	4.0	4.6	1.8
Communications	0	376	0	0	0
Finance, property & business services	17	47,522	1.2	1.5	0.9
Public administration & defence	12	16,545	2.4	2.8	1.7
Community services	66	107,182	2.1	1.3	2.4
Recreation, personal & other services	75	33,628	7.4	6.0	8.4
Total	663	438,755	5.0	6.4	3.3

The industries where the female rate of occupational skin diseases exceeds the male rate is in community services which takes in health care and recreation, and personal and other services which takes in hairdressing.

Table 6.3. Rates of skin disorders. 1988-1991. exempt and non-exempt employers

Occupation	*Total Cases (3 years)*	*Workforce Total*	*Rate per 10,000 person years Males & Females*	*Rate per 10,000 person years Males*	*Rate per 10,000 person years Females*
Managers & administrators	11	46,529	0.8	1.0	0.2
Professionals	8	58,650	0.4	0.3	0.6
Para-professionals	19	34,707	1.8	1.2	2.4
Tradespersons	247	62,238	13.2	11.9	22.0
Clerks	4	67,673	0.2	0.2	0.2
Salespersons & personal service workers	28	72,461	1.3	1.3	1.3
Plant & Machine Operators & Drivers	81	34,777	7.8	7.5	9.3
Labourers & related workers	265	67,644	13.1	6.4	9.8
Total	663	444,379	5.0	6.4	3.3

In reviewing individual case histories, it was noted that people handling fleeces or animal skins were at risk of occupational skin infection. Animal handlers and meat workers were at risk of developing warts and several instances of rubbing safety shoes resulted in cases requiring more than 5 days off work.

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7. *Infectious & parasitic diseases*

ANDREW LANGLEY

Causes, incidence & classification - risk factors - nature of conditions - incidence by occupation & industry - prevention.

Summary

In the workplace, people may be exposed to a range of trivial and sometimes severe infectious and parasitic diseases. There are about eighty reported cases of occupation-related infectious diseases each year in South Australia particularly affecting "community services" (which includes health care and teaching), 'agricultural', and 'manufacturing" (which includes meatworking) industries.

Introduction

There are a wide range of infections associated with occupations. With some the mode of transmission and opportunity clearly indicate that the infection has arisen in the workplace, (e.g. orf in a shearer or Q fever in a slaughterman); in others the infection may have arisen in the workplace or in the community (e.g. tuberculosis in a migrant nurse).

Occupational infections can be placed in two general categories:

- infections in which contact with animals, their byproducts or excreta put humans at risk. If these infections are transferred from vertebrate animals they are known as zoonotic infections e.g. brucellosis, Q Fever, leptospirosis;
- infections in which people occupationally in close contact with other people, their biological fluids or excreta are at risk, eg hepatitis B, tuberculosis, scabies.
- infections arising from pathogens in the general environment in a susceptible person eg tetanus in an unimmunised person; Legionnaires' Disease in an immunocompromised person; tinea in a person working in gumboots so that their skin is macerated.
- infections arising from environmental conditions so that the normal microbiological flora of the body are 'unbalanced' and infections arise eg candida causing paronychia (a chronic infection around the cuticle of the nail) with wet work in the hospitality industry; pseudomonas otitis externa in a swimming instructor.

Some of the key facts about infections are:

- there is a difference between infection and disease: Infection is the entry and multiplication of an infectious agent in the body. The infection may cause no symptoms at all, minor symptoms or severe illness. It is only when there are symptoms that a person is considered to have dis-ease. With infections there are nearly always very specific tests to indicate infection but these do not always indicate how severe the disease is. With brucellosis a meatworker may have had no symptoms or felt that he/she had a mild flu. On the other hand, the illness may have been severe with fevers, shaking and extreme weakness;
- it may be difficult to pin down exactly when (or where) the person contracted the infection. The incubation period is usually somewhat variable. With a short incubation period it is usually easier to pin down the likely time of contact than with a long incubation period (e.g. the incubation period

for staphylococcal food poisoning is 2-4 hours but for Hepatitis B it can vary from 6 weeks to 6 months);

- diseases will have specific sources, means of transmission and host subjects which are invariable. Theories of how an infection was contracted must fit these constant parameters. For instance hepatitis A has a human source and a human host and is most likely to have passed between them by faecal-oral transmission. A nurse will therefore not have developed hepatitis A from a patient having coughed towards him or her.

An occupational connection may be relatively easy to detect with some infectious and parasitic diseases. A number of these diseases such as Q-fever and leptospirosis have long been acknowledged to be occupational diseases; while other diseases can be attributed to occupation because of their infectious nature, and have been acquired by workers in institutions such as schools and hospitals, where the diseases have occurred in students or patients. It is important to ensure that exposed people are aware of the risks, understand control procedures and can recognise the early symptoms of relevant infectious diseases. Some of the diseases are unusual and may not be recognised by a medical practitioner at initial consultation. To overcome this, some meatworkers carry a card to hand to their doctor indicating that they work at an abattoirs, are exposed to zoonotic diseases and suggesting that certain blood tests be performed if they present with a feverish illness.

For some of the relatively uncommon and specific infectious diseases there may be a lesser degree of under-reporting than for other categories such as occupational respiratory disease. Overall, there is still likely to be a substantial understatement of the true incidence of these occupational diseases since, as in the other disease categories, the Australian Bureau of Statistics data only include cases which have been accepted as work-caused for compensation purposes and which have led to more than 5 lost working-days. Many common infectious diseases such as the common cold may be difficult to attribute specifically to the work environment as there may be numerous opportunities for exposure in the general environment.

Within South Australia most occupational infections will be seen in meatworkers (e.g. brucellosis), animal handlers (e.g. leptospirosis in dairy farmers) health workers (e.g. hepatitis B, scabies) and education workers (e.g. mumps, chicken pox).

There are about eighty reported cases of occupation-related infectious diseases each year in South Australia and there is likely to be some degree of under-reporting. The incidence for any one disease can vary markedly. There were four or five cases of brucellosis per year in SA through the 1970's, 22 cases over a 5 month period in one meatworks in 1979-80 and no cases in 1984 and 1985. Australian cattle herds were free of bovine tuberculosis and brucellosis by 1992 thus effectively eliminating the risk of these infections for meatworkers.

While the data from WorkCover enabled 26 Q fever cases to be identified from July 1988 to June 1991, data from the Communicable Disease Control Unit identified 45 cases for the three year period from January 1989 to December 1991. Not all of these people may have required more than five days off work and it is known that many cases in a 1994 epidemic at a small abattoirs in rural South Australia did not seek workers compensation. Marmion (in press) has identified the cost of screening and vaccination program in an abattoir of 500 workers as equivalent to the cost of seven cases of acute Q fever or less than half the cost of an average case of chronic Q fever. Trialled in South Australia, the use of Q fever vaccine is the only effective means of preventing infection in workers. It has been shown to offer a high level of protection to workers. The Australian Taxation Office does not recognise Q fever vaccination as a deduction in the way it recognises protective equipment: it is being approached to vary this non-recognition.

Weinstein (1991) estimated the risk of Q fever in an unvaccinated meat worker to be approximately 1 per 300 per annum. His study found that "most cases occurred in meat workers (55%), but workers in

the animal transport, farming, dairy and wool industries were also at risk. Importantly, eight cases occurred in known contacts of people in the above categories, indicating that direct contact with the animal reservoir is not always required to contract Q fever".

WorkCover data identified three cases of leptospirosis but Weinstein (1991) identified 26 cases in the five years 1986-1990 with most cases occurring in meat workers (57%) with farmers and stock transporters also at risk. Vaccination of cattle against leptospirosis has been applied to cattle in South Australia. This prevents obvious disease in the cattle but does not affect the carriage of the organisms and their subsequent shedding in the urine so that an occupational risk to workers remains.

South Australia was declared brucella-free on 1 January 1988 (Weinstein, 1991) but two cases of brucellosis were reported in the WorkCover data, one of which was proposed to be a recurrence.

Thirty nine cases of chicken pox (Varicella - Herpes zoster) reported over the three year period from workplace exposures. Like measles, tuberculosis and colds, chicken pox is spread by the airborne or respiratory route and prevention is aimed at guarding coughs and sneezes and strict hand hygiene.

Twenty one cases of scabies were reported. As the infectivitiy of scabies will cease within 24 hours of appropriate treatment it is an unclear why periods in excess of five days time off work were required.

Two cases of whooping cough were identified. Until the advent of an IgM diagnostic test in 1988 whooping cough (pertussis) was considered rare in adults. The identification of these cases is a reflection of ascertainment rather than the identification of new disease patterns.

Sixteen cases of orf, a transient viral ulcerating condition, were identified amongst people shearing or handling fleeces.

No cases of HIV infection requiring more than 5 days off work were identified from the data although one instance of infection is understood to have occurred.

One case of "viral hepatitis" was identified but the occupational mechanism of infection wasn't provided.

The concept of Standard Precautions (previously called universal precautions) has been an important concept in South Australian health care to minimise the risk of disease by routes apart from respiratory. All bodily fluids are treated as potentially infectious and all non contact skin and mucous membranes are acknowledged as potential sites of entry for infection. Skin is seen as a mode of transmission of organisms from one site to another. In a general sense, handwashing is the single most important means of preventing the spread of infection and is most important with the agents responsible for diseases such as hepatitis A, brucellosis and diarrhoeal diseases. Handwashing is effective not so much in killing infectious agents as by diluting them and flushing them from the skin. Plain soap is usually just as good as *sophisticated* antiseptics and less likely to cause dermatitis or allergic reactions. Importantly, there must be adequate provision of facilities to enable the practise of adequate hygiene.

Blood borne diseases such as hepatitis B, hepatitis C and human immunodeficiency virus (HIV) can be prevented by maintaining intact skin (eg preventing needle stick injuries) and maintaining physical barriers.

Vaccine is available to protect against hepatitis B. It has been used extensively in the workforce in South Australia. While immunisation has been shown to be very effective for hepatitis B, better infection control may have had more effect on rates of infection than the vaccine.

At-risk industries must develop adequate Infection Control Policies encompassing: staff training; methods of implementing, monitoring, evaluating and improving procedures; effective management of cases of infection; identification and action on risks; and the development of a safe environment eg. by the use of disposable materials, reducing the load of organisms in the environment with good housekeeping and using appropriate forms of disposal.

Industries

Incidence varies between 0 (communications) and 5.5 (agriculture, forestry, fishing and hunting) per 10,000 person years. Rates for industries above the mean for males (1.8) were: agriculture, forestry, fishing and hunting (5.5), community services (4.4) and manufacturing (2.5). For males the rates varied between 0 (communications, finance, property and business services) and 7.4 (agriculture, forestry, fishing and hunting). Industries above the mean for males of 1.7 were agriculture, forestry, fishing and hunting (7.4), manufacturing (3.2) and community services (2.7). For females the rates varied between 0 and 5.1 (community services which was the only industry above the mean of 2.0).

Occupations

Incidence varies between 0.06 (managers and administrators) to 4.5 (paraprofessionals). Occupations above the mean of 1.8 are paraprofessionals (4.5), labourers and related workers (2.9), tradespersons (2.8) and professionals (2.6). For males the rates varied between 0 (clerks) and 3.2 (tradespersons). Occupations above the mean rate of 1.7 were tradespersons (3.2), paraprofessionals (2.5) and labourers and related workers (3.1). For females the rates varied between 0 (several occupations) and 6.2 (paraprofessionals). Occupations above the mean rate of 2.0 were paraprofessionals (6.2), professionals (4.1) and labourers and related workers (2.7).

The occupation category of 'tradespersons' includes meatworkers; 'para-professionals' includes registered nurses (enrolled nurses are classed as 'tradespersons); and 'professionals' includes teachers. When reviewing individual case studies, these classifications help to account for the data. Similarly, interpretation of data is assisted by the understanding of the inclusion of the meatworking industry under 'manufacturing' and health workers and teachers under 'community services'.

Compared to the data in "The Human Cost Of Work" (Gun and Langley, 1987) which covered the years 1982-1985, the rate of claims increased by almost 50% in the period 1988-1991 from about 54 to about 80 reported cases each year (the average workforce remained about the same).

Table 7.1. Infectious & parasitic diseases by industry

Industry	*Total Cases (3years)*	*Workforce Total*	*Rate per 10,000 person years Males & Females*	*Rate per 10,000 person year Males*	*Rate per 10,000 person years Females*
Agriculture, forestry, fishing & hunting	7	15,188	5.5	7.4	0.7
Mining	1	3,523	0.9	1.0	0
Manufacturing	19	79,244	2.5	3.2	0.5
Electricity, gas & water	0	6,916	0.4	0.5	0
Construction	0	20,262	0.5	0.6	0
Wholesale & retail trade	1	93,420	0.2	0.3	0.07
Transport & storage	1	14,949	0.4	0.6	0
Communications	0	376	0	0	0
Finance, property & business services	0	47,522	0.06	0	0.1
Public administration & defence	1	16,545	0.4	0.3	0.5
Community services	39	107,182	4.4	2.7	5.1
Recreation, personal & other services	1	33,628	0.2	0.2	0.1
Total	242	438,755	1.8	1.7	2.0

Table 7.2. Rates of infectious and parasitic diseases. 1988-1991.

Exempt and non-exempt employers

Occupation	*Total Cases (3 years)*	*Workforce Total*	*Rate per 10,000 person years Males & Females*	*Rate per 10,000 person years Males*	*Rate per 10,000 person years Females*
Managers & administrators	1	46,529	0.06	0.09	0
Professionals	45	58,650	2.6	1.3	4.1
Para-professionals	47	34,707	4.5	2.5	6.2
Tradespersons	53	62,238	2.8	3.2	0
Clerks	8	67,673	0.4	0	0.5
Salespersons & personal service workers	25	72,461	1.1	0.5	1.5
Plant & machine operators & drivers	4	34,477	0.4	0.5	0
Labourers & related workers	59	67,644	2.9	3.1	2.7
Total	242	444,379	1.8	1.7	2.0

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8. Psychological disorders

R T GUN

Definition of stress - consequences of stress - suicide & occupation - compensation claims - stress claims in the public sector - stress & heart disease - stress & cancer.

Summary

Psychological stressors are any factors in the work environment which lead the subject to experience arousal and aversion. The sequelae of these reactions can be impaired productivity or safety, mental disorders eg anxiety state, unsafe behaviours eg excess alcohol intake, suicide, and possible organic disease.

Analysis of suicide rates by occupation showed the highest rates in unskilled labourers, and other blue collar workers. Individual occupations with high suicide rates include artists and related professionals, male and female nurses, farmers and agricultural workers. Contrary to expectations, the suicide rate was below average for male professional health practitioners, which includes medical practitioners. For the equivalent category in females the rate was above the average for females, but the estimate was based on very few cases.

In contrast with suicides, claims for work-caused stress problems are relatively infrequent in blue collar workers, the highest rates in both sexes occurring in professionals and paraprofessionals.

Since the introduction of WorkCover, the number of claims for work-related stress in South Australia has trebled. The claims are mostly in the public sector, with the rate in State Government employees about 3 times that of the overall workforce. Male occupations with high claim numbers include teachers and education managers, police, prison officers, welfare paraprofessionals and bus and train drivers, and in females teachers and education managers, welfare paraprofessionals, registered nurses and prison officers. Commonwealth Government employees have a claims rate about intermediate between that of the overall workforce and State Government employees.

Some evidence suggests an that psychosocial stressors can affect several conventional heart disease risk factors, eg blood pressure, smoking habits, cholesterol levels, catecholamine levels (causing coronary artery spasm, arrhythmias, and platelet clumping).

There is also some epidemiological evidence of an association between occupational stress and cancer.

Introduction

This chapter deals with the consequences of occupational stress.

An operational definition of stress is required. In conventional engineering terms, stress refers to external forces acting on a body or thing. The response of the body or thing to stress is referred to as strain.

Similarly, in biological terms, stress generally refers to the external forces which act upon and require a readjustment, or coping mechanism, of the body on all or some levels (physical, psychological, behavioural, social), and in the following discussion stress will be used in this sense.

Psychological stressors, which are the subject of this chapter, are any factors in the work environment which lead the subject to experience arousal and aversion. Although they may include physical factors

(e.g. heat and cold), this discussion principally concerns factors whose impact is mainly psychological, e.g. factors such as fear of dismissal, fear of failure, fear of injury, excessive workload, strained management-staff relationships or lack of job autonomy.

The *perception of stress* has a number of concomitants - psychological, physiological and behavioural.

Such responses may be beneficial. The perception of stress may be accompanied by a motivation to behaviour which will counter or help to cope with the stress, and by physiological responses which facilitate the coping behaviour.

On the other hand, the perception of stress may lead to adverse effects:

- the perception of stress itself causes unwanted psychological responses of arousal and aversion
- there may be an accompanying impairment of motor performance; in the work environment the consequence of this may be falling productivity or unsafe behaviour.
- failure to cope can lead to neurotic or psychotic disorders, such as anxiety or depression.
- some behaviours may occur which are damaging to health, e.g. smoking, excess alcohol intake, inadequate food intake.
- stress may cause or contribute to diseases, such as coronary artery disease, peptic ulcer or asthma and possibly cancer.

The first of these responses, ie arousal and aversion, is intermediate between the external stressors and the other responses, such as anxiety state, drug use or heart disease. Thus it is not the actual stressor which cause the stress-related effect but the individual's response to it. Particular stimuli evoke arousal and aversion in some individuals but not in others. For a disorder to be defined as stress-related, the stress factor in the environment must be perceived as stressful by the subject.

Psychological consequences of stress

There is a spectrum of psychological consequences of occupational stress. They range from mere awareness or perception of the stressors, to discomfort, through a range of symptoms such as insomnia, anxiety, depressions and anorexia, to suicide.

It is very difficult to quantify the effect of neurotic disorders from occupation, because the diagnosis rests entirely on interviewing the person affected. Interview of such an employee or patient is likely to consist of questions relating to stressors in the work environment, e.g. "Do you have sufficient time to complete your work?" or "Do you feel that management puts you under pressure?"; and questions on the *response* to such job stressors may ask "Do you experience anxiety or insomnia?". While positive responses to these questions might lead to a conclusion that job stress causes the anxiety state, such a response pattern might be given a reverse interpretation, i.e. the existence of an anxiety state leads the person to perceive that there is insufficient time to complete the day's work, or that there is undue pressure from management. In other words it is a matter of interpretation whether the stressful job situation gives rise to anxiety state or whether a predisposition to anxiety leads the individual to perceive the job as stressful.

Quantification would be easier if there were more tangible effects of occupational stress than simply asking the individual if he/she finds work stressful. Alcoholism is a possible measurable outcome of stress, but in fact it would be very difficult to evaluate its relationship to stress, especially occupational stress, since alcohol intake is known to correlate strongly with other occupational factors such as employment in hotels and breweries, which may be quite unrelated to stress.

Suicide is no doubt a tangible outcome with an unequivocal nexus to stress. Study of the relationship between suicide and occupation/industry might therefore be expected to give an index of the degree of

stress in various occupations, although such a manifestation would only affect a minority of people at the extremity of the response spectrum.

Unfortunately the national death statistics failed to include occupational data until 1993. Thus only data for that one year are available for analysis of suicide by occupation. However we have also obtained mortality data for South Australia for the period 1990-93, and this has also been analysed for suicides according to occupation. The rates were calculated using for denominators the numbers employed in different occupational categories in the 1991 Census. The data are not strictly comparable: the Census data are based on the respondent's job at the time of interview, whereas occupational category in the death record is based on the longest-held occupation in the life of the deceased.

The analyses of suicides by occupation for Australia for 1993 and SA 1990-93 are shown in Table 8.1. In 1993 there were 2083 suicides in Australia, of whom 1689 were males and 394 were females. Occupational information was available for 1194 males and 158 females. In South Australia 1990-93 there were 685 suicides, and occupational information was unavailable in 229. Of the 456 cases included in the analyses, 387 were males and 69 were females. The estimates of most industry- and occupation-specific rates in females are unstable because of the low number of suicides.

Table 8.1. *Suicide rates by occupation, Australia, 1993, and South Australia 1990-93. Rates expressed as suicides/100,000 person-years.*

	Males		*Females*	
	Australia 1993	*SA 1990-93*	*Australia 1993*	*SA 1990-93*
Managers & administrators				
Managers & administrators nfd	22.1	36.9*	6.9*	0
General managers	20.1	6.3*	0	42.4*
Specialist managers	13.2	10.2*	7.8*	0
Farmers & farm managers	47.7	19.0	6.1*	7.0*
Managing supervisors sales & service	29.4	35.4	4.8*	10.2*
Managing supervisors, other	37.5	25.9*	0	0
Total managers and administrators	27.8	20.3	5.8	6.2*
Professionals				
Professionals n.f.d.	0	55.8*	0	0
Natural scientists	24.1	13.7*	11.8*	0
Building professionals	38.4	4.6*	26.2*	91.2*
Health practitioners	24.2	33.3*	15.9*	7.7*
School teachers	20.2	16.2*	3.9*	2.0*
Other teachers & instructors	11.7	12.8*	4.9*	0
Social professionals	38.8	42.2*	5.0*	11.9*
Business professionals	13.9	2.6*	5.7*	11.0*
Artists & related professionals	69.2	76.6*	21.9*	16.8*
Miscellaneous professionals	26.4*	0	6.8*	24.4*
Total professionals	25.8	19.0	7.1	7.3*
Para-Professionals				
Para-professionals n.f.d.	73.9*	19.8*	0	120.2*
Medical & science tech. officers & tech	6.8*	19.4*	12.2*	0
Engineering & building associates & tech	7.9*	3.8*	16.0*	0
Air & sea transport technical workers	46.7*	0	152.7*	0
Registered nurses	55.9*	42.2*	16.3	26.5
Police	9.4*	8.7*	20.3*	0
Miscellaneous para-professionals	20.5	26.8*	1.6*	0

Total para-professionals	19.9	16.5	12.3	17.9
Tradespersons				
Tradespersons n.f.d.	29.3*	20.0*	0	0
Metal fitting & mach tradespersons	31.6	31.9	0	0
Other metal tradespersons	49.7	75	0	0
Electrical & electronic tradespersons	28.1	28.2	0	105.9
Building tradespersons	40.2	43.1	0	0
Printing tradespersons	34.5*	0	0	0
Vehicle tradespersons	37.3	34.0	0	0
Food tradespersons	59.1	35.8*	9.5*	0
Amenity horticultural tradespersons	36.0	21.3*	0	51.1
Miscellaneous tradespersons	47.2	77.6	5.7*	14.7*
Marine construction tradespersons	0	0	0	16,666*
Total tradespersons	39.4	41.8	5.6*	14.8*
Clerks				
Clerks n.f.d.	35.5	44.5*	9.9	35.2
Stenographers & typists	0	0	1.8*	1.6*
Data processing & business mach.ops	12.0*	20.4*	3.6*	0
Numerical clerks	4.6*	0	3.7*	0
Filing, sorting & copying clerks	7.8*	0	4.2*	0
Material recording & despatch clerks	3.7*	10.3*	4.1*	0
Receptionists, telephs & messengers	10.8*	0	4.9*	7.6*
Miscellaneous clerks	4.2*	0	5.8*	5.7*
Total clerks	11.5	12.2	4.4	7.0
Salespersons & personal service workers				
Salespersons & personal service wrkrs	79.3	91.2*	38.3*	3.1*
Investment, insurance & real estate	19.1	18.0*	10.6*	0
Sales representatives	19.6	15.0*	7.6*	0
Sales assistants	13.9	11.6*	2.8*	5.9*
Tellers, cashiers & ticket salespersons	16.8*	0	.96*	0
Miscellaneous salespersons	22.6	39.3*	7.4*	0
Personal service workers	46.1*	31.6*	7.4*	3.9*
Total salespersons & pers srvc wrkrs	21.0	20.3	4.8	3.7*
Plant & machine operators & drivers				
Plant & machine ops & drivers nfd	123.7	162.3*	220.3*	0
Road & rail transport drivers	38.9	43.1	7.1*	0
Mobile plant ops(except transport)	20.0	21.5*	0	0
Stationary plant operators	40.7	26.4*	0	0
Machine operators	17.2	24.8*	0	0
Total plant & machine ops& drivers	33.5	37.2	3.9*	0
Labourers & related workers				
Labourers & related workers n.f.d.	230.7	393.6	6.1*	65.2*
Trades assistants & factory hands	34.0	20.6*	1.6*	0
Agricultural labourers & related wrkrs	54.6	64.2	5.1*	13.2*
Cleaners	17.9	31.7*	4.6*	2.2*
Construction & mining labourers	20.4	24.9*	0	0
Miscellaneous labs & related work	29.9	33.5	4.6*	6.3*
Total labourers & related workers	47.8	55.9	4.1	5.9*

** indicates that the rate estimate is based on fewer than 10 cases and is therefore unstable.*

The *national suicide rate* in males for all occupations combined (ie excluding people not in the labour force) was 31.4/100,000 person-years. Of the major occupational categories, all 3 blue-collar categories were over the average and all the white-collar categories were under it. The highest rate was in labourers, whose suicide rate was more than 50% above the average. Within occupational categories, the highest rates were in workers about which there was little detail eg "labourers & related workers nfd" and "plant and machine operators & drivers nfd" had by far the highest rates. This may reflect difficulty in obtaining detailed occupational data on suicide victims.

Amongst occupational subcategories, (male) registered nurses had a high suicide rate - about 3 times the rate for other paraprofessionals; however the estimate was based on a small number of cases. Amongst professional workers, by far the highest rate was in artists and related professionals. The rate in health practitioners, which would include doctors, was somewhat lower than the average for professionals, a surprising finding given previous findings that doctors have a high suicide rate. Farm work appears to carry a high suicide risk; agricultural workers had a higher rate than other labourers, and farmers and farm managers had a higher rate than other managers. Amongst tradesmen, food tradesmen had a high rate of suicide.

The *State-based data* for males showed a very similar pattern for major occupational categories. Health practitioners showed a somewhat higher rate than for other professionals, but the estimate was based on small numbers. Farmers did not have a high suicide rate in these statistics, but agricultural workers had a higher rate than most other labourers, as in the national data. Again, the rate for nurses was high.

Fewer than one-half of female suicides occurred in female members of the labour force. The suicide rate was only one sixth of the male workforce, and accordingly the rates for occupational categories and subcategories are based on small numbers and therefore subject to considerable error. However, a high suicide rate for artists and related professionals is apparent. Registered nurses also had a high rate.

Compensable mental disorders

Another index of occupational stress is the rate of successful claims for work-related mental disorders. The number of such claims in successive years in South Australia since 1980-81 is shown in Table 8.2.

Table 8.2. Numbers of successful claims for mental disorders, 1980-81 to 1990-91, South Australia.

Year	*Males*	*Females*
1980-81	16	11
1981-82	17	12
1982-83	31	28
1983-84	68	33
1984-85	54	32
1985-86	96	85
1986-87	108	63
1987-88	na	na
1988-89	273	261
1989-90	405	342
1990-91	439	391

The figures for 1988-89 and thereafter represent the period after the introduction of WorkCover. Part of the explanation for the large increase in claims from 1988-89 may lie in the wording of the

WorkCover legislation, which was interpreted (by the SA Supreme Court) to allow a claim on the basis of even a perception that the workplace or the work was stressful (and provided, of course, that a doctor certified that the worker was thereby unfit to work). The criteria for acceptance of stress-related claims were made more stringent in 1992.

An analysis of successful claims for the period 1988-89 to 1990-91 by occupational category is given in Table 8.3.

Table 8.3. *Rates of mental disorders (number of successful claims/10000 person-years) by occupational category by gender, 1988-89 to 1990-91.*

Occupation	*Males*	*Females*
Managers & administrators	9.7	10.1
Professionals	27.5	43.1
Para-professionals	54.3	31.8
Tradespersons	5.1	5.0
Clerks	23.7	13.7
Salespersons & personal service workers	6.9	7.7
Plant & machine operators & drivers	15.1	16.6
Labourers & related workers	9.8	8.0
Total	15.3	16.4

For major occupational categories the claims rates for males in the blue-collar categories are all below the average for all occupations, whereas all white-collar categories except managers and administrators are above the overall average, ie the rate of stress claims is inversely related to the risk of suicide. In females that claims rate showed a similar occupational profile as in males.

WorkCover data were also scanned for individual occupations with high numbers of stress-related claims. The highest numbers in male workers were in the following occupations:

teachers & education managers	175
police	49
prison officers	42
welfare paraprofessionals	30
bus and train drivers	67

The highest numbers in females were:

teachers & education managers	266
welfare paraprofessionals	53
registered nurses	51
prison officers	35

Analyses of claims rates for males and females according to industry category are shown in Table 8.4.

Table 8.4. *Rates of mental disorders (number of successful claims/10000 person-years) by industry category by gender, 1988-89 to 1990-91.*

Industry	*Males*	*Females*
Agriculture, forestry, fishing & hunting	0.3	0.7
Mining	14.4	11.1
Manufacturing		
Food, beverages & tobacco	4.5	5.8
Textiles, clothing & footwear	9.0	7.3
Wood, wood products & furniture	3.5	14.0
Paper, paper products, printing & publishing	7.4	8.7
Chemicals, petroleum & coal products	3.6	0
Non-metallic mineral products	5.9	12.5
Basic metal products	3.6	9.3
Fabricated metal products	2.8	12.7
Transport equipment	4.5	12.0
Other machinery & equipment	5.3	9.4
Miscellaneous manufacturing	7.4	8.5
Total manufacturing	4.7	8.3
Electricity, gas and water	15.0	4.2
Construction	11.4	10.0
Wholesale & retail trade	3.5	4.8
Transport & storage		
Road transport	59.7	39.8
Air transport	17.2	0
Services to transport	15.1	5.0
Storage	15.6	14.0
Total transport & storage	35.3	16.8
Finance, property & business services	9.0	9.9
Public administration & defence	14.3	23.0
Community services		
Health	34.9	16.2
Education, museum & library services	57.8	44.9
Welfare & religious institutions	48.7	42.9
Other community services	100.8	58.4
Total Community Services	58.7	30.7
Recreation, personal & other services	7.2	5.3
Total	15.3	16.1

Claims rates in the public sector

Data on stress-related claims for the State Government workforce were obtained from the Government Workers' Rehabilitation and Compensation Office. This excludes employees of the SA Health Commission, ETSA and the State Transport Authority (TransAdelaide). Data on the employees of the SA Health Commission and local government were obtained from WorkCover, and data on Commonwealth Government employees in South Australia were obtained from Comcare. Comparative figures for these categories, with corresponding data for the overall SA workforce, are shown in Table 8.5.

Table 8.5. *Rates of mental disorders (number of successful claims/10000 person-years), government and Statewide, 1988-89 to 1990-91.*

Sector	*Males*	*Females*
Overall SA workforce[1]	15.3	16.4
State Government [2]	47.7	52.8
SA Health Commission	54.6	25.8
Local government	18.4	12.3
Commonwealth Government[3]	27.1	39.4

1 Covers all categories, including private sector, but excludes Commonwealth employees

2 excludes SA Health Commission, ETSA and STA

3 excludes Australia Post and Telecom

Clearly the public sector is incurring most of the stress claims. The claims rate for stress-related problems for employees of the State Government is about 3 times the rate for the overall State workforce. Commonwealth Government employees have a claims rate about intermediate between that of the overall workforce and State Government employees.

The contribution of occupational stress to disease

Because exposure to stress is difficult to measure, and because of the methodological difficulties in relating health outcomes to psychological stressors, it is not possible to estimate the impact of occupational stressors on the occurrence of organic disease. Following the work of Selye on "adaptation", it was considered that prolonged stress could lead to failure of the adaptive response to stress, leading to empirical studies based on the hypothesis that prolonged stress could reduce the resistance or immunity to disease. However the link between stress and disease risk is not proven. Indeed a recent study on Vietnam veterans with post-traumatic stress disorder were found to have *enhanced* cell-mediated immunity compared with veterans without post-traumatic stress disorder. [1]

Cardiovascular disease (especially coronary artery disease) and cancer are disease categories which have received attention as being possibly stress-related. Since these diseases account for over one-half of the deaths in the population, any stress-related factors which might contribute to their incidence, even if small in proportion, could be contributing to a substantial absolute number of deaths. More importantly, if occupational stressors were controllable, there would be a significant potential for public health benefit through intervention in the workplace.

Coronary artery disease (CAD)

The most important risk factors in the causation of CAD, smoking, hypertension and raised serum cholesterol, do not fully explain the variations in the occurrence of CAD within and between populations. Because of the high incidence and mortality from CAD, especially in Western countries, there has been intensive investigation into two possible roles of stress: in the development of CAD itself, ie of the degenerative arterial disease (atherosclerosis) which leads to heart attacks, and in the incidence of heart attacks in individuals with underlying CAD. (Of course stress is not the only possible CAD risk factor under study. For example, a substantial amount of epidemiological and experimental evidence has accumulated in recent years on childhood influences in the later development of CAD.)

Typically the highest incidence of heart disease occurs in the lowest occupational categories (ie manual unskilled workers), and the excess incidence in these workers is not fully explicable on the basis of their higher smoking prevalence. It is of considerable importance to identify any job-related

factors which might account for this excess. In a prospective study, Karasek and associates identified a combination of excess job demand and lack of job-decision latitude as occupational risk factors which increase the risk of heart disease.[2] These findings have been widely used not only to infer a link between these job attributes and not only heart disease, but to use them as an indices of job stress and its psychological consequences. Although a more recent prospective study (also co-authored by Karasek) came to contrary conclusions to the earlier study,[3] a recent review has concluded that there is a body of evidence that strongly suggests a causal association between job strain and CAD.* However the authors point out that further aspects of the job environment such as job insecurity, need to be tested for their possible role in causation of CAD.[4]

In 1988 a working party of the National Heart Foundation reported on stress and cardiovascular disease.[5] Its conclusion was that there is little evidence for concluding in individual cases that the continuing chronic excess of any particular job may have contributed directly to heart disease. The working party considered that the question was more open on the relationship between acute life stresses and heart ischaemic heart disease. It was also concluded that there is good evidence that stress and its psychological consequences do affect several conventional risk factors, eg on blood pressure, smoking habits, cholesterol levels, catecholamine levels (causing coronary artery spasm, arrhythmias, and platelet clumping).

Whilst the evidence of a link between stress and CAD is not proven, it is still far from disproven. It is of interest that the eminent psychologist Hans Eysenck has concluded from review of data from the Framingham study and other epidemiological studies that stress not only is six times as predictive of CAD as smoking, cholesterol level and blood pressure and important contributor than cigarette smoking to the incidence of CAD, but is much more accessible to prevention.[6]

Cancer

It is not only heart disease which Eysenck associates with psychosocial stress. He, and others, have contended that a link exists between stress and cancers.[6,9]

The Melbourne colorectal study, a large case-control study, of possible risk factors for bowel cancer, found a 2.5-fold increase in risk associated with work-related problems in the 5-year period prior to diagnosis.[7] More recently a Swedish study came to similar conclusions in relation to stressful event in the preceding 10 years. The stressful associations and the relative risks included:

death of a spouse	1.5 (statistically significant)
death of a child	0.6
death of a dear friend or relative	0.9
divorce	1.5
unemployment > months	2.2
serious occupational problems	5.3 (statistically significant)
serious financial problems	0.5
change of residence . 200km	3.2 (statistically significant)

The nature of the serious occupational problems was not made clear.[8]

The authors hypothesise that possible mechanisms may be behavioural, ie the stress leads to excess alcohol use, or immunological.

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* *It should be noted that one of the authors of the review was also a co-author of Karasek in the publication proposing the thesis under review.*

In commenting on the Swedish study, Kune, a co-investigator of the Melbourne study, emphasised that it is not the stressful event themselves but the experience of extreme upset which causes the response. Events "are just what they are" and it is their effects, measured by how we think about them, that are important. [9]

Some studies have linked stress with cancers of the lung, breast and stomach, although other studies have failed to demonstrate any such link.[9]

Discussion

The data on stress-related claims and suicides add up to the following: there has been (literally) an exponential increase in the number of stress claims; the incidence of these claims has been mostly in the public sector; and the occurrence of stress-related claims between broad occupational categories is inversely related to the occurrence of suicide.

Drawing conclusions on these findings is difficult because it is not clear what the statistics are actually measuring. The stress-related data are based on claims which are in turn based on a medical assessment, but the doctor has no tangible means of identifying psychological disorders, since, unlike diseases such as measles, melanoma or malaria, there is no disease *process* to be identified. Under current (1995) criteria for stress claims, certifying doctors are required to identify a mental disorder listed in the DSM-3 (the manual accepted as the standard for psychiatric diagnoses), but for the 3-year period 1988-89 to 1990-91 the claims data represent virtually all workers who chose to claim for even a perception of work-related stress. Clearly then at least some of the increase in the number of claims has been related to the relatively loose criteria which applied up to 1992.

However this fails to explain the increase which occurred before 1988 or the preponderance of claims in the public sector. Whilst it is tempting to blame poor management, it is difficult to understand how this situation should be so different from the early 1980s and before then, when stress claims were infrequent. A common view is that work stresses have increased in recent years as a result of "flatter" management structures and "downsizing", producing fewer employees with heavier responsibilities, and higher stress levels.[10] Yet it is unlikely that these changes have been greater in the public sector.

A possible explanation is that work in the public sector is more stressful. Whilst it is intuitively reasonable to accept that dealing with schoolchildren and social security and Commonwealth Employment Service clients is stressful, it would be difficult to prove or disprove that these are more stressful than private sector jobs. The suicide data in fact strongly suggest that the greatest stresses are borne by unskilled manual workers, an occupational group which is under-represented in the public sector and in the compensation statistics for stress-related claims.

A provisional explanation for these findings is (i) all work is stressful sometimes, (ii) downsizing and changing management practices may have increased occupational stresses in recent years, (iii) increased awareness of health and safety issues have lead to an increasing perception in the workforce that it is the responsibility of management to prevent occupational stresses and to accept the financial consequences if it should fail to do so, (iv) claims are relatively infrequent in the private sector because individuals who cannot cope with work pressures are either inhibited from making claims because of job insecurity or are forced out of the organisation. This is not to imply that some public sector employees are not making unjustified claims, but it is almost impossible to identify which ones. As discussed earlier, it is not the job stressor which causes the problem, but the individual's arousal-aversion response to it. The occurrence of stress-related problems depends on whether the individual can or cannot cope, and there is no objective measure by which the individual's perception of being able or unable to cope can be verified.

The occupational distribution of suicides does not necessarily reflect the influence of work. For example the high suicide rate in labourers may partly reflect a selection effect: people with severe psychiatric illness tend to gravitate to low-status jobs - if they can obtain work at all.

Indeed it is probable that employment factors are relatively unimportant in the causation of suicide. A study of all suicide cases for one year in the SA Coroner's office indicated that problems with personal relationships (eg marriage breakdown) and psychiatric illness are overwhelmingly the most important risk factors. Nevertheless, the data analysed here suggest an increased suicide risk in occupations which the worker cannot leave behind when going home, such as artists and related professionals and farmers. A high suicide rate for actors has also been reported elsewhere.[11]

From the viewpoint of prevention, it is important that continued attention be given to the possible role of occupational stressors in the occurrence of serious disease such as CAD and cancer. Given that psychological stressors can affect CAD risk factors, and that work contributes to the total burden of stress experienced by all individuals, there is potential for future research in this area to play a significant role in disease prevention.

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9. Adverse reproductive outcomes in relationship to work

SCOTT DUNDAS

Occupational reproductive effects - hazardous agents - infertility - spontaneous abortion - malformations

Summary

Reproductive impairments are common in society with many known and unknown non-occupational risk factors presenting a challenge in relating apparent increases in adverse outcomes to agents in the workplace.

In addition, methodological problems in reproductive research abound, explaining a general lack of consistency in findings.

Despite a general lack of consistency, several agents are regularly implicated. These include solvents, various heavy metals, ionising radiation, and waste anaesthetic gases.

Maternal physical exertion is an often overlooked association but the scientific consensus does not support a causal association with visual display terminals.

Some South Australian data are available but generally they can only point to associations worthy of further study.

Introduction

Reproductive impairments are common in any society. They include infertility, foetal loss (spontaneous abortion), malformations, low birth weight, neonatal death, and some childhood cancers.

Accurate data on normally expected obstetric outcomes are more difficult to obtain than would be expected. Methodological problems abound when studying humans, but in this case changing a classification/definition dramatically changes the measured outcome. For example, how low a birth weight is classified as abnormal; how long a delay in achieving pregnancy is defined as "infertility"?

Similarly, more sophisticated methods of detection alter the pick-up rate. Foetal loss is much more common when sensitive hormonal methods of pregnancy detection are used. Clinically recognised abortions occur in about 15% of pregnancies but with the use of human chorionic gonadotrophin assays, which can detect pregnancies much earlier, a figure of around 40% is shown to be more accurate.

Maternal exposures have been much studied over time but attention is swinging to equal concern for male exposures. A much quoted example is that of dibromo-chloropropane (D.B.C.P.) a nematocide once used in SA. which is associated with a reduction in sperm count increasing in proportion to the number of years of exposure.[1] Lead is a commonly used substance with known adverse male reproductive effects.

It is often overlooked just how radically different germ cell formation is between the sexes. Females are born with their full number of egg cells and, in addition, the first reduction division (where the amount of DNA is halved to allow combination with the male portion of DNA) has already occurred by the time of birth. In contrast, the male has all stages of sperm development present at any one time. This may make male reproduction vulnerable equally throughout time, whereas the egg cells are likely

to be more vulnerable only when actively dividing, as when a few egg cells undergo their second division in each monthly cycle. The full sperm generation cycle takes approximately 70 days.

Problems with research

A great many studies have explored the relationship between reproductive outcomes and various occupational exposures. Generally, there is a lack of consistency in the findings. This is not surprising given the many methodological difficulties.

Background non-occupational risk factors abound. These include age, previous pregnancies, general health, smoking, alcohol, nutrition, and many infections, such as rubella, cytomegalovirus, and toxoplasmosis.

Biases occur. For example, fertile women may tend to leave work early such that the population of women at work may be less fertile than the population at large. The much reported "healthy worker" effect cannot easily be excluded.

It is also likely that women who have suffered a traumatic pregnancy event would be influenced by this and recall more exposures because they are motivated to explain the outcome for peace of mind. This is the much recognised "recall bias" encountered in most retrospective studies. Also, linking effects to a particular exposure is very difficult if there is a "critical window". This describes a period of time, possibly only a few hours long, during which the foetus is vulnerable to a particular developmental outcome.

Agents with a strong influence may be hard to detect if they act very early in pregnancy, as very early miscarriage is often undetected. Because of this, malformation rates may be a poor measure of teratogenicity with a badly damaged foetus aborting so early that the spontaneous abortion appears to be a late, heavy menstrual period.

Background variation is significant; it is difficult to obtain large enough numbers of research subjects; and problems occur with grouping of study outcomes.

If rare outcomes are grouped together for larger numbers to study, information may be lost, but studies of individual rare outcomes lack statistical power.

Studies requiring volunteer response can introduce bias, and studies involving active cooperation do not obtain wide representation (studies of semen quality obtain a 40% response rate). Ambiguity exists; what is a normal sperm count?

Occupation/job title does not adequately describe exposure, it is really a surrogate for exposure.

Birth defects are generally more fully reported by exposed women because they suspect a link.

Nonetheless, several exposures are viewed as having strong associations with adverse reproductive outcomes.

Effects of maternal exposure

The monthly probability of pregnancy has been estimated to be 20-25% in healthy couples not practising contraception. Several occupational exposures have been reported to be associated with menstrual irregularities, and to the extent that these may be mediated by hormonal-like mechanisms, this may signal an alteration in fertility. Examples of these occupational exposures include: fluoride (superphosphate manufacture); solvents (e.g. toluene and perchlorethylene); some heavy metals; and shift work.

Solvent exposure (styrene, aliphatic hydrocarbons, toluene, xylene, chloroform) and glycol ethers are reported as an association with spontaneous abortion. So too are certain heavy metals, anaesthetic gases, and anti-neoplastic (anti-cancer) pharmaceuticals. Anti-neoplastic agents damage DNA. leading to a very plausible mechanism of action. Similarly, ionizing radiation is capable of chromosomal

damage, but human evidence of effects at current dosage levels is lacking and not expected to be forthcoming.

Physical exertion is often overlooked. Many studies make this link, and a major Montreal study, involving approximately 56,000 women and 100,000 pregnancies, confirmed this association in all six employment sectors studied.[2]

Organ development in the growing foetus is particularly intense and probably most vulnerable in the first 60 days after conception. The development of the C.N.S., the eyes, and external genitalia extends well beyond this time and this may explain the relatively higher prevalence of congenital abnormalities of these organs.

Approximately 2% of newborn children are found to have a major congenital malformation. Minor defects, and defects evident later add 4-5% to this figure.

Theoretical mechanisms for congenital malformations include: chromosomal damage; altered cell adhesion, membrane permeability, gene expression, energy utilisation, pH, or electrolyte balance; or other uncertain mechanisms.

Chromosomal defects are more prevalent in infants dying in utero (about 0.6%), and more prevalent again in spontaneously aborting foetuses (about 30%).

Malformation rates are about 40% higher for boys than girls mainly because of a higher rate of abnormalities of male external genitalia.

Specific linkages with malformations have been found for solvents, especially aromatic hydrocarbon solvents, with suggestions of increases in cardiac, urinary tract, and oro-facial abnormalities.

Lead, methyl mercury, and ionizing radiation are also considered to be teratogens (agents causing congenital malformations). The Minamata Bay disaster in Japan in which methyl mercury entered the food chain causing horrendous neurological abnormalities in offspring, often when the mother was unaffected, shows the vulnerability of the foetus across the placenta.

Some occupations, not necessarily associated with identified agents, are the focus of intense study with respect to malformations. Examples include agricultural and horticultural workers, nursing staff, and operating theatre personnel.

Data on possible causal factors for low birth weight and premature delivery (not the same thing) are even less consistent. Alcohol, tobacco smoke (perhaps from nicotine or carbon monoxide, the latter with significant occupational overtones), and many health behaviours are clearly important.

Physical exertion and postural factors seem relevant, as do certain manufacturing and service occupations. Lead (often studied), agricultural work, solvent use, and the electronic semi-conductor industry also have positive associations in some studies.

It is plausible that a proportion of childhood cancers are caused by parental occupational exposure via genetic means, or by interference with foetal development as substances exert actions after crossing the placenta.

Definitive evidence that transplacental carcinogenesis occurs exists with the unfortunate story of diethylstilboestrol (a female hormone given to women and which years later caused genital tract cancer in their offspring).

The incidence of childhood cancer has been increasing over 20 years or so; this has been both for boys and girls, and across ethnic groups. This has been most obvious for brain cancer, leukemia, and certain lymphomas.

Hydrocarbon exposure, paints, and agricultural occupation again feature with possible links to childhood cancer. As always, the results of studies are frustratingly inconsistent and therefore open to interpretation.

The issue of visual display terminals and adverse reproductive outcomes is often raised. The balance of scientific opinion favours the view that no plausible causal agent has been identified, and the very powerful reporting and recall biases can explain the positive associations in these studies.

Effects of paternal exposure

Paternal contribution to adverse outcomes could arise from direct sperm DNA effects, from the indirect mechanism of transmissible agents in the seminal fluid, or by maternal exposure to agents imported from the workplace by the father.

An explanation for the excess cases of leukemia and lymphoma in children whose fathers worked at Sellafield nuclear plant[3] could be either a direct DNA effect or transmission of a radionuclide in the seminal fluid, representing the first two mechanisms outlined above.

Can semen analysis assist in resolving the contribution of paternal factors?

The sperm count, sperm motility, and sperm morphology (shape) have value, particularly in investigating infertility. Unfortunately, there is considerable variability between individuals making the interpretation of results more difficult.

Nonetheless, strong correlations have been demonstrated between such tests and the interval to first pregnancy, and total number of living children, in a 20 year follow up of nearly 800 men attending for investigation of possible infertility[4].

Heat (hot tubs, tight underwear, etc) is often implicated in lay discussions. Interestingly, a study of outdoor workers in Texas showed a reduction in sperm counts and motile sperm in summer compared to winter, with those workers having lower values suffering the greater reduction in test results between winter and summer[5].

European lead workers in the 1800s noticed evidence of infertility, spontaneous abortion, stillbirth, and malformations. The exposure may well have been at doses toxic to adults. Recent studies confirm the likelihood of these links.

Ethylene dibromide, used as a fumigant and scavenger in petrol; solvents yet again (particularly in the aircraft industry); and carbon disulphide (little used now), have been implicated in altered sperm counts and increased risk of certain malformations.

The offspring of male anaesthetists appear to have an increased risk of congenital abnormalities and spontaneous abortion, possibly due to exposures to anaesthetic agents[6].

Population based studies provide data for further exploration, but many associations will occur merely by chance. Plausible biological relationships, and causal links have to be sought. One such study using 20 categories of malformation and 58 paternal occupational classifications and representing over 14,000 births, found associations with solvents, wood products, metals, pesticides, and electrical, printing, and textile occupations.[7]

South Australian data

Pregnancy outcome data for SA. is available from publications of the Pregnancy Outcome Unit of the South Australian Health Commission [8].

Data for 1993 are derived from a denominator of nearly 20,000 live births.

These data do not address the previously discussed issue of spontaneous abortion, as the 3.7% threatened miscarriage rate only relates to late pregnancy and actual spontaneous abortion is not recorded.

In 1993, from the nearly 20,000 births, there were 123 stillbirths, 72 neonatal deaths, and a perinatal mortality rate of 4.2 per 1000 when criteria of a birth-weight of more than 1000 grams or a gestation

of more than 28 weeks are used. Congenital abnormalities represented 2.4% with 487 reported cases. 13 sentinel congenital abnormalities involved the central nervous system, 21 the gastro-enterological system, 29 were cleft lip/palate deformities, and 52 cases involved the male external genitalia.

Another source of SA. data uses SA. cancer registry and perinatal statistics data to study links between perinatal characteristics and subsequent childhood cancer.[9]

Tracing of 235 cases of childhood cancer arising in children born in the years 1981-1993 yielded valuable information. When compared with control children, it was found that a threatened miscarriage increased the risk of childhood cancer by approximately twofold.

This was due to the increased risk associated with chromosomal abnormality, mainly Down's syndrome, which elevated the risk 36 times.

These data show that genetic change is linked, in substantial part, with spontaneous abortion, cancer, and malformations.

The observation that young mothers imparted lower risks to their offspring may reflect lower cumulative exposure to DNA damaging mutagens, because of their shorter-to-date lifespan.

The pregnancy and lifestyle study (PALS) studied associations between occupational and environmental exposures and reproductive outcome.[10]

The study was a prospective South Australian study on 585 "normal" couples planning a pregnancy. The volunteer couples completed several staged questionnaires and were invited to undergo various clinical and laboratory investigations. Several emotive questions were added, and the responses compared between couples who had previously suffered an adverse outcome of pregnancy, and those who had not. The answers were similar, and the investigators felt that this diminished the likelihood that certain biases were occurring.

The PALS study found an association between male infertility and exposure to "dusts". As these dusts were various, including metals, woods, inks, paints, concrete, and ceramics it is hard to draw a firm conclusion from these data.

Male trade occupations were associated with an increased risk of spontaneous abortion in their partners.

Little information on female occupation was forthcoming. Only the visiting of industrial worksites where "metal" or "chemical" pollution existed showed an association with spontaneous abortion, but on the basis of only 14 reports, unclear definition of terms, and self assessment of exposure, this is at best suggestive.

Domestic and hobby factors were reported, for example male exposure to glues, oil paints, and oven cleaners; female exposure to home renovation; and trunk X- rays in both sexes.

The PALS study, as do many, raises many interesting questions but chance and non-causal associations have to be excluded.

Addressing deficiencies in reproductive outcome data

- Better delineation of study factors, coupled with more precise data collection, will enable advances in this field.
- Population studies should better define the normal frequency of obstetric events.
- More precise job and industry classifications should be used in research studies, and in hospital and registry information collection. This applies to the occupations of both men and women. Where causal associations are suspected, detailed exposure measurement should follow. Too often, we are forced to rely on the best guess of the individuals concerned.

- Prospective studies on spontaneous abortion, using the most sensitive laboratory methods should be commissioned in selected industries, where detailed occupational hygiene data can be collected at the same time. In this way, perhaps single research questions can be better answered, rather than multiple issues being unsatisfactorily addressed.

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10. Miscellaneous outcomes from chemical exposures

R T GUN

Hospital admissions for the effects of chemical exposures at work - compensable injuries - the occurrence of systemic diseases caused by occupational exposure to chemicals.

Summary

In the period 1988-89 to 1990-91 there were 91 hospital admissions in South Australia for poisoning by chemicals occurring in a farm, mine, quarry of factory. The commonest single causal agent was carbon monoxide. There were several admissions for the effects of organophosphate pesticides and herbicides. Ironically, there were no cases specified as being caused by organochlorine pesticides, which the Commonwealth and State Government ministers of agriculture have chosen to ban.

Over the 3-year period there were 660 compensable injuries leading to more than 5 days of lost time which resulted from the use of chemicals. Slightly more than one-half were chemical burns or burns from the ignition of chemicals, and about 12% were poisonings.

A survey of physicians specialising in haematology, nephrology and neurology indicated that the incidence of non-malignant systemic disease of the blood, kidney or nervous system from occupational use of chemicals is virtually nil.

Introduction

Over recent decades there has been some improvement in the conditions under which many of the recognised dangerous chemicals have been used, so that some of the severe forms of chemical poisoning have become infrequent. Thus for example clinical lead poisoning, mercury poisoning and silicosis are now uncommon due to better exposure controls, and some hazardous substances, such as asbestos and β-naphthylamine, are no longer used because of their severe effects on health. However, this has not allayed concern that the prolonged use of some chemicals may give rise to adverse effects in the long term, although the exposures are not sufficient to cause any immediate effect. For example, although lead exposures are now rarely high enough to cause clinically recognisable lead poisoning, there is evidence that exposure levels now being experienced may, if continued, give rise to effects of insidious origin, such as hypertension, kidney disorders, and adverse reproductive effects.

This concern has been exacerbated by the large number of chemicals introduced into industry in the last 50 years. Unlike pharmaceuticals, many chemicals in industrial use have, until recently, been introduced without adequate assessment of their possible effects on human health. Foremost amongst these concerns has been the possibility that some of these chemicals may cause cancer; another has been the possibility of adverse reproductive outcomes such as still births, spontaneous abortion and congenital birth defects. These matters have been addressed in earlier chapters. This chapter addresses 2 other types of chemical-related problems - effects of acute exposures (eg from accidents, spillages and burns) and some other diseases, viz blood disorders, kidney disorders and neurological disorders.

Accidental poisonings

South Australian hospital separation data for the 3-year period 1988-89 to 1990-91 were scanned for admissions classified "Accidental poisoning by other solid and liquid substances, gases and vapours" (ICD-9 codes E860-869). These cases are categorised according to the place of occurrence, and for the purposes of this analysis, poisonings occurring on a farm, in a mine or quarry, or in industrial premises were selected. There were 375 separations in the 3-year period, 229 males and 146 females. Of these, 101 occurred on a farm, in a mine or quarry, or in industrial premises. The places of occurrence are shown in Table 10.1.

Table 10.1. Hospital separations in South Australia, 1988-89 to 1990-91, by place of occurrence.

	Males	*Females*
Farms	24	1
Mines or quarries	2	0
Industrial premises	62	12
Total	88	13

The data may not accurately reflect the number of occupation-related separations, since some farm poisonings may not be occupational, and the analysis has excluded cases occurring on a street or highway, in public buildings or residential institutions, some of which may have been occupational. Another source of underenumeration is the significant proportion (31%) of cases in the hospital separation data in which the place of occurrence has not been entered in the record.

The chemical agents responsible for the poisonings are shown in Table 10.2. The most common cause of these hospital admissions is carbon monoxide poisoning. There were a number of poisonings from the effects of organophosphate pesticides and herbicides. Ironically, there were no cases specified as being caused by organochlorine pesticides, which the Commonwealth and State Government ministers of agriculture have chosen to ban.

Table 10.2. Occupation-related hospital separations in South Australia, 1988-89 to 1990-91, by chemical agent.

	Males	*Females*
Carbon monoxide	21	0
Exhaust gases	6	2
Other gas or vapour	19	4
Organophosphate pesticides	4	1
Herbicides	6	0
Other pesticides	3	0
Caustic or alkali	7	2
Acids	2	0
Glues	0	2
Petroleum products and solvents	5	1
Metals	3	0
Food/plants	3	0
Lead paint	1	0
Other paint	1	0
Sulphur dioxide	1	0
Other solids or liquids	6	1
Total	88	13

Chemical-related fatalities

South Australian mortality data from the ABS for the years 1989 to 1993 were scanned for chemical-related deaths. There were 23 deaths coded "Accidental poisoning by other solid and liquid substances, gases and vapours" (ICD-9 codes E860-869), 21 males and 2 females. The data set does not indicate which fatalities if any occurred at work, and in most of these cases the data on the occupation of the deceased are inadequately described.

Compensable injury caused by chemicals

WorkCover statistics for the 3-year period showed that there were 395 injuries leading to more than 5 days of lost time in which the causative agent was a basic chemical, and a further 224 from "other chemical products".

The chemical agents responsible are shown in Table 10.3. Slightly more than one-half of these injuries are caused by chemical burns or burns from the ignition of chemicals, and about 12% are poisonings. The remaining 40% are not true chemical effects, but are musculoskeletal injuries occurring in the manual handling of chemicals.

Table 10.3. Injuries caused by chemicals, 1988-89 to 1990-91, by agency of injury.

	Males	*Females*
Basic chemicals		
Industrial gases	68	14
Arsenic and arsenic compounds	2	0
Chlorine	2	0
Lead compounds	5	0
Plastic materials & synthetic resin	177	23
Radioactive material	1	0
Acid	15	4
Other basic chemicals	83	1
Total basic chemicals	353	42
Other chemical products		
Insecticides, fungicides, herbicides	3	0
Paints, varnish, solvents	15	1
Asphalt, tar, bitumen	6	0
Smoke, fire, flame	60	1
Other chemicals	108	30
Total other chemicals	192	32
Total	545	74

Carbon monoxide poisonings

Some additional information was obtained on cases of carbon monoxide poisoning seen at the Royal Adelaide Hospital, where they are treated in the Hyperbaric Unit. A study of 100 consecutive admissions to the RAH for carbon monoxide poisoning has been reported by Professor DF Gorman and associates[1]. Of the 100 consecutive cases admitted between 1986 and 1989, 30 were occupational in origin. These included exposure from vehicles, generators and exhausts, mines, kilns, diving and a laboratory accident. No cases were fatal.

From 1986 to 1993 there were 652 cases of carbon monoxide poisoning admitted to South Australian hospitals. The proportion of these cases which were occupation-related is not known. In 1993 there

were 37 fatal carbon monoxide poisonings reported to the South Australian coroner. All except one were suicides.

Diseases related to chemical exposures

To estimate the incidence of work-related diseases of the blood, the kidney and the nervous system, a questionnaire was sent to physicians practising in the appropriate specialties seeking the numbers of cases of the relevant diseases seen in the previous 3 years. The diseases of interest were those included in the schedule of Sentinel Health Events (Occupational) developed by the US National Institute for Occupational Health and Safety.[2] This schedule includes diseases which may be occupation-related if occurring in workers in certain occupations or in certain industries. The questionnaires sent out are in Appendix 1.

Diseases of the blood & blood-forming organs

The questionnaire addressed the following conditions: aplastic anaemia, methaemoglobinaemia, agranulocytosis or neutropenia, and non-autoimmune haemolytic anaemia. Questionnaires were sent to the 3 major teaching hospitals, and 2 were returned. No cases were reported in association with the occupations or exposures of interest. One respondent commented that careful histories are taken as a rule, but that in fact all of these conditions are extremely rare.

Renal disorders

The questionnaire addressed the occurrence of acute or chronic renal failure. Questionnaires were sent to the renal units of the 3 major teaching hospitals, and responses were obtained from all 3. 2 replies stated that no cases of renal failure from the occupations or exposures cited had been seen in their renal units in the previous 3 years. The third respondent stated that such information is virtually impossible to obtain retrospectively, and suggested a prospective analysis.

Neurological disorders

Questionnaires were sent to 22 neurologists, of whom 15 replied. The questionnaire addressed the following: inflammatory and toxic neuropathy, mononeuritis and mononeuritis multiplex, cerebellar ataxia, and toxic encephalitis. The conditions of principal concern were conditions related to chemical exposures. One respondent had seen 2 council workers with symptoms of neuropathy who had been exposed to a fungicide, although the association with the exposure was not established. No other neurologists reported any neurological disorder of occupational origin. Some respondents said that they would not expect to see any such cases because of the specialist nature of their practice eg paediatric neurology.

Several neurologists had seen cases of carpal tunnel syndrome from cumulative trauma. The incidence of this condition is discussed separately in Chapter 3.

Discussion

These data give an insight on the occurrence of chemical-related morbidity which is quite contrary to the popular conception. For those occurrences described in the WorkCover data set as "accidents", there is a significant component caused by the occupational use of chemicals. In 3 years there were no fewer than 619 chemical-related injuries leading to more than 5 days of lost time. Although many of these cases were really musculoskeletal injuries incurred in the manual handling of chemicals, over 60% were burns and poisonings.

In contrast the evidence of chronic disease from long-term, low-level exposure to chemicals is scant (aside from the questions of cancer, respiratory disease and skin disease, which are discussed in separate chapters). Thus the diseases of occupational origin listed in the schedule of Sentinel Health

Events (Occupational) do not represent any meaningful list of actual problems threatening the workforce in South Australia (nor, presumably, in Australia as a whole). This is not to say that the chemicals identified in the schedule are not capable of causing the respective sentinel health events, but with the current exposure levels prevalent in Australian industry, the occurrence of significant levels of chronic disease is low. Of course it is possible that occupational diseases are being missed by physicians, but given the almost unanimously negative response to the questionnaires, it is clear that there are not a large number of unexplained diseases which merely await identification of an occupational cause; certainly not of diseases of sufficient severity to come to the notice of specialist physicians or requiring admission to hospital.

The hospital separation data show a significant number of admissions from occupational use of chemicals - 101 over a 3-year period. Although the morbid conditions arising from these poisonings is not known, it is likely that they did not result in chronic disease, otherwise they would have been so categorised. Rather these cases were almost certainly acute episodes arising from unanticipated events such as spillages, gas escapes, or ignition of chemicals, rather than the result of continuing low-level exposure. Certainly this would be the case with carbon monoxide poisonings, the most important single chemical exposure in the statistics.

The implications of these findings are of considerable importance for the prevention of chemical morbidity. The Model Regulations and Code of Practice for the Control of Workplace Hazardous Substances adopted by Worksafe Australia are suited more to the control of the chronic effects of chemical exposure. They require identification of hazardous chemicals, assessment of exposure - including quantitative measurement if considered necessary - and control measures to keep exposures below levels likely to result in toxic effects. Such a risk assessment procedure is based on the assumption that the future effects of the chemicals can be predicted from a knowledge of (i) the toxic effects of the chemicals, including dose-response relationships, and (ii) the prevailing exposures. However, many of the most serious effects arise from exposures which are *not* predictable in this way; rather they arise from unanticipated engineering or administrative failure. Moreover, these events are possibly commoner than the occurrence of chemically-caused disease. As shown in the foregoing analyses, apart from the chemically-related dermatoses and respiratory diseases, diseases from the occupational use of chemicals are relatively uncommon. The current regulatory strategy thus requires a greater focus on problems arising from acute exposures. The anticipation of such problems lies not so much in identifying current exposures, but from recognising situations which have led to events such as spillages, fires and acute poisonings in the past.

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11. Deficiencies in data collection

K McCAUL, R T GUN

SA Central Cancer Registry - Inpatient Separation Information System - Workers' compensation data - Sentinel Health Events.

Epidemiology traditionally uses data from two sources. Firstly, there is data which is collected for other purposes, but, because of its population coverage, has immense value in descriptive epidemiology. The second source of data is that which is collected by epidemiologists themselves and which may come from cross-sectional surveys or short studies and is usually collected for a specific purpose.

This report has relied on data from the first source and in this chapter each of the data sources are briefly outlined and problems associated with their use in occupational epidemiology are discussed.

South Australian Central Cancer Registry

The South Australian Central Cancer Registry has been in existence since 1977. Notification by hospitals and pathology laboratories is compulsory and believed to be complete. Regular linkages to mortality data supplied by the Registrar of Births, Deaths and Marriages helps to ensure that information regarding deaths is up to date.

Occupation is not a notifiable aspect of cancer registration and occupation is usually entered onto the registry when an individual's deaths is recorded. The source of this information is therefore the Registrar of Births, Deaths and Marriages. Efforts to improve this aspect of the CCR are unlikely to be successful since the purpose of the registry is primarily to register the diagnosis of cancer.

Improvement in the specification of occupation and in occupational history could, however, be achieved via hospital-based cancer registries which exist within each of the major teaching hospitals in this State. These registries are site-specific and record detailed information on pathological characteristics and treatment. It might be possible to augment those registries which deal with sites for which there is a strong occupational component in their aetiology, with detailed occupational information.

Inpatient Separation Information System (ISIS)

Inpatient Separation Information System (ISIS) is operated by the South Australian Health Commission and contains information on all separations from South Australian hospitals. The Commission holds such data back to 1984, although private hospitals were not fully incorporated into the system until 1988.

This is the only source of morbidity data in the State which is collected at a population level. There are, however, a number of problems with this data which lead to bias and confounding.

The data describes hospital admissions thus conditions which are not severe enough to require inpatient treatment in a hospital are not represented (for example, hearing loss). Outpatient treatment is not recorded, therefore conditions such as asthma, for example, where treatment will generally be given in outpatient departments, will go unrecorded. Since only the more severe asthmatics are admitted, the data in ISIS on asthma will be quite biased and underestimate the population level of this condition.

While occupation is recorded on admission, this is only a self-report and no effort is made to collect a more detailed occupational history. In addition, people over the age of 65 years will often give their occupation as 'pensioner' which for the purposes of the chartbook is non-informative.

Taking a more detailed occupational history would not necessarily involve asking many occupation-related questions. Even asking 'what is the longest held occupation' would provide more information than is currently available.

While the principal condition for admission is presumably recorded accurately, the other presenting conditions which include underlying conditions and other conditions unrelated to admission may not. Since the history is taken on admission by an intern, it cannot be assumed that the same degree of inquiry is applied in all admissions.

Workers' compensation data

Compensation statistics have been used for monitoring occupational injury trends mainly because they are the only statistics available for most occupational injuries and diseases. However they are not a true reflection of the extent of occupational morbidity. There are many other factors other than the occurrence of disease or injury which drive a worker's decision whether or not to claim compensation - for example, whether the worker is in a type of job where continued work is possible in spite of injury or impairment. Another issue is the disinclination of many workers to claim compensation to which they are legally entitled. A feeling of job insecurity may inhibit workers from claiming compensation; this is suggested by the relatively higher rates of many types of injury claims in public sector employees, in whom the feeling of job insecurity is probably less than in the private sector. Survey data from Connecticut tend to confirm the impression that workers compensation claims understate the incidence of occupational disease and injury. Data collected in that State in 1992, as part of the Bureau of Labor Statistics annual industry survey, showed much higher rates of musculoskeletal disorders, for example, than workers' compensation data for the same year. Thus to obtain a better indication of the true incidence of occupational morbidity, it would be desirable to survey the incidence directly.

The Worksafe Australia National dataset has added a further dimension to the problem. Data are being compiled on a national basis by Worksafe Australia, using a standardised data classification system, the National Data Set. This national uniformity has been bought at the cost of loss of some information, the most serious problem being the abandonment of the ICD system for classifying disease or injury. Thus, for example, many forms of respiratory disease will now be grouped under the unhelpful rubric "other diseases of the respiratory system", and similar problems will occur in the case of musculoskeletal disorders. As a result, it will not be possible to compare all future compensation statistics with those in this volume and those of the 1980s, which appeared in *The Human Cost of Work*. It is probably impractical to expect WorkCover to maintain two sets of statistics, one with ICD-9 and one with the Worksafe Australia data set. The only remedy would be for Worksafe Australia to revert to the ICD-9 or its successor, the ICD-10.

Some other specific concerns became evident during our examination of worker's compensation statistics.

- A number of self-insuring agencies, eg: the Government Workers and Rehabilitation Office, the Local Government Association and Telecom were unable to advise the total number of people employed by gender or broad occupational groups, making it impossible to compute accurate lost-time injury rates.
- Data relating to noise-induced hearing loss are incomplete, since data are only routinely collected on disease or injury resulting in loss of 5 or more work days, which is rarely the case for hearing loss.

While WorkCover maintains hearing-loss data for people insuring through the Corporation, no information is available on the very substantial fraction of the workforce employed by self-insurers.

Sentinel Health Events (Occupational)

This system of health surveillance was developed in the US National Institute of Occupational Safety and Health (NIOSH). Data sets, such as mortality statistics from death certificates, are scanned for cause of death - injury - occupation combinations which match known occupational diseases eg nasal cancer in furniture workers. In this volume we have used this system to survey specialist physicians to estimate the occurrence of renal, neurological and haematological systems, and have not found the method very fruitful. Indeed NIOSH itself has virtually abandoned the system for the same reasons. To detect occurrences of less common occupational diseases, it would be preferable to undertake hazard surveillance rather than to continue with outcome surveillance. Thus, for example, if information becomes available on beryllium disease in a particular industry or process, it would probably be more cost-effective to identify such industries or processes and to evaluate exposures and undertake health surveillance on the workers involved, rather than waiting for the occurrence of overt disease to appear in a survey of doctors or hospital statistics such as the ISIS system.

However, for the more common occupational diseases, it would still be worthwhile surveying physicians. We found the survey of respiratory physicians quite useful, even though it was *ad hoc*, and putting such a system on a formal basis would probably have the cooperation of respiratory physicians and provide information which could be of practical benefit in disease prevention, as has been the experience of such a system in the United Kingdom. A similar benefit would almost certainly come from such a notification system for occupational dermatoses.

Conclusion

Descriptive occupational epidemiological studies rely heavily on routinely collected records of medical treatment, employment and occupational exposure. It is the case that the data used in this chartbook are less than ideal for the task of clearly elucidating the role of occupation in adverse health outcomes. Devising the optimum occupational health surveillance system is a subject in itself, but the following recommendations should be considered.

1. With regard to cancer registration, improvement in the specification of occupation and in occupational history could be achieved via the hospital-based cancer registries rather than the Central Cancer Registry. Those registries which deal with sites for which there is a strong occupational component in their aetiology, could be augmented with more detailed occupational information. One relatively inexpensive activity would be to focus on cancers which are relatively uncommon and which are frequently occupation-related: examples would be sinonasal cancers (wood and furniture, leather, nickel, chromium) and scrotal cancers (coal tar pitch, mineral oils). This process could operate in the same manner as the Australian Mesothelioma Register, except that by operating on a State basis it could lead to prompt workplace intervention.
2. To supplement information obtained from compensation data, information should be obtained on the actual occurrence of disease and injury. Four options appear possible:
   (i) An industry survey similar to that of the US Bureau of Labor Statistics. This is a survey of a probability sample of industries, who provide information from their OSHA logs on work-related diseases and injuries in their employees.
   (ii) Addition of questions on occupational diseases and injuries in the SA Health Omnibus survey

(iii) The inclusion of an occupational disease and injury supplement in an Australian Health Survey, similar to those produced from time to time by the UK Office of Population Census and Statistics

(iv) Inclusion of occupational disease and injury data in the periodic Labour Force Surveys undertaken by the ABS. This has already been done by the ABS in New South Wales in 1993.[1] (This survey also showed that compensation statistics underestimate injury rates.)

3. Appropriate physicians be periodically surveyed for the occurrence of work-related respiratory disease and skin disease.

4. For less common systemic diseases related to occupation, hazard surveillance may be preferable to disease surveillance.

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12. Overview

R T GUN

This volume makes an examination of the occurrence and distribution of occupational injury and disease in South Australia, and of the trends following the introduction of the Occupational Safety, Health and Welfare Act of 1986. This legislation, modelled on the principles proposed by the Robens Committee of the UK Parliament in 1971, marked an important shift in the manner in which safety in the workplace is to be achieved. Previous legislation relied on specific regulations, each targeting a specific problem, enforced by an external, ie government, inspectorate which could prosecute employers for breach of a regulation. Current legislation on the other hand is based on self-regulation by employers, who are required themselves to identify any hazard in their workplaces - whether or not they are covered by a regulation - and to institute controls. Statutory codes of practice are enacted to guide employers on the procedure for identifying hazards and assessing risk. The occurrence of an accident is *prima facie* evidence that the employer has failed to exercise duty of care, irrespective of whether a regulation has been breached, and in the event of prosecution the onus is then on the employer to show that action was taken, as far as was reasonably practical, to prevent the accident.

At the time of presenting its report, the Robens Committee had no data to show that its proposals would improve Workplace safety. The only indication can come from death and injury trends after the proposals were put into effect. The proposals were enacted in the UK in 1974, and in the following years there was a steady reduction in the rate of occupational fatalities. However this could not be shown to be attributable to the Robens legislation, since a parallel fall in the occupational fatality rate occurred in the US, which has continued to rely on control by prescriptive regulation.[1]

Injury experience in SA

The legislation modelled on the Robens philosophy came into law in South Australia in 1987, and the appearance of injury statistics for subsequent years gives some indication of the effects of the legislation. Most of the data go only to 1990-91, so that it is probably too early to expect significant changes, as the infrastructure of the new system needs time to be put into place. However some inferences can be drawn, helped by the availability of data on fatalities up to 1993-94.

The interpretation of the data is complicated by the WorkCover scheme, which was also introduced in 1987. Not only did the administration of workers' compensation change, but the responsibility for compiling statistics passed from the Australian Bureau of Statistics (ABS) to WorkCover itself. The ABS and WorkCover have both published data only on injuries which resulted in more than 5 days of lost time.

Following the introduction of WorkCover there was a sharp increase in the reported injury rate - from an average of 24.3/1000 person-years in the 3-year period preceding WorkCover to 29.7/1000 person-years in the first 3-year period for which WorkCover data are available. This increase is a consequence of the WorkCover legislation itself rather than any failure in the new Health and Safety law. Examination of the available statistics for all workers' compensation claims indicated that the total number of claims has remained constant, and that the reported rate increase is due to an increase in the proportion of claims which result in more than 5 days of lost time.

The total lost time and the average lost time per accident also showed a steep increase, but again this is likely to be due to the WorkCover legislation and its administration rather than an increase in the actual number of severe injuries.

Fatalities

The fatality statistics are suggestive of a reduction in death rates from injury in the 3-year period to 1993-94. It may be premature to claim a real improvement as a result of the legislation, as fatality data frequently require upward revision. However the fatality rate has been sustained below 3.0 per 100 000 person-years for 3 successive years, which has not happened previously in the period since 1975-76.

Of course it is not only the law which determines safety performance in the workplace. Other factors include the penalties imposed for non-compliance, as well as economic factors, the commitment of management, the availability of sound advice on injury prevention etc. The resultant of all these influences can only be assessed by the examination of statistics from year to year, and for this to be done it is important that data from year to year be comparable. Comparison of statistics has been made difficult by the passing of responsibility from the ABS to WorkCover. From May 1991 WorkCover has adopted the criteria of the National Data Set, promulgated by Worksafe Australia, and it is important that the drive for national uniformity does not impair the capacity to monitor the performance within individual States over time, so that any problems with the occupational safety law, or its administration or enforcement, can be remedied.

Musculoskeletal injuries

Statistics on musculoskeletal injuries, such as for back injuries and RSI, offer the opportunity to monitor the performance of the manual handling regulation and Code of Practice, which were introduced in South Australia in 1990. As with the Robens legislation itself, regulations pursuant to the new Act also place the onus on employers to identify the hazards in their own workplaces. In the case of manual handling, there is a Code of Practice which recommends a procedure to be followed by employers. The Code of Practice purports to enable employers to identify hazards and assess the risks from manual handling, yet there is no evidence that it will do so.

The data so far do not indicate any downturn in the rate of back injuries, and the number of RSI claims has sharply increased. Again, it is premature to expect an improvement in the rate of manual handling injuries, but there is reason to doubt whether a system based on codifying risk assessment is sufficiently versatile to anticipate the large range of situations which give rise to manual handling injuries. The Code of Practice seeks to limit the physical loads which workers must handle, reduce the physical stress in handling given loads, and reduce the frequency with which such loads are handled. Yet these physical demands have been steadily reducing for many years, without any reduction in the rate of back injury. It is likely that many back injuries are not caused by excessive physical loads - which workers can often regulate for themselves - but by events which are unlikely to be anticipated in the assessment as prescribed in the Code of Practice.

Thus the prevention of manual handling injuries may require a more flexible assessment procedure, which takes into account not just the physical parameters of a job, but the *situations* likely to result in a manual handling injury; this might best be learnt from an information system based on what circumstances have caused injuries in particular manual handling systems in the past. Such a system might need to be based on a system of keywords, which would enable personnel carrying out risk assessment to identify potential risks from events which have occurred elsewhere.

Diseases & injuries caused by chemicals

A number of important observations may be made from the data in relation to the effect of chemicals.

With the decreasing occurrence of severe illness from chemicals, eg lead poisoning, attention has focussed the use of chemicals in amounts insufficient to cause acute illness but if continued at low levels may lead to serious diseases in the long term. Foremost among these concerns has been fear of chemically-induced cancer, based on the presumption that even low-level exposures can cause a finite number of cancers.

It is these concerns which have driven the regulations for the control of hazardous substances. Again, the regulations are supported by a Code of Practice which aims to provide employers with the means for identifying hazards and assessing risk from chemicals, and they focus on the chemicals themselves rather than the situation in which they are used. The codified assessment procedure includes identifying hazardous chemicals, (from a list and/or an algorithm), obtaining information about its effects and what are safe levels of exposure, measuring the exposure and reducing it to safe levels. It is argued that if exposures are then maintained within safe limits, long-term disease can be prevented.

The data in this volume put the problem of chemical morbidity into perspective. The commonest problem from chemicals is not insidious disease, but acute injury. In the 3-year period there were approximately 400 chemical injuries, mostly burns or poisonings, which resulted in more than 5 days of lost time, and 102 hospital admissions for the effects of chemical exposures occurring at a workplace. On the other hand, it is clear from the questionnaire sent to specialist physicians that systemic diseases other than cancer caused by chemicals are very uncommon. Even chemical-related cancer is less frequent than acute chemical injury, although the outcome is often more serious.

It is likely that acute chemical injuries will not be prevented by the rote application of a code of practice. Analogous to the manual handling problem, effective prevention may lie not just in examining the properties of the chemicals themselves but the *situations* which give rise to injury. For example, carbon monoxide poisoning arises from administrative or engineering failure, and cannot be anticipated from measuring the carbon monoxide exposure when the system is not in failure mode. To anticipate such chemical injuries, as opposed to diseases, may require an information system based on hazardous situations which have arisen previously, and which employers may access to anticipate potentially dangerous situations in their own workplaces.

For the control of current cancer risks, it is not certain that the present strategy will identify the principal hazards identified in the analysis presented here. Of the recognised carcinogens present in South Australian workplaces, many are identified not as a particular exposure but simply by industry, such as boot and shoe manufacturing, wood and furniture industries, painting and the rubber industry. It is not clear that the industries concerned have all been alerted to the potential risks in their workplaces, nor that they are likely to pay the necessary attention to these problems under the generic principles of the Code of Practice.

Of equal concern is the lack of information on whether the workers industries identified in this survey (Chapter 2) are actually experiencing an excess cancer risk. This question could be partly addressed by a simple cancer surveillance system.

The data also indicate that skin diseases are probably the most common problems from occupational use of chemicals. The incidence rates derived from compensation statistics probably represent a substantial degree of under-reporting.[2] An alternative to WorkCover statistics should be considered for surveillance of skin disease. The Epi-Dem system operating in the UK commends itself by its simplicity. Dermatologists and occupational physicians are surveyed periodically for cases of occupational dermatoses they have seen, together with the causative agent. The patient's date of birth and initials are provided, thus preventing double counting whilst retaining anonymity.

The survey of chemicals also revealed that it is difficult to find where many of the carcinogenic and otherwise hazardous chemicals are being used. Much of the use of substances such as cadmium, acids, cobalt, MOCA and mercury occurs in small workshops. This would not matter if the employers and workers concerned were aware of the potential risks and acted upon them, but it is the small workshops where Codes of Practice are least likely to achieve penetration. Some selective hazard surveillance is required.

Stress

The findings in relation to stress suggest that the workers' compensation data are not a true indication of the morbidity due to occupational stress. The overwhelming preponderance of stress claims in the public sector may mean that employment in that sector is more stressful than in private enterprise, but it is more probable that public sector employees are less inhibited from making stress claims by the possible adverse effects they would have on their careers .

The suicide data suggest that it is blue collar workers, particularly the unskilled, who experience the maximum stress. However the data may reflect selection factors; for example, people with psychiatric illness - a strong predictor of suicide risk - may gravitate towards unskilled work.

Comparatively little attention has been paid to the possible relationship between occupational stress and cancer. Epidemiological evidence has been accumulating that the continuing stresses of employment can be an important predictor of several types of cancer. Possible mechanisms include behaviour change (eg to alcohol use) or alteration of the immune mechanism (although the response of the immune system to psychological stressors is not well understood). It is quite possible that stress is a more important mediator of cancer than chemicals.

Solar radiation

SA Cancer Registry data clearly indicate an excess of cancer of the lip in outdoor workers. The Anti-Cancer Foundation is now very active in the prevention of cancers caused by solar radiation, but there is a probable need to ensure that the campaign extends to all outdoor workers and their employers.

References

1. Gun RT. Regulation or self-regulation: is Robens-style legislation a formula for success? J Occup Health and Safety, Australia and NZ 1992; 8:383-388.
2. Keegan K. Occupational skin disorders among workers in the Department of Transport, South Australia. Research Dissertation, Department of Community Medicine, University of Adelaide, 1994.

Appendices

Appendix 1

Questionnaire to respiratory physicians

1. *Silicosis*

(a) How many cases of silicosis have been seen in your Department since 1989?_____

(b) Please list, where possible, the occupations or activities responsible in each case.

2. *Asbestosis*

(a) How many cases of asbestosis have been in your Department since 1989?_____

(Do not include cases of pleural plaques without any demonstrable lung disease)

(b) Please list where possible, the occupations or activities responsible in each case.

3. *Lung Cancer*

(a) Over this time how many cases have been seen in your Department of asbestosis complicated by carcinoma of the lung?_____

(b) Over this time how many cases have been seen in your department of lung cancer, in the absence of asbestosis, in patients who had been occupationally exposed to asbestosis? (For reference, a list of industries with the most significant asbestos exposure is included in the next question)_____

(c) The following is a list of industries which accounted for 84% of the cases with known asbestos exposure in the Australian Mesothelioma Surveillance Program, 1980-85. If you have been able to identify any asbestos-exposed *lung cancer* cases for Question 3(b), please write the number of cases next to the industry classification in the list below.

Asbestos mining and milling
Asbestos or asbestos product manufacturing, installation or transport (including asbestos-cement)
Insulation work
Construction or demolition
Shipping or stevedoring
Boiler fabrication
Railways fabrication, repair or maintenance
Steel or non-ferrous smelting
Engineering fabrication or repairs
Power-station construction or maintenance
Other (please specify)

Thank you for your assistance

Appendix 2

Questionnaire to haematologists

Haemolytic anaemia, non-autoimmune

Occupation	*Agent*	*No. cases*
Leather Industry	copper sulphate	[ ]
Electrolytic Processes	arsine	[ ]
Smelting of arsenic-containing ore	arsine	[ ]
Primary Plastics Manufacture	trimellitic anhydride	[ ]
Dye, Celluloid or Resin Industry	naphthalene	[ ]
any other occupation with exposure to any of the agents in column 2 (state occupation if known):	state which of the above agents:	
		[ ]
		[ ]
		[ ]
		[ ]
any of the occupations in column 1 with a suspected agent not mentioned in column 2 (state which occupation):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]

Aplastic anaemia

Occupation	*Agent*	*No. cases*
Explosives Manufacture	trinitrotoluene	[ ]
Radiologists	ionising radiation	[ ]
any other occupation with exposure to any of the agents in column 2 (state occupation if known):	state which of the above agents:	
		[ ]
		[ ]
		[ ]
		[ ]
any of the occupations in column 1 with a suspected agent not mentioned in column 2 (state which occupation):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]

Methaemoglobinaemia

Occupation	*Agent*	*No. cases*
Explosives and Dye Manufacture	acromatic amino and nitro compounds Eg. aniline	[ ]
Rubber Workers	aniline, o-toluidine nitrobenzene	[ ]
any other occupation with exposure to any of the agents in column 2(state occupation if known):	state which of the above agents:	
		[ ]
		[ ]
		[ ]
		[ ]
any of the occupations in column 1 with a suspected agent not mentioned in column 2 (state which occupation):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]

Agranulocytosis or neutropaenia

Occupation	*Agent*	*No. cases*
Explosives Manufacture	phosphorus	[ ]
Pesticide Workers	phosphorus & inorganic arsenic	[ ]
Pharmaceutical Workers	inorganic arsenic	[ ]
Miners, Smelters, Boot and Shoe Workers	benzene	[ ]
any other occupation with exposure to any of the agents in column 2 (state occupation if known):	state which of the above agents:	
		[ ]
		[ ]
		[ ]
		[ ]
any of the occupations in column 1 with a suspected agent not mentioned in column 2 (state which occupation):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]

Appendix 3

Questionnaire to nephrologists

Acute or chronic renal failure

Occupation	*Agent*	*No. Cases*
Plumbers	Inorganic lead	[ ]
Battery Makers	Inorganic lead	[ ]
Solderers	Inorganic lead	[ ]
Battery Makers	Inorganic mercury	[ ]
Jewellers	Inorganic mercury	[ ]
Dentists	Inorganic mercury	[ ]
Fire Extinguishers	Carbon tetrachloride	[ ]
Antifreeze manufacture	Ethylene glycol	[ ]
any other occupation with exposure to any of the agents in column 2 (state occupation if known):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]
any of the occupations in column 1 with a suspected agent not mentioned in column 2 (state which occupation):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]

Appendix 4

Questionnaire to neurologists

Toxic encephalitis

Occupation	*Agent*	*No. cases*
Battery Workers	Lead	[ ]
Smelter Workers	Lead	[ ]
Foundry Workers	Lead	[ ]
Battery Makers	Inorganic & Organic Mercury	[ ]
Fungicide Formulators	Inorganic & Organic Mercury	[ ]
any other occupation with exposure to any of the agents in column 2 (state occupation if known):	state which of the above agents:	
		[ ]
		[ ]
		[ ]
		[ ]
any of the occupations in column 1 with a suspected agent not mentioned in column 2 (state which occupation):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]

Carpal tunnel syndrome

Occupation	*Agent*	*No. Cases*
Meat Packers	Cumulative Trauma	[ ]
Deboners	Cumulative Trauma	[ ]
any other occupation with exposure to the agent stated in column 2 (state occupation if known):	state agent:	
		[ ]
		[ ]
		[ ]
		[ ]
any of the occupations in column 1 with a suspected agent not mentioned in column 2 (state which occupation):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]

Cerebellar ataxia

Occupation	*Agent*	*No. cases*
Chemical Workers	Toluene	[ ]
Battery Makers	Organic Mercury	[ ]
Fungicide Formulators	Organic Mercury	[ ]
any other occupation with exposure to any of the agents in column 2 (state occupation if known):	state which of the above agents:	
		[ ]
		[ ]
		[ ]
		[ ]
any of the occupations in column 1 with a suspected agent not mentioned in column 2 (state which occupation):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]

Mononeuritis of the upper limb & mononeuritis multiplex

Occupation	*Agent*	*No. cases*
Dental Technician	Methyl methacrylate monomer	[ ]
Poultry Processing	Cumulative Trauma	[ ]
Turkey Processing	Cumulative Trauma	[ ]
Meatpackers	Cumulative Trauma	[ ]
Deboners	Cumulative Trauma	[ ]
any other occupation with exposure to any of the agents in column 2 (state occupation if known):	state which of the above agents:	
		[ ]
		[ ]
		[ ]
		[ ]
any of the occupations in column 1 with a suspected agent not mentioned in column 2 (state which occupation):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]

Inflammatory & toxic neuropathy

Occupation	*Agent*	*No. cases*
Pest Exterminator	Arsenic/arsenic compounds	[ ]
Pharmaceutical Forumulators	Arsenic/arsenic compounds	[ ]
Furniture Refinishers	Hexane	[ ]
Plastics Worker	methyl n-butyl ketone	[ ]
Explosives Worker	Trinitrotoluene	[ ]
Plastics Worker	Tri-o-cresyl phosphate	[ ]
Hydraulics Worker	Tri-o-cresyl phosphate	[ ]
Battery Worker	Inorganic Lead	[ ]
Smelter Worker	Inorganic Lead	[ ]
Foundry Worker	Inorganic Lead	[ ]
Dentists	Inorganic Mercury	[ ]
any other occupation with exposure to any of the agents in column 2 (state occupation if known):	state which of the above agents:	
		[ ]
		[ ]
		[ ]
		[ ]
any of the occupations in column 1 with a suspected agent not mentioned in column 2 (state which occupation):	state name of agent:	
		[ ]
		[ ]
		[ ]
		[ ]