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3 Horton AW, Boleswicz LC, Barstow AW, Butts CK. Comparison of the promoting activity of pristane and α,α',α'-trihalo-2,4,6-trinitrotoluene in skin carcinogenesis with their physical effects on micellar models of biological membranes. Biochim Biophys Acta 1991;1077:147-53.

Digestive tract neoplasms among employees with past exposure to brominated dioxins

Previously, we reported biomonitoring, immunological, and other clinical findings for a group of 42 employees potentially exposed to polybrominated dibenz-p-dioxins (PBDDs) and furans (PBDFs) during extrusion blending of resins containing poly- brominated diphenyl ether (PBDE) flame retardants.1,2 In five years since the clinical study was completed, two cases of digestive tract neoplasms have come to our attention within this group of employees and an additional digestive neoplasm was reported in a techni- cian who performed analyses in support of the production operation including testing by pyrolytic methods. The two cases in the earlier study group had the highest observed 2,3,7,8-tetabromodibenzo-p-dioxin (2,3,7,8-TBDD) concentrations in blood lipids among the 42 persons surveyed.

Case 1 was diagnosed to have squamous cell carcinoma of the oesophagus in January 1994 at the age of 57 and died nine months later. This person worked as an extruder operating throughout a 17-year period when PBDEs were used in the extrusion operations (1977-89). He had the highest record- ed 2,3,7,8-TBDD concentration (527 parts per trillion (ppt) measured in 1989) of any person in the study population. Other dioxin and furan congeners were increased as well with a 2,3,7,8-TBDD concentration of 176 ppt and total hepta- plus octa-TBDD concentra- tions of 10 000 ppt. Other factors potentially related to this diagnosis included a history of smoking about one pack of ciga- rettes a day for 40 years and consumption of two to three bottles of beer a day.

Case 2 was diagnosed to have adenocarcino- noma of the duodenum, a relatively rare type of cancer, in April 1994 at the age of 49 and died in July 1994. He had performed laboratory work support work since 1977. Potential contact with PBDDs and PBDFs would have occurred between 1977 and 1989. Postmortem microscopic examination showed a non-detectable 2,3,7,8-TBDD concentration, and a 2,3,7,8-TBDF concentration of 10 ppt. Marginally higher concentrations of the measured congeners were contained in a fat sample as well as detectable concentrations of both 2,3,7,8-TBDD (0-8 ppt) and 1,2,3,7,8-penta-BDD (2-3 ppt).

Case 3 was diagnosed to have tubular adenoma of the rectum in September 1995 at the age of 48. He had also been employed as an extruder operator during the entire period of PBDE use. His initial 2,3,7,8- TBDD blood lipid concentration in 1989 was 425 ppt.

Case 1 is of particular interest because of our earlier findings of increased digestive and respiratory cancer among people with high chlorinated dioxin exposures who were also cigarette smokers.1 The data presented here for cases representing occurrences where dioxin concentrations were less clear- ly higher than background (case 2) or where the tumour diagnosed was a relatively com- mon benign neoplasm (case 3).

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Factors affecting recognition of cancer risks of nuclear workers

Editor—I have a problem with Kneale and Stewart's approach to statistical analysis and that is the fact that the more statistical tests they run on the same data, the more likely there is of encountering spurious or artefactual associations. For this reason the more nested tests, correction factors, and optimised values introduced into tests, the more likely the results will be significant but of no practical consequence. Surely the Hanford data have now been worked to death.

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Author's reply—Both the analyses of Hanford data which O'Donnell finds so unnecessary, and the 1991 analysis of Oak Ridge data by Wing et al.1 have produced evidence of incompatibility between the records of nuclear workers recently released by the
United States Department of Energy (DOE data) and the records of A-bomb survivors that are the mainstay of radiation safety regulations in this country and elsewhere (A-bomb data). Also, analyses of the Hanford data have been performed for one variable in a relative risk model—that is, the one which measures the effect that the age when exposed has on the subsequent cancer risk, which should have a constant value whatever the source of the radiation—there is incompatibility not only between A-bomb data and DOE data but also between Hanford and Oak Ridge data and between Hanford data for different exposure periods. 

What is generating O'Donnell is not the numbers of times that Hanford data have been analysed (which is far fewer than the number of analyses on the A-bomb data) but the number of independent variables in the Keneale and Stewart relative risk model. Correct assessment of these variables is essential for future tests of important hypotheses, such as whether young people are more or less sensitive to cancer effects of radiation, and whether A-bomb survivors apart from their radiation dose are or are not representative human beings. So it is clearly important not to stint on the process of recognising false elements in DOE and A-bomb data.

Finally, we have a special reason for making this point, as we hope shortly to publish a paper which shows that the A-bomb survivor who most resembled the survivors—that is, the survivors who had multiple acute injuries, such as burns, purpura and epilation—differs in several important respects from the much larger number of survivors who had no such injuries.


Mesothelioma in a community in the north of England

Editor—Muir, who wrote a brief essay on the subject of bias in the field of occupational health in the final issue of the British Journal of Industrial Medicine, will find this paper useful for teaching purposes. He might take issue with the authors on the literary style and presentation of historical facts, and on their analysis of data. We are informed by correspondence in 1939 to produce gas mask filters. Reference to Defence of the Realm powers for initiating asbestos work might be misread by the reader as a plea in mitigation for the heavy harvest of disease arising from post-war exposures. The introduction cites Bertram Mann as making some reference to problems resulting from asbestos exposure. The paper is concerned with the tendency to rout out the text of his 1978 Royal College of Physicians Milroy lecture, will find that this chest physician had a lot to say about the amount of disease that he came across in this small part of his catchment area.

The discussion section states that: "In common with many asbestos factories, working conditions in respect of asbestos dust were poor, especially in the early years of its operation." This might be misread to imply that conditions in its latter days were acceptable. Sir Alan Marre's (The Ombudsman) report in 1976 of its inquiry into Acre Mill, although concerned solely with the quality of maladministration, did not find that the factory was a cause for concern. The authors' statement that; "The factory closed in 1970 and has since been demolished," is false: it was occupied by another manufacturer for several more years, in a not entirely decontaminated state.

In the discussion section we are informed that between 2000 and 3000 people worked at the factory, although the material and methods section is not explicit on this point. One may assume that the authors did not have access to the nominal roll of employees: otherwise they would surely have used the Registrar General's facilities for tracing and flagging the total population. No researcher should be faulted when making the best of limited data, provided the necessary caveats are presented prominently. In their calculations, the authors gave an average incidence of tumour in Calderdale over the period 1966-94 as "12.5/5 million persons/year". This result is from the dilution by the overwhelming non-exposed population. When the factory population is studied separately, the rate works out not surprisingly as between 524 and 786 cases of mesothelioma per million person-years, depending on which extreme estimate of factory population is taken. For the non-exposed, the rate works out as 3.2 million person years, which is a higher rate than one would like to note.

The authors state that there were no neighbourhood cases of asbestos related disease. Yet of the 17 cases of malignant mesothelioma reported in the population exposed to asbestos, eight had exhibited excessive amounts of amphibole in their lung tissues. We are not informed of how this might have been acquired. It is possible that despite the Pennine geography and meteorology exposure conditions that were healthier with respect to malignant mesothelioma than in the "dust bowls" of Barking and North Western Cape province.

Agropus, the fibre counts in the lungs (which commonly means the parenchyma, rarely the pleura, and even more rarely the bronchi), toxicologists look at the science of xenobiotic disease differentially from pathologists, mineralogists, statisticians, and physicians. Physicians looking at disease in the pleura or peritoneum are content to relate it to the amount and type of fibre in the lung parenchyma rather than the type and quantity that has reassembled to the critical tissues. (Yet it is chrysotile rather than the amphiboles that is more often reported by pathologists to be found juxtaposed.) Again, although physicians are content to compare the toxicokinetics and mode of action of any two fibres that have not been conducted on the various asbestos species to relate their carcinogenic effects, dose for dose, fibre for fibre when equal dimensions are involved, basic crude studies have insisted that they can share similar carcinogenic power. The courts are led to think that one can differentiate between the tumour caused by asbestos and the tumour not so caused, on an estimation of parenchymal fibre content. This is despite the wide confidence limits that need to be placed around an estimate involving uncertainties in sampling and in counting, and despite the overlap in the distribution curves for lung fibre content between people with a history of asbestos exposure and those with no ascertainable exposure. For the authors' more sanguine attitude to chrysotile, the reader would be well advised to await the forth-coming report of chrysotile in the Environmental Health Criteria series published as part of the International Programme on Chemical Safety. This has had a stormy passage but is due out soon.
Factors affecting recognition of cancer risks of nuclear workers.

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