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Environmental Medicine
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CRITIQUE

*Draft Final Baseline
Public Health
Risk Assessment*

*New Bedford Harbor
Feasibility Study*

October 12, 1989

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1. Introduction

This critique of the "Draft Final Baseline Public Health Risk Assessment New Bedford Harbor Feasibility Study" (Ryan and E.C. Jordan Company 1989) was prepared by Dr. Rudolph J. Jaeger, Ph.D., DABT of Environmental Medicine Incorporated. Dr. Jaeger's Curriculum Vitae is attached as Appendix 1.

As a result of previous unrestricted use, PCBs are frequently found in the environment. The New Bedford Harbor (NBH) is known to be contaminated by PCBs as well as other toxic materials, e.g. oil, grease, gasoline, unburned and partially combusted hydrocarbons (i.e. Polycyclic Aromatic Hydrocarbons, PAHs), lead, cadmium and copper among other substances. While of considerable importance to public health, issues related to these other toxic substances have been excluded from the present review.

A site-specific cleanup by "operable unit" has been proposed for the NBH. The cleanup will be limited in scope and will not remove a large number of other materials from the NBH and thus, the present risk assessment by E.C. Jordan creates a false degree of assurance concerning the proposed risks, the utility of any cleanup which removes only a portion of the referenced materials and the degree of risk reduction to be expected by the public, but not to be fulfilled for the Harbor as a whole by a proposed "operable unit cleanup" even following the expenditure of large sums of money.

The Risk Assessment (RA) document cited above was prepared at the request or under the direction of Ebasco Services Incorporated by Elizabeth A. Ryan of the E.C. Jordan Co. The calculations presented in the document were performed by the contractor, E.C. Jordan, using spreadsheet analysis done in Lotus 1-2-3 (version 2.01) format. Magnetic disk copies of 6 spread sheets were provided by the EPA to Dr. Jaeger.

2. Summary of Opinion

Based on my review of the RA, its associated tables and spreadsheets and from my review of other literature and opinions in this matter, I conclude that the RA document is incorrect. As a whole, the RA seeks to apply unreasonable and overly large estimates of exposure (estimates of dose greater than are likely to be true) coupled with incorrect and inflated estimates of cancer potency leading to exaggerated estimates of cancer risk (larger probability values than likely to be true). The calculated risk estimates are, therefore, wrong. The reason given within the RA and elsewhere to consistently make such an error is the stated need to protect public health and never to underestimate risk. Thus, the estimates are called "conservative." Risk assessment, as a mature science, must develop estimates which neither overstate nor understate the chance of harm. The document concludes, based on its scenarios, that a significant risk now exists even though such a conclusion is not the most likely from the available data. The public health evidence, at least for the PCB class of contaminants, contradicts the assertion that a health risk does, in fact, exist in New Bedford Harbor from PCBs.

3. Demographics

The population described in Table 1 of this review (Table 2-1¹ of the RA) asserts that most of the population in the affected area resides in New Bedford to the west and south of the "hot spot." Based on the population distribution map (figure 2-3 of the RA, reproduced here as Figure 1 of this review), the geographic distribution of population lies almost totally to the southwest of the harbor. The majority of the population is likely to be far removed from the "hot spot." A summary and tabulation of the population data by compass quadrant are shown in Table 2. The "center of demographic gravity" (the area where the majority of the population occurs) appears to be toward the southwest, an area removed by a significant distance from the "Hot Spot."

The RA presents an exposure assessment that is driven by selected extreme or average PCB exposures derived largely from the area defined as a "Hot Spot" which lies in Area I, the northern most portion of the harbor and near the AVX/Aerovox facility. It is concluded by the RA that persons, particularly children, who live near or who may travel to the "Hot Spot" area, will receive significant PCB exposures by skin contact or ingestion. The area that such children would need to access is below the Wood Street Bridge and the

¹ The values given in this table were checked for accuracy and errors were found. The correct values are shown in table 1.

surrounding environs north of the Coggeshall Street Bridge. Persons visiting this area, specifically those coming into contact with the sediments contained in them, are postulated as being likely to come into contact with PCB contaminated sediments derived from the "Hot Spot" and surrounding mud flats. Based on personal observation of the area by this reviewer, namely, the low lying mud flats which are adjacent to the harbor and the Aerovox/AVX facility as well as the surrounding property, it is my opinion that few persons of the kind described by the RA as being at particular risk are likely to be attracted to the sediments in this area for any legitimate or recreational purpose.

4. Land Use

The industrial facilities and low lying wetlands that surround the Harbor's headwaters are not attractive to children under 6 years of age; children are blocked from free access by the presence of industries and fences on the west side of the "hot spot" area and the soils (mud flats) will not support the bearing weight needed for free locomotion over the surface. Wading is also listed as a possible activity in the area. Only in grassy wet-lands (located on the east side of the harbor) is there a possibility of free movement and in such areas, if traveled or ridden on by older children, the grassy mat and other natural fibrous debris provide an effective shield or barrier precluding exposure to the underlying sediments.

5. Principal Exposure Pathways

It is clear that exposure scenarios which suggest significant contact by ingestion, inhalation and dermal exposure, with proper selection of risk potency factors, could lead to the erroneous conclusion that a hazard exists. Hazard is the product of dose times risk per unit dose. Dose is only estimated from a series of "What-if" calculations that are subject to varying degrees of uncertainty. Such estimates lack the means for verification. In the present context, these daily doses are said to be added and divided by the time period in question to yield an average daily dose which is then used to calculate risk.

5.1. Estimation of an Average Daily Dose

The calculation for a source based Average Daily Dose (ADD) for any toxicant, in the present case, PCBs, is usually performed as follows:

$$\text{ADD} = \frac{\text{(Total Intake of the PCB mixture)}}{\text{(Body Weight) * (Averaging Period)}}$$

The averaging period may be a single exposure interval (less than a day such as might occur with a young child eating dirt on a single occasion), multiple exposures during a longer period of time such as might be related to food ingestion, repeated visits to a contaminated site or ingestion of water (e.g. weekly/monthly/annually over a number of years) or a lifetime (70 year) average daily dose.

The doses of PCBs received via different routes of exposure are assumed by the RA to be additive in terms of the average daily dose over time. The factor of exposure route is not given special weight in the estimate of risk or the ratio that an exposure might exceed a standard or guideline value. The exception to this is given as a Toxicokinetic Factor (TKF) where each route is assigned a different degree of uptake likelihood, i.e. from 0 to 100%.² The choice of a TKF is usually done on "empirical" grounds and except for animal data, few studies exist in man that the RA might use as a basis for its assumptions.

For the New Bedford Harbor risk characterization, and as noted above, only PCB exposures estimated in the RA are being considered in this critique, although other toxicants are found in the harbor³. These include a variety of Oil and Hazardous Materials (OHMs⁴) as well as metals (e.g. other hazardous materials) such as copper, lead and cadmium.

The calculation of a source based ADD for the intake of PCBs from water or sediment by adults or children has been attempted in the RA. The methods are based on spread sheet systems that employ Lotus 123 as the calculation engine. The average daily dose, whether it occurs daily, weekly, monthly and so on will determine the estimate of risk. The calculation of the ADD due to the ingestion with water, sediment, food or air may be calculated in the following way:

² We observe that a recently released Record of Decision (ROD) for another Superfund site, namely Wells G&H in Woburn, MA (ROD issued 9/14/89) lists the following assumptions for PCBs: Ingestion absorption factor 0.3 and Dermal absorption factor 0.02. For lifetime exposure, the incidental ingestion rate is given as 54 mg/day for soil with a frequency of 100 exposures per year. The dermal contact rate is given in the ROD as 790 mg/day.

³ Another consulting firm, TERRA Inc., has highlighted the issue of Polycyclic Aromatic hydrocarbons as being significant contaminants within the Harbor, which the present RA chooses to ignore and yet, the PAH surrogate is considered to be more potent and at least as carcinogenic as the PCBs.

⁴ The terminology of OHM is being used since it is referenced in the Massachusetts Contingency Plan (MCP).

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$$ADD_{\text{receptor}} = ADD_{\text{water}} + ADD_{\text{sediment}} + ADD_{\text{food}} + ADD_{\text{air}}$$

ADD_{water} , ADD_{sediment} , ADD_{food} and ADD_{air} are the daily dose estimates from water exposure, whether in household drinking water or surface water, sediment exposure, food intake and air exposure. Over a period of time, these values can be the basis for averaged estimates of dose. Each medium, respectively, may potentially contain PCBs as well as other OHMs which could also pose their own specific health risk. The daily dose estimates can be defined as the following:

$$ADD_{\text{water}} = ADD_{\text{water-ingestion}} + ADD_{\text{water-dermal uptake}}$$

while

$$ADD_{\text{surface water}} = ADD_{\text{sw-ingestion}} + ADD_{\text{sw-dermal uptake}}$$

and

$$ADD_{\text{sediment}} = ADD_{\text{sed-ingest}} + ADD_{\text{sed-dermal uptake}} + ADD_{\text{sed-inhaled}}$$

and

$$ADD_{\text{food}} = ADD_{\text{biota}} + ADD_{\text{dairy}} + ADD_{\text{meat}} + ADD_{\text{other}}$$

The subject compounds or mixtures of compounds could be ingested from contaminated drinking water ($ADD_{\text{water ingestion}}$), whether due to drinking tap water or surface water (e.g. $ADD_{\text{sw ingestion}}$), sediment ($ADD_{\text{sediment dermal uptake}}$) or food, e.g. biota and other food sources which may contain PCBs or other OHMs (ADD_{food}). They may also be dermally absorbed (as estimated by $ADD_{\text{water dermal}}$), or inhaled following volatilization or vaporization/mist/dust formation from the subject source (e.g. $ADD_{\text{sediment inhalation}}$). Of these parameters, the RA only considers ingestion of biota (food), dermal contact with sediments and ingestion

of sediments as being important (see screening scenario calculations in Tables A-1 through A-6).

5.1.1. Ingestion of Contaminated Sediment

For ingestion of contaminated sediment, the following applies:

$$ADD_{\text{sediment ingestion}} = (\text{OHM}_{\text{sed}} * \text{AI} * \text{BAF} * \text{D} * \text{C}) / (\text{BW}_{\text{avg}} * \text{AP})$$

where OHM_{sed} (in the present case, PCBs) is the PCB concentration in sediment during the exposure period i.e. in units of ug/kg (ppb or comparable), where AI is the average daily amount ingested, e.g. in grams of sediment per day, where BAF is the bioavailability factor for ingested sediment (called TKF by the RA), i.e. sometimes estimated at 100% for GI uptake (the reader should note that other EPA documents cite 30% as an acceptable uptake factor for ingested PCBs), where D is the duration of exposure in days or fraction thereof, e.g. to account for time spent at other locations, where C is a units conversion factor, where BW_{avg} is the average body weight i.e. 10 kg for young children, 40 kg for older children and 50 to 70 kg for adult females and males, respectively, and AP is the averaging period in units of days.

5.1.2. Dermal Absorption of PCBs from Sediment

For dermal absorption of OHM via sediment, the equation that might be used is given below:

$$ADD_{\text{sed dermal uptake}} = (\text{OHM}_{\text{sed}} * \text{SA} * \text{PC} * \text{F} * \text{D} * \text{C}) / (\text{BW}_{\text{avg}} * \text{AP})$$

where $\text{OHM}_{\text{sediment}}$ is the representative concentration of the OHM in the sediment at the exposure point during the period of exposure (in units of mass/volume); SA is the skin surface area in contact with the sediment during the periods of exposure (in units of area); PC is the permeability constant; F is the number of exposure events during the exposure period; D is the duration of exposure and C is a units conversion factor. Other factors are as previously defined.

5.1.3. Inhalation of OHM in Air

The ADD received via inhalation of volatile or dust borne OHM ($ADD_{inhalated}$) may be estimated using the equation presented for the inhalation of gaseous or particulate OHM in air

$$ADD_{inhalated} = (OHM_{air} * VR * BAF * F * C) / (BW_{avg} * AP)$$

where OHM_{air} is the representative concentration the OHM in the air at the exposure point during the period of exposure (in units of mass/volume); VR is the daily respiratory volume for the receptor of concern during the period of exposure (in units of inspired volume/day) and all other units are as stated previously.

The total ADD for the uptake of aqueous and sediment bound OHM is not easily calculated for each OHM and for each potential human receptor, namely that of an adult, young child and older child. There are numerous qualitative and quantitative problems in such calculations. The present effort by the RA contractor is a crude and exaggerated approximation of the true risk which is likely to be much less than the stated value.

The constants for the ADD equations for ingestion, dermal uptake, inhalation and other parameters for each human receptor may be taken from a variety of sources. One such source is the Massachusetts Contingency Plan (MCP) Guidance Document. Other documents are also known to be available and offer a variety of estimates. The guidance documents available do not always agree and thus, the choice of assumption leads to variation in the estimate.

Example of Risk Calculation Parameters⁵

<u>Parameter</u>	<u>Adult</u>	<u>Young Child</u>	<u>Older Child</u>
AP	55 Years	5 Years	10 Years
BAF ⁶	Variable	Variable	Variable
BW _{avg}	70 kg	40 kg	10 kg
F	Variable	Variable	Variable
D	Variable	Variable	Variable
PC	Unavailable ⁷	Unavailable	Unavailable
SA ⁸	1.8 m ²	0.688 m ²	1.19 m ²
AI	Variable	Variable	Variable
VI	2 liters/day	1 liter/day	1 liter/day
VR ⁹	20 m ³ /day	5 m ³ /day	20 m ³ /day
C	As Required	As Required	As Required

In cases where parameters are not known, both the RA, other reviewers and the present critique have attempted or will attempt to apply default assumptions. When no default assumptions are available for those parameters necessary to solve the equations for estimation of ADD by ingestion, dermal routes or other

⁵ The estimates given above are taken from the RA and from applicable portions of the MCP guidance document. Other values can be found and thus, no single estimate should be considered correct except as it specifies a range or a set of conditions.

⁶ The Bioavailability factor or TKF will depend on the material being assessed. "Conservative" estimates frequently use 100% for GI tract and inhalation values when no better estimate are available. As noted previously in the text, EPA guidance has used 30% for GI tract uptake and 2% for dermal uptake for PCBs. Inhalation uptake will depend on the degree of deposition and subsequent solubilization.

⁷ The permeability constant used in any given calculation must be based on published numbers for the dermal penetration of the subject OHM. In general, permeability data for the subject class of chemicals are not available.

⁸ The issue of surface area is usually taken as fixed but is variable with age and sex. There is a normal distribution among children and adults that should be considered when this factor is estimated.

⁹ This value is quite variable but it is usually taken as fixed. Deposition of particles and gases will often vary with respiratory rate and rhythm as well as depth. The true respiration rate and corresponding degree of deposition will depend on the activity level of the individual.

routes of exposure, a conservative or reasonable basis for approximation, e.g. an estimate, is usually employed. There is no basis to conclude that such assumptions are, in fact, correct for any given individual. By this approach which is also suggested in the MCP as well as EPA guidance documents, the RA seeks to make its estimate of dose a conservative overestimate, and as such, it is likely to overstate the true risk. From this, the ADDs for ingestion and dermal exposure are claimed by the RA to be reasonable and through the choice of scenario, "conservative." I judge this combination to be a semantic implausibility.

In Table 2-2 of the RA, a total average daily dose of 6.8×10^{-2} mg/kg-day is proposed, based upon screening scenarios given in Tables A-1 through A-6.

Using computer spreadsheet techniques similar to those used by the authors of the RA and with the data taken from the tables, I was unable to replicate the exact values given by the authors. I conclude that these estimates are likely to be no more accurate than the parameter estimates which underlie them.

I was provided with examples of Lotus 1-2-3 spreadsheet files which were employed by the contractor to prepare the tables. I was able to review these, both with Lotus 1-2-3 and with PlanPerfect, a Lotus compatible spread sheet of comparable precision. I conclude that the values in the Tables are accurately transferred. I do not believe that these estimates are realistic or even correct. As to their correctness, they appear to be mathematically consistent but I conclude that they are substantial overestimates of exposure opportunity, exposure dose and as a consequence, they misstate the true risk.

It appears from the RA that 98.7% of the average exposure dose, as stated in the RA, is postulated to occur over a relatively short period in a person's life, either 3 years or 10 years. The postulated direct contact with sediments and resulting dermal uptake of PCBs by 6 to 16 year old children is stated by the RA to lead to an average daily dose 5.7×10^{-2} mg/kg-day for the 10 year period from age 6 to 16 years of age. This is 84% of the estimated dose but it is not clear if this degree of exposure is likely over a lifetime. While such juvenile and teen age persons are capable of travel to the contaminated area, there exists no objective data to support the conclusion that such contacts do, in fact, occur with any regularity or at all.

The second most significant exposure that is postulated by the RA is given for children from 0 to 6 years of age who are expected to ingest sediments at a postulated dose of 1×10^{-2} mg/kg-day for a period of 3 years. This is stated to be 14.7% of the dose derived from the screening scenario estimates. In order

to get such ingestion exposures, the juveniles (ages 0 to 6 years of age) must receive sediment contact as posed by the screening scenario. Even so, such exposures could not occur year round. Young children might engage in "sediment-play" from May through October but even this is unlikely. Such children (ages 3 to 6) are rarely allowed to roam freely and certainly are not likely to engage in unsupervised play in or on a mud flat within an industrial area known to be contaminated by PCBs, among other substances, e.g. oil, gasoline, PAHs, glass, metal, sewerage, water hazards, etc.

6. Quantitative Exposure Assessment

6.1. Sediment

The most contaminated material in the harbor is said to be the sediment and mud located in the northern part of the harbor. The highest degree of contamination is most frequently underwater and unlikely to be accessible to children, large or small. Access to this area is highly limited and the postulated significant contact with highly contaminated materials is not likely to occur at the levels or with the frequency listed in the RA.

It is noted in the RA that the screening scenario dose estimates are not used for the quantitative risk assessment given in section 4.0 of the RA, "Public Health Characterization." Yet, the accuracy of the screening scenarios and their verification by an external reviewer should support the assumptions that are carried throughout the document. Without having the exact formulas, constants and equations used by E. C. Jordan for all of the estimates, it is not possible to verify the accuracy or even correctness of the large number of tables and risk estimates given in this RA.

6.1.1. Direct Contact with Sediments

In the screening scenario (Table A-2), the dermal toxicokinetic factor is listed as being 0.5 (50% uptake) and this value applies to sediments containing PCBs at concentrations greater than 1% (>10,000 ppm). This numerical estimate is different from the 0.07 (7%) value given elsewhere in the document as being applicable to sediments of lower PCB content. Both values are said to be derived from EPA estimates and thus, to the extent that the Agency is correct about any of these dermal absorption estimates, they could be legitimate factors to use in a screening scenario. However, if the PCB concentration were incorrect or over-estimated as cited in Table A-2 (i.e. not over 1%, 10,000 ppm PCBs, as reported by Battelle) by as little as a factor of 2, the resulting re-estimate of dose by dermal contact would be decreased fifteen fold as a consequence of the stated assumptions. The true average daily dose value from

this screening survey could be lower still if more accurate (or true) values were employed. Thus, an 84% dose fraction from sediment exposure is not true or correct and the actual value is likely to be much lower than the stated value.

The reader should refer to Table 2-5 where the actual shoreline concentrations are given and the value cited for the entire area is listed as 6,393 ppm (maximum value). The mean value is much lower by a factor of approximately 17 fold (378 ppm). This difference is smaller for the cove area where the maximum value is listed as 399 ppm while the mean value is 286 ppm. It is this cove area which is cited by the RA as the area where close approach to contaminants is most likely for small children since the cited area is said to be adjacent to a public playground.

6.1.2. Ingestion of Sediment

Controversy exists over the degree to which young and older children ingest sediments, dirt and other materials in their home and play environment. The RA goes to secondary sources to choose an applicable value for "pica" type dirt and sediment exposure. The value used, 0.5 g per exposure, developed by LaGoy in 1987, is still considered by some to be excessively large. Recent EPA guidance indicates that 200 mg per day may be an acceptable estimate. The stated value may be more appropriate for household dust and backyard dirt but is less likely to be true for soils, sediment or mud derived from hydrated soils found in New Bedford Harbor. The true value is likely to be less than 500 mg in any case.

6.2. Biota

The ingestion of biota is listed by the RA to include winter flounder, softshell clams ("steamers") and lobster. The ameliorating effect of cooking is not considered by the RA and it may be a substantial modifier of dose, e.g. fried versus broiled foods.

The scenarios developed for each species (clams, winter flounder, and lobster) assumed that 100 percent of the seafood diet was from one of these species (Page 4-49, section 4.2.3.3). Each scenario was performed for each seafood source and the risk was estimated. This is not a valid assumption for this area with its substantial ocean fishing industry and large variety of available seafood. The RA in Table 2-8 on page 2-28 demonstrates the prevalence of various seafood consumption patterns for New Bedford area residents. Notwithstanding the inherent limitations of a prevalence (cross sectional) study of residents of Greater New Bedford participating in a PCB Health Effects Study, the sum of lobster, flounder (also included in this category are scup, tautog, fluke flounder,

cod, and sea trout), and clams (quahogs also included) total 53 percent of the seafood consumption of the group sampled. Therefore, these three species probably total less than 50% of the seafood consumed by the area residents. This fact alone invalidates the exaggerated assumptions made by the RA.

The magnitude of the derived risk for the ingestion route of exposure is driven by the inclusion of tomalley (lobster hepatopancreas) with its concentration of PCB content (Pruell et al. 1988). The deletion of this factor or modification of the estimated uptake from this source would result in a reduction in exposure in children and adults by at least a factor of 6.2. If the tomalley is not considered, then lobsters taken from Area III would meet the applicable FDA guideline. Even a lobster taken from Area I would meet the FDA criteria if whole body PCB concentrations are determined (1131.4 ppb or 1.13 ppm in a large lobster taken from Area I, according to Hillman et al. 1987).

The draft RA, page 2-33 (Table 2-10), cites the median PCB concentration in edible muscle and in hepatopancreas from lobsters in Area III. The origin of these estimations, BOS 1987, is not given in the Bibliography (Appendix B) of the RA report. I have obtained a copy of this report and the value given is actually the arithmetic average of two measurements in whole body, edible tissue and lobster hepatopancreas from Area III. The use of the descriptive statistic, median, is correct when a number of values are enumerated and is correctly used for the other values but not for Area III which has but two measures. Since the PCB concentrations appear to have been averaged from Area III, it is the mean concentration that was used as the basis for the ingestion dose estimates. These may or may not be representative of all areas or even other areas.

Whether children of New Bedford eat tomalley, the true frequency that lobster is available or if young children even eat lobster at all, is not established by the RA. Such dietary practices are not common to young children in my experience but New Bedford Harbor may be a special case which might justify such assumptions. Where commercial fishing is an important livelihood, the sale of a cash crop like lobster may be more important for the family livelihood. The children of parents who fish recreationally might eat lobster on occasion but the scenarios given for this practice are clearly not reasonable.

Table 4-9 presents the carcinogenic risk estimates for the ingestion of biota in children, older children, and adults in New Bedford. The variation of the risk estimates should be noted. Within the column representing incremental carcinogenic risk for 70 year exposure (lifetime), the variation between the probable exposure conditions and the conservative exposure conditions for a given exposure frequency (daily, weekly, or monthly) are as great as an order of magnitude. It is striking that the proposed scenarios are based on PCB

concentrations which fall within the FDA guideline of 2 ppm (Maxim and Harrington 1984). Accordingly, these scenarios suggest lifetime incremental cancer risks of parts per hundred with exaggerated ingestion rates for sea food currently considered "safe" for sale in interstate commerce.

6.3. Air

The exposure to PCBs from air is considered by the RA to be a principal exposure pathway. Based upon the screening scenario, the stated fraction of average daily dose is 0.025%. For the purpose of the screening calculation, the RA assumes that all PCBs that are inhaled are also absorbed. That is, the Toxicokinetic Factor (TKF) is "assumed" to be 1.00 (100%) but no supporting basis is cited by E. C. Jordan. This assumption is wholly incorrect since particles, gases and vapors are rarely absorbed to this extent. Much lower fractions are warranted, e.g. 30% or less for deposition of respirable particles, and only after deposition occurs, can there be any chance for absorption by the lungs or GI tract. With such a consideration taken into account, a lesser contribution would be correct for PCBs entering the body via the respiratory route.

The stated values for airborne concentration of PCBs as derived by NUS do not permit one to judge what the source of such PCB concentrations might be. The RA lacks a complete citation to this data source. If the sediment were the source, the PCBs in the air might be bound to soil and dust and thus, might be less bioavailable. It is possible that these levels were monitored in the vicinity of a PCB facility while it was in operation. The use of this value as general air value for the entire New Bedford area is wholly unwarranted. It is certainly not appropriate to use such a value for lifetime exposure.

6.4. Surface Water

6.4.1. Contact with Surface Water

Dermal contact with surface water is listed by to RA as being a small contributor to the average daily dose. This component of the exposure pathway is correctly identified as constituting an insignificant factor in the dose estimate, more so if the underlying assumptions about sediment contact are correctly stated. The screening scenario given in Table A-3 lacks sufficient data for the verification of the value given in the table. The equation given in Table 2-3 does not include a set of parameters that might be employed to correctly assess exposure to PCBs by the dermal contact route. No permeability constant is given in the RA which would allow computation of this source contribution.

6.4.2. Ingestion of Surface Water

Surface water ingestion is not considered to be a principal exposure pathway as stated by the RA. The data given in the table and in equation in Table 2-3 are adequate to estimate the exposure dose from surface water. However, using data and assumptions from the RA, I was unable to verify the estimate given in the RA.

6.5. Other Exposure Considerations

The RA claims that "In-utero and neonatal exposure to PCBs are significant." Yet such an exposure route could not be quantified ("quantitatively evaluated") according to the RA. While the statement could be true if significant exposure were known to occur, the population of New Bedford has not been shown to have significant elevations of blood PCB levels. This absence of elevated blood level suggests that significant exposures are not now occurring. No basis for the RA statement about "In-utero and neonatal exposure to PCBs" is given by the authors. Those exposures which might occur among pregnant females would require contact with PCBs, most likely as sediment or from consumption of heavily contaminated (and illegally taken) biota. There is little evidence to support the assertion that human females, who may also be pregnant, will experience greater health risks to themselves from PCBs or that they will produce more severely affected off-spring if increased PCB exposure occurs. The studies of women who ate sport fish in Michigan and elsewhere are regarded as suggestive evidence of an adverse health effect of PCBs on the unborn fetus, but there appears to be little conclusive evidence. The Greater New Bedford PCB Health Effects Study (GNBHES, Massachusetts Dept. of Public Health et al. 1987), performed by the Massachusetts Department of Public Health (MDPH), suggests that increased exposure is not easily documentable in the general New Bedford Harbor population.

It is of interest to note that PCBs are thought to exert their effects following metabolism. Young children, especially, the conceptus, the fetal, and neonatal stages of development are known to have less of the enzymes necessary to activate PCBs to their toxic form. Thus, it is not clear that PCBs will, with any certainty based on metabolic information alone, have a greater toxic effect on the fetus or the newborn child versus their effect in adults. That is, an understanding of the biochemical mechanism of PCB toxicity and its proposed carcinogenic mode of action refutes the assertion of increased sensitivity of the younger person since biotransformation is required for the expression of toxicity.

7. Toxicity Assessment

The assessment of PCB toxicity is taken from its toxicity profile found in Appendix D of the RA. This profile, characterized as "TOXICOLOGICAL EVALUATIONS", is a review of the toxicologic literature on this class of compounds. Under the toxicity heading, "HUMAN", the most frequently found text statement is as follows: "Pertinent data were not located in the available literature." Such a conclusion supports the suggestion that most of the adverse effects are based on speculation from animal studies.

7.1. Dose Response Considerations

In the case of PCBs, it is acknowledged by many recognized experts that a congener specific analysis of risk is warranted. The information for such an analysis is generally lacking. However, proposals for the generation of such data have been made (Rodricks 1989). Further, the EPA has generally taken the position that negative results in PCB bioassays still contain sufficient information that supports their conclusion that a health risk exists. Thus, negative data are not able to displace positive findings even if there are compelling reasons to disallow the positive results, e.g. Norback and Weltman (Norback and Weltman 1985) used animals that were partially hepatectomized early in life. This fact alone flaws this study. Further, and unusual in this reviewer's experience, the animals were allowed to live for 29 months rather than the standard period of 24 months usually employed in such bioassays (TERRA 1989). In the case of cancer bioassays of lower chlorinated congener mixtures, the position is taken and reasserted as true by the RA, that Aroclor 1260 is the most solidly based standard or guideline for cancer risks to be estimated. The resulting estimate of risk was recently raised from that generated using the data of Kimbrough et al., 1975 (4.34 per mg/kg-day) to 7.7 per mg/kg-day¹⁰. The use of this cancer risk estimate is stated to be based on an analysis found in the RA Appendix E (Norton 1989) wherein Susan Braen Norton states:

"I discussed the results of the analysis with Jim Cogliano of the Carcinogen Assessment Statistics and Epidemiology Branch (formerly CAG). CAG's current cancer potency factor for PCBs (7.7 (mg/kg/d)¹) is based on the Norback and Weltman (1985) bioassay using rats. This bioassay has a stronger experimental design than either the 1975 Aroclor 1260 bioassay or the NCI Aroclor 1254 bioassay. Hence, the 1985

¹⁰ Others have noted that the estimates of risk have been reconsidered by a number of authors, K.S. Crump among them, and their view is that various correction factors are misused in the calculation of risk from the available bioassay data. Using the body weight correction, the value of 0.61 (mg/kg/day)¹ is given as a more proper estimate of risk.

bioassay currently provides the best estimate of cancer potency for any PCB mixture. Because, as described above, the PCB mixture in seafood does not closely resemble any commercial PCB product, there are uncertainties in using any commercial product to estimate the potency of the mixture in seafood. Since the potency factor for Aroclor 1260 of $7.7 \text{ (mg/kg/d)}^{-1}$ is the most scientifically defensible estimate of PCB potency currently available, I would recommend that it be used to assess risks from ingesting seafood from New Bedford Harbor."

The reader can see from this quote that Susan Braen Norton is poorly informed about the quality of cancer bioassay protocols. The use of this statement by the RA contractor, E.C. Jordan and Ms. Ryan, as a basis for the choice of cancer potency factor is poorly developed. It would appear that policy considerations within the EPA Regional Office warrant and require the use of this value. The other values, as noted here and elsewhere, are all lower than the chosen value and have been judged by this reviewer and others as being of better scientific quality for use in risk assessment. The issue of quality of the science in the underlying bioassay is not discussed by the authors of the RA and the use of Norton as an authority is ill advised.

7.2. Applicable or Relevant and Appropriate Requirements (ARARs)

The non-carcinogenic risks estimated by the RA are largely based on Longer Term Health Advisory values judged by the Office of Drinking Water to be safe for exposure to PCBs in drinking water sources, i.e. 0.001 mg/l for a 10 kg child and 0.0035 mg/l for a 70 kg adult. Using methods described in SPHEM, these values are converted to weight doses based on 1 and 2 liter daily rates of water ingestion. The resulting value is not an estimate of toxicity but rather an estimate of the safe dose, given the uncertainties in the assumptions. The Hazard Advisory (HA) values used for comparison with exposure estimates should not be used for the purpose of assessing risk but only for judging the degree to which an advisory level is exceeded. There is no way that the RA can purport to know when toxic effects will be manifest above the stated HA. An HA serves as a guideline and it contains a large margin of safety from a NOEL or NOAEL. Thus, it cannot and should not be described as a measure of toxicity. Certainly, the estimated exceedance of the HA cannot be used as a measure of risk per se.

8. Public Health Risk Characterization

Throughout this document, the use of the term "risk" in the probability sense, is only correct when applied to estimates of carcinogenic risk and even then, the estimates are larger than necessary, as previously discussed. In the case of non-

carcinogenic risk, the increased ratio above one is not an excess above a probable risk level but rather, the value represents the fractional excess above a stated Health Advisory (HA) or "Reference Level" (RL) level. As outlined in Section 4, these so-called "Risk Ratios" represent the ratio of the assessed exposure divided by the "Reference Level" (see Table 4-1). The applicable RL values are taken from the ARARs given within the RA.

The reader should further note that the long-term health advisory which is given in the source document applies to Aroclor 1016. The ODW criteria document (Environmental Criteria and Assessment Office (ECAO) and Office of Drinking Water 1987) states that the chronic studies of Bleavins et al (1980), Auerlich and Ringer (1977) and Barsotti and Van Miller (1984) were used to estimate the long-term HA value. All three studies provided data on the toxicity of a form of PCBs not known to be in the sediments or biota of New Bedford Harbor. Clearly, the classes of PCB congeners vary in the environment and the rationale for using a single cancer potency estimate, namely one based on Aroclor 1260, should be different or at least made more clear than the basis for using an HA value defined for Aroclor 1016. There should be a calculus that allows such parameters to be adjusted for various congeneric mixtures. This was the case for various dioxin isomers where congener or isomer specific potency estimates were assigned by a variety of regulatory bodies around the world.

8.1. Methodology

8.1.1. Estimating Non-carcinogenic Risk

There is no calculation in the RA which accurately estimates this parameter. The values given in the RA for non-carcinogenic risk are the ratio of the dose, above or below a standard of one kind or another, e.g. usually an RfD or long-term/chronic health advisory or other guideline value. As cited in the RA,

" . . . The risk ratio best reflects the potential noncarcinogenic risk when comparisons are made to standards or criteria that are based on the same exposure assumptions as the exposure dose. For example, acute exposure doses should be compared to 1- or 10-day health-based criteria and chronic exposure doses to longer-term criteria. However, for many contaminants in this risk assessment, the only criteria available to evaluate noncarcinogenic risks were those based on lifetime exposure. RfDs and MCLGs are criteria that define an acceptable daily exposure of a contaminant, assuming a 70-year exposure duration. Therefore, comparing an average daily dose derived for a chronic (10-year) or acute exposure to the RfD or MCLG may overestimate the actual risk. In such instances, the significance of the risk ratio value requires further evaluation. For

this report, the toxicity endpoints and the magnitude of the uncertainty associated with the criteria development were considered in evaluating these potential risks."

Notwithstanding the statement above, it is clear that the use of an RfD, a corresponding long-term HA or any similar standard based on safe exposure does not measure risk, merely ratio of safe dose to proposed (estimated) dose.

8.1.2. Estimating Carcinogenic Risk

Estimates of cancer risk are usually done by the mathematical product of dose times risk per unit dose. The EPA uses the q_1' as the basis for this conversion, namely $7.7 \text{ (mg/kg/day)}^{-1}$. Thus, a dose of 1 mg/kg/day is presumed, from this potency value, to pose a risk of 7.7 cancers per million population exposed for a 70 year lifetime. Correspondingly lower doses and shorter times of exposure are presumed to have mathematically related reductions in the risk estimate applied to them, e.g., a 10 year exposure would pose one-seventh the risk. There is no policy which states that risks incurred early in life are greater than similar risks posed later in life. Thus, such estimates are usually regarded as "conservative" first approximations that are not likely to underestimate the true risk. It is assumed that cancer potency among agents with similar mechanisms of carcinogenesis can be added to accumulate a total risk. While this may be true, in the case of the PCBs in New Bedford Harbor, they are the only agents which pose an assumed carcinogenic risk for which the underlying data is sufficient to perform a quantitative risk assessment.

8.1.3. Estimating Multi-toxic risk

The RA states that all risks assessed in the document are assumed to be additive. This is consonant with EPA policy as well as the guidance developed by the National Academy of Sciences (Board On Environmental Studies and Toxicology 1988) among others (US Environmental Protection Agency (EPA) 1986, Paustenbach 1989). As noted above, the only putative carcinogen in New Bedford Harbor that can be "quantitatively assessed" are the PCBs. Data regarding lead and cadmium, while being suspected of posing a carcinogenic risk, are not sufficiently well developed as to be quantified.

8.1.3.1. Non-carcinogens

The value calculated for the various substances in the Harbor are assumed, based on the RA, to have similar toxic end-points and reference is made in the RA to Appendix D, the toxicity profiles. In referring to the toxicity profiles, I find little basis to conclude that these agents have similar end points.

8.2. Quantitative Risk Estimation

8.2.1. Non-Carcinogenic Risk

8.2.1.1. Sediment

Two locations are given by the RA as being likely for exposure of children to sediment via the ingestion scenario. Within Area I and applying the conservative assumptions (see Table 4-3), the cove area (RR = 11), the Upper Estuary (RR=175) and the area wide estimate (RR = 175) are all deemed to pose or otherwise present a possibility of excess exposure risk. Only lead (versus copper and cadmium) rises to the level of significance above an RR of 1. As regards older children and adults, the maximum concentration scenario given in Table 4-4 suggests that excess exposure to PCBs (RR = 8) occurs in older children and adults (RR = 4.6) when an Area I maximum concentration scenario is estimated. The mean concentration values for RR are 2, 0.47 and 0.28 for child, older child and adult, respectively.

8.2.1.2. Biota

For the ingestion of biota, the RA postulates a daily, weekly and monthly ingestion scenario. In area I, there were few lobsters captured and thus, the RA claims that clams and flounder are the principal PCB biota sources. On a once weekly basis, the scenario suggests that clams and flounder, taken from area I, when ingested, will result in RR values greater than 1 in all cases (younger child, older child and adult). As postulated by the RA, daily ingestion leads to proportionately greater RR values. With the conservative scenario applied, the RR values increase by a factor of three greater than the probable scenario (weekly) and even more for the daily scenario. However, the likelihood of all fish being consumed from catches in area I is highly unlikely for a Harbor that is as commercially and industrially developed as this one is and thus, daily and weekly meals are more likely to arise from areas that are harvested more easily and have lesser degrees of contamination. Fishing in such areas, either by commercial fisherman or by "sport" enthusiasts who are putting food on their table, will be more likely than in poor fishing areas such as the New Bedford inner harbor.

Children of females with a history of fish consumption during pregnancy, where PCBs were found to be elevated in blood samples taken at birth, were studied. These youngsters had elevated blood levels of PCBs that were related more to nursing history than to any other factor. Most important, the children were found to eat less than one fish meal per year (Jacobson et al. 1989) and thus, fish consumption was not a significant factor in this population. Whether this is

because children eat little fish as a rule as my experience suggests or whether their mothers chose not to give them fish is not known. In any case, this study with its suggestion of low fish consumption by children weakens the daily, weekly and monthly single fish ingestion scenarios presented by the RA. Among Finnish women who were found to have elevated levels of PCBs in their breast milk, the levels in their milk was reported not to be correlated with the history of fish ingestion. (Mussalo-Rauhamaa et al. 1988).

The issue of exaggerated fish consumption patterns by adults and children, the specific fish availability with its degree of contamination and the calculation from this set of assumptions that there now exists an increased risk of cancer as a result leads the RA to conclude that a substantial cancer risk does exist from this exposure pathway. However, in the opinion of this reviewer, the evidence accumulated to date on this subject is far from conclusive.

8.2.1.3. Air

No meaningful estimates were given in the RA for the non-carcinogenic RR values. It appears that few, if any, applicable standards (ARARs) exist for this exposure route.

8.2.2. Carcinogenic Risk Evaluation

8.2.2.1. Sediment

8.2.2.1.1. Ingestion

Based on the estimates of dose given in the screening scenario and the equations used in the spread sheet calculation, the ingestion of sediment by children has been calculated in the RA to lead to the degree of cancer risk as given in the various tables attached to the RA. The estimated dose times the cancer potency factor leads to an estimate of risk as found in Tables C-24 and C-25 among others. As noted previously, this route is given as 14.7% of total average daily uptake and is applied only to the data for area III. The amount of uptake is overstated as is the opportunity for exposure.

Among the spread sheets provided by the EPA and E. C. Jordan, table C-101 (which does not appear as a separate table in the RA) purports to correctly calculate the risk from daily ingestion of sediment by a child. This table includes a "most probable" and "realistic worst" case scenario. Both of these are wrong and over stated. The area considered is the cove area and the values chosen for the combined estimate, namely sediment ingestion and contact, are likely to overestimate risk by a factor in excess of 25,000.

8.2.2.1.2. Contact

Contact with sediments is stated by the RA to be the most important risk factor. According to the RA in table 2-2 (Page 2-15), the fraction of daily dose due to this route, namely 84%, occupies tables C-1 through C-18. The reviewer notes that the various age classes are estimated as follows: adult exposure is projected to occur over 55 years while the young child (0-5 years of age) is exposed for a portion of 5 years and the older child may be exposed during the ages of 6 to 16 (10 years). Additional tables detailing the risks due to sediment contact appear in C-58. As noted above, the 7% uptake factor for PCBs is greater than the EPA estimate used in the Woburn Wells G & H analysis, namely 2%.

As given above for ingestion, the choice of parameters in table C-101 (spreadsheet provided but not included in the RA) leads to a net overestimate of risk for ingestion and contact by 25,000 fold.

8.2.2.2. Biota

Biota exposure is estimated to occur with a variety of scenarios for the variety of fish said to be eaten locally. These species, namely winter flounder, lobster and clams are postulated as being in the diet on a daily, weekly or monthly basis in table C-26 through C-57. The daily scenario and the amounts given are excessive for the national average and may be above average even for the NBH area.

The values given in table C-52 utilize lobster concentrations of 0.213 ug/g (most probable) and 0.351 ug/g (worst case). Clearly, such concentrations do not exceed the FDA standard of 2 ppm (2 ug/g). Such meals are not likely to be available on a daily basis for either older children or adults. Certainly, such exposures would not be available for the 70 year life span required by the risk estimation equation.

8.2.2.3. Air

The estimates of inhalation related risk for PCBs are given in tables C-19 through C-21. In the case of table C-21, the realistic worst case is described for an adult living in "all areas" of New Bedford. The estimated chronic body dose is given as 1.3×10^{-4} mg/kg/day (1.3E-4 as given in the RA) using a potency estimate of 7.7 (mg/kg/day)¹. The resulting estimated risk which must be assumed to be lifetime risk is given as 1.48×10^{-4} .

It is not possible that adults will experience daily, 24 hour per day exposures, of the magnitude listed throughout all areas of New Bedford Harbor. A large

fraction of time will be spent at the lower background level and most adults are unlikely to live within such exposure areas for their entire lifetime.

9. Conclusions

The RA draft document purports to estimate risks and assess hazards of multiple agents. The agents in question act at different sites, have different mechanisms of action and lack the underlying similarity of toxicity/mechanism for the contractor to be able to legitimately apply their aggregate sum to the task at hand, namely, the determination of "multi-toxic risk." In the case of carcinogenesis, the RA estimates doses in ways that should be correct and verifiable. They clearly are not. The RA estimates risks based on EPA guidance which is different for different locations and is generally overstated. That is, a number of bioassays are available but only the largest potency estimate has been used. This practice is usually justified by the need to protect public health and never to underestimate potential risk. Further, while such choices might be acceptable if this were the best quality study for the purpose, this was not the case in this document. Finally, the RA concludes that a range of risks exists in the New Bedford Harbor area from moderately acceptable (greater than a risk of 1 in 100,000) to extremely large (on the order of 1 in 10,000 or less) and therefore, unacceptable.

The frequency that a standard/guideline is exceeded is called a risk ratio when the true meaning of this proportion is best interpreted as an excess above a safe or acceptable standard level. The true meaning of the "Risk Ratio" is lost in the document and based upon this review, it is my judgement that the authors have little basis to conclude just how great, if any, a non-carcinogenic risk exists from the conditions found in the New Bedford Harbor.

A risk assessment contains the following basic premise: where toxic or otherwise hazardous chemicals are known to exist in the environment, they may pose a definable human health risk, that risk can be accurately quantified and the resulting estimate can be used to determine the magnitude of a remediation effort. The GNBHES found little evidence of excessive exposure to PCBs (as evidenced by elevated PCB blood levels) and the population appeared no different from other US populations with much less likelihood of PCB exposure. It appears that the "risk" tabulations in the RA are an effort to justify the existence of a risk, not an effort to correctly and properly determine if such a risk now exists.

There exists little scientific data which allows the true estimate of environmental PCB mixture-based health risk. Data for Aroclor 1016 (HA basis) and Aroclor 1260 (Cancer Risk) are freely interchanged in the RA and each is characterized

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as representing "Risk." Assumptions about Aroclor 1254 and 1242, which were negative or minimally supportive in chronic bioassays, are been cast in a "supportive" role. This appears to be EPA policy. Mixture information is lumped into broad classes and the EPA Regional office acknowledges that biota PCB levels are orders of magnitude removed from the chosen surrogate. Other risk assessments in neighboring areas used lower potency estimates, e.g. Quincy Harbor where an Aroclor 1254 basis was chosen. The entire supporting basis for the present cancer risk estimate is summed up by the EPA itself when the spokesperson for the EPA Exposure Assessment Group concludes:

" . . .the 1985 bioassay currently provides the best estimate of cancer potency for any PCB mixture. Because, as described above, the PCB mixture in seafood does not closely resemble any commercial PCB product, there are uncertainties in using any commercial product to estimate the potency of the mixture in seafood. Since the potency factor for Aroclor 1260 of 7.7 (mg/kg/d)¹ is the most scientifically defensible estimate of PCB potency currently available, I would recommend that it be used to assess risks from ingesting seafood from New Bedford Harbor."

This type of data analysis, notwithstanding the many tables provided in the RA and the elaborate mathematical formulae applied, does not, in my opinion, form an adequate basis for the degree and extent of a multi-million dollar environmental cleanup. Much of the draft RA is an attempt to justify risk management, rather than an objective listing of postulated risks which can be mathematically verified and are likely to be correct. Certainly, the 1985 PCB bioassay of Norback and Weltman provides only one point of reference (and a scientifically weak one at that) that might be applied to estimate environmental risk from this particular PCB formulation. The absence of a reasonable assessment of other "competing" risks or mixtures with diminished potency based on congener specification causes the present assessment to be flawed and incomplete.

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TABLE 1

POPULATION DISTRIBUTION IN THE NEW BEDFORD HARBOR AREA

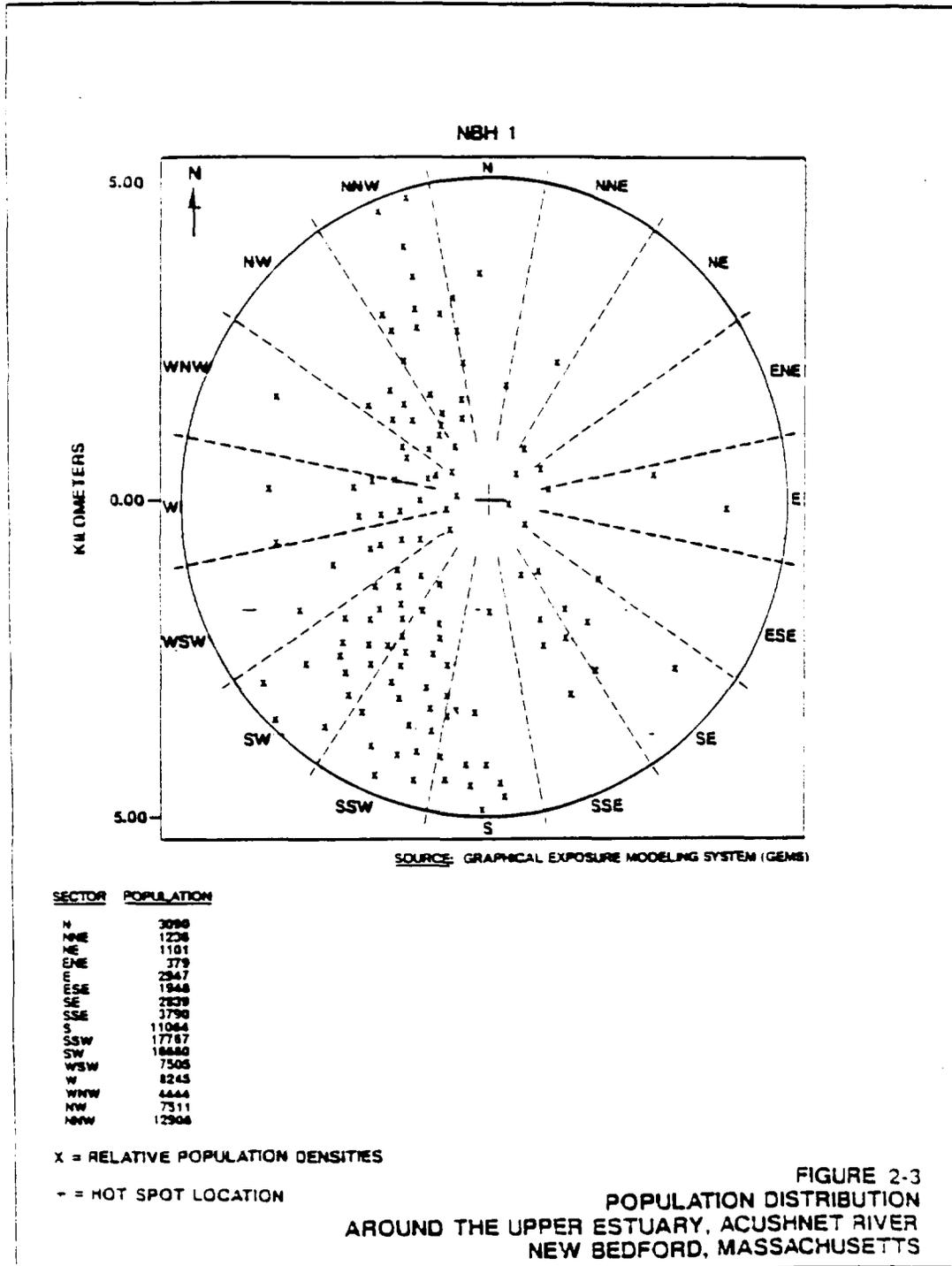
Age (Years)	New Bedford	Correct Value	Acushnet	Correct Value	Dartmouth	Correct Value	Fairhaven	Correct Value	Correct Total	Incorrect Total
<u>Males</u>										
0-5	4268		264		657		466		5655	
6-16	8007		873		2256		1412		12548	
17-44	17452		1708		4832		2985		26977	
45-64	10257		1014		2644		1793		15708	
>65+	5889		401		1237		937		8464	
<u>Females</u>										
0-5	3941		288		573		429		5231	
6-16	7959		780		2087		1303		12129	
17-44	18782		1722		5495		2942		28941	
45-64	12181		1075		2911		1966		18133	
>65+	10007		579		1781		1526		13893	
<u>Totals</u>										
0-5	8209	8209	552	552	1230	1230	895	895	10886	
6-16	15966	15966	1653	1653	4343	4343	2715	2715	24677	
17-44	36239	36234	3340	3430	8929	10327	5927	5927	55918	54435
45-64	22438	22438	2089	2089	5555	5555	3759	3759	33841	
>65+	15896	15896	980	980	3018	3018	2463	2463	22357	
POPULATION TOTAL		98743		8704		24473		15759	147679	

TABLE 2

Population Distribution

COMPASS SECTOR	POPULATION	PERCENTAGE	FRACTION	QUADRANT FRACTION
N	3090	3.1070%		
NNE	1236	1.2428%	4.3498%	
NE	1101	1.1071%		
ENE	379	0.3811%	1.4882%	5.8380%
E	2947	2.9632%		
ESE	1948	1.9587%	4.9220%	
SE	2839	2.8546%		
SSE	3790	3.8109%	6.6655%	11.5875%
S	11064	11.1250%		
SSW	17767	17.8649%	28.9899%	
SW	16680	16.7719%		
WSW	7505	7.5464%	24.3183%	53.3081%
W	8245	8.2904%		
WNW	444	0.4464%	8.7369%	
NW	7511	7.5524%		
NNW	12906	12.9771%	20.5295%	29.2664%
TOTALS	99452	100.0000%	100.0000%	100.0000%

FIGURE 1



1988-08

APPENDIX 1

CURRICULUM VITAE

RUDOLPH JOHN JAEGER

Principal Scientist
and President

Environmental Medicine, Inc.

PERSONAL DATA

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Present Academic Position:	Research Professor
Appointed:	1983
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Message Telephone:	(914) 351-2300
Date and Place of Birth:	January 17, 1944 Weehawken, New Jersey

Education

High School: Stevens Academy,
Hoboken, New Jersey
Diploma, 1962

Undergraduate
Education: Rensselaer Polytechnic Institute,
Troy, New York,
Biology, B.S. 1966

Fairleigh Dickinson University
Teaneck, New Jersey
Non-degree, Clinical Psychology, 1963, 1964

Graduate
Training: Johns Hopkins University, School of Hygiene
and Public Health, Baltimore, Maryland
Ph.D., Biochemical Toxicology, 1971

Post-Doctoral
Training None Required

Post-doctoral
Courses: Harvard University,
School of Public Health, Boston, Massachusetts
Auditor, Respiratory Physiology, 1978

Tufts University
Asbestos Information Center
Asbestos Inspector/ Asbestos Management Plan
January 11-15, 1988

PROFESSIONAL AFFILIATIONS

Society of Toxicology

Society of Toxicology of Canada

American Academy of Clinical Toxicology

American Industrial Hygiene Association

American Conference of
Governmental Industrial Hygienists

American Chemical Society

American Association of Pathologists

American Association for the
Advancement of Science

Sigma Xi (Harvard - Radcliffe Chapter)

Middle-Atlantic Section,
Society of Toxicology

Toxicology Forum
(Individual Member)

Inhalation Toxicology Specialty Section,
Society of Toxicology

Society for Risk Analysis

American Society for Testing and Materials

HONORS

Research Career Development Award,
National Institute of Environmental
Health Sciences, NIH. DHHS
1978-1983

Leslie Silverman Award, New England
Section, American Industrial Hygiene
Association, 1980

EDITORIAL SERVICE

Editorial Boards: Journal of Toxicology and Environmental Health
Journal of the American College of Toxicology
Boston Bulletin on Chemicals and Disease
NYU Medical Center Health Letter

LICENSURE AND CERTIFICATION

Certification: Diplomate, American Board of Toxicology, 1980
Recertified by Re-examination, 1985 1990

Certified/Registered Environmental Assessor (CEA)
State of California

Type III Asbestos Consultant
State of Vermont

Board Eligible: Professional Standards Review,
American Academy of Toxicological Sciences

American Board of Industrial Hygiene
Toxicologic Aspects of Industrial Hygiene

Accreditation: Asbestos Inspector/ Asbestos Management Planner,
Asbestos in Schools, AHERA

TEACHING EXPERIENCE

From 1972 through 1979, Rudolph J. Jaeger was a member of the staff and faculty of the Harvard University School of Public Health, holding the rank of Research Associate in Toxicology, Assistant Professor of Toxicology and Associate Professor of Toxicology. Since 1979, Professor Jaeger has been at New York University where he is currently Research Professor of Environmental Medicine at New York University Medical School. He continues to teach at Harvard University where he is Visiting Lecturer on Industrial Toxicology and Environmental Medicine in the Department of Environmental Health Sciences and Physiology in the School of Public Health. He has been invited to lecture at Clark University in Worcester, MA, the University of Rhode Island in Kingston, RI, the John B. Pierce Foundation of Yale University in New Haven, CT, Hunter College in Manhattan, Ramapo College in New Jersey, the New York State Division of Laboratories in Albany, NY and the AT&T Bell Laboratories in Murray Hill, NJ and Lisle, IL. He has spoken before numerous professional societies (regional sections of the AIHA, ACS, Radiological Physics Society, etc.) and the US Department of Justice, Assistant Attorneys-General, training meeting on Superfund and toxic tort litigation. A more specific listing of Dr. Jaeger's experience is given in the following sections.

ACADEMIC, INDUSTRIAL, GOVERNMENTAL APPOINTMENTS
AND CONSULTATIVE EXPERIENCE

1971-1973	Research Associate In Toxicology	Harvard School of Public Health
1972-1975	Analytic Consultant	Children's Hospital, Boston Blood Preservation Laboratory
1972-1979	Consultant and Lecturer on Industrial Toxicology	Boston Poison Control Information Center
1973-1978	Assistant Professor of Toxicology	Harvard School of Public Health
1974-1977	Instructor and Course Co-ordinator, Toxicology and Product Safety Evaluation	Northeastern University
1974-1975	Analytic Consultant	General Tire and Rubber Company, Akron, Ohio
1975-1979	Reviewer, Ad Hoc Grants Review Committee	Division of Advanced Environmental Research and Technology National Science Foundation Washington, D.C.
1975-1979	Consulting Toxicologist Cancer Bioassay Reviewer	Energy Resources Company Cambridge, Mass.
1975-1979	Chairman, Medical Area Subcommittee on Laboratory Safety	Harvard Medical School
1975-1978	Member, Subcommittee on Doctoral Program and Committee on Admissions and Degrees	Harvard School of Public Health
1976	Expert Witness - Toxicology of Kepone	Office of Regional Solicitor, U.S. Department of Labor, Philadelphia, PA
1976-1977	Consultant to the Plaintiff-Toxicology of Kepone	Hundley, Taylor, and Glass, Richmond, VA
1976	Review Consultant. Criteria Document on PCBs	NIOSH

1976	Lecturer of Industrial Toxicology	G.E. Short Course. Harvard School of Public Health
1977-	Consulting Toxicologist. Chemical Health Committee	Polaroid Corporation Cambridge, Mass.
1977	Instructor, Post Graduate Course, Toxicologic Evaluation of Industrial Chemicals by Rapid, in vitro methods.	American Occupational Medical Association
1977	Review Consultant, Criteria Document of Vinyl Monomers	National Institutes of Occupational Safety and Health
1977-1978	Panel Member. Subcommittee on Flammability of Di-electrics	National Academy of Engineering, NAS-NRC
1977-1978	Member. Microbiologic and Oncologic Biohazards Committee	Harvard Medical School and Harvard School of Public Health
1978	Expert Witness, Styrene	U.S. Department of Labor OSHA
1978	Expert Witness and Toxicology Consultant on Dimethyl Amino-propionitrile (NIAX ESN)	U.S. Department of Labor OSHA
1978	Consultant on Toxicology and Industrial Hygiene of Phosphine	Boston Poison Control Center and United States Coast Guard
1978-1979	Associate Professor of Toxicology	Harvard School of Public Health
1979-1983	Associate Professor of Environmental Medicine	New York University Medical School
1978-1979	Lecturer on Toxicology, ATT Medical Director's course on Industrial Hygiene and Toxicology	Bell System Technical Education Center Lisle, Il.
1979	Criteria Reviewer and Committee Member, Water Quality Standards Division	Environmental Protection Agency, Cincinnati, Ohio
1979	Lecturer on Toxicology	General Electric Co. Schenectady, New York
1979-	Visiting Lecturer on Environmental and Industrial Toxicology	Harvard School of Public Health

1979	Consultant on Curriculum and Career Opportunities in Toxicology	Northeastern University Department of Pharmacology School of Pharmacy and Allied Health Professions Boston, Massachusetts
1980 - 1984	Criteria Document Review Vinylidene Chloride Acrylonitrile Trichlorethylene Perchloroethylene	Environmental Protection Agency, Washington, D.C.
1980-1981	Quality Assurance Review Panel, Document Review	Clement Associates Washington, D.C.
1980	Consultant	Occupational Health Services Inc., Cambridge, MA
1980	Consultant	Metropolitan Life Insurance Co., New York, NY
1981	Lecturer	Bell Laboratories, Murray Hill, NJ
1981	Review Consultant, Human Reproductive Toxicity, Council on Environmental Quality	Clement Associates Washington, DC and CEQ
1982	Councilor	Middle Atlantic SOT
1982	Invited Lecture, Proposed Inhalation Toxicity Testing Guidelines	Toxicology Forum, Aspen, CO
1982	Expert and Rapportuer, Criteria Document Task Group, Styrene Health Effects	World Health Organization International Program on Chemical Safety, Helsinki
1983	Councilor, Member of Program Committee	Inhalation Toxicology Specialty Section
1983 - date	Research Professor	Institute of Environmental Medicine, New York University Medical Center. New York, NY
1983	Invited Speaker	Institute of Occupational Health, Helsinki, Finland

1983	Invited Speaker	United Rubber Workers Akron, OH
1983	Consultant	American Cyanamid Co. Wayne, NJ
1983 - 1985	Consultant	AT&T-Bell Laboratories Murray Hill, NJ
1983 - 1984	Associate Scientist	Clement Associates Arlington, VA
1983	Consultant to Defendant and Expert Witness on Perchloroethylene	Goodwin, Procter and Hoar Boston, MA Inter-state Uniform Co. Woburn, MA
1983	Consultant and Expert Witness Structure Activity Relationships	Life Systems Inc. Cleveland, OH Office of Toxic Substances, USEPA Washington, DC
1983 - 1984	Consultant	Patterson, Bellknap Webb and Tyler New York, NY
1983	Consultant and Expert	Hale and Dorr Boston, MA
1983	Consultant	Mueller's Macaroni Co. Jersey City, NJ
1983 - 1984	Consultant to Plaintiff Expert Witness Metals and Organics	Attorney-General State of New York
1984 - 1986	Consultant and Expert Creosote, Pentachloro- phenol and Dioxins	Southern California Edison Co., Rosemead, CA and Visalia, CA
1984	Consultant	Karch and Associates Washington, DC
1984 - 1985	Consultant To Plaintiff Expert Witness Superfund US v. Hooker Chemical Co.	US EPA and US Dept. of Justice Washington, DC Niagara Falls, NY
1984	Consultant Safety Evaluation Consumer Product	Plymouth Rubber Co. Canton, MA

1984	Invited Speaker Combustion Toxicology of Plastics	Second Annual Fire Engineering Conference Manhattan College, Fire Engineering Institute Riverdale, NY
1984	Consulting Toxicologist Ground Water and Air Toxics Programs - Risk Assessment	Environmental Research and Technology Concord, MA
1984	Consulting Toxicologist Ground Water	Combustion Engineering Windsor, Ct.
1984	Document Reviewer Health Effects of PCBs and Dioxins	Electric Power Res. Inst. Palo Alto, CA
1985	Consultant	Albert Orgain, IV Esq. Richmond, VA
1985-89	Consulting Toxicologist Consumer Labeling Art Supplies and Inks	Esselte Letraset Moonachie, NJ
1985	Consulting Toxicologist	New York Telephone Co. New York, NY
1985	Consulting Toxicologist	Spengler Environmental Consultants
1985	Lecturer on Industrial Toxicology	Organic Chemistry R&D AT&T Bell Labs, Murray Hill, NJ
1985	Consulting Toxicologist Air Toxics Regulations	Env. Res. Technol and Connecticut Business and Industry Alliance, Hartford, CT
1985	Product Registration, PMN and Process Importation- Toxicology	Gallafent & Co. London, England
1985	Review - Biochemical Toxicology & Effects Ranking	US EPA Environmental Criteria and Assessment Office, Cincinnati, OH
1985	Food Safety Review Smoked Meats	William Casagrande Casa Market Bergenfield, NJ

1985	Review - Health Effects of Municipal Solid Waste Incineration	Penobscot Energy Resource Company, Orrington, Maine Cambridge Analytical, Boston, MA
1985	Health and Safety - Galvanized iron pipes for drinking water	The Wadleigh Law firm Manchester, NH
1986	Wood Preservation Chemicals and Soil Contamination	Gardere and Wynne, Dallas, TX
1986	Toxicology Consultant	Townley and Updike
1986	Plaintiff's Expert	Timothy Ellis, Esq. Brown, Terrell, Hogan and Ellis, Jacksonville, FL
1986	Document preparation for Science Advisory Board on perchloroethylene	Goodwin, Proctor, & Hoar Boston, Mass. Unifirst Corp.
1986	Development of Health and Safety Plan for Waste disposal site	Remediation Technology, Inc. Pittsburgh, Pa.
1986	Toxicology Consultant	Womble, Carlyle, Sandridge & Rice Winston-Salem, NC
1986	Document Service	Jones, Day, Reavis, & Pogue Washington, DC
1986	Consultant: product labeling process importation	Esselte Letraset UK London, England
1986-89	Toxicology Consultant	Ropes and Gray Boston, MA
1986	Toxicology Consultant	Union Carbide Corp Law Department Danbury, CT
1986-89	Toxicology Consultant	Goodwin, Procter and Hoar Boston, MA
1986	Toxicology Consultant	Lord & Taylor, NY
1986	Toxicology Consultant	IBM Corporation Armonk, NY
1986	Indoor Air Pollution Health Risk assessment	Ramapo College, Mahwah, NJ

1986-87	Air Pollution and Hazardous Waste Risk Assessment	So. Calif. Edison Co. Venice, CA
1986	Toxicology Consultant	Greenberg, Dauber and Epstein Newark, NJ
1986	Document Review NJ Water Quality	Hackensack Water Co. Harrington Park, NJ
1987	Toxicology Consultant	Shaheen, Cappiello, Stein & Gordon Concord, NH
1987	Toxicology Consultant Special Advisory Committee Methyl Chloroform	State of Michigan Department of Natural Resources Lansing, MI
1987	Toxicology Consultant	Monsanto Agricultural Company St. Louis, Mo.
1987	Toxicology Consultant	Walton, Lantaff, Schroeder & Carson, Miami, Fla.
1987	Toxicology Consultant	Akin, Gump, Strauss, Hauer & Feld, Washington, DC
1987	Toxicology Consultant	Williams & Connolly Washington, DC
1987	Toxicology Consultant	AMAX Mineral Resources, Inc. Golden, Colorado
1987	Toxicology Consultant	Griffith & Burr Philadelphia, Pennsylvania
1987-89	Toxicology Consultant Product Registration	Hyperion Catalysis International, Boston, Ma.
1987	Toxicology Consultant	Ryan, Ryan, & Hickey Stamford, CT
1987	Toxicology Consultant Asbestos Hazard Evaluation	Fort Lee Board of Education Fort Lee, New Jersey
1987	Hazard Communication Training Programs	Bell Communications Research Piscataway, New Jersey
1988-89	Toxicology Consultant Asbestos Hazard Evaluation	Palisades Park Board of Education, Palisades Park, NJ
1988-89	Toxicology Consultant Asbestos Hazard Evaluation	Ridgewood Board of Education, Ridgewood, NY

1988	Toxicology Consultant	McGuire Woods Battle & Boothe Richmond, Va.
1988	Toxicology Consultant	Kehoe, Doyle, Playter & Novick Boston, Ma.
1988-89	Toxicology Consultant	Sidely & Austin Manhattan, New York
1988	Toxicology Consultant	Griffin, Rainwater, & Draper Crossett, Arkansas
1988	Toxicology Consultant	Gubman Sitomer Goldstein & Edlitz, Manhattan, New York
1988	Toxicology Consultant	Schoeman, Marsh, Updike, & Welt, Manhattan, New York
1988	Toxicology Consultant	Arter & Hadden Cleveland, Ohio
1988-89	Toxicology Consultant	Smith, Helms, Mulliss, & Moore, Greensboro, NC
1988	Toxicology Consultant	Holme Roberts & Owen Denver, Co.
1989	Toxicology Consultant	Lockheed Aeronautical Systems Co., Burbank, Ca.
1989	Toxicology Consultant	Electro Signal Laboratory Parsippany, NJ
1989	Toxicology Consultant	Diamond & Associates Montpelier, Vt.
1989	Toxicology Consultant	Scanlon, Howley, Scanlon, & Doherty, Scranton, Pa.

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