Mortality and Cancer Incidence in New Zealand Meat Workers

A thesis presented in partial fulfilment of the requirements for the degree of

Doctor of Philosophy

In Epidemiology

At Massey University, Wellington, New Zealand

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99/94 Work related risk of cancer in meat workers

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Abstract

Background:

Several studies have suggested increased risks of cancers of the lung and lymphohaematopoietic tissue associated with work in the meat industry. The evidence for lung cancer is reasonably consistent, although few studies have controlled for smoking. Increased risks of lymphohaematopoietic cancers have been found consistently in case-control studies, including several conducted in New Zealand, but not in cohort studies. This project aimed to ascertain whether there is an increased risk of these cancers in workers employed in the New Zealand meat processing industry, and to identify what exposures are associated with any increased risks.

Methods:

Two cohorts, 4,064 individuals assembled from union records and 6,647 individuals assembled from company records, were followed from 1988 until 2000. Exposure status was assigned according to a job-exposure matrix. The observed number of deaths and cancer registrations was compared with expected numbers using five-year age-specific rates for the New Zealand population. Subgroup analyses evaluated the effect of duration of exposure to selected agents.

Results:

Vital status was determined for 93% (union) and 92% (company) of the total possible person-years. In the union cohort, mortality from all causes (SMR 0.86) and all cancers (SMR 0.88) were reduced, with no elevation observed for the cancers of *a priori* interest. Mortality from all causes (SMR 1.12) and all cancers (SMR1.12) were elevated in the company cohort, with a significant excess of lung cancer (SMR 1.79) and an excess of non-Hodgkin's lymphoma (SMR 1.45). Subgroup analyses showed significant trends of increasing risk with duration of exposure to biological material.

Conclusions:

The union cohort exhibited a strong healthy worker effect, with no increase in mortality or cancer incidence. By contrast, excess risks for all cause and cancer mortality and incidence, and for lung and lymphohaematopoietic cancers, were observed in the company cohort. This is unlikely to be due to confounding by smoking, and the strong dose response relationship suggests the effect is related to occupational exposures.

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In particular I am indebted to Professor Neil Pearce, Director of the Centre for Public Health Research at Massey University in Wellington, who acted as my principal supervisor in this research project. It was Neil who originally sparked my interest in occupational cancer epidemiology by showing it to be both a challenging endeavour in its own right and also a means of providing an evidence base on which to promote improvements in working conditions. Without his encouragement, and occasional cajoling, over the last few years I doubt I would have completed this study. I am also especially grateful to Alistair Woodward of the Department of Public Health at Wellington School of Medicine who acted as my second supervisor, and who reviewed drafts and was always willing to provide advice when asked.

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records, and their fortitude for granting me access to these when their employer had refused to support the study.

I also thank Barbara Aberdeen from Jacques Martin NZ Ltd and V. Alan Langford from National Provident for providing access to the records of the old meat industry superannuation scheme for linkage with union membership records, as without this help there would have been no union cohort. Several people associated with Richmond Ltd also deserve acknowledgement for their help with locating and accessing personnel records, with answering numerous queries about these records, and also with providing unfettered access to plants. In particular I acknowledge the assistance of Marlene Bishop, Linda Codyre, Gary Williams and Mike Finucane. Dr Michael Short and Penny French from Dannevirke Surgery also volunteered invaluable help with checking historical records from these plants, and their help increased the size of the available dataset.

I also wish to thank Jim Fraser, Chris Lewis and Suzette Laws from the NZ Health Information Service who either facilitated or did the electronic and manual matching of our datasets with the NZHIS database. This type of study would also not be possible without the support of organisations like Work and Income New Zealand (WINZ) and the Electoral Enrolment Centre for providing access to data linkage to ascertain vital status of study participants, and I acknowledge the support of both organisations. Mike O'Rourke from WINZ deserves special thanks for his willing assistance with the project.

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My greatest debt is to my family, and in particular my wife Sally and daughter Sylvie who have supported me during the entire difficult process, and to my mother Rhoda and late father Ian McLean who are responsible for having instilled a sense of the value of education.

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Abbreviations

ATLL	Adult T-cell leukaemia/lymphoma
BIV	Bovine immunodeficiency virus
BPSV	Bovine popular stomatitis virus
BLV	Bovine leucosis virus
BSE	Bovine spongiform encephalopathy
CI	Confidence interval
COPD	Chronic obstructive pulmonary disease
CTS	Carpal tunnel syndrome
CWD	Chronic wasting disease of deer
GaLV	Gibbon ape leukaemia virus
HIV	Human immunodeficiency virus
HPV	Human papilloma virus
HTLV	Human T-cell lymphotropic virus
IARC	International Agency for Research on Cancer
ICD	International classification of diseases
IRD	Inland Revenue Department
JEM	Job-exposure matrix
JSRV	Jaagsiekte sheep retrovirus
MOR	Mortality odds ratio
MPMV	Mason-Pfitzer monkey retrovirus
MRL	Maximum residue level
MTB	mercaptobenzothiazole
NHI	National Health Index (number)

NIOSH	US National Institute for Occupational Safety and Health
NMDS	National Minimum Data Set
NZHIS	New Zealand Health Information Service
ODTS	Organic dust toxic syndrome
OR	Odds ratio
PAH	Polycyclic aromatic hydrocarbons
PC LTAS	Life Table Analysis System for personal computer
PCPV	Pseudocowpoxvirus
PCR	Polymerase chain reaction
PIR	Proportionate incidence ratio
PMR	Proportionate mortality ratio
PPCS	Primary Producers Cooperative Society
PPVO	Parapoxvirus orf
PVNZ	Parapoxvirus of red deer in New Zealand
RR	Relative risk
SIR	Standardised incidence ratio
SMR	Standardised mortality ratio
SRV	Simian retrovirus
STLV	Simian T-cell lymphotropic virus
TCMTB	2-(thiocyanomethylthio) benzothiazole
TDE	Transmissible degenerative encephalopathies
TSFE	Time since first employed
vCJD	New variant Creutzfeldt-Jakob Disease
WHO	World Health Organisation
WINZ	Work and Income New Zealand

Chapter 1 Introduction

1.1 Background

A number of studies have suggested an increased risk of cancers of the lung and larynx, and of leukaemia and lymphoma, among butchers and slaughterhouse workers. These findings have come from investigations conducted in several countries, using a combination of study designs including analyses of routinely collected mortality and incidence data, proportionate mortality and incidence studies, and case-control and cohort studies. While increased risks for these cancers have been suggested by these studies, the available evidence is inadequate to conclude either that the risk for any specific cancer is real or to implicate any specific exposure.

Case-control studies, and particularly a series of such studies conducted in New Zealand, have consistently shown elevated risks for cancers of the lung, larynx and lymphohaematopoietic system amongst meat workers, with the highest risks associated with animal slaughter or contact with raw meat. In a number of these studies the elevated risk of lung cancer persisted after adjustment for smoking, and in others where indirect methods were used to control for smoking the excess observed was greater than would be attributable to smoking. By contrast, the meat workers cohort studies that have been conducted previously in Europe and the USA have produced results that tend to contradict those of the case-control studies, although these studies have been limited by study size or by relatively crude exposure assessment. The cohort studies have shown only small increases in lung cancer relative risk, generally within the range that could be attributable to differences in

smoking rates, although higher risks have been observed among workers in abattoirs in contact with live animals or freshly slaughtered meat. These studies have provided little evidence, however, of any increase in cancers of the lymphohaematopoietic system.

In March 1999 the International Agency for Research on Cancer (IARC) invited researchers in Europe, North America and Oceania to participate in an international multi-centre collaborative project, in which each country would conduct one or more historical cohort studies using a standardised protocol but a pooled analysis would be conducted by IARC, to investigate cancer risks in meat workers. As the earlier New Zealand case-control studies had provided much of the evidence for elevated risk of the cancers of the lymphohaematopoietic system in meat workers, and as no cohort study had been conducted to date in New Zealand, it was decided to initiate two New Zealand cohort studies that could contribute to the larger IARC collaborative study. One of these was based on company personnel records and the other was based on union membership records.

Because these cohorts involved different data sources and methods they are, in general, presented and analysed separately here, particularly since their findings differ in some respects. Nevertheless, it was intended that both cohorts would contribute to the IARC collaborative study. This had been the case with previous New Zealand contributions to IARC collaborative studies, for example two cohorts that had been identified through different data sources (one of pesticide production workers and one of pesticide applicators) for a study of phenoxy herbicides. At the time of writing, the IARC project has not proceeded and the two studies are presented and analysed here

as "stand alone" studies, although the data for both will be made available to IARC if and when they proceed.

As well as the approximately 20,000 people employed in the wholesale meat processing sector, a similar number is employed in the retail meat sector, and although these employees represent only 2 to 3% of the national workforce the study of cancers attributable to exposures in this occupation has broader public health significance. Occupational causes of cancer are disproportionately important in public health terms primarily because of the potential for primary prevention, through regulation and improvement in work processes, provided that the cause(s) can be identified and control measures developed. They result from exposures that are involuntary and undesirable, and their control does not involve the challenges of overcoming addictions or deeply embedded behaviours such as smoking. This research has the potential to contribute to the identification of those exposures responsible for any increased risks in meat workers, and of the control measures necessary to prevent future exposure. The study's potential significance also extends beyond the meat industry to others in the community with contact with animals.

1.2 Objectives

The design of the study undertaken involved the investigation of mortality and cancer incidence in two historical cohorts of workers employed in the meat processing industry in New Zealand.

The primary objectives of this project were:

- to ascertain whether there is an increased risk of cancers of the lung, larynx or lymphohaematopoietic system in workers employed in the New Zealand meat processing industry

- to assess whether there are excess risks for any other cancer sites

- to identify what exposures are associated with any increased risks.

The longer-term aim was that any increased risk of cancer in meat workers could be eliminated, through the development and implementation of preventive measures.

1.3 Thesis outline

This thesis, which describes the rationale, data collection, follow-up and analyses of the two historical cohort studies of mortality and cancer incidence in New Zealand meat workers is organised as follows.

Chapter 2 contains a review of previous studies of cancer in meat workers published in the scientific literature, and the available evidence on the association between work in the meat industry and cancer available. The chapter contains a description of the published studies grouped into the four main study types used, and within each of these study types the evidence concerning risks for lung cancer, cancers of the

lymphohaematopoietic system and other cancers is considered separately. This is followed by a summary and review of the strength of the evidence for a causal association between work in the meat industry and increased risks for each cancer type.

Chapter 3 includes a process description for the New Zealand meat industry, and from a combination of a review of the available literature on occupational exposures, extrapolation from the food hygiene and quality literature related to meat processing, discussions with workers and management in the industry and walk-through occupational hygiene surveys of several plants, describe potential biological, chemical and physical and psychosocial exposures in the industry.

Chapter 4 describes the overall design of the two historical cohort studies conducted. This chapter addresses the rationale for the choice of study design, the consultation and ethical approval processes followed, the methods by which the two study populations were assembled, and the development of a job-exposure matrix used to categorise workers according to potential biological or chemical exposures for subgroup analyses. It also covers the methods used to ascertain vital status of the cohort members, and to analyse the data.

The main results of the two studies are presented in Chapter five. This chapter begins with a description of the study populations and an analysis the vital status ascertainment and loss to follow-up, and then the main analyses describing the results of the mortality and cancer incidence follow-up are presented. This is followed by the results of a series of stratified analyses to investigate the effect of specific exposures

on mortality from all causes, from all cancers, and from lung cancer and lymphohaematopoietic cancer. The examination of the relationship between duration of employment and mortality is also presented. Similar analyses performed to examine cancer incidence are also presented.

The thesis concludes with a discussion of the overall findings of the two studies, the limitations of the data with respect to cohort definition, cohort follow-up, confounding and the quality of exposure data, and conclusions in chapter six.

Chapter 2 - Previous studies of cancer in meat workers

2.1 Introduction

There is considerable epidemiological evidence of increased risks of several types of cancer among people occupationally exposed to meat and meat products. Similar associations have also been found in studies in agricultural occupations generally, and in specific occupations such as farmers, animal handlers, and veterinarians.

In this chapter the evidence available from published studies concerning the association between work in the meat industry and specific cancer types is reviewed. The chapter begins with a brief description of the methods used to review the literature, then follows with a description of the studies, grouped into the four main study types conducted: (i) analyses of routinely collected mortality and incidence data; (ii) proportionate mortality and incidence studies; (iii) cohort studies; and (iv) case-control studies. Within each of these study types the evidence concerning risks for lung cancer, cancers of the lymphohaematopoietic system, and other cancers is considered separately.

This is followed by a brief review of studies of other relevant populations including farmers, veterinarians and workers involved in the slaughter of poultry. Finally, I give a summary and review of the strength of the evidence for a causal association between work in the meat industry and increased risks for each cancer type. Neither the exposures that occur in the meat industry, nor possible explanations for the associations with cancer risk, are considered in depth here since they will be addressed in Chapter 3.

2.2 Methods

The publications on the epidemiology of cancer in meat workers reviewed in this chapter were obtained through searching MEDLINE (US National Library of Medicine) from 1966. Variations on the key words "cancer", "lung cancer", "meat workers", "butchers", "abattoir workers", "farmers", and "veterinarians" were used in this literature search, and relevant articles cited in those identified through this search were also obtained. The precision of risk estimates contained in the various studies was evaluated using 95% confidence intervals. Where these were not reported they were calculated using Rothman's formula for standardised mortality ratios [Rothman, 2002] available at [<http://www.oup-usa.org/epi/rothman/>], and Cornfield confidence intervals for odds ratios as used in EpiInfo version 6 [Dean et al, 1990]. In some instances the published risk estimates from the various studies were pooled to generate an overall summary estimate.

2.3 Analyses of routine mortality and incidence records

The earliest reports of increased cancer risks in meat workers were of lung cancer excesses in butchers, which first appeared in the literature in 1982 (table 2.1). These were short reports from several countries based on analyses of routinely collected data on mortality and cancer incidence, using the occupational categories recorded in these datasets.

Lung Cancer

The first report to highlight the association between work as a butcher and lung cancer was a review of Danish occupational mortality data for the period 1970-1975 [Lynge, 1982]. In the same year analyses of routinely collected national cancer mortality and incidence data in England and Wales, Denmark and Sweden were reported, and these showed consistent excess

relative risks in the range of 1.16 to 2.53 in all but one of nine groups studied [Fox et al, 1982]. It was subsequently noted that similar excesses of lung cancer mortality had previously been reported in the occupational mortality reports of both the 1951 and 1961 censuses in England and Wales [Griffith, 1982]. The most striking feature of these short reports was the consistency of the findings of elevated risks that had occurred over a 30-year time period in four different countries.

A more recent Italian study of lung cancer mortality identified industries and occupations at risk through an occupational disease surveillance programme based on record-linkage between census records and death certificates [Lagorio et al, 1995]. All economically active individuals aged between 18 and 64 years who appeared in the 1981 Italian census (i.e. 13 million subjects) were followed up for mortality to 1989. Lung cancer relative risks by industry sector and job title were estimated as mortality odds ratios, with increased mortality among individuals whose occupation was recorded as animal slaughter in the food processing sector (RR=2.78, $p<0.05$, 6 observed), or as animal slaughter regardless of economic sector (RR=2.45, $p<0.05$, 7 observed). The risk estimates obtained in this study for the specific job title of "slaughterer" are similar to the higher estimates seen in the early reports for those categorised as "butchers in slaughterhouses", compared with those for "butchers employed elsewhere" and "unskilled workers in slaughterhouses". Aggregating the data from the early analyses of routine records according to these exposure categories gives pooled estimates that reflect this distinction between animal slaughter and other contact with meat. The pooled estimate for "butchers in slaughterhouses" (SMR 1.71, 95% CI 1.45 – 2.01) exceeds that for "butchers employed elsewhere" (SMR 1.29, 95% CI 1.21 – 1.38) and for "unskilled workers in slaughterhouses" (SMR 1.14, 95% CI 0.98 – 1.32).

The observation that smoking and curing of meat was a common process in the meat industry, and the knowledge that the chemical exposures generated by these processes were either recognized or suspected carcinogens in other settings, led to the formulation of the hypothesis that these chemical exposures were causal factors. An additional hypothesis proposed at this time was that the virus responsible for the well recognized high prevalence of warts among butchers might be responsible for any excess in lung cancer risk [Pegum, 1982]. These exposures are discussed further in chapter 3.

Other cancers

Another early study of routine data investigated leukaemia incidence by occupation in the Portland-Vancouver metropolitan area in the United Sates. In this study 1,678 leukaemia cases, occurring in residents aged 16–75 years over the 15-year period from 1963-1977, were compared with the age-standardized rates in the general population at 1970 [Morton & Marjanovic, 1984]. Cases were classified according to the standard US Bureau of Census occupation and industry coding system, from the information recorded on the hospital records and death certificates from which cases were identified, and annual age-standardised rates within occupation codes were compared with the overall population rates. A significant excess risk of leukaemia was observed for males whose occupation was recorded as "Food, meat processors" (26.6 per 100,000, $0.01<p<0.05$, 9 cases), with the elevation being most noticeable for non-lymphatic leukaemia (19.7 per 100,000, $p<0.01$, 6 cases). When occupation was examined in more detail, the work category of "meat cutters and wrappers" was found to be the most strongly associated with risk of leukaemia (SIR 2.25, 95% CI 0.73 – 5.25, 5 cases), lymphatic leukaemia (SIR 1.95, 95% CI 0.22 – 7.04, 2 cases) and non-lymphatic leukaemia (SIR 2.48, 95% CI 0.50 – 7.25, 3 cases).

Table 2.1 Analyses of routinely collected data for lung cancer in meat workers

Reference	Country and study period	Study type and population (Risk measure)	Observed cases	Relative Risk	95% Confidence Interval
[Fox et al, 1982]	England and Wales 1966-1967	Incidence in all male butchers (PIR)	286	1.3	1.1 – 1.4
	England and Wales 1968-1970	Incidence in all male butchers (PIR)	253	1.2	1.1 – 1.4
	England and Wales 1970-1972	Mortality in all male butchers (SMR)	260	1.2	1.0 – 1.3
	Denmark 1970-1975	Mortality in butchers in slaughterhouses (SMR)	9	2.5	1.2 – 4.8
	Denmark 1970-1975	Mortality in butchers employed elsewhere (SMR)	32	1.7	1.2 – 2.3
	Denmark 1970-1975	Mortality in unskilled workers in slaughterhouses (SMR)	16	0.9	0.5 – 1.4
	Sweden 1961-1973	Incidence in butchers in slaughterhouses (SIR)	65	1.8	1.4 – 2.3
	Sweden 1961-1973	Incidence in butchers employed elsewhere (SIR)	12	1.3	0.7 – 2.2
	Sweden 1961-1973	Incidence in other staff in slaughterhouses (SIR)	52	1.5	1.1 – 1.9
[Griffith, 1982]	England and Wales 1951	Mortality in meat and fish curers and smokers (SMR)	10	2.0	1.0 – 3.6
	England and Wales 1951	Mortality in slaughterhouse workers (SMR)	17	1.3	0.8 – 2.0
	England and Wales 1959-1963	Mortality in butchers and meat cutters (SMR)	436	1.3	1.2 – 1.4
[Lynge et al, 1983]	Denmark 1970-1975	Mortality in self employed butchers (SMR)	17	1.6	2.0– 2.5
	Denmark 1970-1975	Mortality in skilled butchers in butcher shops (SMR)	9	2.5	1.3 – 4.6
	Denmark 1970-1975	Mortality in skilled butchers in slaughterhouses (SMR)	15	1.7	1.0 – 2.8
	Denmark 1975-1980	Mortality in self employed butchers (SMR)	30	1.8	1.2 – 2.5
	Denmark 1975-1980	Mortality in skilled butchers in butcher shops (SMR)	5	0.9	0.3 – 1.9
	Denmark 1975-1980	Mortality in skilled butchers in slaughterhouses (SMR)	21	1.5	2.0 – 2.3
	Denmark 1975-1980	Mortality in unskilled workers in slaughterhouses (SMR)	35	1.1	0.8 – 1.5
	Denmark 1970-1980	Mortality in self employed butchers (SMR)	47	1.7	1.3 – 2.2
	Denmark 1970-1980	Mortality in skilled butchers in butcher shops (SMR)	14	1.5	0.9 – 2.4
	Denmark 1970-1980	Mortality in skilled butchers in slaughterhouses (SMR)	36	1.6	1.1 – 2.2
	Denmark 1970-1980	Mortality in unskilled workers in slaughterhouses (SMR)	51	1.0	0.8 – 1.3
[Lagorio et al, 1995]	Italy 1981-1982	Mortality in animal slaughterers in the food processing sector (MOR)	6	2.8	$P < 0.1$
	Italy 1981-1982	Mortality in animal slaughterers in any economic sector (MOR)	7	2.5	$P < 0.1$

2.4 Proportionate mortality and Cancer incidence studies

In a number of the early investigations of the cancer risks associated with work in the meat industry, where the available denominator data were either not available or not comparable with the numerator data, proportional measures of mortality or incidence were calculated (table 2.2). For example, for two of the periods in which excess lung cancer among male butchers in England and Wales was reported, risk estimates were presented as proportionate incidence ratios (PIRs) with the expected number of lung cancers calculated on the basis of age specific proportions of all cancer registrations [Fox et al, 1982]. For the period 1966-7, the PIR was estimated as 1.27 (286 cases), with a similar risk estimate for the period 1968-70 (PIR 1.20, 253 cases).

By contrast, no excess of lung cancer was found in a proportionate mortality study comparing death certificate data for the period 1950 – 1979 for white male butchers and meat cutters (working both within or outside of slaughterhouses) with that for other deaths among white males in Washington State [Milham, 1982]. Milham reported a proportionate mortality ratio (PMR) of 1.07 for butchers and meat cutters working in slaughterhouses, and 0.97 for butchers and meat cutters working outside slaughterhouses.

The findings of a proportionate mortality study conducted on 233 deaths occurring between 1965 and 1980 in male members of the meat cutters' union in Baltimore were reported in a letter to the Lancet published at the same time as these initial reports of increased lung cancer risks in butchers [Johnson & Fischman, 1982]. Using 1975 mortality data from the US general population adjusted for age at death and ethnicity to derive expected values, they found an excess mortality from all cancers with 56 observed against 48.22 expected (PMR 1.16). Excess mortality was also found for lung (PMR 1.54, 27 cases) and bladder cancer (PMR 2.24, 3

deaths). Though the numbers were small, this investigation also suggested an association with lymphohaematopoietic cancer: excess mortality was indicated for lymphosarcoma and reticulum cell sarcoma (PMR 2.67, 2 deaths), myeloid leukaemia (PMR 3.75, 3 deaths) and multiple myeloma (PMR 2.74, 2 deaths).

Johnson then reported the results of a historical cohort study of 28,901 members of a Baltimore meat cutters' union employed between 1949 and 1979, including 13,844 white male members [Johnson et al, 1986a], 7,261 white female members [Johnson et al, 1986b] and 5,362 non-white male members [Johnson, 1989] followed from July 1949 to the end of 1980. For the overall cohort, and within each of these sub-cohorts, internal comparisons were possible through separate analyses carried out for subgroups defined according to lifetime work in the following job-categories: abattoirs, meat-packing plants in which no slaughtering was done, meat and delicatessen departments of grocery stores/supermarkets, chicken slaughtering plants, and companies outside the meat industry. Proportionate mortality ratios were calculated by comparing mortality data in this cohort with the general US population.

Among white male members of the union, statistically significant elevated PMRs were found for all cancers (PMR 1.15, $p<0.005$, 151 deaths), and for cancer of the buccal cavity and pharynx (PMR 2.00, $p<0.005$, 23 deaths), large intestine (PMR 1.40, $p<0.05$, 42 deaths) and lung (PMR 1.32, $p<0.005$, 151 deaths). Statistically significant elevated PMRs were found for Hodgkin's Disease in abattoir workers (PMR 2.92, $p<0.05$, 4 deaths), for buccal cavity and pharynx (PMR 2.93, $p<0.05$, 6 deaths), lung cancer (PMR 1.57, $p<0.05$, 31 deaths) and bone cancer (PMR 9.37, $p<0.005$, 3 deaths) in meat-packing plant workers, and for lung cancer (PMR 1.78, $p<0.005$, 23 deaths) in non-meat industry workers.

For all white female union members a significant elevation was found for lung cancer (PMR 2.10, $p<0.005$, 38 deaths), while death from breast cancer was lower than expected (PMR 0.65, $p<0.05$, 27 deaths). Those employed in meat packing plants experienced a significant elevation in mortality from lung cancer (PMR 3.30, $p<0.005$, 12 deaths), while those employed in the meat department of supermarkets experienced significant elevations in mortality from lymphohaematopoietic cancers (PMR 2.17, $p<0.05$, 10 deaths), and lung cancer (PMR 2.18, $p<0.005$, 14 deaths).

Table 2.2 **Proportionate Mortality and Proportionate Incidence studies of cancer in meat workers.**

Cancer site (ICD 9th revision)	Reference	Country and study period	Population studied (Risk measure)	Observed cases	Relative Risk
All Cancers (140-209)	[Johnson & Fischman, 1982]	Baltimore US 1965 - 1980	Meat cutters union members (PMR)	56	1.16
	[Johnson et al, 1986a]	Baltimore US 1949 -1980	White male union members (PMR)	401	1.15
	"	"	White male union members in abattoirs (PMR)	125	1.21
	"	"	White male union members in meat packing plants (PMR)	76	1.25
	"	"	White male union members in supermarkets (PMR)	126	1.12
	"	"	White male union members in non-meat plants (PMR)	43	1.07
	"	"	White female union members (PMR)	178	1.09
	"	"	White female union members in abattoirs (PMR)	29	1.05
	"	"	White female union members in meat packing plants (PMR)	36	1.14
	"	"	White female union members in supermarkets (PMR)	67	1.22
	"	"	White female union members in non-meat plants (PMR)	29	0.83

Lung cancer (162)	[Fox et al, 1982]	England and Wales 1966 -1967	Incidence in all male butchers (PIR)	286	1.27
	"	England and Wales 1968 -1970	Incidence in all male butchers (PIR)	253	1.20
	[Milham, 1982]	Washington US 1950 -1979	Mortality in butchers and meat cutters working in slaughterhouses (PMR)	28	1.07
	"	"	Mortality in butchers and meat cutters not working in slaughterhouses (PMR)	89	0.97
	[Johnson & Fischman, 1982]	Baltimore US 1965 -1980	Meat cutters union members (PMR)	27	1.54
	[Johnson et al, 1986a]	Baltimore US 1949 -1980	White male meat cutters union members (PMR)	151	1.32
		"	White male members working in meat packing plants (PMR)	31	1.57
		"	White male union members working in supermarkets (PMR)	46	1.27
		"	White male union members working in the non-meat industry (PMR)	23	1.78
		"	White female union members (PMR)	38	2.10
		"	White female union members in meat packing plants (PMR)	12	3.30
		"	White male union members in meat department of supermarkets (PMR)	14	2.18
Buccal cavity and pharynx (140-149)	[Johnson et al, 1986a]	Baltimore US 1949 -1980	Mortality in white male union members (PMR)	23	2.00
	"	"	White male union members working in meat packing plants (PMR)	6	2.93
Bone (170)	[Johnson et al, 1986a]	Baltimore US 1949 -1980	White male union members in meat packing plants (PMR)	3	9.37
Large intestine (153)	[Johnson et al, 1986a]	Baltimore US 1949 -1980	White male union members (PMR)	42	1.40
Breast (174)	[Johnson et al, 1986a]	Baltimore US 1949 -1980	White female union members (PMR)	27	0.65
	"	"	White female union members in meat packing plants (PMR)	8	0.56

Bladder cancer (188)	[Johnson & Fischman, 1982]	Baltimore US 1965 -1980	Meat cutters union members (PMR)	3	2.24
All Lymphohaematopoietic (200-208)	[Johnson et al, 1986a]	Baltimore US 1949 - 1980	White male meat cutters union members (PMR)	34	0.92
			White male members working in abattoirs (PMR)	13	1.24
			White male union members in meat packing plants (PMR)	4	0.62
			White male union members in supermarkets (PMR)	12	0.66
			White male union members in the non-meat industry (PMR)	3	0.66
			White female union members (PMR)	10	2.17
			White female union members in abattoirs (PMR)	2	0.89
			White female union members in supermarkets (PMR)	10	2.17
Lymphosarcoma and reticulosarcoma (200)	[Johnson & Fischman, 1982]	Baltimore US 1965 - 1980	Meat cutters union members (PMR)	2	2.67
Hodgkin's disease (201)	[Johnson et al, 1986a]	Baltimore US 1949 -1980	White male union members (PMR)	9	1.70
			White male union members in abattoirs (PMR)	4	2.92
			White male union members in supermarkets (PMR)	2	1.09
Myeloid leukaemia (205)	[Johnson & Fischman, 1982]	Baltimore US 1965 -1980	Union members (PMR)	3	3.75
Multiple myeloma (203)	[Johnson & Fischman, 1982]	Baltimore US 1965 – 1980	Union members (PMR)	2	2.74

2.5 Cohort studies

Following publication of the first studies of routine mortality and cancer incidence records, a number of cohort studies of butchers and meat workers in Germany [Doerken & Rehpenning, 1982], the United States [Johnson et al, 1986a, 1986b, 1995; Johnson, 1989, 1991, 1994], England and Wales [Coggon et al, 1989; Coggon & Wield, 1995], Switzerland [Guberan et al, 1993], and Sweden [Boffetta et al, 2000] have been published. These cohort studies have provided the opportunity not only to evaluate risks for cancer of the lung and the lymphohaematopoietic system, but also to assess whether meat workers were at increased risk for other cancers. Within the limitations of cohort size, and the validity of the exposure data available, they have also allowed specific potential risk factors to be investigated. These studies are reviewed below in chronological order, rather than by disease type, since these studies involved all cancer sites. Risk estimates are presented below as reported in the original articles, while in the summary of all cohort studies presented in table 2.3 the risk estimates include estimated confidence intervals where these were not reported.

Doerken and Rehpenning reported that a more than two-fold excess risk of lung cancer (i.e. 36 cases in butchers but only 15 among bakers, $p<0.01$) had been a chance (and previously unreported) finding in a study in which they compared the mortality experience of a cohort of 398 butchers with that of 399 bakers in Hamburg for the period 1954-1966 [Doerken and Rehpenning, 1982]. Johnson also reported standardised mortality ratios (SMRs) for the Baltimore meat cutters' union cohorts (for which proportionate mortality rates were also reported), with expected rates based on the general US population. Among white male members of this cohort [Johnson et al, 1986a] a statistically significant elevation in risk was found for all cancers (SMR 1.29, $p<0.005$, 401 deaths), which included a statistically significant excess of lung cancer (SMR 1.37, $p< 0.005$, 151 observed). A similar excess of lung

cancer (SMR 1.98, p<0.005, 38 observed) was seen in white female union members [Johnson et al, 1986b]. The lung cancer risk was significantly elevated for both males (SMR 1.88, p<0.005, 31 observed) and females (SMR 4.02, p<0.005, 12 observed) with lifetime employment in meatpacking plants, and for females with lifetime employment in either the meat departments of grocery stores/supermarkets (SMR 1.95, p<0.05, 14 observed). The highest lung cancer rate for white males, however, was found in the comparison group employed in the non-meat companies (SMR 2.15, p<0.005, 23 observed). For the 5,145 non-white males in this cohort [Johnson, 1989], however, there was a significant excess of lung cancer among abattoir workers (SMR 2.1, p<0.005, 43 observed), but not in the other occupations (including the non-meat companies).

The white male union sub-cohort [Johnson et al, 1986a] also experienced 34 deaths from lymphohaematopoietic cancers (SMR 0.95, NS). There were non-statistically significant elevations for Hodgkin's disease (SMR 1.49, NS, 9 observed) and cancer of other lymphatic tissue (SMR 1.49, NS, 11 observed). When categorised according to jobs, work in an abattoir was associated with an elevated risk of Hodgkin's disease (SMR 2.37, NS, 4 observed), while work in meat departments of grocery stores/supermarkets was associated with an elevated risk of cancer of other lymphatic tissue (SMR 1.54, NS, 4 cases) with all four deaths being due to multiple myeloma. There was no increase in risk of lymphohaematopoietic cancer (SMR 0.89, 12 cases) among white women [Johnson et al, 1986a], although there was an elevation (SMR 1.93, NS, 10 cases) among those who had worked only in the meat departments of grocery stores/supermarkets. This included three cases of myeloid leukaemia and four of non-Hodgkin's lymphoma, which in both instances was roughly three times that expected on the basis of US national rates. No excess of lymphatic and haematopoietic cancers was observed in the non-white male union members.

A cohort of 1,610 UK male slaughterhouse workers, employed for at least six months at one of three companies during the period 1946 to December 1971, was followed to the end of 1986 [Coggon et al, 1989]. Death rates were compared with expected rates based on the national population, and internal comparisons were made between exposure categories based on job title. All cause mortality was lower than in the general population (SMR 0.86, 95% CI 0.77 – 0.98, 271 deaths), as was mortality from respiratory disease (SMR 0.72, 95% CI 0.47 – 1.05, 27 deaths), digestive disease (SMR 0.50, 95% CI 0.14 – 1.27, 4 deaths), cerebrovascular disease (SMR 0.65, 95% CI 0.38 – 1.04, 17 deaths) and ischaemic heart disease (SMR 0.86, 95% CI 0.68 – 1.06, 84 deaths). An excess mortality from all cancers (SMR 1.09, 95% CI 0.87 – 1.34, 87 deaths) was observed, due largely to excesses of cancers of the lung (SMR 1.33, 95% CI 0.96 – 1.79, 42 deaths), stomach (SMR 1.62, 95% CI 0.86 – 2.77, 13 deaths) and liver (SMR 5.62, 95% CI 1.16 – 16.42, 37 deaths). A small excess of cases of cancers of the lymphohaematopoietic system was also found, with 8 deaths observed compared with 5.2 expected, although the estimates for specific subtypes lacked precision. Non-significant elevations were observed for Hodgkin's Disease (SMR 2.41, 95% CI 0.29-8.72, 2 cases), all leukaemia (SMR 1.42, 95% CI 0.29-4.16, 3 deaths), and multiple myeloma (SMR 2.44, 95% CI 0.30-8.83, 2 cases).

When cohort members were categorised according to exposure to live animals; warm (freshly slaughtered) meat; chilled meat; and bacon process and products, the highest lung cancer risk (SMR 1.84, 95% CI 1.15 – 2.79, 22 deaths) was observed among workers who had worked with warm meat. Although information on smoking was not available, two different indirect methods were used to control for confounding by smoking. Firstly, a comparison of death rates from other tobacco related cancers (i.e. cancers of the oral cavity, oesophagus, pancreas, larynx, kidney and bladder) found that these were not elevated, and it was noted that deaths

from non-malignant respiratory disease were reduced (SMR 0.72). Secondly, an adjustment made to the expected number of deaths from lung cancer in proportion to the SMRs for lung cancer in local authority areas yielded essentially unchanged risk estimates. Both methods suggested that the observed elevation of lung cancer risk could not be attributed to confounding by smoking.

Johnson reported updated analyses of subgroups of the original cohort from the Baltimore meat cutters' union, namely workers in the meat department of supermarkets [Johnson, 1994], abattoirs and meatpacking plants [Johnson et al, 1995] The additional 9 years of follow-up of the 10,841 union members who ever worked in the meat department of supermarkets, and the inclusion of 766 additional deaths in the analyses, confirmed the increased risk estimate for lung cancer in women (SMR 1.60, 95% CI 1.10 – 2.20, 40 cases) but not in men (SMR 1.10, 95% Cl 0.90 - 1.30, 102 cases). The difference in risk observed between genders was considered likely to be due to exposure to thermal decomposition products from the hot-wire cutting of plastic film, as women did the vast majority of meat wrapping in supermarkets. The additional follow-up of those union members who had worked in abattoirs (5,522) or meatpacking plants (4,589) revealed statistically significant elevations in risk of lung cancer in both abattoir workers (SMR 1.40, 95% CI 1.2 – 1.6, 167 cases) and meatpacking plant workers (SMR 1.50, 95% CI 1.3 – 1.8, 130 cases), with the increase being apparent in both genders and ethnic groups studied. Lung cancer risk was virtually identical, however, in the group who had worked exclusively in non-meat companies (SMR 1.30, 95% CI 1.0 – 1.6, 94 observed). Thirty-two deaths from lymphohaematopoietic cancers were also observed, but the only specific types to show a (non-statistically) significant excess risk were Hodgkin's Disease and multiple myeloma [Johnson et al, 1995]. In a mortality odds ratio (MOR) analysis using the non-meat workers as a reference group, an elevation in risk among abattoir workers was

observed for non-Hodgkin's lymphoma (MOR = 5.20, p>0.14, 6 exposed cases), for Hodgkin's disease (MOR = 6.00, p>0.11, 6 exposed cases), for multiple myeloma (MOR = 2.30, p>0.24, 9 exposed cases), and for all leukaemia (MOR = 2.30, p>0.17, 11 exposed cases).

A cohort of 552 butchers (cattle and sheep) and 310 pork butchers, working in Geneva during the period 1901 to 1969, was followed to 1990 [Guberan et al, 1993]. This cohort experienced a significant increase in all cause mortality (SMR 1.21, 90% CI 1.13-1.30), and in cancer mortality (SMR 1.27, 90% CI 1.10-1.44) and incidence (SIR 1.45, 90% CI 124-169). Specific sites in which incidence was elevated included colorectal (SIR 2.56, 90% CI 1.81-3.53, 27 observed), liver (SIR 2.77, 90% CI 1.38-4.99, 8 observed), lung (SIR 1.52, 90% CI 106-210, 26 observed) and prostate cancer (SIR 1.80, 90% CI 1.24-2.53, 24 observed). Although no elevation was observed for lymphohaematopoietic cancers overall, an eight-fold excess risk of leukaemia (5 cases compared with 0.6 expected) was noted among butchers born before 1900 - a period in which it was noted that all butchers slaughtered their own stock.

A group of 4,018 males whose occupation was listed as butcher in the 1961 census of England and Wales were traced through the National Health Service central death register with follow-up to 1992 [Coggon & Wield, 1995]. Mortality from all cancers was lower than in the general population (SMR 0.96, 95% CI 0.89 – 1.04, 691 deaths), and no excess in mortality from lung cancer was found (SMR 1.01, 95% CI 0.90 – 1.13, 294 deaths). These findings provided no support for the earlier findings of lung cancer excesses in a UK meat workers cohort [Coggon et al, 1989]. However, the authors noted several limitations of the study including the high proportion of study subjects who could either not be traced or for whom only a probable match could be made. There was also no information available on duration of employment of study subjects, and internal comparisons could therefore not be made.

A number of cohort studies have been conducted using Swedish census data and record linkage with the Swedish National Cancer Register and the National Register of Causes of Death. One such study of oesophageal cancer and occupation using record linkage between the 1960 census and the Cancer Registry found elevated incidence (SIR 2.1, $p<0.01$) among males whose occupation was recorded as butcher [Chow et al, 1995]. A similar cohort of 25,049 men recorded as working as butchers or meatpackers, or as being employed in butcher shops, meat processing or packing, in either the 1960 or 1970 Swedish censuses was followed up for cancer incidence and mortality for the period 1971 to 1989 through record linkage with the Swedish National Cancer Register and the National Register Of Causes Of Death [Boffetta et al, 2000]. There was overlap in this cohort with the early report by Fox et al [1982], although the follow-up period was considerably longer. A statistically significant increase in lung cancer (SIR 1.30, 95% CI 1.20 – 1.50, 314 cases) and laryngeal cancer (SIR 1.70, 95% CI 1.20 – 2.30, 47 cases) was observed among those employed as butchers or meat workers in either the 1960 or 1970 censuses. An elevated risk of lung cancer was also observed for butchers working specifically in the meat industry (RR 1.40, 95% CI 1.10 – 2.00, 43 cases), using other workers (excluding those employed in animal-related jobs) as a reference group. However, this risk was not significantly elevated for butchers working in other industries, or for those working as mechanics, maintenance workers, service workers and white-collar workers in the meat industry. For laryngeal cancer an excess risk was observed for butchers in other industries (RR 4.30, 95% CI 1.40 – 13.20, 3 cases), but not for either butchers or non-butchers in the meat industry. A non-significant increase in non-lymphocytic leukaemia (SMR 1.30, 95% CI 0.90-1.80) was observed for all those employed as butchers or meat workers at the 1960 or 1970 census.

Table 2.3 Cohort studies of cancer in meat workers

Cancer (ICD-9 code)	Reference	Exposure category	Observed cases	SMR	95% confidence interval
All cancers (140-208)	[Johnson et al, 1986a]	white males in union	401	1.3	1.2– 1.4
	[Johnson et al, 1986b]	women in union	178	1.1	1.0 – 1.3
	[Johnson et al, 1986a]	white males in abattoir	125	1.3	1.1 – 1.5
	[Johnson et al, 1986a]	white males in meatpacking plant	76	1.6	1.3– 2.0
	[Johnson et al, 1986a]	white males in supermarket	126	1.2	1.0 – 1.4
	[Johnson, 1989]	non-white males in abattoir	43	1.2	0.9 – 1.6
	[Johnson, 1989]	non-white males in meat packing plant	32	1.2	0.8 – 1.7
	[Coggon et al, 1989]	meat industry	87	1.1	0.9 – 1.3
	[Guberan et al, 1993]	self employed butchers	157	1.3	1.1 – 1.4
	[Johnson, 1994]	women in supermarket	153	1.0	0.9 – 1.2
	[Johnson, 1994]	men in supermarket	290	1.0	0.9 – 1.2
	[Johnson et al, 1995]	abattoir	455	1.1	1.0 – 1.3
	[Johnson et al, 1995]	meat packing plant	343	1.2	1.1 – 1.3
	[Coggon & Wield, 1995]	butchers	691	1.0	0.9 – 1.0
	[Boffetta et al, 2000]	butchers or meatpackers	2417	1.1	1.0 – 1.1
Buccal cavity and pharynx (140-149)	[Johnson et al, 1986a]	white males in union	23	2.3	1.5 – 3.3
	[Guberan et al, 1993]	self employed butchers	6	1.0	0.4 – 2.0
	[Johnson, 1994]	women in supermarket	3	1.5	0.3 – 4.4
	[Johnson, 1994]	men in supermarket	15	1.8	1.0 – 3.0
	[Johnson et al, 1995]	abattoir	12	1.1	0.6 – 1.9
	[Johnson et al, 1995]	meat packing plant	17	2.0	1.2 – 3.2
	[Boffetta et al, 2000]	butchers or meatpackers	73	1.1	0.8 – 1.3
Oesophagus (150)	[Johnson, 1989]	non-white males in abattoir	2	0.8	0.2 – 2.6
	[Johnson, 1989]	non-white males in meat packing plant	6	3.1	1.3 – 6.4
	[Guberan et al, 1993]	self employed butchers	9	1.4	0.7 – 2.5
	[Chow et al, 1995]	butchers		2.1	($p < 0.01$)
	[Johnson et al, 1995]	abattoir	19	1.7	1.0 – 2.6
	[Johnson et al, 1995]	meat packing plant	16	1.7	1.0 – 2.8
	[Coggon & Wield, 1995]	butchers	15	0.7	0.4 – 1.2
	[Boffetta et al, 2000]	butchers or meatpackers	38	1.3	0.9 – 1.8

Stomach (151)	[Coggon et al, 1989]	meat industry	13	1.6	0.9 – 2.8
	[Guberan et al, 1993]	self employed butchers	15	1.3	0.8 – 2.0
	[Johnson, 1994]	females in supermarket	3	0.9	0.2 – 2.7
	[Johnson, 1994]	males in supermarket	12	1.0	0.5 – 1.8
	[Coggon & Wield, 1995]	butchers	72	1.0	0.8 – 1.2
	[Boffetta et al, 2000]	butchers or meatpackers	160	1.1	1.0 – 1.3
Colon (153)	[Johnson et al, 1986a]	white males in union	42	1.6	1.2 – 2.2
	[Johnson, 1994]	females in supermarket	8	0.6	0.3 – 1.2
	[Johnson, 1994]	males in supermarket	36	1.5	1.1 – 2.1
	[Johnson et al, 1995]	abattoir	36	1.0	0.7 – 1.4
	[Johnson et al, 1995]	meat packing plant	35	1.4	1.0 – 2.0
	[Coggon & Wield, 1995]	butchers	38	0.8	0.6 – 1.2
	[Boffetta et al, 2000]	butchers or meatpackers	160	1.0	0.8 – 1.2
Rectum (154)	[Coggon et al, 1989]	meat industry	4	1.1	0.3 – 2.8
	[Coggon & Wield, 1995]	butchers	39	1.2	0.9 – 1.7
	[Boffetta et al, 2000]	butchers or meatpackers	142	1.2	1.0 – 1.4
Colon, rectum (153,154)	[Guberan et al, 1993]	self employed butchers	25	1.8	1.2 – 2.5
Liver (155)	[Coggon et al, 1989]	meat industry	3	5.6	1.2 – 16.4
	[Guberan et al, 1993]	self employed butchers	8	1.7	0.9 – 3.1
	[Coggon & Wield, 1995]	butchers	2	0.5	0.1 – 1.7
	[Boffetta et al, 2000]	butchers or meatpackers	44	0.9	0.7 – 1.2
Pancreas (157)	[Guberan et al, 1993]	self employed butchers	6	1.2	0.5 – 2.3
	[Coggon & Wield, 1995]	butchers	26	0.9	0.6 – 1.3
	[Boffetta et al, 2000]	butchers or meatpackers	78	1.1	0.8 – 1.3
Larynx (161)	[Guberan et al, 1993]	self employed butchers	6	1.8	0.8 – 3.6
	[Coggon & Wield, 1995]	butchers	5	0.8	0.3 – 1.9
	[Boffetta et al, 2000]	butchers or meatpackers	47	1.7	1.2 – 2.3

Lung (162)	[Doerken & Rehpenning, 1982]	butchers	-	"2 fold"	-
	[Johnson et al, 1986a]	white males in union	151	1.5	1.3 – 1.8
	[Johnson et al, 1986b]	females in union	38	2.2	1.6 – 3.0
	[Coggon et al, 1989]	meat industry	42	1.3	1.0 – 1.8
	[Johnson, 1989]	non-white males in abattoir	23	2.1	1.4 – 3.1
	[Johnson, 1989]	non-white males in meat packing plant	9	1.0	0.5 – 1.9
	[Guberan et al, 1993]	self employed butchers	35	1.2	0.9 – 1.6
	[Johnson, 1994]	females in supermarket	40	1.6	1.1 – 2.2
	[Johnson, 1994]	males in supermarket	102	1.1	0.9 – 1.3
	[Johnson et al, 1995]	abattoir	167	1.4	1.2 – 1.6
	[Johnson et al, 1995]	meat packing plant	130	1.5	1.3 – 1.8
	[Coggon & Wield, 1995]	butchers	294	1.0	0.9 – 1.1
	[Boffetta et al, 2000]	butchers or meatpackers	314	1.3	1.2 – 1.5
Bone (170)	[Johnson et al, 1986a]	white males in meat packing plant	3	10.4	2.9 – 27.8
	[Johnson et al, 1995]	abattoir	2	1.5	0.2 – 5.4
	[Johnson et al, 1995]	meat packing plant	4	4.2	1.1 – 11.0
Prostate (185)	[Coggon et al, 1989]	meat industry	3	0.8	0.2 – 2.2
	[Guberan et al, 1993]	self employed butchers	23	1.7	1.1 – 2.3
	[Coggon & Wield, 1995]	butchers	71	1.2	1.0 – 1.5
	[Boffetta et al, 2000]	butchers or meatpackers	523	1.1	1.0 – 1.2
Bladder (188)	[Guberan et al, 1993]	self employed butchers	5	0.9	0.4 – 1.9
	[Johnson et al, 1995]	abattoir	14	1.7	0.9 – 2.9
	[Johnson et al, 1995]	meat packing plant	8	1.5	0.7 – 3.0
	[Coggon & Wield, 1995]	butchers	34	1.1	0.7 – 1.5
	[Boffetta et al, 2000]	butchers or meatpackers	191	1.2	1.0 – 1.3
Kidney (189)	[Coggon & Wield, 1995]	butchers	12	1.1	0.6 – 1.9
All Lymphohaematopoietic (200-208)	[Johnson et al, 1986a]	white males in union	34	1.0	0.7 – 1.3
	[Johnson et al, 1986b]	females in union	12	0.9	0.5 – 1.5
	[Guberan et al, 1993]	self employed butchers	9	1.2	0.6 – 2.1
	[Johnson, 1994]	females in supermarket	17	1.4	0.8 – 2.2
	[Johnson, 1994]	males in supermarket	17	0.6	0.3 – 1.0

Hodgkin's disease (201)	[Johnson et al, 1986a]	white males in union	9	1.5	0.7 – 2.7
	[Coggon et al, 1989]	meat industry	2	2.4	0.3 – 8.7
	[Guberan et al, 1993]	self employed butchers	2	2.5	0.4 – 7.9
	[Coggon & Wield, 1995]	butchers	2	0.8	0.1 – 2.9
	[Boffetta et al, 2000]	butchers or meatpackers	11	0.8	0.4 – 1.4
Non-Hodgkin's lymphoma (200,202)	[Johnson et al, 1986b]	females in union	5	0.8	0.3 – 1.8
	[Johnson et al, 1986a]	white males in union	16	1.1	0.6 – 1.7
	[Guberan et al, 1993]	self employed butchers	2	1.3	0.2 – 3.9
	[Coggon & Wield, 1995]	butchers	7	0.7	0.3 – 1.4
	[Boffetta et al, 2000]	butchers or meatpackers	66	1.0	0.8 – 1.2
Multiple myeloma (203)	[Coggon et al, 1989]	meat industry	2	2.4	0.3 – 8.8
	[Coggon & Wield, 1995]	butchers	5	0.6	0.2 – 1.5
	[Boffetta et al, 2000]	butchers or meatpackers	30	0.8	0.6 – 1.2
Leukaemia (204-208)	[Johnson et al, 1986b]	females in union	5	1.0	0.4 – 2.1
	[Johnson et al, 1986a]	white males in union	8	0.6	0.3 – 1.1
	[Coggon et al, 1989]	meat industry	3	1.4	0.3 – 4.2
	[Guberan et al, 1993]	self employed butchers	5	1.7	0.7 – 3.5
	[Johnson, 1994]	females in supermarket	6	1.3	0.5 – 2.8
	[Johnson, 1994]	males in supermarket	6	0.5	0.2 – 1.2
	[Coggon & Wield, 1995]	butchers	8	0.5	0.2 – 1.1

2.6 Case control studies

Case-control studies are efficient in time and cost, allow for the investigation of a wide range of putative risk factors, and are ideally suited to the investigation of rare diseases which have long induction periods such as cancer [Dos Santos Silva, 1999]. They have been used extensively to investigate the association between cancer and work in the meat industry, and have provided insight into the specific exposures that may be responsible for any increased cancer risks. A distinction is made in the studies reviewed here according to the source of exposure information, which ranges from routine data sets to more detailed information obtained from questionnaires or personnel records in case-control studies nested within cohorts. A summary of the results from all studies is presented in table 2.4.

Lung cancer

The first of the studies of lung cancer in butchers to control for smoking involved the reanalysis of a previously collected case-control series of 1190 white male lung cancer patients admitted to Roswell Park Memorial Institute in Buffalo, New York during the period 1957-65 [Vena et al, 1982]. The 2,124 controls were selected from white male non-respiratory, non-infectious and non-cancer patients admitted during the same period. Twenty-one of the lung cancer cases, and 32 controls, had ever been employed in meat-cutting or packing related industries, giving an odds ratio of 1.1, which reduced to 1.0 when adjustment was made for smoking.

Another case-control study investigated lung cancer in butchers and slaughterhouse workers registered in the Swedish national census of 1960 [Gustavsson et al, 1987]. All males who had died of lung cancer between 1971 and 1982 were selected as the case group. This group was

compared with two reference groups, namely all males who had died of any type of cancer and a random sample of males who had died from any cause. Information on work history, occupational exposures and smoking was obtained from next of kin. The detailed work history information allowed study participants to be categorized according to exposure to a number of putative risk factors including work with live animal care, in the bleeding area, on the killing floor, or with meat cutting, processing, curing, smoking, chilling and packaging. No statistically significant increase in the rate of lung cancer was found to be associated with any of the occupational categories studied. Gustavsson et al [1982] concluded, therefore, that tobacco smoking was likely to have been the predominant factor contributing to the overall excess of lung cancer that had been found in other studies of abattoir workers.

A New Zealand Cancer Registry based case-control study evaluated cancer risks in the 19,904 males registered during the period 1980-84, using registrants for all other sites as the reference group [Reif et al, 1989]. Analyses were restricted to those aged 20 years or more, and to those who had an occupation recorded. Excess risks for lung cancer (OR 1.30, 95% CI 1.06 – 1.58, 135 exposed cases) and laryngeal cancer (OR 2.01, 95% CI 1.19 – 3.39, 15 exposed cases) were observed in those whose occupation was recorded as "meat worker". In evaluating the potential for confounding by smoking it was noted that other smoking related sites did not show any consistent elevation in risk, with cancers of the oesophagus (OR 1.38) and pancreas (OR 1.45) marginally increased while cancers of the bladder (OR 0.70), oral cavity (OR 0.66) and pharynx (OR 1.07) were not. When data on smoking and occupation from the 1981 census was analysed, however, the food and beverage workers occupational group (which included meat workers) was found to be more likely than the overall full time male work force to be a current or ever smoker. The risk attributable to the higher smoking rates among this group was estimated, according to the method of Axelson [Axelson, 1978], to be 1.20 for lung cancer and

1.16 for laryngeal cancer. It was concluded, therefore, that at least a part of the increased risk observed was likely to be due to cigarette smoking.

Johnson [1991] reported a case-control study of lung cancer nested within the cohort study of 28,901 members of the Baltimore meat cutters' union. Of the 289 cohort members who had died of lung cancer, 60 were selected at random for the case group, which was compared with a control group of 60 selected from among all other deceased members of the cohort who had not been diagnosed with lung cancer. Information on exposures and potential confounders for all study subjects were obtained from next of kin using telephone interviews. Although of limited statistical power this study did suggest an excess risk of lung cancer among those who had ever worked in the meat industry (OR 3.6, 95% CI 0.7 – 17.9, 16 cases), with a strong suggestion of a dose response based on the time between first exposure in the meat industry and diagnosis. It also found a strong association between contact with raw meat for a period ≥ 5 years (OR 7.4, 95% CI 1.3 – 43.0, 32 cases), and contact with raw meat in an abattoir for a period of ≥ 5 years (OR 13.1, 95% CI 2.0 – 86.7, 11 cases), both of which supported Coggon's earlier findings of increased risk associated with exposure to warm (freshly slaughtered) meat.

A West German case-control study of occupational risk factors for lung cancer found a two fold excess of lung cancer (OR 1.97, 95% CI 0.98 – 3.97, 29 cases) associated with the industry classification "production and processing of meat and poultry meat products", and also with the job title "butcher" (OR 2.08, 95% CI 0.97 – 4.44, 25 cases), in both instances after adjustment for smoking and asbestos exposure [Jockel et al, 1998]. In a study of incidence data for all adult males from five of the eight Swiss cancer registries for the period 1980-1993, Bouchardy et al [2002] used a case-referent approach in which for each cancer type studied, all other cancers were used as referents. A range of potential confounders, including

socioeconomic status, was controlled for in the analysis. For the occupational category "butchers and related occupations", no elevation was found for cancer of the lung (OR 0.80, 95% CI 0.60 –1.00, 69 cases).

Lymphohaematopoietic cancers

A series of case-control studies based on the New Zealand Cancer Registry also investigated the association between employment in the meat industry and lymphohaematopoietic cancers. The New Zealand Cancer Registry based case-control study of 19,904 males found a non-statistically significant elevation in risk for leukaemia (OR 1.45, 95% CI 0.90 – 2.31, 19 exposed cases), which was more pronounced for the specific subtype acute myeloid leukaemia (OR 2.12, 95% CI 1.09 – 4.12, 9 exposed cases) [Reif et al, 1989]. Another study from this series of case-control studies based on the New Zealand Cancer Registry assessed the risk of leukaemia among agricultural workers [Pearce et al, 1986]. This study compared 546 male leukaemia patients aged more than 20 years, and registered in the period 1979-1983, with four controls per case chosen at random from the Cancer Registry and matched on age and year of registration. Exposure assessment was relatively crude, being based solely on the routinely coded cancer registry record of current or most recent occupation, which would be likely to cause random misclassification of exposure with the potential to bias the observed risk towards unity. Notwithstanding this, a statistically significant excess risk for acute myeloid leukaemia was observed for those whose occupation was listed as meat worker (OR 2.51, 95% CI 1.19-5.30, 9 exposed cases).

The same relatively crude exposure measure of the current or most recent occupation recorded on the cancer registration had been used in another from this series of case-control studies, which found that workers in agricultural occupations were at increased risk of developing non-

Hodgkin's lymphoma (ICD 202) and multiple myeloma [Pearce et al, 1985]. In a follow-up study conducted to investigate this finding further, telephone interviews with both cases and controls were used to obtain a full occupational history, although the emphasis was again on occupations with the potential for exposure to phenoxyherbicides and chlorophenols [Pearce et al, 1986a]. This study compared 83 cases of non-Hodgkin's lymphoma (ICD 202) registered on the NZ Cancer Registry in the period 1977-1981 with two control groups, namely: (i) 168 other cancer patients matched for the same year of registration and a birth date within two years of that of the case, and (ii) 228 general population controls selected at random from the electoral roll. The odds ratio estimates for those individuals who had worked in the pelt department of meat works were elevated in comparisons with both the other cancer controls (OR 2.3, 90% CI 0.7-7.6) and the population controls (OR 4.1, 90% CI 1.1-14.0), although this was based on only 4 exposed cases. The odds ratio estimates, however, were also elevated for the larger group who had ever worked in the meat processing industry as a whole, with a pooled estimate calculated by combining both control groups also elevated (OR 1.8, 90% CI 1.1-3.1).

An expanded case-control study [Pearce et al, 1987] investigated the association previously found between non-Hodgkin's lymphoma and agricultural occupations by using an enlarged case group; 100 cases of lymphosarcoma and reticulosarcoma (ICD 200) during the period 1977-1981 were added to the previously studied group of 83 registrants with non-Hodgkin's lymphoma other than lymphosarcoma and reticulosarcoma (ICD 202). As with previous studies other cancer registrants were used as controls, and telephone interviews were conducted to obtain an occupational history. The previous finding of an excess risk associated with employment in a meat works was more strongly supported (OR 1.8, 90% CI 1.2-2.6, 43 exposed cases) for all non-Hodgkin's lymphoma, and again the excess risk was not confined to

pelt department workers (OR 1.9 90% CI 0.9-4.0, 10 exposed cases) or tannery workers (OR 0.5, 90% CI 0.1-1.8, 2 exposed cases). Pearce et al concluded that, as the excess risk found with meat works employment was not confined to the pelt department workers, their study provided little support for the *a priori* hypothesis of an association between chlorophenol exposure and NHL and instead raised the alternative hypothesis of exposure to oncogenic viruses.

The risk estimate for all leukaemias was also elevated (OR 1.5, 95% CI 0.9 - 2.3, 19 exposed cases) in the New Zealand registry-based study of cancer in meat workers, although it lacked precision [Reif et al, 1989]. In cell-type specific analyses, however, the earlier finding [Pearce et al, 1986] of an excess of acute myeloid leukaemia was reproduced (OR 2.1, 95% CI 1.1 - 4.1, 9 exposed cases) with stratification by age showing a higher risk (OR 2.7, 95% CI 1.2 - 6.1, 6 exposed cases) for men aged 20-64 years than for older men. An excess risk was also apparent for chronic myeloid leukaemia (OR 2.1, 95% CI 0.8 - 5.7, 4 exposed cases) and acute lymphatic leukaemia (OR 2.5, 95% CI 0.8 – 8.0, 3 exposed cases), but the estimates lacked precision due to the small numbers. However, the study did not reproduce the earlier finding [Pearce et al, 1987] of an excess risk of non-Hodgkin's lymphoma. The reasons for this are unclear, though one possibility is that the random misclassification of exposure through the use of routinely coded occupation data caused a bias toward the null, in which case the finding of an elevated risk for leukaemia in this study is particularly notable.

Whittaker presented case notes from a planned case-control study of acute lymphoblastic leukaemia in Wales, which showed that 5 of 33 cases gave an occupational history of working in an abattoir or as a butcher [Whittaker, 1991]. It was noted that none of the control subjects, who were selected from patients admitted to the same hospitals and were matched for age, sex

and area of residence, had worked with meat either as a butcher or slaughterhouse worker. In another case-control study, based on death certificate records in 16 US states, 5,147 male leukaemia deaths were compared with 51,470 males who died from other causes [Loomis and Savitz, 1991]. They found a non-statistically significant increase in risk of acute lymphocytic leukaemia (OR=2.2, 95% CI 0.7-7.0) for the occupational group "butchers and meat cutters" using the last known occupation listed on the death certificate.

A case-control study nested within the Baltimore union cohort investigated a range of risk factors for lymphohaematopoietic cancers within the meat processing industry [Metayer et al, 1998]. All deaths from these cancers that had occurred during the original study period of 1949 to 1980, plus an additional four subsequent deaths for which death certificates were available, formed the case group. Controls were selected at random from cohort members who had died of any other cause. As the response rate from the control group was low, an additional control group that had been used in the earlier nested case-control study of lung cancer was added to achieve a 1:2 case-control matching ratio. Interviews were conducted with next of kin of cases and controls to obtain information on lifetime occupational exposures, and non-occupational factors such as diet, medical conditions, medications used and leisure activities. The information on employment history was supplemented with information contained in the union records.

For all lymphohaematopoietic cancer an increase in risk was associated with ever having worked in the meat industry (OR 2.2, 95% CI 0.8-6.3, 44 exposed cases), which was stronger for those with over 5 years exposure (OR 2.9, 95% CI 1.0-8.6, 32 exposed cases) than for those with less than 5 years exposure (OR 2.1, 95% CI 0.6-7.1, 11 exposed cases). When categorised according to type of job, the strongest risk observed was for those who ever worked in an

abattoir (OR 2.8, 95% CI 0.8-9.5, 12 exposed cases) or in the meat department of a supermarket (OR 2.7, 95% CI 0.9-8.1, 25 exposed cases), but not in a meatpacking plant (OR 0.9, 95% CI 0.3-3.3, 7 exposed cases). The increased risk associated with work in an abattoir was largely confined to those with more than 5 years exposure (OR 3.7, 95% CI 1.0-14.6, 8 exposed cases), but this pattern was not evident in the supermarket workers. The specific subtype with the strongest increase in risk was non-Hodgkin's lymphoma among those who had ever worked in an abattoir (OR 12.0, 95% CI 1.1-130.6, 5 exposed cases).

A much higher elevation in risk was observed among those who had ever worked slaughtering animals (OR 5.3, 95% CI 1.0-27.0, 5 exposed cases) than among those who worked as a meat cutter but did no slaughtering (OR 1.7, 95% CI 0.5-5.5, 13 exposed cases). A higher risk was also observed for those classified as ever working with raw meat (OR 2.5, 95% CI 0.9-7.2, 37 exposed cases), confined to those who worked with raw meat in an abattoir (OR 3.4, 95% CI 0.9-12.8, 9 exposed cases) or supermarket (OR 3.2, 95% CI 1.0-9.8, 22 exposed cases) and not in a meat packing plant (OR 0.9, 95% CI 0.2-3.6, 5 exposed cases). The risk associated with handling raw meat was stronger for those with exposure of more than 5 years (OR 3.1, 95% CI 1.0-9.3, 25 exposed cases) than for those with less than 5 years (OR 1.6, 95% CI 0.4-6.1, 6 exposed cases).

A New Zealand case-control study, which contrasted the occupational and environmental exposure histories of acute leukaemia cases with population controls, has confirmed the elevation of risk for leukaemia among abattoir workers and certain butchers [Bethwaite et al, 2001]. This study compared 110 incident leukaemia cases identified from referrals to one of six treatment centres between 1989 and 1991 with 199 general population controls. Detailed occupational exposure histories were obtained by interview, and the departments and job tasks

undertaken in an abattoir were divided according to whether or not there was direct contact with live animals or animal tissues. The following job/task descriptions were classified as "animal contact": stock control (pen and move stock from trucks into works yards), stock cleaner (clean down stock before slaughter), slaughterer, meat inspector, pelt remover, butcher/boner on killing chain, carcass grader, offal packer and grader, butcher/boner on chilled carcass, meat packers (wrap and store chilled meat) and small goods workers. Freezing store workers and packers were not classified with this group, as these workers only handle and load frozen, wrapped and sealed meat.

Work in an abattoir for a period exceeding two years was found to be associated with an increased risk of acute non-lymphocytic leukaemia (OR 4.6, 95% CI 1.2-17.5, 8 exposed cases), and of acute lymphoblastic leukaemia (OR 6.2, 95% CI 1.7-23.3, 4 exposed cases). The increased acute non-lymphocytic leukaemia risks were confined to those workers having contact with animals or animal products (OR 6.8, 95% CI 1.4 – 32.3, 6 exposed cases). Work as a butcher was also associated with increased acute leukaemia risk (OR 2.9, 95% CI 1.1 – 7.2, 17 exposed cases), confined to butchers in abattoirs (OR 4.8, 95% CI 1.1 – 20.0, 7 exposed cases) and persons who butchered stock on a farm (OR 8.2, 95% CI 0.9 – 77.4, 5 exposed cases), but no increased risk was found for work as a retail/wholesale butcher or meatpacker (OR 1.2, 0.4 – 3.6, 6 exposed cases). This finding of an increased leukaemia risk associated with employment in the meat industry, which was confined to abattoir workers with over two years employment in the industry and to persons whose jobs involved contact with animals or animal tissue, added weight to the hypothesis that biological exposures may be responsible.

In a US case-control study of occupational exposures and non-Hodgkin's lymphoma, cases of small cell diffuse lymphomas (N=185), follicular lymphomas (N=268) and large cell diffuse

lymphomas (N=526) were selected from 8 state population-based cancer registries, and 1,659 controls frequency matched on registry and date of birth in 5-year categories were selected at random from the general population [Tatham et al, 1997]. Telephone interviews were conducted with all living cases and controls to obtain information on lifestyle, medical history and a detailed work history. After controlling for a number of potential confounders, including smoking, an association between work in the meat packaging/processing industry and follicular lymphomas was found (OR 1.6 95% CI 0.99 – 2.66). Some evidence of a dose-response relationship was also observed with the risk associated with exposure for ≤ 2 years (OR 1.2, 95% CI 0.6 – 2.2) increasing significantly for those with > 2 years exposure (OR 2.1, 95% CI 1.1 – 4.2).

In the Swiss Cancer Registry based study [Bouchardy et al, 2002], elevated risks were also observed for non-Hodgkin's lymphoma (OR 1.7, 95% CI 1.0 – 3.0, 15 cases), lymphoid leukaemia (OR 2.4, 95% CI 1.2-5.0, 9 cases) and for chronic lymphatic leukaemia (OR 2.7, 95% CI 1.3-5.7, 9 cases) in the occupational category "butchers and related occupations".

Other cancers

An incidental finding of a 1984 study of soft tissue sarcoma and exposure to phenoxyherbicides and chlorophenols in New Zealand was an increased risk observed in abattoir workers (RR 2.80, 90% CI 1.3-6.3, 19 exposed cases) [Smith et al, 1984]. In this study the 112 incident male cases of soft tissue sarcoma reported to the New Zealand Cancer Registry between 1976 and 1980 comprised the cases, and a similar number of controls were selected at random from all other patients in the Cancer registry with the same year of registration and with no more than two years difference in age. Telephone interviews were conducted to obtain information on occupational exposures, focusing primarily on occupations known to involve exposure to phenoxyherbicides or chlorophenols. The meat industry was

studied due to the use of 2,4,6-TCP in the pelt departments of meat works and in tanneries. However, while an elevated risk was observed for meat workers, this association was not specifically with the chlorophenol exposure. Only 6 of the 19 exposed cases worked in either a pelt department or a tannery, and of these the potential for exposure to chlorophenols could be independently confirmed in only two cases. It was thought likely that the association was with the meat processing industry overall, rather than with chlorophenol exposure. In a review of the New Zealand studies of meat workers, previously unreported findings from a case-control study of soft tissue sarcoma were presented [Pearce et al, 1988]. An increased risk (OR 1.6, 90% CI 0.9 – 3.0, 11 exposed cases) was observed, which when pooled with the earlier finding [Smith et al, 1984] gave an overall relative risk estimate of 1.9 (90% CI 1.2 – 3.1).

The only other cancer site for which a statistically significant increase amongst meat workers has been reported in more than one case-control study is laryngeal cancer. A Uruguayan study of laryngeal cancer, which was part of a multi-centre collaborative study coordinated by IARC, reported an increased risk associated with work as a butcher [De Stefani et al, 1998]. This study compared the exposure of 112 cases with that of 509 controls who were patients with other cancers, reporting a high participation rate (96.1%) in face-to-face interviews conducted shortly after admission to hospital. This interview covered sociodemographic variables, smoking, alcohol consumption and a detailed work history. Exposure as a butcher was reported by 10 cases and 16 controls, and an increased risk was observed (OR 2.8, 95% CI 1.1 – 7.2) after adjustment for a number of potential confounders including smoking and alcohol consumption. This study thus supported the earlier finding of excess risk for laryngeal cancer (OR 2.0, 95% CI 1.2 – 3.4, 15 exposed cases) among New Zealand meat workers [Reif et al, 1989], but not the Swiss cancer registry study finding (OR 1.2, 95% CI 0.6-2.4, 9 cases) [Bouchardy et al, 2002].

Table 2.4 Case control studies of cancer in meat workers

Site (ICD9 Code)	Study	Exposure category	Exposed Cases	Risk Estimate	95% Confidence Interval
Larynx (161)	[Reif et al, 1989]	Male meat workers	15	2.0	1.2 – 3.4
	[De Stefani et al, 1998]	Butchers	10	2.8	1.1 – 7.2
	[Bouchardy et al, 2002]	Butchers and related occupations	9	1.2	0.6 – 2.4
Lung (162)	[Vena et al, 1982]	Meat cutting or packing related occupations	21	1.0*	
	[Reif et al, 1989]	Male meat workers	135	1.3	1.1 – 1.6
	[Gustavsson et al, 1987]	Live animal care	-	1.5	0.5 – 4.5
		Bleeding area	-	1.0	0.4 – 2.4
		Killing area	-	0.9	0.4 – 2.0
		Chilling room	-	0.9	0.6 – 1.5
		Meat cutting	-	0.4	0.2 – 1.0
		Meat processing	-	0.6	0.3 – 1.4
		Meat curing	-	1.3	0.6 – 3.1
		Smokehouse operation	-	0.9	0.4 – 2.0
		Packaging	-	0.9	0.3 – 2.6
		Other work	-	1.1	0.5 – 2.6
	[Johnson, 1991]	Ever worked in meat industry	Not reported	3.6*	0.7 – 17.9
		Worked in meat industry			
		<5 yrs before diagnosis	16	1.6*	0.6 – 4.4
		5-10 yrs before diagnosis	23	2.7*	1.0 – 7.1
		≥10 yrs before diagnosis	44	5.5*	1.5 – 20.0
		Ever had contact with raw meat for ≥ 5 years	32	7.4*	1.3 – 43.0
		Ever had contact with raw meat in abattoirs for ≥ 5 years	11	13.1*	2.0 – 86.7
	[Jockel et al, 1998]	Production and processing of meat and poultry products	29	2.0*	1.0 – 4.0
		Butchers	25	2.1	1.0 – 4.4
	[Bouchardy et al, 2002]	Butchers and related occupations	69	0.8	0.6 – 1.0
Soft tissue sarcoma (171)	[Smith et al, 1984]	Ever worked in meat works	19	2.8	1.3 – 6.3
	[Pearce et al, 1988]	Ever worked in meat works	11	1.6	0.9 – 3.0

Lymphohaematopoietic (200 – 208)	[Metayer et al, 1998]	Ever worked in meat industry	44	2.2	0.8 – 6.3
		< 5 years exposure	11	2.1	0.6 – 7.1
		> 5 years exposure	32	2.9	1.0 – 8.6
		Ever worked in abattoir			
		< 5 years exposure	12	2.8	0.8 – 9.5
		> 5 years exposure	8	3.7	1.0 – 14.6
		Ever worked in meatpacking	7	0.9	0.3 – 3.3
		Ever being a butcher (killing)	5	5.3	1.0 – 27.0
		Ever a meat cutter (no killing)	13	1.7	0.5 – 5.5
		Worked with raw meat	37	2.5	0.9 – 7.2
		< 5 years exposure	6	1.6	0.4 – 6.1
		> 5 years exposure	25	3.1	1.0 – 9.3
NHL (202)	[Pearce et al, 1986a]	Ever worked in meat works	19	1.9	1.0 – 3.7
NHL (200, 202)	[Pearce et al, 1987]	Ever worked in meat works	43	1.8	1.2 – 2.9
NHL (200,202)	[Bouchardy et al, 2002]	Butchers and related occupations	15	1.7	1.0 – 3.0
	[Metayer et al, 1998]	Ever worked in meat industry	20	7.3	0.9 – 60.2
		Ever worked in abattoir	5	12.0	1.1 – 130.6
Follicular lymphomas (202.0)	[Tatham et al, 1997]	Ever worked in meat packing/processing	29	1.6	1.0 – 2.6
		< 2 years	14	1.2	0.6 – 2.2
		> 2 years	14	2.1	1.1 – 4.2
Multiple myeloma (203)	[Reif et al, 1989]	Male meatworkers	7	1.0	0.5 – 2.0
Leukaemia (204-208)	[Reif et al, 1989]	Male meatworkers	19	1.5	0.9 – 2.3
AML (205.0)	[Reif et al, 1989]	Male meatworkers	9	2.1	1.1 – 4.1
AML (205.0)	[Pearce et al, 1986b]	Ever worked in meat works	9	2.5	1.2 – 5.3
ALL (204.0)	[Loomis & Savitz, 1991]	Routinely coded occupation	-	2.2	0.7 - 7.0
Acute Leukaemia	[Bethwaite et al, 2001]	Ever worked in an abattoir	14	2.3	1.0 – 5.2
		≤ 2 years	3	0.8	0.2 – 3.1
		> 2 years	11	4.9	1.5 – 15.6
		animal contact	7	5.2	1.2 – 22.2
		no animal contact	7	1.5	0.5 – 4.2
		Ever worked as a butcher	17	2.9	1.1 – 7.2
		in abattoir	7	4.8	1.1 – 20.0
		farm kills	5	8.2	0.9 – 77.4
		retail butcher	6	1.2	0.4 – 3.6
Leukaemia	[Bouchardy et al, 2002]	Butchers and related occupations	14	1.4	0.8 – 2.5
Myeloid (205)			5	1.2	0.5 – 3.1
Lymphoid (204)			9	2.4	1.2 – 5.0
Chronic lymphatic (204.0)			9	2.7	1.3 – 5.7

* Adjusted for smoking.

2.7 Studies of other relevant populations

Agriculture workers

Studies of several related agricultural occupations have shown similar excess cancer risks, particularly for lymphatic and haematopoietic cancers. For example there have been a number of studies of cancer risks in farmers [Blair et al, 1985; Pearce & Reif, 1990; Blair & Zahm, 1991, 1995]. The overall cancer incidence and mortality in farmers is considerably lower than in the general population, due largely to reduced risks for cancers of the lung, bladder, oesophagus, colon, rectum, liver and kidney. The reduction in overall cancer risk among farmers, and in risk of specific cancers such as lung, oesophageal and colon, has been attributed to a generally healthy lifestyle (with low rates of smoking and alcohol consumption), as well as to a protective effect of physical exercise [Pearce & Reif, 1990].

A higher than average risk for lip, brain, stomach and prostate cancer, malignant melanoma, and cancer of the lymphatic and haematopoietic system (including Hodgkin's disease, leukaemia, non-Hodgkin's lymphoma and multiple myeloma), however, has also been a consistent finding. A number of potential exposures in agricultural occupations have been proposed as possible causes of the increased risk of specific cancers such as those of the lymphatic and haematopoietic system in farmers, although none explain all the excesses observed. The three main hypotheses that have been proposed are: (i) exposure to agricultural chemicals (and in particular organochlorines), (ii) exposure to oncogenic animal viruses, and (iii) chronic exposure to as yet unidentified agents (but possibly animal proteins, organic dusts, pesticides) that cause perturbation of the immune system [Pearce & Reif, 1990; Kristensen et al, 1996].

The series of New Zealand Cancer Registry based case-control studies conducted in the 1980s that investigated cancers in farmers found significant increases in risk of multiple myeloma (OR 1.7, 95% CI 1.0 – 2.9) [Pearce et al, 1986b], and in particular among sheep farmers (OR 1.9, 95% CI 1.0 – 3.6) and those with a history of exposure to beef cattle (OR 1.7, 95% CI 1.0 – 2.9), and a non-statistically significant increase in risk of leukaemia (OR 1.24, 95% CI 0.93 – 1.66) which was strongest among those working in livestock farming (OR 3.00, 95% CI 1.23 – 7.32) [Pearce et al, 1986]. No excess risk among farmers was observed in studies of non-Hodgkin's lymphoma [Pearce et al, 1986a; Pearce et al, 1987] or soft tissue sarcoma [Smith et al, 1984].

An Italian population-based case-control study of non-Hodgkin's lymphoma and chronic lymphocytic leukaemias, found a significantly elevated risk (OR 1.8, 95% CI 1.2 – 2.6) for individuals working in agricultural occupations associated with animal breeding [Amadori et al, 1995]. These risks were primarily for leukaemia (OR 3.1, 95% CI 1.1 – 8.3) and for NHL (OR 2.2, 95% CI 1.2 – 4.3). A subsequent reanalysis of these data found a strong association with exposure to cattle (OR 1.7, 95% CI 1.2 – 2.6), sheep (OR 2.4, 95% CI 1.4 – 4.0) and pigs (OR 1.8, 95% CI 1.2 – 2.7) [Nanni et al, 1996]. A similar association between exposure to farm animals and hairy cell leukaemia (OR 2.0, 95% CI 1.2 – 3.2) has been observed in a Swedish case-control study [Nordstrom et al, 1998].

Subsequent studies have confirmed the lower overall cancer risk in male (SIR 77, 95% CI 73-81) and female (SIR 92, 95% CI 85-99) farmers in Norway [Kristensen et al, 1996]. There were no major differences in cancer risk between males and females in this study, with elevated risk for lip cancer, leukaemia and Hodgkin's disease apparent in both genders, although female farmers had a noticeable elevation in bladder cancer (SIR 128, 95% CI 72-

207) which stood out when compared with the lower risk for males (SIR 73, 95% CI 60-88). A recent meta-analysis of 32 studies of multiple myeloma published between 1981 and 1996 confirmed the earlier reports of excess risks for this cancer, yielding a combined estimate of relative risk of 1.23 (95% CI 1.14 – 1.32) [Khuder & Mutgi, 1997]. An updated proportionate mortality analysis of cancer in Iowa farmers for the years 1987 - 1993 showed the same clear pattern of deficits for all cancers, and for lung and liver cancer, but excess deaths for multiple myeloma (PMR 1.17, 95% CI 0.98 – 1.40), non- Hodgkin's lymphoma (PMR1.09, 95% CI 0.96 – 1.23), and Hodgkin's disease (PMR 1.62, 95% CI 1.04 – 2.54) [Cerhan et al, 1998].

Veterinarians

Veterinarians are an occupational group that experience similar exposures to many in the agricultural occupations, namely to pesticides, animal remedies and zoonotic microorganisms. Their cancer risk has also been studied [Blair & Hayes, 1980, 1982; Kinlen, 1983; Miller & Beaumont, 1995] and recently reviewed [Fritschi, 2000] and there is some evidence that they experience similar patterns of risk to those in meatworkers and other agricultural occupations. They experience no overall excess of cancer, and significantly lower risks of lung cancer, but do appear to experience increased risks of melanoma and cancer of the lymphatic and haematopoietic system. In the largest study conducted, a proportionate mortality study of 5,016 white male veterinarians dying between 1947 and 1977, a statistically significant PMR of 1.5 (112 deaths) was found for all lymphatic and haematopoietic cancers combined [Blair & Hayes, 1982]. These included statistically significant elevations for Hodgkin's disease (PMR 1.9, 18 deaths) and cancer of other lymphatic tissue (PMR 1.9, 26 deaths). When the different professional specialties were examined, a significant elevation in all lymphatic and haematopoietic cancer was observed in meat inspectors (PMR 3.4, 8 deaths), which is consistent with the increased risks seen in meat workers. A case-control study of multiple

myeloma, in which 12,148 cases frequency matched by age, race and gender with 5 controls per case, also found a statistically significant risk (OR 3.2, 95% CI 1.1 – 9.0, 6 exposed cases) in veterinarians compared with all other occupations [Figgs et al, 1994].

Poultry workers

Another occupational group with similar exposures are workers engaged in the slaughter and processing of poultry. The Baltimore union cohort included a sub cohort of members with lifetime employment in this industry, which was included in the original PMR and SMR analyses as well as in an updated analysis [Johnson et al, 1997]. A total of 2,639 workers in poultry slaughtering plants were followed from 1949 to 1989, with standardised mortality ratios calculated with reference to US general population rates. In addition a comparison was made with the mortality experience of 6,081 unexposed workers from the same union who had worked only in non-meat industries to derive estimates of relative risk. No statistically significant increase in risk was observed in the comparison with general population rates, although in the comparison with the non-meat industry workers statistically significant increases were observed for cancer of the oesophagus (RR 4.7, 95% CI 1.1 – 22.5, 6 deaths) and of the lymphohaematopoietic system (RR 2.9, 95% CI 1.0 – 8.1, 8 deaths).

2.8 Summary

The studies reviewed above are analyses of routinely collected data, proportionate mortality or incidence studies, case-control studies of specific cancer sites, or cohort studies involving relatively small numbers or inadequate exposure data. Thus, the available evidence for increased risk for specific types of cancers is preliminary. The strength of the evidence for

these associations is summarised below taking account of potential sources of bias or confounding.

All cancers

A small excess risk for all cancers has been observed in all but one [Coggon & Wield, 1995] of the reported cohort studies of butchers, meatpackers or meat industry workers [Johnson et al, 1986a, 1986b, 1995; Johnson, 1989; Coggon et al, 1989; Guberan et al, 1993; Boffetta et al, 2000]. This observation is of interest as it is normal to observe lower relative mortality in occupational cohort studies, when comparisons are made with those in the general population, due to the "healthy worker effect" that arises because healthy people are more likely to gain employment and to remain in employment [McMichael, 1976]. Although the healthy worker effect is generally less pronounced in studies of cancer, it is nevertheless still normal to observe SMRs for all cancers below unity in occupational cohorts [Checkoway et al, 1989]. This suggests a small excess risk of all cancers for those working in abattoirs or meat cutting and packing.

Lung cancer

The evidence for an increased risk of lung cancer among people occupationally exposed to meat and meat products is reasonably consistent, having been reported in several countries across different time periods and in a range of study types, including both cohort and case-control studies. There is a possibility that part of the observed increase may be due to confounding by smoking, but sufficient studies that have adjusted for this either directly or indirectly have indicated that the excess is over and above that which could be attributed to smoking or other lifestyle factors. A number of chemical exposures occurring in the meat

processing industry originally thought to be risk factors have also been tested and excluded, and the strongest association is found with those involved directly with the slaughter of animals.

A review of the literature on lung cancer among butchers concluded that, as the majority of studies reported excess risks and that as those studies reporting excess risk tended to be of better quality, butchers in general do have an excess risk of lung cancer [Kristensen & Lynge, 1993]. While the majority of studies reviewed lacked individual smoking data it was considered that the use of indirect control methods, including analysis of risk for other smoking related diseases in the study population and adjustment for differences in smoking rates between exposed and unexposed groups, provided sufficient evidence that the excess risk of lung cancer in butchers exceeded the likely excess risk that could be attributed to smoking. After evaluating the evidence for the hypothesis that chemical exposures including nitrite from curing processes, products of incomplete combustion from smoking processes and thermal decomposition products from plastics used in meat wrapping were the cause of this excess risk it was concluded that there was little evidence in support of any of them. They concluded instead that the remaining hypothesis of an aetiological role for a biological exposure, such as to a papilloma virus, was supported most strongly by the available evidence and that it warranted further investigation.

Studies of lung cancer in meat workers published since that review have provided consistent support for the association, and taken together they provide strong support for the hypothesis that the association is strongest for work involving the preparatory handling of livestock and then specifically the slaughter of animals, rather than the general work of meat cutting performed by butchers. The one new analysis of routine records from Italy suggested a strong

and statistically significant risk (MORs of 2.78 and 2.45) related specifically to those employed as slaughterers [Lagorio et al, 1995], while the UK cohort of a broader occupational grouping of butchers experienced no excess in lung cancer mortality [Coggon & Wield, 1995]. The large Swedish cohort found a small but statistically significant overall excess (SIR 1.3) among those in the broad occupational category of "butchers or meatpackers", or "employed in butcher shops, meat processing or packing". The relative risk, using other workers excluding those employed in animal-related jobs as a reference, was higher for butchers employed in the meat industry (RR 1.4, 95% CI 1.1 – 2.0) than it was for those working as either a butcher outside the meat industry (RR 1.2, 95% CI 0.6 – 2.5) or in other jobs within the meat industry (RR 1.1, 0.8 – 1.6) [Boffetta et al, 2000].

Tobacco smoking is a strong potential confounder in any study of the association between work in a particular occupation and the risk of lung cancer risk, and must be considered when interpreting these findings. As with the studies reviewed previously by Kristensen and Lynge, however, there is sufficient evidence in the more recent studies to suggest that the increase observed is greater than that which could be attributed to smoking. Although these excesses observed in the Swedish cohort are within the range that could be attributed to different smoking rates, it would be reasonable to assume similar smoking rates within these groups. In addition, several studies have controlled for smoking including the case-control study nested within the Baltimore meat cutters' union cohort. This study provided support for the existence of an excess of lung cancer associated with ever having worked in the meat industry (OR 3.6, 95% CI 0.7-17.9), and showed that this excess was unrelated to smoking [Johnson, 1991]. The case-control study from Germany also reported a doubling of the lung cancer risk among workers in the production and processing of meat and poultry meat products and among butchers, after adjustment for both smoking and asbestos exposure [Jockel et al, 1998].

Although the numbers were small, the case-control study nested within the Baltimore meat cutters' union cohort also observed a strong dose response based on duration of employment in the meat industry and on duration of exposure to unwrapped raw meat [Johnson, 1991]. The highest risks observed were associated with working in an abattoir (OR 5.3, 95 % CI0.4-64.1), and specifically in the stockyard or kill/dress area of an abattoir (OR 5.6, 95% CI 0.6-49.8), although risks were also elevated for those who had ever worked in a meat packing plant (OR 7.9, 95% CI 0.4-163.7). Those most at risk were those who had ever had contact with raw meat in an abattoir for more than 5 years (OR 13.1, 95% CI 2.0-86.7).

Overall these studies support the hypothesis that meat workers are at a significantly elevated risk of lung cancer, which is greater than that which could be attributed to smoking. It also appears that the risk is associated most strongly with animal handling, and with animal slaughter and exposure to freshly slaughtered meat, which supports the hypothesis of an aetiological role for biological exposures.

Cancers of the lymphohaematopoietic system

The lymphohaematopoietic cancers are a heterogeneous group of malignancies, and although a large number of epidemiological studies have investigated risk factors for leukaemia, lymphoma and multiple myeloma the aetiology of these cancers is still poorly understood [Constantini et al, 2001]. The difficulties inherent in interpreting studies of any disease are exacerbated in the case of studies of these malignancies by the use of different classifications or groupings of the various subtypes, which in many cases are on the basis of strict coding according to the International Classification of Diseases but in others are based on attempts to combine groups of distinct pathological entities. The relative rarity of these cancers limits the

precision of risk estimates for the specific subtypes in all but very large studies. In addition, if the effect of any risk factor is similar across all subtypes then the maximum effect would be observed by taking all lymphohaematopoietic cancers as the outcome of interest, while if it affects only one or two of the subtypes then its effect on the entire class of cancers of the lymphohaematopoietic system would be diluted.

Elevated risks of lymphatic and haematopoietic cancers amongst workers in the meat industry have been observed in all the study types reviewed here, although with these relatively rare cancers the numbers of cases were invariably small in cohort studies and the relative risks were generally lower than those observed in case-control studies. The risk factors that have been evaluated as potential causes of increased risk have generally been the same as those proposed for lung cancer, i.e. chemical and biological exposures. The results of studies of lymphohaematopoietic cancers overall have been inconsistent, but suggest an increased risk for those with either exposure to the slaughtering process or longer duration of exposure in the meat industry.

Although no excess in proportionate mortality (PMR 0.9) for all lymphohaematopoietic cancers was observed among the white male members of the Baltimore union cohort [Johnson et al, 1986a], an excess (PMR 2.2) was observed in white female members [Johnson et al, 1986b]. Among the males an excess (PMR 1.2) was apparent only in those union members working in abattoirs, although all the excess in females occurred amongst those working in supermarkets. Analyses of standardised mortality ratios for the union cohort showed no overall excess for males (SMR 1.0) or females (SMR 0.9), while those for the Geneva cohort showed an excess (SMR 1.2) among self employed butchers [Guberan et al, 1993]. For those studies reporting estimates only for specific subtypes, an approximate estimate of overall

lymphohaematopoietic cancer risk can be constructed by pooling the reported results. Using this method excess risk (SMR 1.5, 95% CI 0.7 – 2.9) was observed in abattoir workers [Coggon et al, 1989] and in those members of the Swedish cohort employed long term (i.e. at both the 1960 and 1970 censuses) as butchers (SIR 1.3, 95% CI 0.9 – 2.0) [Boffetta et al, 2000].

The case-control study of lymphatic and haematopoietic cancers nested within the Baltimore union cohort, although potentially subject to selection bias due to the very low response rate from next of kin of the initial control group selected, found an excess of lymphohaematopoietic cancers among those who had ever worked in the meat industry but not in meat packing [Metayer et al, 1998]. In particular this study suggested a dose response based on duration of employment in the industry, with higher risks being associated with employment in abattoirs, with employment as a slaughterer but not a meat cutter, and with contact with raw meat in abattoirs but not in meat cutting plants. This study, however, also found elevated risk in those employed in the meat department of supermarkets, with significant elevation for those either working with raw meat, wrapping meat, being a meat cutter, or working with fumes. These findings for supermarket workers are not consistent with the other studies, and may be due to chance or other risk factors present in the supermarket environment.

The most consistent reports of increased risk for any of the subtypes of the lymphohaematopoietic cancers have been for leukaemia, although this is also not a single disease entity, but rather is a heterogeneous group of both myeloproliferative and lymphoproliferative disorders. While no excess was observed amongst the Baltimore union cohort [Johnson et al, 1986a, 1986b] or in UK butchers [Coggon & Wield, 1995], excesses have been observed in analyses of routine data [Morton & Marjanovic, 1984], in cohorts of

abattoir workers [Coggon et al, 1989], self employed butchers [Guberan et al, 1993] and butchers and meat workers [Boffetta et al, 2000], as well as in case-control studies of meat workers [Reif et al, 1989; Pearce et al, 1986a; Loomis & Savitz, 1991; Bethwaite et al, 2001] or butchers and related occupations [Bouchardy et al, 2002]. The case-control study nested within the Baltimore union cohort found an elevated risk associated with work in the meat industry, and in particular among those working with raw meat in an abattoir, although these estimates were based on small numbers and were imprecise [Metayer et al, 1998]. The New Zealand case control study of acute leukaemia has provided the strongest support for the existence of an association between these cancers and work in the meat industry [Bethwaite et al, 2001]. This study suggested that the risk is associated most strongly with contact with animals and with work as a butcher involved in slaughtering animals, but not in retail butchers who work essentially as meat cutters, and demonstrated a strong dose-response relationship based on duration of exposure.

The evidence for associations between work in the meat industry and the lymphomas is much less consistent. An elevated risk of non-Hodgkin's lymphoma was first reported in the Baltimore meat cutters' union [Johnson & Fischman, 1982], and subsequently in individuals who had worked in the meat industry in two New Zealand case-control studies [Pearce et al, 1986a; Pearce et al, 1987]. No elevation has been observed in any of the cohort studies conducted [Johnson et al, 1986a, 1986b, 1995; Coggon et al, 1989; Coggon & Wield, 1995; Guberan et al, 1993; Boffetta et al, 2000], although in all instances numbers have been small. Two recent case-control studies have again found significant associations between work as a butcher or related occupation and NHL [Bouchardy et al, 2002] and work in meat packaging/processing and follicular lymphoma [Tatham et al, 1997]. Similarly for Hodgkin's disease a strong elevation in risk has been observed in abattoir workers in cohort studies

[Johnson et al, 1986a; Coggon et al, 1989] and of self employed butchers [Guberan et al, 1993], while no association was observed in studies of the broader occupational classification of butchers [Coggon & Wield, 1995; Boffetta et al, 2000]. No elevation in risk was observed in the two case-control series that used other cancer registrants as controls [Reif et al, 1989; Bouchardy et al, 2002] and no other case-control studies of this specific cancer have reported any association with work in the meat industry.

In contrast to the increase in multiple myeloma that has been observed in farmers [Khuder & Mutgi, 1997], there is little evidence for any increase in risk associated with work in the meat industry. Five studies have evaluated risk for multiple myeloma; with one study reporting an excess based on only two cases [Coggon et al, 1989], while the others reported slight deficits [Coggon & Wield, 1995, Boffetta et al, 2000, Reif et al, 1989, Bouchardy et al, 2002].

Thus, the available evidence suggests a small excess in risk of lymphohaematopoietic cancers associated with work in the meat industry, which is most strongly associated with exposure to the slaughtering process, and which is most likely to manifest as a leukaemia. However, these findings are most consistent in case-control studies and there is less evidence of an increased risk in cohort studies.

Other cancers

Associations between work in the meat industry and a number of other cancers have been observed in the studies reviewed here, although in most cases these have been isolated observations or there is potential for confounding by smoking.

The specific cancer for which the strongest evidence exists for an excess associated with work in the meat industry is laryngeal cancer, with an approximate doubling of risk having been observed in two cohorts [Guberan et al, 1993; Boffetta et al, 2000] and two case control studies [Reif et al, 1989; De Stefani et al, 1998]. While neither cohort study could control for smoking or alcohol intake, in the New Zealand case control study census records of smoking rates by occupation were used to calculate the likely contribution of smoking to the increased risk observed and it was found to be negligible [Reif et al, 1989]. Information on both smoking and alcohol intake were obtained through interview in the Uruguayan study, and reported risk estimates were adjusted for both factors [De Stefani et al, 1998]. This evidence suggests that meat workers are at increased risk for laryngeal cancer.

Other cancers for which excesses have been reported in more than one study include oesophageal cancer, rectal and colorectal cancer, and prostate cancer. A marginal excess of oesophageal cancer has been observed in the US union cohort in both abattoir workers [Johnson et al, 1995] and meat packing plant workers [Johnson, 1989; Johnson et al, 1995] and in butchers in Switzerland [Guberan et al, 1993] and Sweden [Boffetta et al, 2000]. No case control study has reported any association between work in the meat industry and oesophageal cancer, however, and smoking could account for the excess observed in these cohorts. Similar small excesses of both prostate and colorectal cancers have been observed in cohort studies [Coggon et al, 1989; Coggon & Wield, 1995; Guberan et al, 1993; Boffetta et al, 2000] and of prostate cancer in the Swiss cancer registry based case control study [Bouchardy et al, 2002]. However none of these studies could control for potential confounding by the consumption of animal fat. There is no consistent evidence for an association between work in the meat industry and any other cancer type.

Chapter 3 - Exposures in the meat industry

3.1 Introduction

Most commercial meat processing in developed countries is conducted as a high volume commodity production process in a factory environment. Stock is moved from holding pens to be stunned immediately prior to slaughter, after which it moves through the plant on a mobile production line of overhead conveyors (referred to as the "chain") with individual workers performing specialised tasks in a series of clearly defined process steps to break the animal down into its component parts. It remains a labour intensive industry with only limited automation of a few operations such as head, hock and pelt removal, and the use of power saws in a few cutting operations.

Meat processing plants in New Zealand are commonly referred to as "freezing works", a term derived from the significant export trade of frozen meat which began with the departure of the first refrigerated shipment on the sailing ship Dunedin in February 1882. Work in freezing works is predominantly seasonal, with the length of season varying from approximately nine months in the north to six or seven months in the southernmost regions. Other factors such as climatic conditions and stock availability may also influence the length of each season, and the level of production varies over the course of the season with an extra chain or shift being added in some works to increase capacity at it's height.

In spite of the seasonal nature of the work, however, the New Zealand meat industry workforce is remarkably stable with annual turnover of no more than 10-15%. In addition, once employed in the industry, workers tend not to change jobs due to strict redundancy provisions in union collective agreements. These apply the principle of "first on last off", thus providing a strong incentive for workers to maintain any seniority they have earned by accumulating service in the same job.

Published literature on exposures of meatworkers is sparse. The assessment of potential exposures presented in this chapter has been developed from a review of the limited published literature on exposures in meat workers obtained through searching computerised databases of the medical and veterinary literature, and in particular MEDLINE (US National Library of Medicine) since 1966. To supplement the limited amount of exposure information available, much of the information on exposure contained in this dissertation is based on interviews with workers, unions, and management in the industry. There has also been extrapolation about the prevalence and magnitude of exposure, and of the potential for transmission of infectious agents, made from the more extensive literature on microbiological quality within the freezing works environment contained in the food quality and veterinary literature.

As expected in an agricultural industry, the primary exposures are to biological agents, including bacterial and viral infectious agents as well as non-infectious bio-aerosols. Exposures to some chemical agents are associated with specific products or processes such as the treatment of hides and pelts, and there is also recognised potential for

exposure to animal remedies and agricultural compounds including hormone growth promoters and dieldrin. While comprehensive information about routes of transmission and prevalence of worker exposure is available for some of the zoonotic infections such as leptospirosis and brucellosis, little is known about the potential for human exposure to animal viruses with known oncogenic potential such as bovine leukaemia virus (BLV).

In the absence of such data it is difficult to develop exposure profiles apart from assigning job titles and/or work areas to fairly broad categories in nominal (yes/no) or ordinal (low, medium, high) rankings of jobs with respect to potential for exposure to factors such as:

- live animals,
- animal pelts or hides,
- the slaughter process or freshly slaughtered meat,
- animal urine,
- gastrointestinal microflora through animal faeces or gut contents,
- blood-borne infectious agents.
- specific process chemicals

It is also possible to further categorise workers according to their exposure to ovine or bovine stock, as individual freezing works either process a single species or operate discrete chains and further processing lines for each species.

3.2 Process description

Meat processing is a significant industry in New Zealand contributing almost 20% of the country's export receipts, with approximately 20,000 people directly employed in meat processing and packing plants in 1998 [www.maf.govt.nz/statistics]. The predominant species processed in the New Zealand meat industry is sheep, with significantly fewer cattle, while other species such as pigs, deer, goats and horses are processed in only small numbers in specialised abattoirs. Total slaughter numbers by species in New Zealand in 1998 are shown in Table 3.1 below:

Table 3.1 Total numbers of stock slaughtered in New Zealand in 1998

Species	Total number slaughtered (%)
Lambs	27,062,560 (71)
Adult sheep	5,865,686 (15)
Calves and vealers	1,402,689 (4)
Adult cattle	2,465,658 (7)
Pigs	776,856 (2)
Deer	412,059 (1)
Goats	144,647 (<1)
Horses	4,246 (<1)
Total	38,134,401 (100%)

All stock processed in New Zealand freezing works has been reared on pastoral farms. Once transported to the freezing works, the stock received for slaughter is held temporarily in holding paddocks before transfer to roofed stockyards attached to the main freezing works. The process that follows is essentially the same for sheep and cattle,

although where there are any differences these are noted in the following process outline. From the stockyards animals are driven along a race that leads up to the entry to the slaughterhouse.

The first process undertaken within the slaughterhouse is the stunning of the animal with an electrical stunner, or in the case of cattle with a bolt stunner gun that drives a pin into the animal's head. Most meat produced in New Zealand is killed in accordance with the requirements of the Muslim religion, by a "Halal Sticker" who bleeds the animal by sticking the jugular arteries in the neck prior to the throat being cut. The animal is then inverted and hung by its rear hocks to the moving overhead chain conveyor that transports it through the remainder of the process, and the draining blood is collected in stainless steel drip trays for further processing.

As the animal moves along the overhead conveyor system butchers and slaughterhouse labourers or assistants make a series of cuts to facilitate placement of a clip on the intestines to prevent discharge of gut contents, and to facilitate the removal of the pelt by an automatic pelt remover. In modern works an automatic decapitator removes the head, and the front hocks are severed in an automatic hock remover. Gut contents and edible organs are removed for further processing in the "gut and bung" section, with the viscera remaining intact provided the clip remains in place.

At several stages during the slaughter and evisceration process the carcass is rinsed with hot water and/or steam for hygiene purposes, and slaughter floor personnel wash knives

and their hands in a hot water and detergent solution between carcasses to prevent the transfer of microbial contamination. The overhead conveyor system transports the carcasses along the chain past trimmers and graders who cut off diseased, bruised or otherwise defective tissue, and mark carcasses according to quality criteria. Government meat inspectors check both meat and offal for evidence of transmissible disease and of faecal or other types of contamination. Ovine carcasses remain intact to this stage, while bovine carcasses are split in half vertically along the spinal column before further processing.

Carcasses are then transferred on the overhead conveyor to the cooling floor or chillers where they are conditioned for at least 12 hours prior to either transfer to freezing chambers or to the boning or cutting room for further processing. Where further processing is carried out, the carcass is broken down using power saws into front and hindquarters, and individual parts are deboned and divided into prime cuts by hand. The product is then wrapped, chilled, frozen or otherwise prepared for despatch.

Edible offal (otherwise known as smallgoods or fancy meats), which includes kidneys, livers, brains, hearts and tongues are processed in a separate department. For example tongues may be processed by either boiling or tinning, or preserving in brine solutions. Some inedible offal is also recovered, for example the casings (or intestines) are passed through rollers or 'runners' to remove faecal material and then cured in salt. Unwanted by-products are digested or rendered in a separate operation to produce meat and bone meal.

Animal hides and pelts are partially processed in separate departments in freezing works by treating them to remove wool or hair in the fellmongery, and then partially preserving them prior to their transport to separate tanneries that are stand-alone operations. Depilation, or separation of wool or hair from the skin, occurs on the paint table where a solution of sodium sulphide containing calcium hydroxide is applied to the inside of the skin. This solution penetrates through the skins, dissolves the roots, and the hair or wool may then be removed. Wool removed in this way is referred to as 'slipe wool'. The skins are then immersed for a further period in a solution of lime and sodium sulphide, known as the 'liming' process, to remove any unpulled wool or hair and also the epidermal layer of the skin. The pH of the treating solution is then lowered and protein enzymes are added to soften the pelt in the 'bating' process. The addition of salt and sulphuric acid to the solution, i.e. the pickling process, preserves the pelt during storage prior to tanning. The use of preservative chemicals such as trichlorophenate and β-naphthol was common; however these have been replaced by substitutes containing alternative fungicides such as sodium mercaptobenzothiazole (MTB), 2-(thiocyanomethylthio) benzothiazole (TCMTB), boric acid and carbendazim.

As in any large industrial operation there are a number of both skilled and unskilled maintenance staff employed to repair and maintain the plant. A number of trades are represented, including boiler attendants, carpenters, electricians, engineering fitters, painters and plumbers, as well as workers engaged in cleaning, laundry and stores. These workers perform the tasks routinely associated with their trades in any industrial plant,

although their work in this industry does entail significant contact with biological wastes, ammonia used as a refrigerant and emissions from welding and gas cutting of the stainless steel that is used extensively in these plants for hygiene reasons.

3.3 Potential exposures

Potentially hazardous exposures in the meat industry include a range of biological exposures (animal diseases including infectious zoonotic microorganisms, animal proteins and dusts), as well as a limited range of chemical exposures either from chemicals used in the process or maintenance of plant and equipment, or from residues of animal remedies or pesticides etc used on pasture on farms. There are also well-recognised physical and psychosocial stresses associated with work in this industry, including musculoskeletal injuries, machine pacing of work and shiftwork. The literature on anything other than the zoonoses, however, is sparse or non-existent.

As the process is labour intensive the workers may come into intimate contact with animal faecal matter or urine, and the blood and internal organs of animals. The physical nature of the work in abattoirs also means that cuts and abrasions are frequent, and as workers' hands are constantly wet so also is dermatitis, all of which afford microbial agents easy access into the body through the skin. There is also potential for the generation and transmission of bio-aerosols, and airborne transmission is known to play a significant role in the spread of microbial contamination throughout the abattoir environment [Rahkio & Korkeala, 1997]. Furthermore, although there is limited use of

power tools in meat processing, their use for selected operations such as carcass splitting means that there is the potential for the generation and dispersion of bio-aerosols containing blood and other tissue in much the same manner as that which has been observed in operating theatres during surgical operations [Jewett et al, 1992; Nogler et al, 2001].

In addition to the process exposures, maintenance workers in the meat industry experience the exposures common to their specific trades including the emissions from welding of stainless steel, asbestos lagging, ammonia used as a refrigerant gas, and entry into confined spaces in which there are at times elevated levels of methane (and the consequent displacement of oxygen) produced by the decomposition of organic material.

3.3.1 Infectious Biological Exposures

Biological agents may be derived from plant or animal matter, or from microorganisms, and can be either infectious or non-infectious. Non-infectious biological agents can be further divided into viable organisms, biogenic toxins produced by bacterial or fungal metabolism, or biogenic allergens such as proteins from animal skin, hair from furs and protein from faecal material or urine. The effects of exposure to biological agents are known to include contagious infectious diseases, acute toxic effects, allergies and cancer [Douwes et al, 2003].

Both bacteria and viruses are recognised causes of cancer, in animals and humans. Examples include various oncogenic retroviruses such as bovine leukaemia virus (BLV),

which is closely associated with the development of leukaemia and lymphoma in both sheep and cattle, and the avian leukosis viruses (ALVs) that cause a wide variety of neoplasms in various avian species [Burmeister, 2001]. In humans the spirochaete *Helicobacter pylori* is involved in the development of gastric cancer [IARC, 1994], and the Human Papilloma Virus types 16 and 18 are associated with invasive cervical cancer [IARC, 1995].

For many other viruses there is strong evidence supporting a causal role in the development of cancer, although in many cases malignant progression has not been documented in the absence of additional carcinogens. For example although hepatitis B virus is a recognised cause of liver cancer in humans, the large discrepancy in rates between the West and certain regions of Africa and Asia is due in part to the interaction between the virus and aflatoxin [Malkin, 2002]. There is a similar synergy in carcinogenesis observed in cattle where infection with bovine Papillomavirus BPV-2 or BPV-4 result respectively in the development of bladder and alimentary tract cancers, but only where naturally occurring immuno-suppressants and mutagens are also present [IARC, 1995]. Viral infection in these cases appears to be a component of the development of cancer, but insufficient on its own. This is not unusual, however, as virtually all established carcinogens (e.g. tobacco smoke, asbestos) are neither necessary nor sufficient carcinogens, but rather affect one or more steps of a multistage process.

3.3.1. (i) Zoonoses

Transmission of zoonotic infections from animals (or their waste products) to meat workers has been common in the New Zealand meat industry, including bacterial and viral infections such as brucellosis [Glass, 1963], leptospirosis [Thornley et al, 2002; Terry et al, 2000], butchers' warts caused by HPV [Jennings et al, 1984] and Parapoxvirus orf [Robinson & Peterson, 1983]. These infectious agents may be transmitted by direct contact between the source and host, by airborne transmission from aerosols generated during the slaughtering process, or through direct contact with the urine of infected animals that is released involuntarily throughout the slaughter process. They may enter the human body through skin, mucous membranes, or by inhalation or ingestion.

For airborne transmission of bioaerosols, the size of the aerosol produced by a process is critical to the extent to which infectious agents may be dispersed. Larger aerosols with an equivalent aerodynamic diameter greater than approximately 10μm will affect only the local area immediately adjacent to their source, while smaller aerosols less than approximately 5μm in diameter may remain airborne for longer periods and may travel considerable distances from the immediate source of infected material. Particle size, and therefore the potential extent of dispersion once airborne, has a significant bearing on whether only those immediately adjacent to a process are exposed or whether exposure is more widespread throughout the plant.

While there are no reports in the literature of assessment of particle size distributions in this environment, the evidence of the airborne spread of microbial contamination within abattoirs [Rahkio & Korkeala, 1997] and of bio-aerosol dispersion in operating theatres [Jewett et al, 1992; Nogler et al, 2001] would suggest that these aerosols are small. In an experiment in which a strain of *Pseudomonas fluorescens* was used as a marker to model the movement of bovine central nervous system material within the abattoir environment following use of a captive bolt stunner gun, the marker organism was subsequently found to be widely dispersed within the slaughter floor, including on the hands of meat workers [Daly et al, 2002]. Even those infectious agents that are the most fragile when outside their natural host organism, e.g. the retroviruses that remain viable for only a short time (minutes to hours) when airborne [Dinter & Morein, 1990], are likely to remain viable for longer periods when incorporated in a matrix of blood or other tissue in bio-aerosols.

Other environmental factors, such as the constantly wet hands of freezing workers, can also play a role in transmission of infectious agents. The severely abraded and continuously wet skin on the hands of workers in an Australian abattoir were found to enhance colonisation of *Staphylococci* to the extent that the workers themselves became the primary source of microbiological contamination of carcasses [Vanderlinde et al, 1999]. In order to minimise the risk of cross contamination of carcasses, however, workers in the New Zealand meat industry employed on the slaughter floor are not permitted to wear gloves as protection against either knife injuries or infectious agents [Legg et al, 1999]. Under current Food Assurance Authority regulations meat processing workers are also not permitted to wear barrier creams as protection against skin irritation.

In the following section the zoonoses known to affect freezing workers are described. Although not necessarily related to cancer or other causes of mortality, these zoonoses are discussed to demonstrate the potential for the transmission of infectious biological agents to freezing workers, and to describe the pathways of transmission.

Bacterial zoonoses

Brucellosis or undulant fever is a systemic infection caused by bacteria belonging to the genus *Brucella*, the main members of which are *B. abortus* associated with infection in cattle, *B. ovis* associated with infection in rams, *B. melitensis* associated with infection in sheep and goats and *B. suis* associated with infection in pigs. It is predominantly an occupational disease of those working with infected animals or their tissues, such as farm and abattoir workers or veterinarians. Transmission of the infection is by ingestion of unpasteurised milk or dairy products, or by direct skin contact with infected tissues, blood, urine, vaginal discharges, aborted foetuses and especially placentas from infected animals. In the meat-processing environment, transmission through the respiratory tract when the infectious organism becomes airborne has also been described [Rodriguez Valin et al, 2001]

Although once widespread in New Zealand, a decline in brucellosis prevalence from approximately 4/100,000 of population in the early 1950s to about 1/100,000 by the late 1960s was the result of the introduction of pasteurisation of milk. At that time almost

50% of cases were in farm workers or their families, reflecting the main routes of transmission which were the handling of infectious tissue and the consumption of unpasteurised milk, while fewer than 5% of cases were freezing workers [Glass, 1963]. A Brucella eradication programme was introduced in 1969, based on intensive herd testing, vaccination of herds and the selective culling of reactor cattle. During the period in which reactor cattle were culled and slaughtered, transmission of the *B. abortus* infection from cattle to workers in the meat industry was common, and disease rates immediately increased again to approximately 4/100,000 in the early 1970s, primarily among males residing in the regions in which freezing works were situated due to the inevitable increased exposure of those who slaughtered reactor cattle [Glass, 2002].

By 1989, bovine brucellosis had been eradicated in New Zealand [MacDiarmid, 1994; O'Neil, 1995], although there was a lag in the decline in prevalence rates due to a pool of chronic cases that continued to present. Although a few locally acquired brucellosis cases have been notified since the eradication of the animal reservoir, they are believed to have been misdiagnoses made either despite inadequate laboratory findings or based on false positive reactions with serological tests due to cross-reactivity between *Brucella abortus* and *Yersinia enterocolitica* 0:9 [Matheson, 1993].

Leptospirosis is another zoonosis that is predominantly occupational in origin, and is considered New Zealand's most common occupationally acquired infectious disease [Thornley et al, 2002]. It is an acute febrile illness caused by leptospires, which are members of the order *Spirochaetales*. The reservoir of infection includes cattle and pigs,

and a number of wild animals, and humans are infected through direct or indirect contact of the skin (especially if abraded) with urine from infected hosts. In the abattoir environment there is involuntary release of urine by animals after stunning and slaughter, and spillage of urine over the carcass may occur. Meat workers who handle offal, and especially the bladder and kidneys, also risk contact with urine or viable spirochaetes. A serological survey of 1000 meat inspectors, for example, found a prevalence of 10.2% with minimum titres of 1:24, a level considered indicative of previous leptospiral infection [Blackmore et al, 1979].

Subsequent surveys of both meat inspectors and meat workers found a similar prevalence of seropositive individuals in each group, with the highest prevalence in meat workers found to be amongst those working on the slaughter floor [Blackmore & Schollum, 1982]. The average annual incidence of leptospirosis in New Zealand remains high relative to other temperate developed countries, at least 4.5 times that reported in Portugal, Australia and Ireland in the 1990s. The highest rates of notified cases (confirmed by laboratory results showing a single titre ≥ 400 on the microscopic agglutination test, a greater than fourfold rise in titres between two sequential specimens, or isolation of leptospires from clinical specimens) occur in the occupational category of meat processing workers, with a crude incidence rate of 163.5/100,000 [Thornley et al, 2002].

Infection with *Helicobacter pylori,* a spiral, flagellated, gram-negative bacterium that colonises the gastrointestinal tract, has been associated with gastric cancer in humans

[IARC, 1994]. The only recognised routes of transmission of *H. pylori* are person-to-person, via oral-oral and oral-faecal routes, and there is little evidence for the existence of non-human reservoirs [Fox, 1995]. Notwithstanding this, New Zealand meat workers and meat works veterinarians have been found to have significantly elevated titres when compared with the general population [Morris et al, 1986], although it is possible that this may be due to antigenic cross-reactivity in workers' sera due to their constant exposure to other large spiral gastric Helicobacter-like organisms common in the gastrointestinal flora of animals [Fox, 1995]. A non-significant increase in stomach cancer (OR 1.26, 95% CI 0.9 – 1.8) has been observed in New Zealand meatworkers [Reif et al, 1989], and an increase of borderline significance (RR 1.33, 95% CI 1.0-1.80) has been observed in butchers and meat preparers in Sweden [Aragones et al, 2002].

Viral zoonoses

In addition to these bacterial zoonoses, there are a number of viral infectious agents known to infect freezing workers. Butchers and abattoir workers are known to have a higher prevalence of warts than non-meat handlers [Finkel & Finkel, 1984], with the highest prevalence in workers who slaughter cattle and pigs [Jennings et al, 1984]. These warts are thought to be due to the human papilloma virus, and in particular the subtype HPV-7 that is found almost exclusively in meat handlers and which appears to be transmitted directly from raw meat rather than through person to person contact [Keefe et al, 1994].

At the time of the initial reports of an association between work in the meat industry and

lung cancer it was suggested that a possible cause may have been the viral warts common in butchers [Pegum, 1982], although in one subsequent investigation no DNA from HPV-7 (or other HPV types associated with laryngeal and genital cancers) was found in histological material from the lung tumours of 40 butchers and 26 controls [Al-Ghamdi et al, 1995]. It was concluded from this that there was no evidence that HPV infection was a significant cause of lung cancers in butchers.

Parapoxvirus orf (PPVO) is a zoonotic DNA virus that causes a highly contagious pustular dermatitis primarily localised in the mouth and nostrils of sheep and goats, known as contagious ecthyma or "scabby mouth". The fact that this virus can repeatedly reinfect sheep, in spite of a vigorous inflammatory and host immune response to the infection, has provoked an interest in the underlying mechanisms by which it escapes from the ovine host's protective immune response. PPVO is thought to inhibit the host's immune responses by inducing apoptosis in a significant number of antigen-presenting cells at the site of exposure to the virus, thus preventing a primary T-cell response. The reduced antigen presentation does not result in a general immunosuppression, and there is a compensatory non-specific systemic activation of the immune system following the localised suppression of the immune response [Kruse & Weber, 2001].

When Parapoxvirus infections are transmitted to humans they also induce proliferative disorders, usually benign epitheliomas or occasionally histiocytomas [zur Hausen, 2001]. The virus is very resistant to physical factors, except ultraviolet light, and may persist in the environment or on animal coats. The predominant mode of transmission to humans from live animals is direct contact with the mucous membranes of infected animals, or

with the lesions on the animal. Infection of workers in the New Zealand meat industry with orf virus is well documented, although the prevalence is low, with 4% of those working on the mutton chain affected in one survey [Robinson & Petersen, 1983]. In the abattoir environment the highest risk of infection was found to be associated with the handling of pelts and/or wool, and 95% of the resulting lesions were on the hands. The observed risk of contracting orf did not decrease with the number of years employed at the meatworks, and cases of reinfection were reported. Other members of the genus Parapoxvirus include bovine papular stomatitis virus (BPSV) and pseudocowpoxvirus (PCPV), which are both maintained in cattle and infect humans, and parapoxvirus of Red Deer in New Zealand (PVNZ), which is not known to infect humans [Mercer et al, 1997].

Novel zoonoses

Outbreaks of encephalitis among Malaysian pig farmers and Singaporean abattoir workers in the late 1990s were found to be caused by the previously unknown Nipah virus [Parashar et al, 2000]. Investigations in Singapore following the outbreak among abattoir workers showed that the main risk factor for infection was contact with pigs that were infected and shedding the virus, and in particular direct contact with pig urine and faeces. Other individuals such as meat inspectors and public butchers who had no contact with live animals, or therefore with infectious body excretions, appeared to be at reduced risk of infection. In addition, although serological findings were similar in both abattoirs tested, there was a significantly lower proportion of overt disease among workers in one abattoir in which plastic face shields were worn. Their use was thought to have reduced the viral load to which workers were exposed, thereby allowing the virus to be more

efficiently handled by the immune system [Chan et al, 2002]. This finding is suggestive of airborne transmission of the infectious agent, and of inhalation or contact with mucous membranes as the main portals of entry into the body.

Another example of a novel zoonotic disease was the escalation of the numbers of new variant Creutzfeldt-Jakob disease (vCJD) cases in relatively young people that occurred in the late 1990s in the United Kingdom, which is thought to have resulted from exposure to beef infected with bovine spongiform encephalopathy (BSE). This represents the only known example of the transmission of one of several known fatal transmissible degenerative encephalopathies (TDEs) from animals to humans [Taylor, 2000]. In these diseases the transmissible agent is believed to be a modified infectious form of the host animal's normal protein that resists catabolic destruction by proteolytic enzymes, and is also able to survive the temperatures reached in the rendering process used to manufacture meat and bone meal. The transmission of this agent from animal-to-animal, and also animal-to-human, is believed to be primarily through a dietary route. An evaluation of national mortality records for the period 1979 – 1996 in England and Wales found no evidence of occupational transmission to groups with potentially high exposure such as people working in animal husbandry and slaughter [Aylin et al, 1999], although it was noted that the follow-up period in this investigation may have been too short for disease to manifest following the BSE outbreak if the incubation period for this disease is longer than 15 years [Alperovich, 1999].

New Zealand has been confirmed as free from the transmissible spongiform encephalopathies of animals, including BSE, scrapie of sheep and goats, and chronic wasting disease of deer (CWD), through a testing programme which includes ante-mortem inspection for clinical signs in live cattle at freezing works, and prionics testing of all cattle that are dead on arrival or that die in cattle yards at freezing works, plus a proportion of cattle presented for rendering or pet food manufacture [Sabirovic, 2002].

3.3.1 (ii) Oncogenic Retroviruses

Of special interest in this context are a number of retroviruses from the genera *Alpharetrovirus*, *Betaretrovirus Gammaretrovirus* and *Deltaretrovirus* that have been identified as the cause of malignant diseases in animals. There are also members of the *Lentivirus* genus that are known to cause chronic immunodeficiency diseases, and immunosuppression is believed to be associated with non-Hodgkin's lymphoma [Burmeister, 2001]. Some of these viruses are present in healthy animals that are processed in the meat industry.

The oncogenic animal retroviruses include bovine leukaemia virus (BLV), which infects both sheep and cattle and is closely associated with the development of leukaemia and lymphoma in both species. BLV is genetically related to human T-cell lymphotropic virus type 1 (HTLV-1), which causes adult T-cell leukaemia/lymphoma (ATLL) in humans, and it induces lymphomas in approximately 5% of infected cattle and in all experimentally infected sheep [IARC, 1996]. Other oncogenic animal retroviruses

include sheep enzootic nasal tumour virus (ENTV), which causes tumours of the upper respiratory tract in sheep, and Jaagsiekte sheep retrovirus (JSRV) that causes ovine pulmonary adenomatosis, a transmissible lung cancer in infected sheep that resembles human bronchioalveolar carcinoma. Several avian leukosis viruses (ALVs) are also known to cause a wide variety of neoplasms in various avian species [Burmeister, 2001]. Members of the *Lentivirus* genus include bovine immunodeficiency virus (BIV), which as its name suggests causes chronic immunodeficiencies in cattle, and Maedi-Visna virus (MVV), which causes chronic interstitial pneumonia and demyelinating leukencephalomyelitis in sheep.

As is the case in most countries, both of the known bovine retroviruses have been found in New Zealand stock. Serological evidence of infection with BLV has been reported in New Zealand cattle, although the prevalence of infection was low (0.05%) [Parrish et al, 1981]. An enzootic bovine leucosis eradication scheme based on the testing and culling of all positive animals to slaughter was implemented in 1997. To date a total of 8,579 BLV positive animals have been culled to slaughter, resulting in a reduction of infected herds from a peak of 928 (6.3%) to 159 (1.2%) by May 2002 [Hayes, 2002]. Sheep have been shown to be highly susceptible to experimental infection with the BLV virus, however there is no evidence of natural infection in New Zealand sheep and a survey of 677 ram serum samples taken in 2000 found no samples positive for anti-BLV antibodies [Reichel, 2000]. The *Lentivirus* BIV has also been detected in New Zealand cattle [Horner, 1991], but extensive serological testing since the early 1980s has confirmed the

absence of Maedi-visna virus from New Zealand sheep flocks [Thornton & Motha, 1995].

Several retroviruses infect lower primates, including Mason-Pfitzer monkey retrovirus (MPMV) and Simian retrovirus (SRV) which both cause immunosuppression but not malignant diseases in infected animals, Gibbon ape leukaemia virus (GaLV) which causes myeloproliferative disorders in Gibbons and Simian T-cell lymphotropic viruses (STLVs) which cause T-cell lymphoma in Old-World monkeys. In spite of the large number of known animal oncoretroviruses, only four genuine human retroviruses have been isolated, i.e. human immunodeficiency virus (HIV) types 1 and 2 and human T-cell lymphotropic virus (HTLV) types 1 and 2 [Burmeister et al, 2001]. Of these, HTLV-1 and HIV-1 are the only human retrovirus known to cause cancer, namely adult T-cell leukaemia/lymphoma (ATLL) [Johnson et al, 2001] and Kaposi's sarcoma [IARC, 1996].

The transmission of the exogenous oncogenic retroviruses may be either horizontal or vertical. The virus may exist as infectious particles shed by infected organisms, although in the general environment these viruses are very fragile and can survive outside the host organism for minutes to hours only [Dintzer & Morein, 1990]. Transmission between animals may be horizontal through close contact including inhalation of infectious particles in the case of Jaagsiekte sheep retrovirus (JSRV), or in the case of BLV by inadvertent iatrogenic transfer of infected blood [Burmeister, 2001]. Vertical transmission through ingestion of infected milk is also common. The predominant modes of transmission for the human retroviruses such as HIV and HTLV, i.e. contaminated blood

products (including needle sharing amongst intravenous drug users), infected mother's breast milk (as the virus may be present in white blood cells in the milk) and sexual intercourse, depend on the transfer of infected blood cells [Lowis et al, 2001]. Some of the animal retroviruses including JSRV have been shown experimentally to be capable of infecting human cells [Rai et al, 2000], and serological testing has shown cross species retroviral transmission with the infection of humans with simian retrovirus [Lerche et al, 2001] and simian foamy virus [Brooks et al, 2002; Sandstrom et al, 2000] occurring among people occupationally exposed to nonhuman primates.

There is, however, little epidemiological or experimental evidence of zoonotic viral causes of human malignancies. Moreover, in a recent study, cell samples taken from 44 patients with various malignant haematological diseases were tested for evidence of putative human oncoretroviruses using consensus polymerase chain reaction (PCR) primers developed from genome regions of known animal retroviruses [Burmeister et al, 2001]. These PCR primers were capable of specifically amplifying type C and D exogenous animal retroviruses, without amplifying human endogenous retroviral elements, but no human homologues of nucleotide sequences of known animal oncoretroviruses (or related previously undetected human retroviruses) were found.

3.3.2 Non-infectious bio-aerosols

In addition to the infectious agents and other viable organisms present in the slaughterhouse environment, it is likely that workers in those parts of the process where live animals are handled will also experience exposure to bio-aerosols including fungi and bacterial endotoxins emanating in particular from the animal hides, proteins from the animal skins, hair or wool, and proteins from animal urine and faecal material. Considerably elevated levels of these agents have been found in swine and poultry confinement and in wool mills [Simpson et al, 1999] and in dairy cattle barns [Kullman et al, 1998], although the levels could be expected to be significantly lower in the less enclosed environment of the freezing works stockyards.

The most frequently recorded effects of these exposures in a wide range of occupations have been both allergic and non-allergic respiratory symptoms including organic dust toxic syndrome (ODTS), chronic obstructive pulmonary disease (COPD) and asthma [Douwes et al, 2003]. It has also been suggested that chronic exposure to animal proteins may set up a low-grade chronic inflammation that results in either chronic antigenic stimulation and/or macrophage activation, both of which adversely affect myelopoiesis [Pearce et al, 1986b; Papadaki et al, 2001]. It has also been hypothesised, however, that the relatively low incidence of lung cancer in farmers [Mastrangelo et al, 1997] and cotton textile workers [Levin et al, 1987] may be due to a protective effect of exposure to bacterial endotoxin through a host factor, the Tumour Necrosis Factor, produced by

alveolar macrophages. No clear evidence of an association between chronic antigenic stimulation and cancer has emerged.

3.3.3 Chemical Exposures

Although the main potential exposures in the meat industry are biological in origin, much of the focus on exposures in earlier epidemiological studies of cancer in the meat industry was on a range of chemical exposures that were present in traditional butcher shops or small abattoirs and meat processors. These included products of incomplete combustion such as polycyclic aromatic hydrocarbons (PAHs) from smoking meat, nitrosamines formed from nitrite used for curing meat [Gustavsson et al, 1987], and fume known to contain substances such as benzene, phthalic anhydride and phthalates (that are known or suspected to be carcinogenic in other settings) which are generated by thermal decomposition when heat is applied to cut and seal the plastic film that is used to wrap meat [Johnson et al, 1986b].

Exposure measurements taken in traditional meat processing plants in Denmark and Sweden indicated levels of PAHs in very close proximity to the smokehouse that were similar to those found in other industries in which excess lung cancer risk has been observed [Gustavsson et al, 1987]. Smoking of meat, however, is not a process carried out in the majority of large meat processing plants so this exposure would be experienced by relatively few meat workers. The consumption of nitrite cured meat has also been proposed as a potential source of exposure to carcinogens due to the formation of nitrosamines, but it is unlikely that the use of nitrite in curing vats would result in

significant exposure by inhalation and only a small proportion of meat workers are employed in meat preservation [Coggon et al, 1989].

None of the above exposures are features of the New Zealand meat processing industry, although as in any industrial process there is a range of potential chemical exposures associated with production in this industry. These include exposures experienced by those relatively small groups of employees engaged in maintenance and repair, or involved in specific processes such as the preparation of pelts and hides in the fellmongery or salting of casings. There is potentially more general workforce exposure to the various chemicals used in cleaning, and to residues of animal therapeutic agents or pesticides carried on or by the animals.

Maintenance workers in this industry experience the exposures common to their specific trades, with a particular emphasis in this industry on the emissions from welding of the stainless steel used for hygienic surfaces and food vessels and on fugitive emissions or leaks of the ammonia used as a refrigerant gas. An acute hazard for maintenance workers in this industry is entry into confined spaces in which there are at times elevated levels of methane (and consequent displacement of oxygen) produced by the decomposition of organic material. As in all industrial processes, the maintenance workers can experience intimate contact with waste products of the process, in this case with biological material with the attendant heightened risk of exposure to infectious agents, or intense exposure to the products of thermal decomposition of process chemicals such as when welding through residues of the chlorophenols used in the past as preservatives for pickled pelts.

The fellmongering process involves significant potential for chemical exposures of the employees in these areas, to both acute reactions to contact with acid or alkaline solutions and chronic effects of the gases, vapours and mists released into the atmosphere. The depilatory agent applied to ovine pelts is an aqueous solution of calcium hydroxide and sodium sulphide that is stable under the alkaline conditions maintained during the painting and liming stages of the process. With the addition of salt and sulphuric acid to reduce the pH for the deliming and pickling stages, however, hydrogen sulphide gas may be liberated into the workroom air [unpublished report, Mason, Leather And Shoe Research Association]. The addition of ammonium chloride or ammonium sulphate to the liming solution for the deliming process may also result in the liberation of ammonia gas. Chlorophenols have been used in the past as anti-fungal treatments in the preservation of pickled pelts and hides, and exposure would have occurred through both skin contact and inhalation. They have been replaced by substitutes containing alternative fungicides such as sodium mercaptobenzothiazole (MTB), 2-(thiocyanomethylthio) benzothiazole (TCMTB), boric acid and carbendazim.

There is extensive use of cleaning agents in freezing works due to particularly stringent food hygiene requirements for export abattoirs. Individual workers wash their hands and boots each time they enter processing areas, and slaughter floor personnel wash their hands between each carcass processed in a hot water and detergent solution, all of which may damage the skin and afford microbial agents easy access into the body through the skin. There are, in addition, cleaning crews assigned to thoroughly clean each department

daily on each shift, with the effectiveness of the cleaning programme being audited regularly with a series of hygiene swabs taken from a variety of surfaces weekly to test aerobic plate count and for *E coli*.

The detergents used for cleaning plant and equipment and sanitising food contact surfaces may be both alkaline or acid, and solutions of various strengths of sodium hypochlorite. The exposure to these products would inevitably be most intense for the cleaning crews, but the hygiene requirements affect the entire workforce who must regularly use these products either on themselves, their tools or their immediate work area. There is some evidence of an association between exposure to chlorination by-products in drinking water, such as the trihalomethanes and haloacetates that are produced from chemical interactions between chlorine and organic chemicals in water, and certain types of cancer. The evidence is strongest for bladder cancer, and elevations in risk have also been observed for colon and rectal cancer although the results are not consistent [Cantor, 1997]. Inhalation and dermal exposures, particularly when water is heated, have been implicated as significant routes of exposure associated with this risk and there is the potential for similar exposures to occur during cleaning in meat processing plants.

A number of natural and synthetic hormones have been licensed for use in New Zealand for animal growth promotion in cattle over the period covered in this study. These include oestradiol-17 beta, trenbolone, oestradiol benzoate, progesterone, testosterone and zeranol. A product containing zeranol has also been registered for use as a growth promoter in lambs, but has not been marketed or sold [personal communication, Neil

Kennington, Agricultural Compounds and Veterinary Medicines Group, Ministry of Agriculture and Forestry Food Section, 2002]. These products are administered orally or through implants, with license conditions specifying that a veterinarian or a technician supervised by a veterinarian must administer them, and minimum withholding periods prior to slaughter are specified. No specific maximum residue levels (MRLs) have been set for the hormonal growth promoters progesterone, testosterone, oestradiol or the synthetic analogue trenbolone [Ministry of Agriculture and Forestry Food Section, 2002]. Residue monitoring in a sample of 300 cattle detected no growth promoters apart from 46 samples which tested positive for zeranol, thought to be the result of the metabolism of zearalenone, which is a mycotoxin produced by a fungus (Fusarium) present in herbage [MAF, 2001].

Notwithstanding these requirements, there is the potential for exposure of freezing workers to these hormones or their metabolites through cutaneous or respiratory contact during slaughter and processing of treated beef. Residues of hormone growth promoters and other animal remedies have been found in a variety of animal tissues tested, including loin, liver, kidney, fat, and muscle [Lange et al, 2001; Daeseleire et al, 1992]. They are also present in bovine urine [Daeseleire et al, 1992; Haughey et al, 2001], to which workers on the slaughterboard are routinely exposed.

Exposure to additional levels of natural or synthetic hormones may disturb the endocrine system and increase the risk of hormone-dependent cancers such as breast or prostate cancer [Zacharewski, 1998]. It has been hypothesised that both high standardised

mortality ratios from prostate cancer and high ratios of male births to butchers were related to the use of androgens as growth promoters in meat production, and that relatively low ratios of male births to butchers coincided with a period in which oetrogens were the most commonly used hormone growth promoters [Lloyd et al, 1987]. In addition two of the hormone growth promoters, oestradiol-17 beta and zeranol, have been shown to suppress expression of a tumour suppression gene (protein tyrosine phosphatase gamma or PTP gamma) in human breast tissue in vitro [Liu et al, 2002].

Organochlorine pesticides, including DDT applied to pasture to control grass grub, lindane used as an insecticide for the control of lice in cattle, ectoparasites in sheep and grass grub in pasture, and aldrin and dieldrin were both used in sheep sprays or dips to control ectoparasites, have been used widely in the past in New Zealand agriculture [Buckland et al, 2001]. Their use was phased out by the mid 1970s, but residue monitoring programmes still routinely detect DDT and its metabolites and dieldrin in animals tested [MAF Biosecurity, 2000]. The direct exposure of meat industry workers that would have occurred through the handling of treated stock would have ceased following the withdrawal of these products, however the residue monitoring indicates that the potential for exposure through contact with meat products remains.

3.3.4 Physical and psychosocial exposures

Ergonomic hazards related to the fast paced, repetitive and high-force manual work involved in meat processing, and the resultant cumulative trauma disorders, have been

well documented. In the late 1980's for example the incidence of disorders due to repeated trauma in the US meatpacking industry was approximately seventy five times that of US industry as a whole [Sheridan, 1991]. In a Danish study which compared the prevalence of carpal tunnel syndrome (CTS) in a low risk reference group working in a chemical factory with that for slaughterhouse workers, prevalence rates up to five times those in the reference group were found for certain slaughterhouse workers. While 1.6% of the reference group had either previously been operated on for CTS or exhibited current symptoms typical of the disorder in combination with positive neurophysiological signs, 5.1% (PR 3.23, 95% CI 1.3-7.99) of the workers engaged in slaughter but not deboning operations and 7.8% (PR 4.91, 95% CI 2.03 – 11.81) of those engaged in deboning operations had CTS [Frost et al, 1998]. A similar study found the prevalence of carpal tunnel syndrome among workers in a modern Canadian meat packing plant to be 21% [Gorsche et al, 1999].

Cutting or piercing injuries to the hand and arm are also common in the meat processing industry, and one New Zealand study of national hospital discharge data for the period 1979-1988 found a significant increase over that period from a hospitalisation rate of 3.3 per 1000 to 5.3 per 1000 meat workers [Laing et al, 1997]. No reason for this increase was evident, and it did occur even during a period in which the use of protective clothing had increased. Other physical exposures in the industry, which include ultraviolet light used in certain areas to inhibit microbial growth, noise and vibration, and the electromagnetic radiation associated with electrical stunning and power sources for the

production line conveyors, are unremarkable when compared with most industrial operations. None of these physical hazards have been associated with cancer.

The possible role of work-related psychosocial stress in the elevation of lung cancer risk among butchers and slaughterhouse workers has been suggested as warranting further study [Kristensen & Lynge, 1993]. The excess lung cancer risk in butchers was observed in England and Wales at the 1951 census, which indicated that aetiological factors must have been present in this environment for a long time [Griffith, 1982]. The slaughtering, processing and meat packing industry has long been associated with a high incidence of accidents, injuries and illnesses, and recognised as a stressful working environment [Sinclair, 1906].

In more recent times the industry has been transformed and restructured to increase production rates and organise tasks within the meat processing industry according to the principles of 'scientific management'. Workers perform repetitive specialised tasks on the machine paced chain, with average chain speeds in New Zealand having increased between 1980 and the present from approximately seven and a half to eight and a half or nine carcasses per minute. Over this same period the average working day has increased from eight to ten hours, while staffing levels on the chain have reduced [personal communication, Stephens, 2002]. There has been only limited implementation of job redesign, such as job rotation, to ameliorate the effects of the repetitive and fast paced work. In addition to the stress imposed by these elements of the work organisation in this industry, other stressors include the seasonal nature of employment, frequent industry

restructuring and plant closures, limited job security apart from that afforded by the collective strength of the unionised workforce, and an historically adversarial industrial relations environment. There are no further reports of investigations of the association between stress and cancer in this industry, and no clear evidence of any strong association between the two has subsequently emerged.

3.4 Summary

Published literature on exposures of meat workers is sparse. What is available would suggest that the primary exposures are likely to be to biological agents, including infectious bacteria and viruses, and non-infectious bio-aerosols. The infectious agents known to exist in this environment include recognised causes of cancer in both animals and humans, such as the bovine retroviruses bovine leukosis virus (BLV) and bovine immunodeficiency virus (BIV), human papilloma virus (HPV) subtypes and *Helicobacter pylori*. Direct exposures to chemical agents associated with specific processes such as the treatment of pelts and hides or plant maintenance, and inadvertent exposures to residues of pesticides and animal remedies, are also possible but are likely to be less widespread in this environment.

The transmission of zoonotic infections, such as leptospirosis and brucellosis, has frequently been documented in New Zealand meat workers. Known routes of transmission of these infections include direct contact between source and host, particularly via contact with mucous membranes or damaged skin, or airborne

transmission through inhalation of bio-aerosols. Inhalation is also the primary exposure route for the non-infectious bio-aerosols, for which effects such as both allergic and non-allergic respiratory symptoms including organic dust toxic syndrome (ODTS), chronic obstructive pulmonary disease (COPD) and asthma have been recorded. There has also been speculation that chronic exposure to these bio-aerosols may set up a low-grade chronic inflammation that may result in chronic antigenic stimulation.

Without explicit data on worker exposure in this industry it is not possible to develop precise exposure profiles for the purposes of defining internal comparison groups. It is possible, however, to develop broad exposure categories to rank job titles or work areas according to their potential for exposure to a range of biological and chemical agents.

Chapter 4 - Study Design

4.1 Overview

This thesis describes two historical cohort studies undertaken to examine mortality and cancer incidence among a group of workers employed in the New Zealand meat processing industry. One cohort was based on members of the New Zealand Meat Workers and Related Trades Union Incorporated (the "Union Cohort") whereas the other was based on personnel records for three freezing works from two separate companies (the "Company Cohort"). Information on the potential exposure to various chemical or biological agents was used to develop a job exposure matrix to enable individual study subjects to be assigned to exposure categories. Study subjects were traced forward from 01/01/1988 until date of death, emigration or the end of the study period (31/12/2000), and their mortality and cancer incidence established by computerised searching of national records. Death and/or cancer registration rates were compared with New Zealand national rates, and subgroup analyses were made for various subgroups of workers defined according to the assigned exposure categories.

4.2 Choice of study design

A combination of study designs has been used previously to investigate cancer risks among meat workers, i.e. studies based on routinely collected mortality and incidence data, proportionate mortality and incidence studies, and case-control and cohort studies. While increased risks of all cancers, and in particular of cancers of the lung, larynx and

lymphohaematopoietic system have been suggested by these studies, the available evidence is inadequate to conclude either that the risk for any specific cancer is real or to implicate any specific exposure. Of the study designs applied the analyses of routinely collected data [Lynge, 1982; Fox, 1982; Griffith, 1982; Lagorio et al, 1995; Morton & Marjanovich, 1984] were useful in generating hypotheses, but were of limited value in assessing whether there was a true exposure-outcome relationship at an individual level. The measures of proportionate mortality and incidence [Fox, 1982; Milham, 1982; Johnson & Fischman, 1982, Johnson et al, 1986a; Johnson et al, 1986b; Johnson et al, 1987] also need to be interpreted with caution due to the potential biases inherent in such analyses [Checkoway et al, 1989].

For the study of cancer and other diseases of long induction, population-based case-control studies are usually very efficient, particularly for investigating rare diseases [Breslow & Day, 1980]. They allow a wide range of exposures that might be related to the disease to be evaluated simultaneously and are, therefore, particularly useful for screening hypotheses regarding occupational exposures that may warrant more intensive inquiry in subsequent industry based studies [Checkoway et al, 1989]. Case-control studies have the added benefit of providing the opportunity to obtain detailed information both on the exposure(s) of interest and on potential confounders, but they may be more susceptible to bias and in particular selection bias (e.g. selection of an appropriate control group) and information bias (e.g. accurate measures of past exposures). Furthermore, they are not suitable for investigating exposures that are rare in the source population unless the exposure is responsible for a large proportion of cases. Where the putative exposure is

rare in the general population and is responsible for only a small proportion of the cases of any specific cancer, and where it is possible to identify a specific group with that exposure, the historical cohort study is the most efficient study design for evaluating cancer risk. As multiple health outcomes can be examined, this study design will also provide the clearest picture of the overall health experience of that group [Checkoway et al, 1989].

A benefit of the historical cohort study design is that, provided that an appropriate cohort can be identified and that historical exposure information exists, recall bias is eliminated and selection bias is usually minimised. This is because exposure status is ascertained, and because exposed and unexposed individuals are enrolled into the study population, before the outcome of interest has developed. Confounding may still occur due to the so-called "healthy worker effect", which manifests as lower overall morbidity and mortality in the working population being studied when comparisons are made between an occupational cohort and the general population. This occurs because only relatively healthy people are able to gain employment, and to remain in employment, whereas the general population includes a wider range of people including those too ill to work [Checkoway et al, 1989].

There is, in addition, the potential for information bias (other than recall bias) in historical cohort studies where classification of exposure or outcome is invalid [dos Santos Silva, 1999]. However, this is likely to involve non-differential misclassification because exposure status is ascertained before the outcome of interest has developed, and

subjects therefore have the same chance of their exposure status being misclassified regardless of their outcome status. This is relatively common in historical cohort studies of occupational groups because, as records of actual measurements of individual exposure rarely exist, individuals are often categorised as exposed or non-exposed on the basis of surrogate measures such as job title or work area. The use of surrogate measures of exposure such as these, which attribute the same exposure estimate to each individual within the same job title, will introduce (non-differential) misclassification as even workers performing the same job experience significant variability in average exposure levels [Boleij et al, 1995]. Where non-differential misclassification of the exposure status of study subjects exists, the risk estimates will be biased toward the null, thereby underestimating the strength of association between that exposure and outcome [Copeland et al, 1977]. Differential misclassification of exposure on the other hand, which occurs where the classification of exposure status is dependent on the outcome status of individual study subjects and which can bias the risk estimates in either direction, is less likely in historical cohort studies of occupational cohorts where exposure data is collected on the study population before the outcome is known.

Another limitation of the historical cohort study design is the fact that there is potential for uncontrolled confounding where information on factors such as tobacco smoking is lacking. The potential confounding effect of smoking is often overestimated, however, as differences in smoking rates between groups of manual workers are usually small. Even for lung cancer the differences in smoking status are unlikely to account for a relative risk of greater than 1.5 in studies involving a comparison with national mortality rates

[Axelson, 1978], and the confounding effect of smoking is even weaker for internal dose-response analyses [Siemiatycki et al, 1988].

The cohort studies conducted previously to examine cancer risks among meat workers in the USA [Johnson et al, 1986a, 1986b, 1995], the UK [Coggon et al, 1989; Coggon & Wield, 1995] and in Switzerland [Guberan et al, 1993] have been limited primarily by their study size, or in the case of the one large Swedish cohort [Boffetta et al, 2000] by the relatively crude exposure data based on occupation listed at census. Possibly because of these limitations, these cohort studies have produced results that contradict those of the case-control studies, showing only a small increase in lung cancer risk (albeit within the range that could be attributable to differences in smoking rates) and little evidence of any increase in cancers of the lymphohaematopoietic system.

As no cohort study of New Zealand meat workers had been done previously, it was considered appropriate to conduct studies here primarily to establish whether the elevated risks for cancers of the lymphohaematopoietic system found in earlier case-control studies could be replicated with this study design, and also to examine the associations between specific exposures and any increased cancer risk. Although the processing of meat and meat products is a significant industry in this country's predominantly agricultural economy, and is a significant employer with 20,000 people directly employed in meat processing and packing plants, the exposure of interest is still very rare with less than 1% of the adult population working in this industry. While the earlier New Zealand case-control studies have shown consistently elevated risks for cancers of the

lymphohaematopoietic system [Pearce et al, 1985; Pearce et al, 1986; Pearce et al, 1987; Reif et al, 1989] and a clear dose-response [Bethwaite et al, 2001], these have been based on only small numbers of exposed cases and relied on participants' recall of job titles and exposure.

Historical cohort studies provide the opportunity, therefore, to evaluate the disease experience of a large cohort with known exposure. It also permits the classification of individual study subjects according to exposure based on a work history compiled from historical records, with less potential for information bias than exists with the case-control study design.

There are also a number of reasons why New Zealand is a good place to do this type of study. Compared with many other countries, the meat processing industry in New Zealand has a relatively stable workforce with annual turnover of only 10 to 15%, as well as a relatively stable population for which acceptable rates of follow-up can be achieved. A National Cancer Registry with compulsory registration, first established in 1948, also provides reliable cancer incidence data for an aetiological study of this type [New Zealand Health Information Service, 2002] and death registration data are also considered to be valid and virtually complete [Brown & Frankovitch, 1998]. Two historical cohort studies were, therefore, conducted to examine mortality and cancer incidence, and to investigate associations between disease and a range of exposures, among workers employed in the New Zealand meat processing industry.

4.3 Consultation and ethical approval process

Although historical cohort studies focus on the comparison of disease rates in the study population with those in the general population, or between subgroups of the study population, and not on individual study subjects, there are a number of reasons why these studies are still subject to the standard ethical requirements for health research [Health Research Council, 1993].

In a purely practical sense it is necessary to obtain names and unique identifiers such as dates of birth of individual study subjects, and then to follow individuals through searches of death records and the Cancer Registry in order to establish the death and cancer incidence rates within the study cohorts. Where any personal information was originally collected for another purpose, and where it was obtained from sources other than the individual concerned, as it was in the case of this research, its use is also subject to the provisions of the Privacy Act 1993. In addition, as the study involves the use of personal health information, there is a requirement to comply with the principles of the Health Information Privacy Code 1994 which was issued under the Privacy Act 1993. This Act makes specific allowance, in Part II Information Privacy Principles, for personal health information to be used for the purposes of health research through an exemption from the general requirements that applies where;

> (f) Compliance is not reasonably practicable in the circumstances of the particular case; or

(g) That the information

(i) will not be used in a form in which the individual concerned is identified; or

(ii) will be used for statistical or research purposes and will not be published in a form that could reasonably be expected to identify the individual concerned;

As funding for this research was sought from the Health Research Council, it was also a requirement under Sections 25 and 31 of the Health Research Council Act 1990 that it must be subjected to independent ethical assessment. In addition, as this was a nationwide (or multicentre) study the special provisions relating to obtaining approval from each of the regional ethics committees were also applied.

It is not possible in an historical cohort study to gain written informed consent from individual study subjects for release of their personal health information, as no actual contact is made with individual study subjects. As the records used to identify the study population are up to thirty years old, a significant number of the study subjects will have died or emigrated or their whereabouts will be unknown. In lieu of individual consent, we sought permission for the release of personal information from both the meat industry employers as holders of that information and the trade unions that have a specific legal authority under current employment legislation to represent the interests of their members. A standard application for ethical approval for this study was made to the

Wellington Ethics Committee, and approval for the project "Work related risk of cancer in meat workers (Approval 99/94)" was granted on 28 September 1999. In order to meet obligations under the ethical approval that was granted, once the follow-up of death records and the cancer registry using names and dates of birth was completed, the list of names of study subjects was stored separately in locked filing cabinets, and ID numbers only were included in the files for subsequent analysis. It was also agreed that nothing would be published in a form that could reasonably be expected to identify any individual study subject.

The purpose, methods and aims of the study, and the issues related to privacy and ethical approval, were discussed with a number of organisations during consultation in the preparatory stages of the study. As well as informing those parties with a potential interest in the research, this consultation provided the opportunity to seek either simple endorsement of the study or more active support such as agreement to provide access to personnel or membership records or to information on exposure.

The four large meat companies, i.e. Richmond Ltd, the AFFCO Group, the Alliance Group and PPCS (the Primary Producers Cooperative Society), as well as the Meat Industry Association of New Zealand, were all contacted initially by letter, with subsequent telephone contact where no response was received. Positive responses, and agreement to provide support for the study, were received from Richmond Ltd and the AFFCO Group. The Alliance Group and PPCS both advised, however, that they would

provide no support of any kind, while the Meat Industry Association queried the value of the research and the potential adverse effect of any findings on trade in meat.

The two trade unions representing workers in the industry were also consulted. The Meat and Related Trades Union of Aotearoa Incorporated has coverage of most North Island meat processing plants, while the New Zealand Meat Workers and Related Trades Union Incorporated has coverage of most South Island plants. These unions represent most blue-collar workers in the meat processing industry, the exception being those workers in the skilled trades such as maintenance fitters and electricians who are represented by their own unions. Unions in the meat industry have maintained virtually 100% membership, even during the period of workplace ballots on union membership under the Labour Relations Act 1987 and essentially voluntary unionism under the Employment Contracts Act 1991. Both unions offered support for the project, and the historical membership records of the Canterbury Marlborough Westland branch of the Meat Workers Union subsequently contributed exposure information to one of the cohorts studied 9the other union did not have sufficient historical records). The Occupational Safety and Health Service of the New Zealand Department of Labour was also advised of the project.

4.4 Study Population

The study population in this historical cohort study consisted of two separate dynamic cohorts. One cohort, the "Union Cohort", was assembled from the membership records of the Canterbury Marlborough Westland branch of the New Zealand Meat Workers and

Related Trades Union covering the period 1969 to 1998. The "Company Cohort" was assembled from historical employment records obtained from three individual meat processing plants, covering the period 1981 to 1998. These were the Alliance Lorneville, and the Richmond Takapau and Richmond Oringi sub-cohorts.

4.4.1 The Union Cohort

The Union Cohort was based on the annual membership lists of the Canterbury Marlborough Nelson West Coast Branch of the New Zealand Meat Workers and Related Trades Union for the years 1969 to 1998. This union represented all workers, apart from those employed in management or in the trades associated with maintenance of the plant and equipment, from all meat processing plants located in the South Island north of the Waitaki River.

These union membership records had been compiled annually, as members signed on at the beginning of each season, from the butts of union membership tickets filled in by hand and distributed by the union delegate for each distinct work area or department. The butts of completed union ticket books were collected by the union branch office, and used to compile a typed and bound annual membership list complete for every year since 1916. Information contained in this record included surname, up to three initials of given names, meat processing plant, and work area or department. In large departments this was further broken down into subgroups according to tasks, for example among the

slaughterboard employees separate classifications would be given to butchers, slaughtermen and slaughterhouse labourers.

Thus the union membership records contained a complete work history for each member for the period that they were employed in the industry. Electronic files containing these annual membership records were converted to Microsoft ACCESS, and merged to produce a file containing a total of 157, 238 records, which represented 34,887 unique individuals. This file was sorted electronically by surname and first initial in order to consolidate the entries into unique records for individual workers, detailing each individual's work history, and a unique ID number was allocated to each. Further manual sorting and checking of this file was necessary to complete this consolidation, primarily where minor errors in spelling of names had prevented an electronic match. In these instances individuals were matched on criteria that included the similarity of names and/or initials, meat processing plant name, work department or type of job, and geographical location.

However, the union records did not include full names (only initial(s) and surname) or date of birth. These were obtained by linking the union file to records of membership of a national meat industry superannuation scheme, operated under the National Provident Fund, which had accepted new members from the time of its establishment in 1973 until 1992 when it was replaced by a competing Meat Industry Superannuation scheme. A file containing 17, 684 individual records of members of the meat processing industry scheme was provided by the scheme's administrators Jacques Martin Ltd, as at year-end 1991

which was the date at which membership would have been at its peak. This file included full names, dates of birth, gender, residential address and contributing employer.

The final file assembled, where a clear match of surname and at least first initial was made between the superannuation and union membership files, thus contained the unique personal identifiers necessary for follow-up. This file also contained a full work and exposure history (by job title or work area, and by species slaughtered) for a total of 4,064 study subjects (or 12% of the 34,887 people on the union file). The period of eligibility for inclusion in this sub-cohort was from 1974 to 1991, with work history records available for eligible study subjects from 1969 to 1998. Thus, this cohort consisted of individuals who were members of the Canterbury Marlborough Nelson West Coast branch of the New Zealand Meat Workers Union, and were also members of a meat industry superannuation scheme. It does not, therefore, represent all union members, but rather just those who joined the superannuation scheme (which as noted above represented only 12% of the union members). It would be likely to be more representative of individuals who regarded their employment in this industry as long term than of casual or short-term employees.

4.4.2 The Company Cohort

The Company Cohort was based on three sub-cohorts of workers from specific freezing works. The first sub-cohort was assembled from copies of annual printouts of employee masterfiles from a single freezing works located in Lorneville (Alliance Lorneville) over

the period 1986 to 1998, which the company had provided each year to the site union branch for the purposes of reconciling union membership and fee deduction records. The Lorneville plant was built in 1960, initially with six mutton chains and one beef chain, and now operates four mutton chains each on a double shift while the processing of beef ceased in 1998. This plant employs up to 1800 staff at the peak of the season. The records available were full personnel records, which contained full name and date of birth and a full work and exposure (by job title and/or work area and species) history. The paper records were photocopied then scanned and read into Microsoft ACCESS, with a manual check of the entire file undertaken subsequently to confirm the accuracy of the conversion to electronic form. Particular care was necessary to check the accuracy of the conversion of numerals contained in dates of birth and job title codes. Once converted to electronic form these files were merged then sorted electronically by surname and first initial to consolidate entries into individual records with a work history. Additional electronic sorting used the employee number that had been assigned to each worker by the company payroll section, and also the Inland Revenue Department (IRD) tax number, supplemented this consolidation into unique records. A unique ID number was then allocated to each of the 3430 study subjects contained in the Lorneville file.

The second and third sub-cohorts were assembled from similar annual employee master files provided by the management of two separate freezing works, owned by the same company (Richmond Ltd) and located at Oringi and Takapau in southern Hawkes Bay. Both works process sheep exclusively. Annual printouts of masterfiles of employees at the end of each financial year were available from both plants, for the period 1981 to

1996 at Takapau and 1987 to 1998 at Oringi. The records available from these plants included the IRD tax numbers for each employee, which subsequently proved useful in the follow-up conducted to ascertain vital status of individuals not recorded as having died. In addition to the sorting on surname and initial to consolidate the file into unique records, including work histories for individuals, both the IRD number and the unique pay number assigned to individuals at the time of first hire were used for sorting. The format of the printouts of personnel records meant that it was not feasible to simply scan records for conversion to electronic form. A Microsoft ACCESS file was set up for each plant, and data for every third or fourth year was entered manually. In order to compile the files for the intervening years, these records were then copied and amended manually by making the necessary deletions or additions to account for the roughly 10% annual labour turnover. On completion of the transcription of these records it was noted that for 1,179 of the Takapau sub-cohort records, and for 333 of the Oringi sub-cohort records, no date of birth had been recorded. The files of employees with missing dates of birth were manually checked against medical records by staff of the medical centres at both sites, and in this way an additional 1000 dates of birth were added to the Takapau file and another 138 to the Oringi file. The final sub-cohort files contained 1,196 individuals from Oringi and 2,057 individuals from Takapau.

4.4.3 Combination of the cohorts

In the initial analyses, the Union Cohort and the three company sub-cohorts were analysed separately. In subsequent analyses, the three company sub-cohorts were

combined into a single Company Cohort. When this was done a check was made for people who had worked in more than one plant, and their records were consolidated (to avoid counting workers and deaths more than once). This only involved 36 workers and two deaths. Similarly, in some analyses the Union and Company Cohorts were combined and their records were consolidated, but this only involved 24 workers and no deaths. The years of employment and of follow-up of the meat workers sub-cohorts are shown in Table 4.1 below.

Table 4.1 Years of employment and follow-up for the meat workers sub-cohorts

Sub-cohort	N	Years of employment	Years of follow-up
Union	4,064	1974 – 1991	1988 –2000
Lorneville	3,430	1986 – 1998	1988 –2000
Oringi	1,196	1987 – 1998	1988 –2000
Takapau	2,057	1981 – 1996	1988 –2000

All cohorts were followed from 01/01/1988. For the Union Cohort and for one of the company sub-cohorts (Takapau) follow-up could have included earlier years, but this was not done so that follow-up could be standardised across the various cohorts and also because of difficulties with electronic matching of deaths and cancer registrations in the earlier years. In particular, the National Minimum Dataset, which utilises National Health Index (NHI) numbers to link and store deaths, cancer registrations and hospital admissions in electronic form, is only complete from 1988. In any case, the numbers of deaths prior to 01/01/1988 were small (estimated as 20 in the Union Cohort, 10 in the Takapau sub-cohort, and zero in the Lorneville and Oringi sub-cohorts).

4.5 Exposure assessment

Information on the potential exposure of workers to various chemical or biological agents was used to develop a job-exposure matrix. Although published literature on exposures of meatworkers is sparse, a compilation of potential exposures was developed from a review of the limited published literature on exposures in the meat industry obtained through searching computerised databases of the medical and veterinary literature, and in particular MEDLINE (US National Library of Medicine) since 1966. To supplement the limited amount of information contained in the published literature, interviews with workers, unions, and management in the industry provided additional information on exposure. There was also extrapolation made about the likely prevalence and magnitude of exposure, and of the potential for transmission of infectious agents, from the more extensive food quality and veterinary literature on microbiological quality within the freezing works environment.

The ideal historical cohort study of occupational cancer would have historical exposure measurements for individual subjects for each putative risk factor, but this is rarely possible and in this instance it is not clear what the relevant exposures are. Published literature on the types of exposure experienced by meat workers is sparse, with no quantitative information reported on the prevalence and magnitude of exposures. As discussed in Chapter 3, the potential exposures in the New Zealand meat processing industry are likely to be predominantly biological in origin, including both bacterial and viral infectious agents and non-infectious bio-aerosols, although information on modes of

transmission and prevalence of exposure is available for only a limited number of the common zoonoses. The chemical exposures examined in early studies of lung cancer among butchers and other meat workers, such as PAHs, nitrates and thermal decomposition products generated from hot wire cutting of plastic film wrap, are not features of the New Zealand meat processing industry. As in any industrial process, however, there are a range of potential chemical exposures associated with maintenance and repair of plant and equipment, and with the chemicals used in cleaning or in specific processes such as the fellmongering of animal skins. There is also the potential for exposure to residues of animal therapeutic agents, growth hormones or pesticides carried on or by the animals being processed. The biological exposures of potential significance with respect to cancer and other forms of mortality in the meat industry can be subdivided into six distinct categories based on the opportunity that is provided for the transmission of a biological agent from the animal to meat workers. This is represented in tabular form below, detailing the source of exposure and known modes of transmission as well as giving known examples (see Table 4.2). A similar table lists the main potential chemical exposures in the New Zealand meat industry, with a description of the likely sources and exposure circumstances (see Table 4.3).

An industry specific job-exposure matrix (JEM) has been constructed, therefore, by cross-tabulating a list of job titles present in the industry with a list of the agents to which workers carrying out those jobs may potentially be exposed. For any given job title, the probability of exposure to the specific agent is depicted as either + or - (i.e. exposed or not exposed), or the likely intensity or frequency of exposure is depicted by ordinal

rankings (i.e. low, medium, high). Separate matrices are presented in tabular form for potential biological exposures (see Table 4.4) and potential chemical exposures (see Table 4.5).

Work in the meat industry is seasonal, with the length of the season varying from approximately nine months in the north to six or seven months in the southernmost regions. Climatic conditions and the availability of stock may influence the length of each season, and also the level of production at different stages of the season. A second chain (or shift) is added soon after the start of each season, and in busy seasons a third chain may be added to cope with the volume at the height of the season. The third chain may operate for relatively short periods, and individuals employed on this chain may work for no more than six to eight weeks in a year compared to the six to nine months of the majority of the workforce, although the numbers in this category are relatively small. Information on the period worked within each season was available only for the two smallest company sub-cohorts, so it was not possible to include a minimum period of work in the definition of "season" used to determine duration of exposure for study participants. Any individual who was recorded as having worked within a specific season, either by virtue of joining the union in the case of the Union cohort or by having a record of employment in the Company cohort, was therefore classified as having worked for a season.

Table 4.2. Biological exposure categories in the meat industry

Exposure variable	Source of exposure and routes of transmission	Documented examples
Live animal contact	Live animals may be shedding bacteria or viral particles, and occupational exposure provides the opportunity for skin contact with infectious tissue and for inhalation of both infectious agents and non-infectious bio-aerosols. There is also potential for the exposures associated with the animal pelt or hide outlined below.	Brucellosis Simian retrovirus Nipah virus Leptospirosis
Pelts or hides	Animal hides and skins harbour a range of non-infectious matter including fungi and bacterial endotoxins, proteins from the skin, hair, wool, and from urine or faeces, as well as infectious agents. Exposure occurs through inhalation or skin contact	Biogenic allergens Orf virus
Slaughter process – and handling freshly slaughtered meat	The process of slaughtering animals entails some potential for the exposures associated with contact with live animals and their pelts and hides, with additional potential for contact with diseased animal tissue and blood borne pathogens either through direct contact, percutaneous inoculation through cuts or puncture wounds, or inhalation.	Leptospirosis Brucellosis HPV BSE
Urine	Live animals in the stockyards urinate freely, and in the slaughterboard from stunning to evisceration involuntarily release of urine is common. This may contaminate animal pelts, hides and carcasses and infectious agents contained in urine may remain viable in these media. Transmission may occur directly through inhalation, ingestion or contact between splash droplets and mucous membranes, or through contact with damaged skin.	Leptospirosis Brucellosis
Gastrointestinal microflora and faeces	Traces of animal faeces will be present on live animals and on pelts and hides, and infectious agents may survive in this medium. During the stunning and slaughter process there is potential for involuntary defaecation, and slaughterboard personnel engaged in evisceration risk direct exposure, while processing of casings involves removal of faecal matter.	Helicobacter pylori Nipah virus
Blood	The highest potential for direct exposure to blood occurs at the sticking and slaughter processes at the start of the chain, although the potential remains until after evisceration. Transmission may be through either airborne dispersion, direct contact with damaged skin or mucous membranes, or percutaneous inoculation through cuts or puncture wounds.	Brucellosis Human and animal retroviruses

Table 4.3. Chemical exposure categories in the New Zealand meat industry

Exposure variable	Likely source and exposure circumstances
Welding emissions	Maintenance and repair of plant and equipment. The emission will include significant concentrations of nickel and chromium due to the stainless steel used throughout food processing plants.
Refrigerant gases	Ammonia is used as a refrigerant in the meat industry. Acute exposure to leaks will mainly affect maintenance workers, but in major events may affect the entire workforce. Workers in the chiller and freezer areas may experience longer term exposure to lower levels resulting from fugitive emissions.
Cleaning chemicals	Used extensively throughout the processing areas. Each employee washes prior to each entry to restricted areas, and washes tools and work surfaces frequently. Cleaning gangs exposed full time.
Hormone growth promoters	Almost certainly in beef only. Limited information about trends in use over time, so assume no change. Residues in most tissues, and released in urine, so workers in stockyards, on slaughter floor and in meat cutting potentially exposed.
Organochlorines in felmongering	Used as anti-fungal agent to preserve pickled pelts prior to transport to tanneries. Pelts soaked in baths containing aqueous solution of PCP. Significant opportunity for skin contact and dermal uptake. High potential for exposure to both PCP and products of thermal decomposition (e.g. dioxins and furans) when welding contaminated metal surfaces during maintenance and repair. Withdrawn from use in 1990.
Organochlorines as pesticide residues	DDT, dieldrin and lindane were widely used in sheep and cattle dips, and applied to pasture for grass grub control, during the 1950s and 1960s. Their use progressively declined through to the late 1970s. Exposure may occur through contact with residues on the wool or hair, or in animal tissue through the slaughtering and cutting process.

Table 4.4 Potential biological exposures in Job Areas and Titles within the Freezing Works

Area code	Area	Job code	Job title	Combined code	Live animal contact		Animal pelts or hides		Slaughter or raw meat		Urine		Faeces		Blood	
100	Stockyard	0	Unknown	100	+	2	+	2	-	0	+	2	+	2	-	0
100	Stockyard	1	Shepherd	101	+	2	+	2	-	0	+	2	+	2	-	0
100	Stockyard	2	Stockwash	102	+	2	+	1	-	0	+	2	+	2	-	0
200	Slaughterboard	0	Unknown	200	-	0	+	1	+	2	+	1	+	1	+	1
200	Slaughterboard	1	Stunner	201	+	1	+	1	+	1	+	1	+	1	-	0
200	Slaughterboard	2	Sticker	202	+	1	+	1	+	2	+	1	+	1	+	2
200	Slaughterboard	3	Butcher	203	-	0	-	0	+	2	+	1	+	1	+	2
200	Slaughterboard	4	Assistant	204	-	0	-	0	+	2	+	1	+	1	+	2
200	Slaughterboard	5	Gut and Bung	205	-	0	-	0	+	2	+	2	+	2	+	1
200	Slaughterboard	6	Trimmer	206	-	0	-	0	+	2	+	1	+	2	+	1
200	Slaughterboard	7	Grader	207	-	0	-	0	+	2	+	1	+	1	+	1
300	Cooling Floor	0	Unknown	300	-	0	-	0	+	1	+	1	-	0	+	1
300	Cooling Floor	1	Chillerhand	301	-	0	-	0	+	1	+	1	-	0	+	1
300	Cooling Floor	2	Bagging	302	-	0	-	0	+	1	+	1	-	0	+	1
400	Meat cutting	0	Unknown	400	-	0	-	0	+	1	+	1	-	0	+	1
400	Meat cutting	1	Boning/Cutting	401	-	0	-	0	+	1	+	1	-	0	+	1
400	Meat cutting	2	Wholesale Butchers	402	-	0	-	0	+	1	+	1	-	0	+	1
500	Freezers	0	Unknown	500	-	0	-	0	-	0	-	0	-	0	-	0
500	Freezers	1	Chamberhand	501	-	0	-	0	-	0	-	0	-	0	-	0
500	Freezers	2	Despatch	502	-	0	-	0	-	0	-	0	-	0	-	0
600	Processing	0	Unknown	600	-	0	-	0	-	0	-	0	+	1	-	0
600	Processing	1	Edible offal	601	-	0	-	0	-	0	+	1	+	1	-	0
600	Processing	2	Inedible offal	602	-	0	-	0	-	0	+	1	+	2	-	0
600	Processing	3	Blood	603	-	0	-	0	-	0	-	0	-	0	+	2
600	Processing	4	Casings/Runners	604	-	0	-	0	-	0	-	0	+	2	-	0

600	Processing	5	Rendering	605	-	0	-	0	-	0	-	0	-	0	-	0
600	Processing	6	Curing/Bacon	606	-	0	-	0	+	1	-	0	-	0	-	0
700	Fellmongery	0	Unknown	700	-	0	+	2	-	0	-	0	-	0	-	0
700	Fellmongery	1	Paint Table	701	-	0	+	2	-	0	-	0	-	0	-	0
700	Fellmongery	2	Wool room	702	-	0	+	2	-	0	-	0	-	0	-	0
700	Fellmongery	3	Pelt house	703	-	0	+	2	-	0	-	0	-	0	-	0
800	Plant Services	0	Unknown	800	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	1	Boilers	801	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	2	Carpenters	802	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	3	Cleaners	803	-	0	-	0	-	0	+	1	+	1	-	0
800	Plant Services	4	Electricians	804	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	5	Engineering fitters	805	-	0	-	0	-	0	+	1	+	1	+	1
800	Plant Services	6	Greasers	806	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	7	Laundry	807	-	0	-	0	-	0	+	1	+	1	-	0
800	Plant Services	8	Painters	808	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	9	Plumbers/Drainage	809	-	0	-	0	-	0	+	2	+	2	+	1
800	Plant Services	10	Yard gang	810	-	0	-	0	-	0	+	2	+	2	+	1
800	Plant Services	11	Stores	811	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	12	Cooperage	812	-	0	-	0	-	0	-	0	-	0	-	0
900	Admin/mgmt	0	Unknown	900	-	0	-	0	-	0	-	0	-	0	-	0
900	Admin/mgmt	1	Personnel	901	-	0	-	0	-	0	-	0	-	0	-	0
900	Admin/mgmt	2	Quality control	902	-	0	-	0	-	0	-	0	-	0	-	0
900	Admin/mgmt	3	Corporate	903	-	0	-	0	-	0	-	0	-	0	-	0
900	Admin/mgmt	4	R&D	904	-	0	-	0	-	0	-	0	-	0	-	0

Legend: + = exposed, - = not exposed

0 = no exposure, 1 = low or medium exposure, 2 = high exposure.

Table 4.5 Potential chemical exposures in Job Areas and Titles within the Freezing Works

Area code	Area	Job code	Job title	Combined code	Welding emissions		Refrigerant gases		Cleaning chemicals		Animal remedies		Hormone growth promoters		Organo-chlorines	
100	Stockyard	0	Unknown	100	-	0	-	0	-	0	+	1	+	1	+	1
100	Stockyard	1	Shepherd	101	-	0	-	0	-	0	+	1	+	1	+	1
100	Stockyard	2	Stockwash	102	-	0	-	0	+	2	+	1	+	1	+	1
200	Slaughterboard	0	Unknown	200	-	0	-	0	+	2	+	2	+	1	+	1
200	Slaughterboard	1	Stunner	201	-	0	-	0	+	2	+	2	+	1	+	1
200	Slaughterboard	2	Sticker	202	-	0	-	0	+	2	+	2	+	2	+	1
200	Slaughterboard	3	Butcher	203	-	0	-	0	+	2	+	2	+	2	+	1
200	Slaughterboard	4	Assistant	204	-	0	-	0	+	2	+	2	+	2	+	1
200	Slaughterboard	5	Gut and Bung	205	-	0	-	0	+	2	+	2	+	3	+	1
200	Slaughterboard	6	Trimmer	206	-	0	-	0	+	2	+	2	+	2	+	1
200	Slaughterboard	7	Grader	207	-	0	-	0	+	2	+	2	+	2	+	1
300	Cooling Floor	0	Unknown	300	-	0	+	1	+	1	-	0	-	0	-	0
300	Cooling Floor	1	Chillerhand	301	-	0	+	1	+	1	-	0	-	0	-	0
300	Cooling Floor	2	Bagging	302	-	0	+	1	+	1	-	0	-	0	-	0
400	Meat cutting	0	Unknown	400	-	0	-	0	+	1	+	1	-	0	+	1
400	Meat cutting	1	Boning/Cutting	401	-	0	-	0	+	1	+	1	-	0	+	1
400	Meat cutting	2	Wholesale Butchers	402	-	0	-	0	+	1	+	1	-	0	+	1
500	Freezers	0	Unknown	500	-	0	+	1	-	0	-	0	-	0	-	0
500	Freezers	1	Chamberhand	501	-	0	+	1	-	0	-	0	-	0	-	0
500	Freezers	2	Despatch	502	-	0	+	1	-	0	-	0	-	0	-	0
600	Processing	0	Unknown	600	-	0	-	0	-	0	-	0	+	1	-	0
600	Processing	1	Edible offal	601	-	0	-	0	-	0	+	0	+	1	-	0
600	Processing	2	Inedible offal	602	-	0	-	0	-	0	+	0	+	1	-	0
600	Processing	3	Blood	603	-	0	-	0	-	0	-	0	-	0	-	0
600	Processing	4	Casings/Runners	604	-	0	-	0	-	0	-	0	-	0	-	0

600	Processing	5	Rendering	605	-	0	-	0	-	0	-	0	-	0	-	0
600	Processing	6	Curing/Bacon	606	-	0	-	0	-	0	-	0	-	0	-	0
700	Fellmongery	0	Unknown	700	-	0	-	0	-	0	-	0	-	0	+	2
700	Fellmongery	1	Paint Table	701	-	0	-	0	-	0	-	0	-	0	+	2
700	Fellmongery	2	Wool room	702	-	0	-	0	-	0	-	0	-	0	+	2
700	Fellmongery	3	Pelt house	703	-	0	-	0	-	0	-	0	-	0	+	2
800	Plant Services	0	Unknown	800	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	1	Boilers	801	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	2	Carpenters	802	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	3	Cleaners	803	-	0	-	0	+	2	-	0	-	0	-	0
800	Plant Services	4	Electricians	804	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	5	Engineering fitters	805	+	2	-	0	-	0	-	0	-	0	+	2
800	Plant Services	6	Greasers	806	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	7	Laundry	807	-	0	-	0	+	2	-	0	-	0	-	0
800	Plant Services	8	Painters	808	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	9	Plumbers/Drainage	809	-	0	-	0	-	0	+	2	+	2	-	0
800	Plant Services	10	Yard gang	810	-	0	-	0	-	0	+	1	+	1	+	1
800	Plant Services	11	Stores	811	-	0	-	0	-	0	-	0	-	0	-	0
800	Plant Services	12	Cooperage	812	-	0	-	0	-	0	-	0	-	0	-	0
900	Admin/mgmt	0	Unknown	90	-	0	-	0	-	0	-	0	-	0	-	0
900	Admin/mgmt	1	Personnel	901	-	0	-	0	-	0	-	0	-	0	-	0
900	Admin/mgmt	2	Quality control	902	-	0	-	0	-	0	-	0	-	0	-	0
900	Admin/mgmt	3	Corporate	903	-	0	-	0	-	0	-	0	-	0	-	0
900	Admin/mgmt	4	R&D	904	-	0	-	0	-	0	-	0	-	0	-	0

Legend: + = exposed, - = not exposed

0 = no exposure, 1 = low or medium exposure, 2 = high exposure

4.6 Ascertainment of vital status

Study subjects were traced forward from their date of first employment (or 01/01/1988 if employment started before that date) until their date of death, date of emigration or the last day of follow-up (31/12/2000). Their mortality and cancer incidence was established by a combination of electronic and manual matching with national records for deaths and cancer registrations through the New Zealand Health Information Service (NZHIS). Electronic files for each sub-cohort containing full name, date of birth and ID number were submitted to the NZHIS to be linked to the NZHIS database. Exact matches on name and date of birth were supplemented by "near matches" involving minor differences in spelling of the name or in the date of birth (e.g. it is common for the day and month of birth to be correct, but for the year of birth to be incorrect in personnel records). The matching was used to determine the National Health Index (NHI) number for each study participant. NHI numbers were identified for 94.9% of the union cohort, 92.8% of the Lorneville sub-cohort, 88.0% of the Oringi sub-cohort and 81.5% of the Takapau sub-cohort. It was expected that not all study participants would have an NHI number since these are only "issued" when a person has contact with the hospital system or dies. This information was then linked to the NZHIS National Minimum Data Set (NMDS) to identify deaths and cancer registrations. Those cohort members not registered as having died during the study period were followed to verify vital status by record linkage with both the 2001 New Zealand Electoral Roll and with the client records of the Department of Work and Income (WINZ). In addition, record linkage with hospital discharge records held by the NZHIS (using the NHI number) and with current union membership files was used to supplement the vital status information obtained from the main matches. For

those cohort members who had died during the study period, the last date of follow-up was the date of death. For all other cohort members the last date of follow-up was the most recent date available from their work history, hospital discharge records, WINZ records, the Electoral Roll or Union membership records.

The matching with NZHIS records was done using surname, first names and date of birth to identify the national Health Index (NHI) number. This was then used to link with hospital admissions, cancer registrations and death registrations in the National Minimum Data Set (NMDS). If an exact match (on name and date of birth) was not obtained, then Telnet was used to identify near matches; in each instance it identified people with the same surname (and a similar date of birth) or the same date of birth (and a similar surname). The operator then made a decision as to whether there was a sufficiently near match, and this was then checked against the full information on the NHI database. The matching with the Electoral Roll and WINZ records was done in a similar manner. Exact matches on full name and date of birth were accepted, and for those remaining, exact matches on year of birth, surname, and first name were accepted. Exact matches on name (but not date of birth) were then manually checked for year of birth and domicile address. For the WINZ records, the Internal Revenue Department (IRD) Numbers were available for the Union and the Lorneville cohorts and this was used in combination with name and date of birth.

4.7 Data analysis

The initial analyses conducted in this study were of mortality based on external comparisons using New Zealand mortality rates. For each cause of death standardised

mortality ratios (SMRs) were calculated as the ratio of observed to expected deaths. Expected deaths were computed by multiplying the person-years, stratified by gender, five-year age bands (15-19, 20-24, 25-29,.....85+ years) and calendar year in single years, by the New Zealand national rates using the NIOSH PC LTAS programme [Steenland et al, 1998]. The national rates for mortality, for the period 1988 to 2000, were derived from the WHO Mortality Database [WHO, 2001]. 95% confidence intervals for the SMRs and SIRs were calculated under the assumption that the observed numbers of deaths or registrations follow a Poisson distribution [Checkoway et al, 1989]. Separate analyses were conducted for the Union Cohort, each Company subcohort and the combined Company Cohort.

Further stratified analyses were conducted separately, for the Union Cohort and the Company Cohort, for selected causes in subgroups of workers defined according to exposure. The first such analyses examined all cause mortality, and mortality from all cancer, lung cancer, lymphohaematopoietic cancer, non-Hodgkin's lymphoma and leukaemia by age at risk, duration of employment and time since first employed. Internal comparisons were also made, for both the Union Cohort and the Company Cohort, based on the assigned exposure categories of department "ever worked" in, and of potential biological and chemical exposures.

A further series of analyses examined mortality by duration of exposure in selected departments or in jobs that entailed specific biological or chemical exposures. These analyses investigated all cause mortality and mortality from all cancers, and cancers of the lung and lymphohaematopoietic system in those departments or jobs which contained sufficient numbers of cases. Duration of exposure was defined as either no

exposure or as one of three categories based on duration of employment in years, with different category definitions used for the Union (1-4, 5-19, 20+ years) and the Company cohorts (1-4, 5-14, 15+ years) in order to include adequate numbers in each category. An analysis of trend was included here, with the p-value derived according to the method described elsewhere [Pearce & Cryer, 1986].

Separate analyses were conducted to examine cancer incidence. Standardised incidence ratios (SIRs) were calculated in the same manner as for SMRs, with expected numbers of cases being computed from national cancer registration data also derived from the WHO statistical information service [WHO, 2001]. Similar stratified analyses as were conducted in the mortality analyses were conducted for selected cancers in both cohorts, to examine cancer incidence by age at risk, duration of exposure and time since first employed and also by duration of exposure in selected exposure categories (again with an analysis of trend). As numbers of cases were too small to give stable estimates of risk in a comparison of no, medium and high exposure to individual biological agents, an analysis using combined exposure to animal urine, faeces and blood as an index of exposure to "biological agents" was conducted. This was conducted for both the Union and Company cohorts, and also for both cohorts combined, for all cancer, and for cancers of the lung, lymphohaematopoietic system, non-Hodgkin's lymphoma and leukaemia.

Chapter 5 - Results

5.1 Introduction

This chapter presents the main results of the two cohort studies. It begins with a description of the study populations with respect to factors such as duration of employment, age at risk, time since first employed, calendar period and length of follow-up. This is followed by an analysis of the vital status ascertainment and loss to follow up. The main analyses are then presented as a series of tables describing the results of the mortality follow-up for the Union Cohort, for each company sub-cohort and for the Company Cohort. This is followed by the results of separate analyses on the union and Company Cohorts that were conducted to investigate the effect of specific exposures. All cause mortality, cancer mortality, and the specifc cancers of *a priori* interest (namely lung cancer and lymphohaematopoietic cancers) are evaluated through stratification on age at risk, duration of employment and time since first employed. Further analyses evaluate the effect of department ever worked in, and of potential biological and chemical exposures, on cause-specific mortality. The relationship between duration of employment and mortality is then examined in selected exposure categories.

Similar analyses were performed to examine cancer incidence. The incidence of selected cancers for each cohort is presented, followed by a similar examination of the incidence of all cancers, lung cancer, lymphohaematopoietic cancers, non-Hodgkin's lymphoma and leukaemia in both cohorts stratified on age at risk, duration of

employment and time since first employed. In the final analyses, the relationship between the incidence of selected cancers and duration of exposure to selected departments and to biological or chemical agents is examined.

5.2 Description of Cohorts

Table 5.1 presents a description of the Union Cohort, the three company sub-cohorts and the overall combined Company Cohort. The Union Cohort contained 4,064 individuals, while the Company Cohort comprised 6,647 individuals assembled from the Alliance Lorneville (3,430), Richmond Oringi (1,196) and Richmond Takapau (2,057) sub-cohorts. The exclusions from the analysis, 148 (4%) from the Union Cohort and 221 (3%) from the Company Cohort, were made primarily where individuals had a last known date of follow-up that was prior to the start of follow-up at 01/01/1988.

Follow-up of members of the Union Cohort involved 47,651 person-years of observation, while for members of the Company Cohort it involved 63,160 person-years. The average length of follow-up of Union Cohort members was longer at 12.2 years, compared with 9.8 years for those from the Company Cohort. The gender distribution varied between the union and company cohorts, with the Union Cohort being almost exclusively male (99%) while 18% of the Company Cohort was female.

Table 5.1 Characteristics of the cohorts

	Union Cohort	Company Cohorts			
		Lorneville	Oringi	Takapau	Combined
	N (%)	N (%)	N (%)	N (%)	N (%)
In database	4,064	3,430	1,196	2,057	6,647
Exclusions:	148 (4)	36 (1)	38 (3)	147 (7)	221 (3)
Missing date of birth or gender	0	32	38	41	111
Last date of follow-up <01/01/1988	143	4	-	103	107
Included in analysis	3,916 (96)	3,394 (99)	1,158 (97)	1,910 (93)	6,426 (97)
Gender:					
Male	3,859 (99)	2,938 (87)	918 (79)	1,415 (74)	5,239 (82)
Female	57 (1)	456 (13)	240 (21)	495 (26)	1,187 (18)
Total person-years of follow-up	47,651	33,134	10,388	19,933	63,160
Mean length of follow-up (years)	12.2	9.8	9.0	10.4	9.8
Mean duration of employment (years)	13.2	8.9	5.9	4.7	7.1
Mean age at hire (years)	28.1	29.1	33.0	30.0	30.1

A description of each cohort stratified according to factors such as duration of employment, age at risk, time since first employed, calendar period and length of follow-up is presented in Table 5.2 below. Further differences between the Union and Company Cohorts are evident from this stratification. For example, more than 50% of the person-years of observation in the Company Cohort were for individuals with

Table 5.2 Distribution of person-years

	Union Cohort	Company Cohorts			
		Lorneville	Oringi	Takapau	Combined
Years	p-yrs (%)	p-yrs (%)	p-yrs (%)	p-yrs (%)	p-yrs (%)
Duration of employment					
1-4	7,685 (16)	14,169 (43)	5,509 (53)	13,871 (70)	33,225 (53)
5-9	9,049 (19)	6,609 (20)	3,085 (30)	4,125 (21)	13,860 (22)
10-14	9,535 (20)	5,012 (15)	1,446 (14)	1,475 (7)	7,946 (12)
15-19	11,955 (25)	3,516 (11)	347 (3)	348 (2)	4,205 (7)
20+	9,427 (20)	3,828 (11)	0 (0)	114 (1)	3,924 (6)
Total	47,651 (100)	33,134 (100)	10,387 (100)	19,933 (100)	63,159 (100)
Age at risk					
<25	1,051 (2)	3,294 (10)	1,338 (13)	2,342 (12)	6,948 (11)
25-34	7,894 (17)	10,587 (32)	3,329 (32)	6,817 (34)	20,621 (33)
35-44	13,370 (28)	9,631 (29)	2734 (26)	6,165 (31)	18,447 (29)
45+	25,337 (53)	9,621 (29)	2,986 (29)	4,609 (23)	17,143 (27)
Total	47,651 (100)	33,134 (100)	10,387 (100)	19,933 (100)	63,159 (100)
Time since first employed					
<5	2,205 (5)	9,289 (28)	3,698 (36)	5,185 (26)	18,031 (29)
5-9	4,960 (10)	7,993 (24)	3,681 (35)	7,081 (35)	18,660 (30)
10-14	7,894 (17)	6,632 (20)	2,301 (22)	5,410 (27)	14,303 (22)
15-19	10,705 (22)	4,153 (13)	707 (7)	2,115 (11)	6,974 (11)
20+	21,887 (46)	5,067 (15)	0 (0)	142 (1)	5,191 (8)
Total	47,651 (100)	33,134 (100)	10,387 (100)	19,933 (100)	63,159 (100)
Calendar period					
1988-1990	11,416 (24)	6,245 (19)	1,720 (17)	3,902 (20)	11,829 (19)
1991-1995	18,714 (39)	12,281 (37)	4,000 (38)	7,760 (39)	23,934 (38)
1996-2000	17,522 (37)	14,608 (44)	4,667 (45)	8,271 (41)	27,396 (43)
Total	47,651 (100)	33,134 (100)	10,387 (100)	19,933 (100)	63,159 (100)

fewer than 5 years employment in the industry, whereas 84% of the person-years in the Union Cohort were for individuals with 5 or more years of employment. The distribution of age at risk shows that the Union Cohort was also older than the Company Cohort, primarily because the union membership records from which the cohort was assembled began in 1970 whereas the company sub-cohorts were enumerated from employment records from the mid to late 1980s onwards. This

difference in cohort definition was also reflected in the different distribution of the calendar periods of observation, with the Union Cohort having a slightly higher percentage of individuals in the 1988-1990 period, i.e. 24% compared with 19%. There was an even more marked difference between the two cohorts in time since first employment, with almost 60% of the person-years of the Company Cohort being within 10 years of first employment, compared with only 15% of the Union Cohort. By contrast almost 50% of the person-years in the Union Cohort were more than 20 years since first employment compared with only 8% in the Company Cohort.

5.3 Vital status ascertainment and loss to follow-up

The results of the follow-up of the study populations are shown in Table 5.3 below. Of the 3,916 individuals from the Union Cohort included in the study, 3,350 (86%) were still alive at the end of the study period, 246 (6%) had died, and 320 (8%) had incomplete follow-up. From the 51,088 theoretical person-years of follow-up that the Union Cohort would have accumulated (i.e. if those lost to follow-up had been followed until 31/12/2000) the total number of person-years of observation achieved was 47,651 (93.3%).

Because of the lower average age of individuals in the company sub-cohorts there were fewer deaths among members of the Company Cohort. Of the 6,426 individuals included in the analysis, 5,134 (80%) were still alive at the end of the study period, 227 (4%) were deceased and 1065 (16%) were lost to follow-up. The achieved follow-up as a proportion of possible person-years of observation, at 63,166 compared with 69,014 person years or 91.53%, was similar to that achieved in the Union Cohort.

Table 5.3 – Follow-up and Vital Status ascertainment

	Union Cohort	Company Cohorts			
		Lorneville	Oringi	Takapau	Combined
Vital status at 31/12/2000	N (%)	N (%)	N (%)	N (%)	N (%)
Alive	3,350 (86)	2,804 (83)	811 (70)	1,546 (81)	5,134 (80)
Deceased	246 (6)	132 (4)	35 (3)	61 (3)	227 (4)
Lost to follow-up	320 (8)	458 (13)	312 (27)	303 (16)	1,065 (16)
Total	3,916	3,394	1,158	1,910	6,426
Person-years	47,651	33,134	10,388	19,933	63,160
Total possible person-years	51,088	35,394	11,305	22,649	69,014
Achieved follow-up (as % of total possible person-yrs)	93%	94%	92%	88%	92%

5.4 Cause-specific mortality

The findings for total and cause-specific mortality are presented below in Tables 5.4.1 (Union Cohort), Tables 5.4.2 to 5.4.4 (company sub-cohorts) and Table 5.4.5 (combined Company Cohort).

5.4.1 Cause-specific Mortality - Union Cohort

The observed all cause mortality in the Union Cohort was significantly below expected (SMR 0.86, 95% CI 0.76 – 0.98), and there was also a deficit in mortality from all malignant neoplasms (SMR 0.88, 95% CI 0.71 – 1.10), consistent with a

Table 5.4.1 Cause-specific Mortality – Union Cohort

Cause of death (ICD 9th revision)	Observed	Expected	SMR	95% confidence interval Lower	Upper
ALL CAUSES	246	284.93	**0.86***	0.76	0.98
ALL MALIGNANT NEOPLASMS (140-208)	84	94.92	**0.88**	0.71	1.10
Oral cavity and pharynx (140-149)	0	2.27	**0.00**	0.00	1.63
Oral cavity (141 - 145)	0	1.06	**0.00**	0.00	3.49
Nasopharynx (147)	0	0.36	**0.00**	0.00	10.23
Oesophagus (150)	4	3.27	**1.22**	0.33	3.13
Stomach (151)	4	4.39	**0.91**	0.25	2.33
Colon (153)	5	9.44	**0.53**	0.17	1.24
Rectum (154)	6	6.39	**0.94**	0.34	2.04
Liver, specified as primary (1550)	0	1.94	**0.00**	0.00	1.90
Gallbladder (156)	0	0.54	**0.00**	0.00	6.82
Pancreas (157)	4	3.80	**1.05**	0.29	2.69
Peritoneum (158)	1	0.25	**4.00**	0.10	22.21
Nose and sinuses (160)	0	0.10	**0.00**	0.00	36.35
Larynx (161)	0	0.76	**0.00**	0.00	4.84
Lung (162)	23	23.20	**0.99**	0.63	1.49
Pleura (163)	0	1.15	**0.00**	0.00	3.22
Bone (170)	0	0.32	**0.00**	0.00	11.47
Soft tissue (171)	0	0.51	**0.00**	0.00	7.23
Melanoma (172)	5	4.02	**1.24**	0.40	2.91
Other skin (173)	1	0.66	**1.51**	0.04	8.41
Breast (174-175)	0	0.38	**0.00**	0.00	6.48
Female genital organs (179 - 184)	0	0.13	**0.00**	0.00	28.99
Prostate (185)	4	5.78	**0.69**	0.19	1.77
Other male genital organs (186, 187)	0	0.34	**0.00**	0.00	7.18
Bladder (188)	2	1.64	**1.22**	0.15	4.40
Kidney (189)	6	2.68	**2.24**	0.82	4.87
Eye (190)	0	0.15	**0.00**	0.00	24.24
Brain (191)	4	4.01	**1.00**	0.27	2.55
Thyroid (193)	0	0.17	**0.00**	0.00	22.35
Other endocrine glands (194)	0	0.14	**0.00**	0.00	26.24
Ill defined (195, 199)	6	5.69	**1.06**	0.44	2.18
Lymphatic and haematopoietic tissue (200-208)	5	8.85	**0.57**	0.18	1.32
Non-Hodgkin's lymphoma (200, 202)	3	3.98	**0.75**	0.21	2.01
Hodgkin's disease (201)	0	0.30	**0.00**	0.00	12.34
Multiple myeloma (203)	0	1.55	**0.00**	0.00	2.39
Leukaemia and aleukaemia (204-208)	2	3.02	**0.66**	0.13	2.12
BENIGN NEOPLASMS (210-239)	1	0.89	**1.12**	0.03	6.24
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	6	12.66	**0.47***	0.20	0.98
MENTAL DISORDERS (290-319)	2	1.93	**1.03**	0.13	3.74
DIS. OF NERVOUS SYSTEM (320-359)	0	4.69	**0.00***	0.00	0.79
DIS. OF CIRCULATORY SYSTEM (390-459)	98	108.86	**0.90**	0.73	1.10
Hypertension (401-405)	1	2.12	**0.47**	0.01	2.62
Ischaemic heart disease (410-414)	77	76.87	**1.00**	0.79	1.25
Pulmonary circulation and other heart diseases (415-429)	10	9.98	**1.00**	0.48	1.84
Cerebrovascular (430-438)	10	13.35	**0.75**	0.36	1.38
DIS. OF RESPIRATORY SYSTEM (460-519)	13	15.31	**0.85**	0.48	1.41
Pneumonia (480-486)	2	1.73	**1.16**	0.14	4.17
Influenza (487)	0	0.06	**0.00**	0.00	58.38
Bronchitis, emphysema and asthma (490-493)	11	4.66	**2.36***	1.18	4.23
Pneumoconioses (500-508)	0	0.17	**0.00**	0.00	21.54
DIS. OF DIGESTIVE SYSTEM (520-579)	5	6.47	**0.77**	0.29	1.69
DIS. OF URINARY SYSTEM (580-599)	0	1.91	**0.00**	0.00	1.94
DIS. OF SKIN (680-739)	0	1.03	**0.00**	0.00	2.39
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	3	0.44	**6.80***	1.40	19.87
EXTERNAL CAUSES (E800-999)	34	33.03	**1.03**	0.71	1.44
Transport accident (800-848)	12	12.73	**0.94**	0.49	1.65
Other accidents (850-888, 890-929)	2	6.57	**0.30**	0.04	1.10
Suicide (950-959)	10	11.84	**0.84**	0.40	1.55
Homicide and other violence (960-999)	10	1.79	**5.60****	2.68	10.30

* $p < 0.05$
** $p < 0.01$

healthy worker effect. There was also a deficit for diseases of the circulatory system (SMR = 0.90, 95% CI 0.73 – 1.10, 98 deaths), as well as a significant deficit for the category of diseases of the endocrine system and blood (SMR 0.47, 95% CI 0.20 – 0.98, based on 6 cases). A significant elevation was observed for deaths in the non-malignant respiratory disease category of bronchitis, emphysema and asthma (SMR 2.36, 95% CI 1.18 – 4.23). There was also a significant elevation in deaths from symptoms and ill-defined conditions (SMR 6.80, 95% CI 1.40 – 19.87, based on 3 deaths).

No statistically significant elevations in risk were observed for any specific cancer types, although there was an excess of cancer of the kidney (SMR 2.24, based on 6 deaths). Of other diseases of *a priori* interest the 23 deaths from lung cancer were close to expected (SMR 0.99, 95% CI 0.63 – 1.49), and there were fewer deaths from cancers of lymphatic and haematopoietic tissue than expected (SMR 0.57, 5 deaths), and no cases of cancer of the larynx were observed.

5.4.2 Cause-specific Mortality - Company sub-cohorts

In contrast to the experience of the Union Cohort, mortality from all causes exceeded expected rates in each of the Lorneville, Oringi and Takapau company sub-cohorts (SMRs 1.12, 1.04 and 1.15 respectively). An excess relative risk of death from all malignant neoplasms was also observed in Lorneville and Takapau (SMRs 1.32 and 1.08 respectively), although with only 6 deaths from cancer the Oringi sub-cohort had fewer than expected (SMR 0.58). Also in contrast to the Union Cohort there was an excess of mortality from diseases of the circulatory system in the Lorneville and

Table 5.4.2 Cause-specific Mortality – Lorneville cohort

Cause of death (ICD 9th revision)	Observed	Expected	SMR	95% confidence interval Lower	Upper
ALL CAUSES	132	117.60	**1.12**	0.94	1.33
ALL MALIGNANT NEOPLASMS (140-208)	47	35.58	**1.32**	0.97	1.76
Oral cavity and pharynx (140-149)	1	0.81	**1.24**	0.03	6.89
Oral cavity (141 - 145)	1	0.38	**2.63**	0.07	14.60
Nasopharynx (147)	0	0.15	**0.00**	0.00	24.74
Oesophagus (150)	2	1.07	**1.86**	0.23	6.72
Stomach (151)	2	1.64	**1.22**	0.15	4.40
Colon (153)	4	3.34	**1.20**	0.33	3.06
Rectum (154)	1	2.20	**0.45**	0.01	2.52
Liver, specified as primary (1550)	0	0.73	**0.00**	0.00	5.03
Gallbladder (156)	1	0.21	**4.84**	0.12	26.89
Pancreas (157)	0	1.36	**0.00**	0.00	2.72
Peritoneum (158)	0	0.11	**0.00**	0.00	33.54
Nose and sinuses (160)	0	0.04	**0.00**	0.00	83.22
Larynx (161)	1	0.23	**4.28**	0.11	23.77
Lung (162)	17	7.74	**2.20***	1.28	3.52
Pleura (163)	0	0.37	**0.00**	0.00	10.11
Bone (170)	0	0.19	**0.00**	0.00	19.08
Soft tissue (171)	0	0.25	**0.00**	0.00	14.56
Melanoma (172)	2	1.70	**1.18**	0.14	4.26
Other skin (173)	0	0.21	**0.00**	0.00	17.38
Breast (174-175)	0	1.11	**0.00**	0.00	2.23
Female genital organs (179 - 184)	0	0.51	**0.00**	0.00	7.24
Prostate (185)	2	1.76	**1.14**	0.14	4.10
Other male genital organs (186, 187)	0	0.21	**0.00**	0.00	11.58
Bladder (188)	2	0.53	**3.74**	0.45	13.52
Kidney (189)	0	0.98	**0.00**	0.00	3.78
Eye (190)	0	0.06	**0.00**	0.00	62.57
Brain (191)	2	1.68	**1.19**	0.14	4.30
Thyroid (193)	2	0.07	**27.74****	3.36	100.1
Other endocrine glands (194)	0	0.07	**0.00**	0.00	53.12
Ill defined (195, 199)	2	2.04	**0.98**	0.20	3.14
Lymphatic and haematopoietic tissue (200-208)	5	3.60	**1.39**	0.45	3.24
Non-Hodgkin's lymphoma (200, 202)	3	1.59	**1.89**	0.52	5.04
Hodgkin's disease (201)	0	0.15	**0.00**	0.00	24.42
Multiple myeloma (203)	0	0.54	**0.00**	0.00	6.82
Leukaemia and aleukaemia (204-208)	2	1.32	**1.51**	0.30	4.85
BENIGN NEOPLASMS (210-239)	0	0.37	**0.00**	0.00	9.91
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	1	5.25	**0.19***	0.02	0.89
MENTAL DISORDERS (290-319)	1	1.19	**0.84**	0.02	4.69
DIS. OF NERVOUS SYSTEM (320-359)	1	2.35	**0.43**	0.01	2.37
DIS. OF CIRCULATORY SYSTEM (390-459)	42	38.12	**1.10**	0.79	1.49
Hypertension (401-405)	0	0.77	**0.00**	0.00	4.81
Ischaemic heart disease (410-414)	27	26.02	**1.04**	0.68	1.51
Pulmonary circulation and other heart diseases (415-429)	5	3.91	**1.28**	0.41	2.99
Cerebrovascular (430-438)	10	6.18	**1.62**	0.78	2.98
DIS. OF RESPIRATORY SYSTEM (460-519)	6	3.22	**1.86**	0.68	4.05
Pneumonia (480-486)	0	0.63	**0.00**	0.00	5.83
Influenza (487)	0	0.02	**0.00**	0.00	152.0
Bronchitis, emphysema and asthma (490-493)	6	1.78	**3.38***	1.23	7.36
Pneumoconioses (500-508)	0	0.06	**0.00**	0.00	62.02
DIS. OF DIGESTIVE SYSTEM (520-579)	7	3.11	**2.25**	0.90	4.64
DIS. OF URINARY SYSTEM (580-599)	1	0.75	**1.33**	0.03	7.41
DIS. OF SKIN (680-739)	1	0.42	**2.37**	0.22	10.99
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	0	0.23	**0.00**	0.00	15.83
EXTERNAL CAUSES (E800-999)	26	24.10	**1.08**	0.71	1.58
Transport accident (800-848)	9	9.89	**0.91**	0.42	1.73
Other accidents (850-888, 890-929)	2	4.07	**0.49**	0.06	1.77
Suicide (950-959)	10	8.80	**1.14**	0.54	2.09
Homicide and other violence (960-999)	5	1.30	**3.83***	1.24	8.96

* $p < 0.05$
** $p < 0.01$

Oringi sub-cohorts, while the observed number was close to expected in the Takapau sub-cohort, indicating that there was no obvious healthy worker effect operating in the company sub-cohorts.

5.4.2 (i) Cause-specific Mortality - Lorneville sub-cohort

In addition to the elevated all cause and all cancer mortality observed in the Lorneville cohort, significant excesses were observed for lung cancer (SMR 2.20, 17 deaths), thyroid cancer (SMR 27.74, based on only 2 deaths), and for bronchitis, emphysema and asthma (SMR 3.38, 6 deaths). Non-significant elevations were also observed for several cancers, namely oesophageal (SMR 1.86, 2 deaths), bladder (SMR 3.74, 2 deaths), lymphatic and haematopoietic tissue (SMR 1.39, 5 deaths), non-Hodgkin's lymphoma (SMR 1.89, 3 deaths) and leukaemia (SMR 1.51, 2 deaths). Non-malignant diseases with non-significant excesses included cerebrovascular disease (SMR 1.62, 10 deaths) and diseases of the digestive system (SMR 2.25, 7 deaths). As was observed in the Union Cohort, a significant deficit in deaths from diseases of the endocrine system and blood (SMR 0.19) was found in the Lorneville cohort although this was based on only 1 death. A similar non-significant deficit was also observed for diseases of the nervous system (SMR 0.43, based on 1 death).

5.4.2 (ii) Cause-specific Mortality - Oringi sub-cohort

In the Oringi sub-cohort, the observed number of deaths was close to that expected (SMR 1.04, 35 deaths). A deficit of cancer deaths (SMR 0.58, 6 deaths) was observed, however, with two from cancer of the colon (SMR 2.07), and one each from cancers

Table 5.4.3 Cause-specific Mortality – Oringi cohort

Cause of death (ICD 9th revision)	Observed	Expected	SMR	95% confidence interval Lower	Upper
ALL CAUSES	35	33.62	**1.04**	0.73	1.45
ALL MALIGNANT NEOPLASMS (140-208)	6	10.34	**0.58**	0.21	1.26
Oral cavity and pharynx (140-149)	0	0.22	**0.00**	0.00	16.67
Oral cavity (141 - 145)	0	0.11	**0.00**	0.00	34.53
Nasopharynx (147)	0	0.04	**0.00**	0.00	89.69
Oesophagus (150)	0	0.29	**0.00**	0.00	12.96
Stomach (151)	0	0.46	**0.00**	0.00	8.06
Colon (153)	2	0.96	**2.07**	0.25	7.49
Rectum (154)	0	0.62	**0.00**	0.00	5.91
Liver, specified as primary (1550)	1	0.21	**4.71**	0.12	26.14
Gallbladder (156)	0	0.06	**0.00**	0.00	61.74
Pancreas (157)	0	0.38	**0.00**	0.00	9.67
Peritoneum (158)	0	0.03	**0.00**	0.00	111.22
Nose and sinuses (160)	0	0.01	**0.00**	0.00	259.94
Larynx (161)	0	0.06	**0.00**	0.00	58.91
Lung (162)	1	2.14	**0.47**	0.01	2.60
Pleura (163)	0	0.11	**0.00**	0.00	35.25
Bone (170)	0	0.06	**0.00**	0.00	62.25
Soft tissue (171)	0	0.08	**0.00**	0.00	47.67
Melanoma (172)	0	0.49	**0.00**	0.00	7.58
Other skin (173)	0	0.06	**0.00**	0.00	64.04
Breast (174-175)	0	0.53	**0.00**	0.00	4.64
Female genital organs (179 - 184)	1	0.25	**4.07**	0.10	22.59
Prostate (185)	0	0.44	**0.00**	0.00	8.35
Other male genital organs (186, 187)	0	0.06	**0.00**	0.00	43.00
Bladder (188)	0	0.15	**0.00**	0.00	24.95
Kidney (189)	0	0.28	**0.00**	0.00	13.33
Eye (190)	0	0.02	**0.00**	0.00	222.7
Brain (191)	0	0.49	**0.00**	0.00	7.47
Thyroid (193)	0	0.02	**0.00**	0.00	156.3
Other endocrine glands (194)	0	0.02	**0.00**	0.00	169.1
Ill defined (195, 199)	1	0.59	**1.71**	0.16	7.97
Lymphatic and haematopoietic tissue (200-208)	0	1.04	**0.00**	0.00	3.55
Non-Hodgkin's lymphoma (200, 202)	0	0.46	**0.00**	0.00	5.42
Hodgkin's disease (201)	0	0.05	**0.00**	0.00	82.80
Multiple myeloma (203)	0	0.15	**0.00**	0.00	25.03
Leukaemia and aleukaemia (204-208)	0	0.39	**0.00**	0.00	6.26
BENIGN NEOPLASMS (210-239)	2	0.11	**18.22***	2.21	65.76
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	3	1.52	**1.98**	0.55	5.28
MENTAL DISORDERS (290-319)	0	0.35	**0.00**	0.00	10.60
DIS. OF NERVOUS SYSTEM (320-359)	1	0.69	**1.45**	0.04	8.03
DIS. OF CIRCULATORY SYSTEM (390-459)	16	10.46	**1.53**	0.87	2.49
Hypertension (401-405)	0	0.22	**0.00**	0.00	16.96
Ischaemic heart disease (410-414)	8	7.06	**1.13**	0.49	2.24
Pulmonary circulation and other heart diseases (415-429)	7	1.10	**6.36****	2.55	13.11
Cerebrovascular (430-438)	1	1.39	**0.72**	0.02	3.99
DIS. OF RESPIRATORY SYSTEM (460-519)	1	1.49	**0.67**	0.06	3.13
Pneumonia (480-486)	0	0.18	**0.00**	0.00	20.43
Influenza (487)	0	0.01	**0.00**	0.00	501.3
Bronchitis, emphysema and asthma (490-493)	1	0.50	**2.00**	0.05	11.10
Pneumoconioses (500-508)	0	0.02	**0.00**	0.00	234.9
DIS. OF DIGESTIVE SYSTEM (520-579)	0	0.67	**0.00**	0.00	3.66
DIS. OF URINARY SYSTEM (580-599)	0	0.21	**0.00**	0.00	17.83
DIS. OF SKIN (680-739)	0	0.13	**0.00**	0.00	18.81
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	0	0.07	**0.00**	0.00	52.78
EXTERNAL CAUSES (E800-999)	6	7.17	**0.84**	0.31	1.82
Transport accident (800-848)	3	2.96	**1.01**	0.21	2.96
Other accidents (850-888, 890-929)	2	1.18	**1.70**	0.21	6.15
Suicide (950-959)	0	2.62	**0.00**	0.00	1.41
Homicide and other violence (960-999)	1	0.40	**2.53**	0.06	14.05

* $p < 0.05$
** $p < 0.01$

of the liver, lung, female genital organs and ill-defined sites. A significant elevation were observed for deaths from benign neoplasms (SMR 18.22, based on only 2 deaths), while a highly significant elevation was observed for the category pulmonary circulation and other heart diseases (SMR 6.36, 7 deaths). In contrast to what had been observed in the Union Cohort and the Lorneville sub-cohort, an excess was observed for the category diseases of the endocrine system and blood (SMR 1.98, 3 deaths).

5.4.2 (iii) Cause-specific Mortality - Takapau sub-cohort

In addition to the elevated mortality from all causes (SMR 1.15, 61 deaths), and from all malignant neoplasms (SMR 1.08, 17 deaths), members of the Takapau sub-cohort experienced a highly significant excess of deaths from external causes (SMR 1.82, 24 cases). Non-significant elevations were observed for several cancer types, including stomach (SMR 2.80, 2 deaths), pancreas (SMR 3.60, 2 deaths), lung (SMR 1.65, 5 deaths) and malignant melanoma of the skin (SMR 2.42, 2 deaths). Diseases of the circulatory system contributed the expected number of deaths (SMR 0.98, 15 cases), although an excess was observed for the non-malignant respiratory disease category (SMR 1.41, 3 deaths), primarily due to the two deaths from the category bronchitis, emphysema and asthma (SMR 2.62). Of the other diseases of *a priori* interest there was only one death from non-Hodgkin's lymphoma (SMR 1.39) and no deaths from laryngeal cancer.

Table 5.4.4 Cause-specific Mortality – Takapau cohort

Cause of death (ICD 9th revision)	Observed	Expected	SMR	95% confidence interval Lower	Upper
ALL CAUSES	61	53.15	**1.15**	0.88	1.47
ALL MALIGNANT NEOPLASMS (140-208)	17	15.68	**1.08**	0.63	1.74
Oral cavity and pharynx (140-149)	1	0.34	**2.94**	0.08	16.36
Oral cavity (141 - 145)	1	0.16	**6.20**	0.16	34.45
Nasopharynx (147)	0	0.07	**0.00**	0.00	52.40
Oesophagus (150)	1	0.41	**2.45**	0.06	13.59
Stomach (151)	2	0.72	**2.80**	0.34	10.11
Colon (153)	1	1.39	**0.72**	0.02	3.99
Rectum (154)	0	0.90	**0.00**	0.00	4.12
Liver, specified as primary (1550)	0	0.33	**0.00**	0.00	11.29
Gallbladder (156)	0	0.09	**0.00**	0.00	40.42
Pancreas (157)	2	0.56	**3.60**	0.44	12.99
Peritoneum (158)	0	0.06	**0.00**	0.00	65.45
Nose and sinuses (160)	0	0.02	**0.00**	0.00	162.0
Larynx (161)	0	0.09	**0.00**	0.00	43.43
Lung (162)	5	3.03	**1.65**	0.54	3.86
Pleura (163)	0	0.15	**0.00**	0.00	25.33
Bone (170)	0	0.10	**0.00**	0.00	36.49
Soft tissue (171)	0	0.13	**0.00**	0.00	27.77
Melanoma (172)	2	0.83	**2.42**	0.29	8.72
Other skin (173)	0	0.08	**0.00**	0.00	46.59
Breast (174-175)	1	0.99	**1.02**	0.09	4.73
Female genital organs (179 - 184)	0	0.45	**0.00**	0.00	8.18
Prostate (185)	0	0.59	**0.00**	0.00	6.27
Other male genital organs (186, 187)	0	0.11	**0.00**	0.00	23.05
Bladder (188)	0	0.20	**0.00**	0.00	18.63
Kidney (189)	0	0.42	**0.00**	0.00	8.81
Eye (190)	0	0.03	**0.00**	0.00	135.7
Brain (191)	0	0.81	**0.00**	0.00	4.56
Thyroid (193)	0	0.03	**0.00**	0.00	110.3
Other endocrine glands (194)	0	0.04	**0.00**	0.00	99.04
Ill defined (195, 199)	1	0.86	**1.16**	0.11	5.42
Lymphatic and haematopoietic tissue (200-208)	1	1.65	**0.61**	0.02	3.38
Non-Hodgkin's lymphoma (200, 202)	1	0.72	**1.39**	0.13	6.47
Hodgkin's disease (201)	0	0.08	**0.00**	0.00	46.52
Multiple myeloma (203)	0	0.22	**0.00**	0.00	17.08
Leukaemia and aleukaemia (204-208)	0	0.63	**0.00**	0.00	3.91
BENIGN NEOPLASMS (210-239)	0	0.17	**0.00**	0.00	21.34
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	0	2.43	**0.00**	0.00	1.01
MENTAL DISORDERS (290-319)	0	0.61	**0.00**	0.00	6.10
DIS. OF NERVOUS SYSTEM (320-359)	1	1.18	**0.84**	0.02	4.69
DIS. OF CIRCULATORY SYSTEM (390-459)	15	15.38	**0.98**	0.55	1.61
Hypertension (401-405)	1	0.32	**3.12**	0.08	17.35
Ischaemic heart disease (410-414)	12	10.21	**1.18**	0.61	2.05
Pulmonary circulation and other heart diseases (415-429)	0	1.75	**0.00**	0.00	2.11
Cerebrovascular (430-438)	2	2.05	**0.98**	0.12	3.52
DIS. OF RESPIRATORY SYSTEM (460-519)	3	2.14	**1.41**	0.39	3.75
Pneumonia (480-486)	1	0.26	**3.89**	0.10	21.60
Influenza (487)	0	0.01	**0.00**	0.00	352.4
Bronchitis, emphysema and asthma (490-493)	2	0.76	**2.62**	0.32	9.48
Pneumoconioses (500-508)	0	0.03	**0.00**	0.00	150.5
DIS. OF DIGESTIVE SYSTEM (520-579)	0	1.01	**0.00**	0.00	2.45
DIS. OF URINARY SYSTEM (580-599)	0	0.33	**0.00**	0.00	11.28
DIS. OF SKIN (680-739)	0	0.20	**0.00**	0.00	12.43
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	1	0.12	**8.30**	0.21	46.09
EXTERNAL CAUSES (E800-999)	24	13.16	**1.82****	1.17	2.71
Transport accident (800-848)	9	5.54	**1.62**	0.74	3.08
Other accidents (850-888, 890-929)	3	2.12	**1.42**	0.29	4.14
Suicide (950-959)	6	4.74	**1.26**	0.46	2.75
Homicide and other violence (960-999)	6	0.74	**8.14****	2.97	17.73

* $p < 0.05$
** $p < 0.01$

5.4.3 Cause-specific Mortality - Combined Company Cohort

In contrast to the experience of the Union Cohort, the combined Company Cohort experienced excess mortality from all causes (SMR 1.12, 227 deaths), with major contributions from all cancers (SMR 1.12, 69 deaths) as well as from diseases of the circulatory system (SMR 1.15, 73 deaths), diseases of the respiratory system (SMR 1.10, 10 deaths), diseases of the digestive system (SMR 1.49, 6 deaths), and from external causes (SMR 1.27, 56 deaths).

Among the cancers, significant excess mortality was observed for lung cancer (SMR 1.79, 23 deaths). Excess deaths were also observed for several specific cancers, including cancer of the thyroid (SMR 15.55, 2 deaths), the oral cavity (SMR 3.09, 2 deaths), oesophagus (SMR 1.70, 3 deaths), bladder (SMR 2.28, 2 deaths), and non-Hodgkin's lymphoma (SMR 1.45, 4 deaths).

Excesses were observed for mortality from diseases of the circulatory and respiratory systems, and the excesses were marked for pulmonary circulation and other heart diseases (SMR 1.78, 12 deaths), cerebrovascular disease (SMR 1.56, 13 deaths) and highly significant for bronchitis, emphysema and asthma (SMR 2.97, 9 deaths).

The overall reduction in risk of death from diseases of the endocrine system and blood, and from diseases of the nervous system, that was seen in the Union Cohort was also observed in the Company Cohort. There were 4 deaths from diseases of the endocrine system and blood (SMR 0.44) and 3 from diseases of the nervous system (SMR 0.71). There were only 2 deaths from leukaemia (SMR 0.86).

Table 5.4.5 Cause-specific Mortality – Combined Company Cohort

Cause of death (ICD 9th revision)	Observed	Expected	SMR	95% confidence interval Lower	Upper
ALL CAUSES	227	203.57	**1.12**	0.98	1.27
ALL MALIGNANT NEOPLASMS (140-208)	69	61.38	**1.12**	0.88	1.42
Oral cavity and pharynx (140-149)	2	1.36	**1.47**	0.18	5.30
Oral cavity (141 - 145)	2	0.65	**3.09**	0.38	11.17
Nasopharynx (147)	0	0.26	**0.00**	0.00	14.21
Oesophagus (150)	3	1.76	**1.70**	0.35	4.98
Stomach (151)	4	2.80	**1.43**	0.39	3.65
Colon (153)	6	5.68	**1.06**	0.39	2.30
Rectum (154)	1	3.71	**0.27**	0.01	1.50
Liver, specified as primary (1550)	1	1.27	**0.79**	0.02	4.39
Gallbladder (156)	1	0.36	**2.81**	0.07	15.59
Pancreas (157)	2	2.29	**0.87**	0.11	3.16
Peritoneum (158)	0	0.20	**0.00**	0.00	18.55
Nose and sinuses (160)	0	0.08	**0.00**	0.00	45.59
Larynx (161)	1	0.38	**2.63**	0.07	14.62
Lung (162)	23	12.87	**1.79***	1.13	2.68
Pleura (163)	0	0.61	**0.00**	0.00	6.02
Bone (170)	0	0.35	**0.00**	0.00	10.48
Soft tissue (171)	0	0.46	**0.00**	0.00	7.99
Melanoma (172)	4	3.00	**1.33**	0.36	3.41
Other skin (173)	0	0.35	**0.00**	0.00	10.61
Breast (174-175)	1	2.62	**0.38**	0.04	1.78
Female genital organs (179 - 184)	1	1.21	**0.83**	0.02	4.61
Prostate (185)	2	2.78	**0.72**	0.09	2.59
Other male genital organs (186, 187)	0	0.38	**0.00**	0.00	6.57
Bladder (188)	2	0.88	**2.28**	0.28	8.23
Kidney (189)	0	1.66	**0.00**	0.00	2.22
Eye (190)	0	0.10	**0.00**	0.00	36.06
Brain (191)	2	2.97	**0.67**	0.08	2.43
Thyroid (193)	2	0.13	**15.55***	1.88	56.15
Other endocrine glands (194)	0	0.13	**0.00**	0.00	28.82
Ill defined (195, 199)	4	3.48	**1.15**	0.39	2.74
Lymphatic and haematopoietic tissue (200-208)	6	6.26	**0.96**	0.35	2.09
Non-Hodgkin's lymphoma (200, 202)	4	2.75	**1.45**	0.49	3.45
Hodgkin's disease (201)	0	0.27	**0.00**	0.00	13.48
Multiple myeloma (203)	0	0.90	**0.00**	0.00	4.10
Leukaemia and aleukaemia (204-208)	2	2.34	**0.86**	0.17	2.75
BENIGN NEOPLASMS (210-239)	2	0.65	**3.07**	0.37	11.07
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	4	9.16	**0.44**	0.15	1.04
MENTAL DISORDERS (290-319)	1	2.13	**0.47**	0.01	2.61
DIS. OF NERVOUS SYSTEM (320-359)	3	4.21	**0.71**	0.15	2.09
DIS. OF CIRCULATORY SYSTEM (390-459)	73	63.73	**1.15**	0.90	1.44
Hypertension (401-405)	1	1.30	**0.77**	0.02	4.27
Ischaemic heart disease (410-414)	47	43.13	**1.09**	0.80	1.45
Pulmonary circulation and other heart diseases (415-429)	12	6.73	**1.78**	0.92	3.11
Cerebrovascular (430-438)	13	8.35	**1.56**	0.83	2.66
DIS. OF RESPIRATORY SYSTEM (460-519)	10	9.06	**1.10**	0.57	1.96
Pneumonia (480-486)	1	1.07	**0.94**	0.02	5.21
Influenza (487)	0	0.04	**0.00**	0.00	87.91
Bronchitis, emphysema and asthma (490-493)	9	3.03	**2.97****	1.36	5.64
Pneumoconioses (500-508)	0	0.10	**0.00**	0.00	37.14
DIS. OF DIGESTIVE SYSTEM (520-579)	6	4.02	**1.49**	0.62	3.07
DIS. OF URINARY SYSTEM (580-599)	1	1.28	**0.78**	0.02	4.34
DIS. OF SKIN (680-739)	1	0.75	**1.34**	0.12	6.23
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	1	0.42	**2.37**	0.06	13.18
EXTERNAL CAUSES (E800-999)	56	44.20	**1.27**	0.96	1.65
Transport accident (800-848)	21	18.30	**1.15**	0.71	1.75
Other accidents (850-888, 890-929)	7	7.33	**0.95**	0.38	1.97
Suicide (950-959)	16	16.08	**1.00**	0.57	1.62
Homicide and other violence (960-999)	12	2.42	**4.95****	2.56	8.65

* $p < 0.05$
** $p < 0.01$

5.5 Mortality from selected causes by age, duration of employment and time since first employed

The results of separate stratified analyses to investigate the effect of the exposure variables age at risk, duration of employment and time since first employed on all cause mortality, cancer mortality and the cancers of *a priori* interest are shown in tables 5.5.1 (Union Cohort) and 5.5.2 (Company Cohort) below.

In the Union Cohort no clear pattern was evident to suggest any relationship between mortality from any of the specific causes and age at risk, duration of employment or time since first employment. For lung cancer, the SMR increased with increasing age, but no pattern was evident with either duration of employment or time since first employed. Small numbers of deaths from cancer of lymphatic and haematopoietic tissue, and the specific histological subtypes, precluded any meaningful analysis of the effect of exposure. The 5 deaths that did occur were in the older age groups, but no trend with increasing duration of employment or time since first employed was evident.

In the Company Cohort there was a small increased relative risk of all cause mortality with increasing age at risk, but no clear increase with either duration of exposure or time since first employed. For all cancer there was a moderate, but inconsistent, increase with all three variables. Deaths from lung cancer only occurred in the highest age bands, although there was no clear increase with age at risk. However, a clear but inconsistent increase with both increasing duration of employment and time since first employed was evident for lung cancer, with highly significantly increased SMRs

Table 5.5.1 **Mortality from selected causes according to age, duration of employment and time since first employed in the Union Cohort.**

	All Causes		All cancer		Lung Cancer	
	O/E	SMR	O/E	SMR	O/E	SMR
Total	246 / 284.93	**0.86***	84 / 94.92	**0.88**	23 / 23.20	**0.99**
Age (years)						
< 25	3 / 1.96	**1.53**	0 / 0.08	**0.00**	0 / 0.00	**0.00**
25 – 34	6 / 11.13	**0.54**	0 / 1.26	**0.00**	0 / 0.02	**0.00**
35 – 44	21 / 24.89	**0.84**	2 / 4.79	**0.42**	0 / 0.46	**0.00**
45 – 54	39 / 56.42	**0.69***	17 / 18.09	**0.94**	2 / 3.73	**0.54**
55 – 64	106 / 112.21	**0.95**	38 / 42.38	**0.90**	8 / 11.50	**0.70**
65 +	71 / 78.32	**0.91**	27 / 28.31	**0.95**	13 / 7.49	**1.13**
Duration of employment (years)						
1 -4	40 / 35.85	**1.12**	13 / 10.95	**1.19**	5/ 2.57	**1.95**
5 – 9	32 / 39.58	**0.81**	7 / 11.98	**0.58**	1 / 2.77	**0.36**
10 – 14	24 / 46.73	**0.51****	8 / 15.00	**0.53**	3 / 3.55	**0.85**
15 – 19	90 / 87.16	**1.03**	35 / 29.78	**1.18**	9 / 7.56	**1.19**
20 – 24	48 / 60.34	**0.80**	18 / 21.53	**0.84**	5 / 5.41	**0.92**
25 +	12 / 15.28	**0.90**	3 / 5.68	**0.53**	0 / 1.31	**0.00**
TSFE (years)						
<10	20 / 20.00	**1.00**	4 / 4.81	**0.83**	3 / 1.00	**2.99**
10 – 14	18 / 27.60	**0.65**	4 / 7.96	**0.50**	0 / 1.78	**0.00**
15 – 19	35 / 52.23	**0.67***	16 / 16.40	**0.98**	4 / 3.98	**1.01**
20 – 24	84 / 84.40	**1.00**	31 / 28.80	**1.08**	6 / 7.30	**0.82**
25 – 29	68 / 76.47	**0.89**	24 / 27.82	**0.86**	9 / 6.98	**1.29**
30+	21 / 24.23	**0.87**	5 / 9.15	**0.55**	1 / 2.17	**0.46**

	Lymphohaematopoietic		Non-Hodgkin's lymphoma		Leukaemia	
	O/E	SMR	O/E	SMR	O/E	SMR
Total	5 / 8.85	**0.57**	3 / 3.98	**0.75**	2 / 3.0197	0.66
Age (years)						
< 25	0 / 0.03	**0.00**	0 / 0.01	**0.00**	0 / 0.02	0.00
25 – 34	0 / 0.33	**0.00**	0 / 0.11	**0.00**	0 / 0.17	0.00
35 – 44	0 / 0.72	**0.00**	0 / 0.33	**0.00**	0 / 0.31	0.00
45 – 54	1 / 1.96	**0.51**	1 / 0.98	**1.02**	0 / 0.56	0.00
55 – 64	3 / 3.43	**0.88**	1 / 1.53	**0.66**	2 / 1.18	1.70
65 +	1 / 2.38	**0.42**	1 / 1.03	**0.97**	0 / 0.78	0.00
Duration of employment (years)						
1 -4	0 / 1.08	**0.00**	0 / 0.47	**0.00**	0 / 0.39	0.00
5 – 9	0 / 1.20	**0.00**	0 / 0.53	**0.00**	0 / 0.44	0.00
10 – 14	1 / 1.44	**0.69**	1 / 0.65	**1.55**	0 / 0.50	0.00
15 – 19	2 / 2.66	**0.75**	1 / 1.18	**0.85**	1 / 0.90	1.11
20 – 24	1 / 1.92	**0.52**	1 / 0.89	**1.13**	0 / 0.62	0.00
25 +	1 / 0.56	**1.80**	0 / 0.25	**0.00**	1 / 0.17	5.98
TSFE (years)						
<10	0 / 0.57	**0.00**	0 / 0.23	**0.00**	0 / 0.24	0.00
10 – 14	0 / 0.83	**0.00**	0 / 0.36	**0.00**	0 / 0.31	0.00
15 – 19	0 / 1.55	**0.00**	0 / 0.68	**0.00**	0 / 0.55	0.00
20 – 24	1 / 2.53	**0.40**	1 / 1.14	**0.87**	0 / 0.86	0.00
25 – 29	3 / 2.45	**1.23**	2 / 1.14	**1.76**	1 / 0.76	1.31
30+	1 / 0.93	**1.07**	0 / 1.43	**0.00**	1 / 0.29	3.41

Table 5.5.2 **Mortality from selected causes according to age, duration of employment and time since first employed in the Company Cohort.**

	All Causes		All cancer		Lung Cancer	
	O/E	SMR	O/E	SMR	O/E	SMR
Total	227 / 203.58	**1.12**	69 / 61.38	**1.12**	23 / 12.87	**1.79***
Age (years)						
< 25	9 / 9.78	**0.92**	0 / 0.53	**0.00**	0 / 0.01	**0.00**
25 – 34	19 / 25.90	**0.73**	2 / 3.18	**0.63**	0 / 0.06	**0.00**
35 – 44	37 / 30.69	**1.21**	7 / 6.80	**1.03**	2 / 0.65	**3.10**
45 – 54	41 / 40.70	**1.01**	12 / 14.30	**0.84**	4 / 2.66	**1.50**
55 – 64	72 / 58.03	**1.24**	34 / 22.61	**1.50**	11 / 5.84	**1.88**
65 +	49 / 38.49	**1.27**	14 / 13.97	**1.00**	6 / 3.66	**1.64**
Duration of employment (years)						
1 -4	63 / 69.00	**0.91**	14 / 16.89	**0.83**	3 / 2.82	**1.07**
5 – 9	50 / 44.23	**1.13**	16 / 13.37	**1.20**	4 / 2.77	**1.44**
10 – 14	46 / 33.63	**1.37***	12 / 11.09	**1.08**	6 / 2.43	**2.47**
15 – 19	31 / 22.61	**1.37**	11 / 7.71	**1.43**	3 / 1.77	**1.69**
20 – 24	15 / 15.71	**0.95**	3 / 5.51	**0.54**	1 / 1.33	**0.75**
25 +	22 / 18.39	**1.20**	13 / 6.81	**1.91**	6 / 1.75	**3.43****
TSFE (years)						
<10	83 / 79.51	**1.04**	22 / 19.42	**1.13**	4 / 3.39	**1.18**
10 – 14	46 / 46.01	**1.00**	12 / 14.48	**0.83**	5 / 2.97	**1.68**
15 – 19	42 / 32.10	**1.31**	12 / 10.90	**1.10**	5 / 2.41	**2.07**
20 – 24	24 / 18.20	**1.32**	6 / 6.36	**0.94**	1 / 1.50	**0.6**
25 – 29	20 / 14.48	**1.38**	11 / 5.30	**2.08***	6 / 1.33	**4.52****
30+	12 / 13.27	**0.90**	6 / 4.93	**1.22**	2 / 1.27	**1.58**

	Lymphohaematopoietic		Non-Hodgkin's lymphoma		Leukaemia	
	O/E	**SMR**	**O/E**	**SMR**	**O/E**	**SMR**
Total	6 / 6.26	**0.96**	4 / 2.75	**1.45**	2 / 2.34	**0.86**
Age (years)						
< 25	0 / 0.18	**0.00**	0 / 0.04	**0.00**	0 / 0.14	**0.00**
25 – 34	0 / 0.81	**0.00**	0 / 0.27	**0.00**	0 / 0.44	**0.00**
35 – 44	1 / 0.84	**1.19**	0 / 0.40	**0.00**	1 / 0.35	**2.85**
45 – 54	0 / 1.43	**0.00**	0 / 0.72	**0.00**	0 / 0.40	**0.00**
55 – 64	4 / 1.83	**2.19**	3 / 0.82	**3.65***	1 / 0.62	**1.61**
65 +	1 / 1.18	**0.85**	1 / 0.51	**1.97**	0 / 0.39	**0.00**
Duration of employment (years)						
1 -4	2 / 2.07	**0.97**	2 / 0.87	**2.30**	0 / 0.87	**0.00**
5 – 9	1 / 1.35	**0.74**	1 / 0.59	**1.70**	0 / 0.51	**0.00**
10 – 14	1 / 1.05	**0.95**	0 / 0.47	**0.00**	1 / 0.37	**2.71**
15 – 19	1 / 0.70	**1.42**	0 / 0.32	**0.00**	1 / 0.24	**4.23**
20 – 24	1 / 0.50	**2.02**	1 / 0.23	**4.39**	0 / 0.16	**0.00**
25 +	0 / 0.60	**0.00**	0 / 0.28	**0.00**	0 / 0.19	**0.00**
TSFE (years)						
<10	2 / 2.32	**0.86**	2 / 0.94	**2.14**	0 / 1.00	**0.00**
10 – 14	1 / 1.44	**0.69**	0 / 0.66	**0.00**	1 / 0.52	**1.94**
15 – 19	2 / 1.03	**1.95**	1 / 0.48	**2.07**	1 / 0.34	**2.95**
20 – 24	0 / 0.57	**0.00**	0 / 0.26	**0.00**	0 / 0.19	**0.00**
25 – 29	1 / 0.46	**2.15**	1 / 0.21	**4.66**	0 / 0.15	**0.00**
30+	0 / 0.43	**0.00**	0 / 0.20	**0.00**	0 / 0.14	**0.00**

occurring in those with both the longest duration of employment and time since first employed. Only six deaths from cancers of lymphatic and haematopoietic tissue were observed in this cohort, and again there was no consistent association between mortality rates and duration of employment or time since first employed.

5.6 Distribution of the study population by potential exposures

The distribution of the study population by the department "ever worked" in, and by potential biological and chemical exposures (based on tables 4.2 and 4.3 in Chapter 4), is shown in Table 5.6 below.

The majority of the study participants had worked in the slaughterboard at some time: 62% of the Union Cohort, and 44% of the Company Cohort. As noted in chapter 4, most jobs on the slaughterboard entail significant potential for contact with freshly slaughtered meat, and animal urine, blood and faecal matter. A further 30% of the Union Cohort, and 10% of the Company Cohort, had worked in departments associated with the processing of meat wastes and offal in which the potential for similar exposures is present. The largest remaining category was the 20% of the Union Cohort, and 28% of the Company Cohort, who had worked as meat cutters or boners – work which entails a relatively reduced potential for the biological exposures.

Relatively few of the workers studied had ever worked in the stockyards (with the potential for live animal contact), namely 15% of those in the Union Cohort and only 4% of those in the Company Cohort. Over 25% of the Union Cohort had worked in

Table 5.6 **Distribution of the study population by department ever worked in and by potential biological and chemical exposures**

Exposure	Union Cohort		Company Cohort	
	N (%)	Person-years (%)	N (%)	Person-years (%)
Total	3,916 (100)	47,651 (100)	6,426 (100)	63,160 (100)
Department				
Stockyard	571 (15)	6,933 (15)	250 (4)	2,611 (4)
Slaughterboard	2,441 (62)	29,316 (62)	2,802 (44)	28,069 (44)
Cooling floor	373 (10)	4,477 (9)	153 (2)	1,395 (2)
Meat cutting	778 (20)	9,098 (19)	1,785 (28)	15,682 (25)
Freezers	493 (13)	5,934 (12)	450 (7)	4,697 (7)
Meat processing	1,181 (30)	14,179 (30)	665 (10)	6,393 (10)
Fellmongery	598 (15)	7,145 (15)	219 (3)	2,217 (4)
Plant services	143 (4)	1,554 (3)	939 (15)	8,778 (14)
Admin/management	11 (0)	143 (0)	362 (6)	3,481 (6)
Biological exposures				
Live animal contact	571 (15)	6,933 (15)	250 (4)	2,611 (4)
Pelts or hides	1,116 (28)	13,409 (28)	558 (9)	5,360 (8)
Slaughter/handling freshly slaughtered meat	3,342 (85)	40,617 (85)	4,887 (76)	47,533 (75)
Urine	3,362 (86)	40,786 (86)	5,246 (82)	50,901 (81)
Gastrointestinal microflora and faeces	3,139 (80)	37,811 (79)	4,115 (64)	40,699 (64)
Blood	2,940 (75)	35,473 (74)	4,890 (76)	47,130 (75)
Chemical exposures				
Welding emissions	0 (0)	0 (0)	46 (1)	450 (1)
Refrigerant gases	777 (20)	9,450 (20)	568 (9)	5,709 (9)
Cleaning chemicals	2,924 (75)	35,308 (74)	4,598 (72)	44,471 (70)
Animal remedies	3,263 (83)	39,539 (83)	4,982 (78)	48,062 (76)
Hormone growth promoters	3,008 (77)	36,322 (76)	3,571 (56)	35,330 (56)
Organochlorines	3,396 (87)	41,153 (86)	5,137 (80)	49,859 (79)

jobs with potential for contact with animal pelts and hides, i.e. either on live animals in the stockyards or in the fellmongery, while only 9% of members of the Company Cohort had worked in these settings. By contrast, most members (64 to 86%) of both cohorts had worked in jobs with a high potential for exposures to freshly slaughtered meat, and animal urine, blood and faecal matter.

Altogether 15% of the Company Cohort, and 4% of the Union Cohort, had ever been employed in plant maintenance departments. Of these, very few had potential chemical exposures. For example, less than 1% of the Company Cohort, and no one in the Union Cohort, had worked in jobs with potential exposure to welding emissions. Largely due to the high proportion of workers engaged in slaughterboard or meat cutting and boning activities, the majority of the study population had the potential for exposure (albeit at low levels) to chemical residues such as organochlorines, animal remedies and to cleaning chemicals. As the hormone growth promoters had been used in beef cattle only, and as the majority of the works covered in this study processed sheep, this exposure was rare.

The major differences between the two cohorts relate to the absence of administration/management personnel and the limited number of maintenance workers in the Union Cohort, as these personnel would be either not unionised or members of other unions. The Union Cohort also contained a significantly higher proportion of workers in fellmongeries, and in meat processing and cool store or freezer operations, through the union's coverage of workers in stand alone further processing operations not attached to large meat works. Compared with the company sub-cohorts, a larger proportion of the members of the Union Cohort had a work history that included working in more than one department by virtue of their having been followed on the basis of their union membership as they changed employer.

5.7 Mortality by department "ever-worked" in

The results of a series of sub-cohort analyses, for both the Union and Company Cohorts, of mortality by department "ever-worked" in are presented in Tables 5.7.1 and 5.7.2 below.

5.7.1 Mortality by department – Union Cohort

Table 5.7.1 shows mortality by department "ever worked" in for the Union Cohort. Among the 571 individuals who had ever worked in the stockyards, there was a significant deficit in mortality from all causes (SMR 0.56, 95% CI 0.38 – 0.81, 29 deaths), and a comparable deficit in both cancer deaths (SMR 0.55, 95% CI 0.26 – 1.01, 10 deaths) and mortality from diseases of the circulatory system (SMR 0.59, 12 deaths). There was no significant excess mortality from any specific disease type, and the diseases of *a priori* interest in this study all showed a deficit.

The 2,441 workers who had ever worked on the slaughterboard experienced mortality at close to expected rates, i.e. mortality from all causes (SMR 0.92, 161 deaths), all cancers (SMR 0.95, 55 deaths) and from diseases of the circulatory system (SMR 0.91, 61 deaths). Elevated mortality was observed for cancer of the pancreas (SMR 1.72, 4 deaths) and kidney (SMR 1.84, 3 deaths), and for malignant melanoma of the skin (SMR 1.63, 4 deaths) as well as for the non-malignant respiratory disease category bronchitis, emphysema and asthma (SMR 2.11, 6 deaths), although in all cases the increases were non-significant. In spite of the elevated mortality from

bronchitis, emphysema and asthma, there was no strong corresponding increase in lung cancer mortality (SMR 1.13, 16 deaths).

The cooling floor category only included 373 individuals. All cause mortality was below expected (SMR 0.91, 28 deaths) in this group, although this included a higher than expected (albeit non-significant) number of cancer deaths (SMR 1.41, 14 deaths). Mortality from diseases of the circulatory system was below expectation (SMR 0.84, 10 deaths). Elevated, but non-significant, excesses were observed for both prostate (SMR 3.05) and kidney cancer (SMR 6.76) both with 2 deaths.

The 778 individuals who had ever worked in meat cutting departments experienced a significant deficit in all cause mortality (SMR 0.62, 95% CI 0.41 – 0.91, 26 deaths), and also a substantial deficit in cancer deaths (SMR 0.60, 8 deaths). Non-significant elevations were observed for cancers of the kidney (SMR 5.15) and brain (SMR 3.19), although in both instances this was based on only 2 deaths. There was also a deficit in lung cancer mortality (SMR 0.66, 2 deaths), and no cases of cancers of lymphatic and haematopoietic tissue.

A significant deficit in all cause mortality (SMR 0.59, 95% CI 0.38 – 0.88, 24 deaths) was also experienced by the 493 individuals who had ever worked in freezers. Cancer deaths were similarly reduced (SMR 0.58, 8 deaths), as was mortality from diseases of the circulatory system (SMR 0.76, 12 deaths). Excess risk was observed for mortality from bronchitis, emphysema and asthma (SMR 2.98, 2 deaths), but there was no corresponding excess of lung cancer (SMR 0.87, 3 deaths) observed. There were no deaths from lymphohaematopoietic cancers.

Table 5.7.1 Mortality by Department ever worked in – Union Cohort

Cause of death (ICD 9th revision)	Stockyards			Slaughterboard			Cooling floor		
	O/E	SMR	95% CI	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	29 / 51.74	**0.56****	0.38 – 0.81	161 / 174.51	**0.92**	0.79 – 1.08	28 / 30.79	**0.91**	0.60 – 1.32
ALL MALIGNANT NEOPLASMS (140-208)	10 / 18.14	**0.55**	0.26 – 1.01	55 / 57.76	**0.95**	0.72 – 1.24	15 / 10.60	**1.41**	0.79 – 2.33
Oesophagus (150)	0 / 0.63	**0.00**	0.00 – 5.85	2 / 2.00	**1.00**	0.12 – 3.62	1 / 0.37	**2.71**	0.07 – 15.05
Stomach (151)	1 / 0.82	**1.23**	0.03 – 6.81	1 / 2.69	**0.37**	0.01 – 2.07	1 / 0.48	**2.09**	0.05 – 11.59
Colon (153)	0/ 1.84	**0.00**	0.00 – 2.01	3 / 5.72	**0.52**	0.11 – 1.53	1 / 1.08	**0.93**	0.02 – 5.17
Rectum (154)	1 / 1.24	**0.81**	0.02 – 4.48	4 / 3.89	**1.03**	0.28 – 2.63	1 / 0.72	**1.40**	0.04 – 7.77
Pancreas (157)	1 / 0.72	**1.38**	0.04 – 7.68	4 / 2.32	**1.72**	0.47 – 4.41	1 / 0.42	**2.36**	0.06 – 13.13
Lung (162)	1 / 4.55	**0.22**	0.01 – 1.22	16 / 14.10	**1.13**	0.65 – 1.84	3 / 2.65	**1.13**	0.23 – 3.31
Melanoma (172)	1 / 0.71	**1.41**	0.04 – 7.85	4 / 2.46	**1.63**	0.44 – 4.16	0 / 0.42	**0.00**	0.00 – 8.81
Prostate (185)	2 / 1.12	**1.79**	0.22 – 6.47	3 / 3.57	**0.84**	0.17 – 2.46	2 / 0.66	**3.05**	0.37 – 11.02
Bladder (188)	0 / 0.32	**0.00**	0.00 – 11.60	1 / 1.01	**0.99**	0.03 – 5.50	1 / 0.18	**5.42**	0.14 – 30.13
Kidney (189)	1 / 0.51	**1.98**	0.05 – 10.97	3 / 1.63	**1.84**	0.38 – 5.37	2 / 0.30	**6.76**	0.82 – 24.40
Brain (191)	0 / 0.72	**0.00**	0.00 – 5.16	2 / 2.45	**0.82**	0.10 – 2.95	0 / 0.42	**0.00**	0.0 – 8.72
Ill defined (195, 199)	0 / 1.10	**0.00**	0.00 – 2.24	5 / 3.45	**1.49**	0.57 – 3.27	1 / 0.65	**1.54**	0.14 – 7.20
Lymphatic and haematopoietic tissue (200-208)	1 / 1.62	**0.62**	0.02 – 3.43	1 / 5.41	**0.18**	0.01 – 1.03	0 / 0.96	**0.00**	0.00 – 3.86
Non-Hodgkin's lymphoma (200, 202)	1 / 0.73	**1.37**	0.12 – 6.38	1 / 2.43	**0.41**	0.04 – 1.92	0 / 0.43	**0.00**	0.00 – 5.68
Leukaemia and aleukaemia (204-208)	0 / 0.55	**0.00**	0.00 – 4.52	0 / 1.85	**0.00**	0.00 – 1.33	0 / 0.32	**0.00**	0.00 – 7.70
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	0 / 2.26	**0.00**	0.00 – 1.09	5 / 7.75	**0.65**	0.25 – 1.41	0 / 1.34	**0.00**	0.00 – 1.84
MENTAL DISORDERS (290-319)	0 / 0.30	**0.00**	0.00 – 12.35	0 / 1.21	**0.00**	0.00 – 3.06	0 / 0.19	**0.00**	0.00 – 19.47
DIS. OF CIRCULATORY SYSTEM (390-459)	12 / 20.34	**0.59**	0.31 – 1.03	61 / 66.75	**0.91**	0.70 – 1.17	10 / 11.95	**0.84**	0.40 – 1.54
DIS. OF RESPIRATORY SYSTEM (460-519)	1 / 2.89	**0.35**	0.01 – 1.92	7 / 9.40	**0.74**	0.30 – 1.54	1 / 1.70	**0.59**	0.02 – 3.26
Bronchitis, emphysema and asthma (490-493)	1 / 0.87	**1.15**	0.03 – 6.40	6 / 2.85	**2.11**	0.77 – 4.59	1 / 0.52	**1.93**	0.05 – 10.71
DIS. OF DIGESTIVE SYSTEM (520-579)	0 / 1.55	**0.00**	0.00 – 2.38	3 / 3.96	**0.76**	0.21 – 2.02	1 / 0.71	**1.41**	0.04 – 7.86
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	0 / 0.07	**0.00**	0.00 – 50.29	2 / 0.27	**7.36**	0.89 – 26.59	0 / 0.05	**0.00**	0.00 – 81.90
EXTERNAL CAUSES (E800-999)	6 / 4.55	**1.32**	0.48 – 2.87	28 / 20.45	**1.37**	0.91 – 1.98	1 / 3.05	**0.33**	0.01 – 1.82

* $p < 0.05$
** $p < 0.01$

Cause of death (ICD 9th revision)	Meat Cutting			Freezers			Processing		
	O/E	SMR	95% CI	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	26 / 41.68	**0.62***	0.41 – 0.91	24 / 40.52	**0.59****	0.38 – 0.88	76 / 91.05	**0.83**	0.66 – 1.05
ALL MALIGNANT NEOPLASMS (140-208)	8 / 13.23	**0.60**	0.26 – 1.19	8 / 13.90	**0.58**	0.25 – 1.13	28 / 31.05	**0.90**	0.60 – 1.30
Oesophagus (150)	1 / 0.44	**2.27**	0.06 – 12.62	0 / 0.49	**0.00**	0.00 – 7.56	1 / 1.07	**0.94**	0.02 – 5.21
Stomach (151)	1 / 0.63	**1.60**	0.04 – 8.87	1 / 0.64	**1.56**	0.04 – 8.67	2 / 1.42	**1.41**	0.17 – 5.08
Colon (153)	0/ 1.28	**0.00**	0.00 – 2.88	0 / 1.40	**0.00**	0.00 – 2.65	2 / 3.11	**0.64**	0.08 – 2.33
Rectum (154)	0 / 0.88	**0.00**	0.00 – 4.22	1 / 0.94	**1.06**	0.03 – 5.89	2 / 2.09	**0.96**	0.12 – 3.45
Pancreas (157)	0 / 0.53	**0.00**	0.00 – 7.00	0 / 0.56	**0.00**	0.00 – 6.60	2 / 1.24	**1.61**	0.20 – 5.81
Lung (162)	2 / 3.05	**0.66**	0.08 – 2.37	3 / 3.47	**0.87**	0.18 – 2.53	8 / 7.63	**1.05**	0.45 – 2.07
Melanoma (172)	0 / 0.64	**0.00**	0.00 – 5.79	0 / 0.57	**0.00**	0.00 – 6.49	1 / 1.28	**0.78**	0.02 – 4.35
Prostate (185)	0 / 0.71	**0.00**	0.00 – 5.17	0 / 0.86	**0.00**	0.00 – 4.31	0 / 1.88	**0.00**	0.00 – 1.97
Bladder (188)	0 / 0.21	**0.00**	0.00 – 17.58	0 / 0.24	**0.00**	0.00 – 15.34	0 / 0.54	**0.00**	0.00 – 6.88
Kidney (189)	2 / 0.39	**5.15**	0.62 – 18.61	1 / 0.40	**2.53**	0.06 – 14.06	3 / 0.87	**3.44**	0.71 – 10.07
Brain (191)	2 / 0.63	**3.19**	0.39 – 11.51	1 / 0.57	**1.77**	0.05 – 9.81	1 / 1.28	**0.78**	0.02 – 4.34
Ill defined (195, 199)	0 / 0.77	**0.00**	0.00 – 3.19	1 / 0.843	**1.19**	0.11 – 5.53	3 / 1.87	**1.61**	0.45 – 4.29
Lymphatic and haematopoietic tissue (200-208)	0 / 1.32	**0.00**	0.00 – 2.80	0 / 1.27	**0.00**	0.00 – 2.91	2 / 2.84	**0.71**	0.09 – 2.55
Non-Hodgkin's lymphoma (200, 202)	0 / 0.60	**0.00**	0.00 – 4.10	0 / 0.58	**0.00**	0.00 – 4.28	1 / 1.28	**0.78**	0.07 – 3.63
Leukaemia and aleukaemia (204-208)	0 / 0.45	**0.00**	0.00 – 5.51	0 / 0.42	**0.00**	0.00 – 5.85	1 / 0.96	**1.05**	0.10 – 4.87
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	0 / 1.97	**0.00**	0.00 – 1.25	0 / 1.79	**0.00**	0.00 – 1.38	2 / 4.04	**0.50**	0.10 – 1.59
MENTAL DISORDERS (290-319)	1 / 0.33	**3.08**	0.08 – 17.10	0 / 0.25	**0.00**	0.00 – 15.09	0 / 0.58	**0.00**	0.00 – 6.35
DIS. OF CIRCULATORY SYSTEM (390-459)	9 / 15.02	**0.60**	0.27 – 1.14	12 / 15.87	**0.76**	0.39 – 1.32	35 / 35.22	**0.99**	0.69 – 1.38
DIS. OF RESPIRATORY SYSTEM (460-519)	0 / 1.99	**0.00**	0.00 – 1.86	3 / 2.22	**1.35**	0.28 – 3.96	4 / 4.96	**0.81**	0.22 – 2.06
Bronchitis, emphysema and asthma (490-493)	0 / 0.64	**0.00**	0.00 – 5.80	2 / 0.67	**2.98**	0.36 – 10.76	3 / 1.50	**1.99**	0.41 – 5.83
DIS. OF DIGESTIVE SYSTEM (520-579)	1 / 0.91	**1.10**	0.10 – 5.13	0 / 0.94	**0.00**	0.00 – 2.63	2 / 2.10	**0.95**	0.19 – 3.05
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	0 / 0.08	**0.00**	0.00 – 49.43	0 / 0.06	**0.00**	0.00 – 62.40	0 / 0.14	**0.00**	0.00 – 26.85
EXTERNAL CAUSES (E800-999)	7 / 6.40	**1.09**	0.44 – 2.25	1 / 3.93	**0.25**	0.01 – 1.41	4 / 9.39	**0.43**	0.12 – 1.09

* $p < 0.05$
** $p < 0.01$

Cause of death (ICD 9th revision)	Fellmongery			Plant Services		
	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	40 / 45.75	**0.87**	0.63 – 1.19	7 / 11.25	**0.62**	0.25 – 1.28
ALL MALIGNANT NEOPLASMS (140-208)	14 / 15.52	**0.90**	0.49 – 1.51	3 / 3.93	**0.76**	0.16 – 2.23
Oesophagus (150)	1 / 0.54	**1.87**	0.05 – 10.36	0 / 0.14	**0.00**	0.00 – 27.02
Stomach (151)	0 / 0.72	**0.00**	0.00 – 5.16	0 / 0.18	**0.00**	0.00 – 20.92
Colon (153)	1 / 1.56	**0.64**	0.02 – 3.55	0 / 0.39	**0.00**	0.00 – 9.43
Rectum (154)	2 / 1.05	**1.90**	0.23 – 6.88	0 / 0.27	**0.00**	0.00 – 13.89
Pancreas (157)	0 / 0.62	**0.00**	0.00 – 5.92	0 / 0.16	**0.00**	0.00 – 23.48
Lung (162)	2 / 3.84	**0.52**	0.06 – 1.88	1 / 0.98	**1.02**	0.03 – 5.69
Melanoma (172)	1 / 0.64	**1.56**	0.04 – 8.66	1 / 0.16	**6.42**	0.16 – 35.65
Prostate (185)	1 / 0.93	**1.07**	0.03 – 5.92	0 / 0.25	**0.00**	0.00 – 14.90
Bladder (188)	1 / 0.27	**3.76**	0.10 – 20.86	0 / 0.07	**0.00**	0.00 – 52.60
Kidney (189)	3 / 0.44	**6.81***	1.40 – 19.91	1 / 0.11	**9.12**	0.23 – 50.67
Brain (191)	0 / 0.65	**0.00**	0.00 – 5.71	0 / 0.15	**0.00**	0.00 – 24.26
Ill defined (195, 199)	0 / 0.94	**0.00**	0.00 – 2.62	0 / 0.24	**0.00**	0.00 – 10.29
Lymphatic and haematopoietic tissue (200-208)	2 / 1.43	**1.40**	0.17 – 5.05	0 / 0.35	**0.00**	0.00 – 10.48
Non-Hodgkin's lymphoma (200, 202)	1 / 0.65	**1.53**	0.14 – 7.14	0 / 0.16	**0.00**	0.00 – 15.57
Leukaemia and aleukaemia (204-208)	1 / 0.48	**2.09**	0.19 – 9.75	0 / 0.12	**0.00**	0.00 – 20.90
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	2 / 2.05	**0.98**	0.20 – 3.13	0 / 0.50	**0.00**	0.00 – 4.98
MENTAL DISORDERS (290-319)	1 / 0.29	**3.47**	0.09 – 19.30	0 / 0.07	**0.00**	0.00 – 53.39
DIS. OF CIRCULATORY SYSTEM (390-459)	12 / 17.71	**0.68**	0.35 – 1.18	2 / 4.40	**0.45**	0.06 – 1.64
DIS. OF RESPIRATORY SYSTEM (460-519)	2 / 2.44	**0.82**	0.10 – 2.96	0 / 0.64	**0.00**	0.00 – 5.76
Bronchitis, emphysema and asthma (490-493)	2 / 0.75	**2.66**	0.32 – 9.61	0 / 0.19	**0.00**	0.00 – 19.37
DIS. OF DIGESTIVE SYSTEM (520-579)	1 / 1.05	**0.95**	0.09 – 4.44	0 / 0.26	**0.00**	0.00 – 9.51
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	1 / 0.07	**14.30**	0.36 – 79.43	0 / 0.02	**0.00**	0.00 – 229.2
EXTERNAL CAUSES (E800-999)	7 / 4.83	**1.45**	0.58 – 2.99	2 / 1.00	**2.00**	0.24 – 7.23

* $p < 0.05$
** $p < 0.01$

Mortality from all causes (SMR 0.83, 76 deaths), all cancers (SMR 0.90, 28 deaths) and diseases of the circulatory system (SMR 0.99, 35 deaths) were below expectation in the 1,181 individuals who had ever worked in meat processing. Non-significant excesses for malignancies of the stomach (SMR 1.41, 2 deaths), pancreas (SMR 1.61, 2 deaths), kidney (SMR 3.44, 3 deaths) and other ill-defined or unspecified sites (SMR 1.61, 3 deaths) were observed in this group, and also for the non-malignant respiratory disease category bronchitis, emphysema and asthma (SMR 1.99, 3 deaths). Again there was no corresponding increase in lung cancer (SMR 1.05, 8 deaths). There was one death each from non-Hodgkin's lymphoma and leukaemia, but the overall mortality from cancers of lymphohaematopoietic tissue was below expectation (SMR 0.71).

The 598 individuals who had worked in fellmongeries had experienced no increase in all cause mortality (SMR 0.87, 40 deaths), or from the major disease categories all cancers (SMR 0.90, 14 deaths) or circulatory system disease (SMR 0.68, 12 deaths), although an excess risk of mortality from external causes (SMR 1.45, 7 deaths) was observed. Of the specific cancer types a significant excess in mortality from cancer of the kidney (SMR 6.81, 3 deaths) was observed, as well as non-significant excesses for cancer of the rectum (SMR 1.90, 2 deaths) and of lymphohaematopoietic tissue (SMR 1.40) based on one death each from non-Hodgkin's lymphoma and leukaemia. Bronchitis, emphysema and asthma deaths exceeded expectation (SMR 2.66, 2 deaths), but there was no corresponding excess of lung cancer (SMR 0.52, 2 deaths).

Only 7 of the small group (143 individuals) who had worked in plant services had died from any cause, and there was no excess mortality from any disease category apart from 2 deaths from external causes (SMR 2.0).

5.7.2 Mortality by department – Company Cohort

Table 5.7.2 shows the corresponding findings for the Company Cohort. Only 250 members of the Company Cohort had worked in stockyards, of whom 10 had died (SMR 0.87). Five of these deaths had been from cancer (SMR 1.42), of which 2 were from other ill defined or unspecified sites (SMR 9.89, 95% CI 1.97 – 31.71) and 1 was non-Hodgkin's lymphoma (SMR 6.29). The only other category with elevated risk was external causes (SMR 1.52, 3 deaths).

All cause mortality in the 2,802 individuals who had worked on the slaughterboard was significantly elevated (SMR 1.23, 95% CI 1.01 – 1.48, 113 deaths). Excess mortality was also observed for all cancers (SMR 1.23, 34 deaths), and for diseases of the circulatory system (SMR 1.36, 40 deaths), although these excesses were non-significant. A significant excess was observed for lung cancer (SMR 2.24, 13 cases), and non-significant excesses for cancers of the stomach (SMR 2.32, 3 deaths), colon (SMR 1.96, 5 deaths) and prostate (SMR 1.57, 2 deaths). The excess mortality from lung cancer was matched by an excess in bronchitis, emphysema and asthma (SMR 2.21, 3 deaths). Mortality from cancers of lymphohaematopoietic tissue were below expected (SMR 0.70, 2 deaths).

Table 5.7.2 Mortality by Department ever worked in – Company Cohort

Cause of death (ICD 9th revision)	Stockyards			Slaughterboard			Cooling floor		
	O/E	SMR	95% CI	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	10 / 11.49	**0.87**	0.42 – 1.60	113 / 92.04	**1.23***	1.01 – 1.48	6 / 3.67	**1.63**	0.60 – 3.56
ALL MALIGNANT NEOPLASMS (140-208)	5 / 3.53	**1.42**	0.46 – 3.31	34 / 27.58	**1.23**	0.85 – 1.72	3 / 1.18	**2.55**	0.53 – 7.46
Oral cavity (141-145)	1 / 0.04	**24.84**	0.63 – 138.0	1 / 0.30	**3.37**	0.09 – 18.73	0 / 0.01	**0.00**	0.00 – 322.5
Oesophagus (150)	0 / 0.12	**0.00**	0.00 – 31.25	1 / 0.81	**1.23**	0.03 – 6.86	0 / 0.03	**0.00**	0.00 – 140.3
Stomach (151)	0 / 0.17	**0.00**	0.00 – 21.84	3 / 1.29	**2.32**	0.48 – 6.78	0 / 0.05	**0.00**	0.00 – 75.58
Colon (153)	0 / 0.34	**0.00**	0.00 – 10.89	5 / 2.55	**1.96**	0.64 – 4.59	0 / 0.10	**0.00**	0.00 – 35.69
Pancreas (157)	0 / 0.14	**0.00**	0.00 – 26.06	0 / 1.04	**0.00**	0.00 – 3.54	0 / 0.04	**0.00**	0.00 – 92.31
Lung (162)	0 / 0.82	**0.00**	0.00 – 4.53	13 / 5.80	**2.24***	1.19 – 3.83	1 / 0.22	**4.58**	0.12 – 25.44
Melanoma (172)	0 / 0.17	**0.00**	0.00 – 21.96	1 / 1.39	**0.72**	0.02 – 4.00	1 / 0.06	**17.64**	0.45 – 97.98
Prostate (185)	0 / 0.20	**0.00**	0.00 – 18.66	2 / 1.28	**1.57**	0.19 – 5.66	0 / 0.03	**0.00**	0.00 – 112.6
Bladder (188)	0 / 0.06	**0.00**	0.00 – 62.66	1 / 0.40	**2.50**	0.06 – 13.91	1 / 0.01	**76.25***	1.93 – 423.6
Brain (191)	0 / 0.17	**0.00**	0.00 – 22.07	1 / 1.37	**0.73**	0.02 – 4.07	0 / 0.06	**0.00**	0.00 – 65.55
Thyroid (193)	0 / 0.01	**0.00**	0.00 – 519.2	0 / 0.06	**0.00**	0.00 – 64.92	0 / 0.01	**0.00**	0.00 – 1412
Ill defined (195, 199)	2 / 0.20	**9.89***	1.97 – 31.71	2 / 1.56	**1.29**	0.26 – 4.12	0 / 0.06	**0.00**	0.00 – 38.50
Lymphatic and haematopoietic tissue (200-208)	1 / 0.36	**2.79**	0.07 – 15.48	2 / 2.84	**0.70**	0.09 – 2.54	0 / 0.12	**0.00**	0.00 – 31.66
Non-Hodgkin's lymphoma (200, 202)	1 / 0.16	**6.29**	0.57 – 29.34	1 / 1.26	**0.79**	0.07 – 3.70	0 / 0.05	**0.00**	0.00 – 47.39
Leukaemia and aleukaemia (204-208)	0 / 0.13	**0.00**	0.00 – 19.12	1 / 1.04	**0.96**	0.09 – 4.47	0 / 0.04	**0.00**	0.00 – 55.87
BENIGN NEOPLASMS (210-239)	0 / 0.04	**0.00**	0.00 – 101.6	0 / 0.30	**0.00**	0.00 – 12.51	0 / 0.01	**0.00**	0.00 – 304.7
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	0 / 0.53	**0.00**	0.00 – 4.62	3 / 4.23	**0.71**	0.20 – 1.89	0 / 0.17	**0.00**	0.00 – 14.90
DIS. OF NERVOUS SYSTEM (320-359)	0 / 0.21	**0.00**	0.00 – 17.48	2 / 1.89	**1.06**	0.13 – 3.81	0 / 0.08	**0.00**	0.00 – 44.55
DIS. OF CIRCULATORY SYSTEM (390-459)	1 / 4.06	**0.25**	0.01 – 1.37	40 / 29.32	**1.36**	0.97 – 1.86	0 / 1.01	**0.00**	0.00 – 3.66
DIS. OF RESPIRATORY SYSTEM (460-519)	0 / 0.55	**0.00**	0.00 – 6.77	4 / 4.04	**0.99**	0.27 – 2.53	1 / 0.15	**6.76**	0.17 – 37.55
Bronchitis, emphysema and asthma (490-493)	0 / 0.17	**0.00**	0.00 – 21.41	3 / 1.36	**2.21**	0.46 – 6.45	1 / 0.05	**18.47**	0.47 – 102.6
DIS. OF DIGESTIVE SYSTEM (520-579)	0 / 0.24	**0.00**	0.00 – 15.10	2 / 1.83	**1.09**	0.13 – 3.95	0 / 0.07	**0.00**	0.00 – 53.19
EXTERNAL CAUSES (E800-999)	3 / 1.97	**1.52**	0.31 – 4.45	27 / 19.64	**1.37**	0.91 – 2.00	2 / 0.87	**2.30**	0.28 – 8.32

* $p < 0.05$
** $p < 0.01$

Cause of death (ICD 9th revision)	Meat Cutting			Freezers			Processing		
	O/E	SMR	95% CI	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	35 / 35.99	**0.97**	0.68 – 1.35	13 / 14.26	**0.91**	0.49 – 1.56	23 / 23.00	**1.00**	0.64 – 1.50
ALL MALIGNANT NEOPLASMS (140-208)	12 / 10.36	**1.16**	0.60 – 2.02	2 / 3.98	**0.50**	0.06 – 1.82	6 / 7.03	**0.85**	0.31 – 1.86
Oral cavity (141-145)	0 / 0.10	**0.00**	0.00 – 37.40	0 / 0.05	**0.00**	0.00 – 79.05	0 / 0.07	**0.00**	0.00 – 50.77
Oesophagus (150)	0 / 0.22	**0.00**	0.00 – 17.05	0 / 0.12	**0.00**	0.00 – 30.26	0 / 0.21	**0.00**	0.00 – 17.68
Stomach (151)	1 / 0.45	**2.23**	0.06 – 12.39	0 / 0.20	**0.00**	0.00 – 18.68	0 / 0.32	**0.00**	0.00 – 11.63
Colon (153)	2 / 0.89	**2.25**	0.27 – 8.12	0 / 0.37	**0.00**	0.00 – 9.92	0 / 0.65	**0.00**	0.00 – 5.67
Pancreas (157)	1 / 0.35	**2.89**	0.07 – 16.04	0 / 0.16	**0.00**	0.00 – 23.85	1 / 0.26	**3.80**	0.10 – 21.12
Lung (162)	2 / 1.75	**1.14**	0.14 – 4.13	1 / 0.84	**1.19**	0.03 – 6.63	4 / 1.52	**2.63**	0.72 – 6.72
Melanoma (172)	1 / 0.56	**1.77**	0.05 – 9.85	1 / 0.22	**4.52**	0.11 – 25.10	0 / 0.32	**0.00**	0.00 – 11.50
Prostate (185)	1 / 0.25	**4.05**	0.10 – 22.53	0 / 0.17	**0.00**	0.00 – 22.30	0 / 0.38	**0.00**	0.00 – 9.85
Bladder (188)	1 / 0.11	**9.35**	0.24 – 51.93	0 / 0.05	**0.00**	0.00 – 67.79	0 / 0.11	**0.00**	0.00 – 33.50
Brain (191)	0 / 0.56	**0.00**	0.00 – 6.62	0 / 0.22	**0.00**	0.00 – 16.74	0 / 0.32	**0.00**	0.00 – 11.55
Thyroid (193)	1 / 0.03	**38.18**	0.97 – 212.1	0 / 0.01	**0.00**	0.00 – 456.3	0 / 0.01	**0.00**	0.00 – 259.5
Ill defined (195, 199)	0 / 0.55	**0.00**	0.00 – 4.47	0 / 0.23	**0.00**	0.00 – 10.84	0 / 0.40	**0.00**	0.00 – 6.19
Lymphatic and haematopoietic tissue (200-208)	0 / 1.11	**0.00**	0.00 – 3.32	0 / 0.44	**0.00**	0.00 – 8.41	0 / 0.71	**0.00**	0.00 – 5.23
Non-Hodgkin's lymphoma (200, 202)	0 / 0.49	**0.00**	0.00 – 5.07	0 / 0.20	**0.00**	0.00 – 12.51	0 / 0.31	**0.00**	0.00 – 8.02
Leukaemia and aleukaemia (204-208)	0 / 0.44	**0.00**	0.00 – 5.61	0 / 0.16	**0.00**	0.00 – 15.18	0 / 0.26	**0.00**	0.00 – 9.33
BENIGN NEOPLASMS (210-239)	0 / 0.12	**0.00**	0.00 – 30.15	0 / 0.04	**0.00**	0.00 – 86.86	1 / 0.08	**13.19**	0.33 – 73.29
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	1 / 1.65	**0.61**	0.06 – 2.83	0 / 0.69	**0.00**	0.00 – 3.58	0 / 1.00	**0.00**	0.00 – 2.47
DIS. OF NERVOUS SYSTEM (320-359)	0 / 0.89	**0.00**	0.00 – 4.16	0 / 0.30	**0.00**	0.00 – 12.15	0 / 0.45	**0.00**	0.00 – 8.13
DIS. OF CIRCULATORY SYSTEM (390-459)	7 / 9.02	**0.78**	0.31 – 1.60	3 / 4.38	**0.68**	0.14 – 2.00	8 / 7.48	**1.07**	0.46 – 2.11
DIS. OF RESPIRATORY SYSTEM (460-519)	2 / 1.30	**1.54**	0.19 – 5.56	1 / 0.53	**1.89**	0.05 – 10.49	2 / 1.12	**1.78**	0.22 – 6.43
Bronchitis, emphysema and asthma (490-493)	2 / 0.49	**4.06**	0.49 – 14.66	1 / 0.19	**5.24**	0.13 – 29.09	2 / 0.36	**5.59**	0.68 – 20.18
DIS. OF DIGESTIVE SYSTEM (520-579)	1 / 0.63	**1.59**	0.14 – 7.40	1 / 0.27	**3.66**	0.09 – 20.36	1 / 0.46	**2.17**	0.06 – 12.07
EXTERNAL CAUSES (E800-999)	11 / 10.54	**1.04**	0.52 – 1.87	6 / 3.56	**1.69**	0.62 – 3.67	4 / 4.60	**0.87**	0.24 – 2.23

* $p < 0.05$

** $p < 0.01$

Cause of death (ICD 9th revision)	Fellmongery			Plant Services			Administration		
	O/E	SMR	95% CI	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	11 / 8.55	**1.29**	0.64 – 2.30	38 / 32.83	**1.16**	0.82 – 1.59	7 / 8.34	**0.84**	0.34 – 1.73
ALL MALIGNANT NEOPLASMS (140-208)	5 / 2.47	**2.02**	0.66 – 4.73	11 / 10.02	**1.10**	0.55 – 1.96	2 / 2.80	**0.71**	0.09 – 2.58
Oral cavity (141-145)	0 / 0.03	**0.00**	0.00 – 135.2	0 / 0.11	**0.00**	0.00 – 34.13	0 / 0.02	**0.00**	0.00 – 162.6
Oesophagus (150)	1 / 0.08	**12.13**	0.31 – 67.39	1 / 0.31	**3.19**	0.08 – 17.75	0 / 0.06	**0.00**	0.00 – 64.84
Stomach (151)	0 / 0.12	**0.00**	0.00 – 31.03	0 / 0.46	**0.00**	0.00 – 8.06	0 / 0.11	**0.00**	0.00 – 34.58
Colon (153)	0 / 0.23	**0.00**	0.00 – 15.92	0 / 0.96	**0.00**	0.00 – 3.83	0 / 0.25	**0.00**	0.00 – 14.85
Pancreas (157)	0 / 0.10	**0.00**	0.00 – 37.89	0 / 0.39	**0.00**	0.00 – 9.58	0 / 0.09	**0.00**	0.00 – 40.96
Lung (162)	1 / 0.56	**1.79**	0.05 – 9.92	5 / 2.27	**2.21**	0.71 – 5.16	0 / 0.51	**0.00**	0.00 – 7.19
Melanoma (172)	1 / 0.12	**8.24**	0.21 – 45.79	0 / 0.45	**0.00**	0.00 – 8.12	0 / 0.12	**0.00**	0.00 – 31.82
Prostate (185)	0 / 0.15	**0.00**	0.00 – 24.95	0 / 0.51	**0.00**	0.00 – 7.18	1 / 0.09	**10.81**	0.27 – 60.08
Bladder (188)	0 / 0.04	**0.00**	0.00 – 88.26	0 / 0.16	**0.00**	0.00 – 23.82	0 / 0.03	**0.00**	0.00 – 112.3
Brain (191)	0 / 0.12	**0.00**	0.00 – 30.97	0 / 0.46	**0.00**	0.00 – 8.02	1 / 0.12	**8.55**	0.22 – 47.52
Thyroid (193)	1 / 0.01	**216.94****	5.49 – 1205	0 / 0.02	**0.00**	0.00 – 184.6	0 / 0.01	**0.00**	0.00 – 520.1
Ill defined (195, 199)	0 / 0.14	**0.00**	0.00 – 17.28	1 / 0.58	**1.72**	0.16 – 8.01	0 / 0.16	**0.00**	0.00 – 15.67
Lymphatic and haematopoietic tissue (200-208)	0 / 0.26	**0.00**	0.00 – 14.35	3 / 1.00	**2.99**	0.62 – 8.75	0 / 0.26	**0.00**	0.00 – 14.13
Non-Hodgkin's lymphoma (200, 202)	0 / 0.11	**0.00**	0.00 – 22.10	2 / 0.44	**4.54**	0.91 – 14.56	0 / 0.11	**0.00**	0.00 – 22.61
Leukaemia and aleukaemia (204-208)	0 / 0.10	**0.00**	0.00 – 25.67	1 / 0.37	**2.70**	0.25 – 12.60	0 / 0.10	**0.00**	0.00 – 23.65
BENIGN NEOPLASMS (210-239)	0 / 0.03	**0.00**	0.00 – 136.8	1 / 0.10	**9.82**	0.25 – 54.55	0 / 0.03	**0.00**	0.00 – 126.5
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	0 / 0.38	**0.00**	0.00 – 6.52	0 / 1.44	**0.00**	0.00 – 1.71	0 / 0.34	**0.00**	0.00 – 7.25
DIS. OF NERVOUS SYSTEM (320-359)	1 / 0.17	**6.07**	0.15 – 33.73	0 / 0.64	**0.00**	0.00 – 5.80	0 / 0.19	**0.00**	0.00 – 19.74
DIS. OF CIRCULATORY SYSTEM (390-459)	4 / 2.87	**1.40**	0.38 – 3.57	10 / 10.72	**0.93**	0.45 – 1.72	4 / 2.29	**1.75**	0.48 – 4.47
DIS. OF RESPIRATORY SYSTEM (460-519)	0 / 0.41	**0.00**	0.00 – 9.05	1 / 1.52	**0.66**	0.02 – 3.66	0 / 0.42	**0.00**	0.00 – 8.87
Bronchitis, emphysema and asthma (490-493)	0 / 0.13	**0.00**	0.00 – 28.83	0 / 0.49	**0.00**	0.00 – 7.53	0 / 0.14	**0.00**	0.00 – 26.39
DIS. OF DIGESTIVE SYSTEM (520-579)	1 / 0.17	**5.83**	0.15 – 32.39	0 / 0.66	**0.00**	0.00 – 5.57	0 / 0.16	**0.00**	0.00 – 22.88
EXTERNAL CAUSES (E800-999)	0 / 1.77	**0.00**	0.00 – 2.08	14 / 6.63	**2.11***	1.15 – 3.54	1 / 1.77	**0.57**	0.01 – 3.15

* $p < 0.05$
** $p < 0.01$

Of the 153 individuals that had worked on the cooling floor, 6 had died (SMR 1.63). This included 3 cancer deaths (SMR 2.55), i.e. one each of lung, bladder and malignant melanoma of the skin, and also 2 deaths from external causes (SMR 2.30). All cause mortality in the 1,785 individuals who had worked in meat cutting was close to expectation (SMR 0.97, 35 deaths), as was cancer mortality (SMR 1.16, 12 deaths). There was a deficit in mortality from diseases of the circulatory system (SMR 0.78, 7 deaths). An excess in mortality from the disease category bronchitis, emphysema and asthma (SMR of 4.06, 2 deaths), was not matched by an excess of lung cancer (SMR 1.14, 2 deaths). There were no deaths from lymphohaematopoietic cancers among this group.

The 450 individuals who had ever worked in the freezers had experienced no excess mortality from all causes (SMR 0.91, 13 deaths), from cancers (SMR 0.50, 2 deaths), or from diseases of the circulatory system (SMR 0.68, 3 deaths). There was only 1 lung cancer death (SMR 1.19) and no cases of cancers of lymphohaematopoietic tissue. Mortality from all causes (SMR 1.00, 23 deaths), all cancers (SMR 0.85, 6 deaths) and from diseases of the circulatory system (SMR 1.07, 8 deaths) was also close to expected rates among the 665 individuals who had worked in meat processing departments. An excess of lung cancer (SMR 2.63, 4 deaths) was matched by an excess in the disease category bronchitis, emphysema and asthma (SMR 5.59, 2 deaths). The small group who had worked in the fellmongeries (N = 219) experienced non-significant excesses of mortality from all causes (SMR 1.29, 11 deaths), all cancers (SMR 2.02, 5 deaths) and from diseases of the circulatory system (SMR 1.40,

4 deaths). Small numbers precluded any meaningful examination of mortality from specific causes in this group.

The 939 individuals who had worked in plant services had experienced a marginal increase in mortality from all causes (SMR 1.16, 38 deaths) and all cancers (SMR 1.10, 11 deaths), and a significant excess in mortality from external causes (SMR 2.11, 95% CI 1.15 – 3.54) based on 14 deaths. Mortality from diseases of the circulatory system was close to expectation (SMR 0.93, 10 deaths). Of the diseases of *a priori* interest there were excesses for lung cancer (SMR 2.21, 5 deaths), for lymphohaematopoietic cancers (SMR 2.99, 3 deaths) and for non-Hodgkin's lymphoma (SMR 4.54, 2 deaths).

The very small group (N = 362) in the administration/management departments experienced low overall (SMR 0.84, 7 deaths) and cancer mortality (SMR 0.71, 2 deaths), but a non-significant excess in mortality from diseases of the circulatory system (SMR 1.75, 4 deaths).

5.8 Mortality by Biological Exposures

5.8.1 Mortality by Biological Exposures - Union Cohort.

Table 5.8.1 shows mortality by potential biological exposures in the Union Cohort. Of the potential biological exposures identified, the effect of contact with pelts or hides, slaughter or raw meat, and with animal urine, faeces and blood was examined. Live animal contact was excluded from this analysis, as the only group with this exposure

Table 5.8.1 Mortality by potential biological exposures – Union Cohort

Cause of death (ICD 9th revision)	Live animal contact			Pelts or hides			Raw meat		
	O/E	SMR	95% CI	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	29 / 51.74	**0.56****	0.38 – 0.81	66 / 91.79	**0.72****	0.56 – 0.92	183 / 205.37	**0.89**	0.77 – 1.03
ALL MALIGNANT NEOPLASMS (140-208)	10 / 18.14	**0.55**	0.26 – 1.01	24 / 31.62	**0.76**	0.49 – 1.13	64 / 67.80	**0.94**	0.73 – 1.21
Oesophagus (150)	0 / 0.63	**0.00**	0.00 – 5.85	1 / 1.09	**0.91**	0.02 – 5.08	3 / 2.34	**1.28**	0.27 – 3.75
Stomach (151)	1 / 0.82	**1.23**	0.03 – 6.81	1 / 1.44	**0.69**	0.02 – 3.86	2 / 3.15	**0.63**	0.08 – 2.29
Colon (153)	0 / 1.84	**0.00**	0.00 – 2.01	1 / 3.19	**0.31**	0.01 – 1.74	3 / 6.71	**0.45**	0.09 – 1.31
Rectum (154)	1 / 1.24	**0.81**	0.02 – 4.48	3 / 2.15	**1.40**	0.29 – 4.08	5 / 4.56	**1.10**	0.36 – 2.56
Pancreas (157)	1 / 0.72	**1.38**	0.04 – 7.68	1 / 1.27	**0.79**	0.02 – 4.39	4 / 2.72	**1.47**	0.40 – 3.76
Lung (162)	1 / 4.55	**0.22**	0.01 – 1.22	3 / 7.86	**0.38**	0.08 – 1.12	18 / 16.51	**1.09**	0.65 – 1.72
Melanoma (172)	1 / 0.71	**1.41**	0.04 – 7.85	2 / 1.27	**1.57**	0.19 – 5.67	4 / 2.91	**1.38**	0.38 – 3.52
Prostate (185)	2 / 1.12	**1.79**	0.22 – 6.47	3 / 1.92	**1.56**	0.32 – 4.56	4 / 4.15	**0.96**	0.26 – 2.46
Bladder (188)	0 / 0.32	**0.00**	0.00 – 11.60	1 / 0.55	**1.82**	0.05 – 10.13	2 / 1.18	**1.70**	0.21 – 6.14
Kidney (189)	1 / 0.51	**1.98**	0.05 – 10.97	4 / 0.89	**4.49***	1.22 – 11.48	4 / 1.92	**2.08**	0.57 – 5.33
Brain (191)	0 / 0.72	**0.00**	0.00 – 5.16	0 / 1.29	**0.00**	0.00 – 2.87	3 / 2.88	**1.04**	0.22 – 3.04
Ill defined (195, 199)	0 / 1.10	**0.00**	0.00 – 2.24	0 / 1.91	**0.00**	0.00 – 1.29	5 / 4.05	**1.23**	0.47 – 2.71
Lymphatic and haematopoietic tissue (200-208)	1 / 1.62	**0.62**	0.02 – 3.43	3 / 2.87	**1.04**	0.22 – 3.05	1 / 6.37	**0.16***	0.01 – 0.87
Non-Hodgkin's lymphoma (200, 202)	1 / 0.73	**1.37**	0.12 – 6.38	2 / 1.30	**1.54**	0.31 – 4.92	1 / 2.87	**0.35**	0.03 – 1.63
Leukaemia and aleukaemia (204-208)	0 / 0.55	**0.00**	0.00 – 4.52	1 / 0.97	**1.04**	0.09 – 4.83	0 / 2.18	**0.00**	0.00 – 1.13
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	0 / 2.26	**0.00**	0.00 – 1.09	2 / 4.07	**0.49**	0.10 – 1.58	5 / 9.13	**0.55**	0.21 – 1.20
MENTAL DISORDERS (290-319)	0 / 0.30	**0.00**	0.00 – 12.35	1 / 0.56	**1.80**	0.05 – 9.99	1 / 1.43	**0.70**	0.02 – 3.88
DIS. OF CIRCULATORY SYSTEM (390-459)	12 / 20.34	**0.59**	0.31 – 1.03	23 / 35.78	**0.64***	0.41 – 0.97	69 / 78.14	**0.88**	0.69 – 1.12
DIS. OF RESPIRATORY SYSTEM (460-519)	1 / 2.89	**0.35**	0.01 – 1.92	3 / 5.01	**0.60**	0.12 – 1.75	8 / 11.00	**0.73**	0.34 – 1.37
Bronchitis, emphysema and asthma (490-493)	1 / 0.87	**1.15**	0.03 – 6.40	3 / 1.52	**1.97**	0.41 – 5.76	7 / 3.34	**2.10**	0.84 – 4.32
DIS. OF DIGESTIVE SYSTEM (520-579)	0 / 1.21	**0.00**	0.00 – 3.06	1 / 2.13	**0.47**	0.01 – 2.61	3 / 4.64	**0.65**	0.13 – 1.89
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	0 / 0.07	**0.00**	0.00 – 50.29	1 / 0.14	**7.38**	0.19 – 40.99	2 / 0.32	**6.22**	0.75 – 22.44
EXTERNAL CAUSES (E800-999)	6 / 4.55	**1.32**	0.48 – 2.87	11 / 8.94	**1.23**	0.61 – 2.20	31 / 24.69	**1.26**	0.85 – 1.78

* $p < 0.05$
** $p < 0.01$

Cause of death (ICD 9th revision)	Urine			Faeces			Blood		
	O/E	SMR	95% CI	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	208 / 240.78	**0.86***	0.75 – 0.99	206 / 228.13	**0.90**	0.78 – 1.04	183 / 206.44	**0.89**	0.76 – 1.03
ALL MALIGNANT NEOPLASMS (140-208)	72 / 79.96	**0.90**	0.71 – 1.13	68 / 76.17	**0.89**	0.69 – 1.13	63 / 68.14	**0.92**	0.71 – 1.18
Oesophagus (150)	4 / 2.75	**1.45**	0.40 – 3.72	3 / 2.62	**1.14**	0.24 – 3.35	3 / 2.35	**1.28**	0.26 – 3.73
Stomach (151)	3 / 3.70	**0.81**	0.17 – 2.37	3 / 3.52	**0.85**	0.18 – 2.49	2 / 3.17	**0.63**	0.08 – 2.28
Colon (153)	4 / 7.94	**0.50**	0.14 – 1.29	4 / 7.58	**0.53**	0.14 – 1.35	3 / 6.74	**0.44**	0.09 – 1.30
Rectum (154)	6 / 5.38	**1.11**	0.41 – 2.43	5 / 5.13	**0.97**	0.32 – 2.28	5 / 4.58	**1.09**	0.35 – 2.55
Pancreas (157)	4 / 3.20	**1.25**	0.34 – 3.19	4 / 3.05	**1.31**	0.36 – 3.35	4 / 2.73	**1.46**	0.40 – 3.74
Lung (162)	20 / 19.51	**1.03**	0.63 – 1.58	20 / 18.62	**1.07**	0.66 – 1.66	17 / 16.58	**1.03**	0.60 – 1.64
Melanoma (172)	4 / 3.40	**1.18**	0.32 – 3.01	4 / 3.21	**1.25**	0.34 – 3.19	4 / 2.92	**1.37**	0.37 – 3.50
Prostate (185)	4 / 4.86	**0.82**	0.23 – 2.11	3 / 4.64	**0.65**	0.13 – 1.89	4 / 4.17	**0.96**	0.26 – 2.45
Bladder (188)	2/ 1.38	**1.45**	0.18 – 5.22	1 / 1.32	**0.76**	0.02 – 4.20	2 / 1.18	**1.69**	0.21 – 6.11
Kidney (189)	5 / 2.26	**2.21**	0.72 – 5.17	4 / 2.15	**1.86**	0.51 – 4.77	4 / 1.93	**2.07**	0.57 – 5.30
Brain (191)	3 / 3.39	**0.88**	0.18 – 2.59	2 / 3.21	**0.62**	0.08 – 2.25	3 / 2.90	**1.03**	0.21 – 3.02
Ill defined (195, 199)	5 / 4.79	**1.05**	0.40 – 2.29	6 / 4.56	**1.32**	0.55 – 2.71	5 / 4.07	**1.23**	0.47 – 2.69
Lymphatic and haematopoietic tissue (200-208)	2 / 7.48	**0.27***	0.03 – 0.97	3 / 7.09	**0.42**	0.09 – 1.24	1 / 6.40	**0.16***	0.01 – 0.87
Non-Hodgkin's lymphoma (200, 202)	2 / 3.37	**0.59**	0.12 – 1.91	2 / 3.19	**0.63**	0.13 – 2.01	1 / 2.88	**0.35**	0.03 – 1.62
Leukaemia and aleukaemia (204-208)	0 / 2.56	**0.00***	0.00 – 0.97	1 / 2.42	**0.41**	0.04 – 1.93	0 / 2.19	**0.00**	0.00 – 1.13
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	5 / 10.72	**0.47**	0.18 – 1.02	5 / 10.1288	**0.49**	0.19 – 1.08	5 / 9.19	**0.54**	0.21 – 1.19
MENTAL DISORDERS (290-319)	1 / 1.65	**0.61**	0.02 – 3.36	0 / 1.54	**0.00**	0.00 0 2.39	1 / 1.44	**0.69**	0.02 – 3.85
DIS. OF CIRCULATORY SYSTEM (390-459)	83 / 91.75	**0.90**	0.72 – 1.12	84 / 87.29	**0.96**	0.77 – 1.19	70 / 78.53	**0.89**	0.70 – 1.13
DIS. OF RESPIRATORY SYSTEM (460-519)	9 / 12.86	**0.70**	0.32 – 1.33	11 / 12.28	**0.90**	0.45 – 1.60	8 / 11.03	**0.73**	0.31 – 1.43
Bronchitis, emphysema and asthma (490-493)	8 / 3.92	**2.04**	0.88 – 4.02	9 / 3.74	**2.41***	1.10 – 4.57	7 / 3.36	**2.09**	0.84 – 4.30
DIS. OF DIGESTIVE SYSTEM (520-579)	3 / 5.45	**0.55**	0.11 – 1.61	4 / 5.19	**0.77**	0.21 – 1.97	3 / 4.66	**0.64**	0.13 – 1.88
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	2 / 0.38	**5.32**	0.64 – 19.22	2 / 0.35	**5.67**	0.69 – 20.46	2 / 0.32	**6.18**	0.75 – 22.31
EXTERNAL CAUSES (E800-999)	33 / 28.39	**1.16**	0.80 – 1.63	31 / 26.09	**1.19**	0.81 – 1.69	31 / 24.85	**1.25**	0.85 – 1.77

* $p < 0.05$
** $p < 0.01$

were the stockyard workers already examined in the previous analysis by department ever worked in.

As can be seen in Table 5.8.1 a total of 1,116 members of the Union Cohort were categorised as having worked in contact with pelts or hides, and this group experienced a significant deficit in all cause mortality (SMR 0.72, 95% CI 0.56 – 0.92, 66 deaths). Mortality from diseases of the circulatory system was also significantly lower than expected (SMR 0.64, 95% CI 0.41 – 0.97, 23 deaths), and there was a similar (but non-significant) deficit in mortality from all cancer (SMR 0.76, 24 deaths). Of specific cancer types, excess mortality was observed for cancers of the rectum (SMR 1.40, 3 deaths), prostate (SMR1.56, 3 deaths) and kidney (SMR 4.49, 95% CI 1.22 – 11.48, 4 deaths), as well as for malignant melanoma of the skin (SMR 1.57, 2 deaths) and non-Hodgkin's lymphoma (SMR 1.54, 2 deaths). An excess of mortality from the disease category bronchitis, emphysema and asthma (SMR 1.97, 3 deaths) contrasted with a large deficit in deaths from lung cancer (SMR 0.38, 3 deaths).

The 3,342 individuals who had worked in departments with the potential for exposure to animal slaughter/handling freshly slaughtered meat experienced a significant deficit in all cause mortality (SMR 0.85, 95% CI 0.73 – 0.97), and a similar (but non-significant) deficit in mortality from all cancers (SMR 0.90, 72 deaths) and from diseases of the circulatory system (SMR 0.89, 82 deaths). The only specific cancer sites for which any excess was observed were kidney (SMR 2.65, 95% CI 0.97 – 5.77, 6 deaths), bladder (SMR 1.44, 2 deaths) and oesophagus (SMR 1.45, 4 deaths), while a highly significant deficit in mortality from lymphohaematopoietic cancer (SMR

0.13, 95% CI 0.01 – 0.74, 1 death) was observed. The almost 2-fold excess in mortality from bronchitis, emphysema and asthma (SMR 1.78, 7 deaths) contrasted with the lack of any excess in lung cancer mortality (SMR 1.07, 21 deaths).

The same pattern of significantly low overall mortality (SMR 0.86, 95% CI 0.75 – 0.99, 208 deaths), and deficits in mortality from all cancer (SMR 0.90, 72 deaths) and from diseases of the circulatory system (SMR 0.90, 83 deaths) was evident among the 3,362 individuals who had worked in jobs entailing potential exposure to animal urine. Also evident was the same pattern of a 2-fold excess of deaths from bronchitis, emphysema and asthma (SMR 2.04, 8 deaths) in contrast to a lung cancer mortality rate close to expectation (SMR 1.03, 20 deaths). Both patterns repeated themselves for those members of the groups who had potential exposure to gastrointestinal microflora or faeces and for exposure to animal blood. Also evident for all three groups was a deficit in cancers of lymphohaematopoietic tissue, which in the case of those with exposure to urine and blood was significantly reduced (SMR 0.27 and 0.16 respectively.

5.8.2 Mortality by Biological Exposures - Company Cohort.

Table 5.8.2 shows the corresponding findings for the Company Cohort. Among the relatively few individuals (N = 250) with exposure to live animal contact there was a deficit of all cause mortality (SMR 0.87, 10 deaths), but an excess of cancer mortality (SMR 1.42) based on only 5 deaths. Two of these deaths were from the category ill-defined or other unspecified sites (SMR 9.89, 95% CI 1.97 – 31.71), and one non-Hodgkin's lymphoma death occurred compared with 0.16 expected.

Table 5.8.2 Mortality by potential biological exposures – Company Cohort

Cause of death (ICD 9th revision)	Live animal contact			Pelts or hides			Raw meat		
	O/E	SMR	95% CI	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	10 / 11.49	**0.87**	0.42 – 1.60	22 / 21.90	**1.00**	0.63 – 1.52	146 / 126.93	**1.15**	0.97 – 1.35
ALL MALIGNANT NEOPLASMS (140-208)	5 / 3.53	**1.42**	0.46 – 3.31	10 / 6.50	**1.54**	0.74 – 2.83	44 / 37.69	**1.17**	0.85 – 1.57
Oral cavity (141-145)	1 / 0.04	**24.84**	0.63 – 138.0	1 / 0.07	**13.81**	0.35 – 76.70	1 / 0.39	**2.54**	0.06 – 14.10
Oesophagus (150)	0 / 0.12	**0.00**	0.00 0 31.25	1 / 0.22	**4.64**	0.12 – 25.77	1 / 1.03	**0.97**	0.03 – 5.41
Stomach (151)	0 / 0.17	**0.00**	0.00 – 21.84	0 / 0.31	**0.00**	0.00 – 11.81	4 / 1.73	**2.31**	0.63 – 5.92
Colon (153)	0 / 0.34	**0.00**	0.00 – 10.89	0 / 0.61	**0.00**	0.00 – 6.01	6 / 3.42	**1.75**	0.64 – 3.82
Pancreas (157)	0 / 0.14	**0.00**	0.00 – 26.06	0 / 0.26	**0.00**	0.00 – 14.31	1 / 1.38	**0.72**	0.02 – 4.02
Lung (162)	0 / 0.82	**0.00**	0.00 – 4.53	1 / 1.48	**0.67**	0.02 – 3.75	15 / 7.55	**1.99***	1.11 – 3.28
Melanoma (172)	0 / 0.17	**0.00**	0.00 – 21.96	1 / 0.31	**3.20**	0.08 – 17.76	3 / 1.93	**1.56**	0.32 – 4.55
Prostate (185)	0 / 0.20	**0.00**	0.00 – 18.66	0 / 0.38	**0.00**	0.00 – 9.65	2 / 1.54	**1.30**	0.16 – 4.70
Bladder (188)	0 / 0.06	**0.00**	0.00 – 62.66	0 / 0.11	**0.00**	0.00 – 33.40	2 / 0.51	**3.95**	0.48 – 14.25
Brain (191)	0 / 0.17	**0.00**	0.00 – 22.07	0 / 0.31	**0.00**	0.00 – 11.92	1 / 1.90	**0.53**	0.01 – 2.93
Thyroid (193)	0 / 0.01	**0.00**	0.00 – 519.2	1 / 0.01	**77.49***	1.96 – 430.5	1 / 0.08	**12.22**	0.31 – 67.90
Ill defined (195, 199)	2 / 0.20	**9.89***	1.97 – 31.71	2 / 0.37	**5.37***	1.07 – 17.23	2 / 2.10	**0.95**	0.19 – 3.05
Lymphatic and haematopoietic tissue (200-208)	1 / 0.36	**2.79**	0.07 – 15.48	1 / 0.67	**1.49**	0.04 – 8.28	2 / 3.92	**0.51**	0.06 – 1.84
Non-Hodgkin's lymphoma (200, 202)	1 / 0.16	**6.29**	0.57 – 29.34	1 / 0.29	**3.41**	0.31 – 15.89	1 / 1.73	**0.58**	0.05 – 2.70
Leukaemia and aleukaemia (204-208)	0 / 0.13	**0.00**	0.00 - 19.12	0 / 0.25	**0.00**	0.00 – 9.98	1 / 1.47	**0.68**	0.06 – 3.18
BENIGN NEOPLASMS (210-239)	0 / 0.04	**0.00**	0.00 – 101.6	0 / 0.07	**0.00**	0.00 – 52.71	0 / 0.41	**0.00**	0.00 – 8.96
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	0 / 0.5339	**0.00**	0.00 – 4.62	0 / 0.99	**0.00**	0.00 – 2.49	4 / 5.81	**0.69**	0.23 – 1.64
DIS. OF NERVOUS SYSTEM (320-359)	0 / 0.21	**0.00**	0.00 – 17.48	1 / 0.41	**2.41**	0.06 – 13.41	2 / 2.74	**0.73**	0.09 – 2.64
DIS. OF CIRCULATORY SYSTEM (390-459)	1 / 4.06	**0.25**	0.01 – 1.37	6 / 7.52	**0.80**	0.29 – 1.74	47 / 38.22	**1.23**	0.90 – 1.64
DIS. OF RESPIRATORY SYSTEM (460-519)	0 / 0.55	**0.00**	0.00 – 6.77	0 / 1.05	**0.00**	0.00 – 3.52	7 / 5.34	**1.31**	0.53 – 2.70
Bronchitis, emphysema and asthma (490-493)	0 / 0.17	**0.00**	0.00 – 21.41	0 / 0.33	**0.00**	0.00 – 11.21	6 / 1.85	**3.25***	1.19 – 7.08
DIS. OF DIGESTIVE SYSTEM (520-579)	0 / 0.24	**0.00**	0.00 – 15.10	1 / 0.45	**2.22**	0.06 – 12.31	4 / 3.24	**1.24**	0.34 – 3.16
EXTERNAL CAUSES (E800-999)	3 / 1.97	**1.52**	0.31 – 4.45	3 / 4.20	**0.71**	0.15 – 2.09	37 / 29.63	**1.25**	0.88 – 1.72

* $p < 0.05$
** $p < 0.01$

Cause of death (ICD 9th revision)	Urine			Faeces			Blood		
	O/E	SMR	95% CI	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	179 / 158.04	**1.13**	0.97 – 1.31	166 / 138.67	**1.20***	1.02 – 1.39	170 / 143.73	**1.18***	1.01 – 1.38
ALL MALIGNANT NEOPLASMS (140-208)	56 / 47.25	**1.19**	0.90 – 1.54	51 / 41.71	**1.22**	0.91 – 1.61	52 / 42.80	**1.22**	0.91 – 1.59
Oral cavity (141-145)	2 / 0.50	**4.02**	0.49 – 14.51	2 / 0.45	**4.4**	0.54 – 16.03	1 / 0.45	**2.22**	0.06 – 12.33
Oesophagus (150)	1 / 1.33	**0.75**	0.02 – 4.19	1 / 1.25	**0.80**	0.02 – 4.45	1 / 1.19	**0.84**	0.02 – 4.66
Stomach (151)	4 / 2.16	**1.85**	0.50 – 4.73	3 / 1.94	**1.55**	0.32 – 4.52	4 / 1.96	**2.04**	0.56 – 5.21
Colon (153)	6 / 4.35	**1.38**	0.50 – 3.01	5 / 3.89	**1.29**	0.42 – 3.01	6 / 3.92	**1.53**	0.56 – 3.33
Pancreas (157)	1 / 1.75	**0.57**	0.01 – 3.17	1 / 1.58	**0.63**	0.02 – 3.51	1 / 1.58	**0.63**	0.02 – 3.51
Lung (162)	19 / 9.71	**1.96****	1.18 – 3.06	20 / 8.96	**2.23****	1.36 – 3.45	19 / 8.73	**2.18****	1.31 – 3.40
Melanoma (172)	3 / 2.36	**1.27**	0.26 – 3.72	1 / 2.04	**0.49**	0.01 – 2.73	3 / 2.16	**1.39**	0.29 – 4.07
Prostate (185)	2 / 2.03	**0.99**	0.12 – 3.57	2 / 2.01	**0.99**	0.12 – 3.59	2 / 1.81	**1.11**	0.13 – 3.99
Bladder (188)	2 / 0.66	**3.05**	0.37 – 11.03	1 / 0.62	**1.62**	0.04 – 8.98	2 / 0.59	**3.41**	0.41 – 12.31
Brain (191)	1 / 2.33	**0.43**	0.01 – 2.38	1 / 2.02	**0.50**	0.01 – 2.75	1 / 2.13	**0.47**	0.01 – 2.61
Thyroid (193)	1 / 0.10	**9.86**	0.25 – 54.81	0 / 0.09	**0.00**	0.00 – 43.34	1 / 0.09	**10.92**	0.28 – 60.69
Ill defined (195, 199)	4 / 2.66	**1.51**	0.50 – 3.58	4 / 2.37	**1.69**	0.56 – 4.01	3 / 2.4	**1.25**	0.35 – 3.34
Lymphatic and haematopoietic tissue (200-208)	6 / 4.87	**1.23**	0.45 – 2.68	6 / 4.27	**1.41**	0.51 – 3.06	5 / 4.43	**1.13**	0.37 – 2.64
Non-Hodgkin's lymphoma (200, 202)	4 / 2.15	**1.86**	0.62 – 4.43	4 / 1.88	**2.13**	0.71 – 5.05	3 / 1.95	**1.54**	0.43 – 4.10
Leukaemia and aleukaemia (204-208)	2 / 1.82	**1.10**	0.22 – 3.52	2 / 1.58	**1.27**	0.25 – 4.07	2 / 1.66	**1.21**	0.24 – 3.87
BENIGN NEOPLASMS (210-239)	0 / 0.51	**0.00**	0.00 – 7.25	1 / 0.44	**2.27**	0.06 – 12.64	0 / 0.46	**0.00**	0.00 – 7.97
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	4 / 7.18	**0.56**	0.19 – 1.33	3 / 6.27	**0.48**	0.13 – 1.28	4 / 6.54	**0.61**	0.20 0 1.45
DIS. OF NERVOUS SYSTEM (320-359)	2 / 3.33	**0.60**	0.07 – 2.17	2 / 2.80	**0.71**	0.09 – 2.58	2 / 3.06	**0.65**	0.08 – 2.36
DIS. OF CIRCULATORY SYSTEM (390-459)	54 / 48.47	**1.11**	0.84 – 1.45	53 / 44.56	**1.19**	0.89 – 1.56	54 / 43.77	**1.23**	0.93 – 1.61
DIS. OF RESPIRATORY SYSTEM (460-519)	7 / 6.80	**1.03**	0.41 – 2.12	6 / 6.21	**0.97**	0.35 – 2.10	7 / 6.11	**1.14**	0.46 – 2.36
Bronchitis, emphysema and asthma (490-493)	6 / 2.32	**2.59***	0.95 – 5.64	5 / 2.06	**2.43**	0.79 – 5.68	6 / 2.10	**2.86***	1.05 – 6.23
DIS. OF DIGESTIVE SYSTEM (520-579)	3 / 3.08	**0.98**	0.20 – 2.85	3 / 2.77	**1.08**	0.22 – 3.17	3 / 2.78	**1.08**	0.22 – 3.15
EXTERNAL CAUSES (E800-999)	50 / 35.75	**1.40***	1.04 – 1.84	44 / 29.14	**1.51***	1.10 – 2.03	45 / 33.00	**1.36***	1.00 – 1.83

* $p < 0.05$
** $p < 0.01$

All cause mortality was as expected among those individuals (N = 558) with exposure to pelts and hides (SMR 1.00, 22 deaths), but cancer mortality was elevated (SMR 1.54, 10 deaths). No more than one of these deaths was attributed to any specific cancer site, however, apart from 2 deaths from the category ill-defined or other unspecified sites (SMR 5.37). Deaths from diseases of the circulatory system (SMR 0.80, 6 deaths) or from external causes (SMR 0.71, 3 deaths) were in deficit.

A much larger group (N = 4,887) within the company cohort were potentially exposed to the slaughter process and/or fresh raw meat. A slight excess in mortality from all causes (SMR 1.09, 156 deaths), all cancers (SMR 1.04, 44 deaths), and from diseases of the circulatory system (SMR 1.16, 50 deaths), respiratory system (SMR 1.34, 8 deaths), digestive system (SMR 1.46, 4 deaths) and from external causes (SMR 1.27, 42 deaths) was observed. Of the specific cancer sites a significant excess for lung cancer (SMR 1.76, 15 cases) was matched by a highly significant excess of deaths from the non-malignant respiratory disease category bronchitis, emphysema and asthma (SMR 3.38, 7 deaths). Non- significant excesses were observed for cancers of the stomach (SMR 2.06, 4 deaths), colon (SMR 1.56, 6 deaths) and bladder (SMR 3.50, 2 deaths). Fewer deaths than expected were recorded for cancers of the lymphohaematopoietic system.

In the 5,246 individuals with exposure to animal urine, mortality from all causes (SMR 1.13, 179 deaths), all cancers (SMR 1.19, 56 deaths), diseases of the circulatory system (SMR 1.11, 54 deaths (and from external causes (SMR 1.40, 50 deaths) was elevated. A highly significant excess in lung cancer mortality (SMR 1.96, 19 deaths) was matched by a significant excess of bronchitis, emphysema and asthma (SMR

2.59, 6 deaths). Other cancers in excess were the oral cavity (SMR 4.02, 2 deaths), stomach (SMR 1.85, 4 deaths), colon (SMR 1.38, 6 deaths), bladder (SMR 3.05, 2 deaths) and non-Hodgkin's lymphoma (SMR 1.86, 4 deaths).

There was a significant excess in all cause mortality among the 4,115 people in the group with exposure to gastrointestinal microflora and faeces (SMR 1.20, 166 deaths), and an excess in mortality from all cancers (SMR 1.22, 51 deaths). Mortality from diseases of the circulatory system showed a similar elevation (SMR 1.19, 53 deaths), as did mortality from external causes (SMR 1.51, 44 deaths). A highly significant elevation in lung cancer mortality (SMR 2.23, 20 deaths) was matched by a non-significant excess of mortality from bronchitis, emphysema and asthma (SMR 2.43, 5 deaths). Other cancer sites with excess mortality included the oral cavity (SMR 4.40, 2 deaths), stomach (SMR 1.55, 3 deaths), ill-defined or other unspecified sites (SMR 1.69, 4 deaths) and lymphohaematopoietic tissue (SMR 1.41, 6 deaths) – in particular non-Hodgkin's lymphoma (SMR 2.13, 4 deaths).

A virtually identical pattern was observed for the 4,890 individuals with exposure to blood, with similar levels of excess risk for mortality from all causes (SMR 1.18, 170 deaths), cancer (SMR 1.22, 52 deaths), circulatory disease (SMR 1.23, 54 deaths) and external causes (SMR 1.36, 45 deaths). Lung cancer mortality was also highly significantly elevated (SMR 2.18, 19 deaths), matched by a significant excess in bronchitis, emphysema and asthma mortality (SMR 2.86, 6 deaths). Stomach (SMR 2.04, 4 deaths), colon (SMR 1.53, 6 deaths) and bladder (SMR 3.41, 2 deaths) cancer mortality was elevated, as was non-Hodgkin's lymphoma (SMR 1.54, 3 deaths).

In summary, consistent elevations in all cause, all cancer, circulatory disease, and lung cancer mortality were observed in those exposed to the slaughter process and/or fresh raw meat, urine, gastrointestinal microflora and faeces and blood. These groups also experienced significant excesses in lung cancer mortality, although in all cases this was matched by an excess in mortality from bronchitis, emphysema and asthma. Mortality from non-Hodgkin's lymphoma was also consistently elevated in these groups.

5.9 Mortality by Chemical Exposures

5.9.1 Mortality by Chemical Exposures - Union Cohort

Table 5.9.1 shows mortality by potential chemical exposures in the Union Cohort. Amongst the Union Cohort members with exposure to refrigerant gases (N = 777), cleaning chemicals (N = 2,924), Animal remedies (N = 3,263) and organochlorines (N = 3,396) there were deficits in mortality from all causes, all cancers, and from all other major disease categories apart from deaths from external causes. Lung cancer mortality was close to expectation in all groups, despite an approximately 2-fold excess in mortality from bronchitis, emphysema and asthma which in the case of the exposed to organochlorines was significant (SMR 2.26, 9 cases). The only specific cancer site for which excess mortality was observed was the kidney, with a significant elevation in those exposed to both refrigerant gases (SMR 4.93, 3 cases) and organochlorines (SMR 2.61, 6 cases). There was a consistent deficit in mortality from cancer of the colon and stomach across exposure categories, and also for mortality from cancers of lymphohaematopoietic tissue.

Table 5.9.1 Mortality by potential chemical exposures – Union Cohort

Cause of death (ICD 9th revision)	Refrigerant gases			Cleaning chemicals		
	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	47 / 63.04	**0.75***	0.55 – 0.99	183 / 205.31	**0.89**	0.77 – 1.03
ALL MALIGNANT NEOPLASMS (140-208)	21 / 21.57	**0.97**	0.60 – 1.49	64 / 67.75	**0.94**	0.73 – 1.21
Oesophagus (150)	1 / 0.75	**1.33**	0.03 – 7.39	3 / 2.34	**1.28**	0.27 – 3.75
Stomach (151)	2 / 0.99	**2.03**	0.25 – 7.31	2 / 3.15	**0.64**	0.08 – 2.29
Colon (153)	1 / 2.17	**0.46**	0.01 – 2.56	3 / 6.71	**0.45**	0.09 – 1.31
Rectum (154)	2 / 1.46	**1.37**	0.17 – 4.95	5 / 4.56	**1.10**	0.36 – 2.56
Pancreas (157)	1 / 0.87	**1.16**	0.03 – 6.42	4 / 2.72	**1.47**	0.40 – 3.77
Lung (162)	4 / 5.36	**0.75**	0.20 – 1.91	18 / 16.49	**1.09**	0.65 – 1.73
Melanoma (172)	0 / 0.88	**0.00**	0.00 – 4.20	4 / 2.91	**1.38**	0.38 – 3.52
Prostate (185)	2 / 1.32	**1.52**	0.18 – 5.48	4 / 4.15	**0.96**	0.26 – 2.47
Bladder (188)	1 / 0.37	**2.69**	0.07 – 14.94	2 / 1.17	**1.70**	0.21 – 6.15
Kidney (189)	3 / 0.61	**4.93***	1.02 – 14.41	4 / 1.92	**2.08**	0.57 – 5.33
Brain (191)	1 / 0.88	**1.14**	0.03 – 6.31	3 / 2.89	**1.04**	0.21 – 3.04
Ill defined (195, 199)	2 / 1.31	**1.53**	0.31 – 4.90	5 / 4.05	**1.24**	0.47 – 2.71
Lymphatic and haematopoietic tissue (200-208)	0 / 1.97	**0.00**	0.00 – 1.88	1 / 6.37	**0.16***	0.01 – 0.87
Non-Hodgkin's lymphoma (200, 202)	0 / 0.89	**0.00**	0.00 – 2.76	1 / 2.86	**0.35**	0.03 – 1.63
Leukaemia and aleukaemia (204-208)	0 / 0.66	**0.00**	0.00 – 3.75	0 / 2.18	**0.00**	0.00 – 1.13
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	0 / 2.78	**0.00***	0.00 – 0.89	5 / 9.14	**0.55**	0.21 – 1.20
MENTAL DISORDERS (290-319)	0 / 0.39	**0.00**	0.00 – 9.52	1 / 1.43	**0.70**	0.02 – 3.87
DIS. OF CIRCULATORY SYSTEM (390-459)	20 / 24.50	**0.82**	0.50 – 1.26	69 / 78.09	**0.88**	0.69 – 1.12
DIS. OF RESPIRATORY SYSTEM (460-519)	3 / 3.44	**0.87**	0.18 – 2.55	8 / 10.97	**0.73**	0.31 – 1.44
Bronchitis, emphysema and asthma (490-493)	2 / 1.05	**1.91**	0.23 – 6.89	7 / 3.34	**2.10**	0.84 – 4.32
DIS. OF DIGESTIVE SYSTEM (520-579)	1 / 1.45	**0.69**	0.02 – 3.83	3 / 4.63	**0.65**	0.13 – 1.89
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	0 / 0.09	**0.00**	0.00 – 39.69	2 / 0.32	**6.21**	0.75 – 22.43
EXTERNAL CAUSES (E800-999)	2 / 6.37	**0.31**	0.04 – 1.13	31 / 24.74	**1.25**	0.85 – 1.78

* $p < 0.05$
** $p < 0.01$

Cause of death (ICD 9th revision)	Animal remedies			Organochlorines		
	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	198 / 232.33	**0.85***	0.74 – 0.98	213 / 244.68	**0.87**	0.76 – 1.00
ALL MALIGNANT NEOPLASMS (140-208)	6 / 77.08	**0.86**	0.66 – 1.09	73 / 81.34	**0.90**	0.70 – 1.13
Oesophagus (150)	4 / 2.65	**1.51**	0.41 – 3.86	3 / 2.80	**1.07**	0.22 – 3.13
Stomach (151)	3 / 3.57	**0.84**	0.17 – 2.46	3 / 3.77	**0.80**	0.16 – 2.33
Colon (153)	4 / 7.65	**0.52**	0.14 – 1.34	4 / 8.08	**0.49**	0.14 – 1.27
Rectum (154)	5 / 5.19	**0.96**	0.31 – 2.25	6 / 5.49	**1.09**	0.40 – 2.38
Pancreas (157)	4 / 3.09	**1.30**	0.35 – 3.31	4 / 3.26	**1.23**	0.33 – 3.14
Lung (162)	18 / 18.77	**0.96**	0.57 – 1.52	18 / 19.85	**0.91**	0.54 – 1.43
Melanoma (172)	4 / 3.29	**1.22**	0.33 – 3.11	5 / 3.46	**1.45**	0.47 – 3.38
Prostate (185)	3 / 4.68	**0.64**	0.13 – 1.88	4 / 4.95	**0.81**	0.22 – 2.07
Bladder (188)	1 / 1.33	**0.75**	0.02 – 4.17	2 / 1.41	**1.42**	0.17 – 5.12
Kidney (189)	4 / 2.18	**1.84**	0.50 – 4.69	6 / 2.30	**2.61***	**0.95 – 5.67**
Brain (191)	3 / 3.28	**0.92**	0.19 – 2.68	3 / 3.45	**0.87**	0.18 – 2.55
Ill defined (195, 199)	5 / 4.61	**1.09**	0.41 – 2.38	5 / 4.87	**1.03**	0.39 – 2.25
Lymphatic and haematopoietic tissue (200-208)	2 / 7.22	**0.28**	0.03 – 1.00	4 / 7.61	**0.53**	0.14 – 1.35
Non-Hodgkin's lymphoma (200, 202)	2 / 3.25	**0.62**	0.12 – 1.97	3 / 3.43	**0.88**	0.24 – 2.34
Leukaemia and aleukaemia (204-208)	0 / 2.47	**0.00**	0.00 – 1.00	1 / 2.60	**0.39**	0.04 – 1.80
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	5 / 10.36	**0.48**	0.18 – 1.06	6 / 10.90	**0.55**	0.23 – 1.13
MENTAL DISORDERS (290-319)	1 / 1.60	**0.63**	0.02 – 3.47	2 / 1.67	**1.20**	0.15 – 4.33
DIS. OF CIRCULATORY SYSTEM (390-459)	80 / 88.47	**0.90**	0.72 – 1.13	81 / 93.46	**0.87**	0.69 – 1.08
DIS. OF RESPIRATORY SYSTEM (460-519)	8 / 12.40	**0.65**	0.28 – 1.27	10 / 13.08	**0.76**	0.37 – 1.41
Bronchitis, emphysema and asthma (490-493)	7 / 3.78	**1.85**	0.74 – 3.81	9 / 3.99	**2.26***	1.03 – 4.29
DIS. OF DIGESTIVE SYSTEM (520-579)	3 / 5.26	**0.57**	0.12 – 1.67	4 / 5.55	**0.72**	0.20 – 1.84
SYMPTOMS AND ILL-DEFINED CONDITIONS (780-799)	2 / 0.36	**5.50**	0.67 – 19.87	3 / 0.38	**7.88***	1.63 – 23.04
EXTERNAL CAUSES (E800-999)	33 / 27.51	**1.20**	0.83 – 1.69	34 / 28.53	**1.19**	0.83 – 1.67

* $p < 0.05$
** $p < 0.01$

5.9.2 Mortality by Chemical Exposures - Company Cohort.

Table 5.9.2 shows the corresponding findings for the Company Cohort. Apart from the small group with exposure to refrigerant gases (N = 568) who experienced deficits in mortality from most causes, members of the Company Cohort with potential exposure to cleaning chemicals (N = 4,598), Animal remedies (N = 4,982) and organochlorines (N = 5,137) experienced similar mortality patterns to the overall cohort.

There was a consistent excess in mortality from all causes, from all cancers, and from diseases of the circulatory system and external causes, and also a significant excess in mortality from the non-malignant disease category bronchitis, emphysema and asthma. There was a consistent deficit in mortality from non-malignant diseases of the endocrine system and blood in all chemical exposure categories in the Company Cohort

Lung cancer mortality was significantly elevated in all three groups, with standardised mortality ratios ranging from 1.86 to 1.98. Mortality from cancers of the stomach, colon and bladder was elevated in each of these exposure categories, while mortality from non-Hodgkin's lymphoma was elevated in those exposed to animal remedies and organochlorines.

Table 5.9.2 Mortality by potential chemical exposures – Company Cohort

Cause of death (ICD 9th revision)	Refrigerant gases			Cleaning chemicals		
	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	15 / 16.75	**0.90**	0.50 – 1.48	151 / 133.18	**1.13**	0.96 – 1.33
ALL MALIGNANT NEOPLASMS (140-208)	3 / 4.77	**0.63**	0.13 – 1.84	45 / 39.67	**1.13**	0.83 – 1.52
Oral cavity (141-145)	0 / 0.05	**0.00**	0.00 – 68.65	1 / 0.41	**2.42**	0.06 – 13.43
Oesophagus (150)	0 / 0.14	**0.00**	0.00 – 26.76	1 / 1.08	**0.92**	0.02 – 5.13
Stomach (151)	0 / 0.23	**0.00**	0.00 – 16.13	4 / 1.81	**2.21**	0.60 – 5.65
Colon (153)	0 / 0.44	**0.00**	0.00 – 8.37	6 / 3.62	**1.66**	0.61 – 3.61
Pancreas (157)	0 / 0.18	**0.00**	0.00 – 20.40	1 / 1.46	**0.69**	0.02 – 3.81
Lung (162)	1 / 0.98	**1.02**	0.03 – 5.68	15 / 7.99	**1.88***	1.05 – 3.10
Melanoma (172)	1 / 0.26	**3.87**	0.10 – 21.48	3 / 2.01	**1.49**	0.31 – 4.37
Prostate (185)	0 / 0.19	**0.00**	0.00 – 19.98	2 / 1.62	**1.23**	0.15 – 4.45
Bladder (188)	1 / 0.06	**15.93**	0.40 – 88.51	2 / 0.54	**3.73**	0.45 – 13.47
Brain (191)	0 / 0.26	**0.00**	0.00 – 14.30	1 / 1.98	**0.50**	0.01 – 2.80
Thyroid (193)	0 / 0.01	**0.00**	0.00 – 369.8	1 / 0.09	**11.53**	0.29 – 64.03
Ill defined (195, 199)	0 / 0.27	**0.00**	0.00 – 9.14	3 / 2.22	**1.35**	0.37 – 3.61
Lymphatic and haematopoietic tissue (200-208)	0 / 0.52	**0.00**	0.00 – 7.13	2 / 4.11	**0.49**	0.06 – 1.76
Non-Hodgkin's lymphoma (200, 202)	0 / 0.23	**0.00**	0.00 – 10.63	1 / 1.81	**0.55**	0.05 – 2.57
Leukaemia and aleukaemia (204-208)	0 / 0.19	**0.00**	0.00 – 12.79	1 / 1.54	**0.65**	0.06 – 3.03
BENIGN NEOPLASMS (210-239)	0 / 0.05	**0.00**	0.00 – 72.20	0 / 0.43	**0.00**	0.00 – 8.54
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	0 / 0.80	**0.00**	0.00 – 3.09	4 / 6.07	**0.66**	0.22 – 1.57
DIS. OF NERVOUS SYSTEM (320-359)	0 / 0.36	**0.00**	0.00 – 10.17	2 / 2.86	**0.70**	0.09 – 2.53
DIS. OF CIRCULATORY SYSTEM (390-459)	3 / 5.02	**0.60**	0.12 – 1.75	48 / 40.14	**1.20**	0.88 – 1.59
DIS. OF RESPIRATORY SYSTEM (460-519)	2 / 0.63	**3.17**	0.38 – 11.44	7 / 5.64	**1.24**	0.50 – 2.56
Bronchitis, emphysema and asthma (490-493)	2 / 0.23	**8.76***	1.06 – 31.64	6 / 1.94	**3.09***	1.13 – 6.72
DIS. OF DIGESTIVE SYSTEM (520-579)	1 / 0.32	**3.14**	0.08 – 17.46	3 / 2.57	**1.17**	0.24 – 3.42
EXTERNAL CAUSES (E800-999)	6 / 4.20	**1.43**	0.52 – 3.11	40 / 30.94	**1.29**	0.92 – 1.76

* $p < 0.05$
** $p < 0.01$

Cause of death (ICD 9th revision)	Animal Remedies			Organochlorines		
	O/E	SMR	95% CI	O/E	SMR	95% CI
ALL CAUSES	170 / 148.88	**1.14**	0.98 – 1.33	181 / 156.11	**1.16**	0.99 – 1.34
ALL MALIGNANT NEOPLASMS (140-208)	53 / 44.51	**1.19**	0.89 – 1.56	58 / 46.44	**1.25**	0.95 – 1.62
Oral cavity (141-145)	2 / 0.47	**4.27**	0.52 – 15.40	2 / 0.49	**4.06**	0.49 – 14.65
Oesophagus (150)	1 / 1.25	**0.80**	0.02 – 4.45	2 / 1.32	**1.51**	0.18 – 5.46
Stomach (151)	4 / 2.04	**1.96**	0.53 – 5.01	4 / 2.14	**1.87**	0.51 – 4.77
Colon (153)	6 / 4.08	**1.47**	0.54 – 3.20	6 / 4.27	**1.41**	0.51 – 3.06
Pancreas (157)	1 / 1.65	**0.61**	0.02 – 3.37	1 / 1.73	**0.58**	0.02 – 3.21
Lung (162)	17 / 9.12	**1.86***	1.09 – 2.98	19 / 9.59	**1.98****	1.19 – 3.10
Melanoma (172)	2 / 2.23	**0.90**	0.11 – 3.24	3 / 2.34	**1.28**	0.27 – 3.75
Prostate (185)	2 / 1.91	**1.05**	0.13 – 3.78	2 / 2.04	**0.98**	0.12 – 3.55
Bladder (188)	2 / 0.62	**3.24**	0.39 – 11.69	2 / 0.65	**3.07**	0.37 – 11.08
Brain (191)	1 / 2.21	**0.45**	0.01 – 2.52	1 / 2.31	**0.43**	0.01 – 2.41
Thyroid (193)	1 / 0.10	**10.49**	0.27 – 58.28	2 / 0.10	**20.30****	2.46 – 73.28
Ill defined (195, 199)	4 / 2.50	**1.60**	0.54 – 3.81	4 / 2.61	**1.53**	0.51 – 3.64
Lymphatic and haematopoietic tissue (200-208)	6 / 4.59	**1.31**	0.48 – 2.85	5 / 4.80	**1.04**	0.34 – 2.43
Non-Hodgkin's lymphoma (200, 202)	4 / 2.03	**1.98**	0.66 – 4.70	3 / 2.12	**1.42**	0.39 – 3.78
Leukaemia and aleukaemia (204-208)	2 / 1.72	**1.17**	0.23 – 3.74	2 / 1.79	**1.12**	0.22 – 3.58
BENIGN NEOPLASMS (210-239)	0 / 0.48	**0.00**	0.00 – 7.65	0 / 0.50	**0.00**	0.00 – 7.36
DIS. OF ENDOCRINE SYSTEM & BLOOD (240-289)	4 / 6.78	**0.59**	0.20 – 1.40	4 / 7.11	**0.56**	0.19 – 1.34
DIS. OF NERVOUS SYSTEM (320-359)	2 / 3.15	**0.64**	0.08 – 2.29	3 / 3.29	**0.91**	0.19 – 2.67
DIS. OF CIRCULATORY SYSTEM (390-459)	54 / 45.70	**1.18**	0.89 – 1.54	58 / 48.18	**1.20**	0.91 – 1.56
DIS. OF RESPIRATORY SYSTEM (460-519)	6 / 6.39	**0.94**	0.34 – 2.04	6 / 6.71	**0.89**	0.33 – 1.95
Bronchitis, emphysema and asthma (490-493)	5 / 2.18	**2.29**	0.74 – 5.36	5 / 2.28	**2.19**	0.71 – 5.12
DIS. OF DIGESTIVE SYSTEM (520-579)	3 / 2.90	**1.03**	0.21 – 3.03	4 / 3.04	**1.31**	0.36 – 3.36
EXTERNAL CAUSES (E800-999)	45 / 33.63	**1.34**	0.98 – 1.79	45 / 35.26	**1.28**	0.93 – 1.71

* $p < 0.05$
** $p < 0.01$

5.10 Mortality by duration of exposure

A series of analyses were performed to investigate the relationship between mortality and duration of employment in selected departments or in jobs that entail potential exposure to selected biological or chemical agents. These analyses investigated all cause mortality, mortality from all malignant neoplasms and cancers of the lung and lymphohaematopoietic tissue. This association was evaluated only in those departments or specific exposures with sufficient numbers of cases. Once again, separate analyses were performed for the Union Cohort and for the Company Cohort.

5.10.1 Mortality by duration of exposure - Union Cohort.

The results from this analysis for the Union Cohort are presented in Tables 5.10.1 to 5.10.4 below. As seen previously in the analysis of cause-specific mortality in the Union Cohort (Table 5.4.1), this group had significantly lower overall mortality, with deficits in all cancer, and in lung and lymphohaematopoietic cancers. When stratified by department and specific exposures this pattern persisted, and as seen in Table 5.10.1 the low overall mortality was consistent across all the exposure variables examined regardless of duration of employment. The same pattern was repeated for mortality from all cancers, as seen in Table 5.10.2, with no apparent relationship between duration of exposure and any of the exposure variables. While lung cancer mortality was observed to be elevated among those who had been employed in the slaughterboard or in meat processing, and also in those who had worked in jobs with potential for exposure to the slaughter process and/or fresh raw meat, animal urine, gastrointestinal microflora and faeces, blood and to cleaning chemicals, in comparison

Table 5.10.1 **All cause mortality in the Union Cohort according to employment duration in selected departments and exposure categories.**

	Duration of employment (years)								
	Not exposed		1-4		5-19		20+		
	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR	p for trend
Department									
Slaughterboard	85 / 110.42	**0.77**	57 / 64.27	**0.89**	81 / 83.41	**0.97**	23 / 26.84	**0.86**	0.38
Meat processing	170 / 193.88	**0.88**	37 / 47.83	**0.77**	29 / 34.34	**0.85**	10 / 8.89	**1.13**	0.65
Biological exposures									
Slaughter/meat	63 / 79.56	**0.79**	58 / 63.43	**0.91**	94 / 108.35	**0.87**	31 / 33.59	**0.92**	0.61
Urine	38 / 44.15	**0.86**	57 / 60.72	**0.94**	117 / 137.03	**0.85**	34 / 42.82	**0.79**	0.52
Faeces	40 / 56.80	**0.70**	57 / 62.28	**0.92**	115 / 124.00	**0.93**	34 / 41.84	**0.81**	0.70
Blood	63 / 78.49	**0.80**	58 / 64.32	**0.90**	94 / 108.52	**0.87**	31 / 33.60	**0.92**	0.64
Chemical exposures									
Cleaning chemicals	63 / 79.62	**0.79**	58 / 62.77	**0.92**	94 / 108.72	**0.87**	31 / 33.82	**0.92**	0.66
Animal remedies	48 / 52.60	**0.91**	51 / 60.13	**0.85**	116 / 132.97	**0.87**	31 / 39.23	**0.79**	0.63
Organochlorines	33 / 40.25	**0.82**	57 / 63.65	**0.90**	122 / 141.20	**0.86**	34 / 39.83	**0.85**	0.98

Table 5.10.2 **Mortality from all malignant neoplasms in the Union Cohort according to employment duration in selected departments and exposure categories.**

	Duration of employment (years)								
	Not exposed		1-4		5-19		20+		
	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR	p for trend
Department									
Slaughterboard	29 / 37.16	**0.78**	20 / 20.97	**0.95**	27 / 27.14	**1.00**	8 / 9.65	**0.83**	0.72
Meat processing	56 / 63.87	**0.88**	16 / 16.05	**1.00**	10 / 11.74	**0.85**	2 / 3.25	**0.62**	0.67
Biological exposures									
Slaughter/meat	20 / 27.12	**0.74**	20 / 20.65	**0.97**	34 / 35.12	**0.97**	10 / 12.03	**0.83**	0.76
Urine	12 / 14.96	**0.80**	20 / 19.67	**1.02**	40 / 44.86	**0.89**	12 / 15.43	**0.78**	0.67
Faeces	16 / 18.75	**0.85**	18 / 20.13	**0.89**	40 / 40.97	**0.98**	10 / 15.07	**0.66**	0.62
Blood	21 / 26.79	**0.78**	19 / 20.93	**0.91**	34 / 35.17	**0.97**	10 / 12.03	**0.83**	0.79
Chemical exposures									
Cleaning chemicals	20 / 27.17	**0.74**	20 / 20.41	**0.98**	34 / 35.23	**0.97**	10 / 12.12	**0.83**	0.78
Animal remedies	18 / 17.8362	**1.01**	17 / 19.44	**0.87**	38 / 43.59	**0.87**	11 / 14.05	**0.78**	0.56
Organochlorines	11 / 13.58	**0.81**	21 / 20.54	**1.02**	40 / 46.50	**0.86**	12 / 14.31	**0.84**	0.75

Table 5.10.3 **Lung cancer mortality in the Union Cohort according to employment duration in selected departments and exposure categories.**

	Duration of employment (years)								
	Not exposed		1-4		5-19		20+		
	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR	p for trend
Department									
Slaughterboard	7 / 9.10	**0.77**	6 / 5.09	**1.18**	7 / 6.62	**1.06**	3 / 2.39	**1.25**	0.54
Meat processing	15 / 15.57	**0.96**	4 / 3.89	**1.03**	3 / 2.92	**1.03**	1 / 0.82	**1.22**	0.82
Biological exposures									
Slaughter/meat	5 / 6.69	**0.75**	7 / 5.04	**1.39**	7 / 8.51	**0.82**	4 / 2.96	**1.35**	0.72
Urine	3 / 3.69	**0.81**	8 / 4.77	**1.68**	8 / 10.95	**0.73**	4 / 3.79	**1.06**	0.61
Faeces	3 / 4.58	**0.66**	6 / 4.81	**1.25**	10 / 10.06	**0.99**	4 / 3.75	**1.07**	0.79
Blood	6 / 6.62	**0.91**	6 / 5.11	**1.17**	7 / 8.52	**0.82**	4 / 2.96	**1.35**	0.76
Chemical exposures									
Cleaning chemicals	5 / 6.71	**0.75**	7 / 4.97	**1.41**	7 / 8.53	**0.82**	4 / 2.98	**1.34**	0.73
Animal remedies	5 / 4.43	**1.13**	7 / 4.72	**1.48**	7 / 10.61	**0.6**	4 / 3.44	**1.16**	0.58
Organochlorines	5 / 3.35	**1.49**	7 / 4.96	**1.41**	7 / 11.38	**0.62**	4 / 3.52	**1.14**	0.38

Table 5.10.4 **Mortality from malignant neoplasms of lymphatic and haematopoietic tissue in the Union Cohort according to employment duration in selected departments and exposure categories.**

	Duration of employment (years)								
	Not exposed		1-4		5-19		20+		
	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR	p for trend
Department									
Slaughterboard	4 / 3.44	**1.16**	0 / 1.98	**0.00**	1 / 2.56	**0.39**	0 / 0.88	**0.00**	0.25
Meat processing	3 / 6.01	**0.50**	1 / 1.49	**0.67**	1 / 1.06	**0.95**	0 / 0.29	**0.00**	0.93
Biological exposures									
Slaughter/meat	4 / 2.48	**1.61**	0 / 1.94	**0.00**	1 / 3.33	**0.30**	0 / 1.11	**0.00**	0.11
Urine	3 / 1.37	**2.19**	0 / 1.86	**0.00**	2 / 4.20	**0.48**	0 / 1.42	**0.00**	0.13
Faeces	2 / 1.76	**1.13**	0 / 1.92	**0.00**	3 / 3.80	**0.79**	0 / 1.36	**0.00**	0.53
Blood	4 / 2.45	**1.64**	0 / 1.96	**0.00**	1 / 3.33	**0.30**	0 / 1.11	**0.00**	0.11
Chemical exposures									
Cleaning chemicals	4 / 2.48	**1.61**	0 / 1.92	**0.00**	1 / 3.34	**0.30**	0 / 1.11	**0.00**	0.11
Animal remedies	3 / 1.63	**1.84**	0 / 1.84	**0.00**	2 / 4.09	**0.49**	0 / 1.30	**0.00**	0.17
Organochlorines	1 / 1.24	**0.81**	0 / 1.94	**0.00**	2 / 4.35	**0.46**	2 / 1.32	**1.52**	0.19

to those who had never experienced these exposures. As seen in Table 5.10.3, however, while mortality was elevated in these groups there is no clear dose-response. Most of the cases of lymphohaematopoietic cancers occurred among those individuals who had never experienced the specific exposures evaluated, as seen in Table 5.10.4, and for the small numbers of deaths that did occur there is no clear pattern.

5.10.2 Mortality by duration of exposure - Company Cohort

The results of the corresponding analyses for the Company Cohort are presented in Tables 5.10.5 to 5.10.8 below.

In contrast to the Union Cohort, an increasing risk of mortality from all causes appears to be related to the duration of exposure to work in the slaughterboard department, and to exposure to a number of the potential biological and chemical exposures experienced by those working on the slaughterboard. As seen in Table 5.10.5, a clear elevation in risk is associated with exposure to the slaughter process and/or fresh raw meat, animal urine, faecal matter and blood, as well as to cleaning chemicals, animal remedies and organochlorines. In all cases this excess risk appears to increase with increasing duration of exposure. No clear association was observed between work in the meat processing and plant services department and excess risk, and neither was any increasing risk with increasing duration of employment evident.

For all cancers, a significant excess mortality amongst those with more than 20 years employment in the slaughterboard was observed. As seen in table 5.10.6, however, no excess above the rate experienced by those who had never worked on the

Table 5.10.5 **All cause mortality in the Company Cohort according to employment duration in selected departments and exposure categories.**

	Duration of employment (years)								
	Not exposed		1-4		5-14		15+		
	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR	p for trend
Department									
Slaughterboard	114 / 111.53	**1.02**	31 / 33.77	**0.92**	48 / 36.06	**1.33**	34 / 22.22	**1.53***	0.01
Meat processing	204 / 180.57	**1.13**	9 / 9.44	**0.95**	5 / 7.13	**0.70**	9 / 6.43	**1.40**	0.93
Plant services	189 / 170.74	**1.11**	18 / 15.48	**1.16**	15 / 10.76	**1.39**	5 / 6.59	**0.76**	0.78
Biological exposures									
Slaughter/meat	71 / 61.07	**1.16**	45 / 53.53	**0.84**	69 / 53.47	**1.29***	42 / 35.50	**1.18**	0.32
Urine	48 / 45.54	**1.05**	55 / 61.73	**0.89**	79 / 60.55	**1.31***	45 / 35.76	**1.26**	0.08
Faeces	61 / 64.91	**0.94**	51 / 53.75	**0.95**	67 / 24.22	**2.27****	48 / 33.70	**1.42***	<0.001
Blood	57 / 59.85	**0.95**	55 / 58.44	**0.94**	72 / 51.97	**1.39****	43 / 33.32	**1.29**	0.03
Chemical exposures									
Cleaning chemicals	76 / 70.39	**1.08**	48 / 53.35	**0.90**	65 / 50.22	**1.29***	38 / 29.61	**1.28**	0.12
Animal remedies	57 / 54.69	**1.04**	55 / 58.67	**0.94**	72 / 56.79	**1.27**	43 / 33.42	**1.29**	0.10
Organochlorines	46 / 47.46	**0.97**	53 / 59.80	**0.89**	76 / 58.06	**1.31***	52 / 38.25	**1.36***	0.01

* $p < 0.05$
** $p < 0.01$

Table 5.10.6 **Mortality from all malignant neoplasms in the Company Cohort according to employment duration in selected departments and exposure categories**

	Duration of employment (years)								
	Not exposed		1-4		5-14		15+		
	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR	p for trend
Department									
Slaughterboard	35 / 33.80	**1.04**	9 / 8.81	**1.02**	11 / 11.30	**0.97**	14 / 7.46	**1.88***	0.09
Meat processing	63 / 54.36	**1.16**	3 / 2.42	**1.24**	1 / 2.32	**0.43**	2 / 2.29	**0.87**	0.44
Plant services	58 / 51.36	**1.13**	1 / 3.94	**0.25**	6 / 3.69	**1.63**	4 / 2.39	**1.67**	0.31
Biological exposures									
Slaughter/meat	25 / 23.69	**1.06**	11 / 12.45	**0.88**	17 / 15.04	**1.13**	16 / 10.21	**1.57**	0.16
Urine	13 / 14.13	**0.92**	11 / 15.39	**0.71**	23 / 19.28	**1.19**	22 / 12.59	**1.75***	0.01
Faeces	18 / 19.67	**0.92**	12 / 13.77	**0.87**	17 / 16.33	**1.04**	22 / 11.61	**1.90****	0.01
Blood	17 / 18.58	**0.92**	12 / 14.81	**0.81**	20 / 16.30	**1.23**	20 / 11.69	**1.71***	0.02
Chemical exposures									
Cleaning chemicals	24 / 21.71	**1.11**	11 / 13.49	**0.82**	18 / 15.82	**1.14**	16 / 10.36	**1.55**	0.18
Animal remedies	16 / 16.87	**0.95**	12 / 14.75	**0.81**	20 / 18.07	**1.11**	21 / 11.70	**1.80***	0.02
Organochlorines	11 / 13.94	**0.79**	11 / 14.91	**0.74**	23 / 18.17	**1.27**	24 / 13.37	**1.80****	<0.01

* $p < 0.05$
** $p < 0.01$

Table 5.10.7 **Lung cancer mortality in the Company Cohort according to employment duration in selected departments and exposure categories.**

	Duration of employment (years)								
	Not exposed		1-4		5-14		15+		
	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR	p for trend
Department									
Slaughterboard	10 / 7.07	**1.42**	2 / 1.57	**1.28**	6 / 2.44	**2.46***	5 / 1.79	**2.79***	0.14
Meat processing	19 / 11.35	**1.67**	2 / 0.43	**4.67**	1 / 0.50	**2.02**	1 / 0.60	**1.67**	0.92
Plant services	18 / 10.61	**1.70**	0 / 0.75	**0.00**	2 / 0.90	**2.24**	3 / 0.62	**4.85***	0.09
Biological exposures									
Slaughter/meat	8 / 5.32	**1.50**	3 / 2.09	**1.44**	6 / 3.10	**1.94**	6 / 2.37	**2.54***	0.29
Urine	4 / 3.16	**1.27**	2 / 2.65	**0.75**	8 / 4.10	**1.95**	9 / 2.97	**3.04****	0.04
Faeces	3 / 3.91	**0.77**	3 / 2.51	**1.19**	8 / 3.59	**2.23***	9 / 2.87	**3.14****	0.01
Blood	4 / 4.14	**0.97**	3 / 2.59	**1.16**	7 / 3.39	**2.07**	9 / 2.75	**3.27****	0.02
Chemical exposures									
Cleaning chemicals	8 / 4.88	**1.64**	2 / 2.31	**0.87**	7 / 3.28	**2.13**	6 / 2.40	**2.50***	0.25
Animal remedies	6 / 3.75	**1.60**	3 / 2.55	**1.18**	6 / 3.82	**1.57**	8 / 2.16	**3.71****	0.06
Organochlorines	4 / 3.28	**1.22**	2 / 2.58	**0.77**	8 / 3.83	**2.09**	9 / 3.18	**2.83****	0.05

* $p < 0.05$
** $p < 0.01$

Table 5.10.8 **Mortality from malignant neoplasms of lymphatic and haematopoietic tissue in the Company Cohort according to employment duration in selected departments and exposure categories.**

	Duration of employment (years)								
	Not exposed		1-4		5-14		15+		
	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR	p for trend
Department									
Slaughterboard	4 / 3.42	**1.17**	1 / 1.03	**0.98**	0 / 1.12	**0.00**	1 / 0.70	**1.43**	0.82
Meat processing	6 / 5.56	**1.08**	0 / 0.29	**0.00**	0 / 0.23	**0.00**	0 / 0.19	**0.00**	0.49
Plant services	3 / 5.26	**0.57**	0 / 0.47	**0.00**	2 / 0.33	**6.03***	1 / 0.21	**4.85**	<0.01
Biological exposures									
Slaughter/meat	4 / 2.34	**1.71**	1 / 1.49	**0.67**	0 / 1.49	**0.00**	1 / 0.93	**1.07**	0.47
Urine	0 / 1.39	**0.00**	2 / 1.85	**1.08**	2 / 1.88	**1.07**	2 / 1.14	**1.75**	0.24
Faeces	0 / 1.99	**0.00**	2 / 1.62	**1.23**	2 / 1.59	**1.26**	2 / 1.06	**1.89**	0.15
Blood	1 / 1.83	**0.55**	1 / 1.76	**0.57**	2 / 1.61	**1.24**	2 / 1.06	**1.89**	0.20
Chemical exposures									
Cleaning chemicals	4 / 2.15	**1.86**	1 / 1.60	**0.62**	0 / 1.56	**0.00**	1 / 0.95	**1.06**	0.43
Animal remedies	0 / 1.67	**0.00**	2 / 1.77	**1.13**	2 / 1.76	**1.14**	2 / 1.07	**1.88**	0.20
Organochlorines	1 / 1.46	**0.69**	2 / 1.79	**1.12**	2 / 1.80	**1.11**	1 / 1.21	**0.82**	0.99

* $p < 0.05$
** $p < 0.01$

slaughterboard was evident in those with less than 20 years exposure. A similar excess was observed in those with more than 5 years employment in plant services, although there was no clear increase with increasing duration of employment. Apart from those with exposure to the slaughter process and/or fresh raw meat, all the biological and chemical exposures evaluated appeared to be associated with excess cancer risk and the risk increased with increasing duration of employment. This trend was strongest for the potential biological exposures in animal urine, faeces and blood and for potential chemical exposure to residues of animal remedies and organochlorines.

As seen in Table 5.10.7, a similar pattern was evident for lung cancer mortality. A clear risk was associated with work on the slaughterboard, and with exposure to animal urine, gastrointestinal microflora and faeces and blood, as well as with the chemical exposures evaluated. For animal urine, faeces and blood there was a significant trend of increasing risk with increasing duration of exposure, with highly significant excesses for those with more than 20 years exposure. Although numbers were small, there also appeared to be a trend of increasing risk of cancers of the lymphohaematopoietic system with increasing duration of employment in jobs with potential for exposure to animal urine, faeces and blood as seen in Table 5.10.8.

5.11 Cancer Incidence

The results of the follow-up for cancer incidence are presented in Table 5.11.1 for the Union Cohort and Table 5.12.1 for the Company cohort. The results of the analysis of incidence of all cancers, lung and lymphohaematopoietic cancers, and of non-

Hodgkin's lymphoma and leukaemia by age at risk, duration of employment and time since first employed are presented in Table 5.11.2 (Union Cohort) and Table 5.12.2 (Company Cohort) below. The results of the analysis of the relationship between the incidence of selected cancers and duration of exposure in selected exposure categories for the Union Cohort are presented in Tables 5.11.3 to 5.11 7, and for the Company Cohort in Tables 5.12.3 to 5.12.7.

5.11.1 Cancer Incidence - Union Cohort

Table 5.11.1 Incidence of selected Cancers – Union Cohort

Site (ICD 9th revision)	Observed	Expected	SIR	95% confidence interval Lower	Upper
ALL MALIGNANT NEOPLASMS (140-208)	186	203.78	**0.91**	0.79	1.05
Oral cavity and pharynx (140-149)	8	6.57	**1.22**	0.53	2.40
Oral cavity (141 - 145)	4	3.04	**1.31**	0.44	3.13
Oropharynx (146)	2	0.12	**16.71***	2.02	60.34
Nasopharynx (147)	1	0.66	**1.53**	0.04	8.47
Oesophagus (150)	4	3.47	**1.15**	0.31	2.95
Stomach (151)	6	5.95	**1.01**	0.37	2.20
Colon (153)	20	20.61	**0.97**	0.59	1.50
Rectum (154)	15	14.21	**1.06**	0.59	1.74
Liver, specified as primary (1550)	0	2.79	**0.00**	0.00	1.32
Gallbladder (156)	0	0.80	**0.00**	0.00	4.64
Pancreas (157)	3	4.01	**0.75**	0.15	2.19
Nose and sinuses (160)	1	0.38	**2.62**	0.07	14.55
Larynx (161)	3	2.53	**1.19**	0.24	3.47
Lung (162)	24	26.87	**0.89**	0.57	1.33
Bone (170)	1	0.61	**1.63**	0.04	9.06
Soft tissue (171)	0	3.22	**0.00**	0.00	1.15
Melanoma (172)	18	23.47	**0.77**	0.45	1.21
Other skin (173)	1	0.30	**3.32**	0.08	18.42
Breast (174-175)	2	1.31	**1.53**	0.19	5.51
Female genital organs (179 - 184)	3	0.37	**8.02***	1.65	23.46
Prostate (185)	28	34.25	**0.82**	0.54	1.18
Testis (186)	2	4.08	**0.49**	0.06	1.77
Bladder (188)	12	8.37	**1.43**	0.74	2.51
Kidney (189)	7	6.05	**1.16**	0.46	2.38
Eye (190)	1	0.76	**1.33**	0.03	7.36
Brain (191)	3	4.42	**0.68**	0.14	1.99
Thyroid (193)	0	1.26	**0.00**	0.00	2.92
Other endocrine glands (194)	0	0.24	**0.00**	0.00	15.24
Ill defined (195 - 199)	8	7.39	**1.08**	0.47	2.13
Lymphatic and haematopoietic tissue (200-208)	14	17.12	**0.82**	0.45	1.37
Non-Hodgkin's lymphoma (200, 202)	9	7.77	**1.16**	0.53	2.20
Hodgkin's disease (201)	0	1.07	**0.00**	0.00	3.45
Multiple myeloma (203)	0	2.57	**0.00**	0.00	1.44
Leukaemia and aleukaemia (204-208)	5	5.71	**0.88**	0.33	1.92

* $p < 0.05$

The relative risk of incidence of all cancers in the Union Cohort (Table 5.11.1) was similar to that for mortality seen in Table 5.4.1 (i.e. SIR = 0.91 compared with SMR = 0.88).

As would be expected, the number of cases of cancers in sites that have a high fatality/case ratio, such as lung cancer, was little different from the number of deaths. There was a higher number of cases of cancers of the lymphohaematopoietic system than the number of deaths seen in the mortality analysis, although the SIRs for all lymphohaematopoietic cancers, non-Hodgkin's lymphoma and leukaemia are all close to expected. The only cancer type for which there was a significant excess was cancers of the female genital organs (SIR 8.02, 95% CI 1.65 – 23.46, 3 cases).

In the stratified analysis presented in Table 5.11.2 there was no indication of any increase in the relative risk of any of the cancers examined with increasing age at risk, duration of employment or time since first employed. In the analyses of duration of employment by selected exposure categories (Tables 5.11.3 to 5.11.7), no significant trend of increasing relative risk with increasing duration of employment was evident for any cancer site.

Table 5.11.2 **Incidence of selected cancers according to age, duration of employment and time since first employed among members of the Union Cohort.**

	All Cancer		Lung Cancer		Lymphohaematopoietic	
	O/E	SIR	O/E	SIR	O/E	SIR
Total	186 / 203.78	**0.91**	24 / 26.87	**0.89**	14 / 17.12	**0.82**
Age (years)						
< 25	0 / 0.35	**0.00**	0 / 0.01	**0.00**	0 / 0.07	**0.00**
25 – 34	5 / 5.18	**0.97**	0 / 0.04	**0.00**	1 / 0.79	**1.27**
35 – 44	10 / 15.02	**0.67**	0 / 0.67	**0.00**	0 / 2.05	**0.00**
45 – 54	36 / 40.42	**0.89**	1 / 4.30	**0.23**	3 / 4.29	**0.70**
55 – 64	89 / 85.63	**1.04**	13 / 13.55	**0.96**	7 / 6.22	**1.13**
65 +	46 / 57.18	**0.81**	10 / 8.31	**1.20**	3 / 3.70	**0.81**
Duration of employment (years)						
1 -4	26 / 24.14	**1.08**	4 / 2.99	**1.34**	3 / 2.14	**1.40**
5 – 9	17 / 26.68	**0.64**	1 / 3.22	**0.31**	0 / 2.42	**0.00**
10 – 14	29 / 32.50	**0.89**	3 / 4.05	**0.74**	2 / 2.86	**0.70**
15 – 19	57 / 61.61	**0.93**	9 / 8.63	**1.04**	5 / 4.99	**1.00**
20 – 24	43 / 45.92	**0.94**	5 / 6.32	**0.79**	1 / 3.66	**0.27**
25 +	14 / 12.94	**1.08**	2 / 1.67	**1.20**	3 / 1.06	**2.84**
TSFE (years)						
<10	10 / 11.31	**0.88**	2 / 1.16	**1.72**	2 / 1.22	**1.64**
10 – 14	15 / 18.14	**0.83**	0 / 2.05	**0.00**	1 / 1.76	**0.57**
15 – 19	35 / 34.36	**1.02**	5 / 4.50	**1.11**	1 / 3.10	**0.32**
20 – 24	54 / 58.99	**0.92**	8 / 8.36	**0.96**	3 / 4.94	**0.61**
25 – 29	65 / 61.30	**1.06**	8 / 8.09	**0.99**	6 / 4.68	**1.28**
30+	7 / 19.68	**0.36**	1 / 2.71	**0.37**	1 / 1.42	**0.71**

	Non-Hodgkin's Lymphoma		Leukaemia	
	O/E	**SIR**	**O/E**	**SIR**
Total	9 / 7.77	**1.16**	5 / 5.71	**0.88**
Age (years)				
< 25	0 / 0.02	**0.00**	0 / 0.04	**0.00**
25 – 34	1 / 0.32	**3.16**	0 / 0.27	**0.00**
35 – 44	0 / 1.00	**0.00**	0 / 0.62	**0.00**
45 – 54	3 / 2.12	**1.41**	0 / 1.26	**0.00**
55 – 64	3 / 2.80	**1.07**	4 / 2.14	**1.87**
65 +	2 / 1.52	**1.32**	1 / 1.39	**0.72**
Duration of employment (years)				
1 -4	2 / 0.95	**2.10**	1 / 0.72	**1.39**
5 – 9	0 / 1.09	**0.00**	0 / 0.81	**0.00**
10 – 14	1 / 1.30	**0.77**	1 / 0.94	**1.06**
15 – 19	4 / 2.27	**1.76**	1 / 1.66	**0.60**
20 – 24	0 / 1.68	**0.00**	1 / 1.22	**0.82**
25 +	2 / 0.49	**4.12**	1 / 0.36	**2.80**
TSFE (years)				
<10	2 / 0.53	**3.75**	0 / 0.40	**0.00**
10 – 14	0 / 0.80	**0.00**	1 / 0.57	**1.76**
15 – 19	1 / 1.44	**0.69**	0 / 0.99	**0.00**
20 – 24	2 / 2.28	**0.88**	1 / 1.62	**0.62**
25 – 29	4 / 2.11	**1.90**	2 / 1.62	**1.24**
30+	0 / 0.62	**0.00**	1 / 0.51	**1.97**

Table 5.11.3 **Cancer incidence in the Union Cohort by employment duration in selected departments and exposure categories.**

	Duration of employment (years)								
	Not exposed		1-4		5-19		20+		
	O/E	SIR	O/E	SIR	O/E	SIR	O/E	SIR	p for trend
Department									
Slaughterboard	74 / 79.44	**0.93**	43 / 44.70	**0.96**	51 / 58.45	**0.87**	18 / 21.19	**0.85**	0.61
Meat processing	133 / 137.17	**0.97**	29 / 34.75	**0.83**	18 / 25.04	**0.72**	6 / 6.83	**0.88**	0.86
Biological exposures									
Slaughter/meat	54 / 57.28	**0.94**	39 / 44.12	**0.88**	71 / 75.82	**0.94**	22 / 26.56	**0.83**	0.74
Urine	31 / 31.45	**0.99**	42 / 42.12	**1.00**	81 / 96.18	**0.84**	32 / 34.03	**0.94**	0.66
Faeces	40 / 40.07	**1.00**	47 / 43.35	**1.08**	70 / 87.51	**0.80**	29 / 32.86	**0.88**	0.27
Blood	55 / 56.50	**0.97**	37 / 44.76	**0.83**	72 / 75.96	**0.95**	22 / 26.56	**0.83**	0.76
Chemical exposures									
Cleaning chemicals	54 / 57.36	**0.94**	39 / 43.63	**0.89**	71 / 76.03	**0.93**	22 / 26.76	**0.82**	0.70
Animal remedies	38 / 37.66	**1.01**	42 / 41.19	**1.02**	77 / 93.87	**0.82**	29 / 31.07	**0.93**	0.49
Organochlorines	22 / 29.06	**0.76**	44 / 44.01	**1.00**	88 / 99.30	**0.89**	32 / 31.42	**1.02**	0.57

Table 5.11.4 **Lung cancer incidence in the Union Cohort by employment duration in selected departments and exposure categories.**

	Duration of employment (years)								
	Not exposed		1-4		5-19		20+		
	O/E	SIR	O/E	SIR	O/E	SIR	O/E	SIR	p for trend
Department									
Slaughterboard	9 / 10.47	**0.86**	5 / 5.84	**0.86**	7 / 7.68	**0.91**	3 / 2.88	**1.04**	0.76
Meat processing	16 / 17.99	**0.89**	4 / 4.54	**0.88**	2 / 3.39	**0.59**	2 / 0.95	**2.12**	0.56
Biological exposures									
Slaughter/meat	7 / 7.66	**0.91**	6 / 5.82	**1.03**	7 / 9.83	**0.71**	4 / 3.56	**1.12**	0.98
Urine	5 / 4.20	**1.19**	6 / 5.49	**1.09**	8 / 12.62	**0.63**	5 / 4.56	**1.10**	0.74
Faeces	5 / 5.25	**0.95**	5 / 5.52	**0.91**	9 / 11.63	**0.77**	5 / 4.48	**1.12**	0.43
Blood	8 / 7.57	**1.06**	5 / 5.91	**0.85**	7 / 9.84	**0.71**	4 / 3.56	**1.12**	0.97
Chemical exposures									
Cleaning chemicals	7 / 7.68	**0.91**	6 / 5.74	**1.05**	7 / 9.86	**0.71**	4 / 3.59	**1.12**	0.99
Animal remedies	7 / 5.07	**1.38**	5 / 5.37	**0.93**	7 / 12.29	**0.57**	5 / 4.15	**1.21**	0.71
Organochlorines	5 / 3.86	**1.30**	6 / 5.72	**1.05**	9 / 13.09	**0.69**	4 / 4.21	**0.95**	0.54

Table 5.11.5 Lymphohaematopoietic cancer incidence in the Union Cohort by employment duration in selected departments and exposure categories

	Duration of employment (years)								
	Not exposed		1-4		5-19		20+		
	O/E	SIR	O/E	SIR	O/E	SIR	O/E	SIR	p for trend
Department									
Slaughterboard	8 / 6.61	**1.21**	3 / 3.80	**0.79**	2 / 5.02	**0.40**	1 / 1.70	**0.59**	0.23
Meat processing	12 / 11.66	**1.03**	1 / 2.90	**0.34**	1 / 2.03	**0.49**	0 / 0.53	**0.00**	0.28
Biological exposures									
Slaughter/meat	6 / 4.70	**1.28**	4 / 3.72	**1.07**	3 / 6.54	**0.46**	1 / 2.16	**0.46**	0.14
Urine	4 / 2.60	**1.54**	4 / 3.60	**1.11**	4 / 8.16	**0.49**	2 / 2.76	**0.73**	0.23
Faeces	5 / 3.41	**1.47**	3 / 3.74	**0.80**	4 / 7.35	**0.54**	2 / 2.62	**0.77**	0.37
Blood	6 / 4.63	**1.30**	4 / 3.78	**1.06**	3 / 6.55	**0.46**	1 / 2.16	**0.46**	0.14
Chemical exposures									
Cleaning chemicals	6 / 4.70	**1.28**	4 / 3.70	**1.08**	3 / 6.56	**0.46**	1 / 2.17	**0.46**	0.14
Animal remedies	4 / 3.10	**1.29**	5 / 3.51	**1.42**	3 / 7.98	**0.38**	2 / 2.53	**0.79**	0.20
Organochlorines	1 / 2.39	**0.42**	5 / 3.75	**1.33**	5 / 8.44	**0.59**	3 / 2.55	**1.18**	0.87

Table 5.11.6 Non-Hodgkin's lymphoma incidence in the Union Cohort by employment duration in selected departments and exposure categories.

	Duration of employment (years)								
	Not exposed		1-4		5-19		20+		
	O/E	SIR	O/E	SIR	O/E	SIR	O/E	SIR	p for trend
Department									
Slaughterboard	5 / 5.00	**1.00**	2 / 1.71	**1.17**	1 / 2.28	**0.44**	1 / 0.78	**1.29**	0.82
Meat processing	8 / 5.28	**1.52**	1 / 1.32	**0.76**	0 / 0.92	**0.00**	0 / 0.24	**0.00**	0.20
Biological exposures									
Slaughter/meat	4 / 2.13	**1.88**	2 / 1.67	**1.20**	2 / 2.97	**0.67**	1 / 0.99	**1.01**	0.39
Urine	2 / 1.17	**1.71**	2 / 1.62	**1.23**	3 / 3.70	**0.81**	2 / 1.27	**1.58**	0.94
Faeces	3 / 1.55	**1.94**	2 / 1.70	**1.18**	2 / 3.33	**0.60**	2 / 1.19	**1.68**	0.79
Blood	4 / 2.10	**1.91**	2 / 1.70	**1.18**	2 / 2.98	**0.67**	1 / 1.00	**1.00**	0.39
Chemical exposures									
Cleaning chemicals	4 / 2.13	**1.88**	2 / 1.66	**1.20**	2 / 2.98	**0.67**	1 / 1.00	**1.00**	0.39
Animal remedies	2 / 1.40	**1.43**	3 / 1.58	**1.90**	2 / 3.62	**0.55**	2 / 1.17	**1.72**	0.76
Organochlorines	0 / 1.08	**0.00**	3 / 1.68	**1.78**	4 / 3.84	**1.04**	2 / 1.17	**1.71**	0.57

Table 5.11.7 Leukaemia incidence in the Union Cohort by employment duration in selected departments and exposure categories

	Duration of employment (years)								
	Not exposed		1-4		5-19		20+		
	O/E	SIR	O/E	SIR	O/E	SIR	O/E	SIR	p for trend
Department									
Slaughterboard	3 / 2.20	**1.36**	1 / 1.27	**0.79**	1 / 1.67	**0.60**	0 / 0.57	**0.00**	0.30
Meat processing	4 / 3.89	**1.03**	0 / 0.96	**0.00**	1 / 0.68	**1.48**	0 / 0.18	**0.00**	0.92
Biological exposures									
Slaughter/meat	2 / 1.57	**1.27**	2 / 1.25	**1.60**	1 / 2.17	**0.46**	0 / 0.72	**0.00**	0.15
Urine	2 / 0.87	**2.30**	2 / 1.20	**1.66**	1 / 2.72	**0.37**	0 / 0.92	**0.00**	0.32
Faeces	2 / 1.14	**1.75**	1 / 1.24	**0.80**	2 / 2.45	**0.82**	0 / 0.88	**0.00**	0.25
Blood	2 / 1.54	**1.30**	2 / 1.27	**1.57**	1 / 2.18	**0.46**	0 / 0.72	**0.00**	0.18
Chemical exposures									
Cleaning chemicals	2 / 1.57	**1.27**	2 / 1.24	**2.85**	1 / 2.18	**0.46**	0 / 0.72	**0.00**	0.18
Animal remedies	2 / 1.04	**1.92**	2 / 1.18	**1.70**	1 / 2.65	**0.38**	0 / 0.84	**0.00**	0.08
Organochlorines	1 / 0.80	**1.25**	2 / 1.26	**1.59**	1 / 2.80	**0.36**	1 / 0.85	**1.18**	0.05

5.11.2 Cancer Incidence - Company Cohort

The overall cancer incidence among members of the Company Cohort (Table 5.12.1) was close to expected (SIR 0.95, 143 cases), and the relative risk was lower than that for cancer mortality (SMR 1.12, 69 deaths) shown in Table 5.4.5. Lung cancer incidence was significantly elevated (SIR 1.70, 26 cases), and cancers of the lymphohaematopoietic system were close to expected. Cancer of the testis was also significantly elevated (SIR 1.93, 11 cases).

Table 5.12.1 Incidence of selected Cancers – Company Cohort

Site (ICD 9th revision)	Observed	Expected	SIR	95% confidence limits Lower	Upper
ALL MALIGNANT NEOPLASMS (140-208)	143	150.34	**0.95**	0.80	1.12
Oral cavity and pharynx (140-149)	5	4.45	**1.12**	0.36	2.62
Oral cavity (141 - 145)	0	2.00	**0.00**	0.00	1.23
Nasopharynx (147)	0	0.53	**0.00**	0.00	6.99
Oesophagus (150)	3	1.94	**1.55**	0.32	4.53
Stomach (151)	5	3.77	**1.33**	0.43	3.10
Colon (153)	16	12.90	**1.24**	0.71	2.01
Rectum (154)	6	8.63	**0.70**	0.25	1.51
Liver, specified as primary (1550)	1	1.89	**0.53**	0.01	2.94
Gallbladder (156)	1	0.54	**1.86**	0.05	10.31
Pancreas (157)	2	2.48	**0.81**	0.10	2.91
Nose and sinuses (160)	0	0.30	**0.00**	0.00	12.21
Larynx (161)	4	1.36	**2.95**	0.80	7.55
Lung (162)	26	15.29	**1.70***	1.11	2.49
Bone (170)	1	0.74	**1.36**	0.03	7.55
Soft tissue (171)	2	2.68	**0.75**	0.09	2.70
Melanoma (172)	12	21.82	**0.55***	0.28	0.96
Other skin (173)	0	0.24	**0.00**	0.00	15.57
Breast (174-175)	5	9.49	**0.53**	0.17	1.23
Female genital organs (179 - 184)	7	4.15	**1.69**	0.68	3.47
Prostate (185)	9	16.92	**0.53**	0.24	1.01
Testis (186)	11	5.69	**1.93***	0.96	3.46
Bladder (188)	3	4.92	**0.61**	0.13	1.78
Kidney (189)	3	3.96	**0.76**	0.16	2.22
Eye (190)	0	0.60	**0.00**	0.00	6.21
Brain (191)	1	3.80	**0.26**	0.01	1.46
Thyroid (193)	3	1.63	**1.84**	0.38	5.38
Other endocrine glands (194)	0	0.27	**0.00**	0.00	13.61
Ill defined (195 - 199)	5	4.81	**1.04**	0.34	2.43
Lymphatic and haematopoietic tissue (200-208)	11	13.56	**0.81**	0.41	1.45
Non-Hodgkin's lymphoma (200, 202)	6	6.07	**0.99**	0.36	2.15
Hodgkin's disease (201)	1	1.31	**0.76**	0.02	4.24
Multiple myeloma (203)	0	1.66	**0.00**	0.00	2.23
Leukaemia and aleukaemia (204-208)	4	4.53	**0.88**	0.30	2.10

* $p < 0.05$

Table 5.12.2 **Incidence of selected cancers according to age, duration of employment and time since first employed in the Company Cohort.**

	All Cancer		Lung Cancer		Lymphohaematopoietic	
	O/E	SIR	O/E	SIR	O/E	SIR
Total	143 / 150.34	0.95	26 / 15.29	**1.70***	11 / 13.56	**0.81**
Age (years)						
< 25	2 / 2.31	**0.86**	0 / 0.02	**0.00**	0 / 0.47	**0.00**
25 – 34	13 / 13.92	**0.93**	0 / 0.12	**0.00**	0 / 1.96	**0.00**
35 – 44	20 / 23.59	**0.85**	1 / 0.88	**1.13**	1 / 2.64	**0.38**
45 – 54	32 / 35.10	**0.91**	5 / 3.14	**1.59**	1 / 3.28	**0.31**
55 – 64	56 / 46.86	**1.20**	14 / 6.98	**2.01***	9 / 3.35	**2.69****
65 +	20 / 28.57	**0.70**	6 / 4.15	**1.44**	0 / 1.87	**0.00**
Duration of employment (years)						
1 -4	40 / 48.07	**0.83**	3 / 3.45	**0.87**	2 / 4.95	**0.40**
5 – 9	32 / 33.04	**0.97**	7 / 3.31	**2.12**	3 / 2.95	**1.02**
10 – 14	26 / 25.75	**1.01**	5 / 2.82	**1.77**	0 / 2.19	**0.00**
15 – 19	21 / 17.01	**1.23**	4 / 2.07	**1.93**	3 / 1.42	**2.11**
20 – 24	5 / 11.88	**0.42***	2 / 1.56	**1.29**	2 / 0.97	**2.06**
25 +	19 / 14.60	**1.30**	5 / 2.09	**2.40**	1 / 1.08	**0.92**
TSFE (years)						
<10	42 / 52.94	**0.79**	7 / 4.07	**1.72**	4 / 5.46	**0.73**
10 – 14	39 / 36.55	**1.07**	6 / 3.56	**1.69**	1 / 3.24	**0.31**
15 – 19	31 / 25.23	**1.23**	4 / 2.84	**1.41**	3 / 2.12	**1.42**
20 – 24	7 / 13.77	**0.51**	1 / 1.75	**0.57**	1 / 1.14	**0.88**
25 – 29	10 / 11.30	**0.89**	6 / 1.57	**3.83****	1 / 0.86	**1.16**
30+	14 / 10.55	**1.33**	2 / 1.51	**1.33**	1 / 0.73	**1.37**

	Non-Hodgkin's Lymphoma		Leukaemia	
	O/E	SIR	O/E	SIR
Total	6 / 6.07	0.99	4 / 4.53	**0.88**
Age (years)				
< 25	0 / 0.12	**0.00**	0 / 0.20	**0.00**
25 – 34	0 / 0.80	**0.00**	0 / 0.66	**0.00**
35 – 44	0 / 1.24	**0.00**	1 / 0.85	**1.18**
45 – 54	1 / 1.64	**0.61**	0 / 0.96	**0.00**
55 – 64	5 / 1.51	**3.31***	3 / 1.15	**2.61**
65 +	0 / 0.77	**0.00**	0 / 0.70	**0.00**
Duration of employment (years)				
1 -4	1 / 2.17	**0.46**	1 / 1.66	**0.60**
5 – 9	2 / 1.33	**1.51**	1 / 0.98	**1.02**
10 – 14	0 / 1.00	**0.00**	0 / 0.73	**0.00**
15 – 19	1 / 0.65	**1.53**	1 / 0.47	**2.12**
20 – 24	1 / 0.45	**2.25**	1 / 0.32	**3.09**
25 +	1 / 0.48	**2.07**	0 / 0.37	**0.00**
TSFE (years)				
<10	3 / 2.38	**1.26**	1 / 1.81	**0.55**
10 – 14	0 / 1.49	**0.00**	1 / 1.07	**0.94**
15 – 19	1 / 0.97	**1.03**	1 / 0.71	**1.41**
20 – 24	0 / 0.53	**0.00**	1 / 0.38	**2.64**
25 – 29	1 / 0.39	**2.56**	0 / 0.29	**0.00**
30+	1 / 0.32	**3.17**	0 / 0.26	**0.00**

Table 5.12.3 **Cancer incidence in the Company Cohort according to employment duration in selected departments and exposure categories.**

	Duration of employment (years)								
	Not exposed		1-4		5-14		15+		
	O/E	SIR	O/E	SIR	O/E	SIR	O/E	SIR	p for trend
Department									
Slaughterboard	74 / 82.54	**0.90**	20 / 23.89	**0.84**	25 / 27.21	**0.92**	24 / 16.71	**1.44**	0.06
Meat processing	123 / 134.08	**0.92**	7 / 6.55	**1.07**	7 / 5.27	**1.33**	6 / 4.44	**1.35**	<0.001
Plant services	122 / 126.76	**0.96**	4 / 10.40	**0.38***	12 / 7.99	**1.50**	5 / 5.19	**0.96**	0.52
Biological exposures									
Slaughter/meat	54 / 55.32	**0.98**	28 / 35.32	**0.79**	35 / 37.13	**0.94**	26 / 22.58	**1.15**	0.37
Urine	34 / 32.97	**1.03**	32 / 43.10	**0.74**	43 / 46.47	**0.93**	34 / 27.80	**1.22**	0.03
Faeces	38 / 49.60	**0.77**	30 / 37.04	**0.81**	38 / 38.49	**0.99**	37 / 25.21	**1.47***	<0.01
Blood	44 / 43.26	**1.02**	30 / 41.26	**0.73**	39 / 40.03	**0.97**	30 / 25.79	**1.16**	0.24
Chemical exposures									
Cleaning chemicals	51 / 50.65	**1.01**	28 / 37.92	**0.74**	37 / 38.88	**0.95**	27 / 22.90	**1.18**	0.29
Animal remedies	38 / 39.67	**0.96**	33 / 41.20	**0.80**	40 / 43.62	**0.92**	32 / 25.85	**1.24**	0.15
Organochlorines	34 / 35.08	**0.97**	31 / 41.57	**0.75**	44 / 44.29	**0.99**	34 / 29.40	**1.16**	0.16

* $p < 0.05$
** $p < 0.01$

Table 5.12.4 Lung cancer incidence in the Company Cohort by employment duration in selected departments and exposure categories.

	Duration of employment (years)								
	Not exposed		1-4		5-14		15+		
	O/E	SIR	O/E	SIR	O/E	SIR	O/E	SIR	p for trend
Department									
Slaughterboard	13 / 8.32	**1.56**	2 / 1.91	**1.05**	5 / 2.92	**1.71**	6 / 2.14	**2.80***	0.21
Meat processing	21 / 13.57	**1.55**	2 / 0.52	**3.85**	2 / 0.55	**3.63**	1 / 0.65	**1.53**	0.64
Plant services	20 / 12.59	**1.59***	0 / 0.92	**0.00**	3 / 1.04	**2.89**	3 / 0.74	**4.05***	0.07
Biological exposures									
Slaughter/meat	10 / 6.26	**1.60**	3 / 2.55	**1.17**	6 / 3.69	**1.63**	7 / 2.80	**2.51***	0.30
Urine	6 / 3.68	**1.63**	2 / 3.24	**0.62**	8 / 4.85	**1.65**	10 / 3.52	**2.84****	0.07
Faeces	5 / 4.62	**1.08**	3 / 3.06	**0.98**	8 / 4.25	**1.88**	10 / 3.37	**2.97****	0.02
Blood	6 / 4.83	**1.24**	3 / 3.17	**0.95**	7 / 4.04	**1.73**	10 / 3.25	**3.07****	0.03
Chemical exposures									
Cleaning chemicals	10 / 5.72	**1.75**	2 / 2.83	**0.71**	7 / 3.91	**1.79**	7 / 2.83	**2.47***	0.28
Animal remedies	8 / 4.39	**1.82**	3 / 3.12	**0.96**	6 / 4.51	**1.33**	9 / 3.27	**2.75****	0.21
Organochlorines	6 / 3.80	**1.58**	2 / 3.16	**0.63**	8 / 4.56	**1.76**	10 / 3.77	**2.65****	0.09

* $p < 0.05$
** $p < 0.01$

Table 5.12.5 Lymphohaematopoietic cancer incidence in the Company Cohort by employment duration in selected departments and exposure categories.

	Duration of employment (years)								
	Not exposed		1-4		5-14		15+		
	O/E	SIR	O/E	SIR	O/E	SIR	O/E	SIR	p for trend
Department									
Slaughterboard	7 / 7.36	**0.95**	1 / 2.40	**0.42**	0 / 2.38	**0.00**	3 / 1.42	**2.12**	0.44
Meat processing	8 / 12.12	**0.66**	1 / 0.66	**1.51**	0 / 0.46	**0.00**	2 / 0.32	**6.25***	0.01
Plant services	7 / 11.45	**0.61**	1 / 1.08	**0.93**	2 / 0.64	**3.12**	1 / 0.39	**2.56**	0.03
Biological exposures									
Slaughter/meat	7 / 4.88	**1.44**	1 / 3.58	**0.28**	0 / 3.25	**0.00**	3 / 1.86	**1.62**	0.97
Urine	2 / 2.89	**0.69**	1 / 4.41	**0.23**	4 / 4.01	**1.00**	4 / 2.26	**1.77**	0.05
Faeces	0 / 4.40	**0.00****	2 / 3.79	**0.53**	4 / 3.32	**1.20**	5 / 2.05	**2.44**	<0.001
Blood	3 / 3.80	**0.79**	2 / 4.16	**0.48**	2 / 3.51	**0.57**	4 / 2.10	**1.91**	0.13
Chemical exposures									
Cleaning chemicals	6 / 4.48	**1.34**	2 / 3.82	**0.52**	0 / 3.38	**0.00***	3 / 1.88	**1.60**	0.97
Animal remedies	2 / 3.49	**0.57**	2 / 4.20	**0.48**	3 / 3.76	**0.80**	4 / 2.11	**1.90**	0.06
Organochlorines	4 / 3.02	**1.33**	2 / 4.26	**0.47**	2 / 3.89	**0.51**	3 / 2.39	**1.26**	0.76

* $p < 0.05$
** $p < 0.01$

Table 5.12.6 Non-Hodgkin's lymphoma incidence in the Company Cohort by employment duration in selected departments and exposure categories.

	Duration of employment (years)								
	Not exposed		1-4		5-14		15+		
	O/E	SIR	O/E	SIR	O/E	SIR	O/E	SIR	p for trend
Department									
Slaughterboard	4 / 3.28	**1.22**	1 / 1.07	**0.94**	0 / 1.08	**0.00**	1 / 0.65	**1.55**	0.84
Meat processing	5 / 5.43	**0.92**	0 / 0.29	**0.00**	0 / 0.21	**0.00**	1 / 0.14	**7.02**	0.08
Plant services	3 / 5.13	**0.58**	1 / 0.47	**2.11**	1 / 0.29	**3.48**	1 / 0.18	**5.60**	0.01
Biological exposures									
Slaughter/meat	4 / 2.17	**1.84**	1 / 1.57	**0.64**	0 / 1.48	**0.00**	1 / 0.85	**1.17**	0.48
Urine	0 / 1.29	**0.00**	1 / 1.93	**0.52**	3 / 1.81	**1.65**	2 / 1.04	**1.93**	0.09
Faeces	0 / 1.96	**0.00**	1 / 1.67	**0.60**	3 / 1.50	**2.00**	2 / 0.93	**2.14**	0.04
Blood	1 / 1.69	**0.59**	2 / 1.83	**1.10**	1 / 1.59	**0.63**	2 / 1.06	**1.88**	0.41
Chemical exposures									
Cleaning chemicals	3 / 1.99	**1.51**	2 / 1.68	**1.19**	0 / 1.54	**0.00**	1 / 0.86	**1.16**	0.52
Animal remedies	0 / 1.55	**0.00**	2 / 1.85	**1.08**	2 / 1.70	**1.18**	2 / 0.97	**2.06**	0.16
Organochlorines	2 / 1.34	**1.49**	2 / 1.87	**1.07**	1 / 1.76	**0.57**	1 / 1.09	**0.91**	0.63

Table 5.12.7 Leukaemia incidence in the Company Cohort by employment duration in selected departments and exposure categories.

	Duration of employment (years)								
	Not exposed		1-4		5-14		15+		
	O/E	SIR	O/E	SIR	O/E	SIR	O/E	SIR	p for trend
Department									
Slaughterboard	3 / 2.47	**1.22**	0 / 0.79	**0.00**	0 / 0.79	**0.00**	1 / 0.47	**2.11**	0.78
Meat processing	2 / 4.04	**0.50**	1 / 0.22	**4.50**	0 / 0.15	**0.00**	1 / 0.11	**8.80**	0.01
Plant services	3 / 3.82	**0.79**	0 / 0.36	**0.00**	1 / 0.22	**4.63**	0 / 0.13	**0.00**	0.53
Biological exposures									
Slaughter/meat	3 / 1.65	**1.82**	0 / 1.19	**0.00**	0 / 1.07	**0.00**	1 / 0.62	**1.62**	0.88
Urine	2 / 0.98	**2.04**	0 / 1.47	**0.00**	1 / 1.33	**0.75**	1 / 0.75	**1.33**	0.86
Faeces	0 / 1.47	**0.00**	1 / 1.26	**0.79**	1 / 1.11	**0.90**	2 / 0.69	**2.90**	0.05
Blood	2 / 1.28	**1.56**	0 / 1.39	**0.00**	1 / 1.16	**0.86**	1 / 0.70	**1.43**	0.72
Chemical exposures									
Cleaning chemicals	3 / 1.51	**1.99**	0 / 1.28	**0.00**	0 / 1.12	**0.00**	1 / 0.63	**1.60**	0.84
Animal remedies	2 / 1.18	**1.70**	0 / 1.40	**0.00**	1 / 1.25	**0.80**	1 / 0.70	**1.43**	0.76
Organochlorines	2 / 1.02	**1.96**	0 / 1.42	**0.00**	1 / 1.29	**0.78**	1 / 0.80	**1.25**	0.89

* $p < 0.05$
** $p < 0.01$

As seen in Table 5.12.2 no consistent strong increase with increasing age at risk, duration of employment or time since first employed was observed in this cohort for any specific cancer examined, although the lung cancer relative risk appeared to increase with increasing age at risk, and the non-Hodgkin's lymphoma relative risk increased with increasing duration of employment.

In the analyses of the effect of duration of employment in specific exposure categories, however, cancer risk was seen to be associated with certain exposures. A clear increase in overall cancer incidence was seen among those who worked on the slaughterboard and in meat processing, and also in those exposed to animal urine and faeces, as seen in Table 5.12.3. There was also a consistent increase in risk for lung cancer incidence with duration of employment on the slaughterboard and in plant services, or in jobs with potential exposure to animal urine, faeces and blood, plus the cleaning chemicals, animal remedies and organochlorines, as seen in Table 5.12.4. This trend was significant only for those with exposure to animal faeces and blood. However, in all cases apart from those individuals exposed to animal faeces and blood, lung cancer was also elevated in those not exposed.

Although the numbers were smaller, there was also a clear trend of increasing risk of lymphohaematopoietic cancers with duration of work in meat processing or plant services or of exposure to animal urine and faeces as seen in Table 5.12.5. There was also an apparent increase in non-Hodgkin's lymphoma risk with increasing duration of exposure to work in plant services or to animal urine and faeces, but not with any

chemical exposures, as seen in Table 5.12.6. With only four cases of leukaemia, interpretation of the analysis shown in Table 5.12.7 is difficult. It does appear, however, that there is a clear difference in risk between those with exposure to work in meat processing and to animal faecal matter and those with no exposure, with a trend of increasing risk with increasing duration of exposure.

5.12 Cancer Incidence by level of exposure

In general the numbers were too small to contrast high versus medium exposure to individual biological agents (Table 4.4), so a combined analysis for the three main categories of biological exposures used in tables 5.14 and 5.15 (animal urine, faeces and blood) was conducted. High exposure was defined as work in any of the jobs that involved high exposure to any of these three factors, and medium exposure was defined as any medium (but not high) exposure. Table 5.13 shows the findings for total cancer, lung cancer, lymphohaematopoietic cancers, non-Hodgkin's lymphoma and leukaemia for no, medium and high exposure to biological agents in the Union Cohort, the Company Cohort, and also in the two cohorts combined. No clear pattern of increasing relative risk of cancer with increasing "biological" exposure is evident for any cancer type, apart from lung cancer in the Company Cohort which has a significantly elevated relative risk in the high exposure category (SIR 1.97, 95% CI 1.21 – 3.04, 18 cases).

Table 5.13 Cancer incidence by exposure to biological material from animal urine, faeces or blood.

		Union Cohort		Company Cohort		Combined Cohorts	
	Exposure	**O/E**	**SIR**	**O/E**	**SIR**	**O/E**	**SIR**
All Cancer	None	25 / 25.17	**0.99**	20 / 25.74	**0.78**	60 / 60.69	**0.99**
	Medium	24 / 24.79	**0.97**	33 / 44.83	**0.74**	40 / 57.30	**0.70**
	High	137 / 153.82	**0.89**	90 / 79.77	**1.13**	229 / 236.13	**0.97**
Lung Cancer	None	4 / 3.36	**1.19**	3 / 2.86	**1.05**	8 / 5.54	**1.44**
	Medium	3 / 3.19	**0.94**	5 / 3.27	**1.53**	9 / 7.63	**1.18**
	High	17 / 20.32	**0.84**	18 / 9.16	**1.97****	33 / 28.99	**1.14**
Lymphohaematopoietic	None	3 / 2.11	**1.42**	0 / 2.23	**0.00**	3 / 4.40	**0.68**
	Medium	2 / 2.13	**0.94**	1 / 3.66	**0.27**	5 / 5.88	**0.85**
	High	9 / 12.88	**0.70**	10 / 7.67	**1.30**	17 / 20.40	**0.83**
Non-Hodgkin's Lymphoma	None	2 / 0.95	**2.11**	0 / 0.99	**0.00**	2 / 1.97	**1.02**
	Medium	1 / 0.97	**1.03**	1 / 1.64	**0.61**	3 / 2.64	**1.14**
	High	6 / 5.85	**1.03**	5 / 3.44	**1.45**	10 / 9.23	**1.08**
Leukaemia	None	1 / 0.70	**1.43**	0 / 0.75	**0.00**	1 / 1.48	**0.68**
	Medium	1 / 0.71	**1.41**	0 / 1.22	**0.00**	2 / 1.96	**1.02**
	High	3 / 4.30	**0.70**	4 / 2.56	**1.56**	6 / 6.81	**0.88**

* $p < 0.05$
** $p < 0.01$

Chapter 6 Discussion

6.1 Introduction

These historical cohort studies were undertaken to examine mortality and cancer incidence in a group of workers employed in the New Zealand meat processing industry, and also to investigate associations between specific exposures and any increased cancer risk identified.

The available epidemiological evidence suggests an increased risk of cancers of the lung and larynx, and of lymphohaematopoietic tissue, associated with work in the meat processing industry and, in particular, with work involving animal handling, animal slaughter and exposure to freshly slaughtered meat. The evidence for lung cancer is reasonably consistent, having been reported in several countries across different time periods and in a range of study types including both cohort and case-control studies. Sufficient studies have also adjusted for smoking, either directly or indirectly, to conclude that the excess is probably over and above that which could be attributed to smoking. The case-control studies that have examined cancers of lymphohaematopoietic tissue have consistently shown increased risks, although there is less consistency in the findings from cohort studies.

The cohort studies that have examined cancer risk in meat workers in the USA [Johnson et al, 1986a, 1986b, 1995], the UK [Coggon et al, 1989; Coggon and Wield, 1995], and in Switzerland [Guberan et al, 1993] have been limited by study size, or in

the case of a large Swedish cohort [Boffetta et al, 2000] by relatively crude exposure data.

The case-control studies that have evaluated associations between lung cancer and exposures in the meat industry in the USA [Vena et al, 1982; Johnson 1991], Sweden [Gustavsson et al, 1987], New Zealand [Reif et al, 1989], Germany [Jockel et al, 1998] and Switzerland [Bouchardy et al, 2002] have found inconsistent results. No relationship was found with chemical exposures [Gustavsson et al, 1987], although a dose-response with duration of exposure to raw meat has been observed [Johnson, 1991]. Those that examined cancers of the lymphohaematopoietic system have more consistently shown increased risks [Pearce et al, 1985; Pearce et al, 1986; Pearce et al, 1987; Reif et al, 1989; Loomis & Savitz 1991; Bouchardy et al, 2002] and also a dose-response relationship where this was examined [Metayer et al, 1998; Bethwaite et al, 2001].

Only case-control studies have been conducted to date in New Zealand. These have found a small increase in lung and laryngeal cancer (Reif et al, 1989), with the excess greater than that which could be attributed to smoking. They have also found a consistently elevated risk for cancers of the lymphohaematopoietic system [Pearce et al, 1985; Pearce et al, 1986; Pearce et al, 1987; Reif et al, 1989], with a clear dose-response relationship for leukaemia based on duration of exposure to biological agents [Bethwaite et al, 2001].

As no historical cohort study of meat workers had been conducted in New Zealand, this study was undertaken to establish whether there was increased risk of cancer

incidence or mortality in this occupational group. In particular, the aim was to confirm whether the earlier findings for lung and lymphohaematopoietic cancer in New Zealand case-control studies could be replicated in a cohort study, and also to assess the possible role of various exposures. The processing of meat for export is a significant industry in New Zealand, directly employing approximately 20,000 people, so it was anticipated that a cohort of sufficient size could be assembled to permit analysis of the relatively rare lymphohaematopoietic cancers.

The identification of a study population proved more difficult than anticipated, however, due in part to the reluctance of some companies to provide support for the investigation. Even where support was given, it was found to have been common practice to routinely purge electronic personnel records to delete employees who had resigned, and old hard copy employment records had often been disposed of. Nevertheless, two distinct sources of historical records of employment in the New Zealand meat industry were identified and these permitted the enumeration of two quite separate cohorts.

One cohort (the "Union Cohort") was based on annual membership records of the Canterbury Marlborough Nelson West Coast branch of the New Zealand Meat Workers and Related Trades union, but included only those individuals who were also members of the meat industry superannuation scheme. The final Union Cohort was assembled through a computerised matching of these two datasets, and a total of 4,064 individuals were included. The other cohort (the Company Cohort"), consisting of 6,647 individuals, was assembled directly from historical personnel records obtained from three freezing works owned by two large meat processing companies. Because

of the different methodology used in the enumeration of the two cohorts, and because of significant differences between the cohorts with respect to factors such as gender distribution, age at risk, duration of employment, time since first employment and length of follow-up, they were examined separately.

6.2 Summary of findings

The findings of this study are summarised below, firstly for overall mortality and then for mortality from, and incidence of, the cancers of *a priori* interest in the two cohorts.

6.2.1 Overall mortality

The observed all cause mortality in the 3,916 Union Cohort members included in the analysis was significantly below expected (SMR 0.86, 95% CI 0.76 – 0.98, 246 deaths), as was mortality from most other major disease categories including diseases of the circulatory system (SMR 0.90, 98 deaths) and respiratory system (SMR 0.85, 13 deaths), consistent with a strong healthy worker effect. There was also a deficit in mortality from all cancers (SMR 0.88, 84 deaths), and in all cancer incidence (SIR 0.91, 186 cases).

In contrast to the experience of the Union Cohort, there was elevated mortality from all causes (SMR 1.12, 227 deaths) in the 6,426 members of the Company Cohort included in the analysis. There were major contributions from all cancers (SMR 1.12, 69 deaths), although overall cancer incidence was close to expected (SIR 0.95, 143

cases), as well as from diseases of the circulatory system (SMR 1.15, 73 deaths), respiratory system (SMR 1.10, 10 deaths) and digestive system (SMR 1.49, 6 deaths), and from external causes (SMR 1.27, 56 deaths).

6.2.2 Cancers of *a priori* interest - the Union Cohort

The study focused on the cancers of *a priori* interest, namely lung cancer and lymphohaematopoietic cancers (in particular non-Hodgkin's lymphoma), and the findings of the analyses carried out for these cancers in the two cohorts are outlined below.

The 23 deaths from lung cancer in the Union Cohort were close to expected (SMR 0.99, 95% CI 0.63 – 1.49), even though a significant elevation was observed for deaths from the non-malignant respiratory diseases often related to smoking, namely bronchitis, emphysema and asthma (SMR 2.36, 95% CI 1.18 – 4.23). Of the other diseases of *a priori* interest there were fewer deaths from cancers of lymphatic and haematopoietic tissue than expected (SMR 0.57, based on 5 deaths).

In the stratified analyses performed to investigate the effect of the exposure variables age at risk, duration of employment and time since first employed on all cause mortality, cancer mortality and lung and lymphohaematopoietic cancers no relationship was apparent, nor was there any pattern evident in the analysis of cancer incidence by these criteria. For lung cancer the SMR increased with increasing age, but no pattern was evident with either duration of employment or time since first

employed. Small numbers of deaths from cancer of lymphatic and haematopoietic tissue limited interpretation of the effect of exposure. The five deaths that did occur were in the older age groups, but no trend with increasing duration of employment or time since first employed was evident.

The lower than expected mortality in the Union Cohort persisted in the analysis by stratification on department ever worked in (see Table 5.7.1), although there was a small reduction in the deficit in all cause and cancer mortality among those who had worked on the slaughterboard. The stratification on exposure to potential biological and chemical exposures also produced no change in the observed mortality from any of the cancers of *a priori* interest.

In the analyses performed to investigate the relationship between mortality and duration of exposure to specific exposure categories (Tables 5.10.1 to 5.10.4), lung cancer mortality was elevated in those who had ever been employed on the slaughterboard or in meat processing when compared to those who had never worked in these departments. When this analysis was repeated for lung cancer incidence, a small but consistent increase in risk with increasing duration of employment on the slaughterboard was apparent but the trend was not significant. This pattern was not evident in those ever employed in jobs categorised as having potential for exposure to the slaughter process and/or fresh raw meat, and animal urine, faeces and blood, or to cleaning chemicals. While mortality was elevated in these groups, no clear dose-response was evident. No association was evident for cancers of the lymphohaematopoietic system.

In summary, therefore, there was no evidence of elevated risk of any of the cancers of *a priori* interest among members of the Union Cohort. The internal comparisons provided no evidence of an association between work in this industry and excess risk of these cancers, although there was a small but consistent increase in relative risk of lung cancer incidence with increasing duration of employment on the slaughterboard.

6.2.3 Cancers of *a priori* interest - the Company Cohort

In contrast to the experience of the Union Cohort, significant excess mortality from lung cancer (SMR 1.79, 23 deaths), and excess mortality from non-Hodgkin's lymphoma (SMR 1.45, 4 deaths), was observed in the Company Cohort. Excess mortality from diseases of the circulatory and respiratory systems was also observed, and the excesses were marked for pulmonary circulation and other heart diseases (SMR 1.78, 12 deaths) and for cerebrovascular disease (SMR 1.56, 13 deaths), and highly significant for bronchitis, emphysema and asthma (SMR 2.97, 9 deaths), indicating no healthy worker effect operating in this cohort.

A clear (but inconsistent) increase in lung cancer mortality was evident with both increasing duration of employment and time since first employed (Table 5.5.2), with highly significantly increased SMRs occurring in those with both the longest duration of employment and time since first employed. A similar pattern was evident in the analysis of lung cancer incidence (Table 5.12.2). Only six deaths from cancers of lymphatic and haematopoietic tissue were observed in this cohort, and there was no consistent association between mortality and duration of exposure or time since first employed. The eleven cases in the incidence analysis showed a trend of increasing

risk with increasing duration of employment (Table 5.12.2), however small numbers again limited interpretation for specific subtypes.

The stratification by department ever worked in (Table 5.7.2) showed that the excess all cause mortality in the Company Cohort was primarily due to a significantly elevated risk in those who had worked on the slaughterboard (SMR 1.23, 113 deaths). Although the numbers were small, excesses were also observed for the cooling floor (SMR 1.63, 6 deaths) and fellmongery (SMR 1.29, 11 deaths). The excess in overall cancer mortality was also primarily due to elevated risk in the slaughterboard (SMR 1.23, 34 deaths). Significant trends of increasing mortality from all causes with increasing duration of employment (Table 5.10.5) were seen in the slaughterboard ($p=0.01$), and in those with exposure to animal faeces ($p<0.001$) and organochlorines ($p=0.01$). Similar significant trends for all cancer mortality were seen (Table 5.10.6) in those with exposure to animal urine ($p=0.01$), faeces ($p=0.01$) and blood ($p=0.02$), and to animal remedies ($p=0.02$) and organochlorines ($p<0.01$).

In the analysis of lung cancer mortality by departments (Table 5.7.2) significant elevations were observed in the slaughterboard workers (SMR 2.24, 13 deaths), with smaller contributions also from meat processing (SMR 2.63, 4 deaths) and plant services (SMR 2.21, 5 deaths). The risk of dying from lung cancer was highly significantly elevated for those with potential exposure (see Table 5.8.2) to animal urine (SMR 1.96, 19 deaths), animal faeces (SMR 2.23, 20 deaths) and blood (SMR 2.18, 19 deaths), and significantly elevated for those with exposure to slaughter/raw meat (SMR 1.99, 15 deaths). Lung cancer mortality was also significantly elevated in those potentially exposed to cleaning chemicals (SMR 1.88, 15 deaths), animal

remedies (SMR 1.86, 17 deaths) and organochlorines (SMR 1.98, 19 deaths), as seen in Table 5.9.2. Significant trends of increasing lung cancer mortality with increasing duration of employment (Table 5.10.7) were also observed for those with exposure to animal urine ($p=0.04$), faeces ($p=0.01$) and blood ($p=0.02$) with the relative risk in those with more than 15 years employment in these exposure categories more than trebled.

There was a clear and consistent trend of increasing relative risk of lung cancer incidence with increasing duration of employment (see Table 5.12.4) on the slaughterboard and in plant services, although in neither was the trend significant. There was, however, a significant trend for those with potential exposure to animal faeces ($p=0.02$) and blood ($p=0.03$), with the risk trebled for those with more than 15 years employment. In the analysis of the combined effect of exposure to biological material from animal urine, faeces and blood a clear dose-response for lung cancer incidence is evident in Table 5.13.

Of the six deaths in this cohort from cancers of lymphohaematopoietic tissue, as seen in Table 5.7.2, three were individuals who worked in plant services (SMR 2.99) and two of the four who died from non-Hodgkin's lymphoma worked in plant services (SMR 4.54). A significant trend ($p<0.01$) of increasing mortality from cancers of the lymphohaematopoietic system with duration of employment in plant services was also observed (Table 5.10.8). As seen in Table 5.8.2, mortality from non-Hodgkin's lymphoma was non-significantly elevated in those with exposure to animal urine (SMR 1.86, 4 deaths), faecal matter (SMR 2.13, 4 deaths) and blood (SMR1.54, 3 deaths), but not in those with potential exposure to slaughter/raw meat (SMR 0.58, 1

death). There were also excess deaths from non-Hodgkin's lymphoma in those with potential exposure to animal remedies (SMR 1.98, 4 deaths) and organochlorines (1.42, 3 deaths), as seen in Table 5.9.2, although these are exposures that are experienced in common with the biological exposures and it was not possible to separate their effects.

In the incidence analyses a significant increased incidence of lymphohaematopoietic cancers with increasing duration of employment was observed (in Table 5.12.5) in those employed in meat processing ($p=0.01$) and plant services ($p=0.03$), and in those exposed to animal faeces ($p<0.001$). As seen in Table 5.12.6, non-Hodgkin's lymphoma incidence increased with increasing duration of employment in plant services ($p=0.01$) and exposure to animal faeces ($p=0.04$). Leukaemia incidence was also observed to increase significantly ($p=0.01$) with increasing duration of employment in meat processing (Table 5.12.7).

In summary, therefore, significantly elevated lung cancer mortality and incidence was observed in the Company Cohort, and also excess mortality from non-Hodgkin's lymphoma. Lung cancer risk increased with increasing duration of employment, and was most strongly associated with biological exposures associated with animal urine, faeces and blood that are present in the slaughterboard, meat processing and plant services departments - where the increase with increasing duration of employment was even stronger. The analysis of lymphohaematopoietic cancer mortality provided less evidence of elevated risk associated with work in this industry because numbers were small, although there was an association with increasing exposure to work in

plant services and meat processing. This effect also appears to be associated most strongly with exposures to biological material in animal urine, faeces and blood.

6.3 Limitations of the data.

As in any observational epidemiological study of this type there are a number of potential sources of bias.

6.3.1 Cohort definition.

The two cohorts included in this investigation were defined in different ways, one based on membership of a Union and the other on workers employed in three separate freezing works, which resulted in the assembly of two distinct study populations. Information was assembled from the two sources (union or company records) in order to yield sufficient overall numbers. In general the two cohorts have been analysed separately because they involve different data sources and different types of workforce. It is useful, however, to compare and contrast the findings of the two studies in order to assess their consistency.

The Union Cohort was based on union membership records, but comprised only those individuals who were also members of a meat industry superannuation scheme. The union membership records, compiled annually from 1969 to 1998, contained a total of 157, 238 discrete records. When consolidated into unique records for individual workers, detailing each individual's work history, this contained 34,887 unique individuals. When matched electronically to the file containing individual

superannuation scheme members, this yielded a total cohort of 4, 064 individuals. Therefore, a very select group of only 12% of the eligible union members were included in the study. It would be reasonable to assume that that this group would differ materially from the remainder of the union members excluded from this cohort. The act of making provision for their future by joining the superannuation scheme would suggest an interest in their own future wellbeing, which may manifest in their being more likely to modify lifestyle factors that would impact on their health. They are almost exclusively male, and are a very stable workforce with an average duration of employment almost double that of the members of the Company Cohort (13.2 compared with 7.1 years).

The Union Cohort is, therefore, a very select group, and as can be seen from both the mortality and cancer incidence analyses it exhibits the typical "healthy worker effect". The Company Cohort also consists of a population that was healthy enough to obtain and retain employment, and would normally be expected to exhibit a healthy worker effect, but overall mortality in this cohort was elevated.

6.3.2 Follow-up of the cohorts.

The follow-up of both cohorts to determine mortality and cancer incidence was through computerised searches of New Zealand Health Information Service records, and for additional vital status information through computerised searches of the pension records of Work and Income New Zealand (WINZ) and of the electronic electoral roll. Registration of deaths is thought to be virtually complete in New Zealand [Brown & Frankovich, 1998], with registrations coming from multiple

sources including certificates of causes of death from doctors or coroners, post-mortem reports from private pathologists and hospitals, and death registration forms completed by funeral directors [New Zealand Health Information Service, 2003]. New Zealand has had a cancer registration scheme operating since 1948 and cancer registration is also believed to be virtually complete in New Zealand, although it is recognised that for certain types of cancer (mainly melanoma of the skin, cancers of the female breast and early bowel cancers) there was significant under-reporting prior to the introduction of the Cancer Registry Act 1994.

However, even during the period 1990 – 1993 when an assessment of the accuracy and completeness of registration of childhood cancers in New Zealand was made using capture-recapture methods, the New Zealand Cancer Registry was found to ascertain 97% of incident cases [Dockerty et al, 1997]. It is unlikely, therefore, that there is any significant information bias related to incomplete ascertainment of outcome in this study, with the qualification that the matching between the cohorts and national mortality and cancer datasets included manual matching by New Zealand Health Information Service staff based on similarity of name and date of birth.

Incompleteness of follow-up is a potential source of selection bias in cohort studies, but only where the degree of incompleteness differs in the groups being compared. Ascertainment of vital status in this study was by the same method for all members of both cohorts, and was therefore unlikely to introduce significant bias. As the Union Cohort was a more stable workforce the follow-up was more complete (with only 8% of participants lost to follow-up compared with 16% in the Company Cohort), although the difference was less marked when comparing the percentage of theoretical

person-years (93.27% compared with 91.52% in the Company Cohort). This rate of follow-up is comparable to that achieved in recent New Zealand occupational cohort studies, which ranged from 90% [Firth et al, 1999] to 95% of person-years [Fawcett et al, 2002], and is at the level that is reasonably achievable given the lack of a national population registration system in New Zealand.

A feature of the New Zealand meat industry is its export orientation, and the workforce includes Muslim migrants brought into the country to perform Halal slaughtering to satisfy the requirements of certain markets. Most of these individuals return to their country of origin at completion of their contracts, and are therefore lost to effective follow-up concerning health outcomes. This is a relatively small number of individuals, but equally they are employed in a job with a high putative risk of exposure to biological agents e.g. live animal contact, blood, and the slaughter process. It is also possible that other migrants such as those from the Pacific Islands would return to their countries of origin on retirement, and would also effectively be lost to follow-up. There is no available source of information in New Zealand that would allow quantification of loss to follow-up through emigration.

6.3.3 Confounding

While there is no source of data on ethnicity in these cohorts, given the regional differences it would be reasonable to assume significant differences between the two with respect to the proportion of Maori in the study populations. The Union Cohort was based in Canterbury and would be unlikely to include a higher proportion of Maori than the general population, which was 14.5% in 1996 [www.stats.govt.nz],

while the two Hawkes Bay sub-cohorts in particular could be expected to contain a higher proportion of Maori. In a recent study of meat workers from this area who experienced involuntary job loss, it was established that 51% and 45% of workers employed in two freezing works in the Hawkes Bay during the period 1986 to 1994 were Maori [Keefe et al, 2002]. The proportion of Maori in the larger Lorneville sub-cohort is likely to be below that in the Hawkes Bay sub-cohorts, but still greater than in the general population. Assuming that 25% of the Lorneville sub-cohort are Maori, and using the higher 50% estimate from the study of Keefe et al [2002] in the two Hawkes Bay sub-cohorts, the overall proportion in the Company Cohort would be likely to be in the range 35-40% Maori or just over twice the proportion in the general population.

As in most historical cohort studies, no source of information on smoking among cohort members was available so there is the potential for uncontrolled confounding. This is particularly relevant to the findings related to lung cancer, although the effect of confounding by smoking in studies of occupational cancer is often relatively weak, as differences in smoking rates between groups of manual workers are fairly small. For example 65.4% of food and beverage workers had ever smoked compared with 59.6% of the total full time labour force (and 46.8% current smokers versus 37.7% respectively) in 1981 New Zealand census data [Reif et al, 1989]. Even where comparisons are made with national mortality rates, the most extreme differences in smoking status are unlikely to account for a relative risk of greater than 1.5 [Checkoway et al, 1989]. In the earlier New Zealand study the odds ratio for lung cancer attributable to the higher smoking rates among meat workers was estimated using the method of Axelson [Axelson, 1978] to be 1.20 [Reif et al, 1989].

It is possible that the higher smoking rates among workers in this occupational class are attributable solely to the higher proportion of Maori in the meat industry, as it has been estimated that 50% of the adult Maori population were current smokers in the 1990s [Pomare et al, 1995]. Mortality from lung cancer among adult Maori males in the period 1987 to 1991 was 1.4 times that of non-Maori New Zealanders, while for adult Maori females it was 2.8 times. Using the assumption that the Company Cohort (20% female) was 40 % Maori, compared with approximately 15% in the general population, the relative risk of lung cancer attributable to ethnicity can be calculated to be:

Rate in cohort	=	60*1 + 32*1.4 + 8*2.8	= 127.2
Rate in general population	=	85*1 + 7.5*1.4 + 7.5*2.8	= 116.5

Relative risk = 127.2/116.5 = 1.09

Thus, the high smoking rates in this cohort would only account for a relative risk of lung cancer of about 1.2, while the high proportion of Maori would only account for a relative risk of about 1.1. These cannot be simply multiplied together because each "bias" partly depends on the other, but these estimates do show that the total confounding effect of smoking and ethnicity on lung cancer relative risk in the company cohort is likely to be less than 1.25.

Another method of assessing the potential impact of differences in smoking rates on findings of excess lung cancer is to examine mortality from cancers of the oral cavity, oesophagus, larynx, bladder, pancreas, and kidney. These cancers are recognised as being related to smoking [Stewart and Kleihues, 2003], and would also be elevated in

a cohort with higher rates of smoking. A summary estimate of mortality from these cancers in the Company Cohort can be calculated (SMR = 1.31, 10 deaths), which suggests that a contribution to the excess lung cancer is from smoking (and/or ethnicity), although there is no excess in incidence of these cancers (SMR = 0.90, 15 cases). Mortality data would normally be used in such comparisons to avoid the possibility of bias through incomplete ascertainment of incident cancers compared with mortality records; however as noted previously registration to the New Zealand Cancer Registry is virtually complete. The inconsistency between the mortality and incidence summary estimates, while the lung cancer SMR and SIR do not differ substantially, casts doubt on the magnitude of the contribution made by smoking. It should also be noted that one of the smoking related cancers, laryngeal cancer, has also been found in other studies to be associated with exposures in this industry [Guberan et al, 1993; Boffetta et al, 2000] and also to have been elevated above levels that could be attributed to smoking [Reif et al, 1989; De Stefani et al, 1998].

6.3.4 Exposure data.

As noted in the discussion of exposures in the meat industry in Chapter 2, the published literature on exposures of meat workers is sparse. The exposure categories established for this study were, therefore, based largely on workplace observations and on extrapolation from published literature related either to food quality in meat works or to occupational exposures present in similar environments.

The cohort members were classified primarily according to the department they had ever worked in, although this was extended to include specific biological and

chemical exposures within these departments. Biological exposures such as live animal contact, the slaughter process and contact with raw meat, animal urine, faeces and blood, and to pelts and hides were classified separately, and different jobs were grouped according to their potential for these exposures. In addition, a range of potential chemical exposures, different from those related to curing or wrapping of meat products that have been the focus of previous studies, were included in the exposure assessment part of this study. These included welding emissions, refrigerant gases, cleaning chemicals, and residues of hormone growth promoters and organochlorine pesticides found on animal pelts, meat or urine.

It was also intended that the classifications would extend to an estimate of the likely intensity of each exposure, and individual job titles were assigned intensity ratings. This was never included in the analyses performed in this study, however, due in some instances to small numbers in each category, and in the company cohorts in particular to job codes which identified department or work area only and not specific job titles. In some categories the numbers involved were too small to include, for example no one in the Union Cohort and only 46 in the Company Cohort were classified as exposed to welding emissions. Similarly the hormone growth promoters were used only in cattle, while the vast majority of individuals in both cohorts processed only sheep meat.

There was considerable overlap in the exposure categories used. For example the only individuals deemed to have live animal contact were those already classified on the basis of their employment in the stockyards, but there were also clear distinctions between departments where the potential biological exposures in particular were

applied. For example those employed on the slaughterboard will have potential exposure to the slaughter process and to raw meat, as well as to urine, faeces, blood and possibly to chemical residues. When the classification is based on the potential biological exposure, however, this will span departments. For example animal faeces will be encountered in the stockyards, the slaughterboard and in the processing of casings or runners in the meat-processing department, as well as by those involved in plant cleaning operations, laundering of staff clothing, and in plumbing and drainage work. The animal urine exposure will occur primarily in the stockyards and slaughterboard, and also in certain plant services jobs, whereas blood contact is most likely in the slaughterboard and certain meat processing operations. Similarly, contact with animal pelts and hides will occur in both stockyards and in fellmongering operations.

In the absence of precise exposure information, individual study subjects must be categorised according to exposure on the basis of the best information available. In this study it was possible to compile a full work history for virtually all study subjects, from either company personnel or union membership records, in many cases down to the level of job title. The job title, therefore, could be used as a surrogate to represent a defined profile of exposures that those doing that specific job could be expected to have experienced. Even among workers with the same job title, however, it is recognised that there is considerable "between worker" variability, in both a quantitative (i.e. level of exposure) and a qualitative (agents of exposure) sense, which is in addition to the "within worker" or day-to-day variability commonly observed in occupational exposures [Boleij et al, 1995]. This means that the use of job titles to characterize exposure has limitations, as it may reflect dose in only a limited way, and

misclassification of exposure is inevitable when using job title as a surrogate of exposure. This misclassification is non-differential, however, so its effect would be to dilute any true association between the exposure and the outcome and consequently lead to an underestimation of the strength of that association [Checkoway et al, 1989].

While the primary categorisation of exposure in this study was based on workers' departments and job titles, grouping these into categories based on potential for exposure to the range of biological and chemical agents represents a more detailed exposure assessment than has been used in previous meat worker cohorts. Exposure categorisation in these earlier studies has been based on very broad categories such as exposure to live animals, warm meat, chilled meat or bacon process and products [Coggon et al, 1989], to ever having worked in abattoirs, meatpacking plants, meat department of supermarkets, chicken slaughtering plants and non-meat companies [Johnson 1989; Johnson 1994; Johnson et al, 1986; Johnson et al, 1995] or in the large Swedish cohort to having been classified as butchers or meat workers in one or more consecutive censuses [Boffetta et al, 2000].

In addition to the more detailed exposure assessment used in this study, the two study populations are relatively large compared to the 1,610 member cohort from the United Kingdom [Coggon et al, 1989] and the 882 member cohort from Switzerland [Guberan et al, 1993]. The Baltimore union cohort studied by Johnson and colleagues consisted of 28, 901 individuals, analysed as sub-cohorts of 13,844 white males [Johnson et al, 1986a], 7,261 white females [Johnson et al, 1986b] and 5,145 non-white males [Johnson 1989] although each sub-cohort included workers from non-meat companies as comparison groups. The Swedish cohort included 25,049

individuals with follow-up from 1971 to 1989, although as noted above this study was limited by exposure assessment. The current study, therefore, combines a more detailed exposure assessment with two cohorts of sufficient size for examination of all but the most rare cancers. It was not possible, however, to isolate the effects of the different exposures (for example blood and urine) because of significant overlap and/or relatively small numbers in certain categories.

The prevalence and magnitude of exposure to certain agents, both biological and chemical, will have changed over time in the New Zealand industry. For example brucellosis exposure of meat workers engaged in the slaughter of cattle would have increased dramatically with the introduction of the eradication programme in 1969, would have continued at an elevated rate until the early 1980s while reactor cattle were being selectively culled for slaughter, then would have been eradicated entirely by the end of the 1980s [personal communication, Glass, 2002]. More recently, a similar programme of selective culling of BLV infected cattle, which began in 1997, would have increased the potential for exposure of meat workers engaged in the slaughter of cattle to this known animal oncogenic retrovirus [Hayes, 2002].

Exposure to a range of organochlorines would also have been widespread in the 1950s, both through pesticide residues (DDT, dieldrin and lindane) present on animals being slaughtered and through the use of chlorophenols as fungicides in pelt preservation in fellmongeries. The residues would have progressively reduced with the phasing out of the use of these pesticides, although as noted in Chapter 3 low levels of exposure may still occur, and the chlorophenols used in pelt preservation would have been withdrawn by about 1990 when a general prohibition on their

importation and use was implemented in New Zealand [Buckland et al, 2001]. The period of observation in this study did not permit examination of possible trends associated with these changes. It is possible, however, that the increasing relative risk observed with increasing duration of exposure could be related to either the latency period between disease induction and manifestation, or to an effect related to an exposure that occurred in the past but which is now reduced or eliminated.

6.4 Summary

Notwithstanding the limitations of these two studies associated with the potential for misclassification of exposure and uncontrolled confounding by ethnicity and smoking, there are two key findings of this study that are of considerable interest. The first key finding is that there is an excess of lung cancer in the Company Cohort, for which there is a strong dose-response relationship based on duration of exposure in certain departments and which is most strongly associated with exposures to biological material in animal urine, faeces or blood. The second finding is that despite very small numbers there is evidence of a dose-response relationship between both mortality from, and incidence of, cancers of the lymphohaematopoietic system with increasing duration of work in meat processing and plant services, and particularly with increasing exposure to animal faeces. This effect appears to exist for non-Hodgkin's lymphoma, and possibly also for leukaemia.

These findings for lung cancer in the Company Cohort are consistent with previous findings for meat workers from previous cohort studies, although the overall lung cancer SMR of 1.79 is higher than all the studies except from the SMR of 2.1 reported

by Johnson et al [1989] for non-white males in abattoirs. The level of risk observed in the internal analyses in this study, i.e. up to a three fold elevation for specific departments or exposures with the longest duration, is also higher than any reported previously in cohort studies. The exposures found to be most closely associated with excess lung cancer risk in this study are also similar to those identified previously, although this study indicates that the strongest associations are with biological material from animal urine, faeces and blood rather than the association with the slaughter process and contact with raw meat implicated in previous studies. The findings for lymphohaematopoietic cancers in this study are not strong, and are similar to the findings of earlier cohort studies.

Case-control studies conducted previously have reported highly variable relative risks for lung cancer, with most being lower than the levels observed in this study. The only previous case-control study that reported analyses of the effect of dose [Johnson, 1991] reported higher levels of relative risk associated with duration of contact with raw meat, i.e. up to a relative risk of 13.1 in those with contact with raw meat in abattoirs for a period in excess of 5 years. Apart from the potential for information bias in a case-control study, it is also possible that a similar effect in the New Zealand study would have been attenuated by non-differential exposure misclassification. By contrast several case-control studies, including a series conducted in New Zealand, have observed significant excesses in risk of lymphohaematopoietic cancers, although few cohort studies have observed excess deaths or cases due to small numbers. This study has not found the excess risk of lymphohaematopoietic cancers observed in previous New Zealand case-control studies, although the association between

observed between increasing dose and increasing risk suggests that the effect may be real.

There is no obvious reason for the disparity between the findings for the Union and Company cohorts, although each cohort was defined in a different way and this resulted in the assembly of two distinct study populations. The final Union cohort was a very select group of individuals comprising only 12% of the potential cohort, that is of those identified in union membership records who were also members of the meat industry superannuation scheme. It would be reasonable to assume, therefore, that the act of making provision for their retirement would suggest an interest in their own future well being that may manifest in their being more likely to modify potentially adverse lifestyle factors. There is also a possibility of differences between the two cohorts with respect to both ethnicity and smoking status, the two most powerful potential confounders for lung cancer, although no data on either was available. The differences between the two cohorts with respect to either smoking or ethnicity are unlikely to exceed those in comparisons with the general population, however, so the overall confounding effect would be insufficient to account for the differences observed. Furthermore, while these potential sources of bias could account for the strong healthy worker effect evident in the Union cohort, they could not explain the absence of the dose response relationship that was observed in the Company cohort. There were, however, a number of differences between the cohorts with respect to characteristics that may reflect differences in their potential for exposure. For example the average duration of employment in the Union cohort was almost double that in the Company cohort, and significantly fewer (16% compared with 53%) had worked for less than 5 years. Given the status that seniority affords in this industry, it is possible that members of the Union cohort were more likely to work in the cleaner jobs with

relatively lower exposure to biological agents. Union cohort members had also worked in a greater variety of departments than those in the Company cohort. While it is possible that their greater job mobility was due to the manner in which the cohort was assembled, i.e. following individuals through union membership as they changed employers, it may also reflect a more mobile workforce in which exposure contrasts were reduced.

6.5 Concluding remarks

In conclusion, therefore, this study has demonstrated a significant excess of lung cancer in the Company Cohort. It is not possible to completely rule out the possibility of confounding by smoking and/or ethnicity, but as demonstrated it is highly unlikely that either is sufficient to account for more than a small part of the excess observed. The strong dose-response relationship observed also supports the hypothesis that the effect is related to occupational exposures, and in particular to some component of the biological material contained in animal urine, faeces and blood. The study has also provided some limited support for the previous findings of excess risks of leukaemia and non-Hodgkin's lymphoma associated with work in the New Zealand meat industry, due primarily to the association observed between increasing risk and increasing duration of exposure to the same biological material.

While the observed number of deaths from lung and lymphohaematopoietic system cancers in this study is relatively small, the public health significance of occupational cancer risks such as these is considerably higher than the raw numbers suggest. While not as great a burden as that from smoking and diet, occupational cancers result from

exposures that are both involuntary and theoretically preventable through the elimination of exposure. Furthermore, it is significant that these occupational risks are borne disproportionately by different sectors within society, specifically by groups with lower socio-economic status and by ethnic minorities as was the case in the cohorts studied. Nevertheless, during the period for which the Company cohort was under observation twenty-three deaths from lung cancer were observed, compared with thirteen expected on the basis of comparison with national rates. If a third of this excess could be attributed to differences in rates of smoking and/or ethnicity, this leaves between six and seven lung cancer deaths that could be attributed to occupational exposure. This equates to approximately 25-30% of the lung cancer deaths occurring in this group of workers being attributable to occupational exposures, which is consistent with estimates of the proportion in other blue collar working populations [Boffetta et al, 1995]. Given the size of the meat industry worldwide, and the number employed, the absolute number of cases arising from an effect of the magnitude found in this study would be significant.

Appendix 1

Ethics Committee Approval

Wellington Ethics Committee

99/94 Work related risk of cancer in meat workers

Wellington Ethics Committee

Room 425, Fourth Floor
Community & Support Services
Wellington Hospital
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Phone (04) 385 5999 ext. 5185
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6 October, 1999

Mr Dave McLean
HRC Training Fellow
Department of Medicine.
WELLINGTON SCHOOL OF MEDICINE

Dear Ms McLean

99/94 - Work related risk of cancer in meat workers

Your application for Ethics Committee approval for the above study was considered by the Ethics Committee at its meeting on 28 September 1999.

The key ethical issue in the study is whether you as researcher should be given the names of people who have worked in the meat industry in order to match these with names on the Cancer Registry. The Committee agreed that it is clearly impossible for you to obtain informed consent from all these people, at least 10,000 and probably considerably more than this, for consent to study their data. The Committee was also satisfied that the importance of the study justified the granting of ethical approval for their names to be given to you so that the study can be carried out.

Consequently, ethical approval for the above study is granted by the Wellington Ethics Committee.

It is a condition of Ethics Committee approval that you provide a brief progress report no later than October 2000 and at the completion of the study a copy of any report/publication for the Committee's records. Please notify the Committee if the study is abandoned or changed in any way.

We wish you well with your research.

Yours sincerely

Sharron Cole
CHAIRPERSON

Please include the reference number and study title in all correspondence.

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