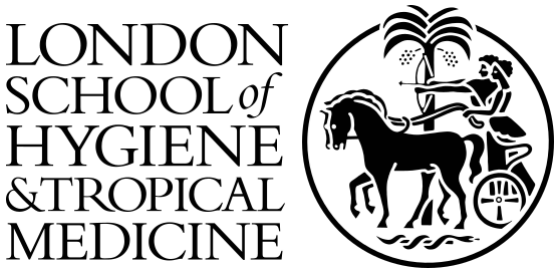

MORTALITY PATTERNS AMONG CIVILIAN WORKERS IN ROYAL NAVAL DOCKYARDS

by

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ABSTRACT

This is a study of asbestos related disease in civilian workers at 3 Royal Naval Dockyards, namely: Devonport, Chatham and Portsmouth. Past work in these dockyards, along with Rosyth in Scotland (undertaken by the Institute of Naval Medicine and the Medical Research Council), has shown that just under 5% of this workforce might be expected to have radiographic abnormalities due to asbestos exposure. In the early 1970s workers in all 4 of these dockyards were invited to participate in health surveys, in which chest x-rays were performed and a health/employment history questionnaire given.

This work is an exact 17 year follow-up of these health surveys, analysing cause specific mortality its time trends and their correlates. The working population of the 3 dockyards, including female industrial workers and 'outstation' male workers was 32,931. However, excluding female workers and absolute non-responders reduced this to 28,265 male workers. The trace rate of this population, over the 17 years, was 97.3% (Rosyth with a rate of less than 70% was excluded from this analysis), 18% of the population traced were found to be dead. The mortality patterns of this cohort were inspected on a yearly basis by the use of a regionally adjusted SMR analysis. Expected rates were calculated, using the OPCS historic mortality data files, to provide a reference set of background mortality levels.

The striking result from this study is one of no excess risk due to lung cancer at the three dockyards, producing SMRs of: 99 (95%CI: 87-122) at Devonport, 85 (95%CI: 70-101) at Chatham, and 94 (95%CI: 81-106) at Portsmouth [$X^2 = 1.8$, $P > 0.1$]. However, an excessive risk was seen for pleural mesothelioma that produced SMRs of: 1983 (95%CI: 1505-2461) at Devonport, 1638 (95%CI: 1049-2437) at Chatham, and 1042 (95%CI: 693-1506) at Portsmouth [$X^2 = 8.4$, $P < 0.025$]. Excesses were also seen for peritoneal mesothelioma and asbestosis. No obvious relationships were seen when analysing lung cancer mortality by employment and asbestos exposure variables. A dose-response of lung cancer mortality to smoking habit was the only clear relationship found. Log-linear modelling supported the SMR findings of no overall excess or deficiency of lung cancer mortality compared to an excess of mesothelioma deaths. These results and their significance are discussed.

"If you poison your boss a little bit each day it's called murder; if your boss poisons you a little each day it's called a Threshold Limit Value."

J P Keogh

NUMEROUS ORIGINALS IN COLOUR

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Chapter 1: INTRODUCTION

1.1 General Introduction.

This work is concerned with asbestos exposure and asbestos-related disease in shipbuilding and repair. In particular it is concerned with the risk of disease and death among civilian employees of the four Royal Naval Dockyards: Devonport, Chatham, Portsmouth and Rosyth. At any time in the post World War II period there have been some 50,000 civilians employed in these four yards, all potentially with some risk of asbestos exposure from the insulating materials used in shipbuilding and repair. In this study the mortality patterns of these civilian workers will be considered with regard to intensity and duration of such exposure.

Historically this country has always had a strong seagoing and Royal Naval tradition, from the early days of the Spanish Maine and the Spanish Armada, to Lord Nelson and Trafalgar, to more recently the World Wars and, lastly, to the Falklands and Gulf Wars. Throughout these times and conflicts there have been four main Royal Dockyards serving the fleet in this country at Devonport, Chatham, Portsmouth and Rosyth (figure 1.1). These dockyards in past times built and repaired Royal Ships of the Line, battleships of the Royal Navy. However, as iron replaced wood the nature of these yards changed. Now the main work of Royal Dockyards is the refitting and repairing of navy ships, and tourism. This century has seen many changes, with not the least of these being the modernization of the Royal Navy and with it the closure and partial closure of Naval Dockyards. Linked with this closure are the appearances of Naval Museums, Historic Dockyards, as at Chatham and Portsmouth. These Historic Dockyards bring money into the exchequer and tourists into the dockyards, giving an insight into both naval history and past dockyard working conditions.

Royal Naval Dockyards.

FIGURE 1.1: Royal Naval Dockyards.

Consequently in this century, the work of the Royal Dockyards has been mostly in refit and repair rather than shipbuilding, this last activity now being commonly undertaken in civilian contract yards. When civilian/commercial yards undertake refits, the extent of these can differ from the naval version. Naval refits are more frequent and extensive than merchant refits, routinely involving the complete removal and replacement of all insulating material in machinery spaces and both the environmental and structural insulation. As the removal of this material produces more dust than its application these should be considered very important differences.

When combined with the many engineering, construction, and workplace differences between naval and merchant ships, naval shipping containing much more machinery in smaller compartments and having no portholes, it is likely that the overall exposure to asbestos dust was higher in Naval Dockyards than in their commercial counterparts. It should be noted that asbestos has no respect for frontiers or nationalities and that in this respect the shipbuilding risk of asbestos exposure has been worldwide, from the UK to the USA to Japan.

1.2 Asbestos Materials in Shipbuilding.

The asbestos materials used in Royal Navy ships are largely the same as those used in Merchant ships only the amount applied differs. The various types of asbestos fibre used, have included chrysotile (white serpentine asbestos) and the iron silicates, crocidolite (blue asbestos) and amosite. Changes in the amount and type of material used may explain the emergence of asbestos-related diseases in dockyards, in particular asbestosis, lung cancer and mesothelioma.

Until the early 1950s most of the machinery insulation aboard naval ships was in the form of asbestos mattresses, that is, magnesia sections containing an amount of amosite asbestos (commonly 15-30% amosite), and asbestos cloth made entirely of chrysotile asbestos. These materials provided adequate insulation to the operating steam temperatures of below 750°F that existed until the 1950s. A

certain amount of crocidolite asbestos would have been used in asbestos board and in magnesia sections as an available replacement for amosite. Crocidolite was also used in a sprayed asbestos process from the end of the war until the mid 1960s when this process was stopped.

Over the 1950s steam temperatures rose to 850°F and more efficient insulation was required. Magnesia sections were replaced by amosite sections (containing 90-95% amosite). A decreasing stock of amosite lagging was still used through the early 1960s with calcium silicate sections (containing 10-15% amosite) introduced as its replacement in this period. Operating temperatures aboard ship had by this time reached 950°F.

From 1968, only materials containing chrysotile asbestos were used in naval dockyards. Presently shipping worldwide contains little asbestos insulation, man-made mineral fibres are now used as its substitute. Obviously, the use of large amounts of amosite section and crocidolite in spraying processes up to the mid 1960s implies a corresponding increase in potential asbestos exposure from insulation removal, over the preceding decades.

There have been many uses of asbestos containing materials in ships, apart from heat insulation. Table 1.1 lists some of these other materials. This table has been arranged into those uses producing dust in their handling and those not. The non-dusty uses are those that would not usually produce dust unless the materials were ground, polished or sawn. We see from table 1.1, that although most of the asbestos aboard ship has been used for heat insulation it could also be found in sound and electrical insulating materials.

TABLE 1.1*: Asbestos materials used in Naval Dockyards.

Dusty	Non dusty
Blankets	Cloth (treated)
Cement	Condenser packing
Cloth (untreated)	Sheets (compressed fibre)
Cord	Gaskets
Fibre	Oilproof jointing
Millboard	Compressed fibre jointing
Packing fibre	Graphite packing
Rope	Rings
Soft sound insulation	Compressed sound insulation
	Jointing strips
	Tape
	Tubing
	Twine
	Washers
	webbing
	Coated electric wire

1.3 Asbestos Processes and Trades in Royal Naval Dockyards.

There have been three main asbestos processes employed in Royal Naval Dockyards: Asbestos spraying, lagging and sound insulation.

Asbestos spraying was used for environmental insulation, the sealing of outer wall and bulkhead surfaces; the spray consisted of a mixture of asbestos fibre and cement applied to a thickness of 2-4 ins. From the mid 1960s, the existing crocidolite material has been extensively removed during refits and replaced by glass fibre and other forms of man-made mineral fibre. The dust concentrations during application and removal have been established as being very high (for removal, many thousands of times higher than the current UK standards).[1,2]** Workers employed as asbestos sprayers and painters were responsible for the application and removal of this 'sprayed asbestos'.

---

* Adapted from: Harries PG (1967) "Asbestos Hazards in Naval Dockyards" Ann Occup Hyg **11**:135-145.

** The referencing throughout this work is unique to each chapter, each forming a separate section of the bibliography.

In the past, laggers were employed to insulate all hot surfaces in machinery spaces aboard ship with preformed 'asbestos' sections, covering them with asbestos cloth. The application and removal of these heat insulating materials would involve both cutting and fixing of the sections and cloth by hand and would produce high localized dust concentrations.

As with lagging, the application and removal of sound insulation involved the cutting and fitting by hand of asbestos boards of various types and thicknesses with again high localized dust concentrations. Sound insulation work would have been undertaken by joiners not laggers.

Other dockyard 'asbestos trades' have included shipwrights, sailmakers, boilermakers, masons, mattress makers, labourers and storemen. Of these, shipwrights and boilermakers cut and fitted asbestos boards in either bulkheads or boiler casings. Masons applied asbestos cement over sprayed asbestos. Sailmakers carried out water pipe and ventilation duct insulation with asbestos cloth. Mattress makers produced mattresses filled with amosite fibre and made from asbestos cloth. Labourers were employed to clear up asbestos debris and storemen issued asbestos materials to all the 'asbestos workers'.

In all of the above trades and processes there would have been a potential asbestos exposure hazard, but the main hazard would occur during what has been termed the vigorous tearing down of old material.[1] The very nature of Royal Navy ships, with long thin watertight corridors, confined machinery spaces and a maze of pipes and fitments would only augment this problem.

1.4 Project Introduction.

The origin of the asbestos disease problem associated with shipbuilding and repair can, therefore, be traced, in part, to the enormous quantities of asbestos materials used by the industry from the start of World War II. During this war period the concern was to build and repair enough ships to win the war; health problems from materials used in construction took second place.

However, along with increased ship production came the requirement to reduce accidents and health hazards that might slow manufacturing. The known hazards then included silica dust, welding fumes, solvents, lead, mercury and asbestos, with asbestos being considered the least dangerous. The risks associated with lead, silica dust and welding received far more attention in the 1940s, they were better understood by the medical and industrial health community and were believed to be more dangerous and widespread.[3,4,5,6] The stage was set, therefore, for future asbestos-related health problems to surface as the other diseases were controlled.

The work of Sheers and Templeton at Devonport Dockyard reported in 1968, highlighted this problem and acted as the catalyst for many Royal Naval research projects. These projects were all undertaken with the aim of improving the health of dockyard workers and settling the asbestos problem (they are reviewed extensively in chapter 2).

In the Devonport study 1,414 men, representing a 10% random sample of the dockyard workforce was drawn.[7] From this sample it was seen that laggers and sprayers with up to 20 years of continuous asbestos exposure had the highest prevalence of asbestos-related disease, asbestosis. Asbestosis was also seen in a variety of intermittently exposed trades. It was also noted that 10 cases of mesothelioma had occurred among the workforce in the 3 years prior to this study. By 1980 Sheers was reporting on 96 mesothelioma cases in Devonport Dockyard; the incidence of mesothelioma correlating with time from first exposure and dockyard occupation.[8]

Also in 1980, Rossiter and Coles reported on the striking finding of an elevated mortality risk of mesothelioma and pulmonary fibrosis among Devonport dockyard workers, but with no obvious accompanying increased risk for lung cancer.[9] In this study 6,292 male workers were identified from dockyard records and their mortality experience followed from January 1947 to the end of 1978. Of these workers 1,043 (16.6%) had died; 31 from mesothelioma, 9 from pulmonary fibrosis and 84 from lung cancer. The number of expected deaths, obtained using estimated South West England mortality rates, was: 998, 0.4, 0.03 and 100 respectively, with associated standardised mortality ratios (SMRs) of 104 for all causes, 7700 ($P < 0.0001$) for mesothelioma, 32000 ($P < 0.0001$) for pulmonary fibrosis and 84 for lung cancer. These results were striking since much higher lung cancer death rates, along with increased mesothelioma rates, had previously been observed in Belfast shipyards and among American insulation workers; occupational groups which were considered to have similar levels of asbestos dust exposure as dockyard workers.[10,11] However, these results were not completely unexpected. A proportional mortality study for the period 1958-1967 comparing Devonport dockyard workers with other Plymouth males showed only a slight (but not statistically significant) excess of lung cancer cases.[12] Nevertheless, the question remains, why in this Royal Naval Dockyard was the risk of lung cancer, a known asbestos-related disease, not significantly different from that observed in the general population when elevated risks were observed for the other known asbestos-related diseases? Was this an artefact, a result produced simply by statistical chance, or does it imply that the asbestos exposures were somehow not high enough to allow lung cancer to develop, but could generate mesothelioma and asbestosis?

Rossiter and Coles commented in this report that the pattern of dockyard asbestos use was such, reaching its peak between 1950 and 1960, that the effect of mesothelioma on mortality may just be starting in the Naval Dockyards. They made no comment on the absence of an excess lung cancer risk. In general it can be said that through the 1960s and 1970s a rising incidence of asbestos-related disease was seen in Royal Naval Dockyards and a large number of deaths reported from mesothelioma of the pleura or peritoneum, with many thousands of Royal Naval dockyard workers appearing potentially at risk. Blot and Fraumeni in 1981 commented that 420,000

American shipyard workers might die of asbestos-related disease because of exposures in the 1940s.[13] They suggested that mesothelioma now occurs exclusively in shipyard/dockyard workers and will probably continue to do so into the 1990s.

Over the period 1972-1973 all workers of the four Royal Naval Dockyards Devonport, Chatham, Portsmouth and Rosyth were invited to have a chest x-ray taken and complete a respiratory questionnaire. The results of these with employment history information will be used in this thesis to examine the relationship between dockyard occupation, exposure to asbestos, smoking habits and cause of death. A particular emphasis will be given to lung cancer and mesothelioma in an attempt to answer the questions produced in the work of Rossiter and Coles. Presented here is an exact 17 year follow-up of the civilian workforce employed in the Royal Naval dockyards during the period 1972-1973.

1.5 Project Aims.

The general aims of this work are twofold:

1. To identify dockyard mortality patterns, across time, and relate these to dockyard occupation, personal medical history, dockyard asbestos exposure, the prevalence of x-ray abnormalities and smoking habits.

2. To assess the relationship between asbestosis, lung cancer and mesothelioma mortality and dockyard employment, and place this relationship into the content of the 'asbestos' literature.

The specific question addressed in this thesis, its null hypothesis, arises directly from the work presented in 1980 by Charles Rossiter and Ruth Coles.[9] Simply stated it is: that there is no excess lung cancer risk in Royal Naval dockyard workers. Subsidiary questions concerning the level of this risk in relation to the mesothelioma risk (i.e. why was the lung cancer risk much lower than the mesothelioma risk) will also be considered.

Chapter 2: LITERATURE REVIEW OF ASBESTOS RELATED DISEASE IN SHIPBUILDING AND REPAIR.

2.1 Introduction.

Asbestos is the collective name given to a group of minerals that are fibrous silicates sharing the common property of high resistance to destruction by physical or chemical means. These minerals include chrysotile and the amphibole group of amosite, crocidolite, anthophyllite, tremolite and actinolite. Chrysotile is the softest type of asbestos and is used in most woven asbestos products (figure 2.1). Amphiboles are harsher, with more bulk, and are more readily used in asbestos-cement and insulation products (figures 2.2 and 2.3). There are three diseases that may commonly develop because of exposure to these minerals: asbestosis, mesothelioma and bronchial carcinoma.

The risk of mesothelioma appears to be greatest in those exposed to crocidolite, slightly less after exposure to amosite, and much less after exposure to chrysotile. Brief exposure to amosite and crocidolite seems to carry a high risk of lung cancer, while prolonged exposure to chrysotile and anthophyllite much less of a risk. Asbestosis may develop from exposure to any type of asbestos, however, amosite appears more fibrogenic than chrysotile and tremolite.[1,2,3,4]

The study of asbestos-related disease has generated a vast amount of literature. It would not be practicable to provide here an exhaustive analysis of this literature; this review will concentrate on setting the scene for the asbestos-related health problems observed in naval dockyards. It will initially consider a historical overview of asbestos-related disease, then more specifically these problems in relation to shipbuilding and repair (with an emphasis on Royal Naval Dockyards studies), and will finally consider the very broad prevailing asbestos health related issues of present day concern. In most of the early reports no differentiation of

asbestos fibre type was made. Where feasible, such differentiation is made in this work.

In 1965 Wagner gave a good description of the etiology of asbestosis.[5] He described how due to its fibrous nature asbestos dust does not follow the same physical laws as other dusts, and that its characteristics allow long fibres to be inhaled and retained in lung tissue. The primary lodging site is in the alveoli arising directly from the respiratory bronchioles. The diameter and length of the fibre are factors resulting in aggregation on this site. He described how the fibrosis later spreads down into the alveolar ducts and atria, resulting in a linkage of the lesions to form a widespread fibrotic network in the lung. Wagner stated that asbestosis is not a sudden explosive diathesis following a dormant period, rather a slow insidious disease. This description can be equally applied to the other asbestos diseases, lung cancer and mesothelioma.

FIGURE 2.1: Chrysotile - white asbestos

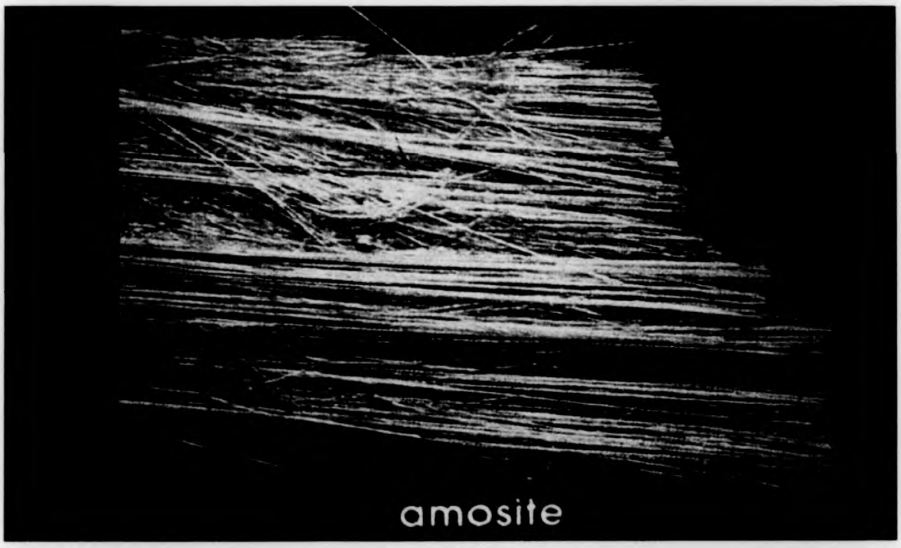

FIGURE 2.2: Amosite - varies from white to yellowish brown in colour.

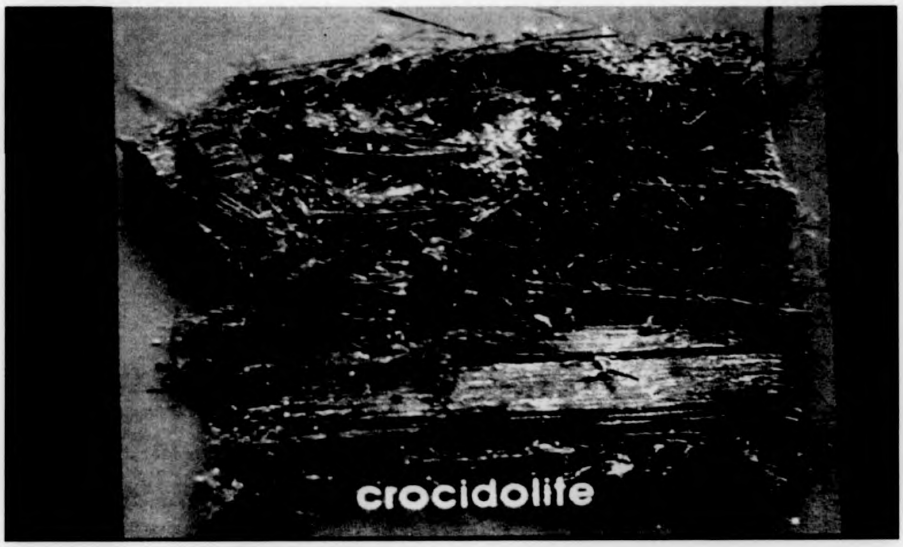

FIGURE 2.3: Crocidolite - blue asbestos.

2.2 Historical Overview.

The Lady Inspectors of Factories in their 1898 report to the Chief Inspector of Factories and Workshops expressed the first concern about the hazards of asbestos dust.[6] To quote: "three occupations can easily be demonstrated as a danger to the health of the workers, ascertained through injury to the bronchial tubes and lungs: asbestos sifting and carding, silk opening and counting, and hemp spinning".

The first reported case of disease associated with the inhalation of asbestos dust was that of pulmonary fibrosis in an asbestos textile worker described by Montague Murray to the Departmental Committee on Compensation for Industrial Diseases in 1906.[7] This worker had been employed for 10 years in the carding room of a textile factory and was the last survivor of a team of 10, all working in the carding room, each having died at around the age of thirty. This man first came to the attention of Murray in 1899 and died, aged 34, in 1900. No evidence was found at postmortem of pulmonary tuberculosis.

The second case, reported by Cooke in 1924, was of a woman aged 33 who died in 1924 having worked with asbestos for 18 years.[8] Postmortem here revealed extensive fibrosis of the lungs, with pulmonary tuberculosis. Both these cases were important, since their publication directed attention to the possibility that inorganic dusts containing little or no free silica might be productive of extensive pulmonary fibrosis. Until then only the opposite view point had been considered, with silicosis occupying the attention of researchers. Cooke's case, though slightly weakened by the presence of a tuberculous infection, was of greater importance, however, as it described the discovery of 'curious' bodies resembling asbestos fibre in the lung tissue. In 1927 Cooke was to call the disease "pulmonary asbestos".[9]

In 1928 Seiler reported the case of an asbestos worker with pulmonary fibrosis for which no other obvious infectious or occupational cause, other than asbestos dust, was found.[10] This report resulted in the Home Office (Factory Department) undertaking investigations into the effects of asbestos dust on the lungs. This case had the four vital conditions necessary to establish a relationship between the inhalation of asbestos dust and the development of fibrosis, namely:

1. Work involving exposure to asbestos dust.
2. The existence, demonstrable clinically and radiologically, of definite pulmonary fibrosis.
3. The absence of previous or present infections known to cause pulmonary fibrosis (for example, tuberculosis, influenza, or pneumonia).
4. The absence of previous or present work involving exposure to other dusts, which might cause pulmonary fibrosis.

By 1930 Merewether and Price, as a result of these Home Office investigations, had found a correlation between the incidence of the disease and the duration and intensity of dust exposure.[11] From this study, 95 of the 363 asbestos textile workers examined clinically showed definite pulmonary fibrosis and a further 21 precursive signs. Of 133 workers examined radiologically, 52 showed signs of diffuse fibrosis and 22 early signs of fibrosis. The authors did not find any evidence of excess pulmonary tuberculosis among the asbestos workers (including those with asbestosis). A dose-response relationship in the development of asbestosis was suggested: fibrosis taking less than 10 years to develop with high exposure to asbestos, and between 15 to 25 years with low dust exposure.

Merewether and Price, in the same report, also reviewed the dust concentrations in textile factories, making numerous recommendations for dust suppression that formed the foundations of the Asbestos Industry Regulations of 1931.[12] Before these regulations were enforced, asbestosis was also recognised as a compensable disease under the 1930 Workmen's Compensation Act (Silicosis and Asbestosis).

Again in 1930, Merewether gave a more detailed account of the clinical findings of the Home office investigations carried out over 1928-29.[13] He described impairment of percussion note and reduced chest expansion as important physical signs of asbestosis, together with scattered fine rales (dry crackling sounds) at the bases and axillae. He also mentioned cyanosis, dyspnoea, finger clubbing, cough and sputum. The radiological changes were described as occurring in four stages:

Stage	I	-	increased linear striations;
	II	-	fairly definite fine dusty stippling;
	III	-	coarser mottling with increased linear striations;
	IV	-	gross lesions with pleural changes and displacements due to the pull of fibrosing lesions.

To sum up developments, by 1930 it may be said that:

> The signs, symptoms, x-ray appearances, and pathological aspects of asbestosis were well recognised.
>
> The disease had been found to be fatal in a number of reported cases.
>
> The disease was capable of progressing after exposure to asbestos dust had ceased.
>
> People still actively employed in asbestos mining, milling and manufacturing showed a high prevalence of asbestosis. Approximately half of those with 10 or more years in the industry were diagnosed as having asbestosis.

In reviewing the pathology and histopathology of asbestosis in 1933 Gloyne described the finding of tough, old, pleural adhesions and reported a 'ground glass' effect to the pleura as thickening increased, with the pleura becoming stiff, yellow and horn like.[14] He noted that 'asbestos' bodies and fibres were found in the lungs of persons with asbestosis and that fibres were commonly found in the upper respiratory tract of asbestos workers. He also reported having seen a case of 'squamous carcinoma of the pleura' with asbestosis.

In a comprehensive three-part article covering the years 1933-34 Merewether summarised the state of asbestosis knowledge, and noted the difficulty of making a diagnosis and what he coined as the "insidiousness of the disease".[15] He suggested that a minimum fibrosis-producing amount of asbestos had to be retained in the lung in order to produce disabling fibrosis, and that a certain development period was necessary before the fibrosis became disabling. He stressed that if dust concentrations could be kept below a certain level, the development of disabling asbestosis would not occur over an average working lifetime. The level he considered appropriate was the amount of dust produced by flyer spinning without dust exhaust. In effect Merewether recommended that flyer spinning of asbestos was acceptable uncontrolled.

Wood and Gloyne in 1934 analysed 100 cases of asbestosis, 12 of whom were autopsied; of these 2 also had lung cancer.[16] Dyspnoea was excessive in the 100 cases and they suggested that asbestosis was a mono-symptomatic disease. It was noted that asbestosis was associated with tuberculosis to a much lower percentage level than was the case for silicosis.

In North America, the Industrial Health Service of the Metropolitan Life Insurance Company carried out similar investigations to the Home Office study of Merewether. This was reported in 1935 by Lanza et al.[17] They examined 126 workers from asbestos mines and mills out of whom 67 had radiological signs of asbestosis, and another 37 doubtful signs. They could not correlate the development of asbestosis with dust exposure through lack of past dust data. The authors stated, without any supporting evidence, that asbestosis was clinically milder than silicosis.

Lynch and Smith in 1935 presented a case history of pulmonary asbestosis with associated bronchial carcinoma.[18] The subject had worked in a cotton mill for 22 years and at an asbestos mill for 21 years. Their conclusion was that the carcinoma was due to chronic bronchial irritation bought on by occupational dust

FIGURE 2.4: Stripping an asbestos carding machine, before the use of exhaust ventiation (circa 1940).

exposure. Figure 2.4 shows the dust created in stripping and cleaning a carding machine.*

By 1938 Gloyne and Merewether reported that of the 95 cases with asbestosis, and 5 other cases of asbestosis with tuberculosis, found in the 1928 Home Office investigations 23 had died.[19] Of these, 12 had died from asbestosis, 9 from asbestosis and tuberculosis and one from asbestosis and carcinoma of the pancreas. The last case, whose underlying case of death was not recorded, was stated to have a "considerable degree of asbestosis" on autopsy. They noted that asbestosis was comparable with the most serious silicosis risks with respect to length of exposure that will cause a fatal degree of fibrosis. They also reported that the association between asbestosis and tuberculosis was less than that between silicosis and tuberculosis.

In 1938 Dreessen et al published a study of American asbestos textile plants that recommended that dust concentrations should be kept below 5 million particles per cubic foot (mppcf)**.[20] This study was also reported on by Sayers and Dreessen in 1939.[21] Their work was based on a group of 511 workers from which only 3 doubtful cases of asbestosis were found. It was unfortunate that of the 511 workers examined only 66 had worked with asbestos for more than 10 years; 333 had worked with asbestos for less than 5 years, and that, as the authors reported, 150 workers had been dismissed from the plants before the survey started as they were suspected cases of asbestosis. This large loss of men unfortunately reduced the value of this study.

Dreessen's study was, however, one of the first in which dust counts were undertaken at various stages of textile manufacture to estimate asbestos exposure to workers. Dust concentrations ranged from 5-75 mppcf without exhaust control

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* Photograph courtesy of the 'Donald Hunter slide collection' held at the London School of Hygiene and Tropical Medicine.

** They recommended 5 mppcf, 8 hours per day, 40 hours per week for a working lifetime as a safe level of exposure.

and between 0.7-7 mppcf with control. The concentrations being highest for willowers, pickers, carders and cloth weavers, these along with spinners and twisters being more liable to develop severe asbestosis. Their value of 5 mppcf being used over the next thirty years as a tentative threshold limit.

In January 1943 the German Federal Government declared asbestosis in combination with lung cancer a compensable occupational disease.[22] This was the first government to acknowledge that lung cancer occurs in clinically slight cases of asbestosis as well as in well-developed asbestosis.

In a thesis submitted to Glasgow University, Wyers in 1946 put forward the case that asbestosis was a clinical disease, not radiographical, and that x-ray change should be used only to confirm the clinical findings.[23] He suggested that the 'ground glass' effect may have been due to poorer techniques and was in fact innumerable small opacities. He speculated that as the disease had changed from an acute to a chronic disease, it might emerge as a neoplastic disease as lower dust concentrations enabled people to live long enough to develop malignant tumours.

In 1947 Wegelius reported on the prevalence of asbestosis in 126 workers from the asbestos mines and factories of Finland.[24] From this group 94 were described as Stage I asbestosis, 23 as Stages II and III and 9 as advanced Stage III, pleural and pericardial thickening being present in this last group. The radiological stages were given here as:

Stage	I	-	very fine network in the middle of the basal fields.
	II	-	denser picture with numerous small nodules.
	III	-	marked shadowing of middle and lower fields with confused heart shadow.

Smith in 1949 performed an analysis on data from current literature, on the etiologic relationship of pneumoconiosis with reference to silicosis, asbestosis and pulmonary cancer.[25] He stated that evidence incriminating silicosis and asbestosis as an aetiologic agent in the development of lung cancer was

inadequate, but that such data should be reviewed frequently and cautioned against the use of insignificant data with respect to the referent community.

Published later in 1949, the Annual Report of the Chief Inspector of Factories for the year 1947, showed that of 235 cases of asbestosis reported 31 (13%) had lung cancer present either as a cause of death or as a concomitant.[26] In addition to these one case, a male aged 77, was diagnosed at postmortem as having 'sarcoma' of the lung.

The association of carcinoma of the lung with asbestosis was highlighted in 1955. The Annual Report of the Chief Inspector of Factories, showed that 24% of 222 male and 12% of 143 female asbestos workers with asbestosis had accompanying lung cancer for the period 1922-55.[27] Doll in the same year presented the postmortem findings of 105 workers from a single asbestos works.[28] Of these, 75 had asbestosis, 15 having associated lung cancer, of the remaining 30 without asbestosis, 3 had lung tumours. The 15 with 'asbestosis cancer' had all worked for periods of 9-23 years before 1933.

In 1960, Leathart monitored the long term progression of asbestosis in 10 subjects.[29] All had worked with chrysotile and amosite fibres. Sputum was routinely examined for asbestos and tubercle bacilli and the subjects were regularly x-rayed and examined clinically. Lung function tests were also performed. From this group, 'asbestos' bodies were found in 8 subjects; no tubercle bacilli was found. From the x-rays, the presence of hilar enlargement distinguished disease stage II from stage I, and the loss of cardiac outline indicated disease stage III. From the lung function tests, compliance was seen to be 20% of normal, vital and diffusing capacity 56% of normal and maximum voluntary ventilation was 45% of normal. Leathart concluded that the vital capacity of workers exposed to asbestos should be measured routinely as a decrease may warn of impending asbestosis. He also noted that lung function changes relate well to clinical abnormality, but not to radiological appearance.

The work of Wagner et al in South Africa in 1960 revealed the first major link between asbestos and mesothelioma.[30] Although considered uncommon, mesothelioma was found in 33 histologically proven cases, 28 of which had mainly a non-occupational association with the Cape amphibole asbestos field. Also in South Africa, Hurwitz reported in 1961 on the radiological changes in asbestosis, among asbestos exposed workers. He stated that pleural changes far outnumbered the cases of lower zone parenchymal fibrosis more commonly reported.[31]

To sum up, by the early 1960s it was known that:

> Lung function tests along with radiographs were a good aid in the diagnosis of asbestosis.
>
> As factory dust levels were lowered, the attack rate of severe asbestosis was lowered. Consequently, asbestos workers begun living long enough to develop asbestos-induced cancer.
>
> Mesothelioma occurs with 'slight' asbestos exposure.

In a series of articles from 1963 to 1968 Leathart confirmed and strengthened his earlier findings, but also suggested that diffusing capacity not vital capacity should now be used to aid diagnosis of asbestosis.[32,33,34] He further suggested that in asbestosis uneven alveolar perfusion existed, and that a restrictive ventilatory defect may have caused dyspnoea in cases with bronchitis. Leathart pointed out that reduced diffusing capacity limited exertion in subjects with 'pure' asbestosis.

Through 1964-65 the studies on insulators by Selikoff et al, finally confirmed the cancer hazard from occupational exposure to asbestos.[35,36,37] In this work lung cancer mortality was 7 times higher than expected, and mortality from gastrointestinal cancer 3 times its expected rate. The authors reported that asbestosis was seen radiologically in 86% of 392 workers with more than 20 years employment.

Enterline and Kendrick conducted a study in 1967 of 21,755 workers from three American asbestos products industries.[38] From the asbestos textile, asbestos

building products and asbestos friction materials industries excess mortality rates were found for respiratory cancer and asbestosis. For the asbestos textile industry, excess rates were also found for digestive cancer and cor pulmonale. Physical inspection of the plants showed the textile factories to be the most dusty.

Also in 1967 the Advisory Panel on 'Problems arising from the use of Asbestos' presented their report and recommendations to HM Senior Medical Inspector.[39] This report described the increasing number of asbestosis cases, particularly in workers not covered by the asbestos regulations (e.g. pipe laggers). It recommended extending the scope of existing regulations while reporting that bronchial carcinoma appeared to be a complication of asbestosis rather than asbestos exposure and that there was strong evidence linking asbestos exposure with the development of mesothelial tumours.

The panel also emphasized the importance of fibre counts in the assessment of the environment and reported on one textile mill were the standard use of exhaust ventilation gave the following results:

TABLE 2.1: Fibre counts from an asbestos textile mill.

Process	Particles counts per cubic centimetre	Fibre counts per cubic centimetre
Carding	400-600	7.7
Beaming	150	4.5
Bag Slitting	100	4.3
Mechanical bagging	125	3.8
Weaving	120	1.9
Plaiting	150	3.8

The panel suggested that the above values should be regarded as immediate goals, but not as standards. Apart from carding, these values for particles not fibres were in the order of 15-45% lower than the American 1938 tentative limit of 5 mppcf (1 mppcf = 35 particles per cubic centimetre).

Correlation of environmental data with the mortality experience of 1,014 asbestos textile workers for the years 1931-1967 was undertaken by Doll and Knox in 1968.[40] Analysis of trends in mortality showed a decrease in lung cancer and other deaths associated with asbestosis with reduction in length of employment before 1933, and an increase in lung cancer and other deaths, without asbestosis, with age. The authors concluded that the occupational risk of bronchial carcinoma had been largely eliminated, but that their data were insufficient to estimate the degree of any other remaining risk.

This work of Doll and Knox, finding a considerable excess of lung cancer in workers exposed before 1933 when the asbestos industry control regulations were enforced, but none in persons exposed for 10 years or more in the most dusty areas since then, was augmented in a study by Newhouse in 1969 at another textile factory.[41] In this study of over 4,500 workers employed between 1st April 1933 (the implementation date of the regulations) and 1st May 1964, Newhouse showed an excess risk of lung cancer mortality for heavily exposed workers followed for at least 16 years with less than 2 years employment. Both studies highlighted the importance of latency considerations when dealing with asbestos exposed cohorts.

A synergistic interaction between cigarette smoking and asbestos exposure was shown in 1968 by Selikoff et al.[42] The authors showed that "asbestos workers who smoke have 92 times the risk of dying of bronchogenic carcinoma as men who neither work with asbestos nor smoke". Weiss in 1971 considered the interactive effects between smoking and x-ray abnormalities.[43] Here the prevalence of pulmonary fibrosis increased with amount and duration of cigarette smoking and with duration of exposure to asbestos dust.

In 1970 a standard of 2 fibres per cubic centimetre (f/cc) for chrysotile dust was adopted in Britain; this followed from a review of the work of Knox undertaken in 1968 by the subcommittee on asbestos of the British Occupational Hygiene Society.[44] The subcommittee concluded that if the risk of clinically significant

disease was to be kept below 1%, for a working life of 50 years, then workers should not be exposed to dust concentrations higher than 2 f/cc. This was one of the many standards set that has lead ultimately to the limits of 0.5 f/ml for chrysotile and 0.2 f/ml for amosite and crocidolite, for daily average exposure, in the United Kingdom today.

The work of Doll, Knox and Newhouse presented in 1968-1969 acted as a spur to numerous studies but most notably to the work of McDonald in Canada examining Quebec chrysotile miners, and in the United Kingdom in the work of Harries in Royal Naval Dockyards. Harries work is considered in detail in section 2.4 of this review. McDonald's study of the Quebec industry was started in 1966, its aims were to relate dust exposure to mortality, radiographic appearances, respiratory symptoms, and lung function; the first reports of this study were presented in 1971-1972.[45,46,47,48]

In this work, Rossiter with McDonald investigated x-ray changes in chrysotile mill and mine workers from Quebec. At Thetford Mines, where dust levels were high (148-780 mppcf 'cumulative dust exposure'), they found that the prevalence of radiologic change was correlated with total dust level and with age; at Asbestos Mine, which was less dusty (101-300 mppcf), the main factors were age and years of exposure. Pleural calcification was common at Thetford but virtually absent at Asbestos. From these studies it was also seen that the overall mortality of the mill and mine workers was lower than expected for the population of Quebec but in the highest dust exposure category comprising 5% of the cohort, the age standardised death rate was 20% higher than in other groups. This excess was largely accounted for by an excess of bronchial carcinomas, 3 cases of mesothelioma were also found. McDonald's work among Quebec miners and millers is considered in detail in section 2.5 of this thesis.

Selikoff et al in 1972 reported on the mortality experience of 230 workers employed at an asbestos products factory.[49] These workers had been employed during the war years in the manufacture of insulation, using amosite fibres, for

shipbuilding and repair. Total deaths were twice the number expected, the excess death rate being limited to two categories, cancer and asbestosis. Fourteen deaths were from asbestosis, 25 from lung cancer and 5 from mesothelioma (2 pleural, 3 peritoneal). It was concluded that occupational exposure to amosite can be associated with a serious cancer hazard, and its continued industrial use requires rigorous control.

In 1973, papers by Weill et al and Jones et al together produced a concise summary of the common radiological observation of the long latency of pleural effects of asbestos (particularly calcification), the frequent finding of extensive pleural abnormalities without parenchymal disease, and the correlation of progressive parenchymal disease, asbestosis, with heavy dust exposure.[50,51] Jones and Sheers in their paper also drew the following conclusions about pleural plaques:

1. There is ample evidence of an association between pleural plaques and all types of exposure to asbestos, and to all types of asbestos fibre. Asbestos is not the only cause of plaques but is certainly the most common.
2. There is insufficient evidence to establish a direct cause-effect relationship between pleural plaques and asbestos dust.
3. The prevalence of plaques is not related to total dust exposure; but, given a minimum initial exposure, the prevalence depends on age.
4. Pleural plaques are not harmful clinically, but they act as a useful marker to possible asbestos exposure.
5. The pathogenesis of plaque formation is unknown.

Webster in 1973 updated the information on the occurrence of mesotheliomas in South Africa.[52] It was reported that 158 out of 360 recorded cases had definite exposure to asbestos but only 88 of these came from the mining areas where exposure to only one type of asbestos could be assumed. Of the 88 cases 84 had been exposed to crocidolite and four to amosite.

Newhouse and Berry in 1976, reporting on a group of workers exposed to chrysotile and a mixture of amphiboles, used a model relating mesothelioma risk

with time since first employment to predict mesothelioma mortality rates in man to the year 2000.[53] They estimated a mortality rate of 7-11% per annum for male and 9-12% for female asbestos textile workers by 2000 AD. This model in many refined forms has been used extensively since then, in particular by Peto.[54,55,56] All of the models used make use of the fact that the relationship of mesothelioma to asbestos differs in several ways from the relationship for lung cancer, the hazard appearing to be more strongly dependent on the type of asbestos, to be largely or wholly unaffected by smoking and independent of the age at which exposure first occurs.

In 1979 Irwig et al reported studies on 1,144 men from South African crocidolite mines and 548 who were involved in the mining of amosite.[57] It was claimed that pleural abnormalities visible on chest radiographs were significantly more frequent among amosite workers than among those who had worked with crocidolite. There were no other differences in recorded pathology between the two groups.

By 1979 Hammond et al and Berry et al had carried the asbestos-smoking interaction a step further; to an increased risk of asbestosis.[58,59] Hammond's work clearly showed that a multiplicative relationship may exist between smoking and asbestos (see table 2.9, in section 2.5.3), it further showed that mortality from asbestosis was 2.8 times higher for asbestos workers who smoked than for their nonsmoking colleagues. Berry after allowing for age showed that there were significantly fewer signs of asbestosis in nonsmokers and light smokers than in heavier and ex-smokers, for men first exposed to asbestos after 1950.

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By the 1970s it was known that:

> A high proportion of cancer deaths occur among subjects with asbestosis.
>
> Asbestos acts synergistically with smoking to produce nonmalignant non-infectious pulmonary disease, although the synergism is not as strong as it is for lung cancer.
>
> Asbestos-related cancer occurs after exposure to dust containing a high proportion of large asbestos fibres, i.e. fibres longer than 5 microns (5μm).
>
> Removal from exposure is unlikely to halt the progression of pleural changes once they have developed.
>
> Amphibole asbestos, in particular amosite and crocidolite, appears more hazardous to health then chrysotile asbestos.

From the early 1970s the need for information on the occurrence of diseases related to asbestos, and their relationship to exposure levels was evident, and many studies followed, particularly in the USA and Britain. To review these here, extensively, would not be practicable as over 200 papers on diseases related to asbestos and its exposure have been published per annum. To summarise, however, the principal disorders related to asbestos exposure include:

> Diffuse interstitial pulmonary fibrosis, termed asbestosis. Lung cancer and mesothelioma of the pleura and peritoneum. Pleural plaque formation and diffuse pleural thickening. It may also cause other cancers, including possibly cancers of the larynx and the gastrointestinal tract, and conceivably a wide range of others (figure 2.5).

Figure 2.6, taken from the ILO Encyclopaedia of Occupational Health and Safety (1983), outlines the rise in production of asbestos worldwide with the dates of acceptance of the causal relation between asbestos and the above diseases. Since the 1980s the use of asbestos in the industrialised world has diminished, with the increasing use of man-made mineral fibres as a substitute material. However, in the developing world (e.g. India and South America) the use of asbestos continues unabated. The worldwide use of asbestos may therefore have slowed but has clearly not stopped altogether. The following sections of this review will now consider asbestos-related disease in shipbuilding and repair, and also the present day concerns generated by past and present asbestos exposure.

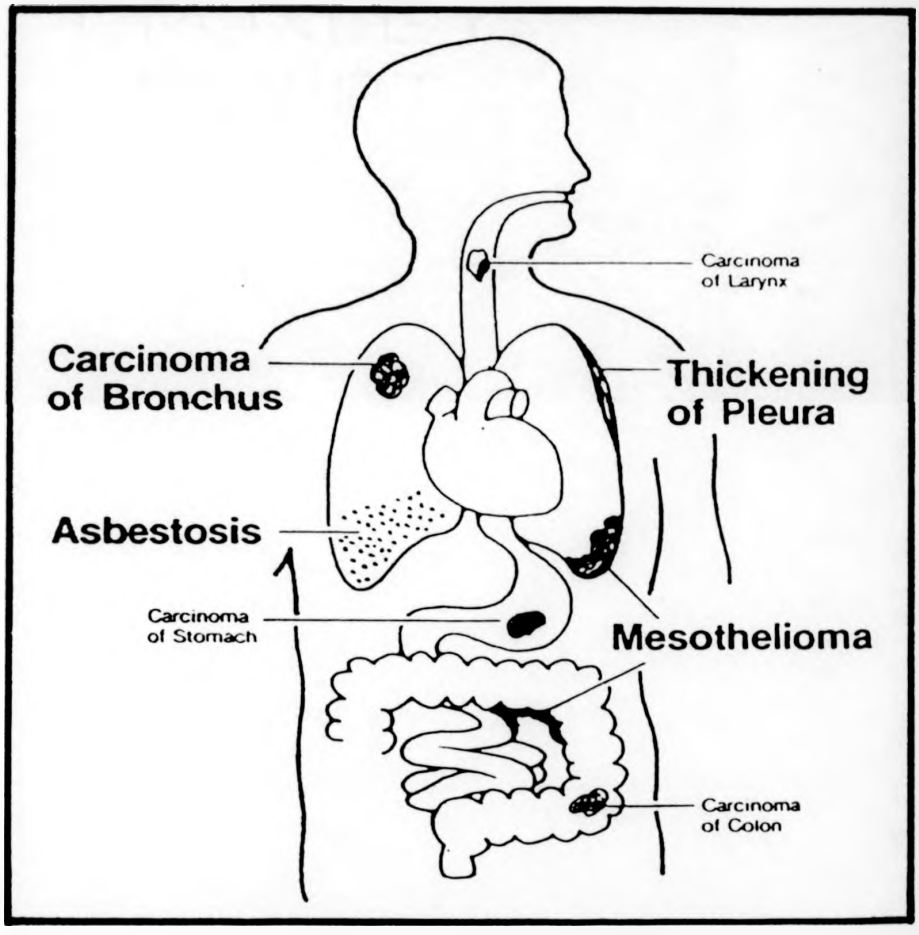

FIGURE 2.5: Principal asbestos related diseases and conditions, and their sites in the human body.

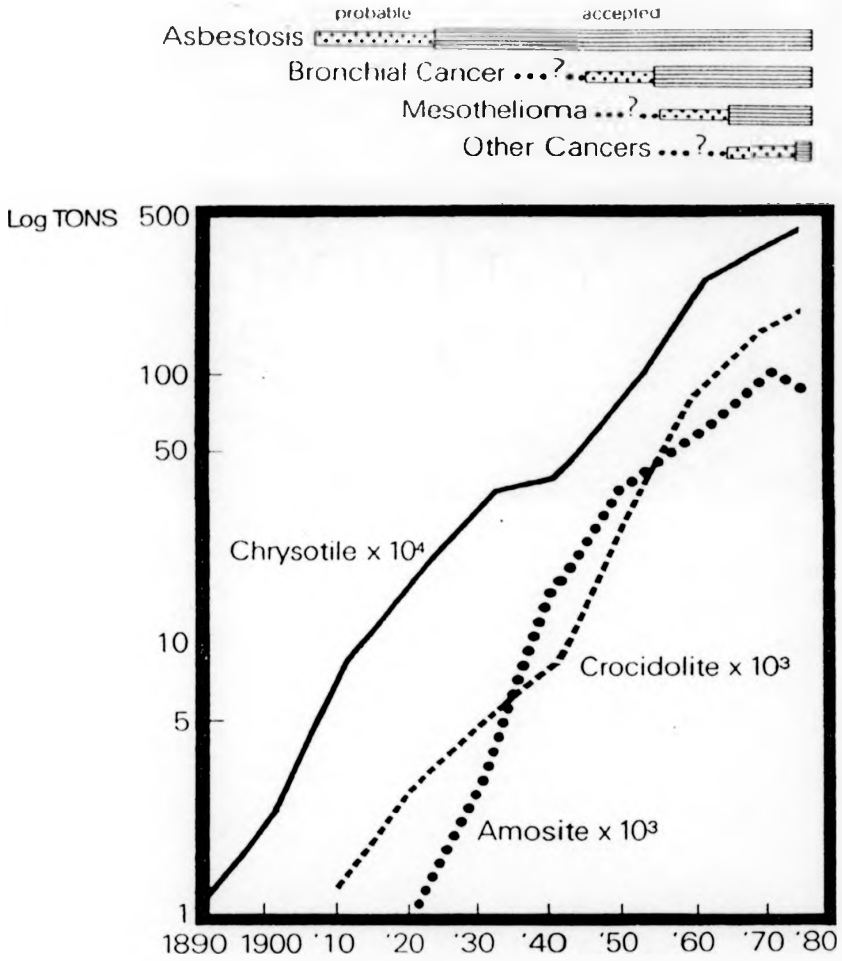

FIGURE 2.6: Rise of production of asbestos 1890-1980, and the dates of acceptance of the causal relationship between asbestos and various diseases.

2.3 Asbestos Related Disease in Shipbuilding and Repair.

Health problems arising from the use of fibrous materials in Royal Naval Dockyards are not a new phenomenon. In a letter dated 17 June 1891, to the Fleet Surgeon, Naval Surgeon G Kirker* reported on a workman suffering discomfort in his chest owing to breathing in the dust of cotton silicate, a fibrous silicate, used as a woven covering for boilers and steam pipes.[60] Eventually cotton silicate was replaced by a better insulator, asbestos.

There was a gradual increase in the use of asbestos in British Dockyards and American Shipyards over the next 30-40 years, followed by a massive increase in use during World War II. Between 1939-45 over 6,500 vessels, all containing asbestos, were built in American yards.[61] At the peak of the war more than 1,700,000 workers were employed in this industry; throughout the war a total of over 4,500,000 men and women were employed in shipbuilding and repair.[62]

Expansion of the British and American shipbuilding and ship repair industries in the 1930s mirrored that of Germany. The first published reports linking asbestosis with shipbuilding coming from German researchers. Kuhn in 1940 reported on a fatal case of asbestosis that had occurred in a German shipyard insulation worker.[63] He described how workers in German shipyards were rotated out of asbestos insulation handling after 2 years of work.

A United States Maritime Commission field survey in 1944, by Dreessen and Fleischer, at a civilian contract shipyard revealed 2 pipe coverers (asbestos laggers) with x-ray evidence of well-established asbestosis and 4 others with possible asbestosis, in all 38 pipe coverers were examined.[64] They concluded that conditions in this yard presented a very real asbestosis hazard.

---

* Kirker was the surgeon in Keyham Yard, now North Yard, HM Naval Base, Devonport.

In August 1945, HM Chief Inspector of Factories issued a warning to the shipbuilding and ship repair industry over the danger to health from the increasing use of asbestos in ships.[65] This warning stressed that while asbestos dust had no immediately obvious effects on health, serious problems were likely to develop subsequently. Among the suggestions made were the need for better ventilation, damping the dust and the quick cleaning up of asbestos debris. The use of dust respirators was recommended for men fitting or removing dry asbestos, and during the spraying of asbestos.

Fleischer et al in 1946 conducted x-ray examinations and dust surveys at four US Naval Shipyards, this work was also reported on by Drinker in 1947.[66,67] Total dust concentrations in these yards ranged from 7-32 mppcf, with an average on-ship exposure of 11-142 mppcf. They reported the lower values in units of 'mppcf asbestos dust'. Of the 1,074 workers examined, 948 had less than 5 years employment in the pipe covering industry and only 54 had more than 10 years employment. Three cases of asbestosis were found, all in workers with more than 20 years of employment. They concluded that pipe covering in the shipbuilding industry was not hazardous. This conclusion conflicted with the earlier statement of Dreessen and Fleischer in 1944 of a very real hazard. The low asbestosis prevalence, shown by Fleischer and Drinker, was clearly an artifact caused by their inadvertent dilution of the at-risk population with briefly exposed workers. Although these early studies did not stand the test of time epidemiologically, they did represent the best occupational health methods of their era.

The use of respiratory protection for asbestos exposed workers in American shipyards during World War II was reviewed by Stoll et al in 1951, who noted that protection was advised but not insisted upon.[68] This was despite the US Navy's publication in 1943 of the minimal requirements of health and safety in contract shipyards.[69] This document required the segregation of dusty work, use of exhaust ventilation and respirators, and periodic medical examinations of workers handling asbestos insulation materials. Stoll also reported on a 40 year old asbestos pipe coverer with bronchogenic carcinoma in association with

pulmonary asbestosis. The authors noted that amosite was the most widely used form of asbestos in shipyards during the War.

In an account of the health hazards of asbestos at Portsmouth Royal Naval Dockyard, Walters in 1959 noted that only one case of asbestosis had been reported among pipe coverers between the years 1946-58, with a further 6 possible cases unconfirmed by the Pneumoconiosis Medical Panel.[70] Over the same years no cases were reported among asbestos sprayers.

Morgan in 1964 described a case of 'rheumatoid pneumoconiosis' associated with asbestosis in an arc-welder, exposure to asbestos was traced to welding of asbestos lagged pipes in the interior of ships.[71] Also in 1964 Marr reported on 5 workers from the Long Beach Naval Shipyard compensated for asbestosis, one of whom had died of the disease.[72] This study looked at the irregular exposure to asbestos occurring during its installation and removal in ship repair or overhaul. Total dust counts were given as 1-5 mppcf. These 'low' counts do not appear to give an adequate indication of the hazard, conflicting with the observation that "the work environment appears extremely dusty, respirator filters often clogging after an hour's work removing insulation". The shipyard also stated that amosite was seldom applied due to the excessive dust created during its removal!

In a follow up study, at one of Fleischer's four shipyards, Murphy and Ferris in 1966 analysed 101 pipe coverers and 94 age-matched controls from departments with minimal asbestos exposure.[73] Of 68 pipe coverers with 5 or more years exposure significant differences were seen on comparison to their controls for symptoms of cough, phlegm, wheezing and shortness of breath. Over 25% of these workers were diagnosed as having asbestosis; no cases occurred with fewer than 8 years of exposure, the average length of exposure was 20 years. The study emphasized that low concentrations of asbestos can lead to pulmonary fibrosis and supported the need for lower threshold limit values. This work was extensively referred to by Murphy and Ferris in subsequent publications.[74,75,76]

In 1967 Anton reported on 12 cases of multiple pleural plaques in shipyard workers.[77] He suggested that the presence of pleural plaques might be a sign of a milder form of asbestosis and considered that pleural fibrosis might have been responsible for earlier researchers description of a 'ground glass' effect in the lower lung fields. The frequency of asbestos bodies in the lungs was studied by Ashcroft in 1968, in a shipbuilding area where asbestos was used in insulation for boilers, pipes and bulkheads.[78] Asbestos bodies were found in 20% of all routine necropsy smears but there was no evidence of classical asbestosis.

Sheers and Templeton in 1966 carried out x-ray examinations of every tenth worker employed at Devonport Royal Naval Dockyard, this work was also reported on by James in 1971.[79,80] From the 1,414 workers examined with possible exposure to asbestos, 4 had pulmonary fibrosis due to asbestosis, 11 extensive pleural fibrosis, 48 limited pleural plaques, 2 bronchogenic carcinoma and 1 mesothelioma. The intermittent nature of asbestos exposure in ship overhaul was suggested as the reason why pleural changes overshadowed parenchymal disease, exposure being insufficient to cause asbestosis except in high risk groups.

The work of Sheers and Templeton at Devonport Dockyard, and of Walters at Portsmouth Dockyard was extended in a series of Royal Naval Dockyard Asbestosis Research Project reports, to cover the four Royal Naval Dockyards: Devonport, Chatham, Portsmouth and Rosyth. This work together with other hygiene and medical reports from naval surgeons form a core of the published material from the United Kingdom for the 1970s and is considered in section 2.4 of this review.

By the late 1960s it was increasingly clear that:

> The risk of disease was not confined solely to workers in mining and manufacturing asbestos. It extended to those workers who used asbestos products and in particular to insulation workers and dockyard workers.
>
> That in the period of World War II through to the 1960s there was extensive use of asbestos in dock and shipyards with little control and minimum precautions in its use.

Horn in 1969 published a summary of the protective measures to be used when working with asbestos containing materials in shipbuilding.[81] The use in Germany of suction devices was highlighted, with the recommendation that suction be improved so that asbestos dust concentrations in room air of 30 particles per cubic centimetre for particles up to 10 micrograms is not exceeded. A stipulation against employment of workers with chronic respiratory diseases or youths aged under 18 in jobs handling asbestos materials was given. The author concluded that "strict prophylactic measures for handling materials containing asbestos must be administered because there is no effective treatment for asbestosis".

The chance finding of asbestos bodies without asbestosis in the lungs of a shipyard worker who had not worked directly with asbestos was recorded in the Netherlands, in 1969, by Stumphius.[82] This led Stumphius in 1971 to an investigation into the relationship between asbestos bodies in the sputum, occupation, and mesothelioma in the shipyard at Vlissingen on Walcheren Island.[83] Examination of the sputum of 277 workers revealed asbestos bodies in 60%, the frequency varying from 39% in workers with no obvious exposure to asbestos to 100% among those with slight but definite exposure. Between 1962-68, 25 cases of mesothelioma were discovered on Walcheren; the attack rate for mesothelioma was estimated to be 100 per 100,000 males per year, 100 times higher than the rate for Dutch provinces with heavy industry. For different occupational categories in the shipyard the rates varied from 50 for 'clean work' to 280 for men with some exposure to asbestos dust.

The occupational classification used by Stumphius was based on exposure to both iron oxides or fumes and to asbestos. The categories used were:

I Iron Vapour: Apparently negligible asbestos exposure - welders and cutters.

II Iron Oxides (Rust): Some exposure to asbestos - fitters, welders, plumbers, etc. employed after launching or during repairs.

III Iron Vapour and Rust: Apparently negligible asbestos exposure - platers, shipwrights, and foundrymen employed on initial construction.

IV Men exposed to other dust, including some asbestos, not exposed to iron vapour or rust: Painters, carpenters, etc. employed after launching or during repairs.

V Clean Work; Not apparently exposed to iron vapour or rust nor to asbestos dust.

As can be seen by these categories, no workers were regarded as working solely with asbestos or as being heavily exposed to its dust. Those men employed by contractors on asbestos insulation work were not studied. Stumphius aired the suspicion that iron oxide might be an important cofactor in the genesis of shipyard mesothelioma.

Evidence of chest disease noted in the x-rays of shipbuilding and engineering workers at an industrial clinic was shown, by Fletcher in 1971, to correlate with trade within the industry.[84] Joiners in particular were noted to have a high prevalence of pleural plaques, plaques occurring in approximately 30% of all joiners, followed to a lesser extent by caulkers, burners, drillers, laggers, and sheet metal workers. For joiners, the use of asbestos board containing 30% amosite in sound insulation was suggested as the probable cause of illness.

Ahlman and Siltanen in 1971 presented annotated tables of 56 cases of pulmonary asbestosis occurring among insulation workers in Finland during 1938-68.[85] In this work they also presented data on Finish shipyard breathing zone exposures to asbestos dust. For pipe coverers, exposures ranged from 34-92 f/cc (fibres longer than 5 microns), total particle counts ranged from 26-50 mppcf. For workers sawing asbestos boards exposures ranged from 86-220 f/cc; 45-97 mppcf.

These concentrations emphasise the importance of dust suppression methods, with the large number of cases suggesting past ignorance of the health hazards of insulation work.

Also in 1971, Whitwell and Rawcliffe in a retrospective study of 52 pleural mesothelioma patients from three Merseyside hospitals confirmed occupational exposure to asbestos in 80% of the cases, the most common industry involved being shipbuilding and repair.[86] The lungs of patients with industrial mesothelioma showed basal asbestosis in 17% of cases and excessive asbestos bodies in most others. The interval from first exposure to asbestos to the appearance of mesothelioma ranged from 13-63 years, with a reported mean of 42 years.

Elmes in 1971 reported on a high incidence of lung fibrosis in Belfast insulation workers.[87] From 165 workers, over a 27 year period, 98 had died and from these 40 had radiological or postmortem evidence of extensive fibrosis (excluding tuberculosis). These workers were all trade union members who had worked either in the Belfast shipyards or on large Northern Ireland construction sites. Two other studies concerned with the health of Belfast insulators were presented by the same team of researchers in 1971.[88,89] From these studies there was evidence suggesting that the pleural fibrosis was due to asbestos exposure in childhood, with many of the insulators family homes being in the shipyard area.

From the International Labour Office (ILO) publication on Safety and Health in Shipbuilding and Ship Repairing published in 1972 we are given the following asbestos exposure information by Cross:[90]

TABLE 2.2: Ranges of dust concentrations (data grouped across numerous yards).

Process monitored	f/cc
Insulation rip-out	159 - 353
Spraying, undamped	150 - 1500
Spraying, damped	1 - 4.7
Pipe covering	11.2 - 61
Hand sawing board, without exhaust	14 - 58
Hand sawing board, with exhaust	1.1 - 3.4
Power sawing, without exhaust	63 - 200
Power sawing, with exhaust	1.4 - 4.5

These data were supplied to Cross by industry and government in the UK. It should be noted that for 'rip-out' the values given were averages; the maximum was 3815 f/cc for sweeping and bagging debris. This value is approximately 20,000 times the current UK limit for amosite and crocidolite exposure and was obtained at Devonport Dockyard.[91] In the same ILO publication Nicholson reported on electron microscopy findings that showed that some processes yielded only small fractions of airborne fibres longer than 5 microns (e.g. cutting asbestos block, 3.5%; removal of pipecovering, 5.9%).[92] Nicholson believed exposures could be kept below 0.7 f/cc with the use of improved industrial hygiene practices.

At this point, the mid 1970s, it was clear that:

> There was a high prevalence of pleural plaques in ship and dockyard workers and that asbestos bodies were being seen with and without asbestosis in these workers.
>
> That asbestos-related diseases were occurring in 'non-asbestos' shipyard trades and were also present from childhood exposures.

In 1976, Stumphius continued his initial work on Walcheren Island, reporting that 52 cases of mesothelioma had occurred in shipyard workers during 1962-76.[93] None of the cases was a typical asbestos worker but the majority had worked in the same areas as asbestos workers, their exposure was of an indirect occupational type. No asbestosis was seen. As a result of Stumphius work the Dutch authorities stopped the use of asbestos insulation aboard Royal Dutch Navy ships.

Blot with Harrington in 1978 investigated the high lung cancer rate among male residents in coastal areas of Georgia USA.[94] This study consisted of 458 lung cancer cases and 553 controls (subjects hospitalised for reasons other than lung cancer) from 11 coastal counties. The occupational risk of lung cancer was calculated for 16 industrial categories, including shipbuilding. It was found that 95 cases (21%) and 80 controls had worked in shipyards during World War II. The relative risk for shipbuilding adjusted for smoking, age, race, occupation and county of residence was estimated to be 1.6; a synergistic relationship was found between shipyard employment and smoking. The authors concluded that asbestos and other shipyard exposures account in part for the excess mortality from lung cancer in coastal Georgia.

Renke in 1978 reported on a study which examined lung function in 60 Polish shipyard workers, 21 male and 39 female, over a four year period.[95] Two cases of asbestosis were diagnosed in this group over the study period; the average duration of asbestos exposure being 5.8 years for men and 10.7 years for women. Renke noted on an increase in the number of obstructive and restrictive ventilation disturbances across the 4 years.

In a continuation of their previous work Murphy and Ferris in 1978 re-evaluated their cohort of 101 pipe coverers and 94 controls.[96] Clinical findings indicative of asbestosis were noted in 16% of the pipe coverers compared to 9% in the earlier surveys. Asbestosis was considered present if three of the following chest abnormalities were found: bibasilar fine rales, irregular x-ray opacifications, forced vital capacity less than 80% of normal, or single breath diffusing capacity less than 80%. Eight pipe coverers had died since the original study, 3 of confirmed and 1 of suspected asbestosis, 1 from peritoneal mesothelioma and 1 suffered a fatal myocardial infarction; his lungs showing slight pulmonary fibrosis. Three pipe coverers had retired because of pulmonary insufficiency. Asbestosis was 11 times more common among pipe covers than their controls. The authors noted that the increasing prevalence of asbestosis was in spite of a massive reduction in the amount of asbestos used in the shipyard since 1969.

Blot continued his earlier work on lung cancer producing a succession of reports over 1979-81. In the first of these, lung cancer mortality rates over 20 years were calculated for 49 US shipyard counties, 80 urban counties of similar size but with no involvement in the construction of cargo vessels or warships served as controls.[97,98] For females, 80% of the shipyard counties had higher lung cancer rates than the control counties; for males the cancer rates for all shipyard counties were at least 30% higher than their controls. In his later work, increased lung and laryngeal cancer risk among shipyard workers from Virginia USA employed before 1950 was reported.[99] The relative risk, adjusted for smoking, was 1.7. This was of the same order of magnitude among those who had only worked in shipbuilding during the war years as among those whose careers were spent in the industry. This was followed by a study of 61 cases of mesothelioma, 47 from shipyards in Virginia.[100] The median time between tumour diagnosis and first shipyard employment was 34 years. The conclusion from these studies was that shipyard exposures to asbestos are implicated as being partially responsible for the parallel clustering of mesothelioma and lung cancer in shipyard areas.[101]

The incidence of bronchial carcinoma in shipyard workers from Barrow-in-Furness with pleural plaques was reported by Edge in 1979.[102] Pulmonary asbestosis was verified in chest x-rays of 429 male shipyard workers, this group was compared to age-matched males from Carlisle with no asbestos exposure. Over a 7 year period 127 deaths occurred among the shipyard workers, 19 from bronchial carcinoma and 23 from mesothelioma; in the control group 74 men died, 4 from bronchial carcinoma and none from mesothelioma.

The hazardous effects of asbestos exposure were again highlighted by Selikoff et al in 1980 in a study of 286 ship repair workers.[103] The workers were retired or still active with at least 20 years of exposure; x-ray evidence of asbestosis was found in 86%, 5 cases of previously undiagnosed lung cancer were reported. Selikoff noted that ships constructed before 1975 contained extensive amounts of

asbestos and pointed out that the risks were not limited to ship repair workers, they also extend to crew members who performed emergency repairs at sea.

During World War II, women were encouraged to replace men in shipyards with many becoming welders and insulators; upon demobilization they returned to other work and consequently had only limited durations of asbestos exposure. Boylen et al in 1983 presented one of the first studies on the frequency of asbestos-related diseases in such women.[104] In this study 60 women were considered, 13 of whom had sufficient asbestos exposure to produce stigmata after 40 years.

Kilburn in a series of reports during 1985-86 continued the investigation into asbestos-related disease among female shipyard workers and extended this to include family contacts of shipyard workers.[105,106,107] In his first report 71 female workers who had been employed before May 1961 were studied; 15 had x-ray signs of asbestos disease, of which 7 had irregular opacities in the lung parenchyma and 8 pleural disease (2 had pleural calcifications). Chronic bronchitis was diagnosed in 21% and symptoms of dyspnoea and wheezing in 71% and 42%, respectively. These prevalences were 50% higher than in the wives of male shipyard workers. In his later work the incidence of asbestosis in 338 male and 81 female workers (each with at least 20 years asbestos exposure) and their families was investigated. The family members included 280 wives, 144 daughters and 81 sons. Among the workers there was x-ray evidence of asbestosis in 185 males and 15 females. Among their family members with no occupational exposure to asbestos, the disease was found in 31 wives, 6 sons and 3 daughters. This was higher than the corresponding incidence in a control group taken from the general population. From this study, it was concluded that family members of shipyard workers face a serious asbestosis risk along with the workers of either sex.

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To summarise, by the early 1980s:

> It was clear that the asbestos disease problem that has caused so much public concern could be traced, in part, to the use of asbestos aboard ships. Contributing to this problem was its massive use during World War II and immediately thereafter.
>
> That first impressions of the problem suggested that only those workers continuously involved with asbestos were at risk. In the dockyards these would have been mattress workers, laggers, joiners, sailmakers working with asbestos cloth, and asbestos sprayers and strippers. However, experience has shown, that first impressions can be false, with light and intermittent exposure, indirect occupational exposure and non-occupational exposure carrying serious risks.

It is obvious that ships are not factories and that shipbuilding and repair conditions, and their asbestos exposures, must differ from those in asbestos mills and factories. These conditions in Royal Naval Dockyards are highlighted in the following section.

2.4 Royal Naval Dockyard Asbestosis Studies.

During the late 1960s and early 1970s a series of research projects were supported jointly by the Institute of Naval Medicine (MOD), Alverstoke, and the Medical Research Council (MRC) Pneumoconiosis Unit, Penarth. These projects have been termed the 'Royal Naval Dockyard Asbestosis Studies'. They were set up to investigate and answer the medical and hygiene problems associated with asbestos use in Royal Naval Dockyards. This section will concentrate on these studies, and in particular the work of Harries at Devonport Dockyard.

Harries was to bring to the attention of the scientific community the need for better preventive measures in asbestos handling. In a series of reports he looked at dust concentrations and dust sampling techniques involved in asbestos application and removal.[91,108,109,110,111] In this work, the highest fibre concentrations were found in bagging and sweeping up debris and in the removal of sprayed crocidolite asbestos. Fewer fibres were observed in the removal of asbestos section and board (table 2.3). All debris had to be carried out by hand, through narrow passageways and vertical ladders (figures 2.7 and 2.8).

TABLE 2.3: Ranges of dust concentrations (data from Devonport dockyard).

Process monitored	f/cc
Storerooms	0.1 - 36
Application of amosite sections	9 - 40
Application of asbestos cloth	0.05 - 0.26
Removal of amosite sections	29 - 1040
Removal of sprayed asbestos	112 - 1906
Removal of asbestos acoustic board	48 - 683
Bagging asbestos debris	106 - 3815

As mentioned in the last section, the high value found for bagging waste is approximately 20,000 times the current UK limit for amosite and crocidolite exposure. It should also be borne in mind that these fibre counts are generally

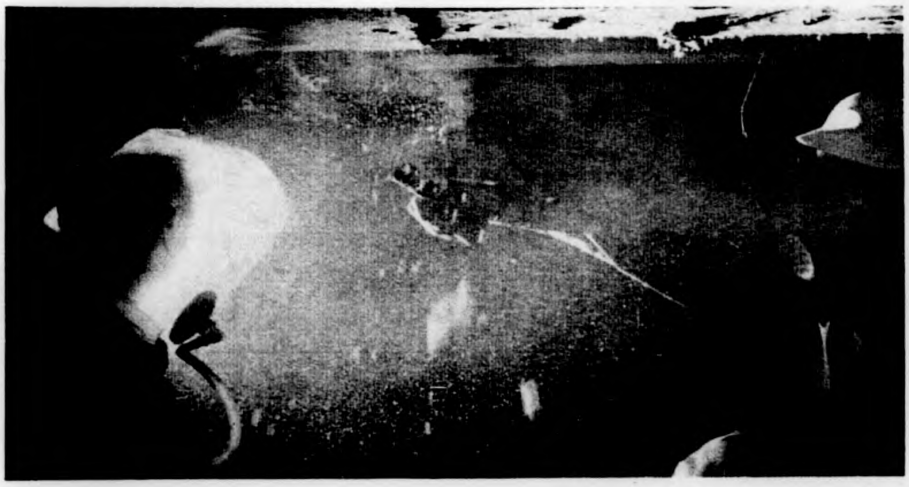

FIGURE 2.7: Removal of sprayed crocidolite asbestos from a deckhead.

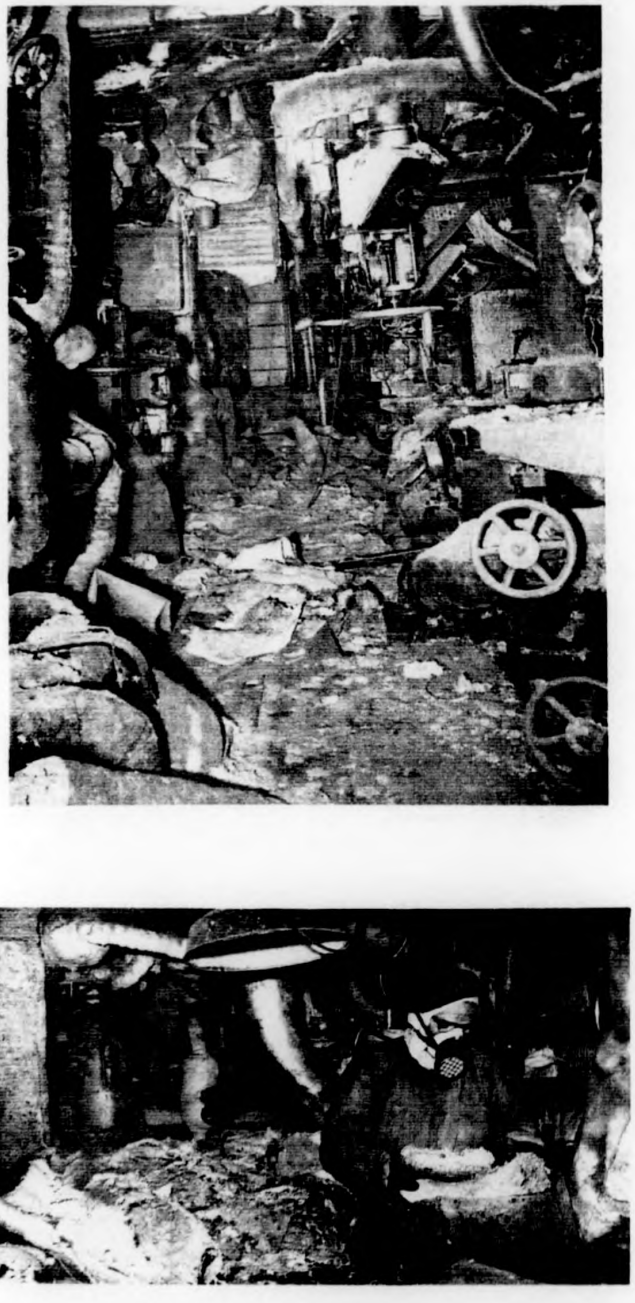

FIGURE 2.8: Asbestos debris in a boiler room.

much higher then those stressed as immediate goals in 1967 by the government advisory panel on asbestos who suggested a maximum range of 1.9-7.7 f/cc.[39]

It was also seen that average dust levels were much higher in boiler rooms then in engine rooms (table 2.4). A boiler room would have more insulating material, but would also have at least two levels and it was common place for debris to fall 3-4 metres to the deck creating high levels of dust in the general atmosphere.

TABLE 2.4: Dust concentrations during application and removal of asbestos lagging (f/cc).

	General Atmosphere Mean value Range	Breathing Zone Mean Value Range
Removal:		
Boiler rooms	171 0.04 - 1062	97 25 - 220
Engine rooms	88 0.16 - 3021	91 2 - 490
Application:		
Boiler rooms	22.4 1.0 - 61	16.8 0.1 - 68
Engine rooms	2.1 0.1 - 14	7.3 0.04 - 40

Additionally, in the above work Harries noted that adequate preventive measures were not introduced into Naval Dockyards until 1967; before this it was common to see asbestos waste left scattered about ships for most of the refit period (sometimes up to 3 years). This clearly shows that the health risks associated with asbestos dust and waste were not fully appreciated. Lumley in 1971 emphasized this point in a survey of sprayed asbestos in dockyard buildings.[112] In storehouses, roof insulation was found to be unsealed and damaged. Crocidolite dust concentrations of 0.3-52.6 f/cc were obtained from these floors. Roof dampness and birds nesting in the roof spaces were seen as the main reasons for asbestos debris falling onto ledges, boxes and the floor (figure 2.9 illustrates the extent of this debris).

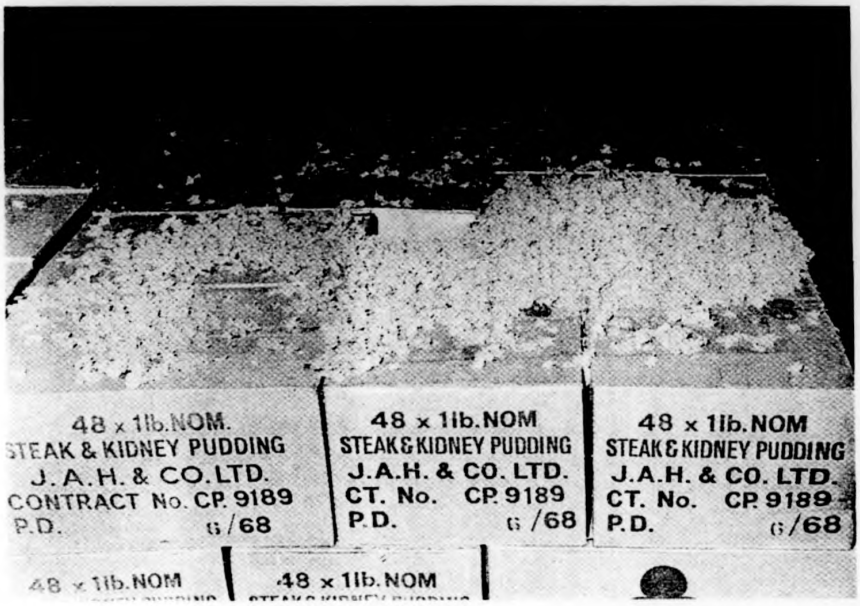

FIGURE 2.9: Asbestos debris on storeroom boxes.

Harries also made an evaluation of respirators and respirator usage, with the following observations: for respirators to be efficient they had to be of good fit, comfortable to wear, and the workers had to be encouraged to wear them. In fact, almost the opposite was true with little encouragement given and no attempt made to see that a respirator fitted a worker's face. Consequently, it was common for respirators not to be worn completely, if at all, over a working shift. It was, therefore, practically impossible to measure respirator efficiency in reducing asbestos dust exposure. Added to this was Harries commentary that many of the respirators issued were unfortunately fitted with fume cartridges and not dust filters.[110] This situation was soon to change with enforced use of air-stream hoods and better quality dust respirators.

The measures then taken to reduce the asbestos hazards, are described in the above publications and by the Ministry of Defence in 1970.[113] They included a review of the use of asbestos, and where possible the introduction of substitute materials (i.e. man-made mineral fibres and plastics) and enforcement of a Code of Practice.

The Code of Practice consisted of the following safeguards:

1. To isolate asbestos work, and restrict entry to those properly protected.
2. To reduce the amount of dust created by asbestos work by improving work methods and the materials themselves.
3. To protect all workers whether they worked directly with asbestos or not.
4. To keep a register of all men directly employed on asbestos work.
5. To ensure that all Registered Asbestos Workers have regular medical supervision.
6. To provide protective clothing adequate for the degree of risk involved and to provide changing and washing facilities.
7. To carry out regular dust monitoring whenever asbestos is handled.

The medical supervision of 'asbestos workers', before implementation of the code of practice, was shown in the above work by Harries to apply only to sprayers and laggers, all others were unsupervised. The Navy policy, from 1949 onwards, was

confused with recommendations of monthly, six monthly and annual examinations, enforcement of which was uncertain. From 1965 laggers were given annual medical examinations. The following articles show how ineffective this protection was for this group of workers, sizeable proportions developing asbestosis.

Before moving on to the dockyard medical reports it should be noted that by the early 1970s the following had occurred:

> Sprayed crocidolite asbestos was completely removed from Royal Navy ships in the 1960s. It was removed, not for health reasons, but when it was found to be increasing the top weight of warships.
>
> All forms of amphibole asbestos were prohibited from use after 1968. Only chrysotile asbestos and man-made mineral fibres were used from then on.

A detailed study of the clinical and radiological changes occurring in men exposed to asbestos at Devonport was reported on by Harries in 1971.[110] This supported, and was founded on, the work of Sheers and Templeton in 1966, which was further continued by Harries et al in 1972 to cover Portsmouth, Chatham and Rosyth.[79,114] Here the prevalence of radiographic asbestos-related abnormalities was related to both dockyard occupation and duration of exposure (table 2.5 shows prevalence by dockyard). No association was found between smoking habits and the incidence of parenchymal or pleural disease due to asbestos. Pleural abnormalities were reported 10 times more frequently than paranchymal disease. However, concern was expressed over the uncertainty of the prognosis in men with pleural abnormalities. At Devonport 37 men developed pleural mesothelioma and 128 asbestosis cases were reported since 1965.

A further component of Harries initial study was a proportional mortality analysis.[110] In this analysis, which compared male Devonport dockyard workers with a control group of other Plymouth males, it was observed that lung cancer had only a slightly raised mortality rate that was not statistically different from that in the general public.

TABLE 2.5: Prevalence of radiographic asbestos abnormalities (%) in 10% random sample.

Dockyard	No. Examined	No. with abnormality	Prevalence
Devonport	1414	63	4.5
Portsmouth	1017	32	3.1
Chatham	765	25	3.3
Rosyth	660	12	1.8
All yards	3856	132	3.4

The randomly selected 10% samples of the population at Devonport, chosen by Sheers and Templeton, at Portsmouth, Chatham and Rosyth, chosen by a team of investigators (including Sheers and Harries) formed the baseline data for future 'Royal Naval Dockyard Asbestosis Studies' reported on in the late 1970s and early 1980s. In these studies 10% samples and the complete workforce of the dockyards were scrutinised for radiographic abnormalities, morbidity and mortality.

Harries et al in 1975, in the first Royal Naval report on the asbestosis studies, summarised asbestos research in naval dockyards and reported on morbidity at Devonport, Chatham, Portsmouth and Rosyth.[115] Here the entire workforce of the four yards was examined radiographically, using mobile radiographic units. The results confirmed those of the 10% sample and gave an overall prevalence of radiographic asbestos abnormalities of 4.6% (table 2.6). In both tables 2.5 and 2.6 the term radiographic abnormalities is given to these workers with x-ray evidence of pleural thickening, pleural calcification, or either suspected or definite pulmonary fibrosis. Tuberculosis (active and clinically inactive) was not included in this definition. The higher prevalence seen at Devonport and Portsmouth is explained by the larger number of major warship refits undertaken at those dockyards. For pleural thickening the risk in smokers was shown to be approximately 25% greater than that for nonsmokers.

TABLE 2.6: Prevalence of radiographic asbestos abnormalities (%) in entire workforce.

Dockyard	No. Examined	No. with abnormality	Prevalence
Devonport	10165	468	4.6
Portsmouth	6779	386	5.7
Chatham	4828	179	3.7
Rosyth	2803	101	3.6
All yards	24575	1134	4.6

Evidence of an occupational effect due to asbestos exposure was sought by Lumley in 1976 when comparing cancer registrations for Devonport dockyard workers with those for Plymouth men in the same age groups for 1960-69.[116] The overall death rate was similar in both groups. However, an excess of cancers of the respiratory tract was found in this period (confined to pleural tumours). Twenty-two cases of pleural mesothelioma were recorded in the dockyard compared to only 3 in other Plymouth males. A trend of increasing annual excess of stomach and gastrointestinal registrations was also observed in the dockyard group.

Harries and Lumley in 1977 presented a survey of Registered Asbestos Workers (RAWs) in the four dockyards.[117] In this survey 990 male RAWs were studied, these included laggers, sprayers, storemen and workers indirectly exposed to asbestos (i.e. boilermakers, shipwrights, electrical fitters, etc). It was found that radiographic, clinical and physiological abnormalities associated with exposure to asbestos only occurred in older men exposed before enforcement of the Code of Practice.

In a sample of 1200 men aged 50-59 years from all four dockyards, Rossiter and Harries in 1979 confirmed that smoking partly accounts for the increasing dockyard prevalence rates of radiographic, clinical and lung function

abnormalities.[118] However, the prevalence of pleural calcification, seen to be related to duration of asbestos exposure, was highest in nonsmokers. The most extensive disease was seen in laggers and sprayers employed before 1957.

Sheers, also in 1979, reported on a follow-up of his 1966 cohort.[119] It was shown that of 971 workers with normal radiographs in 1966, 96 (9.9%) had developed nonmalignant asbestos-related abnormalities by 1977. Thirty-eight deaths from mesothelioma had occurred during the study interval among men aged 35 to 64 with 20 or more years of asbestos exposure.

The incidence of mesothelioma among workers at Devonport dockyard was further scrutinized in 1980 by Sheers and Cole.[120] In this work, asbestos exposure histories were ascertained for 96 dockyard mesothelioma cases; the cases were classified as continuously or intermittently exposed to asbestos. The latency period from first exposure averaged 41 years; the shortest latency was associated with continuous exposure. Mesothelioma incidence was found to be correlated with both time from first exposure and occupation.

A nine year follow-up of men at Devonport dockyard was presented in 1980.[121] In this Rossiter et al showed that despite the almost complete removal from exposure to asbestos in 1968, radiographic parenchymal abnormalities increased in a group of 253 workers initially seen in 1966 and followed for the next 9 years. Progression of disease was greater for smokers than nonsmokers. Also in 1980 Rossiter and Coles undertook a mortality study in Devonport dockyard.[122] In this study highly elevated risks were observed for mesothelioma and pulmonary fibrosis, but no clear excess risk was present for lung cancer. As was mentioned in chapter 1 this was a very striking result, but not completely unexpected. The proportional mortality study of Harries in 1971 had already shown that there could well be only a negligible lung cancer risk at Devonport dockyard.[110] These studies, and the questions arising from them, effectively form the foundation of this thesis.

In another follow-up study, this time at Devonport and Portsmouth dockyards, McMillan et al in 1980 reported on the attack rates of pulmonary and pleural lesions due to asbestos dust.[123,124] The overall prevalence of asbestos-related lesions was over 13% at both yards, the prevalence rate being higher for current and ex-smokers than for nonsmokers. McMillan and Rossiter continued this work at Devonport dockyard and reported in 1982 on the development of parenchymal fibrosis in men with pleural lesions.[125] Here they concluded that the lesions may not be merely "markers" of previous asbestos exposure but may be identifiers of future parenchymal fibrosis.

McMillan in further work, at the Medical Research Unit of Devonport Dockyard over 1979-84, looked at the health of dockyard welders in relation to other trades, i.e. boilermakers, drillers, fitters, joiners, painters, plumbers and shipwrights.[126,127,128,129,130,131,132]

One of McMillan's conclusions was that there was no evidence of an association between shipyard exposure to welding fumes and gases and an increased risk of mortality due to respiratory diseases and gastrointestinal cancers. The risk of asbestos-related disease, apart from mesothelioma, among the welders being accredited to the heavy asbestos exposure during shipyard welding. It was shown that less than 1% of welders developed parenchymal fibrosis but that 13% had nonmalignant pleural lesions.

Wagner et al in 1986 reported on 333 necropsies on workers from Devonport dockyard.[133] These had been collected from 1966 to 1982. An exposure rating based on each worker's occupation and the number of years employed was formed. Severity of asbestosis correlated with exposure rating and fibre count. The fibre counts of crocidolite and amosite fibres found in lung sections increased with severity of asbestosis, the counts for chrysotile remained constant. Mesothelioma was seen to occur in those with minimal or slight asbestosis and low exposure rating, pulmonary carcinoma with moderate to severe asbestosis and high

rating. It was concluded that amphibole lung content may be correlated with severity of asbestosis.

In summary by the 1980s it was seen that:

> The prevalence of asbestos-related disease, particularly asbestosis and mesothelioma, was increasing in Royal Naval Dockyards.
>
> Before the mid 1960s there was no suggestion of any type of pneumoconiosis occurring in these yards, due to the long latency of these diseases. This must have been the main reason why energetic preventive measures were not undertaken earlier.
>
> By the 1980s sufficient time had elapsed for the effects of asbestos exposure to be seen in multiple diseases. This was aided by more intensive and improved medical investigations.

All of the studies in this section have shown that in Royal Naval Dockyards workers were exposed to asbestos, not only directly, but also by working in close proximity to other workers handling asbestos products. Before enforcement of the Code of Practice, those workers handling asbestos may well have used some form of respiratory protection and come under a limited medical supervision scheme, those working nearby were completely unprotected.

Since the 1980s the attention of the medical and scientific community has begun to focus more on the health effects of the asbestos replacement material, man-made mineral fibres. However, certain controversies concerning asbestos exposure still remain. Namely, is chrysotile asbestos less carcinogenic than the amphiboles, is lung cancer really only a complication of asbestosis, and is there a threshold dose below which asbestos is non-carcinogenic? These questions will be considered in the following section. The removal of asbestos from buildings insulated with asbestos material and the public's concern with possible environmental exposure will also be considered.

2.5 Current Concerns and Controversies in Asbestos Related Disease.

This section will consider the asbestos health related issues, debated in contemporary medical and scientific literature. It will focus on the concepts of cancer thresholds, linear dose-response relationships, the carcinogenic potential of different asbestos fibre types and dimensions, and the issue of fibrogenicity/carcinogenicity - which one comes first? It will consider the pros and cons of each side of the present debate. Specifically, the following questions will be addressed:

1. Is lung cancer a complication of pulmonary fibrosis (asbestosis), or a risk for exposed individuals with or without asbestosis? This question has been posed in many ways by researchers and linked with question two below. Browne in 1986 asked the question "is asbestos or asbestosis the cause of the increased risk of lung cancer in asbestos workers?"[134] McDonald, at the 1991 British Occupational Hygiene Society Annual Conference, more directly asked "does asbestos cause lung cancer in the absence of fibrosis?"[135]

2. Is there a threshold dose of asbestos exposure beneath which asbestos is non-carcinogenic, and is a linear dose-response relationship for lung cancer and mesothelioma realistic?

3. Is chrysotile asbestos less carcinogenic than the amphiboles? This question has sometimes been rephrased in the literature as "are the amphiboles alone responsible for asbestos disease risk" and termed the "amphibole hypothesis".[136,137] The current debate concerns the contamination of chrysotile asbestos with fibrous tremolite and whether this is the cause of an increased lung cancer risk in textile plants, and a high rate of mesotheliomas in asbestos miners and millers.[138]

Consideration will also be given to secondary questions, which concern smoking as a co-factor, and how the strikingly different dose-response gradients of risk observed, in the assorted asbestos trades and industries (over many studies), can be interpreted.

High exposures to asbestos are clearly hazardous. The public controversy today concerns risk at low doses, and how large that risk is. The question arising from this is whether high occupational risks can be extrapolated to low public risks (the form of the dose-response relationship clearly has great importance in this question). An associated question commonly asked is; are we increasing exposure to the general public by improperly removing asbestos from buildings that is best left in place? This point will also be considered.

2.5.1 Background to the Controversies.

The link between asbestosis and lung cancer bears directly on the relationship between the amount of exposure to asbestos and risk of lung cancer (i.e. the dose-response relationship). At the low doses of exposure (below 0.001 fibres per millilitre of air) that are a public health issue today, there are no data that directly address this point, however, several possibilities exist. If asbestosis develops first (and then sharply increases the risk of lung cancer), the dose-response relationships for lung cancer and asbestosis should be similar. Many scientists now believe that exposures above a certain threshold are needed for asbestosis to develop, with 25 f/ml years being the best judgement of lifetime occupational exposure, below which clinical manifestations cannot advance.[139,140] If lung cancer is directly connected to asbestosis, its risk should also be zero below some threshold of exposure. Conversely, if asbestos can cause lung cancer in the absence of asbestosis, an individual might incur some risk even at low levels of exposure.

The relationship between asbestos dose and carcinogenic risk is clearly important. A linear relationship between dose and risk suggests that if the dose is halved, the risk is halved. This also implies that some risk remains, even at very low levels of asbestos exposure. In a nonlinear relationship, the risk may fall off faster at low doses, perhaps exhibiting a threshold exposure below which the risk is zero. Consequently, a linear dose-response relationship would imply higher (more

conservative) levels of cancer risk at lower doses than a threshold or other nonlinear (perhaps sigmoid) model. For this reason alone the linear dose-response model has been supported by scientists and regulatory agencies.[3,141] In 1984 the National Academy of Sciences concluded in a review document that although a linear dose-response assumption may not be always justified it should lead to an appropriate upper bound for risk assessments of asbestos.[142]

Differences in lung cancer and mesothelioma risk have been shown with both fibre type and industrial process in numerous studies, and these studies have in turn been extensively reviewed.[143,144,145,146,147,148] From these reviews, the overall influence of asbestos exposure on health is clearly very substantial. An indication of the extent of this influence is shown in table 2.7, which summarizes the results of the cohort studies considered by McDonald and McDonald in 1987.[146,147]

TABLE 2.7*. Excess mortality in cohorts exposed to asbestos.

Disease group	Chrysotile only	Amphiboles only	Chrysotile-amphibole mixtures
Pneumoconiosis (taken from 21 studies)	83/4996 1.7%	102/2008 5.1%	254/6471 3.9%
Lung cancer (taken from 42 studies)	102/5827 1.8%	137/2187 6.3%	772/10882 7.1%
Mesothelioma (taken from 40 studies)	12/5476 0.2%	78/2187 3.6%	422/10904 3.9%
Total excess	3.6%	14.9%	14.9%

Each cell in this table shows the excess number of deaths for each disease group divided by the total number of deaths, with the proportional mortality expressed below. This meta-analysis shows that even without data on age, sex, smoking and

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* Adapted from: McDonald JC (1990) "Cancer risks due to asbestos and man-made fibres" Recent Results in Cancer Research **120**:122-131.

asbestos exposure intensity there is a consistent pattern of mortality across asbestos fibre grouping. The excess mortality of cohorts exposed to the amphiboles and chrysotile-amphibole mixtures appears similar but several times worse than those exposed to chrysotile alone. This large difference is partly explained by the rarity of mesothelioma in the chrysotile only cohorts.

2.5.2 Different Dose-Response Gradients.

As cited by McDonald in 1990, a more reliable indication of risk would be obtained from those studies that have attempted to estimate individual asbestos exposures in terms of intensity and duration.[149] When considering only lung cancer, this reduced the number of cohorts from those seen in the meta-analysis to 11 industrial populations presented by McDonald and McDonald in 1987.[147] Table 2.8 and figure 2.10 are taken from this publication. The exposure estimates from these studies were, however, considered to be usually scanty and inadequate in quality and little more than informed guesses.[147,149] There was also the associated problem of conversion from total respirable dust particles to fibre concentrations, considered problematic at best, with no one simple conversion factor.[144]

From the review of McDonald and McDonald, summarized by table 2.8 and figure 2.10, two features are evident. The first is that the exposure-response relationships appear linear and, if expressed as relative risks, pass through the origin. The second feature is that the gradients vary greatly with industrial process and fibre type. The risk of lung cancer from chrysotile exposure is seen to be far greater in the manufacture of textiles than in cement or friction products manufacture. Similarly, a difference of equal magnitude is exhibited between miners and millers of chrysotile and miners and millers exposed to tremolite. There were too few female lung cancer cases (23 over three industrial cohorts) to allow any interpretation. Although the relationships for mesothelioma were not as well quantified, higher risks were also observed in the same type of industry (e.g. mining, textiles and cement plants) when exposure included amphibole fibres rather than chrysotile alone.[146]

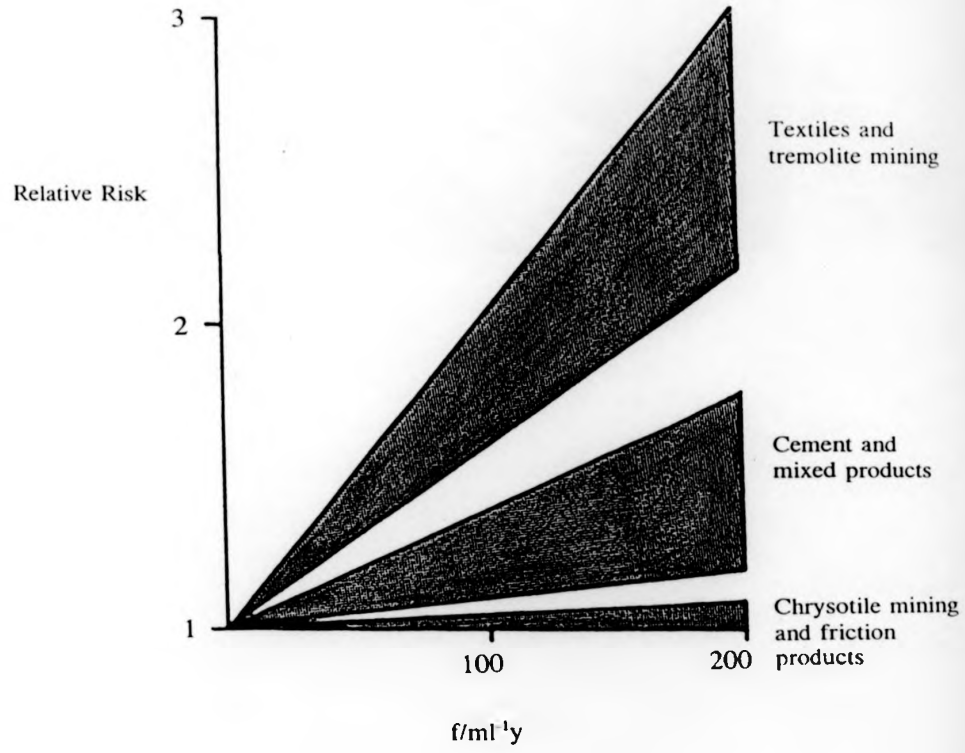

FIGURE 2.10: **Relative risk of lung cancer for different industrial processes and fibre types.**

TABLE 2.8: Cohort studies with individual estimates of accumulated exposure for male workers.

Reference	Place	Fibre	Lung cancer deaths	Increase in relative risk per f/ml^{-1} year
Mining and milling				
McDonald et al, 1979 [150]	Quebec	Chrysotile	230	0.0004
Amandus et al, 1987 [151]	Montana	Tremolite	20	0.006
McDonald et al, 1986 [152]	Montana	Tremolite		0.01
Textiles				
Dement et al, 1982 [153]	South Carolina	Chrysotile	26	0.01
McDonald et al, 1983 [154]	South Carolina	Chrysotile	59	0.01
McDonald et al, 1983 [155]	Pennsylvania	Mixed	53	0.0009
Peto et al, 1985 [156]	Rochdale	Mixed	93	0.01
Building materials				
Henderson and Enterline, 1979 [157]	USA	Mixed	63	0.001
Hughes and Weill, 1980 [158]	Louisiana	Mixed	51	0.004
Finkelstein, 1984 [159]	Ontario	Mixed	26	not linear
Albin et al, 1984 [160]	Sweden	Mixed	16	not linear
Friction products				
Berry and Newhouse, 1983 [161]	UK	Mixed	143	effectively zero
McDonald et al, 1984 [162]	Connecticut	Chrysotile	73	effectively zero

For lung cancer the picture seen in figure 2.10 had lead Liddell et al earlier to conclude, for the Quebec study, that "there was a clear direct relationship, which may well be linear, between excess lung cancer mortality and total dust exposure".[163] This conclusion was for workplace exposures with little thought given to possible future extrapolations to the very low levels of airborne asbestos fibre concentration experienced by the general public, or as Gaensler termed in 1992 to the following "pandemic of mediagenic disease".[164]

When considering asbestos friction product manufacture, table 2.8 highlights a remarkable finding. Although environmental measurements were similar in order of magnitude to those in the chrysotile textile plants, there was little if any convincing evidence of excess lung cancer in either the USA or UK cohorts. Also, the exposures classified as mixed for the study of Berry and Newhouse, in

fact consisted entirely of chrysotile asbestos, except for one well-defined work area were crocidolite asbestos was used for two short periods before 1944.[161] In a follow-up study at this factory Newhouse and Sullivan in 1989 reported on 13 mesothelioma deaths, 11 of which occurred to workers known to have been exposed to crocidolite in this work area.[165] No excess risk was observed for lung cancer in the workers followed-up from 1941 to 1986, and it was reported that since 1950 the asbestos fibre concentrations in air were maintained at between 0.5 and 1.0 f/ml.

Nicholson in 1986 showed, in a report to the EPA, that risk estimates for lung cancer derived from 14 asbestos-exposed cohorts varied by approximately two orders of magnitude.[145] This in part could reflect statistical variation, differences in hazards associated with different fibre types and dimensions, inaccuracies in exposure estimates, and/or the use of inappropriate lung cancer rates in calculating standardized mortality ratios. Nonetheless, wide variation was seen in the fractional increased risk of lung cancer for a one year exposure to one fibre per millilitre in all fibre groups (see figure 2.11). This report substantially covered the same ground as the review of McDonald; 10 of the 14 studies were also reviewed by McDonald. The EPA report, however, also included a separate section on insulation workers exposed to amosite asbestos.

In figure 2.11, considering chrysotile exposure, the highest unit exposure risk was found among workers in a textile plant that used chrysotile whereas the lowest risk was observed among chrysotile mine and mill workers; matching the pattern observed in figure 2.10. It was suggested that these differences may be related to differences found in the fibre size distributions (fibres being longer and more curly in mining and milling), and that fibre length and diameter strongly influences the potential for fibres to produce lung cancer.[145,162,166] A further unsubstantiated suggestion has been that the high lung cancer rates seen in American textile plants were related to the use of a co-carcinogen, mineral oil spray that was used in these plants over a 30 year period to reduce airborne dust levels.[167] A further observation was that tremolite asbestos contaminated the

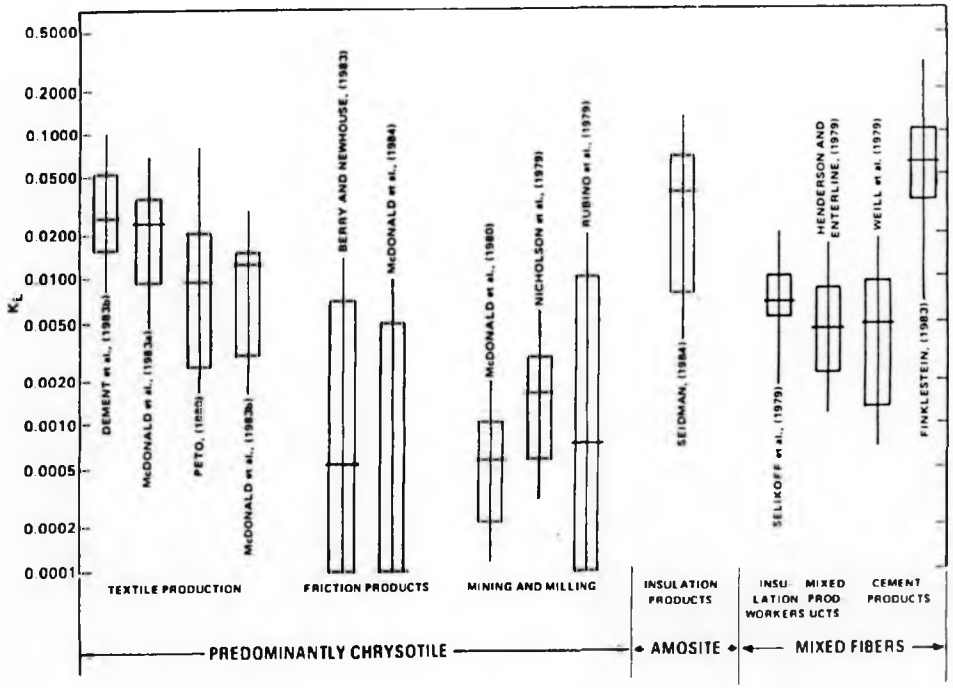

FIGURE 2.11: Values of K_L, the fractional increase in lung cancer per f-y/ml of exposure. The open bar reflects the estimated 95% confidence limits associated with measures of response. The line represents the uncertainties associated with measures of exposure.

chrysotile asbestos used in these plants, with the suggestion that this may be partially responsible for the high lung cancer risk.[167] Figure 2.11 shows 95% confidence limits for the unit exposure and indicates that there are highly significant differences between the different estimates. Nicholson pointed out "that differences between studies using the same fibre type far exceed those that exist between studies using different fibres".

The review of Nicholson, therefore, attempted a formal analysis of the variation seen between the different cohorts. It presented explicit confidence intervals for each estimate K_L, the fractional increase in lung cancer per fibre-year/ml of exposure, allowing for statistical variation and assuming a two-fold uncertainty in exposure estimates. In some cohorts, adjustments were made for suspected biases (i.e. the use of inappropriate lung cancer rates). When excluding the significantly lower risks per unit exposure for chrysotile mining and milling this analysis gave a geometric mean for K_L of 0.01, with an associated 95% confidence interval of 0.004 to 0.027 (i.e. approximately a factor of 2.5). This was based on an analysis of variance of 11 separate estimates. Nicholson estimated that the factor would be approximately 10 (i.e. a 10-fold range of uncertainty) for any unstudied exposure circumstance, with the proviso that it may be greater.

The only study to have a significantly higher unit risk than 0.01, in the 11 studies used in its estimation, was the Ontario asbestos cement products factory reported on by Finkelstein; a study considered to have questionable exposure estimates.[145] Nicholson observed that the data illustrated in figure 2.11 provides no evidence of a substantially different lung cancer unit exposure risk among different fibre types (after excluding studies of chrysotile miners and millers).

Data presented on the unit risk for mesothelioma (K_M) closely parallelled that found for lung cancer; very similar ratios of the unit exposure risks of mesothelioma and lung cancer were observed irrespective of the type of exposure experienced (although the magnitude of these varied greatly across fibre grouping). Suggesting that similar factors affect the variability of mesothelioma risk as affect

lung cancer risk. The best estimate for K_H was found to be 1.0×10^{-8}, with a 20-fold range of uncertainty. Nicholson noted that peritoneal mesothelioma was largely associated with amphibole exposure, whereas pleural mesothelioma was associated with exposure to chrysotile and crocidolite. Doll and Peto in their 1985 review concluded that peritoneal mesothelioma is rarely or never caused by chrysotile exposure, and that crocidolite and amosite are more dangerous (fibre types) than chrysotile when used in the same way.[144]

From the 1991 review undertaken by the Health Effects Institute (HEI) the clearest difference between the effects of different fibre types is seen in the proportion of mesotheliomas that are present in the peritoneum.[148] Almost all cases among chrysotile workers (usually with some exposure to crocidolite or tremolite) or among crocidolite miners are pleural, whereas workers with some amosite exposure have suffered similar and sometimes higher risks of peritoneal than pleural mesothelioma. The only exception appears to be female gas mask workers exposed mainly to crocidolite, among whom several mesotheliomas were peritoneal.[168,169,170] The possibility of some amosite exposure in these workers was not discussed by the authors of these reports. The inference is that most peritoneal mesotheliomas are caused by amosite exposure. For pleural mesothelioma a direct comparison was undertaken of workers employed for similar durations to different forms of asbestos in varying asbestos industries. This comparison displayed evidence of a higher risk for amphiboles than for chrysotile, supporting the conclusion of Doll and Peto.

There are marked differences between cohorts in the ratio of excess lung cancer to pleural mesothelioma.[144] The lowest reported ratio based on substantial numbers of mesothelioma cases occurred in British Dockyard workers exposed to a mixture of asbestos types.[122] This was the study of Rossiter and Coles at Devonport Dockyard, reporting a high mesothelioma risk with no corresponding excess of lung cancer, which was the foundation of this thesis. The highest occurred at a South Carolina chrysotile textile plant were there was a marked excess of lung cancer and no pleural mesothelioma.[153,154] These data have been

almost universally accepted as demonstrating that amphiboles, particularly crocidolite, cause a disproportionate mesothelioma risk.

Both Doll and Peto, and Nicholson showed that among men the ratio of excess lung cancer to pleural mesothelioma is approximately three times greater for chrysotile than crocidolite, varying from at least four for chrysotile to between one and two for crocidolite, with substantially lower ratios for women. It should be noted, however, that such pooling of often inconsistent cohort data is of dubious value. In fact, it conceals the most extreme inconsistencies, most notably the marked excess of mesothelioma without any detectable excess of lung cancer observed among British Dockyard workers, and in the subgroup of friction product workers with crocidolite exposure studied by Newhouse.[122,161,165]

The opposite view is that nearly all mesotheliomas are caused by amphibole exposure and that chrysotile causes a negligible mesothelioma risk. The only strong evidence against the inference that mesothelioma is almost never caused by chrysotile alone is the observation of substantial numbers of cases among Quebec chrysotile miners and millers. It has, however, been suggested that these are related to the presence of fibrous tremolite in the raw material.[138,171] Tremolite was found to compose less than 1% of the raw fibre but more than half of the long fibres ($>5\mu m$) found in the lung tissue of workers at autopsy.[167] Similarly, high levels of crocidolite were found in lung tissue from British textile workers who were exposed mainly to chrysotile but suffered a high incidence of mesothelioma.[172] The evidence that chrysotile rarely causes pleural mesothelioma is consequently consistent but not conclusive.

2.5.3 Smoking as a co-factor.

Any discussion of dose-response relationships for asbestos and cancer is complicated by the fact that asbestos seems to exert its effect synergistically with cigarette smoke, that is, the cancer risk is more than that from asbestos exposure and smoking considered separately. The usual reference for this is the 1979 study of insulation workers undertaken by Hammond et al.[58] Table 2.9 is adapted from that study. A further complication is that from the numerous asbestos studies appraised above most considered only male workers employed initially over the period 1940 - 1960, a time when male industrial workers were known to be cigarette smokers. Therefore, the number of asbestos workers with lung cancer who had never smoked in these cohorts is small and their smoking histories very uncertain.

TABLE 2.9: Risks of lung cancer caused by asbestos exposure and smoking (multiplicative relationship).

Asbestos/smoking group	Death rate*	Mortality difference	Mortality ratio	Predictive equation
Nonsmoker, non asbestos exposed	11.3	0.0	1.00	1
Nonsmoker, asbestos exposed	58.4	47.1	5.17	5
Smoker, non asbestos exposed	122.6	111.3	10.85	11
Smoker, asbestos exposed	601.6	590.3	53.24	55

* rate per 100,000 person years

The third column of table 2.9 records the mortality ratios (i.e. the relative risks) of asbestos exposure with and without smoking. Based on this, Hammond et al suggested that the risks of asbestos and cigarette smoking combine in a multiplicative fashion. In fact the relative risks can be simplified to the following expression:

$$\text{Relative Risk} = (1 + a)(1 + c) = 1 + a + c + ac.$$

Where a and c represent asbestos exposure and cigarette smoking and the term ac the synergistic effect between smoking and asbestos ($a = 4$, $c = 10$). Therefore, compared with a nonsmoker with no asbestos exposure, a smoker has a 11-fold higher risk for lung cancer, a nonsmoking asbestos worker has a 5-fold higher risk, and a smoker with asbestos exposure has a 55-fold higher risk. The results of this predictive equation agree remarkably well with the data (table 2.9). No other study has produced such a good fit to an equation like this that includes a multiplicative term. By contrast, McDonald in 1980 found something closer to an additive effect, in which the relative risk is close to the sum of risks from smoking and asbestos considered separately (table 2.10).[173]

TABLE 2.10: Risks of lung cancer caused by asbestos exposure and smoking (additive relationship).

Smoking group	Asbestos exposure		
	Little	Moderate	Heavy
Nonsmokers	1.0	2.0	6.9
Moderate smokers	6.3	7.5	12.8
Heavy smokers	11.8	13.3	25.0

Berry et al in 1985 pooled the results from 6 cohort studies (table 2.11) and expressed the relative asbestos effect (the ratio of the relative risk in nonsmokers to smokers) for each.[174] Except the study of Quebec miners and millers expected numbers of cases in smokers and nonsmokers were obtained from data outside the cohort database. For the three American studies this was obtained from samples matched for education and occupation from data collected by the American Cancer Society. For the remaining UK studies it was obtained from the results of Doll on lung cancer in British doctors.

From table 2.11 a multiplicative relationship between smoking and asbestos exposure is seen when the 95% confidence interval is significantly greater than one. Therefore, a multiplicative interaction is detected by this method only for

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which is directly cytotoxic, effectively absorbs hydrocarbons and this probably accounts for the higher (multiplicative) risk of lung cancer in individuals who have histories of asbestos and cigarette smoking.[176] Antman additionally speculated that the lack of any relationship between cigarette smoking and malignant mesothelioma most likely reflects the earlier development and death from lung cancer in smokers, removing them from the risk pool for mesothelioma. An alternative, and more reasonable explanation, is that translocation may move fibres, but not the hydrocarbons.

When considering the probability of causation (i.e. the probability that asbestos caused any particular case of mesothelioma or lung cancer) the following appears to be the case for mesothelioma and lung cancer:

For mesothelioma, which is so rare that whenever it develops in an exposed individual the cause is usually assumed to be asbestos, asbestos appears to be the causative factor. Against this hypothesis, however, are cases of mesothelioma and lung cancer occurring in a Turkish village attributed to exposure to erionite, a locally obtained fibre used in building materials.[177] Also against this argument are the cases of spontaneous mesothelioma occurring in the general public (i.e. the background level of mesothelioma). Pelnar in 1988 reviewed cases of non-asbestos related mesothelioma; in this work spontaneously occurring mesothelioma included mesotheliomas of the tunica vaginalis testis, pericardium, anterior mediastinum, and myocardium.[178] It was also noted that the Salk polio vaccine, used by injection in the early 1960s, was contaminated with the SV40 virus, a virus that was shown to cause mesothelioma in laboratory animals. It was pointed out by McDonald in 1985 that the background level of mesothelioma incidence (non-asbestos related) was approximately 2 cases per million, supporting the argument that mesothelioma is a rare disease despite the SV40 virus and the more spontaneous forms of mesothelioma.[179]

Lung cancer is more difficult to attribute simply to asbestos because of the strong synergistic relationship between asbestos and smoking. Lung cancer in a

nonsmoker would normally be attributed to asbestos when there is a history of asbestos exposure. With smokers the situation is not as clear. A nonsmoker exposed to asbestos is less at risk of lung cancer than a smoker with no exposure to asbestos. The inference from this is that smoking is the main causative factor, however, the combined risks of smoking and asbestos exposure far exceed those of smoking alone (by a factor of 5). Sound public health practice, whatever the form of the smoking/asbestos interaction, is accordingly to advise asbestos-exposed individuals not to smoke.

2.5.4 Fibre Dimensions.

Considering now the dimensions of the asbestos fibre, and the associated question of what are the critical fibre dimensions for carcinogenicity? The original fibre size, for regulatory purposes*, was set long before there was any consensus on biologically critical sizes and was based on the use of the membrane filter and light microscope methods. Each of the asbestos fibre types appears to possess its own size range in airborne and tissue evaluations with amosite fibres generally being the thickest in diameter followed by crocidolite and then chrysotile (the finest).[180,181] Industrial hygienists currently classify fibres of asbestos as fibres that are greater than 5 microns (5μm) in length with a diameter greater than 0.25μm having an aspect ratio (length/diameter) greater than 3:1. This classification is often used to evaluate whether fibres are possibly carcinogenic (those longer than 5μm are commonly taken as effective carcinogens). Generally, shorter fibres are considered less effective per fibre than long fibres ($>5\mu$m) because the macrophages, the white blood cells, can envelop and remove these but not the long fibres.

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* The original regulatory fibre size for asbestos fibres was: fibres with a minimum length of 5μm, a diameter less than 3μm, and a length to diameter ratio greater than 3:1. The HSE regulatory document was MDHS 39 issued in 1984, superseded in 1988 by MDHS 39/2, and finally superseded in 1990 by MDHS 39/3.[182,183,184]

Lippmann has comprehensively reviewed the available hygiene and medical data and concluded that fibres of different shapes and sizes cause different diseases, dependant on a certain critical minimum length that varies with disease.[185,186] This length was stated to be 2μm for asbestosis, 5μm for mesothelioma, and 10μm for lung cancer. For asbestosis and lung cancer, where the fibres must be retained in the lungs, fibres with a diameter less than 0.15μm appeared to pose no risk; for mesothelioma the hazard seemed linked to diameters finer than 0.5μm. These conclusions on the biologically critical size of asbestos fibres support the regulatory guidelines that fibres longer than 5 μm are effective carcinogens, however, they have not been conclusively established. McDonald et al in 1989 performed an autopsy study on 78 mesothelioma cases and matched controls.[187] In this study it was observed that long amphibole fibres ($\geq 8\mu$m) differentiated effectively between cases and controls, whereas no difference was observed for chrysotile or short fibres. Suggesting here a minimum critical fibre length for mesothelioma of 8μm.

It seems very probable that the proportion of airborne or tissue fibres of different size will vary with the origin and treatment of the asbestos, whether it is being mined or milled, and what it is being used to manufacture. As previously mentioned it is also probable that the physical attributes of the varying fibre types and their dimensions may control their carcinogenic potential.[144,162] The debate being that the curlier chrysotile fibres found in mining and milling are less carcinogenic than the chrysotile fibres found in textile factories (these in turn being less carcinogenic than the sharper amphiboles), curlier fibres being more easily cleared from the upper bronchus. Timbrell showed that chrysotile fibres have a large effective diameter because of their curliness and so tend to be intercepted and deposited at the bifurcations of airways before they penetrate lung tissue.[188] Against this hypothesis are animal studies which show that chrysotile is more harmful than the amphiboles.[189,190] The inference is that in humans, because of their longer life span, chrysotile fibres are removed from the lungs before disease can develop. Clearly fibres dissolve according to chronological time, but interactions with cells occur in relation to biological time,

two very different time scales. Using life span as an index, human biological time runs approximately 30 times slower than that of a rat.

In all considerations of lung fibre dimension and burden the carcinogenic mechanism is important. If asbestos fibres are primary carcinogens, then retention is important. If they transport chemical carcinogens or activate some latent virus, then fibre retention may not be relevant. Whatever the case it may still be speculated that fibre type and dimension play a part in explaining the variable dose-response gradients seen across the numerous asbestos cohorts.

2.5.5 Low Level Environmental Exposure.

With respect to low level environmental asbestos exposure the 1991 review undertaken by the Health Effects Institute compiled the available American non-litigation data, both published and unpublished.[148] From this the concentrations of asbestos fibres longer than 5μm generally showed average concentrations in the order of 0.00001 f/ml for outdoor rural air (except near asbestos containing rock outcroppings) and average concentrations up to 10-fold higher in the outdoor air of urban environments. However, outdoor urban average concentrations above 0.0001 f/ml were reported close to major roads (presumably due to vehicle braking) and near building demolitions. Data on ambient indoor levels of asbestos from direct transmission electron microscopy measurements were averaged from a number of buildings and building types (see figure 2.12). From 1,377 air samples (obtained from 197 buildings) the overall average concentrations (by building type) ranged from 0.00019 to 0.00051 f/ml. The overall recommendation from this was that asbestos containing materials in well-maintained buildings are unlikely to expose office workers and other general building occupants to toxic levels of asbestos fibre concentration, with the suggestion that it was better not to remove such material. This review felt that exposure to radon and environmental tobacco smoke was more of a health threat.

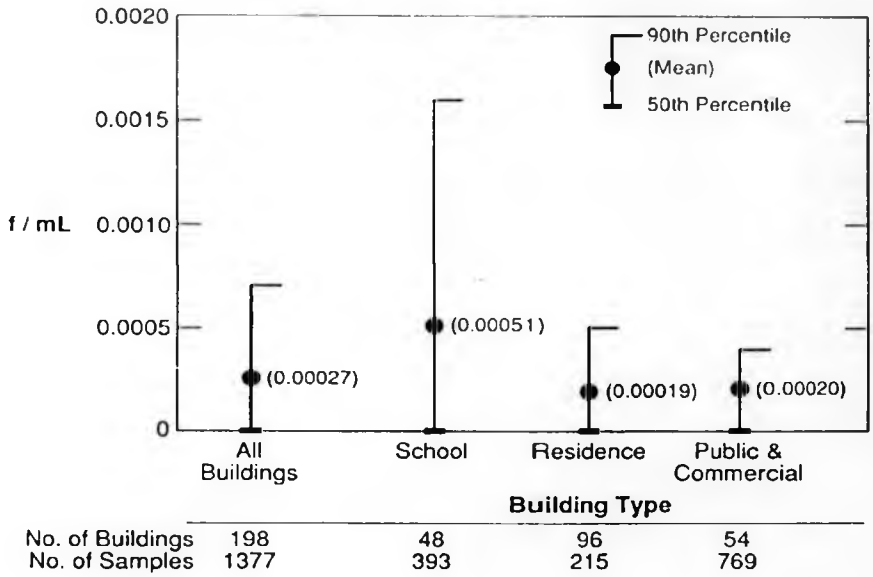

FIGURE 2.12: Summary of average airborne asbestos fibre concentrations in public and commercial buildings.

Burdett et al in 1989 had undertaken a similar summary of UK buildings.[191] The data supported the above recommendation, with indoor asbestos concentrations in the same range for well-maintained buildings (< 0.001 f/ml). However, it was observed that after asbestos had been removed from buildings the average airborne concentrations increased for several months after removal. In certain cases the airborne levels were approximately 40 times higher than normal background levels 6 months after removal (figure 2.13). The authors concluded that the management (in place) of undamaged asbestos material may be preferable to large scale removal projects. In which case it would be unlikely that levels would exceed 0.0005 f/ml in normal occupancy. This view has wide support, with large scale removal seeming to give more risk from increased exposure. Peto addressing environmental health students in April 1994, theorised that among construction workers exposed to asbestos during removal operations the risk would eventually materialise in higher mesothelioma mortality rates.[192]

These low level environmental exposure ranges should be placed in context, table 2.12 attempts this. From this table typical average exposure ranges are illustrated for various settings with a crude ranking of exposure (taking building occupancy exposures as the baseline measure). From this we can see that past occupational exposures carried a very high risk when compared to present day general public exposures. The present day exposures being in the order of 4×10^{-6} smaller than dockyard and insulation exposures and 3×10^{-5} less than those seen in textile factories, mining and milling, and asbestos cement manufacture. The data taken from Burdett et al support Peto's theory showing that construction workers may well face an elevated risk of asbestos-related disease.[191] In Third World countries strip mining and milling of asbestos continues unabated (particularly in South America and India) with limited control measures and not surprisingly an increasing risk of asbestos-related disease matching that seen in industrialized countries 20 years ago.[193,194]

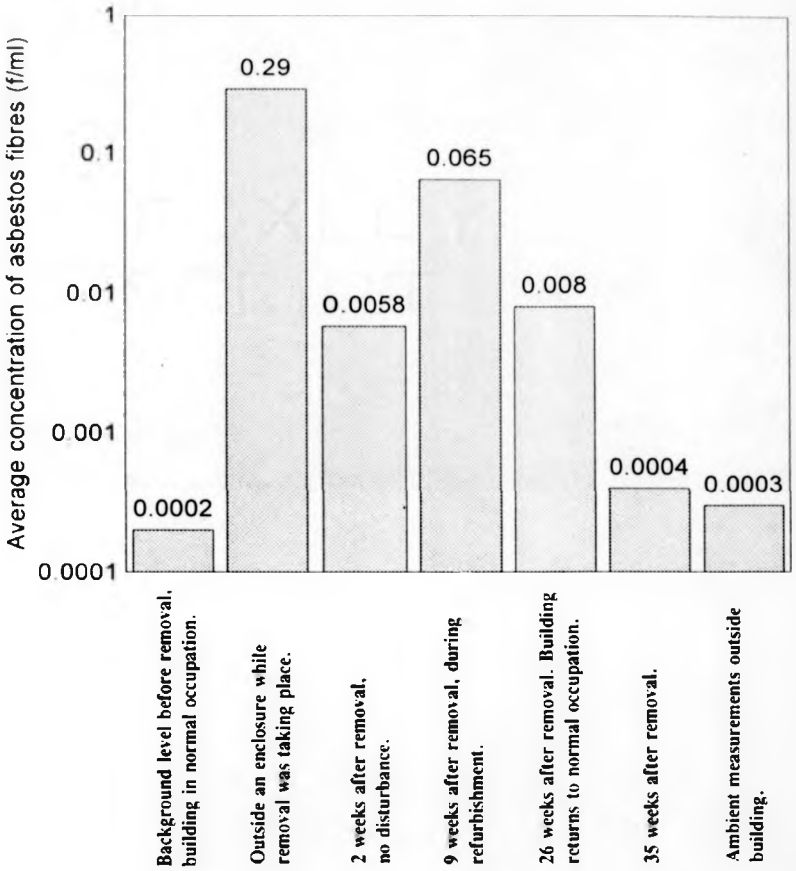

FIGURE 2.13: Average TEM concentrations of asbestos fibres ($>5\mu m$) before, during and after an asbestos removal programme at a six-storey school building.

TABLE 2.12: Potential asbestos exposure ranges.

Occupational setting	Average exposure f/ml	Exposure ranking
Building in normal occupation	<0.001	1
Construction workers (during asbestos removal, from figure 2.13)	0.29	290
Historical and potentially present Third World exposures:		
Asbestos miners and millers	4-50	27000
Textile workers	30	30000
Asbestos cement workers	6-60	33000
Dockyard workers	10-500	255000
Insulation workers	50-500	275000

Peto in 1989 predicted that for an average fibre concentration of 0.001 f/ml the life long risk is of the order of 1 in 100,000 for 10 years building occupancy.[195] The number of individuals exposed to this level of airborne asbestos are unknown. However, Peto assumed that 1 in 5 of the population are exposed and concluded that this would imply approximately one death per year in the UK, 5 per year in the USA, or correspond to an average loss of expected life of about 15 minutes. For comparison, more than 100 deaths occur in the city of Leeds each year due to transport accidents.[196] Peto's calculation would imply that low level environmental asbestos exposure with concentrations less than 0.001 f/ml presents a negligible public risk. It should be noted that asbestos concentrations in buildings, except during asbestos removal, are seldom much higher than concentrations in the air outside and that much of this small risk is related to the entry of outdoor fibres into the buildings with the ventilation air. The risk estimate would obviously increase when removal activity increases cumulative doses to both workers and occupants (and for individuals involved in other asbestos work). Peto concluded that the campaign to eliminate all asbestos on the grounds that 'one fibre can kill', besides being a cost-benefit absurdity, may actually increase the public risk.

2.5.6 The Principal Controversies.

In this section I will consider the arguments both for and against each of the principal asbestos controversies.

Controversy 1 concerns the possibly that lung cancer occurs in asbestos exposed workers as a complication of asbestos-induced fibrosis and not as a primary pathologic event. The issue could further be stated as whether fibrogenesis plays a role in carcinogenesis independent of exposure dose? The question addressed here, however, is:

> *Is lung cancer a complication of asbestosis, or a risk for exposed individuals with or without this disease?*

When considering disease prevention, if lung cancer risk was simply limited to those individuals with asbestosis then primary and secondary prevention of lung cancer could be obtained by both preventing sufficient exposure to induce fibrosis (assuming a threshold level exists for asbestosis) and then by focusing on those who have (or have a chance of developing) asbestosis. If pulmonary fibrosis is not the root cause of lung cancer, then the shape of the lung cancer dose-response relationship (whether linear or sigmoid, etc.) clearly becomes more significant and its implications applied to a larger population of exposed individuals both with and without non-malignant pulmonary disease.

The arguments that have been presented in support of lung cancer being a complication of asbestosis are as follows:

> In laboratory inhalation experiments close relationships have been observed between the degree of asbestosis and the incidence of lung tumours in animals exposed to asbestos.[187,197,198,199] Also in laboratory experiments, asbestos fibres do not appear to be cancer initiators in any of the standard in vitro tests suggesting an alternate route for the observed in vivo carcinogenesis.[200,201,202,203]

In one of the largest and most detailed of the published asbestos cohort studies (the Quebec chrysotile miners and millers) most of the lung cancers occurred in a subgroup of individuals with prior radiographic evidence of asbestosis.[204] The study of Louisiana asbestos cement product workers, though much smaller, supports this finding.[158,205] When reviewing the published mortality data from the remaining asbestos cohort studies (including those without exposure-dose data) a close association is observed between the relative risks of lung cancer and non-malignant respiratory disease.[3,139,143,144,145,148]

Lung cancers in asbestos exposed workers are seen to frequently originate in the lower lobes; since the interstitial fibrosis of asbestosis normally begins in the lower lobes an aetiological connection is suggested.[134,206,207,208,209,210] An excess of adenocarcinomas (the cell variant bearing the least relation to smoking) has also been observed in subjects with confirmed asbestosis, suggesting a potential mechanistic relationship.[207,208,211] Certain other fibrotic lung diseases unrelated to asbestos (e.g. idiopathic pulmonary fibrosis) are associated with interstitial pneumonitis (the typical pathologic lesion in asbestosis) and are also associated with an increased risk of lung cancer.[212,213,214] This may imply that the lesion, not asbestos fibres, is the basis of succeeding lung cancers.

The arguments against lung cancer being a complication of asbestosis are as follows:

Asbestos fibres are not seen to have tumour-initiating properties in standard laboratory tests, but experimental animal models have demonstrated an in vitro tumour-promoting effect.[215,216,217] These have included direct fibre acceleration of squamous metaplasia and intracellular fibre-born delivery of polyaromatic hydrocarbons. These models would support a theoretical mechanism for asbestos carcinogenesis independent of any fibrogenic effect.

Lung cancers appear to occur in excess in heavily exposed individuals without clinical or pathologic evidence of asbestosis; giving no support to the argument that lung cancer is simply a complication of fibrosis.[172,218]

In the Quebec chrysotile miners and millers study a limited amount of the excess lung cancer mortality occurred in individuals without prior

radiological evidence of asbestosis.[204] In the study of American friction products workers exposed to chrysotile asbestos (undertaken by the same research team as the Quebec project, viz. Professors JC McDonald and AD McDonald) it was further suggested that asbestos may be a complete carcinogen for mesothelial cells but only a promoter for bronchial cells.[162] A study of short term American amosite insulation workers also displayed a clear excess of lung cancer in workers with no demonstrable mortality from asbestosis.[219] In this study it was also concluded that asbestos acts as a classical tumour promoter (requiring earlier interaction with an initiator before malignant change can take place).

With the reported excess of adenocarcinomas in subjects with asbestosis, squamous cell tumours have also been observed in asbestos exposed workers.[218,220] It is therefore likely that the phenomenon of adenocarcinoma being secondary to asbestosis would explain only a relatively small portion of the total cancer burden associated with asbestos exposure.

Finally, the multiplicative relationship between asbestos and tobacco is hard to equate with the view that fibrosis, which if at all, is only minimally related to smoking, is the primary pre-neoplastic lesion.

From these arguments the current data clearly supports the idea of a close biological relationship existing between these two respiratory tract toxicities of asbestos. However, the evidence that asbestosis is a required precursor to lung cancer remains at best unclear (there appears to be equally good evidence to both support and reject this hypothesis). It consequently remains plausible that asbestos could be carcinogenic without the interposition of asbestosis. From the available data a simple inference could be that where fibrogenic dust goes so goes carcinogenic dust. From this if it is assumed that the risk of asbestosis increases with increasing total dose of asbestos it cannot be firmly established whether an increase in lung cancer risk is due to cumulative asbestos exposure or the asbestosis itself. Because the effects of asbestos exposure combined with smoking increase the risk, any assessment of lung cancer risk must allow for smoking habits of the asbestos workers and comparisons. Until this is done the influence of asbestosis on the risk of developing lung cancer cannot be clearly established. Finally the question "Is it statistical?", meaning is this controversy really a

statistical artefact (as it is very difficult to disentangle the causation from the association, when lung cancer has a number of causes) will also need to be addressed.

Controversy 2 concerns the likelihood of there being a threshold dose of asbestos below which there is no carcinogenic effect. This is a question of great importance, particularly to regulatory agencies. If a threshold can be shown for individuals occupationally exposed to asbestos then most of the anxieties about current environmental exposures are misplaced. Current environmental exposures are many orders of magnitude less then past occupational exposures and the anxieties are concerned with extrapolations of the linear dose-response model to these low levels, and the associated 'one fibre can kill' theory. Conversely, if a threshold does not exist then this would be evidence against the idea that asbestosis is a prerequisite for the development of lung cancer (again assuming that asbestosis itself requires some minimal threshold dose). The question addressed here then is:

> *Is there a threshold dose of asbestos exposure beneath which asbestos is non-carcinogenic, and is a linear dose-response relationship for lung cancer and mesothelioma realistic?*

The arguments that have been presented in support of a threshold for lung cancer are as follows:

> If lung cancer is a complication of asbestosis (despite the arguments presented above), then the evidence that induction of asbestosis requires at least some minimum threshold dose of exposure would be prima facie evidence for a cancer threshold as well.
>
> Reviewing the published asbestos studies that provided information on individual duration and intensity of exposure revealed evidence of a threshold of exposure below which the risk of lung cancer does not appear to be raised.[221] Of the studies reviewed the study of UK friction product workers failed to show a significant excess at any measured dose,

and several other studies in dockyards and asbestos cement factories showed no excess cancers despite other asbestos-related effects.[122,161,165,222,223,224,225] One well-studied general population, non-mining residents of Quebec asbestos mining regions, has been shown to have higher than normal ambient exposures to asbestos but with no suggestion of an increased cancer risk.[180,226,227] This would suggest a possible threshold effect in these settings.

A linear dose-response relationship is used by many regulatory agencies. Unfortunately, the inaccuracies in exposure estimates will tend to smooth curves and obscure thresholds, making a threshold response appear linear.[221,228]

Asbestos fibres do not appear to be tumour initiators.[200,201,202,203] Consequently there is no compelling theoretical reason for assuming absence of a threshold. In animal experiments spontaneous lung neoplasms periodically develop.[190,199,208] The carcinogenic response to low dose exposures in the laboratory has accordingly not been positive, giving some support to the idea of a dose threshold.

It is difficult to believe that the physiological defence and repair mechanisms are without effect. Such mechanisms must surely prevent or reduce disease at low dust concentrations generating an effective threshold.

The arguments against a dose threshold are:

In the cohort mortality studies with dose information showing possible threshold levels, a linear dose-response has nevertheless also been demonstrated suggesting perhaps that methodological problems and not biological discontinuities are the reason for the apparent threshold observed. In particular, errors and bias in exposure assessment and in death certification may obscure excesses in the least exposed subjects.

In the 11 asbestos studies reviewed by McDonald and McDonald in 1987 there is no clear evidence of a single potential threshold level.[147] Figure 2.14 illustrates the problem of detecting whether the 11 fitted dose-response lines show evidence of a threshold. Mortality ratios at zero exposure range from approximately 0.5 to 1.5, none passing through the origin. The explanation for this is presumably the incomparability of the reference populations.[149]

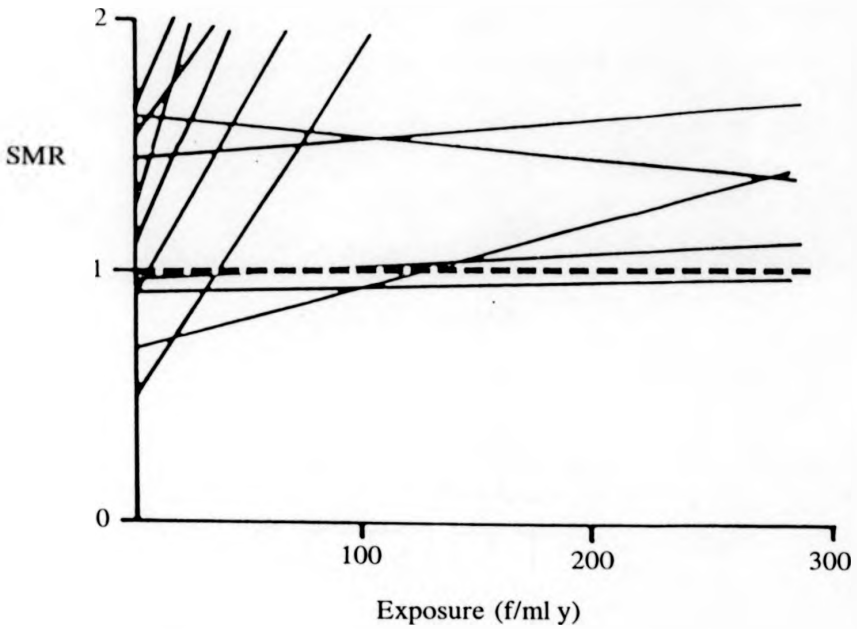

FIGURE 2.14: Lung Cancer SMR by exposure to asbestos fibres (exposure-response relationships from 11 studies).

In two asbestos cohorts with steep linear dose-response gradients (the chrysotile textile workers studied by Dement and the amosite insulation workers studied by Selikoff), workers with very low exposure (less than 5 f/ml years in one, and less than 1 month exposure in the other) had an increased cancer risk.[153,219,229] These studies and a further study of Australian crocidolite miners where asbestosis occurred at very low levels of exposure, therefore, produced evidence in favour of a linear no threshold model.[228,230] This last point argues against the view that pulmonary fibrosis requires a minimal threshold dose. The inference being that even if asbestosis is a precursor for asbestos-induced lung cancer, there may still be no safe exposure threshold in terms of lung cancer risk.

There is growing evidence that asbestos is a direct tumour promoter.[215,216,217] Linked with this is the knowledge that asbestos fibres can remain in the lung for many years. This provides a possible basis for concluding that a threshold is unlikely, as each fibre may confer a finite promotional effect. Animal inhalation studies have also shown a clear linear dose-response relationship for all levels of asbestos dose.[190,198]

The arguments for and against a dose threshold for cancer are each seen to lack conclusive evidence. For the argument against the threshold effect the strongest evidence is given by the studies of Dement and Selikoff were workers with low exposures, or limited duration of exposure, are seen to have a positive cancer risk. These analyses are weakened, however, by being standardized mortality studies in which the comparison with the general population could falsely elevate relative risks. Lamm et al in 1988 pointed out that finding an excess of lung cancer among workers with short durations of exposure and employment is common because of the effect of undocumented past exposures in previous employment.[231] This would also weaken the argument. The clearest evidence for a threshold is the absence of a significant excess of lung cancer at the lowest doses in the published occupational studies. Unfortunately, given the difficulties of past dose assessment and possible misclassification biases, it would be an error to over interpret these findings. On the other hand the biological argument for a threshold is fairly compelling but somewhat speculative.

In all the published studies exposure is commonly expressed as the simple product of duration of employment and average dust concentration, the effect of these two components being used almost interchangeably. From these studies duration of employment is normally well documented but average dust concentrations are approximate at best. Considering this point the linear nature of the fitted dose-response lines might be more easily explained by duration of exposure with only a limited contribution from concentration.[147] This would be a problem since it is the relationship to dust concentration that is required to be generalised. McDonald in 1990 presented an analysis of 58 cases of lung cancer in the Quebec cohort with less than 2 years employment and of 98 controls matched for smoking habit, year of birth, date of employment, gross service and mining area.[149] No relationship (either linear or threshold) was seen in this analysis between dust concentration and lung cancer risk after allowing for duration of exposure (see table 2.13). This clearly underlines the problem in the linear dose-response models, is it duration of employment or average dust concentration that has a straight line effect? Only future studies will be able to definitely answer this point.

TABLE 2.13: Lung cancer in men employed less than 2 years.

Concentration (f/ml)	Cases (n)	Controls (n)	Risk Ratio
<10	27	44	1
10-29	8	18	0.7
30-99	16	18	1.4
100+	7	18	0.6

It is worth noting that where estimates of exposure are given, the threshold for increased risk of lung cancer appears to be somewhere between 25-100 f/ml years.[221] The lower end of this range matches the threshold for asbestosis suggested by the Ontario Royal Commission (25 f/ml years; equivalent to inhaling about 30,000 million fibres in a lifetime).[3] A threshold for asbestos-related lung

cancer at or above the threshold for asbestosis does not prove that the risks are linked. However, it is consistent with the hypothesis that an increased risk of lung cancer occurs in individuals with asbestosis. It should be noted that further work has been undertaken which implies that this threshold level for asbestosis may be too high. In an autopsy study of South African amphibole asbestos miners, significant proportions of the workforce exposed to fibre concentrations of 2 f/ml or less were seen to develop asbestosis. It was concluded that if there is an average fibre concentration to which workers can be exposed for prolonged periods without developing asbestosis it is certainly less than two fibres at the working place.[232]

Regulatory agencies have taken a utilitarian view of the dose-response relationship. They have chosen to use a linear (non-threshold) model since the risk estimates from this would lead to the most conservative upper limits for risk assessment at low levels of asbestos exposure. Unfortunately, this view point has also caused much public anxiety when it resulted in the 'one asbestos fibre can kill' theory. A theory difficult to apply under a threshold or sigmoid dose-response model. Which is the correct, or the best, dose-response model? Epidemiologically this question can only be answered by the long-term follow-up of those cohorts of workers who have had well-quantified but low-level exposures (e.g. present day construction workers involved in asbestos removal).

The arguments above also apply to mesothelioma, although here there are no data on the form of the relationship to cumulative dust exposure. Nonetheless these tumours because of their highly specific relationship to asbestos and their distinctive clinical features provide a measure of environmental impact. During the past two to three decades the patterns of mesothelioma incidence in North America and the United Kingdom have been very similar. Mortality attributed to mesothelioma has increased steeply in men but remained relatively unchanged in women. In the UK mortality rose from 4 per million in 1968 to 8 per million in 1978 for men, whereas the incidence in females remained steady at 2 per million.[146] As was mentioned in section 2.5.3 McDonald has calculated that a

normal background level of mesothelioma incidence is approximately 2 per million.[179] The deduction from this is that the rate for males reflects an occupational effect, whereas that seen in females represents a more ambient environmental exposure. As there have certainly been some cases in women resulting from both occupational and domestic exposure, the absence of any increased risk over these years is striking. It suggests that cases due to very low level exposure are too few to be detectable. Supporting this is also the idea of a threshold for mesothelioma, although this has not been clearly proven.[233]

Browne in 1987 when reviewing the 'one fibre' theory summarised an address given by Professor M Kuschner at the WHO 1986 Copenhagen conference on man-made mineral fibres.[234] Professor Kuschner is reported to have concluded that it is his belief that fibres (presumably all mineral fibres) have a single pathological effect, not three separate pathologies, namely cancer of the lung, mesothelioma of the pleura and lung fibrosis. If this is the case then the threshold/non-threshold controversy for lung cancer also applies to mesothelioma. Whatever the case the arguments both for and against a dose threshold or a linear model for asbestos-related cancer are each seen to lack conclusive evidence.

Controversy 3 concerns the possibility that chrysotile asbestos is less potent as a lung carcinogen than amphibole asbestos. In a testimony to NIOSH, Lemen in 1990 concluded that chrysotile is much more chemically and biologically reactive than amphibole fibres.[235] He further concluded that there is no compelling evidence at this time to justify different public health policies for different asbestos fibre types, with the scientific evidence suggesting that fibre shape and size are the most critical factors in the pathogenicity of asbestos. The arguments below will address this point, however, clearly the question of chrysotile's potency is very important not only to regulatory agencies, and the many individuals exposed to it, but also to the asbestos mining and manufacturing interests.

Manufacturers would obviously benefit financially from an official report that said chrysotile is a less potent carcinogen than amosite or crocidolite and that

mesothelioma does not occur among workers who used their chrysotile products. Equally the asbestos mining and manufacturing interests would very much like to prevent their remaining markets (in third world countries) from adopting policies to phase out the use of chrysotile.

However, the question addressed here will ignore the financial and political views and will consider the scientific issue:

Is chrysotile asbestos less carcinogenic than the amphiboles?

The background to this controversy has been thoroughly explored in sections 2.5.1 and 2.5.2. The following arguments for and against chrysotile being less carcinogenic than the amphiboles are mainly derived from the material already given in section 2.5.2. The arguments in support of chrysotile being less potent are as follows:

The lower rates of lung cancer observed in heavily exposed chrysotile miners and millers, compared to those found in other industries with workers heavily exposed to either amphibole or a mixture of asbestos types. Suggesting pathogenic differences in fibre types, with chrysotile being the least pathogenic.

In asbestos cement manufacturer workers exposed to chrysotile alone have been observed to have no excess risk of lung cancer.[236,237] In friction product manufacture, an industry that has largely used only chrysotile asbestos, there is very little convincing evidence of excess lung cancer occurring.[161,162,165] The conclusion from one UK study was that under good environmental conditions products containing chrysotile can be manufactured with no detectable excess mortality.[165]

In animal studies chrysotile fibre clearance from the lung appears to be greater than that for amphibole fibres.[199] Human autopsy studies have shown that low levels of chrysotile dust are retained in the lungs (relative to apparent dose of exposure) when compared to amphibole levels.[167,238] Suggesting that chrysotile has greater pulmonary clearance, which leads to a basis for its possible lower pathogenicity.

The accumulated evidence favours chrysotile fibres being less potent as a cause of mesothelioma than the amphiboles.[122,153,154,239] Suggesting a generally lower level of carcinogenicity for chrysotile fibres.

The arguments that have been presented against chrysotile being less carcinogenic are as follows:

The high rate of lung cancer seen among textile workers exposed exclusively to chrysotile.[153,154] These studies have generated the steepest linear dose-response gradients. For mesothelioma the substantial number of cases observed in Quebec miners and millers, predominantly exposed to chrysotile, argues against chrysotile being generally less carcinogenic than the amphiboles.[139,150,204]

Animal studies have shown that chrysotile is as potent as crocidolite in inducing mesothelioma after intrapleural injection and pulmonary neoplasms after inhalation exposure.[240] Animal studies have also shown a higher level of carcinogenicity for chrysotile at any given dose compared to amosite and crocidolite.[190,199]

The analysis undertaken by Nicholson in 1986 clearly shows that certain chrysotile using industries (namely mining and milling) have significantly lower lung cancer risks than other asbestos industries.[145] Equally, from this work and that of McDonald and McDonald, and Doll and Peto, there is evidence to conclude that chrysotile alone has a limited mesothelioma producing potential.[144,146] The view of the Asbestos Institute was put forward by Dunnigan in 1988, who stated that amphiboles are mainly responsible (presumably for lung cancer and mesothelioma), whereas chrysotile alone has little or no mesothelioma producing potential.[241] These commentaries are very similar but Dunnigan's view appears to close the case. In fact, the contemporary literature suggests, but does not prove, that a contaminant of some chrysotile ores, tremolite, has caused the mesothelioma seen in asbestos miners and millers.[138,167] A truer conclusion would be that processed chrysotile has not been shown alone to cause mesothelioma; animal studies have been of little assistance in resolving this question. As mentioned earlier in this chapter, the evidence that chrysotile rarely causes mesothelioma is consequently consistent but not conclusive. A similar

conclusion could be drawn for lung cancer, with the qualification that high risks have often been observed in chrysotile textile workers. It has been suggested that the fibre size distributions found in this industry (the fibres are commonly shorter and sharper than the chrysotile fibres found in mining) strongly affect the fibres potential for producing lung cancer.[145,166] A further possibility concerns tremolite contamination. The American textile factories were mainly supplied with Quebec asbestos, therefore, the same problem of tremolite contamination holds for the textile workers as for the miners and millers (but reasonably with different fibre dimensions).[167] In the chrysotile textile plants, a flotation process was used to remove the tremolite from the chrysotile before weaving the cloth. However, United States lawyers have failed to find any evidence that the cloth contained any tremolite.[242] So where did the tremolite go? Could the flotation process have liberated the tremolite to be inhaled? Unfortunately, there appears to be no past environmental exposure data from the textile plants which analysed fibre type in this work area.[242]

In conclusion, from animal studies chrysotile fibres are seen to be extremely carcinogenic, more so than the amphiboles, but are also cleared very efficiency from the lungs. In human studies low levels of chrysotile dust are seen to be retained in the lungs and there is evidence that chrysotile fibres may have less carcinogenic potential than amphibole fibres. This produces a theory where the longer human life span allows chrysotile fibres to be removed from the lungs before carcinogenesis can fully develop. The paradox here is that chrysotile on simple biological grounds might be evaluated to be both more or less harmful than the amphiboles. The conservative and most sensible view in these circumstances would be to support Lemen's view and conclude that it is to soon to define a specific differential fibre factor, although the evidence is building up in favour of chrysotile being less carcinogenic.

2.5.7 Summary.

There is clear agreement medically, scientifically and epidemiologically that exposure to asbestos can cause asbestosis, lung cancer and mesothelioma. However, wide disagreement exists in the medical and scientific literature on the shape of the dose-response curve (particularly at low levels of exposure), on the biologically relevant measure of dose, and on the relationship between carcinogenic potential and the fibrogenic properties of asbestos fibres. The inadequacies of historic exposure estimates, the use of inappropriate rates in calculating standardized mortality ratios, the effect of statistical variation, and the differential effect of variable fibre types (and other contaminants) may all have played a role in obscuring both the shape of the dose-response curve, the effect of a threshold, and the potency of chrysotile asbestos. These factors are likely also to partially explain the distinct dose-response gradients of lung cancer risk seen in the assorted asbestos trades and industries.

A WHO consultation meeting was held in 1992 to address the current debate on the interpretation and relevance to humans of data derived from various animal models to assess fibre carcinogenicity.[243] The meeting concentrated on man-made fibres nonetheless the conclusions could be applied to all mineral fibres. From this meeting it was felt that to compare risks extrapolated from animal models, the full specifications of the fibrous materials used in the model needs to be allowed for. Comparisons on a simple fibre number basis were considered misleading because of the different size distributions of the fibres and the uncertainty about the fraction responsible for biological activity. It was also felt that comparisons of exposure-dose-response relationships should be made using a range of dose parameters until the most appropriate ones are defined. It is clear that these conclusions also hold for asbestos fibres and would help in answering the current asbestos controversies. It is equally clear that for asbestos a large amount of animal data has accumulated with results that are at odds with the human data from cohort studies. Possibly the answer is for further cohort investigations that include a full mineralogic analysis, as well as detailed

information on the full distribution of fibre sizes occurring to the exposed population. This research is needed both at very low levels of asbestos exposure and also potentially at very high levels of airborne fibre concentration to confirm either a linear relationship, a sigmoid relationship (which is biologically plausible and consistent with the available data), or a threshold effect. To date it is conceivable that the linear model for lung cancer risk is valid only over a middle range of asbestos exposures, which probably cannot be applied to the current ambient environmental levels.

To conclude this review, the specific diseases primarily associated with dockyard asbestos exposure have been shown to be asbestosis and mesothelioma. There is however only limited evidence that lung cancer is associated with this form of exposure. This evidence comes mostly from studies outside of the naval dockyards. For example, from Belfast and Barrow-in-Furness shipyards, from American insulation workers, and from studies of Coastal North America. As mentioned in section 1.4 the lack of a statistically significant excess of lung cancer cases in naval dockyard workers may well be a statistical artefact or it may suggest that the asbestos exposure patterns somehow did not allow lung cancer to develop, but could induce asbestosis and mesothelioma. The following chapters will consider this point.

For all asbestos-related diseases the essential feature in prevention is the strict use and control of all materials containing asbestos. This statement holds for both past dockyard occupational exposures and the public environmental exposures causing concern today. However, in the shipbuilding and repair industry the damage has already been done, with the past uncontrolled use and removal of vast quantities of asbestos containing material. Ramazzini in his *Treatise on the Diseases of the Tradesmen* urged his fellow physicians to care for the well-being of workers and to "see to it that, so far as possible, they should exercise their callings without harm".[244] This credo was written nearly 280 years ago. In this work Ramazzini also recognized the need for dust control and ventilation long before the invention of industrial exhaust machines. If society had adopted Ramazzini's credo at the start of the industrial revolution the shipbuilding asbestos problem might have been avoided.

Chapter 3: MATERIALS AND METHODS.

3.1 MATERIALS.

3.1.1 Introduction.

Medical surveys were carried out at each of the four Royal Naval Dockyards: Devonport, Chatham, Portsmouth and Rosyth over the period 1972-1973. These were morbidity surveys and commenced on the following dates: for Devonport 1st March 1972, for Chatham 4th September 1972, for Portsmouth 5th March 1973, and for Rosyth 1st August 1973. These surveys form the baseline data of this dissertation and their inception dates are taken as the enumeration dates of this work. In this study the workers enumerated at each dockyard are followed for exactly 17 years. Consequently, the follow-up ended on the following dates for each dockyard: for Devonport 28th February 1989, for Chatham 3rd September 1989, for Portsmouth 4th March 1990, and for Rosyth 31st July 1990.

The study population in these morbidity surveys included all male employees, and female industrial employees on the dockyard payroll on these dates who had been employed for at least six months. Each worker was asked to complete and return a health questionnaire and to attend for a chest radiograph (small 100mm chest x-rays were used). Non-responders were identified and reissued with questionnaires and invited to attend once again for a radiograph. A further recall procedure was used for subjects with radiographs showing possible asbestos related abnormalities. These workers were given a more detailed questionnaire and a full size chest radiograph was taken.

Definition of the study population at each dockyard occurred approximately one month before the issue of questionnaires, and approximately three months before the radiographic screening. It is possible that the delay between the definition of the population and the start of these cross-sectional studies may have affected the

TABLE 3.1: Response Rates*.

Dockyard	Survey Population	Either X-ray Responders	Either Questionnaire Responders	X-ray and Questionnaire	Absolute Non-Responders
Devonport:					
In-yard males	13185	11107 (84%)	11568 (88%)	10289 (78%)	798 (6%)
Outstation males	1079	782 (72%)	762 (71%)	647 (60%)	182 (17%)
Female workers	-	-	-	-	-
Total	14264	11889 (83%)	12330 (86%)	10936 (77%)	980 (7%)
Chatham:					
In-yard males	6694	5205 (78%)	4465 (67%)	4004 (60%)	1028 (15%)
Outstation males	-	-	-	-	-
Female workers	434	270 (62%)	200 (46%)	166 (38%)	130 (30%)
Total	7128	5475 (77%)	4665 (65%)	4170 (58%)	1158 (16%)
Portsmouth:					
In-yard males	10255	7877 (77%)	6904 (67%)	6122 (58%)	1597 (16%)
Outstation males	1256	466 (37%)	444 (35%)	246 (20%)	592 (47%)
Female workers	35	27 (77%)	4 (11%)	4 (11%)	8 (23%)
Total	11546	8370 (72%)	7352 (64%)	6372 (55%)	2197 (19%)
Rosyth:					
In-yard males	6580	4782 (73%)	3420 (52%)	2925 (44%)	1303 (20%)
Outstation males	496	209 (42%)	187 (38%)	124 (25%)	224 (45%)
Female workers	261	157 (60%)	105 (40%)	85 (32%)	84 (32%)
Total	7337	5148 (70%)	3712 (51%)	3134 (43%)	1611 (22%)
All Dockyards:					
In-yard males	36714	28971 (79%)	26357 (72%)	23340 (64%)	4726 (13%)
Outstation males	2831	1457 (51%)	1393 (49%)	1017 (36%)	998 (35%)
Female workers	730	454 (62%)	309 (42%)	255 (35%)	222 (30%)
Grand Total	40275	30882 (77%)	28059 (79%)	24612 (61%)	5946 (15%)

* This table is reproduced from: Harries PG, Rossiter CE, Coles RM (1976) "Royal Naval Dockyards Asbestosis Research Project, Report No. 1, December 1975" Institute of Naval Medicine, CRWP Report 1/76, Gosport.

response rates (see section 3.1.7). The rates are given in table 3.1. The non-response rate varied from 7% at Devonport to 22% at Rosyth.

3.1.2 Basic Data.

In this work the data obtained from the questionnaire responses and chest radiographs have been combined with payroll/employment information and death certificate information to form the basic dataset for each dockyard. The format of this data is given in table A1.1. Briefly, the data consists of the following: a unique identifier, formed using the individuals national insurance number and the first three letters of the surname, the date of birth, year of employment, occupational code, medical history, cause and date of death. Smoking habits were determined from the questionnaire responses of the workforce. Section 3.1.7 discusses the practical problems experienced in the creation of each dataset.

The initial characteristics of the morbidity data, i.e. prevalence rates of respiratory illness, etc., have been described in detail elsewhere by Harries et al.[1]

3.1.3 Questionnaires.

The questionnaires used in the morbidity surveys were based on the Medical Research Council (MRC) Questionnaire on Respiratory Symptoms (1966). Questions included details of personal medical history, smoking history and employment history. Those workers who were recalled for full size x-ray were given a more detailed doctor-administered questionnaire. Copies of the health questionnaires are given in Appendix 1.

The doctor administered ('controlled') questionnaire more closely followed the format of the MRC questionnaire, asking detailed questions on past illnesses, cough, phlegm and breathlessness, and included a section on physiological tests. When responses were available to both questionnaires, those from the 'controlled' questionnaire were used in preference to those from the self-administered

questionnaire. This was seen to give little if any resulting bias for the majority of the questionnaire derived variables, except possibly in the case of the question concerning breathlessness with had the lowest calculated sensitivity of test. In table 3.2 the questions concerning cough, breathlessness and smoking history have been used for illustrative purposes to show the level of agreement between the responses to the controlled and free questionnaire. The responses shown in this table are for those workers with both self-administered and controlled questionnaires (i.e. 4,762 workers across all 4 dockyards). When taking the doctor controlled result as the 'gold standard' the diagnostic sensitivities and specificities were seen to be high for symptoms of cough and smoking habit; exceptionally so for cough (a perfectly valid diagnostic test would have a sensitivity and specificity both equal to 100%). For smoking habit, ex-smokers and those with unknown habit where included in the smoking group. For breathlessness it could be speculated that the sensitivity was reduced simply because it is very difficult for the workers to accurately define their own breathlessness (even given reasonable guidelines, see question 14 from the self-administered questionnaire in appendix 1).

TABLE 3.2: Questionnaire responses.

	Controlled questionnaire			Sensitivity	Specificity
	Cough				
	Yes	No	Total		
Yes	937	75	1012	95.5%	98.0%
No	44	3706	3750		
Total	981	3781	4762		
Self-administered questionnaire	**Breathlessness**				
	Yes	No	Total		
Yes	327	346	673	68.5%	91.9%
No	150	3939	4089		
Total	477	4285	4762		
	Smoking				
	Yes	No	Total		
Yes	963	17	980	89.3%	99.5%
No	115	3667	3782		
Total	1078	3684	4762		

Table 3.3 shows the type of questionnaire and x-ray film used by dockyard. The slight differences seen in tables 3.1 and 3.3 are due to record duplication and some cases of prior enumeration at another dockyard. The difference in take-up rates of large film (400mm) x-ray and controlled questionnaire seen in table 3.3 are indicative of technical and equipment problems in the radiographic machinery and not abnormalities.[1] This was particularly the case at Rosyth with approximately 19% of the workforce recalled for controlled questionnaires, but 49% receiving large film x-rays. Overall, just under 9% of the dockyard population (from all four yards) appear to have received large film x-rays due to equipment failure of the 100mm (small film) mobile x-ray unit.

TABLE 3.3: Type of x-ray and questionnaire (male workers).

Dockyard	X-ray		Questionnaire	
	Small Film only	Large Film	Self-completed only	Controlled
Devonport	9554	2335 (19.6%)	10431	1899 (15.4%)
Chatham	4283	920 (17.7%)	3829	634 (14.2%)
Portsmouth	6135	2204 (26.4%)	5798	1548 (21.1%)
Rosyth	2543	2448 (49.0%)	2926	681 (18.9%)
All yards	22515	7907 (26.0%)	22984	4762 (17.2%)

From the medical history section of the questionnaires the following questions will be considered in this study. Do you usually cough during the day or night at work? Do you usually bring up any phlegm from your chest first thing in the morning in winter? Do you get short of breath when walking with people of your own age on level ground? These questions are associated with the known symptoms of asbestos related respiratory illness: cough and breathlessness. The following two linked questions will be considered for chest-illness. During the past three years have you had any chest illness which has kept you from your usual activities for as much as a week? Did you bring up more phlegm than usual in any of these illnesses? Table 3.4 shows the positive responses to these questions by dockyard.

TABLE 3.4: Medical History Questions.

Dockyard	Cough	Phlegm	Breathlessness	Chest-illness
Devonport	2523 (20.5%)	2836 (23.0%)	1102 (8.9%)	1119 (9.1%)
Chatham	784 (17.6%)	836 (18.7%)	305 (6.8%)	319 (7.1%)
Portsmouth	1620 (22.1%)	1638 (22.3%)	608 (8.3%)	585 (8.0%)
Rosyth	688 (19.1%)	782 (21.7%)	254 (7.0%)	223 (6.2%)
All yards	5615 (20.2%)	6092 (22.0%)	2269 (8.2%)	2246 (8.1%)

Smoking habits were also discerned from the questionnaire responses and the groups shown in table 3.5 were identified. Only 0.9% of the returned questionnaires were without smoking history. If a worker had given up smoking for less than a month before completion of the questionnaire he was considered a smoker. Smokers were further subdivided by amount of tobacco smoked per day into the following groups: < 15 gms/day, 15-24 gms/day, and ≥ 25 gms/days. The amount of tobacco was determined by calculating cigarette-gramme equivalents; 1 manufactured cigarette = 1 gm. The following scale was used to convert other smoking types.

1 oz tobacco in hand rolled cigarettes/week = 4 gms/day
1 oz tobacco in pipes/week = 4 gms/day
1 large cigar/day = 5 gms/day
1 small cigar/day = 2 gms/day

TABLE 3.5: Smoking Status.

Dockyard	Non-smokers	Ex-smokers	Smokers	Unknown
Devonport	2838 (23.0%)	2488 (20.2%)	6842 (55.5%)	162 (1.3%)
Chatham	1081 (24.2%)	879 (19.7%)	2444 (54.8%)	59 (1.3%)
Portsmouth	1700 (23.1%)	1635 (22.3%)	3970 (54.0%)	41 (0.6%)
Rosyth	956 (26.5%)	675 (18.7%)	1973 (54.7%)	3 (0.1%)
All yards	6575 (23.7%)	5677 (20.5%)	15229 (54.9%)	265 (0.9%)

3.1.4 Occupational History.

Employment histories were sought from the questionnaire responses, the workers being asked to list and code all jobs held since leaving school. A list of dockyard jobs was given with a list of non-dockyard jobs thought to be in someway associated with exposure to asbestos and dusty environments such as mines and quarries. Table 3.6 gives this list and the occupational code numbers. The workers chronologically listed their employment to the nearest year.

To enable comparison with the past work of Sheers and Templeton[2] the occupational codes, of the workers last held dockyard trade, were used to form the following four occupational groups:

Group 1.	All registered asbestos workers (RAWs).
Group 2.	Electrical fitters, burners, welders, riveters, caulkers, drillers, shipfitters, plumbers, coppersmiths.
Group 3.	Shipwrights, engine fitters.
Group 4.	All dockyard trades not in groups 1, 2 and 3.

TABLE 3.6: Occupational code numbers.

Dockyard employment		Other employment	
01	Labourer or skilled labour afloat	30	Royal Navy engine or boiler room branch
02	Lagger afloat	31	Royal Navy - other than code 30
03	Lagger ashore or in mattress shop	32	Civilian Shipyard
04	Asbestos storeman	40	lagger or insulation worker with asbestos
05	Asbestos sprayer or stripper	41	Any other job with asbestos
06	Sailmaker lagger	42	Coal miner - underground
07	Mason afloat	43	Coal miner - surface
08	Welder afloat	44	Any other mine work
09	Boilermaker afloat	45	Foundry work
10	Engine fitter afloat	46	Steelworks
11	Electrical fitter afloat	47	Quarrying
12	Painter afloat	48	Pottery
13	Coppersmith afloat	49	Cotton, Flax, Hemp Mill
14	Plumber afloat	50	Refractory brick works
15	Joiner afloat	51	Masons yard
16	Burner, Riveter, Caulker, Driller	52	Any other dusty job
17	Foundry worker	53	Any job exposed to irritant gas or chemical fumes
18	Shipfitter afloat	60	All other jobs not listed above
19	Shipwright afloat	61	Unemployed
20	All other dockyard jobs not listed above		
22	Any other dusty job		

Group 1, the registered asbestos workers, consists of laggers, sprayers, masons, sailmakers, painters and asbestos storemen. This group differs from the other three, in that, it forms the core of the register of dockyard asbestos workers. Registers were formed in the late 1960s of all workers in the trades listed under group 1. This was to enable those workers heavily exposed to asbestos to be monitored in the dockyards even if they subsequently stopped working with asbestos. All RAWs supplied questionnaires and x-rays.

Clearly, these four groups may give fairly ambiguous measures of asbestos exposure, they only allow for the last job held and therefore only that exposure. In practice workers exposed to asbestos and affected by asbestos disease can be found in all the groups. Promotion and job transfer would account for this. Table 3.7 gives a breakdown of the occupational groups by dockyard. In all, only 3.0% of the questionnaire responses omitted any mention of employment history.

TABLE 3.7: Occupational Group by Dockyard.

Dockyard	Occupational Group 1	2	3	4	Unknown
Devonport	591 (4.8%)	1392 (11.3%)	1315 (10.7%)	8781 (71.2%)	251 (2.0%)
Chatham	196 (4.4%)	461 (10.3%)	462 (10.4%)	3255 (72.9%)	89 (2.0%)
Portsmouth	304 (4.1%)	747 (10.2%)	707 (9.6%)	5292 (72.0%)	296 (4.0%)
Rosyth	102 (2.8%)	281 (7.8%)	264 (7.3%)	2751 (76.3%)	209 (5.8%)
All yards	1193 (4.3%)	1429 (10.4%)	2748 (9.9%)	20079 (72.4%)	845 (3.0%)

3.1.5 Asbestos Exposure Rating.

The crude classification of asbestos exposure by occupational groups can be improved upon. For instance, a grading of High, Medium and Low exposure may be made by job type and years in service. For this, occupational codes 02-06 and codes 40 and 41 could form the high group; codes 01, 07-16, 18, 19, 22, 30 and 32 could than be considered as the medium group; the residue would then form the

low group. This takes us from four to three groups! A better approach would be to code asbestos exposure for each dockyard trade and produce an exposure rating.

This rating would take the form:

$$\text{Exposure rating} = \text{Exposure code} \times \text{Years in that job.}$$

So a rating could be found for each worker by selecting the occupational codes given in the questionnaire and multiplying the years spent in those jobs by a number taken as the exposure code for that particular trade. Table 3.8 gives exposure codes used for all dockyard occupations. The codes were chosen by qualified dockyard industrial hygienists and medical officers.[3] These codes were in fact empirically chosen, without verification with fibre counts. However, the hygienists involved believed these to be the best relative estimates available for each dockyard trade.[4,5] These numbers can therefore only be considered to give informed relative guesses ('guestimates') of dust exposure; however, they go further towards a more accurate reflection of asbestos exposure than, for example, describing a worker as a lagger when in fact he has lagged for only one year and worked as a labourer ashore for thirty.

TABLE 3.8: Asbestos Exposure Codes.

Exposure Code	Occupation
1	Office work, messengers, outstation personnel.
2	All industrial work ashore, non-industrial supervisory staff with very occasional exposure afloat.
5	Foundry workers.
10	Labourer or skilled labourer afloat, joiner, coppersmith, riveter, plumber, caulker, burner, driller, shipfitter, engine fitter, electrical fitter.
12	Boilermaker, shipwright, welder.
15	Painter, mason.
20	Sailmaker lagger, asbestos storeman.
25	Lagger afloat and ashore, asbestos sprayer or stripper.

Table 3.9 gives the calculated exposure rating for each dockyard. From this it can be seen that 4.7% of the responders had an unknown rating. Most of the workforce had a rating of less than 100 (e.g. were employed less than 10 years as labourers, or less than 4 years as laggers). Only 610 workers had a rating over 400 (e.g. were employed as laggers for over 16 years).

TABLE 3.9: Exposure rating by Dockyard.

Dockyard	Exposure Rating					
	<100	100-	200-	300-	400+	Unknown
Devonport	7851 (63.7%)	1966 (15.9%)	1077 (8.7%)	559 (4.5%)	275 (2.2%)	602 (4.9%)
Chatham	2965 (66.4%)	649 (14.5%)	363 (8.1%)	211 (4.7%)	113 (2.5%)	162 (3.6%)
Portsmouth	4482 (61.0%)	1205 (16.4%)	696 (9.5%)	449 (6.1%)	183 (2.5%)	331 (4.5%)
Rosyth	2687 (74.5%)	418 (11.6%)	169 (4.7%)	85 (2.4%)	39 (1.1%)	209 (5.8%)
All yards	17985 (64.8%)	4238 (15.3%)	2305 (8.3%)	1304 (4.7%)	610 (2.2%)	1304 (4.7%)

In addition to the calculated exposure rating, estimates of individual periods of asbestos exposure are available. These estimates were obtained from additional questions present only in the controlled questionnaire, and are illustrated in table 3.10. From this table it can be seen that 3,792 (9.6%) of the workforce assessed their period of asbestos exposure, with only 778 (2.0%) reporting more than 30 years of exposure. The form of exposure, whether to asbestos materials only or to asbestos dust and debris is unknown. However, 35,746 (90.4%) of the workforce did not supply these estimates. The percentages shown in table 3.10 are based on the 4,762 workers with controlled questionnaire information.

TABLE 3.10: Period of asbestos exposure by dockyard.

Dockyard	Asbestos exposure period (yrs) < 10	10-	20-	30+
Devonport	514 (27.1%)	445 (23.4%)	369 (19.4%)	293 (15.4%)
Chatham	173 (27.3%)	113 (17.8%)	112 (17.7%)	114 (18.0%)
Portsmouth	350 (22.6%)	291 (18.8%)	273 (17.6%)	312 (20.1%)
Rosyth	166 (24.4%)	127 (18.6%)	81 (11.9%)	59 (8.7%)
All yards	1203 (25.3%)	976 (20.5%)	835 (17.5%)	778 (16.3%)

3.1.6 Chest Radiographs.

All workers were invited to attend for chest x-rays. Small 100mm films were used, each film being read by two members of a panel of four readers. All subjects with films showing possible asbestos related abnormalities or technical faults were recalled for further investigation with large (400mm) films. Table 3.3 shows the x-ray type used.

The readers scored each film according to the classification given in table 3.11. Each reading was then allocated to one of the following x-ray groups.

X-ray score		X-ray group
00 or 19	1.	Normal and technical faults.
01 or 03	2.	Pleural thickening.
02 or 04	3.	Pleural calcification.
05 or 06	4.	Suspected or definite pulmonary fibrosis.
07 or 08	5.	Active or clinically significant healed pulmonary tuberculosis. Not primary complex.
09 to 18	6.	All other abnormalities.

The prevalence of x-ray abnormalities are calculated by counting ½ for each reading and allocating that reading to the x-ray group. Therefore, a film with two readings of 00 would score 1 in group 1, a film with readings 00 and 01 would

score ½ in groups 1 and 2 respectively. Table 3.12 gives these prevalences by dockyard. In this table the more sensitive large film reading has been used, when recorded, in place of the small film score, to obtain the most accurate measure of disease prevalence.

TABLE 3.11: Classification for Chest Radiographs.

Code Numbers	Disease
00	Normal
	Suspected Asbestos Abnormalities
01	Limited pleural thickening without calcification
02	Limited pleural thickening with calcification
03	Extensive pleural thickening without calcification
04	Extensive pleural thickening with calcification
05	Pulmonary fibrosis suspected
06	Pulmonary fibrosis definite
	Other diseases
07	Tuberculosis active
08	Tuberculosis inactive
09	Malignant tumours
10	Other tumours including thyroid enlargement
11	Lymphadenopathies
12	Cardiovascular disease
13	Pulmonary infections
14	Pleural effusions
15	Emphysema
16	Simple unilateral costophrenic occlusion
17	Bilateral occlusion of costophrenic angles
18	Other abnormalities
19	Technical faults, poor position etc.

TABLE 3.12: X-ray Abnormalities by Dockyard.

Dockyard	X-ray group 1	2	3	4	5	6
Devonport	10171 (85.6%)	467 (3.9%)	147 (1.2%)	119 (1.0%)	364 (3.1%)	621 (5.2%)
Chatham	4468 (85.9%)	177 (3.4%)	53 (1.0%)	41 (0.8%)	152 (2.9%)	312 (6.0%)
Portsmouth	7019 (84.2%)	414 (5.0%)	120 (1.4%)	60 (0.7%)	198 (2.4%)	528 (6.3%)
Rosyth	4263 (85.4%)	221 (4.4%)	43 (0.9%)	36 (0.7%)	124 (2.5%)	304 (6.1%)
All yards	25921 (85.2%)	1279 (4.2%)	363 (1.2%)	256 (0.8%)	838 (2.7%)	1765 (5.8%)

It should be clearly noted that the x-ray reading scale used predated that of the full ILO U/C 1971 classification which is considered one of todays standard methods. The classification used here (and used previously in dockyard surveillance studies[2,6]) was employed over the period 1972-1973, the full ILO classification was however only published in 1972.[7] The initial studies effectively started too early to implement this new ILO classification; the readings being undertaken quickly after the x-ray examination using the then well established naval reading scale.[4] This researcher is unaware why the UICC/Cincinnati 1968 classification, or the short ILO U/C 1968 classification was not used. It could be speculated that this was for the same reason that the full ILO classification was not implemented; possibly due to established techniques and/or naval policy.

TABLE 3.13: Comparison of reading methods.

		Large film reading positive			Overall agreement	Agreement among films positive at standard level
		ILO standard level of abnormality				
		Yes	No	Total		
Small film positive ILO standard level of abnormality	Yes	71	12	83	90.2%	56.8%
	No	54	537	591		
Small film positive screening reading	Yes	80	43	123	86.9%	64.0%
	No	45	506	551		
Large film positive screening reading	Yes	87	57	144	85.9%	69.6%
	No	38	492	530		
		Screening level of abnormality				
		Yes	No	Total		
Small film positive screening reading	Yes	83	40	123	84.4%	57.6%
	No	61	490	551		

Subsequently attempts were made to validate what was then considered to be a screening method (the method described in this work) with the ILO classification.[8,9] This validation was undertaken by Sheers et al in 1978 on a group of 674 men randomly drawn from all four dockyards for which both x-ray film types (100mm and 400mm) were available. This study was designed to attempt to decide which type of film (small or large), which method of classification, and how many film readers should be employed to achieve a satisfactory level of surveillance for asbestos workers. The study consisted of a controlled trial by five readers using the full ILO classification, which was compared to the earlier screening classification (undertaken by two readers as earlier documented). Table 3.13 shows the study sensitivities obtained.

In order to compare the overall results of the two reading methods (i.e. to calculate the sensitivities seen in table 3.13) a common reference standard was defined. The standard was set as the ILO classification with large films. The standard level was defined as: width b or more for pleural thickening, grade 2 or more for pleural calcification and category 1/1 or more for small opacities. This standard was chosen to deliberately exclude the lowest categories of small opacities and the lowest grading of pleural thickening and calcification in order to avoid the problems caused by borderline abnormalities. Table 3.13 clearly demonstrates that there is a deficiency in the detection of abnormalities by small film, when large and small films are read to the same standard (giving sensitivities of only 56.8% and 57.6%). This is despite the fact that the screening reading includes all grades of abnormality (codes 1 to 6 in table 3.11) found by either reader. When considering only the ILO classification for small films the sensitivity was seen to increase when lower categories and grades of abnormality were scored (and when the number of readers was reduced). The sensitivities obtained from this approach were: 72.8%, 80.0% and 92.8% (table 4, Sheers et al[9]). These were for any level of abnormality found by 3 readers, by 2 readers, and by any reader, respectively. This approach resembles the situation in the screening classification (i.e. 2 readers with any level of abnormality), when all grades of abnormality recorded by any reader on the small films would lead to recall of the subject for re-examination on

large film. Table 3.13 also highlights a problem with the screening readings. When these are compared to the standard reading a deficiency is seen in the results of the screening reading regardless of film size (sensitivities of 64.0% and 69.6%).

The conclusion of this comparison study was that the screening classification showed a deficiency, independent of film size, of at least 30% in the detection of asbestos-related radiographic abnormalities. It was further concluded that for adequate diagnostic sensitivity the ILO U/C classification, with a minimum of 3 readers, appears to be essential (having a minimum sensitivity of approximately 73%). This creates limitations in the use of the dockyard screening classification, even though this method had a sensitivity of 64% for small films and approximately 70% for large films. A possibility would be to reread all the radiographs to the full ILO classification, a massive and costly operation. However, these dockyard radiographs are no longer available.[5] Accordingly the classification used in this work, with its estimated sensitivity of approximately 70%, is that used in the original dockyard surveillance studies. A final recommendation of the comparison study, on the grounds of cost-effectiveness, was the use of small films in large-scales studies were the abnormality rate is expected to be low, with a recall procedure used for the more sensitive (and expensive) large films.

The analysis of Sheers et al emphasised that the small film, regardless of the classification method used, had a much poorer diagnostic sensitivity then the large (i.e. a sensitivity of only 57.6% when using the dockyard reading scale). For this reason the large film reading will be used in the following analysis when both readings are present.

Table 3.12 gives an overall prevalence of dockyard asbestos-related abnormalities of 6.2% (groups 2, 3 and 4 combined). This result is somewhat higher than the prevalence found by Harries in 1972 and 1975 (illustrated in tables 2.5 and 2.6).[1,6] Here the prevalence of asbestos-related abnormalities was quoted as being 3.4% (in a 10% sample) and 4.6% (in the entire population). By comparing the total number of workers examined (24,575 in 1975 against 30,422 in this

work), it is clear that some 5,847 workers were excluded in 1975. By inspection of table 3.1, after allowing for enumeration errors (e.g. female workers coded as male workers), it is probable that the 1975 analysis included only those workers with both paired x-ray and questionnaire results. When the calculation was repeated allowing for this the prevalence of 6.2% was reduced slightly to 5.8%. However, the 1975 Naval report also quoted prevalence rates of radiographic abnormalities for workers with either large or small films (excluding outstation workers). Table 3.14 shows these rates. By estimation from this table there appears to be a total prevalence of x-ray abnormalities of 6.0% (again combining groups 2, 3 and 4). A result in very close agreement with the value of 6.2% presented here (which includes outstation workers). It should be noted that if group 3 (i.e. pleural calcification) is omitted in the calculation of prevalence the results here closely match those of the 1975 study (i.e. 5.0% verses 4.6%). Section 3.1.7 will further consider issues of data integrity and possible associated study restrictions.

TABLE 3.14: Prevalence rates of x-ray abnormalities by Dockyard.

Dockyard	Number of workers x-rayed	X-ray group 1	2	3	4	5	6
Devonport	11107	86.2%	3.6%	1.3%	1.1%	3.0%	4.8%
Chatham	5205	84.2%	3.2%	1.0%	0.6%	2.4%	8.5%
Portsmouth	7877	85.8%	4.9%	1.4%	0.6%	1.8%	5.5%
Rosyth	4782	85.4%	3.9%	1.0%	0.7%	2.6%	6.5%

3.1.7 Study/Data Restrictions.

The Ministry of Defence (Navy) had available in the early 1970s, information only on current occupation for its civilian employees. Records of earlier jobs within or outside the dockyards, and their start dates, were not available. In fact, the occupational codes used in the definition of the morbidity surveys were obtained from the dockyard payroll grade codes. This crude occupational code has many

drawbacks, not the least of these being the absence of any measure of intensity of asbestos exposure (i.e. length and type of exposure). Fortunately, the health questionnaires were augmented with questions on workers occupations since starting employment. The occupational codes used in this work are, therefore, based on the questionnaire data.

The use of questionnaire responses to assess occupation, and also respiratory illness and smoking habits, confines us to the questionnaire responders. However, radiographic abnormalities, as seen on either type of film, form an important part of this work, so responders to x-ray examination must also be considered. Both considerations restrict the following analysis to those workers who were not 'absolute non-responders' in the initial cross-sectional studies, that is, approximately 85% of the defined population. It should be noted that the use of 100mm film rather than full sized films, and reading to the simplified x-ray classification, rather than to the ILO U/C classification may lead to a possible underestimation of the prevalence rates of asbestos abnormalities. This would be the case even though men were recalled for large films when there was any suspicion of asbestos related abnormally on the small film. This would clearly weaken the value of the x-ray groups illustrated in table 3.12, the results of which have to be used with caution.

From table 3.1 it can be seen that male employees fall into two categories, 'in yard' and 'outstation' workers, 2831 (7.0%) males being outstation workers. Outstation workers were dockyard employees working, at that moment, in dockyard outstations (substations outside the main yard); in yard workers were those working at the main dockyard. The workforce would have been very mobile with much interchange and exchange between these categories. Consequently no differentiation is made in this work between in yard and outstation workers.

It should be noted that in all dockyards, the outstation workers had the highest non-responder rates. This is likely to be due to the survey entry policies, cross-sectional studies being limited to one time point, and the outstation workers being

more geographically difficult to pursue after study definition. The time delay of up to two months between issue of questionnaires and the x-ray screening must have exacerbated this.

The 730 female employees, who formed less than 2% of the original surveys, were not followed-up in this study. Overall females had a high non-response rate 30% (from table 3.1). Therefore, only 508 'responded' females were omitted due to this exclusion. This was felt to produce little resulting bias and eliminated the need for female expected mortality rates to be calculated.

As in most mortality studies undertaken in the United Kingdom, details of all workers were submitted to the Office of Population Censures and Surveys (OPCS) to be flagged so that subsequent mortality could be recorded and analysed. The follow-up rate for Rosyth was particularly poor as the records submitted to the OPCS had to be passed to the equivalent office in Scotland if the required Health Service Number was believed to be of Scottish origin. No attempt was made to resubmit details of those not flagged at the first search. Table 3.15 shows the trace rates for the four dockyards over the 17 year period.

TABLE 3.15: Number of male workers in the Dockyards.

Dockyard	Total male workers	OPCS flagged	No. of deaths/ emigrations	No. of valid responders*	OPCS flagged	No. of deaths/ emigrations
Devonport	14264	97.5%	2744 (19.2%)	13283 (93.1%)	98.3%	2441 (18.4%)
Chatham	6691	96.8%	1484 (22.2%)	5663 (84.6%)	97.5%	1147 (20.3%)
Portsmouth	11507	93.7%	2358 (20.5%)	9319 (81.0%)	95.7%	1794 (19.2%)
Rosyth	7076	69.8%	926 (13.1%)	5549 (78.4%)	77.0%	733 (13.2%)
All yards	39538	91.3%	7512 (19.0%)	33814 (85.5%)	94.0%	6115 (18.1%)

* No. of male workers with either x-ray or questionnaire information.

Because of the low follow-up rate at Rosyth (69.8%), in comparison with the other dockyards, and because of the overall lower response rates seen in table 3.1, Rosyth has been removed from all subsequent analyses in this dissertation. This has the advantage of eliminating the need for Scottish mortality rates in the following analysis and the use of the 'Scottish OPCS'. The number of Dockyards actively analysed was, therefore, restricted to the three British yards (Devonport, Chatham, and Portsmouth) with 28,265 male workers followed-up for a 17 year period. The overall proportion traced by OPCS in these yards was 97.3%.

It can be seen that the main data and study restriction in this investigation is its use and reliance on the morbidity surveys study base. These cross-sectional studies were not planned as the first phase of a longitudinal study and this has resulted in restrictions on the use of the cross-sectional dockyard population of 40,275 male and female workers. These restrictions, the exclusion of female workers and the initial surveys non-responders and finally the exclusion of Rosyth Dockyard reduce the study population to 28,265 male civilian workers. Of these 5,031 (18.0%) have died and 351 (1.2%) emigrated over the study period (table 3.16).

TABLE 3.16: Number of Deaths/Emigrations in the 3 British Dockyards.

Dockyard	Responders			Non-responders		
	No. of workers	Deaths	Emigrations	No. of workers	Deaths	Emigrations
Devonport	13283	2292 (17.3%)	149 (1.1%)	981	295 (30.1%)	8 (0.8%)
Chatham	5663	1046 (18.5%)	101 (1.8%)	1028	321 (31.2%)	16 (1.6%)
Portsmouth	9319	1693 (18.2%)	101 (1.1%)	2188	540 (24.7%)	24 (1.1%)
All yards	28265	5031 (18.0%)	351 (1.2%)	4197	1156 (27.5%)	48 (1.1%)

From table 3.16 we can see that the emigration rates for responders and non-responders are almost identical. However, the same is not true for the rates of death, the non-responders appear to have a higher death rate. This may be a measure of self-selection bias, the unhealthy workers deselecting themselves from

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the initial studies. Selection bias is considered in section 3.2.5 and the non-responder death rates studied in chapter 4.

When considering the final dockyard datasets generated for this work, a limitation is apparent. Lung function information is missing. Physiological tests were undertaken and recorded in the initial surveys, however, these were not available to this researcher. These tests were only performed for those workers recalled for the large x-ray and 'controlled' questionnaire, and should have been assembled into the separate dockyard data files at that time. These files were received at the London School of Hygiene and Tropical Medicine (LSHTM), by myself in 1986, from the MRC Pneumoconiosis Unit, Penarth. They were initially compiled at the Medical Research Unit of Devonport Dockyard. The files consisted of computerised magnetic tapes, computer listings and a haphazard number of both free and controlled questionnaires. This with the death certificates, which were reported directly to LSHTM from 1986, comprised all the available 'raw' dockyard data. Unfortunately the physiological data was missing. This omission is likely to have happened during the exchange of data across its many sites. The final LSHTM datasets created by myself therefore omitted all physiological data.

Unfortunately, a further problem existed with the raw data. The magnetic tapes holding the radiographical information were all found to be corrupted and unreadable. To remedy this entailed many visits to Penarth and Devon, where further computer listings (and coding sheets) were eventually obtained and the x-ray scores were then reentered onto computer file. During the data entry of the x-ray and death certificate information, range and consistency checking was performed along with double entry of all data. By this technique data entry errors were minimised and an overall data entry error rate of less than 0.5% was achieved. The integrity of the remaining magnetic tape information was confirmed by undertaking a re-inputting of a 10% sample of the data documented on original Devonport Research Unit computer listing. Table 3.17 illustrates the final cohort numbers obtained in this re-generation of the data in relation to the first reported morbidity study. Apart from the point raised in section 3.1.6 concerning the

prevalence of x-ray abnormalities the characteristics of the final LSHTM datasets match those earlier itemised by Harries.[1] The very slight difference illustrated for male workers in table 3.17 is simply due to past record duplication; the larger difference for females is probably due to initial x-ray coding errors.

TABLE 3.17: Overall cohort numbers.

All Dockyards	Population	Either X-ray Responders	Either Questionnaire Responders	X-ray and Questionnaire	Absolute Non-Responders
1975 Naval report [1]:					
Male workers	39545	30428 (77%)	27750 (70%)	24357 (62%)	5724 (14%)
Female workers	730	454 (62%)	309 (42%)	255 (35%)	222 (30%)
This study:					
Male workers	39538	30422 (77%)	27746 (70%)	24354 (62%)	5724 (14%)
Female workers	711	319 (45%)	287 (40%)	203 (28%)	308 (43%)

3.2 METHODS.

3.2.1 Introduction.

The main objective of this study is to consider the total mortality (generally, mortality from the causes listed in table A1.2) and mortality from the specific causes shown in table 3.18, with regard to dockyard occupation, length and intensity of asbestos exposure, however measured, for the workers described in section 3.1, followed-up for 17 years. The specific causes of death detailed in table 3.18 have been shown 'a priori', in the literature review, to be related to asbestos exposure. As was stated in chapter one, lung cancer and mesothelioma will be considered throughout with a view to addressing the research question of this work. Asbestosis has not been excluded from this question, however, since only 10 deaths were observed in the 17 year follow-up period for this disease definitive results are unlikely.

The objectives will be considered in the following ways:

1. Use of standardised mortality ratios (SMRs) as traditionally used in cohort studies.

2. Use of log-linear models, to explore the ways in which confounders relate to one another and to the exposure.

3. Use of logistic regression on nested case-referent data, evaluating smoking habits, using conditional maximum likelihood estimation.

4. Use of mathematical modelling to estimate the relationship between mesothelioma death rate and time since first exposure.

Where possible, these methods and their results are compared and contrasted. Time trends are isolated and analysed to give a view of the mortality patterns with time. The effect of confounders (i.e. age, length of employment, etc.) on mortality will be measured and, if possible, removed.

A major problem in epidemiology is the existence of competing risks. These have to be allowed for when analysing mortality from a specific cause of death. Consideration will be given to this. As a consequence the results from analyses of cause specific mortality must be interpreted very cautiously. Selection bias is also a problem in epidemiology, particularly in occupational epidemiology. Consideration will also be given to this and its most common form, the healthy worker effect.

TABLE 3.18: Specific Causes of Death.

Causes of Death	Deaths (all 3 yards)		ICD (9th revision)
All Causes	5031		0 - 999
All Neoplasms	1729	(34.4%)	140 - 239
Cancer of Stomach	160	(3.2%)	151
Cancer of Peritoneum (Mesothelioma)	13	(0.3%)	158
Cancer of Lung	575	(11.4%)	162
Cancer of Pleura (Mesothelioma)	118	(2.3%)	163
Diseases of the Circulatory System	2259	(44.9%)	10 - 18
Diseases of Pulmonary Circulation	36	(0.7%)	415 - 417
Diseases of the Respiratory System	459	(9.1%)	460 - 519
Bronchitis, Emphysema and Asthma	119	(2.4%)	490 - 493
Asbestosis	10	(0.2%)	501
Pulmonary Fibrosis	4	(0.1%)	515

3.2.2 Standardised Mortality Ratios (SMRs).

The most elementary technique used to analyse cohort survival data is to calculate fixed-period survival rates for various subgroups of the cohort and use these rates to determine which of the subgroups have a better expectation of life.[10] However, this approach has a certain weakness - it ignores mortality patterns during the period of follow-up. This method may be further criticised for making it cumbersome to compare two populations because of the multiplicity of the subgroups needed.[11]

In all cohort studies subgrouping has to be undertaken. This grouping will be by exposure level and all other factors expected to influence mortality. The most

important of these factors is age. This important determinant of mortality obviously changes with time and clearly in a cohort subjects move from one age group to another every year and generally die in an age group different to the one they started in. To show the true effect of age in these studies the age distribution of the deaths has to be divided by the corresponding age distribution of the person-years at risk. The technique of using person-years at risk, and the construction of age and cause specific mortality rate tables, leading to the use of standardised death rates, avoids, to some extent, the problem of multiple subgroup comparisons and overcomes the weaknesses of using simple fixed-period survival and fatality rates.

Thus in a cohort, the age distribution changes each year and this has to be reflected when calculating age-specific death rates. This is achieved by making each subject contribute a value to the person years at risk for each age group and year moved through in the study. The contribution will be one, a fraction, or zero, depending on whether the subject survives, dies during, or dies before that age and year. The subject contributes zero person-years to the ages he lived through before the start of the study. This process of calculating person-years at risk is often illustrated by a Lexis diagram.[12,13] In these diagrams a subjects life in a study is represented by a 45° line passing through the two time axes, age group and calendar time period.

Considering this 45° line, suppose we have N_o subjects in a cohort which is followed for a maximum period of T years. Let t_{oi} be the time in the study for a subject who dies or is lost to follow-up before the end of the study. So for each subject we have the following person-years at risk (y_{oi}):

$$y_{oi} = \begin{cases} t_{oi}, & \textit{if the individual dies during the study} \\ T, & \textit{otherwise.} \end{cases}$$

The person-years at risk for the whole cohort is, therefore, given by:

$$Y_0 = \sum_{i=1}^{N_0} y_{0i}.$$

Stratifying into K age groups and J periods, and considering the number of person-years spent in each age-group/period cell, say y_{ijk} for subject i, gives the following total person-years at risk for each cell:

$$y_{jk} = \sum_i y_{ijk}, \quad j=1,2,\ldots,J; \quad k=1,2,\ldots,K.$$

If the total number of deaths occurring in the cohort is D, an estimate of the crude death rate in the cohort is given by:

$$\hat{\lambda}_c = \frac{D}{Y_0}.$$

If a distribution of the deaths by age at death and by calendar year of death is formed, with the same grouping as a table of age/calendar person-years at risk formed in a Lexis diagram, we can compute the age-period specific death rates as:

$$\hat{\lambda}_{cjk} = \frac{d_{jk}}{y_{jk}}, \quad j=1,2,\ldots,J; \quad k=1,2,\ldots,K,$$

where d_{jk} is the number of deaths occurring in age group k and period j. Age-cause specific death rates can also be obtained using this procedure, limiting the calculation to the cause of death of interest. This is equivalent to treating the deaths from other causes as censored observations. An example of the mechanics of the person-years at risk calculation, applied to a cohort of asbestos workers, is given by Doll.[14]

Commonly in cohort studies it is necessary to compare mortality patterns across subgroups (i.e. calendar year period, cause of death, etc.). The problem of multiple comparisons is removed by calculating a single measure of mortality for each subgroup. To avoid comparison of two subgroups with different age

distributions, this single statistic has to be standardised. Liddell suggested numerous methods for this standardisation.[15] The basis of all these methods is the comparison of the mortality of a defined group of subjects with the mortality that would have been expected if the group had experienced death rates similar to those of the community of which the group is a part. The expected mortality is usually calculated using published national or regional death rates (see section 3.2.7 for a discussion on the rates used in this analysis).

The most commonly used mode of standardisation is the 'indirect' method. In this method age specific death rates of the 'standard' population (national or regional, etc.) are multiplied by the number at risk in the appropriate age groups in the study cohort to give the cohorts expected deaths. The total number of these expected deaths, over all ages, divided into the number of observed deaths produces the Standardised Mortality Ratio (SMR). This is the method used in this dissertation. This ratio should be equal to 1 (or 100%) if the mortality in the study cohort equals that of the standard population.

To compute the death rate in the chosen standard population for a particular age group and year entails dividing the number of deaths in that age group during the year by the midyear population of the age group. For example:

$$\lambda_{ojk} = \frac{d_{ojk}}{P_{ojk}}, \; j=1,2,\ldots,J; \; k=1,2,\ldots,K.$$

For periods that are greater than one calendar year an average age-specific annual death rate is obtained from:

$$\hat{\lambda}_{ojk} = \sum_{j=1}^{m} d_{ojk} \; / \; \sum_{j=1}^{m} P_{ojk}.$$

To obtain the expected number of deaths in the j–k^{th} cell, i.e. for age group k and period j, that cells population death rate is multiplied by the cohorts person-years at risk contribution.

$$E_{jk} = \lambda_{ojk} \times y_{jk}$$

and from this we obtain:

$$E_j = \sum_k E_{jk}$$

which is the expected number of deaths over all ages in period j. A more formal description of the properties of this person-years method has been given by Berry.[16]

If D_j is the observed number of deaths in the cohort during the same period j, the SMR for the cohort for that period is then given by:

$$SMR_j = 100 \times \frac{D_j}{E_j}.$$

SMRs for cause-specific mortality are calculated by the above method by using cause specific death rates in place of an all-cause rate.

Mortality in the study is then compared with mortality in the standard population by testing departures of the SMR from 1 (or 100%). This is commonly undertaken by constructing confidence intervals around the SMR. There are various methods and assumptions used in calculating these confidence intervals. One suggested by Gilbert is to assume that D is a Poisson variable with expectation E.[17] The statistic

$$(D-E)^2/E,$$

can then be treated as a X^2 variable with 1 degree of freedom. If E is small, this Normal approximation to the Poisson loses accuracy and Poisson tables should be used. An alternative is to use the following variance estimate,

$$Var(SMR) = \frac{D}{E^2}$$

to create a confidence interval for the SMR. The generalised $(1-\alpha)100\%$ confidence limits for this would be:

$$[(\frac{Z^{\frac{\alpha}{2}}}{2} - \sqrt{D})^2 / E, \quad (\frac{Z^{\frac{\alpha}{2}}}{2} + \sqrt{D})^2 / E] .$$

This can be simplified as:

$$SMR \pm (Z^{\frac{\alpha}{2}} \times \frac{SMR}{\sqrt{D}}) ,$$

for a 95% confidence interval this becomes:

$$(D \pm 1.96 \times \sqrt{D}) / E.$$

Again this approximation to the Poisson distribution can lose accuracy for small numbers. In this study the exact Poisson distribution was used in the formation of the SMR confidence limits when the observed deaths were less than 30 ($D < 30$).

The SMR and its confidence interval produced by the above technique compare the cohort mortality with the mortality pattern found in the standard population. In cohort studies this technique is sometimes used in the comparison of subgroups of the same cohort. Here the comparison of each group to the standard population would become a comparison between subgroups. This procedure has been criticised for not allowing for the separate age structures of each subcohort or strata.[18]

A more formal approach for comparing subcohorts is given by using an extension of Armitage and Berry's test of trend in proportions.[19] The test needed in the comparison of subgroup SMR's is a test of homogeneity of these ratios (leading to

a test of trend). If there are K subgroups (strata) to be compared, the test would then take the following form:

$$H_0: b_1 = ... = b_K$$

where b_k is the coefficient of the k^{th} binary indicator variable representing membership to subgroup k. Let,

$$\hat{E}_k = E_k \times \frac{D}{E} \quad \text{where} \quad \sum_k \hat{E}_k = D.$$

The test statistic, for the test of homogeneity, with $K-1$ degrees of freedom becomes:

$$X^2_{K-1} = \sum_k \frac{(D_k - \hat{E}_k)^2}{\hat{E}_k}.$$

If the groups correspond to levels of a single quantitative covariable with values X_k, a test for trend in the SMR is given by:

$$X^2_1 = [\sum_k X_k (D_k - \hat{E}_k)]^2 / [\sum_k X_k^2 \hat{E}_k - (\sum_k X_k \hat{E}_k)^2 / D].$$

An example of the application of tests of homogeneity and trend applied to subgroups of a cohort of smelter workers exposed to arsenic has been given by Breslow and Day.[20]

A major objective of this study is to compare subcohort mortality patterns with each other. This will produce many multiple comparisons. How can we overcome this and the potential problem of varying age distributions in the subgroups? The problem is exacerbated if further subclassification on explanatory variables is attempted. One solution is to use a more parametric approach, that is, regression modelling to analysis mortality, in effect to smooth the data across the subgroups. Subsequently, the methods we now consider will be regression type procedures.

3.2.3 Poisson Regression.

The most important use of an SMR is in the comparison of the mortality of a study cohort with that of a standard population from which the cohort was drawn. If a cohort can be characterised by other covariates, and an SMR is available for each of the subgroups resulting from the cross-classification of these covariates, it may be of interest to test the following.

1. That the SMRs are all equal.
2. That the SMRs are all equal to 1 (or 100%).
3. That the SMRs show a trend with increasing levels of an ordered categorical variable (i.e. exposure level).

Since the subgroups of the cohort are likely to have different age distributions, it may be necessary to use age and calendar year or follow-up period as additional classifying variables so that in the comparison of the different subgroups, the residual age and period effects are excluded.

To this end Poisson regression is considered in this section. In this form of regression a mathematical model is constructed in which the logarithm of the incidence rate is modelled as a linear combination of a set of risk factors. Poisson regression is used here since it can be formulated as an extension of the SMR method, and because the rate ratio estimates produced are unaffected by small numbers in particular strata. The number of deaths in each cross-classified subgroup is assumed to have an independent Poisson distribution.

Several authors have suggested a method for testing for homogeneity and trend in SMRs by way of regression parameters.[17,21,22] They have suggested the following model:

$$\lambda_j(i) = \exp(Z'_j(i)\,b)\,\lambda^*_j(i)$$

where $\lambda_j(i)$ is the death rate for the j^{th} individual in the follow-up period i, and $Z'_j(i)$ is that individuals covariate vector. From this,

$$\lambda_j^* = \lambda_j^*[t_{1j}(i),\ t_{2j}(i)]$$

is the rate determined by the individuals age $t_{1j}(i)$ and calendar period $t_{2j}(i)$ at that time. The model is similar to that proposed by Cox in 1972.[23] However, Cox's hazard is replaced here by a known constant, i.e. the threshold rate. Indeed theoretical analysis has shown an inherent link between Poisson regression and the proportional hazards model.[24]

With classification into K subgroups such that $Z'_j(i) = Z'_k$ the log-likelihood can be written as:

$$L(b) = \sum_k [D_k Z_k' b - \exp(Z_k' b) E_k],$$

where D_k is the total number of deaths in subgroup k and E_k is the expected number depending on the person-years at risk in subgroup k. This log-likelihood is similar to that when D_k are independent Poisson variables with means,

$$\exp(Z_k' b) E_k,$$

where E_k are constants. This implies that standard routines for Poisson regression can be used once a distribution of observed and expected deaths is obtained. For example, in the statistical package GLIM you have only to specify that D_k have Poisson errors, invoke the logarithmic link function and introduce $logE_k$ in the model as OFFSETS.

The present data are cross-classified, so the covariate vector associated with each cell is in general a set of indicator variables showing membership/non-membership of the various levels of the factors of interest and their interactions. Because of this the linear predictor, $Z'_k b$, can be written using the ANOVA notation outlined by Bishop.[25] The fitted model has the form:

$$\log E(D_{ijk}) = \log E_{ijk} + \alpha + \alpha_1(i) + \alpha_2(j) + \alpha_3(k) + \alpha_4(ij) + \alpha_5(ik) + \alpha_6(jk) + \alpha_7(ijk)$$

where, for example, there are I age-groups, J follow-up periods, and K categories of asbestos exposure. The use again of GLIM ensures a unique solution by constraining to zero the parameters associated with terms involving the first level of any of the factors. All interaction terms which involve the first level of any of the factors are also set to zero. Testing the models goodness-of-fit can be based on the deviance found in GLIM, which is equivalent to the likelihood-ratio statistic:

$$G^2 = -2\log(\max L),$$

where G^2 is approximately distributed as a Chi-square variable with $h - m$ degrees of freedom, $h = I \times J \times K$, and m is the number of parameters in the model.

The model can be described by the h data points with h parameters. This would be fitting a 'saturated' model. More commonly a model is obtained with less than h parameters by including only those factors and interactions that are significant - in order to explain the underlying structure with as few parameters as possible, i.e. building a parsimonious model. In this work a forward selection approach will be used: after fitting the constant term, the factor which produces the largest change in G^2 will be included, followed by the second and then the third, etc. Interaction between the factors with significant main effects will be considered in the following way. If there are at least two significant two-factor interactions than a three-factor interaction will be considered, and so forth. Interaction and effect modification is considered in section 3.2.6.

Significance testing for specific α terms in the model can then be based on the difference between the G^2 statistics for two 'nested' models, one of which does not contain the α term being tested. This difference between the G^2 statistics has an approximate Chi-square distribution with degrees of freedom equal to the difference in degrees of freedom of the two models. In GLIM the comparison of different models is undertaken through 'Analysis of Deviance'.[26]

Testing for homogeneity and trend in the SMR can then be undertaken by the method outlined in the previous section. The parameters of the fitted model are interpretable as logarithms of the relative risks, i.e. the α_k, $k=1,\ldots,n$, parameters are the logarithms of the SMRs in the k^{th} subgroup relative to that in the first subgroup. Anti-logging these parameter estimates yields the mortality rates for particular subgroups relative to the first. This clearly takes the same form as that of the relative risks obtained using the traditional SMR method. For example, the relative risk associated with the k^{th} subgroup is given by:

$$\phi_k = SMR_k / SMR_0 .$$

This is perhaps the major reason for the assumption of the multiplicative form of the model. Models specifying non-multiplicative relationships result in parameters not easily related to the relative risk, and may also pose estimation problems because of the range restrictions that have to be imposed on the parameters.

3.2.4 Logistic Regression.

Logistic regression techniques are employed in this thesis on nested case-control data sampled from the cohort population. Conditional maximum likelihood estimation is applied. This method is the modelling analogue of the Mantel-Haenszel stratification procedure.

The sampling method employed in this study is incidence density sampling. In this each case is matched to one or more controls selected at random from a set of subjects who are still at risk (the risk set) at the time that the case was identified. The risk set includes subjects who may subsequently develop the disease. Nested case-control data will yield unbiased estimates of the relative risk from the full cohort.

The parameter of interest in this type of study is the odds ratio. This ratio has the same value whether calculated from the disease or exposure probabilities. That is, the ratio of the odds of being exposed for the cases against the odds of exposure for the controls (exposure odds ratio) is equal to the ratio of the odds of having the disease in the exposed against the unexposed subjects (disease odds ratio). In the analysis the odds ratio estimates the increase in the odds of disease for the k^{th} factor (for example, exposure level) relative to the baseline level, $k=0$, of that factor.

Logistic regression produces a mathematical model in which the log odds is modelled as a linear combination of a set of risk factors. This model is derived from the mathematical function:

$$f(y) = 1/(1 + e^{-y}) \quad where \quad -\infty < y < +\infty,$$

$f(y)$ ranges from zero to one, as a sigmoid curve, as y increases from negative to positive infinity. This regression technique is also known as linear logistic regression. The word linear referring to the property that the logit transformation,

$$logit\ f(y) = \log\left[\frac{f(y)}{1-f(y)}\right] = y$$

is linear in y. The properties of this transformation are, therefore, used in epidemiology to model the risk of disease development during some specified time interval as a function of various independent variables known or suspected to be related to disease development.

If we now consider that Y is the odds of a subject being a case and that p is the proportion of subjects who are cases. This implies that:

$$Y = \frac{p}{(1 - p)}.$$

If we now consider the logarithm of the odds. A linear model can be created with the logit as the dependent variable in the following equation:

$$\log\left[\frac{p}{(1-p)}\right] = \alpha + \alpha_1 x_1 + \ldots\ldots \alpha_k x_k,$$

where x_j, $j=1, \ldots, k$ are the set of k explanatory variables. From this we have a linear model sometimes called a multiple logistic regression model, multiple in its number of k independent variables. So, the logit is the logarithm of the odds and logit differences are in turn logarithms of odds ratios. In GLIM you would perform a logistic analysis by specifying Binomial errors and declaring the link function to be logit. The goodness-of-fit of the generated models is then tested by scrutinising the deviances produced in GLIM.

Confidence intervals can then be used to test if the odds ratio produced is statistically different from unity. Miettinen has suggested an approximate confidence interval of the form:[27]

$$\psi^w, \qquad where \;\; w = (1 \pm Z^{\frac{\alpha}{2}}/\sqrt{\chi^2})$$

and χ^2 is the Mantel-Haenszel chi-square. An alternative is to calculate an estimate of the variance of the log odds and use the following logit confidence limits:

$$\exp(\log(\psi) \pm Z^{\frac{\alpha}{2}}\sqrt{Var[\log(\psi)]}).$$

A limitation of the multiple logistic model is the multiplicative relationship of separate explanatory variables in the model with each other. In the model each variable contributes towards the sum that is the log odds of disease. The different variables then have a multiplicative relationship with each other with regard to the rate of disease occurrence. This is equivalent to assuming a constant ratio measure of effect for a given factor over categories of the other factors.

3.2.5 Bias, Confounding and the Healthy Worker Effect.

Selection bias, as defined by Rothman, is the distortion in result that occurs from the techniques by which study subjects are chosen from the total population that could theoretically be studied.[28] In theory every occupational epidemiology study should include every worker ever employed in a specific industry followed up to the end of their life. In practice most studies use a subset of this information and from these subsets bias may occur. Bias may also be increased by various methodological errors, for example, considering subjects lost to follow-up as alive at the end of the study.

The most common form of selection bias in occupational studies is the healthy worker effect. It occurs when 'healthy' individuals are more likely to gain employment and to remain employed than the 'non-healthy'. In part this is due to self-selection, the individuals must be sufficiently healthy to seek work, and in part to employer selection, i.e. the use of pre-employment medical screenings. The effect can be typified by considering the general population. This population includes people who are too sick to work or who have been refused employment on health grounds and also employed people. Consequently, mortality rates in workforces are often lower than those of the general population. The healthy worker effect is the term sometimes used to refer to this phenomenon.

The healthy worker effect is of concern in the interpretation of occupational mortality studies because adverse effects of exposures at work may be wholly or partly masked by an apparent deficit of mortality in the workforce when compared to the general population. The inclusion of all person-time experience of every worker ever employed in a particular industry reduces bias, but it does not remove the bias resulting from initial selection of healthy workers into employment.

Healthy worker effects were first described by Ogle in 1885.[29] He saw the "considerable standards of muscular strength and vigour" required of individuals in certain occupations as introducing a "great flaw in all calculations of death rates in different industries". In brief three factors have been recognised in the healthy worker effect:[30]

1. The selection of healthy members from the source population.
2. The survival in the industry of healthier workers.
3. The length of time for which the population is followed.

Therefore, it is possible to characterise the healthy worker effect according to the events on which it operates, the start and end of work.

TABLE 3.19: SMRs by duration of follow-up in asbestos workers (taken from McMicheal[18]).

Causes of Death	Follow-up (years)		
	0-4	5-9	10-15
All Causes	95	112	123
All Neoplasms	100	136	153
Non-cancers	83	100	106

It has been shown in many studies that the mortality of employed persons, compared with the general population, is lowest during the period immediately after starting employment (table 3.19).[18,30,31] A common characteristic of these studies has been low SMRs for the early years of follow-up, with these ratios approaching unity (or 100) and beyond as follow-up continued. The relative mortality advantage of employed persons then diminishes with length of follow-up. Conversely, the advantage is also most pronounced in workers with the longest duration of employment. This is attributable to the survival in the workforce of relatively healthier workers.[31]

The healthy worker effect is obviously most marked in chronic diseases that are readily detectable. McMicheal argued that heart disease is therefore more likely to be selected against than cancer, with its "long-deferred clinical manifestations", in the recruitment and retention of active workers.[18] This in turn would result in a reduction in 'non-cancer' mortality in comparison to 'cancer' mortality shortly after study definition in an occupational cohort (table 3.19). Fox and Collier also showed differential patterns in SMRs according to the cause of death examined.[30] In their study of polyvinyl chloride production workers, SMRs for respiratory diseases were lowest and increased more slowly with time since entry to the industry (table 3.20). SMRs in this study were highest for cancer, those for circulatory disease were intermediate. Another example of differential patterns in SMRs is given by the OPCS Longitudinal Survey.[32] In this survey SMRs in employed males were lower for respiratory and circulatory diseases than for malignant neoplasms, all increasing with time since cohort definition.

TABLE 3.20: SMRs by time since entering the industry (taken from Fox and Collier[30]).

Causes of Death	Time since entering the industry			
	0-4	5-9	10-14	15+
All Causes	37	63	75	94
All Neoplasms	45	71	94	112
Circulatory Disease	22	70	85	91
Respiratory Disease	21	39	31	93

In Fox and Collier's study of the polyvinyl chloride manufacturing industry a survival effect was shown by separating workers who survived 15 years, from study definition, according to whether they were still in the industry or not. The all cause mortality rate among those who left the industry was approximately 50% higher than those still employed. Other studies have supported the hypothesis that the healthy worker effect is strongest during active employment and rapidly disappears following the cessation of employment, particularly if this occurs before

the usual retirement age.[33,34] It has even been suggested that the term "active worker effect" be used in place of heathy worker effect.[35]

Whether the effect is called 'healthy worker', 'active worker', 'comparison bias', as suggested by Hernberg[36], or health-related selection bias, it is often observed in occupational cohorts. Its main characteristic are initial low mortality following study definition becoming less apparent with time. The effect is, therefore, time-dependent. This suggests a method of managing this form of bias, e.g. by investigating the way in which SMRs change with time since entry into a workforce, as opposed to using the overall magnitude of SMRs at any one point in time. As an alternative to this, regression models as outlined in section 3.2.3 may be used to eliminate any confounding due to the healthy worker effect, i.e. due to length of employment, length of follow-up and age.

Selection bias and confounding can be viewed as aspects of the same phenomenon: distortions that the investigator hopes to prevent or, if necessary to remove from the data. A method outlined by Rothman to counteract these is to consider any bias that can be controlled in the analysis as confounding.[28] Confounding can be considered as a mixing of the effect of the study factor/exposure of interest with the effect of other risk factors. A confounder is, therefore, a factor in someway associated with both exposure and disease. In occupational studies time-related factors are good predictors of disease states and will be confounders if they are also associated with exposure. For example, calendar year will be a confounder if disease incidence and exposure patterns vary over time. Another example of this concerning both bias and confounding would be if more recently employed workers (with better workplace conditions and lower levels of exposure), followed for a short period, are compared to workers employed in a previous period (with higher exposure levels).

Confounding can be controlled in the study design, in the analysis or in both. Controlling during study design can involve three methods:

1. Randomisation. That is, random allocation to exposure categories. This is not possible in occupational studies.

2. Restriction. Restricting the study to narrow the range of potential confounders (e.g. restricting a study to workers aged less than 45). This method as well as restricting confounding may also restrict the usefulness of a study.

3. Matching. Matching study subjects on potential confounders. This is the preferred method of control, its main limitation is that it is expensive in time and project costs. It is effective in cohort studies and very effective in nested case-control studies. It should be noted that matching does not remove the confounder, but aids in its control during analysis.

Controlling confounding during analysis involves the simultaneous control of all confounding factors. This is undertaken by stratifying the data according to the levels of the confounders and calculating an effect estimate that summarizes the information across each subgroup (strata) of confounder. The study size clearly restricts the number of confounders that can be simultaneously controlled. The ideal method of stratifying for all confounders has to be limited by the data, and care taken not to over stratify or produce too many strata cells with no data. The problem of over stratification can be solved by regression modelling, allowing for the simultaneous control of confounders by smoothing the data across subgroups.

Control of confounding is important and requires careful use of prior knowledge, and also inference from the observed data. Most occupations involve exposure to more than one potential risk factor and the possibility of confounding by other occupational exposures must be considered in the context of each study.

3.2.6 Effect Modification (Statistical Interaction).

In epidemiology the concept of effect modification and interaction is more difficult to define than in experimental studies. The aetiology of a disease is often very complex, with many causative and confounding factors. For cancer, carcinogens may act at different stages independently of each other, but each stage is required to occur before the next one can start. The question is therefore, can occupational epidemiology and its use of probability theory to identify risk factors provide an insight into the biological mechanisms of disease causation? A following question is whether an interaction term in a regression model can be interpreted on any biological scale?

Other factors which complicate the issue are the statistical models used to analyse epidemiological data. The commonest model used is logistic regression, where the odds of disease, transformed by the logarithmic function, are related to a linear combination of risk factors. The logistic model assumes that the risk factors combine on a multiplicative scale to estimate the risk of disease. Thus, even when no interaction term (product term) is present in the model, the effect of the individual terms is additive on the log scale, i.e. multiplicative on the non-log scale. This leads to the question of whether there is independence of the risk factors if additivity on a log scale applies. The same question applies to poisson regression (log-linear modelling) since it also measures interaction on this scale.

Clearly to demonstrate biological interaction by means of statistical models requires knowledge of the underlying biological model and reasoning, and cannot be done mechanically. Helping to clarify the situation, Rothman in 1980 suggested that interaction in epidemiology can be defined in four broad contexts: statistical, biological, public health and individual decision making.[37] His definitions are as follows:

Statistical interaction. This is dependent on the mathematical model fitted to the data. If the regression coefficient for the product term of the risk factors is significantly different from zero and the goodness-of-fit statistic shows that presence of the interaction term improves the fit, then statistical interaction is present and should be included in the model.

Biological interaction. This is present when two or more causative factors act interdependently to produce the disease. Two categories are of interest: 1) those in which the aetiological factors act interchangeably in the same step in a multiple process and thus the effect of each factor adds to the effect of the other. 2) those that act at different steps in the process. For example, smoking may cause damage to lung cells and subsequent exposure to dust acts on those cells to increase the probability of lung cancer. The two categories correspond to the statistical models in which the combined effect of exposure factors is assumed to be additive or multiplicative, respectively. The best fitting of the two models can suggest the nature of the interrelation between the causal factors.

Public-health interaction. This is present when the effect of one risk factor on the disease occurrence is modified by the presence of another and when departure from the additivity of the individual effects is observed. For statistical models, applied to test the combined effect, it means that a significant interaction term is present in the model that assumes additivity of relative risks, and that the model that assumes multiplicativity of the effects may fit the data significantly better than the model that assumes additivity of the effects.

Individual-decision making. This involves evaluating personal risk of disease considering that the causal factors acting in combination lead to an increased/decreased risk of disease above/below the sum of the individual risks.

Considering now a visual assessment of interaction. By plotting calculated relative risks against the exposure of interest, a trend in a dose-response relationship would be detected. If we choose a continuous variable for exposure X and a dichotomous

(0,1) indicator variable for exposure Y, then by plotting the relative risks for each exposure Y against the exposure X, an indication of whether an interaction affect is present or not can be detected. For example, if the exposure Y modifies the effect of the exposure X, then effect modification (interaction) is present. Figure 3.1 shows some possible relationships.

The detection of the presence of an effect modification depends to some extent upon the transformation scale applied when analysing the data. When we try to infer the presence of an interaction from the data plotted in figure 3.1 in terms of the additive or the multiplicative models, the following can be observed:

1) [Figure 3.1a]: The lines are parallel, therefore, there is no evidence of an interaction on the additive scale, but there is a negative interaction on the multiplicative scale.

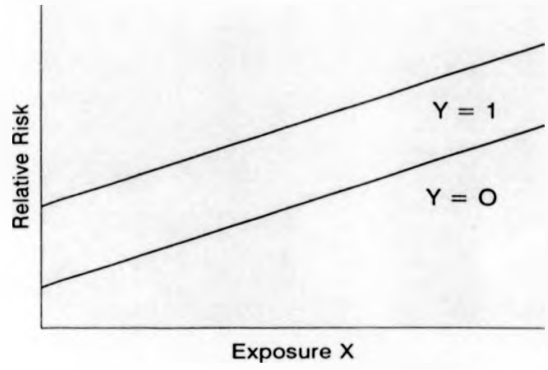
2) [Figure 3.1b]: There may be no interaction on the multiplicative scale, but a positive interaction on the additive scale.

3) [Figure 3.1c]: An interaction is present, but may be detected only by means of indicator variables for the exposure X, in which case an additive relative risk model is more likely to detect the interaction.

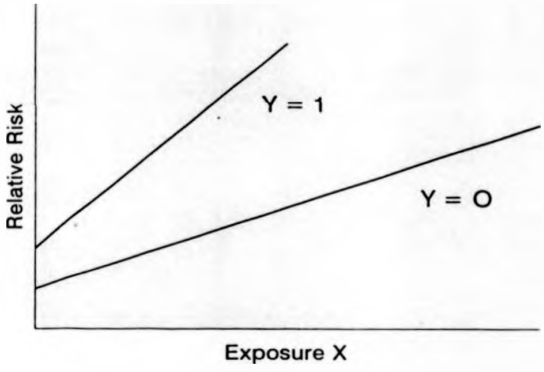
Epidemiology should go beyond pure statistical modelling. The idea of effect modification and statistical interaction, linked as it is to an arbitrariness in the choice of model, can lack interpretation in occupational epidemiology since it does not rest on a definite theoretical foundation. Without implication for inference beyond the model, a purely statistical notion of interaction does not contribute to the study of occupational epidemiology. If the occurrence of interaction were simply a statistical construction that allowed contradictory interpretations from the same data, it would not contribute to scientific and medical knowledge.

FIGURE 3.1: The trends in relative risk that can be observed with two risk factors.

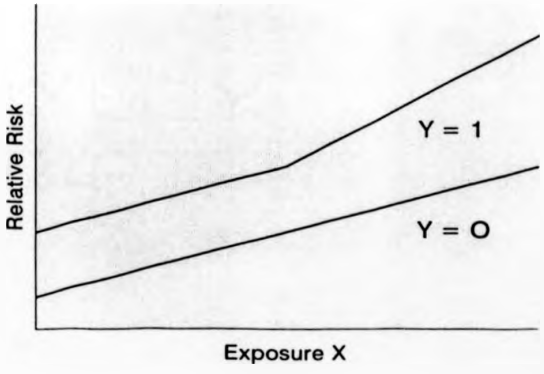
a)



b)



c)



3.2.7 Disease Groupings and Regional Adjustment Factors.

This section is concerned with the standard death rates used in this thesis and in particular their formation from the OPCS 'Historic Mortality Data Files' and the use of regional adjustment factors.

In the analysis of cohort mortality a pivotal role is frequently played by published national death rates (this was outlined in section 3.2.2). Clearly, their use restricts the researcher to the published structure of these rates. The Office of Population Censuses and Surveys (OPCS) recognised this limitation and in 1979 created their historic deaths file. This file contains death information by age, sex, and International Classification of Disease (ICD) code for England and Wales on a yearly basis from 1901. This data file is, therefore, a basic building block that when linked with corresponding population data can be formed to fit research project needs. The ICD codes recorded (i.e. 7th, 8th or 9th revision of ICD) are those in operation at the time of registration of death.

For this work OPCS supplied annual national death information for the years 1972 to 1988. This period presents two problems in the construction of a death rate database. First, it overlaps two revisions of the ICD, the 8th revision was in force through 1968-1978, the 9th revision has been used since 1979. Secondly, the follow-up period in this study is 17 years and based on the enumeration dates presented in section 3.1.1 we have a study period extending between 1972 and 1990.

The last problem was solved by using the death rates constructed for 1988 as 'best' estimates for those of 1989 and 1990. Considering the first problem, that is the overlapping ICD revisions. All observed deaths in this study have been coded by a nosologist to the 9th revision of the ICD. The death rates generated from the OPCS file for the years 1972-1978 are based on 8th revision disease groupings. Since these have to be compared with 9th revision observed deaths approximate disease groupings have been used for certain conditions. Table A2.1 lists the causes of death used in this study with their attendant 9th revision grouping and its

8th revision estimate. From this table it can be seen that three disease groupings are principally affected by this approximation, namely, diseases of pulmonary circulation (9th ICD: 415-417, estimated 8th: 426 and 450), chronic obstructive pulmonary disease (9th ICD: 490-496, estimated 8th: 490-493 and 518) and pneumoconiosis (9th ICD: 500-508, estimated 8th: 515-516). All three of these groups are new to the 9th revision. Of these only disease of pulmonary circulation is used as a specific cause of interest in this study (table 3.18), this is to give an estimate of *cor pulmonale* mortality, a major constituent of the 9th revision grouping of codes 415-417. The disease grouping of bronchitis, emphysema and asthma (8th and 9th ICD: 490-493) is used as a surrogate for chronic obstructive pulmonary disease. The disease grouping of pneumoconiosis is considered only as a general grouping of interest, whereas, its included condition asbestosis (9th ICD: 501, 8th ICD 515.2) is obviously considered as a specific cause.

The disease grouping of pneumoconiosis (9th ICD: 500-508) was mainly classified to codes 515 and 516 in the 8th revision. However, aspiration bronchopneumonia and aspiration pneumonitis, coded as part of 8th revision groups 485 and 486, are included in the 9th revision pneumoconiosis group under code 507 (pneumonitis due to solids and liquids).[38] The 8th revision codes, 485 and 486, are both broad codes covering unspecified bronchopneumonia and pneumonia. It is impossible to singly subtract the effect of aspiration bronchopneumonia and aspiration pneumonitis from these groups and include them in the construction of the 1972-1978 death rates for pneumoconiosis. The construction of pneumoconiosis death rates, from the OPCS historic deaths file, will, therefore, only produce approximate estimates of the true rates. This is particularly true for all disease groupings that are new in the 9th revision of the ICD.

Apart from changes to the classifications, new groupings etc., changes were also made in the rules for the coding of the underlying cause of death from the 8th to 9th revision. The change that most affects this study is the removal of the precedence in coding for asthma when reported with bronchitis. This has resulted in an increase in the number of reported asthma cases and a decrease in the number with bronchitis from 1979. This rule change is effectively covered by using the

broad disease group of bronchitis, emphysema and asthma. However, any change in coding rule adds extra problems to the use of death rates across ICD revision. They tend, obviously, to make changes also to the measurement of disease frequency that can be both elusive and subtle and can only be counteracted by using broad disease groups.

The England and Wales death rates constructed for this study are given in appendix 2. These rates are in 5 year age groups starting at age 10 and continuing until age 84, ages over 84 are grouped together to form a '85 plus' group. In total 50 disease groups are given in this appendix.

Ideally, the standard population used in calculating expected mortality should be one that is similar in both demographic and environmental characteristics, apart from exposure history, to the cohort population. Attempts to achieve this include using standard populations that are of the same sex, race and geographical location as the occupational cohort. The standard population death rates created and used in this study are male rates for the whole of England and Wales. To produce rates that more closely characterise the cohort, i.e. regional rates instead of national rates, was not possible when using the OPCS historic deaths file. This data file was only available for the whole of England and Wales.

As an approximation to the direct formation of regional rates an alternative is needed. This alternative can take the form of applying a regional adjustment to the calculated national death rates. Considering the study period of this work, i.e. 1972 to 1990, the microfiched area mortality information from the last decennial supplement of the OPCS that covers the years 1979 to 1983, approximately the midyears of this study, can be used to produce these regional adjustment factors.[39] From the decennial supplement SMRs are given for the standard regions of England and Wales, i.e. South East, North West, etc., for all ages, for the age group 15 to 64 and for the age group 65 plus. They are also given for both sexes and for selected causes of death. These SMRs, represented as proportions and not percentages, can be used as weighting factors to adjust the national rates

into regional estimates, for the relevant ages and causes of death. Table A2.2 gives the regional adjustments used in this thesis.

From table A2.2 it can be seen that many causes of death considered in this study have no applicable adjustment factor from the 1979-1983 OPCS data. For these the nearest available OPCS area information is used. This is the data from the OPCS 1989 area mortality statistics microfiche.[40] Where the 1979-1983 information is present it is used to produce an adjustment, when absent the 1989 data have been used. For 18 causes of death, indicated in table A2.2, no adjustment factor was possible. Mesothelioma of the pleura and peritoneum and also asbestosis are included among the causes with no regional adjustment factor.

The regional adjustment factors used are for the South West and South East standard regions. The south west adjustment is used for Devonport dockyard and the south east for both Chatham and Portsmouth dockyards. When no adjustment is given, i.e. for ages 10 to 14 and for causes of death with no obvious factor, unity is used as the factor.

There are several reasons why national or regional death rates may not be completely appropriate to the group under study. For example, the social-class structure or the smoking habits of the group may differ from those of the region. Another reason is the healthy worker effect, due to the selective mechanisms that contribute to deciding an individuals occupation. As mentioned in section 3.2.5 the national population contains some people not fit enough to engage in certain types of work. These people contribute to the death rates of the national population but are inappropriate for comparison with a working population of which they could not be members. The difficulty of interpretation that arises because the comparisons may not be completely appropriate is an inevitable feature of the epidemiological approach, as opposed to the experimental one - but, of course, the experimental approach makes the assumption that the rat in his artificial environment is comparable to man in his 'free' environment.

Chapter 4: MORTALITY PATTERNS IN ROYAL NAVAL DOCKYARDS.

4.1 Introduction.

This chapter describes the results of standardised mortality ratio (SMR) and Poisson regression analyses performed to investigate dockyard mortality patterns with regard to occupation, exposure to asbestos and time. Consideration is given to the effect of self-selection into the study base, i.e. the mortality rates of study non-responders, as defined in the last chapter, are appraised and compared to responder rates. Attention is also given to the healthy worker effect and its influence on mortality. This involves examining associations between mortality and the factors of greatest relevance to health-related selection, namely time since employment and duration of employment. Before describing the results of these analyses on the specific causes of death given in table 3.18, the overall mortality in the workforce is summarised for the 50 disease groups given in table A1.2. Lung cancer and pleura mesothelioma are given special consideration throughout this chapter in an attempt to address the questions arising from the past dockyard studies (i.e. the finding of a non-significant lung cancer risk accompanied by a very significant mesothelioma risk[1,2]). Peritoneal mesothelioma and asbestosis will also be considered in this light, however, the small number of reported deaths from these causes (13 and 10 respectively) make any conclusive results unlikely.

The analyses performed in this chapter were undertaken only for those workers traced by the Office of Population Censuses and Surveys (OPCS): 27,502 workers across the three dockyards, that is, 97.3% of the study base. Workers that emigrated were considered lost to follow-up at their date of emigration. Censoring was undertaken for subjects who reached the age of 85, i.e. 85 was considered the age above which the reliability of data concerning the events of interest would be considered inadequate. This censoring prevents the bias that would result from allowing subjects to contribute time at risk to the rate denominator of the SMR,

when they are not in fact at risk of suffering an event that could be both observed and counted in the numerator. This had the effect of reducing the number of observed deaths in the study by 5, i.e. from 5,031 to 5,026. The results for each dockyard are reviewed independently. The main results tables are given in appendix 3. Appendix 4 contains supplementary results.

4.2 Cause specific mortality for 50 disease groups.

This section is concerned both with the overall mortality pattern shown in the 50 disease groups and the effect of regional adjustment on this pattern. The results for the three dockyards are given in tables 4.1 to 4.3.

From these tables it can be seen that the workforce experienced mortality rates that were, generally, 13-18% lower than national rates; the all-cause SMR was 87 for Devonport (95%CI: 83-91), 85 for Chatham (95%CI: 80-91) and 82 for Portsmouth (95%CI: 78-85)*. The effect of applying a regional adjustment was to increase these SMRs to the following levels; 97 (93-101), 91 (86-97) and 88 (83-92) for Devonport, Chatham and Portsmouth respectively. The observed mortality being generally significantly lower than expected, both with and without regional adjustment. The variation in all-cause SMR across dockyard was also found to be statistically significant [$X^2 = 9.7$, $P < 0.01$]**.

For the disease group 'all-neoplasms' a statistical deficit is not seen. This is best illustrated by considering the regionally adjusted all-neoplasm SMR. For the three dockyards in the order Devonport, Chatham and Portsmouth*** we have the following SMRs: 117 (109-125), 107 (96-118) and 98 (90-106). Devonport

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* Throughout, 95% confidence intervals will henceforth be shown in parentheses, e.g. (83-91) in place of (95%CI: 83-91).

** Chi-squared tests for a difference between SMRs will be given, when appropriate, inside square brackets [ ].

*** This order of Devonport, Chatham and Portsmouth will be used throughout the remainder of this work.

when they are not in fact at risk or suffering an event that could be both observed and counted in the numerator. This had the effect of reducing the number of observed deaths in the study by 5, i.e. from 5,051 to 5,046. The results for each dockyard are reviewed independently. The main results tables are given in appendix 3. Appendix 4 contains supplementary results.

4.2 Cause specific mortality for 50 disease groups

This section is concerned both with the overall mortality pattern shown in the 50 disease groups and the effect of regional adjustment on this pattern. The results for the three dockyards are given in tables 4.1 to 4.3.

From these tables it can be seen that the workforce experienced mortality rates that were generally 15-18% lower than national rates; the all-cause SMR was 87 for Devonport (95%CI: 83-91), 85 for Chatham (95%CI: 80-91) and 82 for Portsmouth (95%CI: 78-85)*. The effect of applying a regional adjustment was to increase these SMRs to the following levels: 97 (93-101), 91 (86-97) and 88 (83-92) for Devonport, Chatham and Portsmouth respectively. The observed mortality being generally significantly lower than expected, both with and without regional adjustment. The variation in all-cause SMR across dockyard was also found to be statistically significant: $[X^2 = 9.7,\ P < 0.01]$**.

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*** This order of Devonport, Chatham and Portsmouth will be used throughout the remainder of this work.

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* Throughout, 95% confidence intervals will henceforth be shown in parentheses, e.g. (83-91) in place of (95%CI: 83-91).

** Chi-squared tests for a difference between SMRs will be given, when appropriate, inside square brackets [ ].

*** This order of Devonport, Chatham and Portsmouth will be used throughout the remainder of this work.

TABLE 4.1: Devonport Dockyard. Cause specific mortality for 50 disease groups, with and without regional adjustment.

Causes of Death	Without regional adjustment Obs	Exp	SMR	95% CI	With regional adj. SMR	95% CI
All Causes	2289	2629.8	87	83- 91	97	93-101
Infectious and Parasitic Diseases	6	12.5	48	18-105	56	21-122
Tuberculosis	2	5.4	37	5-135	57	7-206
All Neoplasms	789	758.9	104	97-111	117	109-125
Ca. Lip, Oral Cavity and Pharynx	5	11.1	45	15-105	51	16-118
Ca. Digestive Organs and Peritoneum	244	220.9	110	97-124	118	103-132
Ca. Oesophagus	31	26.0	119	77-161	127	82-172
Ca. Stomach	81	66.9	121	95-147	145	113-176
Ca. Peritoneum (mesothelioma)	10	1.4	731	351-1344		
Ca. Respiratory System	318	312.7	102	90-113		
Ca. Lung	241	301.3	80	70- 90	99	87-112
Ca. Pleura (mesothelioma)	66	3.3	1983	1505-2461		
Ca. Bone, Tissue, Skin and Breast	14	11.9	118	65-198		
Ca. Genito-urinary Organs	84	87.6	96	75-116	90	71-110
Ca. Prostate	40	41.9	95	66-125	89	61-117
Ca. Other and Unspecified Sites	74	60.3	123	95-151		
Ca. Lymphatic and Haematopoietic Tissue	42	46.5	90	63-118	94	66-122
Benign Neoplasms	3	2.2	138	28-402	172	36-504
Unspecified Neoplasms	5	5.4	93	30-216		
Endocrine and Nutritional Diseases	29	28.4	102	68-147	101	67-145
Diseases of Blood and Blood-forming Organs	3	5.2	58	12-169	63	13-184
Diseases of the Nervous System	26	34.7	75	49-110	75	51-116
Diseases of the Circulatory System	1152	1310.4	88	83- 93	95	90-101
Hypertensive Disease	17	24.9	68	40-109	74	43-119
Ischaemic Heart Disease	868	924.0	94	88-100	102	95-109
Diseases of Pulmonary Circulation	28	12.3	227	151-329	232	154-335
Cerebrovascular Disease	148	205.8	72	60- 84	78	66- 91
Diseases of the Respiratory System	192	255.4	75	65- 86	95	82-109
Acute Respiratory Infections	1	2.8	36	91-199		
Other Disease of Upper Respiratory Tract	0	0.2	-	0-1836		
Pneumonia and Influenza	39	79.6	49	34- 64		
Bronchitis, Emphysema and Asthma	45	106.8	42	30- 54	60	43- 78
Chronic Obstructive Pulmonary Disease	85	156.8	54	43- 66	72	57- 88
Pneumoconiosis	7	3.6	197	79-406	352	141-726
Coalworkers Pneumoconiosis	0	1.8	-	0-203		
Asbestosis	7	0.4	1718	690-3538		
Silicosis	0	0.3	-	0-1308		
Other Diseases of the Respiratory System	8	11.3	71	30-139		
Pulmonary Fibrosis	2	3.0	68	8-245		
Diseases of the Digestive System	33	65.9	50	33- 67	56	37- 76
Diseases of Oesophagus and Stomach	11	21.6	51	25- 91		
Diseases of the Genito-urinary System	11	24.7	45	22- 80	51	25- 91
Diseases of the Skin and Subcutaneous Tissue	1	1.0	102	3-568	91	2-506
Diseases of the Musculoskeletal System	1	7.3	14	0- 76	15	0- 83
Symptoms, Signs and Ill-defined Conditions	0	2.4	-	0-153	-	0-199
Accidents, Poisonings and Violence	35	104.3	34	22- 45	34	23- 46
Transport Accidents	5	32.1	16	5- 36		
Accidental Poisoning	2	4.1	49	6-177		
Accidental Falls	1	10.8	9	0- 51		
Suicide and Self-inflicted Injury	10	30.5	33	16- 60		

TABLE 4.2: Chatham Dockyard. Cause specific mortality for 50 disease groups, with and without regional adjustment.

Causes of Death	Without regional adjustment Obs	Exp	SMR	95% CI	With regional adj. SMR	95% CI
All Causes	1045	1223.9	85	80- 91	91	86- 97
Infectious and Parasitic Diseases	6	5.5	109	40-237	106	39-231
Tuberculosis	0	2.3	-	0-158	-	0-154
All Neoplasms	365	351.0	104	93-115	107	96-118
Ca. Lip, Oral Cavity and Pharynx	3	4.9	61	13-178	72	15-211
Ca. Digestive Organs and Peritoneum	103	102.0	101	81-120	112	90-134
Ca. Oesophagus	8	11.9	67	29-132	73	31-144
Ca. Stomach	34	31.1	109	72-146	122	81-162
Ca. Peritoneum (mesothelioma)	2	0.6	338	41-1222		
Ca. Respiratory System	146	144.3	101	85-118		
Ca. Lung	116	139.1	83	68- 99	85	70-101
Ca. Pleura (mesothelioma)	24	1.5	1638	1049-2437		
Ca. Bone, Tissue, Skin and Breast	6	5.3	114	42-249		
Ca. Genito-urinary Organs	38	42.4	90	61-118	90	61-118
Ca. Prostate	20	21.1	95	58-146	90	55-140
Ca. Other and Unspecified Sites	47	27.4	171	122-220		
Ca. Lymphatic and Haematopoietic Tissue	18	21.0	86	51-135	86	51-137
Benign Neoplasms	1	1.0	104	3-580	116	3-646
Unspecified Neoplasms	3	2.5	121	25-354		
Endocrine and Nutritional Diseases	8	13.4	60	26-118	62	27-123
Diseases of Blood and Blood-forming Organs	0	2.5	-	0-147	-	0-151
Diseases of the Nervous System	12	16.3	74	38-129	76	39-133
Diseases of the Circulatory System	487	610.2	80	73- 87	89	81- 96
Hypertensive Disease	14	11.4	123	67-206	124	68-208
Ischaemic Heart Disease	360	424.7	85	76- 93	94	84-103
Diseases of Pulmonary Circulation	4	5.8	69	19-177	66	18-170
Cerebrovascular Disease	71	99.8	71	55- 88	84	65-104
Diseases of the Respiratory System	107	124.0	86	70-103	93	75-110
Acute Respiratory Infections	2	1.3	155	19-559		
Other Disease of Upper Respiratory Tract	0	0.1	-	0-3672		
Pneumonia and Influenza	23	38.8	59	38- 89		
Bronchitis, Emphysema and Asthma	35	50.9	69	46- 91	77	51-102
Chronic Obstructive Pulmonary Disease	62	76.1	82	61-102	89	66-111
Pneumoconiosis	0	1.7	-	0-212	-	0-512
Coalworkers Pneumoconiosis	0	0.9	-	0-404		
Asbestosis	0	0.1	-	0-1992		
Silicosis	0	0.3	-	0-2639		
Other Diseases of the Respiratory System	3	5.5	55	11-160		
Pulmonary Fibrosis	1	1.4	72	2-399		
Diseases of the Digestive System	14	30.3	46	25- 78	49	27- 82
Diseases of Oesophagus and Stomach	7	10.2	69	28-142		
Diseases of the Genito-urinary System	11	11.9	92	46-165	94	28-142
Diseases of the Skin and Subcutaneous Tissue	0	0.5	-	0-777	-	0-771
Diseases of the Musculoskeletal System	1	3.5	28	1-159	30	1-166
Symptoms, Signs and Ill-defined Conditions	0	1.1	-	0-335	-	0-271
Accidents, Poisonings and Violence	22	44.5	49	31- 75	51	32- 77
Transport Accidents	6	13.7	44	16- 96		
Accidental Poisoning	2	1.7	117	14-422		
Accidental Falls	1	4.9	21	1-115		
Suicide and Self-inflicted Injury	6	12.8	47	17-102		

TABLE 4.3: Portsmouth Dockyard. Cause specific mortality for 50 disease groups, with and without regional adjustment.

Causes of Death	Without regional adjustment Obs	Exp	SMR	95% CI	With regional adj. SMR	95% CI
All Causes	1692	2072.7	82	78- 85	88	83- 92
Infectious and Parasitic Diseases	7	9.3	75	30-155	73	29-151
Tuberculosis	2	3.8	52	6-189	51	6-184
All Neoplasms	575	604.2	95	87-103	98	90-106
Ca. Lip, Oral Cavity and Pharynx	12	8.7	138	71-240	164	85-286
Ca. Digestive Organs and Peritoneum	164	175.6	93	79-108	104	88-120
Ca. Oesophagus	27	21.1	128	84-186	139	92-203
Ca. Stomach	45	52.6	86	61-111	95	68-123
Ca. Peritoneum (mesothelioma)	1	1.0	99	2-551		
Ca. Respiratory System	251	248.3	101	89-114		
Ca. Lung	218	239.1	91	79-103	94	81-106
Ca. Pleura (mesothelioma)	28	2.7	1042	693-1506		
Ca. Bone, Tissue, Skin and Breast	8	9.0	88	38-174		
Ca. Genito-urinary Organs	42	71.9	58	41- 76	58	41- 76
Ca. Prostate	26	35.6	73	48-107	70	46-102
Ca. Other and Unspecified Sites	62	48.2	129	97-161		
Ca. Lymphatic and Haematopoietic Tissue	33	36.2	91	60-122	92	97-161
Benign Neoplasms	2	1.6	122	15-439	136	61-124
Unspecified Neoplasms	1	4.3	23	1-130		
Endocrine and Nutritional Diseases	17	23.0	74	43-119	77	45-124
Diseases of Blood and Blood-forming Organs	4	4.2	95	26-242	97	26-248
Diseases of the Nervous System	15	27.7	54	30- 89	56	31- 92
Diseases of the Circulatory System	820	1037.0	79	74- 84	88	82- 94
Hypertensive Disease	24	18.9	127	81-189	129	83-193
Ischaemic Heart Disease	582	731.4	80	73- 86	88	81- 95
Diseases of Pulmonary Circulation	4	9.5	42	11-108	40	11-103
Cerebrovascular Disease	122	164.3	74	61- 87	88	72-104
Diseases of the Respiratory System	160	200.2	80	68- 92	86	73- 99
Acute Respiratory Infections	5	2.1	240	78-559		
Other Disease of Upper Respiratory Tract	0	0.2	-	0-1530		
Pneumonia and Influenza	29	59.0	49	33- 71		
Bronchitis, Emphysema and Asthma	39	81.2	48	33- 63	54	37- 71
Chronic Obstructive Pulmonary Disease	78	126.2	62	48- 76	67	52- 82
Pneumoconiosis	3	2.8	106	22-310	251	52-733
Coalworkers Pneumoconiosis	0	1.4	-	0-256		
Asbestosis	3	0.3	905	187-2646		
Silicosis	0	0.2	-	0-1669		
Other Diseases of the Respiratory System	7	9.3	75	30-155		
Pulmonary Fibrosis	1	2.4	43	1-237		
Diseases of the Digestive System	38	51.7	74	50- 97	78	57- 99
Diseases of Oesophagus and Stomach	9	17.0	53	24-100		
Diseases of the Genito-urinary System	9	19.2	47	22- 89	48	22- 90
Diseases of the Skin and Subcutaneous Tissue	2	0.8	252	30-908	250	30-903
Diseases of the Musculoskeletal System	6	6.0	100	37-218	106	39-230
Symptoms, Signs and Ill-defined Conditions	0	1.8	-	0-201	-	0-160
Accidents, Poisonings and Violence	31	72.2	43	28- 58	44	29- 60
Transport Accidents	3	21.4	14	3- 41		
Accidental Poisoning	1	2.7	37	1-205		
Accidental Falls	2	8.0	25	3- 90		
Suicide and Self-inflicted Injury	14	21.3	66	36-111		

showing a clear excess of cancer mortality. The difference in SMRs was significant [$X^2 = 10.5$, $P < 0.01$]. This picture, of SMRs close to, or above, 100 for cancer mortality and a deficiency in all-cause mortality, may imply the existence of the healthy worker effect. Accordingly, non-cancer disease groupings will now be considered and in particular the two groups, diseases of the circulatory system, and diseases of the respiratory system.

For diseases of the circulatory system the following regional SMRs were obtained: 95 (90-101), 89 (81-96) and 88 (82-94) [$X^2 = 3.3$, $P > 0.1$]. For diseases of the respiratory system we have: 95 (82-109), 93 (75-110) and 86 (73-99) [$X^2 = 0.1$, $P > 0.1$]. From this there is only a slight suspicion of the healthy worker effect, with the upper limit of the confidence intervals either overlapping 100 or being less than 6% beneath it. In the presence of the healthy worker effect a reduction in 'non-cancer' SMRs could have been expected in comparison to 'cancer' SMRs. However, this phenomenon may be obscured here by the long follow-up period. Accordingly, mortality patterns for the 12 specific disease groups will be scrutinised by time since start of employment for the healthy worker effect in section 4.5.

This pattern of SMRs, the 'non-cancer' SMRs having confidence intervals overlapping 100, generally holds for all subgrouped diseases within the circulatory system grouping, with the exception of disease of pulmonary circulation at Devonport dockyard. In this instance, the SMR is significantly elevated: 232 (154-335). Clearly, this does not support the idea of an important healthy worker effect. However, this disease group is one of the three commented upon in the last chapter as being only an estimated grouping, and therefore having only estimated death rates (section 3.2.7). It is used here as a limited indicator of *cor pulmonale* mortality, a disease suspected 'a priori' to be related to asbestos exposure.[3] Caution should, however, be applied in the over interpretation of this single result.

Within the 'diseases of the respiratory system' grouping, we have the following four interesting groups: 1) Pneumonia and Influenza, 2) Bronchitis, Emphysema and Asthma, 3) Asbestosis, and 4) Pulmonary Fibrosis. From these only bronchitis, emphysema and asthma is regionally adjusted, giving the following SMRs over the three dockyards: 60 (43-78) at Devonport, 77 (51-102) at Chatham, and 54 (37-71) at Portsmouth [$X^2 = 2.4$, $P > 0.1$], generally indicating a deficiency in mortality due to bronchitis, emphysema and asthma. This deficiency is also seen for pneumonia and influenza, the upper limit of the confidence intervals reaching only 89%. For asbestosis, a large excess mortality is clearly seen at two dockyards: the SMR at Devonport is 1718 (690-3538), no deaths from asbestosis were recorded at Chatham, and at Portsmouth the SMR is 905 (187-2646). However, these SMRs are based upon only 7 and 3 deaths, respectively, [$X^2 = 0.9$, $P > 0.1$]. It is therefore likely that this large difference in SMRs has arisen simply due to statistical chance. For pulmonary fibrosis, that is, fibrosis coded without mention of asbestos on the death certificate, the following SMRs were obtained: 68 (8-245), 72 (2-399) and 43 (1-237). These, however, are based upon only 4 deaths across the three dockyards [$X^2 = 0.2$, $P > 0.1$]. Asbestosis and pulmonary fibrosis (9th revision ICD codes 501 and 515), although arguably the same clinical condition without postmortem verification of the death certificate, are analysed separately throughout this work. All death certificates are analysed as coded by the nosologist (the professional death certificate coder) to avoid bias.

Pneumoconiosis is also a subgroup of diseases of the respiratory system. It has a regional adjustment factor and the following SMRs were produced: 352 (141-726) at Devonport, no deaths were recorded at Chatham, and 251 (52-733) at Portsmouth [$X^2 = 0.2$, $P > 0.1$]. Clearly, the mortality due to pneumoconiosis at Devonport dockyard is significantly elevated. However, since this is again one of the estimated disease groups referred to in chapter three, care should be exercised in interpreting this result. Coalworkers' pneumoconiosis and silicosis were also considered among the 50 disease groups. This was to allow for any

effect of previous mining or quarrying employment, however, there were no recorded deaths from these conditions at any dockyard.

The final estimated disease group, that of chronic obstructive pulmonary disease, follows the same pattern as its surrogate group 'bronchitis, emphysema and asthma' with slightly higher regional SMRs of 72 (57-88) at Devonport, 89 (66-111) at Chatham, and 67 (52-82) at Portsmouth [$X^2 = 3.0$, $P > 0.1$]. Showing, as with its substitute grouping, a general deficiency in mortality due to chronic obstructive pulmonary disease. The grouping of bronchitis, emphysema and asthma will be used in place of the estimated group of chronic obstructive pulmonary disease in the analysis of the 12 specific disease groups.

Returning to all-neoplasms, by observation the two disease groups showing clear excess mortality across all three dockyards are cancers of the peritoneum and pleura, the two forms of mesothelioma. For pleural mesothelioma the following unadjusted SMRs are obtained: 1983 (1505-2461), 1638 (1049-2437), and 1042 (693-1506); based on 66, 24 and 28 observed deaths [$X^2 = 8.4$, $P < 0.025$]. For peritoneal mesothelioma we have the following: 731 (351-1344), 338 (41-1222), and 99 (2-551); based on 10, 2 and one death, respectively [$X^2 = 5.9$, $0.05 < P < 0.1$]. Stomach cancer showed a slight excess. Here the adjusted SMRs were: 145 (113-176), 122 (81-162), and 95 (68-123) [$X^2 = 5.2$, $0.05 < P < 0.1$]. Mortality due to lung cancer is not seen to be in excess. The three dockyards show the following regionally adjusted lung cancer ratios: 99 (87-112) at Devonport, 85 (70-101) at Chatham, and 94 (81-106) at Portsmouth [$X^2 = 1.8$, $P > 0.1$]. Lung cancer appears to be neither significantly in excess or in deficit at these dockyards, but would appear to be similar with the background population rate. Whereas, both forms of mesothelioma and asbestosis show potentially a large excess risk.

Regional adjustments were not available for the three disease groups that at this point show a clear excess mortality - namely asbestosis, and mesothelioma of both the pleura and peritoneum. By examination of the other disease groups it is

apparent that any adjustment applied would very probably increase the calculated SMR. Only in four disease groups at Devonport and Portsmouth and in three at Chatham were the SMRs reduced by the regional adjustment. However, regarding asbestosis and the two forms of mesothelioma, would any form of regional adjustment be appropriate? Would this be comparing like with like? These are rare diseases and it is likely that the majority of reported cases in OPCS 'coastal' regions (here the south west and south east regions) are concentrated around dockyard conurbations. If this is the case no regional adjustment should be undertaken. For lung cancer, the other 'known' asbestos related disease, a regional adjustment is both available and used. This has its main affect at Devonport dockyard, taking the lung cancer SMR from 80 (70-90) to 99 (87-112), i.e. taking the ratio from a statistically significant deficiency to being no different from that expected for the south west regional population. This affect is also seen at Chatham dockyard. Since lung cancer is a relatively more 'common' disease, the regional adjustment was judged to be required.

When considering the 50 disease groups there is slight evidence of a difference in mortality between the dockyards for certain diseases. Possibly, with the mortality rates at Devonport being higher than Chatham, which in turn may be higher than Portsmouth. However, the variation in SMRs across dockyard is not consistent over all groups, and the pattern of disease for each dockyard is very similar. As a footnote to this section, mortality due to accidents, poisoning and violence was significantly reduced in all three dockyards, as was disease of the digestive system.

4.3 Non-responder mortality.

The fifty disease groups were also scrutinised for those who were absolute non-responders in the cross-sectional surveys. The results are given in tables A3.1 to A3.3. From these tables, it is readily apparent that the non-responders experienced higher overall mortality rates than their colleagues who responded. The former were 7-41% higher than expected from the general population. The all-cause SMR being 141 (125-157) for Devonport, 126 (112-140) for Chatham, and 107 (98-116) at Portsmouth. With regional adjustment these become: 156 (138-173), 135 (120-149) and 114 (104-124) [$X^2 = 19.6$, $P < 0.001$]. The SMRs all being elevated, and generally significantly so (i.e. they are unlikely to be chance statistical findings).

For all-neoplasms, the regionally adjusted SMRs are: 143 (111-176), 130 (104-157), and 125 (106-144) [$X^2 = 1.0$, $P > 0.1$]. Again all statistically elevated from 100. In the case of lung cancer, the SMRs are all in excess of 100, but only Portsmouth reaches statistical significance. The lung cancer SMRs are: 142 (94-207), 121 (78-161), and 139 (106-169) [$X^2 = 0.6$, $P > 0.1$]. Pleural mesothelioma follows the same general pattern for non-responders and responders, with a clear statistical excess appearing at all yards. The SMRs are: 2917 (1070-6349), 1558 (425-3989), and 907 (294-2117); based on 6, 4 and 5 deaths, respectively [$X^2 = 4.1$, $P > 0.1$]. Peritoneal mesothelioma is seen only at Devonport and Portsmouth: 1076 (27-5993) and 457 (12-2544); based on one case at each yard [$X^2 = 0.4$, $P > 0.1$].

This picture of excess mortality, occasionally reaching a statistical excess, is generally observed throughout the disease groups of all-neoplasms, diseases of the circulatory system, and diseases of the respiratory system. It is very apparent for tuberculosis and pulmonary fibrosis, though based on low numbers of observed deaths. The tuberculosis SMRs are: 363 (9-2022), 407 (49-1469), and 109 (3-609) for 4 cases in total [$X^2 = 1.4$, $P > 0.1$]. A single case of pulmonary fibrosis is seen at each dockyard, producing the following SMRs: 404 (10-2248) at Devonport,

333 (8-1856) at Chatham, and 172 (4-956) at Portsmouth [$X^2 = 0.4$, $P > 0.1$]. No cases of asbestosis were reported. No single cause of death was seen to have significantly reduced SMRs.

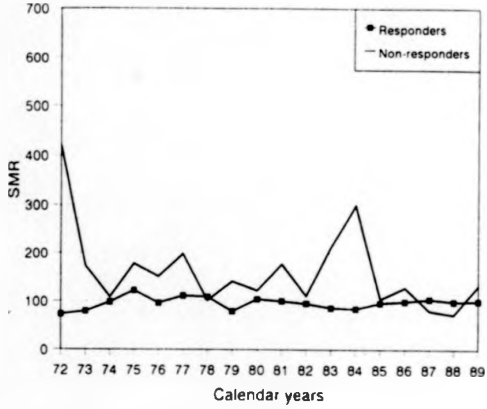
This suggests that an element of the non-responders may have been chronically ill and deselected themselves out of the initial surveys on health grounds. If this hypothesis is correct the non-responder mortality rates may be expected to be high at study definition, declining to 'normal' levels (i.e. responder levels) as follow-up continued. To explore this, non-responder mortality rates were initially examined by period of follow-up for the 12 specific causes of interest given in table 3.18. Table 4.4 gives these results.

From table 4.4 the largest SMRs generally appear to occur in the early years of follow-up (i.e. in either the group < 5 years or the group of 5-9 years of follow-up). There is an indication of significant differences in the SMRs over follow-up period at Devonport and Portsmouth, however, at Chatham the rate appears constant. For example, considering all-cause mortality, at Devonport a chi-squared test of the difference between SMRs approached statistical significance [$X^2 = 5.5$, $0.05 < P < 0.1$], likewise at Portsmouth [$X^2 = 4.6$, $P = 0.1$]. However, at Chatham no difference was seen [$X^2 = 0.05$, $P > 0.1$]. For all-neoplasms only the difference between SMRs at Portsmouth was statistically significant [$X^2 = 6.5$, $P < 0.05$]. For diseases of the respiratory system, the difference in SMRs at Devonport [$X^2 = 6.3$, $P < 0.05$] and Portsmouth [$X^2 = 9.3$, $P < 0.01$] were both significant. The large change in SMR at Devonport for pleural mesothelioma was also significant [$X^2 = 6.5$, $P < 0.03$], but was based on only 6 cases.

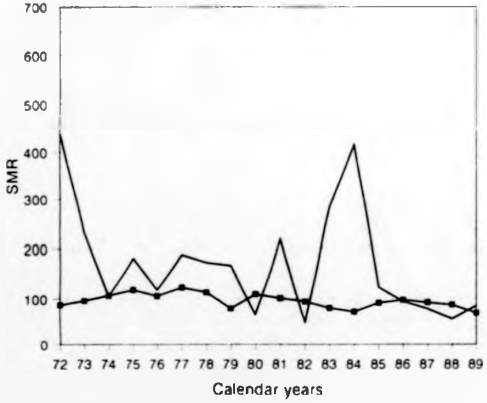
TABLE 4.4: Cause specific mortality by follow-up period and dockyard for initial study non-responders.*

Causes of Death	Follow-up period (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	<5	88	193	153-234	78	137	107-168	126	126	104-148
	5-9	81	146	115-178	94	135	108-163	130	98	81-114
	10+	125	142	117-167	148	133	112-154	276	118	104-132
All Neoplasms	<5	26	205	134-301	22	132	83-201	49	167	120-213
	5-9	18	117	70-185	30	147	99-209	39	98	67-129
	10+	31	128	83-172	39	119	82-157	85	123	96-149
Ca. Stomach	<5	4	319	87-816	2	124	15-449	7	251	101-518
	5-9	1	71	2-397	3	168	35-491	4	118	32-301
	10+	2	103	12-370	7	280	112-577	6	114	42-248
Ca. Peritoneum	<5	1	3235	82-18018	0	-	0-10121	0	-	0-5474
	5-9	0	-	0-12467	0	-	0-10719	0	-	0-5541
	10+	0	-	0-11381	0	-	0-9500	1	1178	30-6560
Ca. Lung	<5	9	177	81-337	10	137	66-251	19	148	89-231
	5-9	8	137	59-271	13	150	80-257	19	114	69-179
	10+	10	123	59-226	11	90	45-161	39	151	103-198
Ca. Pleura	<5	4	9344	2546-23921	1	1826	46-10171	3	2830	584-8272
	5-9	2	3653	442-13188	2	2975	360-10741	0	-	0-2671
	10+	0	-	0-3412	1	742	19-4134	2	651	79-2351
Circulatory System	<5	45	194	138-251	42	154	107-200	52	108	79-137
	5-9	47	162	115-208	46	135	96-174	65	99	75-124
	10+	71	156	120-192	75	140	108-172	123	109	90-129
Pulmonary Circulation	<5	2	868	105-3135	0	-	0-1239	0	-	0-709
	5-9	2	512	62-1850	0	-	0-716	1	98	2-547
	10+	1	239	6-1331	1	197	5-1097	0	-	0-377
Respiratory System	<5	13	293	156-502	10	161	77-296	22	214	134-325
	5-9	10	179	86-328	14	177	97-296	11	75	38-135
	10+	9	103	47-195	15	121	68-199	30	120	81-172
Bronchitis, Emphysema and Asthma	<5	2	90	11-325	3	91	19-265	8	148	64-292
	5-9	4	178	49-457	2	59	7-213	3	49	10-144
	10+	2	80	10-287	8	205	89-404	8	102	44-201
Asbestosis	<5	0	-	0-65776	0	-	0-48884	0	-	0-25250
	5-9	0	-	0-43654	0	-	0-35608	0	-	0-17704
	10+	0	-	0-25513	0	-	0-20942	0	-	0-9634
Pulmonary Fibrosis	<5	0	-	0-5175	0	-	0-4287	0	-	0-2473
	5-9	0	-	0-5683	1	1328	34-7398	0	-	0-2723
	10+	1	896	23-4992	0	-	0-2660	1	336	0-1871

* The quoted SMRs from this table onwards will, where appropriate, be with regional adjustment.



a) All Cause Mortality



b) Circulatory System Mortality

FIGURE 4.1: Standardised mortality ratios by calendar year period for Devonport dockyard.

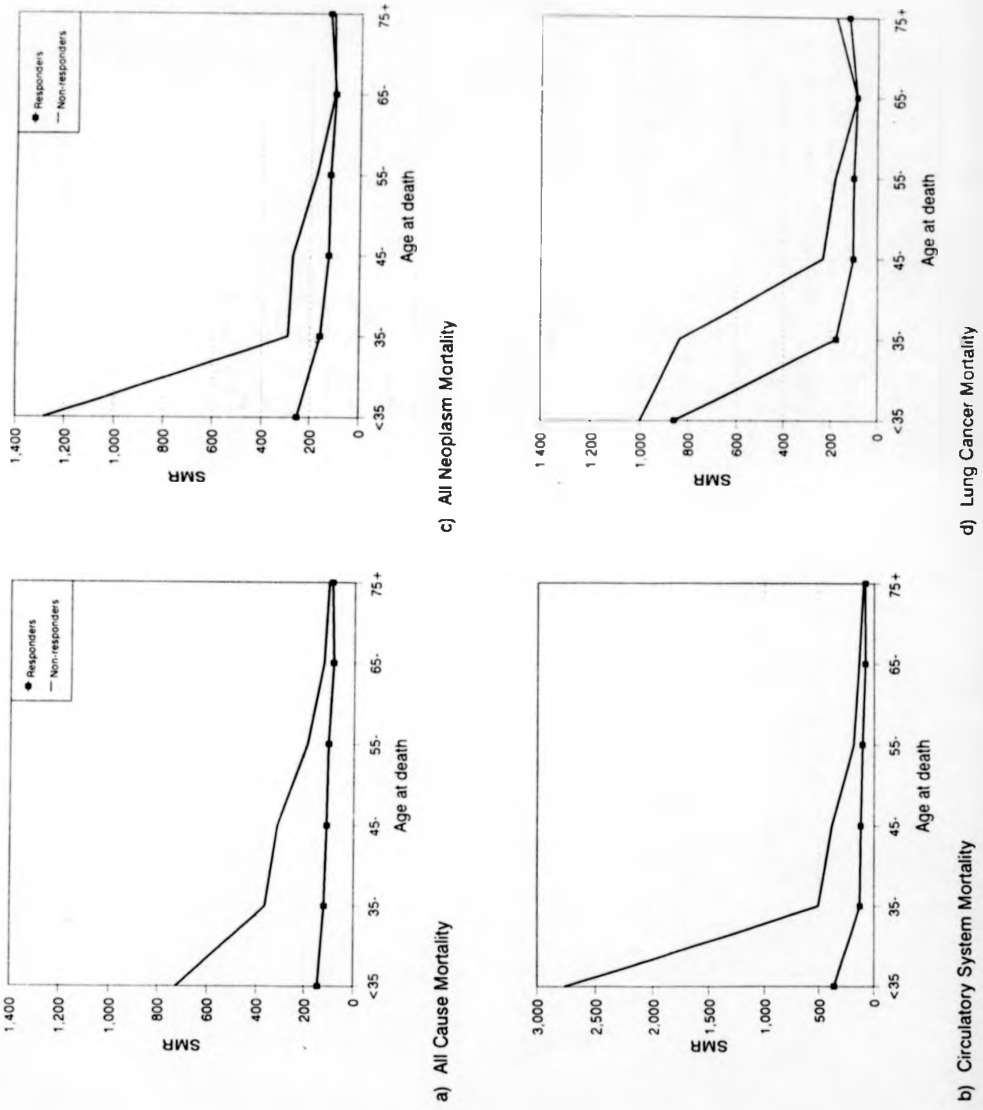
c) All Neoplasm Mortality

d) Lung Cancer Mortality

Annual patterns of mortality for both responders and non-responders, over the 17 year follow-up period, were considered next (these are given in tables A3.4 to A3.6). These clearly illustrate, for each dockyard, a 'fading-away' affect in the non-responder mortality rate with time. Figure 4.1 demonstrates this affect graphically for Devonport dockyard for the disease groups of: all-causes, all-neoplasms, lung cancer, and diseases of the circulatory system. For all-causes a chi-squared test of difference in SMRs for study responders was significant [$X^2 = 28.9$, $P < 0.05$] and a test of a decreasing trend in their SMRs was non-significant [X^2 for trend $= 0.01$, $P > 0.1$], the non-responders produced both a significant difference and trend [$X^2 = 71.1$, $P < 0.001$; X^2 for trend $= 10.3$, $P < 0.005$]. For all-neoplasms the responders produced non-significant results in both tests [$X^2 = 18.7$, $P > 0.1$; X^2 for trend $= 1.2$, $P > 0.1$], and again the non-responders gave significant results [$X^2 = 33.6$, $P < 0.01$; X^2 for trend $= 9.6$, $P < 0.005$]. It may be concluded from these results and observation of figure 4.1, that the responders do indeed represent a baseline level of mortality that the initial surveys non-responders slowly approach over time. These tables and figures then give some evidence to support the idea of certain non-responders being chronically ill at study definition and in turn succumbing to disease and death in the first years of follow-up, the overall non-responder mortality rate then reducing to responder levels.

Age effects on non-responder mortality were considered next. The only information available concerning the age of non-responders was their date of death. Accordingly, mortality rates by age at death are compared for responders and non-responders (these are given in tables A3.7 and A3.8). From these tables there appears to be a slight overall decline in SMR with age at death for study responders and a more substantial decline for non-responders. The rate of decline is affected by the relatively low numbers of observed deaths at the younger ages producing the highest SMRs. Figure 4.2 illustrates this point. After omitting the youngest group (< 35 years of age) the decline in SMR for non-responders was generally still very significant ($P < 0.01$). This again gives support to the idea of

FIGURE 4.2: Standardised mortality ratios by age at death for Devonport dockyard.



certain non-responders being chronically ill, producing much higher than expected SMRs at younger ages in comparison to the responder SMRs.

It is possible that the overall excess mortality rates found in the non-responder group may explain, in part, the deficiency seen in section 4.2 for the study responders. To examine this the following three disease groups were considered: all-causes, all-neoplasms, and lung cancer. By combining the responder and non-responder observed and expected mortality at each dockyard table 4.5 was produced.

TABLE 4.5: Responder and Non-responder mortality for the disease groups: All-Causes, All-Neoplasms and Lung Cancer.

	Dockyard	Obs	SMR	95% CI
All Causes.	Devonport:	2583	101	97-105
	Chatham:	1365	99	93-104
	Portsmouth:	2224	93	89- 97
			[x^2=8.6, P<0.025]	
All Neoplasms.	Devonport:	864	119	111-127
	Chatham:	456	111	101-121
	Portsmouth:	748	103	96-111
			[x^2=8.4, P<0.025]	
Lung Cancer.	Devonport:	268	102	90-114
	Chatham:	150	91	77-106
	Portsmouth:	295	103	91-114
			[x^2=1.7, P>0.1]	

From this table it is clear that an 'all-worker' study would not have generally found a deficit mortality rate in these disease groups. The all-cause SMRs being more or less 100, the all-neoplasm SMRs being raised above 100 and showing no statistical deficit. The lung cancer SMRs show no signs of either a significant excess or deficit in mortality. It is possible that any healthy worker effect to be seen among the study responders may be overshadowed by the absolute deselection effect of the non-responders.

It should be noted that the non-responders had poor OPCS trace rates. In total 527 non-responders from the three yards could not be traced; by dockyard this was: 131 (13.4% of the non-responders) at Devonport, 72 (7.9%) at Chatham, and 324 (14.8%) at Portsmouth*. This is likely to be an artifact of the initial surveys follow-up requirements (non-responders being invited to attend only once more after initial failure to attend), and a problem of definition of the study population. It has the effect of reducing the number of non-responders analysed in this section to 3,670 workers (i.e. to only those non-responders who were successfully flagged at OPCS).

4.4 Cause specific mortality according to questionnaire and x-ray type.

Before progressing to a more detailed analysis of mortality, the study responders are briefly inspected according to questionnaire and x-ray type. Tables A3.9 and A3.10 give the results of this. In these tables the mortality rates of workers replying to the self-administered (free) questionnaire are compared with those responding to both the free and doctor-administered questionnaire, and against those not supplying a questionnaire. For x-ray type, workers with only small x-rays are compared to those with both small and large x-ray, and to those with no x-ray.

By inspection the mortality rates by questionnaire type at Chatham dockyard appear reasonably constant; when tested all differences were non-significant ($P > 0.1$). At Devonport significant differences were found in the SMRs for all-cause mortality [$X^2 = 9.1$, $P < 0.025$], all-neoplasm mortality [$X^2 = 11.7$, $P < 0.005$], mortality due to lung cancer [$X^2 = 10.0$, $P < 0.01$], and pleural mesothelioma [$X^2 = 13.2$, $P < 0.005$]. Similar results were observed for Portsmouth. Generally the pattern of mortality over questionnaire type at Devonport and Portsmouth is variable, no one type being seen to have consistently

* At Rosyth 666 (43.3%) of the non-responders were untraced.

FIGURE 4.3: Standardised mortality ratios by employment-time variables.

higher mortality rates. This variability of rate is also observed over x-ray type for all three dockyards. From this it could be concluded that the questionnaire and x-ray non-responders (i.e. the groups designated 'neither' in tables A3.9 and A3.10) generally experienced similar mortality rates to the questionnaire and x-ray responders (the groups entitled either 'free' and 'both' in table A3.9 or 'small' and 'both' in table A3.10).

The majority of the following analyses will exclude those workers with no questionnaire information. These workers are not initial survey 'absolute non-responders' since they all supplied x-ray information. However, no employment, medical history, or asbestos exposure information was available for these subjects. This reduces the number of observed deaths in the bulk of the following analyses from 5,026 to 4,373.

4.5 Cause specific mortality according to employment-time variables.

This section is concerned with a description of mortality over certain employment variables. These variables are all time related and consist of the following: time since start of employment, length of service, year of start of employment, and age at start of employment. Mortality related to these variables is cited when there are sufficient deaths to allow sensible analysis.

When analysed according to age, the patterns in each dockyard were similar (figure 4.3), with the SMRs generally appearing to be reasonably constant over recruitment age. The results are given in table A3.11; these are illustrated, for Devonport only, in figure 4.4*. For the 12 causes of death analysed, the SMRs commonly showed little change over age at start of employment group, chi-squared tests of a difference in SMRs were mostly non-significant ($P > 0.1$). The

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* Throughout, SMR histograms are presented taking Devonport as a representative dockyard.

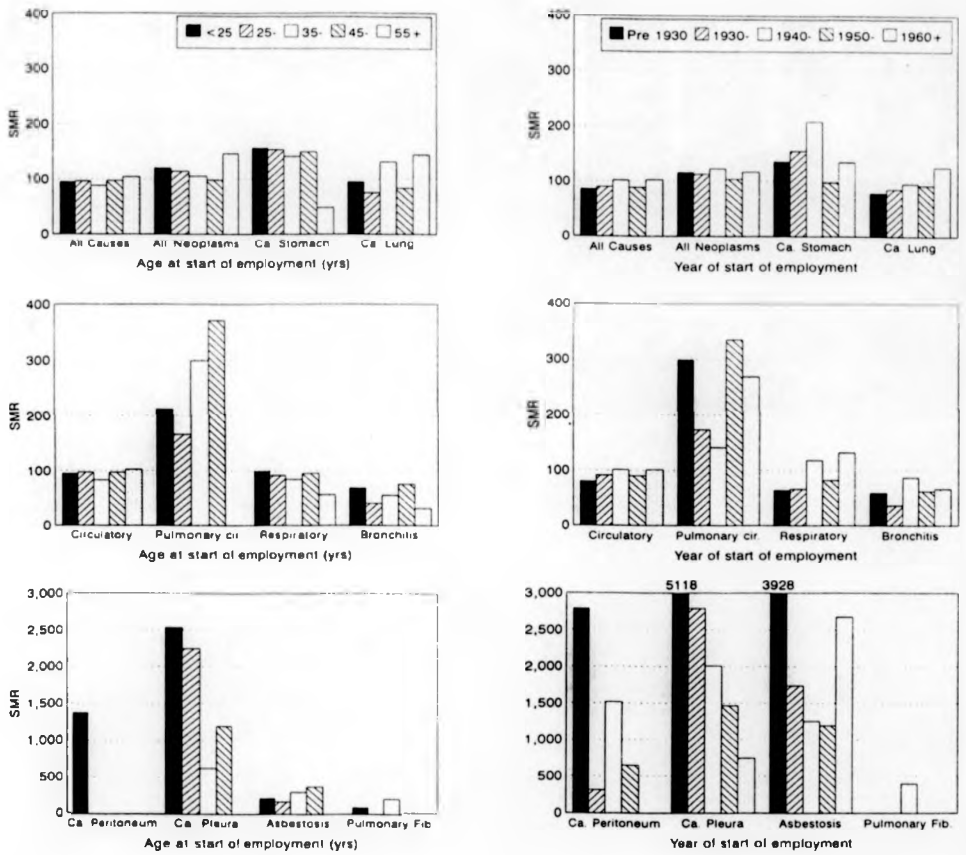

FIGURE 4.4: Standardised mortality ratios by age at start of employment and year of start of employment for Devonport dockyard.

exception to this is lung cancer and pleural mesothelioma both of which approached a statistically significant difference in SMR with age [$X^2 = 9.4$, $P = 0.05$ and $X^2 = 8.2$, $0.05 < P < 0.1$, at Devonport], lung cancer appearing to increase, while pleural mesothelioma appears to decrease with age. Pleural mesothelioma also showed a significant trend in its decreasing SMR [X^2 for trend $= 4.4$, $P < 0.05$]. Both forms of mesothelioma and asbestosis continue to show a clear excess mortality. However, as noted before, the peritoneal mesothelioma and asbestosis SMRs are based on low numbers (similarly for pulmonary fibrosis).

Disease of pulmonary circulation shows a marked affect in table A3.11 and figure 4.4; with high recorded SMRs over age at start of employment group, the SMRs appearing to increase with age. However, this increase is statistically non-significant [e.g. $X^2 = 2.2$, $P > 0.1$, at Devonport]. As mentioned previously this disease group was formed as a surrogate for *cor pulmonale*, which has been considered associated with both lung disease and asbestos exposure.[3] It is possible that overly low estimates of expected numbers has resulted in these huge SMRs with their accompanying wide standard errors (producing chi-squared tests of a difference in SMRs that are non-significant). For this reason, disease of pulmonary circulation will not be considered further, however, it will continue to be tabulated in the appendix tables and illustrated in the forthcoming figures.

Calendar year of employment is considered next (figures 4.3, 4.4 and table A3.12). The associations seen are in slight contrast with those for age at start of employment. Overall, statistical differences are not seen in SMR across calendar period, however, lung cancer, pleural mesothelioma and diseases of the respiratory system are exceptions to this (generally, $P < 0.05$). Another exception is all-cause mortality at Devonport dockyard [$X^2 = 10.4$, $P < 0.05$]. As with age at recruitment a trend in decreasing pleural mesothelioma SMR is seen at Devonport [X^2 for trend $= 14.7$, $P < 0.001$]. The highest SMRs for this disease all occurring before World War II. Mesothelioma SMRs prior to 1930 were: 5118 (2207-10082), 1424 (36-7933), and 5072 (1643-11838); based on 14 deaths. Peritoneal mesothelioma

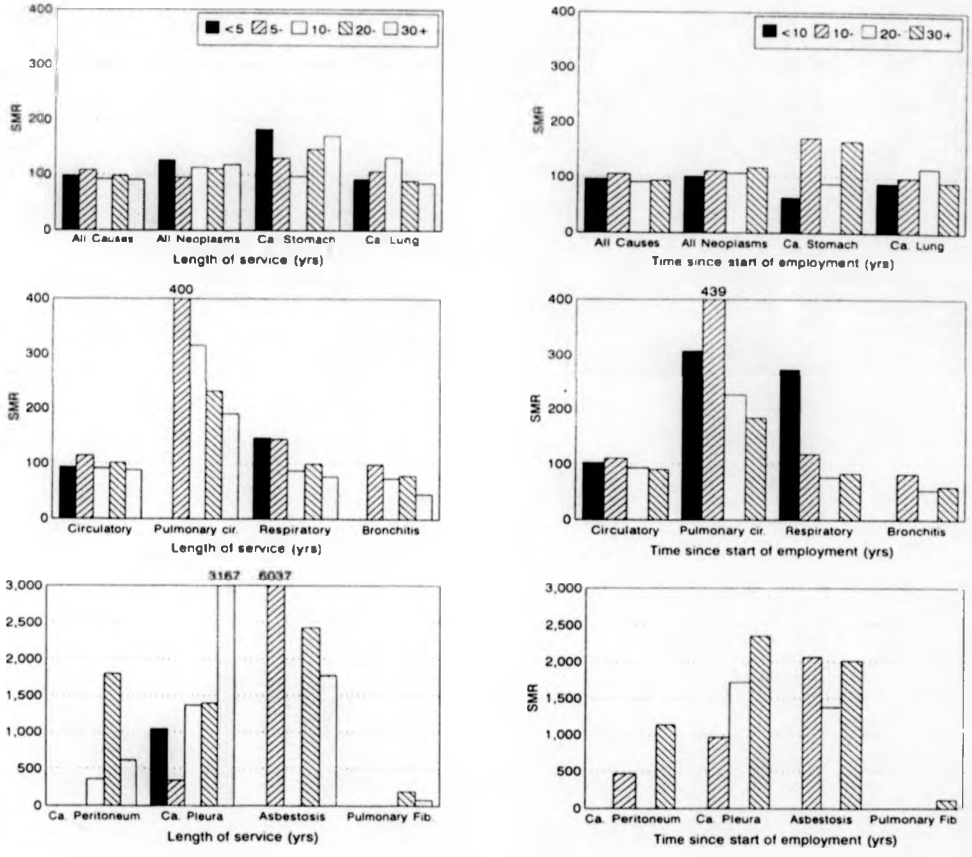

FIGURE 4.5: Standardised mortality ratios by length of service and time since start of employment for Devonport dockyard.

in the same period produced SMRs of 2789 (338-10070) at Devonport and 3206 (81-17857) at Chatham; based on 3 deaths. An increasing trend is also seen at Devonport for diseases of the respiratory system [X^2 for trend = 8.4, $P < 0.005$].

Few notable patterns emerged when SMRs were analysed according to length of service (figure 4.5 and table A3.13). However, an increasing trend in SMR is seen for pleural mesothelioma (generally, $P < 0.01$). By observation of table A3.13 there was a tendency for SMRs to be lowest for individuals employed for 30 years or more. For these workers mortality due to all-causes, diseases of the circulatory and respiratory systems and bronchitis was seen to be significantly reduced from that expected in the regional population for Devonport and Portsmouth.

When analysing mortality by time since first employment (figure 4.5 and table A3.14) only respiratory system diseases showed a significant trend, indicating a decreasing SMR over time [$X^2 = 23.4$, $P < 0.001$ at Devonport, and $X^2 = 20.9$, $P < 0.001$ at Portsmouth]. This decreasing trend was significant only at Devonport [X^2 for trend = 12.7, $P < 0.001$]. A slight deficiency in mortality is seen at more than 30 years since first dockyard employment. This is again for all-causes, diseases of the circulatory and respiratory systems and bronchitis. However, apart from diseases of the respiratory system no other disease group gave any evidence of a systematic increase or decrease in SMR over time since first employment. SMRs for all causes of death combined for the three dockyards were very similar throughout the period of follow-up (figure 4.3), generally being just below 100 and non-significant.

SMRs showed no obvious pattern when time since first employment was analysed by length of service stratum (table 4.6 gives the results for all-cause mortality, tables A3.15 and A3.16 for lung cancer and pleural mesothelioma). There is only a very slight hint that the lowest SMRs fall on the main diagonal of these tables; lowered SMRs on this diagonal are a feature common in studies showing the healthy worker effect. The empty cells to the right of the diagonal would have

referred to individuals who had left dockyard employment (in this study this would be before the enumeration dates), a group whose mortality patterns are unavailable in this study. This is another factor that may aid in obscuring a healthy worker effect and assist in producing the patterns seen at the three dockyards of little, if any, overall excess or deficiency of mortality.

Only for those workers with 30 or more years of service and 40 plus years since first potential exposure were effects being seen with significant declines in all-cause SMR showing at both Devonport and Portsmouth. Chatham dockyard had no result that was either in excess or significantly reduced. No obvious trends in mortality pattern were seen. This result is generally repeated for lung cancer. However, there is an indication of excess mortality occurring at long intervals from first exposure, i.e. for those workers with less than 20 years employment who had first been employed over 30 years ago, with an SMR of 199 (114-324) at Devonport. In fact for those workers with between 10 and 19 years of employment an increasing lung cancer SMR was observed at Devonport with time since first exposure. The SMRs were: 104 (52-186) for 10-19 years since first exposure, 122 (79-164) for 20-29 years, and 199 (114-324) for 30-39 years. However, this was not a statistically significant trend [$X^2 = 3.5$, $P > 0.1$ and X^2 for trend $= 2.8$, $P > 0.1$]. This pattern, also with a non-significant trend, was further observed for those workers with between 20 and 29 years of employment at Chatham.

When considering pleural mesothelioma the numbers become very small for Chatham and Portsmouth (table A3.16), however, it is clear that there is a large excess mortality at all three dockyards for this condition clustered among those workers with long follow-up and long duration of service. For the longest period of service and follow-up the following SMRs are observed: 3337 (2215-4459) at Devonport, 3080 (1725-5080) at Chatham, and 1756 (959-2946) at Portsmouth; based on 34, 15 and 14 deaths respectively. No clear trends are seen in this excess mortality, the SMRs being very variable and singly highly significant. At Chatham one mesothelioma death occurred for a worker within 10 years of first

TABLE 4.6: All cause mortality by length of service, time since first exposure, and dockyard.

Length of service (yrs)	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
<5	51	116 (84-148)	73	86 (67-106)	4	165 (45-422)	-		-	
5 - 9	20	68 (41-105)	143	119 (99-138)	61	105 (78-131)	-		-	
10 - 19	-		108	105 (85-125)	223	87 (75- 98)	81	94 (74-115)	-	
20 - 29	-		-		109	91 (74-108)	257	97 (85-109)	65	120 (91-149)
30+	-		-		-		167	95 (81-110)	721	90 (83- 97)
					CHATHAM					
<5	17	75 (44-121)	38	79 (54-105)	1	39 (1-218)	-		-	
5 - 9	7	65 (26-135)	43	87 (61-114)	22	89 (56-135)	-		-	
10 - 19	-		14	61 (33-103)	57	93 (69-118)	16	69 (40-112)	-	
20 - 29	-		-		33	88 (58-118)	96	92 (73-110)	25	86 (55-126)
30+	-		-		-		66	88 (66-109)	385	95 (85-104)
					PORTSMOUTH					
<5	26	96 (63-140)	43	79 (56-103)	3	173 (36-507)	-		-	
5 - 9	6	50 (18-108)	54	104 (76-132)	23	88 (56-133)	-		-	
10 - 19	-		49	84 (61-108)	143	95 (79-111)	41	91 (63-119)	-	
20 - 29	-		-		87	91 (72-110)	188	78 (67- 90)	58	87 (65-110)
30+	-		-		-		108	103 (83-122)	502	81 (74- 88)

TABLE 4.7: All cause mortality by year of start of employment, time since first exposure, and dockyard.

Year of Start	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
Pre 1930	-		-		-		-		329	86 (72- 99)
1930-	-		-		-		91	81 (64- 97)	493	92 (84-100)
1940-	-		-		53	107 (78-136)	237	102 (89-115)	144	99 (83-115)
1950-	-		44	93 (66-121)	214	84 (72- 95)	175	96 (82-111)	-	
1960+	71	96 (74-118)	280	107 (95-120)	132	99 (82-116)	-		-	
					CHATHAM					
Pre 1930	-		-		-		-		74	91 (70-111)
1930-	-		-		-		56	102 (75-129)	281	97 (86-109)
1940-	-		-		15	75 (42-124)	76	78 (61- 96)	55	85 (62-107)
1950-	-		4	42 (11-108)	58	95 (70-119)	46	90 (64-116)	-	
1960+	24	72 (46-107)	91	82 (65- 99)	40	90 (62-117)	-		-	
					PORTSMOUTH					
Pre 1930	-		-		-		-		97	86 (69-103)
1930-	-		-		-		61	105 (78-131)	331	78 (70- 87)
1940-	-		-		28	70 (46-101)	176	87 (74- 99)	132	86 (72-101)
1950-	-		11	49 (24- 87)	136	90 (75-105)	100	78 (62- 93)	-	
1960+	32	81 (53-109)	135	95 (79-111)	92	110 (88-133)	-		-	

TABLE 4.8: All cause mortality by age at start of employment, time since first exposure, and dockyard.

Age at start (yrs)	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
<25	9	78 (36-149)	46	134 (95-173)	79	108 (84-132)	192	93 (80-107)	613	92 (85- 99)
25-	1	31 (1-173)	28	144 (95-207)	71	93 (71-114)	181	100 (85-114)	161	90 (76-104)
35-	7	83 (34-172)	58	89 (66-113)	138	83 (69- 97)	117	94 (77-111)	12	106 (55-185)
45-	26	90 (59-132)	147	105 (88-122)	99	88 (71-106)	13	93 (50-159)	-	
55+	28	127 (85-184)	45	89 (63-115)	12	126 (65-220)	-		-	
					CHATHAM					
<25	3	80 (17-235)	9	79 (36-149)	16	71 (40-115)	72	95 (73-117)	299	91 (80-101)
25-	-		3	59 (12-173)	18	83 (49-131)	68	83 (63-103)	106	105 (85-125)
35-	3	107 (22-312)	15	92 (51-151)	46	116 (82-149)	37	91 (61-120)	5	142 (46-331)
45-	12	117 (60-204)	37	82 (56-109)	23	73 (46-110)	1	27 (1-153)	-	
55+	6	39 (14- 84)	31	74 (48-100)	10	96 (46-177)	-		-	
					PORTSMOUTH					
<25	3	62 (13-181)	15	97 (54-160)	33	80 (52-107)	124	88 (73-104)	436	80 (72- 87)
25-	1	60 (2-334)	7	76 (30-156)	51	107 (78-137)	130	95 (78-111)	110	84 (68-100)
35-	4	93 (25-238)	38	104 (71-137)	108	95 (77-113)	73	72 (55- 88)	14	105 (58-177)
45-	11	75 (38-135)	62	85 (64-107)	62	93 (70-116)	10	99 (48-182)	-	
55+	13	93 (49-159)	24	78 (50-117)	2	40 (5-146)	-		-	

dockyard exposure (the worker had less than 5 years dockyard employment). No previous occupational history was available for this worker.

The same picture is seen when considering time since first employment by period of first employment (tables 4.7, A3.17 and A3.18). No clear trends emerge from these tables. All-cause mortality together with lung cancer mortality is not seen to be in significant excess or deficit. Pleural mesothelioma, though again suffering from low numbers, can be seen to be generally in great excess, concentrating around those workers with long follow-up, having been first employed before World War II.

When examining age at start of employment strata over time since first employment period a problem arises. These factors are correlated, i.e. there are more recent recruits among younger workers than among older workers, etc. This problem will be addressed by the use of regression modelling (section 4.8). However, by inspection no obvious excess or deficit is seen for lung cancer and all-cause mortality (tables 4.8 and A3.19). Pleural mesothelioma (table A3.20) again shows an excess, mainly for those workers employed at a young age over 30 years ago.

4.6 Cause specific mortality according to health-status variables.

Under this general heading the following factors will be considered: personal medical history, i.e. the prevalence of cough, phlegm, breathlessness and chest-illness, smoking habits and smoking history, and also x-ray grouping. This last category, x-ray group, is considered here since in its grouped form this also represents a health status variable, i.e. the presence or absence of pulmonary fibrosis, tuberculosis, etc. In this sense, x-ray group and the personal medical symptoms may be considered predictive variables of disease outcome.

Cough is examined in table A3.21. From this table it is reasonable to conclude that there is no overall difference seen in this possible symptom of asbestos related

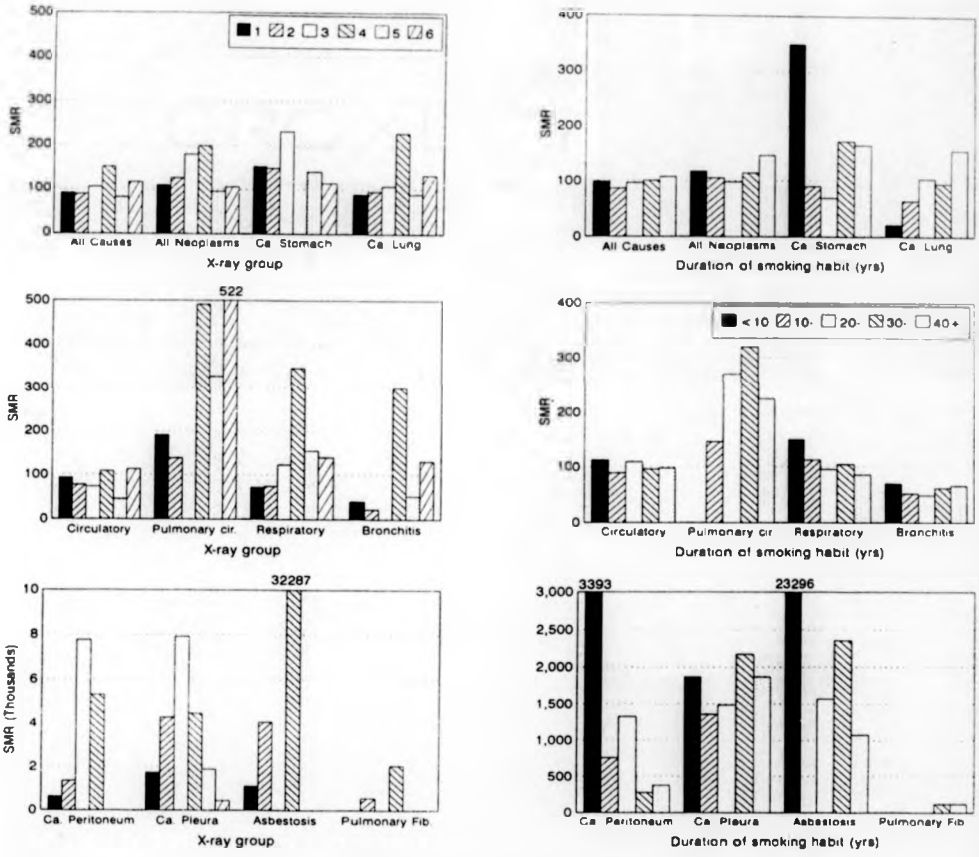

FIGURE 4.6: Standardised mortality ratios by x-ray group and duration of smoking habit for Devonport dockyard.

respiratory illness over the dockyards. For all-cause mortality, the SMRs in each dockyard are significantly in excess for those workers responding positively to the question 'do you usually cough during the day or night at work?'. However, there is no significant difference between the dockyards ($P > 0.1$). The SMRs are: 121 (111-130), 119 (103-135), and 127 (115-139). For those workers responding 'No' to the above question the following significantly reduced SMRs are obtained: 88 (83-93), 83 (76-89), and 73 (68-78). Not surprisingly, there are highly significant differences between the SMRs in the different response-type groups, i.e. between the yes or no response, (commonly, $P < 0.01$). This pattern generally holds across all 12 disease groups. However, an obvious exception is for diseases of the circulatory system. Here the difference over response type has borderline significance at Devonport [$X^2 = 4.0$, $0.05 < P < 0.1$], non-significance at Chatham [$X^2 = 0.6$, $P > 0.1$] and significance at Portsmouth [$X^2 = 15.3$, $P < 0.01$].

The patterns seen for cough are also generally observed for phlegm, breathlessness and chest-illness (tables A3.22 to A3.24). The significant and non-significant results are seen to be very variable over disease group and dockyard, with the likelihood of these being chance findings varying considerably. From tables A3.21 to A3.24 it is reasonable to conclude that the four medical symptoms may be used as predictive variables for lung disease and possibly asbestos related lung disease. Accordingly, these symptoms will be used in the following log-linear modelling.

As expected no obvious patterns or trends emerge over x-ray group, the grouping being a nominal scale (figure 4.6 and table A3.25). The 'normal' x-ray group, group 1, has significantly low overall SMRs for each dockyard: 91 (86-95), 85 (79-91), and 80 (75-85); the variation over dockyard being significant [$X^2 = 9.8$, $P < 0.01$]. Group 4, the pulmonary fibrosis group, generally has significantly raised SMRs. For all-cause mortality the SMRs for group 4 are: 151 (112-191) at Devonport, 125 (77-191) at Chatham, and 151 (101-218) at Portsmouth; with no variation showing across dockyard [$X^2 = 0.6$, $P > 0.1$]. Not surprisingly the highest SMRs for this x-ray group are found in the disease groups of asbestosis

(with an SMR of: 32287 (3919-116917) at Devonport), pulmonary fibrosis (with SMRs of: 2049 (52-11413) and 4637 (117-25829) at Devonport and Chatham, respectively), and the two forms of mesothelioma. Peritoneal mesothelioma occurring only at Devonport with an SMR of 5303 (134-29539), pleural mesothelioma occurring at Devonport and Portsmouth with SMRs of 4456 (539-16085) and 4405 (111-24538). These are all based on very low numbers of deaths. The very high SMR for asbestosis at Devonport dockyard being based upon two reported cases. For lung cancer the following high SMRs were reported: 227 (104-430) at Devonport, 142 (29-415) at Chatham, and 375 (171-711) at Portsmouth. In absolute terms these lung cancer SMRs are smaller than those reported above, but it should still be noted that there is a clear excess risk being reported at Devonport and Portsmouth.

High SMRs are seen for subjects with pleural calcification, i.e. for x-ray group 3, for peritoneal and pleural mesothelioma. Peritoneal mesothelioma being seen only at Devonport in this x-ray group and producing an SMR of 7761 (939-28016), based on two cases. Pleural mesothelioma is seen at all three dockyards with the following SMRs: 7910 (2563-18463), 7894 (955-28497), and 1760 (45-9805); based on 5, 2, and one death respectively. No deaths occurred, as anticipated, in this x-ray group from asbestosis and pulmonary fibrosis. For lung cancer the following non-significant SMRs occurred: 107 (39-234), 110 (23-323), and 52 (11-152).

Excessive SMRs are also seen for x-ray group 2, the workers with pleural thickening. These centre around pleural mesothelioma and asbestosis. For Pleural mesothelioma the following SMRs were produced: 4277 (1844-8426) at Devonport, 2137 (258-7693) at Chatham, and 3726 (1607-7339) at Portsmouth; based on 18 deaths in total. For asbestosis an SMR of 4060 (107-22614) was obtained at Devonport; based on one death. Lung cancer was again seen to be neither in excess or deficit with SMRs of: 95 (52-159), 64 (24-140), and 115 (73-173).

High lung cancer SMRs are generally seen only for x-ray groups 3, 4 and 6; group 6 being a general catchment group of 'all other abnormalities'. Lung cancer SMRs for this group were: 132 (86-195), 137 (75-230), and 123 (81-181). Pleura mesothelioma was reported with the following SMRs: 44 (11-2473), 3138 (647-9172), and 941 (114-3398); based on 6 deaths. There were no cases of asbestosis or peritoneal mesothelioma in group 6. Generally x-ray group 5, those workers with tuberculosis, gave non-elevated SMRs. Apart from pleura mesothelioma with SMRs of: 1894 (391-5538) at Devonport, no deaths at Chatham, and 1264 (32-7041) at Portsmouth. The SMRs for lung cancer were: 89 (44-159), 74 (20-190), and 180 (99-303); with the raised SMR at Portsmouth being very close to statistical significance ($P = 0.056$). From these results it would appear that x-ray groups 2, 3 and 4 (the groups indicating pleural thickening, pleural calcification and pulmonary fibrosis) could provide reasonable predictive properties for asbestos related lung disease. Therefore, these factors will be considered in the subsequent log-linear modelling.

When considering mortality by smoking habits no apparent differences appear between dockyards. However, a gradient of SMR from low in nonsmokers (significantly reduced), to higher in ex-smokers (with no difference from an SMR of 100), to significantly high in current smokers is generally observed over all disease groups in all three dockyards (table 4.9). Smoking habits were defined in the initial 1972-73 surveys; the definition of a current smoker is, therefore, a worker who smoked as much as one cigarette per day, or its equivalent, in the early 1970s. The above mentioned gradient produces a highly statistically significant trend over smoking habit for the following disease groups in each dockyard: all-cause mortality [X^2 for trend $= 82.9$, $P < 0.001$]*, all-neoplasms [X^2 for trend $= 33.1$, $P < 0.001$], lung cancer [X^2 for trend $= 58.1$, $P < 0.001$], diseases of the circulatory [X^2 for trend $= 14.7$, $P < 0.001$] and respiratory systems [X^2 for trend $= 17.1$, $P < 0.001$], and bronchitis [X^2 for trend $= 8.0$, $P < 0.005$]. No trend

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* The tests for trend are for Devonport dockyard. Those for Chatham and Portsmouth were generally lower, however, still highly significant.

TABLE 4.9: Cause specific mortality by smoking habit and dockyard.

Causes of Death	Smoking Habit	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	Non	251	72	63- 80	95	58	46- 70	171	58	49- 66
	Ex	427	75	68- 82	165	70	59- 81	315	68	60- 75
	Current	1415	112	106-118	569	109	100-118	906	104	97-111
	Unknown	37	76	52-101	14	73	40-123	8	83	36-163
All Neoplasms	Non	77	78	61- 96	33	68	45- 92	57	64	47- 80
	Ex	149	91	76-105	60	85	63-106	97	68	55- 82
	Current	489	135	123-147	214	137	118-155	334	126	113-140
	Unknown	13	96	51-165	3	55	11-162	2	67	8-243
Ca. Stomach	Non	10	126	60-231	3	77	16-226	4	56	15-144
	Ex	18	131	78-208	7	120	48-247	12	104	54-182
	Current	48	159	114-205	20	155	95-240	24	112	72-167
	Unknown	2	172	21-619	0	-	0-813	0	-	0-1546
Ca. Peritoneum	Non	1	454	11-2530	1	1097	28-6111	0	-	0-2241
	Ex	2	634	77-2289	0	-	0-3188	1	436	11-2429
	Current	6	822	301-1789	0	-	0-1355	0	-	0- 812
	Unknown	0	-	0-15847	1	123	311-68557	0	-	0-75109
Ca. Lung	Non	6	18	6- 38	3	16	3- 47	7	20	8- 41
	Ex	32	54	35- 72	14	49	27- 82	24	42	27- 63
	Current	183	140	120-160	78	124	97-152	156	148	125-171
	Unknown	2	41	5-148	1	47	1-260	1	84	2-469
Ca. Pleura	Non	13	2511	1337-4294	5	2246	728-5242	7	1605	644-3307
	Ex	14	1776	971-2980	4	1350	368-3455	8	1287	555-2536
	Current	34	1912	1269-2555	12	1775	917-3101	11	911	455-1629
	Unknown	1	1791	45-9978	0	-	0-19160	0	-	0-27732
Circulatory System	Non	131	75	62- 88	49	63	46- 81	92	65	52- 78
	Ex	229	78	68- 88	76	66	51- 81	169	75	63- 86
	Current	703	109	101- 117	257	102	90-115	408	97	87-106
	Unknown	161	72	42- 113	9	97	45-185	5	107	35-249
Pulmonary Circulation	Non	3	176	36- 514	1	120	3-670	1	68	2-378
	Ex	7	238	95- 490	0	-	0-294	0	-	0-152
	Current	17	262	153- 420	3	108	22-317	1	22	1-124
	Unknown	0	-	0- 1407	0	-	0-3505	0	-	0-7391
Respiratory System	Non	18	64	38- 101	7	44	18- 91	5	18	6- 42
	Ex	23	47	29- 70	16	66	38-107	26	56	37- 82
	Current	130	121	100- 141	56	107	79-135	99	118	95-141
	Unknown	4	84	23- 216	1	43	1-241	1	107	3-595
Bronchitis, Emphysema and Asthma	Non	4	39	11- 99	2	32	4-117	1	9	0- 52
	Ex	4	22	6- 56	5	52	17-122	6	33	12- 73
	Current	32	80	52- 107	19	91	55-142	27	82	54-119
	Unknown	1	59	1- 328	1	117	3-654	1	273	7-1523
Asbestosis	Non	0	-	0-6293	0	-	0-14120	0	-	0-7250
	Ex	3	3008	621-8792	0	-	0-9509	0	-	0-4621
	Current	4	1828	498-4679	0	-	0-4303	1	-	17-3711
	Unknown	0	-	0-48336	0	-	0-140652	0	-	0-217149
Pulmonary Fibrosis	Non	0	-	0- 884	0	-	0-1911	0	-	0-1052
	Ex	0	-	0- 510	0	-	0-1256	0	-	0-638
	Current	2	158	15- 457	1	156	4-871	0	-	0-347
	Unknown	0	-	0-5873	0	-	0-15707	0	-	0-31012

TABLE 4.10: Cause specific mortality by smoking amount and dockyard.

Causes of Death	Smoking amount (gms/day)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	< 15	712	88	82- 95	359	94	84-103	485	85	77- 92
	15-24	771	108	100-116	261	100	88-112	471	93	85-102
	≥ 25	360	114	102-126	116	100	82-118	266	101	89-113
All Neoplasms	< 15	236	102	89-115	139	120	100-140	177	102	87-117
	15-24	272	133	117-149	90	115	91-139	159	103	87-119
	≥ 25	131	144	119-168	45	129	92-167	95	119	95-143
Ca. Stomach	< 15	26	134	88-196	13	136	73-233	17	120	70-193
	15-24	32	189	124-255	11	171	85-306	11	89	44-158
	≥ 25	8	107	46-210	3	106	22-310	8	125	54-246
Ca. Peritoneum	< 15	5	1142	370-2664	0	-	0-1918	1	349	9-1945
	15-24	2	480	58-1732	0	-	0-2720	0	-	0-1418
	≥ 25	1	524	13-2918	0	-	0-6031	0	-	0-2670
Ca. Lung	< 15	56	67	49- 84	42	90	63-118	51	73	53- 93
	15-24	98	133	107-159	32	102	67-137	80	130	102-159
	≥ 25	61	186	139-233	18	130	77-206	49	155	112-199
Ca. Pleura	< 15	20	1852	1132-2861	12	2494	1289-4356	12	1559	805-2723
	15-24	19	1865	1123-2912	2	590	71-2131	4	576	157-1474
	≥ 25	10	2134	1025-3925	2	1290	156-4658	3	817	169-2389
Circulatory System	< 15	365	88	79- 97	159	85	72- 99	217	78	68- 89
	15-24	376	103	93-114	126	100	83-118	238	97	85-110
	≥ 25	191	118	101-135	50	90	65-115	123	97	80-114
Pulmonary Circulation	< 15	10	238	114-438	1	49	1-270	0	-	0-124
	15-24	9	248	113-470	2	145	18-522	1	38	1-214
	≥ 25	5	313	101-730	0	-	0-614	0	-	0-274
Respiratory System	< 15	69	97	74-120	33	84	55-112	48	85	61-109
	15-24	67	112	85-138	27	103	68-151	49	100	72-128
	≥ 25	17	66	38-105	12	105	54-183	28	111	74-161
Bronchitis, Emphysema and Asthma	< 15	16	61	35- 98	12	76	39-133	12	55	28- 95
	15-24	15	67	37-110	10	96	46-177	15	78	44-129
	≥ 25	5	51	17-120	2	45	5-161	6	61	22-133
Asbestosis	< 15	3	2170	448-6345	0	-	0-5887	1	1018	26-5670
	15-24	3	2413	498-7055	0	-	0-8586	0	-	0-4245
	≥ 25	1	1781	45-9919	0	-	0-19123	0	-	0-8184
Pulmonary Fibrosis	< 15	1	97	2-542	0	-	0-773	0	-	0-522
	15-24	1	113	3-628	0	-	0-1157	0	-	0-596
	≥ 25	0	-	0-948	1	711	18-3960	0	-	0-1157

whatsoever is seen for stomach cancer and the two forms of mesothelioma ($P > 0.1$, at all yards). Asbestosis and pulmonary fibrosis are again affected by low numbers. It should be noted, that for current smokers significantly elevated lung cancer SMRs were observed as follows: 140 (120-160) at Devonport, 124 (97-152) at Chatham, and 148 (125-171) at Portsmouth; based on 417 deaths.

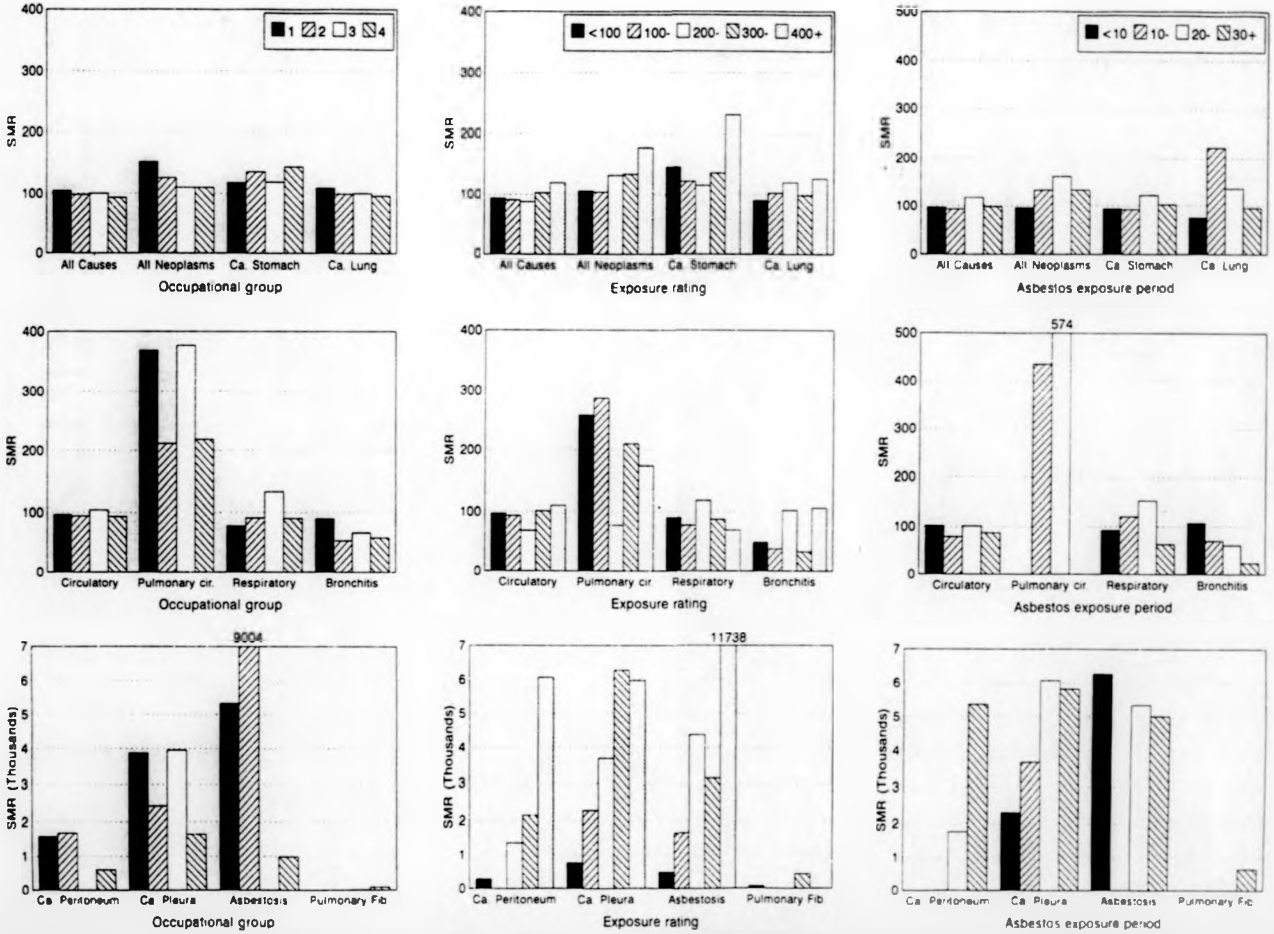
This picture of an increasing risk of death over smoking group is further considered by examining the amount of tobacco smoked (table 4.10). In this table the amount smoked per day by ex-smokers and current smokers is analysed by dockyard. Generally this analysis supports that shown by smoking habit, with its most obvious effect a lung cancer dose-response. At Devonport the lung cancer SMRs were: 67 (49-84) less than 15 gms/day, 133 (107-159) 15-24 gms/day, and 186 (139-233) 25 or more gms/day. At Chatham: 90 (63-118), 102 (67-137), and 130 (77-206). Similarly at Portsmouth: 73 (53-93), 130 (102-159), and 155 (112-199). In table 4.9 mortality gradients were seen with smoking group. However, from table 4.10 there appears to be a more limited gradient (i.e. dose-response) with amount smoked. This is clearly seen at Devonport and Portsmouth for all-cause mortality [X^2 for trend = 78.5 at Devonport and X^2 for trend = 48.3 at Portsmouth, both $P < 0.001$], and lung cancer [X^2 for trend = 74.7 at Devonport and X^2 for trend = 46.1 at Portsmouth, $P < 0.001$]. At Chatham dockyard no specific cause of death produced a significant trend. Pleural and peritoneal mesothelioma have non-significant differences in their SMRs at each dockyard over amount smoked.

The amounts recorded by the workforce may have affected the results here. They appear generally low and have forced the creation of the groups < 15, 15-24, and 25 or more grammes smoked per day, chosen to separate the smokers into approximately equal sized groups. To further scrutinise smoking history, duration of smoking was considered (figure 4.6 and table A3.26). This generally repeated the above findings, with an increasing SMR seen with duration of smoking. Considering Devonport dockyard, significant trends in SMR over smoking period are seen in the following disease groups: all-causes [X^2 for trend = 69.0,

$P < 0.001$], all-neoplasms [X^2 for trend = 42.4, $P < 0.001$], and lung cancer [X^2 for trend = 53.4, $P < 0.001$]. Significant differences, without a statistical trend, were approached in the SMRs for stomach cancer [$X^2 = 7.9$, $0.05 < P < 0.1$], and peritoneal mesothelioma [$X^2 = 8.7$, $0.05 < P < 0.1$], with asbestosis producing a significant difference [$X^2 = 22.3$, $P < 0.001$]. For asbestosis this difference reflects the very high SMR obtained for those workers with less then 10 years duration of smoking habit. However, these results should be treated with caution since they are again based on low numbers of deaths. From figure 4.6 diseases of the respiratory system appears to show a decreasing trend over time, however, this was statistically non-significant ($P > 0.1$). The remaining disease groups all produced non-significant results in tests of a difference between SMRs and tests of trend. Similar results were obtained for Chatham and Portsmouth, supporting the suggestion of a lung cancer time and dose-response.

Mortality has further been considered for these health-status variables, i.e. x-ray group, smoking habit etc., by time since first employment. The results of this examination are given in appendix 4 (tables A4.1 to A4.15). In these tables all-cause mortality, lung cancer mortality and pleural mesothelioma mortality is analysed by health-status variable and time since first dockyard employment. Generally, little clear effect is seen on mortality in these tables, with no obvious novel patterns of an excess or deficiency in all-cause and lung cancer mortality demonstrated. Pleural mesothelioma continues to show excessive SMRs over each variable for each time period; however, based on low observed numbers.

FIGURE 4.7: Standardised mortality ratios by occupational group, exposure group, and asbestos exposure period.



4.7 Cause specific mortality according to 'asbestos' variables.

This section is concerned with the following 'asbestos' variables: occupational group (indicating registered asbestos workers, laggers and sprayers, etc.), asbestos exposure rating, asbestos exposure period, and period of continuous asbestos exposure. The results are given in tables A3.27 to A3.29, and illustrated in figure 4.7.

Considering occupational group (table A3.27), the SMRs commonly showed little change over this grouping. For example, for lung cancer the SMRs found at Devonport were: 109 (56-190) for group 1, 99 (59-154) for group 2, 100 (50-178) for group 3, and 96 (82-11) for group 4. At Chatham these were slightly lower with SMRs of: 79 (26-185), 88 (35-181), 68 (14-200), and 86 (67-105). At Portsmouth: 106 (42-218), 157 (102-230), 77 (33-515), and 90 (75-105); with a significant result being obtained for group 2, the electrical fitters, and burners, etc. The exceptions to this limited change over grouping are pleural mesothelioma and asbestosis. For pleural mesothelioma a decreasing trend in SMR is observed from group 1 to group 4, for all dockyards [X^2 for trend = 4.3, $P < 0.05$; at Devonport]; this is likely to be due to the high number of mesothelioma deaths occurring in group 4, the catchment group of 'all other dockyard trades'. For asbestosis no trend is observed, however, the SMRs are significantly different over occupational grouping at Devonport ($P < 0.01$). A further exception is seen at Portsmouth for stomach cancer which appears to decrease over grouping with SMRs of: 223 (46-653), 150 (48-351), 141 (29-413), and 93 (62-133). However this was not significant [$X^2 = 3.1$, $P > 0.1$ and X^2 for trend = 2.5, $P > 0.05$].

The nature of this occupational grouping, as defined in 3.1.4, with group 1 indicating registered asbestos workers, etc., could well imply that this variable is a good substitute for asbestos exposure. Group 1 indicating very high exposure, group 2 high exposure, etc., down to group 4 which indicates low exposure. This is supported by the results for pleural mesothelioma; however, lung cancer has no

trend whatsoever across this variable [$X^2 = 0.2$, $P > 0.1$; at Devonport], nor indeed does peritoneal mesothelioma, giving no support to this theory.

A potentially better reflection of asbestos exposure is given by considering asbestos exposure rating (table A3.28). In this table increasing asbestos exposure should be seen with increasing rating. There is a general suggestion from the table that mortality increases with increasing rating; implying increasing SMR with increasing asbestos exposure. This is seen at all three dockyards. The following disease groups show increasing trends in their SMRs at Devonport: all-neoplasms [X^2 for trend $= 16.2$, $P < 0.001$], peritoneal mesothelioma [X^2 for trend $= 21.7$, $P < 0.001$], pleural mesothelioma [X^2 for trend $= 50.9$, $P < 0.001$], and asbestosis [X^2 for trend $= 10.4$, $P < 0.005$]. A borderline trend is also seen for all-cause mortality [X^2 for trend $= 3.0$, $0.05 < P < 0.1$]. Lung cancer, unlike the other asbestos related diseases, again has no clear statistical trend [$X^2 = 3.1$, $P > 0.1$]. However, by observation of the SMRs there is a hint of an increasing trend. For example, at Devonport the lung cancer SMRs are: 90 (74-107), 102 (68-135), 120 (79-162), 98 (59-154), and 126 (69-212). At Chatham: 94 (70-117), 65 (31-120), 81 (39-149), 67 (24-145), and 76 (24-176). At Portsmouth: 97 (78-116), 75 (48-112), 68 (39-110), 112 (69-170), and 193 (114-305). This last group at Portsmouth, those workers with an exposure rating of 400 or above, being clearly statistically significant. Pulmonary fibrosis has too few deaths to allow sensible analysis, 3 deaths over two dockyards.

An even better indication of asbestos exposure should be given by the workers own estimated period of asbestos exposure (table A3.29). The only limitation of this variable is the fact that less than 10% of the total workforce estimated this period. From the 3 dockyards 3359 workers responded to this question, out of which 887 (26.4%) have died. From table A3.29 increasing trends in mortality are seen at Devonport for: all-neoplasms [X^2 for trend $= 18.6$, $P < 0.001$], peritoneal mesothelioma [X^2 for trend $= 21.0$, $P < 0.001$], and pleura mesothelioma [X^2 for trend $= 37.4$, $P < 0.001$]. A decreasing trend is seen for lung cancer [X^2 for trend $= 9.0$, $P < 0.05$], this is however not repeated at Chatham and Portsmouth.

The lung cancer SMRs obtained at Devonport are: 75 (30-155), 222 (137-339), 136 (76-225), and 95 (49-166). At Chatham these were: 83 (230212), 27 (1-148), 44 (5-158), and 111 (70-108). At Portsmouth: 116 (56-213), 101 (46-191), 124 (66-213), and 108 (63-174). An excess of lung cancer is therefore seen at Devonport for those workers with between 10 and 20 years of asbestos exposure. Non-significant results were obtained for asbestosis, and again the numbers were too small for pulmonary fibrosis.

From the workers who estimated their asbestos exposure period, 429 from the 3 dockyards, also estimated their period of continuous exposure. From these 104 (24.2%) have died. Table 4.11 shows the mortality by period of continuous exposure for these workers. The numbers are very small, however, there appears generally to be significantly raised SMRs at Devonport and Portsmouth for those workers with more than 10 years of continuous asbestos exposure. Particularly lung cancer has SMRs of: 361 (132-786) at Devonport, and 490 (211-965) at Portsmouth; clearly showing an excess risk for lung cancer in this group of workers. However, only based on 14 deaths. Pleura mesothelioma is once more seen to be very high: 9232 (1117-33328) at Devonport, and 4994 (126-27818); based on 3 deaths. The numbers are to small for peritoneal mesothelioma and asbestosis to allow evaluation.

Mortality has also been appraised for the 'asbestos' variables by time since first employment. The results of this are given in appendix 4 (tables A4.16 to A4.27). In these tables all-cause, lung cancer and pleural mesothelioma mortality is analysed by the 'asbestos' variables and time since first dockyard employment. Similar to the health-status variables, no obvious new patterns of an excess or deficiency in all-cause and lung cancer mortality is generally apparent in these tables. Pleural mesothelioma again continues to show highly elevated SMRs over each variable for each time period. The results are obviously affected by low numbers, particularly in tables A4.25 to A4.27.

TABLE 4.11: Cause specific mortality by period of continuous asbestos exposure and dockyard.

Causes of Death	Continuous asbestos exposure (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	<10	23	117	74-176	16	143	82-233	15	119	67-197
	10+	27	170	112-247	5	108	35-251	18	138	82-218
All Neoplasms	<10	4	71	19-181	9	264	121-502	5	132	43-307
	10+	14	308	168-517	1	73	2-408	14	346	189-580
Ca. Stomach	<10	1	222	6-1239	2	723	88-2611	1	328	9-1882
	10+	1	260	7-1446	0	-	0-3167	0	-	0-1137
Ca. Peritoneum	<10	0	-	0-28311	0	-	0-56870	0	-	0-48353
	10+	1	11216	284-62475	0	-	0-181558	0	-	0-50799
Ca. Lung	<10	1	50	1-280	2	147	18-530	0	-	0-253
	10+	6	361	132-786	0	-	0-666	8	490	211-965
Ca. Pleura	<10	0	-	0-11421	1	6203	157-34550	2	9976	1207-36015
	10+	2	9232	1117-33328	0	-	0-77076	1	4994	126-27818
Circulatory System	<10	12	121	63-212	5	93	30-218	4	67	18-172
	10+	7	85	34-174	2	88	11-317	4	63	17-161
Pulmonary Circulation	<10	0	-	0-3946	0	-	0-6263	0	-	0-6147
	10+	2	2370	287-8554	0	-	0-13985	0	-	0-5573
Respiratory System	<10	5	340	110-793	2	194	24-702	2	186	23-673
	10+	6	427	157-929	2	373	45-1348	0	-	0-316
Bronchitis, Emphysema and Asthma	<10	1	183	5-1017	2	475	57-1714	0	-	0-892
	10+	1	190	5-1058	0	-	0-1752	0	-	0-784
Asbestosis	<10	1	27969	708-155786	0	-	0-188727	0	-	0-166003
	10+	1	36317	919-202285	0	-	0-546965	0	-	0-151071
Pulmonary Fibrosis	<10	1	4308	109-23995	0	-	0-28027	0	-	0-25595
	10+	0	-	0-18285	0	-	0-62290	0	-	0-23521

The 'asbestos' variables have been further scrutinised by smoking habit (tables A3.30, 4.12 and 4.13). In these tables all-cause, lung cancer and pleural mesothelioma mortality is again considered, here for nonsmokers, ex-smokers and current smokers. No obvious patterns occur when considering all-cause mortality (table A3.30). For lung cancer, significantly high SMRs are seen in 'smoking groups', the highest SMRs occurring in current smokers, except at Portsmouth where ex-smokers are seen to have generally high SMRs. Pleural mesothelioma (table 4.13) is affected by the low numbers of observed deaths, but significantly high SMRs are seen once again. These are however not related to smoking habit, there being no clear statistical trends (generally, $P > 0.1$). When considering lung cancer (table 4.12) increasing SMRs across smoking habit, which reach statistical significance, are seen only for occupational group 4. At Devonport these are: 72

(52-92) for nonsmokers, 134 (104-163) for ex-smokers, and 177 (125-230) for smokers [$X^2 = 21.4$, $P < 0.001$ and X^2 for trend $= 21.6$, $P < 0.001$]. At Chatham: 102 (70-135), 96 (61-143), and 137 (77-226) [$X^2 = 1.3$, $P > 0.1$ and X^2 for trend $= 0.6$, $P > 0.1$]. At Portsmouth: 72 (49-94), 119 (88-150), and 152 (103-201) [$X^2 = 11.6$, $P < 0.005$ and X^2 for trend $= 11.9$, $P < 0.001$]. Indicating generally that for occupational group 4 (i.e. the catchment group of all other dockyard workers) there is undoubtedly an increasing trend in lung cancer mortality by smoking habit.

The relationship between lung cancer and pleura mesothelioma to these asbestos variables will be further considered in section 4.8. Here Poisson regression techniques will be used to focus on these variables taking into account other factors such as smoking habit, and x-ray group, etc.

TABLE 4.12: Devonport Dockyard. Lung cancer mortality by smoking habit and 'asbestos' variables.

	Non-smokers		Ex-smokers		Smokers	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
Occupational group						
1	3	88 (18-256)	5	140 (45-327)	3	151 (31-440)
2	2	32 (4-115)	9	143 (65-271)	7	226 (91-466)
3	1	30 (1-166)	3	82 (17-241)	6	289 (106-628)
4	49	72 (52- 92)	79	134 (104-163)	44	177 (125-230)
Exposure rating						
< 100	26	58 (38- 85)	56	141 (104-178)	25	140 (91-207)
100-	7	52 (21-107)	13	119 (63-204)	15	294 (164-484)
200-	9	100 (46-190)	15	159 (89-263)	8	194 (84-383)
300-	5	67 (22-157)	5	80 (26-186)	8	342 (148-675)
400+	5	132 (43-309)	6	164 (60-358)	3	181 (37-530)
Asbestos exposure period (yrs)						
< 10	1	28 (1-155)	4	126 (34-323)	1	64 (2-356)
10-	6	181 (66-394)	7	189 (76-389)	7	449 (180-924)
20-	6	147 (54-321)	6	151 (55-328)	3	156 (32-456)
30+	4	85 (23-218)	3	78 (16-229)	5	256 (83-597)
Continuous asbestos exposure (yrs)						
< 10	-		-		1	220 (6-1228)
10-	4	601 (164-1540)	2	285 (35-1030)	-	

TABLE 4.12 (cont.): Chatham Dockyard. Lung cancer mortality by smoking habit and 'asbestos' variables.

	Non-smokers		Ex-smokers		Smokers	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
Occupational group						
1	2	57 (7-205)	2	124 (15-448)	1	73 (3-761)
2	1	32 (1-176)	4	165 (45-423)	2	163 (20-588)
3	-		1	63 (2-352)	-	
4	38	102 (70-135)	24	96 (61-143)	15	137 (77-226)
Exposure rating						
< 100	25	93 (60-138)	21	123 (76-187)	14	180 (99-303)
100-	5	82 (26-191)	4	90 (25-231)	1	40 (1-225)
200-	7	129 (52-267)	-		-	
300-	3	79 (16-230)	2	68 (8-247)	1	110 (3-612)
400+	-		3	133 (28-390)	2	312 (38-1127)
Asbestos exposure period (yrs)						
< 10	3	131 (27-384)	1	80 (2-444)	-	
10-	-		1	87 (2-483)	-	
20-	-		1	88 (2-491)	1	135 (3-753)
30+	3	129 (27-376)	2	105 (13-379)	1	219 (6-1220)
Continuous asbestos exposure (yrs)						
< 10	1	143 (4-799)	1	279 (7-1556)	-	
10-	-		-		-	

TABLE 4.12 (cont.): Portsmouth Dockyard. Lung cancer mortality by smoking habit and 'asbestos' variables.

	Non-smokers		Ex-smokers		Smokers	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
			Occupational group			
1	3	133 (27-388)	2	104 (13-376)	2	137 (17-493)
2	5	87 (28-204)	16	334 (191-543)	5	173 (56-403)
3	1	26 (1-147)	3	98 (20-286)	2	145 (18-523)
4	39	72 (49- 94)	58	119 (88-150)	37	152 (103-201)
			Exposure rating			
< 100	28	76 (50-110)	41	125 (87-164)	28	175 (116-252)
100-	6	51 (19-111)	10	113 (54-207)	6	119 (44-259)
200-	3	42 (9-122)	7	91 (36-187)	6	124 (46-270)
300-	7	102 (41-211)	12	212 (109-369)	2	70 (8-252)
400+	4	126 (34-323)	9	273 (125-519)	3	293 (61-857)
			Asbestos exposure period (yrs)			
< 10	3	91 (19-266)	3	101 (21-296)	4	284 (77-728)
10-	2	49 (6-177)	7	283 (114-583)		-
20-	4	106 (29-272)	6	196 (72-427)	3	137 (28-399)
30+	3	47 (10-137)	10	212 (102-389)	4	201 (55-514)
			Continuous asbestos exposure (yrs)			
< 10		-		-		-
10-	3	617 (127-1803)	4	574 (157-1470)	1	429 (11-2388)

TABLE 4.13: Devonport Dockyard. Pleural mesothelioma mortality by smoking habit and 'asbestos' variables.

	Non-smokers	Ex-smokers	Smokers
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
	Occupational group		
1	4 8564 (2334-21925)	1 1893 (48-10541)	1 3655 (92-20357)
2	2 2304 (279-8317)	3 3178 (656-9290)	-
3	2 3421 (414-12351)	2 2985 (361-10775)	2 5559 (673-20068)
4	12 1395 (721-2436)	12 1522 (786-2658)	7 2029 (815-4181)
	Exposure rating		
< 100	2 343 (41-1238)	5 908 (294-2120)	2 784 (95-2830)
100-	6 3244 (1189-7061)	3 1800 (371-5261)	-
200-	4 3308 (901-8467)	4 2934 (799-7510)	5 8297 (2688-19365)
300-	5 5589 (1811-13045)	6 7820 (2869-17030)	1 3245 (82-18075)
400+	3 7764 (1602-22697)	-	2 11178 (1353-40353)
	Asbestos exposure period (yrs)		
< 10	1 2078 (53-11573)	1 2330 (59-12977)	-
10-	2 4440 (537-16029)	2 3800 (460-13718)	-
20-	4 7428 (2024-19016)	4 7317 (1994-18732)	1 4017 (102-22372)
30+	3 6034 (1245-17640)	1 2415 (61-13451)	3 13569 (2800-39667)
	Continuous asbestos exposure (yrs)		
< 10	-	-	-
10-	2 23469 (2840-84724)	-	-

TABLE 4.13 (cont.): Chatham Dockyard. Pleural mesothelioma mortality by smoking habit and 'asbestos' variables.

	Non-smokers	Ex-smokers	Smokers
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
	Occupational group		
1	1 3035 (77-16907)	-	-
2	5 13775 (4463-32150)	-	1 6863 (174-38228)
3	1 4219 (107-23501)	-	-
4	5 1321 (428-3082)	2 757 (92-2734)	1 824 (21-4590)
	Exposure rating		
< 100	-	1 535 (14-2981)	-
100-	-	-	1 3357 (85-18696)
200-	5 8416 (2727-19643)	-	-
300-	4 10720 (2921-27443)	1 3366 (85-18749)	1 11519 (291-64163)
400+	2 8471 (1025-30581)	-	-
	Asbestos exposure period (yrs)		
< 10	-	-	-
10-	1 4752 (120-26471)	-	-
20-	2 8720 (1055-31480)	-	-
30+	2 8933 (1081-32247)	-	1 23190 (587-129171)
	Continuous asbestos exposure (yrs)		
< 10	1 11188 (283-62311)	-	-
10-	-	-	-

TABLE 4.13 (cont.): Portsmouth Dockyard. Pleural mesothelioma mortality by smoking habit and 'asbestos' variables.

	Non-smokers	Ex-smokers	Smokers
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
	Occupational group		
1	-	-	-
2	2 2949 (357-10646)	2 3255 (394-11750)	-
3	-	-	1 5193 (131-28927)
4	10 1689 (811-3106)	2 371 (45-1338)	2 721 (87-2604)
	Exposure rating		
< 100	4 973 (265-2490)	-	-
100-	2 1496 (181-5400)	-	1 1534 (39-8542)
200-	2 2345 (284-8465)	3 3432 (708-10033)	-
300-	3 4156 (857-12149)	-	1 3431 (87-19108)
400+	1 3453 (87-19233)	1 3551 (90-19778)	1 9505 (240-52943)
	Asbestos exposure period (yrs)		
< 10	1 2632 (67-14658)	-	-
10-	2 4405 (533-15903)	-	-
20-	-	1 3215 (81-17909)	-
30+	5 8359 (2708-19511)	1 2216 (56-12343)	1 5005 (127-27880)
	Continuous asbestos exposure (yrs)		
< 10	1 10522 (266-58610)	1 17455 (442-97233)	-
10-	-	-	-

4.8 Log-linear modelling.

The modelling presented in this section is for Devonport dockyard only, it has been chosen as a representative dockyard and for the cause of brevity. Chatham and Portsmouth have also been examined and generally have shown the same relationships, however, to a less significant level.

The two disease groups considered are the principal conditions seen to be of interest in this study, namely: lung cancer and pleural mesothelioma. The disease group of 'all-causes' is also examined. These three specific disease groupings have substantially enough numbers of observed deaths to allow sensible modelling. The groups of: pulmonary fibrosis with 2 deaths (at Devonport), asbestosis with 7 deaths, and peritoneal mesothelioma with 10 have not been modelled.

Statistical variation in strata-specific mortality (SMRs) was assessed by comparing the fit of Poisson regression models with and without terms representing the stratifying factor, i.e. by the use of log-linear models for grouped data. The goodness-of-fit of each model was tested by considering the change in 'deviance' (the approximate Chi-squared statistic) found in GLIM; the technique used was that described in section 3.2.3.

When exponentiated, the coefficients gained from the regression analyses are relative standardised mortality ratios (RSMRs), i.e. the exponential coefficients estimate the mortality rate for workers in a particular stratum (subgroup) relative to that in stratum 1. From these RSMRs, tables similar to those given for the SMRs in appendix 3 can be produced. Generally the RSMRs have shown little variation from the SMRs, even when adjusted for statistical interactions and confounding factors, therefore, RSMR tables have not been furnished. In their place, this section will concentrate on describing the appropriateness of fit of the 'employment-time', 'health-status', and 'asbestos' variables considered in sections 4.5 to 4.7, on the regression models.

TABLE 4.14: Devonport dockyard. Goodness-of-fit statistics obtained in Poisson regression models.

All Causes			Lung Cancer			Ca. Pleura		
Model	Δdev	df	Model	Δdev	df	Model	Δdev	df
Constant+			Constant+			Constant+		
ag	22.9**	4	ag	99.8**	4	ag	7.4	4
ys	7.9	4	ys	8.3	4	ys	10.1*	4
ls	6.4	4	ls	9.6*	4	ls	13.3**	4
ts	5.7	3	ts	6.1	3	ts	6.4	3
og	4.1	3	og	4.3	3	og	8.0*	3
ex	6.3	4	ex	5.8	4	ex	14.1**	4
ae	3.7	3	ae	12.4**	3	ae	9.7*	3
ce	5.7*	1	ce	7.9**	1	ce	-	
co	4.6*	1	co	5.6*	1	co	1.7	1
ph	2.6	1	ph	3.9*	1	ph	1.2	1
br	4.0*	1	br	4.2*	1	br	3.9*	1
ch	3.5	1	ch	3.6	1	ch	0.9	1
sm	12.2**	2	sm	25.1**	2	sm	2.5	2
pt	1.8	1	pt	2.7	1	pt	3.9*	1
pc	2.0	1	pc	3.7	1	pc	4.1*	1
pf	3.6	1	pf	4.8*	1	pf	5.7*	1
Constant+ag+			Constant+ag+			Constant+ex+		
sm	12.8**	2	sm	37.6**	2	ls	18.1**	4
ce	6.2*	1	ce	10.9**	1	og	6.7	3
ae	3.8	3	ae	5.5*	3	ae	11.3*	3
pf	0.9	1	pf	2.8	1	pf	4.9*	1
			ls	9.9*	4			
Constant+ag+sm+			Constant+ag+sm+			Constant+ex+ls+		
ce	1.3	1	ce	3.2	1	ae	7.2	3
pf	0.4	1	pf	7.8**	1	pf	3.7	1
			co	8.9**	1			
			br	6.3*	1			
Constant+ag+			Constant+ag+			Constant+ex+		
ag*sm	1.4	2	ag*sm	12.0**	2	ex*ls	6.7	4
ag*ce	0.1	1	ag*ce	0.7	1			
			ag*ae	0.3	4			
			Constant+ag+ ag*sm+					
			co	2.8	1			

Δdev = change in model deviance
df = degrees of freedom

* P<0.05
** P<0.01

Grouped variable definitions:

'Employment' variables.
ts time since start of employment
ls length of service
ys year of start of employment

'Asbestos' variables
og occupational group
ex exposure rating
ae asbestos exposure period
ce continuous asbestos exposure

'Health-status' variables.
co cough
ph phlegm
br breathlessness
ch chest-illness
sm smoking habit
pt pleural thickening
pc pleural calcification
pf pulmonary fibrosis

ag age at death

The results of the main regression models are given in table 4.14. Considering all-cause mortality, the models clearly show that there is a significant variation in the SMRs with age, smoking habit (nonsmokers, ex-smokers, etc.), breathlessness, asbestos exposure period and period of continuous asbestos exposure. It should be noted, as stated in the last section, that the continuous exposure variable is based on a limited number of workers (i.e. 429 in total). Occupation group, asbestos exposure rating and time since start of employment are seen to have no effect, the same is true of the remainder of the descriptive variables used in this modelling. No interaction terms produced statistically significant results. The conclusion for the disease group of 'all-causes' is that age and smoking are the main factors of interest. This result in no way contradicts the results given in earlier sections of this chapter, and supports the conclusions of no excess risk for the majority of the employment and asbestos variables considered.

For lung cancer the smoking dose-response is reinforced, and smoking with age seen to be a very good predictor of death. Age and smoking are in fact seen to have a significant interaction. Asbestos exposure period and period of continuous asbestos exposure are again seen to have significant variation in their SMRs. Cough, phlegm and breathlessness are also seen to be fairly good predictive variables of lung cancer mortality. Radiographs with pulmonary fibrosis present also show a slight predictive value. However, occupational group and asbestos exposure rating are not significant, i.e. the SMRs do not vary over occupational group and exposure rating, supporting the findings of section 4.7. The fact that man is a better predictor of his own health than exposure ratings is shown by the significance and high SMRs for cough, phlegm, breathlessness and chest-illness and supported by the regression analysis.

Pleural mesothelioma is not seen to be related whatsoever to smoking. The only significant variation in SMR seen in the regression models is with the employment and asbestos variables. The main predictive factors for mesothelioma in this dockyard appear to be asbestos exposure rating and length of service in the dockyard. Pulmonary fibrosis on radiograph is again seen to have reasonable

predictive values together with pleural thickening and pleural calcification. The same is true for breathlessness and occupational group.

Generally, the results of the log-linear modelling support the previous SMR analyses, showing that smoking is a highly significant variable in predicting lung cancer death, whereas, occupational grouping is not. The reverse is seen for mesothelioma.

It should be noted that a major advantage of the models used in this section is their ease of implementation using such packages as GLIM or GENSTAT. These packages have the outstanding feature of enabling the user to specify factors and their interactions without having to generate the dummy variables him/herself, providing a useful means of carrying out detailed comparisons of mortality among subgroups defined by a multitude of covariates.

4.9 Summary.

For the three dockyards Devonport, Chatham and Portsmouth, a slight deficiency in all-cause mortality has been observed. Linked with this deficiency is the fact that mortality due to 'all-neoplasms' is not seen to be in either excess or deficit. For example, all-neoplasm SMRs are not generally different from 100, although at Devonport the SMR is statistically raised, i.e. 117 (109-125). Generally, 'non-cancer' mortality is seen to be reduced, with SMRs commonly reduced below 100 (but in most cases not significantly). For asbestosis and the two forms of mesothelioma, large significant excesses in mortality are seen, with SMRs commonly being at least an order of magnitude higher than 100. However, low, non-significant SMRs are seen for pulmonary fibrosis.

It is possible that there is some death certificate confusion between asbestosis and pulmonary fibrosis. They are indeed practically the same medical condition, and it is likely that a mis-coding may have transpired between these conditions. Although it may be argued that we are dealing with an 'asbestos environment', i.e.

the dockyards, and that this fact may have influenced the reported death certificate cause of death, these conditions have been analysed separately in this work. It is hoped by this, that any recording bias by the nosologist will be balanced over all death certificates.

The interesting result of this study is the lack of notable excess lung cancer mortality. In fact, an overall significant deficiency in lung cancer mortality is also absent. For all three dockyards, we generally have neither a statistically significant excess or deficit of lung cancer mortality from that expected in the South West and South East regional populations. This gives little support to any suggestion that dockyard exposure to asbestos is an aetiological factor in lung cancer production.

The disease groups of all-cause and all-neoplasm mortality in the 'absolute non-responders' from the initial surveys, have significantly high SMRs. For lung cancer, SMRs are all high, with the SMR at Portsmouth being statistically elevated from 100. Mesothelioma again shows a huge excess. Overall, an excess mortality is seen in all disease groups, occasionally reaching a statistical excess. It appears that a proportion of the non-responders may have been chronically ill at study definition, in the early 1970s, and deselected themselves out of the surveys for health reasons. These workers were reinvited to attend for interview and radiograph only once more after initial non-response. This deselection of workers also overshadows any healthy worker effect to be seen in the study responders.

Generally then, there was little, if any, overall excess or deficiency of mortality seen in the initial surveys responders, over the three dockyards, apart from the asbestos related diseases of pleural and peritoneal mesothelioma, and asbestosis. These disease groups together with the other 9 specific disease groups were also considered by 'employment-time', 'health-status', and 'asbestos' variables.

When considering mortality by the 'employment-time' variables (length of employment, time since start of employment, etc.), the lung cancer risk appears

to increase with increasing age at start of employment. Whereas, mesothelioma appears to decrease, the youngest workers at start of employment having the highest mortality rates; showing the possible effects of early asbestos exposure. A decreasing trend in mesothelioma SMR is also seen over calendar year of employment, with the highest SMRs occurring for those workers employed before World War II. Lung cancer appears to increase with calendar period (figure 4.4), however, there is no statistical trend evident. Asbestosis and pulmonary fibrosis mortality is based on too few numbers to allow sensible analysis.

An increasing mortality trend is seen for pleural mesothelioma over length of service grouping, the highest mortality rates occurring in those workers with 30 or more years employment. Supporting the suggestion of a long disease latency period. When considering time since first employment, only disease of the respiratory system showed a significantly increasing trend in mortality. No other disease group gave any evidence of a systematic increase or decrease in mortality with time since first employment and possible first exposure to asbestos. A huge excess of mesothelioma was, however, seen at all three dockyards, clustered around those workers with long follow-up and long duration of service. Lung cancer was seen only to be in excess for workers with between 10 and 19 years of employment first exposed to asbestos over 30 years ago.

From the 'health-status' variables (i.e. personal medical history, smoking habits, etc.) cough, phlegm, breathlessness and chest-illness, were seen to be reasonable predictive variables for lung disease. Radiographic grouping was another such variable, and in particular x-ray groups 2, 3 and 4. That is, the groups showing pleural thickening, pleural calcification, and pulmonary fibrosis, present on radiograph. When examining mortality by smoking history, a clear dose-response relationship was seen for lung cancer. The relative risks for lung cancer at Devonport across smoking group are: 1, 3, and 7.8; for nonsmokers, ex-smokers and smokers, respectively (table 4.9). No such relationship was seen for mesothelioma. Asbestosis and pulmonary fibrosis were again affected by low numbers. Considering the 'asbestos' variables, lung cancer shows no pattern

whatsoever across these occupation and asbestos exposure parameters, except when analysed by smoking habit. When this is undertaken there appears to be an elevated lung cancer risk for the workers in occupational group 4 (i.e. the group of 'all other workers'). Lung cancer risks are also present in the small group of 429 workers (i.e. 1.5% of the cohort) who estimated their period of continuous asbestos exposure. Here workers with more than 10 years of continuous exposure appear most at risk. Pleural and peritoneal mesothelioma all show increasing trends with asbestos exposure rating.

In conclusion, there is only limited evidence in this study to link dockyard asbestos dust exposure aetiologically with lung cancer. There is, however, support for the idea of a lung cancer dose-response with tobacco smoking. For both forms of mesothelioma the reverse is seen; no relationship with smoking, but evidence of a relationship with asbestos exposure. The log-linear modelling supported these results. Indeed the two approaches used, the SMR and regression analyses, produced virtually the same results.

Chapter 5: MORTALITY REVISITED.

5.1 Introduction.

This chapter describes the results of nested case-control analyses performed on the workers from the three dockyards to further evaluate lung cancer and mesothelioma mortality, with regard to smoking habit and asbestos exposure. Pleural and peritoneal mesothelioma are grouped in this chapter as one cause of death 'mesothelioma'. The cases are all lung cancer and mesothelioma deaths occurring in the dockyards over the 17 year study period. The controls are matched to the cases by age and year of first dockyard employment. Logistic regression, as described in chapter 3, will be used in this chapter to analysis the case-control data.

The relationship of mesothelioma with time since first possible asbestos exposure, i.e. first dockyard employment, is also scrutinised in this chapter. Section 5.3 summarises a mathematical model relating dockyard mesothelioma incidence over all three dockyards, Devonport, Chatham and Portsmouth, exponentially with time since employment. This model is compared and contrasted to the model suggested by Peto in 1982.[1]

5.2 Nested case-control analysis.

The only conceptual difference between a full cohort study and a case-control study based on the same cohort is that the latter involves a sample of the study base rather than an analysis of the entire study base. There is little loss of precision in a nested case-control analysis compared to a full cohort analysis, indeed the case-control approach is particularly valuable if the study disease is rare or has a long induction period, as often happens with mesothelioma. In essence this chapter is chiefly concerned with mesothelioma. An advantage of the case-

control method over a full cohort analysis is its ability to control confounders in the matching of cases and controls. An associated advantage is therefore its ability to negate the health worker effect.

In the analyses presented here, the controls were matched to the cases by incidence density sampling, according to age and year of first employment (± 1 year for each of these). Four controls per case were employed, giving for mesothelioma: 70 case-control sets at Devonport, 23 at Chatham, and 26 at Portsmouth. For lung cancer 216 case-controls sets were obtained for Devonport, 84 for Chatham, and 183 at Portsmouth. Only 2 mesothelioma deaths were unmatched, and omitted, in these analyses (one at both Devonport and Portsmouth). In total 24 lung cancer deaths were not matched to controls; 7 at Devonport, 12 at Chatham, and 5 at Portsmouth.

Table 5.1 summarises the results of the mesothelioma logistic regression analysis. From this table it can quickly be seen that after matching for age the main effect on mortality across all three dockyards was asbestos exposure rating. At Devonport occupational group was also seen to be significant in the regression model; at Chatham phlegm production takes the place of occupational group and is seen in the model. The other factors considered, cough, breathlessness, and smoking habits, etc., are not seen to have any effect.

When modelling just the exposure rating term the following elevated mesothelioma odds ratios were produced: 1.84 (95%CI: 1.45-2.33) at Devonport, 1.64 (95%CI: 1.12-2.41) at Chatham, and 1.65 (95%CI: 1.17-2.33) at Portsmouth. These show a clear excess mortality due to mesothelioma over all three dockyards, when relating mesothelioma incidence to asbestos exposure rating.

Table 5.2 gives the lung cancer regression results. From this table it is apparent that occupational exposure to asbestos plays only a limited role in the production of lung cancer at the three dockyards, with cigarette smoking appearing to be the predominant casual factor. Other significant factors appear to be symptoms from

TABLE 5.1: Goodness-of-fit statistics obtained in Mesothelioma logistic regression.

Devonport			Chatham			Portsmouth		
Model	Δdev	df	Model	Δdev	df	Model	Δdev	df
og	5.6*	1	og	3.6	1	og	0.6	1
co	0.1	1	co	0.4	1	co	0.6	1
ph	0.5	1	ph	6.1*	1	ph	2.4	1
br	0.2	1	br	0.1	1	br	0.0	1
ch	0.4	1	ch	0.0	1	ch	0.2	1
sm	0.5	1	sm	0.6	1	sm	1.4	1
sa	0.0	1	sa	3.7	1	sa	0.2	1
sd	0.4	1	sd	0.4	1	sd	0.8	1
ex	30.1**	1	ex	7.2**	1	ex	9.1**	1
ls	0.5	1	ls	-		ls	0.0	1
ae	0.2	1	ae	2.8	1	ae	1.8	1
ae+og	31.1**	2	ae+og	7.3*	1			
			ae+ph	9.7**	1			
ae+ae*og	31.5**	3						

TABLE 5.2 Goodness-of-fit statistics obtained in Lung Cancer logistic regression.

Devonport			Chatham			Portsmouth		
Model	Δdev	df	Model	Δdev	df	Model	Δdev	df
og	0.1	1	og	0.1	1	og	3.4	1
co	15.8**	1	co	14.4**	1	co	28.3**	1
ph	16.4**	1	ph	4.7*	1	ph	44.3**	1
br	11.3**	1	br	4.1*	1	br	8.8**	1
ch	0.6	1	ch	0.3	1	ch	1.3	1
sm	51.5**	1	sm	26.7**	1	sm	65.7**	1
sa	15.7**	1	sa	0.8	1	sa	19.0**	1
sd	38.9**	1	sd	19.4**	1	sd	31.0**	1
ex	6.5*	1	ex	0.7	1	ex	6.4*	1
ls	0.0	1	ls	0.3	1	ls	0.0	1
ae	0.6	1	ae	0.0	1	ae	0.4	1
sm+sd	40.1**	2	sm+sd	19.4**	2	sm+ph	87.4**	2
sm+sa	40.2**	2	sm+co	33.5**	2	sm+co	75.5**	2
sm+co	57.9**	2	sm+ph	27.9**	2	sm+sd	51.7**	2
sm+ph	56.7**	2	sm+br	29.1**	2	sm+sa	74.4**	2
sm+br	62.2**	2				sm+br	73.0**	2
sm+ae	54.0**	2				sm+ex	73.5**	2
sm+br+ae	61.6**	3	sm+co+br	34.0**	3	sm+ph+co	88.4**	3
sm+br+co	65.0**	3	sm+co+ph	33.5**	3	sm+ph+sa	89.8**	3
sm+br+ph	63.8**	3	sm+co+sd	24.9**	3	sm+ph+ex	91.4**	3
sm+br+sm*br	63.7**	4	sm+co+sm*co	21.4**	4	sm+ph+sm*ph	87.1**	4

Δdev = change in model deviance
df = degrees of freedom

* P<0.05
** P<0.01

Grouped variable definitions:

og	occupational group	co	cough
ex	exposure rating	ph	phlegm
ls	length of service	br	breathlessness
ae	asbestos exposure period	ch	chest-illness
		sm	smoking habit
		sa	smoking amount
		sd	duration of smoking

the workers personal medical history; with, for example, breathlessness having a significant effect at Devonport, cough at Chatham, and phlegm production at Portsmouth. Asbestos exposure rating is seen to be significant in the regression models at Devonport and Portsmouth, however, less significantly so than medical history.

When modelling only with the term relating to smoking habit the following highly elevated lung cancer odd ratios were produced: 2.55 (95%CI: 1.89-3.44) at Devonport, 2.85 (95%CI: 1.71-4.75) at Chatham, and 3.04 (95%CI: 1.83-5.04) at Portsmouth. Demonstrating an excess lung cancer risk.

Simply, this nested case-control analysis supports the results of chapter 4, showing for lung cancer that cigarette smoking is the dominant factor and not dockyard asbestos exposure (or its near surrogate). The opposite is seen for mesothelioma, no smoking effect, and elevated odds ratios for asbestos exposure rating.

5.3 Mesothelioma modelling.

In contrast to lung cancer, which has been commonly described by a linear dose-response relationship with asbestos exposure[2], mesothelioma is best described by an absolute risk model in which the incidence of both pleural and peritoneal mesothelioma is independent of the age at first exposure (and cigarette smoking) and increases according to a power function of time since first exposure. This model was first suggested and used by Newhouse and Berry in 1976 to predict mesothelioma mortality in a group of asbestos factory workers.[3] They suggested the following model:

$$I_M = b(t - w)^k ,$$

where I_M is the incidence of mesothelioma at time t from first exposure, w is a delay in the expression of the risk, and b and k are empirically derived constants.

The incidence of asbestos induced mesothelioma in rats has also been seen to follow this time course.[4] From the work of Newhouse and Berry it was suggested that k lies between 1.4 and 2 and w between 9 and 11 years, i.e. a quadratic model was suggested with a time delay of approximately 10 years. Peto et al in 1982 further showed that the absolute incidence of mesothelioma was independent of age at first exposure and suggested the model:

$$I_M = bt^k ,$$

with 3.20 as his best estimate of k.[1] This model was based on the data from the study of North American insulation workers undertaken by Selikoff et al in 1979[5], and modelled 236 mesothelioma deaths. The model was seen to fit the data well between 20 and 45 years from first asbestos exposure. However, observed incidence rates for earlier times were less than those projected, and Peto suggested a return to the model of Newhouse and Berry, that is, the use of a quadratic time-dependence with a lag of 10 years. This quadratic expression fitted the data better up to 45 years from first exposure. The analysis of Peto et al excluded workers first employed before 1922 and after 1946, and over the age of 80; the fit to the mortality of the entire group suggested a value of k of about 5.

The model suggested by Peto has been used by Sullivan et al to model the mesothelioma incidence in the Royal Naval Dockyards (see appendix 5: supporting evidence).[6] In this work the mortality experience of workers at Devonport, Chatham and Portsmouth was followed for 10 years, and 56 mesothelioma deaths were accumulated. The best estimate of the power parameter (k) was found to be in close agreement to Peto's value; with $k=3.14$. However, the constant of proportionality (b), a measure of relative mesothelioma incidence, was seen to be nearly an order of magnitude less than that suggested by Peto. Suggesting that the risk of dying from mesothelioma for dockyard workers is much less than that seen for North American insulation workers.

TABLE 5.3: Distribution by time since first employment and age at first employment of 120 mesothelioma deaths.

Age first employed (yrs)		Years since first employment										
		0-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50+
15-24	Person-years	5505	18466	28060	34264	31053	26274	25845	26705	23590	18821	14022
	Observed	0	0	1	0	2	4	8	14	20	23	20
	Expected	0.63		1.94	1.30	3.27	7.66	8.13	10.32	19.68	22.97	20.19
25-34	Person-years	1137	3424	4805	6147	6618	8087	8389	8030	4870	2470	563
	Observed	0	0	0	1	1	7	3	0	3	3	1
	Expected	0.13		0.33	0.23	0.70	2.36	2.64	3.10	4.06	3.01	0.81
35-44	Person-years	750	2720	4444	6214	6542	5576	3587	1477	305	10	
	Observed	1	0	0	0	1	1	1	0	1	0	
	Expected	0.09		0.31	0.24	0.69	1.62	1.13	0.57	0.25	0.01	
45-54	Person-years	920	3287	4822	5011	2994	1218	314	13			
	Observed	0	0	2	1	1	0	0	0			
	Expected	0.10		0.33	0.19	0.31	0.35	0.10	0.00			
55+	Person-years	471	1398	1356	1015	273	11	5				
	Observed	0	0	0	0	0	0	0				
	Expected	0.05		0.09	0.04	0.03	0.00	0.00				
Total	Person-years	8783	29296	43488	52651	47479	41166	38140	36225	28766	21300	14586
	Observed	1	0	3	2	5	12	12	14	24	26	21
	Annual death rate (per 100,000):											
	This study	11.39	0	6.90	3.80	10.53	29.15	31.46	38.65	83.43	122.06	143.98
	Peto (1982)	0	0	0	12.02	32.31	154.17	289.25	525.35	569.35	1080.72	664.45

TABLE 5.4: Devonport Dockyard. Distribution by time since first employment and age at first employment of 70 mesothelioma deaths.

Age first employed (yrs)		Years since first employment										
		0-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50+
15-24	Person-years	3213	10615	15853	19006	16842	13684	13269	12988	10891	8483	5912
	Observed	0	0	0	0	1	3	6	6	7	16	10
	Expected			1.30	1.30	2.58	5.11	6.16	4.53	8.30	15.86	10.55
25-34	Person-years	584	1896	2705	3541	3882	4200	4187	3585	2117	1135	250
	Observed	0	0	0	1	1	5	2	0	2	2	1
	Expected			0.22	0.24	0.60	1.57	1.94	1.25	1.61	2.12	0.45
35-44	Person-years	418	1517	2505	3474	3606	2875	1773	632	110	9	
	Observed	0	0	0	0	1	0	1	0	1	0	
	Expected			0.21	0.24	0.55	1.07	0.82	0.22	0.08	0.02	
45-54	Person-years	476	1800	2667	2751	1654	642	159	7			
	Observed	0	0	2	1	1	0	0	0			
	Expected			0.22	0.19	0.25	0.24	0.07	0.00			
55+	Person-years	223	635	593	440	112	2	5				
	Observed	0	0	0	0	0	0	0	Total number of person-years:			187,918
	Expected			0.05	0.03	0.02	0.00	0.00	Death rate (per 100,000):			37.25
Total	Person-years	4913	16463	24322	29213	26095	21404	19388	17211	13119	9626	6161
	Observed	0	0	2	2	4	8	9	6	10	18	11
	Annual death rate (per 100,000):											
	Devonport	0	0	8.22	6.85	15.33	37.88	46.42	34.86	76.23	186.98	178.54
	All yards	11.39	0	6.90	3.80	10.53	29.15	31.46	38.65	83.43	122.06	143.98

TABLE 5.5: Chatham Dockyard. Distribution by time since first employment and age at first employment of 23 mesothelioma deaths.

Age first employed (yrs)		Years since first employment										
		0-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50+
15-24	Person-years	870	3517	5282	6627	5745	4653	4516	4960	4766	3911	3119
	Observed	0	0	1	0	0	0	0	4	8	4	2
	Expected	0.61		0.66				0.70	2.87	7.25	4.32	1.92
25-34	Person-years	185	576	775	922	850	1179	1334	1692	1110	614	135
	Observed	0	0	0	0	0	0	1	0	1	1	0
	Expected	0.13		0.10				0.21	0.98	1.69	0.68	0.08
35-44	Person-years	111	478	681	827	812	697	522	254	42		
	Observed	1	0	0	0	0	0	0	0	0		
	Expected	0.08		0.08				0.08	0.15	0.06		
45-54	Person-years	162	624	829	843	445	159	41	2			
	Observed	0	0	0	0	0	0	0	0			
	Expected	0.11		0.10				0.01	0.00			
55+	Person-years	101	382	411	313	101	9	5				
	Observed	0	0	0	0	0	0	0	Total number of person-years:			66,191
	Expected	0.07		0.05				0.00	Death rate (per 100,00):			34.75
Total	Person-years	1430	5577	7977	9533	7953	6698	6417	6908	5919	4525	3254
	Observed	1	0	1	0	0	0	1	4	9	5	2
	Annual death rate (per 100,000):											
	Chatham	69.91	0	12.54	0	0	0	15.58	57.90	152.06	110.50	61.46
	All yards	11.39	0	6.90	3.80	10.53	29.15	31.46	38.65	83.43	122.06	143.98

TABLE 5.6: Portsmouth Dockyard. Distribution by time since first employment and age at first employment of 27 mesothelioma deaths.

Age first employed (yrs)		Years since first employment										
		0-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50+
15-24	Person-years	1422	4334	6925	8632	8467	7936	8060	8757	7932	6427	4991
	Observed	0	0	0	0	1	1	2	4	5	3	8
	Expected					0.63	2.43	1.31	2.89	4.08	2.70	7.72
25-34	Person-years	368	951	1325	1683	1886	2708	2869	2753	1643	722	179
	Observed	0	0	0	0	0	2	0	0	0	0	0
	Expected					0.14	0.83	0.46	0.91	0.84	0.30	0.28
35-44	Person-years	221	726	1258	1912	2124	2003	1291	591	153	1	
	Observed	0	0	0	0	0	1	0	0	0	0	
	Expected					0.16	0.61	0.21	0.19	0.08	0.00	
45-54	Person-years	282	863	1326	1417	895	416	114	4			
	Observed	0	0	0	0	0	0	0	0			
	Expected					0.07	0.13	0.02	0.00			
55+	Person-years	146	381	353	261	60						
	Observed	0	0	0	0	0						
	Expected					0.00						
									Total number of person-years:			107,771
									Death rate (per 100,00):			25.05
Total	Person-years	2439	7255	11188	13905	13431	13063	12335	12106	9728	7140	5170
	Observed	0	0	0	0	1	4	2	4	5	3	8
	Annual death rate (per 100,000):											
	Portsmouth	0	0	0	0	7.45	30.62	16.21	33.04	51.40	41.96	154.73
	All yards	11.39	0	6.90	3.80	10.53	29.15	31.46	38.65	83.43	122.06	143.98

This analysis excluded 'outstation' workers and was performed on 20,426 male in-yard workers followed from the early 1970s for 10 years. Their mortality experience was considered up to 49 years from first possible asbestos exposure. In all 193,560 person-years of observation were obtained and used in this model. Figure 5.1 shows the cumulative risk of dying from mesothelioma produced in this work. It has an identical form to figure 1 from Peto et al.[1] Signifying a similar pattern of risk between dockyard workers and insulation workers, though the risk is much smaller in the dockyard workers, i.e. the lifelong risk is 3% in dockyard workers compared to approximately 15% in insulation workers first employed at age 20 (where a lifetime risk of 3% means that 3 mesotheliomas will occur by age 80 in a cohort of 100 men followed to extinction).

TABLE 5.7: Estimates of the power parameter (k) and the constant of proportionality (b).

	Devonport	Chatham	Portsmouth
	For all time since first employment periods:		
Power parameter (k)	3.31	2.04	6.00
Constant (b)	3.96×10^{-9}	3.41×10^{-10}	6.75×10^{-14}
	For all 3 dockyards combined, $k = 3.06$, $b = 8.18 \times 10^{-9}$.		
	Omitting the first 3 periods:		
Power parameter (k)	3.33	1.24	6.00
Constant (b)	3.66×10^{-9}	7.64×10^{-6}	6.75×10^{-14}
	For all 3 dockyards combined, $k = 3.08$, $b = 7.57 \times 10^{-9}$.		

Over the 17 years of follow-up considered in this work 361,880 person-years of observation have accrued, with 120 deaths accumulated over the 3 dockyards from pleural and peritoneal mesothelioma. Table 5.3 shows the distribution of these deaths by time since first employment and age at first employment. The expected values from this table have been calculated on the assumption that mesothelioma

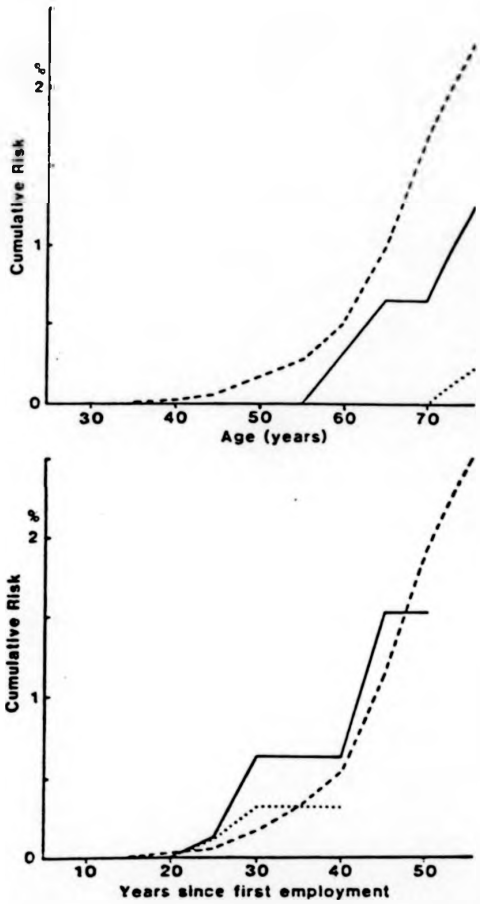


FIGURE 5.1*: Cumulative risk of dying of mesothelioma for workers first employed in the dockyards at age 15-24 (-----), 25-34 (——), or 35 and over (.....) against age (upper graph) and years since first employment (lower graph).

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* Taken from: Sullivan KR, Lam TH, Rossiter CE (1988) "Mesothelioma and Time Since First Employment" Ann occup Hyg 32:491-496.

mortality is independent of age, i.e. the expected numbers are calculated internally by multiplying the overall death rate in each quinquennium since first employment by the number of person-years in each cell in the corresponding column of table 5.3. The fit is very close between the observed and expected deaths (e.g. X^2 goodness of fit = 5.95, df = 9, P > 0.5, for those aged 15-24 at first employment) and the annual death rate is seen to rise analogously to the rate reported by Peto. Tables 5.4 to 5.6 show the distribution of mesothelioma deaths for each dockyard.

A model of the form outlined by Peto was then fitted to the data in table 5.3 to gain the best estimates of the parameters b and k for the 120 mesothelioma deaths. This was undertaken in GLIM using a method of minimisation of fitted deviance, i.e. by minimizing the likelihood-ratio statistic. Table 5.7 shows the estimates produced for each dockyard. The following illustrates the GLIM macro used:

```
$ MACRO MIN
$ CAL px = x**%a $
$ YVAR Y $
$ FIT $
$ FIT +px -%GM $
$ DISP R E $
$ ENDMAC
```

From this table it can be seen that a combined dockyard estimate of the power parameter over all time since first employment periods is given by: $k=3.06$ with an accompanying constant, $b=8.18\times10^{-9}$; this model excludes only those workers over the age of 85. The model was fitted to observed mesothelioma deaths beyond 50 years since first employment using 52.5 as an approximate mid-period value in the modelling. By inspection of tables 5.3 to 5.6 it is apparent that 4 mesothelioma deaths have occurred at very early follow-up periods. At Chatham dockyard a single mesothelioma death occurred with less than 5 years possible dockyard exposure. No previous occupational history was available for these 4 workers. If we assume a minimum latency period of at least 15 years from first exposure, then these 4 deaths should be excluded from a dockyard mesothelioma mortality model. Accordingly, the model parameters were recalculated omitting

the first 15 years since first employment. This resulted in revised estimates of: $k=3.08$ and $b=7.57\times10^{-9}$. It also dramatically increased the constant of proportionality, the measure of mesothelioma incidence, at Chatham.

These parameter estimates are very close, and are also close to the estimate given at 10 years of follow-up. A reasonable estimate of the parameters for dockyard mortality could, therefore, be given by: $k=3.1$ with $b=7.01\times10^{-9}$. This also avoids the spurious precision implied by a second decimal place in the estimates of the power parameter. Table 5.8 shows the effect of the various estimates in the calculation of an annual mesothelioma death rate 30 years after first employment (i.e. $t=30$, in each calculation).

TABLE 5.8: Estimates of an annual mesothelioma death rate.

	Power (k)	Relative incidence (b) $\times10^{9}$	Calculated death rate per 10,000
This study:			
Total follow-up	3.06	8.18	2.71
Minus first 15 years	3.08	7.57	2.68
Sullivan (1988):	3.20*	5.64	3.01
Peto (1982):	3.20	43.7	23.29
Dockyard estimate:	3.1	7.01	2.66

* Peto's value of 3.20 was used in place of the estimated value 3.14 in this publication, in the calculation of b.

From table 5.8 it is clear that the relative mesothelioma incidence, and indeed mesothelioma risk, in any particular cohort could be summarised by the constant b. From this table the insulation workers clearly show a higher risk than the dockyard workers (generated from the larger constant value). It is also clear that the dockyard models have produced compatible estimates of annual death rates, and that a model fitted with an exponential time-dependence appears to be appropriate for dockyard mesothelioma incidence (the fit of observed and expected

deaths were not significantly different at each dockyard). The model is also very robust and allows for the effect of mesothelioma latency, i.e. the removal or inclusion of the first 15 years of follow-up has minimal effect on the model parameters. An obvious limitation of this exponential model of mesothelioma incidence is, however, its lack of any measure of actual asbestos exposure. A model postulated by the World Health Organization modifies the exponential model and suggests a breakdown of the constant parameter (b) into two terms; one for level of asbestos (in fibres per millilitre) and one a constant term characteristic of asbestos type and distribution of fibre dimension.[7] Due to the lack of actual fibre levels in this study it was not possible to implement the WHO model.

The result of this mesothelioma modelling strongly supports the conclusion of Peto in 1982, that mesothelioma mortality is related to time since first employment, and first possible asbestos exposure, and is independent of age. However, the 'relative risk' parameter b is much lower than that observed by Peto for insulation workers and four other occupational groups (namely, asbestos factory workers, chrysotile factory workers, crocidolite miners and amosite factory workers). These cohorts will be considered in the following chapter. This can be linked with the results of chapter 4, and particularly that of no overall excess or deficiency of lung cancer deaths, and suggests that the dockyard workers may well have been exposed to significantly less asbestos than the other occupational groups considered by Peto.

Chapter 6: DISCUSSION AND CONCLUSION.

6.1 Discussion.

A common objective of occupational mortality studies is to assess whether mortality in a particular industry, or workforce, is higher than expected because of adverse exposures in the workplace. In this study a screening process was undertaken in which 50 broad disease groups were inspected. This was followed by an analysis of 12 specific disease groupings that were scrutinised for any possible trends in mortality related to dockyard asbestos exposure. Throughout, however, special emphasis has been given to the two main asbestos-related diseases, i.e. lung cancer and mesothelioma (and specifically pleural mesothelioma). The striking result found has been one of no overall excess in lung cancer mortality, together with excessively high mortality rates for both pleural and peritoneal mesothelioma. Asbestosis has shown high SMRs, but was based on too few observed deaths to allow sensible analysis. These results apply equally to all three dockyards, Devonport, Chatham and Portsmouth.

The only clear lung cancer dose-response found in this study, is its already well-documented relationship with cigarette smoking.[1,2] Additionally, an increasing lung cancer risk (with significant trend) was clearly observed across smoking habit for the workers in occupational group 4. This was the group created by Sheers and Templeton to represent potentially the lowest level of asbestos exposure, but as mentioned in section 3.1.4 these groups may in fact give ambiguous measures of asbestos exposure (with much worker interchange between groups).[3]

No obvious lung cancer relationship was seen with potential asbestos indicators (asbestos exposure rating, occupational group, etc.), or with most employment history factors. However, an indication of excess lung cancer mortality was observed, occurring at long intervals from first possible dockyard exposure (i.e.

for those workers with less than 20 years employment who had been first employed over 30 years ago). This agrees to some extent with the initial results reported on by McDonald for Quebec chrysotile miners and millers.[4] Here workers with 20 or more years employment (considered at least 20 years after start of employment) were seen to have clear excesses; these were the group of workers exposed to the heaviest asbestos dust concentrations. However, little overall excess lung cancer was observed in McDonald's study. Excess lung cancer risk was also observed for those workers with x-ray evidence of pulmonary fibrosis, giving some support to the suggestion of Browne that asbestosis is a precursor of lung cancer.[5] For those workers who assessed their own period of asbestos exposure (i.e. 3359 workers over all yards), a significant excess was seen only at Devonport dockyard. This was observed in workers with potentially 10 to 20 years of asbestos exposure. For the small subgroup of workers (i.e. 429 workers over all yards) who further assessed their period of continuous exposure to asbestos, significantly raised lung cancer risks were observed for those workers with more than 10 years continuous asbestos exposure; however, this was based on a very small number of deaths. These results are, however, suggestive that a subgroup of the dockyard workforce may have been exposed to high levels of asbestos dust some 20 to 30 years ago, producing the lung cancer excesses observed in this study.

Overall, these results do not give much support (except for a small subgroup, who may have been the heaviest exposed) to the hypothesis that dockyard asbestos exposure is linked aetiologically with lung cancer. In the majority of the analyses lung cancer risk was not raised significantly above that expected in the general population.

Conversely, pleural mesothelioma is seen to have no relationship at all with smoking habit, another well-documented fact[6], but is seen in this work to be related to possible asbestos exposure (specifically with asbestos exposure rating). This was a highly significant association, and could be taken as indicating that the

exposure codes used are relevant surrogate measures of exposure. Mesothelioma incidence is also seen to be related to time since first dockyard employment (and first possible dockyard asbestos exposure). Large excess mesothelioma risks were observed to be clustered among those workers with long follow-up and long duration of service (i.e predominantly for workers first employed before World War II). However, significant mortality trends were not observed in these groups. Dockyard mesothelioma incidence also appeared to be well related to a mortality model fitted with an exponential time-dependence. The overall picture is then one of a limited lung cancer risk, with smoking its predominant casual factor, and a high mesothelioma risk related to past asbestos exposure.

The results therefore confirm and strengthen the conclusions drawn from earlier studies undertaken at Devonport Dockyard.[7,8] The conclusions were that there is very little evidence of an overall excess mortality from lung cancer, despite a considerable number of mesothelioma deaths. This result is clearly not in complete accord with the other published studies of asbestos workers that either show excess mortality from both causes, or little excess from either. For example, the work of Hobbs in Australia for crocidolite miners and millers (excess in both), and McDonald in Quebec (little excess) for chrysotile miners and millers.[4,9] The asbestos studies of McDonald were extensively reviewed in chapter two.

From the literature review section of this dissertation (section 2.5), it can be seen that the results from this dockyard cohort are very striking, and at odds with the majority of published asbestos studies. Studies that have suggested that increasing lung cancer risks are clearly observed in cohorts exposed to amphibole asbestos, or mixtures of this with chrysotile asbestos; in the naval dockyards, however, all forms of asbestos (as itemised in chapter one) have been used over the years. Recent studies in and around dockyards have however produced further supporting evidence of a limited lung cancer risk, but high mesothelioma risk.[10,11] In the first of these studies Sanden et al undertook a cohort investigation of 3,893

Swedish shipyard workers.[10] Here the workers were exposed mainly to chrysotile asbestos, and were seen to have no increased risk of lung cancer (SMR of 108, 95%CI:54-109, for workers defined as heavily exposed to asbestos). For pleural mesothelioma the risk was highly elevated (SMR of 1429, 95%CI:620-2810, also for heavily exposed workers). Their results are very similar to the findings of this dockyard study. The conclusion from this Swedish study was that asbestos may have different carcinogenic mechanisms in causing lung cancer and mesothelioma. They felt that asbestos acts as a promoter of lung cancer, but as a complete carcinogen in developing mesothelioma. In the next study mortality from mesothelioma and asbestosis around Plymouth docks was analysed by the Small Area Health Statistics Unit (SAHSU) using a computerised national health monitoring facility.[11] In this work elevated risks were seen within 3 km of the docks. For mesothelioma a high SMR of 841 (95%CI:550-1230) was obtained based on 26 observed deaths, for asbestosis an SMR of 1364 (95%CI:500-2970) based on 6 deaths. This again supports the findings of this thesis.

Table 6.1 illustrates further studies, across varying asbestos industries, where no clear excess lung cancer risk has been observed. By inspection of this table the lowered dockyard risks for lung cancer generally correspond to a raised proportional mortality for mesothelioma. Suggesting that even without an excess lung cancer risk a very real asbestos-related disease problem exists in these workplaces. It should be noted, however, that for the studies cited in table 6.1 longer periods of follow-up may eventually show higher SMRs for lung cancer.

Could it be concluded that it is not asbestos but rather asbestosis that prepares the basis for subsequent malignancy? This was the first controversy considered in chapter two. Would this hypothesis fit the pattern seen in this study of neither an overall excess nor a deficiency of lung cancer deaths, linked with low numbers of asbestosis deaths, but with an excess risk of mesothelioma? We also have an excess of lung cancer deaths in workers with x-ray evidence of previous

TABLE 6.1: Asbestos cohort studies showing no increase in lung cancer risk.

Industry		Number of lung cancer deaths	SMR	Proportional mortality mesothelioma
Dockyard				
UK (Naval)	[7]	84	84	3.0%
UK (Civilian)	[12]	35	104	4.0%
USA	[13]	27	84	0.2%
Sweden	[10]	11	108	6.6%
Asbestos Cement				
Austria	[14]	49	104	0.7%
Belgium	[15]	21	94	0.5%
Sweden	[16]	11	123	-
UK	[17]	28	85	0.6%
UK	[18]	34	89	0.3%
UK	[19]	33	95	2.4%
Mining and Manufacture				
Italy	[20]	22	110	0.5%
USA	[21]	4	93	-

pulmonary fibrosis. For asbestosis to develop, high levels of asbestos exposure are needed, probably above 25 f/ml.[22] The results of this study would only support this hypothesis if it could be proved that dockyard asbestos exposures were less then this asbestosis threshold (i.e. if the exposures were only low enough to allow mesothelioma to develop and not asbestosis and then lung cancer). The problem is that this would be an attempt to prove a negative. A further problem occurs concerning the assumption that exposures should have been less than 25 f/ml. Reported dockyard exposure values from the 1970s considerably exceed this limit. Table 2.3 presented a summary of the available dockyard exposure data, and clearly the processes monitored have a wide variation of possible exposures (ranging from 0.05 to 3815 f/cc). However, in a further report it was stated that "even though all processes involving work with asbestos insulating materials in Naval Dockyards give rise to asbestos dust concentrations of at least 2 f/cc, most processes have dust concentrations of 50 f/cc or more".[23] It is likely therefore that exposures before the 1970s would have been at least as high as this and

probably higher. This, however, cannot be completely substantiated because exposure assessments prior to the work of Harries in the early 1970s were both rarely taken and often not consistently recorded. In fact the studies undertaken by Harries were performed in an attempt to give some idea of the potential past dust concentrations in the dockyard.[23] It consequently appears unlikely that dockyard exposures would be below the threshold value for asbestosis. However, it is possible that the intermittent nature of ship repair may play a role in this pattern of mortality. If it could be hypothesised that the workers were exposed to high peaks of exposure during rip-out operations, followed by low background levels the majority of the time, then their average exposures may possibly fall under this threshold limit. At the moment, however, the lung cancer - asbestosis question remains undecided for this dockyard cohort. The related question of whether there is a threshold dose of asbestos exposure below which asbestos is effectively non-carcinogenic to humans must also, for the same reasons, remain undecided. A recommendation of this work will be the creation of a detailed dockyard asbestos job-exposure matrix; once this is available more conclusive results should be possible.

It should be noted that McDonald and McDonald in a review of asbestos-related lung cancer also felt that despite the pathologic, experimental, radiologic and epidemiologic arguments put forward by Browne[5] (concerning the asbestosis - lung cancer controversy) the basic question, as considered in section 2.5.6 of this thesis, remains unanswered.[24] They stated that there appears to be no certain evidence that fibrogenesis and carcinogenesis in the lung are linked except to the extent that they sometimes share the same aetiologic agents.

A method of overcoming the lack of definitive asbestos fibre measurements presents itself in this study, i.e. the use of the mesothelioma modelling introduced in chapter five. In these models the parameter b, a constant of proportionality, supplies a measure of relative mesothelioma incidence. This in turn could be taken

as a simple measure of (or a guide to) asbestos exposure. The constant of proportionality could therefore be compared across studies to assess likely levels of incidence (and or asbestos exposure).

TABLE 6.2: Estimates of relative mesothelioma incidence.

Reference	Data used		Power parameter k	Constant b $\times 10^{-3}$
This thesis	Naval dockyard workers		3.1	7
Peto [25]	London textile workers	[26]	3.2	49
	American amosite factory	[27]		49
	American insulation workers	[28]		44
	Australian crocidolite miners	[9]		51
	Rochdale textile workers	[29]		29
Hughes [30] & Weill	American insulation workers	[28]	3.2	3
	Rochdale textile workers	[29]		1
	Ontario cement manufacturers	[31]		22
Ontario [22] Royal Commission	American insulation workers	[28]	4.0	0.13
	Rochdale textile workers	[29]		0.07
	Ontario cement manufacturers	[31]		0.21

Table 6.2 presents estimates of relative mesothelioma incidence across many asbestos cohorts (asbestos textile workers, crocidolite miners, asbestos cement workers, etc.). These cohorts have all been cited previously in this thesis. The different fibre types involved in the studies in table 6.2, the relative proportions of each, their dimensions, and the industrial process involved all possibly alter the carcinogenic effects of asbestos. Fibre dimensions and industrial processes are however undoubtedly related. When considering this information the modelling undertaken in table 6.2 appears too simplistic, producing a single value that can be compared across cohorts. However, Peto et al has shown that the most appropriate way of making comparisons statistically between studies is by describing incidence rates after adjustment for time from first exposure (i.e. this comparison technique).[25] When taking the Peto model from table 6.2 as standard it is clear that in absolute terms the dockyard workers mesothelioma risk is much less than that in the other cohorts. This implies that dockyard workers

may have been exposed to significantly less asbestos than the other occupational groups. When considering the modified model of Hughes and Weill (a model which has a fixed power parameter and included duration of exposure information) the results are not so clear, with the risks in insulation and textile workers being of the same order of magnitude as dockyard workers, but the risks among asbestos cement workers being much more. The model of the Ontario Royal Commission has been included for reference only, its use of the slightly larger power term, prohibits direct comparison. A conclusion of this comparison could therefore be that the risk in dockyard workers is either of the same order of magnitude, or indeed much less, than these found in the other cohorts. This is not a very useful result. However, when considering the results of this thesis, i.e. no clear lung cancer risk and a very clear mesothelioma risk, it is probable that the risk for dockyard workers (and their exposure) is much less than that observed in the other cohorts.

A major limiting factor in this study has been both the lack of detailed exposure data as presented earlier, and the use of a non-standard x-ray screening classification. As previously described in section 3.1.6 it is likely that this screening classification will have a sensitivity of approximately 70% when compared to the ILO U/C 1971 classification. Resulting in a possible underestimation of the prevalence rates of asbestos-related abnormalities presented in table 3.12. Recently the ILO classification itself has been criticised for underestimating asbestosis.[32] This study suggests that the sensitivity of the ILO method is between 80% and 90%; it would appear therefore that each method has its limitations. The advantage of the ILO classification however still holds. It is an internationally agreed method of detecting asbestosis and other forms of pneumoconiosis.

Controversy three (section 2.5.6) concerns the amphibole hypothesis or more correctly the question: is chrysotile asbestos less carcinogenic than the amphiboles? This question cannot be directly addressed in this study since the

workforce has potentially been exposed to both chrysotile and amphibole asbestos in many mixed forms (prior to 1968, as described in chapter one); with only very limited availability of asbestos fibre concentration in air measurements. The further use of a job-exposure matrix applied to this cohort, with details concerning type as well as amount of asbestos exposure, may address this issue. Numerous epidemiological studies (reviewed in section 2.5) have demonstrated increased lung cancer risk among past asbestos exposed workers; the evidence from these studies indicates that, except in the textile industry, where other exposures may play a role, the risk from chrysotile exposure is likely to be lower than that from amphiboles. There is no reason to suspect at present that this would be different in Naval Dockyards. Indeed an assumption could be that the lack of an overall lung cancer risk more likely reflects past chrysotile exposure than exposure to either crocidolite or amosite. The finding of Sanden et al of a deficiency of lung cancer cases in a cohort of chrysotile shipyard workers supports this assumption.[10]

6.2 Conclusion and Recommendations.

6.2.1 Conclusion.

The essential feature in preventing any asbestos related disease is, and has been, the control of the amount of asbestos to which individuals are exposed. It can be inferred that through control of dust levels in industry, medical surveillance of workers at risk, reduced use of asbestos and decreased cigarette smoking among exposed workers that asbestosis and related cancers will become increasingly rare. Opinion is that the risk of asbestosis is essentially nil at today's industry standards.[33] No such forecast has been made for mesothelioma, except conceivably for countries where the use of amphibole asbestos is heavily proscribed. For mesothelioma there appears to be no level of exposure to amphibole fibres that is acceptable. For these reasons nations worldwide have imposed standards that effectively prevent the current and future use of crocidolite and amosite.

Unfortunately, as this work has highlighted, in shipbuilding the damage has already taken place with the initial use and subsequent uncontrolled removal of material containing virtually every form of asbestos (but predominantly crocidolite and amosite). At the moment the incidence of asbestos related disease is still increasing worldwide, due to past conditions, and projections made in the 1970s have indicated that the incidence of asbestos-related industrial cancers will only start to decline around the year 2000.[34] However, more recent work has indicated that a further epidemic of asbestos-related disease concentrated around construction workers and present day operations, involving the removal of asbestos from buildings, will continue this increased incidence over the next 20 to 40 years.[35,36]

Recently the concept of a linear dose-response between asbestos exposure and lung cancer has been challenged, with support growing for the idea that a threshold

exists for asbestos related lung cancer.[24,37,38] This concept was considered in detail in section 2.5.6 of this thesis. It was noted that the dose-response may only have been observed in a mid-range of cumulative exposures and from this extrapolation has been made down to the low ambient environmental levels observed today. This has resulted in the 'one fibre can kill' theory and much public and media concern.[39] Information is clearly needed at both the low and high ends of the exposure scale to finally confirm the form of the lung cancer dose-response relationship. When considering lung cancer thresholds, the related question of whether lung cancer is simply a complication of asbestosis, or not, is frequently considered.[5,40] High asbestos dust levels are required for the development of asbestosis, so if asbestosis is a pre-requisite for an increased lung cancer risk, this would imply high past dust exposures.

In this study there is no overall significantly increased (or decreased) lung cancer risk, only a very few recorded asbestosis deaths, and an excess risk for both forms of mesothelioma (pleura and peritoneal). However, a radiographical prevalence of pulmonary fibrosis of 0.9% (i.e. 220 cases over the 3 dockyards) was observed. This would only support the hypothesis of lung cancer being a complication of asbestosis (and potentially also the asbestos lung cancer threshold hypothesis) if it could be assumed that past dockyard asbestos exposures were quite low; low enough generally only to produce mesothelioma. A problem occurs with this assumption, i.e. the fact that recorded dockyard exposures reached approximately 20,000 times the current UK limit for amosite and crocidolite, and also the observation that past dockyard working conditions were very dusty with men described as emerging from the compartments covered from head to foot in dust.[41,42] The intermittent nature of these exposures may help to explain these results, with sharp peaks of exposure followed by periods of possibly only limited background exposure, resulting in lower average exposures. Consequently the lung cancer - asbestosis question, and the related threshold question, remain unresolved; due simply to the lack of consistent and well recorded past asbestos fibre data. However, clearly a threshold below which lung cancer is effectively

non-carcinogenic would explain the observation seen here of an excess of mesothelioma, caused by potentially intermittent exposure, without an accompanying lung cancer excess. In consequence for dockyard workers, the idea of a lung cancer threshold is consistent but not proven conclusively.

It should also be noted that in mortality studies the frequency of asbestosis is likely to be underestimated, due to the difficulties involved in its diagnosis. Diagnosis is made on a history of asbestos exposure together with the clinical, physiological and radiographic features of this condition; each of which may be uncertain. More simply put, the absence of asbestos bodies in the sputum or on bronchopulmonary lavage are against a diagnosis of asbestosis. Accordingly, diagnosis can be made with a high degree of accuracy in advanced cases of the disease, but is uncertain and difficult in the early stages. If the frequency of asbestosis is underestimated this would obviously produce errors in any risk estimation, in particular at lower levels of exposure. In this work asbestosis has been seen to be in significant excess, however based on only 10 reported deaths over the three dockyards. It is possible that some misclassification has taken place with pulmonary fibrosis, resulting in lowered asbestosis SMRs. This would further obscure any relationship between lung cancer and asbestosis.

It should be noted that the establishment of dose-response relationships for asbestos exposure is beset with many problems. Among these should be included the long latent interval between initial exposure and evidence of an adverse effect, the features of which may be difficult to define: the unreliability of diagnosis leading to inexact death certification; the confounding effects of tobacco smoking; exposure to more than one type of asbestos each of which may have a different potential for producing an adverse effect; the inadequacy or even absence of data on past exposure; and until recently the crude methods in use for dust sampling and fibre identification and counting. Precision has, however, increased in epidemiological studies with the development and use of standardised questionnaires on respiratory symptoms and the use of international classifications

for chest radiographs. Exposure assessment, that is, dust sampling methods have been greatly improved; the early practice of recording total particle counts has given way to fibre counting and this has become more precise with improvements in microscopy: the use of the eye piece graticule; phase contrast illumination; membrane filters; and the introduction of transmission and scanning electron microscopy for fibre identification. The use of these new techniques for future dockyard surveillance programmes of all forms of mineral fibre must form one of the recommendations of this dissertation.

To conclude, this study has shown that lung cancer is generally neither significantly in excess, or in deficit, in this dockyard cohort. This implies that the overall lung cancer risk is no different for dockyard workers than for the general population. A subgroup of the cohort, some 429 workers (i.e. 1.5% of the cohort), did however assess their own period of continuous asbestos exposure. From this group elevated lung cancer risks were seen with more than 10 years continuous exposure; however, these were based upon only 14 deaths in total. A relationship was observed between lung cancer risk and smoking, with significant trends showing particularly for the occupational group consisting of 'all other dockyard workers'. Interestingly, this would have been the group with the least asbestos exposure according to the definition of Sheers and Templeton.[3] For mesothelioma, the opposite appears to be true, that is, no apparent relationship seen with smoking, but a significant relationship observed with the surrogate asbestos exposure variable (i.e. with asbestos exposure rating). It is concluded, therefore, that there is no overall excess risk of lung cancer among Naval Dockyard workers, however, there is a very high excess mesothelioma risk.

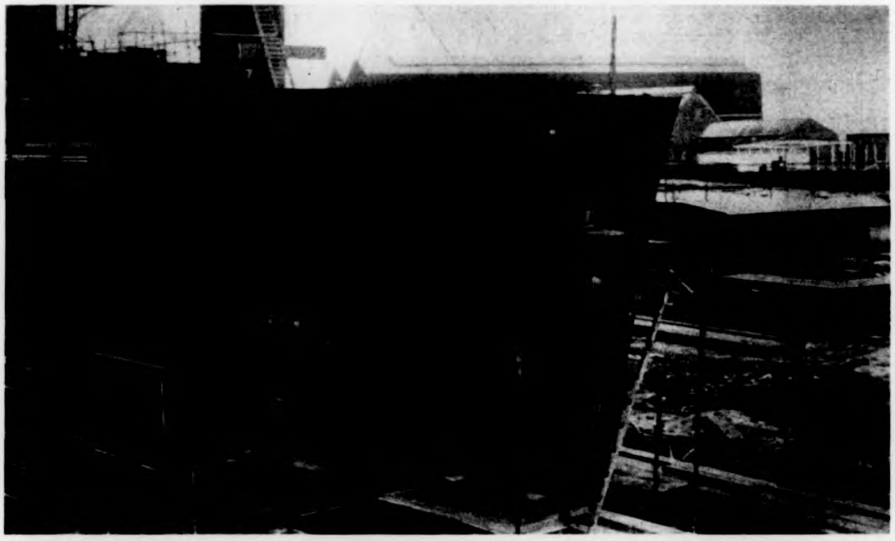

FIGURE 6.1: An active dockyard (circa 1989).

6.2.2 Recommendations.

The findings of this work, notably no clear excess or deficiency of lung cancer coupled with a very clear excess of mesothelioma needs to be confirmed or refuted. To this end further work is needed. The mortality experience of the dockyard workers continues to be recorded, and further follow-ups will be reported. However, these would still have the same limitations as this work: no definite asbestos exposure data (i.e. fibre counts in air); and the use of a non-standard radiographical classification. Ideally past asbestos exposures should be obtained and the x-rays re-read to the full ILO U/C classification. Clearly this would be almost impossible. The exposures were rarely recorded before the 1970s (and would have been static samples measuring particles, not personal samples measuring fibres)[23], and the radiographs are no longer available. A solution would be to investigate the current dockyard workforce. However, the work processes have changed dramatically over the last few decades with more mechanisation and the reduction in the number of workers (and the closure of the dockyards*), also asbestos has been removed from the yards and its use restricted.

A recommendation of this work, therefore, is for the future follow-up mortality studies to be supplemented, as far as possible, with all available work and exposure information. For this, current asbestos exposure information (mid 1970s - to date) should be collected, along with qualitative information (i.e. information derived from interviews with former dockyard workers, current workers employed some 20 years ago, etc.), for as many occupational codes (the codes listed in table 3.6) as is possible. This extra information is essential and should be used to supplement the asbestos exposure codes used in this thesis. From all of this a detailed job-exposure matrix should be created to replace the asbestos exposure rating used here.

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* With the closure of the naval dockyards, the scenes depicted in figures 6.1 and 6.2 of dockyard activity become simply a part of industrial history.

FIGURE 6.2: Dockyard workers.

Enough deaths are now beginning to accrue in the cohort for autopsy studies to be performed. A further recommendation would therefore be for these studies to be undertaken with the view to assessing asbestos lung burden, and characterising the fibre types and dimensions. As for example, in the work of Sebastien et al undertaken for two North American asbestos cohorts, where autopsy data was used to question the amphibole hypothesis considered in section 2.5 of this dissertation.[43] Similar work has in fact previously been undertaken at Devonport Dockyard, a necropsy study undertaken in 1986.[44] However, the recommendation here is for autopsy studies to be undertaken covering workers from all the Naval Dockyards. These studies would again be used to supplement further cohort analyses, with fibre information. It should be emphasised that these autopsy studies would only provide estimates of possible dose and not exposure.

It has been argued by McDonald et al that the risk of lung cancer in relation to asbestos exposure cannot be examined adequately by the subject-years method.[45] The authors stressed that it is becoming increasingly apparent that the linear relationships that have been found between SMRs and cumulative exposure are an oversimplification. They cite the work of Vacek and McDonald, in which a form of conditional logistic regression was used to create an exposure intensity model for assessing lung cancer risk among vermiculite miners[46]. This model assumed that exposure variables (such as exposure intensity, average duration of exposure, and average time since last exposure) had multiplicative effects on lung cancer risk and that the relative risk increased exponentially with exposure duration at a specified intensity.[47] The authors concluded that in order to access exposure response relationships from epidemiological data accurately, exposure intensity as well as duration must be taken into account. Without definite asbestos exposure counts this is not directly possible. However, the model created by Vacek and McDonald could still be applied, in a limited form, to the dockyard cohort. This would be a further recommendation.

One of the recommendations from this work would have been the controlled removal of asbestos from the dockyard environment. However, this process has been underway since the early 1970s, with asbestos being rapidly replaced by man-made mineral fibres (MMMFs). Mineral fibres that in their own right have been shown to be carcinogenic in laboratory animals.[48] Essentially it could be argued that the biological effects of MMMFs are the same as those produced by asbestos fibres, varying only in potency rather than in nature. In epidemiological studies conducted to date, there has been an excess of lung cancer observed in rockwool and slagwool production workers, but no significant excesses observed in glasswool or continuous filament production.[49,50,51] The excesses seen in the rockwool and slagwool industries were for workers heavily exposed in the earlier years of production when exposure levels were less well controlled. There have been no reports of mesothelioma in occupational groups without co-exposures to asbestos. William Bunn, a vice president of the famous American asbestos company, the Manville Corporation (formerly the Johns-Manville Corporation), has questioned the potential toxic consequences involved in the manufacture and use of man-made mineral fibres and has cautioned that they "invoke the sense that we have been here before"![52] If this is actually the case, it leads to a broad recommendation, that of, the highly controlled dockyard use of all mineral fibres, and strict adherence to codes of work practice already in place for asbestos.

Finally, the legacy of asbestos exposure continues even after the closure of the dockyards. Chatham and Portsmouth both closed in the early 1980s. The two remaining dockyards, Devonport and Rosyth, ceased to be operated by the Ministry of Defence in April 1987 when commercial management was introduced; at present Rosyth is threatened with closure. However, this work has shown that mesothelioma risk is currently in excess for the workers of these closed and closing dockyards. The closure of these Dockyards does not close the questions.

BIBLIOGRAPHY

Chapter 1.

1. Harries PG (1968) "Asbestos Hazards in Naval Dockyards" Ann Occup Hyg 11:135-145.

2. Harries PG (1971) "Asbestos Dust Concentrations in Ship Repairing: A Practical Approach to Improving Asbestos Hygiene in Naval Dockyards" Ann Occup Hyg 14:241-254.

3. Drinker P (1942) "Report to the Maritime Commission" U.S. National Archives, Record Group 178, Washington DC.

4. Roche JM (1942) "Report to the Maritime Commission" U.S. National Archives, Record Group 178, Washington DC.

5. Drinker P (1943) "The Health and Safety Program of the United States Navy" J A M A 121:822-823.

6. Corn JK, Starr J (1987) "Historical Perspective on Asbestos: Policies and Protective Measures in World War II Shipbuilding" Amer J industr Med 11:359-373.

7. Sheers G, Templeton AR (1968) "Effects of Asbestos in Dockyard Workers" Brit Med J 3:574-579.

8. Sheers G, Coles RM (1980) "Mesothelioma Risks in a Naval Dockyard" Arch Environ Health 35:276-282.

9. Rossiter CE, Coles RM (1980) "HM Dockyard Devonport: 1947 Mortality Study" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibres" IARC Scientific Publication No. 30, Lyon. pp713-721.

10. Elmes PC, Simpson MJC (1971) "Insulation Workers in Belfast. 3. Mortality 1940-66" Brit J industr Med 28:226-236.

11. Selikoff IJ, Hammond EC, Seidman H (1973) "Cancer Risk of Insulation Workers in the United States" In: Bogovski P, Gilson JC, Timbrell V, Wagner JC (eds.) "Biological Effects of Asbestos" IARC Scientific Publication No. 8, Lyon. pp209-216.

12. Harries PG (1971) "The Effects and Control of Diseases Associated with Exposure to Asbestos in Devonport Dockyard" Institute of Naval Medicine, CRWP Report No. 1/71, Gosport.

13. Blot WJ, Fraumeni JF (1981) "Cancer Among Shipyard Workers" In: Peto R, Schneiderman H (eds.) "Quantification of Occupational Cancer, Banbury Report No. 9" Cold Spring Harbor Laboratory. pp37-46.

1. Gilson JC (1966) "Health Hazards of Asbestos - Recent Studies on its Biological Effects" Trans Soc Occup Med **16**:62-68.

2. Wagner JC, Skidmore JW (1965) "Asbestos Dust Deposition and Retention in Rats" Ann N Y Acad Sci **132**:77-80.

3. Ontario Royal Commission (1984) "Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario" Ontario Ministry of the Attorney General, Toronto.

4. Churg A, Wright J, Wiggs B, Depaoli L (1990) "Mineralogic Parameters Related to Amosite Asbestos-Induced Fibrosis in Humans" Amer Rev Resp Dis **142**:1331-1336.

5. Wagner JC (1965) "The Sequelae of Exposure to Asbestos Dust" Ann N Y Acad Sci **132**:691-695.

6. Annual Report of the Chief Inspector of Factories and Workshops for 1898 (1899) HMSO, London.

7. Departmental Committee on Compensation for Industrial Diseases (1907) "Minutes of Evidence (given in 1906), Appendices and Index" Wyman & Sons, London.

8. Cooke WE (1924) "Fibrosis of the Lungs Due to the Inhalation of Asbestos Dust" Brit Med J 2:147.

9. Cooke WE (1927) "Pulmonary Asbestos" Brit Med J 2:1024-1025.

10. Seiler HE (1928) "A Case of Pneumoconiosis: Result of the Inhalation of Asbestos Dust" Brit Med J 3:982-983.

11. Merewether ERA, Price CW (1930) "Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry" HMSO, London.

12. Asbestos Industry Regulations (1931) "Statutory Rules and Orders, 1931, No. 1140" HMSO, London.

13. Merewether ERA (1930) "The Occurrence of Pulmonary Fibrosis and Other Pulmonary Affections in Asbestos Workers" J industr Hyg **12**:198-222,239-257.

14. Gloyne SR (1933) "The Morbid Anatomy and Histology of Asbestosis" Tubercle **14**:445-451,493-497,550-558.

15. Merewether ERA (1933-1934) "Memorandum on Asbestosis" Tubercle 15:69-81,109-118,152-159.

16. Wood WB, Gloyne SR (1934) "Pulmonary Asbestosis: A Review of 100 Cases" Lancet 2:1383-1385.

17. Lanza AJ, McConnell WJ, Fehnel JW (1935) "Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers" Publ Health Rep 50:1-12.

18. Lynch KM, Smith WA (1935) "Pulmonary Asbestosis III: Carcinoma of Lung in Asbesto-Silicosis" Amer J Cancer 24:56-64.

19. Gloyne SR, Merewether ERA (1938) "Asbestos" In: "Supplement to Occupation and Health. Encyclopedia of Hygiene, Pathology and Social Welfare" ILO, Geneva.

20. Dreessen WC, Dallavalle JM, Edwards TI, Miller JW, Sayers RR (1938) "A Study of Asbestosis in the Asbestos Textile Industry" Public Health Bull No. 241, U.S. Public Health Service, Washington, DC.

21. Sayers RR, Dreessen WC (1939) "Asbestosis" Amer J Publ Health 29: 205-214.

22. Fourth Schedule of Extension of Compensation for Industrial Accidents and Diseases (1943) Reichsgesetzblatt, Part 1, No. 14.

23. Wyers H (1946) "That Legislative Measures have proved generally effective in the Control of Asbestosis" Doctor of Medicine thesis, Glasgow University.

24. Wegelius C (1947) "Changes in the Lungs in 126 Cases of Asbestosis Observed in Finland" Acta Radiolog 28:139-152.

25. Smith LW (1949) "Pneumoconiosis and Lung Cancer" Compensation Med 2:3-10.

26. Annual Report of the Chief Inspector of Factories for 1947 (1949) HMSO, London.

27. Annual Report of the Chief Inspector of Factories for 1955 (1956) HMSO, London.

28. Doll R (1955) "Mortality from Lung Cancer in Asbestos Workers" Brit J industr Med 12:81-86.

29. Leathart GL (1960) "Clinical, Bronchographic, Radiological and Physiological Observations in 10 Cases of Asbestosis" Brit J industr Med 17:213-225.

30. Wagner JC, Sleggs CA, Marchand P (1960) "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province" Brit J industr Med **17**:260-271.

31. Hurwitz M (1961) "Roentgenolic Aspects of Asbestosis" Amer J Roentg **85**:256-259.

32. Leathart GL, Sanderson JT (1963) "Some Observations on Asbestosis" Ann Occup Hyg **6**:65-74.

33. Leathart GL (1965) "The Effect of Asbestosis on Pulmonary Function" Newcastle Med J **29**:30.

34. Leathart GL (1968) "Pulmonary Function Tests in Asbestos Workers" Trans Soc Occup Med **18**:49-55.

35. Selikoff IJ, Hammond EC, Churg J (1964) "Asbestosis and Neoplasia" J A M A **188**:22-26.

36. Selikoff IJ, Churg J, Hammond EC (1965) "The Occurrence of Asbestosis among Insulation Workers in the United States" Ann N Y Acad Sci **132**:139-155.

37. Selikoff IJ, Hammond EC, Churg J (1965) "Report and Recommendations of the Working Group on Asbestos and Cancer" Ann N Y Acad Sci **132**:706-721.

38. Enterline PE, Kendrick MA (1967) "Asbestos Dust Exposures at Various Levels and Mortality" Arch Environ Health **15**:181-186.

39. Memorandum of the Senior Medical Inspector's Advisory Panel (1967) "Problems arising from the use of Asbestos" HMSO, London.

40. Knox JF, Holmes S, Doll R, Hill ID (1968) "Mortality from Lung Cancer and Other Causes among Workers in an Asbestos Textile Factory" Brit J industr Med **25**:293-303.

41. Newhouse ML (1969) "A Study of the Mortality of Workers in an Asbestos Factory" Brit J industr Med **26**:294-301.

42. Selikoff IJ, Hammond EC, Churg J (1968) "Asbestos Exposure, Smoking, and Neoplasia" J A M A **204**:104-110.

43. Weiss W (1971) "Cigarette Smoking, Asbestos and Pulmonary Fibrosis" Amer Rev Resp Dis **104**:223-227.

44. British Occupational Hygiene Society Sub-committee on Asbestos (1968) "Hygiene Standards for Chrysotile Asbestos Dust" Ann Occup Hyg **11**:47-69.

45. McDonald JC, McDonald AD, Gibbs GW, Siemiatycki J, Rossiter CE (1971) "Mortality in the Chrysotile Asbestos Mines and Mills of Quebec" Arch Environ Health 22:677-686.

46. McDonald JC, Becklake MR, Fournier-Massey G, Rossiter CE (1972) "Respiratory Symptoms in Chrysotile Asbestos Mine and Mill Workers of Quebec" Arch Environ Health 24:358-363.

47. Rossiter CE, Bristol LJ, Cartier PH, Gilson JC, Grainger TR, Sluis-Cremer GK, McDonald JC (1972) "Radiographic Changes in Chrysotile Asbestos Mine and Mill Workers of Quebec" Arch Environ Health 24:388-400.

48. Becklake MR, Fournier-Massey G, Rossiter CE, McDonald JC (1972) "Lung Function in Chrysotile Asbestos Mine and Mill Workers of Quebec" Arch Environ Health 24:401-409.

49. Selikoff IJ, Hammond EC, Churg J (1972) "Carcinogenicity of Amosite Asbestos" Arch Environ Health 25:183-186.

50. Weill H, Waggenspack C, Bailey W, Ziskind M, Rossiter CE (1973) "Radiographic and Physiologic Patterns Among Workers Engaged in Manufacture of Asbestos Cement Products" J Occup Med 15:248-252.

51. Jones JSP, Sheers G (1973) "Pleural Plaques" In: Bogovski P, Gilson JC, Timbrell V, Wagner JC (eds.) "Biological Effects of Asbestos" IARC Scientific Publications No. 8, Lyon. pp243-248.

52. Webster I (1973) "Malignancy in Relation to Crocidolite and Amosite" In: Bogovski P, Gilson JC, Timbrell V, Wagner JC (eds.) "Biological Effects of Asbestos" IARC Scientific Publications No. 8, Lyon. pp195-198.

53. Newhouse ML, Berry G (1976) "Predictions of Mortality from Mesothelial Tumours in Asbestos Factory Workers" Brit J industr Med 33:147-151.

54. Peto J, Henderson BE, Pike MC (1981) "Trends in Mesothelioma Incidence in the United States and the Forecast Epidemic Due to Asbestos Exposure During World War II" In: Peto R, Schneiderman H (eds.) "Quantification of Occupational Cancer, Banbury Report No. 9" Cold Spring Harbor Laboratory. pp37-46.

55. Peto J, Seidman H, Selikoff IJ (1982) "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment" Brit J Cancer 45:124-135.

56. Peto J (1983) "Dose and Time Relationships for Asbestos-related Cancers, and Risk Assessment in the General Population" Vesein Deutsche Ingenieure Berichte 475:309-311.

57. Irwig LM, du Toit RSJ, Sluis-Cremer GK, Solomon A, Thomas GR, Hamel PPH, Webster I, Hastie T (1979) "Risk of Asbestosis in Crocidolite and Amosite Mines in South Africa" Ann N Y Acad Sci **330**:35-52.

58. Hammond EC, Selikoff IJ, Seidman H (1979) "Asbestos Exposure, Cigarette Smoking and Death Rates" Ann N Y Acad Sci **330**:473-490.

59. Berry G, Gilson JC, Holmes S, Lewinsohn HC, Roach SA (1979) "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory" Brit J industr Med **36**:98-112.

60. Letter to the Fleet Surgeon from Gilbert Kirker, Surgeon, 17 June 1891. Reprinted in: J Soc Occup Med **30**:139 (1980).

61. Lane F (1951) "Ships for Victory" The Johns Hopkins Press, Baltimore.

62. Bureau of Labor Statistics (1945) "Wartime Employment, Production and Conditions of Work in Shipyards" Bulletin No. 824, US Department of Labor, Washington, DC.

63. Kuhn A (1940) "Erkrankungen der Werftarbeiter der Kriegsmarinewerft Wilhelmshaven unter besonderer Berucksichtigung der Betriebsunfalle und Berufskrankheiten" Arch Gewerbepath Gewerbehyg **10**:133-150. Abstract printed in Bull Hyg **17**:818-821 (1942).

64. Dreessen WC, Fleischer WE (1944) "Report on Investigation of Asbestosis from Amosite Pipe Covering at Bath Iron Works, Bath, Maine" U.S. National Archives, Record Group 52, Washington, DC.

65. Letter from AW Garrett, HM Chief Inspector of Factories (August 1945) "To: The Shipbuilding and Shiprepairing Industry. Subject: Asbestos Insulation aboard Ships" Letter reprinted in: Institute of Naval Medicine, CRWP Report 1/71, Gosport. pp268-269.

66. Fleischer WE, Viles FJ, Gade RL, Drinker P (1946) "A Health Survey of Pipe Covering Operations in Constructing Naval Vessels" J industr Hyg Toxical **28**:9-16.

67. Drinker P (1947) "Health and Safety in Contract Shipyards during the War" Occup Med **3**:335-343.

68. Stoll R, Bass R, Angrist AA (1951) "Asbestos Associated with Bronchogenic Carcinoma" Arch Internal Med **88**:831-834.

69. Minimum Requirements for Safety and Industrial Health in Contract Shipyards (1943) Industr Med Surg **12**:259-263.

70. Walters JD (1959) "Asbestos: A Potential Hazard to Health in the Shipbuilding and Shiprepairing Industries" Diploma in Public Health thesis, London School of Hygiene and Tropical Medicine.

71. Morgan WKC (1964) "Rheumatoid Pneumoconiosis in Association with Asbestosis" Thorax **19**:433-435.

72. Marr WT (1964) "Asbestos Exposure During Naval Vessel Overhaul" Amer industr Hyg Assoc J **25**:264-268.

73. Murphy RLH, Ferris BG (1966) "Respiratory Disease in Workers Exposed to Low Concentrations of Asbestos Fibres" Proceedings of the 15th International Congress on Occupational Health, Vienna. pp229-232.

74. Ferris BG (1971) "Prevalence of Chronic Respiratory Disease - Asbestosis in Ship Repair Workers" Arch Environ Health **23**:220-225.

75. Murphy RLH, Ferris BG, Burgess WA, Worcester J, Gaensler EA (1971) "Effects of Low Concentrations of Asbestos. Clinical, Environmental, Radiologic and Epidemiologic Observations in Shipyard Pipe Coverers and Controls" New Eng J Med **285**:1271-1278.

76. Murphy RLH, Gaensler EA, Redding RA, Belleau R, Keelan PJ, Smith AA, Goff AM, Ferris BG (1972) "Low Exposure to Asbestos" Arch Environ Health **25**:253-264.

77. Anton HC (1967) "Multiple Pleural Plaques" Brit J Radiolog **40**:685-689.

78. Ashcroft T (1968) "Asbestos Bodies in Routine Necropsies on Tyneside: A Pathological and Social Study" Brit Med J **1**:614-618.

79. Sheers G, Templeton AR (1968) "Effects of Asbestos in Dockyard Workers" Brit Med J **3**:574-579.

80. James SAH (1971) "Asbestosis" Radiography **37**:81-85.

81. Horn A (1969) "Protective Measures for Working with Asbestos-Containing Materials in Shipbuilding" Arbeitsschutz **4**:81-84.

82. Stumphius J (1970) "Effects on Health of Exposure to Asbestos" De veiligheid **46**:49-53.

83. Stumphius J (1971) "Epidemiology of Mesothelioma on Walcheren Island" Brit J industr Med **28**:59-66.

84. Fletcher DE (1971) "Asbestos-Related Chest Disease in Joiners" Pro Royal Soc Med **64**:837-838.

85. Ahlman K, Siltanen E (1971) "Exposure of Insulation Workers to Asbestos Dust" Work Environment Health **8**:1-5

86. Whitwell F, Rawcliffe RM (1971) "Diffuse Malignant Pleural Mesothelioma and Asbestos Exposure" Thorax **26**:6-22.

87. Elmes PC, Simpson MJC (1971) "Insulation Workers in Belfast. 3. Mortality 1940-66" Brit J industr Med **28**:226-236.

88. Wallace WFM, Langlands JHM (1971) "Insulation Workers in Belfast. 1. Comparison of a Random Sample with a Control Population" Brit J industr Med **28**:211-216.

89. Langlands JHM, Wallace WFM, Simpson MJC (1971) "Insulation Workers in Belfast. 2. Morbidity in men still at work" Brit J industr Med **28**:217-225.

90. Cross AA (1972) "Practical Methods for Protection of Men Working with Asbestos Materials in Shipyards" In: "Safety and Health in Shipbuilding and Ship Repairing" ILO, Geneva.

91. Harries PG (1968) "Asbestos Hazards in Naval Dockyards" Ann Occup Hyg **11**:135-145.

92. Nicholson WJ, Holaday DA, Heimann H (1972) "Direct and Indirect Occupational Exposure to Insulation Dusts in United States Shipyards" In: "Safety and Health in Shipbuilding and Ship Repairing" ILO, Geneva.

93. Stumphius J (1976) "Cancer Resulting from Shipyard Work on Walcheren Island" World Conference on Health and Safety in the Metal Industry, Oslo, Norway, August 1976.

94. Blot WJ, Harrington JM, Toledo A, Hoover R, Heath CW, Fraumeni JF (1978) "Lung Cancer after Employment in Shipyards during World War II" New Eng J Med **299**:620-624.

95. Renke W, Graczyk M (1978) "Spirometric and Radiologic Estimation of Changes in the Respiratory System of Shipyard Workers Exposed to the Effect of Asbestos Dust during 4 Years of Observation" Bulletin of the Institute of Maritime and Tropical Medicine (Poland) **29**:43-48.

96. Murphy RLH, Gaensler EA, Ferris BG, Fitzgerald M, Solliday N, Morrisey W (1978) "Asbestosis: Observations from a Longitudinal Survey of Shipyard Pipe Coverers" Amer J Med **65**:488-498.

97. Blot WJ, Stone BJ, Fraumeni JF, Morris LE (1979) "Cancer Mortality in U.S. Counties with Shipyard Industries during World War II" Environ Res **18**:281-290.

98. Blot WJ, Fraumeni JF (1979) "Lung Cancer Mortality in the United States: Shipyard Correlations" Ann N Y Acad Sci **330**:313-315.

99. Blot WJ, Morris LE, Stroube R, Tagnon E, Fraumemi JF (1980) "Lung and Laryngeal Cancers in Relation to Shipyard Employment in Coastal Virginia" J Nat Cancer Inst **65**:571-575.

100. Tagnon E, Blot WJ, Stroube RB, Day NE, Morris LE, Peace B, Fraumeni JF (1980) "Mesothelioma Associated with the Shipbuilding Industry in Coastal Virginia" Cancer Res **40**:3875-3879.

101. Blot WJ, Fraumeni JF (1981) "Cancer among Shipyard Workers" In: Peto R, Schneiderman H (eds.) "Quantification of Occupational Cancer, Banbury Report No. 9" Cold Spring Harbor Laboratory. pp37-46.

102. Edge JR (1979) "Incidence of Bronchial Carcinoma in Shipyard Workers with Pleural Plaques" Ann N Y Acad Sci **330**:289-294.

103. Selikoff IJ, Nicholson WJ, Lilis R (1980) "Radiological Evidence of Asbestos Disease among Ship Repair Workers" Amer J industr Med **1**:9-22.

104. Boylen CT, Kilburn KH, Warshaw R, Johnson S (1983) "Asbestosis in Women who Worked in Shipyards" Paper given at the annual meeting of the American Thoracic Society. Abstract published in: Amer Rev Resp Dis (supplement) **127**:184.

105. Kilburn KH, Warshaw R, Thornton JC (1985) "Signs of Asbestosis and Impaired Pulmonary Function in Women who Worked in Shipyards" Amer J industr Med **8**:545-552.

106. Kilburn KH, Lilis R, Anderson HA, Boylen CT, Einstein HE, Johnson S, Warshaw R (1985) "Asbestos Disease in Family Contacts of Shipyard Workers" Amer J Pub Health **75**:615-617.

107. Kilburn KH, Warshaw R, Thornton JC (1986) "Asbestos Diseases and Pulmonary Symptoms and Signs in Shipyard Workers and their Families in Los Angeles" Arch Intern Med **146**:2213-2220.

108. Harries PG (1971) "A Comparison of Mass and Fibre Concentrations of Asbestos Dust in Shipyard Insulation Processes" Ann Occup Hyg **14**:235-240.

109. Harries PG (1971) "Asbestos Dust Concentrations in Ship Repairing: A Practical Approach to Improving Asbestos Hygiene in Naval Dockyards" Ann Occup Hyg **14**:241-254.

110. Harries PG (1971) "The Effects and Control of Diseases Associated with Exposure to Asbestos in Devonport Dockyard" Institute of Naval Medicine, CRWP Report 1/71, Gosport.

111. Harries PG (1973) "Experience with Asbestos Disease and Control in Great Britain's Naval Dockyards" Medical Research Unit, HM Dockyard (Devonport), Paper No. 19. Paper presented at the International Shipyard Health Conference, Los Angeles, December, 1973. Published in: Environ Res **11**:261-267 (1976).

112. Lumley KPS, Harries PG, O'Kelly FJ (1971) "Buildings Insulated with Sprayed Asbestos: A Potential Hazard" Ann Occup Hyg **4**:255-257.

113. Ministry of Defence (NAVY) (1970) "Working with Asbestos" Ministry of Defence Labour Division (Naval).

114. Harries PG, MacKenzie FAF, Sheers G, Kemp JH, Oliver TP, Wright DS (1972) "Radiological Survey of Men Exposed to Asbestos in Naval Dockyards" Brit J industr Med **29**:274-279.

115. Harries PG, Rossiter CE, Coles RM (1976) "Royal Naval Dockyards Asbestosis Research Project, Report No. 1, December 1975" Institute of Naval Medicine, CRWP Report 1/76, Gosport.

116. Lumley KPS (1976) "A Proportional Study of Cancer Registrations of Dockyard Workers" Brit J industr Med **33**:108-114.

117. Harries PG, Lumley KPS (1977) "Royal Naval Dockyards Asbestosis Research Project - Survey of Registered Asbestos Workers" J Royal Naval Med Serv **63**:133-148.

118. Rossiter CE, Harries PG (1979) "UK Naval Dockyards Asbestosis Study: Survey of the Sample Population aged 50-59 years" Brit J industr Med **36**:281-291.

119. Sheers G (1979) "Asbestos-Associated Disease in Employees of Devonport Dockyard" Ann N Y Acad Sci **330**:281-287.

120. Sheers G, Cole RM (1980) "Mesothelioma Risks in a Naval Dockyard" Arch Environ Health **35**:276-282.

121. Rossiter CE, Heath JR, Harries PG (1980) "Royal Naval Dockyards Asbestosis Research Project: Nine-year Follow-up Study of Men Exposed to Asbestos in Devonport Dockyard" J Royal Soc Med **73**:337-344.

122. Rossiter CE, Coles RM (1980) "HM Dockyard Devonport: 1947 Mortality Study" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibres" IARC Scientific Publication No. 30, Lyon. pp713-721.

123. McMillan GHG, Sheers G, Pethybridge RJ (1980) "Attack Rates of Pulmonary and Pleural Lesions Related to Exposure to Asbestos in Two Naval Dockyards" Institute of Naval Medicine, CRWP Report 23/80, Gosport.

124. McMillan GHG, Sheers G, Pethybridge RJ (1980) "Effect of Smoking on Attack Rates of Pulmonary and Pleural Lesions Related to Exposure to Asbestos Dust" Brit J industr Med **37**:268-272.

125. McMillan GHG, Rossiter CE (1982) "Development of Radiological and

Clinical Evidence of Parenchymal Fibrosis in Men with Non-malignant Asbestos-Related Pleural Lesions" Brit J industr Med 39:54-59.

126. McMillan GHG, Heath J (1979) "The Health of Welders in Naval Dockyards: Acute Changes in Respiratory Function during Standardized Welding" Ann Occup Hyg 22:19-32.

127. McMillan GHG, Molyneux MK (1981) "The Health of Welders in Naval Dockyards: The Work Situation and Sickness Absence Patterns" J Soc Occup Med 31:43-60.

128. McMillan GHG (1981) "The Health of Welders in Naval Dockyards: Welding, Tobacco Smoking and Absence Attributed to Respiratory Diseases" J Soc Occup Med 31:112-118.

129. McMillan GHG (1983) "The Health of Welders in Naval Dockyards: Final Summary Report" J Royal Naval Med Serv 69:125-131.

130. McMillan GHG, Pethybridge RJ (1983) "The Health of Welders in Naval Dockyards: Proportional Mortality Study of Welders and Two Control Groups" J Soc Occup Med 33:75-84.

131. McMillan GHG (1983) "The Health of Welders in Naval Dockyards: The Risk of Asbestos-Related Diseases Occurring in Welders" J Soc Occup Med 33:727-730.

132. McMillan GHG, Pethybridge RJ (1984) "A Clinical, Radiological and Pulmonary Function Case-Control Study of 135 Dockyard Welders Aged 45 and Over" J Soc Occup Med 34:3-23.

133. Wagner JC, Moncrieff CB, Coles RM, Griffiths DM, Munday DE (1986) "Correlation Between Fibre Content of the Lungs and Disease in Naval Dockyard Workers" Brit J industr Med 43:391-395.

134. Browne K (1986) "Is Asbestos or Asbestosis the Cause of the Increased Risk of Lung Cancer in Asbestos Workers" Brit J industr Med 43:145-149.

135. McDonald JC (1991) "Current Controversies in Occupational Lung Disease" British Occupational Hygiene Society, Annual Conference, Swansea, April 1991.

136. Mossman BT (1993) "Mechanisms of Asbestos Carcinogenesis and Toxicity: The Amphibole Hypothesis Revisited" Brit J industr Med 50:673-676.

137. Huncharek M (1994) "Asbestos and Cancer: Epidemiological and Public Health Controversies" Cancer Invest 12:214-222.

138. Mossman BT, Bignon J, Corn M, Seaton A, Gee JBL (1990) "Asbestos:

Scientific Developments and Implications for Public Policy" Science **247**:294-301.

139. Acheson ED, Gardner MJ (1979) "The Ill Effects of Asbestos on Health" In: Health and Safety Commission "Asbestos: Final Report of the Advisory Committee (volume 2)" HMSO, London. pp7-83.

140. Browne K (1991) "Asbestos Related Malignancy and the Cairns Hypothesis" Brit J industr Med **40**:73-76.

141. Peto J (1979) "Dose Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards: Part II, Mortality" Ann N Y Acad Sci **330**:195-203.

142. National Academy of Sciences (1984) "Asbestiform Fibers: Nonoccupational Health Risks" National Academy Press, Washington, DC.

143. United States Consumer Product Safety Commission (1983) "Report to the U.S. Consumer Product Safety Commission by the Chronic Hazard Advisory Panel on Asbestos" Directorate for Health Sciences, Washington, DC.

144. Doll R, Peto J (1985) "Effects on Health of Exposure to Asbestos. A Report to the Health and Safety Commission" HMSO, London.

145. Nicholson WJ (1986) "Airborne Asbestos Health Assessment Update" Office of Health and Environmental Assessment, U.S. Environmental Protection Agency (Report No. EPA/600/8-84/003F), Research Triangle Park, NC.

146. McDonald AD, McDonald JC (1987) "Epidemiology of Malignant Mesothelioma" In: Antman K, Aisnar J (eds.) "Asbestos-Related Malignancy" Grune and Stratton, London. pp31-55.

147. McDonald JC, McDonald AD (1987) "Epidemiology of Asbestos-Related Lung Cancer" In: Antman K, Aisnar J (eds.) "Asbestos-Related Malignancy" Grune and Stratton, London. pp57-79.

148. Health Effects Institute-Asbestos Research (1991) "Report of Literature Review Panel" Asbestos Control Report, Cambridge, MA.

149. McDonald JC (1990) "Cancer Risks due to Asbestos and Man-Made Fibres" Recent Results in Cancer Research **120**:122-131.

150. McDonald JC, Liddell FDK (1979) "Mortality in Canadian Miners and Millers Exposed to Chrysotile" Ann N Y Acad Sci **330**:1-9.

151. Amandus HE, Wheeler R (1987) "The Morbidity and Mortality of Vermiculite Miners and Millers exposed to Tremolite-Actinolite. Part II Mortality" Amer J industr Med **11**:15-26.

152. McDonald JC, McDonald AD, Armstrong B, Sebastien P (1986) "Cohort Study of Mortality of Vermiculite Miners Exposed to Tremolite" Brit J industr Med **43**:436-444.

153. Dement JM, Harris RL, Symons MJ, Shy CM (1982) "Estimates of Dose-response for Respiratory Cancer among Chrysotile Asbestos Textile Workers" In: Inhaled Particles V: Proceedings of an International Symposium, September 1980, Cardiff, Wales. Ann occup Hyg **26**:869-887.

154. McDonald AD, Fry JS, Woolley AJ, McDonald JC (1983) "Dust Exposure and Mortality in an American Chrysotile Textile Plant" Brit J industr Med **40**:361-367.

155. McDonald AD, Fry JS, Woolley AJ, McDonald JC (1983) "Dust Exposure and Mortality in an American Factory using Chrysotile, Amosite and Crocidolite in mainly Textile Manufacture" Brit J industr Med **40**:368-374.

156. Peto J, Doll R, Hermon C, Binns W, Clayton R, Goffe T (1985) "Relationship of Mortality to Measures of Environmental Asbestos Pollution in an Asbestos Textile Factory" Ann occup Hyg **29**:305-355.

157. Henderson VL, Enterline PE (1979) "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality" Ann N Y Acad Sci **330**:117-126.

158. Hughes JM, Weill H (1980) "Lung Cancer Risk Associated with Manufacture of Asbestos-Cement Products" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibres" IARC Scientific Publication No. 30, Lyon. pp627-635.

159. Finkelstein MM (1984) "Mortality Among Employees of an Ontario Asbestos-Cement Factory" Amer Rev Resp Dis **129**:754-761.

160. Albin M, Jakobsson K, Englander V, Ranstan J, Welinder H, Westrup C, Moller T (1984) "Mortality and Cancer Morbidity in a Cohort of Asbestos Cement Workers" In: VIth International Pneumoconiosis Conference (Volume 2), September 1983, Bochum, West Germany. pp825-829.

161. Berry G, Newhouse ML (1983) "Mortality of Workers Manufacturing Friction Materials using Asbestos" Brit J industr Med **40**:1-7.

162. McDonald AD, Fry JS, Woolley AJ, McDonald JC (1984) "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant" Brit J industr Med **41**:151-157.

163. Liddell FDK, McDonald JC, Thomas DC (1977) "Methods of Cohort Analysis: Appraisal by Application to Asbestos Mining" J Royal Stat Society (series A) **140**:469-491.

152. McDonald JC, McDonald AD, Armstrong B, Sebastien P (1986) "Cohort Study of Mortality of Vermiculite Miners Exposed to Tremolite" Brit J industr Med **43**:436-444.

153. Dement JM, Harris RL, Symons MJ, Shy CM (1982) "Estimates of Dose-response for Respiratory Cancer among Chrysotile Asbestos Textile Workers" In: Inhaled Particles V: Proceedings of an International Symposium, September 1980, Cardiff, Wales. Ann occup Hyg **26**:869-887.

154. McDonald AD, Fry JS, Woolley AJ, McDonald JC (1983) "Dust Exposure and Mortality in an American Chrysotile Textile Plant" Brit J industr Med **40**:361-367.

155. McDonald AD, Fry JS, Woolley AJ, McDonald JC (1983) "Dust Exposure and Mortality in an American Factory using Chrysotile, Amosite and Crocidolite in mainly Textile Manufacture" Brit J industr Med **40**:368-374.

156. Peto J, Doll R, Hermon C, Binns W, Clayton R, Goffe T (1985) "Relationship of Mortality to Measures of Environmental Asbestos Pollution in an Asbestos Textile Factory" Ann occup Hyg **29**:305-355.

157. Henderson VL, Enterline PE (1979) "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality" Ann N Y Acad Sci **330**:117-126.

158. Hughes JM, Weill H (1980) "Lung Cancer Risk Associated with Manufacture of Asbestos-Cement Products" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibres" IARC Scientific Publication No. 30, Lyon. pp627-635.

159. Finkelstein MM (1984) "Mortality Among Employees of an Ontario Asbestos-Cement Factory" Amer Rev Resp Dis **129**:754-761.

160. Albin M, Jakobsson K, Englander V, Ranstan J, Welinder H, Westrup C, Moller T (1984) "Mortality and Cancer Morbidity in a Cohort of Asbestos Cement Workers" In: VIth International Pneumoconiosis Conference (Volume 2), September 1983, Bochum, West Germany. pp825-829.

161. Berry G, Newhouse ML (1983) "Mortality of Workers Manufacturing Friction Materials using Asbestos" Brit J industr Med **40**:1-7.

162. McDonald AD, Fry JS, Woolley AJ, McDonald JC (1984) "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant" Brit J industr Med **41**:151-157.

163. Liddell FDK, McDonald JC, Thomas DC (1977) "Methods of Cohort Analysis: Appraisal by Application to Asbestos Mining" J Royal Stat Society (series A) **140**:469-491.

164. Gaensler EA (1992) "Asbestos Exposure in Buildings" Clin Chest Med **13**:231-242.

165. Newhouse ML, Sullivan KR (1989) "A Mortality Study of Workers Manufacturing Friction Materials: 1941-86" Brit J industr Med **46**:176-179.

166. Dement JM (1991) "Carcinogenicity of Chrysotile Asbestos: A Case Control Study of Textile Workers" Cell Biology and Toxicology **7**:59-65.

167. Sebastien P, McDonald JC, McDonald AD, Case B, Harley B (1989) "Respiratory Cancer in Chrysotile Textile and Mining Industries: Exposure Inferences from Lung Analysis" Brit J industr Med **46**:180-187.

168. McDonald AD, McDonald JC (1978) "Mesothelioma after Crocidolite Exposure during Gasmask Manufacture" Environ Research **17**:340-346.

169. Jones JSP, Smith PG, Pooley FD, Berry G, Sawle GW, Aggarwal A, Wignall BK, Madeley RJ (1980) "The Consequences of Exposure to Asbestos Dust in Wartime Gasmask Factory" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibres" IARC Scientific Publication No. 30, Lyon. pp637-653.

170. Acheson E, Gardner MJ, Pipard EC, Grime LP (1982) "Mortality of Two Groups of Women who Manufactured Gasmasks from Chrysotile and Crocidolite Asbestos: A 40 year follow-up" Brit J industr Med **39**:344-348.

171. Churg A (1988) "Chrysotile, Tremolite and Malignant Mesothelioma in Man" Chest **93**:621-628.

172. Wagner JC, Pooley FD, Berry G, Seal RME, Munday DE, Morgan J, Clark NJ (1982) "A Pathological and Mineralogical Study of Asbestos-Related Deaths in the United Kingdom" Ann occup Hyg **26**:417-422.

173. McDonald JC (1980) "Asbestos Related Disease: An Epidemiological Review" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibres" IARC Scientific Publication No. 30, Lyon. pp587-601.

174. Berry G, Newhouse ML, Antonis P (1985) "Combined Effects of Asbestos and Smoking on Mortality from Lung Cancer and Mesothelioma in Factory Workers" Brit J industr Med **42**:12-18.

175. Frank AL (1979) "Public Health Significance of Smoking-Asbestos Interaction" Ann N Y Acad Sci **330**:791-794.

176. Antman KH (1993) "Natural History and Epidemiology of Malignant Mesothelioma" Chest **103**(supplement):373S-376S.

177. Baris I, Simonato L, Artvinli M, Pooley F, Saracci R, Skidmore J, Wagner C (1987) "Epidemiological and Environmental Evidence of the Health

Effects of Exposure to Erionite Fibers: A Four-Year Study in the Cappadocian Region of Turkey" Inter J Cancer **39**:10-17.

178. Pelnar PV (1988) "Further Evidence of Nonasbestos-Related Mesothelioma. A Review of the Literature" Scand J Work Environ Health **14**:141-144.

179. McDonald JC (1985) "Health Implications of Environmental Exposure to Asbestos" Environ Health Perspect **62**:319-324.

180. Pooley FD, Clark NJ (1980) "A Comparison of Fibre Dimension in Chrysotile, Crocidolite, and Amosite Particles from Samples of Airborne Dust and from Post-Mortem Lung Tissue Specimens" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibre" IARC Scientific Publication No. 30, Lyon. pp79-86.

181. Berman W, Chatfield EJ (1989) "Interim Superfund Method for the Determination of Asbestos in Ambient Air. Part II: Technical Background Document" United States Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, DC.

182. HSE Occupational Medicine and Hygiene Laboratories (1984) "Asbestos Fibres in Air: Determination of Personal Exposure by the European Reference Version of the Membrane Filter Method" Methods for the Determination of Hazardous Substances, MDHS 39.

183. HSE Occupational Medicine and Hygiene Laboratories (1988) "Asbestos Fibres in Air: Light Microscope Methods for use with the Control of Asbestos at Work Regulations" Methods for the Determination of Hazardous Substances, MDHS 39/2.

184. HSE Occupational Medicine and Hygiene Laboratories (1990) "Asbestos Fibres in Air: Light Microscope Methods for use with the Control of Asbestos at Work Regulations" Methods for the Determination of Hazardous Substances, MDHS 39/3.

185. Lippmann M (1988) "Review: Asbestos Exposure Indices" Environ Res **46**:86-106.

186. Lippmann M (1992) "Asbestos and other Mineral Fibres" In: Lippmann M (ed.) "Environmental Toxicants. Human Exposures and their Health Effects" Van Nostrand Reinhold Co., New York. pp30-75.

187. McDonald JC, Armstrong B, Case B, Doell D, McCaughey WTE, McDonald AD, Sebastien P (1989) "Mesothelioma and Asbestos Fiber Type. Evidence from Lung Tissue Analyses" Cancer **63**:1544-1547.

188. Timbrell V (1973) "Physical Factors as Etiological Mechanisms" In: Bogovski P, Gilson JG, Timbrell V, Wagner JC (eds.) "Biological Effects of Asbestos" IARC Scientific Publication No. 8, Lyon. pp295-303.

189. Walton WH (1982) "The Nature, Hazards and Assessment of Occupational Exposure to Airborne Asbestos Dust: A Review" Ann occup Hyg 25:117-247.

190. Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD (1986) "Inhalation and Injection Studies in Rats using Dust Samples from Chrysotile Asbestos Prepared by a Wet Dispersion Process" Brit J Exp Pathol 67:113-129.

191. Burdett GJ, Jaffrey SAMT, Rood AP (1989) "Airborne Asbestos Fibre Levels in Buildings: A Summary of UK Measurements" In: Bignon J, Peto J, Saracci R (eds.) "Non-Occupational Exposure to Mineral Fibres" IARC Scientific Publication No. 90, Lyon. pp277-290.

192. Peto J (1994) "An Epidemic of Mesothelioma Deaths" Talk given at the London School of Hygiene and Tropical Medicine, 29th April 1994.

193. Levy BS, Seplow A (1992) "Asbestos-Related Hazards in Developing Countries" Environ Res 59:167-174.

194. Mukherjee AK, Rajmohan HR, Dave SK, Rajan BK, Raghavendra RS (1992) "An Environmental Survey in Chrysotile Asbestos Milling Processes in India" Amer J industr Med 22:543-551.

195. Peto J (1989) "Fibre Carcinogenesis and Environmental Hazards" In: Bignon J, Peto J, Saracci R (eds.) "Non-Occupational Exposure to Mineral Fibres" IARC Scientific Publication No. 90, Lyon. pp457-470.

196. Britton M (1990) "Geographic Variation in Mortality, 1979-83" In: Britton M (ed.) "Mortality and Geography: A Review in the mid-1980s. England and Wales" HMSO, London. pp18-26.

197. Wagner JC, Berry G, Timbrell V (1973) "Mesothelioma in Rats after Inoculation with Asbestos and Other Materials" Brit J Cancer 28:173-185.

198. Wagner JC, Berry G, Skidmore JW, Timbrell V (1974) "The Effects of Inhalation of Asbestos in Rats" Brit J Cancer 29:252-261.

199. Davis JMG, Beckett ST, Bolton RE, Collings PF, Middleton AP (1978) "Mass and Number of Fibres in the Pathogenesis of Asbestos-Related Lung Disease in Rats" Brit J Cancer 37:673-688.

200. Price-Jones MJ, Gubbings G, Chamberlain (1980) "The Genetic Effects of Crocidolite Asbestos: Comparison of Chromosome Abnormalities and Sister Chromatid Exchanges" Mutat Res 79:331-342.

201. Light WG, Wei ET (1980) "Surface Charge and a Molecular Basis for Asbestos Toxicity" In: Brown RC, Gormley IP, Chamberlain M, Davis R (eds.) "The In Vitro Effects of Mineral Dusts" Academic Press, London. pp244-251.

202. Miller EC, Miller JA (1981) "Mechanisms of Chemical Carcinogenesis" Cancer **47**:1055-1064.

203. Boyland E (1985) "Tumour Initiators, Promoters and Complete Carcinogens" Brit J industr Med **42**:716-720.

204. Liddell FDK, McDonald JC (1980) "Radiologic Findings as Predictors of Mortality in Quebec Asbestos Workers" Brit J industr Med **37**:257-263.

205. Hughes JM, Weill H (1991) "Asbestosis as a Precursor of Asbestos-Related Lung Cancer: Results of a Prospective Mortality Study" Brit J industr Med **48**:229-233.

206. Jacob G, Anspach M (1965) "Pulmonary Neoplasia Among Dresden Asbestos Workers" Ann N Y Acad Sci **132**:536-548.

207. Kannersteing M, Churg J (1972) "The Pathology of Carcinoma of the Lung Associated with Asbestos Exposure" Cancer **30**:14-21.

208. Whitwell F, Newhouse ML, Bennett DR (1974) "A Study of the Histological Cell Types of Lung Cancer in Workers Suffering from Asbestosis" Brit J industr Med **31**:298-303.

209. Warnock ML, Churg A (1975) "Association of Asbestos and Bronchogenic Carcinoma in a Population with Low Asbestos Exposure" Cancer **35**:1236-1242.

210. Sluis-Cremer GK (1980) "The Relationship Between Asbestosis and Bronchial Cancer" Chest **78**:380-381.

211. Newhouse ML, Berry G, Wagner JC (1985) "Mortality of Factory Workers in East London, 1933-80" Brit J industr Med **42**:4-11.

212. Turner-Warwick M (1978) "Immunology of the Lung" Edward Arnold, London.

213. Becklake MR (1980) "Asbestos-Related Diseases of the Lungs and Pleura" Amer Rev Respir Dis **126**:187-194.

214. Schmachl DFK (1983) "Carcinogenic Aspects of Asbestos" In: "Proceedings of the World Symposium on Asbestos" Canadian Asbestos Information Centre, Montreal.

215. Mossman BT, Craighead JE, MacPherson BV (1980) "Asbestos Induced Epithelial Changes in Organ Cultures of Rat Trachea: Inhibition of Retinyl Methyl Ether" Science **207**:311-316.

216. Mossman BT, Craighead JE (1981) "Mechanisms of Asbestos Carcinogenesis" Environ Res **25**:269-274.

217. Mossman BT, Eastman A, Landesman JM, Bresnick E (1983) "Effects of Crocidolite and Chrysotile Asbestos on Cellular Uptake and Metabolism of Benzo(a)pyrene in Hamster Tracheal Epithelial Cells" Environ Health Perspect **51**:331-340.

218. Roggli VL, Pratt PC, Brody AR (1986) "Asbestos Content of Lung Tissue in Asbestos Associated Disease: A Study of 110 Cases" Brit J industr Med **43**:18-29.

219. Seidman H, Selikoff IJ, Hammond EC (1979) "Short-Term Asbestos Work Exposure and Long-Term Observation" Ann N Y Acad Sci **330**:61-89.

220. Ives JC, Buffler PA, Greenberg SD (1983) "Environmental Associations and Histopathologic Patterns of Carcinoma of the Lung: The Challenge and Dilemma in Epidemiologic Studies" Amer Rev Respir Dis **128**:187-195.

221. Browne K (1986) "A Threshold for Asbestos Related Lung Cancer" Brit J industr Med **43**:556-558.

222. Thomas HF, Benjamin IT, Elwood PC, Sweetnam PM (1982) "Further Follow-up Study of Workers from an Asbestos Cement Factory" Brit J industr Med **39**:273-276.

223. Farebrother MJB, Heller RF, O'Brien I, Azzoparde A, Telfa TP, Young M (1983) "Occupation and Lung Cancer in a Naval Dockyard" Thorax **38**:225.

224. Newhouse ML, Oakes D, Woolley AJ (1985) "Mortality of Welders and Other Craftsmen at a Shipyard in NE England" Brit J industr Med **42**:406-410.

225. Hodgson JT, Jones RD (1986) "Mortality of Asbestos Workers in England and Wales 1971-81" Brit J industr Med **43**:158-165.

226. Pampalon R, Siemiatycki J, Blanchet M (1982) "Environmental Asbestos Pollution and Public Health in Quebec" Union Med Can **111**:475-480.

227. Churg A (1986) "Lung Asbestos Content in Long-Term Residents of a Chrysotile Mining Town" Amer Rev Respir Dis **134**:125-132.

228. Armstrong B (1990) "Effects of Measurement Errors on Estimates of Exposure-Response Relationships" Recent Results in Cancer Research **120**:50-63.

229. Dement JM, Harns RL, Symons MJ, Shy CM (1983) "Exposures and Mortality Among Chrysotile Asbestos Workers. Part II: Mortality" Amer J industr Med **4**:421-433.

230. Cookson WOCM, de Klerk NH, Musk AW, Armstrong BK, Glancy JJ, Hobbs MST (1986) "Prevalence of Radiographic Asbestosis in Crocidolite

Miners and Millers at Wittenbom, Western Australia" Brit J industr Med **43**:450-457.

231. Lamm SH, Levine MS, Starr JA, Tirey SL (1988) "Analysis of Excess Lung Cancer Risk in Short Term Employees" Amer J Epidem **127**:1202-1209.

232. Sluis-Cremer GK, Hnizdo E, du Toit RSJ (1990) "Evidence for an Amphibole Asbestos Threshold Exposure for Asbestosis Assessed by Autopsy in South African Asbestos Miners" Ann occup Hyg **34**:443-451.

233. Ilgren EB, Browne K (1991) "Asbestos-Related Mesothelioma: Evidence for a Threshold in Humans and Animals" Reg Toxicol Pharm **13**:116-132.

234. Browne K (1986) "Asbestos-Related Disease and the 'One Fibre' Theory" Occup Health Rev, Oct/Nov:11-13.

235. NIOSH (1990) "NIOSH Testimony on Abestos by R Lemen, April 26, 1990".

236. Weiss W (1977) "Mortality of a Cohort Exposed to Chrysotile Asbestos" J occup Med **19**:737-740.

237. Ohlson CG, Hogstedt C (1985) "Lung Cancer Among Asbestos Cement Workers. A Swedish Cohort Study and a Review" Brit J industr Med **42**:397-402.

238. Rowlands N, Gibbs GW, McDonald AD (1982) "Asbestos Fibres In The Lungs Of Chrysotile Miners And Millers - A Preliminary Report" Ann Occup Hyg **26**:411-415.

239. Berry G (1986) "Chrysotile and Mesothelioma" In: Wagner JC (ed.) "Accomplishments in Oncology, Vol 1, No. 2, The Biological Effects of Chrysotile" JB Lippincott Co., Philadelphia. pp123-132.

240. NIOSH (1980) "Workplace Exposure to Asbestos. Review and Recommendations" US Department of Health and Human Services.

241. Dunnigan J (1988) "Linking Chrysotile Asbestos with Mesothelioma" Amer J industr Med **14**:205-209.

242. Rossiter CE (1994) A personal communication.

243. WHO (1992) "Validity of Methods for Assessing the Carcinogenicity of Man-Made Fibres. Executive Summary of a WHO Consultation, 19-20 May 1992, Copenhagen" World Health Organization Regional Office for Europe, Copenhagen.

244. Ramazzini B (1713) "De Morbis Artificum Diatriba" Translated by: Wright WC (1940) University of Chicago Press, Chicago.

Chapter 3.

1. Harries PG, Rossiter CE, Coles RM (1976) "Royal Naval Dockyards Asbestosis Research Project, Report No. 1, December 1975" Institute of Naval Medicine, CRWP Report 1/76, Gosport.

2. Sheers G, Templeton AR (1968) "Effects of Asbestos in Dockyard Workers" Brit Med J 3:574-579.

3. Rossiter CE, Coles RM, Jackaman I (1984) "HM Naval Base, Devonport: Lung Cancer and Mesothelioma Case Control Studies" In: Proceedings of the VIth International Pneumoconiosis Conference, Bochum, 1983. Bochum, Bergbau-Berufgenossenschaft, p830-836.

4. Rossiter CE (1993) A personal communication.

5. Harries PG (1994) A personal communication.

6. Harries PG, MacKenzie FAF, Sheers G, Kemp JH, Oliver TP, Wright DS (1972) "Radiological Survey of Men Exposed to Asbestos in Naval Dockyards" Brit J industr Med **29**:274-279.

7. International Labour Office (1972) "The ILO U/C International Classification of Radiographs of the Pneumoconioses, 1971" ILO Occupational Safety and Health Series No. 22, Geneva.

8. Anonymous (1975) "Radiological Methods in the Surveillance of Workers Exposed to Asbestos" Institute of Naval Medicine, CRWP Repot No. 4/75, Gosport.

9. Sheers G, Rossiter CE, Gilson JC, MacKenzie FAF (1978) "UK Naval Dockyards Asbestosis Study: Radiological Methods in the Surveillance of Workers Exposed to Asbestos" Brit J industr Med **35**:195-203.

10. Berkson J, Gage RP (1950) "Calculation of Survival Rates for Cancer" Proceedings of the Staff of the Mayo Clinic 25:270-286.

11. Mauser JS, Bahn AK (1974) "Epidemiology: An Introductory Text" W B Saunders, Philadelphia.

12. Hill ID (1972) "Computing Man Years at Risk" Brit J Prev Soc Med 26:132-134.

13. Elandt-Johnson RC, Johnson NL (1980) "Survival Models and Data Analysis" John Wiley & Sons, New York.

14. Doll R (1955) "Mortality from Lung Cancer in Asbestos Workers" Brit J industr Med **12**:81-86.

15. Liddell FDK (1960) "The Measurement of Occupational Mortality" Brit J industr Med **17**:228-233.

16. Berry G (1983) "The Analysis of Mortality by the Subject-Years Method" Biometrics **39**:173-184.

17. Gilbert ES (1983) "An Evaluation of Several Methods for Assessing the Effects of Occupational Exposure to Radiation" Biometrics **39**:161-171.

18. McMicheal AJ (1976) "Standardised Mortality Ratios and the Healthy Worker Effect" J Occup Med **18**:165-168.

19. Armitage P, Berry G (1987) "Statistical Methods in Medical Research" Blackwell Scientific Publications, Oxford.

20. Breslow NE, Day NE (1987) "Statistical Methods in Cancer Research. Volume II - The Design and Analysis of Cohort Studies" IARC Scientific Publications No. 82, Lyon.

21. Clayton, DG (1982) "Fitting a General Family of Failure-Time Distributions using GLIM" Applied Stat **32**:107-109.

22. Breslow NE, Lubin JH, Marek P, Langholz B (1983) "Multiplicative Models and Cohort Analysis" J Amer Stat Assoc **78**:1-12.

23. Cox DR (1972) "Regression Models and Life Tables" J Royal Stat Soc (B) **34**:187-220.

24. Holford TR (1980) "The Analysis of Rates and Survivorship using Log-linear Models" Biometrics **36**:299-306.

25. Bishop YMM, Fienberg SE, Holland PW (1975) "Discrete Multivariate Analysis: Theory and Practice" MIT Press, Cambridge, MA.

26. Baker RJ, Nelder JA (1978) "The GLIM System Release 3. Generalised Linear Interactive Modelling" NAG, Oxford.

27. Miettinen OS (1976) "Estimability and Estimation in Case-Referent Studies" Amer J Epidemiol **103**:226-235.

28. Rothman KJ (1986) "Modern Epidemiology" Little Brown & Co, Boston, MA.

29. Ogle W (1885) "Letter to the Registrar-General on the Mortality in the Registration Districts of England and Wales during the ten years 1871-80" In:"Supplement to the 45th Annual Report of the Registrar General of Births, Deaths, and Marriages, in England, 1885" p. xxiii.

30. Fox AJ, Collier FF (1976) "Low Mortality Rates in Industrial Cohort

Studies due to Selection for Work and Survival in the Industry" Brit J Prev Soc Med; 30:225-230.

31. Gilbert ES (1982) "Some Confounding Factors in the Study of Mortality and Occupational Exposures" Amer J Epidemiol **116**:177-188.

32. Fox AJ, Goldblatt PO (1982) "Healthy Worker Effect" In: "OPCS Longitudinal Study. Socio-demographic differentials" Series LS No. 1, HMSO, London.

33. Vinni K, Hakrama M (1980) "Healthy Worker Effect in the Total Finnish Population" Brit J Indust Med **37**:180-184.

34. Olsen J, Sabroe S (1979) "Health Selection among Members of a Danish Trade Union" Inter J Epidemiol **8**:155-159.

35. Wen CP, Tsai SP (1982) "Anatomy of the Healthy Worker Effect - A Critique of Summary Statistics Employed in Occupational Epidemiology" Scand J Work Environ Health; **8**(Suppl 1):48-52.

36. Hernberg S (1980) "Evaluation of Epidemiologic Studies in Assessing the Long-Term Effects of Occupational Noxious Agents" Scand J Work Environ Health **6**:163-169.

37. Rothman KJ, Greenland S, Walker AM (1980) "Concepts of Interaction" Amer J Epidemiol **112**:467-470.

38. Office of Population Censuses and Surveys (1983) "Mortality Statistics: Comparison of 8th and 9th Revisions of the International Classification of Diseases" Series DH1 No.10, HMSO, London.

39. Office of Population Censuses and Surveys (1990) "Mortality and Geography: A Review in the Mid-1980s, England and Wales" (Microfiche Supplement) Series DS No.9, HMSO, London.

40. Office of Population Censuses and Surveys (1991) "1989. Mortality Statistics. Area. Microfiche" Series DH5 No.16, HMSO, London.

Chapter 4.

1. Harries PG (1971) "The Effects and Control of Disease Associated with Exposure to Asbestos in Devonport Dockyard" Institute of Naval Medicine, CRWP Report No. 1/71, Gosport.

2. Rossiter CE, Coles RM (1980) "HM Dockyard Devonport: 1947 Mortality Study" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibres" IARC Scientific Publication No. 30, Lyon. pp713-721.

3. Enterline PE, Kendrick MA (1967) "Asbestos Dust Exposures at Various Levels and Mortality" Arch Environ Health **15**:181-186.

Chapter 5.

1. Peto J, Seidman H, Selikoff IJ (1982) "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment" Brit J Cancer **45**:124-135.

2. Liddell FDK, McDonald JC, Thomas DC (1977) "Methods of Cohort Analysis: Appraisal by Application to Asbestos Mining" J Royal Stat Soc (Series A) **140**:469-491.

3. Newhouse ML, Berry G (1976) "Predictions of Mortality from Mesothelial Tumours in Asbestos Factory Workers" Brit J industr Med **33**:147-151.

4. Berry G, Wagner JC (1969) "The Application of a Mathematical Model Describing the Times of Occurrence of Mesotheliomas in Rats Following Inoculation with Asbestos" Brit J Cancer **23**:582-586.

5. Selikoff IJ, Hammond EC, Seidman H (1979) "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976" Ann N Y Acad Sci **330**:91-116.

6. Sullivan KR, Lam TH, Rossiter CE (1988) "HM Naval Bases: Mesothelioma and Time Since First Employment" Ann Occup Hyg **32**:491-496.

7. WHO (1986) "Asbestos and other Natural Mineral Fibres" World Health Organization, Environmental Health Criteria 53, Geneva.

Chapter 6.

1. Doll R, Hill AB (1952) "A Study of the Aetiology of Carcinoma of the Lung" Brit Med J **2**:1271-1286.

2. Doll R, Peto R (1978) "Cigarette Smoking and Bronchial Carcinoma: Dose and Time Relationships among Regular Smokers and Lifelong Non-smokers" J Epidemiol Community Health **32**:303-313.

3. Sheers G, Templeton AR (1968) "Effects of Asbestos in Dockyard Workers" Brit J Med **3**:574-579.

4. McDonald JC, Liddel FDK, Gibbs GW, Eyssen GE, McDonald AD (1980) "Dust Exposure and Mortality in Chrysotile Mining, 1910-75" Brit J industr Med **37**:11-24.

5. Browne K (1986) "Is Asbestos or Asbestosis the Cause of the Increased Risk of Lung Cancer in Asbestos Workers" Brit J industr Med **43**:145-149.

6. Hammond EC, Selikoff IJ, Seidman H (1979) "Asbestos Exposure, Cigarette Smoking and Death Rates" Ann N Y Acad Sci **330**:473-482.

7. Rossiter CE, Coles RM (1980) "HM Dockyard Devonport: 1947 Mortality Study" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibres" IARC Scientific Publication No. 30, Lyon. pp713-721.

8. Rossiter CE, Coles RM, Jackaman I (1984) "HM Naval Base, Devonport: Lung Cancer and Mesothelioma Case Control Studies" In: Proceedings of the VIth International Pneumoconiosis Conference, Bochum, 1983. Bochum, Bergbau-Berufgenossenschaft, p830-836.

9. Hobbs MST, Woodward SD, Murphy B, Musk AW, Elder JE (1980) "The Incidence of Pneumoconiosis, Mesothelioma and other Respiratory Cancer in Men Engaged in Mining and Milling Crocidolite in Western Australia" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibres" IARC Scientific Publication No. 30, Lyon. pp615-625.

10. Sanden A, Javholm B, Larsson S, Thringer G (1992) "The Risk of Lung Cancer and Mesothelioma after Cessation of Asbestos Exposure: A Prospective Cohort Study of Shipyard Workers" Eur Respir J **5**:281-285.

11. Elliot P, Westlake AJ, Hills M, Kleinschmidt I, Rodrigues L, McGale P, Marshall K, Rose G (1992) "The Small Area Statistics Unit: A National Facility for Investigating Health around point sources of Environmental Pollution in the United Kingdom" J Epidem Comm Health **46**:345-349.

12. Newhouse ML, Oakes D, Woolley AJ (1985) "Mortality of Welders and

Other Craftsmen at a Shipyard in NE England" Brit J industr Med **42**:406-410.

13. Kolonel LN, Yoshizawa CN, Hirohata Y, Myers BC (1985) "Cancer Occurrence in Shipyard Workers Exposed to Asbestos in Hawaii" Cancer Res **45**:3924-3928.

14. Neuberger M, Kundi M (1990) "Individual Asbestos Exposure, Smoking and Mortality. A Cohort Study in the Asbestos Cement Industry" Brit J industr Med **47**:615-620.

15. Laquet LM, Van der Linden L, Lepoutre J (1980) "Roentgenographic Lung Changes, Asbestosis and Mortality in a Belgian Asbestos Cement Factory" In: Wagner JC, Davis W (eds.) "Biological Effects of Mineral Fibres" IARC Scientific Publication No. 30, Lyon. pp713-721.

16. Ohlson CG, Hogstedt C (1985) "Lung Cancer Among Asbestos Cement Workers. A Swedish Cohort Study and Review" Brit J industr Med **42**:397-402.

17. Thomas HF, Benjamin IT, Elwood PC, Sweetham Pm (1982) "Further Follow-up Study of Workers from an Asbestos Cement Factory" Brit J industr Med **40**:1-7.

18. Gardner MJ, Winter PD, Pannett B, Powell CA (1986) "Follow-up Study of Workers Manufacturing Asbestos Cement Products" Brit J industr Med **43**:726-732.

19. Hodgson JT, Jones RD (1986) "Mortality of Asbestos Workers in England and Wales, 1971-1981" Brit J industr Med **43**:158-165.

20. Piolatto G, Negri E, La Vecchia C, Pira E, Decarli A, Peto J (1990) "An Update of Cancer Mortality Among Chrysotile Asbestos Miners in Balangero" Brit J industr Med **47**:810-814.

21. Weiss W (1977) "Mortality of a Cohort Exposed to Chrysotile Asbestos" J Occup Med **19**:737-740.

22. Ontario Royal Commission (1984) "Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario" Ontario Ministry of the Attorney General, Toronto.

23. Harries PG (1971) "Asbestos Dust Concentrations in Ship Repairing: A Practical Approach to Improving Asbestos Hygiene in Naval Dockyards" Ann Occup Hyg **14**:241-254.

24. McDonald JC, McDonald AD (1987) "Epidemiology of Asbestos-Related Lung Cancer" In: Antman K, Aisnar J (eds.) "Asbestos-Related Malignancy" Grune and Stratton, London. pp57-79.

25. Peto J, Seidman H, Selikoff IJ (1982) "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis" Brit J Cancer **45**:124-135.

26. Newhouse ML, Berry G (19760 "Predications of Mortality from Mesothelial Tumours in Asbestos Factory Workers" Brit J industr Med **33**:147-152.

27. Seidman H, Selikoff IJ, Hammond EC (1979) "Short-Term Asbestos Work Exposure and Long-Term Observation" Ann N Y Acad Sci **330**:61-76.

28. Selikoff IJ, Hammond EC, Seidman H (1979) "Mortality Experience of Insulation Workers in the United States and Canada. 1943-1976" Ann N Y Acad Sci **330**:91-116.

29. Peto J, Doll R, Hermon C, Binns W, Clayton R, Goffe T (1985) "Relationship Of Mortality To Measures Of Environmental Asbestos Pollution In An Asbestos Textile Factory" Ann Occup Hyg **29**:305-355.

30. Hughes JM, Weill H (1986) "Asbestos Exposure - Quantitative Assessment of Risk" Amer Rev Respir Dis **133**:5-13.

31. Finkelstein MM (1984) "Mortality Among Employees of an Ontario Asbestos-Cement Factory" Amer Rev Respir Dis **129**:754-761.

32. Rockoff SD, Schwartz A (1988) "Roentgenographic Underestimation of Early Asbestosis by International Labor Organization Classification. Analysis of Data and Probabilities" Chest **93**:1088-1091.

33. Rossiter CE, Ellison JMcK (1986) "The Current Asbestos Standards" Occupational Health Review, June/July 1986, pp25-29.

34. Newhouse ML, Berry G (1976) "Predictions of Mortality from Mesothelial Tumours in Asbestos Factory Workers" Brit J Industr Med **33**:147-151.

35. Peto J (1994) "An Epidemic of Mesothelioma Deaths" Talk given at the London School of Hygiene and Tropical Medicine, 29th April 1994.

36. Peto J (1989) "Fibre Carcinogenesis and Environmental Hazards" In: Bignon J, Peto J, Saracci R (eds.) "Non-Occupational Exposure to Mineral Fibres" IARC Scientific Publication No. 90, Lyon. pp457-470.

37. Browne K (1991) "A Threshold for Asbestos-Related Lung Cancer" In: Sluyser M (ed.) "Asbestos Related Cancer" Chichester: Ellis Horwood.

38. Health Effects Institute-Asbestos Research (1991) "Report of Literature Review Panel" Asbestos Control Report, Cambridge, MA.

39. Gaensler EA (1992) "Asbestos Exposure in Buildings" Clin Chest Med **13**:231-242.

40. McDonald JC (1991) "Current Controversies in Occupational Lung Disease" British Occupational Hygiene Society, Annual Conference, Swansea, April 1991.

41. Harries PG (1968) "Asbestos Hazards in Naval Dockyards" Ann Occup Hyg 11:135-145.

42. Harries PG (1971) "The Effects and Control of Diseases Associated with Exposure to Asbestos in Devonport Dockyard" Institute of Naval Medicine, CRWP Report 1/71, Gosport.

43. Sebastien P, McDonald JC, McDonald AD, Case B, Harley B (1989) "Respiratory Cancer in Chrysotile Textile and Mining Industries: Exposure Inferences from Lung Analysis" Brit J industr Med 46:180-187.

44. Wagner JC, Moncrieff CB, Coles RM, Griffiths DM, Munday DE (1986) "Correlation Between Fibre Content of the Lungs and Disease in Naval Dockyard Workers" Brit J industr Med 43391-395.

45. McDonald JC, Liddell FDK, Dufresne A, McDonald AD (1993) "The 1891-1920 Birth Cohort of Quebec Chrysotile Miners and Millers: Mortality 1976-88" Brit J industr Med 50:1073-1081.

46. Vacek PM, McDonald JC (1991) "Risk Assessment Using Exposure Intensity: An Application to Vermiculite Mining" Brit J industr Med 48:543-547.

47. Vacek PM (1990) A personal communication.

48. Stanton MF, Layard M, Tegeris A, Miller E, May M, Kent E (1977) "Carcinogenicity of Fibrous Glass: Pleural Response in the Rat in Relation to Fiber Dimension" J Natl Cancer Inst 58:587-603.

49. Enterline PE, Marsh GM, Henderson V, Callahan C (1987) "Mortality Update of a Cohort of U.S. Man-Made Mineral Fibre Workers" Ann Occup Hyg 31:625-656.

50. Shannon HS, Jamieson E, Julian JA, Muir DCF, Walsh C (1987) "Mortality Experience of Ontario Glass Fiber Workers - Extended Follow-up" Ann Occup Hyg 31:657-662.

51. Simonato L, Fletcher AC, Cherrie J, Anderson A, Bertazzi P, Charnay N, Claude J, Dodgeson J, Esteve R, Frentzel-Beyne R, Gardner MJ, Jensen O, Olsen J, Teppo L, Winkelmann R, Westerholm P, Winter PD, Zocchetti C, Saracci R (1987) "The International Agency for Research on Cancer Historical Cohort Study of MMMF Production Workers in Seven European Countries: Extension of the Follow-up" Ann Occup Hyg 31:603-624.

52. Bunn WB, Bender JR (1991) "Fiber Toxicology and Occupational Health: The Integration of Research and Health Programs" Occupational Problems in Medical Practice (from Pfizer Laboratories) **6**:1-7.

Index to Appendices

APPENDIX 1.

TABLE A1.1: Coding Schedule of Dockyard Datasets.

Variable Name	Position (length)	Description
ID	1-12 (12)	Unique identifer, formed from the national insurance number and first 3 characters of surname.
DOB	13-18 (6)	Date of Birth.
SEX	19 (1)	0 = male, 1 = female.
DOCKYARD	20 (1)	1 = Devonport, 2 = Chatham, 3 = Portsmouth, 4 = Rosyth.
PAYNO	21-25 (5)	Dockyard payroll number.
STATUS	26 (1)	1 = traced and alive, 2 = traced and dead, 3 = traced and emigrated, 4 = untraced.
DOD	27-32 (6)	Date of death.
ICD	33-37 (5)	International Classification of Disease.
XRAY	38 (1)	1 = x-ray taken, 2 = no x-ray, 3 = large x-ray taken.
QUES	39 (1)	1 = questionnaire obtained, 2 = no questionnaire, 3 = controlled questionnaire obtained.
Small x-ray		
SR1	40 (1)	Reader one code.
SS1	41-42 (2)	Reader one score.
SR2	43 (1)	Reader two code.
SS2	44-45 (2)	Reader two score.
Large x-ray		
LR1	46 (1)	Reader one code.
LS1	47-48 (2)	Reader one score.
LR2	49 (1)	Reader two code.
LS2	50-51 (2)	Reader two score.
The variables in this section, and their descriptions, are given in the accompanying questionnaires (see pages A1-3 to A1-10).		
Self Administered Questionnaire		
Personal Medical History	52-64 (13)	0 = yes, 1 = no.
Smoking History	65-81 (17)	0 = yes, 1 = no.
Employment History	82-201 (120)	20 x Job code (2), Start year (2), Stop year (2).
Controlled Questionnaire		
Medical History	202-217 (16)	0 = yes, 1 = no.
Smoking History	218-234 (17)	0 = yes, 1 = no.
Medical History (cont.)	235-243 (9)	0 = yes, 1 = no.
Asbestos Exposure	245-254 (10)	
Employment History	255-314 (60)	10 x Job code (2), Start year (2), Stop year (2).

SELF ADMINISTERED QUESTIONNAIRE.

MEDICAL-IN-CONFIDENCE (when completed)

Naval Dockyard Asbestos Survey

Please answer all questions carefully to the best of your ability either by writing in the boxes provided, or by placing a tick in the appropriate box in some cases.

General Particulars

1. Write in the boxes provided your National Insurance Number (this is shown on your certificate of pay and tax deducted, Form P60).

2. Print in the boxes provided your Surname. If there are not enough spaces continue beyond the boxes.

   Write on the line below your Forename(s)

   _____

3. Write in the space provided your home address:-

   _____

   _____

   _____

4. Write in the space provided your Dockyard or payroll number.

   _____

5. Write in the box provided your National Health Service number (which is shown on your Medical Card.

6. Write in the boxes provided your date of birth.

   Day | Month | Year

7. Write in the boxes provided your height to the best of your knowledge.

   Ft | ins

8. Write in the space provided your weight to the best of your knowledge.

   _____ st _____ lbs

Personal Medical History

9. Have you ever had:-

Place a Tick in the appropriate box

An injury or operation to your chest?	YES	☐	NO	☐
Pleurisy?	YES	☐	NO	☐
Pulmonary tuberculosis?	YES	☐	NO	☐
Bronchitis?	YES	☐	NO	☐
Any other serious chest illness?	YES	☐	NO	☐

10. Have you had a chest X-ray in the last 12 months? YES ☐ NO ☐

If "Yes", please give details required below:-

When? _____

Where? _____

_____

11. Do you usually cough during the day or night at work? YES ☐ NO ☐

12. Do you usually bring up any phlegm from your chest first thing in the morning in winter? (NB Answer "Yes" if this is with your first smoke). YES ☐ NO ☐

13. In the past 3 years how many periods of increased cough and phlegm lasting for 3 weeks or more have you had? Tick appropriate box.

NIL	1	2	3	4 or more
☐	☐	☐	☐	☐

14. Do you get short of breath when walking with people of your own age on level ground? YES ☐ NO ☐

15. During the past 3 years have you had any chest illness which has kept you from your usual activities for as much as a week? YES ☐ NO ☐

16. Did you bring up more phlegm than usual in any of these illnesses? YES ☐ NO ☐

17. How many illnesses like this have you had in the past 3 years? Tick appropriate box

NIL	1	2 or more
☐	☐	☐

Smoking History

Place a Tick in the appropriate Box or insert the answer

18. Have you ever smoked regularly?
(This means as much as one cigarette or one small cigar a day or one large cigar a week, or one ounce of tobacco a month for as long as a year).

If your answer is 'NO' go direct to question No 27.

YES ☐ NO ☐

19. Do you smoke at present?

YES ☐ NO ☐

20. If you have given up smoking

a. was this more than one month ago?

YES ☐ NO ☐

b. how old were you when you last gave up? ☐

21. How old were you when you started smoking regularly? ☐

The next questions are about how much you now smoke, or used to smoke if you have now given up.

22. How many manufactured cigarettes per day? ☐

23. How many ounces of hand rolled cigarette tobacco per week? ☐

24. How many ounces of pipe tobacco per week? ☐

25. How many small cigars per day? ☐

26. How many large cigars per week? ☐

Employment History

27. Please think back over all the different types of work you have done or supervised since you left school both inside and outside the Dockyard. We have listed below various jobs in which we are interested. Please write down in the order in which you did or supervised each job, the code number for that job, the year in which you started the job and the year in which you finished the job. If any of your particular jobs are not included then please use Code 20 for all "other Dockyard jobs, and code 60 for all "other jobs" not listed for employment outside the Dockyard.

Code No	Dockyard Employment
01	Labourer or Skilled Labourer Afloat
02	Lagger Afloat
03	Lagger Ashore or in Mattress Shop
04	Asbestos Storeman
05	Asbestos Sprayer or Stripper
06	Sailmaker Lagger
07	Mason Afloat
08	Welder Afloat
09	Boilermaker Afloat
10	Engine Fitter Afloat
11	Electrical Fitter Afloat
12	Painter Afloat (all grades)
13	Coppersmith Afloat
14	Plumber Afloat
15	Joiner Afloat
16	Burner, Riveter, Caulker, Driller
17	Foundry Worker
18	Shipfitter Afloat
19	Shipwright Afloat
20	All other Dockyard jobs not listed above

Code No	Other Employment
30	Royal Navy Engine or Boiler Room Branch
31	Royal Navy - other than Code 30
32	Civilian Shipyard
40	Lagger or insulation worker (with asbestos)
41	Any other job using asbestos
42	Coal Miner - underground
43	Coal Miner - surface worker
44	Any other mine
45	Foundry work
46	Steelworks
47	Quarrying
48	Pottery
49	Cotton, Flax, Hemp Mill
50	Refractory Brick Works
51	Masons Yard
52	Any other dusty job
53	Any job exposed to irritant gas or chemical fumes
60	All other jobs not listed above
61	Unemployed

	Job Description	Job Code No	Year Started	Year Finished
1st			19	19
2nd			19	19
3rd			19	19
4th			19	19
5th			19	19
6th			19	19
7th			19	19
8th			19	19
9th			19	19
10th			19	19

If there is insufficient space for all your jobs please continue on a separate sheet of paper and place a tick in this box. ☐

CONTROLLED QUESTIONNAIRE.

PACK NO .....

NAVAL DOCKYARD ASBESTOS SURVEY
MEDICAL IN CONFIDENCE (when completed)
Input Code [M][0][5] Col 1-3

National Insurance No. [ ][ ][ ][ ][ ][ ][ ][ ][ ] 8-16

Surname [ ][ ][ ] 17-19

Forenames ..... D M Y

Date of Interview [ ][ ][ ][ ][ ][ ] 20-25

Home Address .....

.....

Dockyard No .....

National Health Service No [ ][ ][ ][ ][ ][ ][ ][ ][ ][ ] 26-36

Day Month Year

Date of Birth .....

Town Country

Place of Birth .....

General Practitioner's Name .....

Address .....

Present Job .....

Interviewer's Name ..... Code [ ] 37

Standing Height cms [ ][ ] 38-39

Weight Kgs [ ][ ] 40-41

Use the actual wording of each question. Put X in appropriate square after each question.
When in doubt record NO.

PREAMBLE: "I am going to ask you some simple questions about your Chest.
Please try to answer the questions wherever possible as YES or NO."

PAST ILLNESSES

			Age	Y	N	
1.	Have you ever had					
	a.	An injury or operation affecting your chest?	....	[ ]	[ ]	49
	b.	Pleurisy?	....	[ ]	[ ]	50
	c.	Pulmonary Tuberculosis?	....	[ ]	[ ]	51
	d.	Bronchitis?	....	[ ]	[ ]	52
	e.	Pneumonia?	....	[ ]	[ ]	53
	f.	Any other serious chest illness?	....	[ ]	[ ]	54
COUGH						
2.	a.	Do you usually cough during the day (or at night when on night-work)? If NO, go to 3.		[ ]	[ ]	55
	b.	Do you cough like this on most days for as much as 3 months each year?		[ ]	[ ]	56
PHLEGM						
3.	a.	Do you usually bring up any phlegm from your chest first thing in the morning in the winter? If NO, go to 3c		[ ]	[ ]	57
	b.	Do you bring up phlegm like this on most days for as much as three months each year?		[ ]	[ ]	58
	c.	Have you ever coughed up any blood? If NO, go to 4.		[ ]	[ ]	59
	d.	When was this? Record each year of occurrence				

BREATHLESSNESS

		Y	N	Col
4. a.	Do you get short of breath walking with people of your own age on the level? If NO, go to 5			60
b.	Do you get short of breath walking at your own pace on the level? If NO, go to 5			61
c.	Do you get short of breath on washing or dressing?			62

CHEST ILLNESSES

5.	During the past 3 years have you had any chest illness which has kept you from your usual activities for as much as a week? If NO, go to 8		63
6.	Did you bring up more phlegm than usual in this/any of these illnesses?		64
7.	How many illnesses like this have you had in the past 3 years? Record number		

TOBACCO SMOKING

8. a.	Have you ever smoked? (This means as much as one cigarette or one small cigar a day, or one large cigar a week, or one ounce of tobacco a month for as long as a year). If NO, go to 10.		65
b.	Do you smoke at present?		66
c.	Have you given up smoking in the last month?		67
d.	How old were you when you started smoking regularly?	Age	68-69
e.	How many manufactured cigarettes do/did you usually smoke per day including the week-ends?	Number	70-71
f.	How much tobacco do/did you usually smoke per day including the week-ends in hand-rolled cigarettes? (Ozs p wk ÷ 4 = gms p day)	gms	72-73
g.	How much pipe tobacco do/did you usually smoke per day including the week-ends? (Ozs p wk ÷ 4 = gms p day)	gms	74-75
h.	How many small cigars do/did you usually smoke per day including the week-ends?	Number	76-77
i.	How many large cigars do/did you usually smoke per week?	Number	78-79

EX SMOKERS ONLY

9.	How old were you when you last gave up smoking?	Age	20-21

CHEST PAIN

			Y	N	
10. a.	Have you ever had any pain or discomfort in your chest?				22
b.	Do you get it when you walk uphill or hurry?				23
c.	Do you get it when you walk at ordinary pace on the level?				24
	If YES to either 10b or c, then				
d.	What do you do if you get it while you are walking?	Stop or slow down			25
	Record stop or slow down if subject carries on after taking Nitroglycerine (Trinitrin)	Carry on			26
e.	If you stand still what happens to it?	Relieved			27
		Not relieved			28
f.	If relieved - How soon?	10 minutes or less			29
		More than 10 minutes			30

ASBESTOS EXPOSURE

Year of First Exposure ☐☐ Year of last exposure ☐☐ Period of exposure (Yrs) ☐☐ Col 31–36

Type of Exposure ..... Continuous (Yrs) ☐☐ Intermittent (Yrs) ☐☐ 37–40

COMMENTS:

OCCUPATIONAL HISTORY

Code No	Dockyard Employment
01	Labourer or Skilled Labourer Afloat
02	Lagger Afloat
03	Lagger Ashore or in Mattress Shop
04	Asbestos Foreman
05	Asbestos Sprayer or Stripper
06	Sailmaker Lagger
07	Mason Afloat
08	Welder Afloat
09	Boilermaker Afloat
10	Engine Fitter Afloat
11	Electrical Fitter Afloat
12	Painter Afloat (all grades)
13	Coppersmith Afloat
14	Plumber Afloat
15	Joiner Afloat
16	Burner, Riveter, Caulker, Driller
17	Foundry Worker
18	Shipfitter Afloat
19	Shipwright Afloat
20	All other Dockyard jobs not listed above
22	Any other dusty job

Code No	Other Employment
30	Royal Navy Engine or Boiler Room Branch
31	Royal Navy – other than Code 30
32	Civilian Shipyard
40	Lagger or Insulation worker (with asbestos)
41	Any other job using asbestos
42	Coal Miner – underground
43	Coal Miner – surface worker
44	Any other mine
45	Foundry work
46	Steelworks
47	Quarrying
48	Pottery
49	Cotton, Flax, Hemp Mill
50	Refractory Brick Works
51	Masons Yard
52	Any other dusty job
53	Any job exposed to irritant gas or chemical fumes
60	All other jobs not listed above
61	Unemployed

AGE	EMPLOYEES NAME	JOB	Job Code	Start Year	Finish Year	
			☐☐	☐☐	☐☐	20–25
			☐☐	☐☐	☐☐	26–31
			☐☐	☐☐	☐☐	32–37
			☐☐	☐☐	☐☐	38–43
			☐☐	☐☐	☐☐	44–49
			☐☐	☐☐	☐☐	50–55
			☐☐	☐☐	☐☐	56–61
			☐☐	☐☐	☐☐	62–67
			☐☐	☐☐	☐☐	68–73
			☐☐	☐☐	☐☐	74–79

CONTROLLED QUESTIONNAIRE (continued).

Input Code [1 | 0 | 2] Col 1-3

Operator's Name ..... Code [ ] [ ] 20-21

Instrument No [ ] 22

	1	2	3	4	5	
FEV 1.0	[ ] [ ] [ ]	[ ] [ ] [ ]	[ ] [ ] [ ]	[ ] [ ] [ ]	[ ] [ ] [ ]	23-37
FVC	[ ] [ ] [ ]	[ ] [ ] [ ]	[ ] [ ] [ ]	[ ] [ ] [ ]	[ ] [ ] [ ]	38-52

Operator's Name ..... Code [ ] [ ] 53-54

Instrument No [ ] 55

	V Insp	Va	TF	
First Test	[ ] [ ] [ ]	[ ] [ ] [ ]	[ ] [ ] [ ] [ ]	56-65
Second Test	[ ] [ ] [ ]	[ ] [ ] [ ]	[ ] [ ] [ ] [ ]	66-75

CLINICAL

General

Hb	[ ] [ ] [ ]	20-22
CVS		
BP	systolic [ ] [ ] [ ] diastolic [ ] [ ] [ ]	24-28
Cyanosis	L [ ] R [ ]	29
RS		
Rales	[ ] [ ]	30
Do Rales clear on coughing	[ ] [ ]	31
Rhonchi	[ ] [ ]	32
Pleural Rub	[ ] [ ]	33
Clubbing	[ ] [ ]	34
Asbestos corns	[ ] [ ]	35

Comments

Small Film No. .....

Large Film No .....

CONTROLLED QUESTIONNAIRE (continued)

Input Code — N | 0 | 2 — Col 1-3

Operator's Name ..... Code — 20-21

Instrument No — 22

	1	2	3	4	5	
FEV 1.0						23-37
FVC						37-52

Operator's Name ..... Code — 53-54

Instrument No — 55

	V Insp	Va	TF	
First Test				56-65
Second Test				66-75

CLINICAL

General

Hb		20-27
CVS		
BP	systolic / diastolic	24-28
Cyanosis	Y / N	29
RS		
Rales		30
Do Rales clear on coughing		31
Rhonchi		32
Pleural Rub		33
Clubbing		34
Asbestos Corns		35

Comments

Small Film No. .....

Large Film No .....

APPENDIX 2.

TABLE A2.1: Causes of Death, and International Classification of Disease (ICD) groupings.

	Causes of Death	9th Revision ICD's	8th Revision where different
*1.	All Causes	000-999	
2.	Infectious and Parasitic Diseases	001-139	001-136
3.	Tuberculosis	010-018	010-019
*4.	All Neoplasms	140-239	
5.	Ca. Lip, Oral Cavity and Pharynx	140-149	
6.	Ca. Digestive Organs and Peritoneum	150-159	
7.	Ca. Oesophagus	150	
*8.	Ca. Stomach	151	
*9.	Ca. Peritoneum (mesothelioma)	158	
10.	Ca. Respiratory System	160-163	
*11.	Ca. Lung	162	
*12.	Ca. Pleura (mesothelioma)	163	
13.	Ca. Bone, Tissue, Skin and Breast	170-175	170-174
14.	Ca. Genito-urinary Organs	179-189	180-189
15.	Ca. Prostate	185	
16.	Ca. Other and Unspecified Sites	190-199	
17.	Ca. Lymphatic and Haematopoietic Tissue	200-208	200-209
18.	Benign Neoplasms	210-229	210-228
19.	Unspecified Neoplasms	230-239	
20.	Endocrine and Nutritional Diseases	240-279	
21.	Diseases of Blood and Blood-forming Organs	280-289	
22.	Diseases of the Nervous System	320-389	
*23.	Diseases of the Circulatory System	390-459	390-458
24.	Hypertensive Disease	401-405	400-404
25.	Ischaemic Heart Disease	410-414	
=*26.	Diseases of Pulmonary Circulation	415-417	426,450
27.	Cerebrovascular Disease	430-438	
*28.	Diseases of the Respiratory System	460-519	
29.	Acute Respiratory Infections	460-466	
30.	Other Disease of Upper Respiratory Tract	470-478	500-508
31.	Pneumonia and Influenza	480-487	470-474,480-486
*32.	Bronchitis, Emphysema and Asthma	490-493	
=33.	Chronic Obstructive Pulmonary Disease	490-496	490-493,518
=34.	Pneumoconiosis	500-508	515-516
35.	Coalworkers Pneumoconiosis	500	515.1
*36.	Asbestosis	501	515.2
37.	Silicosis	502	515.0,515.9
38.	Other Diseases of the Respiratory System	510-519	510-514,517,519
*39.	Pulmonary Fibrosis	515	517
40.	Diseases of the Digestive System	520-579	520-577
41.	Diseases of Oesophagus and Stomach	530-537	
42.	Diseases of the Genito-urinary System	580-629	
43.	Diseases of the Skin and Subcutaneous Tissue	680-709	
44.	Diseases of the Musculosketelal System	710-739	710-738
45.	Symptoms, Signs and Ill-defined Conditions	780-799	780-796
46.	Accidents, Poisonings and Violence	800-999	
47.	Transport Accidents	800-848	800-845
48.	Accidental Poisoning	850-869	850-877
49.	Accidental Falls	880-888	880-887
50.	Suicide and Self-inflicted Injury	950-959	

= Signifies a new code/disease grouping in the 9th revision. An estimate of this grouping is produced for the 8th revision (i.e. for before 1979).
* Signifies a specific cause of interest in this study.

Death Rates for England and Wales.

The following pages (A2-4 to A2-20) give male death rates for England and Wales ($\times 10^{-8}$). For each of the 50 categories, the entries are arranged as follows:

	Year																
	1972	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988
Age																	
10–																	
15–																	
20–																	
25–																	
30–																	
35–																	
40–																	
45–																	
50–																	
55–																	
60–																	
65–																	
70–																	
75–																	
80–																	
85+																	

For calendar years 1989 and 1990 the death rates for 1988 are taken as the best estimate.

ALL CAUSES (0-999)

35991	34278	31983	33383	30567	28437	29592	29292	26999	28699	28058	27196	27631	28888	23004	25683	25858
85571	86133	88901	86581	88227	83960	89706	84624	83453	82009	78210	74437	71446	67598	71290	70907	69931
96986	104218	95736	98588	96222	91413	99503	91677	87383	83105	86376	82322	83900	82288	81956	84781	83008
87170	93058	85245	84309	84812	87968	88091	90225	84203	83936	82800	82714	82492	77249	79915	77653	78272
104809	107444	106824	103729	100104	95446	97440	100479	94320	93847	97114	96237	93329	92236	95306	100558	96033
163803	159267	151328	148404	154347	148446	158049	146852	141751	133445	129617	130256	127849	130880	130024	129445	130116
286804	288499	282728	267197	264380	253693	249682	251833	244213	239210	231084	224199	213311	219520	210896	205479	211965
540835	536551	523186	507197	487229	480447	475984	463586	443777	432117	412250	413771	399072	385713	383876	365309	378299
921001	899348	918544	906514	898510	873767	877604	857405	823303	786985	759441	724209	705064	701417	676012	642418	673282
1556386	1569303	1535683	1449364	1470252	1411761	1440329	1465806	1431588	1391870	1361302	1321007	1266439	1247141	1208293	1183989	1213141
2585267	2494532	2455153	2436590	2474415	2381578	2426021	2422116	2304543	2214705	2167697	2193445	2157160	2175439	2122518	2026523	2108160
4276800	4125598	4066310	3983485	4000528	3858612	3873695	3807042	3675059	3583956	3596309	3617919	3500306	3423729	3329351	3227110	3326730
6801644	6558140	6551825	6399432	6432491	6157365	6169148	6094797	5885875	5795287	5768838	5686622	5446023	5567463	5435020	5277100	5426527
10149179	10002362	9750465	9844031	9993546	9555921	9634381	9612776	9266169	9024084	9002246	8823054	8463804	8789797	8602173	8193035	8528335
14783019	14729549	14644643	14601504	15032373	13970357	14205591	14384128	13970791	13769704	13781500	13508621	12989488	13568764	12997852	12296697	12954437
24291117	23850655	23697256	23542056	24322987	22657785	22765381	23204380	22244856	22615873	22303077	22071590	21130562	22313287	21481654	19159856	20984932

INFECTIOUS & PARASITIC DISEASES (1-139)

415	709	690	820	622	666	430	484	544	451	617	423	662	345	787	763	631
982	1882	1517	492	957	466	606	686	1291	1040	1031	707	1011	836	998	1225	1019
1214	1192	1552	1046	752	1029	961	890	867	527	518	705	680	710	704	889	767
1285	1292	1224	844	1158	944	972	1100	821	882	588	639	964	1045	906	1083	1011
1562	1336	1434	1209	980	1246	1092	1237	1495	910	1134	935	1065	1192	1545	1116	1284
2244	2636	1647	1982	1732	2220	2322	1524	1807	1494	1154	1557	1968	1570	1783	1821	1724
2885	3122	3238	2769	2429	2141	1907	2235	2225	2468	1479	2284	1479	1896	1883	2027	1935
5484	5169	5865	3926	4732	4423	3618	3014	3329	3577	3869	2384	3142	3124	3156	2362	2880
9310	8135	8676	7980	6882	5479	4876	6141	5840	4305	4992	3802	3924	3654	3823	4046	3841
11636	11690	10412	10953	10027	8074	7395	7018	7498	7706	6004	5796	6086	5381	6396	6292	6023
14897	15431	13435	14259	10510	11158	11568	11120	9813	7418	9077	9504	9904	9147	8696	7555	8466
25289	21490	18627	18811	16904	14587	13858	14627	13598	13963	12945	13118	14292	13442	13439	10352	12411
32192	27460	25342	28275	21758	20737	19428	20354	17220	16864	17226	14164	14651	16522	16719	15420	16220
37354	38262	32528	37029	35499	31661	26916	29395	25870	24792	26611	26121	25666	26316	30037	29467	28606
42677	33527	39286	42901	42572	34874	44731	39679	31643	34218	38602	37241	44678	40468	43265	37416	40383
53650	46288	54031	56924	47819	54954	45939	43796	60032	44444	33077	40693	67104	63636	61374	52885	59298

TUBERCULOSIS (10-18)

0	0	0	0	0	95	0	48	0	0	0	53	55	0	0	0	0
58	228	337	164	106	0	51	0	0	142	0	0	0	148	50	0	66
331	341	402	290	174	114	226	111	108	53	155	0	146	47	0	47	31
335	108	213	211	211	111	172	290	235	176	0	58	113	55	160	0	71
611	401	456	318	61	340	55	161	374	321	170	117	177	238	238	176	217
1122	1179	275	479	416	832	888	265	516	498	173	334	164	217	108	114	146
1786	2012	1689	1136	929	500	636	768	765	1128	423	554	336	458	251	405	371
3634	3893	3137	2342	3132	2106	1206	933	724	1387	1460	722	857	852	646	644	714
6341	5488	5742	5839	3675	3219	2926	2047	2137	1220	2243	877	1184	895	1124	1573	1197
8045	7818	6247	6402	6239	4787	4314	2913	3439	2899	2242	2054	2078	1569	1355	1516	1480
10705	10412	9382	9204	6828	6964	7377	3883	4230	2445	3287	2245	2241	2624	2463	2676	2587
17959	14176	12567	12629	9597	8963	8157	6131	5702	5514	4958	5928	3981	4091	4013	3034	3712
23425	20497	17739	20496	15472	14457	12564	8805	5965	7148	7002	4869	5398	5287	4904	5178	5123
27123	26925	23002	22217	24957	20354	16699	11306	9838	9917	11405	8595	9378	9041	9906	7879	8942
29650	22798	25446	29633	24390	17001	26667	16885	12170	11918	12017	15862	13798	14543	12581	10816	12646
25506	27074	23156	31436	29362	34971	18868	12165	18167	20635	6154	12057	17505	21678	16678	12019	16791

ALL NEOPLASMS (140-239)

7271	7139	5421	6320	5214	5136	6215	5713	4401	5059	6064	4868	5074	5111	3995	4259	4455
7795	8214	8710	8308	7707	7515	8435	8232	6695	7993	6607	7726	7222	6642	6136	6841	6539
11371	11409	11435	10109	10241	10348	9888	9118	10293	7857	9218	8157	8740	7907	7886	7209	7667
15367	14971	14580	14869	14899	13820	14415	13493	11669	12646	9226	12605	12416	10227	11401	10261	10629
19359	21315	19814	20619	19053	20902	20416	19149	17882	17068	16724	17296	17033	16386	14379	17034	15933
33378	31285	30609	30761	34638	33019	33946	31743	30066	26839	28913	29867	29735	29553	27820	28563	28645
66360	64720	62641	56080	63594	56376	55093	56289	55544	59450	56573	52745	54790	53409	52347	50269	52008
132946	130832	130251	128728	122347	123640	116850	113224	114616	109708	107023	106423	111175	102677	104935	103515	103709
246037	240493	251108	245232	254694	246233	252943	239235	234955	227579	219577	216129	217427	210365	198501	201978	203614
453311	470320	457909	427998	436233	419120	433687	444290	431795	433298	424810	421161	412869	410806	395064	386172	397347
762914	735506	734219	727348	747526	736884	723950	729856	717114	697098	682683	705979	725873	715550	709635	697859	707681
1146784	1142483	1146168	1126910	1137612	1119508	1127533	1108522	1098167	1098186	1086761	1091633	1111882	1082895	1066449	1055863	1068402
1601781	1591381	1608084	1581183	1599299	1572343	1575966	1558719	1569387	1552937	1574350	1613478	1593963	1590814	1592956	1580437	1588069
1971925	1987955	2019284	2056553	2107573	2078331	2108841	2104579	2081800	2080751	2052704	2093360	2132938	2176784	2167758	2153010	2165850
2187332	2240948	2321875	2367979	2343681	2421099	2407742	2535669	2504260	2563629	2565550	2593793	2801577	2822004	2702976	2738673	2754551
2371152	2381659	2400515	2429057	2441275	2448793	2516817	2619627	2682464	2764286	2775385	2825923	3146608	3274126	3164777	3173678	3204193

CA LIP, ORAL CAVITY & PHARYNX (140-149)

52	51	49	0	48	0	48	97	0	100	51	53	55	0	0	0	0
58	57	0	55	213	104	101	147	96	47	47	188	48	246	50	153	149
0	57	172	0	58	0	170	167	0	105	207	201	49	142	188	234	188
391	54	53	105	158	56	172	116	176	353	118	174	113	55	53	258	122
68	0	261	191	613	340	437	323	320	321	170	117	473	238	119	352	236
491	277	412	342	623	486	410	663	1032	747	462	723	601	487	648	626	587
824	763	1056	781	1429	1356	706	1536	1599	1128	1902	1038	1412	1896	1444	2259	1866
1850	2282	2387	2411	2088	2457	2483	2655	2315	2920	2774	3251	2499	2982	3945	3293	3406
4318	4326	4057	4412	4610	3562	3344	3953	4772	5381	5933	5191	6145	6264	5172	5544	5660
6896	7591	6648	6633	7428	8145	6847	7216	8255	9332	8608	7631	9722	9043	7525	9021	8529
8759	7940	8482	9808	10894	10999	10562	10767	12605	11819	10486	12497	13302	11846	12006	11805	11885
15210	14447	12567	14307	14527	11336	13595	15065	12370	15386	13129	15839	16435	13248	15026	14724	14332
22740	18657	18753	19632	19340	18367	19428	15895	19021	17087	18115	20471	19828	18835	16273	16110	17072
34975	29995	27416	30072	28399	21382	23576	29584	20040	19302	21427	24604	19743	20988	22208	20013	21069
41330	43362	33036	40248	37251	40976	36559	43056	30832	33449	30590	32069	31537	19602	23320	24847	22589
65963	63755	53173	71368	55369	58285	46760	48662	45024	43651	36923	45968	40846	33566	37358	27043	32655

CA DIGESTIVE ORGANS AND PERITONEUM (150-159)

104	101	0	0	48	95	0	145	0	100	51	53	55	115	0	64	59
520	57	281	273	372	104	202	147	335	331	187	47	48	492	249	153	298
938	738	690	988	289	343	735	334	488	580	621	604	583	521	516	468	501
1341	2208	1224	1793	1895	1277	1030	1564	938	1235	705	813	1077	1100	1598	1598	1432
3464	4210	3389	4900	4043	3908	3548	2905	3309	3050	2551	3155	2721	2979	2258	3289	2842
8555	8532	7755	8066	9283	9295	8606	8416	7549	6663	6983	8176	8144	7794	6536	7112	7147
20403	20394	20341	16469	18221	17198	16104	16621	17518	18900	15288	17305	16403	15689	15441	14073	15067
36738	40209	40235	38983	34936	36439	37531	36880	35818	33139	31245	34896	35202	30675	30770	31785	31076
73399	70760	71656	71688	77170	70822	73981	76168	72288	67585	65258	68363	66627	66145	63118	64654	64639
133171	136253	132078	124489	126495	121535	129339	132936	127743	128667	120506	120845	122235	125177	120551	121522	122416
223611	215131	210388	206488	216264	214291	207310	210661	207597	202886	195399	204221	209427	200330	206711	204549	203863
357981	346456	352317	350349	341786	327417	331462	324604	323537	330487	313900	312603	327991	315020	309286	305640	309982
498356	495204	498606	490554	490995	470553	476966	482790	465053	454992	457324	468518	467614	457099	454191	448101	453130
667380	654464	651719	661355	673623	649671	644990	632561	635088	621569	595127	632120	627674	623830	634926	609991	622915
828841	809566	796875	824414	792461	848736	767312	821866	782961	773549	798616	791724	819645	846032	786131	796258	809473
929639	915284	880789	914189	898490	863447	890894	933496	906793	938889	856154	938206	958425	985315	925284	932692	947763

CA OESOPHAGUS (150-150)

0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	49
55	0	0	58	0	0	0	56
112	54	53	105	105	0	114	0
136	334	196	382	306	510	55	269
631	555	549	752	762	555	478	928
1649	1734	2252	1562	1429	1855	1625	1955
4229	3491	4569	4546	4663	3651	3760	2798
6679	7296	7116	7136	9020	8904	9613	8895
11852	14346	14017	13266	14558	16005	14310	15492
20886	21948	20791	20671	22325	23502	25484	24181
30786	28081	31462	32147	32224	32777	34208	33021
45890	41387	43588	41980	38680	39934	46301	44597
53295	51488	59480	60817	58520	62089	60904	55587
62893	74654	66964	69438	64745	73235	65806	69228
90589	74236	72041	76466	88087	64946	78753	78670

CA STOMACH (151-151)

0	0	0	0	0	0	0	48
0	0	0	55	0	0	0	0
166	284	115	349	58	57	57	0
168	646	160	316	421	333	114	232
883	1069	912	1018	796	623	764	484
2594	2150	2471	2119	2217	2705	1981	2121
5496	6798	6334	4401	5288	4210	4732	3422
9911	13358	12480	11227	9952	9900	10429	10117
22128	24404	23532	24329	23385	21164	20759	21107
47191	45848	41890	41342	39664	35724	39849	38133
86465	79476	79336	74236	73878	72565	68321	63896
136797	132460	134938	125673	125022	112830	115867	109223
182466	181842	178662	179158	177445	165304	170195	166495
238401	225083	222119	219479	224828	221217	201768	208404
285714	266428	248214	281734	249667	273758	256774	246518
273527	270742	253859	266780	260906	232306	274815	264396

CA PERITONEUM (Meso) (158-158)

0	0	0	0	0	0	0	48
115	57	0	55	159	0	51	49
0	114	0	116	58	57	113	0
112	377	53	316	105	56	114	58
204	200	130	255	123	283	109	0
70	416	343	273	69	208	273	66
687	208	422	71	572	214	212	210
463	470	546	482	348	632	780	574
1214	904	811	973	735	548	766	918
1293	1366	1602	1234	817	1143	1438	662
1872	1348	1651	1132	1074	1266	1593	706
2932	2619	1961	1325	1057	2285	1667	2190
3151	3416	1901	1729	2418	2014	3025	1830
3093	2598	3950	1571	2367	3906	2358	942
1348	894	6250	2211	3104	1308	3441	1266
1759	2620	858	2549	839	1665	4922	0

0	0	0	0	0	0	0	0	0
48	0	47	0	0	0	0	0	0
0	0	52	101	49	47	141	0	62
117	235	118	116	113	165	107	52	108
107	214	283	292	237	477	238	411	375
710	685	808	834	820	650	1080	1252	994
1529	1551	1832	2007	1681	1896	2009	2143	2016
3907	3942	3212	5057	4427	4971	4160	4582	4571
12107	9040	8971	9505	8291	9396	8771	10264	9477
16028	16896	16492	16436	16179	20925	17910	18801	19212
24448	26736	25980	25369	27543	31789	30322	33527	31879
37372	37887	40856	36731	43181	43639	38264	42388	41430
41981	40652	48122	49906	50782	53090	58738	57422	56416
59574	68532	61172	66397	66469	67485	63429	73432	68115
78702	69973	79752	79655	70959	91053	88371	92371	90598
71880	75397	70769	92690	79504	90909	95397	92548	92951

0	0	0	0	0	0	0	0	0
0	95	47	0	0	49	0	0	16
54	158	104	101	97	47	47	47	47
117	59	118	116	57	0	373	309	227
320	321	510	526	355	596	238	764	532
1419	1121	1270	1613	2022	1407	1242	1536	1395
4380	4937	3593	4222	3092	3007	2950	2780	2912
9334	7810	8030	7731	7783	6817	6814	7588	7073
17235	17004	17508	16890	15916	14989	13568	12886	13814
38247	37752	32043	31991	32136	30491	30025	28808	29774
61755	58934	52352	65105	57616	51807	54410	51000	52405
107466	106990	93922	99213	91058	88934	88754	79243	85643
160158	154791	147699	150160	149152	131182	135310	127043	131178
206595	198335	189390	200202	198585	193736	187410	179956	187034
221907	221838	235615	251724	235545	234587	233200	223619	230468
255134	252381	255385	254710	263311	276923	250167	254207	260432

0	0	0	0	0	57	0	0	19
143	47	0	0	48	0	100	51	50
108	105	104	101	49	95	141	47	94
117	118	0	58	57	55	53	103	70
53	161	0	117	0	119	0	0	39
194	125	115	334	273	217	108	57	127
348	353	282	346	269	327	188	0	171
507	438	219	144	500	781	215	215	403
570	574	362	1097	592	298	675	899	624
1169	990	723	1101	816	822	677	531	676
931	652	1096	898	1012	750	1770	1259	1259
2456	889	826	1360	1021	1169	840	892	967
1688	1675	2223	1992	1652	2864	1226	1956	2015
729	1948	1382	1180	2303	1453	1598	1261	1437
1623	3845	1821	1724	657	2529	3068	1754	2450
1580	2381	3846	754	1459	2098	1334	2404	1945

CA RESPIRATORY SYSTEM (160-163)

104	51	49	0	48	48	0	145	99	50	51	0	0	0	0	64	21
231	57	169	0	159	104	101	49	96	47	94	47	0	98	100	51	83
552	568	230	407	463	286	283	56	108	105	155	50	97	95	141	94	110
1006	1131	905	949	1000	722	744	405	645	1176	176	290	397	385	426	103	304
2513	3074	3063	2100	2144	1869	1747	1829	1281	1391	1531	1227	1360	953	832	1057	947
6171	6867	5559	5674	6304	6035	6898	5832	4194	4421	5078	4116	4209	4493	4646	3869	4336
19372	17897	17948	16256	17149	14415	14268	13269	13695	13893	13668	12459	12303	11702	11926	10540	11389
55306	51151	50259	48970	46628	44724	39447	37957	38712	35766	36210	31862	32060	31740	31774	28420	30644
109897	106011	111604	110289	108238	110959	106583	99322	90592	89611	83201	79330	74771	73005	65367	63755	67375
213619	223167	219944	198072	204709	188340	196303	199073	189585	186921	178807	181745	167285	169345	155918	140626	155296
374083	356554	357877	347341	353740	342486	336407	339952	327299	317493	298067	313702	311285	306568	296368	287817	296917
534451	534898	527718	515764	514967	518190	506885	496365	492236	468428	470162	474784	462229	448178	436771	425843	436930
700822	705689	706285	682430	691285	686100	682527	661750	673945	648984	650811	671019	638907	620994	617811	615650	618151
707828	756731	766264	788600	801635	817229	823772	838138	809619	816540	776568	787159	773445	787698	759386	745982	764355
568733	628073	693304	711632	704656	762860	797849	837062	850710	847366	834304	858966	880092	874486	820804	840398	845229
452067	439301	508576	497876	517617	531224	580804	618005	666667	646032	700000	698568	743982	793007	789860	736178	773015

CA LUNG (162-162)

0	51	0	0	0	0	0	0	99	50	0	0	0	0	0	64	21
173	57	56	0	106	0	0	49	48	0	0	47	0	0	50	51	33
497	511	172	116	405	172	170	0	54	53	155	0	97	47	94	94	78
950	1023	745	738	842	555	515	405	469	882	176	290	340	330	320	52	234
2174	2806	2803	1655	2022	1699	1583	1560	1228	1391	1361	993	1301	953	772	999	908
5960	6382	5284	5400	5542	5411	6079	5368	4065	3736	4617	3726	3662	4222	4322	3528	4024
18204	16648	16962	15049	16292	13345	12926	12361	12444	12130	12400	11213	11496	10329	10984	9382	10231
52399	48130	47941	47180	43705	42407	37247	36234	36469	33358	34239	29550	29632	29894	28260	26272	28142
106928	102008	107484	105424	103494	106507	101846	94381	85535	86024	79222	74212	70995	67338	61319	57537	62064
207298	215956	212495	190204	195796	181909	187949	190798	180918	178791	170416	172793	158602	160227	147490	131150	146289
365474	347191	347144	336100	343767	331962	326934	329891	316471	304777	287112	300681	297188	292922	281976	273572	282823
521715	520632	513369	500927	499472	504657	491711	481738	478375	453042	455196	458070	448550	433762	418105	407371	419746
679178	681119	682970	659835	672066	666074	663564	644483	655374	627541	632252	652318	619740	596431	594739	593556	594908
683084	730987	739080	765260	772590	790707	797839	811004	786664	793519	751339	765420	753044	764127	734622	723290	740679
539982	600358	655357	689960	669623	732781	763011	806669	821501	816993	809541	829310	844941	842871	790120	810582	814524
422164	404367	483705	467290	488255	504580	548811	588808	640600	618254	667692	675961	716265	764336	755837	695313	738495

CA PLEURA (Meso) (163-163)

52	0	0	0	48	0	0	0	0	0	0	0	0	0	0	0	0
58	0	56	0	0	52	51	0	0	0	0	0	0	0	0	0	0
0	0	0	232	58	57	57	0	0	0	0	0	0	0	0	0	0
56	108	53	53	53	167	172	0	0	59	0	0	0	0	0	52	17
204	200	0	191	61	57	55	54	0	0	57	0	0	0	59	59	39
0	208	137	68	277	347	205	265	129	62	115	222	273	217	108	228	184
69	277	211	568	214	428	494	279	487	776	564	554	269	588	502	811	633
859	806	955	344	1322	562	922	718	1013	511	511	1228	1071	1136	1363	787	1095
877	968	999	1362	1269	1438	1463	988	1852	933	868	2120	1333	1864	1874	2547	2095
1939	1822	1282	1928	2525	1715	1986	1655	2614	2545	2170	3375	3266	3811	3988	3942	3913
1348	2247	2177	2565	2455	2849	2431	2295	2199	3424	3052	3817	4771	5098	4925	5194	5072
1741	2167	1693	3003	3170	3076	4035	3066	3597	3646	3030	4761	4900	4189	5226	5979	5131
2740	2234	2914	4075	2297	4147	2908	3202	3714	3686	4001	4869	5508	6388	7133	6559	6693
1190	2834	2556	3142	2582	3906	4519	4711	3644	2833	2938	2696	4278	3875	6870	5358	5367
3594	1788	2232	2211	1774	3923	4731	2533	2434	3076	2185	6552	5585	5375	7057	7015	6482
2639	1747	3431	1699	839	833	3281	0	1580	1587	2308	3014	1459	6993	2668	2404	4021

CA BONE, TISSUE, SKIN & BREAST (170-175)

727	1063	1084	965	813	713
1155	1939	1798	1366	850	1399
1380	1589	1494	1510	1041	800
1285	1346	958	1318	1369	1277
1291	1136	1304	1400	1532	1699
1332	1318	1990	1846	1801	1526
3091	2844	2182	2272	3573	2997
3172	3424	3205	3375	3897	4283
4453	4842	4244	4801	6214	4521
5818	6224	7849	5553	7279	7859
9208	8539	7206	8525	10203	9338
12095	13995	12834	12982	10829	12039
17397	15110	18880	16298	16681	17538
23793	25980	24396	22666	24312	19737
35490	38891	34375	35383	36364	34874
49252	52402	39451	45030	48658	54954

CA GENITO-URINARY ORGANS (179-189)

260	203	99	289	191	95
577	913	955	711	531	829
1711	1760	2011	1336	2083	2058
2738	2639	2767	2953	2738	2442
3193	3207	2607	2736	2879	3229
3576	3746	2539	3555	4087	3885
4671	4925	4364	3336	5216	4639
8722	8391	8320	8747	7864	8706
15516	16980	17415	14143	17906	16918
32467	34765	30116	31238	31197	34224
64306	61573	63499	65636	68508	67184
118563	120903	119964	120198	130569	125220
216575	218237	213761	224349	221927	212940
366643	350968	361989	368492	372849	370888
517071	522128	537946	537373	551220	522232
678980	693450	692110	641461	668624	698585

CA PROSTATE (185-185)

0	0	0	0	0	0
0	0	0	0	53	0
0	0	57	0	0	0
56	0	0	0	0	0
0	0	0	0	0	57
70	69	137	0	0	0
69	208	422	71	143	143
793	738	818	689	696	632
2766	3034	2871	2984	2673	2945
9266	9792	7769	8099	10027	9431
23656	22472	22442	25198	27465	26668
54792	55621	52139	54844	62599	59930
116164	116148	114673	120879	119183	116009
225791	216580	223745	222846	218373	222039
342318	346446	370982	359575	363193	343941
459982	481223	485420	440102	447987	458784

669	872	742	801	1182	582	496	804	666	572	680
1364	1862	1052	2034	1500	1696	1637	1132	1497	1225	1284
1187	1946	1896	1107	1450	1259	1651	1136	1596	1170	1300
915	1390	997	1412	940	1278	1701	1430	1652	980	1354
1801	1667	1655	1766	1928	1519	1597	1430	1723	1880	1677
2049	2319	2645	2491	2655	2836	2624	3031	3025	2731	2929
1836	3073	3476	3173	3523	2492	3092	3138	3452	2896	3162
4399	4520	3039	4161	3358	4768	4356	4473	3443	4080	3998
6409	4377	6339	5524	4847	4899	6219	5891	6297	5094	5760
7258	6951	6329	6292	6438	6824	8461	6801	7299	8339	7479
11652	9973	9898	7744	9703	8157	9036	9072	10620	10074	9922
16841	10423	10527	12807	12119	13701	14394	13637	11293	13296	12742
16403	15323	15982	17869	15226	15160	20599	17623	15716	18412	17250
22200	18466	16396	20365	19008	25110	21882	22603	23167	24582	23450
33118	33770	22312	31142	30226	31379	32852	32880	28230	29524	30211
47580	50284	39494	38095	49231	47476	48140	56643	50700	54087	53810
96	145	49	100	103	106	110	0	121	0	40
960	490	622	615	328	471	144	49	200	357	202
1526	1223	1625	844	725	755	923	994	939	562	831
2746	2374	2463	1529	1175	2207	1474	990	1279	1289	1186
2402	2098	2295	1177	1814	1519	1656	1490	1070	1997	1519
2937	2916	2968	2055	2020	1947	2186	1732	1675	2447	1951
4238	3352	3337	4090	3311	3738	3630	3595	3013	3301	3303
6314	7606	7598	7226	7373	7080	8711	6888	7244	7731	7287
16858	16024	16523	16932	15917	16963	17841	15735	17391	16182	16436
33002	34293	34739	34217	35443	36173	34956	34377	37023	31916	34438
63207	64160	64292	62439	64872	67425	68387	73474	75189	70360	73007
124989	127879	121677	130736	124495	133515	136076	137054	139711	135374	137379
217776	211778	218571	217445	225383	238796	237938	256526	258582	256732	257280
379961	363859	365458	357535	366511	371925	383021	401518	413485	425622	413541
517849	547488	535903	558247	539694	566207	660644	631363	639153	638118	636211
672683	690998	720379	727778	741538	718915	913202	927273	848566	892428	889422
0	0	0	50	0	0	0	0	0	0	0
51	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	50	0	47	47	0	31
0	58	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	59	0	0	0	0
0	0	0	62	0	0	0	0	0	57	19
283	0	209	141	282	138	269	261	126	232	206
780	502	1158	730	803	506	643	710	717	1289	905
3135	3812	3134	3659	2894	3144	4146	3207	4648	2847	3567
9175	8937	11626	10675	10199	11079	11058	11210	11739	11371	11440
26322	26917	26647	24209	26371	27165	28483	28940	33400	31639	31326
57363	60787	60356	62878	61605	69381	73397	72570	76435	73443	74149
121801	117210	123917	124525	126695	137103	137475	154422	153032	155351	154268
235167	226870	225178	221356	232417	231378	243008	259929	271769	282225	271307
335914	356268	346450	363706	352877	376552	435611	422700	437251	450161	436704
443806	459854	483412	492063	486923	477016	630926	656643	617745	630409	634932

CA OTHER & UNSPECIFIED SITES (190-199)

1870	2127	1232	1351	1052	1427	1721	1017
924	913	1461	1148	1329	1140	1263	1029
1822	1703	1494	1685	1620	1658	1469	1279
2515	2693	2554	2162	2369	2442	3203	1911
3260	3675	3454	3182	2573	3455	3494	3711
4838	4093	5422	4648	5404	4786	5328	4838
8106	7492	8094	7667	8789	6565	7840	8799
12026	12016	12480	12880	13293	14042	13196	10691
18350	18077	21035	18944	20178	19452	24800	21530
30384	31501	32119	31855	29563	29151	31017	35816
39677	41273	44509	44361	47181	47005	52058	50481
49386	52731	57398	55021	60838	67926	67801	71910
65479	60307	71338	65317	78206	80934	77943	89194
72329	72744	82017	84829	101549	102796	93713	104579
83109	84041	99107	86245	104213	103313	111828	127058
75638	82096	94340	98556	112416	114072	119770	121655

CA LYMPHATIC & HAEMATOPOIETIC TISSUE (200-208)

3583	2886	2563	3136	2631	2615	3346	2760
3811	3879	3259	4318	3827	3265	3687	3871
4416	4541	4827	3892	4166	4631	4068	3558
5644	4631	5906	5167	4843	4718	5034	4922
5026	5680	5149	5346	4779	5721	6059	5917
6662	5341	5971	6084	6166	6659	7035	5765
8518	9295	7672	7951	8003	7564	8829	8101
12555	10338	10775	10813	11413	11093	12203	10763
17001	16528	18164	18295	16837	17329	18042	15460
26433	26340	24509	24990	24289	26293	26498	23833
36982	38202	36103	39155	35596	39962	36969	37066
50944	51648	54011	50870	55115	51933	58767	52203
69178	68848	70705	73713	69745	76431	73174	70555
87081	85026	96190	89767	93158	84087	107859	103071
98383	102369	112054	118974	103326	92415	126452	107218
92348	115284	120069	133390	119128	109908	143560	134631

BENIGN NEOPLASMS (210-229)

415	304	246	338	144	95	143	0
289	57	506	328	266	363	303	98
221	341	172	116	405	286	283	56
224	162	0	158	316	333	400	290
272	67	326	318	368	340	546	161
912	555	549	137	623	277	410	265
412	763	563	497	643	856	565	279
1189	873	1159	1102	1183	702	568	574
1147	1033	1061	1492	1336	1438	1393	635
1868	1518	1842	2160	2525	1786	1232	927
2396	2397	2702	2112	1764	2137	2599	2030
3482	2257	3832	3533	3170	2548	2894	2365
4384	3679	3294	4075	4352	4147	4188	2287
5472	4724	3020	5162	6024	4317	5697	2261
7188	6705	8036	7961	7539	9154	7742	2111
18470	11354	6861	13594	10067	8326	7383	2433

Death Rates for England and Wales (continued).

1335	1352	1285	899	1158	1321	908	1081	1103
1483	1277	797	1036	1637	1082	948	1072	1034
1950	1055	1450	1410	1408	1231	1173	1358	1254
1876	1941	1528	2730	2381	1649	1918	1598	1721
2829	4173	3005	3681	3430	3992	2555	2526	3024
4904	4483	3924	4950	5302	5142	4916	4950	5002
8133	8322	8102	6853	8067	7845	8097	9266	8402
12446	14453	13214	12427	14780	13136	15493	12385	13671
23859	24394	23730	22446	25614	24758	22114	24573	23815
36390	37610	42387	37714	40151	40356	35894	40255	38835
50842	54777	59081	56050	63182	66127	61875	65717	64573
72550	75240	87679	77446	86668	86012	86421	90487	87640
89589	108220	113136	111652	117537	118075	121601	122900	120858
120969	132460	149300	134311	166338	171779	172711	180429	174973
133874	161092	191551	164828	217148	226367	229211	235604	230394
142180	215873	230769	183120	242888	266434	286191	298077	283567
2027	2154	2724	2593	2758	2470	1998	2225	2231
2582	3453	3327	3957	3322	3149	3043	3573	3255
3684	3744	4039	3575	3593	3314	2910	2996	3073
4046	4353	3585	4647	4762	3959	4049	4073	4027
5551	4494	4876	5493	5145	4767	5110	4816	4897
5936	5231	5944	6174	5630	6279	5078	6316	5891
6535	8604	8032	7822	9076	8368	7909	7066	7781
12156	10438	11024	10548	11782	11219	10831	14031	12027
18090	14564	17002	16743	16805	16033	16417	19404	17284
23801	24956	25244	25974	25976	21897	26262	30248	26135
38575	32768	36388	38240	44603	41610	41712	41791	41704
56321	53984	51506	54805	58289	60004	57676	59611	59097
75183	75162	78684	77349	78101	86023	94851	90104	90326
98743	94563	103508	104314	115169	118825	116313	120233	118457
126166	134179	115805	129655	130420	155232	146364	147033	149543
138231	132540	132308	177091	163384	176224	186791	186899	183304
0	100	51	212	55	57	61	0	39
143	47	0	94	48	49	0	51	33
163	158	259	0	97	95	141	0	78
235	176	176	58	227	165	53	155	124
214	0	170	117	59	298	119	470	295
323	374	404	167	273	217	324	171	237
278	282	423	415	269	327	502	290	373
507	0	365	433	500	568	502	358	476
427	717	651	585	1110	373	600	599	524
1169	1485	868	1247	520	747	1656	1213	1205
1438	978	783	1197	2024	1799	1231	1495	1508
2369	2312	1744	2429	2348	2533	2706	3570	2936
3151	2457	2334	2102	2974	3525	3901	2877	3434
3279	3542	2419	2528	2632	3229	3195	4255	3559
5680	4614	2913	4828	4928	6639	3375	3508	4507
5529	2381	3845	2261	4376	8392	2668	5409	5489

UNSPECIFIED NEOPLASMS (230-239)

156	354	99	241	239	48	191	533
231	342	281	109	159	207	455	392
331	114	345	174	116	286	170	389
224	108	213	264	211	555	172	463
272	267	261	445	123	340	382	484
841	555	412	410	346	69	273	729
962	347	422	852	572	785	706	1117
1388	2148	1432	1446	1044	1194	709	1507
1956	1937	1873	1168	2205	1233	1533	1624
2658	2960	2803	3008	2748	1786	2191	2979
3893	3895	3453	3923	3376	3482	3186	4501
4673	5147	5526	3886	5811	2900	4298	7270
6849	5650	6462	4815	6769	5332	7562	9034
6424	7322	6273	5610	6024	8224	7073	11871
7188	5811	7143	5750	6652	6539	9032	16041
8795	8734	5146	13594	10906	9992	7383	19465

ENDOCRINE & NUTRITIONAL DISEASES (240-279)

1143	1367	1133	1254	1435	999	1530	1114
693	1312	899	1312	1222	1451	1313	1470
662	1135	1207	1394	1041	1086	904	1390
1173	916	1064	1265	1263	1998	1316	1100
1630	1270	1760	2227	1225	1643	1638	1452
2875	3122	2677	2529	2771	2220	2391	2121
3091	4162	4293	4117	4430	3854	4097	3632
4625	5639	6069	4959	5568	4985	5392	4592
8770	7941	8239	7007	8018	9452	8220	6706
12786	13360	13136	13344	13221	11646	12530	11387
19239	21723	20791	19842	23705	22632	18442	19681
34451	37472	37522	35945	33281	35325	35260	32495
54521	57417	58540	57661	54756	54390	55375	54545
96122	84554	81320	87298	83477	82237	80550	81590
130728	130085	141964	115436	122395	115519	117849	106796
158311	144105	143225	168224	166107	151540	133716	132198

DISEASES OF BLOOD & BLOOD-FORMING ORGANS (280-289)

156	456	345	96	335	285	287	242
289	171	337	547	159	518	354	245
55	341	460	523	579	515	678	500
168	269	213	316	421	333	114	521
340	67	0	382	245	397	164	269
280	208	480	547	277	416	137	265
412	486	211	568	429	357	494	349
661	738	887	551	626	702	780	574
1147	1227	874	1492	1336	1301	627	1059
1508	1974	1842	1080	1931	1786	2054	1986
4492	2397	2702	2565	3376	2374	2263	3795
6780	6862	6684	6182	5987	5360	5526	6920
11781	12351	12164	13705	11241	10191	8609	10863
27123	24799	24861	20422	22806	19120	17878	23554
47170	47385	49107	38479	37251	30950	37849	35458
102023	107424	102916	90909	88087	65779	68089	68938

Death Rates for England and Wales (continued).

148	301	462	317	331	287	121	254	220
287	142	234	141	289	344	50	204	199
379	158	259	302	243	284	188	281	251
235	353	705	290	170	440	320	155	305
427	642	624	292	473	238	535	411	394
323	374	1270	612	601	325	810	228	454
834	917	2325	623	403	654	502	347	501
1881	1533	1460	1011	1285	852	932	1074	952
1994	2439	2966	1316	1851	2088	2024	1723	1945
3302	3535	6148	2641	3340	2690	2784	2426	2633
4061	6032	7512	4191	4337	4348	3232	3620	3733
6316	8182	11935	6122	7350	6819	6626	6604	6683
8441	10721	13003	7635	10245	11455	9140	8861	9818
11660	14875	18490	10785	22705	25993	21569	21116	22892
14604	19992	21850	13448	23982	28770	25775	22508	25684
18167	18254	24615	14318	30635	26573	37358	39063	34331
1236	1152	1285	1270	1324	1551	1090	1144	1261
1339	1040	750	1413	722	1378	1497	817	1230
1192	1107	1087	1510	1554	1089	1220	1545	1284
997	1059	1175	1220	1304	1814	2557	1702	2024
1388	1124	1587	993	1360	1788	2674	4170	2877
2710	2179	1616	1613	1804	2111	3025	5462	3532
3198	3385	2607	2353	3227	3792	4770	4807	4456
5137	5328	4234	6286	4855	6817	6455	7016	6762
7407	6027	7597	7677	8217	8203	8471	10788	9154
12933	12230	11284	11446	12840	13302	15953	18118	15791
17934	16710	16198	16164	21470	21517	25781	25106	24134
27283	33529	29838	29735	33483	40912	42930	41317	41719
56275	46795	50011	49131	63450	73797	80361	72727	75628
85079	83407	82426	79373	109905	127704	133887	125433	129008
118458	114187	115805	113103	184297	202656	217858	196726	205746
134281	158730	150769	147702	258206	337762	313542	266827	306043
643	301	257	106	276	287	363	127	259
383	473	281	141	241	344	499	204	349
217	475	414	403	243	379	282	421	360
176	235	235	290	397	330	266	567	387
107	107	340	292	355	536	238	235	336
387	623	289	278	328	487	324	171	327
695	494	493	415	672	523	565	290	459
1013	876	657	795	428	852	430	859	713
1567	1435	1085	1170	1407	1044	900	899	947
2476	1697	2025	1541	1484	2541	2483	2426	2483
3468	4076	3913	3143	4337	4348	4156	4722	4408
6141	6492	6519	7482	7248	6526	7839	7139	7168
11818	10610	10224	10623	13770	15310	13486	13464	14086
20404	20365	22119	17863	29121	31159	28599	29467	29741
37728	39216	32411	35172	50591	59437	60448	49693	56526
74250	62698	58462	50490	117433	123077	135424	105168	121223

DISEASES OF THE NERVOUS SYSTEM (320-389)

2597	2582	2661	2074	2583	2473	1912	1840	2126	2154	1850	1958	2702	1895	2058	2161	2038
4331	4278	5001	5083	4199	3472	4192	4214	3682	3973	4217	3392	4574	5166	4739	5003	4969
3754	4711	5172	4009	3587	4402	3899	3503	3792	3533	3521	3877	4273	4309	3661	4354	4108
2626	4147	4044	3374	3948	3330	3547	3243	3284	3529	3878	2904	3345	3959	3197	3403	3519
3940	4410	4302	3309	3492	3172	3166	3873	3843	3852	3855	4207	4317	4230	3624	4112	3988
4348	4093	6451	4853	4503	3885	4303	4573	4000	3985	4097	4616	3717	5034	5564	5576	5391
5496	5203	6546	5679	6359	5638	5227	6355	4588	4513	6623	5399	5647	7518	5963	6081	6520
8590	7854	7570	7852	7447	7583	7024	8897	7959	7591	6935	5924	8568	7598	8392	8304	8098
11334	10330	10673	9926	10757	10274	9544	10871	9187	8681	10708	8262	10882	10515	11994	8990	10499
14366	14954	14658	15118	13444	14576	14447	16485	14515	15977	13382	13060	16773	17786	15201	15768	16251
22234	20749	22142	19992	21864	20337	21125	23917	19288	22008	20737	20804	23711	28190	26012	25185	26462
33077	34402	32620	31794	33457	32074	30611	33722	32284	31750	34521	34010	49102	47243	45730	43637	45536
52055	54921	51571	50624	54636	50835	50256	56261	51097	55729	59680	61525	81626	91750	87606	87457	88937
78753	75579	77602	82585	80465	80387	73870	86866	89087	100761	108346	104483	162389	167904	173191	151907	164334
123989	110863	101339	112340	106874	98954	100215	138455	123732	146867	146395	166897	268068	289598	284750	258696	277681
125770	134498	132075	118097	110738	134055	147662	161395	156398	182540	210000	228335	419402	429371	422282	377404	409685

DISEASES OF THE CIRCULATORY SYSTEM (390-459)

1662	1975	2119	1303	1339	1759	908	1307	1533	1703	822	1693	827	1493	1150	1017	1220
3753	2966	3203	3170	3242	2540	2829	3528	3396	3169	2624	3298	3178	2116	3093	3267	2825
4747	4655	5402	4764	4687	4574	4407	4503	4713	4219	5230	4078	3884	3740	3098	3839	3559
10002	9209	8407	8225	7423	6771	8523	8513	8502	7705	8286	7319	7314	7148	6979	4744	6290
21193	21649	21704	20364	17828	17786	17250	17804	16761	17496	16384	15075	15318	13466	14557	14567	14196
53783	54523	50580	47987	49255	46337	51089	46918	42583	42219	37338	37597	33452	34263	35815	32091	34056
130315	128746	128730	122595	113898	117177	114211	105594	104484	101410	92222	89776	82487	84526	77956	78300	80260
279701	273612	264525	256009	243162	242154	240866	240080	221129	218102	199591	206054	187290	182063	178238	163433	174578
478108	472658	482117	481705	457273	457329	461860	457998	435083	414622	396252	378811	358010	357047	343928	322370	341115
786094	781691	779175	747628	756518	742569	748716	764978	748641	718841	694901	679800	652145	624094	608398	595330	609274
1294879	1259026	1247542	1244285	1246030	1209069	1264565	1259465	1182133	1133844	1111198	1115917	1089641	1089369	1052024	997875	1046422
2181235	2109977	2087166	2053254	2036714	2000176	2012981	1960322	1891920	1840982	1827121	1858711	1784504	1720826	1664209	1617973	1667669
3498082	3377217	3391662	3306458	3251783	3195639	3213006	3162607	3033652	3013290	2929206	2889233	2783322	2825752	2717454	2638435	2727213
5371401	5238073	5142426	5138016	5056583	4926398	4950884	4928962	4738568	4530016	4540003	4415908	4303554	4429286	4312190	4101639	4281038
8105121	8033080	7861161	7820433	7740133	7206190	7362581	7264669	7092089	6833910	6756373	6528276	6459921	6692697	6324026	6040339	6352354
13759894	13310917	13116638	12882753	12503356	11827644	11931091	11960260	11186414	11144444	10764615	10601356	10380744	10665035	10203469	9170072	10012858

HYPERTENSIVE DISEASE (401-405)

0	0	49	0	0	0	48	48	99	0	0	0	55	0	0	0	0
0	0	56	109	106	52	0	49	0	189	0	0	96	49	150	51	83
221	284	57	116	116	57	113	167	108	158	155	101	0	47	94	94	78
726	323	426	422	211	111	286	58	352	176	118	290	227	220	107	52	126
611	1002	1108	827	735	906	273	1022	534	482	340	234	414	179	59	352	196
1753	1873	1029	2051	1593	1595	1366	1723	1097	810	635	334	328	379	486	455	440
3984	4301	4645	3833	3716	2569	1836	1676	2155	1904	1339	1938	1210	1438	816	1042	1098
8590	7048	8388	7439	5707	5757	5108	4951	4052	4745	4015	2818	3142	2343	2080	1933	2118
12750	13042	13295	12845	11692	9384	7454	8753	6267	7390	5716	4972	5848	4922	5022	4645	4863
23344	20874	22747	19360	16935	16433	14926	14499	12657	12018	12514	10419	10094	7922	8353	7353	7876
40051	36854	35352	32969	30917	30466	30598	24711	23771	21519	21285	18185	17205	16644	16700	12907	15417
67436	56975	58200	57935	56700	48682	45435	43356	36758	35486	33327	30609	28277	28054	26972	21149	25391
93836	93286	84516	86924	73734	80460	72010	63465	60101	55506	46455	47803	46596	42846	40682	37860	40462
154175	141001	136385	122756	116394	101357	111591	96476	81253	79157	70848	73643	65482	61027	53203	48219	54149
181941	185069	174554	169394	164523	150828	145376	134234	113590	106882	104516	85517	88371	94531	78245	65770	79515
294635	230568	229846	211555	208054	213156	178835	194647	153239	138889	136923	122080	131291	110490	101401	92548	101479

ISCHAEMIC HEART DISEASE (410-414)

52	0	49	48	0	0	48	0	49	100	0	53	55	0	61	0	20
231	57	281	109	319	104	51	196	191	47	187	94	193	98	50	102	83
497	738	460	871	926	629	509	612	867	738	673	403	825	379	329	281	329
2459	2962	2395	1951	2316	1943	2460	2722	2815	2765	2174	2033	2551	1814	1598	1392	1601
10800	10825	10363	10882	8822	9176	8079	9790	8701	9845	8957	8239	7866	6256	7130	7930	7105
36673	34892	33491	30487	32698	31285	35585	30219	29357	28831	24700	25195	22083	22788	24363	21394	22848
94868	92952	96213	91574	85173	87990	87795	80522	80222	76375	71720	68180	62387	63869	59377	58609	60618
213559	209438	202332	200703	187974	187109	189216	188563	175470	171679	157103	163500	150660	146560	141587	131076	139741
364838	359416	369265	375178	352442	354521	365657	360440	345346	330105	314933	303283	287015	286801	280135	259365	275433
575420	573326	571406	556730	575578	563661	579391	582853	576598	551573	535552	531000	521746	499066	483483	477219	486589
894370	880599	879231	881026	885846	869431	918518	920660	874461	852054	826199	836713	829538	841505	799369	766567	802480
1410024	1380767	1374332	1364303	1351118	1355448	1370231	1326881	1306606	1272056	1268729	1316879	1280421	1252776	1205693	1193646	1217371
2059315	2014847	2033452	2012224	2018736	1995853	2026640	1977702	1916939	1935336	1900311	1890450	1852831	1909682	1828021	1780783	1839495
2847728	2801842	2739545	2818896	2823580	2818051	2858153	2764650	2719439	2641403	2684638	2636333	2609576	2698095	2666241	2530728	2631688
3708446	3725525	3782143	3832817	3869180	3679163	3819785	3630224	3603651	3571319	3587036	3537586	3587057	3758141	3508131	3378837	3548369
5379947	5468122	5422813	5327103	5323826	5314738	5419196	4882401	4808057	4969841	4834615	4920121	4927790	5223077	5037358	4579327	4946587

DISEASES OF PULMONARY CIRCULATION (415-417)

0	0	49	0	0	0	0	97	49	0	51	106	110	57	0	0	19
0	57	112	164	0	52	51	98	143	142	141	236	48	148	100	153	133
110	170	172	116	58	172	57	56	163	211	362	101	194	142	141	47	110
391	108	0	211	105	111	57	116	176	294	118	349	113	220	160	206	195
272	134	196	255	184	113	164	215	267	161	57	351	118	0	0	235	78
280	902	343	410	277	416	137	596	581	685	173	278	219	217	216	284	239
1305	1110	985	852	500	928	989	489	1529	776	775	623	538	392	251	521	388
1652	1947	1296	1722	1670	1194	1277	2798	1954	1679	2336	1951	571	568	646	501	571
4048	3874	3308	3763	3608	4178	3344	4659	3846	3731	3617	4387	1407	1790	1199	1124	1371
6321	6073	7769	5939	7576	5716	5546	9599	9218	9756	8318	7191	3191	3886	3161	2729	3258
11978	11311	13135	11015	13042	11395	11485	18092	15735	15487	16355	14667	6578	6598	6234	5431	6087
19791	20587	19519	21461	19194	19332	18332	30744	26230	26770	27819	25459	10106	9838	9426	8299	9187
33973	35606	31804	31238	30340	32468	29781	47684	48959	52044	42009	41717	18616	19055	18725	18067	18615
39971	45111	45074	47576	48838	45641	44990	74242	67408	64105	69293	70104	36690	34872	33072	32619	33521
63792	66607	58036	64131	69180	67568	67527	111017	105477	101499	104151	96897	46649	53114	52777	47647	51179
69481	71616	94340	81563	98993	90758	107465	123277	132701	132540	127692	113791	73669	64336	70714	61298	65449

CEREBROVASCULAR DISEASE (430-438)

831	861	739	338	478	713	191	436	593	551	257	476	110	517	242	318	359
1270	856	843	1148	850	726	859	931	1052	1088	422	1225	674	640	998	970	869
1601	1419	2069	1685	1389	1544	1639	1445	1517	896	1864	1460	1117	1089	986	1030	1035
3353	2100	2395	2689	2158	2331	2460	2432	2932	2294	2468	2382	2041	2474	2131	1134	1913
4891	4544	4236	3246	2941	3059	3930	3496	3470	3103	3742	3214	3253	2920	4219	2937	3358
6872	8532	8373	6357	7135	6174	7718	7687	6388	6289	6637	6452	6286	6279	5996	5576	5950
14083	15400	14217	14552	12504	13630	12361	12571	11470	11707	10286	9760	10353	10394	8850	9730	9658
30924	29670	28369	26517	25054	27733	24122	26118	22504	22190	20295	22975	18493	19740	19796	16393	18643
53768	54426	51807	50149	48239	48425	46395	48496	45367	42474	40515	36558	36941	36913	32609	33338	34286
101853	103234	102203	91786	86236	86882	82027	86594	84130	78402	74286	73960	68577	64868	67650	61860	64792
201827	192659	183367	187778	173456	168394	169168	165828	150157	137757	139369	134850	140678	130379	132292	121124	127931
413231	397381	385205	363155	359658	341388	341110	325129	302658	294468	280114	271597	283075	258134	248903	232108	246381
794795	754829	731247	707742	667714	644152	644835	644597	593585	578736	554012	535244	554968	536623	525524	495052	519066
1371163	1338687	1298327	1247756	1192986	1156867	1138310	1166196	1102204	1038073	1015206	957364	1006252	1039070	989775	941223	990022
2348158	2281627	2159375	2089341	2034146	1837838	1852903	1831575	1801217	1716647	1667152	1613103	1700394	1752134	1726296	1616486	1698305
4052770	3851528	3639794	3532710	3404362	3139883	3157506	3180049	2954976	2907937	2819231	2696307	2921225	2997902	2854570	2536058	2796176

DISEASES OF THE RESPIRATORY SYSTEM (460-519)

2597	2532	2710	2171	3348	2045	1721	2130	2522	2003	2210	1799	1489	1091	1211	1271	1191
5139	4563	5170	4099	4837	2747	3586	3185	3539	3311	3187	2450	1878	2362	1746	2144	2084
5410	4995	4597	4648	4455	3773	3334	4003	3088	3638	2952	3625	2816	2131	2535	2294	2320
4582	4524	4363	4587	4528	4052	4119	3069	4046	4294	3761	3717	2438	2694	2451	3094	2746
6725	5412	5475	6873	7229	3908	5459	4626	4430	3906	4422	4149	2957	3396	3862	2819	3359
11570	8740	8441	7929	8590	7839	7377	6163	6194	5667	5771	5061	3553	5142	4646	3755	4514
18616	15954	15695	13133	14291	12488	12007	12571	10497	9027	10356	10175	6790	6995	8034	7008	7345
35946	36182	30074	31476	27977	24573	26321	23606	21491	18467	21390	18424	13638	13775	13198	13387	13453
73939	70437	63230	60919	67215	54932	52734	51249	46222	39532	41239	32902	28280	30276	28636	23150	27354
154575	150979	133921	124875	125084	103530	110168	103012	99608	95723	97794	90836	68577	73163	67800	61026	67329
309253	282322	257299	250622	267357	235895	234387	221516	205143	188865	184443	190601	145883	168016	153225	134976	152072
621312	555576	514260	500839	527734	449385	447943	443286	401526	372999	403874	395880	299510	313657	286981	262984	287874
1175205	1078965	1036113	1024077	1085459	928665	926477	902001	827462	792495	815070	760319	562679	605463	594628	548331	582807
1986914	1948276	1782063	1852334	2029905	1773232	1819646	1761070	1658954	1609881	1628650	1562690	1072228	1156765	1109922	976678	1081121
3053010	3123380	3053125	3121628	3639024	3134263	3148387	3255382	3104665	3043445	3109250	3069655	1991130	2177996	2068119	1760304	2002139
5560246	5623581	5699828	5705183	6895134	6112406	6015587	6189781	6052133	6265079	6404615	6201959	4221007	4704895	4515677	3682091	4300887

ACUTE RESPIRATORY INFECTIONS (460-466)

208	203	345	145	239	95	143	145	198	0	103	159	110	172	0	64	78
520	342	281	437	372	155	152	98	0	236	187	47	96	148	100	51	99
386	284	402	407	0	286	113	167	108	158	52	252	146	0	47	0	15
279	269	372	316	211	333	286	0	352	118	176	58	57	385	53	155	197
272	267	261	318	490	170	164	269	53	0	57	117	118	179	119	117	138
280	416	343	410	416	208	342	331	387	125	58	445	109	162	162	57	127
481	555	704	710	429	571	424	140	348	212	352	415	202	131	628	405	388
1123	873	887	1033	348	843	639	933	289	438	219	433	143	142	143	215	166
1619	1227	1560	908	1604	1301	1115	847	641	646	651	219	518	597	750	225	524
1580	2960	2243	2391	1931	1715	2396	1125	1376	778	651	1101	520	1046	903	910	953
4492	4120	3753	3772	3836	2691	2766	2824	1438	2038	1565	973	1157	2399	2078	1889	2122
9621	8488	5615	6535	4930	4569	4736	3153	3597	3646	2663	2526	2756	2630	2333	1606	2189
18219	13664	12038	12347	13901	8413	9190	7204	5515	6478	7113	4758	3855	4736	5127	4028	4630
29265	26216	19284	22890	20439	16859	17682	13002	11842	9208	9677	11122	8391	9041	7030	6776	7615
50764	45597	46875	40690	43016	32258	39570	29548	27586	21530	21122	18966	11827	18653	19638	10523	16271
88830	89956	88336	83263	107383	84929	68089	79481	58452	59524	47692	37679	43764	46853	37358	25240	36483

OTHER DISEASES OF UPPER RESPIRATORY TRACT (470-478)

52	51	0	145	0	48	0	48	49	0	0	53	0	0	0	0	0
115	228	112	109	0	0	152	0	48	0	0	47	0	148	50	51	83
110	114	172	0	174	0	57	56	0	53	52	50	0	95	141	47	94
0	54	0	158	53	111	114	0	0	118	59	0	0	0	0	52	17
272	67	0	64	0	170	109	161	107	54	57	0	59	0	0	0	0
0	69	69	137	69	277	68	133	65	0	58	0	109	54	0	57	37
69	139	0	0	286	71	0	70	0	0	70	138	0	0	0	0	0
66	67	68	69	70	211	213	144	72	0	73	72	0	0	215	143	119
135	65	250	195	134	137	70	141	71	0	289	73	74	0	0	0	0
72	0	160	231	297	71	0	199	138	141	0	147	74	149	0	303	150
225	150	225	377	77	79	84	265	0	0	235	75	0	0	308	0	102
641	181	178	265	352	88	351	88	439	0	275	97	102	97	93	0	63
411	526	634	123	363	355	582	572	338	223	333	443	441	441	223	345	336
476	945	465	449	0	1028	786	377	1093	0	346	169	0	646	320	630	532
449	447	446	442	887	0	1290	844	811	384	364	690	1314	632	921	292	615
880	1747	1715	850	2517	0	0	811	790	0	769	0	0	0	667	1202	623

PNEUMONIA & INFLUENZA (480-487)

1402	1215	1577	1013	2153	951	908	1114	1483	851	719	582	221	230	545	254	343
3002	2567	3653	2186	3348	1969	2424	1911	2056	1419	1312	1036	241	689	649	562	633
3312	3292	2931	2614	3124	2287	2260	2502	1788	1476	1295	1410	825	663	657	421	580
2515	2639	2714	2636	2948	2387	2288	1448	2170	1823	1410	2033	794	715	639	980	778
3600	3074	3324	4582	4901	2096	3221	2636	2455	1766	2381	1870	1005	1132	1664	1116	1304
6942	4509	4049	3896	4849	4509	3347	3115	3097	2864	3693	2670	984	2436	1513	910	1619
9892	8116	7531	6034	7646	5638	5933	6425	5283	3949	5002	4707	2218	2484	2573	2954	2670
14140	15976	12752	14670	12736	10812	12274	9543	9117	7956	10147	6936	3642	3479	4304	4152	3978
26243	26600	23594	21993	27794	21849	18321	20613	16879	15569	14904	11260	6145	6861	5847	4270	5659
48484	49719	42851	42499	46795	36582	38138	34558	34945	32167	28861	29276	11801	11060	11137	11144	11113
93352	91011	76785	77480	96663	80161	76369	76692	70891	66107	63150	60316	21904	24067	23549	19440	22352
210189	195756	172282	175130	209808	167047	164284	167645	149750	137051	148090	141386	49918	49192	44330	39443	44321
457808	436079	407501	421040	498006	407631	398325	394511	361058	344427	354968	325329	109495	115762	114690	97238	109230
917678	948040	860595	895197	1075301	899054	907466	883738	852432	827519	808018	788675	278052	297546	293338	238418	276434
1759209	1856951	1788839	1871296	2340133	1900174	1904516	1977628	1878296	1842753	1894028	1847931	708607	804932	742559	608594	718695
3782762	3947598	4027444	4022940	5129195	4478768	4320755	4549878	4445498	4619841	4692308	4532027	2344274	2643357	2483656	1991587	2372866

BRONCHITIS, EMPHYSEMA & ASTHMA (490-493)

935	962	739	724	765	856	574	678	742	952	1285	952	1103	402	605	699	568
1039	1198	787	1148	1063	518	707	784	1148	1324	1265	1084	1396	1132	898	1276	1102
1325	1135	862	988	868	915	565	834	813	1529	1191	1359	1359	994	1549	1685	1409
1620	1077	958	1265	1053	833	1087	1274	1173	1765	1880	1278	1361	1320	1279	1547	1382
1630	1804	1369	1400	1225	906	1419	1130	1334	1284	1417	1636	1124	1668	1485	1292	1481
3015	2636	2951	2393	2286	1734	2459	1657	1807	2117	1154	1390	1421	1516	1675	1707	1632
6045	5757	5419	5040	4216	4995	3885	4539	3059	3526	3100	2630	3092	2811	3013	2085	2636
17444	15976	13298	13362	11413	9057	9720	8682	8249	7007	6935	7008	5784	6178	4877	4725	5260
41625	38350	33082	33346	31202	25274	25566	21954	21010	17004	16712	13307	12215	13423	10945	9739	11369
93808	89191	78574	70497	62913	53658	54776	52565	46640	42418	41374	36686	30800	30192	24983	21454	25543
193742	170262	159949	151264	142079	127008	127001	113494	96016	82410	74497	76106	61736	68376	57103	48009	57829
369342	319639	304367	287733	270470	234974	229278	217921	179753	159374	158924	147216	127807	118644	104526	83705	102291
641507	572986	556513	528954	494863	434649	428106	409377	352167	311146	285619	250858	221855	225355	190927	161105	192462
962170	892537	819470	835278	817771	737664	760118	700584	608125	541881	531363	442872	397664	383113	318262	248503	315626
1151842	1109075	1095536	1093764	1102882	1025719	1022366	1038835	939959	846213	806628	712759	659658	619665	525928	405729	517107
1507476	1420087	1408233	1386576	1421141	1304746	1382281	1282238	1224329	1155556	1128462	1019593	927790	933566	834556	586538	784886

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (490-496)

935	962	788	724	765	856	574	678	742	1002	1285	952	1103	402	605	763	590
1155	1255	955	1203	1063	518	758	882	1243	1372	1406	1084	1444	1181	898	1276	1118
1435	1249	919	1046	1099	915	735	945	1029	1529	1295	1611	1457	1042	1549	1732	1441
1676	1508	1011	1265	1211	944	1258	1390	1231	1941	1939	1336	1417	1375	1438	1547	1453
2377	1871	1695	1655	1593	1246	1528	1183	1548	1498	1531	1753	1360	1668	1723	1292	1561
3787	3191	3294	3144	2771	2289	3210	2121	2258	2553	1443	1613	1804	1840	2215	2219	2091
7144	6451	6264	5537	4573	5281	4450	5168	3893	4443	4227	3807	3899	3792	4017	2954	3587
18766	17386	14594	14051	12875	9759	10500	10978	10420	8613	9198	9681	8140	8876	7101	7660	7879
43176	39641	34517	34060	32939	26849	26681	25907	25283	21022	23441	19668	19544	19985	18591	16032	18202
95891	91089	81698	73120	65364	55373	57104	62032	57990	56133	60976	53929	51136	55078	49063	43818	49319
197335	174232	163702	153678	146298	130648	130019	132380	122325	110694	109398	118387	111834	129555	114514	104360	116143
374382	325598	310873	292944	274872	239895	234278	253569	228880	215226	231546	233602	227746	240503	221185	200518	220735
651096	581527	566016	536239	503928	440692	434272	469868	430276	407639	416426	399469	413858	447847	436023	409781	431217
976445	907889	835270	850539	832616	750000	772102	802337	736564	714185	752030	703573	716025	775105	737178	655846	722709
1171608	1130979	1116071	1110571	1118847	1041848	1042581	1173491	1132252	1095732	1109250	1102759	1156702	1232690	1187174	1023385	1147749
1535620	1441048	1431389	1413764	1441275	1328893	1402789	1437956	1433649	1426190	1518462	1478523	1619256	1773427	1772515	1463341	1669761

PNEUMOCONIOSES (500-508)

0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	98
0	0	57	0	0	0	0	56
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	54
0	0	0	0	0	139	0	0
69	69	0	0	143	71	141	70
463	201	205	207	209	211	142	144
742	775	624	973	802	411	488	635
2083	2581	2082	1774	1931	1215	959	1258
4342	5019	2927	5432	3682	3719	3437	2295
11178	10023	9358	8390	7396	6854	6491	6482
17397	15110	12671	11977	16923	10783	9772	11435
20462	17005	13243	21320	17427	18092	18075	20916
10332	16987	16071	18134	15965	21796	16344	20262
12313	12227	12007	16143	15101	14988	17227	21087

COALWORKERS PNEUMOCONIOSES (500-500)

0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
132	67	0	0	70	140	0	0
202	516	375	195	134	137	70	212
1293	1670	961	848	891	286	479	397
2620	3446	1876	3093	1918	2137	1593	706
7422	6953	5526	4769	4490	3339	3421	3241
11507	10905	8363	7779	12692	6043	6166	6975
13562	12045	9526	15934	12694	13569	11002	13755
6289	14752	12946	12826	11530	16129	14194	16041
11434	11354	6003	13594	12584	12490	13126	13788

ASBESTOSIS (501-501)

0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	69	0	0
0	69	0	0	0	71	141	0
132	67	0	69	0	70	0	0
270	65	0	260	67	68	139	71
359	228	320	309	297	286	342	199
75	375	225	528	307	396	335	530
183	90	446	530	264	703	789	526
548	394	380	494	484	829	465	343
238	472	0	898	0	0	196	377
0	0	446	885	443	0	0	844
0	0	858	0	0	0	0	811

Death Rates for England and Wales (continued).

49	0	0	0	0	115	0	0	38
0	47	0	94	0	49	0	0	16
54	53	0	151	49	0	0	0	0
0	59	0	0	0	55	0	0	18
0	54	170	0	0	0	0	0	0
0	125	58	0	0	54	108	284	148
0	141	0	346	134	65	126	116	102
72	73	219	144	214	71	72	143	95
499	430	217	73	296	224	750	225	399
963	1273	1374	1394	1336	448	1129	758	778
2792	3016	2895	2245	2313	2024	2617	1338	1993
5878	4536	5325	4276	4390	5065	4013	3391	4156
11367	11168	10780	10402	10355	10133	9028	7595	8918
15850	18063	16935	18874	16617	15660	17255	15128	16014
15010	17301	18208	22759	23325	24344	26388	25431	25387
22117	34127	17692	25622	31364	33566	38692	29447	33901

0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
142	0	0	0	0	0	150	0	50
138	141	506	514	223	224	151	152	175
1269	897	1174	898	940	1125	1000	472	865
3070	2490	3030	2624	2042	1851	2147	1517	1838
5853	6701	5779	5311	5288	5287	5461	3913	4887
8745	10802	9850	12302	9707	10010	10066	10243	10106
8519	10381	9832	15862	12155	14859	18411	16662	16644
11848	22222	6923	9043	16776	17483	26017	16827	20109

0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	58	19
72	73	219	72	71	0	0	0	0
285	143	217	73	222	149	150	150	149
275	283	289	440	371	0	452	379	277
423	489	391	449	578	225	693	315	411
263	534	459	389	306	877	747	625	749
1013	558	889	996	661	661	1115	1036	937
182	531	1382	843	987	1453	799	1103	1118
0	0	0	0	0	948	614	877	813
0	0	0	2261	0	699	0	1202	633

SILICOSIS (502-502)

0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	70
0	67	68	0	0	0	0	0
67	0	0	0	67	0	70	0
0	304	240	77	0	71	0	66
524	449	150	302	384	317	168	177
1008	1084	1248	971	1057	1054	702	701
2055	1839	634	1235	1209	1185	698	1144
2617	1653	1162	1346	1291	1028	1965	2261
3145	1341	446	885	1774	1308	860	1266
0	873	2573	1699	0	833	2461	1622

OTHER DISEASES OF THE RESPIRATORY SYSTEM (510-519)

0	101	0	145	144	48	48	145
289	171	169	109	53	104	101	196
110	57	115	523	58	286	113	278
112	54	106	158	105	222	172	232
68	134	196	127	245	227	218	323
210	277	412	273	208	347	342	464
550	416	704	710	786	642	848	698
925	1141	1023	964	1253	1615	2057	1866
1349	1485	1810	1752	2539	3425	5225	3106
3591	2201	2883	3008	5868	7073	9038	3840
5989	4195	6230	6035	12888	15114	18107	7060
10354	8397	10695	11304	24476	26626	31488	12350
21096	24570	28637	31856	43515	49413	63518	18411
33547	35664	38336	49820	70353	76275	92927	40701
49865	60349	66964	68554	104656	127289	128602	53609
124890	126638	129503	157179	183725	191507	201805	100568

PULMONARY FIBROSIS (515-515)

0	0	0	0	48	48	48	48
58	0	0	55	0	0	0	0
55	0	0	58	0	0	57	0
0	0	160	53	0	56	0	0
136	0	0	127	0	0	218	54
351	277	275	68	277	69	68	66
412	208	493	142	429	214	212	70
463	537	546	482	487	1123	497	359
675	646	874	1038	1403	959	836	988
2873	2429	2002	1851	2897	1500	2533	1324
3518	3596	3678	3848	3913	3482	3605	1765
4948	7133	5258	6270	5899	4306	6315	3241
9178	7489	8616	10495	8824	11376	10819	4574
9041	12518	14870	12118	13769	11924	10609	6972
10782	12070	17857	11942	15521	10898	15484	10131
14952	4367	9434	11045	15940	13322	4922	13788

Death Rates for England and Wales (continued).

0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	75	0	25
0	0	217	147	0	0	0	0	0
508	408	157	150	217	75	385	79	179
702	267	367	194	612	682	93	178	317
1125	893	778	553	1212	881	780	230	630
1275	885	1210	1348	1481	1292	1598	315	1068
811	1922	728	1379	2300	2213	614	877	1234
1580	1587	1538	1507	1459	2098	1334	1202	1544

0	150	103	53	55	172	61	191	141
191	236	281	141	96	148	50	204	134
108	369	259	151	340	331	141	94	188
293	235	176	290	170	165	320	361	282
267	535	227	409	414	417	357	294	356
387	0	462	334	547	595	648	228	490
973	282	705	761	336	523	690	579	597
1520	1387	1533	1156	1499	1207	1363	1074	1214
2849	1865	1736	1609	1703	2610	2699	2397	2568
4196	5232	5931	4989	3711	5381	5569	4094	5014
7698	7010	7199	8606	8675	9972	10159	7949	9360
12984	12540	15975	13993	14598	16170	15026	18026	16407
18908	22560	25450	19918	24675	26545	29536	29344	28475
41173	40907	41645	40276	53142	58767	54801	59880	57816
50710	65744	66278	76552	89356	96744	91439	92078	93420
91627	125397	127692	128109	182349	207692	182789	171274	187251

0	50	0	0	0	0	0	0	0
48	0	0	0	48	0	0	0	0
0	53	52	101	0	47	0	0	15
0	59	0	58	0	55	53	103	70
53	0	0	0	59	60	0	0	20
65	0	115	0	55	54	0	0	18
209	141	70	138	67	0	314	232	182
217	219	365	217	357	142	72	0	71
712	287	289	146	444	373	525	300	399
825	566	1085	367	891	1420	1505	758	1227
2369	2038	2035	1796	2096	2249	2386	944	1859
3158	2757	3489	3790	3369	4091	3640	5087	4272
5290	5919	4890	4537	6169	6939	7468	7365	7257
10567	8854	6739	8763	11188	12593	11983	13552	12709
8925	9996	11653	11379	15769	16756	14728	16369	15951
9479	7937	14615	10550	22611	16783	18012	21034	18609

DISEASES OF THE DIGESTIVE SYSTEM (520-579)

935	861	887	579	765	618	765	581
982	1312	899	1039	744	777	556	686
1656	1646	919	2091	1620	1315	1074	1612
2235	2747	2607	2425	1895	1998	1945	1795
3396	3742	4302	2864	3431	3512	2948	3389
6591	6382	5696	5947	4849	6104	5737	6163
9274	11307	10698	10435	10218	7778	7769	10126
14669	17856	16571	16186	17538	16921	14544	16144
24219	24663	27277	25821	25924	26644	24730	25625
37997	40914	40368	35789	38847	36510	35262	40185
54200	58277	54192	58091	54929	53652	51471	58247
88144	89391	90196	86108	86811	81986	74116	89516
153288	154382	148632	137795	144808	133428	119707	134134
217702	217525	227928	214991	218589	225123	200786	230639
351303	335717	353571	324193	348115	296425	324731	346138
478452	488210	507719	553951	522651	462115	454471	566910

DISEASES OF OESOPHAGUS & STOMACH (530-537)

156	51	49	48	0	48	96	48
58	456	56	219	106	155	101	98
331	284	57	232	463	172	170	222
559	108	479	369	158	500	229	347
747	1336	652	573	674	680	437	699
1402	1595	1373	1094	554	1179	683	1127
2610	2636	2745	3265	2644	2070	1483	2235
5352	4699	4774	4270	4872	4634	3902	3659
8365	7425	9051	7720	8352	7603	6409	5153
13791	14954	13536	13806	11810	11503	10339	13439
22384	24345	20866	23086	19946	21287	18610	17562
40224	38284	39750	37446	35658	32513	28506	31006
70411	69242	68044	64082	63459	54390	50372	53288
93980	95418	101533	97621	96816	96834	90766	93461
142408	151989	155357	134896	157428	117698	140215	142676
193492	205240	234991	235344	219799	186511	200984	209246

DISEASES OF THE GENITO-URINARY SYSTEM (580-629)

571	304	542	531	335	190	574	145
866	1141	562	711	957	363	556	441
1546	1022	1322	1162	810	743	735	667
1900	1400	1330	1265	1158	1277	801	984
2649	2806	1955	1845	2205	1190	1528	1237
2875	3468	2402	2187	1940	2705	1912	1325
4809	3607	5419	3620	3144	3925	3673	1606
6740	4967	6342	6199	5568	4915	4895	3588
11401	8587	10112	9277	7684	5479	6966	4447
14797	13815	15138	13421	12107	13075	12872	10195
26501	23371	24844	23161	24012	22711	23975	19328
47828	41986	44296	41243	42349	41652	36313	33021
84658	78702	85403	77170	85217	75246	76664	77301
161075	164384	160316	151481	155120	155016	150098	150367
311770	296379	319643	284830	291353	281604	292903	289574
645558	572052	590909	578590	661074	566195	605414	613950

396	701	308	159	276	287	0	381	222
622	520	843	424	770	836	499	510	615
1409	1055	777	604	923	758	845	796	799
1407	1471	1587	1859	1701	1704	1012	1547	1421
2829	3371	3061	3097	2957	2741	3268	3994	3334
6388	5106	5021	4505	5466	5467	5996	5519	5660
10080	9027	10075	9414	9345	9414	9352	8919	9228
15991	14161	13944	13944	14566	14273	13771	15964	14669
21651	25685	20764	19668	20654	24310	21439	18130	21293
40036	35914	36094	32284	31320	35722	33185	33204	34037
56763	58771	57360	51186	54218	55930	56411	53282	55207
89131	76841	79692	76960	77276	79193	83528	81385	81368
133258	132790	131918	117074	126570	130521	125279	125086	126962
223356	218346	200968	199528	205166	222312	212334	204853	213166
340365	346790	374727	328276	348883	366108	364222	347559	359296
520537	568254	542308	560663	601021	665035	646431	557692	623052
0	50	51	0	55	0	0	127	42
239	47	187	141	193	197	100	102	133
325	211	155	201	49	142	235	140	172
352	176	353	290	283	440	160	258	286
907	214	510	351	296	596	178	705	493
839	872	404	834	711	1028	486	683	732
1946	1551	1832	1523	1748	1504	753	985	1080
3111	3358	2555	2240	2785	2059	2510	2362	2310
4843	5453	4269	5922	3702	4623	4573	3521	4239
10387	10251	10488	8658	8461	10687	7751	8112	8850
19457	16710	18077	17212	16771	17019	15930	14796	15915
30704	28637	29288	25459	28583	26885	29211	24897	26997
53686	48247	51789	42935	44834	49234	46478	41657	45789
93460	89782	85537	79879	80454	88634	86595	72644	82624
146856	139562	156227	125172	136662	148277	135011	129787	137691
201422	234127	213077	204974	208607	234965	248833	191106	224968
198	100	206	53	110	57	121	254	144
191	426	281	330	433	148	200	102	150
325	316	155	50	291	189	563	187	313
528	588	940	523	510	330	586	258	391
534	749	680	643	355	358	713	587	552
968	1059	1270	1335	601	650	378	626	551
2364	1269	1902	1454	874	1242	1004	869	1038
2967	2774	1971	2890	1785	2059	1291	1432	1594
4843	4592	3256	3363	3405	3729	3523	2472	3241
9218	8908	9403	7557	5937	5082	5493	4624	5066
17765	14346	16355	14892	12217	11096	12006	10467	11189
32810	34240	32134	29540	27562	21625	24732	19543	21966
65954	64440	65126	60197	52985	56394	50045	52129	52856
148114	148752	141524	135322	125864	136422	129094	120076	128530
268560	281430	292061	270000	257884	275055	274624	250804	266827
584518	584921	602308	574228	658643	644755	615744	519832	593443

DISEASES OF THE SKIN & SUBCUTANEOUS TISSUE (680-709)

0	0	49	0	0	48	0	0
0	114	0	0	53	104	0	49
0	0	57	0	0	57	0	0
0	54	0	0	105	0	57	0
68	0	0	0	123	0	55	0
140	69	69	0	139	69	137	133
275	277	422	71	143	143	212	210
264	67	68	207	70	140	284	144
202	194	187	130	200	0	139	353
718	304	400	540	297	214	616	265
749	824	826	1056	614	475	838	971
1466	1354	1337	1325	1233	264	965	876
2055	1971	3041	1605	2659	2488	2327	2287
2855	5905	4182	5386	4518	2878	3929	4711
8985	4917	6696	8403	6652	5231	6452	9287
14952	16594	23156	14444	13423	7494	16407	17843

DISEASES OF THE MUSCULOSKETELAL SYSTEM (710-739)

104	0	99	96	144	48	96	48
115	342	225	109	106	155	152	392
497	284	172	116	116	343	283	111
168	215	266	105	263	167	229	58
272	267	326	64	123	397	109	108
491	486	549	752	416	624	273	199
687	902	704	568	643	500	777	698
1057	1544	1364	1446	1183	1404	1419	861
1687	2195	1748	2336	2539	1644	1811	1271
3376	3568	3124	3625	3045	2644	3560	2516
5764	5169	5329	5206	5447	5539	5449	5383
10995	8939	8467	8302	7836	7996	9648	7533
16027	13664	15585	15064	12813	13746	12913	13265
19986	25272	23699	21993	24312	24260	25540	22989
45822	31739	38839	42459	35033	37925	41290	52343
99384	77729	87479	92608	93960	91590	101723	109489

SYMPTOMS, SIGNS & ILL-DEFINED CONDITIONS (780-799)

0	0	49	241	96	48	96	97
115	285	225	55	0	104	202	294
386	341	115	465	579	286	226	111
168	700	372	369	263	444	229	463
475	134	521	382	735	113	328	377
140	416	206	205	416	347	888	663
343	277	422	923	500	571	777	768
793	873	477	620	835	421	568	574
1214	516	811	908	668	822	1463	918
718	1139	1121	1388	1188	429	1575	1589
1123	1348	1201	1584	1304	1187	1090	1324
2566	1986	1961	2561	1761	2548	2544	2365
8630	8015	6842	6297	5923	6280	5584	2173
22127	19839	15799	17504	16351	15419	13556	6783
86253	77783	62500	61035	52328	57977	46022	23639
416887	401747	343053	304163	262584	253122	216571	150041

Death Rates for England and Wales (continued).

0	0	0	0	0	0	0	0	0
0	47	0	0	0	49	0	0	16
54	53	0	0	0	0	0	0	0
0	59	0	0	0	55	0	52	35
0	0	57	0	0	0	0	0	0
0	125	0	111	55	0	0	114	38
0	141	70	138	134	65	126	116	102
72	73	365	217	143	213	0	72	95
285	646	145	146	148	373	300	75	249
69	283	217	147	223	299	452	455	402
677	897	470	599	723	675	539	944	719
1053	1245	1194	583	1021	1364	1120	1696	1393
2589	1452	1445	2877	1322	2974	2341	2301	2538
5101	4781	3974	4044	5265	7588	7829	5358	6925
6491	8458	8740	11724	8870	10433	14115	9062	11203
18957	27778	26154	22607	43034	30769	32688	28245	30567
198	150	51	159	110	57	121	191	123
143	95	187	188	48	246	150	153	183
163	211	52	50	291	189	94	94	125
293	176	294	0	397	220	53	155	142
374	107	57	58	177	358	238	0	198
129	560	115	111	273	325	540	114	326
348	846	493	554	807	784	251	579	538
1085	1533	949	722	286	781	932	573	762
2493	1435	1375	1462	1703	1641	1874	1349	1621
2614	2757	2821	1834	3043	3288	3236	3260	3261
4399	5624	5791	4864	6434	5548	6695	5824	6022
7895	7204	10007	9620	12862	13345	11386	9281	11337
14406	12285	15559	13279	21701	19606	21734	19793	20377
24048	25854	24883	25110	36525	36487	34990	38134	36537
40162	54979	44064	47586	77201	84097	81927	70740	78921
94787	124603	110769	127355	179431	206993	220147	177284	201474
148	50	0	0	0	115	303	64	160
239	0	94	94	144	197	150	102	149
217	527	207	352	340	663	469	421	517
410	235	411	465	454	605	639	464	569
374	161	454	409	414	358	59	940	452
839	187	289	278	492	650	810	797	752
834	423	1127	761	471	654	942	1158	918
724	876	730	1156	1499	1420	1291	1432	1381
783	933	1230	1316	1036	1119	1874	1274	1422
825	1767	1374	1614	891	1644	1656	1668	1656
1269	1875	1487	2095	1446	2324	2540	1968	2277
1842	1156	2295	1360	2654	2533	2800	2142	2491
1238	2457	2890	2766	1322	2203	2675	3337	2738
7105	8146	6221	4719	6910	7427	8468	10085	8660
20284	18839	17480	20690	18397	21182	26082	30985	26083
138231	116667	122308	119819	99927	127273	158773	179087	155044

ACCIDENTS, POISONINGS & VIOLENCE (800-999)

15425	13519	12813	14858	11815	11793	12477	13121
57856	56471	58949	58267	61228	60482	63491	58506
61824	68570	59591	63499	64167	60256	68539	61211
45876	49599	42782	43763	44643	49895	48736	52004
40144	42563	42365	40919	40985	38292	40777	43785
42073	41690	39531	39647	41289	39748	44054	42545
41217	46476	39766	43728	41372	39892	40119	47769
44535	47929	48213	45044	47324	45426	49947	44342
48641	46937	49061	49241	51046	49658	47997	47438
58325	60270	58791	49132	53257	52301	52037	56273
62434	61124	63649	61335	59992	54206	60441	60454
67620	66095	68093	60231	59518	58699	66310	62451
99452	92760	93386	83838	88360	81052	88646	80389
135617	152102	132435	132855	131885	116776	133006	132655
248428	221278	251339	208757	212417	203575	213763	210215
430959	454148	403945	437553	428691	385512	387203	396594

TRANSPORT ACCIDENTS (800-848)

8881	7291	6653	7670	6266	6182	7936	7359
42554	41412	43664	41323	45549	46178	48944	43806
35604	41324	33674	35671	35873	35102	42152	35804
23748	24019	19741	19719	19742	21257	20764	21716
17932	18976	16359	14764	15622	13368	15394	14523
16058	15954	14755	12509	13440	12833	13319	13320
14770	17134	11684	13488	12576	11703	13137	13548
13414	16379	12821	13637	10926	11865	13693	11552
15449	16463	13794	13883	15100	13219	14211	14471
19825	22393	18742	14886	14930	15647	16912	16948
23132	20899	21917	17503	19716	18042	20371	18092
23090	21129	22549	19253	16904	17047	20086	16642
32740	32716	31931	23213	24175	23699	27338	21727
46871	44639	43913	41068	37220	28166	38703	29960
75472	51408	70982	51305	51885	48387	54624	43900
68602	71616	66895	64571	65436	74105	57424	41363

ACCIDENTAL POISONING (850-869)

260	101	246	531	191	95	191	242
1674	799	1236	1257	1276	1347	1212	1372
1766	2384	2931	3137	2719	2973	2543	2835
1397	1777	1756	2056	1948	2609	2345	2664
1494	1403	1173	2100	2328	1699	1583	1990
1823	1457	1990	2256	2009	2012	2322	2054
1923	2150	2182	2414	2287	2284	1836	1886
2048	2551	3478	2824	2088	2949	2483	1866
1956	2389	2185	1557	2138	3151	2577	1412
2729	1746	2163	2160	1857	1858	2054	2383
2695	1573	2102	1735	1381	1504	1090	1059
2107	2528	2317	1590	1937	1494	1403	1839
2466	2496	2408	2716	2538	2133	2210	1830
2379	2362	1626	2693	3657	2262	1965	3392
4043	4917	3125	2654	3991	3923	2151	3377
5277	5240	6861	2549	5872	1665	5742	6488

Death Rates for England and Wales (continued).

10978	13323	12282	12434	12850	15047	9867	11634	12182
58680	58125	54639	51258	48337	43540	46994	46506	45680
57533	57055	59137	55687	55739	56531	57125	58284	57313
48610	48409	48716	47514	47228	43600	46137	45839	45192
41849	41520	44390	44992	43116	43913	45276	46696	45295
41809	41472	41724	40656	42416	42382	39650	40683	40905
46437	44076	43821	44923	43630	46087	44125	41235	43815
44645	46423	47598	45661	47554	45445	47482	44312	45746
48928	48142	47388	46428	44936	45414	45952	42403	44589
55995	51608	55913	49013	46905	47007	45752	48291	47016
61585	55836	51021	50737	47929	52257	53948	48717	51640
60707	55407	59310	58984	55227	55328	54130	53632	54363
80135	74268	75572	69935	71271	72585	74008	66053	70882
116415	122366	120270	103809	107601	112367	107685	95178	105076
197566	197232	182811	189655	184297	183686	179503	164864	176017
362559	369048	315385	339864	315828	326573	317545	265625	303247
6033	5910	6629	7619	7390	9534	6417	7883	7944
43807	32208	38425	34816	33219	29617	30581	30834	30344
35322	24731	33194	31016	31268	29544	29994	28744	29427
19233	13411	19040	18994	19560	17154	18380	18769	18101
12651	10326	14400	14199	13130	12513	14557	13451	13507
11291	8220	12119	11290	12298	11204	9669	10982	10618
12930	8674	11272	11490	10555	10852	9415	9787	10018
11143	8832	11024	10765	12139	10722	11620	10380	10907
14600	8538	11793	11845	11179	11633	11019	9589	10747
14583	10604	14250	12400	9945	11210	10008	12433	11217
19203	11738	12755	11674	11133	11396	13468	10153	11672
15703	10939	12578	13215	13985	13637	13252	12672	13187
24086	15635	19671	19697	17405	17513	22403	16686	18867
27874	25146	29376	24267	27147	28253	27001	22849	26034
50304	38447	36781	37241	39093	34145	34060	37708	35304
49763	30952	45385	32404	39387	34266	36691	34856	35271
148	250	308	265	662	345	121	0	155
1387	1561	1593	1837	1926	1181	948	1072	1067
2275	2953	2537	2316	1894	2273	1455	2481	2069
3342	3706	3173	3079	1701	2089	2078	1702	1956
2295	2889	2891	3155	2839	2443	2317	2526	2428
1936	1806	2712	1557	2241	2273	2323	2048	2214
2850	1834	2184	1730	2487	1896	2448	1969	2104
2098	2482	2555	1734	1499	2272	2582	1145	1999
1994	2439	2098	1682	1555	1491	1799	1423	1571
2339	1414	1953	1541	1336	1270	1957	1592	1606
1354	2201	1643	1197	1807	1350	1462	1810	1540
1228	1690	1469	1360	1531	1169	1400	1071	1213
2251	1675	1667	775	991	1101	1672	1611	1461
2551	3719	2419	2022	1974	1776	1917	1418	1703
2840	2691	4370	3793	2957	1265	2455	2046	1922
3160	6349	2308	4521	729	4196	1334	3606	3045

ACCIDENTAL FALLS (880-888)

987	810	1133	917	1005	571	478	533
1444	1597	1630	1366	1754	985	1010	882
2594	2214	2126	1917	2083	1944	1526	1557
2570	2154	1596	1635	1685	2331	1716	1853
2038	2806	2542	1845	1960	2946	2347	2528
2524	2358	2471	1777	2148	2844	2595	1789
3366	2983	2956	2627	2715	2070	2825	4400
3899	4162	4705	3375	3619	3440	3902	4018
5599	4713	5430	5644	5078	4795	4180	3883
6034	8046	6087	5939	5125	5644	6436	6157
8759	7491	8331	9280	6751	6964	8970	9620
11545	11377	12656	11393	10829	8699	12192	12000
25616	22730	22301	21978	21999	19315	21638	18525
48061	57156	46933	46230	46256	44819	45383	45789
114106	108628	125446	107032	101552	95466	98065	91600
292876	310044	262436	286321	271812	235637	240361	240065

SUICIDE & SELF-INFLICTED INJURY (950-959)

260	152	49	96	144	48	287	291
3060	2339	2641	3389	3189	3213	3637	4116
9329	9025	8964	9470	8968	9662	10623	9396
9499	10286	9365	10334	10792	12599	12127	12740
9510	8954	11862	10309	10353	11102	11081	13340
10869	11931	11461	10800	11777	11654	12499	13386
12159	13319	11191	11713	11718	13273	13491	15294
14008	13895	15753	13500	16424	15446	16531	15355
13965	13300	15168	14078	13764	16301	14559	15389
15371	15637	15058	13112	16193	15719	14242	16816
15496	16779	16588	17503	18105	12978	16347	16503
18692	18962	17647	14131	16200	17135	16665	15591
19589	18263	20400	19756	18977	17656	20940	16810
18558	20784	18820	17504	20869	18914	19450	24308
22462	25928	21429	17691	22616	21360	20215	26171
12313	17467	18010	19541	15940	22481	16407	21898

Death Rates for England and Wales (continued).

890	1102	668	635	938	459	545	636	546
1483	3642	1265	1178	1155	886	948	868	900
1246	3533	1864	1712	1505	1752	1502	1358	1537
1994	2412	1469	1568	1701	1155	1438	1392	1328
2402	2782	1871	2104	1952	2026	1248	1762	1678
2258	2366	2078	2392	2186	2869	1783	1764	2138
2920	3032	3170	2769	2891	3269	2887	2606	2920
3256	3869	3139	4118	3213	2343	4017	3078	3146
3703	4879	3979	4021	2887	4176	3898	4270	4114
5710	5797	4774	5430	4527	3587	4214	4928	4243
7783	6358	6965	7409	6506	7197	6388	5903	6496
10001	9249	9916	10689	10208	9059	7839	9281	8726
17558	16194	15670	15160	15202	15200	13375	13809	14128
38076	36657	37843	30839	31589	31644	29877	27734	29751
86410	84198	79388	80690	78187	76510	70881	61093	69494
231438	225397	174615	220799	191831	199301	186791	150240	178777
99	200	206	106	55	115	121	254	163
4017	4162	3655	3957	4333	4280	4240	5258	4592
8993	9808	9528	9617	10633	11979	12955	13014	12649
12138	14705	14691	12953	13777	15065	13639	13458	14054
13505	14018	14287	15017	14254	15492	15746	15800	15679
14840	14883	14658	15628	15305	15426	14153	14851	14810
15850	15726	15147	16751	18017	17389	16006	14421	15938
15051	16496	18981	16328	18708	17965	16210	15320	16498
15597	18439	17942	18279	17619	16257	16942	14609	15936
17197	18522	19458	18050	18480	17487	15953	17436	16958
18611	17933	15807	18185	15253	18668	16546	18180	17798
18423	16009	17077	18171	15619	16852	17732	15795	16793
17558	17199	17559	16820	18506	16962	17499	15880	16780
22226	21427	20563	21739	21882	21472	19971	18122	19855
19473	23068	23307	25172	24967	24976	29150	22216	25447
20537	23016	29231	19593	19694	20280	22015	22837	21710

TABLE A2.2: Regional Adjustment Factors for the South West (SW) and South East (SE) Standard Regions, for the years 1979-83 and 1989.

	Causes of Death	1979 - 83*				1989#	
		SW		SE		SW	SE
		15-64	65+	15-64	65+	All ages	
1.	All Causes	0.89	0.91	0.91	0.95	0.91	0.92
2.	Infectious and Parasitic Diseases	0.88	0.82	1.05	0.99	0.83	1.09
3.	Tuberculosis	0.63	0.70	1.03	1.02	0.62	1.12
4.	All Neoplasms	0.87	0.91	0.92	1.01	0.91	0.94
5.	Ca. Lip, Oral Cavity and Pharynx	0.90	0.88	0.84	0.84	0.77	0.86
6.	Ca. Digestive Organs and Peritoneum					0.94	0.90
7.	Ca. Oesophagus	0.97	0.90	0.88	0.95	1.06	0.88
8.	Ca. Stomach	0.80	0.87	0.88	0.91	0.88	0.87
=9.	Ca. Peritoneum (mesothelioma)						
=10.	Ca. Respiratory System						
11.	Ca. Lung	0.78	0.83	0.89	1.04	0.79	0.93
=12.	Ca. Pleura (mesothelioma)						
=13.	Ca. Bone, Tissue, Skin and Breast						
14.	Ca. Genito-urinary Organs					1.06	1.00
15.	Ca. Prostate	0.94	1.11	1.03	1.05	1.15	1.00
=16.	Ca. Other and Unspecified Sites						
17.	Ca. Lymphatic and Haematopoietic Tissue					0.96	0.99
18.	Benign Neoplasms	0.75	0.88	0.85	0.96	0.83	1.01
=19.	Unspecified Neoplasms						
20.	Endocrine and Nutritional Diseases	1.03	1.00	0.92	0.98	0.99	1.02
21.	Diseases of Blood and Blood-forming Organs	1.04	0.85	1.03	0.95	1.21	0.94
22.	Diseases of the Nervous System	0.96	0.94	0.96	0.97	0.92	1.00
23.	Diseases of the Circulatory System	0.89	0.95	0.87	0.92	0.94	0.88
24.	Hypertensive Disease	0.87	0.97	0.92	1.04	0.93	1.08
25.	Ischaemic Heart Disease	0.90	0.94	0.87	0.93	0.92	0.85
26.	Diseases of Pulmonary Circulation					0.98	1.04
27.	Cerebrovascular Disease	0.84	0.96	0.81	0.86	0.98	0.86
28.	Diseases of the Respiratory System	0.77	0.80	0.89	0.95	0.78	0.93
=29.	Acute Respiratory Infections						
=30.	Other Disease of Upper Respiratory Tract						
=31.	Pneumonia and Influenza						
32.	Bronchitis, Emphysema and Asthma	0.70	0.70	0.85	0.92	0.77	0.91
33.	Chronic Obstructive Pulmonary Disease					0.75	0.92
34.	Pneumoconiosis	0.50	0.59	0.67	0.31		
=35.	Coalworkers Pneumoconiosis						
=36.	Asbestosis						
=37.	Silicosis						
=38.	Other Diseases of the Respiratory System						
=39.	Pulmonary Fibrosis						
40.	Diseases of the Digestive System	0.86	0.92	0.93	0.95	0.91	0.98
=41.	Diseases of Oesophagus and Stomach						
42.	Diseases of the Genito-urinary System	0.82	0.92	0.98	0.99	0.98	0.98
43.	Diseases of the Skin and Subcutaneous Tissue	1.43	0.93	0.96	1.03	0.76	1.10
44.	Diseases of the Musculosketelal System	0.88	0.95	0.84	1.01	1.01	0.88
45.	Symptoms, Signs and Ill-defined Conditions	0.59	1.06	1.48	0.95	1.18	1.05
46.	Accidents, Poisonings and Violence	1.02	0.80	0.99	0.89	0.98	0.96
=47.	Transport Accidents						
=48.	Accidental Poisoning						
=49.	Accidental Falls						
=50.	Suicide and Self-inflicted Injury						

* Taken from the OPCS 1981 decennial supplement microfiche.
\# Taken from the OPCS 1989 area mortality statistics microfiche.
= No adjustment factor possible.

APPENDIX 3.

TABLE A3.1: Devonport Dockyard, non-responders. Cause specific mortality for 50 disease groups, with and without regional adjustment.

Causes of Death	Without regional adjustment Obs	Exp	SMR	95% CI	With regional adj. SMR	95% CI
All Causes	294	209.1	141	125-157	156	138-173
Infectious and Parasitic Diseases	3	0.9	333	69-974	394	81-1151
Tuberculosis	1	0.4	242	6-1350	363	9-2022
All Neoplasms	75	58.3	129	100-158	143	111-176
Ca. Lip, Oral Cavity and Pharynx	2	0.8	262	32-945	295	36-1064
Ca. Digestive Organs and Peritoneum	21	17.1	123	76-188	131	81-200
Ca. Oesophagus	1	1.9	53	1-297	58	1-322
Ca. Stomach	7	5.4	129	52-266	152	61-313
Ca. Peritoneum (mesothelioma)	1	0.1	1076	27-5993		
Ca. Respiratory System	33	24.2	136	90-183		
Ca. Lung	27	23.4	116	76-168	142	94-207
Ca. Pleura (mesothelioma)	6	0.2	2917	1070-6349		
Ca. Bone, Tissue, Skin and Breast	2	0.8	250	30-902		
Ca. Genito-urinary Organs	8	7.4	109	47-214	102	44-202
Ca. Prostate	3	3.8	79	16-230	72	15-211
Ca. Other and Unspecified Sites	6	4.2	141	52-308		
Ca. Lymphatic and Haematopoietic Tissue	2	3.3	60	7-218	63	8-227
Benign Neoplasms	0	0.2	-	0-2445	-	0-2982
Unspecified Neoplasms	1	0.4	251	6-1399		
Endocrine and Nutritional Diseases	3	2.2	135	28-394	134	28-391
Diseases of Blood and Blood-forming Organs	1	0.4	230	6-1283	258	6-1437
Diseases of the Nervous System	4	2.7	150	41-385	159	43-407
Diseases of the Circulatory System	163	104.8	156	132-179	167	141-192
Hypertensive Disease	1	2.1	49	1-271	52	1-289
Ischaemic Heart Disease	113	70.8	160	130-189	172	140-204
Diseases of Pulmonary Circulation	5	1.1	472	153-1100	481	156-1123
Cerebrovascular Disease	23	18.4	125	79-187	134	85-200
Diseases of the Respiratory System	32	23.7	135	88-182	170	111-229
Acute Respiratory Infections	2	0.2	840	102-3032		
Other Disease of Upper Respiratory Tract	0	0.0	-	0-14688		
Pneumonia and Influenza	10	8.1	124	59-228		
Bronchitis, Emphysema and Asthma	8	10.0	80	35-158	115	50-226
Chronic Obstructive Pulmonary Disease	14	13.9	100	55-169	134	73-225
Pneumoconiosis	0	0.3	-	0-1105	-	0-1934
Coalworkers Pneumoconiosis	0	0.2	-	0-1987		
Asbestosis	0	0.0	-	0-12936		
Silicosis	0	0.0	-	0-12908		
Other Diseases of the Respiratory System	1	1.0	104	3-580		
Pulmonary Fibrosis	1	0.3	404	10-2248		
Diseases of the Digestive System	6	5.0	119	44-260	133	49-289
Diseases of Oesophagus and Stomach	2	1.8	111	13-402		
Diseases of the Genito-urinary System	3	2.3	133	27-390	149	31-435
Diseases of the Skin and Subcutaneous Tissue	0	0.1	-	0-4345	-	0-4163
Diseases of the Musculosketelal System	1	0.6	168	4-937	180	5-1005
Symptoms, Signs and Ill-defined Conditions	0	0.2	-	0-2105	-	0-2430
Accidents, Poisonings and Violence	2	6.4	31	4-113	33	4-119
Transport Accidents	1	2.0	50	1-277		
Accidental Poisoning	0	0.2	-	0-1567		
Accidental Falls	0	0.8	-	0-466		
Suicide and Self-inflicted Injury	0	1.8	-	0-210		

TABLE A3.2: Chatham Dockyard, non-responders. Cause specific mortality for 50 disease groups, with and without regional adjustment.

Causes of Death	Without regional adjustment Obs	Exp	SMR	95% CI	With regional adj. SMR	95% CI
All Causes	320	253.4	126	112-140	135	120-149
Infectious and Parasitic Diseases	2	1.1	187	23-674	184	22-663
Tuberculosis	2	0.5	417	50-1505	407	49-1469
All Neoplasms	91	71.1	128	102-154	130	104-157
Ca. Lip, Oral Cavity and Pharynx	0	0.9	-	0-396	-	0-471
Ca. Digestive Organs and Peritoneum	27	20.7	130	86-189	145	95-210
Ca. Oesophagus	2	2.3	86	10-311	93	11-336
Ca. Stomach	12	6.5	183	95-320	204	105-356
Ca. Peritoneum (mesothelioma)	0	0.1	-	0-3363		
Ca. Respiratory System	38	29.5	129	88-170		
Ca. Lung	34	28.5	119	79-160	121	78-161
Ca. Pleura (mesothelioma)	4	0.3	1558	425-3989		
Ca. Bone, Tissue, Skin and Breast	0	1.0	-	0-382		
Ca. Genito-urinary Organs	8	9.0	88	38-174	88	38-174
Ca. Prostate	5	4.7	106	34-247	101	33-236
Ca. Other and Unspecified Sites	10	5.2	191	91-352		
Ca. Lymphatic and Haematopoietic Tissue	8	4.0	199	86-392	201	87-396
Benign Neoplasms	0	0.2	-	0-2036	-	0-2238
Unspecified Neoplasms	0	0.5	-	0-759		
Endocrine and Nutritional Diseases	3	2.7	110	23-322	114	24-334
Diseases of Blood and Blood-forming Organs	0	0.5	-	0-693	-	0-716
Diseases of the Nervous System	6	3.3	184	68-401	191	70-415
Diseases of the Circulatory System	163	127.1	128	109-148	142	120-163
Hypertensive Disease	2	2.4	82	10-296	82	10-296
Ischaemic Heart Disease	107	86.1	124	101-438	136	111-162
Diseases of Pulmonary Circulation	1	1.3	79	2-438	76	192-422
Cerebrovascular Disease	29	22.2	130	87-187	154	103-221
Diseases of the Respiratory System	39	28.4	138	94-181	147	101-193
Acute Respiratory Infections	1	0.3	356	9-1981		
Other Disease of Upper Respiratory Tract	0	0.0	-	0-7344		
Pneumonia and Influenza	12	9.5	126	65-221		
Bronchitis, Emphysema and Asthma	13	11.8	111	59-189	123	65-210
Chronic Obstructive Pulmonary Disease	16	16.9	95	54-154	103	59-167
Pneumoconiosis	0	0.4	-	0-923	-	0-2383
Coalworkers Pneumoconiosis	0	0.2	-	0-1672		
Asbestosis	0	0.0	-	0-10385		
Silicosis	0	0.0	-	0-10994		
Other Diseases of the Respiratory System	1	1.2	84	2-470		
Pulmonary Fibrosis	1	0.3	333	8-1856		
Diseases of the Digestive System	6	6.1	99	36-214	104	38-227
Diseases of Oesophagus and Stomach	1	2.2	46	1-257		
Diseases of the Genito-urinary System	4	2.7	149	40-380	150	41-385
Diseases of the Skin and Subcutaneous Tissue	0	0.1	-	0-3581	-	0-3533
Diseases of the Musculosketelal System	0	0.7	-	0-501	-	0-517
Symptoms, Signs and Ill-defined Conditions	0	0.2	-	0-1722	-	0-1487
Accidents, Poisonings and Violence	3	7.5	40	8-116	42	9-122
Transport Accidents	0	2.4	-	0-156		
Accidental Poisoning	0	0.3	-	0-1347		
Accidental Falls	0	0.9	-	0-391		
Suicide and Self-inflicted Injury	1	2.1	49	1-272		

TABLE A3.3: Portsmouth Dockyard, non-responders. Cause specific mortality for 50 disease groups, with and without regional adjustment.

Causes of Death	Without regional adjustment Obs	Exp	SMR	95% CI	With regional adj. SMR	95% CI
All Causes	532	498.0	107	98-116	114	104-124
Infectious and Parasitic Diseases	2	2.1	95	11-343	93	11-337
Tuberculosis	1	0.9	112	3-625	109	3-609
All Neoplasms	173	141.4	122	104-141	125	106-144
Ca. Lip, Oral Cavity and Pharynx	2	1.9	105	13-379	125	15-452
Ca. Digestive Organs and Peritoneum	45	41.2	109	77-141	121	86-157
Ca. Oesophagus	6	4.7	127	46-276	137	50-298
Ca. Stomach	17	12.7	134	78-214	149	86-238
Ca. Peritoneum (mesothelioma)	1	0.2	457	12-2544		
Ca. Respiratory System	86	58.0	148	117-179		
Ca. Lung	77	56.0	138	107-168	139	106-169
Ca. Pleura (mesothelioma)	5	0.6	907	294-2117		
Ca. Bone, Tissue, Skin and Breast	4	2.0	203	55-521		
Ca. Genito-urinary Organs	11	18.0	61	31-110	61	31-110
Ca. Prostate	4	9.4	43	12-109	41	11-104
Ca. Other and Unspecified Sites	17	10.8	158	92-252		
Ca. Lymphatic and Haematopoietic Tissue	7	8.1	86	35-177	87	35-179
Benign Neoplasms	0	0.4	-	0-1025	-	0-1129
Unspecified Neoplasms	1	1.0	101	3-560		
Endocrine and Nutritional Diseases	4	5.5	73	20-186	75	21-193
Diseases of Blood and Blood-forming Organs	2	1.1	188	23-677	194	23-699
Diseases of the Nervous System	13	6.6	196	104-336	203	108-347
Diseases of the Circulatory System	240	249.9	96	84-108	106	93-120
Hypertensive Disease	4	4.6	87	24-222	87	24-223
Ischaemic Heart Disease	170	171.2	99	84-114	109	93-126
Diseases of Pulmonary Circulation	1	2.4	41	2-230	40	1-221
Cerebrovascular Disease	26	42.9	61	40-89	71	47-105
Diseases of the Respiratory System	63	53.2	118	89-148	127	95-158
Acute Respiratory Infections	0	0.5	-	0-706		
Other Disease of Upper Respiratory Tract	0	0.0	-	0-7494		
Pneumonia and Influenza	8	16.9	47	20-100		
Bronchitis, Emphysema and Asthma	19	21.5	88	53-138	98	59-153
Chronic Obstructive Pulmonary Disease	36	32.5	111	75-147	120	81-160
Pneumoconiosis	0	0.8	-	0-488	-	0-1249
Coalworkers Pneumoconiosis	0	0.4	-	0-904		
Asbestosis	0	0.1	-	0-5003		
Silicosis	0	0.1	-	0-6009		
Other Diseases of the Respiratory System	6	2.3	257	94-559		
Pulmonary Fibrosis	1	0.6	172	4-956		
Diseases of the Digestive System	13	12.2	107	57-182	113	60-194
Diseases of Oesophagus and Stomach	3	4.2	71	15-208		
Diseases of the Genito-urinary System	6	5.1	117	43-255	119	43-258
Diseases of the Skin and Subcutaneous Tissue	0	0.2	-	0-1798	-	0-1776
Diseases of the Musculosketelal System	0	1.5	-	0-250	-	0-258
Symptoms, Signs and Ill-defined Conditions	1	0.4	236	6-1316	200	5-1112
Accidents, Poisonings and Violence	10	14.9	67	32-124	70	34-129
Transport Accidents	1	4.4	23	1-126		
Accidental Poisoning	1	0.5	187	5-1041		
Accidental Falls	2	1.9	108	13-389		
Suicide and Self-inflicted Injury	2	4.2	48	6-172		

TABLE A3.4: Devonport Dockyard. Cause specific mortality for 12 disease groups by calendar year period, for study responders and non-responders.

All Causes

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	50	73	53- 93	29	420	281-603
1973	70	79	61- 98	15	173	97-285
1974	93	98	78-118	10	108	52-198
1975	122	122	100-144	17	178	103-284
1976	104	96	78-114	15	151	85-250
1977	125	111	92-131	20	198	121-306
1978	134	109	91-128	11	101	51-181
1979	104	79	64- 94	16	141	81-229
1980	142	104	87-121	14	122	66-204
1981	142	100	84-117	20	177	108-273
1982	142	95	79-110	13	111	59-190
1983	137	86	72-101	26	213	139-312
1984	139	84	70- 98	36	300	202-397
1985	170	96	81-110	13	104	55-178
1986	183	99	85-114	16	128	73-208
1987	195	104	89-119	10	80	39-148
1988	203	99	85-113	10	72	35-133
1989	34	100	66-134	3	132	27-385
Total	2289	97	93-101	294	156	138-173

All Neoplasms

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	13	71	38-121	11	582	290-1041
1973	20	84	51-130	3	126	26-367
1974	33	127	84-171	4	155	42-396
1975	44	160	113-208	4	150	41-384
1976	37	122	83-162	4	144	39-368
1977	34	107	71-143	7	247	99-508
1978	40	117	80-153	0	-	0-123
1979	33	90	59-121	3	97	20-285
1980	44	113	80-146	5	156	51-364
1981	47	115	82-148	2	63	8-228
1982	52	121	88-154	3	93	19-273
1983	59	128	96-161	5	148	48-346
1984	46	93	66-120	6	175	64-382
1985	64	124	93-154	4	116	32-296
1986	63	117	88-146	7	202	81-416
1987	66	118	90-147	2	57	7-208
1988	82	140	110-170	4	109	30-278
1989	12	125	64-218	1	167	4-932
Total	789	117	109-125	75	143	111-176

Ca. Stomach

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	1	59	1-329	0	-	0-1949
1973	3	133	27-390	1	418	11-2327
1974	3	123	25-361	1	382	10-2126
1975	8	314	135-618	1	379	10-2114
1976	5	182	59-425	1	371	9-2067
1977	1	36	1-201	0	-	0-1411
1978	2	65	8-236	0	-	0-1306
1979	5	157	51-368	0	-	0-1297
1980	5	149	48-349	1	344	9-1917
1981	9	258	118-489	0	-	0-1302
1982	4	118	32-302	0	-	0-1383
1983	3	78	16-228	0	-	0-1267
1984	6	152	56-330	0	-	0-1296
1985	5	128	42-300	0	-	0-1364
1986	8	193	83-380	0	-	0-1349
1987	3	73	15-215	0	-	0-1408
1988	7	157	63-324	2	698	84-2521
1989	3	410	84-1197	0	-	0-7929
Total	81	145	113-176	7	461	61-313

Ca. Peritoneum

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	0	-	0-5185	0	-	0-58373
1973	1	1205	30-6710	1	14485	366-80684
1974	0	-	0-4280	0	-	0-54056
1975	1	1288	33-7174	0	-	0-65498
1976	1	1579	40-8793	0	-	0-83970
1977	1	1176	30-6551	0	-	0-55194
1978	0	-	0-3810	0	-	0-52199
1979	0	-	0-4944	0	-	0-67254
1980	1	1095	28-6101	0	-	0-59993
1981	0	-	0-5503	0	-	0-82675
1982	0	-	0-5616	0	-	0-79844
1983	0	-	0-4240	0	-	0-70300
1984	1	1267	32-7055	0	-	0-76809
1985	0	-	0-4032	0	-	0-70336
1986	0	-	0-4819	0	-	0-90135
1987	3	3992	824-11670	0	-	0-97163
1988	1	1200	30-6686	0	-	0-82656
1989	0	-	0-27366	0	-	0-509200
Total	10	731	351-1344	1	1076	27-5993

Ca. Lung

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	4	56	15-143	5	647	210-1511
1973	7	76	30-156	0	-	0-381
1974	13	128	68-218	2	192	23-693
1975	10	94	45-173	1	94	2-526
1976	15	129	72-213	1	91	2-509
1977	14	114	62-191	3	268	55-783
1978	13	100	53-171	0	-	0-320
1979	6	43	16- 94	2	171	21-617
1980	14	96	52-161	2	165	20-595
1981	11	74	37-132	1	86	2-480
1982	21	136	84-207	1	87	2-482
1983	16	96	55-156	3	249	51-727
1984	9	53	24-100	2	172	21-620
1985	20	113	69-175	1	86	2-482
1986	19	106	64-166	3	266	55-777
1987	20	111	68-171	0	-	0-336
1988	25	130	84-192	0	-	0-317
1989	4	127	35-326	0	-	0-1965
Total	241	99	87-112	27	142	94-207

Ca. Pleura

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	1	1508	38-8399	2	37734	4566-136219
1973	1	1013	26-5640	0	-	0-45523
1974	4	4416	1203-11305	1	14043	355-78221
1975	3	2434	502-7116	0	-	0-36460
1976	2	1399	169-5050	1	9208	233-51287
1977	3	2137	441-6248	1	9413	238-52432
1978	6	3831	1405-8339	0	-	0-32640
1979	2	1520	184-5489	0	-	0-39020
1980	3	1719	355-5026	1	8558	217-47668
1981	3	1652	341-4828	0	-	0-31705
1982	9	5351	2450-10155	0	-	0-34540
1983	5	2002	649-4673	0	-	0-26185
1984	5	1869	606-4362	0	-	0-24799
1985	2	687	83-2480	0	-	0-24017
1986	10	3028	1453-5569	0	-	0-20390
1987	5	1483	480-3461	0	-	0-21562
1988	2	615	74-2220	0	-	0-22243
1989	0	-	0-7024	0	-	0-138567
Total	66	1983	1505-2461	6	2917	1070-6349

TABLE A3.4 (cont.): Devonport Dockyard. Cause specific mortality for 12 disease groups by calendar year period, for study responders and non-responders.

	Circulatory System						Pulmonary Circulation					
				Non-responders						Non-responders		
Year	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
1972	29	87	59-126	15	437	245-721	1	321	8-1788	1	3021	76-16827
1973	41	96	66-125	10	230	110-422	1	247	6-1376	0	-	0-8720
1974	50	107	77-136	5	106	34-246	3	644	133-1883	1	2094	53-11662
1975	60	119	89-150	9	182	83-346	1	208	5-1161	0	-	0-7390
1976	57	106	78-133	6	119	43-258	3	547	113-1600	0	-	0-7230
1977	71	124	95-153	10	189	91-348	2	362	44-1307	1	1912	48-10648
1978	72	114	87-140	10	174	84-321	3	523	108-1528	0	-	0-6931
1979	54	79	58-100	10	168	80-308	4	394	107-1008	0	-	0-4024
1980	78	110	86-135	4	66	18-169	3	295	61-861	0	-	0-4004
1981	74	101	78-123	13	218	116-373	2	179	22-646	1	1038	26-5781
1982	72	94	72-115	3	49	10-144	3	265	55-775	0	-	0-4074
1983	66	80	61-100	18	284	168-449	2	172	21-621	0	-	0-8126
1984	62	72	54- 90	26	415	271-608	0	-	0-655	1	1065	27-5933
1985	83	91	71-111	8	124	53-244	0	-	0-600	0	-	0-7908
1986	91	97	77-117	6	94	34-204	0	-	0-589	0	-	0-8078
1987	88	92	73-111	5	78	25-183	0	-	0-580	0	-	0-8032
1988	92	87	70-105	4	56	15-144	0	-	0-511	0	-	0-7042
1989	12	69	36-120	1	85	2-476	0	-	0-3066	0	-	0-42608
Total	1152	95	90-101	163	167	141-192	28	232	154-335	5	481	156-1123

	Respiratory System						Bronchitis, Emphysema and Asthma					
				Non-responders						Non-responders		
Year	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
1972	7	119	48-246	2	293	35-1057	2	65	8-234	1	272	7-1514
1973	7	94	38-194	2	237	29-856	2	53	6-190	0	-	0-848
1974	10	131	63-241	0	-	0-424	2	51	6-183	0	-	0-812
1975	16	194	111-314	3	328	68-959	1	24	1-134	1	216	5-1204
1976	7	73	29-150	4	396	108-1013	1	23	1-130	0	-	0-816
1977	13	142	76-243	3	309	64-904	0	-	0-89	1	231	6-1287
1978	19	182	110-284	1	91	2-506	4	86	24-221	1	211	5-1173
1979	6	53	19-115	1	85	2-473	2	41	5-149	1	206	5-1145
1980	12	102	53-178	3	256	53-749	1	22	1-121	1	224	6-1247
1981	10	82	40-152	3	263	54-770	5	112	36-262	0	-	0-915
1982	7	50	20-103	4	307	83-785	2	43	5-156	0	-	0-901
1983	7	47	19- 96	1	73	2-404	1	22	1-120	0	-	0-937
1984	12	96	50-168	1	95	2-529	5	112	36-261	1	276	7-1539
1985	10	68	32-124	1	84	2-469	5	103	33-241	0	-	0-995
1986	14	91	50-153	1	85	2-471	5	113	37-263	1	311	8-1733
1987	20	133	81-205	1	87	2-487	5	129	42-301	0	-	0-1356
1988	12	67	34-116	1	71	2-396	2	39	5-142	0	-	0-1001
1989	3	99	20-289	0	-	0-1564	0	-	0-435	0	-	0-6042
Total	192	95	82-109	32	170	111-230	45	60	43- 78	8	115	49-226

	Asbestosis						Pulmonary Fibrosis					
				Non-responders						Non-responders		
Year	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
1972	0	-	0-34323	0	-	0-522308	0	-	0-3620	0	-	0-36482
1973	0	-	0-32413	0	-	0-395299	0	-	0-2941	0	-	0-26693
1974	0	-	0-38685	0	-	0-361651	0	-	0-2693	0	-	0-27241
1975	0	-	0-17974	0	-	0-210879	0	-	0-2431	0	-	0-23598
1976	0	-	0-30429	0	-	0-379734	0	-	0-1975	0	-	0-22300
1977	0	-	0-17531	0	-	0-198398	0	-	0-2379	0	-	0-25635
1978	0	-	0-15896	0	-	0-205032	0	-	0-1804	0	-	0-19347
1979	0	-	0-20148	0	-	0-279759	0	-	0-3120	0	-	0-37204
1980	0	-	0-16493	0	-	0-198595	1	787	20-4382	0	-	0-33422
1981	1	4219	107-23498	0	-	0-230643	0	-	0-3065	0	-	0-34085
1982	0	-	0-13175	0	-	0-181210	0	-	0-2636	0	-	0-34179
1983	2	6947	841-25080	0	-	0-182846	0	-	0-2636	1	8962	227-49919
1984	3	10290	2123-30081	0	-	0-200623	1	539	13-2932	0	-	0-25085
1985	0	-	0-13373	0	-	0-186207	0	-	0-1604	0	-	0-21378
1986	0	-	0-8982	0	-	0-162488	0	-	0-1476	0	-	0-21011
1987	1	2746	69-15293	0	-	0-175534	0	-	0-1419	0	-	0-19766
1988	0	-	0-9864	0	-	0-173902	0	-	0-1318	0	-	0-18882
1989	0	-	0-60669	0	-	0-999999	0	-	0-7380	0	-	0-115531
Total	7	1718	690-3539	0	-	0-12936	2	68	8-245	1	404	10-2248

TABLE A3.5: Chatham Dockyard. Cause specific mortality for 12 disease groups by calendar year period, for study responders and non-responders.

All Causes

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	2	15	2- 56	10	303	145-557
1973	22	53	33- 80	13	124	66-212
1974	33	73	48- 97	6	53	19-116
1975	50	103	75-132	15	127	71-210
1976	47	90	64-115	23	185	117-278
1977	67	124	94-153	15	119	67-197
1978	61	104	78-130	23	170	108-255
1979	67	106	81-132	24	174	112-259
1980	68	104	80-129	13	94	50-161
1981	66	98	74-122	12	84	43-146
1982	64	90	68-113	24	162	103-240
1983	63	84	64-105	27	174	115-253
1984	64	82	62-102	32	210	137-282
1985	71	85	65-105	17	108	63-173
1986	99	117	94-140	21	134	83-206
1987	82	96	76-117	22	142	89-215
1988	75	82	63-100	15	91	51-149
1989	44	67	48- 87	8	71	31-140
Total	1045	91	86- 97	320	135	120-149

All Neoplasms

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	0	-	0-102	4	427	116-1093
1973	7	59	24-123	3	100	21-291
1974	14	107	59-180	0	-	0-112
1975	22	158	99-239	4	117	32-299
1976	16	104	60-170	7	191	77-394
1977	27	168	110-244	5	133	43-312
1978	20	116	71-179	7	178	71-366
1979	25	135	88-200	10	251	120-461
1980	22	113	71-171	5	122	39-285
1981	22	108	68-164	3	70	14-205
1982	21	99	61-151	6	137	50-299
1983	19	84	50-131	5	109	35-255
1984	26	107	70-157	12	258	133-451
1985	24	95	61-141	7	150	60-310
1986	28	109	72-157	8	173	74-340
1987	23	87	55-131	4	86	23-221
1988	26	95	62-139	0	-	0-79
1989	23	120	76-180	1	32	1-176
Total	365	107	96-118	91	130	104-157

Ca. Stomach

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	0	-	0-1045	0	-	0-3862
1973	1	87	2-484	0	-	0-1224
1974	1	79	2-442	0	-	0-1125
1975	7	529	212-1090	0	-	0-1109
1976	1	71	2-394	0	-	0-1064
1977	2	141	17-507	2	594	72-2146
1978	1	65	2-362	1	278	7-1549
1979	4	251	68-642	0	-	0-1044
1980	3	181	37-529	0	-	0-1034
1981	0	-	0-214	0	-	0-1002
1982	2	121	15-435	3	860	177-2514
1983	1	54	1-300	0	-	0-974
1984	2	105	13-379	4	1081	294-2766
1985	0	-	0-198	2	574	69-2074
1986	3	155	32-454	0	-	0-1053
1987	1	53	1-297	0	-	0-1104
1988	3	149	31-435	0	-	0-1060
1989	2	142	17-511	0	-	0-1572
Total	34	122	81-162	12	204	105-356

Ca. Peritoneum

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	0	-	0-29237	0	-	0-128012
1973	0	-	0-10013	0	-	0-45574
1974	0	-	0-9960	0	-	0-44684
1975	0	-	0-10692	0	-	0-54461
1976	0	-	0-13457	0	-	0-67211
1977	0	-	0-9629	0	-	0-45964
1978	0	-	0-8732	0	-	0-43180
1979	0	-	0-11031	0	-	0-56375
1980	0	-	0-9217	0	-	0-49311
1981	0	-	0-12871	0	-	0-69246
1982	0	-	0-12835	0	-	0-66820
1983	0	-	0-9957	0	-	0-58470
1984	2	6007	727-21685	0	-	0-64969
1985	0	-	0-9328	0	-	0-57903
1986	0	-	0-11819	0	-	0-74910
1987	0	-	0-12059	0	-	0-77482
1988	0	-	0-10762	0	-	0-70428
1989	0	-	0-15807	0	-	0-106081
Total	2	338	41-1222	0	-	0-3363

Ca. Lung

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	0	-	0-237	1	240	6-1335
1973	2	40	5-143	2	150	18-540
1974	8	142	61-279	0	-	0-252
1975	8	134	58-264	3	199	41-583
1976	8	123	53-241	4	250	68-640
1977	11	159	79-284	1	61	1-338
1978	6	82	30-179	4	236	64-604
1979	6	77	28-168	3	176	36-516
1980	4	49	13-126	1	57	1-320
1981	7	85	34-174	2	114	14-410
1982	7	82	33-168	3	169	35-493
1983	7	77	31-158	2	108	13-391
1984	8	85	37-168	2	112	14-404
1985	9	94	43-178	2	114	14-411
1986	6	63	23-137	2	117	14-424
1987	4	42	11-107	1	60	2-335
1988	6	60	22-130	0	-	0-220
1989	9	130	59-246	1	89	2-496
Total	116	85	70-101	34	121	80-161

Ca. Pleura

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1972	0	-	0-31267	1	39232	993-218519
1973	0	-	0-8232	0	-	0-37732
1974	1	2434	62-13560	0	-	0-42352
1975	1	1773	45-9877	0	-	0-29624
1976	0	-	0-5783	0	-	0-27892
1977	0	-	0-5818	0	-	0-28192
1978	3	4283	884-12522	0	-	0-26542
1979	3	5119	1056-14966	1	8739	221-48676
1980	1	1337	34-7445	1	7169	181-39933
1981	3	3821	788-11169	0	-	0-25292
1982	3	4138	854-12096	0	-	0-27857
1983	1	955	24-5320	0	-	0-21005
1984	1	889	22-4950	0	-	0-19805
1985	3	2491	514-7281	0	-	0-19513
1986	3	2190	452-6402	1	4574	116-25476
1987	0	-	0-2717	0	-	0-17883
1988	1	764	19-4258	0	-	0-19052
1989	0	-	0-2437	0	-	0-28815
Total	24	1638	1050-2437	4	1558	425-3989

TABLE A3.5 (cont.): Chatham Dockyard. Cause specific mortality for 12 disease groups by calendar year period, for study responders and non-responders.

	Circulatory System						Pulmonary Circulation					
				Non-responders						Non-responders		
Year	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
1972	2	33	4-121	4	257	70-657	0	-	0-5979	0	-	0-22685
1973	14	72	39-121	6	121	44-263	0	-	0-1855	0	-	0-7057
1974	13	61	32-104	4	74	20-189	0	-	0-1562	0	-	0-6099
1975	22	95	60-144	8	140	60-276	0	-	0-1515	0	-	0-5937
1976	21	85	52-130	13	218	116-373	1	362	9-2016	0	-	0-5562
1977	34	130	86-174	10	163	78-299	0	-	0-1326	0	-	0-5537
1978	32	112	73-151	8	120	52-236	0	-	0-1291	0	-	0-5427
1979	36	117	79-155	9	133	61-252	0	-	0-725	0	-	0-3232
1980	38	120	82-158	7	103	42-213	0	-	0-730	0	-	0-3310
1981	34	103	69-138	9	128	58-242	1	180	5-1002	0	-	0-2982
1982	28	82	54-118	14	194	106-326	0	-	0-667	0	-	0-3125
1983	28	77	51-112	17	226	131-362	1	177	4-985	0	-	0-3034
1984	27	71	47-104	10	134	65-247	0	-	0-1332	0	-	0-6392
1985	27	67	44- 98	6	79	29-172	1	332	8-1851	0	-	0-6217
1986	48	119	85-152	10	134	64-246	0	-	0-1225	0	-	0-6358
1987	39	96	66-127	13	176	94-300	0	-	0-1221	1	1721	44-9584
1988	33	75	49-101	10	126	61-232	0	-	0-1098	0	-	0-5834
1989	11	35	18- 63	5	93	30-216	0	-	0-1527	0	-	0-8505
Total	487	89	81- 97	163	142	120-163	4	66	18-170	1	76	2-422

	Respiratory System						Bronchitis, Emphysema and Asthma					
				Non-responders						Non-responders		
Year	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
1972	0	-	0-285	1	270	7-1502	0	-	0-507	1	471	12-2622
1973	1	25	1-137	3	262	54-767	1	45	1-251	0	-	0-583
1974	6	140	51-305	2	168	20-605	2	84	10-304	0	-	0-550
1975	5	106	34-248	2	158	19-570	2	78	9-283	2	288	35-1041
1976	9	164	75-311	2	140	17-504	2	76	9-273	0	-	0-530
1977	6	115	42-250	0	-	0-273	2	78	9-282	0	-	0-558
1978	6	103	38-223	7	458	184-943	3	106	22-311	0	-	0-508
1979	3	46	10-135	4	253	69-648	1	33	1-186	2	277	33-1000
1980	3	45	9-131	0	-	0-238	2	71	9-255	0	-	0-564
1981	4	58	16-148	0	-	0-229	0	-	0-135	0	-	0-585
1982	3	38	8-111	3	163	34-476	0	-	0-130	0	-	0-574
1983	9	108	49-204	2	101	12-366	3	108	22-314	2	320	39-1155
1984	4	57	16-146	4	266	72-680	1	37	1-205	2	350	42-1262
1985	9	108	50-206	1	59	1-330	3	103	21-300	1	171	4-955
1986	12	142	73-248	1	60	1-333	4	153	42-392	0	-	0-732
1987	11	136	68-244	1	62	2-346	4	178	49-457	1	235	6-1309
1988	10	105	51-194	4	209	57-535	4	138	37-352	1	181	5-1008
1989	6	86	32-188	2	151	18-544	1	48	1-266	1	264	7-1469
Total	107	93	75-110	39	147	101-193	35	77	51-102	13	123	65-210

	Asbestosis						Pulmonary Fibrosis					
				Non-responders						Non-responders		
Year	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
1972	0	-	0-199408	0	-	0-999999	0	-	0-19432	0	-	0-76421
1973	0	-	0-69098	0	-	0-311802	0	-	0-6223	0	-	0-22967
1974	0	-	0-78456	0	-	0-299069	0	-	0-5730	0	-	0-22891
1975	0	-	0-38476	0	-	0-170507	0	-	0-5041	0	-	0-19844
1976	0	-	0-65351	0	-	0-296432	0	-	0-4242	0	-	0-18206
1977	0	-	0-37440	0	-	0-165875	0	-	0-5088	0	-	0-21607
1978	0	-	0-34629	0	-	0-167570	0	-	0-3864	0	-	0-16247
1979	0	-	0-43049	0	-	0-217374	0	-	0-6761	0	-	0-31634
1980	0	-	0-37227	0	-	0-177923	0	-	0-6241	0	-	0-28995
1981	0	-	0-35522	0	-	0-181720	0	-	0-6471	0	-	0-28296
1982	0	-	0-30272	0	-	0-150944	0	-	0-5731	1	7431	188-41389
1983	0	-	0-29111	0	-	0-152066	0	-	0-5800	0	-	0-26584
1984	0	-	0-30018	0	-	0-167493	0	-	0-4297	0	-	0-20768
1985	0	-	0-29448	0	-	0-150740	1	951	24-5299	0	-	0-17732
1986	0	-	0-21379	0	-	0-130024	0	-	0-3321	0	-	0-17478
1987	0	-	0-23884	0	-	0-141161	0	-	0-3168	0	-	0-16429
1988	0	-	0-23598	0	-	0-147876	0	-	0-3028	0	-	0-16534
1989	0	-	0-34524	0	-	0-223842	0	-	0-4252	0	-	0-24291
Total	0	-	0-1992	0	-	0-10385	1	72	2-399	1	333	8-1856

TABLE A3.6: Portsmouth Dockyard. Cause specific mortality for 12 disease groups by calendar year period, for study responders and non-responders.

All Causes

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1973	19	36	21- 55	37	253	171-334
1974	48	69	49- 88	18	94	56-149
1975	71	94	72-116	21	104	64-159
1976	66	80	61- 99	22	102	64-155
1977	79	92	72-112	25	115	74-169
1978	98	105	84-126	21	87	54-133
1979	104	104	84-123	30	115	77-164
1980	95	90	72-109	19	71	43-111
1981	87	79	63- 96	26	95	62-139
1982	110	95	77-112	34	121	80-161
1983	103	84	68-100	38	127	86-167
1984	93	72	57- 86	46	148	105-191
1985	115	82	67- 97	29	89	59-128
1986	150	104	87-120	45	138	97-178
1987	145	99	83-115	38	120	82-158
1988	128	80	66- 94	33	93	62-125
1989	148	88	74-102	46	124	88-160
1990	33	110	72-143	4	62	17-159
Total	1692	88	83- 92	532	114	104-124

All Neoplasms

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1973	4	27	7- 68	20	477	291-737
1974	20	100	61-154	7	126	50-259
1975	20	93	56-143	8	136	59-268
1976	22	91	57-138	9	142	65-269
1977	22	86	54-130	3	46	9-134
1978	30	109	73-155	8	112	48-221
1979	26	87	57-128	6	79	29-171
1980	29	91	61-131	7	88	35-181
1981	43	128	90-166	8	97	42-191
1982	47	133	95-171	10	118	57-217
1983	41	108	75-141	13	144	77-246
1984	28	68	45- 98	17	178	104-285
1985	44	102	72-132	3	31	6- 90
1986	48	107	77-137	15	153	85-252
1987	46	100	71-128	12	123	64-215
1988	45	92	65-119	7	68	27-140
1989	50	99	71-126	20	191	116-294
1990	10	112	54-206	0	-	0-204
Total	575	98	90-106	173	125	106-143

Ca. Stomach

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1973	0	-	0-253	5	1201	389-2803
1974	2	105	13-380	1	55	5-1015
1975	2	98	12-354	1	57	4-983
1976	0	-	0-167	0	-	0-620
1977	1	44	1-248	0	-	0-634
1978	1	41	1-227	1	155	4-863
1979	2	79	10-284	2	298	36-1074
1980	3	112	23-327	0	-	0-534
1981	7	248	99-510	1	142	4-789
1982	7	256	103-526	0	-	0-551
1983	5	161	52-376	1	134	3-746
1984	1	31	1-174	0	-	0-487
1985	1	32	1-176	0	-	0-509
1986	3	90	18-262	2	270	33-974
1987	2	61	7-220	2	286	35-1032
1988	3	84	17-245	0	-	0-483
1989	4	107	29-275	1	128	3-714
1990	1	152	4-847	0	-	0-2749
Total	45	95	68-123	17	149	86-238

Ca. Peritoneum

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1973	0	-	0-7523	0	-	0-30675
1974	0	-	0-5725	0	-	0-24265
1975	0	-	0-6425	0	-	0-28955
1976	0	-	0-7901	0	-	0-35516
1977	0	-	0-5860	0	-	0-24410
1978	0	-	0-5174	0	-	0-22759
1979	0	-	0-6613	0	-	0-28128
1980	0	-	0-5407	0	-	0-24179
1981	0	-	0-7606	0	-	0-34388
1982	0	-	0-7548	0	-	0-32094
1983	0	-	0-5684	1	7380	187-41108
1984	0	-	0-6414	0	-	0-30062
1985	0	-	0-5411	0	-	0-26612
1986	1	1772	45-9873	0	-	0-33633
1987	0	-	0-6604	0	-	0-35394
1988	0	-	0-6025	0	-	0-31520
1989	0	-	0-5957	0	-	0-31271
1990	0	-	0-34211	0	-	0-183403
Total	1	99	2-551	1	457	12-2544

Ca. Lung

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1973	1	16	0- 87	7	382	153-786
1974	8	93	40-183	1	41	1-227
1975	6	65	24-142	4	156	42-399
1976	10	98	47-179	2	73	9-262
1977	10	91	44-168	3	105	22-306
1978	14	120	66-202	4	131	36-334
1979	14	112	61-188	2	61	7-222
1980	11	83	41-148	1	30	1-165
1981	16	117	67-190	4	118	32-303
1982	15	105	59-173	9	261	119-495
1983	14	91	50-152	7	191	77-394
1984	11	69	34-123	11	299	149-535
1985	24	145	93-216	2	54	7-196
1986	21	125	78-192	7	193	77-397
1987	13	77	41-131	4	113	31-290
1988	11	61	30-109	3	80	17-235
1989	17	91	53-146	6	160	59-349
1990	2	61	7-221	0	-	0-577
Total	218	94	81-106	77	139	108-170

Ca. Pleura

Year	Obs	SMR	95% CI	Non-responders Obs	SMR	95% CI
1973	0	-	0-6016	1	6705	170-37344
1974	1	1454	37-8097	1	6219	157-34637
1975	2	2171	263-7837	0	-	0-16464
1976	2	1848	224-6671	1	4015	102-22365
1977	1	946	24-5267	0	-	0-15167
1978	0	-	0-3107	0	-	0-13467
1979	0	-	0-3707	0	-	0-16136
1980	0	-	0-2825	0	-	0-12866
1981	6	4351	1595-9471	0	-	0-12326
1982	4	3138	855-8034	0	-	0-13251
1983	3	1577	325-4611	0	-	0-9649
1984	3	1470	303-4296	1	2471	62-13762
1985	0	-	0-1679	0	-	0-8760
1986	2	807	98-2915	0	-	0-7569
1987	1	400	10-2226	1	2158	55-12020
1988	1	415	10-2310	0	-	0-8265
1989	1	414	10-2308	0	-	0-8401
1990	1	2397	61-13353	0	-	0-49489
Total	28	1042	693-1506	5	907	294-2117

TABLE A3.6 (cont.): Portsmouth Dockyard. Cause specific mortality for 12 disease groups by calendar year period, for study responders and non-responders.

	Circulatory System						Pulmonary Circulation					
				Non-responders						Non-responders		
Year	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
1973	11	44	22- 79	10	145	69-266	0	-	0-1467	0	-	0-5142
1974	21	64	39- 97	9	98	45-186	0	-	0-1022	0	-	0-3631
1975	35	97	65-129	7	72	29-148	0	-	0-994	0	-	0-3515
1976	29	74	50-106	7	68	27-140	0	-	0-845	0	-	0-3219
1977	46	111	79-143	18	169	100-268	0	-	0-844	0	-	0-3258
1978	55	121	89-153	12	101	52-176	0	-	0-818	0	-	0-3076
1979	59	120	89-151	13	101	54-172	0	-	0-459	0	-	0-1712
1980	49	95	69-122	10	76	37-140	1	125	3-695	0	-	0-1728
1981	34	63	42- 85	12	89	46-156	0	-	0-414	1	429	11-2388
1982	40	71	49- 93	19	139	84-217	0	-	0-406	0	-	0-1661
1983	45	75	53- 97	17	117	68-187	1	109	3-606	0	-	0-1593
1984	42	67	47- 87	22	146	91-220	1	226	6-1256	0	-	0-3196
1985	57	85	63-107	12	76	39-133	1	202	5-1125	0	-	0-3034
1986	70	101	78-125	22	141	88-213	0	-	0-731	0	-	0-3094
1987	72	103	79-127	15	99	55-163	0	-	0-723	0	-	0-3163
1988	62	81	61-101	16	94	54-153	0	-	0-640	0	-	0-2759
1989	80	99	77-120	19	107	64-167	0	-	0-597	0	-	0-2591
1990	13	90	48-154	0	-	0-120	0	-	0-3326	0	-	0-14843
Total	820	88	82- 94	240	106	93-120	4	40	11-104	1	40	1-221

	Respiratory System						Bronchitis, Emphysema and Asthma					
				Non-responders						Non-responders		
Year	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
1973	3	60	12-177	7	465	186-957	0	-	0-137	4	481	131-1231
1974	7	113	45-233	1	52	1-291	0	-	0-108	0	-	0-343
1975	14	202	110-339	6	291	107-633	3	80	16-234	2	177	21-638
1976	14	171	93-287	4	171	47-438	3	76	16-222	0	-	0-324
1977	10	128	61-235	4	185	50-475	4	104	28-266	2	187	23-674
1978	12	138	71-241	0	-	0-145	0	-	0- 87	0	-	0-301
1979	7	73	29-150	2	69	8-251	4	89	24-228	1	75	2-420
1980	5	49	16-115	0	-	0-127	2	46	6-166	0	-	0-301
1981	4	38	10- 97	3	102	21-299	1	23	1-131	0	-	0-318
1982	13	106	57-182	5	154	50-359	3	67	14-197	1	86	2-481
1983	8	62	27-122	4	113	31-288	2	45	5-163	1	86	2-479
1984	7	64	26-131	3	101	21-296	2	46	6-167	1	88	2-491
1985	10	75	36-138	7	206	83-424	2	42	5-151	2	169	20-608
1986	16	116	66-188	6	177	65-386	5	114	37-267	2	193	23-697
1987	9	67	31-127	4	128	35-328	4	105	29-269	2	234	28-846
1988	10	63	30-115	3	78	16-227	3	60	12-176	1	87	2-482
1989	8	46	20- 91	2	47	6-169	0	-	0- 69	0	-	0-298
1990	3	95	20-278	2	266	32-960	1	104	3-580	0	-	0-1706
Total	160	86	73- 99	63	127	95-158	39	54	37-71	19	98	59-153

	Asbestosis						Pulmonary Fibrosis					
				Non-responders						Non-responders		
Year	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
1973	0	-	0-50171	0	-	0-202636	0	-	0-4989	0	-	0-16980
1974	0	-	0-50293	0	-	0-178483	0	-	0-3715	0	-	0-13484
1975	0	-	0-23483	0	-	0-94893	0	-	0-3306	0	-	0-11765
1976	0	-	0-39009	0	-	0-162672	0	-	0-2634	0	-	0-10334
1977	0	-	0-23420	0	-	0-91531	0	-	0-3209	0	-	0-12983
1978	0	-	0-20736	0	-	0-84808	0	-	0-2452	0	-	0-9172
1979	0	-	0-24919	0	-	0-109651	0	-	0-4173	0	-	0-16433
1980	0	-	0-22150	0	-	0-89669	0	-	0-3877	0	-	0-15140
1981	0	-	0-20218	0	-	0-90156	0	-	0-4093	0	-	0-15202
1982	1	4811	122-26800	0	-	0-76255	1	936	24-5215	0	-	0-14311
1983	1	4559	115-25394	0	-	0-73276	0	-	0-3585	0	-	0-14155
1984	1	4636	117-25822	0	-	0-78261	0	-	0-2671	0	-	0-10379
1985	0	-	0-17296	0	-	0-73255	0	-	0-2127	0	-	0-8683
1986	0	-	0-11632	0	-	0-58809	0	-	0-1958	1	2282	58-12711
1987	0	-	0-13293	0	-	0-65202	0	-	0-1861	0	-	0-8044
1988	0	-	0-12798	0	-	0-64637	0	-	0-1737	0	-	0-7656
1989	0	-	0-12610	0	-	0-66503	0	-	0-1637	0	-	0-7376
1990	0	-	0-72623	0	-	0-392440	0	-	0-9192	0	-	0-42521
Total	3	905	187-2646	0	-	0-5003	1	43	1-237	1	172	4-956

TABLE A3.7: Cause specific mortality by age at death for the 3 dockyards.

Causes of Death	Age at death (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	<35	55	145	107-184	23	131	83-197	26	117	76-171
	35-	85	120	95-146	30	110	74-158	51	120	87-153
	45-	265	110	97-123	85	89	70-108	157	96	81-111
	55-	785	104	97-111	305	95	84-106	554	93	86-101
	65-	867	86	80- 91	466	87	79- 95	725	82	76- 88
	75+	232	92	80-104	136	86	71-100	179	78	67- 90
All Neoplasms	<35	15	251	141-414	4	141	38-362	4	111	30-285
	35-	25	159	103-235	13	206	110-353	14	143	78-240
	45-	82	127	99-154	31	130	84-175	48	105	75-135
	55-	280	122	108-136	110	109	89-130	199	106	91-121
	65-	308	103	92-115	162	97	82-112	262	94	83-105
	75+	79	126	98-154	45	109	77-140	48	79	56-101
Ca. Stomach	<35	1	714	18-3979	0	-	0-5271	0	-	0-4100
	35-	0	-	0-373	0	-	0-922	1	159	4-884
	45-	8	163	70-321	4	212	58-542	1	28	1-155
	55-	30	160	109-230	12	140	72-245	15	96	54-159
	65-	34	130	86-174	16	116	66-188	25	111	72-164
	75+	8	153	66-301	2	61	7-222	3	63	13-185
Ca. Peritoneum	<35	1	2500	63-13925	1	5000	126-27850	0	-	0-18450
	35-	0	-	0-3690	0	-	0-9225	0	-	0-7380
	45-	2	800	97-2888	0	-	0-4100	0	-	0-2171
	55-	6	1200	440-2612	0	-	0-1757	1	263	7-1466
	65-	1	244	6-1359	1	500	13-2785	0	-	0-1085
	75+	0	-	0-9225	0	-	0-12300	0	-	0-9225
Ca. Lung	<35	3	857	177-2506	1	588	15-3276	2	909	110-3282
	35-	5	177	57-412	1	85	2-472	1	53	1-296
	45-	22	104	65-158	6	71	26-155	14	88	48-147
	55-	90	102	81-123	38	90	61-119	80	102	80-125
	65-	97	88	70-105	58	83	62-105	104	90	73-107
	75+	24	123	79-183	12	83	43-145	17	80	46-128
Ca. Pleura	<35	0	-	0-18450	0	-	0-73800	0	-	0-18450
	35-	2	1538	186-5554	1	1667	42-9283	2	2500	303-9025
	45-	11	2245	1120-4016	3	1765	364-5159	4	1176	321-3012
	55-	29	2164	1449-3108	10	1818	873-3344	11	1028	513-1839
	65-	20	1709	1044-2640	8	1379	595-2717	10	971	466-1785
	75+	4	2500	681-6400	2	2000	242-7220	1	667	17-3713

TABLE A3.7 (cont.): Cause specific mortality by age at death for the 3 dockyards.

Causes of Death	Age at death (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	<35	17	363	212-582	7	345	138-710	10	385	185-707
	35-	34	129	85-172	9	95	43-180	21	139	86-213
	45-	151	123	103-142	41	98	68-128	78	98	76-120
	55-	409	107	96-117	150	96	81-112	258	89	78-100
	65-	432	80	73- 88	221	84	73- 95	352	81	72- 89
	75+	109	82	67- 98	59	77	57- 96	101	91	73-109
Pulmonary Circulation	<35	0	-	0-6150	0	-	0-9225	1	2500	63-13925
	35-	0	-	0-1604	0	-	0-4613	0	-	0-2636
	45-	4	412	112-1056	0	-	0-1025	0	-	0-551
	55-	17	425	248-681	1	57	1-315	0	-	0-113
	65-	7	124	50-256	3	98	20-288	3	63	13-184
	75+	0	-	0-2171	0	-	0-499	0	-	0-365
Respiratory System	<35	12	764	395-1335	5	625	203-1459	4	396	108-1014
	35-	13	469	250-803	1	86	2-480	6	333	122-726
	45-	16	136	78-220	6	127	47-277	17	193	112-308
	55-	61	110	82-138	31	115	74-156	58	120	89-151
	65-	65	65	49-81	45	74	53- 96	62	64	48- 80
	75+	25	84	54-124	19	89	53-139	13	44	23- 75
Bronchitis, Emphysema and Asthma	<35	1	250	6-1393	0	-	0-1757	0	-	0-1367
	35-	1	120	3-671	0	-	0-997	0	-	0-647
	45-	5	102	33-238	2	97	12-351	4	105	29-269
	55-	15	59	33- 98	13	101	54-172	16	71	40-115
	65-	19	53	32- 83	16	67	38-108	16	43	25- 70
	75+	4	52	14-133	4	66	18-169	3	36	7-106
Asbestosis	<35	1	13889	351-77361	0	-	0-369000	1	21277	538-118511
	35-	0	-	0-123000	0	-	0-184500	1	25000	632-139250
	45-	0	-	0-9225	0	-	0-36900	0	-	0-2838
	55-	2	1250	151-4512	0	-	0-5271	0	-	0-12300
	65-	3	1765	364-5158	0	-	0-4613	1	667	17-3713
	75+	1	3333	84-18567	0	-	0-18450	0	-	0-12300
Pulmonary Fibrosis	<35	0	-	0-36900	0	-	0-73800	0	-	0-56800
	35-	0	-	0-6150	0	-	0-18450	0	-	0-12300
	45-	0	-	0-1677	0	-	0-5271	0	-	0-2460
	55-	1	103	3-574	0	-	0-900	1	139	4-774
	65-	1	78	2-435	0	-	0-568	0	-	0-345
	75+	0	-	0-946	1	435	11-2422	0	-	0-1054

TABLE A3.8: Non-responder cause specific mortality by age at death for the 3 dockyards.

Causes of Death	Age at death (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	<35	18	726	430-1147	13	425	226-726	9	192	88-365
	35-	11	365	182-654	15	534	299-880	18	240	142-381
	45-	30	312	211-446	15	145	81-239	42	163	113-212
	55-	78	188	146-229	74	137	106-168	153	139	117-161
	65-	118	124	101-147	153	126	106-146	230	100	87-113
	75+	39	105	72-138	50	109	79-139	80	90	70-110
All Neoplasms	<35	5	1282	415-2992	0	-	0-785	2	263	32-950
	35-	2	289	36-1078	4	625	170-1600	5	291	94-678
	45-	7	272	109-561	4	139	38-356	15	207	116-342
	55-	22	176	110-266	22	131	82-198	53	153	112-194
	65-	29	107	71-154	45	121	86-157	74	104	80-128
	75+	10	110	53-202	16	136	78-220	24	103	66-153
Ca. Stomach	<35	0	-	0-36900	0	-	0-73800	0	-	0-36900
	35-	0	-	0-9225	0	-	0-7380	0	-	0-3356
	45-	1	500	13-2785	0	-	0-1604	0	-	0-647
	55-	2	189	23-681	0	-	0-251	6	204	75-444
	65-	3	119	24-347	8	249	107-491	7	117	47-241
	75+	1	132	3-733	4	430	117-1101	4	220	60-562
Ca. Peritoneum	<35	0	-	0-141923	0	-	0-73800	0	-	0-92250
	35-	0	-	0-55074	0	-	0-46125	1	10000	253-55700
	45-	0	-	0-36900	0	-	0-36900	0	-	0-12300
	55-	1	3333	84-18567	0	-	0-12300	0	-	0-5271
	65-	0	-	0-9225	0	-	0-7380	0	-	0-4100
	75+	0	-	0-36900	0	-	0-36900	0	-	0-36900
Ca. Lung	<35	1	1000	253-55700	0	-	0-12300	0	-	0-9225
	35-	1	833	21-4642	2	1667	201-6017	2	606	73-2188
	45-	2	233	28-839	1	96	2-536	8	316	136-623
	55-	9	182	84-346	10	140	67-258	24	166	106-247
	65-	9	88	40-166	14	89	48-149	37	124	84-164
	75+	5	178	58-415	7	170	68-351	6	74	27-162
Ca. Pleura	<35	0	-	0-205000	0	-	0-52714	0	-	0-92250
	35-	0	-	0-76875	0	-	0-41000	0	-	0-36900
	45-	1	5000	0-27850	0	-	0-18450	0	-	0-7380
	55-	2	2857	346-10314	2	2500	303-9025	4	2222	606-5689
	65-	3	3333	688-9744	2	1818	220-6564	0	-	0-1537
	75+	0	-	0-18450	0	-	0-18450	1	1667	42-9283

TABLE A3.8 (cont.): Non-responder cause specific mortality by age at death for the 3 dockyards.

Causes of Death	Age at death (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	<35	8	2759	1190-5434	5	1515	491-3536	3	566	117-1655
	35-	7	506	202-1037	7	722	290-1487	9	345	158-654
	45-	19	386	232-603	5	98	32-230	18	142	84-225
	55-	40	189	131-248	40	152	105-199	68	127	97-157
	65-	70	138	105-170	78	130	101-158	108	95	77-113
	75+	19	98	58-152	28	126	84-182	34	79	52-105
Pulmonary Circulation	<35	0	-	0-94615	0	-	0-41000	0	-	0-92250
	35-	0	-	0-36900	0	-	0-36900	0	-	0-18450
	45-	1	2500	63-13925	0	-	0-9225	0	-	0-3355
	55-	3	1304	269-3813	0	-	0-1230	0	-	0-605
	65-	1	169	4-944	0	-	0-505	1	74	2-410
	75+	0	-	0-2050	1	435	11-2422	0	-	0-900
Respiratory System	<35	2	2222	269-8022	3	2308	476-6746	4	1818	495-4655
	35-	1	909	23-5064	2	1667	202-6017	0	-	0-1153
	45-	2	408	49-1473	4	690	188-1765	4	284	77-726
	55-	9	278	127-527	8	170	73-335	19	206	124-321
	65-	16	156	89-253	18	124	73-196	27	101	67-147
	75+	2	44	5-158	4	62	17-158	9	75	34-143
Bronchitis, Emphysema and Asthma	<35	1	5000	127-27850	0	-	0-9225	0	-	0-6150
	35-	0	-	0-12300	1	2500	63-13925	0	-	0-3690
	45-	0	-	0-1845	1	385	10-2142	2	328	40-1184
	55-	2	128	15-463	3	128	26-375	8	180	78-355
	65-	5	126	41-294	7	115	46-238	7	65	26-134
	75+	0	-	0-313	1	54	138-303	2	59	7-214
Asbestosis	<35	0	-	0-263571	0	-	0-82000	0	-	0-108529
	35-	0	-	0-105429	0	-	0-49200	0	-	0-80217
	45-	0	-	0-61500	0	-	0-4146	0	-	0-73800
	55-	0	-	0-58571	0	-	0-36900	0	-	0-18450
	65-	0	-	0-18450	0	-	0-18450	0	-	0-9225
	75+	0	-	0-36900	0	-	0-52714	0	-	0-36900
Pulmonary Fibrosis	<35	0	-	0-57656	0	-	0-147600	0	-	0-82000
	35-	0	-	0-92250	0	-	0-82000	0	-	0-73800
	45-	0	-	0-36900	0	-	0-36900	0	-	0-12300
	55-	1	1667	42-9283	1	1429	36-7957	0	-	0-2636
	65-	0	-	0-2838	0	-	0-2460	1	357	9-1989
	75+	0	-	0-6150	0	-	0-6150	0	-	0-2838

TABLE A3.9: Cause specific mortality by dockyard for questionnaire type (Free or Controlled).

Causes of Death	Ques. type	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	Free*	1634	94	89- 98	663	89	82- 96	941	79	74- 84
	Both	496	101	92-137	180	92	79-106	459	100	91-109
	Neither	159	119	100-137	202	100	86-113	292	101	89-113
All Neoplasms	Free	540	109	100-118	241	109	95-122	319	89	79- 99
	Both	188	132	113-151	69	116	88-143	171	121	103-139
	Neither	61	161	121-201	55	92	68-116	85	98	77-119
Ca. Stomach	Free	62	152	114-189	21	116	72-177	23	80	51-120
	Both	16	133	76-217	9	183	84-347	17	148	86-237
	Neither	3	96	20-282	4	82	22-209	5	72	23-168
Ca. Peritoneum	Free	5	489	158-1140	2	511	62-1844	0	-	0-586
	Both	4	1506	410-3855	0	-	0-3854	1	-	11-2491
	Neither	1	1267	32-7055	0	-	0-3562	0	-	0-2351
Ca. Lung	Free	154	87	72-101	80	91	71-111	121	86	70-101
	Both	69	132	101-163	16	66	38-107	67	117	89-145
	Neither	18	134	79-211	20	84	52-131	30	88	60-126
Ca. Pleura	Free	37	1495	1013-1976	14	1447	791-2428	12	719	371-1256
	Both	25	3722	2435-5553	7	2835	1138-5840	14	2298	1255-3855
	Neither	4	2122	578-5432	3	1194	246-3491	2	490	59-1770
Circulatory System	Free	850	96	89-102	309	86	77- 96	458	80	73-88
	Both	231	91	79-102	95	86	67-195	216	96	84-109
	Neither	71	105	81-129	96	99	80-119	146	106	89-123
Pulmonary Circulation	Free	19	216	130-337	4	102	28-262	1	17	0-92
	Both	8	310	134-611	0	-	0-346	1	41	1-231
	Neither	1	149	4-829	0	-	0-350	2	137	17-496
Respiratory System	Free	132	90	74-105	63	84	63-105	88	77	61- 94
	Both	43	100	70-130	17	85	50-137	43	95	67-123
	Neither	17	151	88-242	27	131	86-190	29	106	71-153
Bronchitis, Emphysema and Asthma	Free	31	57	37- 77	20	68	41-105	24	54	35- 81
	Both	10	62	30-114	7	88	35-181	11	62	31-110
	Neither	4	97	26-247	8	99	43-194	4	38	10- 97
Asbestosis	Free	4	1342	366-3435	0	-	0-3068	1	495	13-2758
	Both	3	3457	713-10107	0	-	0-11560	0	-	0-4581
	Neither	0	-	0-16295	0	-	0-11172	2	4091	495-14768
Pulmonary Fibrosis	Free	0	-	0-172	1	111	3-616	0	-	0-258
	Both	2	315	38-1139	0	-	0-1500	0	-	0-638
	Neither	0	-	0-2239	0	-	0-1494	1	290	7-1618

* Free = The free questionnaire
Both = Both the free and controlled questionnaires.

TABLE A3.10: Cause specific mortality by dockyard for x-ray type (Small or Large).

Causes of Death	X-ray type	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	Small*	1334	89	84- 93	667	88	81- 94	859	78	73- 83
	Both	622	107	99-115	276	102	87-114	601	102	93-110
	Neither	323	120	107-133	102	94	75-112	232	102	84-108
All Neoplasms	Small	445	103	93-112	228	101	88-114	280	84	74- 94
	Both	230	137	119-154	97	117	94-141	218	120	104-136
	Neither	114	152	124-179	40	123	85-162	77	107	83-131
Ca. Stomach	Small	50	141	102-180	24	130	84-194	16	60	35- 98
	Both	23	162	103-244	9	131	60-249	21	142	88-218
	Neither	8	124	54-245	1	37	1-206	8	135	58-266
Ca. Peritoneum	Small	5	548	178-1279	2	492	60-1776	0	-	0-615
	Both	5	1590	515-3710	0	-	0-2782	1	339	9-1886
	Neither	0	-	0-2612	0	-	0-7142	0	-	0-3219
Ca. Lung	Small	127	83	68- 97	70	79	60- 97	101	78	62- 93
	Both	80	130	101-158	32	95	62-128	86	117	92-142
	Neither	34	125	83-167	14	107	59-180	31	108	70-146
Ca. Pleura	Small	33	1487	979-1994	16	1596	913-2591	10	628	301-1154
	Both	26	3326	2172-4873	7	2069	831-4262	16	1997	1142-3243
	Neither	7	2142	860-4412	1	804	20-4477	2	684	83-2468
Circulatory System	Small	704	91	85- 98	313	86	76- 95	430	82	74- 89
	Both	293	97	86-108	126	95	78-112	289	97	86-108
	Neither	155	113	95-130	48	91	65-117	110	94	77-112
Pulmonary Circulation	Small	17	224	131-359	0	-	0- 93	2	36	4-131
	Both	7	229	92-473	1	68	2-377	2	65	8-234
	Neither	4	280	76-717	3	503	104-1472	0	-	0-286
Respiratory System	Small	102	81	65- 97	64	84	64-105	71	69	53- 85
	Both	57	112	82-141	35	125	84-167	61	106	79-132
	Neither	33	25	87-177	8	68	30-135	28	110	73-160
Bronchitis, Emphysema and Asthma	Small	21	45	28- 69	18	61	36- 96	12	30	16- 53
	Both	13	68	36-116	15	134	75-221	16	70	40-114
	Neither	11	119	59-213	2	43	5-156	11	112	56-200
Asbestosis	Small	2	762	92-27511	0	-	0-3006	3	1588	328-4642
	Both	4	3918	1068-10030	0	-	0-8112	0	-	0-3553
	Neither	1	2326	59-12953	0	-	0-21723	0	-	0-9554
Pulmonary Fibrosis	Small	1	54	1-300	0	-	0-403	1	76	2-425
	Both	1	133	3-743	1	291	0-1623	0	-	0-499
	Neither	0	-	0-1071	0	-	0-2696	0	-	0-1225

* Small = The small 100mm radiograph
Both = Both the small and large radiographs.

TABLE A3.11: Cause specific mortality by age at start of employment and dockyard.

Causes of Death	Age at start (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	<25	939	95	89-101	399	90	81- 99	611	82	75- 88
	25-	442	96	87-105	195	92	80-105	299	92	81-102
	35-	332	88	79- 98	106	103	83-122	237	88	77- 99
	45-	285	97	86-108	73	81	62- 99	145	88	74-103
	55+	85	104	82-126	47	68	49- 88	39	79	54-103
	Unknown	47	141	100-181	23	98	62-148	69	80	61- 99
All Neoplasms	<25	337	120	107-132	156	117	99-135	235	104	90-117
	25-	150	114	96-132	75	119	92-146	95	95	76-114
	35-	113	105	85-124	33	106	70-142	76	93	72-114
	45-	83	98	77-119	23	83	53-125	49	98	71-126
	55+	33	146	96-196	14	73	40-123	14	95	52-159
	Unknown	12	125	65-219	9	129	59-245	21	80	50-123
Ca. Stomach	<25	35	156	105-208	16	151	86-245	23	129	82-193
	25-	17	153	89-245	7	132	53-272	9	111	51-210
	35-	13	141	75-242	3	115	24-336	4	59	16-152
	45-	11	149	75-267	1	44	1-243	3	73	15-212
	55+	1	48	1-269	2	120	15-433	1	80	2-444
	Unknown	1	123	3-688	1	174	4-968	0	-	0-173
Ca. Peritoneum	<25	9	1370	627-2601	2	758	92-2735	1	228	6-1271
	25-	0	-	0-1468	0	-	0-3763	0	-	0-2275
	35-	0	-	0-1933	0	-	0-7367	0	-	0-3027
	45-	0	-	0-2678	0	-	0-9180	0	-	0-5234
	55+	0	-	0-10984	0	-	0-15834	0	-	0-19230
	Unknown	0	-	0-19853	0	-	0-31750	0	-	0-8939
Ca. Lung	<25	93	95	75-114	44	85	60-110	78	88	69-108
	25-	36	75	51-100	22	86	54-130	35	87	58-116
	35-	52	131	95-166	12	94	49-165	36	109	73-144
	45-	26	83	54-121	10	88	42-162	23	113	71-169
	55+	12	144	74-251	5	65	21-153	7	116	46-239
	Unknown	4	114	31-293	3	108	22-315	9	86	39-163
Ca. Pleura	<25	40	2527	1744-3310	17	2570	1497-4115	23	1969	1248-2954
	25-	14	2244	1226-3765	3	1243	256-3633	2	456	55-1645
	35-	3	622	128-1818	1	778	20-4333	1	306	8-1702
	45-	4	1193	325-3054	0	-	0-3514	0	-	0-1972
	55+	0	-	0-5161	0	-	0-7515	0	-	0-7914
	Unknown	1	2280	58-12701	0	-	0-12895	0	-	0-3344

TABLE A3.11 (cont.): Cause specific mortality by age at start of employment and dockyard.

Causes of Death	Age at start (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	<25	467	95	86-104	181	86	74- 99	280	79	69- 88
	25-	230	97	84-109	86	83	66-101	147	92	77-107
	35-	163	83	70- 96	49	97	70-124	124	94	77-110
	45-	150	97	82-113	39	88	60-116	66	82	62-102
	55+	44	102	72-132	24	71	45-106	18	74	44-117
	Unknown	27	158	104-230	12	107	55-186	39	93	64-122
Pulmonary Circulation	<25	10	212	102-390	2	91	11-327	1	27	1-153
	25-	4	166	45-426	1	86	2-478	0	-	0-217
	35-	6	299	110-650	1	177	4-987	1	69	2-383
	45-	6	371	136-807	0	-	0-745	0	-	0-411
	55+	0	-	0-779	0	-	0-905	0	-	0-1310
	Unknown	1	572	14-3184	0	-	0-2938	0	-	0-812
Respiratory System	<25	74	99	76-121	31	78	50-105	49	74	53- 95
	25-	37	91	62-121	21	92	57-141	31	96	62-130
	35-	29	84	56-121	14	130	71-218	26	91	60-134
	45-	27	95	63-138	8	84	36-165	16	91	52-147
	55+	5	56	18-131	5	53	17-124	6	103	38-224
	Unknown	3	103	21-300	1	41	1-229	3	34	7-100
Bronchitis, Emphysema and Asthma	<25	19	68	41-106	10	63	30-116	14	55	30- 91
	25-	6	40	15- 87	8	88	38-173	5	40	13- 93
	35-	7	55	22-113	5	116	38-271	9	81	37-154
	45-	8	75	33-149	3	79	16-231	4	58	16-149
	55+	1	31	1-170	1	29	1-160	3	133	27-387
	Unknown	0	-	0-332	0	-	0-383	0	-	0-108
Asbestosis	<25	4	2303	628-5897	0	-	0-4896	0	-	0-2809
	25-	1	1253	32-6982	0	-	0-11024	0	-	0-6512
	35-	1	1540	39-8576	0	-	0-21523	0	-	0-8150
	45-	1	2033	51-11326	0	-	0-24138	0	-	0-13528
	55+	0	-	0-31711	0	-	0-44173	1	13471	341-75035
	Unknown	0	-	0-65001	0	-	0-99987	0	-	0-25403
Pulmonary Fibrosis	<25	1	86	2-481	0	-	0-716	0	-	0-425
	25-	0	-	0-632	0	-	0-1374	0	-	0-905
	35-	1	204	5-1139	0	-	0-2846	0	-	0-1072
	45-	0	-	0-928	1	850	22-4737	0	-	0-1736
	55+	0	-	0-3238	0	-	0-4108	0	-	0-5580
	Unknown	0	-	0-8618	0	-	0-12872	0	-	0-3430

TABLE A3.12: Cause specific mortality by year of start of employment and by dockyard.

Causes of Death	Year of start	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	Pre 1930	149	86	72- 99	74	91	70-111	97	86	69-103
	1930-	584	90	83- 97	337	98	88-109	392	81	73- 89
	1940-	434	102	92-111	146	80	67- 93	336	85	76- 94
	1950-	433	89	81- 98	108	89	72-105	247	82	72- 92
	1960+	483	103	94-112	155	82	69- 95	259	98	86-110
	Unknown	47	141	100-181	23	98	62-148	69	80	61- 99
All Neoplasms	Pre 1930	56	116	86-146	26	109	71-159	44	133	94-172
	1930-	213	113	97-128	129	123	102-144	148	99	83-115
	1940-	152	123	103-142	59	106	79-133	101	83	67- 99
	1950-	143	104	87-121	40	112	77-147	83	92	72-111
	1960+	152	117	99-136	47	87	62-112	93	119	95-143
	Unknown	12	125	65-219	9	129	59-245	21	80	50-123
Ca. Stomach	Pre 1930	6	136	50-296	6	288	106-627	2	71	9-255
	1930-	25	155	101-229	11	126	63-225	18	148	88-234
	1940-	21	209	129-320	7	155	62-320	11	113	56-202
	1950-	11	98	49-175	2	70	8-253	4	55	15-141
	1960+	14	135	74-227	3	70	14-204	5	82	27-192
	Unknown	1	123	3-688	1	174	4-968	0	-	0-173
Ca. Peritoneum	Pre 1930	2	2789	338-10070	1	3206	81-17857	0	-	0-8778
	1930-	1	310	8-1728	0	-	0-2331	1	458	12-2549
	1940-	4	1518	414-3887	0	-	0-3511	0	-	0-1692
	1950-	2	646	78-2333	0	-	0-4833	0	-	0-2101
	1960-	0	-	0-1216	1	955	24-5319	0	-	0-2338
	Unknown	0	-	0-19853	0	-	0-31750	0	-	0-8939
Ca. Lung	Pre 1930	14	79	43-132	8	81	35-160	15	111	62-184
	1930-	61	86	65-108	39	90	62-118	60	98	73-123
	1940-	43	96	68-125	19	85	51-133	32	66	43- 89
	1950-	45	93	66-121	14	104	57-174	31	89	57-120
	1960+	56	126	93-159	13	63	34-109	41	138	96-180
	Unknown	4	114	31-293	3	108	22-315	9	86	39-163
Ca. Pleura	Pre 1930	8	5118	2207-10082	1	1424	36-7933	5	5072	1643-11838
	1930-	23	2791	1769-4187	14	3408	1862-5719	11	1821	909-3259
	1940-	14	2006	1096-3367	4	1388	378-3554	4	642	175-1644
	1950-	11	1467	732-2625	1	549	14-3055	5	1091	353-2547
	1960+	5	748	242-1747	1	427	11-2378	1	260	7-1448
	Unknown	1	2280	58-12701	0	-	0-12895	0	-	0-3344

TABLE A3.12 (cont.): Cause specific mortality by year of start of employment and by dockyard.

Causes of Death	Year of start	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	Pre 1930	73	80	61- 98	37	92	62-122	44	79	56-103
	1930-	308	91	81-101	157	93	79-108	186	79	67- 90
	1940-	224	101	88-115	58	65	48- 82	169	87	74-100
	1950-	222	90	78-102	51	88	64-112	123	84	70- 99
	1960+	227	101	88-114	76	88	68-108	113	93	76-110
	Unknown	27	158	104-230	12	107	55-186	39	93	64-122
Pulmonary Circulation	Pre 1930	3	299	62-875	2	417	50-1504	1	153	4-854
	1930-	6	173	63-376	1	53	1-296	0	-	0-144
	1940-	3	141	29-413	0	-	0-398	1	50	1-279
	1950-	8	335	145-660	1	166	4-923	0	-	0-246
	1960-	6	269	99-585	0	-	0-389	0	-	0-290
	Unknown	1	572	14-3184	0	-	0-2938	0	-	0-812
Respiratory System	Pre 1930	12	64	33-112	7	70	28-145	6	44	16- 95
	1930-	39	66	46- 87	34	95	63-127	33	67	44- 91
	1940-	40	118	82-155	15	91	51-150	40	111	76-145
	1950-	32	82	53-110	7	62	25-127	21	75	47-115
	1960-	49	132	95-169	16	85	49-139	28	119	79-172
	Unknown	3	103	21-300	1	41	1-229	3	34	7-100
Bronchitis, Emphysema and Asthma	Pre 1930	4	59	16-150	3	76	16-222	0	-	0- 70
	1930-	8	36	16- 72	12	84	43-146	9	47	21- 89
	1940-	11	87	44-156	4	61	17-156	11	78	39-139
	1950-	9	62	29-118	1	23	1-26	7	65	26-133
	1960+	9	66	30-125	7	97	39-200	8	88	38-173
	Unknown	0	-	0-332	0	-	0-383	0	-	0-108
Asbestosis	Pre 1930	1	3928	99-21879	0	-	0-31689	0	-	0-22818
	1930-	2	1737	210-6272	0	-	0-6344	0	-	0-4374
	1940-	1	1251	32-6968	0	-	0-11109	0	-	0-5055
	1950-	1	1191	30-6634	0	-	0-19338	0	-	0-7144
	1960+	2	2676	324-9661	0	-	0-13407	1	2335	59-13007
	Unknown	0	-	0-65001	0	-	0-99987	0	-	0-25403
Pulmonary Fibrosis	Pre 1930	0	-	0-1529	0	-	0-3393	0	-	0-2451
	1930-	0	-	0-430	0	-	0-835	0	-	0-594
	1940-	2	393	48-1417	0	-	0-1725	0	-	0-786
	1950-	0	-	0-632	1	723	18-4025	0	-	0-1041
	1960+	0	-	0-673	0	-	0-1689	0	-	0-1219
	Unknown	0	-	0-8618	0	-	0-12872	0	-	0-3430

TABLE A3.13: Cause specific mortality by length of service and dockyard.

Causes of Death	Length of service (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	< 5	128	98	81-115	56	77	57- 97	72	87	67-107
	5-	224	108	94-122	72	85	65-105	83	92	72-112
	10-	412	92	83-101	87	81	64- 98	233	92	80-104
	20-	431	98	89-107	154	90	76-104	333	83	74- 92
	30+	888	91	85- 97	451	94	85-102	610	84	77- 90
	Unknown	47	141	100-181	23	98	62-148	69	80	61- 99
All Neoplasms	< 5	45	126	89-163	18	87	51-137	26	107	70-157
	5-	55	95	70-120	22	91	57-137	29	109	73-157
	10-	143	113	95-132	28	91	60-131	85	112	88-136
	20-	141	111	93-130	62	119	89-149	96	78	63- 94
	30+	332	118	105-130	171	117	99-134	233	104	91-117
	Unknown	12	125	65-219	9	129	59-245	21	80	50-123
Ca. Stomach	< 5	5	182	59-426	2	122	15-440	1	53	1-298
	5-	6	130	48-283	1	51	1-285	2	97	12-350
	10-	10	97	46-178	0	-	0-150	5	83	27-193
	20-	15	146	82-241	6	142	52-310	8	81	35-160
	30+	41	170	118-222	20	164	100-253	24	131	84-195
	Unknown	1	123	3-688	1	174	4-968	0	-	0-173
Ca. Peritoneum	< 5	0	-	0-4214	1	2501	63-13933	0	-	0-7499
	5-	0	-	0-2732	0	-	0-8085	0	-	0-6790
	10-	1	355	9-1976	0	-	0-5396	0	-	0-2509
	20-	5	1798	583-4198	0	-	0-3749	1	294	0-1664
	30+	3	615	127-1798	1	448	11-2495	0	-	7-1640
	Unknown	0	-	0-19853	0	-	0-31750	0	-	0-8939
Ca. Lung	< 5	11	92	46-165	4	51	14-130	12	131	68-228
	5-	21	106	66-162	8	86	37-169	12	119	62-209
	10-	58	131	98-165	8	70	30-137	32	109	71-147
	20-	40	89	61-117	23	112	71-168	36	75	50- 99
	30+	89	85	67-103	50	83	60-106	87	95	75-115
	Unknown	4	114	31-293	3	108	22-315	9	86	39-163
Ca. Pleura	< 5	2	1040	126-3753	1	1082	27-6024	0	-	0-3026
	5-	1	338	9-1882	0	-	0-3643	0	-	0-2813
	10-	9	1366	625-2591	0	-	0-2377	3	812	168-2374
	20-	10	1395	669-2565	4	1529	417-3914	4	652	178-1670
	30+	39	3167	2173-4161	16	2783	1591-4518	19	2037	1227-3182
	Unknown	1	2280	58-12701	0	-	0-12895	0	-	0-3344

TABLE A3.13 (cont.): Cause specific mortality by length of service and dockyard.

Causes of Death	Length of service (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	< 5	56	93	69-117	24	73	46-108	28	75	50-108
	5-	115	114	93-135	35	90	60-119	37	90	61-119
	10-	205	91	78-103	46	92	65-119	111	92	75-109
	20-	228	101	88-114	66	79	60- 98	172	88	75-101
	30+	450	88	80- 96	208	88	76-100	287	80	71- 90
	Unknown	27	158	104-230	12	107	55-186	39	93	64-122
Pulmonary Circulation	< 5	0	-	0-627	0	-	0-1025	0	-	0-954
	5-	4	400	109-1025	0	-	0-849	0	-	0-858
	10-	7	316	127-651	1	190	5-1061	0	-	0-293
	20-	5	231	75-539	0	-	0-423	1	49	1-274
	30+	10	190	91-350	3	113	23-331	1	26	1-144
	Unknown	1	572	14-3184	0	-	0-2938	0	-	0-812
Respiratory System	< 5	14	146	80-246	7	98	39-202	7	98	39-202
	5-	24	144	92-214	8	92	40-182	11	139	69-248
	10-	32	87	57-117	5	49	16-116	19	80	48-125
	20-	34	99	65-132	14	89	48-149	40	107	74-140
	30+	68	76	58- 94	45	89	63-115	51	69	50- 88
	Unknown	3	103	21-300	1	41	1-229	3	34	7-100
Bronchitis, Emphysema and Asthma	< 5	0	-	0-104	3	111	23-324	3	109	22-317
	5-	6	98	36-213	4	120	33-306	3	97	20-285
	10-	10	73	35-135	0	-	0- 95	5	55	18-127
	20-	10	78	38-144	4	64	17-163	12	82	43-144
	30+	15	45	25- 74	16	79	45-128	12	42	21- 72
	Unknown	0	-	0-332	0	-	0-383	0	-	0-108
Asbestosis	< 5	0	-	0-17809	0	-	0-34127	0	-	0-27530
	5-	2	6037	730-21793	0	-	0-29992	1	6888	174-38369
	10-	0	-	0-4999	0	-	0-23773	0	-	0-8918
	20-	2	2427	294-8761	0	-	0-11995	0	-	0-5069
	30+	3	1774	366-5185	0	-	0-4600	0	-	0-2931
	Unknown	0	-	0-65001	0	-	0-99987	0	-	0-25403
Pulmonary Fibrosis	< 5	0	-	0-2555	0	-	0-4457	0	-	0-3997
	5-	0	-	0-1509	0	-	0-3701	0	-	0-3632
	10-	0	-	0-680	1	832	21-4634	0	-	0-1233
	20-	1	189	5-1054	0	-	0-1803	0	-	0-766
	30+	1	78	2-435	0	-	0-601	0	-	0-400
	Unknown	0	-	0-8618	0	-	0-12872	0	-	0-3430

TABLE A3.14: Cause specific mortality by time since start of employment and by dockyard.

Causes of Death	Time since start of employ.	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	< 10	71	96	74-118	24	72	46-107	32	81	53-109
	10-	324	105	94-116	95	79	63- 95	146	89	74-103
	20-	399	91	82-100	113	90	73-106	256	93	82-105
	30+	1289	93	88- 98	588	92	85-100	897	83	78- 89
	Unknown	47	141	100-181	23	98	62-148	69	80	61- 99
All Neoplasms	< 10	19	101	61-158	8	88	38-174	10	93	45-172
	10-	95	111	88-133	28	81	54-117	50	103	74-131
	20-	132	107	89-125	36	99	67-131	85	104	82-126
	30+	470	117	107-128	229	118	102-133	324	98	87-108
	Unknown	12	125	65-219	9	129	59-245	21	80	50-123
Ca. Stomach	< 10	1	63	2-350	1	123	3-686	0	-	0-402
	10-	12	171	88-298	2	72	9-260	4	102	28-262
	20-	9	88	40-168	2	67	8-243	5	75	24-174
	30+	55	165	121-209	24	151	97-224	31	117	76-158
	Unknown	1	123	3-688	1	174	4-968	0	-	0-173
Ca. Peritoneum	< 10	0	-	0-5432	0	-	0-14368	0	-	0-11262
	10-	1	467	12-2602	1	1472	37-8201	0	-	0-3519
	20-	0	-	0-1308	0	-	0-4750	0	-	0-2239
	30+	8	1133	488-2231	1	329	8-1830	1	196	5-1093
	Unknown	0	-	0-19853	0	-	0-31750	0	-	0-8939
Ca. Lung	< 10	6	89	33-194	3	81	17-238	4	95	26-243
	10-	30	99	67-142	8	60	26-119	25	132	85-195
	20-	50	116	84-148	11	79	40-142	32	100	65-134
	30+	133	91	76-107	71	90	69-111	118	89	73-105
	Unknown	4	114	31-293	3	108	22-315	9	86	39-163
Ca. Pleura	< 10	0	-	0-4385	1	2880	73-16043	0	-	0-7912
	10-	4	965	263-2471	0	-	0-2568	0	-	0-1619
	20-	11	1717	857-3071	0	-	0-2140	5	1284	416-2996
	30+	46	2351	1672-3030	20	2396	1464-3700	21	1396	864-2134
	Unknown	1	2280	58-12701	0	-	0-12895	0	-	0-3344

TABLE A3.14 (cont.): Cause specific mortality by time since start of employment and by dockyard.

Causes of Death	Time since start of employ.	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	< 10	33	102	67-137	10	69	33-126	10	59	28-108
	10-	165	110	93-127	48	87	62-112	70	92	71-114
	20-	206	93	80-105	58	97	72-123	123	93	77-110
	30+	650	90	83- 97	263	84	74- 94	432	82	74- 90
	Unknown	27	158	104-230	12	107	55-186	39	93	64-122
Pulmonary Circulation	< 10	1	307	8-1708	0	-	0-2241	0	-	0-1886
	10-	7	439	176-904	0	-	0-566	0	-	0-433
	20-	5	226	73-527	1	159	4-886	0	-	0-261
	30+	13	184	98-314	3	88	18-258	2	36	4-131
	Unknown	1	572	14-3184	0	-	0-2938	0	-	0-812
Respiratory System	< 10	15	273	153-450	5	157	51-366	9	272	125-517
	10-	29	118	79-169	8	68	29-131	11	75	37-134
	20-	27	76	50-110	8	67	29-131	31	122	79-165
	30+	101	83	67- 99	58	89	66-112	77	72	56- 88
	Unknown	3	103	21-300	1	41	1-229	3	34	7-100
Bronchitis, Emphysema and Asthma	< 10	0	-	0-144	2	127	15-457	2	126	15-455
	10-	8	82	35-161	3	64	13-186	3	49	10-144
	20-	7	53	21-108	3	63	13-185	9	87	40-165
	30+	26	59	39- 87	19	75	45-117	21	52	32- 79
	Unknown	0	-	0-332	0	-	0-383	0	-	0-108
Asbestosis	< 10	0	-	0-37676	0	-	0-87174	0	-	0-68452
	10-	1	2064	52-11497	0	-	0-21004	1	3772	95-21011
	20-	1	1378	35-7678	0	-	0-19438	0	-	0-8257
	30+	5	2013	652-4698	0	-	0-3390	0	-	0-1927
	Unknown	0	-	0-65001	0	-	0-99987	0	-	0-25403
Pulmonary Fibrosis	< 10	0	-	0-3838	0	-	0-8424	0	-	0-7516
	10-	0	-	0-1018	0	-	0-2750	0	-	0-1963
	20-	0	-	0-693	1	659	17-3669	0	-	0-1125
	30+	2	114	14-412	0	-	0-466	0	-	0-277
	Unknown	0	-	0-8618	0	-	0-12872	0	-	0-3430

TABLE A3.15: Lung cancer mortality by length of service, time since first exposure, and dockyard.

Length of service (yrs)	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
				DEVONPORT						
<5	5	128 (42-300)	6	76 (28-166)	-		-		-	
5 - 9	1	36 (1-198)	13	111 (59-189)	7	132 (52-273)	-		-	
10 - 19	-		11	104 (52-186)	31	122 (79-164)	16	199 (114-324)	-	
20 - 29	-		-		12	98 (51-171)	21	77 (48-118)	7	126 (51-260)
30+	-		-		-		16	82 (47-133)	73	86 (66-105)
				CHATHAM						
<5	1	41 (1-226)	3	58 (12-170)	-		-		-	
5 - 9	2	164 (20-591)	3	54 (11-157)	3	119 (25-348)	-		-	
10 - 19	-		2	79 (10-284)	5	76 (25-178)	1	42 (1-234)	-	
20 - 29	-		-		3	66 (14-194)	13	103 (55-177)	7	203 (81-418)
30+	-		-		-		10	103 (49-189)	40	79 (55-104)
				PORTSMOUTH						
<5	3	104 (21-303)	9	148 (68-280)	-		-		-	
5 - 9	1	77 (2-426)	9	152 (69-288)	2	71 (9-257)	-		-	
10 - 19	-		7	102 (41-210)	21	120 (74-183)	4	82 (22-209)	-	
20 - 29	-		-		9	78 (36-147)	21	73 (45-111)	6	77 (28-168)
30+	-		-		-		12	90 (47-158)	75	96 (74-118)

TABLE A3.16: Pleural mesothelioma mortality by length of service, time since first exposure, and dockyard.

Length of service (yrs)	Time since first exposure (employment) 0 - 9 Obs SMR (95% CI)	10 - 19 Obs SMR (95% CI)	20 - 29 Obs SMR (95% CI)	30 - 39 Obs SMR (95% CI)	40+ Obs SMR (95% CI)
			DEVONPORT		
<5	-	2 1468 (178-5300)	-	-	-
5 - 9	-	1 621 (16-3462)	-	-	-
10 - 19	-	1 856 (22-4769)	7 1819 (730-3747)	1 637 (16-3547)	-
20 - 29	-	-	4 2700 (736-6913)	5 1105 (358-2578)	1 864 (22-4812)
30+	-	-	-	5 2354 (763-5495)	34 3337 (2215-4459)
			CHATHAM		
<5	1 4117 (104-22933)	-	-	-	-
5 - 9	-	-	-	-	-
10 - 19	-	-	-	-	-
20 - 29	-	-	-	4 2509 (684-6423)	-
30+	-	-	-	1 1137 (29-6333)	15 3080 (1725-5080)
			PORTSMOUTH		
<5	-	-	-	-	-
5 - 9	-	-	-	-	-
10 - 19	-	-	3 1335 (275-3902)	-	-
20 - 29	-	-	2 1698 (206-6131)	1 262 (7-1458)	1 885 (22-4931)
30+	-	-	-	5 3707 (1201-8653)	14 1756 (959-2946)

TABLE A3.17: Lung cancer mortality by year of start of employment, time since first exposure, and dockyard.

Year of Start	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
DEVONPORT										
Pre 1930	-		-		-		-		14	79 (43-132)
1930-	-		-		-		10	78 (37-143)	51	88 (64-113)
1940-	-		-		8	156 (67-308)	20	82 (50-127)	15	98 (55-162)
1950-	-		1	21 (1-116)	21	81 (50-124)	23	131 (83-196)	-	
1960+	6	89 (33-194)	29	114 (76-164)	21	170 (105-259)	-		-	
CHATHAM										
Pre 1930	-		-		-		-		8	81 (35-160)
1930-	-		-		-		10	139 (67-256)	29	80 (54-115)
1940-	-		-		-		9	75 (35-143)	10	127 (61-233)
1950-	-		1	95 (2-531)	8	116 (50-229)	5	90 (29-210)	-	
1960+	3	81 (17-238)	7	57 (23-118)	3	66 (14-192)	-		-	
PORTSMOUTH										
Pre 1930	-		-		-		-		15	111 (62-184)
1930-	-		-		-		11	145 (72-259)	49	91 (66-117)
1940-	-		-		1	20 (1-112)	14	56 (31- 94)	17	92 (53-147)
1950-	-		2	74 (9-268)	17	95 (55-152)	12	83 (43-145)	-	
1960+	4	94 (26-243)	23	142 (90-212)	14	151 (82-253)	-		-	

TABLE A3.18: Pleural mesothelioma mortality by year of start of employment, time since first exposure, and dockyard.

Year of Start	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
Pre 1930	-	-	-	-	8 5118 (2207-10082)
1930-	-	-	-	1 772 (20-4299)	22 3167 (1985-4796)
1940-	-	-	1 1684 (43-9377)	8 2256 (973-4444)	5 1763 (571-4114)
1950-	-	-	9 2512 (1150-4767)	2 592 (72-2137)	-
1960+	-	4 1108 (302-2837)	1 448 (11-2496)	-	-
			CHATHAM		
Pre 1930	-	-	-	-	1 1424 (36-7933)
1930-	-	-	-	-	14 3995 (2183-6704)
1940-	-	-	-	4 2848 (776-7290)	-
1950-	-	-	-	1 1120 (28-6239)	-
1960+	1 2880 (73-16043)	-	-	-	-
			PORTSMOUTH		
Pre 1930	-	-	-	-	5 5072 (1643-11838)
1930-	-	-	-	2 2881 (349-10400)	9 1684 (771-3195)
1940-	-	-	-	3 1009 (208-2951)	1 361 (9-2008)
1950-	-	-	4 1953 (532-4999)	1 440 (11-2453)	-
1960+	-	-	1 733 (19-4083)	-	-

TABLE A3.19: Lung cancer mortality by age at start of employment, time since first exposure, and dockyard.

Age at start (yrs)	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
<25	-	3 445 (92-1301)	4 95 (26-244)	19 92 (56-144)	67 92 (70-114)
25-	-	2 153 (19-553)	6 74 (27-161)	15 71 (40-118)	13 75 (40-129)
35-	-	11 155 (77-277)	24 125 (80-186)	17 142 (83-227)	-
45-	2 61 (7-219)	9 56 (26-106)	13 118 (63-201)	2 194 (24-701)	-
55+	4 154 (42-393)	5 99 (32-232)	3 423 (87-1238)	-	-
			CHATHAM		
<25	-	-	1 75 (2-420)	8 94 (41-185)	35 83 (56-111)
25-	-	-	1 38 (1-210)	11 100 (50-179)	10 86 (41-158)
35-	-	1 50 (1-281)	4 75 (20-191)	5 104 (34-244)	2 620 (75-2240)
45-	1 76 (2-423)	5 83 (27-193)	4 109 (30-279)	-	-
55+	2 97 (12-351)	2 43 (5-154)	1 115 (3-640)	-	-
			PORTSMOUTH		
<25	-	1 303 (0-15119)	1 36 (8-1686)	9 57 (26-107)	67 97 (74-120)
25-	-	2 297 (36-1073)	6 105 (38-228)	15 81 (46-134)	12 79 (41-137)
35-	-	7 155 (62-320)	15 98 (55-162)	12 102 (53-178)	2 168 (20-607)
45-	1 54 (1-300)	11 113 (56-202)	10 127 (61-233)	1 109 (3-606)	-
55+	3 158 (33-461)	4 109 (30-278)	-	-	-

TABLE A3.20: Pleural mesothelioma mortality by age at start of employment, time since first exposure, and dockyard.

Age at start (yrs)	Time since first exposure (employment) 0 - 9 Obs SMR (95% CI)	10 - 19 Obs SMR (95% CI)	20 - 29 Obs SMR (95% CI)	30 - 39 Obs SMR (95% CI)	40+ Obs SMR (95% CI)
			DEVONPORT		
<25	-	-	3 1948 (402-5695)	8 1891 (815-3725)	29 3014 (2019-4329)
25-	-	1 2426 (61-13513)	6 4177 (1532-9092)	2 745 (90-2689)	5 3006 (974-7017)
35-	-	-	1 446 (11-2483)	1 823 (21-4585)	1 16129 (408-89838)
45-	-	3 1700 (351-4970)	1 890 (23-4956)	-	-
55+	-	-	-	-	-
			CHATHAM		
<25	-	-	-	4 2717 (740-6956)	13 2868 (1527-4905)
25-	-	-	-	1 980 (25-5461)	2 2245 (272-8015)
35-	1 22170 (561-123485)	-	-	-	-
45-	-	-	-	-	-
55+	-	-	-	-	-
			PORTSMOUTH		
<25	-	-	2 2283 (276-8242)	6 2069 (759-4504)	15 1944 (1089-3206)
25-	-	-	2 2353 (285-8495)	-	-
35-	-	-	1 674 (17-3756)	-	-
45-	-	-	-	-	-
55+	-	-	-	-	-

TABLE A3.21: Cause specific mortality by medical symptom - cough.

Causes of Death	Cough	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	Yes	590	121	111-130	210	119	103-135	464	127	115-139
	No	1540	88	83- 93	633	83	76- 89	936	73	68-78
All Neoplasms	Yes	220	157	136-178	77	145	113-177	176	158	135-181
	No	508	102	93-111	233	102	89-115	314	81	72- 90
Ca. Stomach	Yes	18	154	91-243	8	183	79-360	12	133	69-232
	No	60	146	109-182	22	118	74-178	28	90	60-130
Ca. Peritoneum	Yes	2	712	86-2572	0	-	0-4081	0	-	0-1948
	No	7	694	279-1430	2	504	61-1819	1	151	4-839
Ca. Lung	Yes	93	183	146-220	36	168	113-223	85	190	150-231
	No	130	73	60- 850	60	66	49- 83	103	67	54- 80
Ca. Pleura	Yes	16	2338	1337-3796	5	2226	721-5194	4	802	219-2053
	No	46	1873	1332-2415	16	1617	924-2625	22	1236	775-2053
Circulatory System	Yes	266	106	93-119	80	94	73-114	192	109	93-124
	No	815	92	85- 98	311	85	75- 94	482	78	71- 85
Pulmonary Circulation	Yes	10	397	191-730	0	-	0-392	1	52	1-292
	No	17	192	112-307	4	99	27-255	1	15	0- 85
Respiratory System	Yes	68	162	124-201	39	219	150-288	62	175	131-218
	No	107	72	58- 86	41	53	37- 69	69	56	43- 69
Bronchitis, Emphysema and Asthma	Yes	21	134	83-204	17	238	139-382	18	128	76-203
	No	20	36	22- 56	10	33	16- 61	17	36	21- 57
Asbestosis	Yes	3	3536	729-10336	0	-	0-12720	0	-	0-5866
	No	4	1313	363-3413	0	-	0-2968	1	455	12-2536
Pulmonary Fibrosis	Yes	1	163	4-907	0	-	0-1691	0	-	0-823
	No	1	46	1-257	1	107	3-598	0	-	0-237

TABLE A3.22: Cause specific mortality by medical symptom - phlegm.

Causes of Death	Phlegm	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	Yes	678	113	105-122	226	112	98-127	473	122	111-133
	No	1452	89	84- 93	617	83	77- 90	927	74	69- 79
All Neoplasms	Yes	235	137	119-154	74	122	94-149	179	151	129-173
	No	493	106	96-115	236	107	93-121	311	82	73- 91
Ca. Stomach	Yes	23	160	101-239	6	119	44-260	16	166	95-270
	No	55	143	105-181	24	133	85-198	24	78	50-117
Ca. Peritoneum	Yes	2	597	72-2154	0	-	0-3618	0	-	0-1869
	No	7	734	295-1512	2	519	63-1874	1	152	4-849
Ca. Lung	Yes	94	151	120-181	33	134	89-180	94	198	158-238
	No	129	77	64- 91	63	72	54- 90	94	62	50- 75
Ca. Pleura	Yes	15	1834	1027-3025	1	393	10-2189	2	383	46-1383
	No	47	2024	1445-2603	20	2084	1273-3219	24	1366	875-2033
Circulatory System	Yes	315	102	91-113	105	108	87-128	198	105	90-120
	No	766	92	86- 99	286	81	71- 90	476	79	72- 86
Pulmonary Circulation	Yes	8	257	111-506	0	-	0-341	2	98	12-355
	No	19	230	138-359	4	103	28-264	0	-	0-58
Respiratory System	Yes	79	151	118-184	29	142	95-203	63	165	124-206
	No	96	70	56- 83	51	69	50- 87	68	56	43- 70
Bronchitis, Emphysema and Asthma	Yes	27	138	91-201	11	134	67-240	14	93	51-156
	No	14	27	15- 46	16	55	31- 89	21	45	28- 69
Asbestosis	Yes	3	2906	600-8494	0	-	0-11119	0	-	0-5555
	No	4	1420	387-3636	0	-	0-3071	1	463	12-2577
Pulmonary Fibrosis	Yes	1	132	3-733	0	-	0-1476	0	-	0-769
	No	1	49	1-275	1	111	3-619	0	-	0-242

TABLE A3.23: Cause specific mortality by medical symptom - breathlessness.

Causes of Death	Breathless	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	Yes	410	143	130-157	126	149	123-175	278	161	142-180
	No	1720	88	84- 92	717	84	78- 90	1122	76	72- 81
All Neoplasms	Yes	146	179	150-208	35	136	91-181	82	156	122-189
	No	582	105	96-113	275	108	95-120	408	91	82-100
Ca. Stomach	Yes	10	142	68-261	4	186	51-476	7	161	64-331
	No	68	148	113-183	26	124	81-182	33	92	61-123
Ca. Peritoneum	Yes	4	2665	726-6823	1	2436	62-13570	0	-	0-4424
	No	5	439	142-1025	1	224	6-1248	1	130	3-723
Ca. Lung	Yes	53	176	129-223	16	152	87-246	41	191	132-249
	No	170	85	73- 98	80	79	61- 96	147	83	70- 96
Ca. Pleura	Yes	8	2234	963-4400	2	1972	239-7118	3	1389	287-4060
	No	54	1941	1423-2459	19	1707	1028-2666	23	1115	707-1673
Circulatory System	Yes	189	127	109-145	64	155	117-193	127	151	124-177
	No	892	90	84- 96	327	79	71- 88	547	77	71- 84
Pulmonary Circulation	Yes	12	778	402-1360	0	-	0-791	1	107	3-595
	No	15	152	85-251	4	89	24-228	1	13	0-74
Respiratory System	Yes	55	209	154-264	25	283	183-418	55	312	230-394
	No	120	73	60- 86	55	64	47- 81	76	54	42- 66
Bronchitis, Emphysema and Asthma	Yes	22	221	138-335	13	362	192-617	20	283	173-437
	No	19	31	19- 49	14	41	23- 69	15	27	15- 45
Asbestosis	Yes	1	2084	53-11608	0	-	0-26479	0	-	0-12618
	No	6	1781	653-3876	0	-	0-2647	1	395	10-2199
Pulmonary Fibrosis	Yes	0	-	0-990	0	-	0-3423	0	-	0-1685
	No	2	83	10-299	1	96	2-534	0	-	0-206

TABLE A3.24: Cause specific mortality by medical symptom - chest-illness.

Causes of Death	Chest-illness	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	Yes	279	108	95-121	93	128	102-154	165	107	91-124
	No	1851	94	89- 98	750	86	80- 93	1235	83	78- 87
All Neoplasms	Yes	95	129	103-154	22	101	63-152	49	105	76-135
	No	633	112	103-121	288	111	98-124	441	97	88-106
Ca. Stomach	Yes	11	176	88-315	2	110	13-397	1	26	67-147
	No	67	144	109-178	28	132	88-191	39	107	73-141
Ca. Peritoneum	Yes	4	2862	780-7328	0	-	0-9987	0	-	0-4883
	No	5	435	141-1015	2	444	54-2604	1	129	3-716
Ca. Lung	Yes	36	134	90-178	8	90	39-177	27	144	95-210
	No	187	92	79-106	88	85	67-103	161	90	76-103
Ca. Pleura	Yes	5	1467	475-3423	2	2214	268-7994	1	500	13-2783
	No	57	2036	1508-2565	19	1691	1018-2640	25	1203	778-1775
Circulatory System	Yes	119	89	73-105	46	131	93-169	75	101	78-123
	No	962	95	89-102	345	83	74-91	599	83	77- 90
Pulmonary Circulation	Yes	7	514	206-1059	1	253	6-1409	0	-	0-456
	No	20	199	121-308	3	66	14-192	2	26	3- 95
Respiratory System	Yes	47	204	145-262	18	243	144-384	32	208	136-2804
	No	128	77	63- 90	62	71	53- 89	99	69	55- 83
Bronchitis, Emphysema and Asthma	Yes	14	163	89-274	10	332	159-610	15	249	139-410
	No	27	44	29- 63	17	49	29- 79	20	36	22- 55
Asbestosis	Yes	3	6812	1406-19915	0	-	0-31107	0	-	0-14158
	No	4	1174	320-3004	0	-	0-2609	1	390	10-2172
Pulmonary Fibrosis	Yes	0	-	0-1116	0	-	0-4112	0	-	0-1928
	No	2	81	10-294	1	94	2-525	0	-	0-203

TABLE A3.25: Cause specific mortality by x-ray group and dockyard.

Causes of Death	X-ray group	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	1	1428	91	86- 95	674	85	79- 91	985	80	75- 85
	2	123	89	73-105	67	90	68-111	161	101	85-117
	3	55	105	77-133	17	77	45-124	39	85	58-119
	4	56	151	112-191	21	125	77-191	29	151	101-218
	5	96	82	66- 99	60	136	102-171	73	116	90-143
	6	208	116	101-132	104	124	100-147	173	101	86-117
All Neoplasms	1	491	109	99-119	241	102	89-115	328	88	78- 97
	2	50	125	90-159	20	88	54-136	63	128	96-160
	3	27	179	118-261	8	120	52-237	12	85	44-149
	4	21	198	122-302	7	137	55-283	14	240	131-402
	5	32	95	62-127	11	83	41-148	22	115	72-174
	6	54	105	77-133	38	151	103-199	59	114	85-143
Ca. Stomach	1	56	152	112-192	24	125	80-1861	22	74	46-112
	2	5	148	48-346	3	159	34-464	3	75	15-219
	3	3	231	48-675	0	-	0-667	3	260	54-760
	4	0	-	0-398	1	231	6-1287	1	205	5-1143
	5	4	140	38-360	2	180	22-649	0	-	0-235
	6	5	114	37-266	3	142	29-416	8	187	81-369
Ca. Peritoneum	1	6	629	231-1368	2	470	57-1697	0	-	0-544
	2	1	1355	34-7550	0	-	0-10155	0	-	0-4733
	3	2	7761	939-28016	0	-	0-37367	1	48	121-26680
	4	1	5303	134-29539	0	-	0-48582	0	-	0-42079
	5	0	-	0-5937	0	-	0-17277	0	-	0-12401
	6	0	-	0-4020	0	-	0-9555	0	-	0-4628
Ca. Lung	1	142	89	74-103	72	77	59- 95	112	76	62- 90
	2	14	95	52-159	6	64	24-140	23	115	73-173
	3	6	107	39-234	3	110	23-323	3	52	11-152
	4	9	227	104-430	3	142	29-415	9	375	171-711
	5	11	89	44-159	4	74	20-190	14	180	99-303
	6	25	132	86-195	14	137	75-230	26	123	81-181
Ca. Pleura	1	40	1722	1188-2256	16	1517	868-2463	13	719	383-1229
	2	8	4277	1844-8426	2	2137	258-7693	8	3726	1607-7339
	3	5	7910	2563-18463	2	7894	955-28497	1	1760	45-9805
	4	2	4456	539-16085	0	-	0-20063	1	4405	111-24538
	5	3	1894	391-5538	0	-	0-6932	1	1264	32-7041
	6	1	444	11-2473	3	3138	647-9172	2	941	114-3398

X-ray group: 1 = Normal
2 = Pleural thickening
3 = Pleural calcification
4 = Pulmonary fibrosis
5 = Pulmonary tuberculosis
6 = All other abnormalities.

TABLE A3.25 (cont.): Cause specific mortality by x-ray group and dockyard.

Causes of Death	X-ray group	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	1	743	93	86-100	316	83	74- 93	486	82	75- 90
	2	55	77	56-100	33	90	59-121	72	92	71-114
	3	20	73	45-113	11	74	32-146	23	102	65-154
	4	21	108	67-166	8	109	50-207	9	96	44-182
	5	45	74	52- 96	29	134	90-193	34	111	73-148
	6	113	121	99-144	44	107	75-139	86	103	81-125
Pulmonary Circulation	1	15	191	107-315	1	24	1-136	2	32	4-117
	2	1	138	3-768	0	-	0-908	0	-	0-440
	3	0	-	0-1304	0	-	0-3037	1	407	10-2265
	4	1	491	12-2733	0	-	0-3832	0	-	0-3494
	5	2	327	40-1182	0	-	0-1526	1	297	8-1656
	6	5	522	169-1218	0	-	0-795	0	-	0-403
Respiratory System	1	93	72	57- 86	57	73	54- 92	79	69	54- 84
	2	9	74	34-141	6	78	29-170	17	109	63-174
	3	6	123	45-268	0	-	0-155	2	43	5-153
	4	12	346	179-604	4	216	59-552	4	199	54-510
	5	16	155	89-252	15	323	181-532	12	186	96-325
	6	23	140	89-211	17	186	108-298	18	102	61-162
Bronchitis, Emphysema and Asthma	1	19	40	24- 62	14	46	25- 77	13	29	16- 50
	2	1	22	1-123	2	65	8 233	3	49	10-142
	3	0	-	0-202	0	-	0-394	0	-	0-201
	4	4	300	82-767	0	-	0-489	1	124	3-690
	5	2	52	6-189	8	429	185-845	6	237	87-517
	6	8	131	56-257	9	248	114-471	5	73	24-169
Asbestosis	1	3	1096	226-3205	0	-	0-2861	3	1403	290-4103
	2	1	4060	107-22614	0	-	0-29534	0	-	0-13012
	3	0	-	0-41451	0	-	0-103305	0	-	0-46874
	4	2	32287	3919-116917	0	-	0-139031	0	-	0-113961
	5	0	-	0-17832	0	-	0-51140	0	-	0-34569
	6	0	-	0-12103	0	-	0-27641	0	-	0-12772
Pulmonary Fibrosis	1	0	-	0-192	0	-	0-387	1	68	2-379
	2	1	561	14-3123	0	-	0-3927	0	-	0-1845
	3	0	-	0-5311	0	-	0-13042	0	-	0-6298
	4	1	2049	52-11413	1	4637	117-25829	0	-	0-14955
	5	0	-	0-2448	0	-	0-6611	0	-	0-4641
	6	0	-	0-1579	0	-	0-3437	0	-	0-1706

X-ray group: 1 = Normal
2 = Pleural thickening
3 = Pleural calcification
4 = Pulmonary fibrosis
5 = Pulmonary tuberculosis
6 = All other abnormalities.

TABLE A3.26 Cause specific mortality by duration of smoking habit and dockyard.

Causes of Death	Duration of smoking (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	< 10	67	98	75-122	16	56	32- 91	30	63	42- 89
	10-	137	86	72-100	47	79	56-101	76	64	50- 79
	20-	306	96	85-106	92	82	66- 99	165	75	64- 87
	30-	666	100	92-108	264	101	89-113	477	100	91-109
	40+	665	107	99-115	314	106	95-118	472	100	91-109
	Unknown	289	72	64- 81	110	60	49- 71	180	59	50- 67
All Neoplasms	< 10	20	117	72-181	4	53	14-135	9	67	31-128
	10-	46	105	74-135	15	87	49-143	25	72	46-106
	20-	92	98	78-119	34	99	66-132	56	83	61-105
	30-	225	114	99-129	105	130	105-154	171	114	97-131
	40+	254	146	128-164	115	132	108-156	170	120	102-138
	Unknown	91	81	64- 98	37	69	46- 91	59	64	48- 80
Ca. Stomach	< 10	4	348	95-891	1	189	5-1051	2	208	25-750
	10-	3	90	19-263	2	152	18-549	1	38	1-210
	20-	5	69	22-160	2	74	9-267	5	95	31-221
	30-	28	172	114-249	8	121	52-239	13	108	58-185
	40+	26	165	108-242	14	186	101-311	15	125	70-206
	Unknown	12	131	68-229	3	69	14-201	4	54	15-139
Ca. Peritoneum	< 10	2	3393	411-12248	0	-	0-17224	0	-	0-11084
	10-	1	756	19-4210	0	-	0-8151	0	-	0-4372
	20-	3	1325	273-3874	0	-	0-5108	0	-	0-2725
	30-	1	276	7-1538	0	-	0-2830	1	412	10-2293
	40+	1	377	10-2099	0	-	0-3119	0	-	0-1965
	Unknown	1	410	10-2285	2	2008	243-7248	0	-	0-2176
Ca. Lung	< 10	1	21	1-115	0	-	0-149	3	65	13-189
	10-	9	64	29-121	0	-	0- 60	4	32	9- 81
	20-	34	104	69-138	7	53	21-108	16	61	35-100
	30-	70	95	73-117	36	108	73-144	69	113	86-140
	40+	101	156	126-187	49	136	98-174	88	151	120-183
	Unknown	8	20	9- 40	4	19	5- 49	8	22	10- 44
Ca. Pleura	< 10	2	1866	226-6737	0	-	0-9041	0	-	0-5002
	10-	4	1356	370-3472	0	-	0-3535	4	1909	520-4887
	20-	9	1485	680-2819	5	2485	805-5799	6	1552	569-3378
	30-	21	2168	1342-3314	8	2256	973-4443	6	860	315-1871
	40-	11	1867	932-3339	3	1109	229-3243	3	650	134-1901
	Unknown	15	2614	1464-4311	5	2061	668-4809	7	1557	625-3208

TABLE A3.26 (cont.): Cause specific mortality by duration of smoking habit and dockyard.

Causes of Death	Duration of smoking (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	< 10	30	112	76-160	6	55	20-119	10	50	24- 93
	10-	68	89	68-110	24	88	56-131	40	73	50- 96
	20-	179	109	93-125	46	85	60-109	77	72	56- 88
	30-	334	96	86-107	114	89	73-105	224	96	83-108
	40+	321	98	87-109	143	99	82-115	225	97	84-110
	Unknown	149	75	63- 86	58	67	49- 84	98	67	54- 80
Pulmonary Circulation	< 10	0	-	0-1467	0	-	0-3234	0	-	0-1854
	10-	1	146	4-812	0	-	0-1382	0	-	0-703
	20-	4	270	74-692	0	-	0-688	0	-	0-353
	30-	11	319	159-572	0	-	0-266	0	-	0-150
	40+	8	225	97-443	3	174	36-510	1	37	1-208
	Unknown	3	152	31-444	1	106	3-591	1	66	2-366
Respiratory System	< 10	6	150	55-326	1	47	1-262	3	83	17-241
	10-	12	113	58-198	2	42	5-150	6	64	23-139
	20-	21	96	59-146	5	55	18-129	13	71	38-122
	30-	58	105	78-132	27	106	70-155	48	108	77-138
	40+	56	86	63-108	37	105	71-139	55	101	74-127
	Unknown	22	66	41-100	8	44	19- 86	6	21	8- 46
Bronchitis, Emphysema and Asthma	< 10	1	70	2-391	0	-	0-463	0	-	0-271
	10-	2	52	6-188	0	-	0-202	2	56	7-203
	20-	4	49	13-125	3	84	17-245	4	57	15-145
	30-	13	62	33-106	7	69	28-142	11	62	31-112
	40+	16	66	38-108	14	100	54-167	16	75	43-122
	Unknown	5	41	13- 96	3	42	9-124	2	18	2- 66
Asbestosis	< 10	2	23296	2819-84099	0	-	0-105607	0	-	0-52900
	10-	0	-	0-14444	0	-	0-40938	0	-	0-19318
	20-	1	1567	40-8728	0	-	0-17076	0	-	0-8818
	30-	3	2354	486-6881	0	-	0-7801	0	-	0-4113
	40+	1	1073	27-5978	0	-	0-8579	1	1383	35-7705
	Unknown	0	-	0-5566	0	-	0-12776	0	-	0-7015
Pulmonary Fibrosis	< 10	0	-	0-6121	0	-	0-14093	0	-	0-7775
	10-	0	-	0-2195	0	-	0-5975	0	-	0-2945
	20-	0	-	0-1016	0	-	0-2928	0	-	0-1477
	30-	1	117	3-652	0	-	0-1124	0	-	0-623
	40+	1	117	3-650	1	256	6-1428	0	-	0-588
	Unknown	0	-	0-766	0	-	0-1695	0	-	0-1018

TABLE A3.27: Cause specific mortality by occupational group and dockyard.

Causes of Death	Occup. group	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	1	112	105	85-124	38	73	50- 96	50	90	65-115
	2	188	98	84-112	77	114	88-139	132	96	80-113
	3	118	100	82-119	38	93	63-122	73	80	62- 99
	4	1651	93	89- 98	665	88	81- 95	1077	85	79- 89
	Unknown	61	124	93-156	25	104	67-153	68	79	60- 97
All Neoplasms	1	47	153	109-197	15	96	54-158	20	119	73-183
	2	69	126	97-156	36	177	119-235	61	145	109-182
	3	36	110	73-145	14	118	64-198	28	103	68-149
	4	555	110	101-119	237	105	91-118	361	93	84-103
	Unknown	21	149	92-227	87	110	48-217	20	76	46-117
Ca. Stomach	1	3	118	24-346	2	155	19-559	3	223	46-653
	2	6	136	50-296	5	307	99-715	5	150	48-351
	3	3	119	24-347	0	-	0-402	3	141	29-413
	4	61	144	108-181	23	124	78-185	29	93	62-133
	Unknown	5	421	136-982	0	0	0-613	0	-	0-172
Ca. Peritoneum	1	1	1587	40-8842	0	-	0-14697	0	-	0-12085
	2	2	1683	204-6074	0	-	0-9470	1	1305	33-7269
	3	0	-	0-4243	1	3628	92-20206	0	-	0-6577
	4	6	604	222-1316	1	261	7-1451	0	-	0-569
	Unknown	0	-	0-13441	0	-	0-31238	0	-	0-8886
Ca. Lung	1	12	109	56-190	6	79	26-185	7	106	42-218
	2	19	99	59-154	7	88	35-181	26	157	102-230
	3	11	100	50-178	3	68	14-200	8	77	33-515
	4	176	96	82-111	78	86	67-105	139	90	75-105
	Unknown	5	97	32-227	3	102	21-299	8	76	33-149
Ca. Pleura	1	6	3886	1425-8458	1	1590	40-8856	0	-	0-4571
	2	7	2438	979-5021	6	6318	2316-13751	6	2878	1055-6265
	3	8	3962	1708-7805	3	4657	961-13614	2	1388	168-5011
	4	40	1646	1136-2156	11	1143	571-2045	18	1038	615-1640
	Unknown	1	1509	38-8405	0	-	0-12375	0	-	0-3322

Occupational group:
1 = Registered asbestos workers.
2 = Electrical fitters, burners, welders, riveters, caulkers, drillers, shipfitters, plumbers and coppersmiths.
3 = Shipwrights, engine fitters.
4 = All dockyard trades not in groups 1, 2 and 3.

TABLE A3.27 (cont.): Cause specific mortality by occupational group and dockyard.

Causes of Death	Occup. group	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	1	53	97	71-123	19	75	45-117	24	90	58-134
	2	91	94	75-114	31	97	63-131	53	81	59-102
	3	59	104	77-130	14	76	41-127	33	77	51-104
	4	848	93	87-100	312	85	76- 95	525	85	78- 92
	Unknown	30	119	80-170	15	128	72-211	39	93	63-122
Pulmonary Circulation	1	2	369	45-1331	0	-	0-1320	0	-	0-13255
	2	2	212	26-764	0	-	0-1075	0	-	0-544
	3	2	377	46-1362	0	-	0-1942	0	-	0-851
	4	20	219	134-339	4	99	27-255	2	30	4-110
	Unknown	1	392	10-2181	0	-	0-2820	0	-	0-807
Respiratory System	1	7	78	31-162	1	18	0-102	3	58	12-171
	2	14	91	50-152	7	111	45-228	10	81	39-149
	3	11	133	66-238	5	147	48-343	5	64	21-149
	4	138	90	75-105	66	86	65-106	110	88	72-105
	Unknown	5	117	38-274	1	40	1-221	3	34	7-100
Bronchitis, Emphysema and Asthma	1	3	90	19-263	0	-	0-173	1	50	1-278
	2	3	53	11-154	1	40	1-221	5	104	34-242
	3	2	66	8-236	4	150	18-542	0	-	0-123
	4	33	58	38- 78	24	79	50-117	29	60	40- 86
	Unknown	0	-	0-229	0	-	0-369	0	-	0-108
Asbestosis	1	1	5339	135-29736	0	-	0-43716	0	-	0-38516
	2	3	9004	1858-26322	0	-	0-32838	0	-	0-14935
	3	0	-	0-18557	0	-	0-56689	0	-	0-23677
	4	3	985	203-2880	0	-	0-2995	1	22	12-2555
	Unknown	0	-	0-43554	0	-	0-94531	0	-	0-25340
Pulmonary Fibrosis	1	0	-	0-2779	0	-	0-5617	0	-	0-5586
	2	0	-	0-1600	0	-	0-4597	0	-	0-2261
	3	0	-	0-2873	0	-	0-8414	0	-	0-3592
	4	2	90	11-324	1	108	3-599	0	-	0-236
	Unknown	0	-	0-5883	0	-	0-12325	0	-	0-3409

Occupational group:
1 = Registered asbestos workers.
2 = Electrical fitters, burners, welders, riveters, caulkers, drillers, shipfitters, plumbers and coppersmiths.
3 = Shipwrights, engine fitters.
4 = All dockyard trades not in groups 1, 2 and 3.

TABLE A3.28: Cause specific mortality by exposure rating and dockyard.

Causes of Death	Exposure rating	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	< 100	1149	94	88- 99	472	87	79- 94	734	85	79- 91
	100-	316	91	81-101	108	83	67- 99	211	79	69- 90
	200-	224	88	77-100	97	97	78-116	159	82	69- 95
	300-	185	103	88-118	83	115	90-140	139	93	78-109
	400+	125	120	99-141	43	81	57-105	78	105	81-128
	Unknown	131	106	88-125	40	100	69-132	79	83	65-101
All Neoplasms	< 100	367	106	95-116	167	103	88-119	233	89	78-101
	100-	104	104	84-124	43	110	77-143	75	93	72-114
	200-	97	132	106-158	35	114	76-152	59	100	74-125
	300-	70	134	103-166	32	146	95-196	58	125	93-158
	400+	53	178	130-226	15	93	52-154	40	176	121-231
	Unknown	37	105	71-139	18	150	89-238	25	86	56-127
Ca. Stomach	< 100	42	146	102-191	15	114	64-187	14	67	36-112
	100-	10	122	59-225	5	158	51-369	8	123	53-243
	200-	7	115	46-237	4	159	43-408	7	147	59-302
	300-	6	136	50-296	3	164	34-480	6	160	59-349
	400+	6	230	84-500	2	147	18-532	5	264	86-617
	Unknown	7	236	95-486	1	101	3-561	0	-	0-156
Ca. Peritoneum	< 100	2	280	34-1010	2	704	85-2543	0	-	0-807
	100-	0	-	0-1725	0	-	0-5018	0	-	0-2572
	200-	2	1349	163-4869	0	-	0-6966	0	-	0-3683
	300-	2	2141	259-7728	0	-	0-10797	1	1366	35-7610
	400+	3	6072	1253-17751	0	-	0-16397	0	-	0-11227
	Unknown	0	-	0-5310	0	-	0-18322	0	-	0-7961
Ca. Lung	< 100	112	90	74-107	60	94	70-117	100	97	78-116
	100-	36	102	68-135	10	65	31-120	24	75	48-112
	200-	32	120	79-162	10	81	39-149	16	68	39-110
	300-	19	98	59-154	6	67	24-145	21	112	69-170
	400+	14	126	69-212	5	76	24-176	18	193	114-305
	Unknown	10	78	38-144	5	104	34-242	9	77	35-146
Ca. Pleura	< 100	13	761	405-1302	2	288	35-1041	6	498	183-1084
	100-	12	2275	1176-3974	3	1604	331-4688	3	783	162-2288
	200-	14	3678	2010-6172	5	3584	1161-8364	6	2183	800-4751
	300-	15	6284	3519-10364	7	7910	3175-16295	6	2920	1070-6355
	400+	7	5972	2397-12303	3	5410	1116-15816	5	5843	1893-13638
	Unknown	1	593	15-3301	1	1994	50-11108	0	-	0-2956

TABLE A3.28 (cont.): Cause specific mortality by exposure rating and dockyard.

Causes of Death	Exposure rating	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	< 100	603	97	90-105	221	85	74- 96	357	86	77- 95
	100-	166	93	79-107	50	80	58-102	104	81	65- 96
	200-	89	68	54- 82	45	92	65-119	76	80	62- 98
	300-	95	101	81-122	34	96	64-129	67	92	70-114
	400+	60	110	82-138	22	84	53-128	26	71	46-104
	Unknown	68	107	82-133	19	98	59-153	44	95	67-123
Pulmonary Circulation	< 100	16	259	148-420	3	105	22-306	1	23	1-127
	100-	5	287	93-671	0	-	0-555	1	74	2-412
	200-	1	77	2-430	0	-	0-695	0	-	0-371
	300-	2	210	25-756	0	-	0-944	0	-	0-474
	400-	1	173	4-966	0	-	0-1232	0	-	0-898
	Unknown	2	313	38-1129	1	464	12-2582	0	-	0-735
Respiratory System	< 100	93	90	71-108	46	83	59-108	83	100	79-122
	100-	22	77	48-117	10	80	39-148	14	55	30- 93
	200-	25	118	76-174	5	51	17-120	13	69	37-119
	300-	14	87	48-147	12	161	83-281	8	55	24-109
	400+	7	69	28-143	6	101	37-219	9	113	52-214
	Unknown	14	130	71-219	1	24	1-136	4	42	11-107
Bronchitis, Emphysema and Asthma	< 100	19	49	30- 77	16	74	42-120	22	68	43-103
	100-	4	38	10- 97	4	82	22-210	4	41	11-104
	200-	8	102	44-200	1	26	1-143	5	69	22-161
	300-	2	33	4-121	5	167	54-390	3	53	11-154
	400+	4	106	29-271	1	42	1-235	1	32	1-177
	Unknown	4	100	27-255	0	-	0-225	0	-	0- 99
Asbestosis	< 100	1	481	12-2680	0	-	0-4245	1	680	17-3787
	100-	1	1637	41-9118	0	-	0-16907	0	-	0-8028
	200-	2	4358	527-15732	0	-	0-21073	0	-	0-10867
	300-	1	3139	79-17487	0	-	0-30203	0	-	0-13706
	400+	2	11738	1420-42374	0	-	0-44247	0	-	0-29909
	Unknown	0	-	0-17428	0	-	0-56855	0	-	0-22682
Pulmonary Fibrosis	< 100	1	66	2-368	1	151	4-842	0	-	0-353
	100-	0	-	0-868	0	-	0-2389	0	-	0-1152
	200-	0	-	0-1173	0	-	0-3012	0	-	0-1553
	300-	1	429	11-2390	0	-	0-3997	0	-	0-1974
	400+	0	-	0-2631	0	-	0-5285	0	-	0-3823
	Unknown	0	-	0-2362	0	-	0-7484	0	-	0-3105

TABLE A3.29: Cause specific mortality by asbestos exposure period and dockyard.

Causes of Death	Asbestos exposure (yrs)	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
All Causes	< 10	87	97	76-117	32	80	52-108	64	91	68-113
	10-	84	93	73-112	20	65	40-100	76	105	81-128
	20-	118	117	96-138	38	105	71-138	92	110	88-133
	30+	114	98	80-116	48	113	81-144	114	93	76-110
	Unknown	1727	94	90- 99	705	89	83- 96	1054	81	77- 86
All Neoplasms	< 10	25	96	62-142	17	141	82-226	25	115	75-170
	10-	35	133	89-178	7	75	30-154	31	139	90-188
	20-	48	162	116-207	10	90	43-165	34	132	88-176
	30+	45	133	94-172	23	176	111-263	46	121	86-156
	Unknown	575	110	101-119	253	107	94-121	354	90	81-100
Ca. Stomach	< 10	2	94	11-339	4	405	110-1037	5	289	94-675
	10-	2	92	11-334	0	-	0-482	1	56	1-310
	20-	3	122	25-356	0	-	0-402	3	144	30-420
	30+	3	102	21-299	4	366	100-936	6	192	70-418
	Unknown	68	157	120-195	22	114	71-173	25	79	51-117
Ca. Peritoneum	< 10	0	-	0-6989	0	-	0-18000	0	-	0-9704
	10-	0	-	0-6814	0	-	0-23014	0	-	0-9671
	20-	1	1756	44-9780	0	-	0-20013	0	-	0-8848
	30+	3	5363	1107-15677	0	-	0-18631	1	1800	46-10026
	Unknown	5	468	152-1092	2	485	59-1750	0	-	0-543
Ca. Lung	< 10	7	75	30-155	4	83	23-212	10	116	56-213
	10-	21	222	137-339	1	27	1-148	9	101	46-191
	20-	15	136	76-225	2	44	5-158	13	124	66-213
	30+	12	95	49-166	6	111	41-241	17	108	63-174
	Unknown	168	90	76-104	83	89	70-108	139	90	75-105
Ca. Pleura	< 10	3	2316	478-6771	0	-	0-7185	1	962	24-5359
	10-	5	3679	1192-8586	1	2379	60-13253	3	2906	600-8494
	20-	9	6072	2780-11524	2	4077	493-14719	1	865	22-4820
	30+	8	5812	2506-11449	4	7851	2140-20100	9	5915	2708-11226
	Unknown	37	1429	969-1890	14	1371	749-2301	12	665	344-1162

TABLE A3.29 (cont.): Cause specific mortality by asbestos exposure period and dockyard.

Causes of Death	Asbestos exposure	Devonport			Chatham			Portsmouth		
		Obs	SMR	95% CI	Obs	SMR	95% CI	Obs	SMR	95% CI
Circulatory System	< 10	47	102	73-131	12	62	32-109	28	82	55-119
	10-	37	79	54-105	11	73	37-131	34	96	64-129
	20-	53	101	74-128	23	129	82-194	43	105	74-137
	30+	53	87	63-110	17	81	47-130	50	83	60-106
	Unknown	891	95	89-102	328	86	77- 96	519	83	76- 90
Pulmonary Circulation	< 10	0	-	0-814	0	-	0-1732	0	-	0-1040
	10-	2	435	53-1569	0	-	0-2274	1	267	7-1487
	20-	3	574	118-1677	0	-	0-1893	0	-	0-848
	30+	0	-	0-577	0	-	0-1581	0	-	0-557
	Unknown	22	236	148-358	4	96	26-246	1	15	0- 84
Respiratory System	< 10	7	93	37-192	2	50	6-179	5	77	25-179
	10-	9	121	55-229	0	-	0-121	4	58	16-149
	20-	13	154	82-263	2	56	7-202	10	124	60-229
	30+	7	64	26-131	5	112	36-261	13	103	55-177
	Unknown	139	89	74-104	71	89	68-110	99	79	64- 95
Bronchitis, Emphysema and Asthma	< 10	3	108	22-315	2	125	15-450	1	39	1-219
	10-	2	71	9-258	0	-	0-309	1	37	1-205
	20-	2	62	8-224	0	-	0-257	3	95	20-276
	30+	1	24	1-135	2	111	13-399	3	60	12-176
	Unknown	33	57	38- 77	23	73	46-110	27	56	37- 81
Asbestosis	< 10	1	6276	159-34958	0	-	0-55759	0	-	0-29283
	10-	0	-	0-22635	0	-	0-70118	0	-	0-28684
	20-	1	5343	135-29762	0	-	0-57978	0	-	0-24727
	30+	1	5009	127-27903	0	-	0-51393	0	-	0-17193
	Unknown	4	1274	347-3262	0	-	0-2885	1	453	11-2524
Pulmonary Fibrosis	< 10	0	-	0-3271	0	-	0-7548	0	-	0-4328
	10-	0	-	0-3292	0	-	0-9784	0	-	0-4154
	20-	0	-	0-2887	0	-	0-8141	0	-	0-3543
	30+	1	637	16-3549	0	-	0-6667	0	-	0-2323
	Unknown	1	44	1-245	1	104	3-579	0	-	0-235

TABLE A3.30: Devonport Dockyard. All cause mortality by smoking habit and 'asbestos' variables.

	Non-smokers		Ex-smokers		Smokers	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
			Occupational group			
1	29	88 (59-127)	49	142 (102-182)	17	87 (51-140)
2	49	80 (58-103)	70	112 (86-138)	39	128 (88-168)
3	29	85 (57-122)	46	118 (84-153)	23	106 (67-159)
4	584	89 (82- 96)	590	104 (96-113)	268	113 (100-127)
			Exposure rating			
< 100	380	87 (78- 96)	424	109 (99-120)	188	108 (93-124)
100-	103	79 (64- 94)	114	106 (87-126)	59	118 (88-148)
200-	68	79 (60- 98)	91	102 (81-123)	41	105 (73-137)
300-	69	100 (77-124)	64	109 (82-136)	27	125 (83-182)
400+	50	141 (102-180)	36	104 (70-138)	23	151 (96-227)
			Asbestos exposure period (yrs)			
< 10	27	79 (52-116)	37	119 (81-157)	15	100 (56-164)
10-	32	101 (66-136)	31	89 (57-120)	16	106 (60-172)
20-	42	112 (78-146)	38	104 (71-137)	24	136 (87-203)
30+	50	115 (83-147)	28	78 (52-113)	22	123 (77-187)
			Continuous asbestos exposure (yrs)			
< 10	6	96 (35-209)	8	124 (53-244)	6	133 (49-289)
10-	17	268 (156-430)	8	119 (51-234)	1	72 (2-403)

TABLE A3.30 (cont.): Chatham Dockyard. All cause mortality by smoking habit and 'asbestos' variables.

	Non-smokers		Ex-smokers		Smokers	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
			Occupational group			
1	21	72 (45-110)	11	83 (42-149)	5	85 (27-197)
2	39	151 (104-198)	17	83 (43-147)	14	131 (72-220)
3	11	77 (38-138)	12	84 (43-147)	7	126 (51-259)
4	278	91 (80-101)	216	104 (91-118)	395	94 (74-114)
			Exposure rating			
< 100	188	84 (72- 96)	152	105 (89-122)	67	101 (76-125)
100-	42	82 (58-107)	39	105 (72-138)	13	63 (34-108)
200-	57	131 (97-165)	19	72 (43-112)	13	107 (57-182)
300-	41	135 (94-177)	21	89 (55-136)	11	153 (76-274)
400+	14	63 (34-105)	38	94 (55-150)	8	159 (68-313)
			Asbestos exposure period (yrs)			
< 10	15	79 (44-130)	10	97 (47-179)	3	74 (15-216)
10-	11	66 (33-118)	7	75 (30-155)	2	82 (10-296)
20-	21	126 (78-193)	8	89 (38-175)	9	153 (70-290)
30+	27	149 (98-217)	14	93 (51-156)	5	143 (46-334)
			Continuous asbestos exposure (yrs)			
< 10	7	122 (49-252)	7	242 (97-498)	1	162 (4-904)
10-	2	70 (9-254)	2	236 (29-851)	1	109 (3-607)

TABLE A3.30 (cont.): Portsmouth Dockyard. All cause mortality by smoking habit and 'asbestos' variables.

	Non-smokers		Ex-smokers		Smokers	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
			Occupational group			
1	21	114 (71-175)	12	76 (39-133)	12	96 (49-167)
2	43	92 (65-120)	50	127 (92-162)	25	104 (67-154)
3	26	82 (53-120)	20	74 (45-115)	11	91 (45-163)
4	377	85 (76- 93)	369	92 (83-102)	202	100 (86-114)
			Exposure rating			
< 100	247	81 (71- 91)	257	95 (83-106)	136	100 (83-117)
100-	77	79 (61- 97)	63	85 (64-106)	41	98 (68-128)
200-	58	99 (74-125)	48	76 (54- 97)	37	94 (64-124)
300-	47	87 (62-112)	57	127 (94-160)	21	92 (57-140)
400+	35	140 (94-187)	23	86 (55-130)	12	143 (74-250)
			Asbestos exposure period (yrs)			
< 10	26	97 (63-142)	14	59 (32- 98)	17	146 (85-234)
10-	32	96 (63-129)	23	116 (73-174)	13	104 (55-178)
20-	34	113 (75-151)	36	148 (100-196)	18	102 (60-161)
30+	52	103 (75-131)	38	101 (69-133)	15	96 (54-159)
			Continuous asbestos exposure (yrs)			
< 10	6	100 (37-219)	2	57 (7-206)	6	312 (114-678)
10-	9	230 (105-437)	6	110 (40-240)	2	107 (13-385)

APPENDIX 4.

TABLE A4.1: All cause mortality by x-ray group and by time since first exposure.

X-ray group	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
1	47	88 (63-113)	214	97 (84-110)	264	85 (75- 95)	316	91 (81-101)	435	85 (77- 93)
2	2	135 (16-489)	3	34 (7- 98)	24	107 (69-159)	36	97 (66-129)	53	82 (60-104)
3	1	358 (9-1993)	3	220 (45-643)	-		6	70 (26-153)	42	106 (74-139)
4	1	119 (3-664)	7	204 (82-420)	5	71 (23-166)	16	163 (93-264)	27	170 (112-247)
5	2	69 (8-249)	17	123 (71-196)	16	83 (48-135)	18	75 (44-118)	33	74 (49-100)
6	6	114 (42-249)	28	127 (84-183)	39	122 (84-160)	39	98 (67-129)	74	108 (83-132)
					CHATHAM					
1	14	56 (31- 94)	65	74 (56- 93)	75	83 (64-102)	112	80 (65- 95)	238	86 (75- 97)
2	1	185 (5-1028)	2	67 (8-243)	6	99 (36-215)	13	76 (40-129)	40	104 (72-137)
3	-		1	71 (2-393)	-		4	111 (30-284)	11	74 (37-132)
4	-		-		2	147 (18-532)	2	72 (9-259)	13	168 (89-287)
5	1	89 (2-494)	8	184 (79-363)	8	115 (50-227)	15	190 (107-314)	13	120 (64-205)
6	4	210 (57-537)	6	80 (29-174)	11	137 (69-246)	14	99 (54-166)	45	3229 (99-180)
					PORTSMOUTH					
1	13	47 (25- 81)	91	81 (65- 98)	141	80 (67- 94)	182	76 (65- 87)	283	74 (65- 82)
2	4	263 (72-672)	5	61 (20-142)	21	103 (64-158)	44	105 (74-136)	80	95 (75-116)
3	-		1	104 (3-581)	3	109 (23-319)	13	182 (97-311)	21	60 (97-311)
4	-		1	74 (2-413)	7	163 (65-336)	7	120 (48-247)	13	171 (91-292)
5	4	277 (76-710)	5	94 (31-220)	12	125 (65-218)	10	76 (36-139)	25	131 (85-194)
6	4	115 (31-293)	19	122 (73-190)	33	126 (83-1696)	33	103 (68-138)	48	85 (61-109)

TABLE A4.2: Lung cancer mortality by x-ray group and by time since first exposure.

X-ray group	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
1	4 86 (23-220)	17 80 (47-128)	35 116 (77-154)	31 86 (56-117)	38 69 (47- 92)
2	-	-	3 127 (26-370)	3 76 (16-223)	8 115 (50-227)
3	-	1 646 (16-3596)	-	-	5 120 (39-280)
4	-	1 261 (7-1454)	-	4 382 (104-977)	4 238 (65-610)
5	-	3 201 (41-586)	1 51 (1-282)	3 117 (24-341)	3 64 (13-186)
6	1 172 (4-959)	2 84 (10-304)	7 210 (84-432)	4 95 (26-244)	9 123 (56-234)
			CHATHAM		
1	1 37 (1-207)	4 42 (12-109)	7 72 (29-149)	14 84 (46-141)	31 90 (58-122)
2	-	1 267 (7-1486)	-	1 46 (1-256)	3 63 (13-184)
3	-	1 581 (15-3248)	-	-	1 55 (1-305)
4	-	-	-	-	1 104 (3-581)
5	-	1 185 (5-1029)	-	2 215 (26-778)	-
6	-	1 116 (3-648)	3 328 (68-957)	2 114 (14-410)	5 125 (41-293)
			PORTSMOUTH		
1	3 106 (22-309)	12 96 (50-168)	16 80 (46-130)	16 56 (32- 91)	35 73 (49- 97)
2	-	1 96 (2-534)	3 119 (24-347)	4 77 (21-196)	14 133 (73-223)
3	-	-	-	1 108 (3-603)	2 46 (6-166)
4	-	-	-	2 277 (34-1000)	7 737 (296-1517)
5	-	3 436 (90-1276)	1 86 (2-478)	3 190 (39-555)	5 208 (68-487)
6	-	4 201 (55-516)	7 215 (86-444)	5 127 (41-297)	7 100 (40-206)

TABLE A4.3: Pleural mesothelioma mortality by x-ray group and by time since first exposure.

X-ray group	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
1	-	3 973 (201-2845)	8 1680 (725-3310)	5 865 (280-2020)	19 2678 (1612-4182)
2	-	-	2 6818 (825-24613)	1 1833 (46-10211)	5 5812 (1883-13564)
3	-	-	-	-	5 10799 (3499-25204)
4	-	-	-	-	2 10431 (1262-37656)
5	-	-	-	3 8411 (1735-24588)	-
6	-	-	-	-	1 1147 (29-6389)
			CHATHAM		
1	-	-	-	5 2389 (774-5577)	9 2494 (1142-4734)
2	-	-	-	-	2 4121 (499-14876)
3	-	-	-	-	2 11955 (1447-43158)
4	-	-	-	-	-
5	-	-	-	-	-
6	1 53305 (1349-296911)	-	-	-	1 2675 (68-14898)
			PORTSMOUTH		
1	-	-	4 1505 (410-3853)	3 765 (158-2237)	4 749 (204-1919)
2	-	-	1 3790 (96-21112)	1 1671 (42-9305)	6 5416 (1986-11788)
3	-	-	-	-	1 2374 (60-13223)
4	-	-	-	-	1 10660 (270-59379)
5	-	-	-	-	1 3786 (96-21085)
6	-	-	-	1 2323 (59-12941)	1 1416 (36-7887)

TABLE A4.4: All cause mortality by medical history and by time since first exposure.

Medical history	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
Cough	19	124 (75-194)	90	131 (104-158)	116	117 (96-138)	143	120 (101-140)	212	117 (102-133)
Phlegm	25	140 (90-206)	103	123 (100-147)	127	105 (87-123)	160	116 (98-134)	249	108 (95-121)
Breathless	14	162 (89-272)	62	154 (116-192)	79	136 (106-166)	103	155 (125-185)	150	137 (115-159)
Chest-illness	10	128 (61-235)	41	119 (82-155)	51	106 (77-135)	60	102 (76-128)	114	107 (87-126)
					CHATHAM					
Cough	7	116 (46-238)	24	112 (72-166)	23	102 (65-153)	48	132 (95-169)	105	124 (100-147)
Phlegm	5	83 (27-194)	31	128 (83-173)	31	115 (74-155)	46	103 (73-133)	105	110 (89-131)
Breathless	6	195 (72-425)	13	126 (67-215)	16	144 (83-234)	32	192 (125-258)	52	131 (95-166)
Chest-illness	1	56 (1-310)	16	239 (137-388)	9	97 (44-183)	13	78 (41-133)	50	138 (100-176)
					PORTSMOUTH					
Cough	15	157 (88-259)	48	124 (89-159)	81	131 (103-160)	115	135 (110-160)	180	122 (104-140)
Phlegm	13	140 (74-239)	52	129 (94-164)	89	134 (106-164)	103	116 (94-139)	193	118 (101-135)
Breathless	5	118 (38-274)	32	187 (122-251)	52	176 (128-224)	72	181 (139-223)	101	140 (113-168)
Chest-illness	3	81 (17-238)	14	76 (42-128)	44	161 (113-209)	35	97 (65-130)	59	95 (71-119)

TABLE A4.5: Lung cancer mortality by medical history and by time since first exposure.

Medical history	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
Cough	2 137 (17-495)	11 157 (79-282)	19 189 (114-295)	24 194 (124-288)	37 193 (131-255)
Phlegm	4 228 (62-583)	12 139 (72-242)	17 140 (81-224)	25 174 (113-257)	36 147 (99-195)
Breathless	1 107 (3-595)	5 116 (37-270)	13 217 (116-371)	17 243 (142-390)	17 147 (86-236)
Chest-illness	1 126 (3-701)	5 140 (45-327)	10 206 (99-379)	7 114 (46-235)	13 115 (61-197)
			CHATHAM		
Cough	1 145 (4-810)	6 245 (90-533)	4 156 (42-399)	9 200 (92-380)	16 151 (86-245)
Phlegm	2 292 (35-1054)	6 214 (78-465)	1 32 (1-178)	11 198 (99-354)	13 110 (58-188)
Breathless	1 262 (7-1458)	1 78 (2-436)	2 144 (17-519)	4 190 (52-486)	8 162 (70-319)
Chest-illness	-	1 128 (3-715)	-	2 98 (12-353)	5 110 (36-258)
			PORTSMOUTH		
Cough	2 194 (24-701)	16 350 (200-569)	10 135 (65-249)	17 163 (95-260)	36 195 (131-259)
Phlegm	2 200 (24-723)	16 336 (192-546)	11 138 (69-247)	15 138 (78-228)	45 221 (156-286)
Breathless	1 187 (5-1044)	7 328 (132-676)	3 80 (17-234)	11 223 (111-399)	18 201 (119-317)
Chest-illness	-	4 179 (49-458)	7 213 (86-439)	5 115 (37-268)	11 143 (71-256)

TABLE A4.6: Pleural mesothelioma mortality by medical history and by time since first exposure.

Medical history	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
Cough	-	4 1250 (341-3201)	8 1613 (696-3177)	7 1087 (436-2238)	27 3018 (1988-4390)
Phlegm	-	-	2 1188 (144-4287)	3 1468 (303-4292)	9 2992 (1370-5678)
Breathless	-	-	-	1 1111 (28-6189)	7 5221 (2096-10755)
Chest-illness	-	-	-	-	5 3704 (1200-8646)
			CHATHAM		
Cough	-	-	-	1 1984 (50-11053)	4 3962 (1080-10143)
Phlegm	-	-	-	1 1622 (41-9034)	-
Breathless	-	-	-	-	2 4489 (543-16207)
Chest-illness	-	-	-	1 4568 (116-25445)	1 2347 (59-13073)
			PORTSMOUTH		
Cough	-	-	1 1178 (30-6559)	1 801 (20-4464)	2 1050 (127-3789)
Phlegm	-	-	1 1095 (28-6101)	-	1 478 (12-2663)
Breathless	-	-	-	1 1963 (50-10935)	2 2302 (279-8311)
Chest-illness	-	-	-	-	1 1280 (32-7130)

TABLE A4.7: All cause mortality by smoking habit and by time since first exposure.

Smoking habit	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
Non	7	57 (23-118)	43	94 (66-122)	44	65 (46- 84)	49	57 (41- 73)	101	75 (60- 90)
Ex	11	65 (32-116)	50	67 (49- 86)	85	77 (60- 93)	115	85 (70-101)	163	71 (60- 82)
Current	51	117 (85-149)	223	122 (106-138)	265	105 (95-118)	331	113 (101-125)	511	108 (99-118)
Unknown	2	183 (22-660)	8	152 (66-300)	5	63 (20-147)	8	63 (27-125)	11	55 (28- 99)
					CHATHAM					
Non	1	14 (0- 77)	15	52 (29- 86)	18	74 (44-116)	15	50 (28- 82)	43	65 (45- 84)
Ex	6	89 (33-194)	10	43 (21- 79)	21	83 (51-127)	37	72 (49- 95)	90	73 (58- 88)
Current	16	85 (48-138)	70	106 (81-130)	71	97 (75-120)	125	108 (89-126)	270	114 (101-128)
Unknown	1	240 (6-1334)	-		3	98 (20-287)	1	19 (0-108)	7	88 (35-181)
					PORTSMOUTH					
Non	1	15 (0- 86)	16	59 (34- 97)	26	60 (39- 88)	48	72 (51- 92)	66	50 (38- 62)
Ex	5	57 (18-132)	21	51 (32- 79)	66	86 (65-107)	76	65 (50- 80)	138	66 (55- 77)
Current	26	109 (71-160)	107	113 (91-134)	160	105 (89-121)	213	104 (90-118)	355	103 (92-113)
Unknown	-		2	98 (12-353)	4	158 (43-404)	-		1	43 (1-241)

TABLE A4.8: Lung cancer mortality by smoking habit and by time since first exposure.

Smoking habit	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
Non	-		2	53 (6-193)	2	33 (4-119)	1	11 (0- 64)	1	7 (0- 39)
Ex	-		3	39 (8-115)	11	98 (49-175)	10	71 (34-130)	8	33 (14- 65)
Current	6	148 (54-321)	25	137 (88-202)	36	143 (96-190)	42	138 (96-180)	71	141 (108-174)
Unknown	-		-		1	122 (3-682)	-		-	
					CHATHAM					
Non	-		-		-		-		2	24 (3- 88)
Ex	1	124 (3-689)	-		1	35 (1-193)	3	47 (10-138)	9	59 (27-112)
Current	2	96 (12-346)	8	108 (47-213)	10	121 (58-223)	21	148 (91-225)	35	119 (79-158)
Unknown	-		-		-		-		1	105 (3-585)
					PORTSMOUTH					
Non	-		-		2	44 (5-157)	1	13 (0- 71)	3	18 (4- 53)
Ex	-		2	41 (5-146)	3	32 (7- 95)	6	42 (16- 92)	13	50 (27- 86)
Current	2	155 (42-397)	23	209 (133-314)	26	145 (95-212)	30	121 (82-173)	65	151 (114-187)
Unknown	-		-		1	327 (8-1823)	-		-	

TABLE A4.9: Pleural mesothelioma mortality by smoking habit and by time since first exposure.

Smoking habit	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
Non	-	2 3488 (422-12592)	3 2805 (579-8201)	-	8 4332 (1868-8533)
Ex	-	-	3 1907 (394-5576)	2 971 (117-5719)	9 3014 (1380-5719)
Current	-	2 795 (96-2872)	5 1363 (442-3182)	9 1995 (913-3787)	18 2856 (1693-4514)
Unknown	-	-	-	-	-
			CHATHAM		
Non	-	-	-	2 4057 (491-14647)	3 3406 (703-9958)
Ex	-	-	-	-	4 2625 (715-6720)
Current	1 5025 (127-27988)	-	-	3 1819 (375-5318)	8 2702 (1165-5323)
Unknown	-	-	-	-	-
			PORTSMOUTH		
Non	-	-	-	2 1733 (210-6257)	5 2760 (894-6443)
Ex	-	-	1 984 (25-5483)	3 1792 (370-5239)	4 1489 (406-3812)
Current	-	-	4 1849 (504-4735)	1 324 (8-1806)	6 1312 (481-2855)
Unknown	-	-	-	-	-

TABLE A4.10: All cause mortality by smoking amount and by time since first exposure.

Smoking amount (gms/day)	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
< 15	18	85 (50-134)	90	87 (69-105)	141	91 (76-106)	164	87 (74-101)	281	87 (77- 97)
15 - 24	31	118 (76-159)	132	129 (107-151)	133	95 (79-112)	190	114 (98-130)	274	101 (89-113)
25+	13	100 (53-171)	51	97 (71-124)	76	112 (87-137)	92	126 (101-152)	119	113 (93-133)
					CHATHAM					
< 15	13	114 (61-194)	41	93 (65-122)	38	76 (52-100)	71	81 (62-100)	186	102 (87-116)
15 - 24	7	79 (32-163)	28	97 (64-140)	32	96 (63-130)	62	109 (82-136)	127	101 (83-118)
25+	2	36 (4-129)	11	63 (32-113)	22	144 (90-218)	29	123 (82-177)	49	94 (67-120)
					PORTSMOUTH					
< 15	8	61 (26-121)	42	78 (54-102)	86	91 (72-111)	127	90 (75-106)	204	85 (73- 96)
15 - 24	11	84 (42-150)	54	103 (75-130)	92	109 (87-132)	105	89 (72-106)	188	88 (75-101)
25+	12	185 (96-324)	33	113 (74-151)	48	94 (68-121)	57	90 (66-113)	100	100 (80-119)

TABLE A4.11: Lung cancer mortality by smoking amount and by time since first exposure.

Smoking amount (gms/day)	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
< 15	-		10	95 (46-174)	13	83 (44-142)	10	51 (24- 94)	22	64 (40- 97)
15 - 24	5	202 (65-471)	13	128 (68-219)	18	130 (77-205)	26	149 (97-219)	35	121 (81-161)
25+	1	80 (2-445)	5	96 (31-223)	16	235 (134-382)	16	212 (121-344)	22	193 (121-292)
					CHATHAM					
< 15	2	148 (18-534)	3	59 (12-172)	4	70 (19-180)	11	102 (51-183)	21	93 (57-142)
15 - 24	1	103 (3-573)	2	63 (8-228)	5	132 (43-309)	8	115 (49-226)	15	96 (54-158)
25+	-		3	160 (33-468)	2	118 (14-425)	5	172 (56-402)	8	122 (53-240)
					PORTSMOUTH					
< 15	2	130 (16-471)	7	109 (44-224)	10	88 (42-162)	7	41 (17- 85)	22	73 (46-111)
15 - 24	1	71 (2-395)	9	146 (67-278)	11	110 (55-197)	23	160 (101-240)	34	128 (85-171)
25+	1	151 (4-843)	9	269 (123-511)	8	135 (58-266)	6	78 (29-170)	22	176 (110-266)

TABLE A4.12: Pleural mesothelioma mortality by smoking amount and by time since first exposure.

Smoking amount (gms/day)	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
< 15	-	-	3 1406 (290-4109)	6 2181 (800-4748)	11 2661 (1328-4760)
15 - 24	-	1 710 (18-3956)	4 1920 (523-4916)	4 1513 (412-3873)	9 2467 (1129-4682)
25+	-	1 1392 (34-7404)	1 978 (25-5446)	1 850 (21-4732)	7 4631 (1859-9540)
			CHATHAM		
< 15	1 8055 (204-44866)	-	-	2 1683 (204-6077)	9 4017 (1839-7624)
15 - 24	-	-	-	1 1224 (31-6819)	1 631 (16-3516)
25+	-	-	-	-	2 2984 (361-10772)
			PORTSMOUTH		
< 15	-	-	4 3102 (845-7942)	2 975 (118-3521)	6 1916 (702-4170)
15 - 24	-	-	1 842 (21-4689)	1 567 (14-3156)	2 718 (87-2591)
25+	-	-	-	1 1050 (27-5849)	2 1483 (179-5352)

TABLE A4.13: All cause mortality by duration of smoking habit and by time since first exposure.

Duration of smoking (yrs)	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
< 10	6	83 (30-180)	17	99 (58-159)	13	102 (54-174)	9	65 (30-124)	17	102 (59-163)
10-	2	33 (4-118)	37	141 (96-187)	37	89 (60-117)	34	84 (56-112)	26	60 (39- 88)
20-	8	82 (35-161)	38	87 (59-114)	64	95 (71-118)	103	109 (88-130)	91	90 (72-109)
30-	23	113 (71-169)	86	98 (77-119)	126	102 (84-120)	166	108 (92-125)	256	94 (82-105)
40+	23	136 (86-204)	95	115 (91-138)	110	94 (77-112)	134	107 (89-126)	283	106 (94-119)
					CHATHAM					
< 10	2	82 (10-297)	5	80 (26-188)	1	25 (1-141)	3	70 (14-204)	5	44 (14-102)
10-	2	90 (11-327)	7	83 (33-171)	16	138 (79-225)	9	64 (29-122)	13	58 (31-100)
20-	2	75 (9-271)	5	45 (14-104)	10	61 (29-113)	35	109 (73-145)	39	84 (57-110)
30-	10	111 (53-203)	28	96 (64-138)	28	99 (66-143)	54	92 (67-116)	137	105 (88-123)
40+	6	65 (24-141)	35	102 (68-136)	36	94 (63-125)	61	104 (78-131)	166	111 (94-128)
					PORTSMOUTH					
< 10	3	94 (19-275)	8	97 (42-191)	4	44 (12-113)	6	52 (19-114)	8	60 (26-118)
10-	2	63 (8-228)	11	76 (38-136)	19	86 (52-134)	15	50 (28- 82)	26	56 (37- 82)
20-	3	62 (13-181)	17	77 (45-124)	33	84 (55-112)	54	82 (60-104)	51	65 (47- 82)
30-	12	113 (58-197)	43	93 (65-121)	85	112 (88-136)	107	94 (77-112)	211	100 (87-114)
40+	11	102 (51-183)	49	110 (79-141)	85	103 (81-125)	106	105 (85-125)	197	96 (82-109)

TABLE A4.14: Lung cancer mortality by duration of smoking habit and by time since first exposure.

Duration of smoking (yrs)	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
< 10	-		-		1	121 (3-673)	-		-	
10-	-		3	164 (34-480)	2	64 (8-231)	2	52 (6-186)	2	43 (5-154)
20-	-		5	113 (37-264)	6	92 (34-199)	12	124 (64-217)	11	100 (50-179)
30-	1	44 (1-244)	11	111 (56-199)	19	139 (84-217)	14	84 (46-140)	24	80 (51-119)
40+	5	247 (80-576)	9	98 (45-186)	19	156 (94-243)	24	186 (119-276)	42	156 (109-203)
					CHATHAM					
< 10	-		-		-		-		-	
10-	-		-		-		-		-	
20-	-		1	76 (2-422)	1	56 (1-312)	2	54 (7-195)	3	51 (10-148)
30-	1	87 (2-483)	6	162 (60-354)	2	55 (7-200)	10	133 (64-245)	17	103 (60-165)
40+	2	164 (20-593)	1	24 (1-135)	8	173 (74-340)	12	163 (84-284)	24	133 (85-197)
					PORTSMOUTH					
< 10	-		2	526 (64-1899)	-		-		-	
10-	-		-		-		-		3	52 (11-152)
20-	-		4	154 (42-395)	4	90 (25-230)	3	39 (8-113)	5	51 (16-118)
30-	1	76 (2-422)	9	150 (69-285)	9	92 (42-175)	14	98 (54-165)	33	123 (81-165)
40+	3	205 (42-600)	10	174 (83-319)	16	155 (89-251)	19	154 (93-241)	37	149 (101-197)

TABLE A4.15: Pleural mesothelioma mortality by duration of smoking habit and by time since first exposure.

Duration of smoking (yrs)	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
< 10	-	-	-	1 3564 (90-19851)	1 3543 (90-19732)
10-	-	-	1 1170 (30-6516)	1 1132 (29-6304)	2 3069 (371-11078)
20-	-	-	3 2375 (490-6944)	3 1500 (310-4386)	3 1662 (343-4858)
30-	-	1 791 (20-4407)	2 1136 (137-4101)	6 2662 (976-5794)	12 2981 (1540-5207)
40+	-	1 1233 (31-6870)	2 1812 (219-6541)	-	8 3170 (1367-6246)
			CHATHAM		
< 10	-	-	-	-	-
10-	-	-	-	-	-
20-	-	-	-	2 3267 (395-11794)	3 3598 (742-10519)
30-	1 9387 (237-52287)	-	-	1 1246 (32-6939)	6 3398 (1246-7397)
40+	-	-	-	-	3 2129 (439-6223)
			PORTSMOUTH		
< 10	-	-	-	-	-
10-	-	-	1 2183 (55-12158)	2 3208 (388-11581)	1 1469 (37-8182)
20-	-	-	3 4496 (928-13143)	1 780 (20-4347)	2 1527 (185-5512)
30-	-	-	-	1 595 (15-3314)	5 1651 (535-3854)
40+	-	-	1 1266 (32-7049)	-	2 986 (119-3561)

TABLE A4.16: All cause mortality by occupational group and by time since first exposure.

Occupational group	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
1	4 138 (38-353)	16 102 (58-166)	19 69 (41-107)	38 153 (105-202)	35 98 (66-130)
2	3 75 (16-220)	19 135 (81-211)	19 79 (47-123)	43 92 (65-120)	103 100 (80-119)
3	3 77 (16-226)	16 99 (56-160)	27 93 (61-136)	30 82 (56-118)	42 131 (92-171)
4	61 98 (73-123)	270 104 (92-116)	329 93 (83-103)	388 94 (84-103)	599 88 (81- 95)
Unknown	-	3 104 (22-305)	5 150 (49-351)	4 103 (28-263)	7 112 (45-231)
			CHATHAM		
1	1 36 (1-201)	8 89 (38-175)	5 72 (23-169)	8 74 (32-145)	16 73 (42-119)
2	1 71 (2-397)	2 58 (7-210)	11 162 (81-290)	19 118 (71-184)	44 110 (78-143)
3	1 100 (3-557)	4 116 (32-298)	6 105 (39-229)	11 108 (54-193)	16 79 (45-128)
4	21 75 (46-114)	81 78 (61- 95)	89 84 (67-102)	140 85 (71- 99)	333 95 (84-105)
Unknown	-	-	2 318 (39-1149)	-	1 178 (5-991)
			PORTSMOUTH		
1	1 56 (1-310)	5 63 (20-147)	11 92 (46-165)	15 99 (55-163)	18 97 (58-154)
2	-	7 111 (44-228)	16 107 (61-174)	39 117 (80-154)	69 85 (65-105)
3	2 190 (23-685)	7 119 (48-245)	9 49 (22- 92)	23 80 (51-120)	32 88 (57-118)
4	29 82 (55-118)	127 88 (73-103)	220 96 (83-109)	260 83 (73- 93)	441 80 (72- 87)
Unknown	-	-	-	-	-

TABLE A4.17: Lung cancer mortality by occupational group and by time since first exposure.

Occupational group	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
1	-	4 262 (71-671)	2 70 (8-252)	2 78 (9-281)	4 104 (28-267)
2	-	-	2 95 (11-342)	6 125 (46-272)	11 99 (49-177)
3	-	3 253 (52-740)	1 40 (1-224)	2 55 (7-198)	5 143 (46-335)
4	6 100 (37-219)	23 88 (56-132)	44 124 (87-161)	43 99 (70-129)	60 84 (63-105)
Unknown	-	-	1 305 (8-1697)	-	-
			CHATHAM		
1	-	2 186 (23-672)	1 122 (3-680)	-	2 75 (9-271)
2	-	-	-	2 104 (13-374)	5 99 (32-231)
3	-	-	1 213 (5-1184)	-	2 78 (9-283)
4	3 93 (19-273)	6 51 (19-111)	9 76 (35-144)	22 109 (68-165)	38 87 (59-115)
Unknown	-	-	-	-	-
			PORTSMOUTH		
1	-	-	3 208 (43-609)	1 54 (1-299)	3 131 (27-383)
2	-	1 172 (4-959)	1 64 (2-354)	5 126 (41-294)	18 174 (103-275)
3	-	-	1 52 (1-289)	3 89 (18-262)	4 86 (24-221)
4	4 102 (28-260)	24 141 (90-210)	27 100 (66-145)	28 74 (49-107)	56 82 (60-103)
Unknown	-	-	-	-	-

TABLE A4.18: Pleural mesothelioma mortality by occupational group and by time since first exposure.

Occupational group	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
1	-	-	3 6992 (1443-20440)	3 8105 (1672-23695)	-
2	-	-	1 2491 (63-13875)	1 1247 (32-6944)	5 3484 (1129-8132)
3	-	-	-	1 1364 (35-7597)	7 12861 (5163-26493)
4	-	4 1150 (313-2945)	7 1392 (559-2868)	6 960 (352-2089)	23 2612 (1656-3919)
Unknown	-	-	-	-	-
			CHATHAM		
1	-	-	-	-	1 4082 (103-22737)
2	-	-	-	1 3960 (100-22057)	5 9066 (2937-21160)
3	-	-	-	1 5133 (130-28591)	2 7094 (858-25611)
4	1 3393 (86-18897)	-	-	3 1298 (268-3793)	7 1605 (644-3307)
Unknown	-	-	-	-	-
			PORTSMOUTH		
1	-	-	-	-	-
2	-	-	-	3 5258 (1085-15371)	3 2509 (518-7336)
3	-	-	-	-	2 3730 (451-13467)
4	-	-	5 1577 (511-3680)	3 651 (134-1903)	10 1402 9673-2578)
Unknown	-	-	-	-	-

TABLE A4.19: All cause mortality by exposure rating and by time since first exposure.

Exposure rating	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
					DEVONPORT					
< 100	67	95 (72-118)	276	107 (94-119)	254	92 (81-104)	242	92 (80-103)	310	87 (77- 96)
100-	1	155 (4-864)	30	97 (65-138)	83	89 (70-108)	93	102 (81-123)	109	82 (67- 98)
200-	-		2	47 (6-168)	34	80 (53-108)	88	92 (73-111)	100	90 (72-107)
300-	-		-		4	64 (17-164)	49	115 (82-147)	132	101 (84-119)
400+	-		1	380 (962-211741)	20	160 (4-891)	170	156 (91-250)	251	115 (93-136)
Unknown	3	112 (23-329)	15	103 (58-170)	23	110 (70-165)	14	65 (36-109)	29	94 (63-135)
					CHATHAM					
< 100	23	72 (45-107)	87	79 (62- 95)	83	92 (73-112)	89	79 (63- 95)	190	95 (82-109)
100-	-		4	70 (19-179)	14	67 (36-112)	29	89 (60-128)	61	87 (65-108)
200-	-		2	254 (31-916)	11	99 (49-177)	32	98 (64-132)	52	94 (68-119)
300-	-		-		2	190 (23-686)	17	109 (63-174)	64	116 (87-144)
400+	-		-		-		9	155 (71-294)	34	72 (48- 97)
Unknown	1	113 (31632)	2	66 (8-239)	3	124 (26-361)	2	55 (7-197)	9	125 (57-237)
					PORTSMOUTH					
< 100	30	78 (53-112)	131	90 (75-106)	186	103 (88-118)	160	79 (67- 91)	226	76 (66- 86)
100-	2	59 (41-1217)	15	94 (53-155)	44	73 (51- 94)	62	86 (65-107)	88	75 (60- 91)
200-	-		-		19	74 (44-115)	61	86 (65-108)	79	83 (65-101)
300-	-		-		5	129 (42-300)	39	116 (80-153)	95	85 (68-102)
400+	-		-		-		11	118 (59-212)	67	105 (80-131)
Unknown	-		-		2	93 (11-334)	4	202 (55-516)	5	159 (51-370)

TABLE A4.20: Lung cancer mortality by exposure rating and by time since first exposure.

Exposure rating	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
< 100	6 94 (34-204)	25 99 (64-146)	34 125 (83-167)	19 70 (42-109)	28 74 (49-107)
100-	-	4 132 (36-338)	8 89 (38-175)	15 162 (91-267)	9 64 (29-121)
200-	-	-	5 117 (38-273)	14 141 (77-236)	13 109 (58-186)
300-	-	-	2 293 (35-1057)	3 64 (13-187)	14 101 (55-169)
400+	-	1 695205 (17589->999999)	-	1 82 (2-457)	12 122 (63-214)
Unknown	-	-	1 49 (1-272)	1 44 (1-247)	4 124 (34-317)
			CHATHAM		
< 100	3 84 (17-247)	7 57 (23-118)	8 81 (35-160)	18 131 (78-207)	24 98 (63-145)
100-	-	1 163 (4-910)	1 46 (1-256)	2 54 (7-197)	6 68 (25-147)
200-	-	-	2 133 (18-541)	2 50 (6-180)	6 87 (32-188)
300-	-	-	-	1 50 (1-279)	5 73 (24-170)
400+	-	-	-	1 132 (3-735)	4 69 (19-177)
Unknown	-	-	-	-	2 231 (28-834)
			PORTSMOUTH		
< 100	4 98 (27-251)	23 138 (87-207)	25 119 (77-175)	19 78 (47-122)	28 76 (50-109)
100-	-	2 111 (13-399)	2 29 (3-103)	7 83 (34-172)	13 89 (48-153)
200-	-	-	4 130 (36-334)	4 47 (13-119)	8 68 (30-135)
300-	-	-	1 208 (5-1157)	4 94 (26-242)	16 113 (65-184)
400+	-	-	-	3 254 (52-743)	15 189 (106-311)
Unknown	-	-	-	-	1 247 (6-1375)

TABLE A4.21: Pleural mesothelioma mortality by exposure rating and by time since first exposure.

Exposure rating	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
< 100	-	4 1131 (308-2894)	3 748 (154-2185)	3 758 (156-2216)	3 631 (130-1846)
100-	-	-	3 2068 (427-6044)	3 1829 (377-5347)	6 3321 (1218-7229)
200-	-	-	4 7105 (1936-18188)	1 639 (16-3557)	9 5525 (2529-10485)
300-	-	-	-	4 7111 (1938-18205)	11 6314 (3151-11297)
400+	-	-	1 129447 (3275-721018)	-	6 5817 (2140-12706)
Unknown	-	-	-	-	-
			CHATHAM		
< 100	-	-	-	1 627 (16-3494)	1 405 (10-2253)
100-	-	-	-	1 1937 (49-10789)	2 2076 (251-7496)
200-	-	-	-	2 4119 (498-14869)	3 3946 (814-11535)
300-	-	-	-	1 5385 (136-29996)	6 8756 (3210-19058)
400+	-	-	-	-	3 6169 (1273-18035)
Unknown	1 104842 (2653-583972)	-	-	-	-
			PORTSMOUTH		
< 100	-	-	3 1167 (241-3412)	-	3 756 (156-2211)
100-	-	-	1 1140 (29-6347)	1 852 (22-4743)	1 636 (16-3543)
200-	-	-	1 2928 (74-16308)	3 2688 (555-7858)	2 1577 (191-5692)
300-	-	-	-	2 4331 (524-15635)	4 2597 (708-6649)
400+	-	-	-	-	5 7029 (2277-16406)
Unknown	-	-	-	-	-

TABLE A4.22: All cause mortality by asbestos exposure period and by time since first exposure.

Asbestos exposure (yrs)	Time since first exposure (employment)									
	0 - 9		10 - 19		20 - 29		30 - 39		40+	
	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
DEVONPORT										
< 10	7	158 (64-326)	21	123 (76-188)	15	86 (48-141)	13	72 (38-121)	31	94 (61-127)
10-	-		5	59 (19-137)	24	87 (56-129)	26	99 (64-145)	29	104 (70-149)
20-	-		5	121 (39-283)	15	117 (66-193)	35	107 (72-143)	63	124 (94-155)
30+	-		3	74 (15-217)	7	94 (38-194)	13	74 (39-126)	91	104 (83-126)
Unknown	64	94 (71-118)	290	106 (93-118)	338	91 (81-100)	416	97 (87-106)	572	87 (80- 94)
CHATHAM										
< 10	-		5	56 (18-131)	4	91 (25-232)	1	14 (0- 78)	22	129 (81-196)
10-	-		1	58 (1-325)	3	61 (13-177)	5	52 (17-122)	11	76 (38-137)
20-	-		1	173 (4-964)	1	36 (1-199)	16	122 (70-197)	20	102 (62-158)
30+	-		1	92 (2-512)	1	106 (3-589)	6	104 (38-226)	40	116 (80-152)
Unknown	24	80 (51-119)	87	81 (64- 98)	104	92 (74-110)	150	90 (75-104)	317	91 (81-101)
PORTSMOUTH										
< 10	4	157 (43-402)	13	133 (71-228)	11	105 (53-188)	11	77 (39-138)	25	74 (48-110)
10-	1	182 (5-1013)	9	161 (74-306)	21	104 (65-159)	19	79 (47-123)	26	118 (77-172)
20-	-		-		11	86 (43-154)	42	126 (88-164)	39	112 (77-148)
30+	-		1	38 (1-212)	6	78 (28-169)	29	150 (100-215)	78	84 (65-102)
Unknown	27	75 (50-109)	123	85 (70-100)	207	93 (80-105)	236	79 (69- 89)	392	78 (70- 85)

TABLE A4.23: Lung cancer mortality by asbestos exposure period and by time since first exposure.

Asbestos exposure (yrs)	Time since first exposure (employment) 0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
< 10	-	2 117 (14-424)	1 57 (1-318)	-	4 115 (31-294)
10-	-	2 89 (27-815)	7 248 (99-510)	6 222 (81-483)	6 199 (73-433)
20-	-	2 433 (52-1563)	3 214 (44-625)	3 85 (17-248)	7 127 (51-262)
30+	-	-	1 121 (3-675)	1 51 (1-281)	10 108 (52-198)
Unknown	6 98 (36-213)	24 90 (58-134)	38 104 (71-137)	43 97 (68-126)	53 76 (56- 97)
			CHATHAM		
< 10	-	2 189 (23-682)	-	-	2 96 (12-346)
10-	-	-	-	-	1 57 (1-316)
20-	-	-	-	-	2 82 (10-296)
30+	-	-	-	-	6 138 (50-300)
Unknown	3 91 (19-265)	6 51 (19-111)	11 89 (45-160)	24 119 (76-177)	36 83 (56-110)
			PORTSMOUTH		
< 10	-	3 264 (54-772)	1 79 (2-441)	1 56 (1-315)	5 119 (39-278)
10-	-	-	3 121 (25-354)	1 34 (1-192)	5 180 (58-420)
20-	-	-	2 121 (15-438)	5 120 (39-280)	6 138 (51-301)
30+	-	-	-	3 121 (25-354)	14 119 (65-199)
Unknown	4 104 (28-267)	22 133 (83-201)	26 101 (66-148)	27 76 (50-110)	51 81 (59-104)

TABLE A4.24: Pleural mesothelioma mortality by asbestos exposure period and by time since first exposure.

Asbestos exposure (yrs)	Time since first exposure (employment) 0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
< 10	-	1 3960 (100-22055)	-	-	2 4599 (557-16604)
10-	-	-	1 2427 (61-13519)	1 2254 (57-12552)	3 7703 (1589-22518)
20-	-	-	1 5773 (146-32155)	3 6230 (1285-18212)	5 6545 (2121-15276)
30+	-	-	-	1 4822 (122-26860)	7 6842 (2747-14095)
Unknown	-	3 815 (168-2384)	9 1648 (754-3127)	6 882 (323-1919)	18 2061 (1222-3257)
			CHATHAM		
< 10	-	-	-	-	-
10-	-	-	-	-	1 5527 (140-30873)
20-	-	-	-	-	1 7472 (904-26973)
30+	-	-	-	-	4 9581 (2611-24527)
Unknown	1 3228 (82-17979)	-	-	5 2058 (667-4803)	8 1827 (788-3598)
			PORTSMOUTH		
< 10	-	-	-	-	1 2207 (56-12294)
10-	-	-	1 3635 (92-20248)	1 2760 (70-15373)	1 3145 (80-17518)
20-	-	-	-	1 2124 (54-11833)	-
30+	-	-	1 10838 (274-60370)	1 4318 (109-24051)	7 6035 (2423-12432)
Unknown	-	-	3 937 (193-2738)	3 645 (133-1886)	6 898 (329-1955)

TABLE A4.25: All cause mortality by period of continuous asbestos exposure and by time since first exposure.

Continuous asbestos exposure (yrs)	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
< 10	3 276 (57-807)	6 125 (46-272)	4 97 (26-248)	4 139 (38-355)	6 90 (33-196)
10+	-	1 96 (2-533)	3 89 (18-260)	7 130 (52-268)	16 262 (150-426)
			CHATHAM		
< 10	-	5 135 (44-316)	2 123 (15-445)	2 95 (12-344)	7 274 (110-564)
10+	-	-	-	2 104 (13-374)	3 158 (33-462)
			PORTSMOUTH		
< 10	4 422 (115-1081)	3 99 (20-290)	1 51 (1-283)	3 166 (34-484)	4 83 (23-213)
10+	-	-	4 135 (37-345)	5 94 (30-219)	9 216 (99-410)

TABLE A4.26: Lung cancer mortality by period of continuous asbestos exposure and by time since first exposure.

Continuous asbestos exposure (yrs)	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
< 10	-	-	1 243 (6-1356)	-	-
10+	-	1 925 (23-5152)	1 282 (7-1571)	1 182 (5-1014)	3 462 (95-1352)
			CHATHAM		
< 10	-	2 473 (57-1707)	-	-	-
10+	-	-	-	-	-
			PORTSMOUTH		
< 10	-	-	-	-	-
10+	-	-	-	2 301 (36-1086)	6 1116 (409-2430)

TABLE A4.27: Pleural mesothelioma mortality by period of continuous asbestos exposure and by time since first exposure.

Continuous asbestos exposure (yrs)	Time since first exposure (employment)				
	0 - 9	10 - 19	20 - 29	30 - 39	40+
	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)	Obs SMR (95% CI)
			DEVONPORT		
< 10	-	-	-	-	-
10+	-	-	1 22503 (569-125343)	-	1 11541 (292-64286)
			CHATHAM		
< 10	-	-	-	-	1 28759 (728-160187)
10+	-	-	-	-	-
			PORTSMOUTH		
< 10	-	-	1 26106 (660-145413)	-	1 16327 (413-90944)
10+	-	-	-	1 11706 (296-65200)	-

APPENDIX 5.

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