The Case for a Global Ban on Asbestos

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BACKGROUND: All forms of asbestos are now banned in 52 countries. Safer products have replaced many materials that once were made with it. Nonetheless, many countries still use, import, and export asbestos and asbestos-containing products, and in those that have banned other forms of asbestos, the so-called "controlled use" of chrysotile asbestos is often exempted from the ban. In fact, chrysotile has accounted for > 95% of all the asbestos used globally.

OBJECTIVE: We examined and evaluated the literature used to support the exemption of chrysotile asbestos from the ban and how its exemption reflects the political and economic influence of the asbestos mining and manufacturing industry.

DISCUSSION: All forms of asbestos, including chrysotile, are proven human carcinogens. All forms cause malignant mesothelioma and lung and laryngeal cancers, and may cause ovarian, gastrointestinal, and other cancers. No exposure to asbestos is without risk. Illnesses and deaths from asbestos exposure are entirely preventable.

CONCLUSIONS: All countries of the world have an obligation to their citizens to join in the international endeavor to ban the mining, manufacture, and use of all forms of asbestos. An international ban is urgently needed. There is no medical or scientific basis to exempt chrysotile from the worldwide ban of asbestos.

KEY WORDS: asbestos, asbestos cancer pandemic, asbestos-related diseases, ban, cancer, chrysotile, controlled use, disinformation, mesothelioma, product defense. *Environ Health Perspect* 118:897–901 (2010). doi:10.1289/ehp.1002285 [Online 1 July 2010]

The Collegium Ramazzini first called for a universal ban on the mining, manufacture, and use of asbestos more than a decade ago (Collegium Ramazzini 1999). All forms of asbestos are now banned in 52 countries, and safer products have replaced many materials that once were made with it. Nonetheless, a large number of countries still use, import, and export asbestos and asbestos-containing products. In many countries that have banned other forms of asbestos, the so-called controlled use of chrysotile asbestos is exempted from the ban, an exemption that reflects the political and economic influence of the asbestos mining and manufacturing industry lobbies.

All forms of asbestos cause asbestosis, a progressive, debilitating fibrotic disease of the lungs. All forms of asbestos also cause malignant mesothelioma and lung and laryngeal cancers, and may cause ovarian, gastrointestinal, and other cancers (Straif et al. 2009). More than 20 years ago, asbestos was declared a proven human carcinogen by the U.S. Environmental Protection Agency (U.S. EPA 1986), the International Agency for Research on Cancer (1977) of the World Health Organization (WHO), and the U.S. National Toxicology Program (NTP 1980). The scientific community is in overwhelming agreement that there is no safe level of exposure to asbestos (Welch 2007; Welch et al. 2009). Moreover, there is no evidence of a threshold level below which there is no risk of mesothelioma (Hillerdal 1999).

The Asbestos Cancer Pandemic

Occupational exposures to asbestos. About 125 million people around the world are exposed to asbestos in their work environments (WHO 2006), and many millions more workers have been exposed to asbestos in years past. As noted by Stayner et al. (1997), the U.S. National Institute for Occupational Safety and Health (NIOSH) has estimated that current occupational exposures to asbestos, even at the U.S. Occupational Safety and Health Administration (OSHA) permissible exposure limit, will cause five deaths from lung cancer and two deaths from asbestosis in every 1,000 workers exposed for a working lifetime.

In 2000, an estimated 43,000 deaths worldwide resulted from malignant mesothelioma, and a much larger number of lung cancer deaths were due to occupational exposures to asbestos (Driscoll et al. 2005). Population-attributable risk for lung cancer among males exposed to asbestos ranges between 10% and 20% (Albin et al. 1999). An estimated 20,000 asbestos-related lung cancers and 10,000 cases of mesothelioma occur annually across the population of Western Europe, Scandinavia, North America, Japan, and Australia (Tossavainen 2000). The national incidence rates for mesothelioma in Australia are the highest in the world (Leigh and Driscoll 2003).

In the United Kingdom, at least 3,500 people die from asbestos-related illnesses each year, and this number is expected to increase to 5,000 in future years. Asbestos accounts for more than half of the work-related cancer deaths in Great Britain (Rushton et al. 2008). The British mesothelioma death rate is now the highest in the world, with 1,749 deaths in men (1 in 40 of all cancer deaths in men < 80 years of age) and 288 in women in 2005 (Rake et al. 2009). The projected lifetime risk of fatal mesothelioma in all British men born in the 1940s is 0.59%, or about 1 in 170 of all deaths. By 2050, there will have been approximately 90,000 deaths from mesothelioma in

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The Collegium Ramazzini is an international academic society that examines and evaluates critical public health issues in occupational and environmental health. The Collegium derives its name from Bernardino Ramazzini, the father of occupational medicine, a professor of medicine of the universities of Modena and Padua in the early 1700s. One hundred eighty clinicians and scientists from around the world are elected to membership. The Collegium is independent of commercial interests.

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Great Britain, 65,000 occurring after 2001 (Hodgson et al. 2005).

Environmental exposures to asbestos. Nonoccupational, environmental exposure to asbestos from the use of construction materials that contain asbestos is also a serious and often neglected problem throughout the world. In developed countries, large quantities of asbestos remain as a legacy of past construction practices in many thousands of schools, homes, and commercial buildings. In developing countries, where asbestos is used today in large quantities in construction, asbestos-contaminated dust is now accumulating in thousands of communities, with virtually all people burdened with asbestos fibers in their lungs and bodies (Brophy et al. 2007; Kazan-Allen 2005).

Both community-based and industrial exposures to asbestos and asbestiform fibers increase risks for mesothelioma (Pasetto et al. 2005). In a study of women residing in Canadian asbestos-mining communities, Camus et al. (1998) found a 7-fold increase in the mortality rate from pleural cancer. In California, residential proximity to naturally occurring asbestos was significantly associated with increased risk of mesothelioma (Pan et al. 2005); the risk of mesothelioma decreased approximately 6.3% for every 10-km increase in residential distance from the nearest asbestos source. Driece et al. (2009) reported that environmental exposures to asbestos waste on the surfaces of roads and yards in a contaminated community of 130,000 residents in the Netherlands result in several cases of malignant mesothelioma each year. The currently observed increase in female cases of mesothelioma in the United Kingdom, many with no occupational exposure to asbestos, suggests widespread environmental contamination (Rake et al. 2009). In a study in Libby, Montana, (Vinikoor et al. 2010), respiratory symptoms were positively associated with the frequent handling of vermiculite insulation. Residents of this mining community who were children when the mine closed experienced respiratory symptoms associated with asbestos-contaminated vermiculite exposure.

Science and Controversy

Asbestos is a general term applied to certain fibrous minerals of two configurations: serpentine and amphibole. The only type of asbestos derived from serpentine minerals, chrysotile (also known as white asbestos), accounts for 100% of the asbestos used in the world today (Natural Resources Canada 2006). Amphibole minerals include five asbestos species: amosite, crocidolite, tremolite, anthophyllite, and actinolite. Two of these are the most commercially valuable forms: amosite, or brown asbestos, and crocidolite, or blue asbestos. Other minerals sometimes containing fibers that are not defined by industry as asbestos, such as erionite, taconite, and talc, are clearly capable of causing asbestos diseases, as are certain man-made fibers, including some nanofibers (Dikensoy 2008; Ryman-Rasmussen et al. 2009; Sanchez et al. 2009). The thermal and chemical resistance and tensile strength of asbestos fibers gave rise to a burgeoning industry before their detrimental health effects—which often take years and decades to appear—became known.

The asbestos industry has relied on scientific debates over the roles of fiber types, viruses, and genetics in the development of mesothelioma to obfuscate the problem of asbestos-related disease (Castleman et al. 1998). The risk of lung cancer among workers exposed to chrysotile asbestos increases slightly with exposure to longer and thinner fibers (Loomis et al. 2009). However, efforts to use statistical models to characterize relative cancer potencies for asbestos fiber types and sizes have not been able to overcome limitations of the exposure data. Epidemiologic, experimental, and molecular evidence suggests that the arguments for the role of fiber size relative to dose, dose–response effect, and genetic susceptibility are fraught with enormous uncertainties (Terracini 2007; Tomatis et al. 2007). Scientists from NIOSH (2010) contend that the uncertainties have been so great that these estimates should not be used to determine occupational and environmental health policy until the agency can perform further research. The U.S. EPA has rejected and discontinued work on its proposed methods for quantifying potency factors for partitioned asbestos fiber types and sizes (Silverstein et al. 2009).

Concern has been raised that mesothelioma deaths might be partly attributable to poliovirus vaccines used during the 1950s and 1960s that were contaminated with simian virus 40 (SV40), a monkey virus that is tumorigenic in rodents (Leithner et al. 2006; Price et al. 2007). However, sex- and age-specific trends in pleural mesothelioma incidence rates were not consistent with an effect of exposure to SV40-contaminated poliovirus vaccine. In addition, studies reporting a high prevalence of SV40 DNA in human tumors were based on molecular assays that are prone to false-positive results (Lopez-Rios et al. 2004).

Some researchers have suggested that susceptibility to asbestos-related diseases is related to genetic differences between individuals within populations. A study of a mesothelioma clustering in Turkey advocated the role of genetic susceptibility and familial inheritance in the etiology of the disease (Roushdy-Hammady et al. 2001; Saracci and Simonato 2001). A genetic factor identified in three villages in Cappadocia, Turkey, where 50% of individuals die of mesothelioma, may contribute to the high incidence of the disease. In these villages, genetic predisposition for mesothelioma works together with erionite (Carbone and Rdzanek 2004). However, in European studies the low proportion of familial cases does not suggest the influence of a large genetic component for mesothelioma in blood relatives (Ascoli et al. 2007).

Controversies such as these have helped to make the disease experiences of asbestos-exposed workers and people in asbestos-contaminated communities invisible and uncompensated, allowing the asbestos industry to escape accountability (Braun et al. 2003). The problem extends well beyond asbestos. "Product defense papers" are commissioned by a wide range of industries seeking to blunt regulators' efforts and to defeat the cases brought by plaintiffs. Even physician-scientists reporting on hazards of asbestos have been disciplined by their politically motivated governments (Joshi et al. 2009).

Industries have the resources to seed the literature with strategic science that is less likely to be subjected to the same scrutiny routinely applied to science that is explicitly case specific (Boden and Ozonoff 2008). Many articles, published primarily in toxicology journals, are termed "product defense" science articles and are frequently sponsored by asbestos interests such as the defendants in personal injury asbestos litigation in the United States (Axelson et al. 2003; Michaels 2008). These articles are distinguished from other science papers in that they are written by scientific consultants and consulting firms that are approached and paid millions of dollars to publish and promote articles used to try to defeat liability claims (Michaels 2006). General Motors, Ford, and Chrysler sponsored the writing of review articles and meta-analyses of previously published work, and paid almost $37 million between 2001 and 2008 to scientist-consultants at ChemRisk and Exponent, Inc., for presentations of these papers at scientific meetings and expert testimony on the articles (Dietz et al. *v.* ACandS Inc. et al. 2009). These companies were defendants in damage suits brought by mechanics over their asbestos exposures and disease arising from automotive friction materials.

When there is consensus in the public health community about the health effects of a compound—particularly one that is as well researched as asbestos—government agencies and other funders are not interested in additional research that will merely demonstrate what is already known. The only people who have an incentive to continue to fund research on the health effects of chrysotile are those with an economic incentive to raise doubt about its harm. Sponsorship by parties involved in litigation leads to an imbalance in the literature (Michaels and Monforton 2007). As a result, subsequent literature reviews that report a predominance of articles reaching a certain conclusion may then mistakenly report

there is a new "consensus" in the literature when that consensus is an artifact of sponsorship (Michaels 2009). Wealthy sponsors have simply paid to have more papers published.

A Conference on Asbestos and Mesothelioma was held in May 2010 and was sponsored by both plaintiff and defense lawyers who paid scientists to come to a resort center to discuss asbestos issues (Perrin Conferences 2010). The conference discussed matters on which there is broad scientific consensus that are still questioned as part of the defense in litigation seeking to reject compensation. Such conferences can serve to perpetuate the illusion of uncertainty about issues for which there is ample evidence concerning the dangers of all forms of asbestos. Indeed, asbestos interests have a record of seizing opportunities to challenge the carcinogenicity of chrysotile, trying to create the impression that it is still a matter of legitimate scientific debate; this creates doubt about legitimate scientific findings and renders policy interventions unlikely (McCulloch and Tweedale 2008). The complex ties of the asbestos industry with international groups are numerous and problematic (Ashford et al. 2002; Castleman 2001; LaDou 2004).

Chrysotile Asbestos

Chrysotile represents nearly 100% of the asbestos produced and used worldwide today (Natural Resources Canada 2006) and 95% of all the asbestos used worldwide since 1900 (Virta 2005). There is general agreement among scientists and physicians, and widespread support from agencies in countries around the world, that chrysotile causes various cancers, including mesothelioma and lung cancer (Agency for Toxic Substances and Disease Registration 2001; American Conference of Governmental Industrial Hygienists 2001; International Labour Organization 2006; International Social Security Association 2004; National Cancer Institute 2003; NTP 2004; OSHA 1994; United Nations Environment Program 1998; WHO 2006; World Trade Organization 2001].

Early suggestions and industry reports that chrysotile might be significantly less dangerous than other forms of asbestos have not been substantiated. Although chrysotile accounts for almost all the asbestos ever used, the asbestos industry continues to claim that asbestos-related cancers are the result of the amphibole varieties (McCulloch 2006). Defenders of the chrysotile asbestos industry contend that "exposure to chrysotile in a pure form seems likely to present a very low if any risk of mesothelioma" (Gibbs and Berry 2008).

The Chrysotile Institute (Montreal, Quebec, Canada), a registered lobby group for the Quebec asbestos mining industry, takes the position that chrysotile can be handled safely (Chrysotile Institute 2008). Numerous epidemiologic studies, case reports, controlled animal experiments, and toxicological studies refute the assertion that chrysotile is safe (Bang et al. 2006; Landrigan et al. 1999; Lemen 2004b; Lin et al. 2007; Smith and Wright 1996; Stayner et al. 1996; Tossavainen 1997). These studies demonstrate that the so-called controlled use of asbestos is a fallacy (Lemen 2004a; Welch et al. 2009). Workers exposed to chrysotile fiber alone have excessive risks of lung cancer and mesothelioma (Frank et al. 1998; Li et al. 2004; Mirabelli et al. 2008).

The Canadian Cancer Society (2010), the Canadian Medical Association (2009), and the Canadian Public Health Association (2010) oppose the export of asbestos to developing countries. The National Public Health Institute of Quebec has published 15 reports, all of them showing a failure to achieve "controlled use" of asbestos in Quebec itself (Takaro et al. 2010). Pat Martin, a member of Canada's parliament and a former asbestos miner, asks, "If we in the developed world haven't found a way to handle chrysotile safely, how can we expect them to do so in developing nations?" (Burki 2010).

Some countries have banned forms of asbestos no longer in use anywhere, yet they exempt the use of chrysotile. This exemption reflects the close relationship the asbestos industry has with many governments, the lack of public health information and regulation in these countries, and the lack of compensation for asbestos victims (Castleman and Joshi 2007; Greenberg 2005; Kazan-Allen 2003). The toll in most countries still using large amounts of asbestos may never be fully ascertained or recorded.

Current Production and Use of Asbestos

Despite all that is known about the dangerous and adverse health effects of asbestos, annual world production remains at > 2 million tons [U.S. Geological Survey (USGS) 2009]. Russia is now the leading producer of asbestos worldwide, followed by China, Kazakhstan, Brazil, Canada, Zimbabwe, and Colombia. These six countries accounted for 96% of the world production of asbestos in 2007. Russia has mines rich enough in asbestos deposits to last for > 100 years at current levels of production (Encyclopedia of the Nations 2010). Most of the 925,000 tons of asbestos extracted annually in Russia is exported.

All forms of asbestos are now banned in 52 countries (International Ban Asbestos Secretariat 2010), including all European Union member countries. Nonetheless, these 52 countries make up less than one-third of WHO member countries. A much larger number of WHO member countries still use, import, and export asbestos and asbestos-containing products (WHO 2006). These are almost all countries in Asia, Eastern Europe, Latin America, and Africa. Most of the world's people still live in countries where asbestos use continues, usually with few safeguards. More than 85% of the world production of asbestos is used today to manufacture products in Asia and Eastern Europe (Virta 2005). In developing countries, where too often there exists little or no protection of workers and communities, the asbestos cancer pandemic may be the most devastating. China is by far the largest consumer of asbestos in the world today, followed by Russia, India, Kazakhstan, Brazil, Indonesia, Thailand, Vietnam, and Ukraine (United Nations Statistics Division 2009; USGS 2009).

Position of International Agencies on Asbestos

International organizations have condemned the continuing use of chrysotile asbestos. In 2006, the WHO called for the elimination of diseases associated with asbestos. The WHO supports individual countries in developing national plans to ban asbestos and eliminate asbestos-related disease, stating that "the most efficient way to eliminate asbestos-related disease is to stop using all types of asbestos" (WHO 2007). The International Labour Organization (2006) expressed concern about an evolving epidemic of asbestos-related diseases and passed a resolution to promote a worldwide asbestos ban. The World Trade Organization has accepted the conclusion that the "controlled use" of asbestos is a fallacy (Castleman 2002).

The Rotterdam Convention (2005) is an international agreement intended to regulate global trade in dangerous chemicals—chemicals that have been banned or severely restricted because of their hazards to human health or the environment. It was entered into force in 2004, and 131 nations are currently Parties to the Convention. The goal is to protect the world's most vulnerable countries—developing countries and countries with economies in transition—against importation of hazardous pesticides and other listed chemicals without their prior informed consent (PIC).

PIC is the core principle of the Rotterdam Convention. This legally binding procedure requires that governments in all countries be provided full information about the risks to health and the environment of each of the hazardous materials regulated by the Convention before importation. Annex III of the Rotterdam Convention lists the chemicals—40 in number—currently covered by the Convention's PIC requirement: 25 pesticides, 4 severely hazardous pesticide formulations, and 11 industrial chemicals.

Repeated efforts to include chrysotile asbestos under the Rotterdam Convention

have failed, not because its Chemical Review Committee has not recommended the listing of chrysotile, but because of the Convention's requirement for unanimity and as a result of the determined opposition of asbestos mining and manufacturing countries. At the 2008 conference of parties on the Convention, Kazakhstan, Kyrgyzstan, Vietnam, Russia, and Zimbabwe opposed listing chrysotile asbestos in Annex III [IISD (International Institute for Sustainable Development) Reporting Services 2008]. A few asbestos-importing countries thwarted the will of > 100 other countries.

The Need for a Universal Ban on Asbestos

The profound tragedy of the asbestos pandemic is that all illnesses and deaths related to asbestos are preventable. Safer substitutes for asbestos exist, and they have been introduced successfully in many nations. Currently, asbestos cement products account for > 85% of world consumption (Virta 2005), and in about 100 countries, asbestos-containing pipes and sheets are manufactured to be used as low-cost building materials (Tossavainen 2004). However, these asbestos cement water-pipe products could be replaced with ductile iron pipe, high-density polyethylene pipe, and metal-wire–reinforced concrete pipe. Many substitutes exist for roofing as well as interior building walls and ceilings, including fiber-cement flat and corrugated sheet products that are made with polyvinyl alcohol fibers and cellulose fibers. Virtually all of the polymeric and cellulose fibers used instead of asbestos in fiber-cement sheets are > 10 μm in diameter and therefore nonrespirable (WHO 2005). For roofing, lightweight concrete tiles can be made and used in the most remote locations using locally available plant fibers, such as jute, hemp, sisal, palm nut, coconut coir, and wood pulp. Galvanized iron roofing and clay tiles are among the other alternative materials (World Bank Group 2009).

If global use of asbestos were to cease today, a decrease in the incidence of asbestos-related diseases would become evident in approximately 20 years (WHO 2006). The asbestos cancer pandemic may take as many as 10 million lives before asbestos is banned worldwide and all exposure is brought to an end (LaDou 2004). But the world's current production of asbestos continues at an alarming rate; therefore; these figures may not reflect the true burden of this pandemic.

An international ban on the mining and use of asbestos is urgently needed. The risks of exposure to asbestos cannot be controlled by technology or by regulation of work practices. Scientists, physicians, and responsible authorities in countries allowing the use of asbestos should have no illusion that "controlled use" of chrysotile asbestos is an effective alternative to a ban on all use of asbestos (Castleman 2003; Egilman and Roberts 2004). Even the best systems of workplace controls cannot prevent occupational and environmental exposures to products in use, or exposures to asbestos discarded as waste. Safer substitute products are in use in countries all over the world where asbestos is banned.

To protect the health of all—now and in future environments—the Collegium Ramazzini again calls on all countries of the world to join in the international endeavor to ban the mining, manufacture, and use of all forms of asbestos.

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Malignant mesothelioma in Pilbara Aborigines

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Abstract: *Malignant mesothelioma occurred in a female Aborigine after environmental exposure to asbestos. All known cases of the disease in Aborigines in Western Australia were reviewed; all occurred in Pilbara residents. Most were exposed while involved in the transport of asbestos from the Wittenoom crocidolite operation. Based on recent estimates of the size of the Aboriginal population in the Pilbara region, their incidence of this disease (250 per million for ages 15 and over) is one of the highest population-based rates recorded.* (*Aust J Public Health* 1995; 19: 520–2)

Australian Aborigines and Torres Strait Islanders are currently the least healthy identifiable subpopulation in Australia.[1] For all major disease categories, death rates for Aborigines are much higher than for other Australians, and life expectancy is about 20 years less. Although the biggest differences occur in circulatory diseases, infectious diseases, and injuries, neoplasms still rank as the fourth most common cause of death.[1] The causes of these neoplasms are likely to be external factors such as smoking. A notification to the Mesothelioma Register of Western Australia has highlighted a problem that is likely to increase.

Case report

A 55-year-old Aboriginal woman was referred for investigation after she presented to the Aboriginal Medical Service in Roebourne with a two-month history of cough, increasing breathlessness and progressive left sided chest pain. She had physical signs of a left pleural effusion, which was confirmed with a chest X-ray. She was given antibiotic treatment but the effusion increased over six weeks. She had a history of obesity, non-insulin-dependent diabetes mellitus, hypertension, and an iron deficiency anaemia, for which she was receiving oral hypoglycaemic and iron therapy.

Physical examination and plain chest X-ray confirmed that she had a large left pleural effusion. She had a blood haemoglobin level of 13.6 g/dL, white cell count 8.9×10^9/L and an electrolyte sedimentation rate of 23 mm/hour. Plasma creatinine, urea and electrolytes were normal, but liver function tests showed an elevated level of alkaline phosphatase (156 IU/L). Her random blood glucose level was 10.8 mmol/L and glycated haemoglobin was 9.4 per cent, suggesting poor diabetic control. Aspirated pleural fluid was lightly blood-stained, with a protein content of 4.5 mg/L and malignant mesothelial cells which contained glycogen but no epithelial mucin (periodic acid-Schiff positivity removed by diastase) and which were negative to the carcinoembryonic antigen immunoperoxidase reaction. Pleuroscopy revealed multiple white nodules 2 to 3 mm in diameter on both visceral and parietal pleural surfaces. Biopsy of these nodules showed infiltrating malignant pleural mesothelioma of epithelial type. She was discharged from hospital with no specific therapy and died early in 1995.

Environmental history

The woman had been born in a mission about 100 km from Port Hedland. When she was a child, her family moved around the Pilbara where her father worked as a stockman. From the age of 16 she lived with her grandmother for about three years at a mission near Wittenoom and was then a cook at a nearby station, where she lived in open camps and tin sheds about 50 km from the homestead. During her time at the station she visited Wittenoom about once a fortnight for supplies, sometimes attending the races and sleeping overnight. During this period she may also have been exposed to crocidolite at Yampire Gorge (near Wittenoom), which she recalled visiting occasionally. She then spent about five years living in the Onslow area in open-air camps, and for the next 15 years she lived in the Pilbara region and worked intermittently as a cook on sheep and cattle stations. After that, she lived about 20 kms outside Wittenoom, visiting the town periodically to buy stores.

Discussion

This Aboriginal woman, who developed malignant pleural mesothelioma, had only occasional and transient nonoccupational exposure to asbestos during a long period while living in the Pilbara region nearby

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to where both chrysotile (white asbestos) and crocidolite (blue asbestos) were mined. Her first exposure to both forms occurred about 38 years ago.

The records of the Occupational Respiratory Epidemiology group and the Western Australian Mesothelioma Registry contain information on 11 other Aborigines who contracted mesothelioma after 1975 in Western Australia (Table 1). All 12 Aborigines lived in the Pilbara region of Western Australia, but this was the first Aboriginal female to develop the disease. Eight of these people are known to have worked with crocidolite at Wittenoom or Point Samson in the past, and one was a child living near Point Samson where Wittenoom asbestos was loaded on to state ships for transport to Perth. He had played in the storage sheds on the wharves as a child of 10 or 11 years of age. The other two had lived at Wittenoom or nearby but had no documented occupational exposure to asbestos.

Mesothelioma due to nonoccupational exposure to mineral fibres has been well established for many years and has been reviewed elsewhere.[2] The disease has been reported among people exposed through naturally occurring sources of asbestiform minerals, through household contact with asbestos workers and through proximity to asbestos mines or production plants.

Crocidolite has probably caused more mesothelioma than any other of the commercially available forms of asbestos; workers who have been exposed to crocidolite have the highest rates of any occupationally exposed groups.[3] A follow-up study of nearly 5000 people who had lived at Wittenoom but had never worked for the mining company, Australian Blue Asbestos, found 24 cases of mesothelioma to the end of 1992.[4]

The practice of transporting the asbestos from Wittenoom in hessian bags was the cause of at least 8 of the 12 cases reported here and was carried out throughout the life of the mine. Such practices were explicitly banned in asbestos-processing factories by the British Asbestos Regulations of 1931, and strongly criticised again in the Chief Inspector of Factories Report for 1949.[5]

Previous studies of workers at Wittenoom showed that the risk of mesothelioma following crocidolite exposure is dose-dependent and increases exponentially with time from first exposure, indicating that many more cases will continue to occur.[6] It has also been predicted that at least 250 cases of mesothelioma among former Wittenoom workers,[7] or 366 further cases among workers and former residents combined could still arise,[8] even though the median duration of exposure to crocidolite in the workforce of nearly 7000 people was only four months and the median cumulative exposure only 6 fibres per ml-years.[9] The number of Aboriginal people inhabiting the region of Wittenoom and Point Samson over the years, who may have had similar exposures to the reported patient or to former workers or residents has never been documented and the possibility of continuing exposure still exists. The size of the population that received transient exposure during periods of casual employment in the industry is also unknown, as employment records for Aborigines were never made. It is therefore impossible to predict what future incidence of mesothelioma may occur in the people of this region.

Table 1: Aborigines known to the Western Australian Mesothelioma Registry, 1961 to 1994

Diagnosis			Exposure		
Age	Sex	Year	Year	Type	Place
61	M	1975	~1955	Lumping	Roebourne, Pt Samson
48	M	1980	~1955	Trucking	Wittenoom, Pt Samson
54	M	1980	~1956	Trucking	Wittenoom, Pt Samson
45	M	1985	1958	Trucking	Wittenoom
50	M	1986	~1940	Father was crocidolite prospector	Wittenoom
75	M	1987	1965	Pipe-laying	Wittenoom
79	M	1989	~1955	None specific	Wittenoom, Roebourne
52	M	1990	1960	Lumping	Pt Samson
40	M	1992	1962	Playing in storage sheds	Pt Samson
55	M	1993	~1957	Lumping	Pt Samson
55	F	1993	~1957	Regular visits to Wittenoom	Wittenoom
58	M	1994	~1957	Lumping	Pt Samson

The total population of Aborigines in Western Australia is about 40 000, of whom about 6000 live in the Pilbara region where all those affected lived at the time of their exposure. Over the last six years, therefore, the crude incidence rate of malignant mesothelioma has been about 250 per million person–years for those aged 15 and over, 5 to 10 times higher than any of the other population-based rates,[3] apart from the extremely high rate due to exposure to erionite in Karain, Turkey.[10] Unfortunately, it is probable that the risk of mesothelioma resulting from past exposures will continue to increase with time. Studies to establish the likely extent of the problem appear indicated. These should include registration of people thought likely to have been exposed, fibre-counting of any available lung tissue specimens, and estimation, possibly through simulation, of likely airborne exposure levels in and around Wittenoom and Point Samson over the years.

Addendum

A further case of malignant mesothelioma in a 60-year-old Aboriginal man in Western Australia was confirmed by needle biopsy in early 1995. He had worked on a sheep station near Wittenoom and visited the Wittenoom mine workshop for repairs to station machinery for a few hours on about 40 to 50 occasions, staying in the town overnight a few times. He had also been a bulldozer driver in open-cut tin, gold, manganese and tantalite mines in the area. Plain chest X-ray showed a mass at the right lung base, extending into the chest wall and the posterior mediastinum. He developed progressive compression of his oesophagus and inferior vena cava and died 14 weeks later.

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Australian dietary targets in 1995: their feasibility and pertinence to dietary goals for 2000

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Abstract: *To ascertain whether the diet of young students in health-related courses conformed to Australian dietary targets for 1995 and to identify nutritional needs in view of dietary targets for 2000, 246 undergraduate students analysed their weighed diet for three or five days. Eight of ten male participants were meeting the dietary goals for 1995 for fibre and fat intake and six of ten were meeting the goals for 2000. More female participants were meeting the goals for fat, but less than half of the females were meeting the 1995 goals for fibre. Some 84 per cent of females, but only 29 per cent of males, were meeting the 1995 dietary goals for sodium. Few participants derived 10 per cent or less of their energy from saturated fat or 10 per cent of their energy from polyunsaturated fatty acids. Substantial numbers of participants failed to meet the recommended daily intakes of zinc, calcium, magnesium and iron (females). Education of specific subgroups and modifications to food production and processing are proposed.* (*Aust J Public Health* 1995; 19: 522–4)

In 1987, the Australian Department of Health published nutrition targets for improving the health of Australians.[1] The dietary goals for Australia include a reduced consumption of fat, sodium, alcohol and refined sugar and an increased intake of fibre. The taskforce believed that such targets were realistic, and the short-term viability of such a program has been confirmed.[2] Despite deliberations about implementing these guidelines,[3] the adequacy of food choices alone to achieve a substantive change in Australian dietary health has been questioned.[4] Production, processing and distribution, and nutrition along with consumption, all deserve attention in the endeavour to enhance healthy diets.[6] Although a population-based strategy introducing health promoting modifications in food production and processing may achieve a more pervasive outcome than consumer education, an educational approach is consistent with the ethos of self-determination in health care.

This paper reports on how well a convenience sample of 'informed' participants conformed to the 1995 dietary goals of the Australian health care system, identifies sex differences in the dietary habits of participants and recognises the potential for precipitating an untoward dietary imbalance by adherence to dietary goals.

Methods

Analysis of the diets of 126 males and 120 females between the ages of 18 and 35 with an interest in health matters was undertaken in the 10 months before 1995, the due date for the first Australian dietary target. During 1994, students undertaking bachelor-level courses with a health orientation were required to perform, for three to five days, a 'weighed personal dietary analysis' as part of their undergraduate nutrition course. All food and drink consumed during the study period was weighed using a digital scale. Data were analysed using the Diet/3 nutrient-analysis Xyris software. This system is based upon the 1992 NUTTAB database and pro-

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Smoking, exposure to crocidolite, and the incidence of lung cancer and asbestosis

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Abstract

In 1979 all former workers from the Wittenoom asbestos industry who could be traced to an address were sent a questionnaire to determine smoking history. Occupational exposure to crocidolite was known from employment records. Of 2928 questionnaires sent, satisfactory replies were received from 2400 men and 149 women. Eighty per cent of these had smoked at some time and 50% were still smoking. Since that time 40 cases of lung cancer and 66 cases of compensatable asbestosis have occurred in this cohort. The incidence of both lung cancer and asbestosis was greatest in those subjects with the highest levels of exposure to crocidolite and in ex-smokers. Statistical modelling of the joint effects of these exposures on the incidence of each disease indicated that crocidolite exposure multiplied the rates of lung cancer due to smoking and that smoking had no measurable effect on the rates of asbestosis. There was also some evidence that the incidence rate of lung cancer is falling with time.

Crocidolite was mined at Wittenoom in Western Australia from 1937 until 1966. From 1943 until 1966 the principal leases were mined by a single company, Australian Blue Asbestos (ABA), which employed over 6000 people, mostly for short periods. The employment records of the company have formed the basis of a continuing cohort mortality study of the workforce.[1 2] The workforce has been shown to have raised incidence and mortality from asbestosis, lung cancer, and malignant mesothelioma. The increases in incidence of these three known asbestos related diseases have shown clear associations with both level and duration of exposure to asbestos.[1 2 3] Higher than expected mortality from alcohol related diseases and other smoking related diseases has also been recorded as well as excesss mortality from tuberculosis, attributed to the migrant state of the workers.[1]

Cigarette smoking and exposure to one or other form of asbestos are both known to cause lung cancer and most evidence suggests that their effects are multiplicative in its production.[4 5] This is to be expected because smoking and asbestos probably act at different stages in the process of carcinogenesis.[6] The combined effects of smoking and exposure to crocidolite alone on risk of lung cancer have not previously been shown prospectively except by Baker[7] in an earlier study on the Wittenoom workers. That study used only rough groupings of worksite to estimate exposure to crocidolite and was restricted to cases of lung cancer arising in Western Australia.

It has long been known that diffuse interstitial pulmonary fibrosis can be caused by asbestos and that the occurrence of this disease and its severity have declined consistently since the introduction of dust suppression methods throughout industries in which asbestos has been used.[8] Although asbestosis does not occur in the unexposed general population, radiographic abnormalities consistent with asbestosis have been found in 10% or more of members of the families of amosite factory workers and shipyard workers[9 10] and 5% to 30% of occupational cohorts mostly exposed to mixtures of types of asbestos.[11 12] Definite exposure reponse relations between both level and duration of exposure to asbestos and presence of definite radiographic abnormalities have been shown by many authors,[11-14] and similar relations exist for mortality from asbestosis. Many, but not all, studies[15] have also shown that smoking increases the prevalence of abnormal findings on *x* ray films in populations of asbestos workers,[16-20] that smoking may increase the rate of progression of parenchymal asbestosis,[19 21] and that prevalence of abnormal radiographs is also related to age.[13 17 20] One longitudinal study has also shown independent effects of cumulative exposure, smoking habit, and age on the incidence of abnormality.[22]

The Wittenoom cohort provides a unique opportunity to study these exposure response relations for subjects exposed almost exclusively to crocidolite.

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Previous studies on the Wittenoom cohort have shown positive effects of cumulative exposure on the prevalence[23] and the severity of radiographic asbestosis,[24] on mortality from asbestosis,[1 3] and on the rate of progression of established asbestosis.[25]

The aim of this study was to examine the separate and combined effects of smoking and exposure to crocidolite on the incidence of lung cancer and asbestosis in workers exposed only to crocidolite.

Subjects

There were 6500 known employees of the Australian Blue Asbestos Company between 1943 and 1966. A total of 2928 of these workers were traced in 1979 and were sent a questionnaire on smoking and other occupational exposures. Two thousand four hundred men replied. These constitute the cohort for this study. Follow up of this cohort has been maintained since that date.

Methods

Methods of follow up, ascertainment of vital state, and estimation of levels of exposure to crocidolite have been described in full previously.[1 2] Briefly, demographic and basic exposure data were obtained from employment records supplemented, when incomplete, by records of the Perth chest clinic, which performed compulsory pre-employment and subsequent periodic examinations by chest *x* ray film of all employees, and by records of the Western Australian Mineworkers Relief Fund, a compulsory benevolent fund to which the company paid monthly subscriptions deducted from each employee.

Ascertainment of vital state in this cohort of questionnaire responders has been chiefly carried out by periodic mailing since 1979. Where necessary this information has been supplemented by access to death registries in all States of Australia from 1 January 1981 to 31 December 1986, all State and Commonwealth electoral rolls current in 1986 or later, and all Australian telephone directories current in 1986 or later.

A survey of airborne respirable fibres of crocidolite greater than 5 μm in length was carried out at various work sites at Wittenoom in 1966.[26] These measurements were used to obtain estimates of fibre concentrations for all 87 job categories in the various worksites. A subjective ranking of the degree of dustiness of these jobs, both before and after September 1957, when a less dusty mill commenced operation, had been provided by an ex-superintendent of operations at Wittenoom and verified by the industrial hygienist who conducted the 1966 survey.[1] The subjective ranking of each job was combined with the results of the 1966 survey of fibre concentrations to provide an estimate of dust exposure for every job at Wittenoom. The scale was used to estimate fibre concentrations in earlier periods and in jobs not included in the survey.

This study was designed as a case-control study within the cohort of 2400 people.[27] The incident cases of asbestosis and lung cancer were identified by a date of diagnosis after the date of returning the questionnaire up to December 1986. The date of diagnosis of asbestosis for each subject was established through the records of the Pneumoconiosis Medical Panel of Western Australia, which handles workers compensation claims for dust diseases in this State and from death certificates obtained from the Registrars General throughout Australia. The date of diagnosis of lung cancer for each subject was established through the Pneumoconiosis Medical Panel and death certificates as well as through all cancer registries in Australia. Each case of asbestosis and lung cancer was matched to all subjects from the cohort who were not known to have developed asbestosis, lung cancer, or malignant mesothelioma by the year of diagnosis of the case, who were the same age (exact year), and who were known to be alive in the year of the case's diagnosis of asbestosis or lung cancer. Thus subjects could be controls for more than one case and some cases could be controls for other earlier cases. Variables compared between cases and controls were smoking state, average intensity of exposure to crocidolite (fibres (f)/ml), duration of crocidolite exposure (days), time since first exposure (years), year of birth, year of starting work (before 1950, 1950–6, after 1956), cumulative exposure to crocidolite and work site (mill only, mill and elsewhere (including mine and unknown), mine only, mine and elsewhere (not including mill), neither mine nor mill, and unknown.

Smoking habit was categorised as that given on the questionnaire and was assumed not to change throughout the study. For ex-smokers it was therefore assumed that the time since they had last smoked was the time between giving up and the time of diagnosis of their disease or that of the matched case.

Statistical methods

The frequencies of the variables of interest in the matched sets of cases and controls were compared using conditional logistic regression analysis to estimate odds ratios by use of the computer program EGRET.[28] For tabular presentations, because cases were matched to sets of controls of varying sizes, variables were averaged across each control set before taking the overall average.

The odds ratio was taken to approximate the relative risk or rate ratio. Interaction odds ratios were also estimated to examine the goodness of fit of the multiplicative model to the data. A poor fit would be

suggested by interaction odds ratios that were different from one; terms less than one showing that the relative effect of asbestos was less in smokers than non-smokers (or conversely, that the relative effect of smoking was less in those exposed to asbestos than those unexposed) and that the combined effects would be likely to be additive, with terms greater than one implying a combined effect that would be more than multiplicative, as for example in initial analyses of American insulation workers where no lung cancers occurred in non-smokers.[29]

Other workers have generally expressed the relative risks of lung cancer for asbestos workers in terms of their cumulative exposure (the product of duration of exposure and level of exposure summed over all different jobs, sites, etc).[30 31 32] Although this may be an inappropriate measure from the theoretical point of view[33] and is certainly inappropriate for mesothelioma,[3 8] analogous to the inappropriate use of pack-years of cigarette-smoking when assessing risk from tobacco,[34] it has received widespread use and often appears to fit available data better than the separate terms. Accordingly, in this study, different measures of exposure were used in alternative models and the relative goodness of fit of these non-hierarchical models was assessed using differences in the residual deviance.[35]

Results

LUNG CANCER

There were 40 cases of lung cancer and 1799 matched controls. The mean duration of exposure to crocidolite for cases of lung cancer was nearly twice that of the control subjects (table 1). The intensity of exposure to crocidolite and time since exposure did not appear to be different between the cases and controls. The proportion of subjects who had never smoked or who had stopped smoking more than 10 years before replying to the questionnaire was lower in the control subjects than in those with cancer, whereas the proportion of subjects who had recently stopped smoking or who continued to smoke was greater in the lung cancer cases than in the controls.

The relative risk associated with exposure to asbestos was slightly greater in smokers than in non-smokers (table 2). The larger effect of exposure to asbestos in smokers was not significantly different from previous findings of a likely multiplicative model, for which the relative effect of asbestos would be expected to be one.

When all the variables listed in table 1 were included in the same model the relative risk of lung cancer in the current smokers was roughly five (table 3). This was slightly greater in heavier smokers. It rose to 13·9 in subjects who had stopped smoking within six years of the date of diagnosis of the index case and then fell to 7·2 in those who had stopped smoking 6–10 years previously. It then fell to 1·3 with more than 10 years since stopping smoking. Adjustment of the relative risks for both smoking and exposure to crocidolite made no difference to either set of relative risks indicating little or no confounding. These relative risks were all significantly greater than one, except for ex-smokers of longer than 10 years duration. There was no significant interaction

Table 1 Lung cancer: asbestos and cigarette smoke exposure variables

	Cases (n = 40)	*Controls (n = 1799)*
Crocidolite exposure state:		
Mean duration (days)	726	450
Mean intensity (f/ml)	28	24
Mean cumulative (f/ml-years)	49	26
Mean time since first exposed (y)	25	28
Smoking state (%):		
Never smoked	7·5	25
Ex >10 y	7·5	20
Ex 6–10 y	15	8
Ex <6 y	12·5	4
Current <20/day	25	19
Current ⩾20/day	32·5	24

*Mean of the mean of each set of controls.

Table 2 Lung cancer: relative effects of smoking and asbestos exposure

	*Asbestos exposure**			
	Non-smokers†		*Smokers*	
	High	*Low*	*High*	*Low*
Cases	4	2	25	9
Controls	357	399	521	522
Relative risk				
Matched analysis (95% CI)	1·90 (0·62–5·85)		2·62 (1·18–5·79)	
Relative asbestos effect (NS:S)	0·73 (95% CI, 0·11–5·80) (p = 0·74)			

*Low exposure here is less than 10 f/ml-years.
†Non-smokers include ex-smokers of longer than 10 years.

Table 3 Lung cancer: relative risks for combined cigarette smoking and asbestos exposure, all variables included together in a single model

	Relative risk (95% CI)
Asbestos exposure:	
Total cumulative (per $\log_e$(f/ml-years))	1·40 (1·12–1·75)
Time since first exposed (>25 y)	0·48 (0·24–0·98)
Smoking state:	
Never	1·0
Ex >10 y	1·30 (0·25–6·90)
Ex 6–10 y	7·21 (1·63–31·9)
Ex <6 y	13·9 (2·84–67·7)
Current <20/day	4·49 (1·17–17·2)
Current ⩾20/day	5·76 (1·51–22·0)

term between smoking and exposure to crocidolite ($p > 0.4$ in all cases) showing that the multiplicative model fitted the data reasonably well.

The relative risk of lung cancer was significantly related to the duration of crocidolite exposure and also to cumulative exposure to crocidolite. When added to a model including duration of exposure, the effect of intensity of exposure, although small, was almost significant ($p = 0.10$). Relative risks were 1·18 (95% confidence interval (95% CI) 1·04–1·33) per year of exposure and 1·08 (95% CI 0·97–1·20) per 10 f/ml. An apparently better fit to the data was, however, obtained using log (cumulative exposure) in place of the separate terms for intensity and duration of exposure with the residual deviance lower by 3·3 with one less parameter estimated.

The only other significant variable was a term for years since first exposed. The best fit for the data here, indicated by examining the effects of the variable after categorisation, was a single term with a lower relative risk of 0·48 (95% CI 0·24–0·98) for those first exposed more than 25 years ago (table 3).

Table 5 Asbestosis: relative risk for combined cigarette smoking and asbestos exposure, all variables included together in a single model

	Relative risk (95% CI)
Asbestos exposure:	
Total cumulative (per (f/ml-years))	1·033 (1·021–1·045)
Total cumulative^2 (per (f/ml-years)^2)	0·999 (0·999–1·000)
Site of work:	
Neither mine nor mill	1·0
Mill only	2·71 (1·13–6·20)
Mine only	6·99 (3·43–14·23)
Mill and elsewhere	6·45 (2·66–15·65)
Mine and elsewhere	7·60 (1·22–47·43)
Unknown	4·69 (2·18–11·32)
Smoking state:	
Never	1·0
Ex > 10 y	0·99 (0·44–2·23)
Ex 6–10 y	0·95 (0·34–2·67)
Ex < 6 y	2·30 (0·80–6·65)
Current < 20/day	0·72 (0·30–1·76)
Current ⩾ 20/day	1·07 (0·49–2·34)

ASBESTOSIS

The mean duration of exposure to crocidolite was nearly three times as high in the 66 subjects with asbestosis as in the 2647 control subjects (table 4). Intensity of exposure was also higher among cases than controls, as was cumulative exposure. No difference in smoking habits between the two groups was found.

The best fitting model for exposure to asbestos (table 5) included a quadratic term in either duration of exposure or cumulative exposure. As with lung cancer, the model with cumulative exposure was a better fit (a reduction of 2·5 in the residual deviance with one less term estimated).

The only other significant effect was that for work site ($p = 0.005$) with the highest rates associated with work in the mine proper. Smoking had no consistent effect ($p = 0.57$) on the relative risk of asbestosis. The effects of the smoking variables were almost the same even without the adjustment for exposure to asbestos included in table 5, indicating little confounding between the two exposures.

Table 4 Asbestosis: asbestos and cigarette smoke exposure variables

	Cases (n = 66)	*Controls* (n = 2647)*
Crocidolite exposure state:		
Mean duration (days)	1000	394
Mean intensity (f/ml)	35	25
Mean cumulative (f/ml-years)	71	23
Mean time since first exposed (y)	25	26
Smoking status (%):		
Never smoked	21	21
Ex > 10 y	21	19
Ex 6–10 y	9	8
Ex < 6 y	11	6
Current < 20/day	15	20
Current ⩾ 20/day	24	26

*Mean of the mean of each set of controls.

Discussion

This study has shown that smoking state is related to the rate of lung cancer in Wittenoom crocidolite workers. The risk in current smokers was dose related and the risk in ex-smokers was greatest in the period up to six years from stopping. There was a smaller but still highly significant effect of crocidolite exposure on the rate of lung cancer. In our study this was best expressed in terms of the logarithm of total cumulative exposure rather than with separate terms for duration and intensity as was found previously.[3]

Statistical modelling of the effect of smoking and exposure to crocidolite on the risk of lung cancer indicated that these effects acted multiplicatively as reported in cohorts exposed to other forms of asbestos.

Previous studies have found a reduction in relative risk from asbestos associated lung cancer long after exposure has ceased.[36 37] That this should be so is supported by theoretical principles of the multistage theory of carcinogenesis if it is assumed that asbestos acts at a late stage in the disease process and there is elimination of asbestos from the lungs.[6] Because of the way the cohort for this study was defined long after exposure had ceased, any such effect would, however, probably be overemphasised because of survivor effects analogous to the healthy worker effect.

The high relative risks in ex-smokers could be due

to inaccuracies in self reporting or, more likely, to people quitting after either experiencing symptoms or being advised to stop smoking after reporting symptoms. The higher risk for asbestosis among recent quitters supports this interpretation.

Both duration and intensity of exposure to crocidolite as well as cumulative exposure have been shown to be strongly related to the risk of asbestosis. The better fit of the quadratic model and the use of cumulative exposure has also been shown before[13 22] and has some theoretical support from consideration of lung clearance mechanisms.[13]

Other studies have shown that increasing age and smoking have an effect on producing minor grades of abnormality on the chest x ray films even in the absence of exposure to asbestos.[23 38 39] Hence it may not be that smoking is making any difference to the process of asbestosis as has been assumed in the analysis. The radiological abnormalities related to smoking that mimic pneumoconiosis probably result from peribronchiolar fibrosis rather than diffuse interstitial fibrosis. If this is true, a small additive effect of smoking on radiographic abnormalities might be anticipated. Where it is possible to judge, other studies have shown such an additive effect,[10 20] although neither study was able to adjust for level of exposure to asbestos[39] as has been done here. A specific measure of the severity of diffuse interstitial fibrosis would be needed to show with any certainty an effect of smoking on asbestosis.

The strong effect of site of work on incidence of asbestosis has been noted before[3] and is likely to be caused by the greater awareness of asbestosis and claims procedures among miners than among millers and also the possible prejudices of members of the Pneumoconiosis Board in regarding exposure outside the mine and mill as not being heavy.

One problem with this study is that only the smoking history obtained in 1979 was used for analysis and this did not allow for change in smoking habits thereafter. Given the comparatively short follow up this is unlikely to be a major problem. Loss to follow up was a serious problem in other studies of the Wittenoom cohort where the whole cohort of workers was included[2 3] but for the cohort studied here, restricted to those traced and responding, no subject was lost to the end of 1986. It is unikely that the differences between responders and non-responders in the 1979 survey would include differences in their reaction to smoking or to exposure to asbestos.

This study has shown that the effect of crocidolite on the incidence of lung cancer multiplies that of smoking, a finding that is consistent with previous studies of exposure to other forms of asbestos. In the Wittenoom cohort the diagnosis of asbestosis for compensation purposes or as the cause of death is closely related to the degree of exposure to crocidolite but not to smoking habits.

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Vancouver style

All manuscripts submitted to the *Br J Ind Med* should conform to the uniform requirements for manuscripts submitted to biomedical journals (known as the Vancouver style).

The *Br J Ind Med*, together with many other international biomedical journals, has agreed to accept articles prepared in accordance with the Vancouver style. The style (described in full in *Br Med J*, 24 February 1979, p 532) is intended to standardise requirements for authors.

References should be numbered consecutively in the order in which they are first mentioned in the text by Arabic numerals above the line on each occasion the reference is cited (Manson[1] confirmed other reports[2-5] . . .). In future references to papers submitted to the *Br J Ind Med* should include: the names of all authors if there are six or less or, if there are more, the first three followed by *et al*; the title of journal articles or book chapters; the titles of journals abbreviated according to the style of *Index Medicus*; and the first and final page numbers of the article or chapter.

Examples of common forms of references are:

1 International Steering Committee of Medical Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1979;1:532–5.
2 Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosino-phil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;294:687–90.
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Smoking, exposure to crocidolite, and the incidence of lung cancer and asbestosis.

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THE BULLETIN

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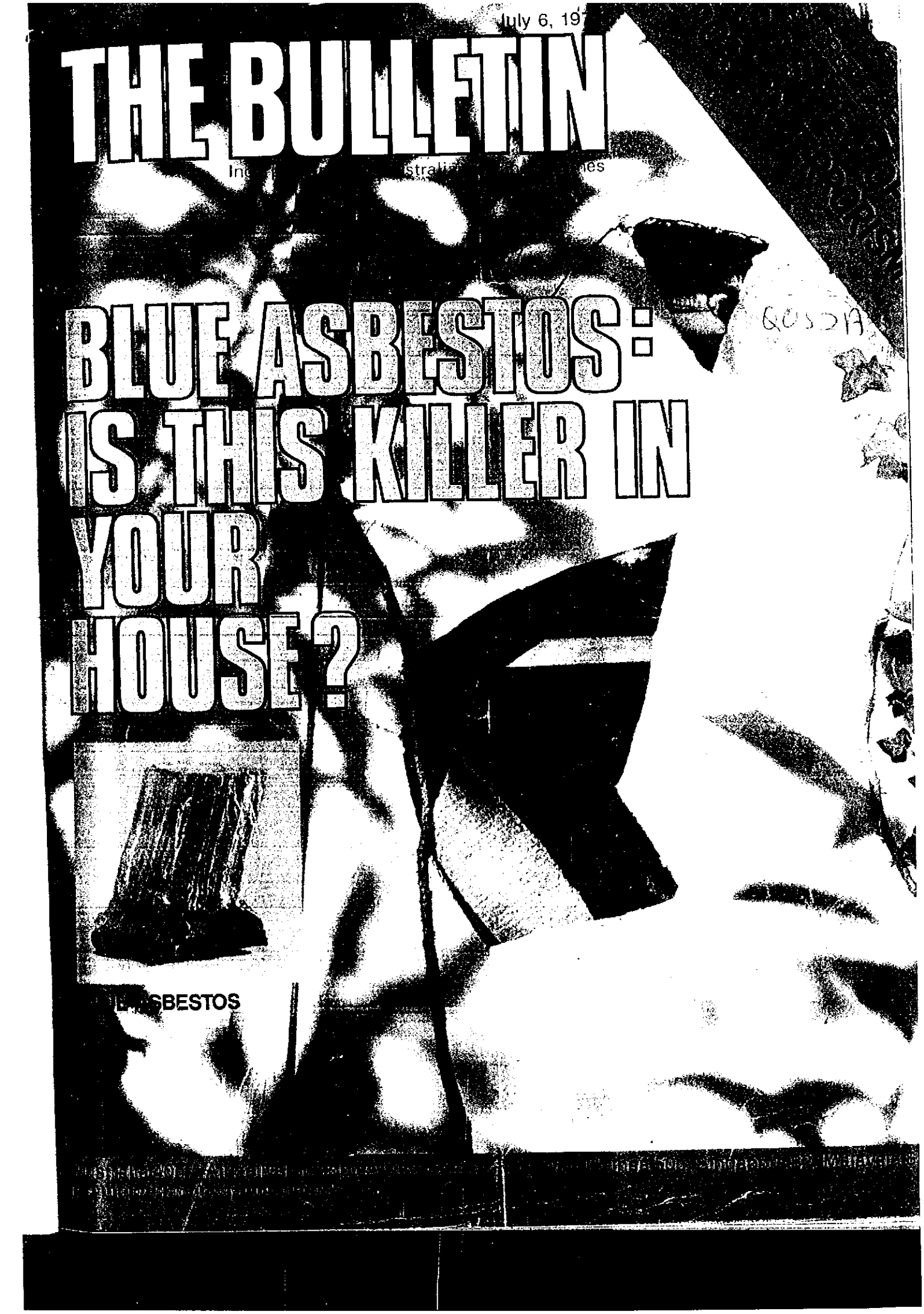
BLUE ASBESTOS: IS THIS KILLER IN YOUR HOUSE?



Is there a killer in your house?

The suburban house made in part with material containing asbestos, is one of the most familiar features of the Australian landscape. Some of the mineral, as pipes or in the roof, can be found in virtually every home. As brake linings and for fire protective clothing it is indispensable.

But along with spreading usefulness has come spreading doubt as to its safety. At first doubts were associated with the mining of the mineral. Now more and more evidence is piling up of the grave danger to people who use materials made from blue asbestos in the course of their work. The greatest danger may be in the building industry, and particularly to those engaged in demolition of older houses and flats.

The Bulletin's TIM HALL has been investigating the history of blue asbestos mining in Australia, talking to academics and medical experts on the present, and possible future, death risk from association with the mineral and finding out how widespread the danger is.

ASBESTOS has been recognised as a major health hazard for a long time. The earliest known risk was the crippling and often fatal hardening of the lungs known as asbestosis which was caused by inhaling the minute asbestos fibres. Then in 1934 it was shown that cancer of the lung could be caused by asbestos dust; and in 1957 a direct relationship was proved between crocidolite, or blue asbestos, and a much rarer cancer called mesothelioma. This affected not the lung itself but the pleura or outside lining of the lung; and occasionally the peritonium.

It added up to a potentially very dangerous mineral, but what kept it from becoming an accepted hazard on a much larger scale was that heavy and prolonged exposure to the fibre was thought to be necessary before there was any danger of getting either asbestosis or lung cancer. With mesothelioma it was different. It was dose-response related only in as much as increased exposure speeded up the time it took the disease to manifest itself. A woman died whose only contact with blue asbestos was the dust which she shook out of her husband's overalls. In addition the first cases in South Africa and England suggested that the dormancy period was very long -- even 30 years and more. For all of this time it was undetectable but once it manifested itself it was invariably fatal.

The discovery of this new risk involving crocidolite should have caused profound concern in every country that used it. Although produced in comparatively small quantities blue asbestos was commonly added to most asbestos products because of the peculiar properties of its long, fine fibres. They are the same properties which make the

Dr Jim McNulty: a tireless critic of the CSR mine

fibres so dangerous to man: they are so fine and sharp that they can go straight through the wall of the lung when they are breathed in and lodge themselves in the pleura.

But apart from a few attempts to have crocidolite banned altogether nothing happened at all. In South Africa which produces most of the world's blue asbestos and where the first cases of mesothelioma were found the victims were nearly all black and the high incidence of the disease passed unnoticed. The mine which employed them, Cape Asbestos Pty Limited, was the wholly owned subsidiary of a British company. The American Government was sitting on huge stockpiles and brought pressure to bear on the manufacture to play down the risk: and European indifference ensured that nothing was done there.

Australia had good reason to be particularly concerned by this new development. It was the only country in the world apart from South Africa that was actually producing blue asbestos; yet in spite of this its only mine continued to work with dust levels that were far above those which were considered safe in any other operation involving asbestos. What happened has been described by Western Australia's Mines Medical Officer, Dr Peter Maguire as "a tragic chapter in our mining history."

Of the 20,000 men who worked there, most of them new migrants, it is estimated that 10 and perhaps even 15 percent are likely to contract mesothelioma. Besides them there are their wives and children and numerous other people who have been exposed to the blue dust in the years since the mine closed and while it was working. Only now as the shortest dormancy periods end is the possible enormity of the tragedy becoming clear.

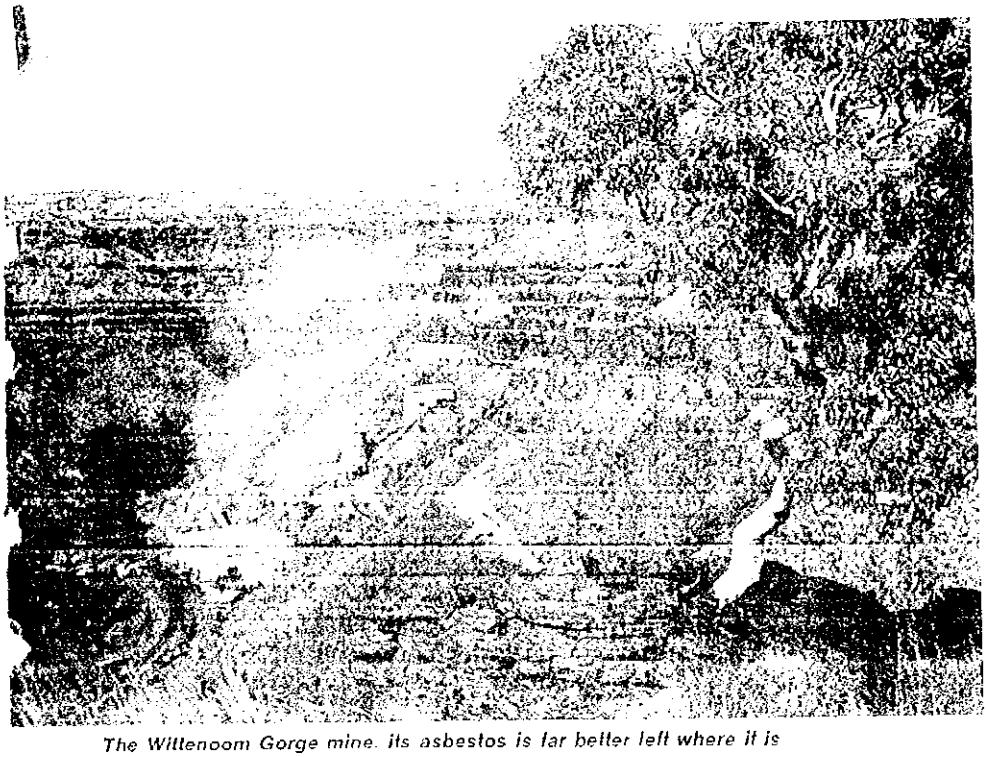
Almost by coincidence the risk involved in all the other uses of asbestos have been questioned too, although hardly at all in Australia. It has now been established for instance that with one exception all the variations of asbestos can to some degree cause mesothelioma. Primarily the dangers are greatest for those who are working with asbestos; but, as Dr Robert Barnes, specialist medical officer with the Worker's Compensation (dust disease) Board in Sydney, points out "environmental exposure to asbestos is a hazard to all and not just to those engaged directly in its manufacture and use."

Few people share the view of the asbestos companies that the risk of exposure from such sources as the demolition of old properties is too small to be worth considering. At a recent meeting of the world's foremost experts in asbestos held in Geneva under the auspices of the ILO it was agreed that "probably the greatest problem in controlling harmful dust exposure occurs during the removal of old insulation and in the demolition of premises and plant." One of the experts attending from Australia was Dr E. S. "Terry" McCullagh, Chief Medical Officer of James Hardie & Company, Australia's largest asbestos producer.

Australia's single blue asbestos mine was at Wittenoom Gorge, 1200 miles north of Perth in the Hamersleys. The deposits were found in 1939 by Lang Hancock who worked at them

half-heartedly until they were taken over in 1943 by CSR. Operating through its wholly owned subsidiary, Australian Blue Asbestos Limited, CSR stayed at Wittenoom for 23 years.

From the time the mine opened it was obvious that it was going to be a dirty operation. The nature of the asbestos itself meant that a dangerously polluted atmosphere was almost unavoidable. In 1958 the first inevitable case of asbestosis was diagnosed there and two years later a laborer died from mesothelioma — the first reported case of this cancer in an asbestos worker outside South Africa.



The Wittenoom Gorge mine. its asbestos is far better left where it is

The case was diagnosed by Dr. Jim McNulty, then a chest physician with the Health Commission, who is now director of public health for the State. From then on he was a tireless critic of the way that CSR operated their mine.

Even by mining standards the turnover of staff at Wittenoom was high, in some years 500 percent. It was because of this that 20,000 men passed through the plant while the work force at any one time never exceeded 500.

In 1959 conditions were so bad that the men put in a claim for an additional allowance of 2/6 per week to compensate for the dust which choked everyone who had to go near the mill and the mine. The claim was opposed by CSR and a board of review spent three days at Wittenoom before deciding in favor of the company and rejecting the claim.

By the end of that year men were contracting asbestosis at Wittenoom after being exposed to the dust for only 12 months and the casualty rate was climbing fast. Of a group of 41 men seen by McNulty, 14 died and 11 were totally disabled. CSR now maintain that there was not enough evidence at the time to prove a special danger beyond doubt; whether this was so or not there were certainly ample grounds for the gravest suspicion about whether it was safe to go on working the mine until the dust levels were brought down to a level which at least was considered safe in other asbestos activities.

"Wittenoom is a dreadful tragedy" says Dr. Janet Elder, senior chest physician at the Sir Charles Gardiner Hospital at the University of Western Australia "because so many of its victims were very young and very fit when they went there. However lax and careless CSR were I don't think they were entirely to blame for not recognising the peculiar dangers of mesothelioma at that stage; but their precautions weren't even sufficient to prevent asbestosis."

There is no doubt that they were told this by McNulty whether they accepted it or not. "I was," he says, "always on their backs." When he diagnosed his first case of mesothelioma in 1960 he explained the significance of it to the company's management; when it was published in the Medical Journal he sent them a copy. It included his conviction that "the relatively short period of exposure to blue asbestos dust confirms the impression . . . that these tumors may arise after transitory exposure to crocidolite."

CSR insist that they or ABA invariably did everything that was recommended by the Health Department and the Mines Department. Correspondence that is still on the Mines Department files hardly supports this. In a confidential letter dated October 2, 1961, to the under secretary for mines, McNulty wrote, "It would appear that repeated advice and warnings of the health hazard from dust have been ineffective and that stronger action will have to be taken."

The Mines Department too was putting what pressure it could on the company. "Twice," says chief inspector of mines for Western Australia, Jack Boyland, "I threatened to close the mine if the company didn't do something about it." But it was an empty threat and presumably the company knew it. Neither the Health Department nor the Mines Department had — or still have — the authority to close the mine no matter how dangerously it is being run. It is a situation which the State mines medical officer Dr. Peter Maguire describes as "disgraceful and quite mad." The Health Department couldn't even prosecute the company because responsibility for the men's welfare and safety was vested in the Mines Department.

Western Australia is not alone in this. In New South Wales are the only two asbestos mines still working in Australia, both producing chrysotile. At Baryulgil, north of Grafton, James Hardie has a small 20-man operation which is in the last year of its life. And at Barraba near Tamworth the unhappy Woodsteef Mine struggles on from day to day, plagued by wildcat strikes and in the hands of the receiver. The New South Wales Occupational Health Department doesn't even have the right to go into a mine, says Dr. Eric Longley, Acting Head of the Department. "We are usually allowed in after considerable wheedling but we've often wished it differently."

After these verbal lashings Australian Blue Asbestos did do something constructive, but it was never enough. Until the day it closed it was the dirtiest mine that McNulty had ever seen in Australia with its dust levels far above any acceptable level of safety. Even when the expensive extractor plant which ABA installed was working properly — which because of poor maintenance it frequently wasn't — the conditions were still deplorable, but in a different place. Jim McNulty remembers it well, "It took the dust out of the mill beautifully — and blew it all over the lawns outside. It was more dangerous to stand out there than it was to work in the mill."

There were other incidents which will still need an explanation. McNulty arrived in the town one day to find that the company had sent a load of tailings with numerous fibres in them to the school playground to fill in small potholes. The children were sitting digging in it. On that occasion he was able to have the tailings removed.

CSR [illegible] of the blue asbestos [illegible] 45 percent of it [illegible] Hancock and most of the rest [illegible] It is difficult to understand why they continued to go on working what had become an unprofitable and embarrassing operation when the blue asbestos could be imported cheaper and in better quality from South Africa. The reason given today by CSR deputy general manager Brian [illegible] is: "We had a responsibility to the people working there to keep the mine going as long as

the mine closed. Like most of the [illegible] people who are still there with him, he admits to being philosophical about the risks but admits that he could easily be harboring mesothelioma. In spite of this he still uses the tailings to make concrete – "but I'm carful not to breathe in very much." Of the townspeople's knowledge of the danger of crocidolite he says: "I haven't heard it told to them and I haven't told them myself. It's just something that never seems to crop up."

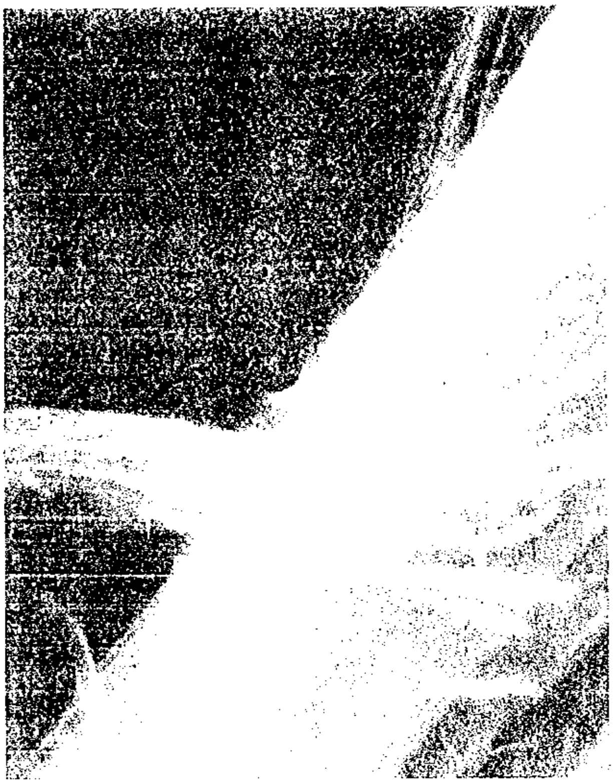
The blue asbestos still turns up in many unlikely places. In Perth last week

Perth [illegible] should have gone further and [illegible] a few illegitimate ones."

CSR has reacted quickly to the suggestion [illegible] by The Bulletin that it was [illegible] in its operation of the mine. Director of the company's corporate research, Dr Max Smythe, has been instructed to make a personal investigation of the whole affair. Perhaps his most revealing remark so far was that there is no question that the mine would have been closed down in a similar situation today.

In the course of his investigation he

Amosite magnified 300 times: it is now being used in place of crocidolite [illegible] is also dangerous



Chrysotile magnified 3000 times: the least dangerous type of asbestos mined in Australia

Crocid[illegible] looks se[illegible]

possible [illegible] the health hazards were not mentioned by the company when it [illegible] its reasons for pulling out of Wittenoom. Instead, it pleaded unprofitability, a low return of asbestos fibre from the ore and difficulty in finding and keeping labor.

On December 30, 1966, it moved out and the following year sold back the [illegible] Hancock for more than a million dollars. Behind them they left townships, roads and paths built almost entirely from blue asbestos and a million tons of tailings. The tailings are surrounded by barbed wire now and there is a Health Department notice warning people not to touch them. But the children still play there and quantities of asbestos still find their way into the town [illegible] concrete. Dr Gorton [illegible] came to Wittenoom 13 years ago as the town's GP with his income guaranteed by CSR, stayed behind when

the owner of a souvenir shop explained that the blue asbestos in her rock samples were so "shaggy" that the fibres were blowing all round the shop and she had had to put it away in a cupboard. When she brought it out to sold for one dollar she blew off the loose dust. She had no idea that it was any more dangerous than a lump of coal and most other proprietors of souvenir shops in the city must think the same.

In hindsight Dr McNulty is not at all sure that he did all that he could. "We were like Cassandra crying that dire results would follow – and nobody listens to people who wail about sad things that are going to happen in the future."

That events have proved him right beyond his worst fears is cold comfort. "Looking back now" he says "I think I tried every legitimate means open to me.

will cross the path of Professor Michael Hobbs, associate professor of occupational health at the University of Western Australia. Hobbs has embarked on the enormous task of trying to track down every one of the men who worked at Wittenoom. Fortunately personnel records had not been destroyed. He hopes to issue a preliminary report before the end of the year.

What happened at Wittenoom highlights the whole question of asbestos pollution which must now be re-examined. If Professor Bryan Gandevia, professor of medicine at Prince Henry Hospital in Sydney is correct, even sawing a sheet containing blue asbestos is enough to ensure that some people will contract mesothelioma.

"If he is right" says McNulty "and there really is only minimal dose response relationship, then there ought to be a complete total world-wide ban on

creedolite for any purpose at all." Some countries are already working towards this end.

Janet Elder agrees "If and I think we've got to accept this there is a link, then blue asbestos shouldn't be used at all. For some time it was cases of respiratory failure that we were seeing from Wittenoom, but now we are getting the cancers and mesotheliomas. I think we're going to get many more mesotheliomas, especially in the next five years as we start to reach the end of the latent period for many of these people."

Magnified 3000 times: This is how the killer ore through a microscope

Professor Hobbs shares her opinion. "The cases cropping up now are early in the incubation period which could herald a real disaster to come."

What happens next depends as much on government as on the self-discipline of the asbestos producing and using companies. With the exception of Queensland there is no legislation of any kind controlling the purchase or use of blue asbestos. Says Dr Longley of the New South Wales Occupational Health Department "We still find people spraying it on ceilings from time to time."

The main difficulty is to control the small unregistered companies whom the department can't even find to educate. Most of them probably know little of the danger involved. It could take a long time for them to find out. Two women who died recently from mesothelioma in London contracted the disease during World War II when they were making gas masks that contained crocidolite. And in Australia two men have also died recently who contracted the disease at about the same time when they were working as welders on pontoons. The handles of their welding rods contained blue asbestos.

The risk is undoubtedly there, although Terry McCullagh of Hardies insists that there is not a worthwhile hazard from the dust in pipes and sheetings.

Most of the evidence now coming available suggests that he might be too optimistic. Dr Bengt Fristedt of the Industrial Medical Clinic in Stockholm investigated 48 building workers by X-ray who were doing house and flat repair work. Six had lung changes considered to be due to asbestos. Said Fristedt echoing the ILO meeting in Geneva, "It is known that dust on building sites and, above all, dust created during demolition and repair work can contain asbestos fibres."

It is very unusual to see anyone involved in demolition work in Australia taking precautions against the dust even when that dust may well contain asbestos fibres.

Meanwhile from Sweden too there is evidence that asbestos dust is affecting not only people who work on the site, but those living near by. At Malmo General Hospital in the south of Sweden two doctors, Inge Hanger-tand and Barbro Seiner, performed autopsies on 97 people for other reasons and discovered that 45 of them had lumps inside their lungs. All these lumps turned out to have been built up around particles of inhaled asbestos dust.

The children at Wittenoom, the souvenir shop keeper with her "shaggy" asbestos, the demolition contractor who ignores the dust and the handy man who saws up a sheet of asbestos are all taking an unnecessary risk. With the sheeting or most other asbestos products which were made until about 1968, 20 percent of the fibre was usually blue. In most cases it has now been replaced by amosite. This has now been shown to be the second most hazardous of the fibres so far as mesothelioma is concerned.

It is not in the interest of the manufacturers that there should be widespread concern about the safety of asbestos, but it is a fact that apart from the work which they carry out to safeguard their own employees they do practically nothing to educate the public to the dangers of asbestos.

There is a danger in waiting too long to act if not always with such tragic results as at Wittenoom. As the ILO concluded, it is unnecessary to wait for a complete understanding or overwhelming proof of one point to make the maximum use of existing knowledge to reduce risks to a minimum in the future.

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Malignant Mesothelioma in Australia, 1945–2000

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Australia has maintained a national malignant mesothelioma register since 1980. The register includes all cases in Australia. Currently 450–600 cases are notified annually in a population of 20 million. Australia has had 6329 cases of mesothelioma in the period 1 January 1945–31 December 2000. A further 680 cases have been notified in the period 1 January 2001–31 December 2001. Annual incidence rates for Australia per million population ≥20 yr (1998) were male, 59.8; female, 10.9; total, 35.4. Incidence rates have been continually increasing in males and females and are the highest reported national rates in the world. While Western Australia has the highest rate (1998, 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 81% of cases with no history of exposure, TEM lung asbestos fibre counts >200000 fibres >2 μm length/g dry lung were obtained, suggesting unrecognized exposure. Australia's high incidence of mesothelioma is related to high past asbestos use, of all fibre types, in a wide variety of occupational and environmental settings. The number of cases in total is expected to be about 18000 by 2020, with about 11000 yet to appear.

Keywords: malignant mesothelioma; incidence; Australia; asbestos exposure; future predictions

INTRODUCTION

Asbestos was mined in Australia for over one hundred years and Australia was the world's highest user per capita of asbestos in the 1950s. Given the ecological relationship between per capita asbestos consumption and mesothelioma incidence (Takahashi *et al.*, 1999), it is no surprise that in the last 20 yr 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 been in operation since 1980. It is the purpose of this paper to 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). The paper updates and enhances previous reports (Leigh *et al.*, 1991, 1997, 1998; Leigh, 1994).

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MATERIALS AND METHODS

Australian Mesothelioma Surveillance Program (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 full occupational and environmental history was obtained for each case, either from the patient or next of kin. The history taking was non-directive but included specific questions on asbestos exposure at the end. 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 tumour for lung fibre content analysis.

Australian Mesothelioma Register

From 1 January 1986, a less detailed notification system has operated, with a short questionnaire on occupational and environmental exposure history, which is followed up assiduously; there is no pathology panel diagnosis and only sporadic lung fibre counts. In the case of New South Wales and Western Australia (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). Incidence rates have been calculated up to the end of 1998 only, because of the up to 2 yr delay in notification experienced while awaiting confirmed diagnosis and reconciliation with the state cancer registries.

RESULTS

The incidence of mesothelioma in Australia

From 1 January 1980 to 31 December 2000, a total of 5671 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 to 2000 inclusive was 6329. A further 680 cases have been notified in the period 1 January–31 December 2001. Notifications show a continuing upward trend in both males and females (Fig. 1). The notifications prior to 1982 were probably the result of bedding in of a new Program and are artificially low (1980, 16; 1981, 104), although a smooth curve of increasing incidence starting from the early 1960s has since been demonstrated by a retrospective search (Fig. 2). The Australian population increased from 14.5 million in 1980 to 20 million in 2001. Mesothelioma incidence rates have increased from 12.8 per million population ≥20 yr age/yr in 1982 to 35.4 per million/yr in 1998 (males and females combined), 50.6 per million/yr (males) and 9.0 per million/yr (female). Figure 3 shows rates by time and sex. If the 1981 figure is accepted it can be claimed that mesothelioma incidence rates have increased 4- to 5-fold in 19 yr 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 rate of liver cancer, and in mortality terms equal to the mortality rates of kidney cancer in males and uterine cancer in females (NSW Cancer Council, 2000). Mesothelioma is no longer a 'rare disease'.

Table 1 shows notifications by state up to 31 December 2000.

Western Australia has the highest incidence (1998 rates: total 52.8, male 96.2, female 9.4) but contrib-

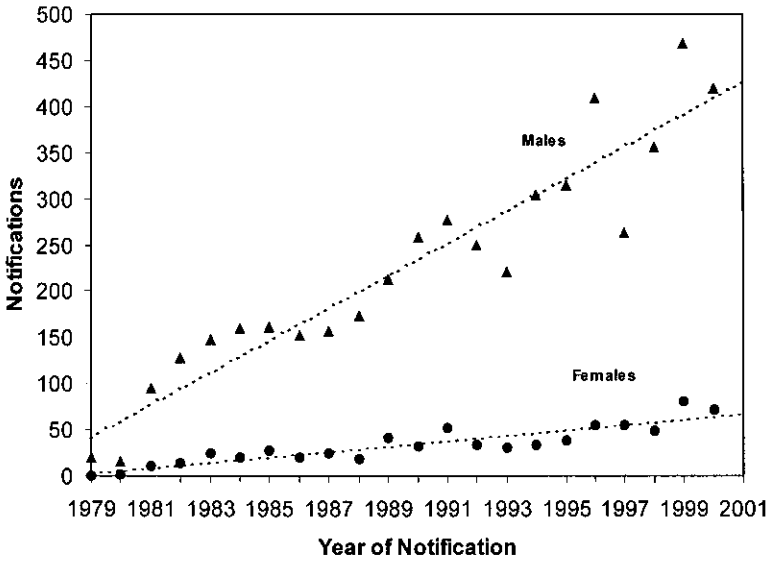

Fig. 1. Australian Mesothelioma Register notifications, 1979–2000 (by sex).

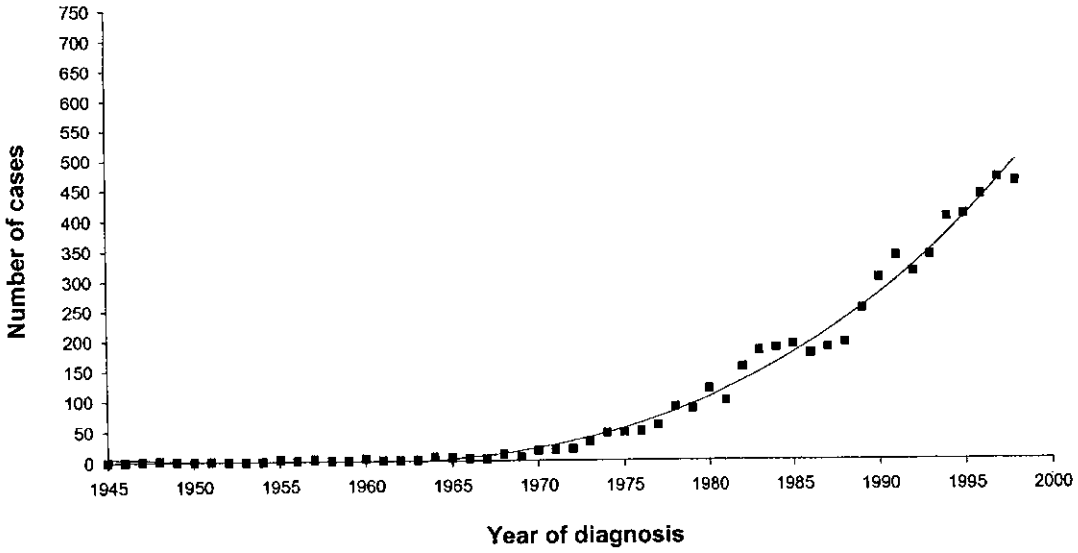

Fig. 2. Incident cases of mesothelioma in Australia, 1945–1998.

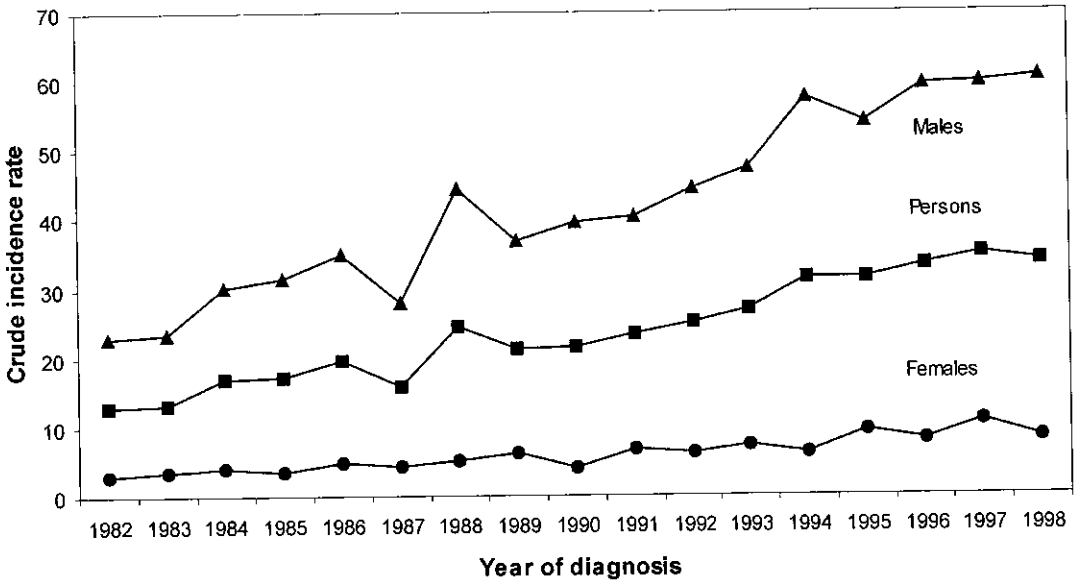

Fig. 3. Time trend of annual incidence rate (per million population ≥20 yr) of mesothelioma in Australia by sex, 1982–1998.

utes 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.

In 93.2% of all Program cases the mesothelioma was pleural in site, 6.5% peritoneal and only 0.3% of cases in other sites. Among men 94.3% were pleural, 5.3% peritoneal; among women 86.3% were pleural, 13.7% peritoneal. These proportions have been generally maintained in Register cases although the female peritoneal proportion has dropped to 10.4%.

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/installation (4%), wharf labouring (3%), power stations (3%), boilermaking (2%), paraoccupational 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 a history of some exposure recorded. Thus only 19% had no known history. Moreover, of this 'no known history' group, 81% had

Table 1. Mesothelioma notifications in Australia, 1980–2000

	NSW	VIC	QLD	WA	SA	TAS	NT	ACT	Totals
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	2118	1078	892	893	557	90	5	38	5671
%	37.3	19.0	15.7	15.7	9.8	1.6	0.1	0.7	100

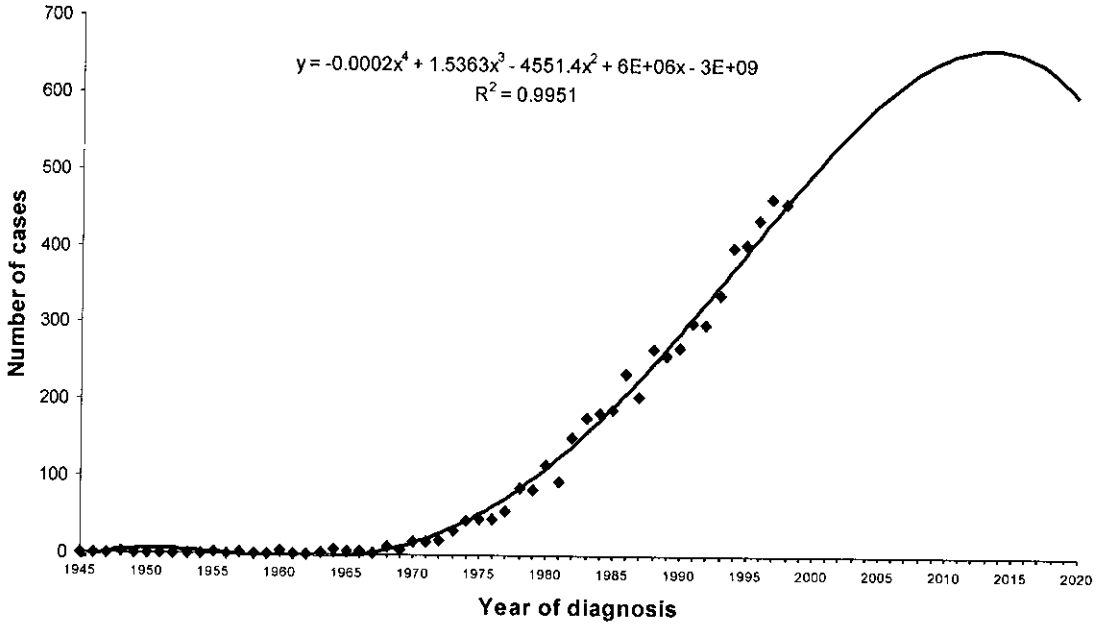

Fig. 4. Incident cases of malignant mesothelioma in Australia 1945–98 and extrapolation to 2020 assuming maximum at 2010.

fibre counts >200000 fibres/g dry lung detected in the lungs, 30% with more than 10^6 fibres/g >2 μm including 'long' (>10 μm) fibres, suggesting that nearly all cases had 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 fibres in the lungs does not negate exposure as fibres may have initiated mesothelioma and then been cleared before death. The shortest duration of exposure was 16 h (waterside worker loading crocidolite fibre (Musk

et al., 1991). Three percent of cases had exposures of less than 3 months. According to history assessment of the first 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 fibre type (Grimwood, 1988). Mean latency from first exposure to presumptive diagnosis was 37.4 yr (Ferguson *et al.*, 1987). The range of latencies was 4–75 yr.

In the cases reported since 1 January 1986, when less detail of history of exposure was sought, 89.9% of males responding to the 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. 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 labour (2%), para-occupational, hobby, environmental (4%), carpenter (4%), builder (6%), navy (3%), plumber (2%), brake linings (2%), multiple (12%).

Risk in particular occupational groups

Approximate lifetime risks in occupational groups exposed 30–50 yr ago were obtained as follows:

Lifetime risk (%) = (70 × number of mesothelioma cases notified 1986–2000 × 100)/(population in occupation category in Australia × 15)

Table 2 shows the results for the major identifiable occupational groups.

Population denominator data was estimated from census data, Australian Bureau of Statistics (ABS) data, cohort data for Wittenoom, defence data for the navy and union data for waterside workers. As the range of latencies was 4–75 yr, the relevant occupational group population sizes were estimated as the mean of the 1933 and 1997 value, except for Wittenoom, navy and waterside workers, where industry, defence and union data was used.

Table 2. Mesothelioma risks in occupational groups

Occupation	Lifetime risk of mesothelioma (%)
Wittenoom mine or mill worker	16.6
Power station worker	11.8
Railway labourer	6.4
Navy/merchant navy	5.1
Wittenoom town	3.1
Carpenter/joiner	2.4
Waterside worker	2.1
Plasterer	2.0
Boilermaker/welder	1.9
Bricklayer	1.8
Plumber	1.7
Painter/decorator	1.2
Electrical fitter, mechanic, electrician	0.7
Vehicle mechanic	0.7
All Australian men	*0.39*
All Australian women	*0.07*

CONCLUSION

The high and increasing incidence of mesothelioma in Australia is due to high asbestos use in the past, combined with poor hygiene practice, relatively high amphibole use in asbestos cement products, slow recognition of chrysotile mesotheliomagenicity 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.

The expected total number of mesothelioma cases in Australia from 1945 to 2020 is estimated to be about 18000, based on models by Berry (1991) and de Klerk *et al.* (1989) for Wittenoom, extrapolated to 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 yr latency from the time of maximum exposure (1970) (Fig. 4). 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.

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. These predictions are consistent on a population and asbestos use adjusted basis with those made for Europe (Peto *et al.*, 1999), Scandinavia, USA, Japan (Takahashi *et al.*, 1999) and New Zealand (Kjellstrom and Smartt, 2000).

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

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Smoking Intervention in Subjects at Risk of Asbestos-Related Lung Cancer

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A smoking intervention counseling program was applied among asbestos-exposed male smokers younger than 65 years of age to examine the effect of an intervention based on risk communication. Intervention subjects (n = 431) were invited to a health status checkup combined with physician-delivered smoking intervention counseling. Control subjects (n = 141) received no intervention. After 1 year, 5% of the responders in the intervention group, versus 3.4% in the control group, had stopped smoking. Corresponding conservative estimates were 3.5% and 2.6%, respectively. The quitters had been exposed to a higher "dose" of asbestos but had smoked less, and for a shorter period, than had the continuing smokers. Counseling by a general physician increased successful quitting threefold, compared to counseling by a physician in a specialized institution. These results suggest a potential for smoking cessation among subjects at high risk of lung cancer due to asbestos exposure. General practice care may be an appropriate setting both for identification of such subjects and for intervention. Am. J. Ind. Med. 31:705–712, 1997. © *1997 Wiley-Liss, Inc.*

KEY WORDS: smoking cessation; reduction of smoking; asbestos workers; general practice

INTRODUCTION

Exposure to asbestos and smoking may synergistically increase lung cancer risk more than to 50–60 times, compared to being an unexposed never-smoker [Hammond et al., 1979]. In two Norwegian studies, lifetime prevalence of asbestos exposure was 36% in a male population aged 18–73 years [Bakke et al., 1990] and 32.5% among men aged 39–48 years [Waage and Hilt, submitted 1997]. In both studies, about 50% reported current smoking, resulting in 15–20% asbestos-exposed smokers in these populations. Tobacco abstinence for 10–15 years reduces the risk of lung cancer among asbestos-exposed subjects by 60–70%, compared to continuing the habit [Hammond et al., 1979; Waage et al., 1993]. Even so, smoking cessation programs have not been widely or systematically applied among asbestos-exposed smokers [Langård, 1992; Langård and Waage, 1990]. One reason may be that tracing of exposed subjects is difficult unless systematic and large surveys are conducted, or company records of formerly exposed workers are available.

In the present study, we wanted to evaluate the effect of an intervention based on communication about the increased lung cancer risk in asbestos-exposed smokers. The intervention was either done in a secondary setting of an institution of occupational health or in a public primary health care setting.

SUBJECTS AND METHOD

Recruitment of Subjects

The study base was responders in a population-based questionnaire survey on asbestos exposure and smoking

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habits in Telemark county in 1982–1983 [Hilt et al., 1986]. In all, 572 males living in five industrialized municipalities who were younger than 65 years of age in 1989 were eligible. The intention was to compile a study group with varying levels of exposure to asbestos and tobacco smoke. Accordingly, all subjects with asbestos exposure of either "light" intensity for 15 years+, "moderate" intensity for 7 years+, or "high" intensity for any period of time, as reported in the questionnaire, were selected (n = 491). A projected lifetime tobacco consumption (as defined below) exceeding 150 kg was also required. To include some subjects with low/medium exposure, we selected 81 subjects with any level of asbestos exposure, whose projected lifetime tobacco consumption could be estimated.

Projected lifetime tobacco consumption

A projected lifetime tobacco consumption was calculated by accumulation of the individual's tobacco consumption to the age of 70, assuming a constant consumption at the reported level (one manufactured cigarette = 1 g, one pack of tobacco for own-rollers = 50 g, one cigar = 3 g). Eligibility for heavy smokers with a short duration of smoking was thus provided.

Allocation to Intervention and Control

Since the industrial structure of the municipalities was similar, study subjects were allocated to intervention (n = 431) or control (n = 141) based on geographical residence. Subjects living in four of the municipalities were selected for intervention, and the remaining were controls. The control group was not contacted and received no information until the evaluation 1 year later.

Intervention Program

The intervention took place between March 1989 and February 1990. Subjects were invited to a free consultation, comprising a brief health status checkup and counseling on smoking cessation. A leaflet explaining the risks of lung diseases related to the combination of asbestos exposure and smoking as well as the benefits of smoking cessation, accompanied the invitation letter. Subjects were urged to respond, or at least report their current smoking status.

The intervention was conducted at the Department of Occupational Medicine at the county hospital and by the public primary health care in one of the municipalities. The intervention was performed according to an outlined procedure. The only deviation was that expired carbon monoxide was not monitored in those who received intervention by the primary health care.

The participants were interviewed on health history, and on cancer among relatives. A brief medical examination was performed. The results have been reported elsewhere [Waage et al., 1996].

Asbestos exposure

The background information on asbestos exposure was supplemented by interview. Exposure intensity was assessed on a scale from 1 (low) to 4 (very high), based on knowledge of the work environment in various occupations, and on the subject's own description. Grade 1 was sporadic exposure (e.g., mechanics and electricians who had handled asbestos-containing gaskets). Grade 2 characterized more frequent exposure (e.g., automobile mechanics, carpenters, and welders). Grade 3 designated daily or almost daily exposure (e.g., work in ship engine rooms or industrial maintenance work). Grade 4 indicated heavy exposure, implying most of the working time (e.g., insulators or masons of industrial furnaces). If the type of work or the exposure intensity had varied, an intensity was assigned for each period. The cumulative asbestos exposure was calculated by adding the products of intensity and years of exposure for each period.

Information on smoking history

Information on weekly tobacco consumption, age of starting the habit, duration, previous quitting attempts, and other smokers living in the home was recorded by interview.

Counseling

The presumed risk of lung diseases was explained to each participant. To motivate smoking cessation, the effect of smoking and asbestos exposure on airway symptoms and lung cancer risk was outlined. The risk of adverse health effects was related to each person's exposure and smoking history, chest radiographic findings, shortness of breath, sputum production, and cough. The benefits of smoking cessation were discussed. As a didactic device carbon monoxide was monitored in expired air [Waage et al., 1992]. A display showed a red column escalating to the exact value (ppm) of carbon monoxide. How to prepare for smoking cessation was discussed (e.g., shift of brand, smoking with the opposite hand, and postponing the "after-meal cigarette"). Whenever the subject was regarded as motivated, a date for quitting within 2–3 weeks was agreed upon. The likelihood of relapse in certain situations (e.g., emotional stress or alcohol consumption was dealt with). Relapse prevention was discussed (e.g., aversive techniques, exercise, and change of immediate surroundings in the case of craving for tobacco). Prescription of nicotine polacrilex was offered. Dietary advice to minimize potential weight gain was routinely given [Coates and Li, 1983]. A brochure with a self-scoring questionnaire was handed out as a motivating device [National Council of Tobacco and Health, 1989].

TABLE I. Description of the Asbestos-Exposed Study Group, Based on Information from the Questionnaire Survey In 1982–1983

	Intervention group (n = 431)				Control group (n = 141)			
	N[a]	Mean	Range	SD	N[a]	Mean	Range	SD
Year of birth	431	1933	1924–1947	5.7	141	1932	1924–1944	5.7
Weekly tobacco consumption (g)	429	105.6	7–500	48.6	139	101.7	42–250	39.9
Number of years smoked	429	30.9	10–58	7.0	140	30.7	4–45	6.8
Projected lifetime tobacco consumption	428	282.8	17–1,508	136.1	138	271.2	114–688	112.3
Number of years exposed to asbestos	311	14.7	<1–37	10.2	102	15.4	<1–41	10.9

[a]Those who had given sufficient information to calculate the given variables. Those remaining had just reported their status of asbestos exposure or smoking.

Total abstinence from tobacco was recommended. However, reducing the consumption was suggested as an alternative for resistant subjects, although it was emphasized that the aim should be to abandon the habit in due course. Each session lasted 30–60 min., and additional appointments were offered. Participants were informed that they would receive postal questionnaires on smoking habits after 3 and 6 months as a follow-up procedure.

Evaluation of Smoking Cessation

After 1 year, a postal questionnaire was sent to all subjects who had initially been invited. They were asked about current weekly tobacco consumption, and the number and duration of any cessation attempts following the invitation letter. After another year, subjects who had actually attended the intervention were invited to a second health examination and to an interview on smoking habits.

In the control group, information on smoking status, weekly tobacco consumption, and the time of cessation (if an ex-smoker) was obtained by a postal questionnaire that was distributed on average 1 year after the intervention. The same leaflet that had been distributed to the intervention group, explaining the risks of lung diseases related to asbestos exposure and smoking, as well as the benefit of smoking cessation, was enclosed with the questionnaire. Also, a contact person was given.

Analysis

Smoking cessation is given in percentages. Ex-smokers were considered as inclined to respond. Hence, nonresponders at each contact were regarded as continuing smokers for calculation of conservative estimates. The relative risk (RR) of smoking cessation was calculated as the ratio between probabilities of cessation, given by the formula $1\text{-}e^{[-IC1]}$ /$1\text{-}e^{[-IC2]}$, where IC is the conservative estimate of incidence of smoking cessation throughout a defined time period [Kleinbaum et al., 1982]. For this purpose, person-time was accumulated by responders and nonresponders. Subjects who reported to have quit smoking at a given contact time contributed person-time until their respective dates for quitting. The cumulative probability of smoking cessation is shown in a Nelson plot [Andersen and Væth, 1988]. Nonresponders were regarded as continuing smokers. Each step in the plot represents an event of quitting.

Test-based 95% confidence intervals were calculated for the RRs. Other 95% confidence limits and P-values were calculated by approximation to the normal distribution. For computations, BMDP version 386, was used [Dixon, 1990].

RESULTS

Effect of the Intervention Program

In the intervention group (Table I), subjects known to be deceased, those with unknown address, or who wished no further contact (n = 12) were omitted from the calculations (Fig. 1). After one reminder, 348 of 419 eligible subjects (83.1%) responded to the invitation. Among these, 232 smokers wished to attend the intervention program, and 49 persons reported to have already stopped smoking. Accordingly, 11.7% among all the invited subjects had ceased smoking during the period 1982–1983 to 1989, resulting in a yearly rate of 1.5%.

At the 1-year evaluation, another three subjects had unknown address or were deceased. Among the remaining 367 smokers that initially had been invited, 70.8% responded. Thirteen subjects reported to have quit, resulting in 3.5% cessation as a conservative estimate, alternatively 5% among the responders (Table II). Twelve of these had attended the program.

In the control group (Table I), eight subjects had unknown address or were deceased, and 106 out of the remaining 133 subjects (79.7%) answered the questionnaire (Fig. 1). Only three subjects reported a cessation date after the start of the intervention program in 1989, resulting in a conservative estimate of 2.6% cessation, or 3.4% among the responders (Table II).

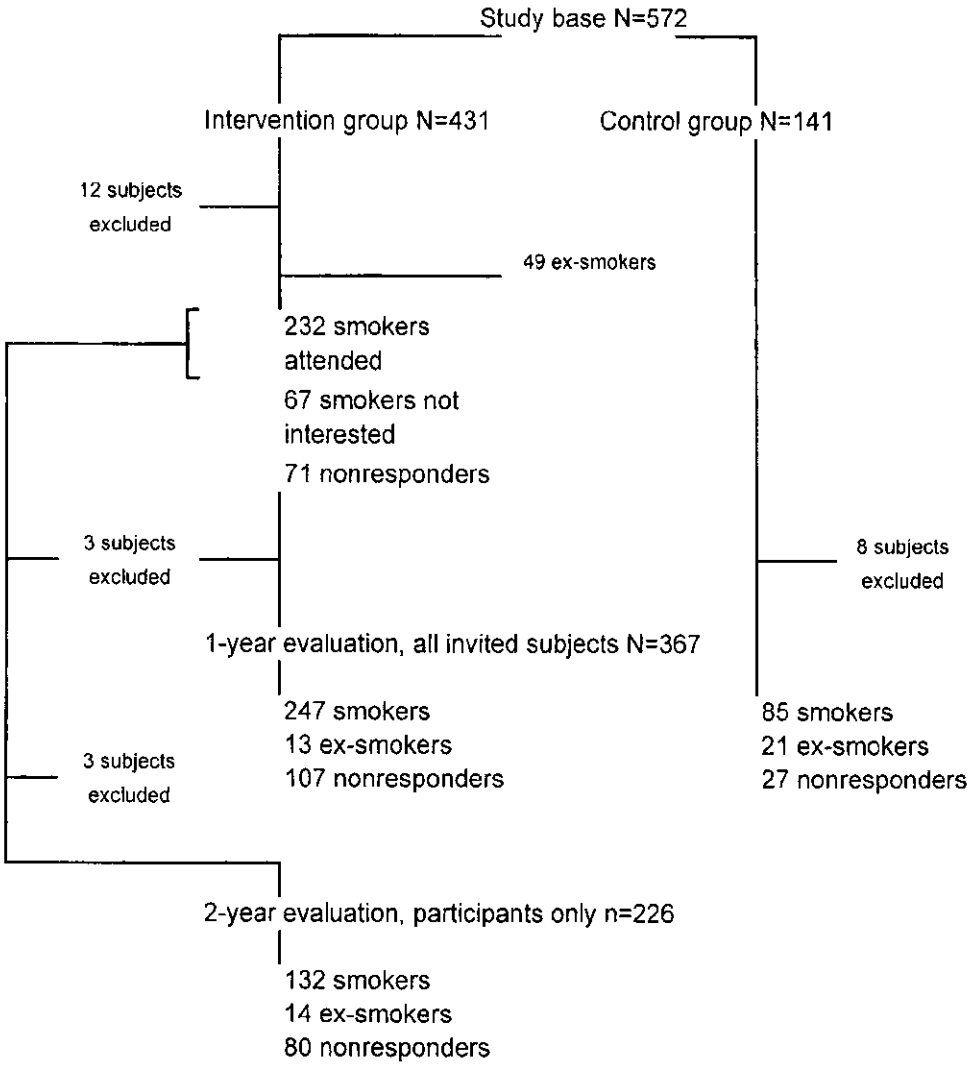

FIGURE 1. Flowchart for the study group of asbestos-exposed smokers in the cessation counseling program.

The RR of cessation was 2.2 (CI = 1.2–4.1) when the probability of cessation following the intervention was compared to the probability of cessation before the intervention in the intervention group, i.e., during the time period 1982/83–1989. When compared to the control group, the RR was 1.7 (CI = 0.5–6.0).

At the 2-year follow-up, 64.6% among the participants in the intervention group responded. Fourteen persons reported ex-smoking (Table III), of whom three had quit during the previous year. During the 2 years, 12 people had stopped smoking for more than 2 weeks but later relapsed. The cessation potential of the relapsers is illustrated in the Nelson plot (Fig. 2). The plot also shows that the intensity of both cessation and cessation attempts was highest just after the intervention, and lapsed after 6–7 months.

Patterns of Smoking Cessation

The quitters had smoked for a shorter period and had initially lower weekly tobacco consumption than the continuing smokers and the relapsers. Conversely, the quitters reported higher asbestos exposure in terms of both duration and cumulative exposure (Table III).

Successful quitting was nearly tripled when the intervention was conducted by the primary health care, compared to a specialized institution of occupational health, viz. 13.5% versus 4.8%. Further analysis (not shown) revealed that this was not related to skewness of other variables listed in Table IV. Previous cessation attempts were associated with successful quitting, and the prescription of nicotine polacrilex enhanced cessation attempts. These three findings were

TABLE II. Proportion of Smoking Cessation and Probability of Cessation in the Asbestos-Exposed Intervention Group and Control Group*

	Before intervention		One year after intervention	
	Intervention group	Control group	Intervention group	Control group
Smokers (N)	299	88	247	85
Ex-smokers (N)	49	18	13 new	3 new
Nonresponders (N)	71	27	107	27
Cessation among responders (%)	14.1	17.0	5.0	3.4
Cessation among all subjects (%)[a]	11.7	13.5	3.6	2.6
Probability of cessation among all subjects[a]	$18.1/10^3$	$22.4/10^3$	$39.3/10^3$	$23.3/10^3$

*Figures are given for the period before intervention (1982/83–1989) and for the year after intervention (1989–1990).
[a]Assuming that nonresponders were smokers.

TABLE III. Summary Descriptions of the 226 Asbestos-Exposed Participants of the Smoking Cessation Program, Related to Continuous Smoking, Quitting, and Nonresponse at the 2-Year Evaluation*

	Smokers (n = 126)	*P*-value[a]	Quitters (n = 14)	Relapsers[b] (n = 12)	Nonresponder (n = 74)
Year of birth	1933 (0.5)		1934 (1.4)	1932 (1.4)	1933 (0.7)
Tobacco consumption (g/wk)	107.4[c] (4.5)	0.1	86.1 (11.7)	72.3 (10.9)	105.9 (5.9)
No. of years smoked	37.7 (0.6)	<0.05	32.5 (2.4)	38.6 (1.4)	37.9 (0.8)
Projected tobacco lifetime dose (kg)	289.5 (12.5)	<0.05	216.7 (30.9)	193.6 (30.5)	285.1 (16.7)
Duration of asbestos exposure (yr)	21.2 (1.0)	<0.001	28.7 (2.0)	23.3 (2.6)	20.6 (1.1)
Cumulative asbestos exposure	43.7 (2.2)	<0.001	67.7 (9.4)	54.9 (7.3)	42.8 (2.7)
Tobacco consumption after 2 years (g/wk)	93.5[d] (4.2)		—	64.7[d] (9.3)	—

*Subjects who reported relapse during the 2 years are presented as a separate group. The exposure information on asbestos and tobacco is from the time of starting the intervention. Mean values with standard error of the mean in parentheses are given.
[a]Comparing mean values among smokers and quitters.
[b]Classified as smokers or nonresponders in Figure 1, since only six of them responded at the 2-year evaluation.
[c]One subject missing.
[d]Figure based on six subjects.

statistically significant. Absence of other smokers in the home, presence of lung or peptic disease, or cancer among first- or second-degree relatives was insignificantly associated with increased percentages of both quitters, and quitters and relapsers combined. Neither ischemic heart disease at any time, nor additional consultations, nor monitoring of expired carbon monoxide increased the likelihood of cessation.

DISCUSSION

We found that 5% among the responders, alternatively 3.5% among all invited subjects, had stopped smoking one year after the intervention. The RR of smoking cessation was RR = 2.2 (CI = 1.2–4.1) by comparison to historical controls. The increase was not significant when compared to the control group [RR = 1.7 (CI = 0.5–6.0)]. The continuing smokers at the 2-year evaluation had reduced their weekly tobacco consumption with an average of 14 g/week.

In contrast to our fairly comprehensive intervention, behavioral counseling lasting 3–5 min. among asbestos-exposed shipyard workers resulted in 8.4% abstinence over a 11-month period, compared to 3.6% after a minimal warning [Li et al., 1984]. An explanation of the modest effect following our intervention could be that the public attention on the adverse health effects of asbestos exposure during the time period from the mid-1970s may have stimulated smoking cessation. Consequently, the present target group may already have been "exhausted." Subjects may thus have been more resistant to antismoking counseling than average smokers [Rose and Hamilton, 1978; Hughes et al., 1981].

In another group of asbestos-exposed smokers (n = 2,627), information-based smoking intervention similar to

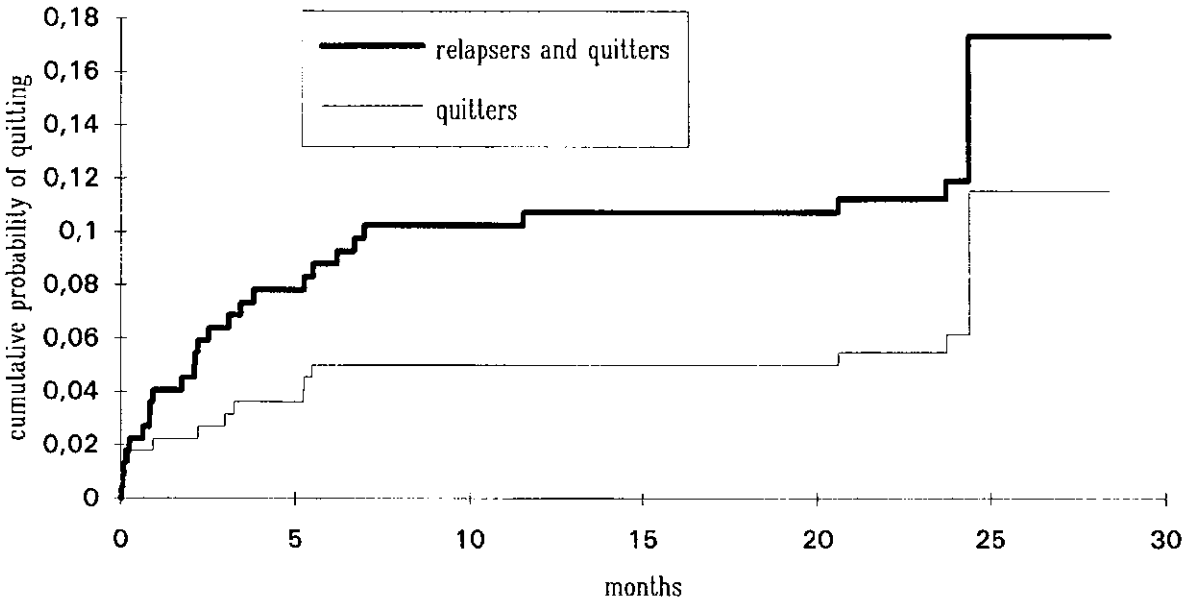

FIGURE 2. Successful quitting (quitters) and quitting attempts (quitters + relapsers), illustrating the effect and potential of the smoking cessation program in asbestos-exposed subjects. The time period was from the intervention until the 2-year evaluation.

ours, was applied [Kilburn and Warshaw, 1990]. After 6–25 months, 29.8% among the 19.2% responders had quit smoking. Telephone interview among 101 of the nonresponders indicated that 17.0% of them had stopped smoking. In historical controls the proportion of cessation was 4.7% for the year prior to intervention. It may be that this intervention had a greater effect than ours. However, the estimate of smoking cessation in nonresponders (17%) seems uncertain. It is likely that quitters are eager to report their success. Accordingly, the vast majority of quitters could be expected to respond to an evaluation. Possibly, nonresponders tend to state the most acceptable outcome, i.e. quitting, upon questioning. In order to deal with this problem in our study, we calculated conservative estimates of smoking cessation, assuming that nonresponders were continuing smokers. The possible bias introduced by assuming that ex-smokers are inclined to report, will be toward the null value. Moreover, the study by Kilburn and Warshaw actualizes the problem with comparison between studies. Participants in an intervention study are likely to be more motivated than the nonattenders. Thus, intervention approaches that attract few but highly motivated participants may seem more effective than other interventions, when only the participants are evaluated.

Those who had abandoned the smoking habit at the 2-year evaluation had the greatest asbestos exposure (Table III). Conversely, the quitters had lower values of the tobacco-related variables than continuing smokers, but at statistically insignificant or borderline significance levels. The results suggest that risk caused by asbestos exposure may motivate for smoking cessation, and indicate an effect of using risk-based estimates in the counseling.

Counseling by the primary health care had a more pronounced effect than counseling by a specialized institution in occupational medicine (Table IV). It seems likely that "the house doctor" is more convincing and thus provides counseling with more authority. The contact between patient and physician is often already established considering that 70% consult a general practitioner each year [Kottke et al., 1988]. At such consultations, subjects at risk can easily be identified by a brief routine interview. Repetitive and reinforced intervention, as well as maintenance can be applied [Kottke et al., 1988; Russell et al., 1988]. Subjects can be reached irrespective of their motivation. Only 27% of the smokers who consult a general practitioner experience anti-smoking advice. Accordingly, the potential for intervention by the primary health care is significant [Silagy et al., 1992].

Coronary heart disease has been reported as a predictor for smoking cessation [Rose and Colwell, 1992; Freund et al., 1992; Burt et al., 1974]. Further, chronic bronchitis may be more prevalent in ex-smokers than in smokers [Kato et al., 1989]. In the present study, it could not be demonstrated that prevalent lung disease, peptic disease, or ischemic heart disease enhanced smoking cessation. Those who have ever had ischemic heart disease may have been subjected to more anti-smoking advice than those with current lung or peptic disease, and may thus constitute a hard core of smokers.

Previous cessation attempts have been shown to facilitate abstinence [Kottke et al., 1988; Daughton et al., 1990], which was also found in the present study. Prescription of

TABLE IV. Percentage of Quitters and "Quitters and Relapsers" After 2 Years Related to Risk Factors at Time of Intervention and Modalities of the Intervention Program Among Asbestos-Exposed Smokers

		Quitters (n = 14)			Quitters and relapsers (n = 26)		
	n	%	SE diff%[a]	Diff% (CL)[b]	%	SE diff%[a]	Diff% (CL)[b]
Cancer among relatives							
Yes	147	6.8	3.4	1.7 (−4.9–8.2)	11.7	4.5	0.3 (−8.4–9.0)
No	79	5.1			11.4		
Current lung disease							
Yes	24	12.5	5.2	7.1 (−3.1–17.3)	16.7	6.9	5.8 (−7.7–19.3)
No	202	5.4			10.9		
Ischemic heart disease at any time							
Yes	29	0	4.8	−7.1 (−16.5–2.3)	6.9	6.4	−5.3 (−17.8–7.2)
No	197	7.1			12.2		
Current peptic disease/symptoms							
Yes	17	11.8	6.1	6.1 (−5.8–18.0)	17.7	8.1	6.7 (−9.1–22.5)
No	209	5.7			11.0		
Stopped smoking previously[c]							
Yes	61	11.5	3.6	7.3 (0.2–14.4)	16.4	4.8	6.7 (−2.7–16.1)
No	165	4.2			9.7		
Other smokers residing in the home [d]							
Yes	59	5.1	5.1	−8.5 (−18.6–1.6)	10.2	5.9	−5.9 (−17.4–5.6)
No	81	13.6			16.1		
Nicotine gum prescription							
Yes	78	9.0	3.4	4.3 (−2.3–10.9)	17.9	4.5	9.8 (1.1–18.5)
No	148	4.7			8.1		
Carbon monoxide monitoring[e]							
Yes	94	3.2	3.1	−3.1 (−9.2–3.0)	4.3	4.3	−10.4 (−18.8–−2.0)
No	95	6.3			14.7		
More than one consultation							
Yes	88	5.7	3.3	−0.8 (−7.3–5.7)	14.8	4.4	5.4 (−3.1–13.9)
No	138	6.5			9.4		
Primary health care setting							
Yes	37	13.5	4.3	8.7 (0.2–17.2)	21.6	5.7	12.1 (0.9–23.4)
No	189	4.8[f]			9.5[g]		

[a]Standard error of the difference between percentages.
[b]Difference between percentages (yes and no). Significant at 95% level if the confidence limits in parentheses are above zero.
[c]Abstinence exceeding 3 months.
[d]Information available on 140 subjects.
[e]Measurements were not obtained for residents in one of the municipalities.
[f]6.5% in the municipality, where subjects with any asbestos exposure and any projected lifetime tobacco consumption were invited, 3.0% and 3.3% in the two other municipalities.
[g]15.2% in the municipality where subjects with any asbestos exposure and any projected lifetime tobacco consumption were invited, 4.5% and 3.3% in the two other municipalities.

nicotine polacrilex may have enhanced smoking cessation and also contributed to its maintenance [Ockene, 1987; Fortmann et al., 1988]. However, the desire for polacrilex in the present study may have been an expression for motivation and not a cause of quitting. Although most subjects expressed that the measuring of expired carbon monoxide with a portable monitor was illustrative, a positive effect as reported by others [Jamrozik et al., 1984] was not found in the present study.

In conclusion, this study indicates that quitting smoking is hard even for those at increased risk of lung cancer due to a combined exposure to asbestos and smoking. For those with greatest asbestos exposure, an individual risk-based intervention resulted in enhanced motivation for smoking cessation. The results should stimulate efforts for further developing an appropriate intervention strategy for asbestos-exposed smokers in order to reduce their high lung cancer risk. Maintenance management of the quitters should be

promoted, and a strategy delivered by the primary health care services seems preferable.

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Increasing incidence of malignant mesothelioma after exposure to asbestos during home maintenance and renovation

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Objective: To determine trends in incidence of malignant mesothelioma (MM) caused by exposure to asbestos during home maintenance and renovation.

Design, setting and participants: Using the Western Australian Mesothelioma Register, we reviewed all cases of MM diagnosed in WA from 1960 to the end of 2008, and determined the primary source of exposure to asbestos. Categories of exposure were collapsed into seven groups: asbestos miners and millers from Wittenoom; all other asbestos workers; residents from Wittenoom; home maintenance/renovators; other people exposed but not through their occupation; and people with unknown asbestos exposure; or no known asbestos exposure. Latency periods and age at diagnosis for each group were calculated and compared.

Results: In WA, 1631 people (1408 men, 223 women) were diagnosed with MM between 1960 and 2008. Since 1981, there have been 87 cases (55 in men) of MM attributed to asbestos exposure during home maintenance and renovation, and an increasing trend in such cases, in both men and women. In the last 4 years of the study (2005–2008), home renovators accounted for 8.4% of all men and 35.7% of all women diagnosed with MM. After controlling for sex and both year and age at diagnosis, the latency period for people exposed to asbestos during home renovation was significantly shorter than that for all other exposure groups, but the shorter follow-up and difficulty recalling when exposure first occurred in this group may partly explain this.

Conclusions: MM after exposure to asbestos during home renovation is an increasing problem in WA, and these cases seem to have a shorter latency period than other types of exposure. MM cases related to renovation will probably continue to increase because of the many homes that have contained, and still contain, asbestos building products.

Malignant mesothelioma (MM) of the pleura or peritoneum is a universally fatal disease predominantly caused by exposure to asbestos. In Australia, the incidence of MM has increased steadily since the early 1960s, initially affecting workers mining and milling raw asbestos and manufacturing asbestos products (the first wave), and then workers who used asbestos products in industry (the second wave). Over the past 20 years, there has been increasing concern about a third wave — people diagnosed with MM after short-term and/or low-level exposure to asbestos in the home or workplace.[1] Home maintenance and renovation involving asbestos-containing building products is one of the activities most frequently associated with this third wave. It is a source of ongoing concern, given the widespread distribution of asbestos-containing products in homes and other buildings in Australian cities and towns.

Two forms of asbestos — serpentine (chrysotile or white asbestos) and amphibole (crocidolite or blue asbestos, and amosite or brown asbestos) — have been mined in and imported into Australia. Chrysotile was the main form of asbestos mined in Australia until crocidolite was mined at Wittenoom in the north of Western Australia, beginning in 1943 and continuing until 1966. More than 60% of the crocidolite produced was used in the manufacturing of asbestos cement products. For many years, Australia also imported both raw asbestos and manufactured asbestos goods. By 1954, Australia was ranked fourth among Western countries (after the United States, the United Kingdom and France) for gross consumption of asbestos cement products. However, on a per capita basis, Australia was top of the list.[2]

After World War II, asbestos cement products were commonly used as a building material in Australia. Asbestos cement products used in building include fibro sheeting; water, drainage and flue pipes; roofing shingles and guttering. Until the 1960s, 25% of all new homes were clad in asbestos cement.[2] The use of asbestos was slowly phased out in the 1970s and 1980s, but it is still found in structures built in the late 1980s. A total ban on the use of any type of asbestos was not introduced in Australia until 2003.

Direct occupational exposure to raw asbestos or asbestos products remains the predominant cause of MM, and the number of cases is not expected to peak until 2020.[3] However, with the ban on mining and asbestos use, the number of occupational cases will decrease over the next 20–30 years.[3] On the other hand, MM cases as a result of non-occupational exposure to asbestos are increasing, and there is little understanding of when, and at what level, this third wave will peak.

We describe here the changing trend in incidence of MM in WA, and the increasing numbers and relative proportions of people with MM whose exposure is unrelated to their occupation, especially those exposed to asbestos during home maintenance and renovation.

Cases were identified from the Western Australian Mesothelioma Register. The Register was formally established in 1982, although earlier

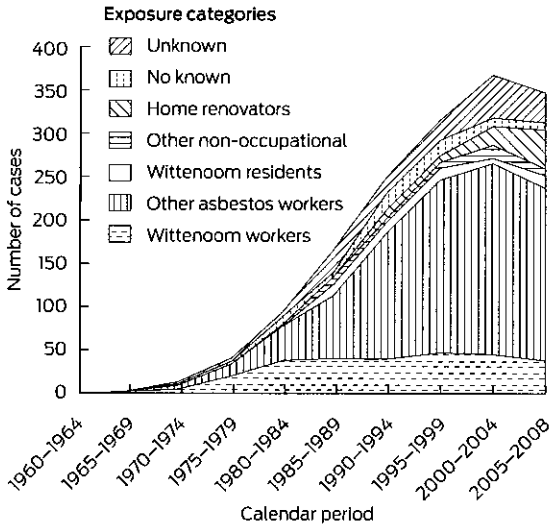
1 Malignant mesothelioma in Western Australia, by exposure category and sex, 1960–2008

Exposure category	Total cases (%)	Men (%)	Women (%)
Asbestos workers			
Wittenoom workers	290 (17.8%)	270 (19.2%)	20 (9.0%)
Other asbestos workers	913 (56.0%)	896 (63.6%)	17 (7.6%)
Non-occupational exposure			
Wittenoom residents	58 (3.6%)	28 (2.0%)	30 (13.5%)
Other non-occupational	50 (3.1%)	13 (0.9%)	37 (16.6%)
Home renovators	87 (5.3%)	55 (3.9%)	32 (14.3%)
Exposure source not identified			
No known	75 (4.6%)	40 (2.8%)	35 (15.7%)
Unknown	158 (9.7%)	106 (7.5%)	52 (23.3%)
Total	**1631 (100%)**	**1408 (100%)**	**223 (100%)**

versions existed from 1960. Since 1960, when the first person was diagnosed with mesothelioma, every MM case in WA has been recorded, and these are now included in the Register. Each case has been reviewed at periodic meetings of the Western Australian Mesothelioma Register Committee, comprising a pathologist, an occupational physician, a respiratory physician, an epidemiologist, the manager of the Western Australian Cancer Registry and a research officer.

The WA Mesothelioma Register entry for each case includes age, sex, and date, as well as methods of diagnosis, histological type, site of disease, date of death, and available history of asbestos exposure. The written report of the pathologist responsible for the cytological or histopathological diagnosis of each case is reviewed to confirm the diagnosis and, in difficult cases, the original diagnostic material is reviewed by the Register's pathologist, and clinical and radiological information is also considered.

2 Malignant mesothelioma in Western Australia, by exposure category and calendar period, 1960–1964 to 2005–2008



Classification of exposure

Until the late 1980s, a questionnaire detailing occupational and non-occupational exposure to asbestos was completed for as many MM cases as possible. However, questionnaires are no longer used, and exposure information is gathered from sources such as clinical notes, doctors' letters, and advocacy groups. If possible, patients are still asked about occupational histories, including descriptions of tasks involving exposure to asbestos, as well as residential or other non-occupational exposure.

In reviewing each case, the Mesothelioma Register Committee seeks to classify the source of asbestos exposure. There are 29 exposure codes (22 occupational, five non-occupational, plus "unknown" and "no known"). "No known" exposure is coded if the person has been intensively questioned, but no source of exposure to asbestos can be identified. "Unknown" exposure is coded if the person has not been questioned at all, or if some source of asbestos exposure has been noted but sufficient details of the exposure are lacking. The five codes for different types of non-occupational or residential exposure include a code for "handyman, home maintenance and do-it-yourself (DIY)" exposure.

If there is more than one source of asbestos exposure, the committee considers the most significant exposure for coding, taking account of when the exposure occurred, and how much exposure was involved. For example, occupational exposure would usually be considered more significant than non-occupational exposure. To be coded as having "handyman, home maintenance and DIY" exposure means that no other source of exposure could be identified. In such cases, exposure has been during "participation in home renovations/home maintenance or as a bystander while such activities occurred". If possible the date of first exposure is also recorded; in some cases, the date of first exposure to any asbestos may be earlier than the date of the most significant exposure.

For this report, the coding was collapsed from 29 categories to seven. There were two occupational categories: asbestos miners and millers from Wittenoom (*Wittenoom workers*); and all other asbestos workers (*other asbestos workers*); three non-occupational categories: residents from the town of Wittenoom (*Wittenoom residents*); handyman, home maintenance and DIY (*home renovators*); and other types of non-occupational exposure (*other non-occupational*); and people whose exposure could not be identified (*unknown*) or who had no known exposure (*no known*).

Statistical analyses

MM cases coded as home renovators, for the whole group and separately for men and women, were grouped by sex, 5-year age-groups from 40 years onwards (with 85 and over as the oldest group), and 5-year periods from 1980 to the end of 2008 (except for the final 4-year period, 2005–2008).

A Poisson regression model was used to examine the changing trend in incidence over time. The log of the WA population was used as the offset variable in the model. Comparisons of age at diagnosis and latency periods between the five exposure groups — two occupational and three non-occupational — were calculated using linear regression, controlling for sex and year of diagnosis. Linear regression analyses were performed with SPSS version 17.1 (IBM, Armonk, NY, USA) and Poisson regression analyses

with Stata version 10.1 (StataCorp, College Station, Tex, USA).

Ethics approval

The study was approved by the Department of Health WA Human Research Ethics Committee.

Between 1960 and December 2008, there were 1631 cases of MM in WA (1408 men and 223 women), 1562 of whom have died. There were 1510 cases of pleural MM (1305 men), 114 cases of peritoneal MM (97 men) and seven cases of MM at other sites (six men).

Occupational exposures were the main source of exposure to asbestos for men (82.8%) but not for women (16.6%) (Box 1). There were about 10% of cases (158/1631) with unknown exposure and about 5% (75/1631) with no known exposure.

Malignant mesothelioma and non-occupational asbestos exposure

A total of 195 cases (96 men) were associated with non-occupational exposures. Fifty-eight cases (28 men) were ex-residents of Wittenoom, 87 (55 men) were home renovators and in 50 cases (13 men) their "other non-occupational" exposure included visiting Wittenoom (15 [nine men]); living with an asbestos worker (22 [three men]) and various other residential exposures, such as dusting asbestos louvres or playing (as a child) in sheds used to store asbestos cement products (13 cases [one man]). For men, non-occupational exposure accounted for 6.8% of all cases; for women, it accounted for 44.4% (Box 1).

The first case of MM associated with exposure attributed to home maintenance and renovation was registered in 1981. Of the 87 cases recorded, 55 men and 32 women, 84 had pleural MM (53 men) and three peritoneal MM (two men). There has been a steady increase in both the number and incidence rates of home maintenance/renovation cases since the mid 1980s (Box 2). Incidence rates for the last two periods (2000–2004 and 2005–2008) were significantly higher than the base rate (1980–1984) (Box 3, Box 4). For both men and women, home renovators now constitute the largest proportion of all non-occupational cases. Between 2005 and 2008, 8.4% of MM cases in men and 35.7% of those in women were attributed to home renovation.

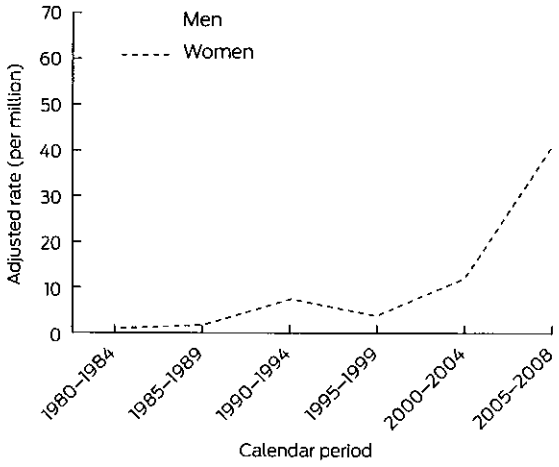
After controlling for sex and both year and age at diagnosis, the latency period for home renovators was found to be significantly shorter than that for all other groups (Box 5). At diagnosis, home renovators were older than the people in the other two non-occupational groups but slightly younger than those in the two occupational groups (Box 5).

The number of cases of MM in WA is still increasing, although the number associated with occupational asbestos exposure appears to be reaching a plateau. However, MM cases associated with home maintenance and renovation have increased markedly over the past 10 years and remain on an upward trend. Most of the exposures reported in this group occurred in the 1960s and 1970s, but many WA homes still contain asbestos building products and home renovations have continued, and possibly increased, since that time. Therefore, the potential for MM cases from home renovation exposure to continue to increase remains a concern. It is not possible to predict for how long this increasing trend will continue, as there are no published data on past or current community exposure to asbestos.

Most MM cases attributed to home renovation have occurred in men, although this type of exposure as a proportion of all cases is much higher in women. For both men and women, there has been a marked increase in MM cases related to exposure from home maintenance and renovation over the past two decades. For men, the proportion of home renovation cases increased from about 3% in the 1990s to over 8% over the last 4 years of the study. For women, home renovation cases have increased from around 5% of all cases in the 1990s to over 35% for the period 2005–2008. Of all known exposures, home maintenance and renovation is the main cause of MM in women. For men, occupational exposures remain the dominant cause of MM, but home maintenance and renovation is the most important non-occupational exposure.

To be included in the home renovator category, people had to have been exposed to asbestos either while performing simple renovations to their homes or, as family members, to have been exposed while these activities took place. The types of reported activities in this category included

3 Age-adjusted increase in relative incidence rates of malignant mesothelioma attributed to asbestos exposure during home renovation — Western Australia



4 Relative change, 1980–1984 to 2005–2008, in incidence rates of malignant mesothelioma attributed to asbestos exposure during home renovation (adjusted for age and sex) — Western Australia

Period	Incidence rate ratios (95% CI)	P
1980–1984	1.00	
1985–1989	1.87 (0.17–20.59)	0.610
1990–1994	8.01 (1.02–62.55)	0.047
1995–1999	4.16 (0.50–34.59)	0.187
2000–2004	13.03 (1.76–96.66)	0.012*
2005–2008	44.96 (6.19–326.32)	0.001*

* Incidence rates for 2000–2004 and 2005–2008 are significantly higher than the base rate (1980–1984). ◆

5 Adjusted mean latency period between exposure and diagnosis of malignant mesothelioma in Western Australia, by exposure category and mean age at diagnosis

Exposure category	Latency period, years (95% CI)*	Age at diagnosis, years (95% CI)†
Wittenoom workers	36.9 (31.4–42.3)‡	68.2 (63.8–72.7)
Other asbestos workers	39.8 (34.3–45.2)‡	70.4 (66.1–74.7)
Wittenoom residents	43.7 (38.0–49.5)‡	57.6 (52.7–62.5)‡
Other non-occupational	39.7 (33.9–45.6)‡	61.6 (56.7–66.7)‡
Home renovators	33.1 (27.5–38.8)	66.5 (61.9–71.1)

* Adjusted for sex, age at diagnosis and year of diagnosis. † Adjusted for sex and year of diagnosis. ‡ Significantly different from home renovation group ($P < 0.05$). ◆

sanding asbestos cement walls in preparation for painting; lifting linoleum floors; replacing "tilux" (asbestos cement used in place of ceramic tiles) in bathrooms; and using asbestos cement sheeting for putting up fences and sheds, extending laundries, and enclosing verandas to create "sleep-outs". Some of these activities, particularly those involving the use of power tools, can produce short-term, high concentrations of asbestos fibres,[4] and major renovation works may increase background fibre concentrations in the medium term, contributing to increased cumulative exposure.[4] In most instances in this series, exposure was limited to a single task, which may have lasted for only a few days.

Based on reported first exposures, the home renovator group had the shortest estimated latency period. Latency periods have mostly been observed to be shorter in groups with occupational rather than non-occupational exposure.[5-7] There are two reasons which may explain why we found a shorter latency period for MM cases with home renovation exposure.

First, there has been a shorter follow-up period of this group. Exposure in the home renovator group started in the 1960s, while exposure for the occupational groups commenced in the mid 1940s and early 1950s. As time from exposure increases so, necessarily, does the average latency period.

Second, recalling when exposure first occurred is difficult, particularly for non-occupational exposures. This is likely to be reflected in the greater proportion of women, compared with men, who could not recall any asbestos exposure ("no known" exposure) (Box 1). This difficulty in recalling non-occupational exposures means that some patients may have been exposed to asbestos before the recorded renovation episode and therefore their latency period has been underestimated.

With the wide use of asbestos products in Australian homes after World War II,[2] the exact number of homes containing asbestos cement or other asbestos products is not known accurately. In one survey, 62.9% of homes in the Australian Capital Territory were found to contain asbestos.[8] It was more common in older homes, with over 70% of homes built before 1965 containing asbestos, but was found in fewer than 1% of homes built after 1984.[8] A recent survey of Australian adults found that over 80% of respondents reported exposure to asbestos either at work or at home.[9]

Home renovation is a common activity in Australia.[10,11] In a survey of home owners in Adelaide, major renovations were undertaken in about 34% of homes over a 5-year period (1986–1991).[11] Renovations were more common in older homes, particularly those over 50 years old.[11] In the 10-year period to 1999, 66% of homes across Australia, built between 1920 and 1949, had been renovated.[10] In the Australian survey on potential asbestos exposure mentioned above, only about a third of respondents reported taking precautions to reduce exposure to asbestos fibres or dust in their homes.[9] Although a Code of Practice for the safe removal of asbestos has been published,[12] advice contained in the code is directed to owners of large buildings and asbestos removalists and not to small operators, individual tradesmen or home renovators.[13]

Our study confirms the rising trend in diagnosis of MM resulting from exposure to asbestos during renovation activities in and around the home. The continued widespread distribution of asbestos cement products in WA homes, and the long latency period between exposure and diagnosis of MM, means that there is likely to be a further increase in cases of MM attributable to home renovations.

Competing interests: No relevant disclosures.

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the extremes of incomprehensibility. The Commission is nevertheless concerned that its arguments should be comprehensible and its recommendations unambiguous—aims best achieved by a clear prose style, unencumbered by jargon and circumlocution. If it is readable, even to the uninitiated, this target will have been reached. No work of literature has ever been written by a committee, and anyone who has experienced the attempts of a group to compose a flowing paragraph knows its impossibility. The style of the final report therefore depends particularly on the secretary, but members are free to offer amendments or even large-scale rewrites if they feel so disposed. This is mostly done in correspondence, and the tactful secretary incorporates these offerings judiciously. Arresting phrases or a few incisive sentences are often interpolated in this way.

The end in sight

Unless there is a determined minority, acrimony recedes by subsequent meetings, for the end is in sight. Giggles sometimes break out and the Commission's own family jokes are heard more often; murmurs of a final party or dinner after the signing ceremony are heard. As the drafts improve in style, compromises over the sticky parts are reached, and at last a final draft is agreed. A very senior civil servant reads it and talks to the Commission about obscurities and difficulties. Then the smooth machinery of Whitehall takes over. The chairman utters grave warnings against "leaks," intended or inadvertent. A date for release has to be considered and press conferences are arranged. The date must not clash with any expected public event, for the Commission does not want its press impact to be diminished. Each member has to state precisely the form in which his name, style, and titles are to be published. While members begin to relax, the chairman tenses. A government reception is given on the day of signing, with cocktail party courtesy from important figures, longing to know what has been said but forbidden to ask. On the day before publication the members each receive a printed copy of the volume, but without its blue cover to indicate its still unofficial status. Finally, publication day dawns, with eager perusal of press response, and then—a slow decline into obscurity.

Occasional Review

The natural history of chronic airflow obstruction

CHARLES FLETCHER, RICHARD PETO

British Medical Journal, 1977, **1**, 1645-1648

Summary

A prospective epidemiological study of the early stages of the development of chronic obstructive pulmonary disease was performed on London working men. The findings showed that forced expiratory volume in one second (FEV_1) falls gradually over a lifetime, but in most non-smokers and many smokers clinically significant airflow obstruction never develops. In susceptible people, however, smoking causes irreversible obstructive changes. If a susceptible smoker stops smoking he will not recover his lung function, but the average further rates of loss of FEV_1 will revert to normal. Therefore, severe or fatal obstructive lung disease could be prevented by screening smokers' lung function in early middle age if those with reduced function could be induced to stop smoking. Infective processes and chronic mucus hypersecretion do not cause chronic airflow obstruction to progress more rapidly. There are thus two largely unrelated disease processes, chronic airflow obstruction and the hypersecretory disorder (including infective processes).

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Introduction

Chronic bronchitis and emphysema are often referred to together as the "British disease" because they are such a common cause of death and disability in Britain. Since their cardinal feature is irreversible obstruction to bronchial airflow, they are often referred to jointly as chronic obstructive pulmonary disease. This term includes chronic obstructive bronchitis and emphysema but excludes asthma or any localised cause of airways obstruction.[1]

Although the number of deaths certified as being due to these conditions has declined in the past 10 years, there were still some 25 000 in England and Wales in 1974. There were also about 1000 deaths due to respiratory heart disease plus an unknown number, perhaps as many as 10 000, certified as being due either to other forms of heart disease or to pneumonia where chronic obstructive pulmonary disease was not certified as the underlying cause of death even though it caused the fatal condition or aggravated a condition that would not otherwise have been fatal. The total mortality attributable to chronic obstructive pulmonary disease is thus about the same as the total mortality attributed to lung cancer. If it were possible to identify all deaths that would not have occurred in the absence of chronic obstructive pulmonary disease it would probably be found that the proportion misleadingly certified as being due to other underlying causes is even larger in other countries, including the USA, than in Britain.[2] Although the certified death rates in other countries are lower than those in Britain, they

therefore represent only a fraction of the total mortality actually attributable to chronic obstructive pulmonary disease.

When airflow obstruction first causes breathlessness that leads a patient to consult a doctor, it is usually sufficiently severe to reduce the forced expiratory volume in one second (FEV_1) to about 1 litre, which is less than half the normal value. Thereafter the course of the condition usually progresses relentlessly over five or more years, with further loss of FEV_1 causing more and more distressing disability and, finally, death from respiratory failure. This often occurs in an episode of bronchial infection complicated by cor pulmonale.

These later phases of the disease have long been well documented[2-5] and it has been found that the severity of airflow obstruction, usually measured by FEV_1, is the main determinant of prognosis.[6-8] Since the damage to the lung appears to be irreversible at this late stage of the disease, any preventive action must be taken much earlier. The essential role of smoking has long been clear,[9-13] but stopping smoking in the terminal stage is too late,[5 6] and general health education has not had much effect on the people (male manual workers) who suffer the greatest risk of this disease.[13] Perhaps it could be more effective if concentrated on potential patients at an earlier stage, but how could they be identified?

In the late 1950s[14] and again more recently[15] it was suggested that such people could be recognised by their having a productive cough (simple bronchitis). Pathologists suggested that mucus hypersecretion encouraged bronchial infection, which caused obstructive damage to bronchioles and alveolar tissues.[16-18] The fatal consequences of infections in terminal patients with terminal obstruction lent plausibility to this latter view, but it remains an unproved hypothesis.

In 1960 the Medical Research Council's committee on the aetiology of chronic bronchitis became concerned with the question of how smoking interacts with other factors in causing airflow obstruction and commissioned a prospective study of respiratory symptoms and changes in ventilatory function over a period of eight years in a large group of working men, few of whom had any clinical disease. The full results of this study were recently published[19] together with some new statistical considerations.[20] We report here a short summary of the methods and main results and conclusions of this study, some of which conflict with current orthodoxy, to stimulate debate in a wider circle than those who will read a specialist epidemiological monograph.

Methods

In 1961 a stratified random sample of men (mostly skilled manual or clerical) aged 30-59 working in West London was taken. Of an initial sample of 1136 men 792 were seen regularly enough over the next eight years to provide sufficient data for analysis. The men were seen every six months, when the following measurements were made.

Mucus hypersecretion was assessed by standard questions about chronic phlegm production and by six-monthly measurements of the volume of phlegm brought up during the first hour after waking on three separate mornings. These two independent measures enabled us to rank the men with respect to chronic expectoration more reliably than has been done in other studies, in nearly all of which single estimates based on questionnaires have been used.

Bronchial infections were assessed by standard questions about chest colds or illnesses in the previous six months during which phlegm production had increased; by recording the purulence of all phlegm specimens posted to us; and by measuring serum antibodies to *Haemophilus influenzae* on one occasion.

Airflow obstruction was estimated by measuring FEV_1. After two practice blows into a spirometer the FEV_1 readings of three subsequent blows were recorded. The maximum of these three was used, contrary to MRC recommendations,[21] because it was definitely more reproducible than the mean (p 164[19]). These six-monthly FEV_1 measurements over eight years allowed us to estimate the average rate of decline of FEV_1 for each man during the study. These estimates are called "FEV_1 slopes." Unfortunately, FEV_1 slopes of individuals could not be measured accurately enough to be useful, but averages of the FEV_1 slopes of groups of a dozen or more men were accurate enough for our analysis of causal factors. To ensure that FEV_1 loss was a valid measure of development of airflow obstruction 18 men with conditions that could cause restrictive loss of FEV_1 were excluded.

Results and comment

SMOKING AND LOSS OF FEV_1

The following conclusions are summarised in figs 1 and 2.

Firstly, we found that FEV_1 declines continuously and smoothly over an individual's life (fig 1). We believe that sudden large irreversible falls are very rare, for the 9190 measurements that we made of the changes in FEV_1 between successive six-monthly surveys were distributed exactly symmetrically about their mean, with no evidence of any "tail" due to sudden substantial losses (p 224[19]). The rate of loss seems to accelerate slightly with aging (p 67[19]).

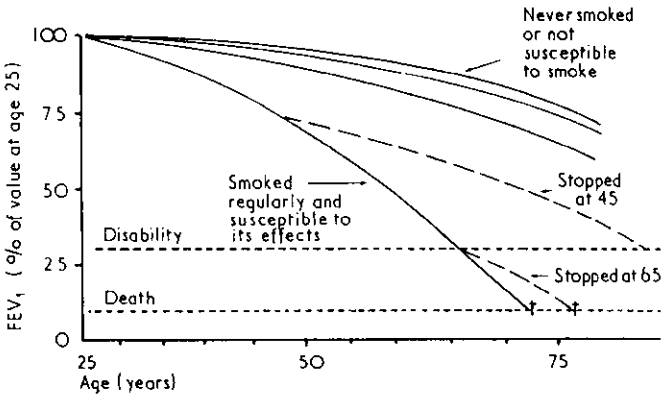

FIG 1—**Risks for various men if they smoke: differences between these lines illustrate effects that smoking, and stopping smoking, can have on FEV_1 of man who is liable to develop chronic obstructive lung disease if he smokes. † = Death, the underlying cause of which is irreversible chronic obstructive lung disease, whether the immediate cause of death is respiratory failure, pneumonia, cor pulmonale, or aggravation of other heart disease by respiratory insufficiency. Although this shows rate of loss of FEV_1 for one particular susceptible smoker, other susceptible smokers will have different rates of loss, thus reaching "disability" at different ages.**

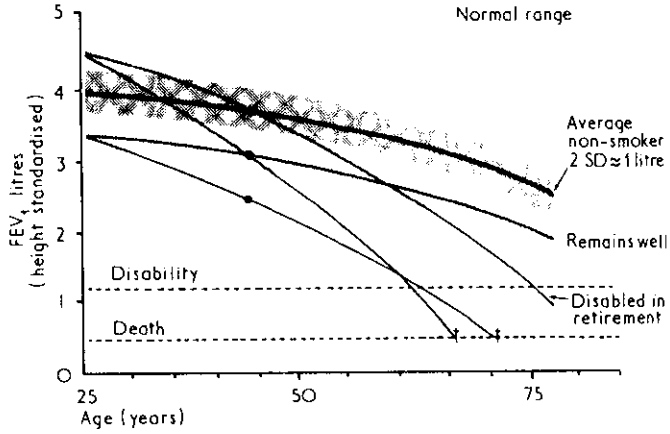

FIG 2—**Identifying susceptible smokers in time to prevent death: various patterns of FEV_1 decline (—) with age that are consistent with certain observations of FEV_1 in middle age (●). Smokers who eventually die of chronic obstructive lung disease have usually already suffered appreciable FEV_1 loss in their 40s. Most smokers whose FEV_1 is already below the normal range for non-smokers by early middle age are thus at grave risk of later death from airflow obstruction unless they stop smoking immediately, while smokers whose FEV_1 is still above average in middle age will probably not get serious obstruction. If, however, FEV_1 at age 25 was originally above average for other men (of the same age and height) then FEV_1 may still lie within the normal range for middle-aged non-smokers even though considerable FEV_1 loss has occurred. It is therefore impossible to be sure of the prognosis of a smoker whose FEV_1 in middle age is just one or two standard deviations below the average for non-smokers, although many of those around two standard deviations below average will become disabled over the coming decades. Other tests may enable those at greatest risk to be detected.**

Secondly, non-smokers lose FEV_1 slowly and almost never developed clinically significant airflow obstruction. None of the 103 non-smokers in our study had any evidence of even moderate obstruction (p 83[18]).

Thirdly, many smokers lose FEV_1 almost as slowly as non-smokers and never develop clinically severe airflow obstruction. They appear to be largely resistant to the effects of smoke on their airflow. Smokers who are more susceptible to these effects develop various degrees of airflow obstruction, which in some ultimately becomes disabling or fatal. "Susceptibility" is not an all-or-nothing attribute: rather, it appears to be a continuum, where the more susceptible a man is the sooner he will be disabled *if he smokes* (p 210[19]).

Fourthly, stopping smoking will, of course, make little difference to the FEV_1 of a non-susceptible smoker whose lungs are not being much affected by his smoking. But it may make all the difference to a susceptible smoker. A susceptible smoker who stops smoking will not recover lost FEV_1, but the subsequent rate of loss of FEV_1 will revert to normal. This finding is based on a small group of men, but it has been reported by Comstock *et al*[22] and is strongly supported by both the low death rate from bronchitis and emphysema among smokers who have given up more than 10 years earlier (observed in the major prospective studies of smoking and health[9-11]) and the minor degrees of emphysema found by pathologists in dead ex-smokers.[23-25] It is, of course, true that severely affected patients derive little benefit from stopping[6 7] because the damage already done to their lungs is by then severe, and merely slowing its further development will not restore adequate function. The quantitative aspects of these effects of smoking on FEV_1 are summarised in the table, where the men aged 50-59 at the start of our study were divided into those who did and those who did not have mild airflow obstruction, as indicated by a slightly low FEV_1 for their age and height. The percentages of men with such airflow obstruction were: 0% of lifelong non-smokers; 28% of ex-smokers (some of whom had probably stopped because of moderate disability); 24% of light smokers (less than 15 cigarettes per day); 46% of heavy smokers (15 or more cigarettes per day). The means of the FEV_1 slopes of non-smokers and of ex-smokers (whether obstructed or not) were similar. The non-obstructed smokers had slightly steeper slopes, and the obstructed smokers had much steeper slopes.

Among smokers who have already developed moderate obstruction, the effect of giving up in early middle age will presumably be to make their subsequent rate of loss of FEV_1 approximate to that of the obstructed ex-smokers in the table instead of that of the obstructed smokers. This twofold difference in mean FEV_1 slope may not seem very impressive, but, as indicated by the line in fig 1 marked "Stopped at 45," it can make the difference between a normal lifespan and premature death. The average effect of stopping is, of course, small since most smokers are not very susceptible and so have normal lungs that do not benefit much from stopping smoking. Those who continue smoking until they are disabled (see, for example, the line marked "Stopped at 65") will also derive little benefit. The important finding is that if those who would eventually die from airflow obstruction stop smoking in early middle age then their subsequent rates of loss of FEV_1 will on average be normal, so that most such individuals will keep well, whereas had they gone on smoking until they became short of breath it would have been too late.

Measuring FEV_1 might thus perhaps be used as a screening test to detect susceptible smokers in middle age, when the fact that the test showed them that smoking was damaging their lungs might help to persuade them to stop. (Care would have to be taken not to imply that smoking is safe for those smokers with normal lung function.) Peak expiratory flow is even quicker and cheaper to measure and so could also be used for screening. The disadvantage of both of these tests for screening (peak flow perhaps even more than FEV_1) is their wide range of normal values. As shown in fig 2, a man whose FEV_1 is near the lower end of the normal range for non-smokers may be at high risk or may be quite free from disease. Such borderline cases could be referred for more detailed lung function tests which might help to discriminate between "low normal" and "low abnormal" FEV_1 values. Preliminary results of a study[26] of functional tests to diagnose small airways disease suggest that the best tests for this purpose would be the airflow rate as forced expiration nears completion —the Vmax 25—and the expiratory nitrogen slope, both of which can[27] be used as field screening tests.

The real effect of smoking on susceptible smokers may be underestimated by looking only at the mean FEV_1 level in all smokers (or the mean $FEV_1/height^3$ in the total column of the table), as is usually done in prevalence surveys. There are two reasons for this. Firstly, smoking has only a small effect on not-very-susceptible smokers, but they, being in a majority, conceal the more severe effect on the most susceptible minority. Secondly, we found that smokers with symptoms tend to cut down their cigarette consumption, so that many of those who are most susceptible, and thus most severely affected, appear among the lighter smokers or the ex-smokers.

EFFECT OF MUCUS HYPERSECRETION AND BRONCHIAL INFECTION

Neither mucus hypersecretion nor bronchial infection cause chronic airflow obstruction to progress more rapidly. This was shown in two ways. Firstly, we found that after adjusting for FEV_1 level, smoking, age, and height there was no independent correlation between FEV_1 slope and indices of either mucus hypersecretion (p 94[19]) or bronchial infections (p 87[19]). This suggests that neither can play any causal part in accelerating the development of chronic airflow obstruction. Since this was a surprising finding, we sought confirmation by looking at changes of FEV_1 level in relation to changes in expectoration and to episodes of bronchial infection in individual men, and no consistent or significant effects were found. The loss of FEV_1 that an individual man suffered from one six-monthly survey to the next was on average the same if a chest cold, chest illness, or attack of sputum purulence intervened as if it did not (p 91[19]). We are forced to conclude that neither mucus hypersecretion nor bronchial infections, as we measured them, play any substantial part in actually *causing* irreversible airflow obstruction. Moreover, the chief anatomical site of chronic mucus hypersecretion (the main bronchi) is different from the (peripheral) usual chief site of fatal airflow obstruction. We therefore feel that chronic airflow obstruction and chronic hypersecretion should cease to be viewed as closely related disease entities (p 141[19]). Both are caused by smoking, but they are otherwise largely unrelated conditions, chronic phlegm production being much less important. The terminology that refers to both conditions as one form or another of "chronic bronchitis" is unfortunately sanctioned by usage, but may lead to confusion: those terms that unmistakably refer to *either* the obstructive *or* the hypersecretory disorder are preferable. Infective processes are related strongly only to the hypersecretary disorder. But can we really dismiss infective processes as early causes of chronic airflow obstruction? Our negative evidence is very strong, and is supported by clinical studies,[28 29] while positive evidence of any effect

Mean FEV_1 1961-9 and FEV_1 slope 1961-9 according to smoking habits among men with and without mild obstruction who were aged 50—59 on entry to study. Data for men in late middle age are tabulated because health benefits obtained by giving up early in middle age depend on subsequent rates of loss later in middle age (ref 19; table G1)*

	With mild obstruction*			Without mild obstruction*			Total		
	% of such men	Mean $FEV_1/height^3$ (cl/m^3)	FEV_1 slope ±1SE (ml/year)	% of such men	Mean $FEV_1/height^3$ (cl/m^3)	FEV_1 slope ±1SE (ml/year)	% of such men	Mean $FEV_1/height^3$ (cl/m^3)	FEV_1 slope ±1SE (ml/year)
Lifelong non-smokers	0			100	65	−42±6	100	65	−42±6
Ex-smokers, 1961-9	28	44	−37±8	72	62	−30±5	100	57	−32±5
Light smokers (average <15 cigarettes/day)	24	41	−62±5	76	62	−42±3	100	57	−47±3
Heavy smokers (average ≥15 cigarettes/day)	46	43	−80±6	54	60	−55±6	100	52	−66±4
All men	29	42	−64±3	71	62	−42±2	100	56	−48±2

*The age-standardised $FEV_1/height^3$ was defined, in units of cl/m^3, by (mean FEV 1961-9)/ $height^3$ + 0·5 (age in 1965-60), and a cut-off point of 50 cl/m^3 was then imposed to define "mild obstruction." This cut-off point represents very mild obstruction indeed, for in a man of 1·71 metres aged 60 it would be 2·5 litres, and even a small percentage of lifelong non-smokers would, in a larger series fall below it.

is virtually non-existent. When challenged to produce evidence to support his contrary opinion,[30] the editor of the *British Medical Journal* could produce no data, just published opinions[21 31]—one of them being that of the MRC committee which organised the present study to test its opinions. If infections are an important cause of irreversible airflow obstruction, it should not be difficult to show this, but it has never been successfully done.* We suggest that those who disagree with, or want more details of, our present conclusions should consult the monograph in which our results are more fully set out and discussed.[19] If it is felt that some point would be clarified by a tabulation or correlation which has not been presented in our monograph, RP can probably provide this quite easily on request, especially if full and precise details of just what is wanted are specified.

The future

Our study has emphasised the importance of smoking in causing airflow obstruction and shown how it might be possible to detect susceptible smokers in time to prevent disability, but many problems remain. What is the basis of susceptibility? It does not seem to lie in overt allergy, for we found no correlation between FEV_1 slope and either sputum eosinophilia or a history of allergic illnesses. Nor does height increase susceptibility, as might be expected from mechanical stresses in the lung, for we, and Cole,[32] found that *percentage* losses of FEV_1 as men of different heights get older are similar. Is susceptibility in any way analogous to α_1-antitrypsin deficiency or due to quantitative differences in leucocyte proteolytic enzymes? Can it be induced by infections in childhood that are associated with impaired lung function?

What causes of obstruction other than smoking are there? The British decline in certified death rates from bronchitis and emphysema over the past three or four decades while cigarette smoking has increased indicates (unless these changes are chiefly due to differences in death certification practice for infective disease) that some important cause or agent must have been declining in severity. Was this just air pollution? The large social class gradient of mortality, which was (unless this, too, was severely biased by nosological artefacts) present long before there was any social class gradient in smoking, suggests that there must be causes related to style of living that have not yet been identified.

Our study has disposed of some misconceptions, and provided a simpler picture of the natural history of airflow obstruction. New ideas to be tested by prospective epidemiology will now be needed to further our understanding of this common, distressing, and often fatal disorder.

The study whose main findings we have described was financed by the Medical Research Council and organised by Dr Cecily Tinker. The analysis, in Sir Richard Doll's department, involved extensive use of the Science Research Council's Atlas Computing Laboratory. We are grateful to the unions, management, and men who participated for eight years, and to many others, particularly Mr I D Hill, Mrs H Joyce, Ms G Mead, Professor G A Rose, Dr F E Speizer, and Ms M Stuart.

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*Clinical histories given by patients with severe airflow obstruction often suggest an onset of the obstruction at the time of a particular chest illness.[3] An example is one man in our study who was admitted to Hammersmith Hospital in cor pulmonale (p 236[19]). On admission to hospital in 1967, he reported that he had been quite fit, even rowing with an amateur rowing club, until 1964, when he had pneumonia, but that after this he had had chronic disabling shortness of breath. Our study records, however, showed that his FEV_1 had already been only 1 litre in 1961 and that in 1961 he had said that he could not keep up with other men when walking on the level because of breathlessness. His FEV_1 declined steadily from 1 litre in 1961 to 0·2 litre just before his death in 1970 in respiratory failure. There was thus no sudden change in FEV_1 level after his pneumonia in 1964. Perhaps this pneumonia increased ventilatory demand on exertion but it did not increase his airflow obstruction. It is obvious how the history given in 1967 could mislead a clinician about the effect of this man's pneumonia on the development of airflow obstruction.

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What might be the cause of pain in the testicles after intercourse?

I think this man probably has referred pain from the lower lumbar spine. Lower lumbar disc pain can be referred to the groin or lower abdomen, including the hemiscrotum of the side in question. Clearly, we are assuming here that there is no clinical abnormality in the testis, cord, or hernial orifice, and that there is no abnormality on abdominal and rectal examination. There may be a history of backache or his testicular pain may be induced by strenuous exercise of the back under other circumstances. Treatment should be on the usual lines for a lumbar disc lesion and in this case his wife should take a more active role during intercourse until such time as a clinical improvement is reached.

My sphygmomanometer has a cuff calibrated to enable one to make reductions on the observed dial reading cccording to the circumference of the patient's arm. I recently saw a patient who was applying for life insurance whose uncorrected diastolic reading was 100 while his corrected diastolic reading was 89. Is one justified in giving the lower reading—that is, with cuff correction—when completing a life insurance report?

I do not believe that one would be justified in giving only the "corrected" lower reading of the blood pressure after measuring the blood pressure in a patient with large arm. It would be reasonable to give the actual blood pressure reading plus the suggested correction for arm size (clearly indicating that this was an extrapolated number and not the one that was actually measured).