

ENVIRON

September 22, 1989

MEMORANDUM

To: Frank Anastasi

From: Susan Youngren *SY*

Subject: Comments on "Draft Final Baseline Public Health Risk Assessment; New Bedford Harbor Feasibility Study, August 1989"

Overall Assessment

The "Draft Final Baseline Public Health Risk Assessment; New Bedford Harbor Feasibility Study, August 1989" (Ebasco 1989) (hereinafter referred to as the "Draft Report") is a comprehensive examination of potential risks to public health under baseline conditions from exposure to PCBs, lead, copper, and cadmium detected in the sediment, surface water biota, and air within the New Bedford Harbor site. The risk was quantitatively estimated from potential exposure to the four contaminants through dermal contact and ingestion of sediments, and ingestion of fish. In addition, a quantitative assessment of risk from potential inhalation of airborne contaminants was performed only for PCBs due to limited air data. A qualitative assessment of risk was performed for dermal contact and ingestion of water.

The assessment is a reasonable examination of the potential current risks to human health under the various exposure assumptions presented within the Draft Report. The report evaluates the appropriate exposure pathways for the appropriate populations of concern. The estimates of risk are conservative, but the assumptions used are within the range of those used in assessments of other sites and accepted by USEPA.

However, ENVIRON believes there are some technical flaws and questionable assumptions used in the Draft Report. Even though these flaws and assumptions do not individually affect the risk estimates appreciably, they should be evaluated prior to using the results presented in the Draft Report as the basis to determine the need for and the extent of remediation at the New Bedford Harbor site. The following summarizes the major areas of concern:

- Inhalation of airborne contaminants is considered a principal pathway of exposure. This conclusion was based on an initial screening of pathways based on exposure to PCBs. However, inhalation of airborne contaminants was found to contribute only 0.025 percent of total dose, while ingestion of aquatic biota, direct contact with sediments and ingestion of sediments contribute greater than 99 percent of the total dose (Table 2-2, pg. 2-15).

It is therefore not evident why this pathway which contributed such a small percentage of total exposure was considered important.

- Various exposure assumptions (e.g., sediment ingestion rates, gastrointestinal factors for metals) are the upper end of the range of estimated values and thus provide the opportunity for an overestimate of risk. It would be more appropriate to estimate risks for both a "typical case" (or average) and "reasonable worst-case" using separate exposure assumptions in each as proposed in USEPA's recently published Exposure Factors Handbook (USEPA 1989a) (e.g., for sediment ingestion rates an average value of 200 mg/day is recommended by EPA). In addition, sensitivity analyses should be performed on the exposure assumptions to determine the effect of the degree of uncertainty associated with the estimated risks.
- The toxicity profile for PCBs (Appendix D, pgs. D-1 through D-36) has various discrepancies and flaws. These are detailed in an attachment to this memo. However, the flaws do not effect the risk estimates presented in the Draft Report.
- The toxicity profile for cadmium includes discussion of an increased risk of cancer of the prostate in workers exposed to cadmium via inhalation (Appendix D, pg. D-39). That conclusion has been refuted (Doll 1985) and the profile should center on the increased risk of lung cancer. This will not however change the cancer potency factor used in the risk estimations.
- The toxicity profile for lead is incomplete (Appendix D, pgs. D-47+). Recent neurologic and behavior studies in infants and young children should be included. In addition, there is no EPA accepted AIC for lead (Table 3-1, pg. 3-4) (USEPA 1988). Work currently in progress in EPA's Office of Air Quality Planning and Standards (OAQPS) supports the use of a biokinetic/uptake model to estimate blood lead levels in children from exposure to specific environmental lead levels (USEPA 1989b). This approach should be developed in this document. The USEPA IRIS (EPA's on-line database) report for lead states the Agency's RfD (reference dose, formerly known as acceptable daily intake or ADI) Group considered it "inappropriate to develop an RfD for inorganic lead" (USEPA 1989c).

Attachment

References

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- Lees, P.S.J., M. Corn, and P.N. Breysse. 1987. Evidence for dermal absorption as the major route of body entry during exposure of transformer maintenance and repairmen to PCBs. *Am. Ind. Hyg. Assoc. J.* 48(3):257-264.
- Taylor, P.R., J.M. Stelma, and C.E. Lawrence. 1989. The relation of polychlorinated biphenyls to birth weight and gestational age in the offspring of occupationally exposed mothers. *Am. J. Epidemiol.* 129(2):395-406.
- U.S. Environmental Protection Agency (USEPA). 1988. June quarterly update for HEA and HEED chemicals. Environmental Criteria and Assessment Office, Cincinnati, Ohio. July 15.
- U.S. Environmental Protection Agency (USEPA). 1989a. Exposure factors handbook. Office of Health and Environmental Assessment, Washington, D.C. EPA/600/8-89/043. May.
- U.S. Environmental Protection Agency (USEPA). 1989b. Review of the national ambient air quality standards for lead: Exposure analysis methodology and validation. Office of Air Quality Planning and Standards, Research Triangle Park, N.C. EPA-450/2-89-011. June.
- U.S. Environmental Protection Agency (USEPA). 1989c. Lead and compounds (inorganic); CASRN 7439-92-1 (06/01/89). Integrated Risk Information System (IRIS) - on-line database.

ATTACHMENT A
COMMENTS ON PCB TOXICITY PROFILE

Appendix B

- o The toxicokinetic factors (TKFs) that are derived for the purpose of modelling absorbed doses of PCBs by direct contact are conservative, but appear reasonable.

Appendix D: Toxicological Evaluations

- o On page D-9 it states that "Dermal exposure to PCBs has not been well-documented in humans." Lees et al. (1987) present fairly good documentation for dermal PCB exposure in transformer repair workers and suggest that it is the major route of exposure of these workers.

- o The first paragraph on page D-17 refers to the results of epidemiological studies of PCB-exposed workers and Yusho victims in Japan (i.e., individuals who ingested contaminated rice oil). The text contains the following passage: Although the results of these studies are suggestive of cause and effect relationship between PCB exposure and cancer,..." This passage gives the impression that the evidence that PCBs are human carcinogens is stronger than it actually is. The Yusho victims also ingested polychlorinated dibenzofurans (PCDF) Congeners in the rice oil, some of which have a considerably higher toxicological potency than do PCBs. Also, none of the epidemiological studies of occupationally exposed cohorts provide compelling evidence of a cause and effect relationship between PCB exposure and human cancer. A similar statement is made on page D-30.

- o The second paragraph on page D-17 refers to the study by Kreiss et al. (1981) in which a direct correlation was reported between increasing serum PCB levels and elevated blood pressure in 458 volunteers who had consumed contaminated fish.

It should be noted that the first contained other contaminants (e.g., DDT) and that this relationship has not been observed in other PCB-exposed populations.

- o On page D-20, Jordan discusses reports of liver damage in PCB-exposed workers. It is important to note that this has only been documented in individuals with very high occupational PCB exposures (and high blood PCB concentrations).
- o The study by Taylor et al. (1984) of women occupationally exposed to PCBs is discussed on page D-26. A recent follow-up to the first study (Taylor et al. 1989) examines the same cohort of women but is based on more detailed interview data. In the more recent paper Taylor et al. report observing significant decrease in the gestational age of offspring as estimated PCB exposure increased. There was also a related decrease in the birth weight of these offspring. The authors note that the magnitude of these effects was small.
- o The results of an occupational epidemiological study of workers at capacitor manufacturing plants is discussed on page D-30. It is important to note that some findings in the most recent study (Brown 1987) are not supportive of a casual relationship between PCB exposure and excess cancer in the study cohort. There was no indication that an increase in risk was associated with an increase in PCB exposure (i.e., no dose-response relationship was observed). Also, risk did not appear to increase with the latency period. Adding further uncertainty to the finding is the fact that none of the cases of "liver and biliary tract cancer" was identified as primary liver cancer, suggesting that the cancers may have originated in organs other than the liver.
- o In the summary on page D-35, it states that "High serum PCB levels among pregnant women have been associated with increased abortions, premature deliveries, and decreased birth weight". This statement (with respect to increased abortions and premature deliveries) is not supported anywhere in the text and no supporting reference is cited on this page. A relationship between serum PCB

levels and the pregnancy outcomes of abortion or premature delivery has not been well documented.

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