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**Review of the "Draft Final Baseline Public Health
Risk Assessment; New Bedford Harbor
Feasibility Study, August 1989"**

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TABLE OF CONTENTS

	<u>Page</u>
1.0 <u>REVIEW OF THE RISK ASSESSMENT</u>	1
1.1 Poor Representation of the Findings	1
1.2 The Issue of Reasonable Exposure	2
1.3 Other Wrong Assumptions in the Risk Assessment	4
1.4 Other Problems in the Risk Assessment Methods	5
2.0 <u>REVIEW OF APPENDIX D</u>	7
3.0 <u>REFERENCES</u>	10

1.0 REVIEW OF THE RISK ASSESSMENT

This review examines the assumptions used in the risk assessment for Area I, PCB exposures. The assumptions for Area I risks have been emphasized because these are related to the largest calculated risks and result in the greatest errors. It is unfortunate that after going to so much trouble to quantify the risks from PCBs, the methods used provide no good estimates of the true risks.

The major flaws identified in this report can be roughly categorized into three groups as follows:

1. The findings from the main report are not properly abstracted into the executive summary.
2. The assumptions regarding frequency of exposure are absurd.
3. Other assumptions used in the calculations are not supported by the literature.

Even if the exposure assessment used assumptions that were aimed at an average exposure, the resulting risk assessment would be extremely conservative. EPA in the "The Risk Assessment Guidelines of 1986" has recognized this fact. The carcinogenicity potency factor used by EPA already has many conservative assumptions built in some of which are listed below:

1. Benign and malignant tumors are counted as cancer.
2. High to low dose extrapolation is done using the most conservative model available.
3. Surface area instead of weight is used for species to species conversion.
4. No threshold dose is used although there is ample evidence that PCBs act by an epigenetic mechanism.
5. All PCBs mixtures are treated as though they are Aroclor 1260. Studies have shown that lower chlorinated PCBs are less potent or do not cause cancer at the doses tested.

1.1 Poor Representation of the Findings

The Executive Summary of the report provides an extremely misleading representation of the findings of the main report. Similar problems are present in Section 4.2.3, the "Risk Summary." Only ranges of risk assessment calculations are given, and arguments are made based upon the upper limits of the ranges. After going through so many different scenarios, the reader is only given the values for the worst cases.

These worst cases are presented as still "realistic." However, the assumptions upon which they are based are clearly unrealistic.

ES-8 "Noncarcinogenic risk estimates for exposure to sediment in Area I exceeded 1 under the majority of scenarios evaluated, and ranged from 0.7 to 200." No differentiation is made between "probable" and "conservative" scenarios. For children 0-5 years of age, "probable" frequency of exposure was not even analyzed. This range includes calculations for 20 exposures per year by children ages 0-5 years in which approximately 1/3 of the body would be in contact with sediment for 24 hours. For adults and older children the assumption is 100 exposures per year in Area I.

ES-9 The same poor representation of the results is given for the carcinogenic risks from dermal exposure in Area I. The higher risks reported have been calculated using the same unrealistic frequency of exposure assumptions, excessive absorption rates, assumption of 24 hour contact, and excessive skin surface areas.

For ingestion of sediment, there is no mention made of the assumptions used and no differentiation is made between "probable" and "conservative" estimates. The higher risks reported in these ranges have been generated assuming 20 ingestions per year of 500 mg sediment in each of years 0-5. These exposure frequencies are inconsistent with "inadvertent" exposure, the amount consumed is too high, and the absorption factor used is not consistent with the literature.

ES-11 For ingestion of biota, there is no differentiation between "probable" and "conservative" scenarios. These ranges presented include assumptions which are clearly absurd, yet there is no attempt to present any kind of a balanced overview of the results. For example, the inclusion of children 0-5 years consuming 4 ounces of seafood, including tomally, daily is included in the calculations.

1.2 The Issue of Reasonable Exposure

The body of the report is extremely confusing regarding the reasonableness of exposure calculations. There are numerous contradictions regarding "likely" exposure scenarios. The rhetoric claims that probable scenarios are used, but the actual numbers used tell a different story. Some examples follow:

ES-4 "These scenarios were based on a various exposure conditions, primarily focusing on areas where exposure was considered likely to occur." Yet, Area I scenarios are then developed for "unlikely" situations. (See 2-5)

2-5 "However, exposure to contaminants by this age class is expected to be limited, given that children under age 5 are generally supervised and have limited mobility. Therefore, they are unlikely to be playing in areas of high contamination."

2-9 The description of the conditions of the upper estuary do not appear to be conducive to exposures. This would certainly be true of the 0-5 year and adult population. The description of the Upper Harbor provides an equally unattractive description of the water's condition.

2-14 One definition of "extremely conservative exposure assumptions" is given: maximum contaminant level, repetitive exposure over 70 years.

21-5 Note to the table. "For example, exposure to children ages 0-6 was evaluated because it is possible that this age class could be exposed 24 hours/day."

2-16 Quantitative exposure assessment is described as "realistic exposure considerations" and "one based on 'average' or probable or moderate exposure conditions, and the other on 'conservative' exposure conditions. Together,, these scenarios provide a range of potential exposure levels, within which the actual exposure for a particular individual would likely fall." However, the same "very conservative" assumptions used in the screening scenarios are used as one set of parameters for the quantitative exposure assessment. The results of these scenarios are the ones reported in the Executive Summary as the upper range results. The more "realistic" or "probable" do not make it to this summary.

2-22 Even though the exposure to sediments in Area I is described to be possible by "inadvertence", 1-20 exposures per year for a lifetime are the numbers used for calculations. The definition of "inadvertence" is "an effect of inattention: a result of carelessness: an oversight, mistake, or fault from negligence." Although it is possible to accept one such episode per year during the years 8-16, it is impossible to describe 20 episodes per year for a lifetime as "inadvertent" exposure. It is impossible to see a child 0-5 years of age being exposed in any way to the sediment in Area I without running a huge risk from drowning. It is difficult to foresee any parent allowing such an exposure.

2-25 Table 2-6 gives a summary of exposure assumptions, which will be evaluated below.

2-26, 2-29 Some very absurd assumptions are made about the quantity of local seafood eaten. "These values were decided after a review of the literature failed to provide a site-specific value applicable to recreational consumption of fish and shellfish." Yet a great deal of literature exists which indicate that fish consumption by adults is between 6-14 g/day divided between locally caught and commercial products. However, EBASCO decided to use 227 g on a daily basis as one assumption.

4-5 The discussion about quantifying parameters that are not directly observed (eg., frequency and duration of exposure) reports to chose ranges within which all individuals receiving exposure would fall. This is not true because exposures of less than one per year have not

been included in the analysis. In Area I for a 0-5 year old child, if a possible exposure does exist, the most likely would be one per lifetime within this age range. The same would also be true for older children and adults.

4-8 "Since there are no recreational areas located within Area I and children (0-5) have limited mobility, exposure to sediment in Area I was estimated to occur between 1 and 20 times per year." This statement does not make sense. If the first part is true, the exposure should be zero to one time per lifetime for an unexpected, truly inadvertent exposure.

4-13 "The risk ratios for these [conservative] scenarios ranged from 6 to 93. The magnitude to which these values exceed 1 indicates that exposure to PCB-contaminated sediment in this area [Area I] presents a public health risk." This statement presents PCBs as a public health hazard for noncarcinogenic health effects. The calculations did not show this to be the case for the "probable" scenarios. Therefore, this statement is attempting to make a case for public health risk because so many of the calculations using "conservative" assumptions give a ratio of greater than one.

A review of the assumptions for chronic exposure is given on Table 2-6. The "conservative" assumption is that a child 0-5 year will have 20 exposures per year to Area I, in which he will have forearms, arms hand, lower legs and feet completely immersed in sediment for 24 hours. Likewise for an older child and adult this is assumed to happen 100 times per year.

Also in this table, the frequency of exposure assumptions are the same for Areas I, II, and III for older children and adults. This contradicts statements made on pages 2-9, 2-10, and 2-18 regarding the likelihood of exposure of Area I versus Areas II and III.

4-15 "For area I, risk ratios were derived assuming only conservative exposure assumptions, since the probable exposure scenarios assumed on 1 exposure per year which represents an acute versus chronic exposure". Therefore, the only calculation done assumes that a child 0-5 years of age would have "inadvertent" exposure every year, 20 times per year to this very unappealing mud. Again, it is hard to imagine parents who would allow even one such exposure in Area I during a any of a child's years 0-5.

Appendix C: The tables in this appendix list the "conservative" estimate as the "realistic worst case." This means that the risk assessors consider 20, 100 and 100 exposures per year in Area I for 0-5, older children, and adults to be "realistic." It is difficult to understand that "inadvertent" exposures could occur with this frequency and be classified as realistic.

1.3 Other Wrong Assumptions in the Risk Assessment

2-20 In Table 2-3, for exposure Via Direct Contact, TKF as used is not a unitless factor. It a rate of absorption, i.e., percent/24 hours. Therefore, absorption must be adjusted for the proportion of a 24 hour day that the sediment is in contact with the skin. The animal absorption studies are reported for 24 hour exposure. Additionally, Shu et al. (1988) found that the four hour absorption rate for TCDD was approximately 0.5%.

2-24 The assumption of a 0.07 TKF for PCBs by dermal absorption is derived at great length in Appendix B. Reference is made to "Jordan," but no citation is given supporting this reference. Although no studies exist which directly measure PCB bound to soil, TCDD literature does exist. However, only one such reference is quoted in Appendix B, which makes these calculations out of date. Shu et al. (1988) reviewed the literature and their own data in a refereed journal article and concluded that dermal absorption of TCDD in soil would be one percent at the greatest. Their data gave 1.5% absorption for 24 hour contact, but the authors concluded that the 24 hour absorption would be less than 1%. The literature shows a 3-10 fold greater absorption by rats compared to humans for similar compounds.

For oral absorption, studies of TCDD bound to soil by Poigner and Schlatter (1980) found 65% and 44% absorption for TCDD bound to soil after 10-15 hours and eight days, respectively. Absorption would be less for more tightly bound TCDD. Umbreit et al. (1986) reported 0.5% and 26% absorption for soils from two different TCDD contamination sites. Shu et al. (1988) reported a mean bioavailability rate of 43% for TCDD in the soil. It is extremely puzzling that Appendix B does not utilize any of these data to derive the TKF.

For inhalation, a value of 1/2 or 1/3 instead of one is usually used for a TKF. Although the tidal volumes quoted in Table 2-4 are correct, only a fraction of this volume reaches the part of the lung where absorption occurs, and only a fraction of the PCB which reaches this area is absorbed.

2-26 LaGoy (1987) estimated "average" ingestions to be 100 mg per day. The amount assumed is five times this for "average" daily exposures.

For biota ingestion 115 grams/meal is assumed for 0-5 years. This is clearly a ludicrous assumption for 0-2 years, and a highly unlikely assumption for 3-5 years of age. To make matters even worse, the assumption is made that this child 0-5 years of age consumes the tomally from a lobster as part of these 115 grams. Next, these risk assessors calculate ingestions for monthly, weekly and even daily consumption.

1.4 Other Problems in the Risk Assessment Methods

Some additional problems are illustrated in the following:

ES-1 "While it is probable that natural processes such as biodegradation and photolysis will result in a decrease in PCB concentrations in sediment and biota, these changes are not expected to be significant over the next 10 years." However, the risk assessment calculations cover the next 70 years.

4-28 "The lifetime risks were estimated by summing the incremental risks associated with exposure during 0-5 years, 6-16 years and 17-70 years." This procedure is only proper if the dosage rate is prorated for lifetime exposures for each of the age groups. For example, the 0-5 years risks would have to be multiplied by a factor of 6 years/70 years.

2.0 REVIEW OF APPENDIX D

This review is confined to the PCB section in this toxicological evaluation. The authors have given a very unbalanced view of the literature. In many instances, only the studies reporting a PCB-related finding have been included without presenting other studies that have looked for but not found such effects. Specific criticisms are given in the following paragraphs.

D-5-D-7 The review of the literature involving oral absorption has omitted some of the most significant literature. Although the document states on D-5, "In addition, matrix effects can significantly alter absorption behavior," there is no inclusion of the pertinent matrix studies. Although no studies exist for PCBs, experiments have been done for TCDD, which would be expected to have similar physical properties. Studies of TCDD bound to soil by Poigner and Schlatter (1980) found 65% and 44% absorption for TCDD bound to soil after mixing the TCDD with the soil for 10-15 hours and eight days, respectively. Absorption would be less for more tightly bound TCDD. Umbreit et al. (1986) reported 0.5% and 26% absorption for soils from two different TCDD contamination sites. Shu et al. (1988) reported a mean bioavailability rate of 43% for TCDD in the soil.

D-7-D-10 The most relevant of the dermal absorption studies of TCDD bound to soil was omitted from this review. Shu et al. (1988) reviewed the literature and their own data in a refereed journal article concluding that dermal absorption of TCDD in soil would be one percent at the greatest. Their data gave 1.5% absorption, but the literature shows a 10 fold greater absorption by rats compared to humans for similar compounds.

D-17 In the first paragraph reference is made to Yusho studies as "suggestive of a cause and effect relationship between PCB exposure and cancer." However, no supporting evidence is given in this report of these claims. On page D-23, in discussing the Yusho incident the report states, "...the potent toxicants polychlorinated dibenzofurans (PCDFs) were also consumed. Thus, the effects cannot be solely attributed to the PCBs themselves."

D-17 Blood pressure was positively correlated with serum PCB levels in one study (Kreiss, 1981); however, the author later characterized the finding as uncertain (Kreiss, 1985). Elevated blood pressures were not reported in 10 other published studies (Emmett, 1988b; Akagi, 1985; Takamatsu, 1984; Stehr-Green, 1986; Fischbein, 1979; Baker, 1980; Chase, 1982; Acquavella, 1986; Smith, 1982; Maroni, 1981).

D-17 "Accidental human ingestion of PCB-contaminated rice oil in Japan and Taiwan resulted in effects similar to those seen following occupational exposure." This statement is not supported by the literature. Several studies have compared Yusho disease to workers with high levels of exposure to PCBs as evidenced by high blood PCB levels. Recently, Kashimoto and Miyata (1986) have reviewed this information

including articles published only in Japanese. They concluded that PCDFs are the only probable explanation for the clinical manifestations of Yusho disease, and that Yusho disease is symptomatically and etiologically different from PCB effects. These conclusions were based upon the following findings comparing Yusho and Yu-Cheng findings (an episode occurring in Taiwan 10 years after Yusho with contaminated rice oil and a similar PCB/PCDF ratio) with occupational exposures to PCBs in Japan:

1. Yu-Cheng patients and Japanese workers had similar PCB blood levels, but the Yu-Cheng patients had severe clinical disease whereas the workers had few if any findings.

2. Five years after the Yusho episode, PCB blood levels were in the normal range, yet there was a persistence of clinical findings. In Japanese workers with highly elevated levels, there were no such findings and even the mild dermal lesions disappeared soon after cessation of exposure.

Additionally, much other information shows the predominance of PCDFs in the Yusho episode. Evaluations of the oil consumed by Yusho patients have shown that the weight ratio of PCB to PCDF was about 200 (Miyata et al., 1977). In contrast, the ratio of PCB to PCDFs in commercial Aroclors is usually greater than 500,000 (Bowes et al., 1975; Rappe et al., 1980). PCDF contamination in the Yusho episode was generated while the PCBs were used as a heat exchange fluid and maintained for a prolonged period at high temperatures.

Several lines of evidence based upon animal toxicity and biochemical markers have shown that the toxicity of PCDFs are very potent compared to the commercial Japanese PCB mixture implicated in the Yusho. Investigators in Japan have concluded that PCDF's were the major causative agent in Yusho (Kashimoto and Miyata, 1986, Masuda and Yoshimura, 1984, Kunita et al., 1984). In contrast, the low concentration of PCDFs in commercial Aroclors would not be expected to contribute to any adverse health effects.

D-19 Enlargement of the liver has been reported in only one study in humans (Maroni, 1981).

D-20 Drinker (1937) reported deaths due to exposures primarily to chlorinated naphthalenes in which one case was also reportedly exposed to PCBs. This is not accurately reflected in the report.

D-20 The study by Kriess et al. (1981) and the Yusho studies have been commented on previously.

D-31 None of the liver/biliary cancers reported by Brown (1987) were primary liver cell cancers. Of the five cancers in this category, one cancer was from the gall bladder, three from the bile ducts, and one metastatic tumor with primary site unknown. An examination of the site-specific mortality showed that an association with cancer of the rectum originally reported by Brown and Jones (1981) did not remain when the additional person-years were added in this study. Associations found in

one study with site specific cancer (or other disease) were contradicted by other studies: Bahn (1976), malignant melanoma; Brown (1987), liver and biliary cancer; and Bertazzi (1987), hematologic and gastrointestinal cancer. The gastrointestinal cancer increase of Bertazzi was not indicative of a liver or biliary cancer increase since there was only one case of each.

D-33 "At this time there is no experimental support for this [PCBs are promoters rather than initiators] hypothesis." This statement does not accurately reflect the literature. Many studies have shown that PCBs act as promoters of cancer when given after initiating (mutagenic) agents. Preston et al., (1981) showed convincingly that Aroclor 1254 was also capable of increasing the incidence of hepatocellular carcinoma initiated by prior treatment with diethylnitrosamine (DEN). Nishizumi (1976, 1980) reported that Kanechlor 500 accelerated the development of liver tumors in rats exposed previously to DEN. These promoting effects also could be produced with DDT and phenobarbital. Kimura et al. (1976) showed that Kanechlor 400 was capable of increasing the incidence of hepatocellular carcinoma when given after 3'-methyl-4-dimethyl-aminoazobenzene (Me-DAB).

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