Comments of General Electric Company on the Human Health Risk Assessment for the General Electric/ Housatonic River Site, Rest of River

(February 2005 Draft)

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April 2005

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1.0 INTRODUCTION

The General Electric Company (GE) is providing these Comments to the U.S. Environmental Protection Agency (EPA) on the February 2005 draft of EPA's *Human Health Risk Assessment for the General Electric/Housatonic River Site, Rest of River* (HHRA) (EPA, 2005). These Comments were prepared on GE's behalf by AMEC Earth & Environmental and BBL Sciences.

The revised HHRA contains a substantial amount of new information and analyses that were not presented in, or have been changed from, the June 2003 draft of the HHRA (EPA, 2003). Some of these additions and changes were intended to address comments made by the peer reviewers on the prior draft, while others were made by EPA on its own initiative. These Comments focus only on such new or changed information and analyses. Moreover, these Comments address only some of the new or changed material in this revised draft. However, GE adheres to and preserves its positions on all points set forth in GE's prior comments (AMEC and BBL, 2003; GE, 2003) on the June 2003 draft HHRA, and reserves the right to raise those points in any future proceeding. In addition, lack of comment herein on other new material or analyses in the HHRA does not necessarily indicate GE's agreement with such material and analyses; GE reserves the right to present any arguments relating to such material and analyses in an appropriate future proceeding.

These Comments contain the following sections:

- Section 2 discusses certain general points in the revised HHRA – namely: (a) its estimation of population sizes for potentially exposed populations; (b) its discussion of the cancer incidence study conducted by the Agency for Toxic Substances and Disease Registry and the Massachusetts Department of Public Health (ATSDR/MDPH); and (c) its application of the Toxicity Equivalency (TEQ) approach to PCBs.
- Section 3 addresses the direct contact assessment. It shows that a number of the changes that EPA has made to the exposure frequencies in the deterministic analyses are not consistent with site characteristics or the available data. It also shows that the new probabilistic analyses added by EPA contain an unwarranted degree of conservatism in some of the distributions used and that its probabilistic model may contain an error in the calculation of a surface area-weighted adherence factor.

- Section 4 addresses the fish and waterfowl consumption assessment, focusing on problems with the new adult and child fish consumption rates used in the deterministic analysis and the child consumption rates used in the probabilistic analysis.
- Section 5 addresses the agricultural products consumption assessment. It notes the speculative nature of some of the HHRA's assertions regarding future agricultural use of the floodplain. It also shows that several of the revised factors used in the deterministic analyses are not justified, and that the probabilistic analyses fail to adequately account for the variability and uncertainties in the risk estimates.
- Finally, Section 6 discusses two points in EPA's new integrated risk characterization chapter – the perspective on TEQ exposures and the evaluation of the breast milk pathway. It shows that the revised HHRA does not adequately address the uncertainties in these evaluations.

In each of these sections, GE presents recommendations for further changes to the final HHRA to make it more scientifically supportable.

2.0 GENERAL

This section addresses three general points relating to the revised HHRA. First, it shows that, in estimating population sizes for potentially exposed populations, the HHRA presents some estimates that are not representative of the size of the user population being evaluated in the risk assessment. Second, it shows that the HHRA needs to clarify its discussion of the cancer incidence study that was conducted by the ATSDR/MDPH (2002). Third, it shows that the revised HHRA has not fully or adequately addressed the problems with the application of the TEQ approach to PCBs.

2.1 Population Estimates

In response to comments received from the peer reviewers, EPA has added an estimation of population sizes for each potentially exposed population. In some cases, however, the general population estimates presented in the revised HHRA do not reflect the size of the user populations being evaluated in the risk assessment. For example, the HHRA estimates that the population of recreational anglers using the Housatonic River is 11,371 individuals (Vol. I, p. 1-33). While that may be a reasonable estimate of the size of the total user population, it is not the size of the subpopulation of anglers (non-sharing consumers) for whom exposure and risk

have been estimated, and in particular is not the size of the subpopulation represented by EPA's Reasonable Maximum Exposure (RME) fish consumption scenario. Thus, it is misleading to present that estimate as the size of the population that may be subject to the levels of potential exposure and risk that are calculated in the HHRA.

The HHRA bases its assumed fish consumption rates for both the RME and central tendency (CTE) risk estimates on data from the Maine angler survey (Ebert et al., 1993) for the fraction of anglers who consume sport-caught fish but do not share their fish with anyone (Vol. IV, p. 4-48). However, the data from that survey indicate a number of differences in fishing and fish consumption behavior between that subpopulation and the total population of Maine anglers. For example, the average amount of fish harvested per fishing trip was 266 grams/day for non-sharing consumers and 413 grams/day for all other fish consumers. Moreover, the 95th percentile "all waters" consumption rate derived by EPA for non-sharing consumers (31 g/day) is higher than that for all fish consumers (26 g/day). Consequently, the assumptions used to estimate potential risks due to fish consumption can only be considered representative of the behaviors of a small subpopulation of anglers who use the river (i.e., non-sharing consumers).

According to the Maine angler survey, 138 individuals who consumed fish during the one-year survey period did not share their fish with anyone else. These represented 8.56 percent of the total survey population of 1,612 individuals. Applying this percentage to EPA's total estimated user population of 11,371 anglers results in a total population of non-sharing consumers of 973 individuals. Since the CTE risk estimates can be considered representative of the average for the total population, the population size for the CTE risk analysis is estimated to be 973 individuals. However, the RME analysis uses the 95th percentile consumption rate for this subpopulation (Vol. IV, p. 4-48) and thus is representative of approximately 0.43 percent of the total user population for the river (8.56 percent times 5 percent). Applying this percentage to the total of population of 11,371 anglers estimated by EPA results in an estimated RME population size of 49 individuals.

The size of the potentially affected population would be larger, but still considerably smaller than EPA's estimate, even if EPA were correct that the non-sharing consumers were representative of the entire population of fish consumers in the Ebert et al. (1993) survey. In that survey, 1,053 of the 1,612 individuals who responded to the survey indicated that they consumed sport-caught fish from any source during the one-year survey period. Thus, 65 percent of the licensed anglers surveyed actually consumed fish during the year. Applying this fraction to EPA's

estimate of 11,371 individuals in the entire Berkshire County angler population results in an estimated population size of 7,391 individuals. It can be considered that the CTE estimate predicted by EPA might be representative of this population. The RME analysis, however, selects the 95th percentile consumption rate and thus is only representative of 5 percent of the consumer population. This results in an estimated population size for the RME analysis of 370 individuals.

Similarly, EPA estimates that there is a population of 3,600 individuals who live in Berkshire County and hunt waterfowl from the primary study area (PSA) (Vol. I, p. 1-35). While GE previously recommended that EPA include a factor to adjust for the fraction of ducks harvested that are non-resident birds (AMEC and BBL, 2003), EPA is instead basing its waterfowl risk estimates only on consumption of resident birds and the PCB concentrations measured in them. To estimate potential exposures to waterfowl consumers, the HHRA assumes that the CTE waterfowl hunter consumes 5.4 meals of resident waterfowl from the PSA each year (Vol. IV, p. 4-84) and that the RME waterfowl consumer ingests 11 waterfowl meals annually from the PSA. EPA assumes that each waterfowl meal is composed of one duck (Vol. IV, p. 4-84). At the same time, however, the HHRA estimates that the size of the resident duck population in the PSA (upon which the exposure point concentration is based) is 120 ducks (Vol. IV, p. 7-14). If there are only 120 resident ducks present in the area, 3,600 waterfowl hunters cannot average even one meal of such ducks during the season. Thus, the ingestion rates used for waterfowl cannot be considered representative of the total waterfowl hunter population estimated by EPA.

Assuming that there are 120 resident ducks in the PSA each year, and using reasonable but conservative assumptions about nesting success, clutch size, and fledging success, one can estimate that there could be as many as 150 resident ducks available for harvest each year without adversely affecting the resident duck population.[1] Based on that total, the CTE consumption rate of 5.4 ducks/year would only allow for an estimated exposed population of 28 consumers of such waterfowl in the PSA. Using the RME consumption rate of 11 ducks/year, it would only be possible for 14 waterfowl hunters to be exposed at the dose rate estimated for the RME. These are likely to be more realistic estimates of the sizes of the potentially exposed

[1] If there are 120 resident ducks, there could be as many as 60 breeding pairs. If it is conservatively assumed that half of the nests are successful (MFWP, 2005; Drilling et al., 2002; Evrard, 2000; Greenwood et al., 1995; Hepp and Bellrose, 1995), each breeding pair averages 10 eggs (EPA, 1993), and 50% of the eggs successfully fledge (Drilling et al., 2002), one can estimate that there are a total of 150 new resident ducks available for harvest each year [30 nests * 10 eggs/nest * 5 fledglings/10 eggs = 150 fledglings].

waterfowl hunter populations that are being evaluated using the exposure assumptions that have been developed for the HHRA.

Estimation of population size is an important exercise to provide perspective for a risk assessment. Risk management decisions need to consider the size of the population that may potentially be exposed at the levels modeled in the risk assessment. EPA's population size estimates are misleading because they imply that all 11,371 anglers and 3,600 waterfowl hunters might have exposures that result in the risk levels estimated in the HHRA, when in fact there are likely to be substantially smaller populations that would experience that potential level of exposure. For this HHRA,, it is important to make risk managers aware that the RME exposures estimated for the fish and waterfowl consumption pathways can only be considered representative of between 49 and 370 recreational anglers and approximately 14 waterfowl hunters, respectively.

2.2 ATSDR/MDPH Study

In its discussion of the ATSDR/MDPH (2002) study, the HHRA states that, while the residents of the Housatonic River Area (HRA) did not have excessive cancer incidence for the majority of six cancer types evaluated between 1982 and 1994, the occurrence of bladder cancer among males in the city of Pittsfield was elevated during that 13-year time period and was elevated in females in one census tract between 1987 and 1994 (Vol. I, p. 1-43). While that statement is consistent with the findings of the ATSDR/MDPH study, it could be incorrectly interpreted to mean that the increased bladder cancer observed in Pittsfield was due to PCB exposure. This perception would not be accurate.

It is important to expand the discussion to clarify that bladder cancer has not been an endpoint of concern for PCB exposure (ATSDR, 2000). While a number of investigators have evaluated standard mortality ratios for bladder and/or urinary tract cancers in humans exposed to PCBs (Kimbrough et al., 1999; Gustavsson and Hogstedt, 1997; Loomis et al., 1997), the standard mortality ratios calculated in these studies have not been reported to be significant. Thus, while ATSDR's regional study may have observed an elevation in bladder cancer in Pittsfield, it cannot be concluded that this elevation is related to PCB exposure. In fact, the ATSDR/MDPH (2002) report concluded that the "[r]eview of the available risk factor information related to cancers that were elevated in the city of Pittsfield suggests that cigarette smoking played a role in the increased rates of male bladder cancer" (p. 30). EPA should clarify this issue to avoid misunderstanding by the public.

2.3 Use of TEQ Approach in Toxicity Assessment

In response to comments received during the peer review process, EPA has revised its application of the TEQ approach to correct for the double-counting of potential risks due to PCBs, which was involved in EPA's previous approach. At the same time, however, EPA has retained its application of the TEQ approach to PCBs (in the main fish and waterfowl consumption assessment and in the sensitivity analyses for the direct contact and agricultural products consumption assessments) despite the substantial comments that were provided both by GE and by peer reviewers showing the flaws in application of the TEQ approach to PCBs. In addition, since the time of the peer review, there have been a number of new developments that have further undermined the appropriateness of the application of the TEQ approach to PCBs. These developments are summarized in a March 15, 2005 letter from GE to Dr. David Eaton, Chair of the National Academy of Sciences (NAS) Committee reviewing EPA's draft Dioxin Reassessment, a copy of which is attached (without its attachments) as Attachment A.

This more recent information confirms that the TEQ approach for PCBs is not appropriate and substantially overestimates potential risks due to PCBs. Consequently, GE again recommends that EPA altogether eliminate the TEQ analysis for "dioxin-like" PCBs from the final HHRA.

3.0 DIRECT CONTACT ASSESSMENT

EPA has made some changes to its deterministic risk assessment for the direct contact exposure pathways and has added probabilistic analyses of direct exposures using a surrogate soil exposure point concentration (EPC) of 1 ppm. GE believes that a number of the changes made to the exposure frequencies in the deterministic assessment are not consistent with site conditions or the available data. In addition, while GE supports the use of probabilistic analyses to provide additional information about potential risks to individuals involved in direct contact activities, the probabilistic analyses presented in the revised HHRA involve distributions that are unjustifiably skewed high, and its probabilistic model appears to contain errors in either calculation or reporting. GE's recommended changes to the direct contact assessment are discussed below.

3.1 Deterministic Analysis – Exposure Frequencies

There have been changes in the exposure frequencies used in the HHRA for many of the exposure areas (EAs) evaluated. In several cases, however, either the magnitude of the change in the frequency or the basis for the change is not supportable. In this connection, while EPA has considered the Housatonic River Floodplain User Survey (HRFUS) data collected by Triangle Economic Research (TER, 2003), it appears that EPA has been somewhat selective in doing so. In particular, it appears that EPA has used the HRFUS data when they support more conservative assumptions about the frequency of use of individual EAs, but has not reduced the exposure frequencies for some EAs for which the HRFUS data clearly indicate little to no use. GE believes that EPA should revisit the exposure frequency assumptions for the following EAs.

EA 18

EA 18 is a portion of a farm property, which the HHRA considers a potential future residential property (Vol. IIIA, p. 5-36). The revised draft HHRA has increased the assumed exposure frequency for this EA from an RME exposure frequency of 90 days/year and a CTE exposure frequency of 30 days/year to the default residential frequency of 150 day/year for both the CTE and RME analyses (Vol. IIIA, p. 5-37).

EA 18 is subject to an agricultural preservation restriction (APR) and, as such, is not likely to be converted for residential use. Moreover, even if this EA were converted to residential use, the default residential frequency of 150 days/year is not reasonable for portions of this area, which are not accessible. Specifically, only seven of the 14 sampling locations used to derive the EPC for this EA can be accessed without crossing open water (see Vol. IIIB, Figure 5-19). Despite the inability to access seven of the sampling locations without crossing open water, the HHRA designates most of them as “walkable” areas and thus gives them a full weight in the use-area weighting approach used to develop the EPC. Because these inaccessible sampling locations have higher PCB concentrations than the accessible areas, their inclusion in the development of the EPC with a weight of 100 percent inflates the estimate of the EPC for the area that would be likely to be accessed on a regular basis even if the EA were used for residential purposes. This, combined with an unreasonable exposure frequency that includes these areas, results in substantially overestimated exposures and risks for this EA.

To solve this problem, GE recommends that EPA either: (a) reduce the exposure frequency for the overall EA to the frequencies used in the prior draft; or (b) subdivide the EA into two different

exposure areas with different EPCs – one that includes all of the land area that could be accessed without crossing open water and the other that includes only those areas that would have to be accessed by boat – and assign a lower exposure frequency to the latter.

EA 42

EPA has reduced the RME exposure frequency for EA 42 from 90 days/year to 60 days/year and has retained the exposure frequency of 30 days/year for the CTE analysis (Vol. IIIA, p. 5-83). While GE agrees that it is appropriate to reduce the RME exposure frequency for this EA, it believes that the exposure the frequencies for the RME and CTE analyses should be further reduced to 30 and 15 days, respectively, to reflect likely low usage of the entire EA.

This parcel has only a very small walkable section that can be accessed without crossing water. The majority of the EA is wadable/difficult with very thick vegetation. In fact, only two of the 15 sampling points used to derive the EPC were obtained from walkable areas that can be accessed without crossing open water (see Vol. IIIB, Figure 5-42). While EPA has assigned a lower use-weighting factor to those areas, the reality is that it is likely that they will receive no usage except if accessed from the river itself.

Very limited use of the floodplain portion of this EA is also supported by the data collected during the HRFUS. No observations of floodplain use in this area were made from the river during the 60 canoe-based survey days, and during the car-based survey, only six cars were observed parked along October Mountain Road, which is at a substantial distance from the floodplain portion of the EA. Consequently, the recreation activities recorded by TER, which were associated with these parked cars, may or may not have occurred within the floodplain itself.

GE believes that EPA should revise its handling of EA 42 in one of two ways. The first way would be for EPA to subdivide EA 42 so that the area being evaluated as EA 42 only includes the walkable area that can be accessed from land without crossing open water. If this approach is taken, it would be reasonable to use the exposure frequencies of 60 and 30 days/year that EPA is currently using, given the change in the area included in the EPC. Alternatively, if EPA does not further subdivide the parcel, EPA should reduce the exposure frequencies to 30 and 15 days to reflect the limited usage that is likely to occur in the majority of the EA.

EA 43

EPA also reduced the RME exposure frequency for EA 43 from 90 days/year to 60 days/year and has retained the former CTE frequency of 30 days/year (Vol. IIIA, p. 5-84). GE supports a reduction in the RME exposure frequency for EA 43 but believes that both the RME and CTE frequency should be reduced further to 30 and 15 days/year, respectively. This is because only a tiny sliver of land is actually walkable and one needs to go down a steep slope to get to it. It is unlikely that this area receives any regular usage.

During 60 days of the canoe-based survey in this area, there were no individuals observed using it. Map 5 of Attachment E of the HRFUS indicates that only one car was observed parked along October Mountain Road during the car survey. The observation point for this automobile was located at a substantial distance from the floodplain portion of the EA. The walking activity recorded by TER associated with the parked car was likely limited to October Mountain Road with no contact with floodplain soils.

The physical characteristics of this EA are very similar to EAs 9 and 29, to which EPA has assigned exposure frequencies of 30 and 15 days for the RME and CTE analyses, respectively. In addition, the HRFUS indicated very little to no use of the area. Thus GE recommends that the exposure frequencies for EA 43 be further reduced to 30 days/year for the RME analysis and 15 days/year for the CTE analysis.

EAs 45, 46, 48, and 54

EPA has added the general recreation scenario to EAs 45, 46, 48 and 54 and assigned exposure frequencies of 90 and 30 days to them for the RME and CTE analyses, respectively (Vol. IIIA, pp. 5-88, 5-90, 5-94, and 5-105). GE believes that these assigned exposure frequencies are too high, given the physical characteristics of the EAs and the use levels observed during the HRFUS.

Access to a substantial portion of EA 45 is blocked by water or by wadable/difficult terrain. During the 60 days of canoe-based counts in the HRFUS, no individuals were observed using the floodplain portion of this EA. The car-based counts reported a total of 11 cars parked along October Mountain Road in this area. These cars were parked a marked distance from any of the floodplain area and close to residences and a garden, which are located outside of the

floodplain along October Mountain Road. Thus, at least a substantial number of these parked cars may have been associated with activities other than recreational activity in this EA.

Much of EA 46 is blocked by open water or is wadable/difficult. During 60 days of canoe-based counts, TER did not observe any individuals engaged in activities within the floodplain. The car-based counts included 5 observations of parked cars and indicated that there were two individuals engaged in waterfowl hunting (which is already being evaluated as a separate exposure scenario for this EA) and three individuals engaged in general recreation. Thus, it appears that the general recreational use of this area is very limited, likely due to the physical characteristics of the EA.

Most of EA 48 is wadable/difficult. During the 60 days of canoe-based counts conducted by TER, no individuals were observed using the floodplain in this EA. The car-based counts indicated that 9 vehicles were observed parked adjacent to the EA along October Mountain Road and that those individuals were engaged in walking, hunting and other recreational activities. Hunters are already being evaluated for this EA. In addition, because of the distance between the parking area along October Mountain Road and the floodplain, as well as the physical characteristics of the EA itself, it is likely that these other recreational activities occurred along October Mountain Road or within October Mountain State Park and resulted in no exposure to floodplain soils.

Except for the boat launch area, EA 54 is wadable/difficult and is not likely to be regularly used except as a boat launch. During 60 days of canoe-based counts, no individuals were observed engaged in floodplain based activities there. A total of nine cars were observed parked in the area during the 60 days of car-based counts. One of these cars was reported to be associated with fishing activity, one with walking, and the other 7 with unknown general recreational activities. It is likely that these 7 cars were associated with the boat launch activity or activities along October Mountain Road or in the adjacent State Forest.

Due to the physical characteristics of these EAs, GE believes that all of these areas should be evaluated as “low use” areas, using exposure frequencies of 30 and 15 days for the RME and CTE analyses, respectively.

EA 55

In the previous draft of the HHRA, EPA evaluated EA 55 using frequencies of 30 and 15 days (for older children and adults, respectively) due to the fact that the area was remote, densely vegetated, and wet. The revised HHRA increases the exposure frequencies to 90 and 30 days (for older children and adults) and adds potential exposure to young children with a frequency of 15 days/year for both the RME and CTE analyses (Vol. IIIA, p. 5-107). The addition of the young child receptor is appropriate given that young children were observed in the EA during the HRFUS. GE believes, however that the revised frequencies assigned to older children and adults are too high, given the conditions reported by EPA for the EA, which have not changed since the 2003 draft.

While EPA has justified this change in frequency based on the fact that it is possible to access this EA from October Mountain Road and the canoe/boat launch to the north (Vol. IIIA, p. 5-107), the EA is still not that likely to be heavily accessed for other recreational purposes due to physical conditions of the EA. During 60 days of canoe-based observations in the HRFUS, a total 5 individuals were observed using the floodplain. A total of 23 cars were reported parked along October Mountain Road toward the southern end of EA 55. At this location, however, the river is located at a substantial distance from October Mountain Road across difficult terrain and thus is not likely to be the focus of the recreational activity associated with these cars. This part of October Mountain Road also provides access to trails leading into October Mountain State Forest. Thus it is likely that most of the activities associated with these cars were related to walking/hiking along October Mountain Road or within the State Forest. Neither of these is located within the floodplain of the river.

In light of the physical conditions in this EA and the types of observations made during the HRFUS, GE recommends that revised frequencies of 60 and 30 days be used for the RME and CTE analyses of older children and adults in this EA. GE supports the use of EPA's current frequency of 15 days for young children.

EA 56

EPA changed the recreational frequencies for this EA from 30 and 15 days/year to 60 and 30 days/year, based on the proximity of the EA to a residence and the Woods Pond footbridge (Vol. IIIA, p. 5-109). GE believes that this frequency is too high. In fact, only two older children were observed bicycling through in this EA during 118 observation days of the HRFUS. While

there is one residential property nearby, the residential property has its own frontage on Woods Pond and that frontage is likely to be preferentially used by those residents. GE recommends that EPA reassign its former frequencies of 30 and 15 days/year for the RME and CTE analyses, respectively, to EA 56.

3.2 Probabilistic Assessment

EPA has added probabilistic analyses of the direct contact pathways, including a Monte Carlo Analysis (MCA) and a Probability Bonds Analysis (PBA). In many cases, the upper bound estimates from the MCA predict higher risks than the RME point estimates. This is due largely to the fact that, in that probabilistic analysis, EPA has modified the underlying distributions for certain parameters, adding degrees of conservatism at the outset that are not appropriate. In addition, it appears that EPA may have made an error in its probabilistic model, specifically related to the development of a factor identified as "X," which is a surface area-weighted adherence factor. If an error in calculation was made, the predicted risk results may be incorrect.

3.2.1 Use of Skewed Distributions

For some of the parameters used in the probabilistic analyses, the revised HHRA uses distributions that are unjustifiably truncated at an artificially high minimum value or are otherwise skewed toward higher values, thus producing overestimates of exposure and risk. These parameters are the soil ingestion rates for certain exposure scenarios, the fraction of soil ingested, and the dermal absorption rate for PCBs.

Soil Ingestion Rate

For certain scenarios (young children, hunters, ATV/dirt bikers), the MCA used in the revised HHRA sets the minimum soil ingestion rate at 50 mg/day (Vol. IIIA, p. 6-11). This approach artificially skews the analysis toward higher ingestion rates and does not reflect the underlying data upon which the soil ingestion rates are based. The Stanek and Calabrese (1992) data for soil ingestion, upon which EPA's point estimate soil ingestion rates for young children are based, indicate that, with the exception of the one pica child involved in the study, the rates of ingestion by 1 to 5 year old children ranged from a minimum rate of 5 mg/day to a maximum of 241 g/day, with a median of 37 mg/day and a mean of 54 mg/day. Thus, even for young children, EPA's approach to the soil ingestion input distribution does not reflect the data upon which it is based and will result in substantially biased estimates of exposure and risk. This bias

is even more pronounced for older children and adults, who are known to have substantially lower soil ingestion rates than young children.

In addition, for the hunter scenario, the revised HHRA not only sets the minimum soil ingestion rate at 50 mg/day but also sets the maximum soil ingestion rate at 200 mg/day (Vol. IIIA, p. 6-11), rather than the upper bound estimate of 100 mg/day that is used in the deterministic analysis. The PBA is even more skewed toward high-end soil ingestion because it sets the maximum rate at 300 mg/day (Vol. IIIA, p. 6-11). As support for these maximum values, the HHRA cites the ingestion rate modeled by Hawley (1985) and the results of the Stanek et al. (1992) adult consumption study, which reported one adult with a soil ingestion rate of 331 mg/day (Vol. IIIA, p. 6-11). The Hawley soil ingestion rate has no empirical basis, was not supported by any direct measurements, and is based on a number of assumptions that have since been determined to be unrepresentative of real-life conditions. EPA's *Exposure Factors Handbook* (1997) describes it as a "conjectural" value. In addition, the 331 mg/day upper bound ingestion rate reported in the Stanek et al. study was reported by those authors to be an unreliable estimate of daily soil ingestion because it reflected three to four days of accumulation rather than a single day of ingestion. One of these authors has since recommended that the 75th percentile of the soil ingestion rate distribution from that study, 49 mg/day, is a more reliable estimate of upper bound soil ingestion by adults (Calabrese, 2003).

For these reasons, GE believes that EPA should revise its probabilistic analyses to be representative of the available data on soil ingestion. For young children, EPA should either directly use the empirical data provided by Stanek and Calabrese (1992) or use a triangular distribution that reflects those data, with a minimum of 5 mg/day, a maximum of 241 mg/day, and a mode of 37 mg/day. For adults and older children, EPA should revise its distribution to have a minimum of 1 mg/day, a maximum of 100 mg/day, and a mode of 10 mg/day, based on the information provided by Stanek et al. (1992).

Fraction Ingested

The HHRA also truncates the input distribution for the fraction of soil ingested from the site and uses a uniform distribution ranging from 50 to 100 percent (Vol. IIIA, p. 6-13). This distribution is also biased and does not reflect the variability that is likely to occur for individuals involved in direct contact activities. For example, there may be many days when individuals are present in exposure areas for only a very brief period of time and do not have any hand contact with soils. Since hand-to-mouth activity is generally considered to be the source for soil ingestion, there

would be no soil ingested from the exposure areas on those days. Similarly, there may be many days during which hunters or other recreators will wear gloves, due to cold temperatures, so that there will be no direct contact between hands and soils. Thus, it is appropriate to allow the input distribution to range between zero and 100 percent to reflect the natural variation that may occur within the exposed population and during different days of activity. Accordingly, EPA should replace its current distribution for fraction ingested with a uniform distribution that ranges from zero to 100 percent.

PCB Dermal Absorption Rate

For its probability bounds analysis, EPA has developed a highly skewed input distribution for the PCB dermal absorption rate, with an interval that ranges from 6 percent to 41 percent. The upper end of the range was derived through MDEP's (Harnois and Smith, 2001) manipulation of the data from the Huntingdon Life Sciences study, subsequently reported by Mayes et al. (2002), and is based on an assumption that is not supported in the published literature. The lower end of the range is based on the upper-bound estimate of absorption derived by EPA (1992a) based on the Roy et al. (1990) study. This value was previously used by EPA (EPA, 1992a) as its upper-bound estimate of the dermal absorption factor for PCBs. Thus, the distribution fails to consider the other data provided by Roy et al. (1990), which indicated that dermal absorption could be as low as 0.6 percent (EPA, 1992a). Consequently, EPA's input distribution does not provide an accurate representation of the range of variability and uncertainty associated with this important exposure parameter.

The Mayes et al. (2002) study evaluated absorption of Aroclor 1260 through the skin of rhesus monkeys, demonstrating absorption factors of approximately 4 percent. During that study, the researchers could account for roughly 59 percent of the Aroclor 1260 dose in the dosing apparatus or on the skin at the end of the dosing, leaving approximately 41 percent of the original dose unrecovered. In their discussion of these data, Harnois and Smith (2001) assumed that the remaining 41 percent of the nominal dose was retained in the skin for later absorption. This is the basis for the upper end of the dermal absorption distribution used in the probabilistic analysis of the HHRA.

Harnois and Smith's (2001) assumption that 41 percent of the dose was retained in the skin is not borne out by experiments conducted by Wester et al. (1993), the study upon which the HHRA bases its point estimate dermal absorption factor of 14 percent. In that study, Wester et

al. also evaluated the percutaneous absorption of Aroclors 1242 and 1254 into human skin *in vitro*. These Aroclors were applied to the skin in soil, mineral oil, or water. The material was left on the skin for 24 hours. The skin surface was then washed once with liquid soap and twice with distilled water. Wash solutions and cells were then analyzed for PCB content. Wester et al. (1993) reported that only 2.6 percent of the Aroclor 1242 applied in soil and 1.6 percent of the Aroclor 1254 applied in soil remained on the skin after the soil was removed and the skin was washed. While Wester et al. did not evaluate Aroclor 1260, it is likely that the amount remaining on the skin would be similar or lower, given the very high binding affinity of Aroclor 1260 to soil particles. These findings appear to indicate that MDEP's assumption that 41 percent of the applied dose would be retained in the skin after washing is unlikely to be true and results in overestimated estimates of absorption.

In addition, that upper-bound absorption factor is not supported by dermal absorption studies of other chlorinated organic compounds (EPA, 2001). As shown in the following table, EPA's (2001) dermal guidance (Exhibit 3-4) recommends the following absorption factors for specific chlorinated organic compounds.

Chlorinated Organic Compound	Dermal Absorption Fraction
PCBs	0.14
Chlordane	0.04
2,4-D	0.05
DDT	0.03
TCDD	0.03
Lindane	0.04
Benzo(a)pyrene	0.13

Clearly, there is uncertainty associated with the dermal absorption factor for PCBs, and GE believes that it is appropriate for EPA to consider the range of possible values in its probabilistic analysis. However, as with the soil ingestion and fraction ingested parameters, the HHRA biases the dermal absorption distribution by using only upper-bound values, rather than evaluating the full range of uncertainty that is evident. At a minimum, EPA should revise the lower bound of the PBA range from 6 percent to 0.6 percent to capture the full range of uncertainty associated with this factor.

3.2.2 Potential Errors in Development of the "X" Factor

The revised HHRA uses a subprogram in its probabilistic analyses that is intended to combine a number of factors, including body surface areas based on body weight and height (adults only), body part-specific adherence factors, and seasonal variations in exposure to produce an input distribution to assist in evaluating potential risks due to dermal exposure for each age and receptor group (Vol. IIIA, pp. 6-15 - 6-16). This subprogram combines distributions for body weights, heights, surface areas for each body part exposed (accounting for seasonal changes), and adherence factors for each body part to derive an input distribution for the single factor, X, that goes into the probabilistic risk calculation. The values estimated by EPA for X are provided in Table 6-3 (Vol. IIIA) of the HHRA.

GE has not been able to duplicate the "X" values derived by EPA. This is likely due, at least in part, to the lack of information provided about the distributions used for soil adherence factors. It may also be due to errors in the approach.

Table 6-4 of the HHRA indicates that EPA has developed "empirical" distributions for the soil adherence factors and it provides minimum and maximum values for these distributions. It does not, however, provide any information about the shape of the distribution itself. Thus, it is not possible to ensure that the inputs for adherence factors used by GE in an effort to duplicate the calculation are or are not correct.

Table 6-4 also states that the equation used to calculate the surface area (SA*) for each body part is $SA^* = a\ BW^b\ H^c$, in which a, b, and c are constants listed in that table. That table does not agree, however, with Table 6-1 in EPA's *Exposure Factors Handbook* (1997), which describes the same methodology. According to Table 6-1 of *Exposure Factors Handbook*, SA is calculated as follows:

$$SA = a_o\ W^{a1}\ H^{a2}$$

Using the female head as an example, EPA (1997) reports that the values for these components of the equation are the following:

$a_o = 0.0256$

$W^{a1} = 0.124$ where W is the body weight (equivalent to BW in HHRA Table 6-4)

$H^{a2} = 0.189$ where H is the height (equivalent to H in HHRA Table 6-4)

The values for a_o agree between these two documents but there is not agreement in the values for "b" and "c." According to Table 6-4 of the HHRA, the value of "b," which is the exponent for the body weight in that equation for the female head, is 0.124. However, EPA (1997) indicates that 0.124 is actually the value of the entire factor W^{a1}, not just the exponent. Similarly, the HHRA reports that the value of "c", which is the exponent for H in Table 6-4, is 0.189. Again, however, EPA (1997) reports that 0.189 is, in fact, the value of the entire factor H^{a2}. In addition, EPA (1997) reports that this approach calculates surface areas in square meters while the HHRA reports them to be in square centimeters. When the units are converted to be consistent, the calculated values do not match.

It is not clear whether EPA has made errors in its calculations. It does not appear that the equation presented in the *Exposure Factors Handbook* is correct because all values in the equation would be constant so that all surface areas would be calculated to be the same, regardless of weight or height. At the same time, however, when AMEC and BBL attempted to use the equation presented in the HHRA, we were not able to derive the numbers that are reported. EPA needs to check its approach to make sure that it has been conducted correctly; and if it has, needs to fix any errors that may be in the equation and provide a more transparent explanation in the text of how these surface area estimates, and the resulting X factor, are derived.

4.0 FISH AND WATERFOWL CONSUMPTION ASSESSMENT

In response to the peer reviewers' comments on the previous draft HHRA, EPA has revised the fish consumption rates for both the deterministic and probabilistic analyses. The HHRA now bases its fish consumption estimates on a subpopulation of anglers from the Maine angler survey (Ebert et al., 1993) – namely, those who did not share any of the fish they caught or obtained with any other individuals. The HHRA then assumes that young children (aged 1 to 6) have fish consumption rates that are one-half the adult rates. The HHRA needs to recognize, however, that the subpopulation of non-sharing consumers is not representative of the total population of fish consumers who may use the study area. In addition, there is evidence that the consumption rate estimates used to evaluate young children do not represent consumption of sport-caught fish by this age group.

4.1 Adult Fish Consumption Rate in Deterministic Assessment

The revised HHRA bases its fish consumption rates on a small subpopulation of individuals who participated in the Maine angler survey (Ebert et al., 1993). The consumption rates are now based only on adults in that survey who consumed 100 percent of the fish that they caught or obtained from other sources (i.e., those who did not share any of that fish with any other individual throughout the one-year survey period) (Vol. IV, p. 4-48). This subpopulation was selected due to EPA's concern that the sharing assumptions that had been used in deriving the consumption rates in the Maine angler survey might be underestimating exposures to adult male consumers who might consume more fish than would women or children in the survey (Vol. IV, p. 4-41). In making this selection, the revised HHRA assumes that this subpopulation of non-sharing consumers is representative of all sport-caught fish consumers.

As discussed in Section 2.1 above, this assumption does not appear to be correct; and EPA should make clear that this subpopulation is likely to be very small and cannot be considered representative of the total population of recreational anglers that use the Housatonic River.

Moreover, in an effort to support its adult fish consumption rates, the HHRA cites the results of the MDPH exposure prevalence study (MDPH, 1997) and the CT Housatonic River creel survey (Ebert et al., 1996) (Vol. IV, pp. 4-50 - 4-53). These comparisons, however, contain errors that need to be corrected before the HHRA is finalized.

The HHRA indicates that the data collected in the MDPH exposure prevalence study support the RME consumption rate of 31 g/day used in the HHRA (assumed to be equivalent to 50 8-ounce meals/year). The rationale for this conclusion is that the 95th percentile frequency of fish meals reported in the MDPH survey was 104 meals/year and that 75 percent of those meals were sport-caught meals, resulting in an estimate of 78 sport-caught fish meals/year (Vol. IV, p. 4-51). This conclusion cannot be drawn from the MDPH survey data due to the way in which the data were collected. The questionnaire used in the MDPH (1997) study does not allow EPA to calculate the fraction of sport-caught fish meals consumed by those individuals. The participants in that study were asked to estimate the frequency of freshwater fish meals they consumed. In the following question, they were asked to indicate how they "usually" obtained those fish. They were not given an option of assigning a fraction of meals as sport-caught or supermarket/grocery store fish. Hence, the respondents were forced to designate all of their fish meals as either sport-caught or supermarket/grocery store fish even though they may have

consumed a mixture of both. Because of the way that the questionnaire was designed, each participant would have had a frequency of either 100 percent sport-caught or zero percent sport-caught. Thus, the HHRA's assumption that 75 percent of the meals were sport-caught actually reflects the fraction of study participants who reported that they "usually" obtained those fish from sport-fishing, not the actual fraction of fish meals consumed that were sport-caught. Consequently, the HHRA's comparison is misleading and should be dropped from the discussion.

In addition, the HHRA incorrectly cites the results of the CT Housatonic River creel survey (Ebert et al., 1996). The HHRA reports that the 95th percentile consumption rate estimates ranged from 21.3 g/day to 32 g/day, depending upon the assumptions made about the number of individuals with whom the fish were shared (Vol. IV, p. 4-52). In fact, the range of 95th percentile rates reported by the authors for different sharing assumptions ranged from 12 g/day (assuming that fish was shared equally among household members) to 32 g/day (assuming that all of the fish harvested were consumed by a single individual). EPA needs to correct this error.

The HHRA goes on, on page 4-52, to make a separate calculation, based on the Maine angler survey consumption rate data and the information on relative gender-specific sizes of all types of fish meals (not just sport-caught fish meals) provided in the EPA's *Exposure Factors Handbook* (1997; Table 10-37). The calculation is intended to demonstrate that men consume more fish than women and that when the total mass of consumable fish reported by each Maine angler (not just the non-sharing individuals) is assumed to be consumed by one male and one female consumer per household and adjusted for relative gender-specific portion size, the same estimate of 31 g/day results.

This comparison is completely artificial because it manipulates the data from the Maine angler survey, making separate assumptions about sharing that are not supported by the data provided by the participants themselves, and then applying a ratio of gender-specific consumption rates that are also not necessarily reflective of long-term consumption behaviors. The data upon which EPA's gender-specific fish consumption rate ratio is based are short-term (3-day diary study) data, and are more representative of portion size for individual meals than they are of long-term consumption rates. These data represent a three-day average consumption rate for only those individuals who consumed fish during the 3-day study period. They did not capture the long-term behavior of those individuals nor did they include individuals who consume fish but did not do so during that 3-day study period. While the ratio of portion sizes may be

appropriate, it does not mean that women have lower long-term consumption rates than men, because long-term consumption is a combination of portion size and meal frequency, and the EPA data that are being used to make this comparison do not provide any information about long-term frequency of fish meals.

A better measure of the relative consumption by males and females is provided in the data for the subpopulation of non-sharing consumers in the Maine angler survey (Ebert et al., 1993) upon which the HHRA bases its consumption rates. These data indicate that the HHRA's unequal sharing assumption may not be representative when sport-caught fish are consumed. In fact, as shown below, the information available from the Ebert et al. (1993) survey for those non-sharing individuals indicates that long-term rates of consumption of sport-caught fish by men and women are very similar.

Fish Consumption Rates (g/day) for Non-Sharing Consumers

	Male	**Female**
Minimum	0.1	0.2
Maximum	182	52.4
Median	2.8	3.7
Mean	8.9	9
75th percentile	8.9	11.5
90th percentile	21	17
95th percentile	31	31

These data indicate that, over a one-year survey period, the non-sharing female fish consumers ate comparable amounts of fish to the amounts eaten by men. In fact, the 95th percentile for non-sharing females was identical to the 95th percentile value for non-sharing males. These data indicate that males and females eat approximately the same amounts of sport-caught fish, so that the assumption used in the Maine angler survey may have been a very reasonable assumption. These data also undermine EPA's rationale for selecting fish consumption rates for this non-sharing subpopulation, and instead support GE's previously recommended 95th percentile consumption rates derived from the Ebert et al. (1993) survey (12 g/day for rivers/streams and 16 g/day for lakes/ponds; see AMEC and BBL, 2003) as likely more representative of the total angler population.

4.2 Child Fish Consumption Rate in Deterministic Assessment

The revised HHRA uses data from EPA's *Estimated Per Capita Fish Consumption in the United States* (2002) to set children's fish consumption rates at 50 percent of the adult fish consumption rates (Vol. IV, pp. 4-53 - 4-54). The rates provided in that document, however, are not representative of long-term consumption rates, but instead are representative of meal sizes since they are short-term measures of consumption from 2-day diaries. As discussed in Section 4.1, such comparisons do not necessarily comport with frequency or long-term consumption behavior.

Researchers at Cornell University (Knuth et al., 1998) evaluated consumption of sport-caught fish by children aged 8 to 14 years. This diary study collected information about children from sport-fishing families who were involved in the New York Sportfishing and Aquatic Resources Education Program (SAREP). These researchers asked the children to record meal-by-meal information for all fish meals eaten between July 1 and October 15, 1996. The source of each fish meal (store, restaurant, or sport-fishing) and a relative portion size for each were also recorded.

According to the raw data provided by the study's authors, these children ate between 0 and 12 total fish meals and 0 to 11 sport-caught fish meals during the survey period of 3.5 months, with average rates of 4 total fish meals and 1 sport-caught fish meal during that time period (Knuth et al., unpublished data). Using the portion size information provided by each individual for each meal consumed, summing all meals consumed to derive a total amount of fish consumed during the survey period, and dividing by the number of days included in the survey period (107 days), AMEC derived a distribution of fish consumption rates for the survey period that ranged from 1 to 22 g/day with a mean of 1.7 g/day and a 95th percentile of 4.3 g/day. These rates were based on data collected for children aged 8 to 14 years and thus are likely to substantially overestimate consumption by children under the age of six, who are likely to have smaller portion sizes than the older children studied by Knuth et al. (1998). In addition, these rates likely overestimate consumption over a one-year period because sport-fishing activities and consumption are likely to be greatest during open water season (the period in which the survey was conducted) so that consumption for many of these children may have been substantially lower during the winter months.

The following is a comparison of the rates derived based on the Knuth et al. (1998) data with the point estimates used in EPA's deterministic analysis. (While the HHRA does not specifically identify consumption rates for older children, it essentially uses the same consumption rates for anyone over the age of 6 years.)

Sport-Caught Consumption Rates (g/day)

	Knuth et al. data	EPA point estimates (bass)	
	8-14 year olds during the Diary Period	1-6 Year Old Children	Older Children/ Adults
Mean	1.7	4.3	8.7
95th %ile	4.3	16	31

It appears that the 95th percentile rate used by EPA to evaluate children aged 1 to 6 years overestimates consumption by this age group by at least a factor of 4, since this age group would be expected to consume smaller amounts of fish than the 8 to 14 year old children included in the Knuth et al. (1998) survey. In addition, the RME adult consumption rate, which is assumed to be applicable to anyone over the age of 6 years, overestimates consumption by 8 to 14 year old children by a factor of 8. In fact, because the Knuth et al. data covered the period between July and October, which is likely to be a much more active fishing period for most anglers than the winter months, even this analysis of the Knuth et al. data is likely to overestimate consumption for this age group.

Although the Cornell study did not collect diary information on children under the age of 8, another phase of the study provided information on the estimated 12-month meal frequencies for younger children in these same families (Knuth et al., unpublished data). Using a very conservative meal size of 4 oz. (the most common meal size reported by the 8 to 14 year old children, who would be expected to eat larger portions than 1 to 6 year old children), the data available for these young children indicated that their mean consumption rate is 2 g/day and their 95th percentile consumption rate is approximately 3.0 g/day. Thus, based on this comparison of long-term rates, it appears that EPA's point estimate consumption rates for young children overestimate mean (CTE) consumption by a roughly a factor of 2 and RME consumption by more than a factor of 5.

Given the availability of specific sport-caught fish consumption information for children aged 8 to 14 years in the Knuth et al. (1998) study, GE recommends that EPA revise its deterministic fish consumption rates for young children to include a mean of 2 g/day and a 95th percentile of 3 to 4

g/day. While the HHRA does not separately evaluate fish consumption by older children, it should discuss the fact that older children are inherently included in the adult fish consumption rates and that these consumption rates overestimate potential exposures for the older children.

4.3 Child Fish Consumption Rate in Probabilistic Assessment

In its probabilistic analyses, the revised HHRA evaluates exposures to young children due to the consumption of fish by developing input distributions for the numbers of meals consumed during the year and the sizes of those meals (Vol. IV, p. 6-28). As discussed above, the Knuth et al. (1998) survey provides information on the frequency of sport-caught meals consumed by 8 to 14 year old diary participants during the survey period. A separate survey instrument used by these authors to screen families and select children for the diary survey asked families to estimate the number of sport-caught and total fish meals consumed by themselves and ALL children in their families (all ages) on an annual basis. The following table provides a comparison of the estimated meal frequencies (meals/year) provided in the family survey for diary participants (aged 8 to 14 years) and for all children aged 1 to 14 years, with the input distributions used in the HHRA's probabilistic analyses (MCA and PBA) for bass consumption in all reaches:

Comparison of Meal Frequencies in Cornell Data and EPA Probabilistic Inputs						
	Meals/Year Based on Family Survey Data Collected by Knuth et al. (1998)				**EPA Probabilistic Analyses for Bass**	
Summary statistic	Diary (8-14 yr-old) participants including those who ate no sport-caught meals	Diary (8-14 yr-old) participants who ate at least one sport-caught meal	All children (all ages) including those who ate no sport-caught meals	All children (all ages) who ate at least one sport-caught meal	MCA Input Distribution for Bass	PBA Input Distribution for Bass
Minimum	0	1	0	1	0.25	0.03
Maximum	20	20	100	100	145	490
Mean	3	6	4	9	13.1	8.3 - 24.3
50^{th} %ile	1	4	0	4		
95^{th} %ile	17	20	20	20		
n	48	27	113	54		

As demonstrated in the above table, there were many children of sport-fishing families who did not consume any sport-caught fish despite the fact that their families did. To be representative of children who do eat sport-caught fish meals, it is most appropriate to base meal frequency estimates on the population of children who were reported to consume at least one sport-caught fish meal. For such 8 to 14 year old children in the diary study, the meal frequencies ranged from 1 to 20 meals/year, with a mean of 6 meals/year and a 95th percentile of 20 meals/year. For all children in these families, including those who did not participate in the diary survey, the meal frequencies for those who ate at least one sport-caught meal ranged from 1 to 100 meals/year, with a mean of 9 meals/year and a 95th percentile of 20 meals/year.

The HHRA's range for the MCA is quite conservative in that it ranges up to 145 meals/year and has a central estimate of 13.1 meals/year, which is 50 percent higher than the arithmetic mean based on the Knuth et al. (1998) data. Moreover, the range for the PBA analysis is highly over-conservative in comparison with the Knuth et al. (1998) data. The range of potential meal frequencies used in the PBA is 0.03 to 490 meals/year and the central estimate is an interval bounded by 8.3 and 24.3 meals/year. The maximum value, which is equivalent to 1.3 fish meals per day, is not supported by available fish consumption data (including adult consumption rates from the Maine angler survey upon which it is purportedly based) and appears to overestimate the maximum meal frequency reported in the Knuth et al. study by a factor of 5. The lower bound of the central estimate interval in the PBA is similar to the arithmetic mean meal frequency observed in the Knuth et al. family survey data, but the upper bound of the interval is higher by nearly a factor of 3. It appears that this approach will yield highly inflated estimates of exposure to young children.

GE recommends that EPA base its children's fish consumption rate distribution on the data provided in the Knuth et al. (1998) study. A distribution based on these data would include a minimum of 1 meal/year, a maximum of 100 meals/year, and a central estimate of 9 meals/year. Use of such a distribution in the probabilistic analyses would yield more representative estimates of exposure and risk.

5.0 AGRICULTURAL PRODUCTS CONSUMPTION ASSESSMENT

EPA has made a number of changes to the agricultural products consumption assessment. To begin with, EPA has added text attempting to support the assumption that the agricultural products consumption pathways evaluated in the HHRA represent reasonably anticipated future

uses (Vol. V, Section 2.1.2). This discussion is based in part on interviews with local farmers and staff at the Pittsfield office of the USDA Farm Services Agency, local agricultural groups as well as personal observations by EPA personnel, contractors, and risk assessors. Much of this information, however, consists of personal opinions, not verified by independent sources, and as such is speculative. GE believes that the HHRA should not rely on such speculations, but should base its discussion on actual data.

In addition, EPA has made changes to its deterministic analysis of the agricultural products consumption pathways and has added probabilistic analyses of these pathways in an attempt to evaluate the variability and potential uncertainties associated with them. As discussed in the following subsections, a number of the revised factors used in the deterministic analysis are not justified, and the probabilistic analyses fail to adequately account for the variability and uncertainties in the risk estimates by using conservative point estimates rather than distributions.

5.1 Deterministic Assessment

At least two of the factors used in the revised HHRA's deterministic assessment of the agricultural consumption pathways are unjustified. First, the revised soil-to-plant transfer factor that is used to evaluate transfer to both exposed plants and fruits fails to take into account the removal of soil as vegetables and fruits are prepared for consumption, and does not even reflect the underlying data upon which it is based. Second, while the point estimate used for the mammalian bioconcentration factor (BCF) may have been reasonable for the range of soil concentrations modeled in the previous draft of the HHRA, it is not appropriate for the higher soil concentrations that are being modeled in the revised HHRA. These points are explained below.

Soil-to-Plant Transfer Factor Used To Evaluate Exposures Via Vegetable and Fruit Consumption

Like the 2003 draft of the HHRA, the 2005 draft explains that "the maximum wet weight soil-to-plant [transfer factor] for corn" was used to estimate total PCBs (tPCBs) in "exposed" surface vegetables and fruits (Vol. V, pp. 4-12, 4-14). Although the basis for this transfer factor (TF) did not change (i.e., use of the maximum value for corn), the number used in the deterministic assessment was increased from 6.4E-04 in the 2003 draft to 1.8E-03 in the current version.

Using the maximum value overestimates the amount of tPCBs on ingested vegetables because it assumes that none of the food items consumed are ever washed. For a daily lifetime exposure, this is an erroneous assumption. This assumption is particularly critical since the TF of 1.8E-03 accounts for the only mechanism for PCBs to be ingested via this exposure pathway. If it is assumed that an individual washes the fruit and/or vegetable before consumption, thereby removing the PCBs and rendering the TF irrelevant, there would be no exposure via this pathway. While the deterministic assessment incorporates a "Produce Loss Factor" for fruit and vegetables to take into account removal during the processing of the food for consumption (e.g., peeling), this loss factor was only included in the CTE analysis (see Vol. V, Table 4-10). Thus, for the RME, the HHRA assumes that 100 percent of the PCBs deposited on the surface of the fruit or vegetable, as estimated by the TF of 0.0018, is consumed. This is an unreasonable assumption, resulting in an overestimation of exposure and risk from this pathway. A reasonable high-end estimate would be that, over time, at least 50 percent of the transported PCBs would be removed as a result of washing before eating, preparation before cooking, or a combination of the two.

In addition, the data presented in Table 4-4, upon which this TF is based, only include the samples with detectable concentrations of tPCBs in unwashed corn stalks. In fact, half of the corn samples analyzed as part of that sampling event had no detectable levels of PCBs (see Vol. V, Table 2-5). Thus, transfer factors based on these samples, if included, would have been zero. The fact that some of the corn stalks had no detectable levels of tPCBs underscores the highly and unnecessarily conservative nature of EPA's use of the maximum value as the TF.

In summary, the revised HHRA: (a) uses the maximum soil-to-plant transfer factor calculated for corn to evaluate all exposures to PCBs in surface vegetables and fruit; (b) assumes that there is no washing of any vegetable or fruit before consumption throughout the exposure period; (c) assumes, for the RME scenario, that vegetables and fruits are never peeled before consumption; and (d) bases the transfer factor on only the corn stalk samples that had detectable levels of tPCBs, thereby ignoring 50 percent of the available data. This combination of assumptions results in an unreasonable exposure scenario. It should be modified to reflect more realistic exposure conditions.

Mammalian Bioconcentration Factor

While the mammalian BCF used in the deterministic assessment has not changed from the 2003 version, the modeled PCB soil concentrations have been altered in the revised draft. This

fact impacts the choice of the mammalian BCF values, which appear to be dependent on the soil concentrations.

Fries (1996) reported a range of BCFs, 1.5 to 3.6, for the transfer of Aroclor 1254 into milk fat, with an apparent inverse relationship between the PCB concentration in feed and the estimated BCF (i.e. the BCF decreases as the PCB concentration increases). In its 2003 draft HHRA, EPA selected the maximum value reported for Aroclor 1254 (3.6) based on the rationale that the range of BCFs (3 to 3.6) was selected from studies in which dietary concentration for the test animals was in the range of dietary concentrations predicted in the assessment (i.e., <1 ppm PCBs) (EPA, 2003, Vol. V, p. 4-14). This value was retained in the 2005 updated HHRA (Vol. V, p. 4-27). However, unlike the June 2003 version of the HHRA, where the maximum floodplain soil concentration modeled was 2.0 ppm, the revised report attempts to model milk and beef concentrations in animals exposed to soil concentrations as high as 25 ppm. Therefore, EPA's justification for using the higher BCF does not apply to the analyses in the revised deterministic approach. Rather, since the revised HHRA is modeling PCB concentrations that range over an order of magnitude, GE believes that the mean of 2.6 of the values from the report by Fries (1996) should be used for the deterministic approach.

5.2 Probabilistic Assessment

In the revised HHRA, EPA has added probability analyses, including an MCA and a PBA, to the agricultural products consumption assessment, which were not presented in the 2003 draft. The purpose of the MCA and PBA was to characterize the variability and, for the PBA, the uncertainty inherent in the deterministic approach (see Vol. V, p. 6-1). To do so, probability distributions of exposure variables replaced some of the point estimates used in the deterministic analyses. Tables associated with Section 6 (Vol. V) of the revised HHRA summarize the inputs used for the MCA and identify the types of distributions assumed for each in the assessment. Unexpectedly, for many (and for some of the scenarios, the majority) of the input variables, the HHRA continues to use point estimates as inputs to the MCA, rather than replacing them with distributions of values. Since the purpose of developing these alternative approaches was to quantify the effect of variability on the risk estimates, it is puzzling that point estimates were retained for so many of the inputs.

This approach is not necessary or warranted. As described in detail below, while site-specific data were not available on many of the inputs and in some cases only limited information exists in the published scientific literature, input distributions can be developed for some of the critical

exposure inputs based on available data. The significance of EPA's choice to use point estimates rather than distributions is clear when evaluated in the context of the Sensitivity Analysis, which is also contained in Section 6. For example, of the three variables that contributed most significantly to the uncertainty and variability in the backyard beef consumption model (soil-to-grass TFs, soil ingestion by farm animals, and human consumption rates), two are represented in the MCA by point estimates rather than distributions (Vol. V. p. 6-33). The benefits that should have been provided by developing the MCA for these exposure pathways are lost when the critical inputs are not changed from the deterministic approach.

Bioavailability of tPCBs from Soil

The HHRA uses a point estimate bioavailability factor of 100 percent in the MCA (Vol. V, p. 6-20). The rationale presented for using this approach is that insufficient data were available for tPCBs that would allow EPA to define the variability in this value or to provide a better estimate. The HHRA itself, however, provides substantial information about the variability associated with this important input to modeled exposures. In fact, it incorporates that variability into the PBA for agricultural products.

EPA's choice to assume that tPCBs in soil are 100 percent bioavailable has a substantial impact on the risk estimates for the agricultural product consumption pathways. This is because, as modeled, the soil ingestion pathway accounts for 55 percent of the risk estimated for the commercial beef consumption pathway, 32 percent of the risk estimated for the consumption of dairy products from backyard farms, and 36 percent of the risk estimated for the consumption of beef from backyard farms. In addition, it accounts for 100 percent of the tPCB intake by free-range poultry and, therefore, 100 percent of the risk estimated for the consumption of meat and eggs from those birds.

These pathways account for the highest cancer risks and noncancer hazards in the agricultural products consumption assessment. While data specific to the bioavailability of PCBs in soil ingested by farm animals are limited, there are data that can be used to develop distributions for the purpose of investigating the impact that EPA's arbitrary assumption of 100 percent has on the risk estimates.

For example, Ruby et al. (2002) reported that the bioaccessibility (a surrogate for oral bioavailability) of low concentrations of polychlorinated dioxins and furans ranged from 19% to 34%. Similar results were reported by Hack and Selenka (1996) for PCBs in a "standardized

gastro-intestinal model." The HHRA itself cites the long-term feeding study in chickens by Stephens et al. (1995) and concludes that "these findings suggest that aging of contaminants in soil may reduce bioavailability" (Vol. V, p. 4-32). Although that study investigated the behavior of dioxins and furans, the HHRA recognizes that these classes of compounds (i.e., persistent, organic, lipophilic compounds) behave similarly in the environment because they share important physical/chemical properties (Vol. V, p. 6-20). In addition, as shown in Table 4-8b of Volume V of the HHRA, EPA recognizes the reduced bioavailability of dioxin-like PCB congeners. That table reports that "predicted absorption" values for the dioxin-like PCB congeners range from 41 to 71 percent.

For the PBA, the HHRA acknowledges that bioavailability is not 100 percent, and in Section 6.5.3.4 explains its decision to set a range for bioavailability of PCBs from soil relative to feed. In fact, the HHRA provides information on a potential range of bioavailability factors that might be used to develop a distribution in the MCA. It is unclear why EPA did not use the information presented in Section 6.5.3.4, or the information included in Attachment I of GE's comments on the 2003 HHRA (AMEC and BBL, 2003), to develop a probability distribution for this important input factor. Since direct soil ingestion is a significant and, in some cases, the only modeled uptake mechanism for farm animals and subsequent human exposures by these pathways, EPA should revise its approach to include an input distribution for this important variable to demonstrate the impact of the variability in this parameter on final risk estimates for the agricultural product consumption pathways.

Soil-to-Grass Transfer Factor

The soil-to-grass transfer factor is also a critical input parameter in the probabilistic exposure assessment for the agricultural product consumption pathways. As shown in the HHRA, the soil-to-grass exposure pathway accounts for 44 percent of the risk estimated for the commercial beef consumption pathway, 68 percent of the risks in the backyard dairy farm scenario, and 64 percent of the risks in the backyard beef consumption scenario (Vol. V, pp. 5-4, 5-5, 5-6).

In the MCA, the HHRA uses a point estimate of 0.036 for this transfer factor (Vol. V, p. 6-19, Figure 6-13), despite information provided in the previous draft HHRA and the PBA discussion in this draft of the HHRA, which indicates that this transfer factor may span an order of magnitude or more. The PBA specifically describes a range from 0.0098 to 0.094 (Vol. V, Table 6-4). Thus, EPA's approach does not consider any of the variability associated with this important parameter.

As stated in GE's Comments on the 2003 HHRA (AMEC and BBL, 2003), the methodology used to obtain the site-specific soil-to-grass transfer factors likely overestimated the actual transfer that is occurring over time. In fact, in the revised HHRA, EPA acknowledges that the grass data "represent an upper bound on exposure concentrations of PCBs for grazing cattle" (Vol. V, p. 4-7). It goes on to state, in Section 7.2.2.1.1 (p. 7-5), that there is a range of literature-based values that span several orders of magnitude, indicating that there is enormous variability associated with this parameter.

There are substantial amounts of data available on soil-to-grass transfer. These include studies discussed by EPA in the HHRA, as well as other studies identified in GE's comments on the earlier draft (AMEC and BBL, 2003). In the 2003 version of the Uncertainty Analysis, EPA provided transfer factors obtained from ATSDR (Section 6.3.2.1) that ranged over two orders of magnitude. However, rather than incorporating the breadth of information available, EPA selected a soil-to-plant transfer factor at the high end of the range found in the scientific literature. Thus, instead of utilizing the advantages, and stated purpose, of the MCA to assess the effect of variability on risk estimates, EPA relied solely on the "upper bound" transfer factor to estimate PCB intake from grass, thereby minimizing the value of the MCA.

GE believes that EPA should revise its approach to incorporate a distribution of soil-to-grass transfer factors based on site-specific and literature-based values. This will allow the MCA to consider the impact of the enormous variability associated with this important parameter and provide more insight into the range of potential exposures and risks that are potentially associated with the exposure pathways that include this transfer route.

Soil-to-Plant Transfer Factor

As discussed in Section 5.1, EPA used the maximum wet weight soil-to-plant transfer factor for corn to estimate tPCBs in "exposed" surface vegetables and fruits. The point estimate TF that was used in the deterministic risk calculations was also used in the MCA (Vol. V, p. 6-16). This maximum value overestimates the amount of tPCBs on ingested vegetables and fruits for all the reasons previously discussed in Section 5.1.

While a "Produce Loss Factor" was used in both the MCA and PBA assessments to account for the removal of soil during the processing of fruits and vegetables for consumption, these assessments did not consider the additional impact that washing would have on the exposure

estimated using the TF. For example, the maximum loss for "exposed vegetables" was assumed to be 0.64 (Figure 6-51), which assumes that 36 percent of the PCBs transferred from soil to the surface of the vegetable, as quantified by the TF point estimate, were consumed. Likewise, for "exposed fruits," the maximum loss was assumed to be 0.41 (Figure 6-53); thus, it was assumed that the ingested PCBs were 59 percent of the total estimated by the TF. The minimum values of these distributions were both set at 0, indicating the potential for no loss of PCBs before consumption. It would seem appropriate in this assessment to evaluate the effect of both washing *and* preparation loss on exposure and risk estimates. While there is a potential that some fruits and vegetables will be consumed without washing, assuming that this never occurs is not a reasonably anticipated occurrence. Rather, including the effect that even periodic washing would have on PCB concentrations, and therefore extending the maximum ranges of the distributions, is consistent both with the purpose of the MCA and with EPA guidance for including high-end, but not worst-case, exposure assumptions (EPA, 1992b, 1995). The potential effects that these two activities have on the deposited PCB concentrations would range from very small impacts (very little removed from either washing or peeling and a loss factor approaching 0) to almost the complete elimination of this as an exposure route (and a loss factor approaching 100 percent).

Mammalian Bioconcentration Factors

For the MCA, the HHRA uses a point estimate of 3.4 to represent the mammalian BCF (Vol. V, p. 6-16), rather than incorporating an input distribution of values based on available data. Tuinstra et al. (1981) orally dosed lactating cows with "lower chlorinated biphenyls and technical grade PCB mixture Aroclor 1260" and reported accumulation factors into milk fat for individual congeners ranging from 0.1 to 5.4, with a mean of 2.22. Fries (1996) reported BCFs into milk fat for Aroclor 1254 ranging from 1.5 to 3.6.

As noted in the HHRA (p. 6-16), BCFs for exposures of less than 1 ppm in the diet exceeded 3.0. However, as discussed previously, unlike the June 2003 draft of the HHRA, where the maximum floodplain soil concentration evaluated was 2.0 ppm, the revised HHRA attempts to model milk and beef concentrations in animals exposed to soil concentrations as high as 25 ppm. Consequently, EPA's rationale for using the higher BCF, based on a dietary concentration range for test animals of <1 ppm PCBs, does not apply to the MCA. Rather, the available information used to derive the point estimate (Fries, 1996) should be considered, along with the data provided by Tuinstra et al. (1981), to develop an appropriate distribution for the mammalian BCF input variable for the MCA.

Steady-State Conditions

In addition to the above instances in which EPA has used point estimates rather than distributions in the MCA, the HHRA's probabilistic analyses fail to take adequate account of variability in animal tissue levels (meat, milk, and eggs) that may result from intermittent PCB exposures. One of the issues raised in GE's comments on the 2003 draft HHRA (AMEC and BBL, 2003) related to the enormous uncertainty associated with EPA's assumption that animals that had contact with PCBs in floodplain soils were at steady-state. In the revised Uncertainty Analysis, EPA acknowledges, at least qualitatively, that fluctuations in concentrations in the diet will result in fluctuations in milk concentrations (Vol. V, p. 7-9). This fluctuation is a result of the fact that non-steady-state conditions exist for lactating animals. In its discussion, EPA concludes that the alterations in milk concentrations that would result from physiological changes would be less than the changes that would result from the variability in contaminant concentrations in feed (Vol. V, p. 7-9). That is not necessarily true.

In an important study cited by EPA for determining milk PCB concentrations, Thomas et al. (1999) reported that under constant exposure conditions, even while PCB intakes increased (because of increased silage consumption), milk concentrations dropped by an average of 25%. It is likely that removal of lactating animals from access to feed containing PCBs (either grass, silage, or soil) for even short periods of time would result in a significant reduction in milk concentrations. Thus, the changes in milk concentrations due to physiological changes may be less than, equal to, or greater than those attributed to fluctuations in concentrations of PCBs in feed. Because of the variability in exposure conditions, a general conclusion about the quantitative impact of this effect on human exposure cannot be reached.

This does not, however, preclude consideration of this variability in the quantitative estimate of exposure and risk associated with the dairy consumption pathways. GE recommends that EPA use a non-steady-state model (e.g., a pharmacokinetic model) in the MCA to address this important source of uncertainty. Models that evaluate intermittent exposures have been used in risk assessment, including the microexposure event simulation for fish consumption in the Housatonic River HHRA (Vol. IV. Section 6.3). Adopting the principles of the microexposure event analysis, and incorporating a consideration of the pharmacokinetics of PCBs in agricultural animals, which can be gleaned from the Thomas et al. (1999) report, would provide a quantitative method for evaluating the impact that this variability has on animal product PCB concentrations, and consequently risks to human consumers.

6.0 INTEGRATED RISK CHARACTERIZATION

EPA has added an integrated risk characterization in Volume 1, Chapter 10 of the HHRA, in response to comments raised by the peer reviewers. This section includes a perspective on the TEQ exposures that are estimated for several of the exposure scenarios and also discusses potential concentrations of PCBs in breast milk. GE believes that these discussions need to be revised to discuss additional uncertainties that potentially affect the conclusions drawn.

6.1 Perspective on TEQ Exposures

In Section 10.2 of Volume I, EPA presents a "Perspective on TEQ Exposure," which compares the exposure levels modeled in the HHRA with background intake levels in the current American food supply that are provided in the published literature. The purpose of this section is to advise the public whether the substitution of agricultural products or fish and waterfowl obtained from the study area would increase intake of TEQ by Housatonic River Area (HRA) residents over TEQ intake that would occur from similar products obtained from national chain grocery stores.

The HHRA compares the TEQ concentrations predicted for milk, beef, and poultry products, based on a 2 ppm soil concentration, with measurements of TEQ in these products as found in the national food supply. It concludes that TEQ concentrations in milk obtained from commercial dairies in the HRA are similar to the national supply, but that milk from backyard farms and beef and poultry from both commercial and backyard farms in the HRA have greater TEQ concentrations than does the national food supply.

In discussing these comparisons, it is important that EPA express the degree of uncertainty associated with the comparisons. For example, Table 10-8 of the HHRA indicates that the PCB-related TEQ concentration predicted to be present in the fat of backyard-raised beef in the HRA is 171 times higher than the PCB TEQ concentration in beef fat in the national food supply. However, as discussed in these comments, the analyses of backyard beef are likely to substantially overestimate concentrations in beef fat due to a combination of highly conservative transfer and bioconcentration factors. In addition, even without the use of input distributions for many of the parameters in the MCA, the predicted risks, and hence exposures, in the MCA range over nearly two orders of magnitude at the 2 ppm soil concentration (see Vol. V, Table 6-15), so that the background level reported by EPA might very well fall within the range of predicted beef fat concentrations. Furthermore, if the MCA had used input distributions for many of the parameters for which it used point estimate values, the range of potential risks

would likely have been even greater. Thus, the HHRA's comparison of its hypothetical and highly conservative exposure estimates for the agricultural pathways to national TEQ data should be qualified to account for the enormous uncertainties associated with the HHRA's predictions.

Finally, the HHRA discusses these agricultural exposures as if they are actually occurring. There are enormous uncertainties, however, associated both with the likelihood of occurrence of the modeled scenarios and with the exposure estimates derived using EPA's approach to the agricultural risk assessment. EPA should revise this section to provide more discussion of the potential uncertainties associated with the predictions, and should note that exposures to agricultural products grown or raised in the HRA may or may not have higher levels of TEQ than are found in the background food supply.

6.2 Breast Milk Pathway

In response to the comments of the peer reviewers, EPA has added an evaluation of the breast milk pathway by predicting estimated PCB and congener concentrations in breast milk and then comparing those estimated concentrations with available data about background concentrations of these compounds in breast milk (Vol. I, Sec. 10.3). EPA has calculated these concentrations using simplistic models, based on the dose levels estimated in the HHRA for adults in the fish consumption, waterfowl consumption, and backyard dairy farm milk ingestion scenarios. The HHRA acknowledges that there are uncertainties associated with the calculation of the breast milk concentrations, particularly as related to the range of half-life estimates available in the published literature (Vol. I, Section 10.3.1.2). It does not, however, adequately address either: (a) the additional uncertainties associated with the use of the simplistic model, which may or may not represent the actual mechanism of the concentration of PCBs into breast milk; or (b) the substantial uncertainties associated with deriving the estimated dose levels for the fish consumption, waterfowl consumption, and backyard dairy milk ingestion scenarios, which are discussed at some length in these comments and in GE's previous comments (AMEC and BBL, 2003; GE, 2003). GE recommends that EPA add a subsection to Section 10.3 of the HHRA that specifically discusses all of the potential sources of uncertainty associated with the breast milk concentration estimates that have been derived.

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Attachment A

Letter from Kevin W. Holtzclaw to Dr. David L. Eaton Re: Comments of the General Electric Company on EPA's Draft Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds

March 15, 2005

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RE: Comments of The General Electric Company on EPA's Draft Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds

Dear Dr. Eaton:

The General Electric Company ("GE") appreciates the opportunity to present to the National Academy of Science's Committee ("NAS Panel") on EPA's Exposure and Human Health Reassessment of TCDD and Related Compounds ("draft Reassessment") its comments and research concerning a fundamental flaw in the draft Reassessment: the belief that certain PCB congeners are the toxic equivalents of dioxin. This belief is contradicted by a substantial body of evidence demonstrating that the internationally recognized criteria for application of the TEQ approach are not met for any of the PCBs. Remarkably, the draft Reassessment does not even mention the vast majority of this evidence. If EPA had considered this evidence, it would have had no choice but to conclude that PCBs are not the toxic equivalents of dioxin and should not be included in the TEQ risk assessment methodology.

This fundamental flaw in the draft Reassessment is important because the application of the TEQ approach to so-called "dioxin-like" PCBs results in a significant overestimate of the risks to human health from exposure to the compounds in question. In the draft Reassessment, EPA calculates that the "dioxin-like" PCBs contribute up to one-third of a typical person's Total Daily Intake of TEQ. Using the TEQ approach, EPA treats this intake as if it is dioxin itself. The net result is that EPA concludes that humans are exposed to more "dioxin", in the form of TEQ, than they are exposed to dioxin per se. Moreover, EPA concludes that the risks arising from PCB exposure, calculated as TEQ, are substantially greater than

they would be if the risks from exposure to PCBs were calculated using the available empirical, PCB-specific data.

As many of the members of the NAS panel are undoubtedly aware, there is a vast literature on the toxicity of PCBs and this class of compounds has been closely regulated by EPA using risk-based standards. Both Reference Doses ("RfDs") and Cancer Slope Factors ("CSFs") have been established by EPA for PCBs and are available on EPA's Integrated Risk Information System ("IRIS") database. The current CSFs for PCBs were established by EPA in 1996. In that year, EPA performed a comprehensive reassessment of the carcinogenicity of PCBs and, based on animal data, established an upper bound CSF for PCB exposure to the higher chlorinated PCB congeners of 2.0 $(mg/kg\text{-}day)^{-1}$ EPA, 1996 (PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures) ("1996 PCB Reassessment"). Risks of exposures to PCBs likely to involve lesser chlorinated PCBs were to be assessed using lower CSFs. Id. Applying the TEQ methodology to PCBs, as urged by the draft Reassessment, would have the effect of increasing the estimated PCB cancer risk by up to 30 times over the upper bound CSF of 2.0 $(mg/kg\text{-}day)^{-1}$. This increase in estimated risk does not result from any error in, or updating of, the 1996 PCB Reassessment -- indeed, the draft Dioxin Reassessment does not even mention the 1996 PCB Reassessment. Instead, the increase in estimated risk is based on a surrogate risk metric, i.e., the CSF for 2,3,7,8-tetrachlorodibenzo-p-dioxin ("TCDD") of 1.4×10^6 $(mg\ TCDD\ TEQ/kg/day)^{-1}$.

The remainder of this letter summarizes the evidence that the NAS panel should consider when determining whether PCBs should be evaluated using the TEQ approach and included in the dioxin reassessment. Some of that evidence is contained in or referenced by Attachments to this letter. Additional evidence has been submitted for publication, and we appreciate the opportunity to provide a summary of this evidence in a presentation to the NAS Panel at the Panel's March 21, 2005 meeting.

Evidence that the TEQ Approach Substantially Over-Predicts the Carcinogenicity of PCBs

EPA's error in applying the TEQ approach to PCBs results largely from the Agency's failure to validate the risk predictions of the TEQ approach for PCBs by comparing them with empirical data on the effects of PCB exposures in animal studies. A basic premise of the TEQ approach is that a given dose of TEQ has equal biological potency irrespective of the chemical mixture from whence it came (van den Berg, et al., 1998). There are at least two ways to test the validity of this premise in the case of PCBs. The first is to calculate CSFs for PCB-derived TEQ in rodents, and to

compare those CSFs to the CSFs derived from rodents exposed to dioxin. The second is to calculate the cancer potency of PCB mixtures using TEQs, and to compare the calculated CSFs to CSFs derived in the 1996 PCB Reassessment for use in assessing human health risks.

Comparison of rodent CSFs

To evaluate the validity of the TEQ methodology in estimating the cancer potency of PCB mixtures, Dr. Russell Keenan and co-workers used the results of two-year cancer bioassays involving four PCB mixtures of known composition that were fed to Sprague-Dawley rats. Those tests are described and their results presented in a journal article that has been submitted for publication and in a short paper that appears in the Dioxin 2003 conference proceedings (Attachment 1a).[1]

In one test, CSFs in rats were determined for the "dioxin-like" components of the four PCB mixtures and compared to that of TCDD (based on a similar two-year cancer bioassay of TCDD in rats). This was done by analyzing the four PCB mixtures to determine their concentrations of dioxin-like congeners and then, for each mixture, determining the total TEQs of the mixture by summing the products of the concentrations of dioxin-like congeners and the TEFs for those congeners, as set forth in the draft Reassessment. The empirically-determined CSF for each mixture was then divided by the TEQ of the mixture to derive the CSF per unit TEQ for the mixture. If the TEQ approach is valid, the CSF per unit of TEQ for each of the PCB mixtures should equal the TEQ of TCDD, i.e., the tests should give equivalent results.

The results of this "litmus test" do not support the TEQ approach. The CSFs for the TEQ in PCB mixtures were not equal to the CSF for TCDD; in fact, the experimentally-determined CSFs for the PCB mixtures based on TEQ varied over a 24-fold range. This discordance demonstrates that the TEQ approach for evaluating cancer risks associated with exposure to PCB mixtures is seriously flawed.

Comparison of human CSFs

In a second test, the human CSFs for three PCB mixtures were determined using the TEQ methodology and compared to the empirically derived CSFs for those mixtures, as cited in the 1996 PCB Reassessment. If the TEQ method were an accurate predictor of the potency of the dioxin-like PCBs in a PCB mixture, then one

[1] Note that similar papers, which are contained Attachments 1b, were presented to the EPA Science Advisory Board in connection with that group's review of the Dioxin Reassessment in 200o and 2001. The current version of the draft Reassessment does not reference this work.

would expect the CSFs determined through the TEQ method to be consistent with the empirically derived CSFs. In fact, the comparisons showed that the TEQ-based CSFs were considerably greater than the empirically-derived CSFs, indicating that the TEQ approach substantially over-predicts the carcinogenic potency of PCB mixtures.

In each case, Dr. Keenan's findings are in sharp contrast to the results that one would expect if the fundamental premise of the TEQ method were true. Each of these analyses indicates that there is a fundamental fallacy associated with the use of the TEQ approach for estimating the carcinogenic potential of PCB mixtures.

Additional evidence from the National Toxicology Program study

The inability of the TEQ method to predict the carcinogenicity of PCB mixtures is confirmed by the results of the recent 2-year bioassays of the National Toxicology Program ("NTP") on TCDD, PCB 126, 2,3,4,7,8-pentachlorodibenzofuran, and a mixture of these three compounds (NTP, 2003), followed by bioassays of PCB 153, a mixture of PCB 153 and PCB 126, and a mixture of PCB 118 and PCB 126 (NTP, 2004). NTP conducted this series of bioassays in female Harlan Sprague-Dawley rats to evaluate the chronic toxicity and carcinogenicity of dioxin, "dioxin-like" compounds, structurally-similar PCBs, and mixtures of these compounds. NTP conducted its evaluation to address "the lack of data on the adequacy of the TEQ methodology for predicting relative potency for cancer risk" (NTP, 2003).

The initial NTP (2003) bioassay results provide evidence of non-additive interactions among "dioxin-like" compounds, inconsistencies in dose-response depending on the dose metric analyzed, and different relative potencies depending on endpoint observed. These results undermine the assumptions essential to the application of the TEQ approach to PCBs. We are preparing two papers for publication, which explore these issues in greater depth and would be pleased to submit these manuscripts to the panel upon their acceptance. A brief summary of our findings will be presented to the Panel on March 21.

Evidence from human studies

Although it is clear that the TEQ approach substantially over-predicts the animal carcinogenicity of PCBs, it should also be noted that the predictions of the TEQ approach for human carcinogenicity are wholly unsupported by available human data. The TEQ approach as set forth in the draft Reassessment ignores the vast body of PCB human epidemiological studies indicating that PCBs are very likely not human carcinogens at all. More than 50 peer-reviewed, epidemiological cancer

studies specific to PCBs have been published over the past 30 years. Many of those studies involved thousands of workers with occupational exposures far greater than those that would result from environmental exposures. None of those studies support a finding that PCBs are human carcinogens. One study, Kimbrough et al. (1999), as updated by Kimbrough et al. (2003), is particularly noteworthy.

Kimbrough et al. (1999) (Attachment 2) represents one of the largest occupational studies ever conducted of a population of workers that was heavily exposed to PCBs. The cohort consisted of 4,062 men and 3,013 women who worked between 1946 and 1977 at two General Electric capacitor manufacturing facilities. Jobs at the two facilities were classified as high or low exposure. The average follow-up time for the workers was 31 years, providing the longest latency period of any PCB-exposure occupational study. The cohort was followed through 1993, providing 120,811 person-years of observation for men and 92,032 person-years observation for women. There were 763 (19%) deceased males and 432 (14%) deceased females. Kimbrough et al. (1999) found that, compared to the general U.S. population, among all workers, including those classified as having the highest PCB exposure, there was no statistically significant increase in deaths due to cancer or any other disease. Moreover, the death rate due to all types of cancer combined was at or below the expected level.

The Kimbrough et al. (2003) (Attachment 3) study followed the cohort through 1998, providing 133,845 person-years of observation for men and 102,139 person-years observation for women. There were 1022 (25%) deceased males and 632 (20%) deceased females. The Kimbrough et al. (2003) update similarly found that, among all workers, including those classified as having the highest PCB exposure, there were no statistically significant increases in deaths due to cancer. There were also no statistically significant increases in cancer or other mortality associated with length of employment or latency.

Golden et al. (2003), a summary paper that discusses the findings of Kimbrough et al., as well as all of the other human evidence relating to the potential carcinogenicity of PCBs, is included in Attachment 4.[2] Golden et al. (2003) concluded that "[a]pplying a weight-of-evidence evaluation to the PCB epidemiological studies can only lead to the conclusion that there is no causal relationship between PCB exposure and any form of cancer . . .." A more detailed review of all the relevant human cancer studies involving exposure to PCBs is included on a compact disk contained in Attachment 6. That review also concluded

[2] Attachment 4 also includes a letter to the editer in reference to the Golden et al. paper and the authors' response to that letter.

that the weight of the human evidence does not support an association, much less a causal relation, between PCB exposure and any type of cancer. All of this information leads inexorably to the conclusion that the TEQ approach, rather than providing a method for more accurate assessment of cancer risk posed by dioxin and so-called dioxin-like PCB congeners, instead would lead to human health risk assessments that unjustifiably exaggerate risk and lead to misallocation of societal resources.

Reasons that the TEQ Approach Does Not Accurately Predict the Human Carcinogenicity of PCBs

Recent studies have investigated why the TEQ approach, as well as animal bioassays, do not accurately predict the human carcinogenicity of PCBs. These studies have thrown considerable light on the differing sensitivities of rodents and humans to PCB exposure. At the March 21st meeting, Dr. Jay Silkworth will present to the Panel data from new studies that show that human liver cells respond differently to both PCB and TCDD than do rat cells. Human cells require higher doses to elicit a response, and the potency of the most potent "dioxin-like" PCB congener (PCB 126) relative to dioxin in human cells is much less than the currently assigned TEF value of 0.1, which is heavily based on data from rodent liver cells. In addition, Dr. Silkworth will present data, based on genomic studies, showing that dioxin elicits responses distinct from PCBs, contrary to the concept of toxic equivalency.

There is No Need to Apply the TEQ Approach to PCBs

It is clear that the TEQ approach is less accurate in predicting the human health risks of PCBs than EPA's traditional methods (RfDs and CSFs) based on empirical, PCB-specific data. We also believe that the justification that EPA has offered for application of the TEQ approach to PCBs is faulty. EPA has suggested that application of the TEQ approach to PCBs is justified as a means of ensuring that risks resulting from PCB congeners that preferentially bioaccumulate in fish tissue are not underestimated. The theory behind this suggestion is the idea that perhaps certain more toxic congeners might accumulate to a greater degree than other less toxic congeners found in the original mixtures, thus enriching the toxicity of the mixture beyond that of the original test material. Hence, according to this theory, the PCB CSF that is based on the original test mixtures might not be protective of potential risks posed by the altered mixture of congeners. This theory would have no validity, however, if the TEQs of environmental mixtures are no greater than the TEQ of the PCB test mixtures upon which EPA's PCB CSF of 2 $(mg/kg\text{-}day)^{-1}$ is based.

This, in fact, is the situation for fish collected from a number of data sets that we have examined. This analysis is described and the results are presented in a journal article that is being submitted for publication. A copy will be provided to the panel upon acceptance by the journal and is summarized below.

EPA's CSFs for PCBs are based on bioassay data from studies of Aroclors 1254, 1242, 1260 and 1016 (EPA, 1996; Cogliano, 1998). According to Cogliano (1998), the TEQ concentration from coplanar PCBs in the Aroclor 1254 mixture used in the bioassays was 46.4 mg TEQ/kg PCB. Similarly, the PCB TEQ in the tested Aroclor 1242, 1260, and 1016 mixtures were lower at 8.1, 7.1, and 0.14 mg TEQ/kg PCB, respectively (Cogliano, 1998). The PCB CSF of 2 $(mg/kg\text{-}day)^{-1}$ is protective for the Aroclor with the greatest TEQ (i.e., Aroclor 1254) and, therefore, is protective for all PCB mixtures of equal or lesser TEQ. It follows, therefore, that the PCB CSF is protective of any exposure to an environmental PCB mixture that has a total TEQ of 46.4 mg TEQ/kg PCB or less.

For numerous fish and soil samples taken from fourteen water bodies noted for their PCB contamination,[3] total PCB concentrations were determined as the sum of all PCB congeners, using one-half the detection limit for non-detected congeners. The total TEQ for each sample was determined as the sum of the data for each coplanar congener (using one-half the detection limit for non-detected congeners) times its respective WHO TEF. Our analysis reveals that these samples from waterbodies noted for their PCB contamination have mean TEQ levels that are statistically significantly lower than 46.4 mgTEQ/kg PCB – the level of TEQ found in the test material upon which EPA's CSF for PCBs is based. Consequently, the use of the PCB CSF developed by EPA in 1996 to evaluate potential cancer risks is more than adequately protective of the carcinogenic potential of the PCB mixtures found in these fish tissues. There is no need to use the TEQ approach to ensure that risks are not underestimated.

* * *

Finally, we would like to call the Panel's attention to two additional issues.

- The Panel should be aware that the use of the TEQ approach for evaluating PCB cancer risks will lead to the conclusion that unacceptable risks exist when, in fact, the PCB congeners of concern are not even present in a sample. Under the TEQ approach, PCB

[3] The Delaware, Hudson, Housatonic, Fox, Kalamazoo, Sheboygan, Spokane, and Christiana Rivers; San Francisco Bay, Newark Bay, Green Bay, and Saginaw Bay; the South California Bight, the Great Lakes, Long Lake (WA) and Dick's Creek (OH).

126 (3,3',4,4',5-pentachlorobiphenyl) is assigned a TEF of 0.1 relative to that of TCDD, designating it as the most potent of the so-called "dioxin-like" PCB congeners. Due to its elevated TEF, PCB-126 may contribute substantially to projected risk estimates, despite the fact that it is usually a minor constituent of the "dioxin-like" PCBs found in environmental residues. Because EPA risk assessment practice calls for the assumption that an undetected chemical is carried through the risk assessment as if it were present at a concentration equal to one-half of its analytical method detection limit, the probability is that undetectable residues of PCB-126 and other PCB congeners will result in elevated risk estimates, even though they are not detected in actual samples. In particular, it appears that the use of the TEQ approach will result in the conclusion that any fish sample, collected at any location, will pose human health cancer risks that exceed EPA's risk benchmark of $1x10^{-4}$, due to the assumed presence of PCB-126, regardless of whether this congener is actually present in the sample. A paper discussing this matter is being prepared for publication and will be submitted to the Panel if possible.-

- The NTP (2003; 2004) bioassays also provide data to illustrate that EPA's (2003) draft Dioxin Reassessment has inappropriately commingled the use of a TEQ approach for PCBs based on administered dose with a CSF for dioxin based on a body burden dose metric. In EPA's draft Dioxin Reassessment, the proposed CSF for TCDD is based on a body burden dose metric. EPA then proceeds to relate the other dioxin-like congeners, including the "dioxin-like" PCBs, to TCDD using TEFs based on administered dose studies. However, as shown in Attachment 5,[4] the combined use of a CSF for dioxin based on body burden with a TEQ approach based on administered dose is incorrect and will serve to artificially magnify the estimated risks. In our analysis based on body burden, the relative cancer potencies of PCB 126, 4-PeCDF, and the TEQ mixture were much lower than predicted using the current WHO TEF scheme. This is not surprising because the TEF scheme was developed based on administered dose comparisons (Van den Berg et al., 1998), and the pharmacokinetics and distribution patterns for other TEQ-contributing compounds are substantially different from those of TCDD (DeVito et al., 1998). This analysis suggests that for carcinogenesis, the WHO TEF values substantially overpredict the cancer potency of 4-PeCDF and PCB 126 on a body burden basis. The current TEF values are based on intake-based assessments and should not be relied upon for assessments of cancer risk on a body burden basis.

* * *

In conclusion, it is of the utmost importance for the Panel to take the time needed to ensure that the risks of PCBs are accurately calculated. Otherwise, we are likely to see a significant misallocation of limited societal resources to address unfounded concerns regarding the safety of the food supply and perceived risks arising from

[4] Attachment 5 has been extracted from a paper that has been submitted for publication.

contaminated sites. Indeed, we believe that prevention of misallocation of resources was one of the principal reasons that Congress asked for this NAS review.

We appreciate your consideration of these lines of evidence that show that the dioxin TEQ approach should not be used to assess the cancer risks of PCB mixtures, and that PCBs should not be included in the dioxin reassessment. We look forward to the opportunity to present our research and to address your questions.

Sincerely,

Kevin W. Holtzclaw

Manager, PCB Issues

The General Electric Company

Attachments

Attachment 1a: Keenan, R., J.Hamblen, J.Silkworth, M.Gray, P.Gwinn, S. Hamilton. 2003. An Empirical Evaluation of the Potency of Dioxin Toxic Equivalents (TEQs) in Several PCB Mixtures. Organohalogen Compounds, 65: 312-315. Proceedings Dioxin 2003 – the 23rd International Symposium on Halogenated Environmental Organic Pollutants and Persistent Organic Pollutants, Boston, Massachusetts, USA. August 24-29.

Attachment 1b: Papers presented to the EPA Science advisory Board in connection with their review of the draft Dioxin Reassessment in 2000-2001.

Attachment 2: Kimbrough, R., M.Doemland, M.LeVois. 1999. Mortality in Male and Female Capacitor Workers Exposed to Polychlorinated Biphenyls. J. Occup. Environ. Med. 41:161-171

Attachment 3: Kimbrough, R., M.Doemland, J.Mandel. 2003. A Mortality Update of Male and Female Capacitor Workers Exposed to Polychlorinated Biphenyls. J. Occup. Environ. Med. 45:271-282.

Attachment 4: Golden,R., J.Doull, W. Waddell, J.Mandel. 2003. Potential Human Cancer Risks from Exposure to PCBs: A Tale of Two Evaluations. Critical Reviews in Toxicology, 33(5):543–580.

Attachment 5: Body Burden Based on Cancer Potencies of Selected Dioxin-like Compounds Are Lower Than Predicted by the Toxic Equivalency (TEQ) Approach (summary of the results of a manuscript that is currently in submission (Gray et al., in submission).

Attachment 6: Compact Disk containing a detailed review of all of the human cancer studies.

References

Cogliano, V.J. 1998. Assessing the cancer risk from environmental PCBs. *Environmental Health Perspectives*. 106 (6): 317-323.

Keenan, R.E., J.M. Hamblen, J.B. Silkworth, M.N. Gray, P.O. Gwinn, and S.B. Hamilton. 2003. An empirical evaluation of the potency of dioxin toxic equivalents (TEQs) in several PCB mixtures. *Organohalogen Compounds* 65: 312-315. *Proceedings Dioxin 2003 – the 23rd International Symposium on Halogenated Environmental Organic Pollutants and Persistent Organic Pollutants,* Boston, Massachusetts, USA. August 24-29.

National Toxicology Program (NTP). 2004a. *DRAFT NTP Technical Report on the Toxicology and Carcinogenesis Studies of 2,3,4,7,8-Pentachlorodibenzofuran (PeCDF) (CAS No. 57117-31-4) in Female Harlan Sprague-Dawley Rats (Gavage Study) (NTP TR 525)*, National Toxicology Program.

National Toxicology Program (NTP). 2004b. *DRAFT NTP Technical Report on the Toxicology and Carcinogenesis Studies of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) (CAS No. 1746-01-6) in Female Harlan Sprague-Dawley Rats (Gavage Study) (NTP TR 521)*, National Toxicology Program.

National Toxicology Program (NTP). 2004c. *DRAFT NTP Technical Report on the Toxicology and Carcinogenesis Studies of 3,3'4,4',5-Pentachlorobiphenyl (PCB 126) (CAS No. 57465-28-8) in Female Harlan Sprague-Dawley Rats (Gavage Study) (NTP TR 520)*, National Toxicology Program.

National Toxicology Program (NTP). 2004d. *DRAFT NTP Technical Report on the Toxicology and Carcinogenesis Studies of a Mixture of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) (CAS No. 1746.01-6), 2,3,4,7,8-Pentachlorodibenzofuran (PeCDF) (CAS No. 57117-31-4), and 3,3'4,4',5-Pentachlorobiphenyl (PCB 126) (CAS No. 57465-28-8) in Female Harlan Sprague-Dawley Rats (Gavage Studies) (NTP TR 526)*, National Toxicology Program.