Ingestion of herring leads to absorption of pristane in humans

Editor—Pristane (2,6,10,14-tetramethylpentadecane) is a branched chain hydrocarbon which is thought to be derived from the phytol moiety of chlorophyll. The compound has been associated with several biological effects. Pristane can induce plasmacytoma,1 and together with some polyunsaturated hydrocarbons, act as promoters in the development of B-lymphoid malignancies and skin tumours in animals.2 Intraperitoneal injections have induced arthritis in mice.4 Pristane occurs in rather high concentrations, 1% to 3% of the body fat, in certain zooplankton and these plankton are assumed to be the primary source of the pristane found in liver oils of sharks and whales.5 Pristane is also found in herring and the concentration in flesh is about 370 μg/g of wet weight.6 Other fish species such as cod have much lower concentrations of pristane in their flesh, most often <1 μg/g of wet weight.4

One of us (BS) volunteered to ingest Atlantic herring as lunch at 11:00 am. Four different meals contained 125, 140, 250, and 310 g of cooked herring. A reference meal was composed of chicken with potatoe, hard bread, and water. The quantitative analysis of pristane was performed with gas chromatography equipped with a flame ionisation detector. Pristane was also identified with gas chromatography-mass spectrometry, operated in electron impact mode. The recovery of the complete clean up method was evaluated from a known standard (dodecylcyclohexane). The detection limit of the method was 300 pg.

The serum concentration of pristane increased to 20-3000 μg/g serum 2-4 hours after the ingestion of herring. Pristane was not detected before and 24 hours after ingestion of herring and it was not detected after the reference meal.

This experiment showed that pristane is clearly absorbed after the ingestion of Atlantic herring. Pristane injected intraperitoneally into mice induces seropositive arthritis in susceptible strains. This agent has been proposed as an experimental model for rheumatoid arthritis.4 Swedish fishermen eat more herring than the general population1 and it is an interesting finding that fishermen had a high prevalence of rheumatoid arthritis when this disease was investigated in 66 large occupational groups in Sweden.6

It is also interesting that pristane is absorbed after ingestion of herring and this absorption should be studied in larger groups of humans after ingestion of herring and other species of fat fish. Further work is also necessary to recognise the possible link between pristane exposure and the occurrence of multiple myeloma and diseases affecting the joints.

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Dietetic tract neoplasms among employees with past exposure to brominated dioxins

Previously, we reported biomonitoring, immunological, and clinical findings for a group of 42 employees potentially exposed to polybrominated dibenz-p-dioxins (PBDDs) and furans (PBDFs) during excursion blending of resins containing polybrominated diphenyl ether (PBDE) flame retardants.1 In the 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 technician 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-tetrabromodibenzo-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 operator throughout his period when PBDEs were used in the extruding operation (1977-89). He had the highest recorded 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-TBDF concentration of 176 ppt and total hepta- plus octa-TBDD concentrations of 10 000 ppt. Other factors potentially relevant to his diagnosis included a history of smoking about one pack of cigarettes a day for 40 years and consumption of two to three bottles of beer a day.

Case 2 was diagnosed to have adenocarcinoma 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 support work since 1974. Potential contact with PBDDs and PBDFs would have occurred between 1977 and 1989. Postmortem measurement of dioxins and furans in blood lipids 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 Cases of cancer observed in this study are not compatible with the null hypothesis. Where cancer concentrations were less clearly higher than background (case 2) or where the tumour diagnosed was a relatively common 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
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,1 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 that they had access to the case records of patients referred to Acre Mill in 1939 to produce gas mask filters. Reference to Defence of the Realm powers for initiating asbestos work might be misconstrued by the reader as a plea in mitigation for the heavy harvesting of disease arising from post-war exposures. The introduction cites Bertram Mann as making some reference to problems resulting from asbestos exposure at Acre Mill. It is stated that the mine was commanded by Sir Alan O’Donnell, who was not the number of times that the authors mentioned the variable ‘cessation of smoking’ in their 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.

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References


Authors’ reply—Greenberg’s repeated concern that parts of our article might be misread is touching but likely to underestimate the degree of commonality of bias. It would be difficult to imagine how the factual statement that Acre Mill was “commandeered by the Home Office in 1939” could be construed as a “plea in mitigation for the heavy harvest of asbestos disease”. Likewise his concern that our observations about working conditions might be misconstrued to “imply that conditions in the latter decades were acceptable”. We surprised this article not with an apportioning blame but to set the medical scientific record straight about the number of cases of mesothelioma found in Calderdale. Even after rereading the article we do not recognise any suggestion that the working conditions at Acre Mill were any better or worse than any other asbestos factories working at that time. Perhaps David Muir might find this letter by Greenberg an interesting example of bias for teaching purposes.

Two of the authors (ATE and DW) worked alongside Bertram Mann for many years and his paper is well known to us. In his classic paper (in which he speculated on asbestos related disease arising from asbestos pollution from the factory) a detailed analysis of tissue proven mesothelioma failed to detect any such case in a person living within two to four square miles of the factory who had not been employed at Acre Mill. Furthermore ATE performed most of the postmortem examinations in the district from 1960 to 1994 and has examined the reports of those not performed personally and is not aware of any case of asbestos related disease occurring as a result of atmospheric pollution from the factory. That despite a high level of exposure to asbestos related disease among the medical profession and in the community whereby suspected cases were readily referred to the local coroner.

We had very good access not only to senior ex-management of the mill but to many other ex-employees who are still alive and who had worked during wartime and later periods leading up to the 1939. It is difficult to imagine how the factual statement that Acre Mill was “commandeered by the Home Office in 1939” could be construed as a “plea in mitigation for the heavy harvest of asbestos disease”. Likewise his concern that our observations about working conditions might be misconstrued to “imply that conditions in the latter decades were acceptable”. We surprised this article not with an apportioning blame but to set the medical scientific record straight about the number of cases of mesothelioma found in Calderdale. Even after rereading the article we do not recognise any suggestion that the working conditions at Acre Mill were any better or worse than any other asbestos factories working at that time. Perhaps David Muir might find this letter by Greenberg an interesting example of bias for teaching purposes.

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