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Malignant Mesothelioma in Australia, 1945–2000

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Background *Australia has maintained a total national malignant mesothelioma case register since 1980. There has been a marked increase in the incidence of mesothelioma in the last 20 years. Currently 450–600 cases are notified annually in a population of 20 million. While the history of the Wittenoom (Western Australia) crocidolite mine and its aftermath is well known, these cases comprise only 5% of the total. This study describes the incidence of mesothelioma in Australia from 1945 to 2000.*

Methods *Using register data, time trends in mesothelioma incidence were calculated. Analyses of incidence are reported by age, sex, anatomical site, and state of notification. Associations with occupational and environmental asbestos exposure histories are described. Lung fiber content measurements were made on a subset of cases.*

Results *Australia has had 6,329 cases of mesothelioma from 1 January 1945 to 31 December 2000. (A further 620 cases were notified in the period from 1 January 2001 to 31 October 2001.) Annual incidence rates for Australia per million population ≥ 20 years (1997) were: male, 59.8; female, 10.9; total, 35.4. Incidence rates have been continually increasing and are the highest reported national rates in the world. While Western Australia has the highest rate (1997 total rate, 52.8), most cases arise from the two most populous eastern states, New South Wales and Victoria. In 88% (male 90%, female 61%) of cases, a history of asbestos exposure was obtained. Exposures occurred in a wide variety of occupational and environmental circumstances. In 80% of cases with no history of exposure, TEM lung asbestos fiber counts $> 200{,}000$ fibers > 2 μm length per gm dry lung were obtained, suggesting unrecognized exposure.*

Conclusions *Australia's high incidence of mesothelioma is related to high past asbestos use, of all fiber types, in a wide variety of occupational and environmental settings. The number of cases in total is expected to be about 18,000 by 2020, with about 11,000 yet to appear.* Am. J. Ind. Med. 41:188–201, 2002. © *2002 Wiley-Liss, Inc.*

KEY WORDS: malignant mesothelioma; incidence; Australia; asbestos exposure; future predictions

INTRODUCTION

Asbestos was mined in Australia for over 100 years and Australia was the world's highest user per capita of asbestos in the 1950's. Given the ecological relationship between per capita asbestos consumption and mesothelioma incidence [Takahashi et al., 1999], it is no surprise that in the last twenty years of the 20th century, Australia has had the world's highest reported incidence of malignant mesothelioma. Australia has one of the world's most complete national surveillance systems for mesothelioma and this has

Abbreviations used: NSW, New South Wales; VIC, Victoria; QLD, Queensland; TAS, Tasmania; SA, South Australia; WA, Western Australia; ACT, Australian Capital Territory; NT, Northern Territory.

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been in operation since 1980. We will describe the history of asbestos use and the incidence of mesothelioma in Australia as a whole, rather than concentrating on the well-known Wittenoom crocidolite mining operation and township in Western Australia [Musk et al., 1992]. This study updates and enhances previous reports [Leigh et al., 1991b, 1997, 1998; Leigh, 1994].

History of Asbestos Production and Use in Australia

Between 1880 and 1889, approximately 47 tonnes of amphiboles were mined at Jones' Creek, near Gundagai, New South Wales, and between 1890 and 1899, about 35 tonnes of chrysotile was mined at Anderson's Creek, Tasmania. South Australia was the first State to mine crocidolite at Robertstown in 1916.

Over the 20th century, there was a gradual increase in asbestos production, with more chrysotile than amphiboles mined until 1939. With the commencement of mining at Wittenoom, Western Australia, in 1937, crocidolite dominated production, until final closure in 1966. New South Wales, the first State to mine asbestos, also produced the largest tonnages of chrysotile (until 1983) as well as smaller quantities of amphiboles (until 1949).

With the closing of the crocidolite mine at Wittenoom in 1966, Australian asbestos production declined to a pre-1952 level. Exports declined from 1967. Imports of chrysotile also started to decline. The earliest records of asbestos imports date from 1929. The main sources of raw asbestos imports were Canada (chrysotile) and South Africa (crocidolite and amosite). About twice as much chrysotile was imported as was mined and half as much crocidolite was imported as was mined. After Wittenoom was closed, a small amount (122 tonnes) of crocidolite was mined in South Australia. In New South Wales, the chrysotile mine at Baryulgil continued production. In 1971, the chrysotile deposits at Woodsreef near Barraba, New South Wales began to be exploited and exports of asbestos fiber expanded as production increased. This operation was open-cast with dry milling.

Australian production of asbestos fiber decreased in 1981 because of the drop in world demand for asbestos and the increased operating costs at the Woodsreef mine. This mine ceased production in 1983 when the dry milling plant could not meet dust control regulations.

Details of Australian asbestos production and imports are shown in Tables I and II. Australian asbestos (crocidolite and chrysotile) was exported to the USA, Japan, UK, and Europe. In particular, Wittenoom crocidolite was exported to the USA and Europe [The Colonial Sugar Refining Company Limited, 1956].

In addition to imports of asbestos fiber, Australia also imported many manufactured asbestos goods, including asbestos cement products, asbestos yarn, cord and fabric, joint and millboard, friction materials and gaskets. The main sources of supply were the United Kingdom, USA, Federal Republic of Germany, and Japan. Apparent consumption of asbestos, the difference between production and imports, and export is shown in Table III and Figure 1. This gives a crude estimate of overall exposure. However the export process itself, bagging, transport, and wharf labor caused much exposure. In Australia, over 60% of all production and 90% of all consumption of asbestos fiber was by the asbestos cement manufacturing industry [Hughes, 1978]. From about 1940 to the late 1960's, all three types of asbestos were used in this industry, crocidolite then being phased out. Amosite use in this industry continued until about 1983. Chrysotile was used until about 1987. Much of this industry output remains in service today in the form of

TABLE I. Asbestos Production in Australia*, 1880–1983

Years (10)	Crocidolite (tonnes)	Chrysotile (tonnes)	Amosite (tonnes)	Total (tonnes)
1880–1889	—	—	26	26
1890–1899	—	20	—	20
1900–1909	—	61	21	80
1910–1919	22	580	23	625
1920–1929	18	3,577	54	3,649
1930–1939	422	1,151	51	1,624
1940–1949	5,619	2,967	750	9,338
1950–1959	63,227	11,511	1	74,739
1960–1969	86,566	8,855	—	95,421
1970–1979	—	394,361	—	394,361
1980–1983	—	160,408	—	160,408
Total	155,874	583,491	926	740,291

*Based on figures supplied by Bureau of Mineral Resources [Hughes, 1978].

TABLE II. Asbestos Imports Prior to 1930 Until 1983 in Australia (in tonnes)*

Year	Chrysotile	Amosite	Crocidolite	Other	Total
19??–1930	—	—	—	—	2,568
1930–1940	—	—	—	—	51,554
1940–1949	—	—	—	—	139,987
1950–1959	186,855	107,509	2,778	16,938	314,080
1960–1969	329,129	81,432	—	24,112	434,673
1970–1979	388,003	87,901	—	79,683	555,587
1980–1983	64,672	8,338	—	4,188	77,198
Total	968,659	285,180	2,778	124,921	1,575,647

*Based on figures supplied by Bureau of Mineral Resources [Hughes, 1978].

TABLE III. Asbestos—Apparent Consumption* in Australia

Year	Production (tonnes)	Imports (tonnes)	Exports (tonnes)	Apparent consumption (tonnes)
1880–1889	26	—	—	26
1890–1899	20	—	—	20
1900–1909	82	—	—	82
1910–1919	625	—	—	625
1920–1929	3,649	2,568	—	6,217
1930–1939	1,624	51,554	1,196	51,982
1940–1949	9,338	139,987	2,410	146,915
1950–1959	74,739	314,080	51,413	337,406
1960–1969	95,421	434,674	44,703	485,392
1970–1979	394,361	555,587	45,523	704,425
1980–1985	160,408	104,324	9,786	154,946
Total	740,293	1,602,774	455,031	1,888,036

*Based on figures provided by Bureau of Mineral Resources [Hughes, 1978].

"fibro" houses and water and sewerage piping. By 1954, Australia was fourth in the world in gross consumption of asbestos cement products, after the USA, UK, and France, and was clearly first on a per capita basis. After World War II to 1954, 70,000 asbestos cement houses were built in the State of New South Wales alone (52% of all houses built). In Australia as a whole, until the 1960's, 25% of all new housing was clad in asbestos cement.

Australia still imports about 1,500 tonnes a year of chrysotile fiber, and about $A13.5 m worth of asbestos products a year, over half as friction material, but also fabricated yarn, fabric, jointing, gaskets, millboard, and asbestos cement products [Victorian Occupational Health and Safety Commission, 1990; NOHSC, 1999]. However, a phase out of new (chrysotile) asbestos use by 2003 is in place. Handling of asbestos in place and removal operations are subject to a strict National Code of Practice. A series of regulations adopted in the late 1970's and early 1980's by the Australian states and territories now impose exposure limits of 0.1 fiber/ml for crocidolite, amosite, and mixtures (all states and territories) and 0.1 (ACT), 0.5 (NSW,VIC) or 1.0 fiber/ml (SA, WA, NT, QLD, TAS) for chrysotile (TWA 8 hr membrane filter method light microscopy, WHO fibers). The chrysotile limit is currently under review.

Exposures in the past were very high in some industries and jobs. For example, up to 25 million particles per cubic foot were documented in asbestos pulverizors and disintegrators in the asbestos cement industry [Roberts and Whaite, 1952]. High concentrations of fibers in mining occupations were also recorded (up to 600 fibers/ml) in baggers at Wittenoom [Major, 1968].

With this background, it was almost certain that Australia would suffer a mesothelioma epidemic of a severe nature.

The first reported case, from Wittenoom, was in 1962 [McNulty, 1962]. Three more cases were reported from Victoria in 1966 [Riddell, 1966], two from Queensland in 1968 [Mortimer and Campbell, 1968], and an additional nine from Victoria in 1969 [Milne, 1969].

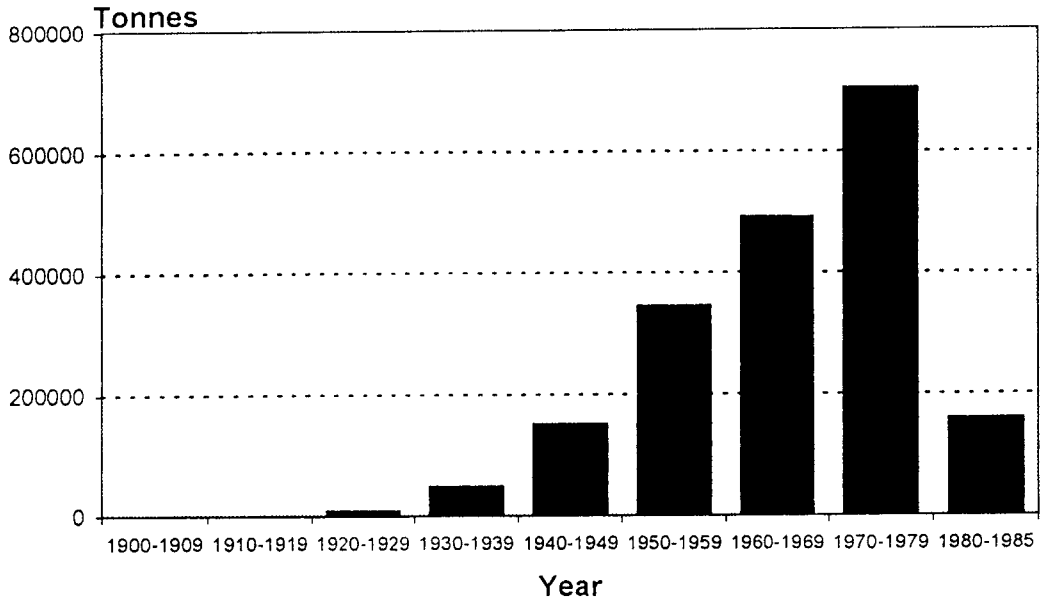


FIGURE 1. Asbestos consumption in Australia (1900–1985).

METHODS

Australian Mesothelioma Surveillance Program

The Australian Mesothelioma Surveillance Program (the Program, as it finally became known) endeavored to correct most of the weaknesses identified in the 1960's and 70's in other such surveillance schemes throughout the world, viz., under-reporting of cases, uncertain diagnosis, poor elucidation of the role of occupational and environmental asbestos exposure, and less than comprehensive coverage [Ferguson et al., 1987].

The Program began on 1 January 1980 after preliminary work from 1977. Formal voluntary notification of cases was actively sought from a network of respiratory physicians, pathologists, general and thoracic surgeons, medical superintendants, medical records administrators, State and Territory departments of occupational health, cancer registries, compensation authorities, or any other source. Notifications from other than the diagnosing physician were confirmed with him/her. After gaining the appropriate consents, a complete occupational and environmental history was obtained for each case, either from the patient or next-of-kin, by a specially trained interviewer. The history taking was non-directive, but included specific questions on asbestos exposure. These histories were coded by two occupational hygienists, who naturally could not be blinded to case status. They also discussed cases together and were thus not independent. The diagnosing pathologist was requested to provide slides and or tissue specimens. These were circulated among a pathology panel for confirmation of diagnosis. Post-mortem examination was actively sought for in every case in order to confirm diagnosis and to obtain lung tissue free of tumor for lung fiber content analysis.

Occupational and environmental exposure was classified as definite, probable, and possible, based on the subjective opinions of the two hygienists. Further grading of intensity of exposure was also very subjective in many cases and duration depended on historical recollection.

Year of presumptive diagnosis was the year in which mesothelioma was first suspected clinically. Year of definitive diagnosis was the year when pathology panel diagnosis was finalized. The panel of five members of the Royal College of Pathologists of Australasia reported individually on the diagnosis as definite, probable, possible and not mesothelioma. The level of consensus was good, with exact agreement or disagreement of only one category in 94% of cases. A scoring system of 1 (definite), 0.75 (probable), 0.5 (possible), 0 (not mesothelioma) was used and the definitive score taken as the score nearest the mean of five. Panel members also classified mesothelioma cell type as epithelial, sarcomatous, mixed, or not agreed.

Lung fiber content was assessed by a local modification of a sodium hypochlorite digestion and filter method. Both light and transmission electron microscopy (TEM) (with energy dispersive x-ray analysis) were used. Fibers were counted by asbestos type and length, but not diameter. All fiber lengths were recorded, but because of uncertainties about filter contaminants by < 2 μm fibers, only fibers > 2 μm (TEM) or > 5 μm (LM) were considered in analyses. These methods gave a sensitivity, corresponding to one fiber counted, of 15,000 fibers/g dry lung (LM) and 200,000 fibers/g dry lung (TEM). Assuming a Poisson distribution of fibers in the counting units, the upper limit of the 95% confidence interval for the population mean, given a zero count obtained, is 3.69. Thus the TEM "detection limit" is about 740,000 fibers/g [Rogers, 1984]. Reports on lung fiber content levels were sometimes used for medico-legal purposes in a fallacious way in that counts were reported as being "within the normal range" as if this excluded an occupational asbestos exposure and liability. The "normal" range was in fact taken from urban hospital patients without mesothelioma, and where fibers were counted, exposure obviously had been received (and not always environmental only as judged by fiber length distribution. It must have sometimes been from occupational sources, but no work histories were available). It was not realized until later that these patients should be treated as non-cases (referents) in a case-referent study.

Detailed analyses of Program data relating lung fiber content, cell type, tumor site, and survival [Leigh et al., 1991a]; lung fiber type and content to risk [Rogers et al., 1991, 1994]; and clinical studies [Driscoll et al., 1993] have been previously reported.

Australian Mesothelioma Register

From 1 January 1986, a less detailed notification system has operated, with a short questionnaire occupational and environmental exposure history (followed up assiduously with up to four letters per case); no pathology panel diagnosis and only sporadic lung fiber counts. In the case of NSW and WA (60% of all Australian notifications), histories are obtained from direct detailed questioning by compensation authorities or cancer registries. Only histologically confirmed cases are accepted and full reconciliation with all state cancer registries and compensation authorities is carried out. This is now known as the Australian Mesothelioma Register, but is a continuation of the Program.

Incidence rates are periodically calculated on cases notified to the Register. An annual report series is produced [NOHSC, 1989–2001]. Cases accepted by the pathology panel in the Program as definite, probable or possible are included. In this report, incidence rates have been calculated up to the end of 1997 only, due to a possible 2-year delay in notification experienced, while awaiting confirmed

diagnosis and reconciliation with the state cancer registries. Incidence rates for 1998 are available in the Australian Mesothelioma Report for 2001 [NOHSC, 2001].

RESULTS

Incidence of Mesothelioma in Australia

From 1 January 1980 to 31 December 2000, a total of 5,671 notifications had been received by the Program and Register. Between 1945 and 1979, there were 658 cases (535 male, 123 female) in Australia [Musk et al., 1989]. Thus the total number of mesotheliomas in Australia from 1945–2000 inclusive is 6,329. (An additional 620 notifications have been received in the period 1 January 2001 to 31 October 2001.) Notifications show a continuing upward trend in both males and females (Fig. 2). The notifications prior to 1982 were probably the result of initial slow acceptance of a new Program and are artificially low (1980:16, 1981:104), although a smooth curve of increasing incidence starting from the early 1960's has since been demonstrated by retrospective search (Fig. 3). The Australian population has increased from 14.5 million in 1980 to 20 million in 2001. Mesothelioma incidence rates in persons over 20 years of age have increased from 11.8 per million per year in 1982 to 35.4 per million per year in 1997 (males and females combined) (59.8 per million per year (males) and 10.9 per million per year (female)). Figure 4 shows incidence rates by time and sex. If the 1981 figure is accepted, it can be claimed that mesothelioma incidence rates have increased four- to five-fold in 19 years in Australia. Both male and female rates have increased, but the male rate is over five times the female rate. These are the highest reported incidence rates in the world [Hillerdal, 1999; Peto et al., 1999; Takahashi et al., 1999; Kjellstrom and Smartt, 2000] and equal to the Australian (NSW) incidence rates of liver cancer and in mortality terms, equal to the mortality rates of kidney cancer in males and uterine cancer in females [New South Wales Cancer Council, 2000]. Mesothelioma is no longer a "rare disease".

Figure 5 shows age-specific incidence for males and females (> 20 years) for the year 1997. Incidence increases with age up to 80 year and then falls, consistent with a cohort effect in that the oldest persons may have missed the heaviest exposure era from 1950–1970. The occurrence of cases in the < 40 year age groups indicates that exposure can occur in children. Figures 6 and 7 show age-specific incidence trends over time for males and females. Generally the time trend of increasing incidence is restricted to age groups over 50 from 1986 onwards, suggesting that occupational exposure controls introduced from the 1970's have been effective. Table IV shows notifications by state up to 31 December 2000.

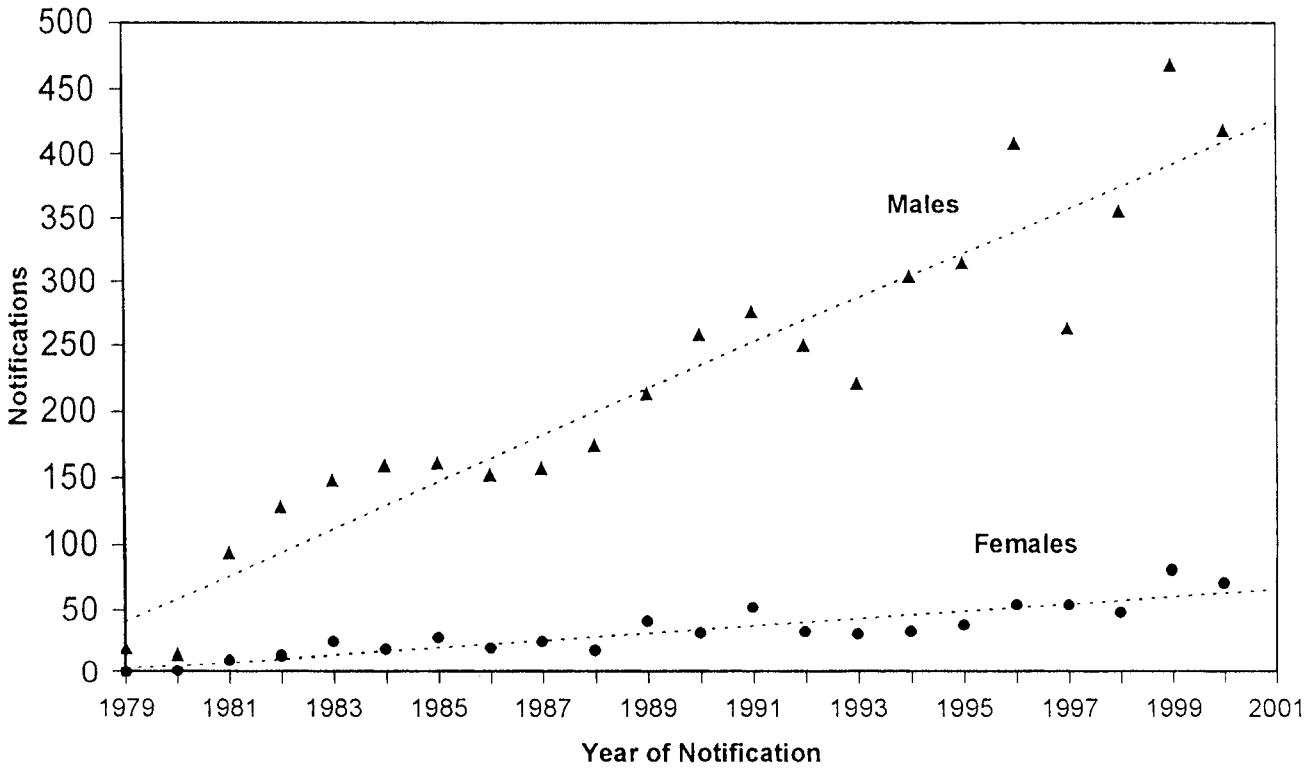


FIGURE 2. Australian Mesothelioma Register Notifications (1979–2000) (by sex).

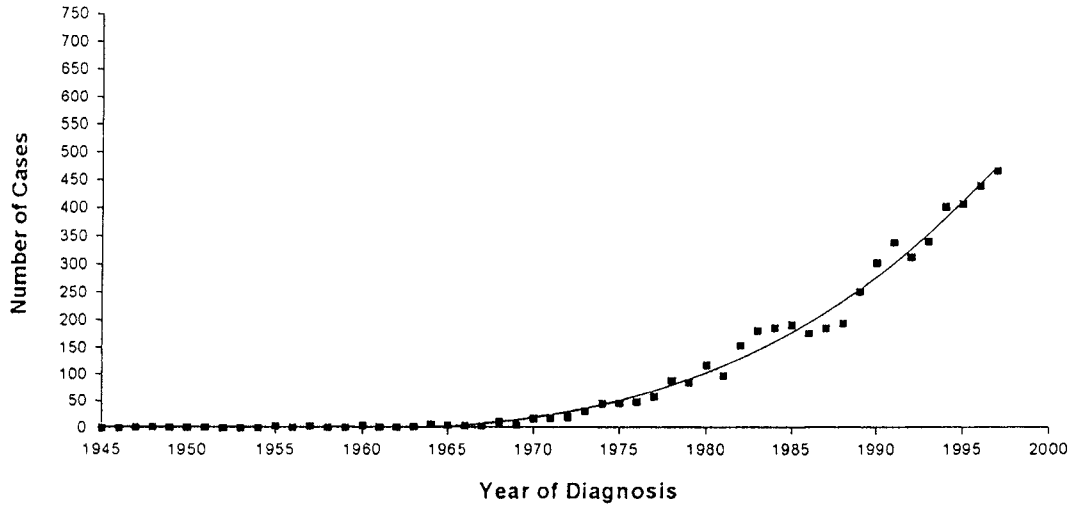


FIGURE 3. Incident cases of mesothelioma in Australia (1945–1997).

Western Australia has the highest incidence (1997 rates: total 52.8 per million/year; male 96.2, female 9.4), but contributes only 15% of the total cases. Wittenoom contributes only 5% of the Australian cases, yet is certainly the most publicized and best known internationally. Most of the cases come from the two most populous and industrialized states, New South Wales and Victoria (Table IV).

In 93.2% of all Program cases, the mesothelioma was pleural in site with 6.5% peritoneal and 0.3% in other sites. Among males, 94.3% were pleural, 5.3% peritoneal; among women, 86.3% pleural, 13.7% peritoneal. These proportions have been generally maintained in Register cases although the female peritoneal proportion has dropped to 10.4%. Of the cases that underwent pathology panel review 96% were confirmed as mesothelioma (73% definite, 17% probable, and 6% possible). The most common occupational exposures were repair and maintenance of asbestos materials (18%), shipbuilding (11%), asbestos cement production (7%), asbestos cement use (7%), railways (6%), Wittenoom crocidolite mining/milling (6%), insulation manufacture/

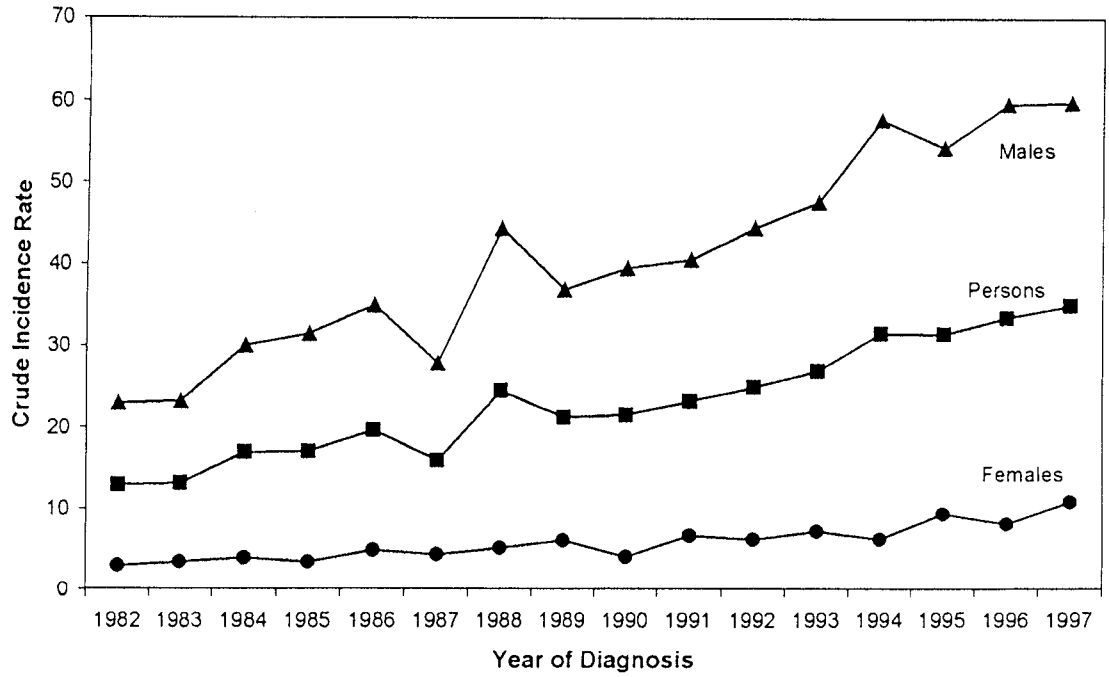


FIGURE 4. Time trend of annual incidence rate (per million population ≥ 20 years) of mesothelioma in Australia by sex (1982–1997).

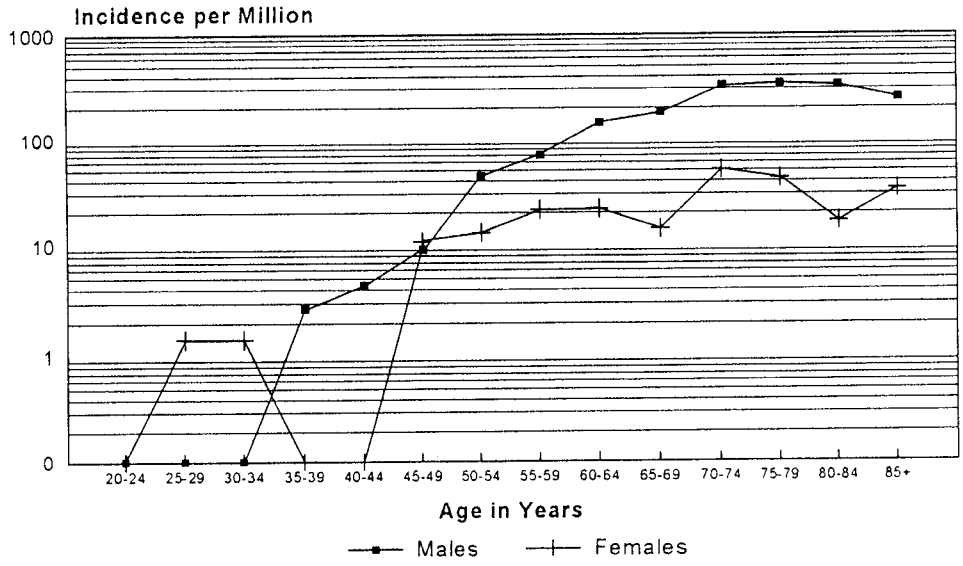


FIGURE 5. Age-specific incidence rates of mesothelioma in Australia (1997) (male and female).

installation (4%), wharf laboring (3%), power stations (3%), boilermaking (2%), para-occupational, hobby, and environmental (15%). When the earlier cases classed as "no history of exposure" were reviewed, it was found that 57 of the 203 so classified actually had history of some exposure recorded. Thus only 19% had no known history. Moreover, of this "no known history" group, 81% had fiber counts $>200{,}000$ fibers/gm dry lung detected in the lungs, 30% with more than 10^6 fibers/g >2 μm including "long" (>10 μm) fibers suggesting that nearly all cases have been exposed. Past exposure is not always recognized as such and this is more likely to be the case in females. Indeed even absence of fibers in the lungs does not negate exposure as fibers may have initiated mesothelioma and then been cleared before

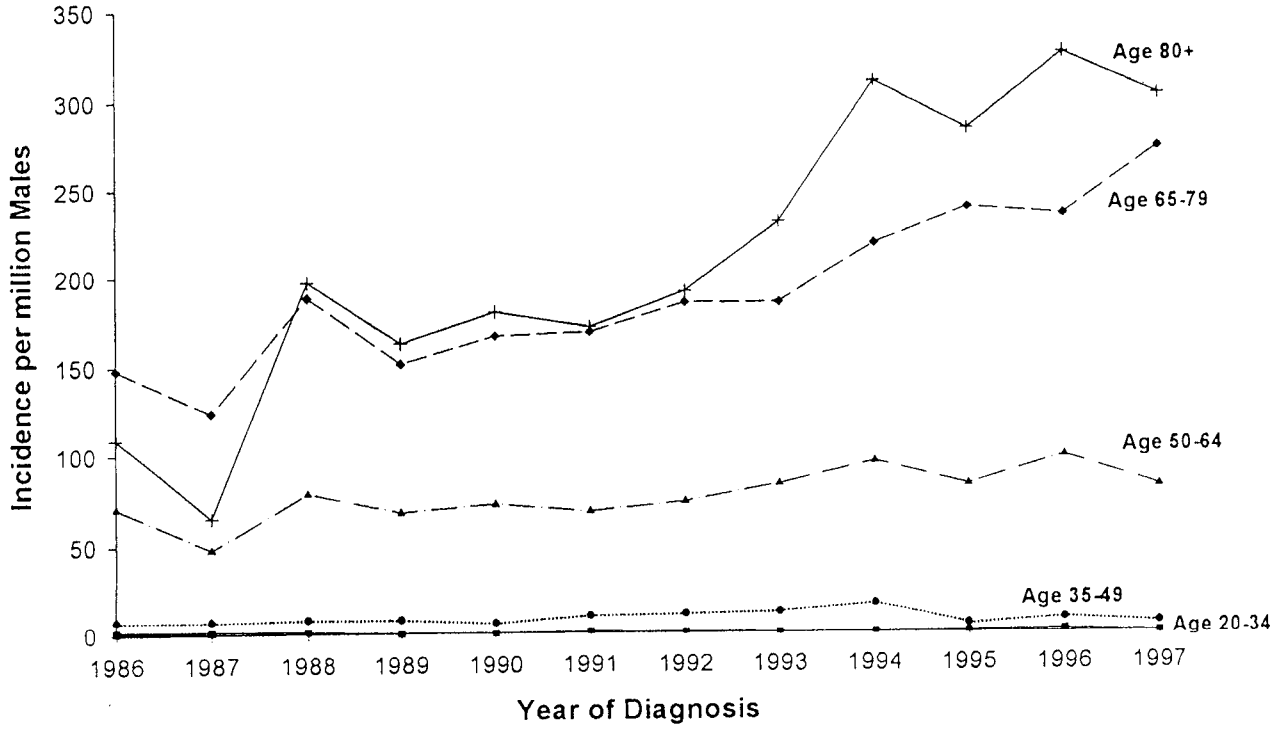


FIGURE 6. Age-specific incidence of mesothelioma in Australia (1986–1997) (male).

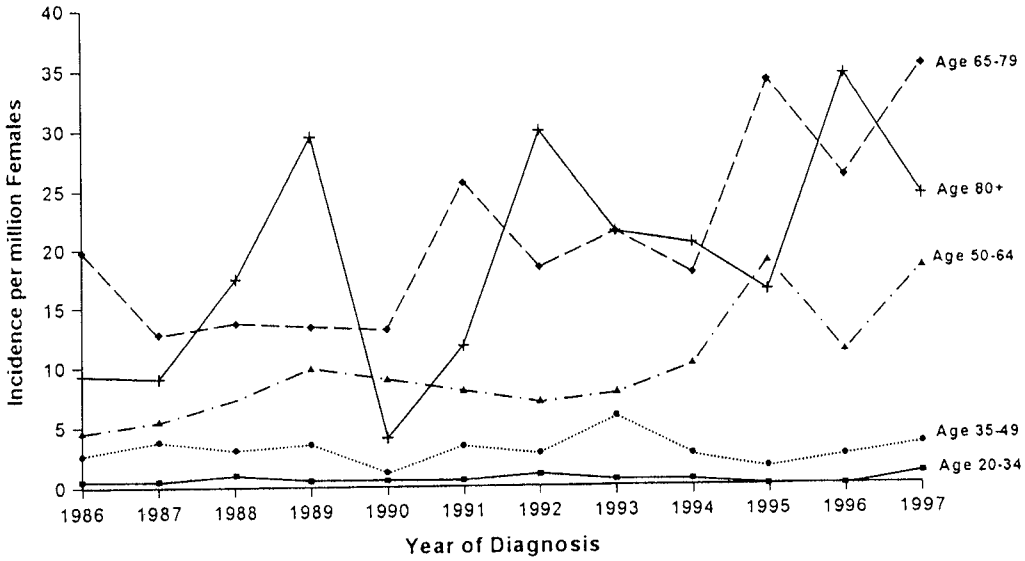


FIGURE 7. Age-specific incidence of mesothelioma in Australia (1986–1997) (female).

TABLE IV. Mesothelioma Notifications in Australia (1980–2000)

	NSW	VIC	QLD	WA	SA	TAS	NT	ACT	Total
1980	15	1	0	0	0	0	0	0	16
1981	51	3	18	22	5	5	0	0	104
1982	90	20	9	0	20	2	0	1	142
1983	53	23	26	46	19	6	0	0	173
1984	76	38	20	26	14	1	1	2	178
1985	71	39	27	30	19	1	0	2	189
1986	46	34	38	32	18	2	1	1	172
1987	54	40	26	28	32	0	0	2	182
1988	57	28	45	23	36	1	0	2	192
1989	124	25	35	44	22	3	0	1	254
1990	111	82	43	26	25	1	0	1	289
1991	105	44	46	66	55	10	0	2	328
1992	117	45	40	37	39	3	1	1	283
1993	99	34	42	47	25	5	0	0	252
1994	151	41	74	32	30	8	0	1	337
1995	124	89	49	33	43	11	1	3	353
1996	87	157	53	127	30	4	1	4	463
1997	107	32	64	82	24	5	0	4	318
1998	160	84	65	66	21	8	0	1	405
1999	252	113	73	79	20	7	0	7	551
2000	168	106	99	47	60	7	0	3	490
ALL	2,118	1,078	892	893	557	90	5	38	5,671
%	37.3	19.0	15.7	15.7	9.8	1.6	0.1	0.7	100

NSW, New South Wales; VIC, Victoria; QLD, Queensland; WA, Western Australia; SA, South Australia; TAS, Tasmania; NT, Northern Territory; ACT, Australian Capital Territory.

Asbestos Exposures as Documented in the Australian Mesothelioma Register (1 January 1986–31 October 2000)

Occupation/source of exposure	Exposed (with no other exposure) (single)	Exposed (with other exposures) (multiple)	Total exposures
[illegible]	1	—	1
[illegible]	12	14	26
[illegible]	11	3	14
Armed forces/wartime	28	16	44
Armed forces/peacetime	6	2	8
Asbestos bagging (not Wittenoom)	8	4	12
Asbestos bags—handled which had contained asbestos	8	—	8
Asbestos clothing—worn	9	4	13
Asbestos covers for cooking	3	—	3
Asbestos dwelling/fence—built/renovated	70	13	83
Asbestos dwelling—lived in	29	8	37
Asbestos products—lived near	11	2	13
Asbestos products factory—worked near	11	—	11
Asbestos mine—worked/lived near (not Wittenoom)	12	6	18
Asbestos product handled in the workplace	42	8	50
Asbestos product manufacturer—worked	106	36	142
Asbestos product—part of workplace or surroundings	26	9	35
Asbestos tailings—played on as a child	8	4	12
Asbestos/or products worker—lived with/washed clothes	42	6	48
Bakery (ovens)	3	—	3
Boilermaker/cleaner/attendant/installer/welder	79	54	133
Brake linings—made/repaired	58	19	77
Brewery	1	—	1
Bricklayer	15	4	19
Brickworks	8	3	11
Builder/Builder's laborer	185	40	225
Carpenter/joiner	224	42	266
Cement factory worker	20	1	21
Chemical engineer	1	—	1
Civil engineer	7	—	7
Concreting	5	3	8
Construction worker	12	2	14
Demolition	5	2	7
Design engineer	2	1	3
Diesel engineer	—	1	1
Dockyard worker	40	23	63
Electrical engineer	5	7	12
Electrical fitter	15	4	19
Electrical mechanic	3	—	3
Electrician	55	12	67
Electroplater	—	1	1
Engineer	26	1	27
Fireproofing	5	—	5
Firedoors	5	—	5
Firefighter	5	3	8
Fitter/turner	51	20	71
Foundry	6	2	8
Furnace	6	—	6
Glassworks/glaziers	6	—	6

TABLE V. *(Continued)*

Circumstances of exposure	Exposed (with no other exposure) (single)	Exposed (with other exposures) (multiple)	Total exposures
Industrial chemist	4	—	4
Industrial engineer	2	—	2
Instrument technician	1	—	1
Insulation	18	4	22
Jeweller	6	2	8
Laboratory technician	6	2	8
Laborer	33	15	48
Lagger	31	16	47
Lagging in workplace	24	4	28
Laundry/dry cleaners	14	5	19
Linesman	9	4	13
Locksmith	1	—	1
Machine fitter	3	1	4
Machine inspector	2	—	2
Machine operator	1	3	4
Machinist	3	—	3
Maintenance carpenter	3	1	4
Maintenance electrician	2	1	3
Maintenance engineer	3	1	4
Maintenance fitter	13	4	17
Maintenance mechanic	3	2	5
Maintenance worker	12	3	15
Marine engineer	9	7	16
Mechanical engineer	5	—	5
Mechanical fitter	7	3	10
Metal fabrication	2	—	2
Metal trades	3	1	4
Metallurgy	1	—	1
Moulder	4	—	4
Navy/merchant navy	160	64	224
Oil refinery	7	2	9
Painter/decorator	37	8	45
Panel beater	9	1	10
Papermill	3	2	5
Patternmaker	6	3	9
Pipes—handled/cut/stored/drilled	24	5	29
Plasterer	16	7	23
Plumbing	56	27	83
Power station worker	86	51	137
Pressure pack manufacturer	1	—	1
Printing	10	—	10
Radiographer	2	—	2
Railways	101	49	150
Renovations/maintenance/lagging in workplace	21	5	26
Roofing	16	4	20
Sheetmetal	14	11	25
Ships—building/repairing/on	75	57	132
Shop fitter	1	—	1
Site visits/inspections	8	7	15

TABLE V. *(Continued)*

Circumstances of exposure	Exposed (with no other exposure) (single)	Exposed (with other exposures) (multiple)	Total exposures
Smelting	1	—	1
Steelworks	13	8	21
Storeman	15	—	15
Stoves	2	—	2
Sugar mill	6	4	10
Tannery	2	—	2
Telephone technician	6	3	9
Tiler	11	1	12
Toolmaker	4	2	6
Trades assistant	17	3	20
Transporting asbestos	14	6	20
Transporting asbestos product	15	2	17
Tire factory	10	4	14
Waterside worker	94	13	107
Weighing trucks	1	—	1
Welder	22	9	31
Whitewash—Greece/Cyprus	4	1	5
Wine making (filters)	1	—	1
Wittenoom (former mining area in Western Australia)	193	51	244
Wood machinist	3	—	3

Summary of asbestos exposures	
Asbestos exposure	
Single	2,608
Multiple	400
Possible	274
No apparent asbestos exposure	457
No response to questionnaire	1,099
Total cases from 1/1/86 to 31/10/2000	4,838
Proportion of respondents with asbestos exposure	$\frac{3,282}{3,739} = 88\%$

death [Becklake and Case, 1994]. The shortest duration of exposure was 16 hr (waterside worker loading crocidolite fiber [Musk et al., 1991]). Three percent of cases had exposure of less than 3 months. According to history assessment of exposure of the first 530 cases by the two hygienists, most cases (55%) had mixed amphibole-chrysotile exposure, 13% amphibole only, 7% amphibole, plus possible chrysotile, 6% chrysotile, with possible amphibole, and 4% chrysotile only, with 15% unknown fiber type [Grimwood, 1988]. Mean latency from first exposure to presumptive diagnosis was 37.4 years [Ferguson et al., 1987]. The range of latencies was 4–75 years.

In the cases reported since 1 January 1986, when less detail of history of exposure was sought, 89.9% of males responding to questionnaire and 61.2% of females gave a history of asbestos exposure (overall 86.4%) (non-response 22% males, 30% females). The pattern of exposure history is changing, and more product, domestic, environmental and para-occupational exposure is apparent, compared to the older traditional industries. Table V shows the circumstances of exposure in cases from 1986–2000. This is a combination of occupation and industry or domestic or environmental circumstance. Exposure occurred in a wide range of occupations and industries and non-occupational settings. Some common exposure histories were: repair and maintenance of asbestos materials (13%), shipbuilding (3%), asbestos cement production (4%), railways (3%), powerstations (3%), boilermaking (3%), Wittenoom (5%), wharf labor (2%), para-occupational, hobby, environmental (4%), carpenter (4%), builder (6%), navy (3%), plumber 2%), brake linings (2%), and multiple (12%).

CONCLUSIONS

The high and increasing incidence of mesothelioma in Australia is due to high asbestos use in the past, combined

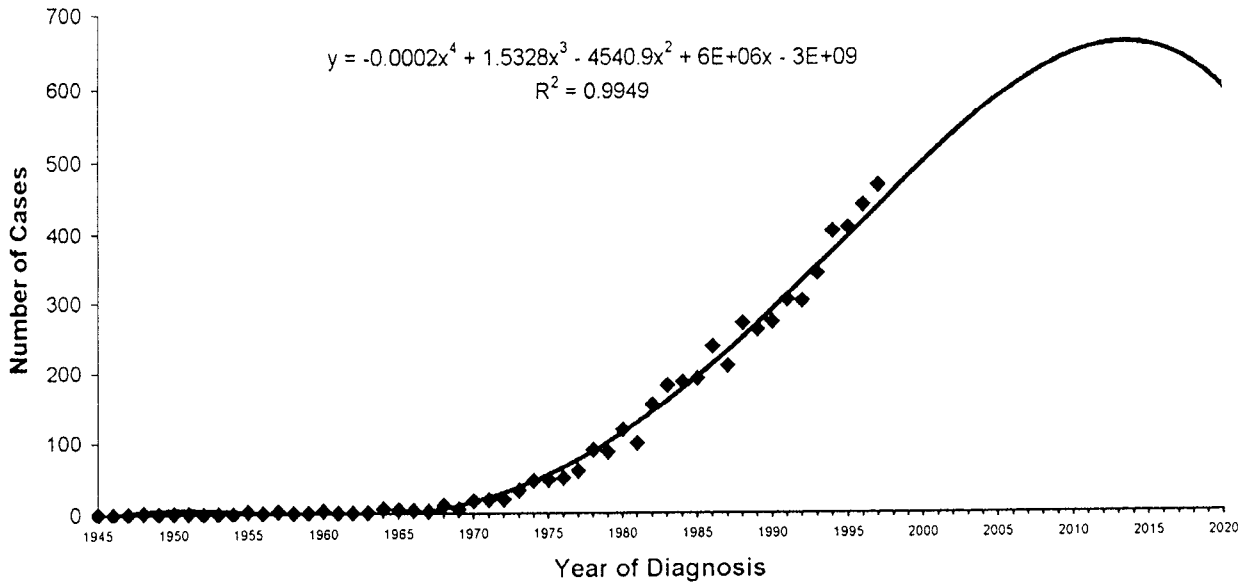


FIGURE 8. Incident cases of malignant mesothelioma in Australia (1945–1997) and extrapolation to 2020 assuming maximum at 2010.

with poor hygiene practice, relatively high amphibole use in asbestos cement products, carcinogenic potency of chrysotile, and excessive focus on Wittenoom to the exclusion of other more common exposures. There was also a reluctance to recognize the causal significance of low occupational and non-occupational exposures and a tendency to ignore or discredit the warnings of scientists, the late Irving J. Selikoff among others.

It is now apparent that nearly all human mesothelioma cases result from asbestos or erionite exposure, which may be of very small magnitude. Other proposed risk factors or co-factors, such as radiation and inflammatory lung conditions are supported only by anecdotal evidence at best. Indeed a recent very large study of mesothelioma after radiotherapy for breast cancer in US nurses is convincingly negative [Neugut et al., 1997]. The case for SV40 virus involvement as a co-factor is still unproven. While there is a dose–response relationship with asbestos exposure, a threshold has not been identified although recent studies have shown it to be less than 0.15 fiber year/ml [Rödelsperger et al., 2001]. If there is a "background incidence rate", it has been estimated to be much less than 1 per million per year [Hillerdal, 1999].

The studies relating lung fiber content and type to mesothelioma risk showed that for Australia as a whole, the greatest risk of mesothelioma was associated with exposure to crocidolite > 10 μm in length. It was difficult to assess the risks associated with chrysotile alone, due to almost universal assessed mixed exposure to amphibole and chrysotile, and the possibility of differential clearance effects. However, in a subset of cases and referents with only chrysotile found in the lungs a significant dose–response trend was found.

The expected total number of mesothelioma cases in Australia from 1945 to 2020 is estimated to be about 18,000, based on models by Berry [1991] and de Klerk et al. [1989] for Wittenoom, extrapolated for Australia as whole (assuming Wittenoom contributes 5% of cases), and direct extrapolation from the best fit to the empirical incidence curve, constrained to have a maximum value at 2010, following a 40 year latency from the time of maximum exposure (1970) (Fig. 8). This will create a heavy clinical and compensation load. Cases will arise from a large variety of occupations and workforces and environmental and para-occupational circumstances. Although classic cohorts related to insulation work and crocidolite mining will have the highest risks, occupations such as carpenters, builders, plumbers, and electricians, because of numbers employed, will generate similar case loads. The risk in brake mechanics is also elevated [WTO, 2000], consistent with chrysotile only causation.

With asbestos related lung cancer estimated to occur at a ratio of 2:1 to mesothelioma [Barroetavena et al., 1996], the expected future case load of asbestos related cancer can be expected to be of the order of 30,000–40,000 by 2020. This prediction is consistent, on a population and asbestos consumption adjusted basis, with those made for Europe [Burdorf et al., 1998; Peto et al., 1999], USA, and Japan [Consensus Report, 2000].

The various Australian state, territory, and federal government preventive, clinical, and compensatory authorities are now developing a national strategy for dealing

with this problem. The elements of this strategy are the phasing out of all new use of asbestos in Australia by the end of 2003, strict control of demolition and removal operations involving asbestos containing materials, screening programs for high risk asbestos exposed groups using spiral CT, support programs for victims of asbestos-related disease, research into better clinical treatments, prophylaxis programs where indicated, and uniform compensation standards based on the principles of the Helsinki criteria Consensus Report [2000].

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