

CORRESPONDENCE

Asbestos and cancer: history and public policy

Sir,—Weller's letter (1992;49:70-2) is so replete with error that it seems to me it would be appropriate to designate him as a negative correspondent—namely, somebody who is wrong more often than chance alone allows him to be.

Weller writes that "it is reasonable to expect that those concerned with mining and processing asbestos should have been alert to the growing body of medical opinion . . . to the established links between asbestos inhalation and serious diseases." One might ask, should they (the asbestos producers and if it comes to that the medical and scientific communities) have known in 1950 on the basis of 30 to 40 published cases of concomitant asbestosis and lung cancer, that there was a cause and effect relation? This was five or more years before Doll published his classic paper.¹ As for mesothelioma, the association between exposure to crocidolite and mesothelioma did not come to light until the paper by Wagner *et al* in 1960.² It was uncertain whether amosite was similarly carcinogenic, and this is what prompted Selikoff to carry out his study, which was subsequently published in 1972.³ Weller tells us that "Johns Manderville" (Johns Mansville?) and other companies should have read Gloyne's pertinent abstract! Gloyne certainly did not write the abstract! Weller does not reference Gloyne's paper, but I assume he refers to the description of two cases of lung cancer and asbestosis that were briefly mentioned in *The Lancet* in 1934⁴ and described more fully in *Tubercle* in 1935.⁵ The 1934 publication of Wood and Gloyne describes 21 cases of active tuberculosis in 100 subjects with asbestosis, an association that was much more statistically significant than the isolated two cases of lung cancer, but with further studies it became evident that tuberculosis and asbestosis were unrelated.

Weller goes on to write that Dreessen *et al* documented the significant risk that occurred in asbestos textile factories in 1938 and urged the elimination of hazardous exposure.⁶ In

reality, Dreessen *et al* realised that some persons in the North Carolina asbestos mills were developing asbestosis, but did not recommend elimination of hazardous exposure although doubtless he was in favour of such a policy. Their conclusion speaks for itself—namely, "that it would seem that if the dust concentration in asbestos factories can be kept below 5 m/p/cft TLV, new cases of asbestosis would not appear." It was not until 1965 and after that it became apparent that Dreessen *et al* were incorrect and new cases of asbestosis were occurring despite adherence to the 5 m/p/cft.^{7,8} Weller then states that the TLV of 5 m/p/cft was established "on the basis of abdominal (intra-peritoneal) injections into guinea pigs." This was not so and nowhere in published work is there such a statement. If Weller were to refer to the American Conference of Governmental Industrial Hygienists (ACGIH) deliberations he would know that the TLV was based on the Dreessen report.⁹ He then goes on to write of the problems distinguishing short and long fibres, but fails to realise that the standard methods of measuring dust exposure between 1930 and 1960 relied on counting particles, and did not separate fibres from other types of particles. The first reliable method of measuring airborne fibres was the British membrane filter, which was not adopted in Britain until the late 1960s. Fibre counting was not adopted by the United States government until 1971, and to this day the standard method of counting fibres (optical microscopy) does not distinguish asbestos fibres from those of cotton, talc, and other vegetable fibres. The ACGIH did not reduce their recommended level to 2 m/p/cft in 1968; their 1968 recommendations appeared as a notice of intended change. It was not until 1970 that the asbestos regulations were amended to 2 m/p/cft.

Contrary to what Weller says, the United States government has never accepted blame for asbestosis contracted in the United States naval shipyards and not a penny has been paid by the United States government to those who have suffered asbestos related impairment, disability, or death as a result of working either for or under contract with the United States navy. Instead, those companies that manufactured the asbestos products have been sued and many have gone out of business as a result. Moreover, the

United States navy was among those who adopted and recommended adherence to the 5 m/p/cft TLV.

Weller might not be aware that the "research" that went into Castleman's book was supported by claimants' lawyers, and indeed it was the same enlightened group of lawyers who invited various federal and state judges to attend a conference sponsored by the Collegium Rammazini, and in order to titillate the judges' interest and perhaps to annul their disinterest and impartiality, offered to waive the \$250.00 registration fee and provide free hotel accommodation.¹⁰

Finally, it would be advantageous to all concerned were Weller to read the papers he quotes in support of his views.

W K C MORGAN
Chest Diseases Unit,
University Hospital, PO Box 5339,
London, Ontario N6A 5A5, Canada.

- 1 Doll R. Morbidity from lung cancer in asbestos workers. *Br J Ind Med* 1955; 12:81-6.
- 2 Wagner J, Sleggs CA, Marchand P. Diffuse pleural mesothelioma and asbestos exposure in North West Cape of Province. *Br J Ind Med* 1960; 17:260-71.
- 3 Selikoff IJ, Hammond EC, Churg J. Carcinogenicity of amosite asbestos. *Arch Environ Health* 1972;25:183-6.
- 4 Wood WB, Gloyne SR. Pulmonary asbestosis: A review of 100 cases. *Lancet* 1934;17:10.
- 5 Gloyne SR. Two cases of squamous carcinoma of the lung occurring in asbestosis. *Tubercle* 1935;17:5-10.
- 6 Dreessen WC, Dallavalle JM, Edwards TI, *et al*. A study of asbestosis in the asbestos textile industry. *Public Health Bulletin* 1938; No 241:1-126. (Washington, DC: US Government Printing Office.)
- 7 Selikoff IJ, Churg J, Hammond EC. The occurrence of asbestos among insulation workers in the United States. *Ann NY Acad Sci* 1965; 132:139.
- 8 Balzer JL, Cooper WC. The work environment of insulation workers. *Am Ind Hyg Assoc J* 1968;29:222.
- 9 American Conference of Governmental Industrial Hygienists Committee on threshold values. *Documentation of threshold limit values*. Cincinnati: ACGIH, 1962:11-2.
- 10 Kelly JM. *Memorandum and Order, Master File*. 83-0268. Philadelphia: United States District Court for the Eastern District of Pennsylvania, 17 June 1991.

Prediction of mesothelioma, lung cancer, and asbestosis in former Wittenoom asbestos workers

The models used by Berry (1991;48: 793-802) to predict future incidence of

Table 1 Calculated number of mesothelioma cases expected from Wittenoom

Year of diagnosis	Year of first employment						Total
	<1940	1940-4	1945-9	1950-4	1955-9	1960-6	
<1980	18.3	6.9	9.5	29.1	20.9	5.1	89.8
*1980-85	3	2	5	18	19	9	56
1986-90	2.7	1.5	4.2	24.9	24.7	12.8	70.8
1991-95) 2.0) 1.3) 3.1) 20.8) 34.2) 16.7) 78.1
1996-00) 1.0) 1.0) 2.8) 15.2) 28.5) 23.1) 71.6
2001-05) 0.3) 0.6) 2.0) 13.9) 20.9) 19.3) 57.0
2006-10))) 1.0) 10.0) 19.0) 14.1) 44.1
2011-15))) 0.3) 6.6) 17.0) 10.0) 33.6
2016-20))))) 5.8) 18.3) 24.1
	27.3	13.3	27.9	138.5	190.0	128.4	525.4

*Observed cases based on Australian Mesothelioma Surveillance Program records.

)Estimated distribution of residual cases.

mesothelioma from the Wittenoom crocidolite mine produce a range of estimates dependent on the values of lag time and lung clearance rates. Although published data suggest a minimum lag period of around 20 years there is considerable spread of another 30 years to cover most cases. Also, the assumption of reduced toxicity due to clearance of fibres from the lung is speculative. de Klerk *et al*¹ have estimated future Wittenoom cases using a model based on lag time and exposure variables but their predictions are somewhat higher than Berry's. As the future occurrence of mesothelioma due to past exposure to asbestos is of great interest to government authorities, industry, workers, and the community, the accuracy of these estimates is of importance.

An alternative predictive approach is proposed using the distribution of time lag period since first asbestos exposure derived from information obtained in occupational histories from the Australian mesothelioma statistics.² Assuming a similar latency distribution in the Wittenoom cases collected in 1980-5, it is possible to calculate the total number of cases expected from the mine and the number expected in each time period. For

example, to calculate the total number of mesotheliomas expected for an employment period 1940-4, use the two observed cases recorded from this period in 1980-5 and the midpoint of employment to yield a latency of 37.5-42.5 years. From the lag data in Ferguson *et al*² prepare a graph of the cumulative percentage of cases *v* lag time since first exposure and use this to estimate that these two cases represent 13% (34% less 21%), hence predicting a total of 13.3. Calculation of the expected cases from the 1940-5 employees occurring in the time period 1991-5 (lag of 52.5-47.5 years) gives 11% (88% less 78%) of 13.3 cases or 1.3. Table 1 shows the overall data set.

The uncertainties in such an approach is that it includes environmentally exposed cases and takes a simplified view that there has been a similar distribution of age, the specific occupations, and exposures during the period of operation of the mine and mill, all of which are known to be incorrect.^{3,4} Also it assumes compatibility between the Wittenoom group and the whole Australian population. The estimates (71) appear reasonable, however, when compared with the 59 cases reported to the Australian Register in 1986-90. These

register records were not used in the calculation as they were not collected with the same rigour as the 1980-5 data and hence cases may have been missed and they are known to contain less detailed information on occupational history.⁵

Table 2 gives a comparison of the estimates from the various methods.

The proportional latency calculation method predicts a total of 525 mesotheliomas from the 6505 male and 411 female workforce and past residents; 366 cases will occur in the period 1986-2020, numbers will peak in the period 1991-1995, and tail off over the next 25 years. This fits comfortably in the range and distribution of most likely values presented by Berry.

A ROGERS

National Institute of Occupational Health
and Safety,
GPO Box 58,
Sydney, NSW 2001,
Australia

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- 2 Ferguson DA, Berry G, Jelihovsky T, Andreas S, Rogers A, Chung Fung S, Grimwood A, Thompson R. The Australian mesothelioma surveillance program 1979-1985. *Med J Aust* 1987;147:166-72.
- 3 Major G. Asbestos dust exposure. In: Major G, ed. Proceedings of the first Australian pneumoconiosis conference in Sydney, 1968: *Joint Coal Board* 1968:467-74.
- 4 Armstrong B, de Klerk N, Musk B, Hobbs M. Mortality in miners and millers of crocidolite in Western Australia. *Br J Ind Med* 1988;45:5-13.
- 5 Leigh J, Corvalan C, Grimwood A, Berry G, Ferguson D, Thompson R. The incidence of malignant mesothelioma in Australia 1982-1988. *Am J Ind Med* 1991;20:643-55.

Non-occupational pneumoconiosis at high altitude villages in central Ladakh

Sir,—Silicosis is a well established, probably underdiagnosed occupational disease,¹ the importance of which may be underestimated as an environmental disease. Several cases of non-occupational silicosis have recently been reported among people living in the Himalayan range by Naboo *et al*.² Another paper³ (BJIM 1991;48:825-9) dealing in greater depth with the radiological characteristics of the disease confirmed the findings of Naboo *et al*,² except that the

Table 2 Comparison of estimates of mesotheliomas

Year of diagnosis	Latency method estimates	Berry estimates adopted from table 7	de Klerk estimates
1987-90	57	40-44	44
1991-95	78	55-65	77
1996-00	72	55-70	102
2001-05	57	45-70	121
2006-10	44	30-60	132
2011-15	34	20-45	121
2016-20	24	10-30	95
	366	265-395	692