CORRESPONDENCE

A critical review of the effect of factory closures on health

Sir,—We read, with interest and dismay, the critical review by Morris and Cook, of the studies on factory closures and health (1991;48:1–8). The authors' statement that "none of the studies fulfil all the criteria for an ideal study, . . ." is sanctimonious when their report itself is peppered with flaws.

By using Index Medicus from 1980 only as their reference source they give a foreshortened perspective. The study by Fisher in 1959, reported in 1965[1], and that by Ziegler in 1964, reported in 1979,[2] have been overlooked. The Michigan study (authors' references[6 8 9]) is not, therefore, the ". . . earliest study reported . . ." although the field work was started in 1965 and not, as stated, in 1967.

In the Danish factory study (authors' reference[10]) there was, in fact, a control group although the investigator referred to it only in a later report.[3] Neither do the authors register the unique feature of the Sardine factory study (authors' reference[11])—namely, that 81% of the study group were female employees.

When appraising the Calne study (authors' references[18–26]) they continue to report inaccurately. We had data on our study group for four years before they could have had any suspicions of job losses (not two years as stated). The study group did have to tolerate, however, two years of insecure employment after the management "rationalised" the plant. In fact the authors seem to fail, repeatedly, to distinguish the phase between the announcement of redundancies and factory closure (often only a few weeks) from the preceding phase of insecure employment which, often in the face of denials by management, workers are able to sense when their employer is is financial difficulties. Both these periods need to be demarcated and contrasted with an even earlier phase of relatively secure employment, which can be the only true baseline in a longitudinal study. In this light the authors' use of the terms "anticipatory" and "pre-closure" as in their table 2 seems obtuse and confusing.

Although we would have preferred it, the control employees in the Calne study did not all work at ". . . a similar factory . . ."[4] and that we reported on spouses and children of employees seems to have been ignored.

We would, however, concur with the listed advantages and disadvantages of factory closure studies although advantage (3) is inadequately expressed. It is possible to recognise and incorporate several distinct phases in the process of job loss as it affects individual workers: (a) that of secure employment in a stable, successful, enterprise; (b) that of insecure employment in a failing industry—a study phase of paramount importance; (c) that between formal announcement of mass redundancy and plant closure; (d) that of actual unemployment; (e) that of continued unemployment or of reemployment (secure or insecure).

That these serial events are each uniquely important in longitudinal research of this nature is now acknowledged and features in study designs.[5 6]

Perhaps, at a time of deepening economic recession in the United Kingdom we could expect that the authors, as professional academics, turn to gamekeeping rather than poaching and perform the large scale factory closure study to which they allude in such idealistic terms. At the very least they owe those, some only amateurs, who have given them the data they dissect, the duty of reporting their studies accurately.

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Authors' reply

We did not intend our conclusions to sound sanctimonious, and we are fully aware of the major difficulties involved in carrying out factory closure studies. The perfect study is unapproachable but this should not blind us to the qualifications that need to be placed on the conclusions of those studies that have been reported. Without such critical evaluation, studies carried out in the current recession are likely to have the same deficiencies.

We thank Beale and Nethercott for pointing out some errors in our review, but would stand by our conclusions. In table 1, the study population of the Sardine factory (Norbest Canning Co) should have been 72 women and 13 men, rather than 72 men and 13 women. In the Calne study it was incorrectly stated that data were collected for only two years before any knowledge about the closure, instead of four years. The control group should have been described as coming from several other local firms. We did attempt to distinguish between data collected at a time when jobs were relatively secure and data collected at a time when there was awareness of the possibility of the factory closure being decided. Finally, in carrying out the review it was our intention to focus on the effects of the last recession and that was the reason for reviewing studies published since 1980. Fortuitously this criterion allowed us to include the Michigan study, which was the forerunner of all the other studies reviewed.

Asbestos and cancer: history and public policy

Sir,—The issues discussed by Castleman (1991;48:427–432) indicate that many problems associated with asbestos dust were known before industrialists responded. Lung cancer and mesothelioma should be put in the context of knowledge of overall risk.

Despite the excellent points made

by Murray, it is reasonable to expect that those concerned with mining or processing asbestos should have been alert to the growing body of medical opinion that, first tentatively and then authoratively, established links between asbestos inhalation and serious diseases. Intelligence gathering in medicine is greatly facilitated by the excellent *Index Medicus* and it is simple to consult this publication for a comprehensive survey of the world medical publications. Journals of Industrial Medicine might reasonably be consulted by the medical staff of major concerns and Johns-Manderville and other asbestos companies subscribed, among other things, to the *Industrial Hygiene Digest*, that carried Gloyne's pertinent abstract.

Following Murray's description of a case of pulmonary fibrosis, reported in 1907 to the Committee on Industrial Hygiene in England, series of cases of asbestosis were described by various observers including Pancoast, Pancoast and Pendergrass, Cooke, who had earlier coined the term, Wood, Haddow, Simson, Lynch and Smith, Ellman, Merewether, Lanza *et al*, Donnelly, McPheeters, and Shull. More definitive studies were conducted by the Pennsylvania Department of Labor and Industry in 1935 and the US Public Health Service and the North Carolina State Board of Health in 1937. The US Public Health Service fully documented the significant risk involved in asbestos textile factories in a report by Dreessen *et al* in 1938[1] and urged elimination of hazardous exposure.

What is clear, from a reading of the major journals and abstracting services is that a Nelsonian eye seems to have been long turned towards the mounting evidence of an industrial hazard. Castleman adduces evidence that the hazard was appreciated and sponsored research suppressed. Legal safeguards, which sluggishly responded to the scientific understanding, seem to have been outrageously flouted. This is the view of the American Courts that have indignantly awarded punitive damages to claimants.

A concentration of 5·0 million parts per cubic foot (mppcf) of air for daily eight hour exposure was adopted by the American Conference of Governmental Industrial Hygienists in 1947 and reaffirmed in their conference of 1964.

This concentration was established on the basis of abdominal (intraperitoneal) injections into guinea pigs, which "may be regarded tentatively as the threshold value for asbestos dust exposure until better data are available." Other evidence, using the same experimental technique, showed that this concentration was only applicable to observations on short fibre asbestos and the concentrations were vigorously criticised as being inadequately stringent for humans. For example Shall (1965),[2] in a conference convened by the New York Academy of Science, pointed out that the industrial plants studied manufactured textiles and the sampling procedure (impinger collection in ethyl alcohol and distilled water) included stone, cotton, and other dust particles and gave only an indirect measure of the risk of asbestosis because of the relative importance of long fibres, whereas the studies had predominantly been on supplies of the short fibre chysolite from Canada. The sampling of subjects was biased, the numbers small, and the controls inadequate. The study was conducted for a mere five years in 333 of the 511 employees investigated, more than half of whom were under 30, the lowest average age of any group studied by the Public Health Service at that time (for comparison the average age was 37·8 in the foundry industry). The sick were missing and the dead were buried. The 76 "controls" used for comparison were office workers in the same factories who were known also to have been exposed to asbestos dust (see Wagner *et al*[3]; Wagner 1963[4]; Hourihane *et al*[5]). Further criticisms included the fact that the exhaust ventilation systems were not standardised and were inappropriate for scientific study. The dust count is conceived as an average but in practice this average often encompasses an enormous range, up to 211 mppcf being recorded, and peak concentrations are probably highly significant because they overwhelm all defence mechanisms with huge lung retentions.

The scientific knowledge of the time was matched by the insight of industrialists. In 1964 CG Addlingley of the British Belting and Asbestos Ltd, Cleckheaton stated that "we do not believe there is any safe limit . . . we are always striving to get right down to zero . . . there is no scientific basis for that limit [5 mppcf] whatever . . ." John Wells of United States Rubber Co, Newnan Ga repeated these views with the observation that "our own conclusion, as we began seeing what was happening in our own process, was that the only safe amount of asbestos dust was zero and that the efforts in terms of achieving that lay basically in engineering, and, secondly, in education. But as far as a safe level of asbestos dust is concerned there is no safe level. The safe level is nil and anything above the safe level represents certain risks."

The New England Journal of Medicine published an editorial in 1965 (1965;272;590–91), which insisted that "certain industrial operations using asbestos can be made safe by engineering control. In other operations this may be so difficult that substitution of a safer material must be considered." In 1968, the American Conference of Governmental Industrial Hygienists reduced their recommended exposure levels to 2 mppcf of air.

Serious doubt exists that even the earlier, less stringent standards, were observed. For example, in the key case (Borel *v* Fiberboard *et al*, 10 September 1973) Borel protested that basic safety equipment, such as masks, had to be specially requested and were intolerable for prolonged wear, and how his clothes were stiff with asbestos dust at the end of each working day. Borel pitifully but convincingly stated that he did not know what asbestosis was and the Court concluded that the manufacturer's warnings were totally inadequate.

The American government accepted its culpability in the Naval shipyards during the second world war, when the war effort condoned unsafe practices (for example, PPG Industries and Corning Glass Works, District Court for the Eastern District of Texas, 1978). There is less justification for industrialists to flaunt safeguards and hazard mens' lives for commercial gain; and worse, they seemed to have suppressed proper research findings that went against their perceived interests, as documented by Castleman.

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Biological monitoring of MDA

Sir,—4,4'-methylenedianiline (MDA) is a primary aromatic amine usually made via the reaction of aniline and formaldehyde. It is used as a hardener in epoxy resin systems. The product produces cholestasis and hepatic necrosis in many animals and caused the so called Epping jaundice when 84 persons ate bread contaminated with it. In industry hepatitis developed in 12 young male workers exposed to MDA.[1] Studies from the National Toxicology programme (NTP)[2] showed that the dihydrochloride salt of MDA is carcinogenic in both sexes of rats and mice, and found cancer of the liver, the thyroid gland, and the haematopoietic system; MDA is structurally similar to benzidine, a known human bladder carcinogen.

The objective of the current study was to measure free and conjugated MDA in the urine of workers as an assessment of exposure.

Method

Urine was collected at the end of a workshift. Until June 1989 MDA was measured in hydrolysed urine with a liquid. chromatographic method and UV detection (210 nm). The detection level was 100 ppb (100 μg/l). In May 1990 the method was changed. After reaction with hydrochloric acid, MDA was measured by high performance, liquid chromatography with electrochemical detection using ethylenedianiline as an internal standard. The detection limit was 2ppb (2 μg/l).

The concentration of urinary creatinine was photometrically estimated with a commercial kit (creatinine—Boehringer Mannheim).

Results below the detection limit were handled as the half of the detection limit.[3]

Results

These are presented in the table.

Discussion

Measurements of MDA were carried out at five different times. With many results below the detection limit it is a problem to calculate an average. Here I used the detection limit/2, a method described by Horning and Reed for use when data are highly skewed and with non-detectable values of more than 30%.[3] The real average must be somewhere between the two results given in parentheses (see table footnote).

After August 1988 working conditions were changed: masks, gauntlets, and disposable paper overalls became obligatory. Results for October 1988 showed a distinct improvement. Nevertheless the management took the decision to totally rebuild the unit. In June 1989 a survey without production was done in the new installation. This showed that 19 of 20 results were below the detection limit. One person had a value of 50 μg/g creatinine. The reason for this was not clear.

At the same time a new method using a liquid chromatographic technique with electrochemical detection of MDA in urine was developed in the medical laboratory of BASF Ludwigshafen with a considerably lower detection limit of 2 ppb. In the workshop the "dirty area" was separated from the "clean area" by a sluice. Additional personal protection equipment was used—namely, total protective PVC suits with uncontaminated air supply from outside. After these modifications had been implemented, biological monitoring was repeated. Results at least the same as in June 1989 were expected but they were disappointing. How was it that with the special dress and supply of air from outside, absorption was still possible? Analysis of the work process step by step showed that by changing protective clothing the outside of the dress contaminated the inside. After better cleaning of the protective clothing and improvements in the procedure for changing clothes, the results for June 1990 showed considerable improvement.

Time of measurement	*No of subjects*	*Median*	*Maximum*	*Average*	*Below DL (%)*
August 1988	91	<DL	4110	236 (215–274)	59*
October 1988	87	<DL	550	98 (63–133)	70*
June 1989	20	<DL	50	50 (2–98)	95*
May 1990	107	71	1416	202 (201–202)	4·5†
June 1990	43	11	366	43 (43–43)	0†

Median, maximum, and average are expressed in μg/g creatinine. DL = detection limit. *Detection limit = 100 ppb; †detection limit = 2 ppb. For the average, the results below the detection limit were handled as DL/2. Between parentheses the first value indicates the average if the results below the detection limit were handled as zero, the second value indicates the average if the results below the detection limit were handled as the value of the detection limit.

Conclusion

Even with extreme individual protection, monitoring of urine for the presence of MDA is recommended as a tool for detecting absorption from all sources. The method can also be used for checking work practices and assessing performance of personal protective equipment.

I am indebted to Dr R Smits and Dr W Will for the analyses of MDA. I express my sincere thanks to all the personnel—management and workers—of the MDA plant. My particular thanks go to Mrs Maes and Mrs Schellemans who organised the study and measured creatinine and to Mrs Andries who brought the manuscript to a readable form.

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