

CORRESPONDENCE

Asbestos and cancer: history and public policy

Sir,—Weller's emotional outpourings on asbestos and cancer (1992;49:70—2) need some response.

Based on the selective use of the work of various researchers over the years and the reported statements of two industrialists he has, in 1992, constructed a theory about the scientific knowledge that existed in the 1960s that leads him to a conclusion that is at odds with the view of two highly respected researchers, both of whom were very active in the 1960s. In the late 1970s they made their judgement on the level and nature of scientific knowledge that existed in the 1960s and thus their views are much more likely to represent the real situation than that put forward by Weller.

Such a response needs only to quote Lee and Selikoff in 1978 when they stated that:

"The decade of the 1960s provides a convenient time at which to terminate a historical review of asbestos disease. With admirable hindsight from the late 1970s we can see that the essential evidence had already been reported, but not yet assembled or vested with sufficient credibility to be entirely convincing. With few exceptions the evidence at that time rested on scattered reports of small numbers of cases, and the cases themselves suffered from being either selected or simply those that happened to come to the attention of the reporter. The population base from which the cases came was seldom mentioned. The significance of pleural changes and the occurrence of mesothelioma in persons without a distinct history of exposure remained in considerable doubt. The idea that asbestos could be at least a cofactor in the production of bronchogenic carcinoma (lung cancer) was far from fully accepted. That parenchymal asbestosis was very likely to occur in those who had been exposed to heavy dosage in the early years of the industry was clear enough, but what effect environmental controls that had been introduced in the 1930s might have upon its future prevalence was not known. The possibility that quite low dosages might have grave consequences 30 or more years after

first exposure was still unproven.

"Many things were needed to confirm the suggestions that were emerging from the studies up to that time. Most importantly, systematic epidemiological investigation was needed of large cohorts drawn from various types of industry, with the inclusion of adequate control populations. Some of these were already organised, but it was too early for the results to be meaningful. We do know that much of the negative evidence stemmed from coming to conclusions prematurely, before the slow processes of carcinogenesis had had a chance to make themselves evident. We now know also that reduction of heavy exposures that lead to early death would reveal such slowly developing diseases as mesothelioma and bronchogenic carcinoma with increasing clarity. But foreknowledge was not available at the time, although some investigators suspected that the auguries were not good. More sophisticated and sensitive ways of recognising the disease process at an early state, before the appearance of marked radiographic changes, were badly needed. A series of international conferences, some already in the planning stages, were to accelerate these developments greatly. Those who felt it was an exciting time were not to be disappointed. The excitement has not even yet entirely dissipated."

Weller's theory relies on his bland statement that "The scientific knowledge of the time was matched by the insight of industrialists." He then produces examples of this connection in the form of two industrialists who are said to have made statements in 1964 that there is no safe level of asbestos exposure. Such was the inconclusive state of the medical debate in 1964, shown by Lee and Selikoff, that it would not have been difficult to produce any number of industrialists who would even have doubted the existence of any link at all between asbestos and the various cancers and it would also not have been difficult to find industrialists who accepted a link but believed that they could "engineer" their way out of the problem. The latter is supported in the extract noted by Weller and taken from an editorial in the *New England Journal of Medicine* (1965;272:590-1) which stated, among other things, that "certain industrial operations using asbestos can be made safe by engineering control."

Enterline also records that in the early 1960s some medical opinion and that of reputable journals were not then convinced of the link between asbestos and the cancers. In these circumstances, why should industrialists as a group have been expected to match "the scientific knowledge of the time"? More importantly why should they have been expected to take action when medical opinion was not even agreed on the link?

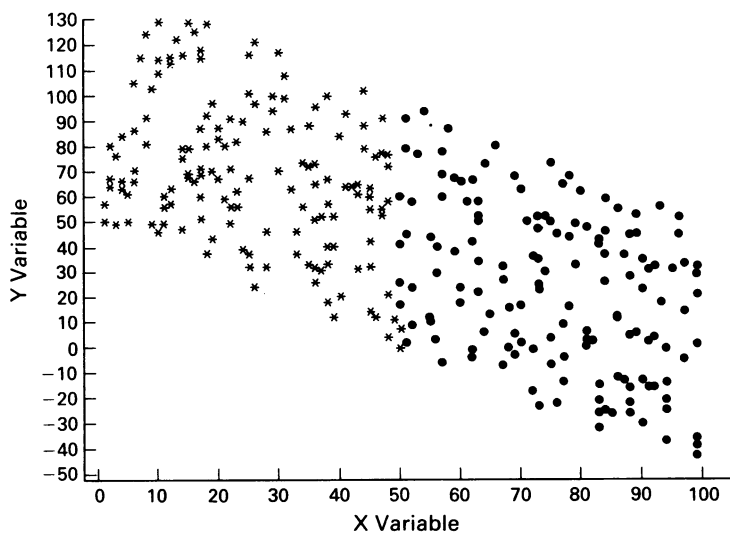
Weller concludes that industrialists flaunted safeguards and hazarded men's lives for commercial gain. Such a conclusion is not reasonable from the evidence he has presented; nor is his claim that industrialists suppressed research findings. It is much more likely that the understandable lack of universal agreement in the medical profession caused a slow response by industrialists to the growing weight of positive and conclusive evidence. In making his comments, Weller has employed all of the old worn out clichés—greed, gain, and conspiracy—which are more suitable for a political statement than one that should be based on scientific principles and fact.

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A cautionary note on the use of the correlation coefficient

Sir,—The product moment correlation coefficient (r) is undoubtedly one of the most frequently used statistics. Originally developed as a measure of the extent of linear relationship between two continuous random variables, X and Y in a sample taken from a bivariate normal population, it is also used as a supplemental statistic (coefficient of determination r^2) in linear regression analysis, where the X variable may or may not be random.¹ More recently, r has been adopted as a measure of agreement (in terms of ranks) between two methods for rating the same quantity.²

The correlation coefficient is easy to communicate as it takes values ranging from 0 for no linear relationship to 1 (–1) for perfect direct (inverse) linear relationship between X and Y . Also, because r is unit free, it can be used to compare relations in variables of diverse units—for example, to determine whether the relation between



dietary and serum cholesterol is stronger than that between body weight and height.

What is not widely recognised, however, is that r is affected by inter-subject variation in the X variable (the standard deviation of the X variable in the sample, s).¹ This means that the sample correlation r is an unbiased estimate of the population correlation ρ only if s is comparable with the standard deviation of X in the population σ ; A numeric example is given to show the pronounced dependence of r on s .

Based on 300 four digit random numbers that I generated by the SAS software,³ I used the first two digits for the X variable and the difference between the last two digits and first two digits plus a constant 50 for Y. Because Y is part of X, clearly the two variables are correlated. Indeed it can be shown theoretically that $r = 0.7$. Next I divided the sample into two subgroups of 150 based on the values of X, subgroup 1 with low X values

and subgroup 2 with high X values (represented by asterisk and solid circle, respectively, in the bivariate scattergram of Y on X; figure). By all appearances, the r value for the subgroups ought to be similar to that for the entire sample. But this is not so (table): r for the entire sample (-0.68) is substantially higher than that for each subgroup (-0.38 and -0.42) simply because s is higher in the entire sample (29.0) than it is in the subgroups (14.2 and 14.5). Note that Spearman's rank correlation is similarly affected by s . Clearly, these results imply that r is generaliseable only to the population from which the sample was taken, provided the sample is reasonably representative so that s is comparable to σ , that r should only be compared among different samples provided their s values are similar, and that r based on the entire sample should not be compared with that computed from subsamples. Thus to assess the correlation between age and blood pressure, it would not be valid to

compare r for the whole sample with that for hypertensives selected from that sample.

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- 1 Oldham PD. Measurement in medicine: the interpretation of numeric data. Philadelphia: Lippincott, 1968 (chapter 6).
- 2 Walker AM, Blettner M. Comparing imperfect measures of exposure. *Am J Epidemiol* 1985;121:783-90.
- 3 SAS language guide, Release 6.03. Cary, North Carolina: SAS Institute, 1988.

Polychlorinated biphenyls: estimated serum half lives

Sir,—Between 1946 and 1977 two plants of the same company located in upstate New York were manufacturing capacitors using polychlorinated biphenyls (PCBs) with aroclors 1254, 1242, and 1016 as their primary dielectric fluids. Almost 7000 employees worked there for at least three months during this period. In 1976 a study was started to determine the possible human health effects of exposure to PCBs by detailed examination of a small group of these workers. One hundred and ninety four workers were selected to include employees whose jobs required direct contact with PCB in zones of high air concentration, were in the immediate periphery of the high exposure zone, or had high but intermittent exposure. Health evaluation included a physical examination, chest x ray film, spirometry, haematological and biochemical analyses, urinalysis, and measurement of serum PCB concentrations.¹ A similar evaluation was conducted in 1979 and 1983 on participants available for re-examination. Serum determinations were performed by Hazelton Raltech (Madison, Wisconsin) with conventional PCB extraction, and packed column gas chromatography, and were reported as Aroclors 1242, 1254, and 1260.¹ Because PCBs were still in use at the time of the 1976 evaluation, and because of other uncertainty about the 1976 data, only data from 1979 and 1983 were used in this analysis. Serum PCB determinations were available from both 1979 and 1983 on 148 persons for aroclors 1242 and 1254,

Mean, standard deviation, and correlations

	n	\bar{X}	s	r	r_s
Entire sample	300	51.0	29.0	-0.68	-0.68
Subgroup 1	150	25.6	14.2	-0.38	-0.33
Subgroup 2	150	76.0	14.5	-0.42	-0.39

\bar{X} = mean of X; s = standard deviation of X; r = product-moment correlation; r_s = Spearman's rank correlation.