MONASH University

2018 HEALTH WATCH

15th REPORT

Australian Institute of Petroleum

MONASH
Centre for Occupational
and Environmental Health

AIP MONASH University Health Watch
MONITORING THE HEALTH OF EMPLOYEES IN THE AUSTRALIAN PETROLEUM INDUSTRY

HEALTH WATCH

The Australian Institute of Petroleum Health Surveillance Program

Fifteenth Report

July 2018

Monash University
Monash Centre for Occupational and Environmental Health (MonCOEH)
Department of Epidemiology and Preventive Medicine (DEPM)

This Fifteenth Report contains an analysis of deaths occurring up to 30th November 2015, and cancers registered up to 31st December 2012.

Monash Centre for Occupational and Environmental Health (MonCOEH)
Department of Epidemiology and Preventive Medicine,
Monash University,
553 St Kilda Road,
Melbourne,
VIC 3004,
Australia.

ISBN 978-0-9804448-2-7

MESSAGE FROM THE CEO OF THE AUSTRALIAN INSTITUTE OF PETROLEUM

For almost 40 years, the Australian Institute of Petroleum (AIP) has sponsored the independent *Health Watch* study to monitor the health of petroleum industry employees in Australia. The study underpins the very longstanding commitment of the industry to the health and wellbeing of their employees.

This internationally recognised research covers over 20,000 past and present employees during their time in the industry and after they leave or retire to track what happens to their health. *Health Watch* is a detailed epidemiological analysis of job types, workplace practices, lifestyle influences, and illness and causes of death. The health of petroleum industry employees is then compared with data for the overall Australian community.

The study provides valuable insights into the influences on employee health, such as the relationship between the incidence of various cancers and working in the industry, and the measurable effects of an employee's lifestyle. The findings of the study assist the petroleum industry to develop workplace policies and programs that are providing safe and healthy working environments, whilst also providing a robust scientific basis for the community to understand the health impacts of exposure to petroleum products.

Since 2005, the *Health Watch* study has been conducted by the Monash Centre for Occupational and Environmental Health (MonCOEH) at Monash University, to take advantage of other epidemiology programs and collaborative research. The Study has been under the direction of Professor Malcolm Sim and his research team including Associate Professor Deborah Glass, Elisa Wood and Anthony Del Monaco.

Importantly, *Health Watch* was expanded in the last decade to provide new employees in participating company worksites across Australia the opportunity to join the study, which resulted in 2,000 employees voluntarily joining *Health Watch*. This new cohort will help to maintain this important longitudinal health study of Australian petroleum industry employees into the future.

AIP is very pleased to receive the 15th *Health Watch* Report which continues to clearly show that petroleum industry employees have better health than the general Australian community and are less likely to die from cancer and from heart, respiratory and digestive diseases. Also most encouraging in this latest study is that the risk of leukaemia is no greater than the general population and has been reducing. There are also few asbestos related lung cancers, and incidences are unlikely to have occurred from working in Australian refineries.

AIP therefore wishes to thank the thousands of employees who continue to participate in *Health Watch* to help enhance understanding of the health impacts of working in the petroleum industry. This internationally respected study continues to be highly valued by petroleum companies and their employees.

Paul Barrett
Chief Executive Officer

HEALTH WATCH PROJECT TEAM

Monash Centre for Occupational and Environmental Health (MonCOEH)
Department of Epidemiology and Preventive Medicine,
Monash University

Study Director	Professor Malcolm Sim
Associate Professor	Deborah Glass
Research Officer	Elisa Wood
Research Fellow / Data Manager	Anthony Del Monaco

The Statistical Consulting Centre, The University of Melbourne

Statistician	Steve Vander Hoorn

HEALTH WATCH ADVISORY COMMITTEE

Dr Andrea James, Chairperson (to September 2017)	Viva Energy Australia Pty Ltd
Dr Raelene Young	Viva Energy Australia Pty Ltd
Mr Nathan Dickens (Secretary)	Australian Institute of Petroleum
Ms Kim Ng	BP Australia Pty Ltd
Dr Marcus Hirschfield	ExxonMobil Australia
Dr Nathan Pastor	ExxonMobil Australia
Mr Rory O'Connor,	Woodside Energy
Dr Arthur Stratigopoulos	Shell Australia Pty Ltd
Mr Andrew Orfanos	Caltex Australia Limited
Dr Max Hills	Chevron Australia Pty Ltd
Mr Michael Collins	Santos Limited
Mr Michael Borowick JP (from March 2018)	Australian Council of Trade Unions

AUSTRALIAN INSTITUTE OF PETROLEUM

COMPANIES WITH EMPLOYEES PARTICIPATING IN *HEALTH WATCH*

Viva Energy Australia Pty Ltd
Caltex Oil Australia (incorporating Total, Ampol Australia and Australian Petroleum Pty Ltd)
BP Australia Pty Ltd (incorporating Amoco)
ExxonMobil Australia Ltd (formerly Esso Australia Ltd and Mobil Oil Australia Limited)
Santos Ltd
Shell Australia Pty Ltd
Chevron Australia (formerly West Australian Petroleum Pty Ltd (WAPET))
Woodside Energy Ltd
Airport Fuel Services
Castrol Australia Pty Ltd (up to 30/06/1994)

ACKNOWLEDGMENTS

Health Watch is funded by the Australian Institute of Petroleum.
The *Health Watch* cohort study was designed by Professor David Christie.
We are indebted to the contact persons in each of the participating companies. *Health Watch* is dependent on them for follow-up information.
We thank the Australian Institute of Health and Welfare for its ongoing co-operation which has made it possible for *Health Watch* to report on the occurrence of cancer and mortality. We also thank the staff of State Death and Cancer Registries for confirmation of information, in particular the staff of the Victorian Cancer Registry for their co-operation in searching their registry.
Thanks to Professor Ian Gordon from the Statistical Consulting Centre, The University of Melbourne, for his statistical oversight.
Finally, we wish to thank the many employees who participated and assisted the team.

CONTENTS

PREFACE **13**

SUMMARY OF LATEST *HEALTH WATCH* RESULTS **14**

1. INTRODUCTION **17**
1.1. Industry Background 17
1.2. Development and Design of the Health Watch Surveillance Program 17
1.3. Reporting Results 20
1.4. Consent and Confidentiality 20
1.5. Ethics Committee Approval 20
1.6. Present Work 21

2. METHODS **22**
2.1. Study Design 22
2.2. Formation and Maintenance of the Cohort 22
2.2.1. Recruitment *22*
2.2.2. Entry to the Cohort *23*
2.2.3. Information Collected at Survey Interviews *23*
2.2.4. Information from Participating Companies *24*
2.2.5. Coding of Employment Data for Assigning Estimates of Hydrocarbon *24*
2.2.6. Participation Rates in Health Watch Surveys *24*
2.2.7. Follow-up *25*
2.2.8. The Special Case of Drivers *25*
2.3. Withdrawal of Members of the Cohort 26
2.4. Health Outcomes 26
2.4.1. Mortality Records *26*
2.4.2. Validation of Mortality Records *27*
2.4.3. Cancer Incidence *28*
2.4.4. Validation of Cancer Incidence *28*
2.5. Statistical Analyses 29
2.5.1. Follow-up Time *29*
2.5.2. External Measures of Comparison: the SMR and SIR *30*
2.5.3. Internal Measures of Comparison: the RMR and RIR *31*
2.5.4. Confidence Intervals and Risk Estimates *31*
2.5.5. The Problem of Multiple Analyses *32*
2.5.6. Confounding Variables *32*
2.5.7. Adjustments to SMR and SIR analyses *32*
2.5.8. Adjustments to RMR and RIR analyses *33*
2.5.9. Time-related Variables *33*
2.5.10. Analysis by Workplace Type *34*
2.5.11. Analysis by Self-Reported Smoking and Drinking Status *34*
2.5.12. Analysis by Job Type *34*

3. GENERAL RESULTS FOR THE COHORT **35**
3.1. The Cohort Population 35
3.1.1. Description of Cohort Population at 30th November 2015 ..... *35*
3.1.2. Person-years of Observation in the Cohort *36*
3.2. Person-years and Mortality and Cancer Incidence Rates 36
3.3. All-cause Mortality – Men and Women 36
3.4. Cancer Incidence and Mortality – Men and Women 37
3.5. Results in Women 39
3.5.1. Mortality by Major Cause for Women *39*
3.5.2. Cancer in Women *40*

3.6. Mortality in Men 41
3.6.1. Mortality among Men by Major Cause 41
3.6.2. All-cause Mortality and Time Relationships 42
3.7. Cancer Incidence among Men 46
3.7.1. Cancer and Time Relationships for Men 47
3.7.2. Workplace Type and Health Outcomes among Men 50
3.8. Non-malignant Disease from Asbestos Exposure 52

4. LIFE STYLE FACTORS AMONG MALE MEMBERS OF THE COHORT 53
4.1. Tobacco Smoking 53
4.1.1. Smoking Status 53
4.1.2. Smoking and All-cause Mortality among Men 54
4.1.3. Smoking and Ischaemic Heart Disease (ICD-10 I20-I25) 55
4.1.4. Smoking and Cancer among Men 55
4.1.5. Smoking and Bladder Cancer (ICD-10 C67) 57
4.1.6. Deaths Attributable to Smoking among Men in the Cohort 58
4.1.7. Effects of Quitting 59
4.2. Alcohol Consumption among Men 60

5. SPECIFIC CANCERS 63
5.1. Mesothelioma (ICD-10 C45) 63
5.2. Lung Cancer (ICD-10 C33-C34) 63
5.3. Melanoma of the Skin (ICD-10 C43) 64
5.4. Prostate Cancer (ICD-10 C61) 66
5.5. Bladder Cancer (ICD-10 C67) 66
5.6. Kidney Cancer (ICD-10 C64-C66, C68) 66
5.7. Leukaemias (ICD-10 C91-C95) 66
5.8. Multiple Myeloma (ICD-10 C90) 68

6. HEALTH OUTCOMES IN SPECIFIC JOBS 70
6.1. All-cause Mortality in Men by Job Group 70
6.2. Cancer Incidence in Men by Job Group 70
6.2.1. Incidence of Cancer and Mortality among Drivers 71
6.3. Cancer Mortality in Men by Job Group 73
6.4. Ischaemic Heart Disease (ICD-10 I20-125) Mortality in Men by Job Group 74
6.5. Mortality from Accidents and Violence (ICD-10 V00-V99, W00-W99, X00-X99, Y00-Y99) in Men by Job Group 74

7. DISCUSSION 76
7.1. Strengths and Weaknesses of the Study 76
7.1.1. Individual Interview Data 76
7.1.2. High Participation Rate 76
7.1.3. Volunteer Bias 76
7.1.4. Unverified Date of Employment 76
7.1.5. Complete Cancer Identification 77
7.2. The Healthy Worker Effect (HWE) 77
7.3. Mesothelioma and other Asbestos-related Conditions 78
7.4. Lung Cancer 78
7.5. Melanoma 79
7.6. Bladder Cancer 80
7.7. Leukaemia 81
7.8. Other Lympho-Haematopoietic Cancers in Men 82
7.9. Myelodysplastic syndrome (MDS) 83
7.10. Regularity of Cohort Reports 83
7.11. Smoking 84

8. CONCLUSIONS 85

9. THE NEW *HEALTH WATCH* COHORT 87
9.1. Introduction 87
9.1.1. Eligibility 87
9.1.2. Survey Implementation 88
9.1.3. Informed consent 88
9.2. Recruitment 88
9.2.1. Company Site Visits 88
9.2.2. On-line Recruitment 88
9.2.3. Email/Postal Invitation to Employees 88
9.2.4. Office-only Employees 88
9.3. Non-Responders 88
9.4. Participation 89
9.5. Results for the Re-Open Cohort 91
9.6. Summary 92
10. ACRONYMS 93
11. REFERENCES 94

Figures

Figure 1: *Health Watch* cohort structure 19

Figure 2: Latest smoking status of the men in the *Health Watch* cohort 53

Figure 3: Relative risk of dying from any cause, of cancer, or of ischaemic heart disease among men for different smoking categories, compared to those who never smoked, adjusted for age and calendar year. 58

Figure 4: Relative risk of dying from lung cancer among men for different smoking categories compared to those who never smoked, adjusted for age and calendar year. 59

Figure 5: Relative risk of dying (all-cause mortality) for men with different levels of alcohol consumption, compared to those men who never drank alcohol and compared to occasional drinkers, adjusted for age, calendar year and smoking 61

Figure 6: All Cause SMR, all cancer SIRs for men in *Health Watch*, plotted at yearly intervals 78

Figure 7: SIRs and cumulative melanoma incidence in men from 1987-2012 80

Figure 8: SIRs for leukaemia, NHL and MM in men from 1987 - 2012 82

Figure 9: Participation from the 2010-2012 6th *Health Watch* Survey 90

Tables

Table 1: State of the cohort as at 30/11/2015 35

Table 2: Distribution of year of birth for *Health Watch* cohort members 36

Table 3: Person-years of observation 36

Table 4: All-cause mortality by sex, adjusted for age and calendar period of follow-up, compared to the Australian population 37

Table 5: All-site cancer incidence, men and women, adjusted for age and calendar period of follow-up, compared to the Australian population 37

Table 6: All-site cancer mortality, men and women, adjusted for age and calendar period of follow-up, compared to the Australian population 37

Table 7: Mortality by major cause for women, adjusted for age and calendar period of follow-up, compared to the Australian population 39

Table 8: Cancer incidence by major anatomical site for women, adjusted for age and calendar period of follow-up compared to the Australian population 40

Table 9: Mortality by major cause for men, adjusted for age and calendar period of follow-up, compared to the Australian population 41

Table 10: All-Cause mortality among men by period of first employment, adjusted for age and calendar period of follow-up, compared to the Australian population 42

Table 11: All-cause mortality among men by period of first employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to those employed from 1985 43

Table 12: Standardised all-cause mortality among men by duration of employment, adjusted for age and calendar period of follow-up, compared to the Australian population 43

Table 13: Relative all-cause mortality among men by duration of employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to those employed for 5-9 years 44

Table 14: Standardised all-cause mortality among men by time since first employment, adjusted for age and calendar period of follow-up, compared to the Australian population 44

Table 15: Relative all-cause mortality among men by time since first employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to the those first employed 5-9 years ago 45

Table 16: Cancer incidence by major anatomical site in men, adjusted for age and calendar period of follow-up, compared to the Australian population 46

Table 17: Cancer incidence and cancer mortality by period of first employment, adjusted for age and calendar period of follow-up, and ever/never smoker compared to those employed after 1985 47

Table 18: Cancer incidence and cancer mortality by duration of employment, adjusted for age and calendar period of follow-up, and ever/never smoker compared to those employed for 5-9 years 48

Table 19: Cancer incidence and mortality by time since first employment, adjusted for age and calendar period of follow-up, and ever/never smoker compared to those first employed 5-9 years ago 48

Table 20: Numbers of male members of the cohort in each workplace type 50

Table 21: All-cause mortality in men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population 50

Table 22: Ischaemic heart disease (ICD-10 I20-I25) mortality for men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population 50

Table 23: Cancer incidence among men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population 51

Table 24: Cancer mortality among men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population 51

Table 25: Source of most recent smoking data for individuals in the cohort. 54

Table 26: Number of 'ever smokers' in each main job category 54

Table 27: All-cause mortality by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked 55

Table 28: Ischaemic heart disease mortality (ICD-10 I20-I25) among men by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked 55

Table 29: Cancer incidence among men by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked 56

Table 30: Cancer mortality among men by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked 56

Table 31: Lung cancer incidence (ICD-10 C33-C34) among men by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked 57

Table 32: Lung cancer mortality (ICD-10 C33-C34) among men by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked 57

Table 33: Bladder cancer incidence (ICD-10 C67) by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked 58

Table 34: All-cause mortality by alcohol category for men, adjusted for age, calendar year and smoking (ever vs never), compared to occasional drinkers (1-7 drinks per week) and total abstainers (0 drinks) 61

Table 35: Ischaemic heart disease mortality (ICD-10 I20-125) by alcohol category, adjusted for age, calendar year and smoking (ever vs never), compared to those who drink 1-7 alcoholic beverages per week. 62

Table 36: Melanoma (ICD-10 C43) incidence in men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population 64

Table 37: Melanoma (ICD-10 C43) incidence among men by period of first employment, adjusted for age and calendar period of follow-up, compared to those employed after 1985 64

Table 38: Melanoma (ICD-10 C43) incidence in men by duration of employment, adjusted for age and calendar period of follow-up, compared to those employed for 5-9 years 65

Table 39: Melanoma (ICD-10 C43) incidence in men by time since first employment, adjusted for age and calendar period of follow-up, compared to those first employed 5-9 years ago 65

Table 40: Melanoma (ICD-10 C43) incidence compared to national rates for men by state of last employment 65

Table 41: Melanoma (ICD-10 C43) incidence compared to state specific rates for men by state of last employment 66

Table 42: Leukaemia (ICD-10 C91-C95) incidence among men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population 67

Table 43: Leukaemia (ICD-10 C91-C95) incidence by period of first employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to those employed after 1985. 67

Table 44: Leukaemia (ICD-10 C91-C95) incidence in men by duration of employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to those employed for 5-9 years 67

Table 45: Leukaemia (ICD-10 C91-C95) incidence in men by time since first employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to those first employed 5-9 years ago 68

Table 46: Multiple myeloma (ICD-10 C90) incidence among men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population 68

Table 47: All-cause mortality among men by Job Group (ever held), adjusted for age and calendar period of follow-up, compared to the Australian population 70

Table 48: Cancer incidence among men by Job Group (ever held), adjusted for age and calendar period of follow-up, compared to the Australian population 71

Table 49: Cancer incidence by selected anatomical site, for drivers by ICD-10 codes, adjusted for age and calendar period of follow-up, compared to the Australian population. 72

Table 50: Relative mortality risk of Ever Drivers compared with Only Ever Office workers. 73

Table 51: Relative cancer incidence of Ever Drivers compared with Only Ever Office workers 73

Table 52: Cancer mortality in men by Job Group (ever held), adjusted for age and calendar period of follow-up, compared to the Australian population 73

Table 53: Ischaemic heart disease (ICD-10 I20-125) mortality in men by Job Group (ever held), adjusted for age and calendar period of follow-up, compared to the Australian population 74

Table 54: Mortality from accident/violence (ICD-10 V00-V99, W00-W99, X00-X99, Y00-Y99) in men by Job Group (ever held), adjusted for age and calendar period of follow-up, compared to the Australian population 74

Table 55: Melanoma (ICD-10 C43) risk over time for men in the *Health Watch* cohort 80

Table 56: Leukaemia incidence for men reported over time in the *Health Watch* cohort 81

Table 57: NHL (ICD-10 C82-C85, C96) and MM (ICD-10 C90) incidence for men in the *Health Watch* cohort from 1987 to 2012 83

Table 58: Criteria for inclusion in 6th *Health Watch* Survey (2010 - 2012) 87

Table 59: Breakdown of existing *Health Watch* members and new participants (including Head and Home Office Employees who were pre-registered into the database)..... 91

Table 60: Participation at worksites. Visited worksites vs online/postal invitation only 91

Table 61: Summary of total cancer and deaths in the Re-open cohort 92

PREFACE

Health Watch is an epidemiological health surveillance program established by the Australian Institute of Petroleum. Since 2005 the study has been executed by researchers from Monash University.

Health Watch consists of a prospective cohort study of all-cause mortality and cancer incidence and a case-control study of leukaemia and benzene exposure. The cohort study was carried out by the University of Melbourne from 1980 to 1998 and by the University of Adelaide from 1999 to 2005, before being transferred to Monash University. Researchers from Monash and Deakin Universities took over responsibility for the nested case-control study in 1999. In 2006, data from *Health Watch* were combined with that of LH cancer cases and controls from similar Canadian and UK studies to carry out a pooled and hence more sensitive analysis of the link between cancer risk and benzene exposure.

Health Watch covers those petroleum industry employees from all major participating oil and gas companies who voluntarily joined the program at their work sites across Australia. About 95% of the industry's employees who were approached to participate from refineries, gas plants, distribution terminals, and production sites, onshore and offshore, have joined *Health Watch* during the major recruitment drives between 1981 and 2000.

Employees in the industry were enrolled in the study by participating in one or more of four industry surveys in the 1980s and 1990s, using a detailed job and health questionnaire. This process obtained information on job tasks, on lifestyle factors including smoking and alcohol, and on health status. An employee was eligible to be included in the cohort analysis following a survey interview or after having served five years in the industry, whichever is later, and remains in the *Health Watch* cohort for life. All participants, including current and ex-employees of participating companies are contacted periodically to obtain an update on health status and employment history. A new cohort commenced in 2010 and current employees of participating companies were invited to participate via either a researcher assisted or online survey.

The employing companies maintain the flow of information on entrants, job changes, resignations and retirements and send regular employee updates to the *Health Watch* study. The study maintains contact with cohort members until death via regular Health Letter updates.

The main output of the study is this report and contains analyses of mortality and cancer incidence. These analyses are carried out by comparing the rates of deaths and cancers in the *Health Watch* cohort with the rates of the general Australian population.

Counts of death and cancer registrations in the general population are obtained from the Australian Institute of Health and Welfare (AIHW), which compiles the National Death Index (NDI) and the Australian Cancer Database (ACD) on behalf of all State Death and Cancer Registries. The Victorian Cancer Registry also provide cancer registrations for that state.

Deaths and cancers in the *Health Watch* cohort are obtained by linking the *Health Watch* data with State and National Death and Cancer Registries.

Cohort and case-control study findings have been published in periodic *Health Watch* reports of which this report is the fifteenth, and in scientific medical and other scientific journals (1-27).

This Report is published on the Australian Institute of Petroleum website (www.aip.com.au) and the MonCOEH website (www.coeh.monash.org/healthwatch.html). Summary reports are distributed to all members of the *Health Watch* cohort.

SUMMARY OF LATEST *HEALTH WATCH* RESULTS

Overview

The *Health Watch* study continues to demonstrate that petroleum industry employees have better health than the general Australian community and are less likely to die of the diseases commonly causing death including cancer, heart disease and respiratory conditions.
For men, death rates in all major disease categories were significantly lower than for the corresponding Australian population. However, there was an increase in the mortality rate of the minor category of lung disease, specifically, asbestosis. A significant reduction in all-cause mortality was seen among men in each workplace type e.g. refinery, terminal and upstream.
For men and women in this industry, the chance of getting cancer was similar to that for the general Australian population. Mortality from cancer was reduced for male *Health Watch* members, however, the cancer mortality rate for the female members was similar to the national rates.
There was no evidence of increasing mortality, cancer risk or increasing cancer mortality with increasing duration of employment. Generally, the chances of dying at any age were very similar no matter where *Health Watch* people work/ed and compared favourably with the rates in all Australian men. The chance of getting cancer was slightly elevated among the drivers, however, the risk of dying from cancer was the same as the national risk for this group.

Status of the cohort

This update of the *Health Watch* cohort was based on national mortality data to 30th November 2015 and cancer incidence data to 31st December 2012. 16,654 men and 1,373 women were included in this report. 3,349 men and 106 women in the cohort had died by the end of 2015. Members who died overseas or whose deaths were not identified on the NDI were not included in these analyses nor were the deaths of the Castrol employees who were excluded from the analysis. *Health Watch* has now accumulated 444,103 person-years of observation in men and 32,864 person-years in women.

Healthy worker effect continues for men and women

The age-adjusted death rate in male and female *Health Watch* members continues to remain significantly lower than that of the general Australian population. The strong *healthy worker effect* identified in previous reports continues to be observed. The chance of developing cancer is similar for people in this industry compared to the general Australian population. However, the mortality from cancer is reduced for *Health Watch* members, significantly so for men.

Results in women

Of the 106 female cohort members who have died, there were 54 deaths from cancer. The risk of cancer mortality in women was similar to that of the general Australian female population. There were 136 diagnosed cancers in women, and this rate was also similar to the national rate for women, i.e., the chance of getting most types of cancer was similar for women working in this industry as it was for the general female population.
The proportion of women in the *Health Watch* program is very small, however and this precludes more detailed analysis of their health outcomes.

Results in men

For men, 3,349 male members of the cohort have died including 1,388 deaths from cancer. However, death rates in all major disease categories - cancer, metabolic, mental, nervous,

circulatory, respiratory, digestive, urinary, and external causes (accidents, violence etc) were significantly lower than for the corresponding Australian population. A significant reduction in all-cause mortality was seen among men in all workplace types e.g. refinery, terminal and upstream.
There was an increase in mortality of the minor category of lung disease, specifically asbestosis. Eleven cohort members have died from asbestosis (SMR 2.27, 95% C.I. 1.19-4.27) and 119 members of the cohort have reported asbestos related illnesses. This was probably an underestimate of the true number because not all members self-report their illnesses and there is no national register for asbestosis as there is for cancer.
Within the cohort, there was evidence of a trend of increasing overall mortality (but not of cancer incidence) by period of first employment (compared with those employed post 1985), however, the mortality in these time periods was lower than the national rates for those corresponding time periods.
There was no evidence of increasing cancer or mortality risk by duration of employment or time since first employment (compared with those under 10 years).

Specific cancers

Three cancers, mesothelioma, melanoma, and prostate cancer continued to occur at statistically significantly higher rates in men working in the industry compared with the general population. Forty-nine incident mesotheliomas have occurred in the cohort (SIR 1.60, 95% C.I. 1.18-2.11), Among mesothelioma cases, 28 of the 49 cases (52%) occurred in refinery workers however only 40% of *Health Watch* members were employed in refineries.
There was a statistically significant increase in the incidence of melanoma in men compared to the general population of Australia. The rates were higher in the sunnier states but when compared with state-based rates, Victoria, New South Wales and Queensland rates were statistically elevated. The melanoma rate did not increase with increasing duration of employment, in fact, the rate decreased with increasing duration (compared to those employed 5-9 years). This suggests that a causal association with the workplace was unlikely, but this finding will continue to be monitored.
Although an increased risk of bladder cancer in the overall cohort was reported in previous *Health Watch* reports (4, 28), this updated analysis showed there was no additional risk of bladder cancer in the cohort compared to the general population. This finding was consistent with what was also observed in the 14th *Health Watch* report. Bladder cancer risk in drivers, whilst slightly lower than the previous report, remained elevated compared to the general population (SIR 1.46, 95% C.I. 0.92-2.21). When compared to office-only workers in the cohort, the risk is over 3 times greater in drivers (RIR 3.20, 95% C.I. 1.45-7.07). This comparison was first made in the 14th *Health Watch* report which also showed a border-line elevated risk of bladder cancer among drivers (RIR 1.60, 95% C.I. 0.96-2.49).(23)

Since the last report, there have been 19 new cases of leukaemia among men in the cohort. However, as identified in the last three consecutive *Health Watch* reports, and contrary to findings in earlier *Health Watch* reports (4, 5, 28, 29) there was again no statistically significant excess of leukaemia in the cohort (SIR 0.80, 95% C.I. 0.62-1.00).

Job group analyses

Health Watch compared members in some particular occupational groups, and a small but statistically significant overall excess in cancer incidence was found in tanker drivers (SIR 1.15, 95% C.I. 1.06-1.24). A similar excess was also observed in the 14th report.(9) The breakdown of major cancer categories among drivers showed that melanoma and prostate

cancer were the only specific cancer types that were statistically significantly elevated compared to the general population. Oesophageal cancer risk was also elevated and was borderline statistically significant (SIR 1.80, C.I. 0.98-3.01). The risk of bladder cancer among drivers was again elevated compared to the general population (as it was in the 14th *Health Watch* report) (23). The risk was not statistically significantly raised (SIR 1.46, C.I. 0.92-2.21) but warrants close monitoring.
Cancer mortality rates, however, were similar to the general population for most occupational groups, including drivers in the current analyses. Refinery operators and Office workers had a significantly lower risk of cancer mortality compared with the general population (SMR 0.88, 95% C.I. 0.78-0.99) and (SMR 0.80, 95% C.I. 0.73-0.87) respectively.

Lifestyle factors

Smoking related diseases i.e. lung cancer, cancer of the lip, oral cavity and pharynx, ischaemic heart disease mortality and chronic obstructive pulmonary disease mortality were lower in *Health Watch* members compared with the general population. However, within the cohort, there was a clear pattern that increasing smoking category was associated with increasing risk of all-cause mortality, specifically of ischaemic heart disease mortality, of overall cancer risk and cancer mortality and especially increased incidence of lung cancer and bladder cancer. Furthermore, it was clear that the risk of mortality and cancer were greatly reduced for ex-smokers compared with those who continued to smoke, however, the risk was still higher compared to those who have never smoked.
Moderate drinkers (1-7 drinks per week) had a lower death rate than total abstainers. Heavy drinking (more than 3 drinks per day), was associated with increased overall mortality.

1. INTRODUCTION

1.1. Industry Background

The petroleum industry became established in Australia in the first decade of the twentieth century when international companies began importing fuels and lubricants. Refineries were built from 1910 onwards and nationwide distribution networks were set up. The distances involved led to considerable cooperation between the competing companies, which were servicing a relatively small, scattered population. World War II was followed by a period of rapid population expansion. Refinery and associated petrochemical plant development took place, with major refineries in three states coming on-stream during the 1950s. Technological development has continued to date in line with the worldwide oil and gas industry. Australian refineries and terminals are technologically advanced although relatively small in capacity. Environmental legislation and emission controls are amongst the most stringent in the world, and this has resulted in changes in technology, e.g. introduction of bottom loading of road and rail tankers and hydrocarbon vapour recovery systems.

Local production of both oil and gas grew rapidly from the 1970s and the production of light crude oil and of natural gas made Australia a net energy exporter. Moreover, in the 1990s, the industry underwent considerable reorganisation leading to refinery operations becoming less labour-intensive, with a significant proportion of work now being undertaken by contractors. Consequently, fewer people are employed by the petroleum companies than when *Health Watch* was established, especially in the refining sector.

Oil production has now been declining over the years as mature oil fields reach exhaustion. The majority of oil reserves are currently located off the coast of Western Australia, in the Northern Territory, Victoria, South Australia and Queensland.

Although the overall production of crude oil has declined since the 2000s, Australia's natural gas production continues to increase. (http://www.miningoilgasjobs.com.au/oil-gas-energy/all-you-need-to-know-about-the-oil---gas-sector/overview-of-australias-oil-and-gas,-energy-industr.aspx)

The downstream petroleum industry is represented by the Australian Institute of Petroleum (AIP) which was founded in 1975. AIP established a Health Committee in the same year.

1.2. Development and Design of the Health Watch Surveillance Program

In 1980, the AIP contracted the (then) Department of Community Medicine at the University of Melbourne to establish an epidemiological health surveillance program to monitor major health outcomes of employees in the industry. The program, called *Health Watch*, has been running continuously since that time, monitoring deaths and cancer incidence in the cohort of people who work or worked in the industry. As Australia's oil and gas development expanded, new companies and projects entered the program. Entry to the existing cohort was closed in 2000, however, a new cohort comprising existing and new employees was established in 2010.

What is a cohort?

A cohort was originally a group of Roman soldiers who marched together. The Health Watch cohort is made up of people who are or have been working in the industry who are marching together through time.

In 1987, an overall excess of lympho-haematopoietic (LH) cancers (all leukaemias, multiple myeloma and all lymphomas except Hodgkin disease) was observed in the cohort. To evaluate the relationship between workplace exposures (specifically benzene) and the excess of these cancers, a nested case-control study was commenced within the cohort in 1988.
In 1999, the University of Melbourne relinquished responsibility for *Health Watch*, and the AIP contracted the University of Adelaide to continue the cohort study. Responsibility for the case-control study was passed to a consortium at Monash University and Deakin University. With the approval of the University of Adelaide's Ethics Committee, information for conduct of the case-control study was provided to the consortium.
In 2005 the AIP transferred custodianship of the *Health Watch* cohort to Monash University's Centre for Occupational and Environmental Health (MonCOEH) in the Department of Epidemiology and Preventive Medicine (DEPM). With the consent of the University of Adelaide's Ethics Committee, Monash University's Ethics Committee and of the State and Territory Cancer Registries and the Australian Institute of Health and Welfare (AIHW), the cohort data were transferred to Monash University.

Although all the major petroleum companies of the AIP joined the *Health Watch* program, participation by individual employees was voluntary. The health outcomes monitored are deaths from any cause and the incidence of cancer. These measures have provided a broad view of the health experience of people working in the participating companies over recent decades. Death and Cancer Registry data is available since 1982 and has allowed mortality and cancer incidence to be recorded and analysed.

Figure 1 is a representation of the *Health Watch* cohort structure as at 30/11/2015.

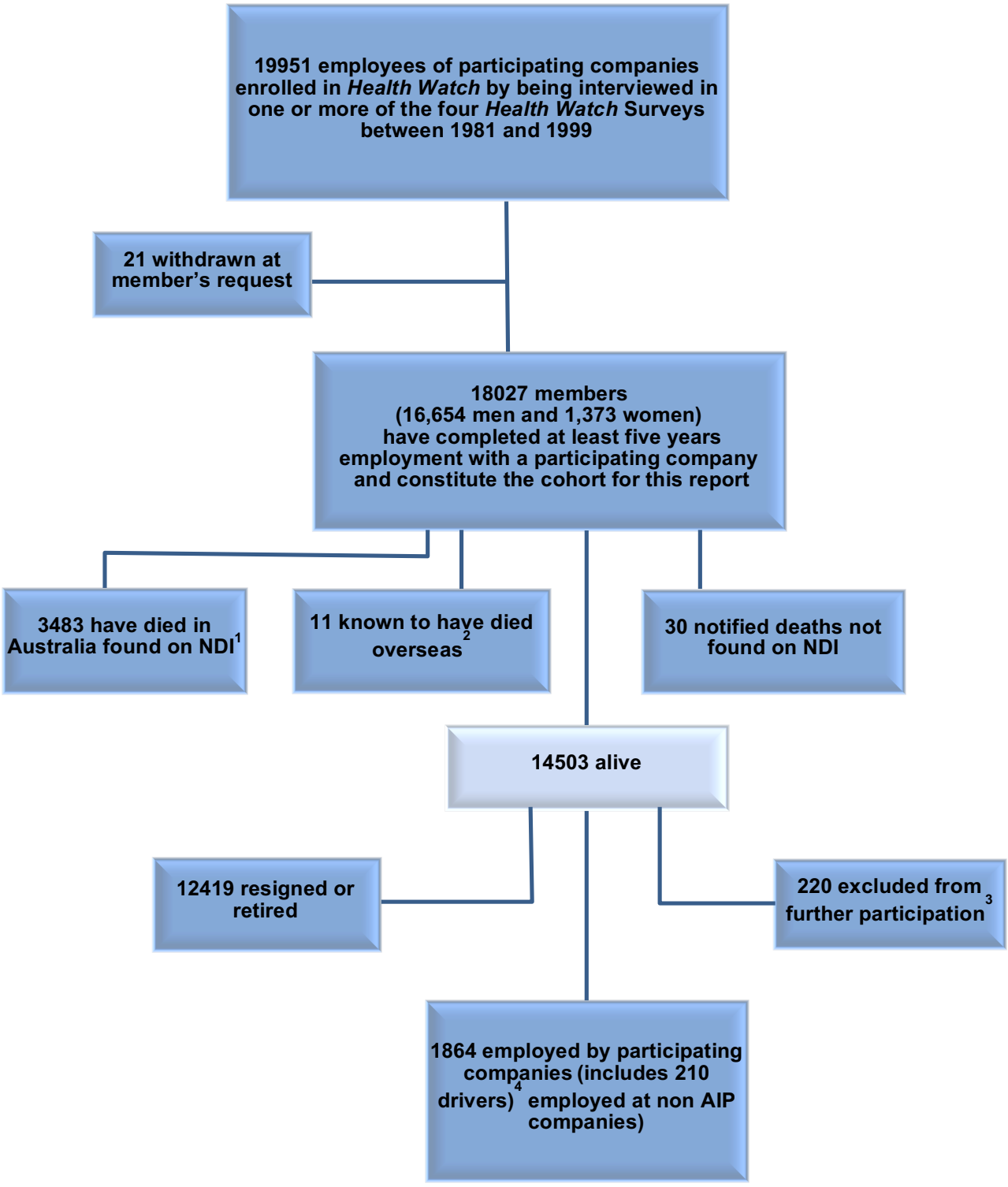

Figure 1: *Health Watch* cohort structure

1. Includes deaths from Castrol employees, however Castrol deaths after 1994 are not included in this analysis
2. One member was previously recorded as overseas death. Has now been matched to the NDI in this analysis
3. Excluded Castrol employees from 1994
4. Section 2.2.8, page 25

1.3. Reporting Results

Results are reported to the *Health Watch* Advisory Committee comprising:

- a representative of the CEO of the AIP
- representatives of petroleum companies participating in *Health Watch*
- a representative of trade unions in the industry
- representatives from the research team at Monash University.

Results are published in this report and will be summarised in leaflets provided to all *Health Watch* employees. These leaflets will also be posted to all individuals who have resigned or retired for whom a current address is available. The leaflets will be prepared by the *Health Watch* project team and will report the current findings of the study in plain language.
Results of this research program have also been published in medical and other scientific journals.(1, 2, 6, 8, 11, 13-20, 22, 24-27)

1.4. Consent and Confidentiality

All information is kept at Monash University and the results are published in such a way that no individual member of the cohort is identifiable. The guidelines for research set out in the DEPM's A Guide to Good Research Practice (30) are followed and only members of the *Health Watch* study team have access to identifiable data. Under the terms of the contract between the AIP and Monash University, all members of the team are bound by formal confidentiality agreements.
All *Health Watch* approaches to cohort members are assessed and approved by the Advisory Committee. Project team members are aware of the need to avoid distress in their dealings with individuals and their families. Medically confidential matters relating to individual members of the cohort are handled within the project by the Study Director who is a medical practitioner.
Health Watch obtains information from members of the cohort, their next-of-kin, relatives, employers, and the AIHW, which maintains the National Death Index (NDI) and the Australian Cancer Database (ACD) on behalf of state cancer registries, and the Victorian Cancer Registry (VCR).
Signed consent was obtained from members of the cohort at interview to obtain relevant information, and specifically to search the cancer registries and to approach employers for job histories. Continuity of consent was obtained at each subsequent survey. Information regarding the consent and its implications was provided to potential entrants at briefing sessions on site, in writing, and at the time of interview. A small number of employees declined to give consent: these employees are still members of the cohort but are not included in Cancer Registry searches (unless deceased).
There are about 900 pilot study members in Victoria who did not complete second or subsequent surveys and were never asked for consent for their names to be searched against the Cancer Registries. Almost all *Health Watch* participants who were asked for consent have agreed to the linkage. With the agreement of the *Health Watch* Advisory Committee, and the relevant Ethics Committees, the Victorian Cancer Registry (VCR) has agreed to continue to match these cohort members.

1.5. Ethics Committee Approval

The *Health Watch* program deals with matters relating to medical and human research ethics, informed consent, and confidentiality. The work of the *Health Watch* cohort study has been approved by the Monash University Human Research Ethics Committee (MUHREC).
In order to obtain identifiable cancer records, approval is also required from the Human Research Ethics Committee (HREC) of the AIHW, and from each of the individual state and

territory cancer registry HRECs and for some states and territories, the Chief Health Officers, Data Custodians or Health Department Privacy Committees. Fortunately, written consent was obtained from members of the cohort at the time of recruitment into *Health Watch* and although the ethics approval process is becoming increasingly complex, ethics approvals from all departments have been granted. Nevertheless, privacy laws currently present several obstacles to the efficient conduct of research, including lengthy procedures applying, reapplying, providing annual updates and responding to Ethics and Privacy Committees of state and national data repositories as each of these have their own forms and processes.

1.6. Present Work

This report is based on the work carried out in the *Health Watch* program in the period 2013-2017. The deaths occurring in the cohort prior to the cut-off date of 30th November 2015 have been ascertained as far as possible, and cohort mortality rates were compared with national rates. This was the latest date for which complete mortality data was available from the AIHW. Registration of all cancers takes longer than death registrations, so that at the time of analysis, national cancer rates were only available for comparison up to 31st December 2012 for all jurisdictions. This extends the previous report by five years in respect of mortality and four years for cancer incidence data.

Mortality data are provided to the AIHW by the Registries of Births, Deaths and Marriages (BDM) and the Australian Bureau of Statistics (ABS). These data are maintained at the AIHW in the NDI. Cancer data is provided by each of the individual state and cancer registries to the AIHW and held in the Australian Cancer Database (ACD).

2. METHODS

2.1. Study Design

The overall design of the *Health Watch* program is that of a prospective cohort study and this cohort has recently been described elsewhere. (26)

Within the cohort, a case-control study was conducted examining the association between benzene exposure and certain cancers of the blood, bone marrow and lymphatic systems known as lymphohaematopoietic cancers (LH cancers). In the past, benzene exposure of cohort members with these cancers was estimated and compared with the estimated exposure of a sample of cohort members who did not have these cancers. The comparison enabled an estimation to be made of any association between these cancers and exposure to benzene. This aspect of the study was carried out by a consortium from Monash and Deakin Universities, and was concluded in 2001. The outcome of the study and the methodology were reported to the AIP in 2001(12), and in peer reviewed literature.(10-13, 15-18) The complete report can be accessed on the AIP website (www.aip.com.au). The case-control study was updated in a collaborative study with cases from two similar overseas petroleum industry cohorts in 2012. These are the UK Institute of Petroleum study and the Canadian Imperial Oil study.(31, 32) The combined case-control study was funded by the Conservation of Clean Air and Water in Europe (CONCAWE) (The Health, Safety and Environment Office of the European Petroleum Industry) (a European refining industry body), the American Petroleum Institute, the Aromatic Producers Association, the Institute of Petroleum; and the Canadian Petroleum Products Institute. The methodology and findings have been published. (20-22, 24, 25, 27)

2.2. Formation and Maintenance of the Cohort

2.2.1. Recruitment

Recruitment to the cohort has been by participation in one or more interviews carried out in four successive surveys, with the last survey ending in 2000. A recruitment drive for a new cohort named the Re-opened cohort was also carried out in 2010-2012. Although 1,041 existing cohort members were identified in the Re-opened cohort, this cohort will be treated as a separate entity and is discussed in detail in Section 9. The following section describes the recruitment to the existing *Health Watch* cohort.

All employees of participating petroleum companies operating in Australia, who worked in refineries, storage and distribution terminals, offshore and onshore production facilities and airports were eligible to become members of the *Health Watch* cohort. Employees working in capital city offices and sites with fewer than ten employees were excluded.

Altogether, four surveys were carried out before the cohort was closed to further entry in 2000. The First *Health Watch* Survey was carried out in 1981-83. The Second *Health Watch* Survey was in 1986-87, the Third *Health Watch* Survey was conducted during 1991-93 and the Fourth *Health Watch* Survey was undertaken between 1996 and 2000. The repeated surveys allowed updating of information for each member of the cohort population still employed, and the recruitment into the cohort of any new employees in the industry since the last survey.

Site rolls were provided by the participating companies and these were used to make contact with each employee to offer them the opportunity to participate in the *Health Watch* study.

During the periodic surveys, entry to the *Health Watch* cohort register was gained through voluntary attendance on site for personal interview with a project team interviewer. Full and informed consent procedures were undertaken for each employee during pre-interview briefings to employees in groups and individually at the time of interview. The major purpose of these briefings was to explain the nature of the study, the implications of entry and the consent procedures, and to provide feedback to existing and prospective cohort participants.

For each survey, interviewers were trained in the application of the questionnaire. *Health Watch* surveys have used almost identical questionnaires and the methodologies have remained comparable, although some changes in technology have occurred. Most of the Fourth Survey was conducted by the University of Melbourne using direct input to portable notebook computers. The interviewer had access to all the previous job history of current members and could accept potential corrections to previous data.
Not all sites were visited for the Fourth Survey, (offshore sites were not included), and further questionnaire responses were obtained by mail and by telephone.

2.2.2. Entry to the Cohort

Petroleum industry employees were admitted to the cohort after completing a survey interview or upon completion of five years of employment with a participating company, whichever was later. Thus, members of the cohort who had already completed five years of employment at the time of their first interview were admitted to the cohort immediately.

2.2.3. Information Collected at Survey Interviews

Demographic information collected at interview included name, gender, date of birth and country of birth.
Employment information was obtained in some detail, in particular, members of the cohort were asked their occupation, the area they worked in, the tasks they performed and the proportion of the working week spent in each area.
During the first two surveys, details were collected by interview on the current job held by each participant. Participants were also asked to identify jobs held for up to five years prior to their first interview. By 1990 it was apparent that more complete job histories were required for the purpose of a nested case-control study of LH cancers within the cohort. Accordingly, in 1991-93 during the Third Survey, all participants were asked at interview about all jobs held during their employment in the petroleum industry. The complete job histories were collected from nearly all current employees who participated. In a few cases, where complete employment histories were not obtained, or later proved to be incomplete, the computerisation of the Fourth Survey allowed gaps in the information to be more easily identified and corrections to be made at the time of re-interview. By this time about 4,000 men and 250 women had retired or resigned from the industry after having worked for at least five years. Their complete job histories had to be collected by including questions relating to this in the periodic Health Letter sent to all retirees, which was undertaken in 1994-95. Retirees were generally longer serving employees than those still employed, and therefore had longer gaps in their previously collected job histories. For many reasons, their complete job histories are likely to be less accurate than those still employed and interviewed in the Third Survey. The response rate from retirees to requests for complete job histories was about 80%. Some job history information for deceased members was completed by surviving partners or family. These more complete job histories were used to assess benzene exposures in the case-control study rather than the more limited information obtained from the survey.
Lifestyle information was also obtained at interview. Standard questions on present and past smoking habits were asked of each participant, and a lifetime smoking history was recorded.
Information on alcohol consumption was also collected during the survey interview. Each cohort member interviewed was asked: "In an average week, on how many days would you have a drink?" and "How many drinks would you usually have on those days?" A drink is defined as a standard measure as served in a hotel or bar. The average number of drinks taken weekly can then be estimated.
Health information, related to current or significant past health problems, was also collected.

2.2.4. Information from Participating Companies

Participating companies have periodically provided lists of new employees, transfers, resignations and retirements. Following the decision to close the cohort to further entry in 2000, companies have continued to provide lists of transfers, resignations and retirements on request. This information from companies is used to compute the date of termination of employment of all members of the cohort and vital status for all current employees. In some cases, addresses have been provided to update the contact details used for the periodic Health Letter (Section 2.2.7, page 25).

2.2.5. Coding of Employment Data for Assigning Estimates of Hydrocarbon

Direct measurements of exposure to hydrocarbons for particular jobs, e.g., in "parts per million in air, time weighted average" were generally unavailable for the several decades of interest to *Health Watch*. In the absence of such information from companies, estimates of exposure have previously been derived from the job details provided at the survey interviews. More details of the coding were included as Appendix 1 to the 9th *Health Watch* report.(4)

A precise job description code was used as the principal exposure index for the cohort analyses, based on collection of a job history from each participant. The job classification developed for the American Petroleum Institute (API) was used, with modifications on the advice of local occupational hygienists in the Australian industry (33, 34).

This ranking for exposure to hydrocarbons was used in the first case-control analysis. Distribution of *Health Watch* person-years across these categories was unequal, with many jobs being placed in the default category in the middle of the range. A more rigorous, quantitative methodology for assessing benzene exposure was developed for the *Health Watch* case-control study. However, the assessment was applied to only the cases and controls in the case-control and pooled studies and was not applied to the cohort as a whole.

The hydrocarbon ranking methodology was established in the early 1980s. During 1994-96 the rankings were revised to take account of changes to workplaces and exposures. The categorisation and groupings are however, now out of date. For example there has been a significant move towards bottom loading of tankers in recent years significantly reducing the exposure to hydrocarbons, perhaps a third of that experienced during top loading.(11)

Therefore, as agreed in a meeting of the HWAC in 2007, the overall hydrocarbon ranking has not been revised and was considered to be out of date and was not used in this or the previous report.

Monash has now developed a list of generic job classifications or titles, which meets the current job descriptions, as a more suitable replacement for the previous hydrocarbon ranking scheme. This classification is used in the re-opened *Health Watch* cohort.

2.2.6. Participation Rates in *Health Watch* Surveys

For the first two surveys, a record was kept of the proportion of employees interviewed. In both surveys, approximately 93% of employees on the site rolls agreed to participate.

It is estimated that 84% of eligible employees were interviewed in the Third Survey.(7)

In the Fourth Survey, not all worksites were visited. Further contact was made by mailing out questionnaires and by telephone, but the response rate was not as high as that obtained by on-site interview. Seventy three percent of *Health Watch* members still employed were re-interviewed in the Fourth Survey, and an additional 1,479 new employees were interviewed. A previous report identified that the incompleteness of the Fourth Survey was largely a result of lack of recruitment of offshore production workers.(7)

In 2003, a Fifth Survey was carried out by the University of Adelaide. This was a questionnaire that was sent to all members of *Health Watch* still employed by participating companies. There

was a 40% response rate and more details of this are provided in the 12th Report.(7) In addition, members of *Health Watch* who were no longer employed by participating companies were sent a reply-paid questionnaire with their 2002 letter. There was a 62% response rate from these cohort members.(7)

2.2.7. Follow-up

Efforts are made to maintain contact with all *Health Watch* cohort members. Retired members are sent periodic Health Letters where they are asked to report changes of address, illnesses and changes in smoking or drinking habits. This is important when updating personal information; in particular it is one of the main means of determining the vital status of cohort members.

A member is considered *Lost to Contact* for whom reliable contact data is not available. If the periodic Health Letter to a *Health Watch* member is unanswered or is 'returned to sender', contact with the employing company does not provide a recent address and a search of the White Pages fails to find a telephone number on which the person can be contacted, the person is considered "lost to contact". The cohort member continues to be included in the cohort analyses however and this status just refers to not having a means of contact.

Following an extensive search by a commercial organisation, Adelaide University located most individuals in the cohort who had been previously classified as lost to contact. Adelaide University estimated in 2003, that 4% of the cohort was lost to contact and the percentage loss of observation time from loss of contact was only 1.3% in men and 4.9% in women.(7) The Department of Immigration and Multicultural and Indigenous Affairs[1] (DIMIA) records identified an individual's last departure or arrival in Australia. For this report, the DIMIA records provided to Adelaide University were reviewed and those who left before 2000 and had not returned by 2003 were assumed to have emigrated.

Most recently, Monash University sent a brief summary of the latest findings in the 14th *Health Watch* report to all members of the cohort with a Health Letter in 2014. This was sent to the entire cohort, either to their place of employment or to a home/postal address if the employee was retired/resigned. There was no attempt to locate those members who did not respond to the 2014 Health Letter. Of the 14,500 letters sent to eligible members of the original cohort, 5,000 (34%) returned the mini survey. It should be noted that non-response does not equate to lost to follow up. Following discussion with the *Health Watch* Advisory Committee it was decided not to attempt another commercial search because of concerns about the privacy legislation.

2.2.8. The Special Case of Drivers

In recent years, most participating companies have outsourced tanker drivers' duties to transport contractors. Many tanker drivers who formerly worked for participating companies now work for contractors but perform identical duties to those previously performed. Although the contractors are not members of the AIP, it has been possible to identify many such tanker drivers from their responses to the Health Questionnaires and Health Letters. In such cases, the drivers have been classified as still "employed" and are included in the analyses.

Numerous other *Health Watch* members have continued to work in the petroleum industry for non-member companies of the AIP. They perform a variety of tasks not readily assigned to

[1] Now The Department of Immigration and Border Protection

the API job coding system, so their non-AIP jobs have not been included in the duration of employment.

2.3. Withdrawal of Members of the Cohort

Twenty-one cohort members have indicated that they wish to withdraw from the study and do not wish to participate further in *Health Watch*. Their follow-up time has been excluded retrospectively (i.e. back to their enrolment), and they have been excluded from the analyses presented here. With the permission of MUHREC, if a person asks to be withdrawn from the cohort, they are sent the necessary formal notice of withdrawal to sign and also a letter explaining that withdrawal means that past contributions are deleted. They are also offered the option of remaining in the cohort, but with no further communication from *Health Watch*.

2.4. Health Outcomes

Health Watch cohort members have provided information on their health in the successive *Health Watch* surveys and in questionnaires sent to members of the cohort who are no longer employed by participating companies. The only health outcomes that have been statistically analysed are cause-specific death rates and cancer incidence rates. These analyses are not based on information supplied by members of the cohort, but from the national mortality and cancer records. However, health information supplied by members of the cohort has proved useful when verifying matches with official records. For example, if there is uncertainty as to whether a name appearing in a death linkage is the same individual as a person with the same name in the *Health Watch* cohort, identification is assisted if the person has notified *Health Watch* of a corresponding illness, or a next of kin has notified of a death. Cancer matches are now clerically reviewed by the AIHW and VCR and only highly certain matches are returned.

2.4.1. Mortality Records

Consideration of all causes of death can provide a broad picture of major health patterns, as these are directly linked to death outcomes. Some medical conditions, where death is not a consequence, e.g. osteoarthritis, cannot be analysed by *Health Watch*, because there is no population registry of these types of diseases. Others, where there is a link between number of deaths and overall morbidity (ill-health), such as ischaemic heart disease (i.e. coronary artery disease) and accidents, can be reliably explored using *Health Watch* information.

For the purpose of mortality analyses, death records are obtained from the NDI, maintained by the AIHW. The NDI is compiled from death records from state registries of Births, Deaths and Marriages, and causes of death, coded by The Australian Bureau of Statistics (ABS). ICD-9 coding was used for deaths occurring up to and including 1996.(35) Deaths occurring from 1997 onwards are coded in ICD-10.(36) The coded deaths by cause are used to compile national annual cause of death statistics (mortality rates).

Periodic searches are made of the NDI by submitting the list of *Health Watch* members, with dates of birth, to the AIHW, which uses a matching algorithm to identify likely and possible matches. These are supplied to *Health Watch* for clerical review and decisions are made on which names on the list are to be accepted as true matches. Matching is sometimes made difficult because many older state death certificates provide only the age (in years) of the person at death, rather than the date of birth. Final decisions on doubtful matches are based on information already held by *Health Watch*, such as notification of death from next of kin or from companies. Previously, it was sometimes necessary to obtain a copy of the death certificate, where certain items of information (e.g. occupation or place of birth) can be compared with information held by *Health Watch*. Changes to the privacy landscape have made it very difficult for anyone other than the next of kin to obtain a copy of the death

certificate. In addition, registration data is now usually more precise so this confirmatory step is no longer required.
The coded deaths identified as true matches are used for comparison with Australian mortality data statistics resulting in the calculation of a comparative index called the Standardised Mortality Ratio (SMR) (Section 2.5.2, page 30). These analyses are undertaken to the time when the NDI is considered to be complete. At the time of this analysis the NDI is considered to have a complete record of all deaths with the cause of death coded up to 30/11/2015, which has therefore been determined as the cut-off date for the mortality analyses included in this report.

2.4.2. Validation of Mortality Records

As discussed in the previous section, mortality analysis is carried out by comparing death rates in the *Health Watch* cohort with national rates. For such an analysis to be valid, the data sets must be comparable. To ensure that mortality ascertainment is complete, it is necessary to ensure that, as best as possible, all deaths known to *Health Watch* are located on the NDI.
Health Watch does not rely solely on NDI linkages to learn of deaths. Notification of deaths in currently employed members of the *Health Watch* cohort may be supplied by the employer. Deaths of members who have left the industry may be notified by next of kin in response to a mail contact or voluntarily via the *Health Watch* freecall number or email address. There were 1094 deaths among eligible *Health Watch* members which have been notified in this manner. Most of them have been subsequently identified through the NDI search, but some have not. *Health Watch* has been notified of 30 members who died before 2015, but who could not be found on the NDI. A further 11 members have died overseas. These deaths have not been included in the analyses because they do not appear on the NDI and therefore cannot be confirmed with a cause of death, but follow up ceases as of the notified death date. One member who was previously reported as an overseas death has been matched to the NDI in this analysis.
Deaths identified from the NDI occurring after the cut-off date of 30/11/2015 have not been included in the analyses.
It is not possible to be certain that all deceased members of the cohort are identified in the matching process. For example, when members of the cohort have changed their name (e.g. women after marriage or divorce) it is possible that their deaths may not be detected. In addition, members who die overseas e.g. after emigration, will not appear on the NDI. Therefore, absence of a person's name from the NDI does not necessarily mean that he/she is alive. As discussed in Section 2.5.1 (page 29), the follow-up time of such members cease from the date of emigration for those who died overseas or the cut-off date 30/11/2015.
In all previous linkages, except the 14th *Health Watch* analysis, all matches from preceding NDI searches were accepted and added to the new matches found from the additional years that were searched in the new linkage. The current linkage covers the entire follow up time of the cohort, not just the additional years of follow up since the last report. This means the entire linkage is repeated and any differences between previous linkages are cross checked to see if any matches have been missed.
Prior to the 2014 analysis, deaths were re-coded at Adelaide or Melbourne University based on individual death certificates. Some data was also coded from ICD10 back to ICD9. In some cases, these manually re-coded deaths did not match what was recorded on the NDI. For this report, only cause of death data obtained from the current NDI linkage has been used to ensure it is consistent with the national population data in these analyses.

2.4.3. Cancer Incidence

A distinguishing feature of the *Health Watch* study, from most cohort studies in the petroleum industry around the world (or indeed any industry), is its ability to consider the occurrence or incidence of cancer which is not necessarily fatal. This is made possible by the existence of population-based cancer registries in all Australian states. Cancer is a notifiable disease in all states and territories and all cancers, except non-melanoma skin cancer, and all deaths are legally notifiable in Australia. Cancer registration has been universal in Australia and represents a complete registry of all cancers since 1982. Written consent has been obtained from most members of the cohort to search for their names on the cancer registries.

Until the mid-1990s, *Health Watch* obtained information on cancer incidence by submitting the names and dates of birth of all members of the cohort to each individual state cancer registry. Since then, matching has been achieved by matching with the ACD, a compilation of data from all state and territory registries from which national cancer incidence data is generated. Additional information from Victoria is obtained directly from the VCR. Only confirmed matches were supplied to *Health Watch* from the ACD and VCR due to privacy restrictions. The uncertain but possible matches were reviewed by the cancer registry and only highly certain matches are released to *Health Watch*.

The analysis is undertaken to the latest time at which the ACD is considered to be complete. In this report, the cut-off date is 31st December 2012, which is three years prior to the available death data, because complete enumeration of cancers takes longer than enumeration of deaths. Cancer cases are grouped into various categories based on the ICD-10 coding system.(36) These categories range from malignancies which include all types of cancers, to broad organ system groupings which contain a range of cancer codes, such as respiratory (C30-C38), or digestive system (C15-C25). Cancers are also categorised into more specific single site sub-categories such as stomach (C16) or bladder (C67). Non-melanoma skin cancers are not generally recorded by the state cancer registries, so for the analyses comparing skin cancer rates in the *Health Watch* cohort with national rates, only melanomas are included.

Data on cancer deaths have been available for many years and is obtained from information on medical certificates of cause of death. However, the major question for studies of the effects of occupational (or other) exposure is how many people actually develop cancer, which is not the same as how many people die from cancer. Cancer death rates relate not just to cancer occurrence, but also to other factors such as the presence of effective treatments, access to health care and presence of co-morbid conditions, which are not directly relevant to the aims of the *Health Watch* study. Since the death rate from cancer is so strongly related to the effectiveness of treatment and other factors, survival rates are now high for many types of cancer. Cancer incidence (i.e. the occurrence of cancer) is therefore a more reliable measure of the cancer rate than deaths from cancer and this outcome is used in the analyses in this report. Cancer mortality figures are also quoted where appropriate.

2.4.4. Validation of Cancer Incidence

Only cancer matches found in the current linkage were used in this analysis. Previous matches that were not matched this time were not included, but any differences in matches between linkages were cross checked. Cancers can be re-coded by the registries to other sites over time (especially those initially coded as "unknown") and can also change between being malignant and non-malignant and vice-versa. Therefore, it is preferable to refresh the entire list of matched cancers in each linkage. This ensures that the cohort cancers can be confidently compared to cancers in the general population. It is, however, possible that numbers in each cancer category may change slightly in each analysis. This difference is insignificant and is unlikely to alter the results.

Another source of cancer notification is death registrations where cancer is given as the cause of death. There were an additional 20 members identified with cancer based on a death notification, but not identified in the cancer registry linkage. Some of these missing cancers can be explained in earlier years as they might have been diagnosed before the commencement of the cancer registries with the resultant death occurring several years later.

Current Cancer and Death Linkage and revised Job Groups
This report analyses deaths occurring up to 30th November 2015, and cancers registered up to 31st December 2012. The current linkage covers the entire time span of the cohort, not just the additional years of follow up since the last report. This provides a more accurate account of the status of the cohort.
The job groups in this report are Drivers, Refinery operators (not including ship personnel), Terminal operators and Maintenance (refinery or terminal based not upstream) and Office workers. Shift work, which was a category used in previous reports, was a self-assessed category and employees in this group also belong in other job groups including operators and maintenance workers. The accuracy of this group is no longer relevant and has been removed from this analysis.

2.5. *Statistical Analyses*

The analyses undertaken for this report was completed in conjunction with MonCOEH and the Statistical Consulting Centre, University of Melbourne using an updated version of the SAS program developed at Adelaide University.
The basic analyses in *Health Watch* are to compare the death and cancer incidence rates of the *Health Watch* cohort with the corresponding rates in the general population by sex and by five-year age groups. The rates are expressed as the number of deaths or new cancers as a proportion of the person-time of follow-up.
The total person-time is the cumulative total of the follow-up time of each individual. For example, if 20 people are each followed up for 10 years, the total person-time would be 200 person-years. If two cancers occurred in these 10 people over that time, the cancer rate would be 2 per 200 person-years.

2.5.1. Follow-up Time

The definition of cohort members' follow-up time (usually expressed in person-years) is critical. Follow-up time commences on admission to the cohort (Section 2.2.2, page 22), which is the date of initial survey interview, or on completion of five years of employment in the industry, whichever is the later.
Follow-up time stops on the date of death or the cut-off date (30/11/2015 for mortality, 31/12/2012 for cancer) or the date of emigration, whichever occurs sooner. Members of the cohort who have emigrated cease to be followed up after leaving Australia. This is because if they die or develop cancer whilst outside Australia, their death or cancer would not appear in the data on which national death and cancer tables are based. Since such deaths or cancers will not be included in the analyses, the person's corresponding follow-up time is excluded from the denominator.
It will be seen that there are two categories of members of the cohort lost to contact:

(i) Those who will be found on the NDI when they die
(ii) Those who will not be found on the NDI when they die.

For example, a person with whom *Health Watch* has lost contact may be living in Australia and be found in the NDI when he or she dies. On the other hand, a person who has emigrated

and remains overseas will never appear in the NDI, and a person who has changed his or her name may be on the NDI under the changed name, and a match with the NDI may never be found.
In most previous *Health Watch* reports, members of the cohort not found to be deceased or not known to have emigrated were treated as living, and all had their follow-up time extended to the cut-off date for the analyses. The inclusion of these people in this way could result in slight over-enumeration of person years, leading to a slight underestimate of the mortality (or cancer incidence) rate relative to the national rate. As discussed in previous *Health Watch* reports, individual cohort participants who were previously considered to be lost to follow up have actually been identified in later matches with the NDI and ACD. It was not therefore considered appropriate to remove these individuals from the cohort follow up.
Adelaide University showed that whether or not the follow-up time of members of the cohort who are lost to contact is or is not included makes very little difference to the result – in men, the estimate varied by 0.01 (i.e. 1%) and in women by 0.02 (2%).(7)

For all analyses in this report the follow up date is up to:

- 30/11/2015 for mortality and 31/12/2012 for cancer
- Date of emigration
- Date of death if found on NDI
- Date of death notification if not found on NDI, but a death has been reported.

2.5.2. External Measures of Comparison: the SMR and SIR

Health Watch compares death and cancer incidence rates in the petroleum industry with the national rates to produce measures called the standardised mortality ratio (SMR) and the standardised incidence ratio (SIR) respectively.
The SMR is a measure of the death rate occurring in the *Health Watch* cohort compared with the death rate occurring in the national population. This ratio can be measured for the whole cohort or any subset of workers, for any particular cause of death, or for all causes. The SMR tabulations show the number of deaths observed in the *Health Watch* population and the calculated expected number which would arise in a group of the same age and sex in the Australian national population.
The expected number is computed from the national rates (by age, sex and year of occurrence) provided by the AIHW (37) and the number of person-years spent by cohort members in each age, sex and year-of-occurrence stratum. Comparison of the observed number of deaths recorded by *Health Watch* to the expected number as shown in the tables produces the SMR. If the deaths in the *Health Watch* cohort are occurring at the same rate as they do in the national population, then the SMR will be 1.0. If the SMR is greater than 1.0 then deaths in the cohort are occurring more frequently than would be expected if national death rates applied to the *Health Watch* population. If the SMR is less than 1.0 then deaths in the cohort are occurring less frequently than they do in the national population. Thus, the SMR forms a measure of the risk of mortality in the *Health Watch* cohort compared to the Australian population as a whole, with age and sex taken into account.
When measuring the risk of developing cancer, the standardised incidence ratio (SIR) is calculated. Incidence measures cancer as it arises, as opposed to when it causes death. All cases of cancer except non-melanoma skin cancers are reported to the relevant state cancer registries by the treating medical specialist. Providing that cancer registration is reliable, as it is in Australia, cancer incidence is a more valid indicator of cancer risk than is cancer mortality.
The SIR is calculated in a similar way to the SMR. To calculate SIRs, calculation of expected

numbers from national cancer incidence is required. The national data are derived from the ACD.(38)
Both SMRs and SIRs are age-standardised because death and cancer rates vary strongly with age. They also vary with sex so rates are calculated separately for men and women.

2.5.3. Internal Measures of Comparison: the RMR and RIR

Health Watch also uses internal comparisons to look at the health effects of working in the petroleum industry. Relative risk is used to examine whether members of a subgroup such as specific job categories, have more or less risk of death or cancer than other subgroups. For any particular exposure or subgroup, a baseline group is chosen, and represented as having a risk of 1.0. All other exposure groups or ranks are then calculated for risk in comparison with the baseline group. The measures of these comparisons are the relative mortality ratio (RMR) when death is the outcome or relative incidence ratio (RIR) when cancer is the outcome.
Where a measure or ranking of exposure can be obtained, a relative risk of death or cancer can be calculated, comparing those who have less exposure to those who have more. Generally, it would be expected that if the exposure is causing the effect, then those with more exposure, in time or intensity, would suffer more effects on their health, and this would show up in the health outcomes. This is known to apply, for example, to the number of cigarettes smoked and the risk of lung cancer.
The reference category for these analyses is usually the least exposed group. For smoking it is people who have never smoked. If the RMR or RIR for any group in *Health Watch* is 1.0 then deaths or cancers, respectively, are occurring at the same rate as they do in the reference group. If the RMR or RIR is greater than 1.0 then deaths or cancers are occurring more frequently than they do in the reference group. If the RMR or RIR is less than 1.0 then deaths or cancers are occurring less frequently than they do in the reference group.

2.5.4. Confidence Intervals and Risk Estimates

The rate ratios, SMR, SIR, RMR and RIR, are accompanied by 95% confidence intervals (95% C.I.). The value attributed to the ratio is actually a statistical estimate of the true ratio, however, the true ratio cannot be known exactly. The spread of estimates of the ratios within which it is 95% certain that the true figure will lie can be calculated. This spread is called the confidence interval.
The choice of 95% confidence intervals is commonly used in health studies, and simply means that the certainty of the result is such that the odds of the true figure lying outside the confidence interval are about 5% or 1 in 20.
Confidence intervals are influenced by the size of the group however. Two groups may have the same point estimate of risk, but the larger group will have narrower confidence intervals. This may mean that larger groups such as onshore production workers have statistically significant results but the same point estimates for smaller groups such as offshore production workers that may not be statistically significant, as in Table 24 . In addition, as deaths or cancer cases accumulate in the cohort and the general population, increased or decreased risk estimates may reach statistical significance.
The importance of this lies in the interpretation of the ratios in terms of risk appraisal. Where a ratio is higher than 1.0 then a risk may be present, but if the lower end of the confidence interval extends below 1.0, then it is possible that the real ratio is 1.0 or less and no risk is present. However, when the lower end of a confidence interval is above 1.0 then we can say with some certainty that an increased risk does exist. This is often described as being a statistically significant result. If both confidence intervals are below 1 then the risk is said to be statistically significantly lower than that of the reference group.

2.5.5. The Problem of Multiple Analyses

In this report, the convention of 95% probability is used to interpret risk estimates. This convention accepts that there is a 1 in 20 chance that an increased or decreased risk has happened by chance. When multiple comparisons are carried out, as in this report, it is possible that some chance findings may be found to be statistically significant. It is important therefore not to accept or dismiss a finding based on a single risk estimate but to interpret the risk estimate in the context of the body of information in this and previous reports and the findings from other studies.(39-43) In this report the risk estimates are discussed with reference to those from previous reports, to aid interpretation.

2.5.6. Confounding Variables

Confounding variables are factors (other than those under investigation) which may affect the cohort health outcomes being studied. Where these factors can have large influences on outcomes, such as with smoking and cancer, it is necessary to account for them in the analyses. Even small differences in exposure to tobacco smoke can cause large differences in lung cancer rates. To cause confounding, a variable has to be a cause of the disease in its own right, and to be unequally distributed between the different groups being compared and not measured or are unable to be adjusted for in the analyses.
Differences in risk between various exposure groups could therefore be masked or falsely calculated if confounding variables are not allowed for.

What is a Confounder?

A confounder is a term used in epidemiological studies in which a group with a particular exposure history is compared with a group without the exposure. In such studies, the presence of a confounder can lead to a misleading result.

To cause confounding, a variable has to be a cause of the disease in its own right, and to be unequally distributed between the different groups being compared.

For example, the lung cancer rate in a group of workers exposed to a carcinogen (say asbestos) may be compared with the rate in a group of workers not exposed. Since smoking can cause lung cancer, smoking prevalence is a potential confounder in this analysis. If the group exposed to asbestos happens to have a higher proportion of smokers than the comparison group, an excess of lung cancer in the former may be incorrectly attributed to the asbestos, whereas it may be partly or wholly due to the difference in smoking. In such a case, the variable "smoking prevalence" is a confounder, where smoking rates are unknown.

2.5.7. Adjustments to SMR and SIR analyses

In the SMR and SIR estimates, adjustment was made for age and calendar year. These variables were used as they are known to have major effects in the Australian population mortality and cancer rates. For example, in the case of calendar year, the incidence rate and mortality rates of many cancers have undergone marked changes over the period since *Health Watch* began. Confounding by sex is avoided by separate analyses of men and women.
Adjustment for tobacco smoking is more difficult. Although *Health Watch* has obtained good smoking data on members of the cohort, comparable data are not readily available for the

general population. Data on smoking prevalence in the Australian population by age group in the mid-1990s are available,(44) and by comparing this with the smoking prevalence of the *Health Watch* cohort, and by using information on the strength of any association between smoking and a particular disease, it is possible to estimate the likelihood that a particular outcome is smoking-related. Another indirect method of estimating whether the smoking prevalence differs from the general population is to examine the cancer rate or death rate from diseases almost exclusively due to smoking, such as emphysema and laryngeal cancer.

2.5.8. Adjustments to RMR and RIR analyses

Because disease rates vary with age and over time in the population, the age at death or cancer diagnosis and the calendar period must be taken into account in the analyses. All RMR and RIR analyses were adjusted for age and calendar period of follow-up and smoking status.
The confounding effect of smoking is more readily dealt with in RMR/RIR analyses, since unlike SMR/SIR analyses; no reference to national smoking rates is required. Therefore, direct adjustment has been made for the confounding effect of smoking in estimating relative rates for all cancers and mortality comparisons. For the purposes of these adjustments, smokers are categorised into two categories – *ever smoked* vs *never smoked*.

2.5.9. Time-related Variables

Analyses were carried out using three time-related variables which assist in examining any occupational cause for excess death rates or cancer rates. These were:

- period of first employment in the industry
- duration of employment in the industry
- time from first employment in the industry.

Period of first employment analyses may provide evidence as to whether exposures in particular calendar periods may have had risks attached to them. Because technology and work procedures, and therefore exposure, have been constantly changing in the industry over the past decades, and over the follow up time of the cohort, health outcomes must be explored to ascertain whether they are related to historical exposures or reflect current risks. If hazardous exposures were present in higher concentrations in the 1970s than in the 1990s, and if some cancer types occurred at a higher rate in the former period, it could be inferred that the exposure may be a cause of that type of cancer.
The analyses for duration of employment in the industry help to investigate whether an excess death or cancer rate may be work-related, even though the specific causal agent in the workplace is not known. The total or cumulative exposure will increase with increasing duration of employment. Therefore, if the workforce is divided into different categories according to duration of employment, the death or disease rate will increase with increasing employment duration if it is related to an exposure at work. Where a person leaves and later returns to work with a participating company, the time away is deducted from the total duration.
Consideration of elapsed time from first employment to diagnosis of cancer or death is an attempt to explore what latency periods might be involved with the development of disease, particularly cancer.
Employment here refers to employment with one of the participating companies (or a contracting company in the case of tanker drivers who were formerly employed by a participating AIP company). The date of commencement was obtained from members of the cohort in the survey interviews. Termination dates are obtained from companies. Duration of employment should not be confused with follow-up time, which continues after members of the cohort cease working for participating companies.

2.5.10. Analysis by Workplace Type

In addition to comparing the overall *Health Watch* cohort with national rates, separate analyses have been performed on different categories of workplace. There are five types of workplace in this report: Refinery, Terminal, Airport, Onshore production and Offshore production. Where a subject has worked in more than one workplace type, he or she is assigned to the workplace worked most recently.

2.5.11. Analysis by Self-Reported Smoking and Drinking Status

Lifestyle choices affect health outcomes, so mortality and cancer incidence have been analysed according to the self-reported rates of smoking and alcohol consumption.

2.5.12. Analysis by Job Type

Analysis of health outcomes for specific categories of job (single Job code) is dependent on there being sufficient members of the cohort who carry out this particular job. The largest groups of employees in the industry are driver, refinery operator, terminal operator, maintenance (terminal and refinery combined) and office (clerical and managerial) and shift worker. The latter three categories are composite groups brought together to allow for job type analyses and have been used in previous *Health Watch* reports.
The job groups formed from the API job codes were revised in the last *Health Watch* report (14th) mainly to remove overlaps and ships' personnel. The revised job groups are also used in this report with the exception of shift workers. Shift workers was a self-assessed category drawn from the questionnaires and employees in this group also belong in other job groups including operators and maintenance workers. This category has also been removed from this analysis due to the lack of consistency between jobs and companies. Accordingly, with the exception of drivers, whose coding has not changed, it is not appropriate to make direct comparisons between the risks for these job groups in this report and the groups in reports prior to the 14th *Health Watch* report. The API job codes grouped in this report are:

- *Drivers* (NB295x)
- *Refinery operators* (not including ship personnel) (BA, BB, BC, HX, IB, PA, PB, PC, RF)
- *Terminal operators* (BA, BB, BC, HX, IB, NA, PA, PC, RF)
- *Maintenance* (refinery or terminal based not upstream) (IB, CA, CB, CC, CD, CE, CF, DA, DB, DC, DD, EA, EB, EC, FA, FB, GX, MX)
- *Office workers* (AX)

3. GENERAL RESULTS FOR THE COHORT

Results are reported for cause of death (mortality) and cancer incidence, for men and women in the cohort. Because of the small number of women, analyses cannot be reliably done to the same level of detail as for men.
The results come from analyses of various occupational factors and categories, smoking, and alcohol, using age-standardised population rates. Diseases and cancer types are identified by standard ICD-10 coding.(36) All statistically significant values are highlighted in bold text throughout the tables. When cancer or mortality counts are less than three, the observed numbers are not reported. This is to keep in line with confidentiality practices and reducing the risk for potentially identifying a cohort member.

3.1. The Cohort Population

3.1.1. Description of Cohort Population at 30th November 2015

There are 16,654 men and 1,373 women in the *Health Watch* cohort population included in the current analyses being those who meet the eligibility criteria for this study. These numbers reflect the preponderance of men employed in the industry. The state of the cohort as at 30/11/2015 is shown in Table 1.

Table 1: State of the cohort as at 30/11/2015

	Men	Women	Total
Died in Australia found on NDI[a]	3,372	111	3,483
Death notifications not found on NDI	28	2	30
Still employed	1,771	93	1,864
Excluded from further participation[b]	160	60	220
Retired from industry	11,312	1,107	12,419
(Of which emigrated[c])	14	3	17
Overseas Deaths before 30/11/2004[d]	11	0	11
Total	16,654	1,373	18,027

a. Includes deaths from Castrol employees, however the Castrol deaths are not included in the analysis.
b. Exclusion due to withdrawal of one company from AIP.
c. Follow-up time of members of the cohort known to have emigrated as of 2001 or known to be resident in New Zealand ceased from estimated departure date.
d. Overseas deaths and death notifications not found on the NDI are not included in the estimates of mortality rates.

The age of the cohort is detailed in Table 2, which shows the distribution of year of birth of the cohort. Although the cohort was closed to recruitment in 2000, an additional 26 men from the last report have reached their five-year qualifying period since the cut-off dates of this analysis because of updates to their employment history. There has been one withdrawal since the last report.
The number of new entrants is now very small in relation to the whole cohort. This means that the cohort is aging as a whole. This factor strongly influences the death rate from most non-infectious diseases, as well as increasing the incidence (rate of occurrence in the population) of many cancers, e.g. prostate cancer. However, when estimates are made of the risk of death or disease from any particular cause in the *Health Watch* population compared with the risk in the general population, allowance is made for the increasing age of the *Health Watch* cohort by comparisons to the rates in the general population with the same age distribution.

Table 2: Distribution of year of birth for *Health Watch* cohort members

Year of Birth	Men		Women		Total	
	N	%	N	%	N	%
1900-1919	78	0.5	1	0.1	79	0.4
1920-1929	1,674	10.1	52	3.8	1,726	9.6
1930-1939	2,875	17.3	144	10.5	3,019	16.7
1940-1949	4,834	29.0	270	19.7	5,104	28.3
1950-1959	4,838	29.1	399	29.1	5,237	29.1
1960-1969	2,106	12.6	396	28.8	2,502	13.9
1970-1979	249	1.5	111	8.1	360	2.0
Total	16,654	100.0	1,373	100.0	18,027	100.0

3.1.2. Person-years of Observation in the Cohort

With each succeeding calendar year, the number of years of observation increases for each surviving member of the cohort population. Each subject completes a person-year of observation for each year since entry into the cohort until death (or emigration). The number of person-years of observation of the cohort is the sum of the person-years contributed by each cohort member. *Health Watch* has now accumulated 444,103 person-years of observation in men and 32,864 person-years in women. The accumulation of person-years by calendar period is shown in Table 3.

Table 3: Person-years of observation

Sex	Number of Cohort members	1981-1985	1986-1990	1991-1995	1996-2000	2001-2005	2006-2010	2011-2015	Total
Men	16,654	29,372	56,650	68,949	74,950	75,177	71,970	67,035	444,103
Women	1,373	1,333	3,026	4,403	5,588	6,283	6,230	6,002	32,864

3.2. Person-years and Mortality and Cancer Incidence Rates

For this report, only 10% of the cohort is classified as employed in an AIP participating company (including contract drivers).

For all analyses in this report (as in most previous reports) only one method of estimating SMR (and SIRs for cancer) was used. The follow-up time of members of the cohort lost to contact was included until the cut-off date of 30/11/2015 for mortality and 31/12/2012 for cancer incidence. Estimates of SMR and SIR could therefore be underestimated because some of those lost to contact could be dead, but the error is most likely to be negligible.

Deaths have occurred in the cohort since the cut-off date of 2015; however, since comparison data were not available for this period, they have not been included in these analyses. They will be included in future analyses as the comparative data becomes available.

3.3. All-cause Mortality – Men and Women

Up to the 30th November 2015, 3349 deaths had occurred in men and 106 deaths in women. The SMR estimate with follow-up time of members of the cohort to the cut-off date is shown in Table 4.

The SMR for men and women continues to show that the death rate in this workforce was significantly lower than in the general population (adjusting for age differences and using yearly rates which account for the general increase in life expectancy occurring in the Australian population in recent decades). This low mortality rate is often noted in working groups and is known as the *healthy worker effect*.(45-47)

Table 4: All-cause mortality by sex, adjusted for age and calendar period of follow-up, compared to the Australian population

Sex	Person-Years	Observed	Expected	SMR	95% C.I.
Men	444,103	3,349	4,321.62	**0.77**	**0.75 - 0.80**
Women	32,864	106	135.98	**0.78**	**0.64 - 0.94**

3.4. Cancer Incidence and Mortality – Men and Women

The incidence of cancer and the mortality rates from cancer are dealt with together in this section. Cancers are classified under the International Classification of Diseases (ICD), Revision 10 by morphological type (i.e. where it arises in the body) and/or by histology (cell type). Cancers occurring in *Health Watch* members are analysed according to workplace type and smoking effects.

Table 5 and Table 6 show the cancer incidence and the cancer mortality in the *Health Watch* population of men and women. The SIR for cancer in men was identical to that of the general population. In women, the SIR was slightly less than women in the general population, but the decrease was not statistically significant. The mortality rate for cancer in men was significantly low in comparison with the general male population (SMR 0.88, 95% C.I. 0.84-0.93).

The low SMR for cancer is probably a reflection of the *healthy worker effect*. As discussed in the section on all-cause mortality (page 36), this is believed to be largely a selection effect, that is, people in good health are more likely to obtain secure employment and to have a longer life expectancy as a group compared with the general population. Other possible factors are the higher standard of living and ready access to medical services for employed workers.

As was discussed in previous *Health Watch* reports, (7, 9) a *healthy worker effect* is clearly demonstrable when mortality is used as the outcome measure, but not when cancer incidence is used. The 12th Report suggested that the *healthy worker effect* could be a consequence of greater survival rather than of a reduced disease incidence.(48)

Table 5: All-site cancer incidence, men and women, adjusted for age and calendar period of follow-up, compared to the Australian population

Sex	Person-Years	Observed	Expected	SIR	95% C.I.
Men	399,633	3,468	3,460.95	1.00	0.97 - 1.04
Women	29,032	136	148.65	0.91	0.77 - 1.08

Table 6: All-site cancer mortality, men and women, adjusted for age and calendar period of follow-up, compared to the Australian population

Sex	Person-Years	Observed	Expected	SMR	95% C.I.
Men	444,103	1,388	1,568.72	**0.88**	**0.84 - 0.93**
Women	32,864	54	55.80	**0.97**	**0.73 - 1.26**

The cancer incidence and cancer mortality data presented in Table 5 and Table 6 are not exactly comparable, as the cancer analysis has been updated only to the end of 2012, whereas the mortality analysis has been updated to the end of 2015 (as is evident from the difference in person-years of observation). Nevertheless, the differences in person-time could not account for the finding that cancer mortality is significantly reduced for men, whereas cancer incidence is not.

The Healthy Worker Effect

One cause of the "healthy worker effect" is the relative social and economic advantage of employed people, especially for people with relatively secure employment. Unemployed people as a whole tend to have lower socioeconomic status. This commonly correlates with lower income, fewer years of education, lower health status and higher age-adjusted mortality rates than employed people.

Hence when the mortality of occupational cohorts is compared with that of the general population, the mortality rate is higher in the latter because it includes many socially disadvantaged people.

Another factor is that people with life-threatening conditions, such as cancer, tend not to seek or obtain employment after diagnosis: this further lowers the mortality rate in the workforce compared with the general population, especially in the years immediately following recruitment of members to the cohort into Health Watch.

3.5. Results in Women

The ability of *Health Watch* to carry out analyses of the risk for women is limited to major groupings of common conditions because of the small number of women in the study population. When cancer or mortality counts are less than three, the observed numbers are not reported. This is to keep in line with confidentiality practices and reducing the risk for potentially identifying a cohort member.

3.5.1. Mortality by Major Cause for Women

Table 7 shows the mortality by major cause for women. Metabolic disease, including diabetes, nervous system diseases, and cerebrovascular disease are new categories that have not been included in previous reports, however the numbers in these categories are still small. Ischaemic heart disease (IHD) and chronic obstructive pulmonary disease (COPD) are subcategories of circulatory and respiratory diseases respectively and are not added twice in 'all cause' mortality. Because of the small number of women in the petroleum industry, and the low counts in all categories except cancer and circulatory mortality, the SMRs for the remaining other categories have wide confidence intervals and so the point estimates may be regarded as unreliable. Mortality from cancer is similar to that of the general female population. A very low mortality rate for *all causes of death* was also reported in the 14th report and this SMR continues to be low.

Table 7: Mortality by major cause for women, adjusted for age and calendar period of follow-up, compared to the Australian population

Cause	ICD-10	Observed	Expected	SMR	95% C.I.
Cancer(Malignant)	C00–C97, D45–D46, D47.1, D47.3, D47.4, D47.5	54	55.80	0.97	0.73 - 1.26
Metabolic	E00-E99	4	5.18	0.77	0.21 - 1.98
Diabetes	E10-E14	3	3.63	0.83	0.17 - 2.42
Nervous System Diseases	G00 – G99	8	5.40	1.48	0.64 - 2.92
Circulatory	I00 – I99	21	34.23	**0.61**	**0.38 - 0.94**
Ischaemic heart disease	I20-I25	9	15.38	0.59	0.27 - 1.11
Cerebrovascular disease	I60-I69	5	9.17	0.55	0.18 - 1.27
Respiratory	J00 – J99	12	10.20	1.18	0.61 - 2.06
COPD[a]	J40-J44	7	5.41	1.29	0.52 - 2.67
Digestive	K00-K93	<3	4.89	0.41	0.05 - 1.48
External Causes (eg accidents, violence, suicide)	V01-Y98	<3	7.86	**0.13**	**0.00 - 0.71**
All other causes		4	12.43	**0.32**	**0.09 - 0.82**
All causes of death		106	135.98	**0.78**	**0.64 - 0.94**

[a]Chronic obstructive pulmonary disease

3.5.2. Cancer in Women

The overall and site-specific cancer incidence rates in women are shown in Table 8. Kidney and Thyroid cancer are new categories that have not been included previously, however, the numbers in these categories are small. Overall, the SIR is slightly lower but not significantly lower than the population rate (SIR 0.91, 95% C.I. 0.77-1.08), based on 136 cases.

Table 8: Cancer incidence by major anatomical site for women, adjusted for age and calendar period of follow-up compared to the Australian population

Malignant neoplasm of:	ICD-10	Observed	Expected	SIR	95% C.I.
Colon	C18	12	11.70	1.03	0.53 - 1.79
Rectum	C19-C21	5	5.90	0.85	0.28 - 1.98
Melanoma	C43	16	15.90	1.01	0.58 - 1.63
Breast	C50	50	49.39	1.01	0.75 - 1.33
Cervix	C53	4	3.90	1.03	0.28 - 2.63
Kidney	C64-C66,C68	3	3.13	0.96	0.20 - 2.80
Bladder	C67	<3	1.35	0.74	0.02 - 4.12
Pancreas	C25	5	2.60	1.92	0.62 - 4.49
Lung	C33-C34	13	10.25	1.27	0.68 - 2.17
Thyroid	C73	4	4.29	0.93	0.25 - 2.39
Leukaemia	C91-C95	4	2.98	1.34	0.37 - 3.43
Other		19	37.25	**0.51**	**0.31 - 0.80**
All Malignant		136	148.65	0.91	0.77 - 1.08

There were four cases of leukaemia in eligible women identified in this cancer registry linkage. None of the cancer types in Table 8 were found to be in statistically significant excess in the cohort. There was a significant reduction in incidence for the *Other Cancers* category, however, all comparisons are based on very small numbers of cancers as indicated by the wide confidence intervals. Because of the small number of female cancers, further analyses cannot be undertaken by workplace types, or time variables.

Results for Women in Health Watch

The proportion of women in the Health Watch program remains very small and this prevents much detailed analysis.

Women in the industry have death rates which are lower than that of women in Australia generally.

No cancer type has occurred in a statistically significant excess, but the numbers of individual cancer types is too low for meaningful analyses.

3.6. Mortality in Men

3.6.1. Mortality among Men by Major Cause

The SMRs for all major categories of cause of death are shown in Table 9. There have been several new categories added for this report, metabolic disease (of which diabetes is a sub-group), mental disease, nervous system diseases (including Alzheimer disease and Parkinson disease), cerebrovascular disease, asthma, liver disease, urinary system disease (including kidney disease) and the sub groups of external causes – accidents and suicides.

Table 9: Mortality by major cause for men, adjusted for age and calendar period of follow-up, compared to the Australian population

Cause of Death	ICD-10	Observed	Expected	SMR	95% C.I.
Cancer (Malignant)	C00–C97, D45–D46, D47.1, D47.3, D47.4, D47.5	1,388	1,568.72	**0.88**	**0.84 - 0.93**
Melanoma	C43	62	60.41	1.03	0.79 - 1.32
Prostate Cancer	C61	145	154.97	0.94	0.79 - 1.10
Metabolic Diseases	E00-E99	95	154.00	**0.62**	**0.50 - 0.75**
Diabetes	E10-E14	66	114.31	**0.58**	**0.45 - 0.73**
Mental Diseases	F00-F99	69	98.52	**0.70**	**0.54 - 0.89**
Nervous system diseases	G00 – G99	94	129.15	**0.73**	**0.59 - 0.89**
Alzheimer disease	G30	26	31.41	0.83	0.54 - 1.21
Parkinson disease	G20-G22	19	31.54	**0.60**	**0.36 - 0.94**
Circulatory Disease	I00 – I994	994	1,377.25	**0.72**	**0.68 - 0.77**
Hypertensive disease	I10-I15	17	30.33	**0.56**	**0.33 - 0.90**
Ischaemic heart disease	I20-I25	636	847.22	**0.75**	**0.69 - 0.81**
Cerebrovascular disease	I60-I69	164	237.03	**0.69**	**0.59 - 0.81**
Respiratory Diseases	J00 – J99	252	333.40	**0.76**	**0.67 - 0.86**
COPD	J40-J44	131	191.80	**0.68**	**0.57 - 0.81**
Asthma	J45,J46	8	12.80	0.62	0.27 - 1.23
Other lung disease	J60-J67	13	5.73	**2.27**	**1.21 - 3.88**
Asbestosis	J61	11	4.61	**2.39**	**1.19 - 4.27**
Digestive Diseases	K00-K93	117	163.24	**0.72**	**0.59 - 0.86**
Liver disease	K70-K77	58	87.98	**0.66**	**0.50 - 0.85**
Urinary Tract Diseases	N00 - N99	36	62.73	**0.57**	**0.40 - 0.79**
Bladder	C67	34	38.74	0.88	0.61 - 1.23
Kidney disease	C64	20	46.99	**0.43**	**0.26 - 0.66**
External Causes (eg accidents, violence, suicide)	V01-Y98	198	291.91	**0.68**	**0.59 - 0.78**
Accidents	V01-X59, Y85-Y86	118	168.67	**0.70**	**0.58 - 0.84**
Suicide	X60-X84	66	101.99	**0.65**	**0.50 - 0.82**
All other Causes		106	142.69	**0.74**	**0.61 - 0.90**
All Causes		3,349	4,321.62	**0.77**	**0.75 - 0.80**

In all major categories, including that for external causes, and for accidents and suicides, there were fewer deaths than expected, and so all the SMRs were below 1.0. The upper limits of

confidence intervals were all below unity (1.0), i.e. mortality rates are significantly lower than in the general male population in all major categories of cause of death. Mortality from Parkinson disease and asthma, which are sub-categories of the nervous system and respiratory system respectively have similar mortality rates to the Australian population.

Asbestosis, a subcategory of lung disease, is significantly elevated compared to the general population (SMR 2.27, 95% C.I. 1.19-4.27).
Ischaemic heart disease (IHD) mortality, based on 636 male deaths, continues to be low with an SMR of 0.75, with the upper limit of the confidence interval at 0.81. This low death rate would suggest that the incidence of IHD itself in this cohort is also low, and comparable with that in the more advantaged groups in Australian society.

3.6.2. All-cause Mortality and Time Relationships

Internal and external comparisons have been carried out for all causes of death combined, to identify any association with the era of first employment in the industry, duration of employment in the industry and time lapse between first employment in the industry and death. All internal analyses have been adjusted for age and calendar period of follow-up and smoking status.

All-cause mortality among men by period of first employment

Table 10 shows an external comparison between male cohort members according to the period of first employment in the industry and the general population. For each period of employment, the SMR is significantly lower compared to the mortality rate of the Australian male population in that time category. SMRs of different subcategories cannot reliably be compared with each other, but this analysis suggests that the generally increasing SMR with earlier date of first employment is due to a low death rate (in absolute terms) in those members of the cohort who entered the industry most recently. This is probably a manifestation of the *healthy worker effect* discussed in Section 3.3 (page 36), which is commonly found to decrease (i.e. the SMR increases), as cohorts are followed over time.

Table 10: All-Cause mortality among men by period of first employment, adjusted for age and calendar period of follow-up, compared to the Australian population

Period of First Employment	Person-Years	Observed	Expected	SMR	95% C.I.
Post 1985	77,315	136	248.34	**0.55**	**0.46 - 0.65**
1975-84	170,585	612	879.81	**0.70**	**0.64 - 0.75**
1965-74	126,877	1102	1,324.56	**0.83**	**0.78 - 0.88**
1955-64	47,884	928	1,133.56	**0.82**	**0.77 - 0.87**
Pre 1954	21,442	571	735.34	**0.78**	**0.71 - 0.84**

Table 11 shows the cohort mortality rates of males, compared internally within the cohort, according to the period of first employment in the industry. The comparisons were made with the category of most recent entrants to the industry – members of the cohort who have started since 1985.

Table 11: All-cause mortality among men by period of first employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to those employed from 1985

Period of First Employment	Person-Years	Deaths	RMR	95 % C.I.
Post 1985	77,315	136		
1975-84	170,585	612	1.16	0.96 - 1.41
1965-74	126,876	1101	**1.38**	**1.14 - 1.67**
1955-64	47,880	926	**1.43**	**1.16 - 1.76**
Pre 1954	21,433	570	**1.40**	**1.12 - 1.75**

Test for heterogeneity P=0.0011 Test for trend P=0.0029

The relative mortality rate for all-causes combined was higher for those entering the industry before 1985, significantly so for those entering the workforce prior to 1975. There was evidence of a trend of increasing all-cause mortality with earlier date of first employment in the industry. This could be a result of a very low absolute mortality rate in the baseline group, and diminishing healthy worker effect over time, as shown by the data in Table 10.

All-cause mortality among men by duration of employment

Table 12 shows the mortality rates of male cohort members according to the duration of employment in the industry compared to the general population. The findings were similar to those relating to period of entering the industry, and were largely due to a low absolute mortality rate in the group employed for 5-9 years. This was also likely to be due to the low mortality rate of those who entered the industry most recently, since those employed longest are likely to also be in the group who entered the cohort in earlier years.

Table 12: Standardised all-cause mortality among men by duration of employment, adjusted for age and calendar period of follow-up, compared to the Australian population

Duration of Employment	Person-Years	Observed	Expected	SMR	95% C.I.
5-9 Years	84,416	228	311.02	**0.73**	**0.64 - 0.83**
10-15 Years	92,200	442	511.62	**0.86**	**0.79 - 0.95**
15-19 Years	82,791	523	663.83	**0.79**	**0.72 - 0.86**
20-24 Years	69,535	551	723.93	**0.76**	**0.70 - 0.83**
≥25 Years	114,988	1605	2,109.78	**0.76**	**0.72 - 0.80**

Table 13 shows the relative mortality rates of males within the cohort according to the duration of employment in the industry. The comparisons were made with the category of shortest duration in the industry - members of the cohort who were employed between five and nine years. It should be noted that individual members of the cohort can contribute to person-years in more than one category as their duration of employment increases.
Compared with the baseline group (employed for 5-9 years), the mortality rate from all causes combined decreases as duration of employment increases. There is now a statistically significant trend of decreasing risk with increasing duration of employment (P=0.003).

Table 13: Relative all-cause mortality among men by duration of employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to those employed for 5-9 years

Duration of Employment	Person-Years	Deaths	RMR	95 % C.I.
5-9 Years	84,416	228		
10-15 Years	92,200	442	1.11	0.94 - 1.30
15-19 Years	82,790	522	0.96	0.81 - 1.12
20-24 Years	69,534	551	0.92	0.78 - 1.08
≥25 Years	114,975	1,602	0.90	0.78 - 1.05

Test for heterogeneity P=0.0107 Test for trend P=0.0028

All-cause mortality among men by time since first employment

Table 14 shows mortality rates for male cohort members according to the time since they first commence employment in the industry compared to the general population. It shows that all groups have lower mortality risk compared to the age standardised general population. In addition, external comparison with the general population shows that there was a very low SMR in the 5-9 years category of 0.49.

Table 15 shows the mortality rates for males, compared internally within the cohort, according to the time since first employment in the industry. The comparisons were made with the category of shortest time - members of the cohort who were employed between five and nine years ago. It should be noted that individual members of the cohort can contribute to person-years in more than one category as their time since first employment increases.
The mortality significantly increased with time since first employment (trend test 0.01) and was highest in the group employed for 25 years or more. The *healthy worker effect* is known to diminish with age and is the most likely explanation for this finding.

Table 14: Standardised all-cause mortality among men by time since first employment, adjusted for age and calendar period of follow-up, compared to the Australian population

Time since first employment	Person-Years	Observed	Expected	SMR	95% C.I.
5-9 Years	40,675	40	80.82	**0.49**	**0.35 - 0.67**
10-15 Years	57,300	99	149.87	**0.66**	**0.54 - 0.80**
15-19 Years	65,576	138	236.49	**0.58**	**0.49 - 0.69**
20-24 Years	68,755	223	350.79	**0.64**	**0.55 - 0.72**
≥25 Years	211,793	2,849	3,503.62	**0.81**	**0.78 - 0.84**

Table 15: Relative all-cause mortality among men by time since first employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to the those first employed 5-9 years ago

Time since first employment	Person-Years	Deaths	RMR	95 % C.I.
5-9 Years	40,675	40		
10-15 Years	57,300	99	1.36	0.93 - 1.99
16-19 Years	65,576	138	1.19	0.80 - 1.75
20-24 Years	68,755	223	1.26	0.85 - 1.85
≥25 Years	211,778	2,845	**1.49**	**1.02 - 2.18**

Test for heterogeneity P=0.0264 Test for trend P=0.0116

Overall Mortality of Men in the Health Watch Cohort

For men, overall death rates were low. Death rates in all major disease categories – circulatory, including heart disease, cancer, respiratory disease, diseases of the digestive system, and external causes (accidents, violence etc.) – were also significantly lower than the corresponding population. There was a significantly elevated death rate for lung disease, specifically death from asbestosis

There was a significant trend of decreasing all-cause mortality with increasing duration of employment.

There was evidence of a trend by time period of first employment and time since first employment. The overall mortality was particularly low for the most recently employed men compared to the general population. It may be the low mortality in the baseline comparative group that explains the apparent increase in mortality in those first employed in earlier years, with the healthy worker effect diminishing as members age.

3.7. Cancer Incidence among Men

Site-specific cancer incidence ratios for men are shown in Table 16. This table lists the number of cases from particular cancers observed in *Health Watch*, the number expected, and the calculated SIRs. The definition of AML has changed since the last report and now includes many sub-categories that were previously included in Acute non-lymphocytic leukaemia (ANLL). For this reason, ANLL was not analysed in this report.

Table 16: Cancer incidence by major anatomical site in men, adjusted for age and calendar period of follow-up, compared to the Australian population

Malignant neoplasm of:	ICD-10	Observed	Expected	SIR	95% C.I.
Lip, Oral cavity and Pharynx	C00-C14	123	151.55	**0.81**	**0.67 - 0.97**
Oesophagus	C15	62	51.63	1.20	0.92 - 1.54
Stomach	C16	84	82.61	1.02	0.81 - 1.26
Colon	C18	261	281.44	0.93	0.82 - 1.05
Rectum	C19-C21	195	193.25	1.01	0.87 - 1.16
Liver	C22	29	46.84	**0.62**	**0.41 - 0.89**
Gallbladder	C23-C24	17	17.59	0.97	0.56 - 1.55
Pancreas	C25	58	70.58	0.82	0.62 - 1.06
Larynx	C32	33	41.75	0.79	0.54 - 1.11
Lung	C33-C34	311	392.57	**0.79**	**0.71 - 0.89**
Melanoma	C90	438	357.95	**1.22**	**1.11 - 1.34**
Mesothelioma	C45	49	30.66	**1.60**	**1.18 - 2.11**
Connective Tissue	C47-C49	15	19.56	0.77	0.43 - 1.26
Prostate	C61	1,072	938.63	**1.14**	**1.07 - 1.21**
Testis	C62	26	21.96	1.18	0.77 - 1.73
Bladder	C67	95	102.12	0.93	0.75 - 1.14
Kidney	C64	100	108.40	0.92	0.75 - 1.12
Eye	C69	12	9.23	1.30	0.67 - 2.27
Brain & Nervous System	C71	59	51.14	1.15	0.88 - 1.49
Thyroid	C73-C75	15	21.39	0.70	0.39 - 1.16
Non-Hodgkin lymphoma	C82-C86, C96	127	130.65	0.97	0.81 - 1.16
Multiple myeloma	C90	46	43.91	1.05	0.77 - 1.40
Leukaemia	C91-C95	74	92.56	0.80	0.63 - 1.00
Acute lymphatic leukaemia	C910	<3	3.38	0.59	0.07 - 2.13
Chronic lymphatic leukaemia	C911	34	40.11	0.85	0.59 - 1.18
Acute myeloid leukaemia[a]	C920, C923–C926, C928, C930, C940, C942, C944–C945	18	25.47	0.71	0.42 - 1.12
Chronic myeloid leukaemia	C921	11	9.73	1.13	0.56 - 2.02
Other leukaemia	C91-95	9	13.87	0.65	0.30 - 1.23
Unspecified cancer site		74	94.22	**0.79**	**0.62 - 0.99**
Myelodysplastic Syndrome[b]	D46	24	20.52	1.17	0.75 - 1.74
Other sites		60	81.43	**0.74**	**0.56 - 0.95**
All Malignant		3,468	3,460.95	1.00	0.97 - 1.04

[a] The definition of AML has changed since the last report.

[b]Myelodysplastic Syndrome (MDS) cases diagnosed after 2003 are included in this analysis only. The National population data set for MDS required to calculate the SMR, is only available from 2003 onwards.

There were statistically significant excess cases of mesothelioma and melanoma of the skin. These excesses were also observed in previous reports.(9, 23)
The excess risk of prostate cancer was also observed in the 14th Report in 2013. The risk of leukaemia continues to decrease in the cohort and has almost reached a significantly lower risk compared with the Australian population. Myelodysplastic syndrome (MDS) was in excess, however the increase was not statistically significant and has decreased since the last report.(23) MDS has been found to be associated with benzene exposure in the petroleum industry.(22)
There was a statistically significant lowering of risk of lung and liver cancer and cancer of the lip, oral cavity and pharynx. These reductions were also observed in previous reports. (9, 23)

3.7.1. Cancer and Time Relationships for Men

Cancer incidence and mortality according to period of first employment

Table 17 shows that there was a slight trend in cancer incidence with period of first employment. The trend of cancer mortality rates with period of first employment was much larger, (p=0.0014), and this trend was stronger than that assessed in the previous report. This could be a result of the very low overall mortality in the baseline group. Although the overall cancer mortality rates are increased in those employed before 1985 compared to the baseline group employed post 1985, there is a significant decrease in cancer mortality when the cohort is compared to the Australian population of comparable age.

Table 17: Cancer incidence and cancer mortality by period of first employment, adjusted for age and calendar period of follow-up, and ever/never smoker compared to those employed after 1985

Period of first Employment	Cancers	Relative Incidence RIR	95 % C.I.	Deaths	Relative Mortality RMR	95 % C.I.
Post 1985	241			57		
1975-84	900	1.07	0.92 - 1.23	298	**1.38**	**1.03 - 1.84**
1965-74	1,168	1.08	0.93 - 1.26	466	**1.54**	**1.15 - 2.07**
1955-64	750	1.15	0.96 - 1.37	354	**1.70**	**1.23 - 2.34**
Pre 1954	409	1.21	0.99 - 1.47	213	**1.80**	**1.27 - 2.54**

Incidence: Test for heterogeneity P=0.3580 Test for trend P=0.0474
Mortality: Test for heterogeneity P=0.0102 Test for trend P=0.0014

Cancer incidence and mortality according to duration of employment

Table 18 shows relative cancer incidence and relative cancer mortality by duration of employment. There was no significant trend in cancer incidence nor cancer mortality with increasing duration of employment.

Table 18: Cancer incidence and cancer mortality by duration of employment, adjusted for age and calendar period of follow-up, and ever/never smoker compared to those employed for 5-9 years

Duration of Employment	Cancers	Relative Incidence RIR	95 % C.I.	Deaths	Relative Mortality RMR	95 % C.I.
5-9 Years	301			89		
10-15 Years	474	0.96	0.83 - 1.11	204	**1.31**	**1.02 - 1.68**
15-19 Years	530	**0.84**	**0.73 - 0.97**	203	0.96	0.74 - 1.23
20-24 Years	605	0.91	0.79 - 1.05	233	1.01	0.78 - 1.30
≥25 Years	1,556	0.98	0.86 - 1.12	659	1.05	0.83 - 1.32

Incidence: Test for heterogeneity P=0.0284 Test for trend P=0.5553
Mortality: Test for heterogeneity P=0.0234 Test for trend P=0.3437

Cancer incidence and mortality by time since first employment

Table 19 shows relative cancer incidence and cancer mortality by time elapsed from first employment to date of cancer diagnosis or death. Note that this is not employment time but rather time elapsed since first employed. The findings were very similar to those according to duration of employment. There was no relationship between cancer incidence and time since first employment. Cancer mortality for all subgroups except those employed for 15-19 years prior to diagnosis were increased relative to those whose cancer arose within nine years of joining the industry. This is likely to be attributable to a low absolute mortality rate in the baseline category of those members of the cohort employed 5-9 years previously.

Table 19: Cancer incidence and mortality by time since first employment, adjusted for age and calendar period of follow-up, and ever/never smoker compared to those first employed 5-9 years ago

Time since first Employment	Cancers	Relative Incidence RIR	95 % C.I.	Deaths	Relative Mortality RMR	95 % C.I.
5-9 Years	51			7		
10-15 Years	133	1.23	0.88 - 1.71	43	**3.11**	**1.37 - 7.08**
15-19 Years	202	1.02	0.73 - 1.43	55	2.24	0.97 - 5.18
20-24 Years	362	1.11	0.79 - 1.55	97	**2.34**	**1.02 - 5.37**
≥25 Years	2719	1.20	0.86 - 1.67	1186	**2.81**	**1.23 - 6.41**

Incidence: Test for heterogeneity P=0.1913 Test for trend P=0.1698
Mortality: Test for heterogeneity P=0.0106 Test for trend P=0.0899

Cancer Incidence and Mortality among Men in the Health Watch Cohort

The chance of getting cancer is the same for men in the petroleum industry as for the general Australian population. This is so for all cancers combined and for most individual cancer types. However, some cancers – mesothelioma, prostate cancer and melanoma of the skin, continue to occur at significantly higher rates compared with the general population. The increased incidence of prostate cancer, together with the absence of an increased prostate cancer mortality, suggest a higher rate of screening compared to the general population. Melanoma incidence did not increase with increasing duration of employment, time since first employment, or time period of first employment, which suggest that workplace factors are not a likely explanation. Mortality from melanoma is similar to the national rates which could indicate that melanoma cases were treated earlier which may improve survival.

There was a significant lowering of the rates of liver and lung cancer and cancers of the lip, oral cavity and pharynx. Leukaemia risk continues to decrease compared with the general population. The age-adjusted mortality rate from all cancers combined was significantly less than in the general population.

Those who worked in the industry in earlier times have not been at a significantly greater risk of developing cancer than those who entered the industry more recently. However, cancer mortality rates were higher for those who entered the industry before 1985 compared to those who started later, and there is a statistically significant trend of higher mortality associated with earlier starting periods.

Cancer and mortality rates do not appear to be related to duration in the industry or time since first employment.

3.7.2. Workplace Type and Health Outcomes among Men

Analyses were undertaken for the five principal workplace types – refineries, terminals, airports, onshore production and offshore production. Men were grouped by the site of their most recent job.

Table 20: Numbers of male members of the cohort in each workplace type

Workplace Type	Number of men in cohort	% of men in cohort	Person-years	% of Person-years
Refinery	6,494	39.0	175,711	39.6
Terminal	6,464	38.8	172,108	38.8
Airport	603	3.6	16,644	3.7
Onshore production	2,347	14.1	59,213	13.3
Offshore Production	725	4.4	19,892	4.5
Total	16,654	100.0	444,103	100.0

The all-cause mortality by workplace type is shown in Table 21. All-cause mortality continues to be significantly lowered in all workplace types, which is consistent with the mortality risk in the cohort overall.

Table 21: All-cause mortality in men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population

Workplace Type	Person-Years	Observed	Expected	SMR	95% C.I.
Refinery	175,711	1,303	1,794.89	**0.73**	**0.69 - 0.77**
Terminal	172,108	1,544	1,837.15	**0.84**	**0.80 - 0.88**
Airport	16,644	126	188.15	**0.67**	**0.56 - 0.80**
Onshore Production	59,213	296	383.71	**0.77**	**0.69 - 0.86**
Offshore Production	19,892	80	116.06	**0.69**	**0.55 - 0.86**
Total	444,103	3,349	4,321.62	**0.77**	**0.75 - 0.80**

Table 22 shows mortality from ischaemic heart disease by workplace type. SMRs were lowered in each workplace type and the difference was statistically significant except for offshore production workers where there were small numbers.

Table 22: Ischaemic heart disease (ICD-10 I20-I25) mortality for men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population

Workplace Type	Person-Years	Observed	Expected	SMR	95% C.I.
Refinery	175,711	238	355.87	**0.67**	**0.59 - 0.76**
Terminal	172,108	316	365.18	**0.87**	**0.77 - 0.97**
Airport	16,644	17	36.99	**0.46**	**0.27 - 0.74**
Onshore Production	59,213	51	68.75	**0.74**	**0.55 - 0.98**
Offshore Production	19,892	14	20.19	0.69	0.38 - 1.16
Total	444,103	636	847.22	**0.75**	**0.69 - 0.81**

Table 23 shows the incidence of cancer in the different workplace types. All five categories of workplace type showed total cancer risks which were no different from that of the general population. This was consistent with the results for the cohort as a whole.

Table 23: Cancer incidence among men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population

Workplace Type	Person-Years	Observed Cancers	Expected Cancers	SIR	95% C.I.
Refinery	158,221	1,383	1,391.42	0.99	0.94 - 1.05
Terminal	155,501	1,488	1,449.79	1.03	0.97 - 1.08
Airport	15,073	143	148.81	0.96	0.81 - 1.13
Onshore Production	52,599	354	352.72	1.00	0.90 - 1.11
Offshore Production	17,765	99	116.54	0.85	0.69 - 1.03
Total	399,633	3,468	3,460.95	1.00	0.97 - 1.04

As shown in Table 24, cancer mortality was lower than population rates in all workplace types, but the difference was statistically significant for refinery workers only. The SMRs for the other production workers were similar to that of refinery workers but did not reach statistical significance.

Table 24: Cancer mortality among men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population

Workplace Type	Person-Years	Observed Cancer Deaths	Expected Cancer Deaths	SMR	95% C.I.
Refinery	175,711	526	642.00	**0.82**	**0.75 - 0.89**
Terminal	172,108	641	667.80	0.96	0.89 - 1.04
Airport	16,644	55	68.53	0.80	0.60 - 1.04
Onshore Production	59,213	131	144.32	0.91	0.76 - 1.08
Offshore Production	19,892	35	45.51	0.77	0.54 - 1.07
Total	444,103	1,388	1,568.72	**0.88**	**0.84 - 0.93**

Health and Workplace Type

The health of male employees as measured from the Health Watch results differed very little between those who worked at the various types of workplaces in the industry, such as upstream production sites and downstream refineries, terminals and distribution sites. That is, the chances of dying, or of getting cancer or heart disease were very similar no matter where men in Health Watch worked.

3.8. Non-malignant Disease from Asbestos Exposure

Apart from its association with certain types of cancer (mesothelioma and lung cancer), asbestos exposure can cause non-malignant conditions, including pleural plaques and asbestosis. Pleural plaques are deposits of fibrous tissue (sometimes becoming calcified) on the pleural lining of the chest cavity. They are the commonest manifestation of asbestos exposure, but in general, they are not disabling.

Asbestosis is a disease affecting the lung tissue itself, and can cause breathlessness and other respiratory symptoms, associated disability such as reduced walking tolerance, and can be fatal. Asbestosis can also lead to an increased risk of lung cancer. There is also increasing evidence that asbestos exposure in itself, even in the absence of asbestosis, can increase the risk of lung cancer, although there is no universal agreement on this conclusion.(49-51)

Eleven members of the *Health Watch* cohort have died from asbestosis, an increase from five deaths in the previous report. This elevation is now statistically significant (SMR 2.39, C.I. 1.19 - 4.27). Although mortality from asbestosis was elevated in the cohort compared to the general population, this does not represent the full picture because over 119 members of the cohort have reported asbestos related illnesses. It is important to note that these self-reported illnesses were not validated and were therefore not included in any analyses. Asbestosis is not necessarily a fatal condition and it is not possible to identify all living cases. Unlike cancer, there is no Australian register for asbestosis or pleural plaques which have not resulted in death.

The 12th Report (7) examined the reporting of asbestos related diseases by members of the cohort and stated:(7) "It is likely that these figures understate the prevalence of effects of asbestos exposure, especially of pleural plaques. Not all members of the cohort reply to the periodic questionnaires. Moreover, since pleural plaques commonly produce no symptoms, they may remain undiagnosed unless the subject has a chest x-ray.

Full enumeration of these effects of asbestos exposure would require a study of different design to *Health Watch*."

Non-malignant Asbestos Diseases

Non-malignant asbestos-related diseases were found in the cohort, both through the mortality data and from self-reports. The significance of the self-reported findings cannot be assessed because there are no suitable comparative data collected in the general population. However, the voluntary nature of the reporting probably underestimates the prevalence in the cohort.

4. LIFE STYLE FACTORS AMONG MALE MEMBERS OF THE COHORT

4.1. Tobacco Smoking

4.1.1. Smoking Status

The smoking status of each member of the cohort used in the analyses in this report was based on smoking habits reported at initial and later interviews. After retirement or leaving the industry, additional information has been derived from postal surveys of all retired and resigned members carried out during 1994, 1996 and 1999 in combination with the Health Letters, the last one being in 2014. The latest information on smoking category was used in the analyses below.

In the 11th *Health Watch* Report, (28) the smoking prevalence was compared with national smoking data based on 1995 estimates, using direct standardisation for age. The *Health Watch* male smoking rate was 24.1% compared with the Australian population rate of 28.2%. On this basis, the age-standardised smoking prevalence was slightly less than in the Australian national population. In the 12th Report, updated figures were used based on the proportion of Australian smoking rates from 1980 to 2001.(44) Age-specific comparisons showed that for older people (over 50) the smoking rates were 5-10% higher in *Health Watch* but for the younger people (under 30) the smoking rates in *Health Watch* were much lower, about half of that in the general population.

Figure 2 shows that a minority of men in the *Health Watch* cohort smoke and comparison with previous reports show that the number of ex-smokers continue to increase.

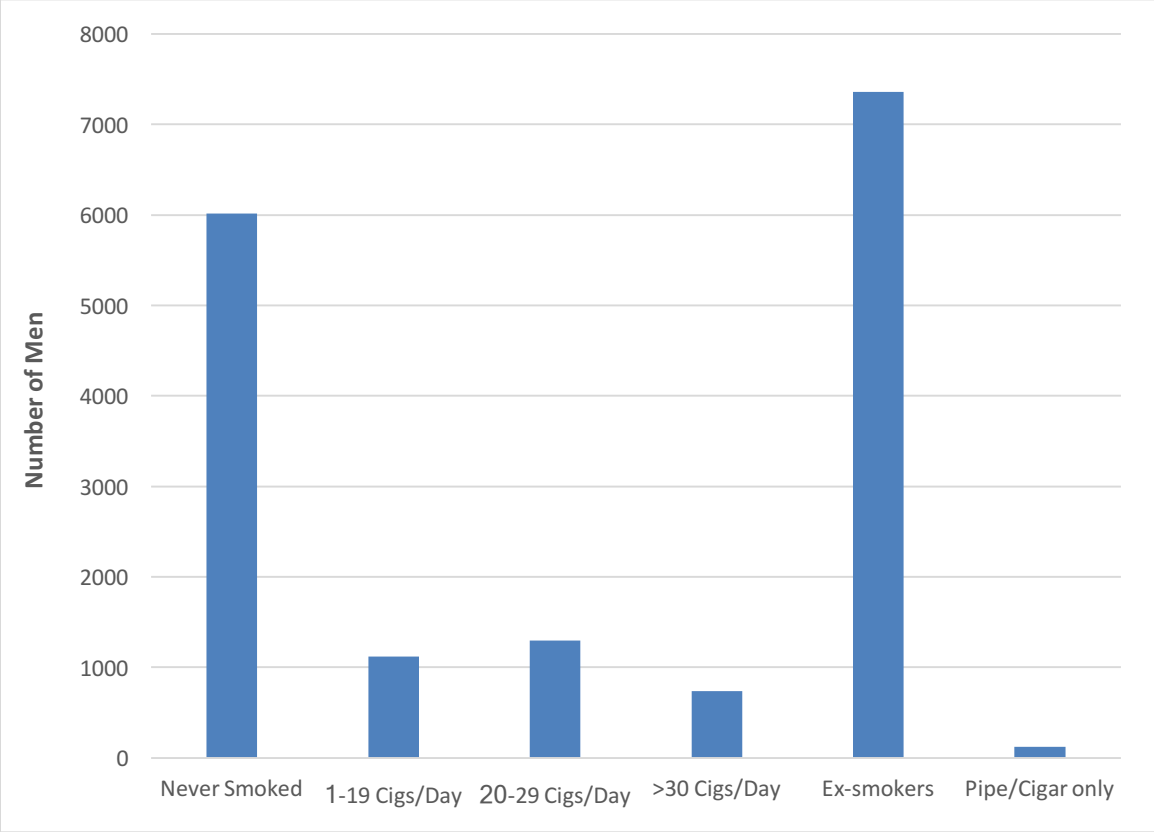

Figure 2: Latest smoking status of the men in the *Health Watch* cohort

Table 25 shows that some of the data used for the smoking analyses were collected as long ago as the early 1980s. It is possible that some of these individuals have changed their smoking habits but have not informed the study team. Many would also now be deceased. Since more individuals reported being smokers than being non-smokers and the trend in Australia has been to quit, it is probable that a survey of current smoking habits would report a reduction in smoking rates. That is, it is possible that fewer of the surviving older people in the cohort, those recruited in Surveys 1 and 2, remain smokers.

Table 25: Source of most recent smoking data for individuals in the cohort.

Source of Data	Period	Number	Percentage
Survey 1	1981-1983	1,408	7.8
Survey 2	1986-1987	1,965	10.9
Survey 3	1991-1993	2,910	16.1
Survey 4	1996-2000	3,029	16.8
Survey 5	2003	1,207	6.7
Health letter updates	Various	7,510	41.7
Total		18,029	100.0

Table 26 is a breakdown of 'ever smokers' in each main job group. The drivers group had the highest proportion of ever smokers at 72.66% while office workers were the least likely to have ever smoked (59.48%). It should be noted that many of these 'ever smokers' could now be ex-smokers.

Table 26: Number of 'ever smokers' in each main job category

Job	Number of Men	Number of Ever Smokers	Percentage
Driver	2,165	1,573	72.66
Refinery	3,209	2,224	69.31
Terminal	2,221	1,555	70.01
Maintenance	3,900	2,586	66.31
Office	5,763	3,428	59.48

4.1.2. Smoking and All-cause Mortality among Men

In Table 27 the relative mortality ratios among men for all-cause mortality are shown according to smoking habit. These tables compare various categories of smokers relative to a baseline of those who have never smoked. The comparison clearly shows a marked increase in age-adjusted mortality with increasing tobacco use. Men smoking up to 19 cigarettes a day have over twice the age-adjusted death rate from all causes combined, compared with those who have never smoked. For those smoking 20-29 cigarettes per day there was nearly a 3 and half fold increase in risk, and there was over a four-fold risk at 30+ cigarettes per day. As in the 14th report, ex-smokers also had a statistically significant increase in mortality risk, although this increase was much less than for current smokers (RMR 1.14, 95% C.I. 1.04-1.24). The trend for increasing age-adjusted mortality with increasing smoking level was highly

statistically significant. This analysis once again showed a strong and steep effect as did the results from previous *Health Watch* reports.

Table 27: All-cause mortality by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked

Smoking Category	Person-Years	Deaths	RMR	95 % C.I.
Never smoked	159,730	782		
1-19 / day	28,015	214	**2.12**	**1.82 - 2.46**
20-29 / day	31,746	424	**3.43**	**3.05 - 3.87**
30+ / day	17,837	295	**4.09**	**3.57 - 4.68**
Ex-smoker	203,778	1,594	**1.14**	**1.04 - 1.24**
Pipe/cigar only	2,983	36	**2.74**	**1.96 - 3.82**

Test for heterogeneity P=0.0000 Test for trend P=0.1277

4.1.3. Smoking and Ischaemic Heart Disease (ICD-10 I20-I25)

Many studies have shown that smoking is a major risk factor for ischaemic heart disease and this is confirmed in the *Health Watch* cohort. Table 28 shows that smoking dramatically affects the chance of dying from heart attack for men in the *Health Watch* cohort. It is reasonable to assume that smoking similarly increases the risk of suffering a heart attack, even if death is not the outcome. Quitting dramatically reduced the risk of ischaemic heart disease and was not statistically elevated for those who have stopped smoking.

Table 28: Ischaemic heart disease mortality (ICD-10 I20-I25) among men by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked

Smoking Category	Person-Years	IHD Deaths	RMR	95 % C.I.
Never smoked	159,730	142		
1-19 / day	28,015	40	**2.20**	**1.55 - 3.13**
20-29 / day	31,746	77	**3.31**	**2.50 - 4.39**
30+ / day	17,837	70	**5.06**	**3.79 - 6.75**
Ex-smoker	203,778	295	1.12	0.91 - 1.36
Pipe/cigar only	2,983	11	**4.40**	**2.38 - 8.14**

Test for heterogeneity P=0.0000 Test for trend P=0.4705

4.1.4. Smoking and Cancer among Men

Table 29 and Table 30 show the relationship between total cancer incidence and total cancer mortality and smoking. As with all-cause mortality, both of these outcomes show a striking and significant increase in risk with increasing tobacco use. Similar to the results reported in the 14th report, ex-smokers appeared to have a slightly greater risk of cancer and cancer mortality compared to those who have never smoked, however this risk was much lower than those who continued to smoke.

Table 29: Cancer incidence among men by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked

Smoking Category	Person-Years	Any Cancer	RIR	95% C.I.
Never smoked	142,939	968		
1-19 / day	25,035	187	**1.29**	**1.10 - 1.51**
20-29 / day	28,649	281	**1.53**	**1.34 - 1.75**
30+ / day	16,168	186	**1.73**	**1.48 - 2.02**
Ex-smoker	184,165	1,824	**1.11**	**1.03 - 1.20**
Pipe/cigar only	2,676	22	1.16	0.76 - 1.78

Test for heterogeneity P=0.0000 Test for trend P=0.0262

Table 30: Cancer mortality among men by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked

Smoking Category	Person-Years	Cancer Deaths	RMR	95 % C.I.
Never smoked	159,730	292		
1-19 / day	28,015	98	**2.42**	**1.92 - 3.04**
20-29 / day	31,746	185	**3.60**	**2.99 - 4.33**
30+ / day	17,837	131	**4.39**	**3.57 - 5.40**
Ex-smoker	203,778	671	**1.29**	**1.12 - 1.48**
Pipe/cigar only	2,983	11	**2.02**	**1.11 - 3.69**

Test for heterogeneity P=0.0000 Test for trend P=0.0239

Table 31 shows the relationship between smoking and lung cancer incidence. For this outcome, the relationship to smoking was very strong; a 16-fold increase in risk in those smoking up to 19 cigarettes per day compared with the risk in those who have never smoked, nearly a 30-fold increase in risk for those who smoke 20-29 cigarettes per day, and over a 50-fold increase in risk for those who smoke 30+ cigarettes per day. Those who reported having quit smoking had over a 7-fold increase in risk compared to those who never smoked. The risks of lung cancer were higher than those reported in the last report, including cigar smokers, however the numbers were very small in this group.

This analysis reaffirms that lung cancer in people who have never been active smokers was a rare disease. There were only 11 lifelong non-smokers in the cohort who have developed lung cancer, a small baseline group so the estimates of increasing risk with increasing tobacco use were approximate only. Nevertheless, one of the great strengths of *Health Watch* is that the smoking histories have been collected prospectively. In most epidemiological studies, smoking histories are collected retrospectively, giving lung cancer cases the opportunity to deny previous tobacco use or to minimise their tobacco consumption.

It should be emphasised that the comparisons in Table 31 showing excess risk are comparisons made within the cohort. The *Health Watch* cohort as a whole has a significantly reduced rate of lung cancer incidence compared with the general male population (Table 16).

Table 31: Lung cancer incidence (ICD-10 C33-C34) among men by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked

Smoking Category	Person-Years	Lung Cancer	RIR	95% C.I.
Never smoked	142,939	11		
1-19 / day	25,035	24	**16.34**	**8.00 - 33.38**
20-29 / day	28,649	56	**29.46**	**15.41 - 56.30**
30+ / day	16,168	60	**53.17**	**27.92 - 101.20**
Ex-smoker	184,165	157	**7.79**	**4.22 - 14.36**
Pipe/cigar only	2,676	3	**15.35**	**4.28 - 55.07**

Test for heterogeneity P=0.0000 Test for trend P=0.0000

Table 32 shows the association between smoking and lung cancer mortality for men. Here the trend was similar to that of lung cancer incidence but the point estimates of relative risk, which are greater than those for lung cancer incidence, were less reliable because the baseline comparison group of non-smokers contained only nine deaths.

Table 32: Lung cancer mortality (ICD-10 C33-C34) among men by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked

Smoking Category	Person-Years	Lung Cancer Deaths	RMR	95 % C.I.
Never smoked	159,730	9		
1-19 / day	28,015	27	**22.13**	**10.40 - 47.10**
20-29 / day	31,746	65	**42.09**	**20.94 - 84.59**
30+ / day	17,837	62	**68.72**	**34.12 - 138.4**
Ex-smoker	203,778	139	**8.53**	**4.35 - 16.75**
Pipe/cigar only	2,983	3	**18.37**	**4.97 - 67.93**

Test for heterogeneity P=0.0000 Test for trend P=0.0000

4.1.5. Smoking and Bladder Cancer (ICD-10 C67)

An analysis was performed on the association between bladder cancer and smoking by smoking category. The results are shown in Table 33.

There was a strong and statistically significant trend of increased bladder cancer incidence with increasing tobacco use. The estimated relative risk in the category of smoking 1-19 or more cigarettes per day is more than two and half times greater than those who have never smoked and was bordering on being statistically significant. The remaining categories all showed a statistically significant increase in bladder cancer, up to more than four times for those who smoked more than 30 cigarettes per day, compared to those who have never smoked. Bladder cancer risk in pipe and cigar only smokers also showed a statistically elevated risk compared to those who never smoked but there were only three cases in this category and the confidence interval is wide.

Table 33: Bladder cancer incidence (ICD-10 C67) by smoking category, adjusted for age and calendar period of follow-up, compared to those who never smoked

Smoking Category	Person-Years	Bladder Cancer	RIR	95% C.I.
Never smoked	142,939	13		
1-19 / day	25,035	5	2.80	1.00 - 7.85
20-29 / day	28,649	7	**2.89**	**1.15 - 7.26**
30+ / day	16,168	6	**4.18**	**1.58 - 11.00**
Ex-smoker	184,165	61	**2.50**	**1.37 - 4.55**
Pipe/cigar only	2,676	3	**11.66**	**3.31 - 40.99**

Test for heterogeneity P=0.0016 Test for trend P=0.0023

4.1.6. Deaths Attributable to Smoking among Men in the Cohort

Health Watch cannot identify which individual deaths are caused by smoking but can provide an indication of the numbers of premature deaths attributable to the smoking habit. The effect is so critical to the future health of those in the cohort, that even crude figures are felt to be worth publishing (Figure 3 and Figure 4).

NB note the difference in scale between Figure 3 and Figure 4.

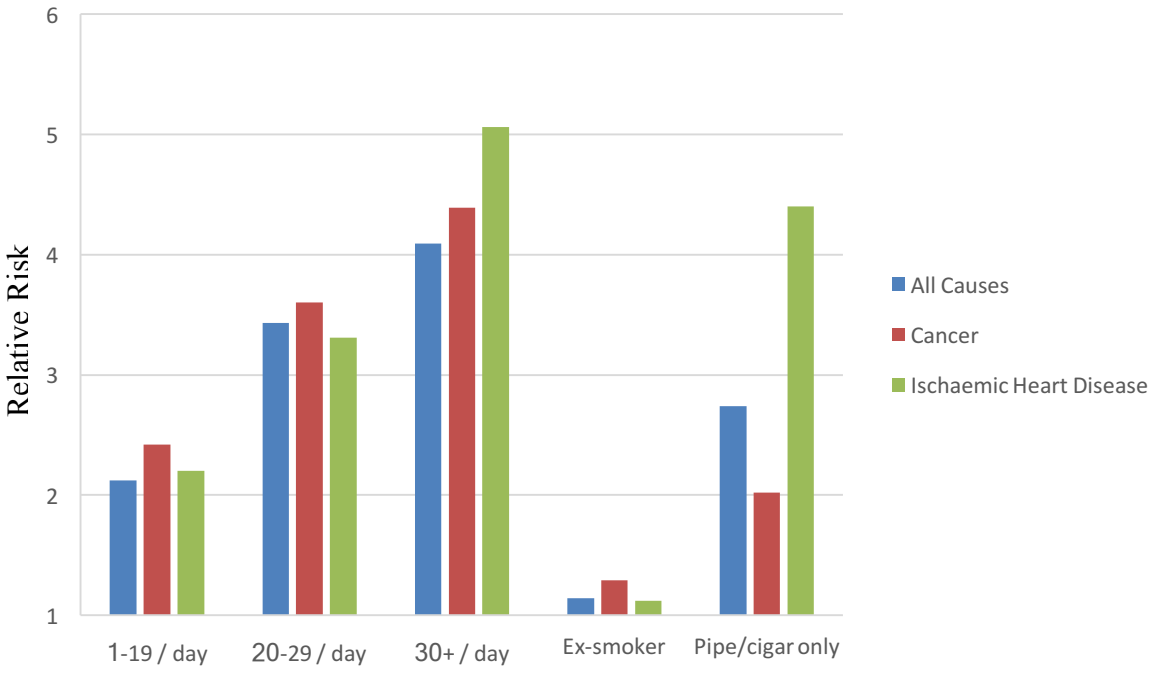

Figure 3: Relative risk of dying from any cause, of cancer, or of ischaemic heart disease among men for different smoking categories, compared to those who never smoked, adjusted for age and calendar year.

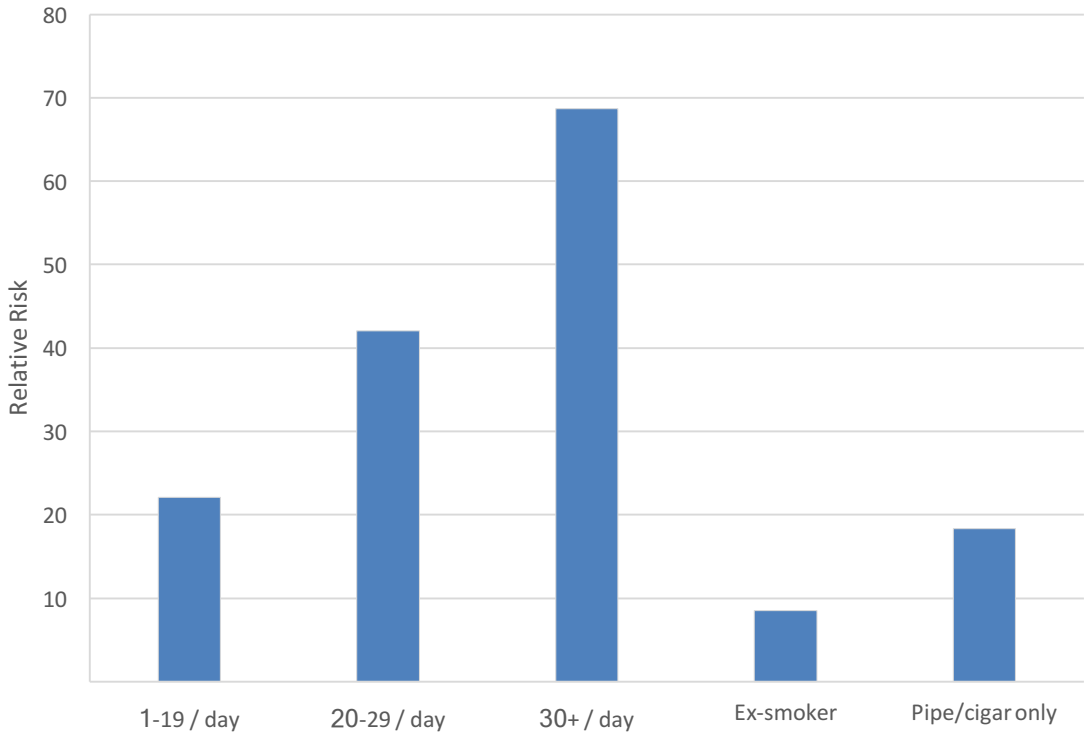

Figure 4: Relative risk of dying from lung cancer among men for different smoking categories compared to those who never smoked, adjusted for age and calendar year.

The effect of smoking on health risks for members of *Health Watch* is demonstrated in the results for lung cancer and ischaemic heart disease, being specific causes of death which can be attributed to smoking. It is likely that other smoking-related diseases are also occurring in the cohort, just as they are in the Australian population as a whole. Crude analyses[2] of the results indicate that smoking probably caused about 49% of the ischaemic heart disease deaths and therefore about 240 men in the cohort have died of heart attacks over the past 38 years due to smoking. Smoking accounts for nearly all lung cancers in the cohort but many other cancers are smoking-related as well.

Altogether it is estimated that smoking has been a contributing factor to about 53% of all male cancer deaths in the cohort, i.e. about 577 men. Combining all-causes of death, it is estimated that smoking has played a part in the deaths of about 1171 men, or 46% of the 2,563 deaths that have occurred in the 'ever smoker' group of the *Health Watch* cohort.

4.1.7. Effects of Quitting

Men who gave up smoking had better outcomes than those who continued to smoke. The effects of quitting are of interest to those in the cohort who have quit, and to those who might be encouraged to do so. The benefit of quitting smoking on mortality and cancer incidence can clearly be seen in the *Health Watch* cohort.

---

[2] The estimates of excess deaths were computed by comparing the actual numbers of deaths with the number expected if the ever-smokers had the same mortality rate as never-smokers. The expected numbers were derived by multiplying the rates for never-smokers by number of person-years of follow-up in all the smoking categories combined. These were not age standardised.

The RMR for deaths from all-causes was about 14% higher in ex-smokers compared with those who have never smoked (RMR 1.14, 95% C.I. 1.04-1.24) and was similar to the results produced in the 14th Report (RMR 1.16, 95% C.I. 1.04-1.30). (23) In the case of death from ischaemic heart disease the RMR in ex-smokers was similar to that of never smokers (RMR 1.12, 95% C.I. 0.91-1.36), and was nearly one-half of the risk relative to those who continued to smoke 1-19 cigarettes per day and much less than those who smoked more. The risk of dying from ischaemic heart disease in ex-smokers was significantly elevated in the 14th Report (RMR 1.30, 95% C.I. 1.03-1.65). (23)

For all cancer deaths combined, the excess risk in ex-smokers was statistically significantly different from that of those who have never smoked (RMR 1.29, 95% C.I. 1.29-1.48) and was slightly higher than what was reported in the 14th Report (RMR 1.20, 95% C.I. 1.02-1.42). (23)

Lung cancer mortality risk in ex-smokers remained significantly higher compared to the risk in those who have never smoked (RMR 8.53, 95% C.I. 4.35-16.75). Whilst the risk was higher in ex-smokers, this risk was less than half of that compared to those who continued to smoke 1-19 cigarettes per day.

Lung cancer incidence risk in ex-smokers also remained higher compared to those who have never smoked (RMR 7.79, 95% C.I. 4.22-14.36) but less than half of the risk in those who continued to smoke 1-19 cigarettes per day.

These data showed a slightly higher risk than that reported previously for all-cause mortality, all cancer mortality and lung cancer incidence among ex-smokers when compared to never smokers. Other studies have shown that the risk of lung cancer in men and women declines as the time since quitting increases.(52-54) There were slight reductions in IHD mortality and lung cancer mortality compared to the rates reported previously.

4.2. Alcohol Consumption among Men

The effects of alcohol consumption on mortality risk produces a "U-shaped" or “J-shaped” curve, as shown in Figure 5. This illustrates how low to moderate drinking results in a reduced risk of all-cause mortality compared to heavier drinking. As drinking consumption increases, so does the risk of mortality. Because many important causes of death from alcohol are also affected by smoking, adjustment has been made in the analysis to allow for the influence of smoking. Table 34 shows the relationship between drinking alcohol and death from all-causes compared to two different reference groups; 1-7 drinks per week and total abstainers. A recent review paper suggested that the comparison group used drastically alters the risk of mortality and the apparent protective effect of alcohol consumption. The paper showed that the protective effects of alcohol disappeared when they made comparisons with occasional drinkers rather than total abstainers. (55) There were similar results in this analysis, that when using the 1-7 drinks per week group as the reference, the protective effects of alcohol disappeared and the risk of mortality for low to moderate drinkers (8-21 drinks per week) was similar to the reference group. Alternatively, consumption of 1-7 drinks appeared to be protective of mortality when total abstainers were used as the reference group, whereas total abstinence appeared to increase the risk of mortality when 1-7 drinks per week was used as the reference group. The total abstainers group also included ex-drinkers which may explain why they were at a significantly elevated risk of mortality when compared to occasional drinkers, i.e. somebody may be an ex-drinker or total abstainer because of an existing medical condition. A significant increase in risk of all-cause mortality was observed in those that reported consuming more than 21 alcoholic drinks per week when either reference group was used. Figure 5 also demonstrates the difference in all-cause mortality risk when using the traditional comparative group of 0 drinks (blue line) and the more recent 1-7 drinks per week (red line). The protective effects of low to moderate drinking is displayed in the area below the 1 in the

Relative Risk axis. This finding has been reported in other studies including a meta-analysis of 34 different studies. (56-58)

Table 34: All-cause mortality by alcohol category for men, adjusted for age, calendar year and smoking (ever vs never), compared to occasional drinkers (1-7 drinks per week) and total abstainers (0 drinks)

Number of drinks per week	Person-Years	Deaths	Compared to 1-7 drinks		Compared to abstainers	
			RMR	95 % C.I.	RMR	95 % C.I.
Nil	80,390	705	**1.15**	**1.04 - 1.28**		
1-7	117,611	716			**0.84**	**0.76 - 0.93**
8-21	139,111	847	1.09	0.99 - 1.21	0.90	0.81 - 1.00
22-35	53,553	446	**1.44**	**1.28 - 1.62**	**1.18**	**1.05 - 1.33**
36-49	28,722	282	**1.70**	**1.48 - 1.95**	**1.40**	**1.22 - 1.61**
50+	24,701	349	**2.28**	**2.00 - 2.59**	**1.86**	**1.64 - 2.12**

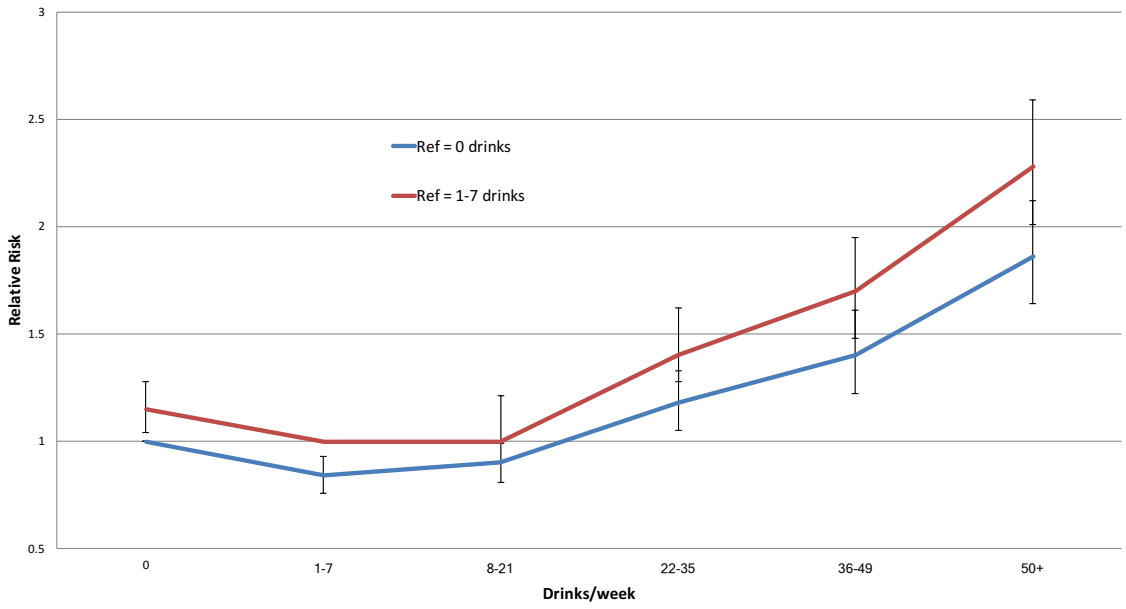

Figure 5: Relative risk of dying (all-cause mortality) for men with different levels of alcohol consumption, compared to those men who never drank alcohol and compared to occasional drinkers, adjusted for age, calendar year and smoking

Table 35 shows the association between alcohol consumption and mortality from ischaemic heart disease using 1-7 dinks per week as the reference group. This analysis was also adjusted for tobacco use, which is an important contributing factor to heart disease. The results show a significant trend of increasing mortality from heart disease with increasing alcohol consumption (P=0.008). There was a statistically elevated risk of mortality from ischaemic heart disease in those consuming more than 35 drinks per week compared with occasional drinkers.

The protective effects of alcohol consumption were not observed in this analysis when using occasional drinkers as the reference group. The previous report used total abstainers as the

reference group and showed a protective effect of alcohol against ischaemic heart mortality up to 35 drinks/week. It is likely that a more sensible approach in analysing the risk of mortality with alcohol consumption is to use occasional drinkers rather than total abstainers as the reference group.

Table 35: Ischaemic heart disease mortality (ICD-10 I20-125) by alcohol category, adjusted for age, calendar year and smoking (ever vs never), compared to those who drink 1-7 alcoholic beverages per week.

Number of drinks/week	Person-Years	Deaths	RMR	95 % C.I.
Nil	80,390	158	**1.32**	**1.05 - 1.66**
Reference	117,611	137		
8-21	139,111	159	1.08	0.86 - 1.35
22-35	53,553	69	1.13	0.84 - 1.51
36-49	28,722	46	**1.41**	**1.01 - 1.97**
50+	24,701	66	**2.12**	**1.58 - 2.84**

Test for heterogeneity P=0.0001 Test for trend P=0.0075

Analyses by Tobacco Smoking and Alcohol Drinking

There is a clear pattern that increasing smoking category is associated with increasing risk of overall mortality, specifically ischaemic heart disease mortality, increasing risk of overall cancer incidence and mortality, and of bladder cancer incidence.

It is clear that all these risks are lower for ex-smokers than continuing smokers.

Moderate drinkers, less than 22 drinks per week, have a similar mortality risk compared to occasional drinkers (1-7 drinks per week). Heavier drinking, more than 21 drinks per week, is associated with increased overall mortality.

There was no reduction in mortality risk from heart disease in those male members of the cohort consuming up to 35 drinks per week compared with occasional drinkers. The opposite was observed in the previous analysis where there was a significant reduction in risk for this group, however, the reference group was total abstainers. This result emphasises the importance of using the correct or most appropriate reference group when doing internal comparisons. By using total abstainers as the reference group, the apparent protective effect of alcohol may be misleading.

5. SPECIFIC CANCERS

5.1. Mesothelioma (ICD-10 C45)

There were 49 mesotheliomas in the cohort. This is a statistically significant excess compared to the general population (SIR 1.60, 95% C.I. 1.18-2.11).
Of the 49 cases of mesothelioma, all but one are now deceased and were identified on the NDI. Of these 48 deaths, 36 were coded as mesothelioma deaths, and five other cases have died but were coded to lung cancer. The remaining seven deaths were coded to various other diseases. There were an additional seven mesothelioma deaths on the NDI not found on the cancer registry, as the death analysis contained additional years of follow up. Mesothelioma deaths before 1997 were coded under the ICD-9 scheme which did not have mesothelioma as a specific code. All cancers have been recoded from ICD-9 to ICD-10 by the cancer registries but the deaths have not been recoded on the NDI.
Mesothelioma risk is strongly related to asbestos exposure. Although the disease is most common in workers who have been heavily exposed, cases do occur in workers whose exposures have been too low to cause asbestosis. Moreover, smoking does not appear to be a risk factor for mesothelioma.(59-61)
Because mesothelioma is nearly always associated with a history of exposure to asbestos, every case should be regarded as significant in itself, irrespective of the statistical significance of the SIR. The occupational histories of the 49 cases of mesothelioma show that 28 of the 49 cases (57%) occurred in refinery workers which make up 39% of the workforce. The dates of hire of these members of the cohort were examined. Nine of the 49 cases entered the industry in the 1950s, 18 in the 1960s, 16 in the 1970s and six in the 1980s and 1990s. Asbestos insulation was used in refineries, particularly in the 1950's and 1960's. This time distribution may be a consequence of measures taken in recent years to eliminate asbestos exposures; on the other hand it may be a consequence of the long induction latency period between exposure and diagnosis of mesothelioma.

5.2. Lung Cancer (ICD-10 C33-C34)

There were 13 cases of lung cancer among women. This rate was slightly higher than the general female population, however, the numbers were small and statistical significance was not achieved. The incidence rate of lung cancer among men in the *Health Watch* cohort is significantly lower than those in the general male population (SIR 0.79, 95% C.I. 0.71 – 0.89). As shown in Table 16 on page 46, the incidence of laryngeal cancer was below that of the general male population, although the reduction was not statistically significant (SIR 0.79, 95% C.I. 0.54-1.11). Cancer of the lip, oral cavity and pharynx was significantly lower than that of the reference population (SIR 0.81, 95% C.I. 0.67-0.97). Chronic obstructive pulmonary disease (COPD) was at least as strongly associated with smoking as was lung cancer. Indeed, these diseases (mainly chronic bronchitis and emphysema) are uncommon in non-smokers. The mortality rate from COPD was significantly reduced (131 deaths vs 192 expected, SMR 0.68, 95% C.I. 0.57-0.81). These figures suggest that the low lung cancer rate in the *Health Watch* population is likely to be due to low average lifetime tobacco use compared with the general population.
All jobs for *Health Watch* cohort members from first employment in the industry to the end of the analysis period were assigned a workplace type based on the company site code. Time in a job was measured as the time between each job or until retirement/resignation.
A comparison of lung cancer incidence was also made between refinery workers and all other workers, adjusted for age, calendar period and smoking. The relative risk was similar for the two groups (RIR 1.03, 95% C.I. 0.80 – 1.33). Little difference in lung cancer incidence was

seen between maintenance workers and all other refinery workers The relative risk was similar in the maintenance group, however, the numbers were small and the confidence intervals were wide (RIR 0.93, 95% C.I. 0.57-1.53).

5.3. Melanoma of the Skin (ICD-10 C43)

Melanoma was one of the commonest cancers in the *Health Watch* cohort, second only to prostate cancer in men and breast cancer in women. There were 16 cases of melanoma in women and the risk was similar to the general female population.

There were 438 melanoma cases in men, and the incidence of melanoma was significantly raised (SIR 1.22, 95% C.I. 1.11-1.34). Melanoma mortality, however, was similar to that of the general Australian population (SMR 1.03, 95% C.I. 0.70-1.32). These findings may suggest a screening bias whereby more cases are detected earlier.

Table 36 shows that the melanoma incidence in men was elevated in most workplace types, and was significantly elevated in refinery and terminal work places where the majority of men work. The lack of statistical significance in the other categories may be due to the relatively low numbers of cohort members in these workplaces.

Table 36: Melanoma (ICD-10 C43) incidence in men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population

Workplace Type	Person-Years	Observed	Expected	SIR	95% C.I.
Refinery	158,221	184	142.39	**1.29**	**1.11 - 1.49**
Terminal	155,501	186	145.92	**1.27**	**1.10 - 1.47**
Airport	15,073	18	14.84	1.21	0.72 - 1.92
Onshore production	52,599	34	40.73	0.83	0.58 - 1.17
Offshore Production	17,765	15	13.83	1.08	0.61 - 1.79
Total	399,633	438	357.95	**1.22**	**1.11 - 1.34**

The following three tables analyse melanoma incidence according to period of first employment, categories of duration of employment, and lapse of time between first employment and diagnosis of melanoma. There was no significant difference between categories and no significant trend in any of these analyses.

The highest rate of melanoma incidence was in members of the cohort employed for the shortest period of time, 5-9 years, whilst those employed for 10-24 years had a significantly lower risk of melanoma. The rationale for this is not clear, but it suggests that a causal association with any exposure in the workplace was unlikely.

Table 37: Melanoma (ICD-10 C43) incidence among men by period of first employment, adjusted for age and calendar period of follow-up, compared to those employed after 1985

Period of first Employment	Person-Years	Melanoma	RIR	95% C.I.
Post 1985	66,412	43		
1975-84	153,538	117	0.99	0.69 - 1.43
1965-74	115,757	145	1.20	0.80 - 1.79
1955-64	44,283	88	1.37	0.85 - 2.21
Pre 1954	19,641	45	1.36	0.79 - 2.37

Test for heterogeneity P=0.4597 Test for trend P=0.1030

Table 38: Melanoma (ICD-10 C43) incidence in men by duration of employment, adjusted for age and calendar period of follow-up, compared to those employed for 5-9 years

Duration of Employment	Person-Years	Melanoma	RIR	95% C.I.
5-9 Years	78,103	63		
10-15 Years	85,222	62	**0.68**	**0.48 - 0.97**
15-19 Years	76,017	64	**0.62**	**0.43 - 0.88**
20-24 Years	62,333	56	**0.55**	**0.38 - 0.80**
≥25 Years	97,807	193	0.87	0.63 - 1.21

Test for heterogeneity P=0.0013 Test for trend P=0.8060

Table 39: Melanoma (ICD-10 C43) incidence in men by time since first employment, adjusted for age and calendar period of follow-up, compared to those first employed 5-9 years ago

Time Since First Employment	Person-Years	Melanoma	RIR	95% C.I.
5-9 Years	40,281	14		
10-15 Years	56,701	34	1.24	0.65 - 2.36
15-19 Years	64,057	44	1.06	0.55 - 2.04
20-24 Years	65,132	48	0.87	0.44 - 1.71
≥25 Years	173,456	298	1.06	0.54 - 2.06

Test for heterogeneity P=0.6106 Test for trend P=0.8391

The excess incidence of melanoma varied by the state of the work site, (see Table 40). Men who worked in Queensland had the highest incidence compared to national data, and those who worked in Victoria and South Australia the lowest (numbers are very small in NT). This suggests a link with sun exposure. It should be noted that some workers may not reside in the same state as they work e.g. off shore workers.

Table 40: Melanoma (ICD-10 C43) incidence compared to national rates for men by state of last employment

State	Person-Years	Observed	Expected	SIR	95% C.I.
VIC	128,084	104	110.96	0.94	0.77 - 1.14
NSW	107,729	141	103.20	**1.37**	**1.15 - 1.61**
NT	2,523	<3	2.34	0.43	0.01 - 2.38
QLD	51,818	88	45.79	**1.92**	**1.54 - 2.37**
SA	46,029	40	40.63	0.98	0.70 - 1.34
TAS	3,701	5	3.84	1.30	0.42 - 3.04
WA	59,726	59	51.16	1.15	0.88 - 1.49
All states	399,633	438	357.95	**1.22**	**1.11 - 1.34**

However, as melanoma incidence can vary by state, further analyses were undertaken using state-based rather than national comparison rates. These are presented in Table 41 and show elevated risks in Victoria, Queensland and New South Wales which was not observed in the previous analysis.(23)

Table 41: Melanoma (ICD-10 C43) incidence compared to state specific rates for men by state of last employment

State	Person-Years	Observed	Expected	SIR	95% C.I.
VIC	128,084	104	82.98	**1.25**	**1.02 - 1.52**
NSW	107,729	141	107.07	**1.32**	**1.11 - 1.55**
QLD	51,818	88	63.90	**1.38**	**1.10 - 1.70**
WA	59,726	59	54.36	1.09	0.83 - 1.40
SA	46,029	40	31.87	1.26	0.90 - 1.71

5.4. Prostate Cancer (ICD-10 C61)

Prostate cancer was the most common cancer in men in the *Health Watch* cohort. There were 1072 cases, up from 730 in the previous report.(23) The incidence was again statistically significantly elevated in the cohort (SIR 1.14, 95% C.I. 1.07-1.21). Prostate cancer mortality, however, was the same as that for the general population (SMR 0.94, 95% C.I. 0.79-1.10). These findings may suggest that members of the cohort are screened for prostate cancer at a greater rate than the general population and may indicate a screening bias whereby more cases are detected earlier, but at a stage when treatment is likely to have a more successful outcome.

5.5. Bladder Cancer (ICD-10 C67)

There were 95 bladder cancers in men which is similar to what is expected, compared to the general male population (SIR 0.93, 95% C.I. 0.75-1.14). The non-statistically significant excesses of bladder cancer seen in the 12th *Health Watch* report (SIR 1.07, 95% C.I. 0.89-1.50) (7) and the 13th Report (SIR 1.11, 95% C.I. 0.85-1.43) was no longer evident.(9) Bladder cancer mortality was also lower in the *Health Watch* cohort (SMR 0.88, 96% C.I. 0.61-1.23) which again could suggest that there is a screening bias since employees are often screened at the workplace. The most recent analysis has shown, however, that bladder cancer among drivers remains elevated but not significantly so (SIR 1.46, 95% C.I. 0.92-2.21).

5.6. Kidney Cancer (ICD-10 C64-C66, C68)

There were 100 cases of kidney cancer among men in the *Health Watch* cohort. This analysis, together with the last analysis has shown that the risk of this cancer has dropped compared to the 12th and 13th reports, (7, 9) now suggesting that the risk is similar to the general population (SIR 0.92, 95% C.I. 0.75-1.12). Drivers were again the exception showing an elevated, but not statistically significant increase of risk of kidney cancer (SIR 1.28, 95% C.I. 0.79-1.96).

5.7. Leukaemias (ICD-10 C91-C95)

There were 74 leukaemia cases found in men, 19 more than were identified in the last report. The risk of leukaemia among men is now less than that of the general population rate bordering on being statistically significantly reduced (SIR 0.80, 95% C.I. 0.63-1.00).
There were, 11 new chronic lymphatic leukaemias, eight new acute myeloid leukaemias and one new chronic myeloid leukaemias since the last *Health Watch* report. In individual leukaemia subtypes, the rates were close to, or less than the population rates.

Table 42 shows that there was no significant excess of leukaemia incidence in any workplace type. When cancer or mortality counts are less than three, the observed numbers are not

reported. This is to keep in line with confidentiality practices and reducing the risk for potentially identifying a cohort member.

Table 42: Leukaemia (ICD-10 C91-C95) incidence among men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population

Workplace	Person-Years	Observed	Expected	SIR	95% C.I.
Refinery	158,221	30	37.41	0.80	0.54 - 1.14
Terminal	155,501	35	38.72	0.90	0.63 - 1.26
Airport	15,073	<3	3.95	0.51	0.06 - 1.83
Onshore production	52,599	5	9.38	0.53	0.17 - 1.24
Offshore Production	17,765	3	3.05	0.98	0.20 - 2.87
Total	399,633	75	92.56	0.81	0.64 - 1.02

The following three tables analyse leukaemia incidence according to period of first employment, categories of duration of employment, and lapse of time between first employment and diagnosis of leukaemia. There was no evidence of a trend in the relative incidence in any of these analyses.

Table 43: Leukaemia (ICD-10 C91-C95) incidence by period of first employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to those employed after 1985

Period of first Employment	Person-Years	Leukaemia	RIR	95% C.I.
Post 1985	66,412	4		
1975-84	153,538	18	1.46	0.48 - 4.47
1965-74	115,757	25	1.74	0.53 - 5.65
1955-64	44,283	19	2.15	0.58 - 7.99
Pre 1954	19,641	9	1.90	0.45 - 8.09

Test for heterogeneity P=0.7875 Test for trend P=0.4596

Table 44: Leukaemia (ICD-10 C91-C95) incidence in men by duration of employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to those employed for 5-9 years

Duration of Employment	Person-Years	Leukaemia	RIR	95% C.I.
5-9 Years	78,103	8		
10-15 Years	85,222	11	1.05	0.42 - 2.63
15-19 Years	76,017	14	1.25	0.51 - 3.08
20-24 Years	62,333	13	1.26	0.50 - 3.19
≥25 Years	97,807	28	1.31	0.56 - 3.10

Test for heterogeneity P=0.9598 Test for trend P=0.4683

Table 45: Leukaemia (ICD-10 C91-C95) incidence in men by time since first employment, adjusted for age, calendar period of follow-up and smoking (ever vs never), compared to those first employed 5-9 years ago

Time since first Employment	Person-Years	Leukaemia	RIR	95% C.I.
5-9 Years	40,281	3		
10-15 Years	56,701	5	1.02	0.23 - 4.48
15-19 Years	64,057	5	0.82	0.17 - 3.88
20-24 Years	65,132	10	1.40	0.31 - 6.33
≥25 Years	173,456	52	1.41	0.31 - 6.49

Test for heterogeneity P=0.8664 Test for trend P=0.4507

5.8. Multiple Myeloma (ICD-10 C90)

There were 46 multiple myeloma cases in men in the cohort. The incidence was again not statistically significantly higher than that in the general population (SIR 1.05, 95% C.I. 0.77-1.40). As shown in Table 46, multiple myeloma was slightly elevated amongst terminal workers and slightly elevated overall (Table 16), however, these elevations were not significant.

Table 46: Multiple myeloma (ICD-10 C90) incidence among men by workplace type, adjusted for age and calendar period of follow-up, compared to the Australian population

Workplace	Person-Years	Observed	Expected	SIR	95% C.I.
Refinery	158,221	19	17.71	1.07	0.65 - 1.68
Terminal	155,501	23	18.45	1.25	0.79 - 1.87
Airport	15,073	<3	1.90	-	-
Onshore production	52,599	4	4.39	0.91	0.25 - 2.33
Offshore Production	17,765	<3	1.44	0.69	0.02 - 3.87
Total	399,633	47	43.91	1.07	0.79 - 1.42

Comments on Specific Cancers in Men

Mesotheliomas, melanoma of the skin and prostate cancer all occurred in statistically significant excess in men in the Health Watch cohort compared to national rates.

There was a statistically significant reduction in lung cancer, liver cancer and cancers of the lip, oral cavity and pharynx and of deaths from COPD which is probably a result of less tobacco consumption by members of the cohort than by the general Australian population.

There was a statistically significant increase in the incidence for melanoma among men in the cohort. For three states, the rate was significantly elevated when compared to relevant state rates. The rate did not, however, increase with increasing duration of employment. On this basis it is unlikely that the excess was caused by a factor in the workplace in this industry.

Bladder and kidney cancers in the cohort remained similar to the general population, as does multiple myeloma. Prostate cancer incidence in the cohort was statistically significantly higher than in the general population, however prostate cancer mortality remained similar to that of the general population.

There was no excess incidence of any leukaemia or of any leukaemia subtype.

6. HEALTH OUTCOMES IN SPECIFIC JOBS

The ability to assess risk in particular jobs as defined by their API Job code is recognised to be a useful method of assessing risk in the industry e.g. refinery workers may have very different jobs having different exposures. However, analysing by API Job code is limited by the numbers of employees in any particular job.
The job groups analysed in this report are:

- *Drivers* (NB295x)
- *Refinery operators* (not including ship personnel) (BA, BB, BC, HX, IB, PA, PB, PC, RF)
- *Terminal operators* (BA, BB, BC, HX, IB, NA, PA, PC, RF) and *Maintenance* (refinery or terminal based not upstream) (IB, CA, CB, CC, CD, CE, CF, DA, DB, DC, DD, EA, EB, EC, FA, FB, GX, MX)
- *Office workers* (AX)

Shift workers was no longer used as a category in this analysis as it self-reported at interview and has not consistently been updated during company updates. Each person's full job history since 1980 was checked and categorised according to whether the person has ever held the particular job classification. Those who have held more than one category appear in both categories in the analysis, so their deaths and person years are counted more than once.
Analysis has been done for some major health outcomes including all-cause mortality, ischaemic heart disease, cancer mortality, and deaths by external cause in addition to cancer incidence. For many other health outcomes, numbers were too low to be reliable.

6.1. All-cause Mortality in Men by Job Group

As shown in Table 47, all-cause mortality for each of these occupations was similar to the all-cause-mortality for the male members of the whole cohort (SMR 0.77, CI 0.75-0.80). Those members who ever worked in offices show an even lower mortality rate.

Table 47: All-cause mortality among men by Job Group (ever held), adjusted for age and calendar period of follow-up, compared to the Australian population

Job	Person-Years	Observed	Expected	SMR	95% C.I.
Driver	62,403	526	611.94	**0.86**	**0.79 - 0.94**
Refinery	88,554	609	818.72	**0.74**	**0.69 - 0.81**
Terminal	58,888	556	572.50	0.97	0.89 - 1.06
Maintenance	105,431	880	1026.75	**0.86**	**0.80 - 0.92**
Office	155,628	1,066	1,564.52	**0.68**	**0.64 - 0.72**

6.2. Cancer Incidence in Men by Job Group

There remained an increase in all-cancer incidence in drivers which was significantly higher than that of the general population (SIR 1.15, 95% C.I. 1.06-1.24), Table 48. This increase was also significant in the last report. (9)

Table 48: Cancer incidence among men by Job Group (ever held), adjusted for age and calendar period of follow-up, compared to the Australian population

Job	Person-Years	Observed	Expected	SIR	95% C.I.
Driver	56,666	598	522.17	**1.15**	**1.06 - 1.24**
Refinery	79,952	724	682.75	1.06	0.98 - 1.14
Terminal	53,319	471	462.03	1.02	0.93 - 1.12
Maintenance	95,251	757	801.00	0.95	0.88 - 1.01
Office	140,111	1208	1243.56	0.97	0.92 - 1.03

6.2.1. Incidence of Cancer and Mortality among Drivers

Table 49 presents the cancer incidence rates for selected major anatomical sites in drivers. There was a small elevation in the total cancer incidence rate among drivers (SIR 1.15, 95% C.I. 1.06-1.24). Although most of the broad cancer categories were somewhat raised among drivers, the only increased cancer risk that was statistically significantly raised was melanoma (SIR 1.35, 95% C.I 1.05-1.69) and prostate cancer (SIR 1.19, 95% C.I. 1.02-1.38). Prostate cancer was elevated in the 14th Report but was not statistically elevated.
There were small numbers in many of the individual cancer types which resulted in wide confidence intervals and unstable point estimates. Cancer of the oesophagus was in excess, (SIR 1.80, 95% C.I. 0.98-3.01) as was cancer of the bladder (SIR 1.46, 95% C.I. 0.92-2.21) but these excesses were not statistically significant. Because there were only 14 and 22 cases respectively, it was not possible to conduct a meaningful analysis in terms of time-related factors.
The lung cancer rate in drivers was lower than that of the general population.

Comparisons of specific cancers and mortality were conducted between cohort members who were ever a tanker driver and those who only ever worked in an office and adjusted for age, calendar period and smoking (Table 50 and Table 51).
All-cause mortality and cancer mortality were similar in these two groups and compared similarly to the results in the 14th Report. Mortality from external causes was statistically elevated among drivers compared with office-only workers (SMR 2.12, 95% C.I. 1.21-3.72). An excess was also observed in the 14th Report but this excess was not statistically elevated. The risk of all-causes cancer in drivers compared with office-only workers have increased since the last report and has now almost reached significance (SIR 1.11, 95% C.I. 0.99-1.25). Leukaemia and bladder cancer again showed a statistically elevated rate in drivers compared to office-only workers, however, the number of cases were small and the confidence intervals were wide.

Table 49: Cancer incidence by selected anatomical site, for drivers by ICD-10 codes, adjusted for age and calendar period of follow-up, compared to the Australian population.

Malignant neoplasm of:	Observed	Expected	SIR	95% C.I.
Lip, Oral cavity and Pharynx	24	22.82	1.05	0.67 - 1.56
Oesophagus	14	7.80	1.80	0.98 - 3.01
Stomach	13	12.33	1.05	0.56 - 1.80
Colon	48	42.54	1.13	0.83 - 1.50
Rectum	36	29.45	1.22	0.86 - 1.69
Liver	4	6.97	0.57	0.16 - 1.47
Gallbladder	<3	2.63	0.76	0.09 - 2.75
Pancreas	12	10.60	1.13	0.59 - 1.98
Larynx	6	6.40	0.94	0.34 - 2.04
Lung	53	58.89	0.90	0.67 - 1.18
Melanoma	72	53.52	**1.35**	**1.05 - 1.69**
Mesothelioma	7	4.68	1.50	0.60 - 3.08
Connective Tissue	3	2.89	1.04	0.21 - 3.04
Prostate	172	144.72	**1.19**	**1.02 - 1.38**
Testis	5	2.67	1.87	0.61 - 4.37
Bladder	22	15.05	1.46	0.92 - 2.21
Kidney	21	16.35	1.28	0.79 - 1.96
Eye	<3	1.38	1.45	0.18 - 5.23
Brain & Nervous System	10	7.68	1.30	0.62 - 2.39
Thyroid	5	3.08	1.62	0.53 - 3.79
Non-Hodgkin lymphoma	17	19.57	0.87	0.51 - 1.39
Multiple myeloma	6	6.61	0.91	0.33 - 1.98
Leukaemia	16	13.76	1.16	0.66 - 1.89
Acute lymphatic leukaemia	<3	0.49	-	-
Chronic lymphatic leukaemia	8	6.08	1.32	0.57 - 2.59
Acute myeloid leukaemia	3	3.76	0.80	0.16 - 2.33
Chronic myeloid leukaemia	<3	1.41	0.71	0.02 - 3.96
Other leukaemia	4	2.03	1.97	0.54 - 5.05
Unspecified cancer site	13	13.86	0.94	0.50 - 1.60
Myelodysplastic Syndrome	3	2.98	1.01	0.21 - 2.95
Other sites	10	11.98	0.83	0.40 - 1.54
All Malignant	598	522.17	**1.15**	**1.06 - 1.24**

Table 50: Relative mortality risk of Ever Drivers compared with Only Ever Office workers.

	Job Type	Current Analysis				14th Report	
		Person-Years	Deaths	RR	95 % C.I.	RR	95 % C.I.
All-cause Mortality (RMR)	Office only	61,338	609				
	Driver ever	62,398	525	1.07	0.95 - 1.21	0.99	0.85 - 1.14
Cancer Mortality (RMR)	Office only	61,338	242				
	Driver ever	62,398	217	1.03	0.85 - 1.24	0.92	0.73 - 1.16
IHD Mortality (RMR)	Office only	61,338	113				
	Driver ever	62,398	95	1.12	0.84 - 1.48	0.99	0.73 - 1.35
External Mortality (RMR)	Office only	61,338	19				
	Driver ever	62,398	37	**2.12**	**1.21 - 3.72**	1.41	0.74 - 2.70

Table 51: Relative cancer incidence of Ever Drivers compared with Only Ever Office workers

	Job Type	Current Analysis				14th Report	
		Person-Years	Cancer	RR	95 % C.I.	RR	95 % C.I.
All Cancer (RIR)	Office only	55,264	591				
	Driver ever	56,665	598	1.11	0.99 - 1.25	1.00	0.88 - 1.15
Prostate Cancer (RIR)	Office only	55,264	188				
	Driver ever	56,665	172	0.94	0.76 - 1.16	0.81	0.63 – 1.04
Leukaemia (RIR)	Office only	55,264	7				
	Driver ever	56,665	16	**2.70**	**1.09 - 6.70**	**5.52**	**1.19 - 25.49**
Kidney Cancer (RIR)	Office only	55,264	15				
	Driver ever	56,665	21	1.44	0.73 - 2.82	1.80	0.84 - 3.86
Bladder Cancer (RMR)	Office only	55,264	9				
	Driver ever	56,665	22	**3.20**	**1.45 - 7.07**	**2.42**	**1.09 - 5.37**

6.3. Cancer Mortality in Men by Job Group

As shown in Table 52, there were no excesses in all-cancer mortality in any of the occupational groups studied including drivers. The cancer mortality rates were significantly lower than the population rates in refinery and office workers and were similar to the national rate for all other job groups. Cancer mortality in the drivers group was statistically lower than national rates in the previous report.(23)

Table 52: Cancer mortality in men by Job Group (ever held), adjusted for age and calendar period of follow-up, compared to the Australian population

Job	Person-Years	Observed	Expected	SMR	95% C.I.
Driver	62,403	217	230.11	0.94	0.82 - 1.08
Refinery	88,554	268	303.78	**0.88**	**0.78 - 0.99**
Terminal	58,888	221	208.59	1.06	0.92 - 1.21
Maintenance	105,431	347	366.87	0.95	0.85 - 1.05
Office	155,628	453	568.20	**0.80**	**0.73 - 0.87**

6.4. Ischaemic Heart Disease (ICD-10 I20-125) Mortality in Men by Job Group

Ischaemic heart disease mortality was significantly lower in the cohort as a whole, compared to the general population (SMR 0.75, 95% C.I. 0.69-0.81).
Table 53 shows that this decrease was also significantly reduced in drivers, refinery workers, and office workers whilst terminal workers were in excess and borderline significantly increased.

Table 53: Ischaemic heart disease (ICD-10 I20-125) mortality in men by Job Group (ever held), adjusted for age and calendar period of follow-up, compared to the Australian population

Job	Person-Years	Observed	Expected	SMR	95% C.I.
Driver	62,403	96	119.19	**0.81**	**0.65 - 0.98**
Refinery	88,554	99	158.35	**0.63**	**0.51 - 0.76**
Terminal	58,888	133	112.99	1.18	0.99 - 1.39
Maintenance	105,431	198	203.07	0.98	0.84 - 1.12
Office	155,628	194	306.67	**0.63**	**0.55 - 0.73**

6.5. Mortality from Accidents and Violence (ICD-10 V00-V99, W00-W99, X00-X99, Y00-Y99) in Men by Job Group

As shown in Table 54, mortality rates from accident/violence in each of the occupational groups analysed was lower than the general population and was similar to that of the cohort as a whole (SMR 0.68, 95% CI 0.59 - 0.78). The mortality rate was highest in the drivers group but similar to the national rates (SMR 0.94, C.I. 0.66-1.29).

Table 54: Mortality from accident/violence (ICD-10 V00-V99, W00-W99, X00-X99, Y00-Y99) in men by Job Group (ever held), adjusted for age and calendar period of follow-up, compared to the Australian population

Job	Person-Years	Observed	Expected	SMR	95% C.I.
Driver	62,403	37	39.53	0.94	0.66 - 1.29
Refinery	58,888	29	38.64	0.75	0.50 - 1.08
Terminal	88,554	44	56.62	0.78	0.56 - 1.04
Maintenance	105,431	44	70.77	**0.62**	**0.45 - 0.83**
Office	155,628	46	102.89	**0.45**	**0.33 - 0.60**

Results for Men by Job Group in Health Watch

Overall mortality rates were lower for men in Health Watch compared with the general population rates in each of the occupational groups studied particularly in office workers. These decreased rates were statistically significant for all groups except terminal operators. Cancer mortality rates were significantly lower in refinery and office workers and similar to the national rates for all other job categories.

Drivers also exhibited a statistically significant increased risk of external causes such as accidents and suicides, compared with office-only workers, while all-cause mortality remained the same between the groups.

Cancer incidence for all job groups was similar to that of the population as a whole except for drivers where the risk of cancer was statistically significantly elevated. Leukaemia, and bladder cancers were significantly elevated in the driver group compared with office-only workers but incidence numbers were small and confidence intervals wide. These cancers were not elevated when compared to the general population.

When compared to the general population, mortality rates from heart disease were significantly lower in drivers, refinery workers and office workers. In terminal operators and maintenance workers the risk was similar to that of the general population

Death from external causes was statistically significantly lower than that of the general population for maintenance and office workers. All other job groups had non-significantly lower rates compared with the general population.

7. DISCUSSION

7.1. Strengths and Weaknesses of the Study

7.1.1. Individual Interview Data

A major strength of *Health Watch* is that there is at least one personal interview record for every subject in the cohort. Written consent has been obtained from members of the cohort to search for their names in periodic searches of the Australian cancer registry data. The interview-based data provides considerable detail about jobs and tasks performed in the industry. It also means that detailed smoking history and alcohol intake is available for each subject although much of this data was collected many years ago and smoking rates may have changed over time.

7.1.2. High Participation Rate

Participation in *Health Watch* was voluntary. This could cause one source of volunteer bias if those motivated to participate had a different health status from non-participants. This is not likely given that recruitment was an active process and participation rates were so high. Site rolls were provided to the survey interviewers, and each individual approached and invited to participate. Refusal to participate was uncommon, and the reason for the missing employees is in most cases difficulty in locating them through temporary absence such as shift work or annual leave. The high participation rates (93%) in the first two surveys make volunteer bias very unlikely. The participation rates were lower in the Third and Fourth surveys (estimated at 84% and 73% respectively). The latter resulted in a lack of recruits to the cohort in the Fourth Survey from offshore production,(7) although this did not significantly alter the composition of the cohort: 4.0% of the cohort were in the offshore production sector, prior to the Fourth Survey and 3.7% afterwards.

7.1.3. Volunteer Bias

Another source of volunteer bias could be the ability of employees to volunteer to participate after becoming ill; that is members of the cohort could have initially refused to participate in a *Health Watch* survey, but having then developed a disease, could then volunteer to participate in a later survey. This could cause an upward bias, i.e. an overestimate, of the mortality rate, but since all mortality rates of all major disease categories and of most individual cancers were lower than expected, this is unlikely to have caused any misleading results. Joining the *Health Watch* cohort after developing cancer cannot affect the cancer analysis, because follow-up time does not commence until the person becomes a cohort member (this is at interview or after five years in the industry whichever is the later). Cancers occurring before this point are excluded from the analysis.

7.1.4. Unverified Date of Employment

A potential weakness of the study is that the date of first employment for members of the cohort is obtained from members at the time of interview. This could affect analyses by time-related variables, i.e. period of employment, duration of employment and time since employment. Unfortunately, the personnel records of most companies have been overhauled in recent years, making access to records from the era when most members of the cohort were first employed very difficult. It has therefore been judged impractical to conduct an audit of the date of employment obtained at interview against dates from company records. Nevertheless, error is likely to be random and hence unlikely to lead to bias. Moreover, errors from imperfect recollection of the year of hire are likely to be small in relation to the size of time-related

categories (e.g. period of employment categories are pre-1954, 1955-64, 1965-74, 1975-84 and post-1985).
Date of termination of employment was obtained from participating companies records. Even here, however, information was not always complete. An audit of those classified as still employed by participating companies disclosed that many were no longer employed.(7) Following a further check of company employment records and other follow-up measures, the errors from this source were minimised.(7) In recent years, company update information has not always been readily forthcoming. A proportion of employment histories may not be up to date for those whose employing companies did not provide complete company update information and for those employees who did not return the latest Health Letter in 2014.

7.1.5. Complete Cancer Identification

Identification of cancer is a major strength of the study as cancer registration is mandatory in all Australian states and territories, and registration is virtually complete. However complete matching cannot be guaranteed due to privacy restrictions in releasing uncertain matches, and some problems have occurred in reconciling information from the ACD held by the AIHW and the state cancer registries which supply the information to it.(62) This has been discussed in Section 2.4.4 (page 28).

7.2. The Healthy Worker Effect (HWE)

In the Australian petroleum industry the *healthy worker effect* is very strong with SMRs for workers in the industry lower than many reported from other occupational cohorts.(63) This may be partly because workers must serve for five years in the industry before entering the cohort. This is a longer qualifying period than for many other occupational cohorts. There is an argument for using a reference population composed of workers with similar demographic characteristics including the likelihood of obtaining and retaining employment rather than the general population.(64)
New research also suggests that higher amounts of overall sitting time or an inactive lifestyle are positively associated with increased overall mortality (65, 66). Many jobs recorded in this cohort are largely active with minimal sedentary periods which also support the strong healthy worker effect in the petroleum industry.
Figure 6 presents the SMRs and SIRs for men in *Health Watch* over time. This analysis was compiled with all new data obtained from the most recent linkage with the cancer and death registries and can only be compared to figures from the 14th *Health Watch* report. All reports prior to the 14th *Health Watch* report simply added the extra years of data rather than re-analysing the entire cohort period with the new available data. A common finding with the *healthy worker effect* is that it decreases as the cohort ages, that is, the SMR tends to increase with time, approaching the general mortality rates of the population. This tendency is becoming evident for men shown by the trend lines in Figure 6. The SIRs for men in the *Health Watch* cohort are increasing and are also becoming closer to population data.

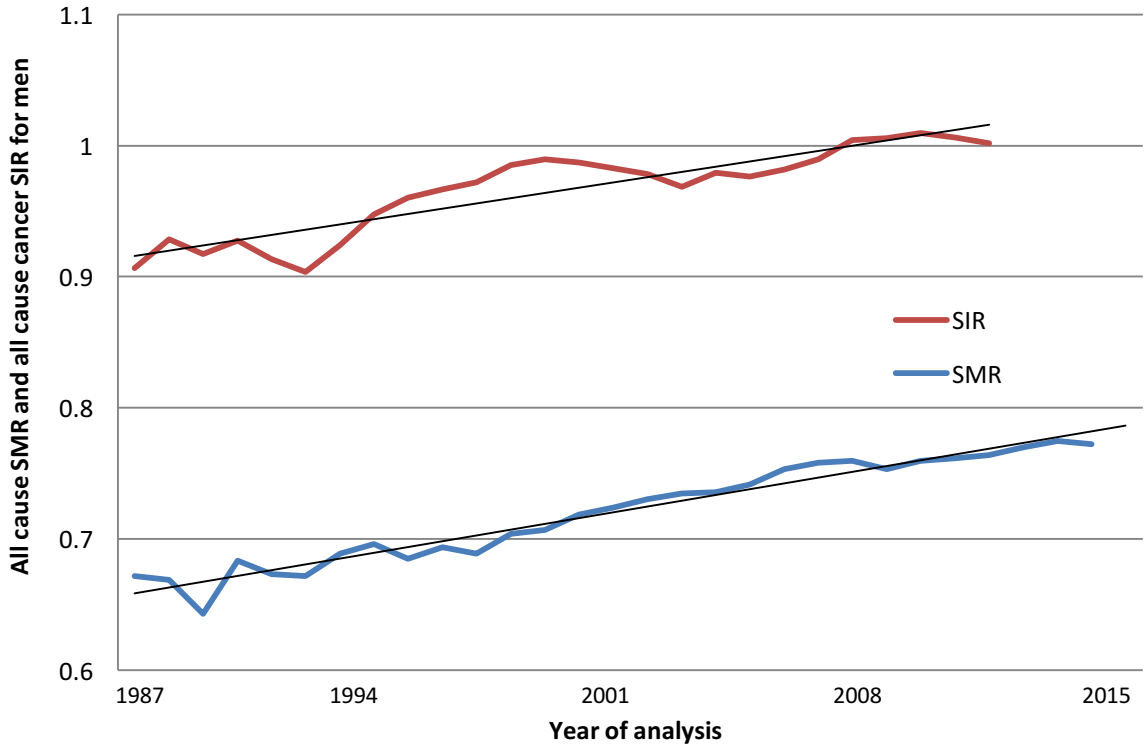

Figure 6: All Cause SMR, all cancer SIRs for men in *Health Watch*, plotted at yearly intervals

The SMRs and SIRs for women were based on very few cases, particularly in the earlier reports. No SIRs for women presented in previous reports have been significantly different to that of the general population but this may be a result of the small numbers.

7.3. Mesothelioma and other Asbestos-related Conditions

There are a number of self-reports of asbestos-related conditions in members of the *Health Watch* cohort such as pleural plaques and asbestosis. Eleven cohort members have died from asbestosis. In addition, the occurrence of 49 cases of mesothelioma was an indication of past asbestos exposure and is consistent with the findings of other studies in oil refinery workers.(67-70) Of course, it is possible that some of these cases were attributable to asbestos exposure prior to entering the petroleum industry but asbestos insulation was used in refineries in particular in the 1950s and 1960s. There was a long latency period between initial exposure to asbestos and occurrence of the disease.(71, 72) Stringent regulations to prevent asbestos exposure have been in place for some years and there is now a much greater awareness of the hazards of asbestos, so that recent exposures are likely have been much less than that which occurred in the 1950s and 1960s. Nevertheless, mesotheliomas can occur after quite low exposures, and it is important that any potential sources of exposure be identified and removed or controlled.

7.4. Lung Cancer

The low lung cancer rate may appear unexpected given the fact that the standardised prevalence of smoking in the cohort was only slightly less than that of the Australian male population when the data was collected (see 4.1.1). However lung cancer risk is also predicted by factors such as the number of cigarettes smoked, age at starting, age at quitting and tar content.(54, 73) Thus although the prevalence of current smokers was similar in the *Health Watch* cohort and the general male population, it is quite possible that the average lifetime tobacco consumption

in the *Health Watch* cohort is much less. This could be because *Health Watch* smokers on average may smoke less than other Australian men, or those who have quit may have done so at an earlier age than in the general population, or if more have quit since their data were collected (Table 25). These factors could be an explanation for the low lung cancer incidence in the *Health Watch* cohort.

Evidence of the low average lifetime tobacco use in the *Health Watch* cohort may also come from analyses relating to other diseases strongly related to smoking such as cancers of the lip, oral cavity and pharynx, laryngeal cancer and chronic obstructive pulmonary diseases which are lower than in the general population. These figures suggest that the low lung cancer rate in the *Health Watch* population is likely to be due to low average lifetime tobacco use compared with the general population.

The occurrence of a number of cases of mesothelioma in the *Health Watch* cohort, and in other studies of oil refinery workers, raises the possibility of an increased asbestos-related lung cancer risk. The concurrence of increased mesothelioma incidence with low lung cancer mortality rates, has been found in several studies in the petroleum industry, (74-77) although other studies have failed to confirm these findings.(78, 79) In some of these studies, the reduced risk of lung cancer could be attributed to lower smoking rates, many studies are confounded by lack of smoking data. Given the low overall lung cancer incidence in *Health Watch* there could be some asbestos-related lung cancers in refinery workers but the number must be small.(68, 74, 80)

This suggestion was investigated by comparing the lung cancer rate in refinery workers compared to the rest of the *Health Watch* cohort (because asbestos exposure was more likely in refineries than other worksites) and within the refinery workers' group by comparing maintenance workers with that in non-maintenance workers (because maintenance workers are more likely than other work groups to have come into contact with the asbestos). An advantage of such an analysis in this cohort is that smoking data, based on individual histories obtained prospectively from every subject in the cohort, are available. The analyses showed that after adjusting for smoking status, there was a minimal increase (not statistically significant) in lung cancer incidence among refinery workers compared to non-refinery workers (SIR 1.03, 95% C.I. 0.80-1.33); and within refineries there was a non-significantly reduced risk for lung cancer incidence in maintenance workers compared to non-maintenance workers within refineries (SIR 0.93, 95% C.I. 0.57-1.53). The analyses were based on small numbers of cancers however.

7.5. Melanoma

Figure 7 presents the SIRs and the cumulative incidence of melanoma among men in *Health Watch*. The analysis examined the state of the cohort every year since 1987 against data from the relevant time period. The risk of melanoma among men has remained significantly higher than that of comparable Australian national rates. As the number of cases increases the confidence intervals become narrower as shown in Table 55.

The significance of the excess melanoma incidence among men in the *Health Watch* cohort is not clear, but no causal association with the workplace is apparent because there was no trend for increasing risk with increasing duration of employment.

Table 40 (page 65) shows that melanoma rates, when compared to national data, vary with state. It is of interest to note that although melanoma incidence was elevated in the cohort, melanoma mortality was the same as that for the general population (SMR 1.03, 95% C.I. 0.79-1.32). These findings may suggest that members of the cohort are screened for melanoma at a greater rate than the general population.

Table 55: Melanoma (ICD-10 C43) risk over time for men in the ***Health Watch*** cohort

Melanoma	1987	1990	1993	1996	1999	2002	2008	2012
Cases	24	48	82	122	176	224	338	438
SIR	1.25	1.26	**1.34**	**1.34**	**1.38**	**1.31**	**1.23**	**1.22**
95 % C.I.	0.80 - 1.86	0.93 - 1.67	**1.07 - 1.67**	**1.12 - 1.60**	**1.19 - 1.60**	**1.15 - 1.50**	**1.10 - 1.36**	**1.11 - 1.34**

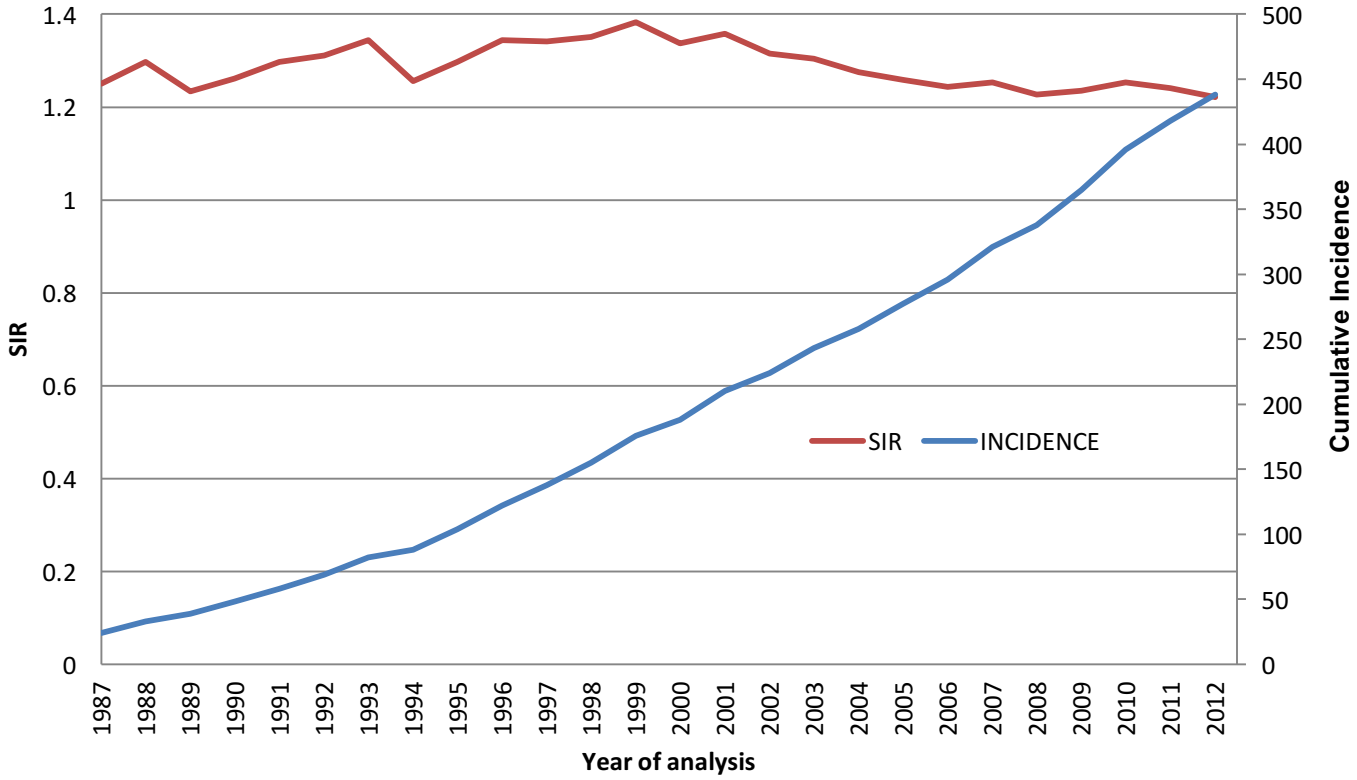

Figure 7: SIRs and cumulative melanoma incidence in men from 1987-2012

A statistically significant excess of melanoma mortality has been reported in UK refinery workers(81, 82) and a non-significant excess in USA refinery workers.(83) The highest SMR for melanoma was reported for Imperial Oil Limited (IOL) employees at upstream operations in Canada: SMR 6.00, 95% C.I. 2.19-13.06.(75) The only other cancer incidence study in the industry (an IOL cohort which overlapped with that reported previously), showed a non-significant excesses of melanoma in both men (SIR 1.25, 95% C.I. 0.82-1.83), and women (SIR 1.46, 95% C.I. 0.83-1.27).(70)

Thus an excess of melanoma in this industry is not unusual, but no occupational cause was apparent. Apart from the well-established association with exposure to solar radiation, melanoma has a tendency to occur in higher socioeconomic groups.(84) There is no reason to suspect socioeconomic status to be of special significance in this cohort, which has mostly excluded senior management.

7.6. Bladder Cancer

Bladder cancer was found in significant excess in the 11th Report (SIR 1.37, 95% C.I. 1.00-1.83).(28) In subsequent reports, the excess was smaller and not statistically significant with the rates being almost on par in last report (SIR 1.01, 95% C.I. 0.81-1.26). The current results

show that bladder cancer risk continues to decrease and is now lower compared to the general population (SIR 0.88, 95% C.I. 0.61-1.23).
Bladder cancer incidence was again found to be significantly elevated among drivers compared with office-only workers, however, the number of cases was small and the confidence intervals were wide (RIR 3.2, 95% C.I. 1.45-7.07).

7.7. Leukaemia

Leukaemia has been a cancer of special concern in this industry because of its association with benzene exposure. The analyses early in the history of the *Health Watch* cohort indicated an excess of LH cancers among men. This is a broad category which includes the leukaemias, multiple myeloma and non-Hodgkin lymphoma, but not Hodgkin disease. Subsequent analyses showed that the excess was mainly from leukaemia cases.(3-5, 29) In our serial reanalyses, the excess was more than 2 fold in 1987 and 1990, and has been reducing over time. The incidence is now lower than the general population and is reaching statistical significance. The successive leukaemia incidences are shown in Table 56 and in Figure 8.

Table 56: Leukaemia incidence for men reported over time in the *Health Watch* cohort

Leukaemia	**1987**	**1990**	**1993**	**1996**	**1999**	**2002**	**2008**	**2012**
Observed	9	16	18	24	30	37	56	74
SIR	2.07	**1.90**	1.30	1.16	1.01	0.90	0.80	0.80
95% C.I.	0.94 - 3.92	**1.09 - 3.09**	0.77 - 2.06	0.74 - 1.72	0.68 - 1.45	0.64 - 1.24	0.61 - 1.04	0.63 - 1.00

It can be seen that there is now no significant excess of leukaemias in the *Health Watch* cohort, in fact, the incidence is lower than the general population rate and is bordering on significance. Moreover, internal analysis within the cohort showed no significant trend in leukaemia incidence with duration of employment. This finding is not unexpected, since "leukaemia" is not a single disease, but a composite of leukaemia types which are in fact different disease entities. Of these, only acute myeloid leukaemia is commonly associated with benzene exposure, however there is some data suggesting that CLL may be associated with benzene exposure.(16, 85, 86)
A number of case-control studies in the industry have been published. In a study of petroleum marketing and distribution workers in the UK, the authors concluded that there was some suggestion of a relation between exposure to benzene and myeloid leukaemia, particularly acute myeloid leukaemia.(31) A case-control study nested within the Canadian cohort in 1996 showed a relationship between duration of exposure to benzene and risk of leukaemia but no association between increasing benzene exposure and risk of leukaemia, but the power of the study was low.(87) On the other hand the nested case-control study in the *Health Watch* cohort has found a strong association with increasing benzene exposure.(13, 14, 16) CONCAWE funded a combined study of three petroleum industry case-control studies. This found no convincing evidence for an association between benzene exposure and AML or other leukaemia subtypes but identified that MDS may be associated with benzene exposure.(22)

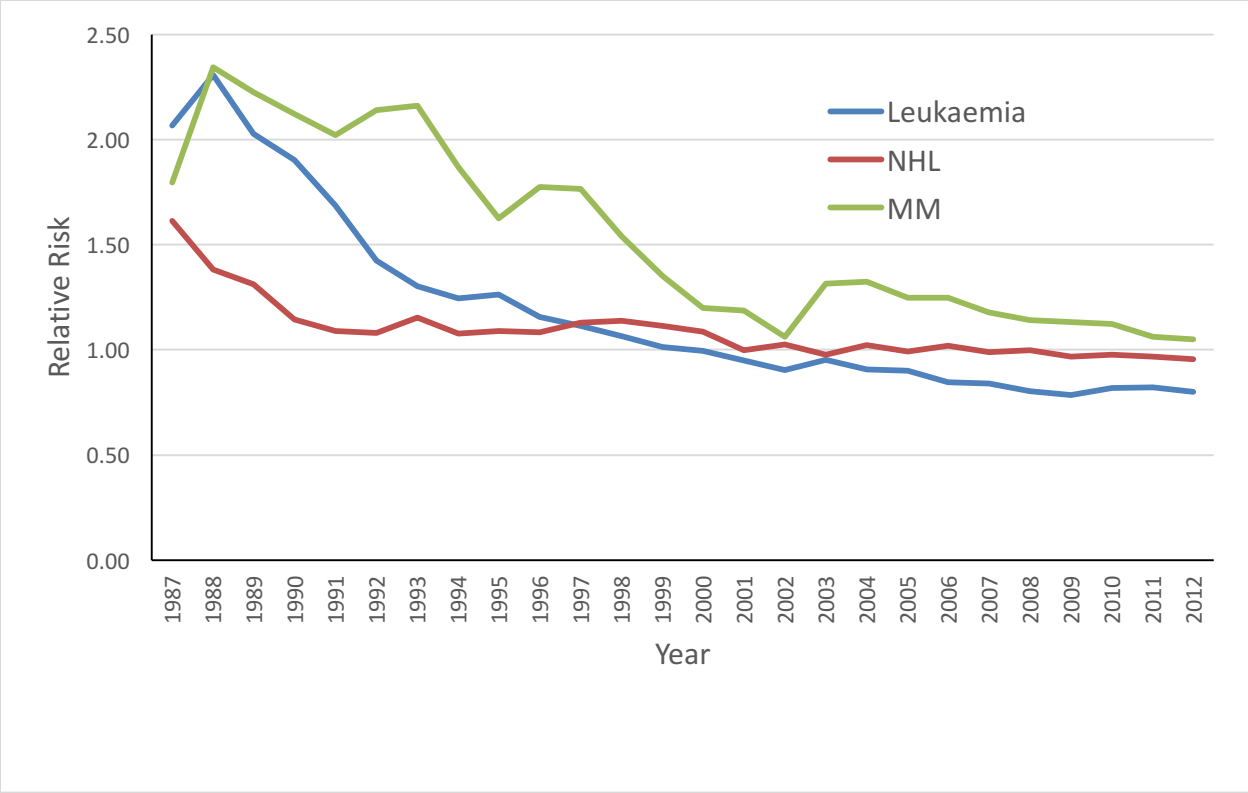

Figure 8: SIRs for leukaemia, NHL and MM in men from 1987 - 2012

7.8. Other Lympho-Haematopoietic Cancers in Men

Other cancers of the blood and bone marrow have been of interest in the *Health Watch* study because of an apparent excess of these conditions in earlier years of follow-up. At that time these conditions were grouped together because the numbers in the specific diseases in this category were very low. In more recent years, non-Hodgkin lymphoma (NHL), multiple myeloma (MM), and leukaemia have been analysed separately as there were larger number of cases as the cohort ages.

Table 57 presents the re-analyses of NHL and MM incidence at various time points. This table shows that the SIRs for both NHL and MM have been declining steadily over the years. There was a statistically elevated rate of MM in 1993, however, the number of cases in the earlier years were small. The steady decline in rates is also shown in Figure 8.

Excess rates of MM have been reported in some studies but not others and there has been much debate in the literature about whether exposure to benzene is associated with increased risk of MM.(88-92)

Table 57: NHL (ICD-10 C82-C85, C96) and MM (ICD-10 C90) incidence for men in the *Health Watch* cohort from 1987 to 2012

		1987	1990	1993	1996	1999	2002	2008	2012
NHL	Cases	10	14	24	34	49	61	98	125
	SIR	1.61	1.14	1.15	1.08	1.11	1.02	1.00	0.96
	95% C.I.	0.77-2.96	0.63-1.92	0.74-1.72	0.75-1.51	0.82-1.47	0.78-1.32	0.81-1.22	0.80-1.14
MM	Cases	3	7	12	15	17	19	36	46
	SIR	1.80	2.12	**2.16**	1.78	1.35	1.06	1.14	1.05
	95% C.I.	0.37-5.25	0.85-4.37	**1.12-3.77**	0.99-2.93	0.79-2.16	0.64-1.66	0.80-1.58	0.77-1.40

7.9. Myelodysplastic syndrome (MDS)

Benzene is a well-known haematotoxin and leukaemogen at a relatively high level of exposure (93-95). In 2017, the International Agency for Research on Cancer (IARC) reconfirmed that benzene causes the acute myeloid leukaemia (AML) subtype in humans and also noted that benzene is likely to be related to other leukaemia subtypes and lymphoid neoplasms.(96) However, some meta-analyses differ in their interpretation of whether previous literature suggests a consistent relationship between benzene and lymphoid neoplasms.(88, 97-99)

A review using meta-regression to examine dose-response relationships suggests that benzene exposure less than 50 ppm-years results in a statistically significant elevated risk of all leukaemias in aggregate.(100) But few quantitative studies have examined risks between specific leukaemia subtypes and exposure to lower concentrations of benzene.(85) There is also sparse literature on specific myeloid tumors, such as myeloproliferative disorders (MPD) and myelodysplastic syndrome (MDS), which can precede and evolve into AML. MDS covers a group of hematopoietic malignancies that have become recognised relatively recently and so has been under-reported in the past. IARC (96) did not mention these myeloid tumours in their recent evaluation of benzene carcinogenicity. The CONCAWE-funded combined study, however, found an association between benzene exposure and MDS at low exposure levels.(22) Recognition of MDS as a discrete disease emerged slowly over the last century with vague and imprecise terminology often used to describe cases of MDS, such as “pre-leukemia,” “subacute leukemia,” “atypical leukemia” etc.(101) Approximately 20% of MDS patients will progress to AML but the two diagnoses are distinct diseases.(101) MDS was first defined in 1976 (102) and the diagnosis formalised in 1982.(103) In 2001, the World Health Organization included it in their LH cancer classification scheme.(104)

MDS was considered a reportable cancer by many cancer registries from 2001 but the reporting is not considered reliable in Australia until 2003 allowing only 10 years of analyses in this report. There were 24 cases recorded in this time frame producing an elevated, but not statistically significant risk in the cohort population (SIR 1.17, 95% C.I 0.75-1.74). The numbers are still quite small and this cancer will be monitored closely in future analyses as more data become available.

7.10. Regularity of Cohort Reports

In the early years of the cohort, until 1987, *Health Watch* reports tended to concentrate on the establishment of and recruitment to the cohort. *Health Watch* reported the state of the cohort on a yearly basis and presented the cancer morbidity and mortality findings. Later reports mainly presented mortality and cancer incidence findings because lower recruitment meant that the composition of the cohort changed little. From 1988 onwards, reports were on a 2-5 year basis, most reports covering a triennium of mortality and cancer incidence results. This report covers an additional five years of mortality and four years of cancer incidence. Recent linkage

to national data has showed limited changes between reports and linkages every five years appear to be sufficient.

7.11. Smoking

Smoking related diseases, lung cancer, cancer of the lip, oral cavity and pharynx, ischaemic heart disease mortality and chronic obstructive pulmonary disease mortality, were lower in the cohort than in the general population (see Section 4.1, Page 53).

However, within the cohort, it can be seen that smoking has a powerful influence on mortality. Altogether it is estimated that smoking has been a contributing factor to the death of 1171 men, or 53% of the 2563 deaths in the 'ever-smoker' group in the *Health Watch* cohort. This includes increases in heart disease, lung cancer, bladder cancer and chronic obstructive pulmonary disease mortality.

The death rate from all causes increased significantly with increasing tobacco use. Compared to non-smokers, those who smoke 30 or more cigarettes a day showed:

- a 4-fold increase in the death rate
- a 5-fold increase in death rate from heart disease
- over a 50-fold increase in incidence of lung cancer

Risk of lung cancer and heart disease was clearly reduced by quitting smoking. Compared to non-smokers, those who quit showed:

- only a slight increase in mortality
- the death rate from heart disease was not significantly raised
- the risk of lung cancer remained raised but dropped to less than 8-fold, less than a seventh the risk for the highest smoking group.

8. CONCLUSIONS

Generally, the chances of dying at any age, or of getting cancer or heart disease were very similar no matter where *Health Watch* people worked, and compared favourably with the rates in all Australian men. The age-adjusted death rate in men and women was significantly lower than the general Australian population. The strong *healthy worker effect* identified in previous studies continued to be observed.
The proportion of women in the *Health Watch* program remained very small and this precluded detailed analysis of contributory factors.

For men, death rates in all major disease categories were significantly lower than for the corresponding Australian population. A significant reduction in all-cause mortality was seen among men in each workplace type e.g. refinery, terminal or upstream.
Smoking related diseases were lower in *Health Watch* members, than in the general population. However, within the cohort, there was a clear pattern that increasing smoking category was associated with increasing risk of all-cause mortality and cancer. Smoking-related diseases are becoming more evident as the cohort ages. Quitting cigarette smoking greatly reduced the risks.

The chance of contracting cancer was similar for men and women in this industry as for all Australians. However, mortality from cancer was reduced for *Health Watch* members, significantly so for men.
There was no evidence of increasing mortality, cancer incidence or increasing cancer mortality with any of the following:

- increasing duration of employment
- increasing time since first employment

There was a significant trend of increasing cancer mortality by period of first employment. This may be due to the very low rate of cancer mortality in the comparison group (Those employed post 1985)

Three cancers, mesothelioma, melanoma, and prostate cancer have been, and still are, occurring at significantly higher rates than in the general population. Prostate cancer occurred at statistically elevated rates for the first time in the previous report and has again in this latest analysis. These are the only cancers in statistical significant excess. Cancer of the bladder and leukaemia are no longer in in excess. Overall, cancer mortality was statistically significantly lower than that of the general population.

Cancer mortality rates were similar across the main job groups, however, cancer incidence was significantly elevated among drivers. The only cancer that was statistically occurring at a higher rate among drivers compared to the general population was melanoma and prostate cancer. Oesophageal cancer risk among drivers has increased since the last report and is now approaching significance (SIR 1.80, 95% C.I. 0.98-3.01). Oesophageal cancer is strongly linked to smoking and alcohol consumption (105, 106) There was an increased proportion of ever smokers among the driver group compared with the entire male cohort. There were 73% of drivers who have recorded ever smoking (this includes ex-smokers), compared with 64% for the all main job groups. The increased smoking rate in drivers compared with the male cohort may account for the excess of oesophageal cancer in this group.

Leukaemia and bladder cancer continues to be statistically elevated in drivers compared with office-only workers and kidney cancer rates were elevated but not statistically so. The number

of cancers in these groups, however, were comparatively low and have produced wide confidence intervals.

The statistically significant increase in the incidence of melanoma in men in the overall cohort was unlikely to be associated with exposure in the workplace because there was no increasing trend with duration of employment. The risk of melanoma remained elevated when the cohort rates were compared with relevant state rates rather than the national rate. The sustained elevation was not observed in the previous report. (23)

Patterns of diseases were stable but the cohort is aging so a larger number of cancers and deaths might be expected to take place over the next five years. Another match to the cancer incidence and mortality data in five years would be of interest.

9. THE NEW *HEALTH WATCH* COHORT

9.1. Introduction

A *Health Watch* Survey was offered to all current petroleum industry employees of Exxon-Mobil, BP, Shell, Caltex, Woodside and Chevron in 2010 to create a new study cohort. This means that members of the original *Health Watch* study who were still employed, as well as new employees, were offered the opportunity to become part of a new study cohort. The decision to offer a survey to new employees was based on the declining proportion of current employees who were members of the existing cohort, in large part through natural attrition linked to an ageing cohort, and recognition by Monash University, AIP and participating petroleum companies, of the importance of maintaining a longitudinal health study of petroleum industry employees in Australia.

9.1.1. Eligibility

The 6th *Health Watch* Survey was a whole of site survey: i.e. all employees of participating companies located at an eligible worksite at the time of survey were invited to take part. This included existing members of *Health Watch* and staff with primarily administrative or management responsibilities. Criteria for inclusion are summarized in Table 58.
An eligible worksite was one which deals directly with the production, refinery or distribution of petroleum products: for example, terminals, refineries and airport fuel depots. Existing members of *Health Watch* working in non-worksite contexts with a participating company (e.g. staff at Head Offices) were also invited to participate; however, their information was not be included in the overall analyses of the new cohort.
Although contractors engaged in activities at eligible worksites were not invited to participate in this survey, some may have completed the questionnaire but were advised that their contact with *Health Watch* will be limited where they are not existing members or participating company employees. Their data will be held but was not included in the study analysis.

Table 58: Criteria for inclusion in 6th *Health Watch* Survey (2010-2012)

Employment Type	Included in survey? YES / NO	Included in new cohort? YES / NO
Current employee at participating company worksite (e.g. trades, technical etc)	YES	YES
Current employee at participating company worksite (work mostly or entirely in site office)	YES	YES
Current employee of participating company AND existing member of *Health Watch*. Previously employed at eligible worksite but now working at Head Office	YES	NO
Current employee of participating company, not previously in *Health Watch* and working at Head Office	NO	NO
Former employee at participating company worksite, now working onsite as contractor. Existing member of *Health Watch*	NO*	NO
Former employee at participating company worksite. Never joined *Health Watch*	NO*	NO
Contractor working long or short term at eligible company worksite	NO*	NO
Former employee at participating company worksite, now retired or working elsewhere. Existing member of *Health Watch*	NO	NO

*Unless they volunteered at a site visit

9.1.2. Survey Implementation

The 6th *Health Watch* Survey broadly followed the format of previous surveys with the primary survey tool being an electronic questionnaire (offline or online). A print questionnaire was available for employees unable or unwilling to complete the survey electronically. Participants could also complete the survey by arrangement remotely via telephone.

9.1.3. Informed consent

Potential participants were provided with an Explanatory Statement which outlined basic information in plain English about the study purpose, participant involvement, confidentiality and data storage and use. It also provided contact details for the project and for the University's research complaints mechanisms through MUHREC. Informed consent was mandatory for participation in the study and was obtained at the point of recruitment by the individual employee completing a Participant Consent Form.

9.2. Recruitment

9.2.1. Company Site Visits

Only onshore facilities received a site visit by *Health Watch*. Employees at offshore facilities received an invitation to complete the survey online; some may have been captured in transit at airports or heliports by arrangement or incidentally at other locations. Generally, only sites with more than 30 employees were assigned a dedicated scheduled site visit; however, some smaller sites were scheduled in conjunction with a visit to a larger site in reasonable proximity.

9.2.2. On-line Recruitment

The interface for the online database mirrored the laptop database. The front end can be accessed via the following website. https://healthwatch.coeh.monash.org

9.2.3. Email/Postal Invitation to Employees

All eligible employees who had a work email address and who did not have the opportunity to complete the survey during a site visit were sent an email invitation to participate in the *Health Watch* study. If an employee did not have a work email, they were sent the same invitation by post to their worksite address. The email invitations were sent to the employees' email address and included a link to a direct entry point for their individual survey. The employee was then prompted to enter their Employee Number to gain access to the explanatory statement, consent form and the survey. Employees who received a postal invitation were prompted to go to the *Health Watch* website and enter their *Health Watch* ID which was provided in the letter.

9.2.4. Office-only Employees

Office-only employees who were existing *Health Watch* participants were identified by participating companies. These members were added to the new database and invited to participate in the 6th *Health Watch* survey.

9.3. Non-Responders

After the completion of the on-site and on-line survey, a list of non-responders was sent to corresponding companies to identify those employees who resigned/retired prior to receiving a *Health Watch* invitation. Returned postal and email invitations were also recorded. Email returns were either a non-deliverable email, or an automated 'out of office' response. These returns were important to calculate participation rates.

9.4. Participation

The sixth *Health Watch* survey was received positively from about half of the employees from Company worksites. Figure 9 illustrates the breakdown of participation. Almost 45 % of employees at Worksites either completed a survey or consented to the study but did not complete the entire survey. Of the 5110 employees from pre-registered worksites, only 19 employees actively refused consent to participate in the study survey, i.e. they logged into the survey site and checked the 'no consent'. It should be noted that non-participation is not indicative of non-consent. There were over 1,000 employees pre-registered into the database who may not have received one or more invitations to the study for various reasons including resigned/retired, moved to different site, postal return to sender, etc. The majority of these employees were those who left their employment prior to one or more of their survey invitations.

There were 129 existing *Health Watch* participants identified at the time as working in a head office or home office site. Fifty six of these employees either completed the survey or at least started the survey. There were 27 contract employees who completed a survey during a site visit. Some of these employees were either existing members of *Health Watch* but now employed as a contractor or were new employees who were keen to participate. As mentioned in Section 9.1.1, the information gathered from these employees will not be included in the overall analyses of the new cohort.

Fifty four company employees also completed a survey who were not included on the company lists but volunteered to participate during a site visit. These employees were not included in the participation calculations because it is unclear how many employees at each site fell into this category. Because they are company employees however, their data will be used in future analyses.

Figure 9: Participation from the 2010-2012 6th *Health Watch* Survey

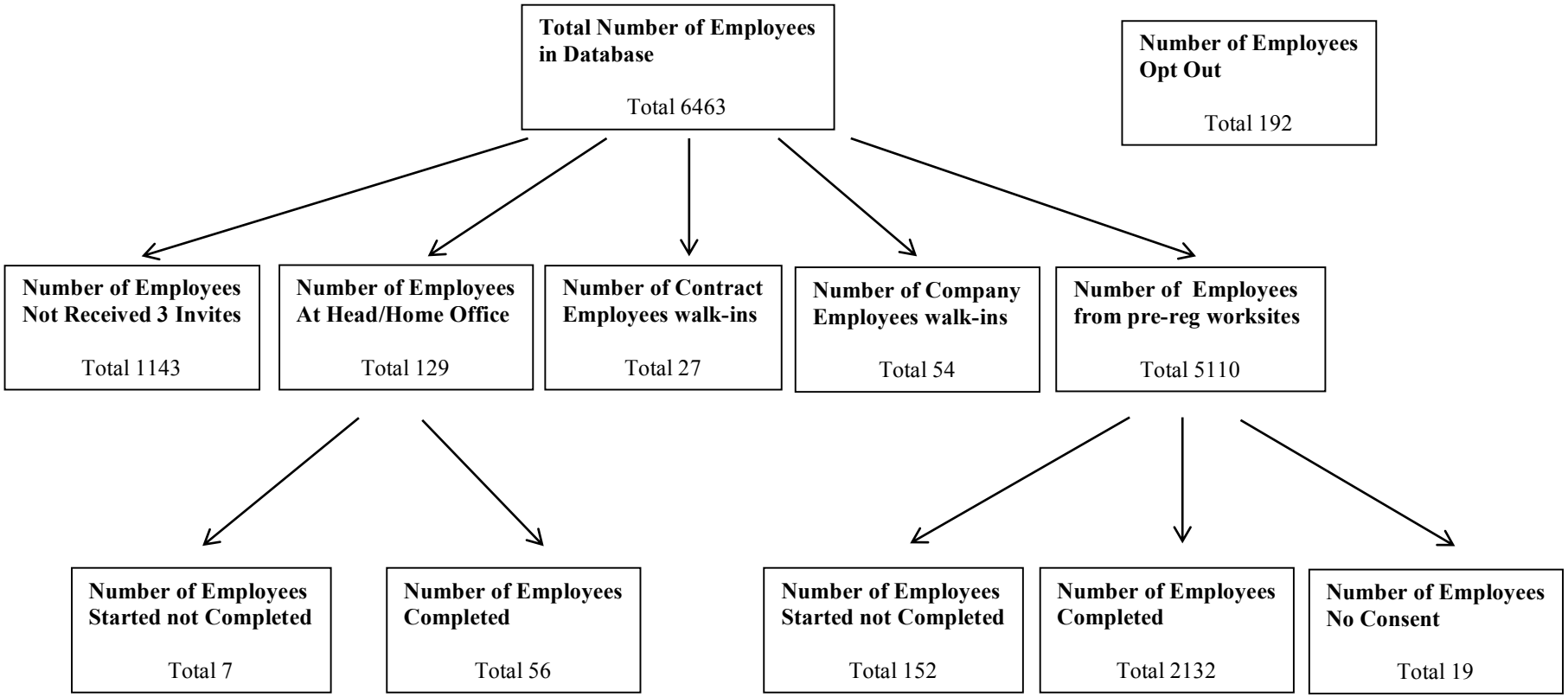

Table 59 shows a breakdown of participation for new and existing *Health Watch* members. Of the 5239 eligible employees, 38% of these were existing *Health Watch* participants. It is interesting to note that 54% of existing *Health Watch* employees either started or completed the survey compared to 39% of eligible new employees. This either illustrated the familiarity and loyalty to the *Health Watch* brand by existing members and or the ever increasing resistance to surveys in general.

Table 59: Breakdown of existing *Health Watch* members and new participants (including Head and Home Office Employees who were pre-registered into the database)

	Elig. No. in DB	*Existing HW Count (%)	New Emp Count (%)	No. Completed survey TOTAL (%)	Existing HW complete survey (%)	New Emps complete survey (%)	Total no. Consents (%)	Existing HW consent (%)	New Emps consent (%)
TOTAL	5,239	1,985 (38)	3,254 (62)	2,188 (42)	1,002 (50)	1,186 (36)	2,347 (45)	1,080 (54)	1,267 (39)

* These figures were calculated by searching the existing *Health Watch* cohort database on Surname and Employee ID or Surname, DOB and the first three letters of first name. There may have been some existing *Health Watch* members that were missed and therefore underestimating these figures.

Visited worksites had a much higher participation rate overall. Fifty six percent of employees either completed or consented to the study at visited worksites, compared to 20% at non-visited sites (Table 60). Office sites were not visited but also had a relatively high participation rate (49%), however, this was due to the majority of these employees being existing *Health Watch* members. Office employees may also be more computer literate than worksite employees, increasing the likelihood of completing an online survey. For those who completed their surveys after an email/postal invitation, participation was higher after the initial invitation. Subsequent reminders to complete the survey produced slightly reduced participation.

Table 60: Participation at worksites. Visited worksites vs online/postal invitation only

	No. in DB	Complete at site visit (%)	Complete after 1 email or postal invite (%)	Complete after 2 emails (%)	Complete after 3+ emails (%)	Total Complete (%)	No Consent online/postal invite (%)	Total consent (%)
Visited worksites ONLY	3,461	1,641 (47)	137 (8)	59 (4)	40 (2)	1,877 (54)	7 (0)	1,951 (56)
Online/postal invites worksites ONLY	1,649	*8 (0)	118 (7)	76 (5)	53 (4)	255 (15)	12 (1)	333 (20)
Office Sites Only	129	*9 (7)	25 (21)	13 (14)	9 (11)	56 (43)	0 (0)	63 (49)

* Employees may have completed a survey during an opportunistic visit to a worksite during a visit by a Monash researcher.

9.5. Results for the Re-Open Cohort

The re-opened cohort was matched to the NDI and ACD in this report and analysed separately. There was a gap of several years between the conclusion of recruitment to the original *Health Watch* cohort and the recruitment to the re-opened cohort which prevented the newer recruits to simply be added to the existing cohort. The number of cancer and deaths recorded in the re-opened cohort were too small to complete any meaningful analyses at this stage. A summary of the death and cancer counts is detailed in Table 61.

Table 61: Summary of total cancer and deaths in the Re-open cohort

		Re-Open Total	Both Cohort	Re-open Only
Men	Total	2,087	1,041	1,046
	Deaths	8	6	-
	Cancer Total	94	70	24
	Cancer Diagnosed Post Recruitment	21	15	6
Women	Total	328	78	250
	Death	-	-	-
	Cancer	10	3	7
	Cancer Diagnosed Post Recruitment	5	-	4

9.6. Summary

The sixth *Health Watch* survey was a whole site survey, open to all employees of participating companies at eligible worksites, including office staff at these sites. The survey broadly followed the format of previous surveys with the primary survey tool being an electronic questionnaire. The survey was administered during a site visit or independently online after an email or postal invitation.

In general, the survey was received positively by most employees. Almost 45 percent of employees at worksites either completed a survey or consented to the study but did not complete the entire survey. Only 19 employees who were invited to take part in the study at the work site actively refused consent.

Participation was higher among existing *Health Watch* members compared to new employees (54% vs 39%). Visited worksites had a much higher participation rate compared to worksites that weren't visited by a Monash researcher (56% vs 20%). It was expected that participation would be greater at visited sites with a Monash presence; however, it was not financially viable to visit many of the remote and smaller sites. It may also appear that those with a prior cancer diagnosis were perhaps more likely to participate, which could bias this cohort given the low recruitment rate. Table 61 shows that a relatively high proportion of participants had a pre-existing cancer at the time of recruitment.

10. ACRONYMS

ABS	Australian Bureau of Statistics
ACD	Australian Cancer Database
ACTU	Australian Capital Territory Union
AEC	Australian Electoral Commission
AIHW	Australian Institute of Health and Welfare
AIP	Australian Institute of Petroleum
API	American Petroleum Institute
BDM	Births, Deaths, Marriages
CI	Confidence Interval
COPD	Chronic Obstructive Pulmonary Disease
CONCAWE	Conservation of Clean Air and Water in Europe
DEPM	Department of Epidemiology and Preventive Medicine
DIMIA	Department of Immigration and Multicultural and Indigenous Affairs
DOB	Date of Birth
HW	*Health Watch*
HWAC	*Health Watch* Advisory Committee
ICD	International Classification of Diseases
IHD	Ischaemic Heart Disease
IOL	Imperial Oil Limited
LH	Lympho-haematopoietic
MonCOEH	Monash Centre for Occupational and Environmental Health
MUHREC	Monash University Human Research Ethics Committee
NDI	Nation Death Index
MDS	Myelodysplastic Syndrome
MPD	Myeloproliferative Disorder
SIR	Standardised Incidence Ratio
SMR	Standardised Mortality Ratio
RIR	Relative Incidence Ratio
RMR	Relative Mortality Ratio
VCR	Victorian Cancer Registry

11. REFERENCES

1. Christie D, Robinson K, Gordon I, Bisby JA. A prospective study in the Australian petroleum industry: I Mortality. Br J Ind Med. 1991;48:507-10.
2. Christie D, Robinson K, Gordon I, Bisby JA. A prospective study in the Australian petroleum industry: II Incidence of cancer. Br J Ind Med. 1991;48:511-4.
3. Christie D, Robinson K, Gordon I, Webley C, Anne. P, Baade A, et al. Health Watch Seventh Annual Report 1987. Melbourne, Australia: The University of Melbourne, Department of Community Medicine; 1988 1998.
4. Bisby JA, Adams GG. Health Watch Ninth Report 1992. Melbourne, Australia: The University of Melbourne, Department of Public Health and Community Medicine; 1993.
5. Bisby JA, Adams GG. Health Watch Tenth Report 1998. Melbourne, Australia: The University of Melbourne, Department of Public Health and Community Medicine; 1999 1999.
6. Gun RT, Pratt NL, Griffith EC, Adams GG, Bisby JA, Robinson KL. Update of a prospective study of mortality and cancer incidence in the Australian petroleum industry. Occup Environ Med. 2004;61(2):150-6.
7. Gun RT, Ryan P, Roder D, Morgenstern M, Pratt N, Griffith E, et al. Health Watch Twelfth Report 2005. Adelaide, Australia: Department of Public Health, Adelaide University; 2005.
8. Gun RT, Pratt N, Ryan P, Roder D. Update of mortality and cancer incidence in the Australian petroleum industry cohort. Occup Environ Med. 2006;63(7):476-81.
9. Glass DC, Del Monaco A, Giuliano K, Friesen M, Cui J, Wood E, et al. Health Watch Thirteenth Report 2007. Melbourne, Australia: Monash Centre for Occupational and Environmental Health (MonCOEH), Department of Epidemiology and Preventive Medicine, Monash University 2007.
10. Glass DC, Adams GG, Manuell RW, Bisby JA. Retrospective exposure assessment for benzene in the Australian Petroleum Industry. Melbourne: Health Watch, Department of Public Health and Community Medicine, University of Melbourne; 1998 1 March 1997.
11. Glass DC, Adams GG, Manuell RW, Bisby JA. Retrospective exposure assessment for benzene in the Australian Petroleum Industry. Ann Occup Hyg. 2000;44(4):301-20.
12. Glass DC, Gray C, Jolley D, Sim M, Gibbons C, Fritschi L. Lympho-haematopoietic Cancer and Exposure to Benzene in the Australian Petroleum Industry. Melbourne: Report to AIP; 2001 May 2001.
13. Glass DC, Gray C, Jolley D, Gibbons C, Sim M. Health Watch Exposure Estimates - Do They Underestimate Exposure? Chem Biol Interact. 2005;153-154:23-32.
14. Glass DC, Gray CN, Jolley DJ, Gibbons C, Sim MR. The Health Watch Case -Control Study of Leukaemia and Benzene- The story so far. Ann N Y Acad Sci 2006;1076:80-9.
15. Glass DC, Sim MR, Fritschi L, Gray C, Jolley D, Gibbons C. Leukaemia Risk and Relevant Benzene Exposure Period-Re: Follow-up time on Risk Estimates. (letter). Am J Ind Med. 2004;45:222-3.
16. Glass DC, Gray CN, Jolley DJ, Gibbons C, Sim MR, Fritschi L, et al. Leukemia risk associated with low level benzene exposure. Epidemiology. 2003;15(5):569-77.
17. Glass DC, Gray C, Jolley D, Gibbons C, Sim M, Fritschi L, editors. The Health Watch Case Control Study. Proceedings of a Conference Institute of Petroleum 19th October 2002; 2003; London.
18. Glass DC, Gray CN, Adams GG, Manuell RW, Bisby JA. Validation of Exposure Estimation for Benzene in the Australian Petroleum Industry. Toxicol Ind Health. 2001;17(4):113-27.
19. Glass DC, Gray CN. Estimating Mean Exposures from Censored Data: Exposure to Benzene in the Australian Petroleum Industry. Ann Occup Hyg. 2001;45(4):275-82.
20. Glass DC, Armstrong TW, Pearlman ED, Verma DK, Schnatter AR, Rushton L. Ensuring comparability of benzene exposure estimates across three nested case-control studies in the petroleum industry in support of a pooled epidemiological analysis. Chem Biol Interact. 2010;184(1-2):101-11.

21. Schnatter AR, Glass DC, Rushton L. Final Report: Pooled Analysis of Petroleum Worker Case-Control Studies. Brussels: CONCAWE; 2012 February 15 2012.
22. Schnatter AR, Glass DC, Tang G, Irons RD, Rushton L. Myelodysplastic Syndrome and Benzene Exposure Among Petroleum Workers: An International Pooled Analysis. J Natl Cancer Inst. 2012;104(22):1724-37.
23. Wood E, Glass DC, Del Monaco A, Vander Hoorn S, Sim M. Health Watch Fourteenth Report Melbourne, Australia: Monash Centre for Occupational and Environmental Health (MonCOEH), Department of Epidemiology and Preventive Medicine, Monash University 2013.
24. Rushton L, Schnatter AR, Tang G, Glass DC. Acute myeloid and chronic lymphoid leukaemias and exposure to low-level benzene among petroleum workers. Br J Cancer. 2013;110: 783-7.
25. Glass DC, Schnatter AR, Tang G, Irons RD, Rushton L. Risk of myeloproliferative disease and chronic myeloid leukaemia following exposure to low level benzene in a nested case-control study of petroleum workers Occup Environ Med. 2014;71(266-274).
26. Glass DC, Wood E, Del Monaco A, Sim M. Cohort Profile: Health Watch-a 30-year prospective cohort study of Australian 5 petroleum industry workers. International Journal of Epidemiology. 2015.
27. Glass DC, Schnatter AR, Tang G, Armstrong TW, Rushton L. Exposure to Benzene in a Pooled Analysis of Petroleum Industry Case-Control Studies. Journal of Occupational and Environmental Health. 2017.
28. Gun RT, Pilotto L, Ryan P, Griffith E, Pratt N, S. E, et al. Health Watch Eleventh Report 2000. Adelaide, Australia: Department of Public Health, Adelaide University; 2000 2000.
29. Christie D, Robinson K, Gordon I, Webley C, Anne. P, Baade A, et al. Health Watch Eighth Annual Report 1988-9. Melbourne, Australia: The University of Melbourne, Department of Community Medicine; 1990.
30. School of Public Health and Preventive Medicine. A Guide to Good Research Practice 5th Edition. Melbourne: Monash University; 2012.
31. Rushton L, Romaniuk H. A case-control study to investigate the risk of leukaemia associated with exposure to benzene in petroleum marketing and distribution workers in the United Kingdom. Occup Environ Med. 1997;54:152-66.
32. Schnatter AR, Armstrong TW, Thompson LS, Nicolich MJ, Katz AM, Huebner WW, et al. The relationship between low-level benzene exposure and leukemia in Canadian petroleum distribution workers. Environ Health Perspect. 1996;104(6):1375-9.
33. American Petroleum Institute. Job code classification system: Part Il - Production operations and marketing/transportation operations. Washington: American Petroleum Institute. OH 26 Industrial Hygiene Subcommittee; 1985.
34. Tabershaw IR. Job code classification system: Part I - Petroleum refineries and selected petrochemical operations. Washington: Occupational Medicine Associates American Petroleum Institute.; 1979.
35. World Health Organisation. International Classification of Diseases. Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death. Ninth Revision Conference 1975 (ICD-9). Geneva: World Health Organization; 1977.
36. World Health Organisation. The International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10). Geneva: World Health Organization; 1992.
37. AIHW. GRIM (General Record of Incidence Mortality) Books, AIHW. 2010 ed. Canberra: AIHW; 2010.
38. AIHW. Cancer incidence projections Australia 2002 to 2011. AIHW; 2005 25 August 2005.
39. Savitz DA, Olshan AF. Multiple comparisons and related issues in the interpretation of epidemiologic data. Am J Epidemiol. 1995;142(9):904-8.
40. Goodman SN. Multiple comparisons explained. Am J Epidemiol. 1998;147(9):807-11.
41. Savitz DA, Olshan AF. Describing data requires no adjustment for multiple comparisons: A reply from Savitz and Olshan. Am J Epidemiol. 1998;147(9):813-4.
42. Thompson JR. Invited commentary: Re: "Multiple comparisons and related issues in the interpretation of epidemiologic data". Am J Epidemiol. 1998;147(9):801-6.

43. Thompson JR. A response to "Describing data requires no adjustment for multiple comparisons". Am J Epidemiol. 1998;147(9):815.
44. Hill D, White V, Scollo M. Smoking behaviours of Australian adults in 1995: trends and concerns. Med J Aust. 1998;168:209-13.
45. Fox AJ, Collier PF. Low mortality rates in industrial cohort studies due to selection for work and survival in the industry. Br J Prev Soc Med. 1976;30:225-30.
46. McMichael A. Standardized mortality ratios and the "healthy worker effect": scratching beneath the surface. J Occ Med. 1976;18:165-8.
47. Monson R. Observations on the Healthy Worker Effect. J Occ Med. 1986;28:425.
48. Gun R. Socioeconomic status and stage of presentation of colorectal cancer. Lancet. 1999;353:409-10.
49. Edelman DDA. Does asbestosis increase the risk of lung cancer? Int Arch Occup Environ Health. 1990;62(5):345-9.
50. Billings CG, Howard P. Asbestos exposure, lung cancer and asbestosis. Monaldi Arch Chest Dis. 2000;55(2):151-6.
51. Mastrangelo G, Ballarin MN, Bellini E, Bizzotto R, Zannol F, Gioffre F, et al. Feasibility of a screening programme for lung cancer in former asbestos workers. Occupational Medicine (Oxford). 2008;58(3):175-80.
52. Halpern M, Gillespie B, Warner K. Patterns of absolute risk of lung cancer mortality in former smokers. J Natl Cancer Inst. 1993;85:457-64.
53. Risch H, Howe G, Jain M, Burch D, Holowaty E, Miller AB. Are female smokers at higher risk for lung cancer than male smokers? A case-control analysis by histologic type. Am J Epidemiol. 1993;138:281-93.
54. Peto R, Darby S, Deo H, Silcocks P, Whitley E, Doll R. Smoking, smoking cessation, and lung cancer in the UK since 1950: combination of national statistics with two case-control studies. BMJ. 2000;321:323-9.
55. Knott CS, Coombs N, Stamatakis E, Biddulph JP. All cause mortality and the case for age specific alcohol consumption guidelines: pooled analyses of up to 10 population based cohorts. BMJ. 2015;350:h384.
56. Makela P, Valkonen T, Poikolainen K. Estimated numbers of deaths from coronary heart disease "caused" and "prevented" by alcohol: an example from Finland. J Stud Alcohol. 1997(58):455-63.
57. Keil U, Chambless L, Doring A, Filipiak B, Stieber J. The relation of alcohol intake to coronary heart disease and all-cause mortality in a beer-drinking population. Epidemiology. 1997;8:150-6.
58. Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, de Gaetano G. Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. Archives of Internal Medicine.166(22):2437-45.
59. McDonald A, Harper A, El Attar O, McDonald J. Epidemiology of primary malignant mesothelial tumors in Canada. Cancer. 1970;26:914-9.
60. Lemen RA, Dement JM, Wagoner JK. Epidemiology of asbestos-related diseases. Environ Health Perspect. 1980;34:1-11.
61. Selikoff IIJ. Lessons for living in a chemical world. Bull Environ Contam Toxicol 1984;33(6):682-95.
62. Hoving J, Del Monaco A, McFarlane E, Fritschi L, Benke G, McKenzie D, et al. Methodological issues in linking study participants to Australian cancer registries using different methods: lessons from a cohort study. Aust NZ J Pub Health. 2005;29:378-82.
63. Hernberg S. Introduction to Occupational Epidemiology. Chelsea Michigan: Lewis; 1992. 230 p.
64. Leonard RC, Kreckmann KH, Lineker GA, Marsh G, Buchanich J, Youk A. Comparison of standardized mortality ratios (SMRs) obtained from use of reference populations based on a company-wide registry cohort to SMRs calculated against local and national rates. Chem Biol Interact. 2007;166(1-3):317-22.

65. Matthews CE, George SM, Moore SC, Bowles HR, Blair A, Park Y, et al. Amount of time spent in sedentary behaviors and cause-specific mortality in US adults. Am J Clin Nutr. 2012;95(2):437-45.
66. van der Ploeg HP, Chey T, Korda RJ, Banks E, Bauman A. Sitting time and all-cause mortality risk in 222 497 Australian adults. Archives of Internal Medicine. 2012;172(6):494-500.
67. Gennaro V, Ceppi M, Boffetta P, Fontana V, Perrotta A. Pleural mesothelioma and asbestos exposure among Italian oil refinery workers.[see comment]. Scand J Work Environ Health. 1994;20(3):213-5.
68. Gennaro V, Finkelstein MM, Ceppi M, Fontana V, Montanaro F, Perrotta A, et al. Mesothelioma and lung tumors attributable to asbestos among petroleum workers.[see comment]. Am J Ind Med. 2000;37(3):275-82.
69. Lewis RJ, Schnatter AR, Katz AM, Thompson FS, Murray N, Jorgensen G, et al. Updated mortality among diverse operating segments of a petroleum company. Occup Environ Med. 2000;57:595-604.
70. Lewis RJ, Schnatter AR, Drummond I, Murray N, Thompson FS, Katz AM, et al. Mortality and cancer morbidity in a cohort of Canadian petroleum workers. Occup Environ Med. 2003;60(12):918-28.
71. Jefferys DB, Vale JA. Malignant mesothelioma and gas-mask assemblers. BMJ. 1978;2(6137):607.
72. Wignall BK, Fox AJ. Mortality of female gas mask assemblers. Br J Ind Med. 1982;39(1):34-8.
73. Wilcox HB, Schoenberg JB, Mason TJ, Bill JS, Stemhagen A. Smoking and lung cancer: risk as a function of cigarette tar content. Prev Med. 1988;17(3):263-72.
74. Tsai SP, Waddell LC, Gilstrap EL, Ransdell JD, Ross CE. Mortality among maintenance employees potentially exposed to asbestos in a refinery and petrochemical plant. Am J Ind Med. 1996;29(1):89-98.
75. Schnatter AR, Theriault G, Katz AM, Thompson FS, Donaleski DL, Murray N. A retrospective mortality study within operating segments of a petroleum company. Am J Ind Med. 1992;22:209-22.
76. Divine BJ, Hartman CM, Wendt JK. Update of the Texaco mortality study 1947-93: part II. Analyses of specific causes of death for white men employed in refining, research, and petrochemicals. Occup Environ Med. 1999;56:174-80.
77. Sorahan T. Mortality of UK oil refinery and petroleum distribution workers, 1951-2003 Occup Med (Oxf). 2007;57(3):177-85.
78. Rosamilia K, Wong O, Raabe GK. A case-control study of lung cancer among refinery workers. J Occup Environ Med. 1999;41(12):1091-103.
79. Satin KP, Bailey WJ, Newton KL, Ross A, Wong O. Updated epidemiological study of workers at two California petroleum refineries, 1950-95. Occup Environ Med. 2002;59:248-56.
80. Finkelstein MM. Maintenance work and asbestos-related cancers in the refinery and petrochemical sector. Am J Ind Med. 1999;36(2):326.
81. Rushton L, Alderson MR. An epidemiological survey of 8 oil refineries in Britain. Br J Ind Med. 1981;38:225-34.
82. Sorahan TT, Nichols LL, Harrington JJM. Mortality of United Kingdom oil refinery and petroleum distribution workers, 1951-1998. Occup Med (Oxf). 2002;52(6):333-9.
83. Satin KP, Wong O, Yuan LA, Bailey WJ, Newton KL, Wen CP, et al. A fifty-year mortality follow up of a large cohort of oil-refinery workers in Texas. J Occup Environ Med. 1996;38(5):492-506.
84. English DR, Heenan PJ, Holman CD, Armstrong BK, Blackwell JB, Kelsall GR, et al. Melanoma in Western Australia 1975-76 to 1980-81: trends in demographic and pathological characteristics. Int J Cancer. 1986;37(2):209-15.
85. Schnatter AR, Rosamilia K, Wojcik NC. Review of the literature on benzene exposure and leukemia subtypes. Chem Biol Interact. 2005;153-154:9-21.

86. Huebner WW, Wojcik NC, Rosamilia K, Jorgensen G, Milano CA. Mortality updates (1970-1997) of two refinery/petrochemical plant cohorts at Baton Rouge, Louisiana, and Baytown, Texas. J Occup Environ Med. 2004;46(12):1229-45.
87. Schnatter AR, Armstrong TW, Nicolich MJ, Thompson FS, Katz AM, Huebner WW, et al. Lymphohaemotopoietic malignancies and quantitative estimates of exposure to benzene in Canadian petroleum distribution workers. Occup Environ Med. 1996;53:773-81.
88. Bergsagel DE, Wong O, Bergsagel PL, Alexanian R, Anderson K, Kyle RA, et al. Benzene and multiple myeloma: Appraisal of the scientific evidence. Blood. 1999;94(4):1174-82.
89. Bezabeh S, Engel A, Morris CB, Lamm SH. Does benzene cause multiple myeloma? An analysis of the published case-control literature. Environ Health Perspect. 1996;104(Suppl 6):1393-8.
90. Goldstein BD. Is Exposure to Benzene a Cause of Human Multiple Myeloma. Ann N Y Acad Sci 1990;609:225-34.
91. Goldstein BD, Shalat SL. The casual relation between benzene exposure and multiple myeloma. Blood. 2000;95:1512-4.
92. Rinsky RA, Smith AB, Hornung RW, Filloon TG, Young RJ, Okun AH, et al. Benzene and leukemia: an epidemiologic risk assessment. N Engl J Med. 1987;316(17):1044-50.
93. Schnatter AR, Kerzic PJ, Zhou Y, Chen M, Nicolich MJ, Lavelle K, et al. Peripheral blood effects in benzene-exposed workers. Chem Biol Interact. 2010;184(1-2):174-81.
94. Crump KS. Risk of benzene-induced leukaemia: a sensitivity analysis of the Pliofilm cohort with additional follow-up and new exposure estimates. J Toxicol & Environ Health. 1994;42:219-42.
95. Hayes RB, Yin S-N, Dosemeci M, Li G-L, Wacholder S, Travis LB, et al. Benzene and the dose-related incidence of hematologic neoplasms in China. J Natl Cancer Inst. 1997; 89(14):1065-71.
96. Loomis D, Guyton KZ, Grosse Y, El Ghissassi F, Bouvard V, Benbrahim-Tallaa L, et al. Carcinogenicity of benzene. The Lancet Oncology.18(12):1574-5.
97. Steinmaus C, Smith AH, Jones RM, Smith MT. Meta-analysis of benzene exposure and non-Hodgkin lymphoma: biases could mask an important association. Occup Environ Med. 2008;65(6):371-8.
98. Kane EV, Newton R. Benzene and the risk of non-Hodgkin lymphoma: A review and meta-analysis of the literature. Cancer Epidem. 2010;34(1):7-12.
99. Alexander D, Wagner M. Benzene Exposure and Non-Hodgkin Lymphoma: A Meta-Analysis of Epidemiologic Studies. J Occup Environ Med. 2010;Epub ahead of print(Feb 3).
100. Vlaanderen J, Portengen L, Rothman N, Lan Q, Kromhout H, Vermeulen R. Flexible meta-regression to assess the shape of the benzene-leukemia exposure-response curve. Environ Health Perspect. 2010;118(4):526-32.
101. Albitar M, et al. Myelodysplastic syndrome is not merely 'pre-leukaemia". Blood. 2002;100(3):791-8.
102. Bennett J, Catovsky D, Daniel M, Flandrin G, Galton D, Gralnick H, et al. Proposals for the classification of the acute leukaemias. French-American-British (FAB) co-operative group. Br J Haematol. 1976;33(4):451-8.
103. Bennett J, Catovsky D, Daniel M, Flandrin G, Galton D, HR. G, et al. Proposals for the classification of the myelodysplastic syndromes. Br J Haematol. 1982;51:189-99.
104. Jaffe ES, Harris NL, Stein H, Vardiman JW, editors. World Health Organization Classification of Tumours: Pathology and Genetics of Tumours of Haematopoietic and Lymphoid Tissues. Lyon: IARC Press; 2001.
105. Sewram V, Sitas F, O'Connell D, Myers J. Tobacco and alcohol as risk factors for oesophageal cancer in a high incidence area in South Africa. Cancer Epidemiol. 2016;41:113-21.
106. Matejcic M, Gunter MJ, Ferrari P. Alcohol metabolism and oesophageal cancer: a systematic review of the evidence. Carcinogenesis. 2017;38(9):859-72.

AUSTRALIAN INSTITUTE OF PETROLEUM
42 Macquarie St, Barton ACT 2600
GPO Box 279, Canberra ACT 2601
(02) 6247 3044
aip@aip.com.au
ABN 11 005 152 581