Ingestion of herring leads to absorption of pristane in humans

Editor—Pristane (2,6,10,14-tetratetramethylpentadecane) is a branched chain hydrocarbon which is thought to be derived from the phytoplankton community, but its importance in the environment is uncertain. The compound has been associated with several biological effects. Pristane can induce plasmacytomas, and together with some polycyclic aromatic hydrocarbons (PAHs) and 

1. Induced absorption and storage in target organs, as well as promoting the development of B-lymphoid malignancies and skin tumours in animals.

2. Intraperitoneal injections have induced arthritis in mice.

Pristane is absorbed 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. Pristane is also found in herring and the concentration in flesh is about 370 μg/g of wet weight. Other fish species such as cod have much lower concentrations of pristane in their flesh, most often < 1 μg/g of wet weight.

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 herring. A reference meal was composed of chicken with potato, bread, and water.

The quantitative analysis of pristane was performed with gas chromatography equipped with a flame ionization detector. Pristane was also identified with gas chromatography-mass spectrometry, operated in electron impact mode. The recovery of the complete clean up method was 85% from muscle and the internal 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. Swedish fishermen eat more herring than the general population 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.

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 fish. Further work is also necessary to scrutinise 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 other clinical findings, and results from a group of 42 employees potentially exposed to polycrrominated dibenzo-p-dioxins (PCDDs) and furans (PCDFs) during extrusion blending of resins containing polychlorinated diphenyl ether (PBDE) flame retardants.

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 neoplasia 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 (23,7,8-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 years in the period when PBDEs were used in the extruding operations (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-TBDD concentration of 176 ppt and total hepta- plus octa-TBDD concentrations of 10 000 ppt. Other factors potentially relevant to this 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 1977. Potential contact with PBDDs and PBDFs would have occurred between 1977 and 1989. Postmortem examination of the possible liver 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 obtained 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 light to moderate smokers. The cases representing occurrences where dioxin concentrations were less clearly higher than background (case 2) or where the tumour diagnosed was a relatively common benign neoplasm (case 3).
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 in terms of the one variable 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 number 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 Kneale 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 than old people, 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 precaution 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 probably had the most radiation of survivors—that is, the survivors who had multiple acute injuries, such as burns, purpura and epilation—differ in several important respects from the much larger number of survivors who had no such injuries.

Reference


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 this community was instructed in 1939 to produce gas mask filters. Reference to Defence of the Realm powers for initiating asbestos work might be misconceived by the reader as a plea in mitigation for the heavy harvest of disease resulting from post-war exposures. The introduction cites Bertram Mann as making some reference to problems resulting from asbestos exposure. We note that Acre Mill was contemplating the task 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 encountered 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 his inquiry into Acre Mill, although concerned solely with the quality of maladministration, did find that the factory was a cause for concern. The authors’ statement that; “The factory closed in 1970 and has since been demolished,” is, we are told, was occupied by another manufacturer for several more years, in a not entirely decontaminated state.

In the discussion section we are informed that between 1000 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/million persons/year”. This results 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 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 see for a town.

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 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 were conducive for the healthier with respect to malignant mesothelioma than in the “dust bowls” of Barking and North Western Cape province.

Approximately 50 fibres counts in the lungs (which commonly means the parenchyma, rarely the pleura, and even more rarely the bronchus), 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 of fibre related to the critical tissues. (Yet is chrysotile rather than the amphiboles that is more often reported by pathologists to be found juxta-plurally.) Again, physicians are content to attribute the occurrence of asbestosis because of its rate of clearance from the body, toxicology requires a better understanding of the toxicokinetics and mode of action of any fibre that has preceded and has not been conducted on the various asbestos species to relate their carcinogenic effects, dose for dose, fibre for fibre when equal dimensions are known. Model studies have indicated 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 parenchy- mal 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.

As for the authors’ more sanguine attitude to chrysotile, the reader would be well advised to await the recently published report on 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.

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Reference


Factors affecting recognition of cancer risks of nuclear workers.

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