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August 31, 1998

Douglas Tomchuk
USEPA - Region 2
290 Broadway
20th Floor
New York, NY 10007-1866

Re: HHRA SOW Comments

Dear Mr. Tomchuk:

Enclosed please find General Electric Company's ("GE") comments on the "Hudson River PCBs Reassessment RI/FS Phase 2 Human Health Risk Assessment Scope of Work." These comments provide a detailed critique of the Scope of Work, and I will not repeat that discussion here. One specific issue, however, deserves emphasis.

One of GE's primary concerns is the Scope of Work's vague, muddled, and at times inconsistent description of the Agency's proposed baseline human health risk assessment. This has increased the difficulty of assessing and commenting on the Scope of Work. Consequently, in addition to pointing out the portions of the Scope of Work with which GE agrees or disagrees, GE's comments provide specific recommendations on how the Agency should conduct the baseline risk assessment. GE also urges the Agency to reissue the Scope of Work to provide a more coherent description of the risk assessment and to respond to the issues raised in GE's comments.

GE welcomes the opportunity to discuss its comments with EPA in greater detail.

Sincerely,

Thomas G. Echikson

cc: Richard Caspe
William McCabe
Melvin Hauptman
Douglas Fischer, Esq.
Marianne Olson
John Cahill
Frank Bifera, Esq.
Walter Demick

COMMENTS OF GENERAL ELECTRIC COMPANY
ON

Hudson River Reassessment RI/FS
Phase 2 Human Health Risk Assessment
Scope of Work
July 1998

August 31, 1998

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FIGURE

Figure 1. Reprint of Figure 9-12 from the EPA Preliminary Model Calibration Report

EXECUTIVE SUMMARY

EPA's Scope of Work for the Human Health Risk Assessment ("SOW") for the Hudson River PCBs Superfund Site ("Site") sets out the Agency's proposed approach for conducting and preparing the baseline human health risk assessment ("HHRA") for the Site. The SOW proposes two risk assessments – one for the Upper Hudson and one for the Mid Hudson – using deterministic and probabilistic methods and standard, IRIS-derived toxicity values. This information will then be used to present a hypothetical statement of the risks associated with the Site against which the risk reduction achieved by various remedial alternatives can be measured.

There is much in the SOW that General Electric Company ("GE") supports. For instance, GE agrees with the Agency's proposal to use Monte Carlo modeling to develop a probabilistic risk analysis capturing both variability and uncertainty associated with the risk estimate. GE also agrees with the Agency's assessment that the primary route of exposure to PCBs is through fish consumption and that other possible exposure routes have little bearing on potential risks.

Despite GE's agreement with much of the general approach described in the SOW, GE has a number of concerns about the Agency's proposal. Many portions of the SOW are vague, internally inconsistent and simply fail to provide enough information to permit one to ascertain the Agency's proposed approach. From the information available, it appears that the Agency intends, in many instances, to use unrealistically high exposure assumptions. Further, the Agency apparently intends to ignore the vast body of epidemiological data that do not show that PCBs cause cancer in humans, instead relying entirely on the uncertain extrapolations derived from laboratory studies of animals. GE's comments point out these and other problems, along with recommendations on how the Agency should conduct the HHRA, convey the risk estimates to the public, and use the results in its remedial decision-making.

Beyond the specific concerns summarized below, there are some general points worth emphasizing. First, because the Site does not extend below the Federal Dam at Troy and in light of the numerous sources of PCBs to the lower River and lower River fish, EPA cannot reasonably rely on the results of the Mid Hudson risk assessment to justify remedial actions in the Upper Hudson. Second, the Agency must emphasize that its risk estimates are hypothetical. The existing catch-and-release requirements in the Upper Hudson, and the consumption advisories in the lower River all significantly limit fish consumption now and into the foreseeable future. If the Agency does not take these facts into account in the baseline HHRA, it must recognize and clearly state that its estimates of risk are completely hypothetical.

1. Exposure issues

- The SOW's method for selecting point estimates for incorporation into the exposure models is vaguely described and implies that the Agency intends to use unrealistic, worst case exposure assumptions. EPA must use more realistic, site-specific and relevant assumptions.

- The SOW's explanation of the Monte Carlo modeling does not clearly explain whether and how the Agency intends to model uncertainty and variability separately. Doing so is necessary to understand the estimated range of exposures as well as the uncertainty in those estimates. EPA should adopt and incorporate the approach set out in GE's previous submissions to the Agency.

- At a minimum, the baseline fish consumption rates must account for consumption limitations posed by factors other than PCBs, including the statewide consumption advisories and the conservation-based fishing restrictions imposed by New York State and the Atlantic Marine States Fisheries Commission. Furthermore, given the lack of good consumption rate data from the Hudson or other comparable New York waters, the best source for recreational angler consumption rates is the assessment of such rates in Maine contained in Ebert, et al. (1993).

- EPA should not consider hypothetical subpopulations of "subsistence" anglers. The available information demonstrates that income level, ethnic background, and commercial and recreational angler status are not relevant for deriving a subpopulation of highly-exposed Hudson River anglers.

- The SOW's proposed approach for assessing species-specific consumption should be replaced with the approach set out in ChemRisk (1995), which provides the appropriate input parameters for the microexposure Monte Carlo analysis.

- EPA should use a mechanistic, time-variable bioaccumulation model to compute average future PCB concentrations in fish. Variability in fish PCB levels should be estimated directly from the NYSDEC database. In addition, for the Mid Hudson, EPA must account for sources of PCBs other than the Upper Hudson.

- GE agrees with the proposal to account for cooking loss of PCBs, which, contrary to the implication of the SOW, is well-supported in the peer-reviewed literature.

2. Toxicity issues

- GE supports the proposal to use Aroclor-based toxicity criteria in light of the more reliable and complete toxicological, epidemiological and analytical databases for Aroclors. Other methods, including congener-based analysis, have critical scientific rely on inconsistencies and inappropriate assumptions.

- The Agency's reassessment of the cancer slope response of PCBs, which is based entirely on rat feeding studies is an important advance. In light of the difficulties and uncertainties associated with extrapolation from rats to humans, GE believes that it is feasible and more appropriate to use a cancer slope factor which takes into account the vast epidemiological database which does not support the proposition that PCBs cause

cancer in humans. GE 's comments present a method for deriving a human-based cancer slope factor that should be used in the HHRA.

- The IRIS-derived reference dose ("RfD") cited in the SOW is also flawed and overly conservative. It is based on a single study of rhesus monkeys and incorporates numerous uncertainty factors when extrapolating to humans. GE's comments present a more defensible and realistic RfD that EPA should use in the HHRA.

- EPA should not proceed with the SOW's proposal to present a qualitative assessment of endocrine disruption in light of the lack of evidence that PCBs have any effects on the human endocrine system.

- EPA should incorporate an analysis of the uncertainty associated with the PCB toxicological criteria incorporated into the risk assessment, just as it intends to do with the exposure assumptions.

3. Risk characterization issues

- Because one goal of the risk management is to determine how exposures for a particular source relate to background, the HHRA must recognize the background levels of PCBs in all individuals.

- The assessment of risks to the "reasonably maximally exposed individual" and the average individual should be based only on the findings of the probabilistic analysis, which is much more powerful than the proposed deterministic analysis. EPA should abandon the proposed deterministic analysis, which would lead to overly conservative risk estimates.

- The baseline HHRA cannot and should not be used to select a remedial decision. The results of the baseline HHRA can only be used to assess the hypothetical risks associated with the "no-action" alternative and, in the context of remedial decision-

making, must be measured against the risks estimated to result after implementation of different remedial options.

SECTION I
INTRODUCTION

General Electric Company ("GE") is pleased to submit these comments on the July 1998 "Hudson River PCBs Reassessment RI/FS Phase 2 Human Health Risk Assessment Scope of Work" ("SOW"). GE supports many aspects of the SOW. For example, GE generally supports the use of Monte Carlo modeling to assess exposure in the Human Health Risk Assessment ("HHRA"). GE also agrees with the Agency's conclusion that the greatest risk of exposure to PCBs in the Hudson River is likely to result from fish consumption and that other exposure routes are not significant.

Nevertheless, the SOW is inadequate in several respects. The SOW fails to explain in sufficient detail the methodology EPA intends to use to complete many of the identified tasks. The SOW also contains significant gaps and inconsistencies, as well as confusing statements and terminology. The SOW provides only a vague description of the relationship between the exposure assessment and the Agency's effort to model future levels of PCBs in fish and the Agency's intended use of Monte Carlo modeling. The SOW proposes to rely entirely on toxicity estimates based on animal studies, ignoring the extensive data from epidemiological studies that do not show that PCBs cause cancer in humans. GE's comments focus on these problems and provide recommendations on how EPA should complete the human health risk assessment ("HHRA") for the Site.

SECTION II
GENERAL COMMENTS

Several broad issues raised by the SOW deserve comment.

A. The site does not extend below the Troy Dam.

First, as GE has previously raised with EPA,[1] the Company disagrees with the Agency's description of the Site as including all 200 miles of the Hudson River between Hudson Falls and the Battery. The documents in the administrative record for the addition of the Site to the CERCLA National Priorities List explicitly limit the reach of the Site to the area above the Federal Dam at Troy, and EPA's post-rulemaking comments to the contrary cannot change this fact. GE's disagreement with EPA on the scope of the Site is particularly important in the context of the HHRA, in light of EPA's proposal to conduct a separate analysis of human health risks from PCBs in the fresh water portion of the lower River, a portion of the River that is not properly considered part of the Hudson River PCBs Superfund Site. From previous correspondence and statements, GE understands that EPA is limiting its analysis to potential remedial actions in the Upper River. Assessing human health risks in the Lower River suggests that the Agency may be attempting to justify a remedial action on the basis of benefits to the Lower River.

Justifying any remedial action in the Upper Hudson River on the basis of benefits to the Lower River would have serious consequences to the scope of EPA's present reassessment. In such circumstances, EPA would be obligated to investigate and evaluate remedial alternatives, such as source control in the lower River; consider the greatly increased number of sources of PCBs (and other contaminants) to fish in the Lower Hudson; and identify the much wider group of parties who rightfully should be classified as PRPs. The presence of other dischargers of PCBs in the Lower River is well known to

[1] See Nov. 6, 1997, letter from Angus Macbeth to Richard Caspe; May 5, 1998, letter from Angus Macbeth to Douglas Fischer.

EPA; the Agency has conducted recent studies of PCB discharges into New York Harbor, including sampling outfalls, and of comparative contributions of PCBs into the Harbor. The Agency made the importance of other contaminants plain in its 1984 ROD, concluding "that detectable levels of dioxin, dibenzofurans, mercury and chlordane (from known and unknown sources) have also been identified in Hudson River fish, and that even if PCBs decrease to an acceptable level, the fishing bans would continue on the basis of these other types of contaminants."

EPA cannot have it both ways. The Agency cannot describe the Site as encompassing the 150 miles from Troy to the Battery and then address only one contaminant and one or two PRPs outside that 150 miles as the sole subjects for remedial consideration. The scope of EPA's Superfund activity at the Site is circumscribed by the characterization and definition of the site which EPA promulgated in its rule-making many years ago.

B. The Agency must provide advanced notice of its intent to use additional data.

The Agency points out in the introduction of each section of the SOW (SOW, at 1, 6, 7) that individual components of the proposed approach may be revised if additional data are identified in the course of preparing the risk assessment. GE agrees that appropriate additional data should be included if they would lead to a more accurate baseline risk assessment. We note, however, that this statement appears to be inconsistent with the Agency's claim that it already possesses all the necessary data to complete the reassessment and, indeed, that no new data will be considered. We trust that the Agency will not ignore new and relevant data in its remedial decision making. Regardless, the Agency should notify the public of any new data it intends to use, indicate how it proposes to incorporate the data in the risk assessment, and, when appropriate, make such data available for review and comment by the public. By releasing this information prior to its use, the Agency will comply with its mandate to include the public in its decision-making process.

In that light, the SOW states that the risk assessment for the Mid River will be based on the ongoing modeling by Drs. Thomann and Farley (SOW, at 17). This work is currently not available for external review and comment. If the Agency intends to use the Thomann/Farley model, it must provide the public an opportunity to review and comment on it. The Thomann/Farley model should also be subject to external peer review consistent with the peer review of EPA's own modeling effort for the Upper Hudson.

C. The Agency must emphasize that the risk estimates presented in the HHRA are hypothetical.

By EPA policy, baseline risk assessments of Superfund sites generally do not consider the effects of administrative and other types of existing controls on exposure (EPA, 1989). To the extent that the assessments provide a starting point for the decision of what remedies (including administrative controls) are necessary at a site, this approach is understandable. However, baseline risk assessments also communicate to the public an understanding of the fundamental nature of the risks that currently exist at Superfund sites.

In the case of the Upper Hudson River, the risks that would occur without fishing restrictions are different from the risks that actually exist today and into the foreseeable future. NYSDEC has established and enforces a ban on keeping of fish in the Upper Hudson.[2] This ban is well publicized and enforced by a conservation officer who patrols the Upper Hudson. This officer interviews each angler he meets, including anglers fishing from shore and from boats. Over a recent three-year period (August 31, 1995 through July 31, 1998), the conservation officer on the Hudson River has checked 1,437 anglers and issued only nine tickets and three warnings for keeping fish (NYSDEC, 1998, attached). This finding confirms that the current ban on keeping fish is extremely effective in controlling exposure to PCBs.

[2] As discussed in Section 3.3 below, consumption of fish from the entire Hudson River is also subject to a general restriction in consumption that is independent of the PCBs in the fish. In addition, stiff, conservation-based restrictions on fishing are in place on the lower River. The advisory and fishing restrictions affect fish consumption by truncating the distribution of consumption rates. Since the advisory and restriction are not functions of PCB contamination, they must be taken into account when assessing fish consumption in the baseline risk assessment.

As to the Lower Hudson, NYSDEC and the Atlantic States Marine Fishery Commission have imposed conservation-based restrictions on keeping fish of many species. These restrictions are imposed to assist in the maintenance of fish stocks, not for reducing exposures to PCBs. Nevertheless, they have the effect of reducing such exposure dramatically. Under the baseline assessment described in the SOW, estimates of fish consumption will be developed under the assumption that there is no ban or restriction on keeping and consuming fish. The findings of an assessment that assumes that all anglers are free to keep and consume as many fish as they desire would not be a fair description of the actual risks facing anglers using the Hudson River. Therefore, the HHRA should clearly state that (1) the fishing restrictions effectively eliminate current and future exposures, thereby eliminating the risks (if any) from the consumption of fish; and (2) the risk estimates produced in the Phase 2 assessment are hypothetical risks that would occur in the absence of the current restrictions on keeping and consuming fish.

SECTION III
EXPOSURE ASSESSMENT

For the exposure assessment, the SOW proposes to develop an exposure scenario using a variety of inputs based both on site-specific data and default assumptions. These will then be used to develop both deterministic and probabilistic estimates of exposure.

GE concurs with many aspects of this approach. For instance, the SOW's proposal for the following aspects of the exposure scenerio are generally sound:

- The definition of the exposed population as those anglers who begin fishing at a specific "start date" ;
- The assumption that an angler fishes from multiple locations along the Upper Hudson River (or Mid Hudson) ;
- The recognition that PCB levels in fish should vary as a function of date, location and species of fish;
- The assumption that an angler catches and consumes a number of different species of fish;
- The consideration of cooking loss;
- The determination of duration of exposure based on site-specific data; and
- Basing fish consumption rates on site-specific information.

The SOW's failure to include details on how the Agency intends to implement these principles is troubling and suggests that the Agency may intend to default to "worst case" exposure assumptions (for example, SOW, at 13).

The SOW's failure to explain the proposed approach for the HHRA adequately also affects other important aspects of the exposure assessment. In particular, the SOW's description of the proposed Monte Carlo modeling is muddled and confusing. We point out these problems below and make recommendations about how the probabilistic modeling should be implemented. We also present recommendations concerning other

aspects of the proposed exposure assessment, including estimation of fish consumption rates, future PCB concentrations in fish, duration of exposure and cooking loss.

A. The proposed approach for determining fish tissue concentrations for the high-end angler is flawed.

The SOW's proposal for selecting fish tissue concentrations for the high-end angler is invalid. As indicated in EPA's Phase 1 Report, fish tissue concentrations increase with the trophic level of the fish. As a result, the fish with the highest levels of PCBs tend to be predator fish, such as the northern pike. These fish species make up a relatively small fraction of the edible fish in the Upper Hudson. It is highly implausible that an angler could catch a sufficient number of these fish to support high levels of fish consumption. It is far more likely that the anglers with high fish consumption rates will consume the more readily available species and will consume multiple species. Thus, the high level of intake will be associated with the average fish concentrations of the more available species.

The HHRA should follow the Guidelines for Exposure Assessment's approach for selecting inputs for the determination of the high-end exposed individual (EPA, 1992). This guidance recommends that values be selected for one or two inputs based on the 95th percentile and that the remaining values be assigned values for typical individuals. Use of extended worst case or even reasonable worst case values for all inputs of a risk assessment results in implausible results (McKone and Bogen, 1991).

Therefore, GE recommends that the inputs to the high-end angler be revised as follows:

- The values for fish intake and duration should be set at the 95th percentile values.
- The median value of body weight (age adjusted) should be used.
- Cooking loss should be based on the most likely estimate of reduction.

- The value for fish concentration should be based on a weighted-average of the best estimates (not UCL) of the mean concentrations of PCBs in the most available species.

B. Monte Carlo Modeling

The SOW provides limited information on EPA's proposed use of Monte Carlo modeling of exposure. GE supports the Agency's proposal to use Monte Carlo modeling, but the SOW's failure to present a thorough and transparent description of the modeling that the Agency intends to use inhibits a detailed critique. Consequently, we present GE's recommendations on how the Agency should proceed.

B.1 The SOW does not provide an adequate description of the model.

The portion of the SOW that discusses the modeling is limited to a few scattered paragraphs and sentences. These fragments contain inconsistent and confusing terminology. Moreover, the limited information provided in the SOW concerning the proposed Monte Carlo modeling suggests that a number of fundamental decisions on the model structure and the inputs to the model have been made, but the details of these decisions have been not been disclosed.

The lack of coherent description is surprising in light of the extensive discussions between EPA and GE concerning this topic and the information that GE has previously provided to the Agency. GE has developed an advanced Monte Carlo model of exposure to PCBs from the consumption of fish in the Upper Hudson River (ChemRisk, 1995e). In October 1995, GE met with EPA and provided a conceptual description of the model, a printout of the computer code, copies of ancillary materials, and electronic copies of the working model. This material outlined the conceptual issues and provided technical material to assist in the evaluation of essential scientific questions in modeling. A peer-reviewed article based on this work has been published on the topic of modeling of exposure to PCBs from the consumption of fish (Keenan et al., 1996). GE urges EPA to

consider, and where appropriate, incorporate these materials into its Monte Carlo modeling effort.

B.2 The SOW does not explain whether and how the Agency intends to model variation and uncertainty.

Monte Carlo modeling is fundamentally the same for any type of equation, involving the repeated use of an equation to produce a range of answers. However, the process of Monte Carlo modeling becomes more complex when models separate uncertainty from variability (Frey, 1993; Hoffman and Hammonds, 1994), and when models consider time-varying exposures such as microexposure event models (Price et al., 1996).

Several statements in the SOW suggest that such complex approaches will be used in the Hudson River assessment. However, the discussion is so scattered and incomplete that the approach the Agency intends to follow is not clear. For example, the SOW states that the exposure assessment portion of the risk assessment will consist of two parts: the first, a standard exposure assessment, and the second, a Monte Carlo analysis (SOW, at 6). The discussion continues that the probabilistic (Monte Carlo) analysis will attempt to capture information on uncertainty and variation. Yet, the discussion only includes a brief description of the steps needed to develop a Monte Carlo model of exposure variation across individuals; it contains no discussion of uncertainty. From this discussion, it is unclear whether EPA intends to assess uncertainty in the Monte Carlo modeling.

Similarly, there are various references in the SOW that allude to "two tier" (SOW, at 10, 11, 12) and "two-stage" (SOW, at 13) Monte Carlo analysis. It is not clear whether the Agency intends these statements to refer to separate analyses of uncertainty and variability or some other sort of analysis. In contrast, in the discussion of risk characterization (SOW, at 15), the SOW states that "[a]n enhanced Monte Carlo analysis will be performed to evaluate variability and uncertainty in exposure parameters, using two phases to distinguish the impacts of variability and uncertainty, where appropriate."

Although this implies separate analyses of uncertainty and variability, the description of the Monte Carlo modeling is insufficient to allow an understanding of how many of the elements will be performed.

GE urges EPA to analyze variability and uncertainty separately. EPA guidance has emphasized the value of separating variability and uncertainty in probabilistic analysis (EPA, 1997a,b). The Agency must recognize, however that this level of analysis poses significant challenges for the Agency. First, such analyses require the use of nested loops in programming (Hoffman and Hammonds, 1994). Since the current modeling recommended by GE requires the use of nested loops (one for each year, and inside of each year a loop for each fish consumed) (ChemRisk, 1995e), the addition of another layer of nested loops poses a significant computational challenge.

Second, all inputs with variability must jointly characterize uncertainty and variability. This may be relatively minor for inputs such as body weights but will pose a significant challenge for factors such as fish consumption, duration, and fish concentration.

Third, the Agency should not arbitrarily reject the consideration of certain sources of uncertainty. If the Agency includes quantitative analysis of uncertainty, then it should strive to include all sources of uncertainty. These sources include:

- uncertainty in the dose response measurements;
- uncertainty in intake rates that results from the use of data on the consumption of fish from multiple bodies of water;
- uncertainty in the stability of fish consumption rates over time;
- uncertainty in modeling fish tissue concentrations; and
- uncertainty in duration of exposure when the effects of cessation are not considered.

B.3 Other issues concerning Monte Carlo modeling

There are several other issues concerning Monte Carlo modeling that require comment.

First, the SOW suggests that an individual's exposure will take into consideration the time-varying nature of exposure concentrations (SOW, at 13). GE supports the consideration of time-varying exposures.

Second, the statement in the SOW that the "90^{th} percentile will be always used" (SOW, at 15) is contradictory to the fundamental nature of Monte Carlo modeling. Monte Carlo modeling requires a random selection of input values from a distribution. Limiting the selection to a single percentile is clearly wrong.

Third, the SOW correctly states that there are no data on an angler's year-to-year variation in fish intake rates in the published literature (SOW, at 15). This raises an important issue on how to account for such potential variation. The SOW correctly states that intake rates over time will not be perfectly correlated because variations in weather, productivity of a fishery, vacation choices, and other factors will influence anglers annual fishing rates on a yearly basis. GE does not agree, however, with EPA's rationale for not allowing the model to vary the ingestion rate from year to year -- namely that such an approach "would assume that there is no correlation between yearly ingestion rates and effectively average the high-end consumers out of the analysis" (SOW, at 16). There are a number of methods by which an angler's year-to-year variation in intake rates can be modeled without assuming that anglers' annual intakes are not correlated. For example, intake can be correlated by allowing the intake rates to vary within a fixed range of percentiles (Price et al., 1997). The uncertainty in annual intake variation is a legitimate issue that should be addressed in modeling long-term dose rates. The Agency's adoption of a modeling approach where the intake rate is fixed as a worst case assumption unnecessarily adds conservatism and fails to use the strengths of Monte Carlo techniques.

Finally, the SOW states that the modeling of fish tissue concentrations may or may not include information on the variation in PCBs in fish of the same species, date, and location (SOW, at 13). This inter-fish variation can be shown to average out in anglers with high levels of fish intake. That is, this source of variation averages out and does not contribute to the variation in doses in anglers consuming large amounts of fish. As a result, all that may be necessary for modeling of fish tissue concentration is an estimate (with uncertainty) of the mean. This factor should be taken into consideration in the development of data on fish tissue levels.

B.4 Recommended approach

As noted above, GE provided EPA with detailed information on a modeling approach for characterizing exposures to anglers (ChemRisk, 1995 a-e) (attached). Based on discussions with the Agency, it was apparent at that time that EPA contractors generally agreed with the approach. GE still believes that this modeling approach is fundamentally correct and should be applied to the Hudson River. This modeling approach includes:

- modeling each fish consumed by each angler;
- defining the angler population of interest as those anglers who would begin using the river at a certain "start date";
- accounting for the temporal changes in the concentration of PCBs in fish;
- modeling exposure duration as a function of the demographics of the angler population;
- accounting for cooking loss;
- modeling the species preference;
- developing estimates of the average daily dose using a PCB specific averaging time; and
- accounting for temporal changes in angler body weight and behavior.

In addition, GE proposes the following additional comments on modeling angler exposures. First, the modeling approach should separate uncertainty from variability. This

should be done using a nested loop approach (Hoffman and Hammonds, 1994; Price et al., 1995). In the outer loop, values are selected from distributions that characterize the uncertainty in the inputs. As discussed above, this uncertainty should include all sources of uncertainty.

Within this outer loop, the model should consist of a microexposure model of variation in the anglers, as described by ChemRisk (1995c). EPA should investigate whether the model can be simplified by replacing distributions of inter-fish variation in concentrations (species, location and date specific) with the mean concentration.

In the case of the Mid Hudson, EPA should revise the model to discriminate between the exposures that hypothetically occur as a result of eating fish that have accumulated PCBs from the Upper Hudson River and those that have accumulated PCBs from other sources (in the Mid River and elsewhere). It will be critical for the Agency to track these two doses separately. This is necessary in order to provide risk managers with a clear understanding of the relationship between PCBs in the river and potential exposures in order to assess the potential effectiveness of remedial options in the Upper River.

EPA should also extend the models of dose to track how exposures from fish affect the human body burdens of PCBs. This analysis will require the incorporation of simple toxicokinetic models of PCB intake and retention into the microexposure Monte Carlo model (Keenan et al., 1997; Avantagio et al., 1998) and data on the background levels of PCBs in anglers. The goal of this analysis is to determine whether the consumption of fish from the Hudson River will change the body burdens of anglers (see Section 5).

C. Fish consumption rates

C.1 Development of a fish consumption rate distribution for recreational anglers

The amount of fish that anglers consume is an important parameter in the estimate of exposure to PCBs from Hudson River sediments. GE agrees with the SOW's conclusion that:

> *Fish ingestion rates are waterbody specific and depend on a number of factors including weather, available fish species, angler (man, woman or child who fishes), preference for specific species, impact of fishing bans, and distance of the angler from the water body.* (SOW, at 7)

This recognition is a significant improvement over the flawed approach taken by EPA (1991) in the Phase 1 Report, where a marine fisheries value of 30 g/day, subject to avidity bias problems (Price et al., 1994), was adopted as an estimate of fish consumption for Hudson River anglers. As GE has explained, the amount of fish consumed by a population of anglers depends on the numbers and types of waterbodies fished and the characteristics of the angler population (ChemRisk, 1995c; 1995e, attached). Fish consumption also depends on factors such as climate, fish species present, productivity, access, and the size of the angler population.

Consumption of fish is constrained by New York State fishing restrictions.

The most important factors affecting consumption of fish from the Hudson River are New York State's strict and effective restrictions on fish consumption in the Hudson River, which result in a significantly lower consumption rate than might be assumed under default exposure scenarios. It can be argued that the baseline HHRA should consider these restrictions in order to provide a site-specific and realistic estimate of exposure. The following paragraphs elaborate on this topic in light of EPA science policy and guidance.

Even apart from the Hudson-specific restrictions and advisories, New York State has issued a statewide general health advisory for eating sportfish from any of New York's freshwaters, "to protect against eating large amounts of fish that haven't been tested or [that] may contain unidentified contaminants" (NYSDOH, 1997). This advisory urges individuals to consume no more than one meal per week (32 g/day) of sportfish taken from any of the state's freshwaters (NYSDOH, 1997). This statewide general health advisory is different from the consumption restrictions placed on the Upper Hudson River. In addition, NYSDEC and the Atlantic States Marine Fisheries Commission impose strict, conservation-based fishing restrictions for various species, including striped bass. Even if EPA disregards the effect of the Upper River consumption ban in developing its rate of fish consumption for use in the baseline HHRA, EPA must address the statewide consumption advisory and the conservation-based fishing restrictions in this context. EPA must do so in light of the fact that these advisories and restrictions were issued independent of the presence of PCBs in the Hudson River. Specifically, any distribution of fish consumption rates selected for use in the baseline Hudson River HHRA must be truncated at 32 g/day to reflect the maximum consumption rate allowed by New York's advisory.

It is appropriate and consistent with EPA guidance for the exposure assumptions in the baseline risk assessment to reflect real-world, current conditions – especially those like the statewide general health advisory that are unrelated to the risk management of the Hudson River PCB Superfund Site. As a general rule, EPA's Risk Assessment Guidance for Superfund ("RAGS") favors the use of site-specific information instead of generic, standardized assumptions. See Risk Assessment Guidance for Superfund, Human Health Evaluation Manual Part A (Interim Final) (July 1989). For example, RAGS recommends examining actual and potential "land use" when characterizing the potentially exposed populations, id. at 6-7, and evaluating a number of site-specific factors that affect the ingestion of chemicals through consumption of fish. Id. at 6-43. Other Superfund guidance provides that, when available, site-specific exposure information should be used in the baseline risk assessment in lieu of standardized, default exposure assumptions. OSWER Directive 9285.6-03, RAGS Volume I: HHEM Supplemental Guidance "Standard Default Exposure Factors" (Interim Final) (March 25, 1991)(Standard exposure

assumptions are to be used only where site-specific values arenot available.). Similarly, the EPA Science Advisory Board ("SAB") recommends the use of site-specific information in Superfund risk assessments where site-specific conditions may be unique, as on the Hudson, and limiting the use of default information to circumstances where exposure parameters are unlikely to vary significantly from site to site. An SAB Report: Superfund Site Health Risk Assessment Guidelines (February 1993, at 2).

Indeed, the whole tenor of EPA's risk assessment approach, as articulated in the above references, is to rely on site-specific information to the greatest extent possible. EPA's Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish: A Guidance Manual (September 1989) (EPA-503/8-89-002) recommends "that local or regional assessments of fishery consumption be performed whenever possible to avoid errors inherent in extrapolating standard values for the U.S. population to distinct subpopulations." (Id., at 54). EPA's Exposure Assessment Guidelines recommend the use of distributional methods of exposure analysis, such as Monte Carlo analysis, because they rely on site-specific data to provide a more precise understanding of the range of actual exposures to an affected population. (57 Fed. Reg. 22889, 22922, May 29, 1992). The SAB similarly recommends that distributional exposure approaches be used in Superfund risk assessments in order to reflect the actual behavior and exposures of those who visit or live near a site because it is "more consistent with the exposure assessment guidelines, and ...in the spirit of the Exposure Factors Guidelines." (Id at 17). In fact, the Commission on Risk Assessment and Risk Management established under the Clean Air Act Amendments of 1990 advocates the use of distributional exposure assessment methods because they incorporate population-specific exposure (receptor-based) information, and not merely assumptions about exposure derived from sources and models. "Risk Assessment and Risk Management in Regulatory Decisionmaking: Volume 2" (1997, at 774-75). The Commission noted that source-based exposure information can be "seriously misleading" as compared to personal measurement results, where individuals in a particular population are monitored for exposure. (Id., at 191).

The distinction between source-based exposure and receptor-based exposure is an important one for EPA to consider, where the pathway from a contamination source (*i.e.*, fish) to a potentially affected population is markedly reduced from that of the generic consumption assumptions on account of New York's statewide health advisory and the conservation-based fishing restriction. Unless the advisory's maximum daily rate of fish consumption (32 g/day) is used to truncate the regional fish consumption distribution selected for use in the Monte Carlo analysis, the risk assessment will fail to reflect the actual receptor-based exposure levels of the potentially affected fishing population.

Using standard fish consumption assumptions would also be contrary to Agency policy. EPA's 1992 Exposure Assessment Guidelines state that, for fish tissue, the following site-specific data are required to characterize exposure: relationship of samples to food supply for individuals or population of interest, consumption habits, and preparation habits (57 Fed. Reg., at 22910). This information is unknown for the Hudson River, largely because of the consumption ban (and subsequent enforcement) on the Upper Hudson, the consumption advisory on the Lower Hudson, the conservation-based fishing restrictions on the lower Hudson, and the statewide general health advisory pertinent to all freshwaters of the state. As it is reasonable to assume that the latter two factors will remain in place for the foreseeable future, regardless of what remedial decision is made for the Hudson River, their effects must be incorporated into exposure information used in the baseline HHRA.

<u>The data from Ebert et al. (1993) should be used to calculate hypothetical fish consumption rates for the Upper Hudson.</u>

If, in spite of the Upper River fish consumption ban and the Lower River advisories, the Agency nevertheless assumes that fish consumption is occurring, then significant changes need to be made to the SOW's proposed approach for estimating consumption rates for recreational anglers. Most importantly, EPA should use the data from Ebert et al. (1993) to calculate hypothetical consumption rates for the Hudson River. Furthermore, the consumption rate distribution derived from Ebert et al. should be

truncated at a maximum value of 32 g/day to reflect the New York statewide health advisory level for freshwater fish consumption (NYSDOH, 1997).

GE disagrees with the SOW's assessment of the surveys that might be used for estimating fish consumption in the Hudson River in the absence of the current restrictions. Of the three surveys of angler behavior on the Hudson River, two are mail surveys of New York anglers in general (NYSDEC, 1990; Connelly et al., 1992), and the Clearwater creel survey (Barclay, 1993) was performed on Hudson River anglers. None of these surveys focused exclusively on fish consumption from the Hudson River. NYSDEC (1990) evaluated fish consumption from all recreational and commercial sources, including self-caught fish from the Hudson. Connelly et al. (1992) evaluated self-caught fish consumption but did not estimate consumption from individual waterbodies. Barclay (1993) collected data on the frequency of self-caught fishmeals but did not calculate a fish consumption rate. In addition, this survey does not contain sufficient information to allow the calculation of a meaningful fish consumption rate for the Upper Hudson River. [3]

Because the available surveys are flawed and cannot be used to assess hypothetical consumption rates in the absence of restrictions, the SOW should base the Hudson River estimates on data from similar bodies of water or from regional data. The selection of a surrogate study depends on the characteristics of the population under consideration and the type of waterbody being evaluated. Specifically, it is critical that the study be focused on the consumption rates of self-caught, freshwater fish over long periods of time. These criteria must be met to ensure that the fish consumption rate closely approximates hypothetical consumption from the Hudson. It would be preferable to use a study that evaluated consumption from a single river that was similar to the Hudson. If a specific waterbody with appropriate characteristics cannot be identified, it would be more appropriate to use estimates generated for flowing waters only. The selected study should

[3] The SOW implies that the Agency intends to create a distribution of fish consumption for the Hudson River by merging data from multiple studies. Such a process is complex, with important logical and statistical issues that must be addressed before proceeding. It is not clear from the SOW that EPA intends to do so. Moreover, the rationale for using data from these angler surveys appears to be founded on the idea that if their results are consistent, EPA can have greater confidence in selecting the Connelly et al. data as its *a priori* favorite (SOW, 8).

have collected data from regionally appropriate waterbodies. In addition, there should be a metric that demonstrates the appropriateness of selection and the uncertainties associated with it.

There are a limited number of studies available in the New York/New England area that provide information on consumption of sport-caught fish from freshwater rivers and streams. The Ebert et al. (1993) and Connelly et al. (1992) studies most closely approximate hypothetical consumption from the Hudson River.[4] Both of these studies evaluated consumption of self-caught freshwater fish by recreational anglers using a mail recall survey. Given these similarities, it is not surprising that both studies reported very similar fish consumption rates. The results of Connelly et al. (1992) indicated that the average New York angler consumes 11 meals per year of self-caught fish from New York's freshwater fisheries. If it is assumed that each meal is 227 grams in size (1/2 pound) (West et al., 1989; NYSDEC, 1990), it can be estimated that the average New York angler consumes self-caught freshwater fish at a rate of 7 g/day. This estimate is very similar to the mean rate of freshwater fish consumption by Maine anglers of 6.4 g/day from all waters reported by Ebert et al. (1993).

Although the Connelly et al. (1992) study is specific to New York State, there are several factors that would require the analyst to make additional assumptions to use the data as the basis for the Hudson River assessment. First, Connelly et al. (1992) only presents a single point estimate value for fish consumption. The use of a distribution of consumption rates is necessary in order to characterize interindividual variability and realistically assess the potential risks to recreational anglers. With only an average consumption rate value, it is not possible to represent the range of recreational anglers accurately, including those anglers who ingest higher amounts of fish. While it may be possible to develop a distribution of consumption rates by going back to the original raw data, additional analysis will be required to complete this task.

[4] Connelly et al. (1996) surveyed Lake Ontario anglers to evaluate the effect of Lake Ontario health advisory recommendations and therefore this study may not be directly relevant to fish consumption in the Upper Hudson.

Second, the mean fish consumption rate determined by Connelly et al. (1992) represents fish eaten from all freshwaters in the State (i.e., lakes, ponds, rivers, and streams). As pointed out in Ebert et al. (1993), intake from rivers and streams is only a fraction of the intake from all freshwaters. In addition, the rate of intake from multiple waterbodies is higher than that from a single water system (Ebert et al., 1994). Given these factors, it is highly likely that the fish consumption rate in Connelly et al. (1992) overestimates the hypothetical fish consumption rate on a single portion of the Upper Hudson River.

Finally, the purpose of the Connelly et al. (1992) study was not to identify a consumption rate for New York anglers. Although questions were asked in the survey regarding fish consumption behaviors, those questions were aimed at estimating how the effect of health advisories altered the consumption behavior of recreational anglers.

While the data from Ebert et al. (1993) are not specific to New York State, these data are readily useable and may provide a more appropriate surrogate for Hudson River anglers than the Connelly et al. (1992) data. Angler demographics and fishing opportunities are similar in Maine and New York, and the mean fish consumption rates are similar for both studies (NYSDEC, 1990; Connelly et al., 1992; Ebert et al., 1993). In addition, Ebert et al. (1993) provides a complete distribution of fish intake rates for flowing waters, i.e., streams and rivers. The Ebert et al. (1993) survey also addresses each of the criteria identified in the SOW (SOW, at 9) to evaluate angler surveys.[5] Thus, the best region-specific data on fish consumption rates are available from Ebert et al. (1993), and GE urges EPA to use these data in the Hudson River risk assessment.

[5] The selection of the most appropriate fish consumption rate is discussed more fully in the paper entitled *Estimating Fish Consumption Rates for the Upper Hudson River* (ChemRisk, 1995c) and in the peer-reviewed journal articles, *The Effect of Sampling Bias on Estimates of Angler Consumption Rates in Creel Surveys* (Price et al., 1994), *Selection of Fish Consumption Estimates for Use in the Regulatory Process* (Ebert et al., 1994), and *Estimating Consumption of Freshwater Fish among Maine Anglers* (Ebert et al., 1993).

The SOW's definition of study population is arbitrary and must be changed.

GE disagrees with the SOW's arbitrary definition of the study population. The SOW truncates the full distribution of hypothetically exposed anglers by defining the lower end of the study population as those anglers "who would consume self-caught fish from the Hudson River at least once per year in the absence of a fishing ban." Many anglers, however, will consume fish at frequencies much less than once per year (ChemRisk, 1991a; Ebert et al., 1993). The SOW's omission of such frequencies thus biases upwards the resulting risk estimates. A better approach would be to define the population as those anglers who consume fish once in the "start year" (the first year of exposure) but not require them to consume fish in every subsequent year. This would allow the model to include anglers who consume fish at lower frequencies than once per year.

For the Mid Hudson, the HHRA should also evaluate its selection of angler consumption studies in the context of potentially short-lived fishing activities, such as fish tournaments or short fish runs (e.g., shad run in the Mid and Lower Hudson River), to determine the potentially exposed populations. Because of these events, it is possible that the estimates of fish consumption for the Upper Hudson cannot be applied categorically to the Mid Hudson. Therefore, surrogate surveys of fish consumption that consider such events are needed to accurately select a distribution of fish consumption rates for this section of the River.

C.2 Angler Subpopulations

The SOW states that no attempt will be made to distinguish subpopulations of "highly exposed or lesser exposed anglers." GE agrees with this approach, which is supported by the available data suggesting that recreational anglers are the appropriate population for the HHRA.

Historically, concerns have been raised over hypothetical subpopulations of anglers who consume greater amounts of self-caught fish than the general recreational angler population, due to their reliance on fishing as a major or sole source of dietary protein for their families (USEPA, 1998); Abraham et al., 1995; Becher et al., 1995; McCormack and Cleverly 1990; West et al., 1991). The term subsistence anglers has been applied to this population.

The characterization of this population has been extremely ambiguous. In North America, Native American populations that have subsistence and treaty rights to certain fisheries (CRITFC, 1994) and Arctic Inuits who, because of tradition and their remote location, rely heavily on native foods obtained from the sea (Kinloch et al., 1992; Coad, 1994) appear to be high consumers. Beyond these fairly well defined populations, clear examples of subsistence anglers are difficult to define.

In order for an individual to consume at high rates, that person must have access to large amounts of the fish and must have either a need or preference to consume locally caught fish in large quantities. There are several factors that could define such a population including:

- low income individuals who must rely on fish for their dietary needs,
- native peoples who have cultural traditions of consuming large quantities of fish,
- commercial anglers who have ready access to large amounts of fish, and
- recreational anglers who have a strong preference for fish

The fish consumption habits of these subpopulations, compared with the distributions of consumption rates for the general recreational angler population, are discussed below.

Income level

Low income, in and of itself, does not lead to high levels of fish consumption. The fish consumption survey literature indicates that there are no significant differences in fish consumption rates among different income groups (Ebert et al., 1993; Connelly et al. 1990; West et al., 1991). Wendt (1986) studied the fish consumption habits of low income families

living in New York State to determine how much freshwater fish they consumed from New York State waters. Based on the reported range of meals and an assumed meal size of 1/2 lb. (227 g), it can be estimated that these individuals consumed at a mean rate of 11 g/day and a maximum rate of 60 g/day. This mean is consistent with the means reported in more recent surveys of New York's recreational anglers (Connelly et al., 1990, 1996) and other recreational anglers in the Northeastern U.S. (Ebert et al., 1993; 1996), while the maximum rate is lower. Thus, low-income populations living in New York State do not have higher rates of fish consumption than recreational anglers in the region.

Ethnic background

There are data indicating that certain localized North American ethnic subpopulations may have higher rates of consumption than the general angler population. Studies of native peoples in the Pacific Northwest of the U.S. and Canada indicate that they rely more heavily on fish as a staple of their diets than does the general population. However, these findings are of little relevance to the Upper and Mid Hudson Rivers since the counties bordering these portions of the River do not have such local ethnic populations.

In addition, when individuals from these same ethnic populations reside in a more heterogeneous and economically developed area, these differences diminish (Wolfe and Walker 1987). While mean consumption rates reported for native peoples living in closer proximity to economically developed areas were higher than the mean values reported for the general recreational populations (NYSDOH, 1993; West et al. 1991; Selikoff et al. 1982; Hutchison and Kraft, 1994; Peterson et al., 1994), their maximum rates were similar. Other comparisons of fish consumption by ethnic background have reported no significant differences among consumption rates for those groups (Ebert et al., 1993; Landolt et al., 1985; Anderson and Rice, 1993).

Commercial Anglers

Since at least 1976, commercial angling has not been practiced on the Upper River and has been severely restricted for many species on the lower River. Nevertheless, this group could, in theory, be a population of concern for the Mid River. Individuals who have commercial fishing licenses have unlimited access to their marketable catch and might be assumed to consume more fish than the recreational angler population. Limited data on the fish consumption activities of freshwater commercial anglers show that such anglers do not eat substantial amounts of the fish that they harvest, due to the fact that the sale of those fish is critical to their household income and their ability to pay for other foods and living expenses. For example, Hubert et al., (1975) studied commercial freshwater fishing activities in Upper East Tennessee during 1973 and reported that, of a total of 94,079 kg of fish commercially harvested by 29 anglers, 2,665 kg were retained for personal use. If this amount of fish is divided among the 29 anglers and their families and assumed to have edible portions of 30 percent, the resulting mean consumption rate is 25 g/day. This mean rate is very similar to mean rates reported for recreational anglers fishing large bodies of water (SCCWRP and MBC, 1994; Ebert et al., 1994). Thus, commercial freshwater anglers do not consume substantially more fish than recreational anglers fishing the same types of waterbodies.

Recreational Anglers

Fish consumption rates among recreational anglers are highly variable. Based on available survey data and on a critical review of the relevant literature, high-level fish consumers in North America are a diverse group that cannot easily be defined or identified by socioeconomic characteristics. With the exception of certain native peoples who have continued to promote their cultural dietary traditions, there are no social or economic characteristics that are associated with the presence of a high fish consuming population. Consequently, EPA has made the correct decision in not developing a separate exposure assessment for this population.

C.3 Species-specific fish ingestion rates

In general, GE agrees with the Agency's attempt to address species-specific fish ingestion rates in the exposure assessment. Anglers typically prefer to catch certain desirable species and to reject others. Moreover, many anglers engage in short-lived fishing activities, as mentioned previously. Since PCB levels in fish vary by species, it is important to capture this angler preference in the estimates of exposure to PCBs. The NYSDEC study and the work by Connelly et al. show that New York anglers preferentially select for certain species in both fishing effort and consumption (NYSDEC, 1990; Connelly et al., 1992). In many cases, the species selected were those that accumulate lower levels of PCBs, often because these most desirable species have relatively low lipid contents as compared to other species present in the Upper Hudson. Since the species of fish sampled by EPA or NYSDEC for PCB tissue analysis are not necessarily consumed by recreational anglers in amounts proportional to their sampling frequencies, the risk assessment for the Upper Hudson should consider both interspecies differences in PCB concentration and angler preferences.

Information on species preference specific to the Upper Hudson River is unavailable. However, data on angler preference in freshwater rivers in New York similar to the Upper Hudson River are available from Connelly et al. (1992).[6] Based on these data, it is possible to identify species preferences among New York anglers that can be used as a surrogate for Hudson River anglers. Connelly et al. (1992) collected information on fishing behaviors (e.g., species caught, waterways fished) and fish consuming behaviors (e.g., species eaten, preparation techniques used) of licensed anglers. In order to use these data for the Upper Hudson, it is necessary to identify rivers and streams with characteristics and species similar to the Upper Hudson. Such an analysis results in a list of fish species likely caught in the Upper Hudson and the probability of how often these species are eaten. By taking this approach, a probability distribution that accurately reflects species consumption preferences of Hudson River anglers can be developed. This

[6] EPA should not include the Connelly et al. (1996) study as it was a survey of Lake Ontario fishers.

issue is addressed in ChemRisk (1995a), which recommends the appropriate input parameters for the microexposure Monte Carlo analysis.

GE is concerned about the SOW's proposed approach for considering species-specific consumption (SOW, at 9-10). The SOW states that species-specific fish ingestion rates will be developed from data collected from multiple studies of anglers. The result of this analysis appears to be some sort of species weighting factors that will be applied to all anglers. This use of a single set of factors implicitly assumes that all anglers will consume the same species and in the same proportions. As the SOW acknowledges (SOW, at 10), this assumption is implausible. Anglers can be expected to vary the species they consume based on their choice of fishing location, tackle, and means of fishing. The SOW proposes to address this uncertainty by running a separate analysis in which anglers will be assumed to consume the species with the highest level of PCBs (SOW, at 13). The SOW does not indicate how the results of the two estimates will be used in assessing the baseline analysis. In any event, this approach for assessing the impact of species choice is invalid because it provides the risk manager with results from two implausible sets of assumptions.

GE is also concerned with the SOW's proposal to combine surrogate fish species preferences for the Upper and Mid Hudson. This approach is unjustified and scientifically invalid as these anglers would be expected to have very different preferences based on the type of fish present in the respective stretches of the river.

D. Determination of future PCB concentrations in fish

D.1 Use of model results

The SOW states that projected PCB concentrations in fish will be determined using the EPA bioaccumulation models (SOW, at 12). There are two components to these predictions: the average concentration and the distribution of concentrations.

Average concentration

The Bivariate Statistical Model is the primary model developed by EPA to estimate mean fish total PCB and Aroclor levels. It is subject to several limitations, as described in GE's comments concerning the Preliminary Model Calibration Report (GE, 1996). One particular source of concern is that the BSM overestimates the observed values at low concentrations (see the enclosed reprint of EPA Figure 9-12). PCB levels in fish are declining, as shown by the trends since 1993 in the upper river. Therefore, it is important to be sure that model predictions do not overestimate the true future levels. As discussed in GE (1996), an alternative modeling methodology must be developed; a time-variable mechanistic bioaccumulation simulation is the preferred alternative.

In addition, the BSM has practically no predictive power within each river reach. For example, in EPA Figure 9-12, there is no relationship between predicted and observed largemouth bass total PCB concentrations in Thompson Island Pool (symbol "D" on the Figure). Because of these problems, the PCB levels predicted by the BSM may not reflect the decline in concentrations in largemouth bass in Thompson Island Pool. This will produce an unrealistically high computed human health risk.

The probabilistic food chain model (PFCM) is based upon linear steady state relationships between sediment, water and fish, as is the BSM. Therefore, the PFCM should be subject to the same bias and lack of predictive power as the BSM in predicting average PCB levels.

Variability

The SOW states that the high-end exposure point concentration for the HHRA will be determined using the 95% Upper Confidence Limit on the mean PCB concentration. The confidence limits of the mean are dependent upon the variance. Therefore, the distribution of PCB concentrations within each fish subpopulation must be estimated. One goal of EPA's modeling efforts was to compute the population variability using the PFCM.

As described in GE, 1996, the PFCM:

- is improperly constructed, calculating variability from uncertainty. This causes model results to have no physical meaning;
- requires the answer to solve the problem;
- incorrectly assumes sediment PCB levels do not change over time; and
- does not take advantage of the substantial information and data that are available concerning the mechanisms of bioaccumulation.

An alternative already suggested by GE is to use the extensive database collected by DEC over the course of 20 years to estimate the shape and parameters of the distributions of PCB levels in fish.

D.2 Selection of Mid Hudson fish species

The SOW states that high-end exposure point concentrations will be estimated using the most contaminated species and data from the most contaminated stretch of the Mid Hudson River (SOW, at 20). Unlike the Upper River, the Mid River contains migratory fish species (striped bass, eels, shad, etc.) that spend significant time away from the Mid River. During this time, the fish have the potential to accumulate PCBs from other sources than the Upper River. EPA must take care interpreting results from such fish when making remedial decisions related to the Upper Hudson.

E. Duration of exposure

The approach proposed to characterize the duration of exposure (SOW, at 10-11, 18-19) is a significant improvement over the approach used in the Phase 1 assessment, which relied on default values. As the SOW notes, mobility is a major consideration in determining the duration of exposure. The Agency's decision to use county mobility as a

surrogate for the probability that an angler will cease using the Hudson because of a new residence location is also appropriate.

However, this approach assumes that a move from one county bordering the Hudson to another does not end exposure, which is invalid for model runs in which the location along the river is considered. Moving from Hudson Falls to Albany will affect the probability of fishing the Thompson Island Pool. Therefore modeling of specific reaches should consider inter-Hudson River county moves.

GE supports the effort to take cessation of angling into the assessment of duration. The SOW, however, does not provide any support for the statment that "generally, anglers are highly dedicated to their sport, and few voluntarily stop fishing." GE has previously provided to EPA evidence from the Maine angler survey showing that cessation is an important factor (ChemRisk, 1995b). GE urges EPA to use these data and, as indicated in the SOW, to perform similar analyses of the data in Connelly et al. (1992).

The SOW fails to discuss the role of lifespan in limiting exposure duration. The population of concern is that group of anglers who would use the Hudson River at an appropriate "start date." Such a population at the time of the "start date" would have an age structure spanning from teenagers to individuals over 65. Therefore, the HHRA must take lifespan into account when determining the distribution of duration.

The HHRA also should model duration of exposure using the methodology described in ChemRisk (1995b). Under this approach the duration is not an input to the model but is directly based on age-specific estimates of cessation, mobility and lifespan. The advantage of this approach is that the age structure of the population is handled in a consistent fashion throughout the model.

F. Cooking loss

GE supports the use of the peer reviewed literature to model the loss of PCBs during cooking, as proposed in the SOW (SOW, at 11). Cooking loss of lipophilic compounds such as PCBs is a real and verified phenomenon that has been repeatedly demonstrated in more than 20 publications in the peer reviewed literature (e.g., Sherer and Price, 1993; Wilson et al. 1998) and forms the basis for fish advisories used by the State of New York (NYSDOH 1996; NYFW, 1995). As discussed in Wilson et al. (1998), the data are more than adequate to allow the modeling of cooking loss as a function of cooking method and in turn the type of fish. GE agrees that the estimates of reduction are subject to uncertainty, and that this uncertainty may warrant consideration in the modeling of exposures.

G. Inhalation exposures

The SOW proposes that risks from inhalation of ambient air will be computed using a deterministic assessment approach (SOW, at 13, 21). As an initial matter, the Agency should recognize that this route of exposure is insignificant. Studies of PCB blood levels in individuals near other Superfund sites have consistently revealed that such individuals do not have excessively high blood levels. GE urges the Agency to abandon this exposure route.

If the Agency still proceeds, it must specify the source(s) of the data it intends to use, when and with what method(s) the data were collected, the quality of the data, or how data will be evaluated with respect to calculating the high-end and central tendency point estimate concentrations. The SOW vaguely describes data collected from sites near the Hudson river, but provides no details concerning these data. The Agency must specify the source(s) of air concentration data and, if not currently publicly available, should make that data available for public review and comment

SECTION IV
TOXICITY ISSUES

A. GE supports the use of Aroclor-based toxicity criteria.

GE supports EPA's use of Aroclor PCBs in lieu of PCB congeners for the human health risk assessment. However, some may claim that an alternative approach should be taken, which we do not support based on critical scientific inconsistencies and inappropriate assumptions. This alternative for evaluating potential risks from exposure to PCBs in environmental matrices consists of the following steps, all of which serve to increase the complexity and the uncertainty of the analysis. First, the concentrations of the 11 "dioxin-like" PCB congeners is converted to 2,3,7,8-tetrachlorodibenzo-p-dioxin toxicity equivalents (TEQs) through the use of one of several TEQ conversion schemes (Ahlborg et al., 1994; EPA, 1989; WHO, 1997) The choice of conversion method is left to professional judgement and can introduce additional uncertainty into the analysis. The carcinogenic risks are then calculated for the TEQs by combining the TEQ concentrations for these congeners with a CSF for 2,3,7,8-tetrachlorodibenzo-p-dioxin of 150,000 $(mg/kg\text{-}day)^{-1}$ or with one of the more recent and more scientifically appropriate values (Keenan et al., 1991). For the non-dioxin-like PCBs, this approach uses the total PCB concentration in conjunction with the CSF for PCBs of 2 $(mg/kg\text{-}day)^{-1}$ to yield the "non-dioxin-like" PCB risk. It then adds these risks together.

To be logically consistent with this approach, the analyst must subtract out the concentrations of the dioxin-like congeners from the total PCB concentrations before making the calculations for the other PCBs. If one fails to do so, then the analysis has additional flaws due to double-counting of the carcinogenic potential of the dioxin-like congeners by including those congeners both in the risk calculation for the TEQs and in the risk calculation for the so-called "non-dioxin-like PCBs." Moreover, even if the analyst subtracts out the concentrations of the dioxin-like congeners in making the risk calculations for the remaining PCBs, this approach would still double-count the carcinogenic potential of the dioxin-like congeners, because those congeners are included

in the CSF for PCBs. The CSF for PCBs of 2 $(mg/kg\text{-}day)^{-1}$ was based on toxicological studies of Aroclor mixtures that contained dioxin-like congeners. Indeed, EPA has attributed much of the so-called carcinogenic potency of PCB mixtures to these congeners (IRIS, 1998). Thus, the CSF of 2 $(mg/kg\text{-}day)^{-1}$ is much too high to represent the carcinogenic potential of the non-dioxin-like congeners. Accordingly, even if the PCB concentrations used for the non-dioxin-like PCBs does not include dioxin-like congeners, the use of a CSF of 2 $(mg/kg\text{-}day)^{-1}$ to calculate the carcinogenic risk of those PCBs represents a double-counting of risks. In fact, unless there were a CSF for non-dioxin-like PCBs, there is no defensible way to use both the TCDD CSF and the PCB CSF in the same assessment.

Furthermore, the toxicological, epidemiological and analytical databases for Aroclor PCBs are more reliable and complete than those for PCB congeners. In summary, the following represent the advantages associated with the use of Aroclor PCBs in lieu of PCB congeners:

- The toxicity studies used to derive the Aroclor PCB CSFs and RfDs include both the coplanar and non-coplanar PCB congeners present in the Aroclor mixtures.

- The concentrations of the coplanar PCB congeners - reputedly the more toxic of the PCB congeners - is known for most of the Aroclor PCBs (e.g., Frame et al., 1996).

- There is a paucity of toxicity data for the non-coplanar PCB congeners.

- The comparability of analytical results can be difficult in PCB congener data since there are inconsistencies in the analytical methods used to quantify coplanar and non-coplanar PCB congeners.

- Aroclor PCB results are the most appropriate to use if there have not been significant changes in the PCB peak patterns.

B. Cancer dose response

GE supports EPA's recent efforts in reassessing the cancer risk of PCBs (EPA, 1996, "the Reassessment") and continues to have an important interest in working with EPA on various issues related to PCB toxicology. Although the Reassessment represents a positive step in evaluating the PCB cancer risk suggested by animal studies, GE believes that EPA needs to conduct additional analyses of existing and forthcoming data if it is to accurately assess and quantify the cancer risk that PCBs pose to humans.

Specifically, GE has previously submitted comments in several rulemakings, urging EPA to consider the numerous epidemiological studies that have been performed on populations with extensive workplace exposure to PCBs. Others have asked EPA to use epidemiological studies to establish a human cancer potency factor. EPA's responses to these comments, as well as statements in the Reassessment and the IRIS database, have been sparse and have suggested strongly that EPA has not thoroughly reviewed the epidemiological studies or considered how they can be used in risk assessment. GE believes that the SOW presents the Agency with a good opportunity to consider this matter more thoroughly.

B.1 The rationale for using epidemiological studies to establish environmental standards

To date, EPA has established cancer slope factors for PCBs based on the results of rat feeding studies. EPA's most recent effort in this regard is the Reassessment, which advocates use of a range of cancer slope factors based on the results of rat studies. Risk managers are to choose a slope factor from within the range based on the regulatory context and the pathway by which humans are expected to be exposed.

Although EPA has historically viewed all positive findings in animal bioassays as suggesting equally serious human health hazards, in reality chemical carcinogens may have tissue-specific effects, and different mechanisms of action and pharmacokinetics.

Additionally, chemicals may differentially exhibit carcinogenic effects under specific animal bioassay conditions that are unrelated to reasonable human exposures. Moreover, as discussed in the reassessment, studies vary in quality and power.

EPA recognizes the difference in potency of chemical carcinogens tested in animal bioassays, but does not evaluate the probability that such chemicals may not be human carcinogens. Many chemicals that have been proven to be carcinogenic at high doses in animal bioassays have not been shown to be carcinogenic in humans at or near environmental or occupational exposure levels. As an example, over 50 percent of approximately 400 to 500 chemicals have tested positive in at least one rodent species at high doses (Ames 1989). However, only approximately 20 chemicals are known to cause cancer in humans (Doll 1984; Paustenbach et al., 1990). Even after accounting for the typical shortcomings of some epidemiology studies (small sample size and poor quantitative knowledge of relatively small exposures), it is clear that many potent rodent carcinogens do not pose an equivalent cancer hazard in humans. (Houk 1990; Kimbrough 1990).

There are several difficulties in estimating human cancer risks from rodent bioassays. Differences in pharmacokinetics and susceptibility to organ toxicity complicate the issue of interspecies extrapolation (MacDonald et al. 1994). Compounds classified as tumor promoters are particularly troublesome in this regard, because they often produce rodent liver tumors in long term bioassays, but are not generally known to cause cancer in humans (Butterworth et al. 1995; Schulte-Hermann 1985). Tumor promoters like PCBs selectively increase the growth of cancerous cells, but do not interact with cellular DNA to cause the initial heritable change that begins the multi-stage process of cancer. The drug Phenobarbital is a classic example of a rodent liver tumor promoter that has not been shown to cause cancer in humans taking this drug for many years (Butterworth et al. 1995).

Another problem caused by the use of animal studies to predict human cancer risk is the need to use a model for extrapolation from high doses to animals to low doses to humans. In the Reassessment, EPA estimated the carcinogenic potency of PCBs by using the linear default method presented in EPA's Proposed Guidelines for Carcinogen Risk Assessment (EPA 1996). This method is likely to overestimate the low-dose carcinogenic risk of PCBs because it assumes that there is a direct linear relationship between the dose of the chemical and a carcinogenic effect. The rationale given by EPA for using a linear low dose extrapolation in the Reassessment is based on the possibility that PCBs might act in concert with other exposures and processes leading to a background incidence of cancer that would be linear at low doses.

Originally, the assumption of linearity was based on an elementary theory of the mechanism of chemical carcinogenesis, in which a single chemical molecule can form an adduct to DNA, and thereby result in cancer.[7] Tumor promotion, however, is characterized as a reversible process and the dose response relationship is expected to be nonlinear, including both a threshold dose level and a maximal response (Pitot and Dragan 1991). EPA's recent cancer guidelines (EPA 1996) allow for nonlinear low dose extrapolation in cases where the available data support a nonlinear mode of action (e.g., nongenotoxic agents).

EPA concedes that there are a number of chemicals which produce a carcinogenic response by mechanisms that may exhibit a non-linear dose response curve at low doses (EPA 1996; Butterworth and Slaga 1987). The increased acceptance of the nonlinearity of dose and effect at low doses is evidenced by a growing consensus among risk assessment practitioners that the linear model is inappropriate for dioxin, thyroid-type carcinogens, nitrilotriacetic acid, trimethylpentane and, presumably, similar non-genotoxic chemicals, (Paynter et al. 1988; Andersen and Alden 1989; Paustenbach 1989; EPA 1992b). Given

[7] While genotoxic chemicals are assumed to be better modeled by a linear dose response assumption (Weisburger and Williams 1987), this is not a proven scientific fact. Ottobonni (1984) suggested that genotoxic agents might also exhibit thresholds at low doses. These thresholds may result from a number of factors including DNA repair mechanisms, cell death, or lethal mutations. Therefore, there is considerable uncertainty in the assumption of low dose linearity for carcinogens.

the uncertainty in cancer dose response modeling, the Agency should reexamine the evidence for carcinogenic risk that can be derived from human epidemiology studies. It has been stated that epidemiologic studies are not as statistically robust as animal studies and, therefore, not as useful (Silbergeld et al. 1988). Although this can be a legitimate concern in some cases, in many cases human epidemiology studies can and should be used to validate, confirm, or set upper bound estimates of carcinogenic potency. In general, when epidemiology data are available, it is not appropriate to accept only the results of mathematical models that analyze rodent data without serious consideration given to the human experience[8] (Cook 1982; Dinman and Sussman 1983; Layard and Silvers 1989). EPA (1996b) appears to recognize this point in its proposed cancer guidelines:

> *Epidemiologic data are extremely useful in risk assessment because they provide direct evidence that a substance produces cancer in humans, thereby avoiding the problem of species to species inference. Thus, when available human data are extensive and of good quality, they are generally preferable over animal data and should be given greater weight in hazard characterization and dose response assessment, although both are utilized.*

In the case of PCBs, EPA can no longer ignore the many clinical and epidemiological studies that do not support the proposition that PCBs cause cancer in humans. GE realizes that toxicologists must be careful in relying on the results of negative epidemiological studies. However, when, as in the case of PCBs, several excellent epidemiological studies have been performed using large numbers of workers heavily exposed to a chemical over a long period of time, and the results of those studies have been negative, GE submits that such results must be factored into, or used in, the derivation of a human cancer potency factor. As discussed below, the epidemiology studies of

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[8] An example of where an animal study yielded implausible results is ethylene dibromide (EDB). In 1982, it was claimed that workers exposed for 8 hrs/day for 40 years to the OSHA threshold limit value (TLV) for EDB of 20 ppm incurred a risk of 999 in 1,000 of developing cancer. However, epidemiological evidence of actual cancer incidence in these workers did not show an increase in the cancer rate (Cook 1993). Although the EDB risks suggested by the low-dose animal models may initially seem plausible, the human epidemiologic evidence makes it clear that these workers are not likely to die prematurely as the model predicted (Hertz-Piciotto et al. 1988).

PCB-exposed cohorts do not indicate that PCB exposure leads to increased mortality, whether based on overall cancer mortality or deaths due to individual cancer types. These findings strongly suggest that human health risks from PCB exposure have been significantly overestimated in current regulations and that EPA should undertake a thorough reevaluation of the actual risks posed by PCB exposures.

In assessing the PCB studies, EPA should use state-of-the-art methodology for interpreting the results of epidemiological studies. This methodology uses a weight-of-the-evidence test and applies what has become known as "causation analysis." The methodology is well recognized within EPA (EPA 1992a; EPA 1996b). At least ten criteria have been proposed for establishing cause and effect relationships (Hill 1965; Evans 1976; Hackney and Linn 1979; Doll 1984; Guidotti and Goldsmith 1986; Mausner and Kramer 1985; Monson 1988; Hernberg 1992). However, as typically applied, the scientific convention applied in weight-of-the-evidence evaluation of epidemiological studies requires (a) the observation of a specific cancer endpoint, and (b) the meeting of six other criteria before a causal relationship between an agent such as PCBs and cancer can be inferred (Hill 1965; Mausner and Kramer 1985; Rothman 1988; Monson 1988; Hernberg 1992; EPA 1985b; IARC 1987; EPA 1996b). The six fundamental criteria are: strength of association; consistency of association; temporally correct association; dose-response relationship; specificity of the association; and coherence with existing information (also called "biological plausibility"). None of the criteria, with the exception of temporality, should be considered as necessary to establish causation. Each of the criteria is important, and causation is established by the weight of the evidence and the degree to which all six criteria are satisfied by the available data. However, the rejection of the association may be made with a high degree of confidence when three of the criteria -- temporality, consistency, and biological plausibility -- are not met (Rothman 1988; EPA 1996b). In addition to considering weight of the evidence, it is important to understand that studies with larger cohorts and numbers of cancer deaths are inherently more important when considering the weight of the evidence than are studies with smaller cohorts and fewer cancer deaths.

B.2 Epidemiological data

The collected evidence from numerous epidemiological studies over the past 20 years fails to demonstrate that PCBs cause cancer in humans, even in populations with much greater exposures than those involved here. A review of the epidemiology data for PCBs is provided below. In general, studies of PCB workers, who were exposed to PCB levels hundreds or thousands of times higher than current environmental levels, have failed to demonstrate a causal association between PCB exposure and cancer. Further, in a recent study by Harvard University researchers, no relationship was found between PCBs and breast cancer among 240 women (Hunter et al., 1997).

The most celebrated incident in which PCBs became suspected of causing cancer in humans is the so-called "Yusho" incident. In 1968, about 1500 persons in Japan became ill after consuming rice oil that was accidentally contaminated with a PCB mixture known as "Kanechlor 400" (Amano et al. 1984). A similar incident, known as "Yucheng," occurred in Taiwan in 1979. Typical symptoms were chloracne, swelling of eyelids, eye discharges, brown pigmentation of the nails and skin, and curling of fingernails and toenails. Signs of the disease were also observed in some offspring of affected mothers. Although the major symptoms disappeared over the next sixteen to twenty years, subsequent studies suggested a possible increase of cancer and adverse developmental and behavioral effects in offspring.

The cause of the incident was extensively studied and the rice oil was found to contain high levels of polychlorinated dibenzofurans ("PCDFs"), a chemical that is 100 to 1,000 times more toxic than PCBs. After finding that workers exposed to much higher levels of PCBs showed minimal adverse health effects, and after performing dose-response studies on the rice oil mixture, Japanese and Taiwanese scientists concluded that PCDFs were the prime causal factor in the Yusho and Yucheng incidents (Kashimoto et al. 1986). ATSDR agrees, finding that "there is inconclusive evidence of cancer in people who were exposed to heated non-Aroclor PCBs during the Yusho and Yu-Cheng incidents, but PCDFs were major contaminants" (ATSDR 1997).

In 1985, Dr. Kimbrough and Dr. Goyer of the National Institutes of Health unequivocally concluded that:

> *The scientific community assumes now that most of the effects observed in these two outbreaks were caused by the ingestion of the polychlorinated dibenzofurans.* (Kimbrough et al. 1985)

Likewise, the Halogenated Organics Subcommittee of EPA's Science Advisory Board reviewed a PCB health advisory from EPA and concluded that:

> *The health effects section suggests that the short-term human exposure to Yusho poisoning is [not] representative of polychlorinated biphenyl toxicosis. Recent studies indicate that the major etiologic agents in Yusho were polychlorinated dibenzofurans rather than polychlorinated biphenyls... Thus, a discussion of the human health effects of polychlorinated biphenyls should not use 'Yusho' as an example. Industrial exposure data more accurately reflect human health effects.* (Doull et al. 1986)

A number of years later in her update of PCB exposure and human health effects, Kimbrough (1995) emphatically stated that:

> *In the poisoning outbreaks, the PCDFs, not the PCBs, caused the adverse human health effects.*

Significantly, this scientific reinterpretation of the Yusho and Yucheng incidents is consistent with data from animal studies that show a relatively low level of acute toxicity — *e.g.*, LD50s ranging from about 1 to 11 g/kg-body-weight in rats, depending on the Aroclor mixture. Moreover, this explanation is consistent with the numerous studies

(discussed below) that show no significant adverse health effects in workers who had been exposed to average levels of PCBs higher than the Yusho patients were.

Subtracting Yusho from the universe of epidemiological studies of the cancer risk of PCBs leaves a number of other studies which can be grouped into three categories: (1) negative studies reporting no statistically significant relationship between exposure to PCBs and cancer (Taylor 1988; Kimbrough et al. 1997, unpublished; Hunter et al. 1997; Zack and Musch 1979; Gustavson 1986; Nicholson et al. 1987), (2) studies that were inconclusive due to small cohort sizes or flaws in study design and data interpretation (e.g., Yassi et al. 1994); and (3) studies which have been cited by some as suggesting a relationship between human exposure to PCBs and cancer (Bahn et al. 1976, 1977; Bertazzi et al. 1987; Brown 1987; Sinks et al. 1991). As will become clear from the following discussion, the studies, whether considered individually or assessed using a weight-of-the-evidence approach, provide virtually no support to the claim that PCBs are human carcinogens.

Inconclusive Studies

Yassi et al. (1994) examined the mortality of 2222 males employed between 1946 and 1975 at a transformer manufacturing plant in Canada. Although some transformers were filled with PCB-containing fluids, the vast majority were filled with mineral oils refined predominantly from naphthenic base crudes (only 85 of 51,000 transformers filled between 1956 and 1975 contained PCB fluids).

This report concludes that neither overall mortality nor total cancer mortality varied significantly from the expected. There were eleven deaths due to pancreatic cancer, a statistically significant excess, but only three of the affected workers worked in transformer assembly. Five of the affected workers worked at the plant for less than one year, and another worked just two years. There were no liver cancers in the cohort.

This study was heavily criticized by Wong (1995) for methodological flaws. Moreover, the Manitoba Workers' Compensation Board awarded compensation to widows of two of the men who died of pancreatic cancer on the basis that the cancers were linked to mineral oil exposures. ATSDR (1997) concluded that the results of Yassi et al. (1994) "must be regarded as inconclusive due to limitations such as exposure to other chemicals and the fact that no medical history of the workers was provided."

Studies Cited as Linking Human PCB Exposure to Cancer

Bahn et al. (1976, 1977) evaluated the incidence of tumors occurring in a New Jersey petrochemical facility where Aroclor 1254 had been used from 1949 to 1957. A significantly increased incidence of malignant melanomas was observed among research and development workers (2 of 31) and refinery personnel (1 of 41). In an update of that same study, NIOSH (1977) observed eight cancers in the total study population (5.7 expected). Three of these tumors were melanomas and two were pancreatic cancers. The incidence of these tumor types was reported to be significantly above calculated expectations, although no data were presented. The results of this study were confounded by the small cohort size, the fact that the workers in this facility were exposed to numerous other chemicals, and the fact that the expected cancer rates were based on U.S. population data rather than on local rates (Bahn et al. 1977; Lawrence 1977). ATSDR (1997) states that the findings of this study should be regarded as inconclusive.

Bertazzi et al. (1987) conducted a retrospective cancer mortality study of 544 male and 1,556 female workers who had been employed for at least one week in the manufacture of PCB-impregnated capacitors in an Italian plant between 1946 and 1978. Mortality was examined for that cohort from 1946 to 1982 and was compared to both national and local mortality rates. Mortality due to all cancers (14 observed vs. 5.5 national and 7.6 local) and due to cancer of the gastrointestinal tract (6 observed vs. 1.7 national and 2.2 local) was significantly increased among male workers. Death rates from hematologic neoplasms and from lung cancer were also elevated, but not significantly. Overall mortality was significantly increased above local rates (34 observed vs. 16.5 local) in the female

population. Total cancer deaths (12 observed vs. 5.3 local) and mortality from hematologic neoplasms (4 observed vs. 1.1 local) were also significantly elevated over local rates in the female population.

These results are limited by several factors, including the small number of cancer cases observed, the limited latency period, lack of pattern or trend when data were analyzed by duration of exposure, and some deaths in males with low potential for direct PCB exposure (ATSDR 1997; Kimbrough 1987). A major problem in the study design was the one week minimum period of employment required for inclusion in the study resulted in the inclusion in the cohort of workers who had no PCB exposure. This makes it difficult to assume that excess cancer cases are attributable to PCB exposures rather than to other factors. This study also did not show a dose-response relationship or any direct relationship between latency and the disease. Moreover, as discussed below, the results of this very small study are dissimilar from the results of much larger and statistically more valid studies of similar worker populations in the United States and Canada. ATSDR (1997) found that the results of this study were inconclusive.

Brown (1987) found an excess risk of cancer of the liver, biliary tract, or gall bladder in 2,588 workers (1,270 male, 1,318 female) from two capacitor factories. The workers had worked for at least three months in areas where they received heavy exposure to PCBs. Exposure was to Aroclors 1254, 1242 and 1016 (Lawton et al. 1981). The workers were also exposed to other chemicals, including trichloroethylene, toluene, and methyl isobutyl ketone.

The first evaluation of this cohort (Brown and Jones 1981) found increased cancer mortality that was not statistically significant. After an additional seven years of observation (Brown 1987), two additional cancers of the liver, gall bladder or biliary tract were observed, making the cancer increase in this combined cancer grouping significant. Among the grouped cancers, four of the five occurred in women from one of the plants. There was no increase in the number of rectal cancers from the previous study. For the total cohort, total mortality and cancer mortality were less than expected. Total cancer

among the cohort at one of the plants was significantly less than expected (18 observed versus 31 expected).

According to ATSDR, limitations and confounding factors in Brown (1987) include the small number of cases and the fact that PCB blood levels were higher in the plant with the lower incidence of cancer (ATSDR 1997). Moreover, the study failed to account for several factors particular to the plant where the increased cancer incidence was noted, including ethnicity (dominant Cape Verde background) and life style (the workers were from a harbor/fishing town where alcohol consumption and smoking behaviors are high). Furthermore, of the five liver grouping cancers, four of the workers had worked at the plant 1.5 years or less and the other worker worked at the plant less than 10 years. Finally, of the five cancers, only one was a primary liver tumor (the type of tumor predicted by animal studies) and at least one had metastasized from another site (and was therefore incorrectly identified as a liver tumor).

Sinks et al. (1991) conducted a retrospective cohort mortality analysis of 3,588 workers who were employed for at least one day at an electric capacitor manufacturing plant between 1957 and 1977. Aroclor 1242 was used in this plant through 1970, and Aroclor 1016 was used from 1970 to 1977. Mortality from all causes and from all cancers was less than expected. A significant increase in mortality rate was observed for skin cancer (8 observed vs. 2 expected) and death rates from brain and nervous system cancers were non-significantly elevated over expected rates. No excess deaths were observed from cancers of the rectum, lung, or liver, biliary tract and gall bladder, or from hematopoietic malignancies. Based on a cumulative dose estimate, which incorporated information on job station history, limited PCB environmental sampling data, and serologic data, the authors were not able to establish a clear relationship between latency or duration of employment and risk for malignant melanoma. Sinks et al. (1991) point out that the skin cancer excesses are not consistent with those of similar studies. The authors also point out that mortality may not be the best index of risk for malignant melanoma, as survival can be affected by differences in health care quality. In addition, other limitations include the lack of evaluation of exposures to other chemicals (metals, solvents, etc.), the relatively short

latency period, the small number of deaths within the cohort, and possible misclassification of brain cancer cases. Citing additional deficiencies in the study, ATSDR (1997) found the results of the study inconclusive.

Negative Studies

By contrast to the inconclusive and confounded results of these studies which are sometimes cited to link PCBs with cancer in humans, the largest study of PCB exposed workers (Taylor 1988) showed no significant increases in mortality or cancers. Taylor (1988) involved a cohort of 6,292 persons employed for at least three months during the period 1946-1976 at the GE Hudson Falls and Ft. Edward facilities. This study showed no increase in cancer mortality or in overall mortality compared to national averages. Deaths due to malignant melanoma, lymphopoietic cancers, or the combination of liver, gallbladder and biliary cancers were not significantly elevated, and brain cancers were well below the expected value. PCB exposure was shown to be negatively associated with (not statistically significant to) cancer mortality (all types combined) and lung cancer (the only cancer outcomes with numbers of cases sufficient to permit a regression analysis). In other words, as PCB exposure increased, the numbers of overall cancer deaths and lung cancer deaths decreased.

Recently, in a follow-up to Taylor (1988), a retrospective mortality study was conducted of the same cohort. (Kimbrough et al. 1997, unpublished). All workers were followed through the end of 1993. The cohort of 4062 white males and 3013 white females contributed 120,811 and 92,032 person years of observation, respectively. There were 763 (19%) deceased males and 432 (14%) deceased females. Death certificates were available for 98.5% of the decedents and only 1.3% of the cohort was lost to follow-up. For comparison, standardized mortality rates (SMRs) were calculated using both U.S. and local county mortality tables.

Overall mortality for the total cohort was significantly lower than that for the general population, as was the mortality for all cancers. The significantly lower SMRs in

the male workers is partly attributable to the low SMRs in the salaried male workers. The overall mortality and mortality due to all cancers of hourly workers was also significantly lower. The dramatically low SMRs in salaried male workers were not as evident in salaried female workers. However, 71% of the male salaried workers had obtained a college education reflecting a socioeconomic factor that is well correlated with decreased mortality (Sortie et al., 1995). Finally, there were no statistically significant increases in mortality due to any of the *a priori* cancer types. The study concluded that there was no evidence that PCB exposure at this plant had resulted in cancer mortality.

Additional recent studies undermine the often-cited link between PCBs and cancer. In Pittsfield, Massachusetts, PCBs were used in manufacturing over an extended period. The Massachusetts Department of Public Health (MDPH) recently issued a registry of the incidence of cancer mortality in Massachusetts from 1987 through 1994. The registry showed no statistically significant increases in cancer incidence (at any level of statistical significance) in Pittsfield or Berkshire County for any of the 23 types of cancers evaluated. MDPH (1997). In fact, the results for all cancer types showed that total cancer incidence in Pittsfield was 10 percent lower than expected based on the state-wide average, and also showed lower-than-expected total cancer rates for other towns in the area.[9] Last year, in what many in the scientific world describe as a definitive result, Hunter et al. (1997) published in the *New England Journal of Medicine* a study focused on the interaction of endocrine disruption and cancer. The study showed no link between PCB exposure and breast cancer. Similar results were reported by Key and Reeves (1994). As Dr. Steven Safe noted in an editorial accompanying Hunter et al. (1997), this study and others "should reassure the public that weakly estrogenic organochlorine compounds such as PCBs, DDT, and DDE are not a cause of breast cancer." (Safe 1997).

[9] Similarly, in a prior letter in 1980, the MDPH had advised the City of Pittsfield that review of the cancer mortality data for 1969-1978 showed no excess cancer mortality in Pittsfield across all causes of cancer, and further showed no excess cancer mortality in the Lakewood neighborhood adjacent to the GE facility. Parker (1980).

Assessment of the Epidemiological Studies of PCB Exposure

Conservatism in hazard identification is manifested when regulatory agencies place an emphasis on data that chemicals might pose adverse effects, and little weight on data that suggest that chemicals fail to cause adverse effects. Emphasizing study data that show adverse health effects in animals while virtually ignoring studies showing no adverse effects does not represent a balance of scientific information. (Nichols and Zeckhauser 1988) Frequently, extraordinary confidence is placed on a study that suggests that a chemical may pose a particular hazard, while only modest consideration is given to the study's quality.

More recently, the scientific community and some regulators have come to accept that not all scientific data are equal, and that only data of similar quality should be compared when drawing conclusions regarding toxic effects based on multiple studies. This philosophy, known as a "weight of evidence" approach, represents an important refinement that should be applicable to both hazard identification and dose response assessment (Sielken 1985; Anderson 1989; Gray et al. 1993). EPA's (1996b) proposed cancer risk guidelines also embrace this philosophy. The benefit of using a "weight-of-evidence" approach is that the results of several high quality toxicity studies will not be disregarded simply because the results of one or two poorly controlled studies have dissimilar findings.

As is clear from the discussion of the PCB epidemiology studies, none of the cancer incidence and mortality studies demonstrates a cause-effect relationship between PCB exposure and cancer.[10] Not only do the individual studies fail to show causation, but the weight of the evidence from the studies taken collectively also fails to establish any such relationship.

[10] It is acknowledged that Rothman et al. (1997) observed a dose-response relationship between PCB serum levels and non-Hodgkin lymphoma; however, the authors pointed out that their "results should be regarded as hypothesis generating," that "our findings require replication, and biological plausibility," and that the matter "needs further investigation." The authors also noted that studies of highly exposed capacitor workers do not support a relationship between non-Hodgkin lymphoma and PCB exposure.

As discussed previously, the scientific convention applied in weight-of-the-evidence evaluation of epidemiological studies requires (a) the observation of a specific cancer endpoint, and (b) the meeting of other criteria (strength of association, consistency of association, dose-response relationship, temporally correct association, specificity of the association, and coherence with existing information (biological plausibility)) before a causal relationship between an agent such as PCBs and cancer can be inferred. None of the criteria, with the exception of temporality, should be considered as necessary to establish causation. Each of the criteria is important, and causation is established by the weight of the evidence and the degree to which all six criteria are satisfied by the available data. However, the rejection of the association may be made with a high degree of confidence when three of the criteria -- temporality, consistency, and coherence with existing information -- are not met. (Rothman 1988; EPA 1996b) In addition to considering weight of the evidence, it is important to understand that studies with larger cohorts and numbers of cancer deaths are inherently more important when considering the weight of the evidence than are studies with smaller cohorts and fewer cancer deaths.

In the PCB studies, small increases in a variety of cancer endpoints were seen in different populations with no common thread, and several studied populations showed no increases at all. The discrepancies can be explained in innumerable ways, including exposures to other chemicals, population life styles, and even chance. Thus, little evidence exists that PCBs are human carcinogens, and the weight of the evidence fails to establish a definitive causal relationship between exposure to PCBs -- even in high concentrations -- and the incidence of cancer in humans.

In 1993, TERRA, Inc. submitted comments on the Great Lakes Initiative that provided a thorough weight-of-the-evidence assessment of what the authors classified as the four "major" cohorts in the PCB epidemiological studies (the Brown, Nicholson, Sinks and Taylor cohorts (TERRA, 1993). GE incorporates these comments by reference. The following tables from that document provide a summary of TERRA's analysis.

Summary of The Major PCB Mortality Studies

Study/Cohort	Were Significant Increases Noted?			
	SMR for All Cancer Deaths	Total Cancers	Malignant Melanoma	Liver/Biliary Cancers
The Brown Cohort				
Brown and Jones 1981 Mortality study of 163 deaths among 2,567 U.S. capacitor workers	89	No	No	No
Brown 1987 Updated mortality of Brown and Jones 1981 analyzing 295 deaths among 2,588 workers	78	No	No	Yes (but limited to females)
Nicholson et al. 1987 Mortality study of 188 deaths among 769 U.S. capacitor workers with ³5 years exposure and ³10 years latency since first exposure	79	No	No	No
Sinks et al. 1992 Mortality study of 192 deaths among 3,588 U.S. capacitor workers	85	No	Yes (but limited to males)	No
Taylor et al. 1988 Mortality study of 510 deaths among 6,292 capacitor workers	95	No	No	No

Evaluation of the Major Cohorts Using Causation Criteria

	Was This Criteria Satisfied?	
Causation Criteria	Liver	Skin
1. Strength of the Association	No*	No*
2. Consistency of the Association	No	No
3. Temporal Relationships	No	No
4. Dose-Response Relationships	No	No
5. Specificity of Association	No	No
6. Coherence of Evidence	No	No

* Denotes a statistical observation that is considered weak because it is confounded by a lack of confirmation in studies of equivalent or greater size, and it lacks confirmation in a study using greater exposure duration and latency criteria for cohort definition.

From its analysis, TERRA concluded that "the available scientific evidence do not support the contention that PCBs are carcinogenic in humans."

Other scientists have reached similar conclusions. For example, Chase et al. (1989), concluded that:

> *There is insufficient evidence to show a causal relationship between PCB exposure and the subsequent development of any form of cancer. In light of the long-term and widespread usage of PCBs in the workplace and, in some cases, the extensive exposures of workers, it is likely that evidence of carcinogenicity in humans would have been observed in the various epidemiological studies discussed above if PCBs were in fact potent carcinogens.*

Similarly, Kimbrough 1988 concluded that:

> *Thus far, no conclusive adverse effects have been demonstrated in people who carry body burdens of PCBs from environmental exposure to trace amounts of*

PCBs... Even workers with exposures two orders of magnitude greater than environmental exposures show no convincing health effects... Thus, despite positive laboratory animal data and except for chloracne, exposure to PCBs has led to no convincing, clinically demonstrable, chronic health effects in humans.

In her 1995 update, Dr. Kimbrough reaches a similar conclusion (Kimbrough 1995).

A recent review of the occupational studies by the American Council on Health and Science also concluded that none of the studies provides evidence that PCB exposure increases cancer risk in humans (Danse et al. 1997). A recent review of studies seeking to determine if there was a relationship between environmental exposures to PCBs and any human health effects, including cancer, found that "none of the 33 studies where exposure had occurred in the natural environment provided positive or suggestive evidence of an association with adverse effect." Swanson et al. (1995).

A fair and careful review of the existing PCB occupational studies leads to the conclusion that there is no credible evidence that PCBs cause cancer in humans, even at exposures that are orders of magnitude greater than environmental exposures. Therefore, GE urges EPA to reassess the human carcinogenicity of PCBs in light of the epidemiological studies.

B.3 Derivation of a cancer slope factor for PCBs from the epidemiological studies

Although the weight of the evidence approach results in the conclusion that there is no credible evidence that PCBs cause cancer in humans, it is still possible to derive a cancer slope factor from the epidemiological studies. In TERRA (1993), the authors derived such slope factors using two approaches.

First, the authors assumed that the results of Brown (1987) showing a statistically significant increase in combined liver and biliary cancers reflected a real measure of cancer potency. The authors then used the observed increase in cancer incidence, along with a

conservative estimate of exposure, to generate a human cancer potency factor. That is, it was assumed that the results of Brown (1987) were representative of human cancer risk, even though other studies of comparable or greater size had failed to duplicate the findings of liver cancer.

Second, the authors used the negative results of the largest study completed at that time (Taylor 1988), along with a conservative estimate of exposure, to calculate an upper confidence on the measured zero risk, thereby placing an upper bound on the risk.

The authors' methodology for estimating exposure and calculating cancer slope factors are described in detail in their paper. It is important to note that the authors estimated exposure using two different methods: estimating the daily dose capacitor workers received using reported workplace exposure estimates and known or estimated absorption; and estimating daily doses received by the capacitor workers using basic pharmacokinetic principles and reported body burdens and estimated tissue half-lives. In both methods, the authors used conservative assumptions to assure that there was little possibility that dose would be overestimated. As one example, when assessing exposure through the inhalation route, the authors used the geometric mean of work place air concentrations measured in the mid- to late 1970s, when PCB use was being phased out. The two methods of estimating exposure arrived at very similar dose estimates.

To be conservative in calculating cancer slope factors from the dose estimates and the results of Brown (1987) and Taylor (1988), and in recognition that there is some degree of uncertainty in the incremental risk rates that were calculated, the authors calculated cancer slope factors using both the measured cancer incidence rate and the 95% upper confidence limit on the incremental risk rate. The calculated cancer slope factors are as follows:

Cancer Slope Factors

Study/Method	Cancer Slope Factor ($mg/kg/day^{-1}$)
Brown (1987) – Measured	5.9×10^{-3}
Brown (1987) - 95% UCL	1.9×10^{-2}
Taylor (1988) – Measured	7.7×10^{-4}
Taylor (1988) - 95% UCL	8.9×10^{-3}

For the reasons discussed throughout these comments, GE believes that cancer slope factors calculated from epidemiological studies can be used to establish environmental standards, including a water quality standard for PCBs. Given that Taylor (1988) is the largest epidemiological study performed to date and is highly relevant to the Hudson River, GE recommends using the measured cancer slope factor from this study as the starting point for establishing environmental standards for PCBs based on cancer risk. As discussed above, the workers studied in Taylor (1988) were exposed primarily to Aroclor 1242 and 1254, with minor exposure to Aroclor 1016. Thus, it is conservative to use the measured cancer slope factor from Taylor (1988) as the cancer slope factor for Aroclor 1242.

Accordingly, General Electric proposes that the SOW for the Hudson River Phase 2 Risk Assessment use a CSF of 7.7×10^{-4} $(mg/kg/day)^{-1}$.

B.4 Summary for cancer toxicity assessment

Although the Reassessment was a positive step in reevaluating cancer risk from exposure to PCBs, GE strongly believes that EPA should use the numerous epidemiological studies that have been performed to date to further assess the true human cancer risk of PCBs. EPA can no longer ignore the many clinical and epidemiological studies that do not support the proposition that PCBs cause cancer in humans. Although

toxicologists must be careful in relying on the results of negative epidemiological studies, when several excellent epidemiological studies have been performed using large numbers of workers heavily exposed to a chemical over a long period of time, and the results of those studies have been negative, such results cannot be ignored.

These comments provide a scientifically valid rationale for using epidemiological studies rather than rodent studies to establish environmental standards for PCBs. They also provide an assessment of the adequacy of the PCB epidemiological studies for evaluating the human cancer risk of PCBs and set forth overall conclusions that can be drawn from those studies using a "weight of the evidence" approach. These comments have further shown how the studies can be used to derive a conservative cancer slope factor for PCBs. GE strongly urges the Agency to use the opportunity presented by the HHRA to consider this matter more thoroughly.

C. Noncancer toxicity values

The noncancer reference dose (RfD) cited in the SOW is flawed and overly conservative, in that it is based on an inappropriate monkey study and overly conservative uncertainty factors. In the SOW, EPA plans to base the noncancer risk assessment using the oral RfD of 2×10^{-5} mg/kg-day (20 ng/kg-day) for Aroclor 1254. This RfD is based on dermal, ocular and immunologic effects in a series of studies of rhesus monkeys reported by Arnold et al. (1993a,b) and Tryphonas et al. (1989; 1991a,b).

The Aroclor 1254 reference dose (RfD) is based on the results of a five year feeding study in rhesus monkeys (Arnold et al., 1993a,b; Tryphonas et al., 1989; Tryphonas et al., 1991a,b). Groups of 16 adult female monkeys ingested gelatin capsules containing Aroclor 1254 (in glycerol, corn oil vehicle) at daily doses of 0, 5, 20, 40, or 80 ug/kg-day for over five years. PCB concentrations in the monkeys had achieved steady state pharmacokinetics by 25 months of exposure, as demonstrated by PCB measurements in blood and adipose tissue (Tryphonas et al., 1989; Mes et al., 1989). The general health and clinical pathology findings in the adult female monkeys dosed with Aroclor 1254 were

reported by Arnold et al. (1993a,b). Clinical signs of toxicity were limited to eye exudate, inflammation, and/or prominence of the tarsal (Meibomian) glands, and changes in finger and toe nails. Significant dose related trends were reported for these clinical signs (Arnold et al., 1993a). The results of an immunologic assessment of the PCB exposed adult female monkeys was reported by Tryphonas et al. (1989, 1991a,b). The most significant finding was a treatment-related decrease in antibody response (IgG, IgM) to sheep red blood cells (SRBC). The LOAEL for clinical signs and immune system effects was 5×10^{-3} mg/kg-day. A total UF of 300 was applied (ten for sensitive individuals, three for interspecies extrapolation, three for minimal effect LOAEL to NOAEL, three for subchronic to chronic study duration) and a RfD of 2×10^{-5} mg/kg-day was calculated.

A number of questions can be raised about EPA's selection and evaluation of the studies from which the Aroclor 1254 RfD was derived. Two critical shortcomings are most important. First, those studies are not appropriate for deriving an RfD because: (a) the clinical relevance of the immunologic changes reported by the researchers has not been demonstrated; (b) the rhesus monkey is not an appropriate model for dermal, ocular, and nail effects of PCBs in humans; and (c) there is compelling evidence to indicate that rhesus monkeys metabolize PCBs in a significantly different way from humans. Second, EPA used inappropriate and overly conservative uncertainty factors in extrapolating from this set of studies to derive an RfD for humans. These points are explained in greater detail in a memorandum prepared by Dr. Russell Keenan and Ms. Carol Gillis, then at ChemRisk, which was attached as Exhibit 96 to GE's May 1, 1998 comments on EPA's proposal to list the Housatonic River site on the National Priorities List (GE, 1998) and is incorporated by reference herein.

Despite changes in immunologic parameters reported by the researchers, clinical relevance of these changes has not been demonstrated. In addition, the rhesus monkey is not an appropriate model for dermal, ocular and nail effects of PCBs in humans. A comparison of effects and body burdens (blood serum levels) seen in workers exposed to PCBs and the effects and associated body burdens reported in rhesus monkey studies indicates that PCBs produce nail changes, ocular effects, and dermal effects at much lower

doses in rhesus monkeys than in humans (Gillis and Price, 1996). In fact, PCBs alone do not produce nail changes, chloracne or ocular effects in humans as demonstrated by the fact that the length of exposures in epidemiological studies would have provided adequate opportunity to demonstrate these effects if they were occurring. These findings suggest that rhesus monkeys are significantly more sensitive and respond differently to PCBs than humans.

More importantly, there is compelling evidence to indicate that PCBs are metabolized differently in humans than in rhesus monkeys and that the metabolism of PCBs may be critical to the overall expression of PCB toxicity (Brown, 1994; Brown et al., 1994). One indication of this difference is the line of evidence suggesting that the patterns of PCB congeners that accumulate in adipose and hepatic tissues of rhesus monkeys chronically exposed to Aroclor 1254 differ from patterns of congener retention in humans exposed to PCBs. Humans produce a retention pattern similar to that observed in *in vitro* studies of P4502B enzyme activity, referred to as P4502B-like metabolism (Brown et al., 1989; 1994). Humans produce a second pattern when exposed to mixtures of PCBs and furans (Masuda et al., 1978; Kunita et al., 1984). This pattern results from metabolism of PCBs by a combination of P4502B-like and P4501A-mediated metabolic pathways. It appears that, in the absence of concurrent exposures to dioxins and furans, PCBs do not induce the P4501A enzymes in humans. Furthermore, studies of PCB induction of P4501A in rodents suggest that such induction, if it occurs in humans, would require exposures of PCBs far higher than have occurred from environmental or historical occupational exposures (Brown et al., 1991).

In contrast to the metabolism of PCBs in humans (in the absence of concurrent exposures to dioxins and furans in the toxic range), a different pattern is observed in rhesus monkeys. Metabolism patterns of PCBs in rhesus monkeys indicate that PCBs are metabolized by means of the P4501A pathway and a second pathway known as the P450RH pathway, which appears to be unique to the rhesus monkey (Brown, 1994). The specific enzymes responsible for metabolizing PCBs in the unusual P450RH pattern observed in monkeys are unclear at this time. However, the differences in enzyme systems

support the finding that PCBs are metabolized differently in rhesus monkeys and humans, and suggest that the rhesus monkey is a poor model for endpoints associated with the activation of P4501A.

Several studies have demonstrated that PCB metabolism is critical to the expression of PCB toxicity in humans (Brown, 1994; Brown et al., 1989; 1991; 1994). For example, induction of P4501A at low PCB doses is associated with dermal, ocular, and nail effects in animals (Brown et al., 1994). In humans, Yusho victims, who were exposed to both PCBs and furans and experienced many of these effects, also displayed P4501A metabolism. Conversely, metabolism of PCBs under the P4502B-like pathway in occupationally exposed human populations is not associated with these effects. In summary, the findings on the metabolism of PCBs suggest that the differences between rhesus monkeys and humans with respect to PCB toxicology may extend beyond dermal, ocular, nail and immunological effects.

In addition to EPA's improper selection of the critical study and toxicological endpoints for establishing the RfD, EPA applied inappropriate uncertainty factors for quantitatively deriving the RfD. According to EPA, an uncertainty factor of three was applied to account for interspecies extrapolation due to the similarities in toxic responses and metabolism of PCBs in monkeys and humans and the general physiologic similarities between the species. This uncertainty factor implies that humans are three times more sensitive than the rhesus monkey for the critical effects on which the RfD is based. However, because the evidence indicates that the rhesus monkey is significantly more sensitive than humans, EPA's uncertainty factor of three to account for interspecies sensitivity is inappropriate. An uncertainty factor equal to or less than one would be more appropriate to address interspecies uncertainty.

In addition, EPA applied an uncertainty factor of three to adjust for study duration. This factor is intended to account for uncertainties related to less-than-chronic exposure and the assumption that a longer exposure duration may result in more pronounced adverse effects as adverse effects at a low dose. In the case of Aroclor 1254, the monkeys were

dosed for greater than 25 percent of their lifetime and steady-state PCB body burdens were achieved (Arnold et al., 1993a,b). This suggests that a longer exposure duration would not result in an increased toxic response. Thus, an uncertainty factor of three is not warranted and should be reduced to a factor of one. In summary, reduction of the uncertainty factors for interspecies sensitivity and study duration from a value of three to one results in a total uncertainty factor of 30, rather than 300. Even if one were to accept EPA's selection of the toxicological study and agree with EPA's evaluation of the critical effects, the necessary adjustments to the total uncertainty factor would argue for a revised chronic RfD no more stringent than 200 ng/kg-day.

D. Endocrine disruption

The SOW currently plans to evaluate "the potential for endocrine effects" in the HHRA. GE disagrees with the need for conducting this evaluation, even if it is limited to "a qualitative assessment of the currently available information on the potential effects of PCBs on the endocrine system," as can be inferred from the SOW. We believe this analysis is unwarranted in light of recent EPA findings on this topic as well as those of independent scientific researchers. For example, it has been suggested that PCBs can interfere with normal endocrine function leading to infertility and other hormone related disorders, although recent reviews suggest that the evidence for these effects is weak and circumstantial (Danse et al., 1997; Golden et al., 1998). Indeed, EPA (1997a) concluded that, with few exceptions (for compounds unrelated to PCBs), "an adverse health effect in humans via endocrine disruption has not been established."

Last year, in what many in the scientific world describe as a definitive result, Hunter et al. (1997) published in the *New England Journal of Medicine* a study focused on the interaction of endocrine disruption and cancer. The study showed no link between PCB exposure and breast cancer. Similar results were reported by Key and Reeves (1994). As Dr. Steven Safe noted in an editorial accompanying Hunter et al. (1997), this study and others "should reassure the public that weakly estrogenic organochlorine compounds such as PCBs, DDT, and DDE are not a cause of breast cancer." (Safe 1997).

GE believes that further qualitative or quantitative evaluation of this topic is not necessary or appropriate in the Hudson River HHRA. As EPA and independent scientific researchers have been unable to find a link between PCB exposure and endocrine disruption, it is not worthwhile to invest the level of effort and resources necessary to further elucidate this topic in the context of a Superfund site risk assessment. Furthermore, by raising it as a potential issue in the Hudson River HHRA, which is itself based on a hypothetical exposure scenario, undue alarm or concern may be generated. This would not serve the public interest.

E. Uncertainty in toxicological criteria

The SOW states the Agency does not intend to evaluate the uncertainty in the dose response criteria (the RfD and the CSF) in the human health assessment (SOW, at 14). The basis for this approach is purportedly consistent "with EPA's policies (EPA, 1997a,b)." These documents are not a statement of the limits of Agency policy. Rather they are a set of guiding principles for the use and evaluation of Monte Carlo modeling. The relevant passage in the documents is as follows:

> *For human health risk assessments, the application of Monte Carlo, and other probabilistic techniques has been limited to exposure assessments in the majority of cases. The current policy, Conditions for Acceptance and associated guiding principles are not intended to apply to dose response evaluations for human health risk assessments until this application of probabilistic analysis has been studied further.* (EPA 1997a, at 2)

It is clear that the EPA's current policy is limited to the evaluation of exposure assessments in human health risk assessments, and that the Agency does not provide the assessor with guidance for the evaluation of Monte Carlo assessments of dose response (toxicity).[11] This is not the same as a policy that forbids the consideration of quantitative

[11] See the identical language in U.S.EPA's Guiding Principals for Monte Carlo Analysis, (EPA, 1997b).

modeling of the uncertainty in dose response. It merely is a statement that the current policy does not address this type of analysis and that the Agency is not offering guidance on the evaluation of this practice.

Therefore, there is no basis for concluding that the uncertainty in toxicity measurements can be set aside or ignored in the evaluation of the uncertainty in risk estimates. GE is unaware of any legal or technical reason to exclude the uncertainty in toxicity criteria. In fact, the Agency's own technical experts have advocated that the Agency consider the uncertainty in toxicity on a case-by-case basis (SAP, 1998).

GE believes that it is critical to account for the uncertainty in toxicity measurements. It has long been recognized that the toxicity portion of the risk assessment, not the exposure assessment, is the greatest source of uncertainty in risk assessment (McKone and Bogan, 1991). Thus, the decision to exclude consideration of such uncertainty has grave impacts on the assessment of risks.

Given the importance of this issue, the only basis for excluding consideration of uncertainty from the HRA is technical infeasibility. However, a number of technical approaches have been suggested for characterizing uncertainty in toxicity criteria. The uncertainty in cancer slope factors has been investigated by several authors (Crouch et al. 1996; Evans et al. 1996a,b). Uncertainty in the RfD has also be the subject of a number of research publications (Slob, 1997; Baird et al. 1996; Price et al., 1997, and Swartout et al. 1998). Swartout et al. (1998) has established a framework for redefining the RfD in probabilistic terms.

Techniques for the integration of uncertainty in toxicity assessments into assessment of exposure uncertainty have also been developed (Carlson-Lynch et al. 1997; Harvey, et al. 1997; Price et al. 1998). In Price et al. (1998), a Monte Carlo model was constructed of the uncertainty and variation in the PCB dose rates of anglers consuming fish from the Tennessee and Clinch Rivers. This model was combined with information on the uncertainty in the toxicity of PCBs to estimate the probability of exceeding the actual

dose that is "protective of sensitive individuals". The result is an assessment that fully discloses the uncertainty in the risk characterization for PCB noncarcinogenic effects and provides the risk manager with the most appropriate basis for decision making.

As discussed elsewhere, the Agency's approach for Monte Carlo analysis is not entirely clear, however, at several points the Agency has indicated that uncertainty will be explicitly modeled (SOW, at 10, 13). Therefore, there appears to be no technical reason why information on uncertainty on the PCB toxicity criteria cannot be evaluated with the uncertainty in exposure.

F. Averaging time

The SOW does not discuss the issue of averaging time. Averaging time is a term in the equation to estimate the average daily dose rate that is used to evaluate non-carcinogenic effects (EPA, 1989). When the dose rate received from a source of contamination is constant over time, the issue of averaging time does not affect the estimate of average daily dose. However, when the dose rate changes over time, the choice of the duration for the averaging time can have a dramatic impact on the estimate of daily dose (Muir et al. 1998). Since the proposed model will address changes in fish tissue concentrations over time, the selection of an averaging time is an important issue for the HHRA.

GE recommends that the averaging time be established based on the half-life of PCBs in humans. Lipophilic compounds exert their chronic effect as a function of the long-term body burden of the compounds. Studies of PCBs in test animals determine doses that are approximately in equilibrium with the body burdens that are associated with the presence or absence of long tem effects. Therefore, it is important that the averaging period is sufficiently long that the dose can come into some sort of equilibrium with the body burdens of the anglers. This suggests that the averaging time be several multiples of the half-life of PCBs in humans. At a minimum, the averaging time should be greater than 10 years.

SECTION V
RISK CHARACTERIZATION ISSUES

A. Consideration of background sources of PCBs

PCBs were used in consumer and commercial products from the 1940s through the 1970s. Because of this widespread use and the persistence of the compounds, PCBs still occur at trace levels in many foods and in many households. As a result, all individuals carry trace levels of PCBs in their bodies (ATSDR, 1997).

One criteria in risk management is to determine how exposures from a particular source relate to the background levels and whether exposure from a certain source will significantly raise an individual's body burdens. For example, if an individual has 2 ppb of a contaminant in his or her blood because of background sources, and exposure at a Superfund Site raises the blood level by 0.01 ppb, then there is likely to be little health benefit to the individual from the control of contamination at the Site. If the exposures at the site doubles or triples the individual's body burden then the potential for causing adverse effects is much higher and the control of the site may be warranted.

The impact of a source on background body burdens can be investigated directly by surveying the blood levels of individuals exposed by certain routes and comparing the results to levels in unexposed populations. Such studies have been performed for exposure to PCBs in soil (Chase et al., 1989; ATSDR, 1987) and exposure from the consumption of fish in the Great Lakes, Tennessee River (ATSDR, 1997b), and in Western Massachusetts (Housatonic River). In the latter two studies, the individuals who consumed fish did not have elevated blood levels of PCBs.

Such direct studies cannot be performed for the Hudson River since the ban on keeping fish has eliminated exposures from fish consumption. However, it is possible to take the output of the exposure models proposed in the SOW and determine the incremental change that consumption would cause in background levels of PCB

(Avantaggio et al. 1997; Keenan et al. 1997). Such an analysis would provide EPA with information on whether the control of PCBs from the consumption of fish would have a significant impact on angler's body burdens.

B. Development of the central and high-end exposure risk estimates

The Agency has indicated that risk management decisions will be made based on the current and future risks to average exposed individual and to the reasonably maximally exposed individual (RMEI) (SOW, at 2). These two risk measurements are proposed to be based on the exposure assessment that consists of a deterministic and probabilistic analysis (SOW, at 6). Thus, EPA is proposing to use two estimates of risk for the average individual and RMEI. The SOW does not clearly state how the two estimates will be used in the risk characterization process.

Deterministic assessments have been historically used at Superfund sites to consider the need to perform remediation and to select a remedial option. At most small sites the deterministic baseline risk assessment would be representative of the site risks. For larger and more complex sites with dynamic physical features (like the Hudson River), a deterministic model is inadequate even to describe "typical" conditions. Probabilistic models, such as Monte Carlo models, are more appropriate in such cases since they have a better chance to capture the variability and the uncertainty which is inherent as sites become more complex.

Consequently, EPA should abandon its proposal to use the findings of the deterministic exposure assessment. As discussed in EPA's Guiding Principles on Monte Carlo (EPA, 1997), the use of probabilistic techniques provides the decision maker with additional insights that the deterministic methods cannot provide. Therefore, the assessment of risks to the RMEI and average individual should be based only on the findings of the probabilistic analysis.

C. The baseline assessment cannot be used to select remedial options.

The baseline risk assessment cannot be used to assess the reduction of risk associated with the implementation of a remedial strategy. Rather, the baseline HHRA examines the hypothetical risk associated with the "no action" scenario. These risks must be measured against the estimated risks that might remain after implementation of various remedial options in order to assess the appropriateness of such actions. Therefore, a separate risk reduction analysis should be completed for each remedial option to focus on the net reduction in risks, if any, associated with the different remedial options.

D. Use of risk finding for small number of anglers fishing hot spots

The SOW describes the assessments of risks associated with fish caught in hot spots as one of the analyses that will be performed (SOW, at 13). However, the procedure to evaluate the results of the risk assessment — i.e., how it will be used in the risk management and feasibility study remedial alternatives assessment — is not presented in the SOW. One area of possible misinterpretation is the extreme upper limit risks that will likely be calculated for the localized areas of elevated contamination. It is important that these risks be put into proper context in the overall risk. This assessment should include a discussion of the areal extent of the elevated concentrations, probability of a suitable fishery, probability of anglers in the same area, angler success, and other relevant factors.

E. Choice of "start date"

According to the SOW, the baseline risk assessment will focus on a population of anglers who begin fishing in 1999 (SOW, at 11). The selection of this date may not be appropriate for the baseline assessment. As discussed above, a separate risk assessment will be needed to evaluate the risks that might remain after various remedial options. These risks will not exist until the remediation has been completed. Given the current schedule for the Hudson River reassessment, this could happen no earlier than 2002 to 2005. The data used in the remedial decision-making process should represent the

conditions that best represent the risks beginning at that time. In order to be consistent with the remedial assessment, the Agency should start the baseline risk assessment clock beginning in 2002 not 1999.

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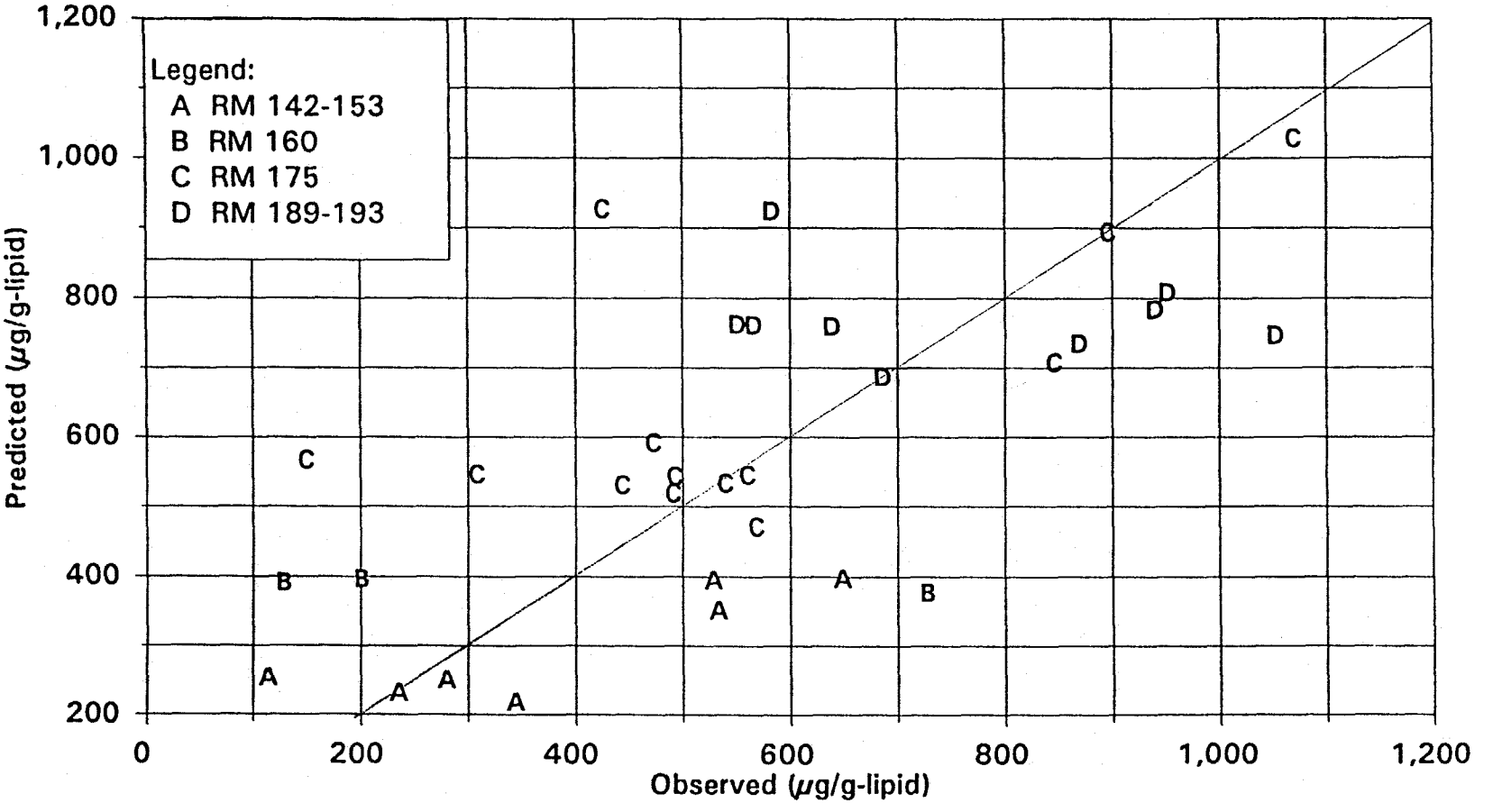
Figure 9-12
Comparison of Observed and Predicted Aroclor 1254 Concentrations in
Hudson River Largemouth Bass (Corrected to NYSDEC 1983 Quantitation Basis)



Source: TAMS/Gradient Database, Release 3.1

10.1896

Figure 9-12 from EPA, 1996. Preliminary Model Calibration Report.

NYSDEC RIVER ENFORCEMENT SUMMARY OF THE CATCH AND RELEASE FISHING PROGRAM (8/31/95 - 7/31/98)

VIOLATION TYPE	TICKETS ISSUED	WARNINGS ISSUED	TOTAL
KEEPING FISH	9	3	12
BAIT FISH/TIP-UPS	5	74	79
NO LICENSE	72	93	165
NAVIGATIONAL/ OTHER	40	28	68
TOTALS	126	198	324
TOTAL FISHERMEN CHECKED TO DATE	**1437**		

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Determining The Intake Of Upper Hudson River Fish By Species

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Determining the Intake of Upper Hudson River Fish by Species

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1.0 INTRODUCTION

In 1991, EPA issued a Phase I Report for the Reassessment RI/FS in which the Agency evaluated the potential human health risks for the Hudson River Superfund site. In this report, EPA (1991) determined that any risks to human health from PCBs in sediment occur through indirect exposure through the fish consumption pathway. Under Superfund Guidance, (EPA, 1989) evaluation of such exposures are specifically required not to consider the impact of any fishing regulations. However, fishing restrictions have been imposed by the State of New York, and thus the estimates of PCB exposure developed for the Upper Hudson River are hypothetical and an overestimation of actual exposures.

In the Phase I risk assessment, EPA (1991) acknowledged that New York anglers do not spend an equal amount of time fishing for each species. Instead, a large majority of time is spent fishing for bass, brown trout, and walleye (NYSDEC, 1990). Although EPA (1991) realized that the NYSDEC information did not specifically reflect fishing preferences of Hudson River anglers, the Agency believed that the statewide values generally reflected efforts on the Hudson. In spite of the availability of species preference data from the New York survey, EPA chose to average the PCB concentration data from all species sampled from the Hudson River to determine a single point estimate (95th percentile) of PCB concentration in fish tissue. EPA defended this decision citing the lack of appropriate sampling data for the species that could be eaten by anglers. Because specific PCB concentration data could not be defined for all species, EPA decided to include the available sampling data on all species of fish in the analysis. In addition, EPA stated that there were not sufficient differences in the PCB levels reported in the various species to warrant a species-specific evaluation of PCB levels in fish.

In the Final Phase 2 Work Plan and Sampling Plan, EPA (1992) stated that it would reevaluate the decision made in the Phase 1 document and possibly refine the estimates of exposure point PCB concentrations in fish to reflect interspecies variability and anglers' preferences for different species. Since the release of the Phase 1 document, additional sampling data on a greater number of species have been collected. In addition, several studies have been released which provide data to support the fact that anglers do select certain species in both their catch and in their consumption habits. This information combined with data on fish species presented in the Phase 1 document can be used to identify the major edible species selected by Hudson River anglers. This paper

presents a discussion of the technical and regulatory issues associated with the determination of the species preferences of Upper Hudson River anglers.

2.0 EXPOSURE CONCENTRATIONS FROM UPPER HUDSON RIVER FISH

Fish PCB Levels

Over 50 different species are known to be present in the Hudson River between Federal Dam and Fort Edward (Malcolm Pirnie, 1984). Earlier sampling efforts were focused on those species that were likely to be consumed by recreational anglers, such as bass, the most desirable game fish in the Hudson River (NYSDEC, 1990). However, recent sampling efforts have been expanded to include other less desirable species.

An examination of the recent sampling data indicates that PCB tissue levels in some species vary over a large range (Table 1). For example, the levels of PCBs in goldfish are estimated to be 20 times greater than levels found in pumpkinseed fish. The variation observed in PCB concentrations in fish is likely affected by the amount of lipid content of the fish. PCBs are highly lipophilic and tend to accumulate in those species with a higher fat content, such as goldfish, carp, or American eel (EPA, 1991). Although fish with more lipids will generally have higher PCB concentrations, there will be variations in individual PCB tissue levels due to the natural distribution within species.

Angler Preferences

Recent studies by the New York State Department of Environmental Conservation (NYSDEC) indicate that New York anglers preferentially select for certain species in both fishing effort and consumption (NYSDEC, 1990; Connelly et al., 1992). Many species (e.g., goldfish, carp) are not desirable sport species and are not likely to be consumed by anglers even if they are caught. In most cases, anglers preferentially fish for and consume species that have low lipid contents and which consequently accumulate lower levels of PCBs. Preferential selection of species by anglers is further supported by a mail recall survey conducted on Maine anglers (ChemRisk, 1992). This survey identified over 15 different species that were caught and consumed by recreational anglers:

Table 1. PCB Concentrations in Selected Species of Fish

	Average PCB Level 1975 - 1988[a]
Carp (goldfish)	137 ppm
White Perch	42 ppm
Bass (largemouth)	27 ppm
Pumpkinseed	10 ppm

a. Data from NYSDEC (1990).

however, over 85% of these fish were represented by only three species. The total intake of PCBs by recreational anglers is therefore, dependent on the concentration of PCBs in only a few select species and not the entire range of PCB concentrations recorded from all species.

3.0 SELECTION OF FISH SPECIES

All species found in the Hudson River will not be consumed by recreational anglers since only a small percentage of the species are considered desirable game fish. Information on species preferences specific to the Hudson is unavailable. However, based on data from Connelly et al. (1992), it is possible to identify species preferences among New York anglers that can be used as a surrogate for Hudson River anglers. Specifically, Connelly et al. (1992) surveyed 2,000 fishing license holders for the year beginning October 1990 and ending on September 30, 1991. Although the survey focused on assessing angler knowledge of the fish health advisories, the survey also was designed to "describe fishing behaviors (e.g., species, waterways) and fish consuming behaviors (e.g., species, preparation techniques used) of licensed anglers." Survey participants provided detailed information on the locations they fished in New York State, the number of fish caught, and the number of fish meals eaten from each of these locations.

An analysis of the data from Connelly et al. (1992) was conducted to select the appropriate information. Because many rivers in New York State are characterized as cold water and fast moving or are stocked with cold water species (e.g., trout), whereas the Upper Hudson is a cool to warm water stream with much slower flow, some of the survey results are not applicable to the Hudson River. Rivers and streams classified by New York State as warm water are likely to contain species similar to the Hudson River. These rivers were identified based on fishing data from New York State and discussions with regional fishery personnel (Table 2). Using the rivers and streams identified in Table 2, a distribution of fish species eaten by New York anglers was determined using appropriate portions of the results of the Connelly et al. survey (Table 3). Although the calculations used to arrive at the values in Table 3 are not presented in this issue paper, they can be provided at a later date.

Although chinook and coho salmon, rainbow trout, and brown trout are not expected to be caught by Upper Hudson River anglers, due to their preference for fast moving, cold waters, these

Table 2. New York State Warm Rivers and Streams Similar to the Upper Hudson

Name	County	Region	Potential Species Present
Allegheny river	Cattaraugus	9	smallmouth bass, muskellunge, northern pike, walleye, catfish, pan fish[a]
Batten Kill river	Washington	5	brown and brook trout
Black river	Lewis	6	largemouth and smallmouth bass, northern pike, walleye, pan fish, bullhead
Buttermut creek - 2	Otsego	4	smallmouth bass, walleye, pickerel, trout
Buttermut creek - 1	Onondaga	7	brown trout, walleye
Chemung river	Chemung	8	largemouth and smallmouth bass, walleye, pickerel, pan fish, bullhead
Chemung river	Steuben	8	largemouth and smallmouth bass, walleye, pickerel, pan fish, bullhead
Chenango river	Broome	7	largemouth and smallmouth bass, northern pike, walleye, pickerel, pan fish, bullhead
Chenango river	Chenango	7	largemouth and smallmouth bass, northern pike, walleye, pickerel, pan fish, bullhead
Chittenango creek	Madison	7	largemouth and smallmouth bass, northern pike, walleye, pickerel, pan fish, bullhead
Chittenango creek	Onondaga	7	largemouth and smallmouth bass, northern pike, walleye, pickerel, pan fish, bullhead
Delaware river	Delaware	4	smallmouth bass, pickerel, walleye, yellow perch, bullhead, pan fish
Delaware river	Orange	3	smallmouth bass, chain pickerel, walleye
Delaware river	Sullivan	3	smallmouth bass, walleye, yellow perch, bullhead, pan fish
East Branch Delaware river	Delaware	4	smallmouth bass, pickerel, walleye, yellow perch, bullhead, pan fish
Genesee river	Livingston	8	smallmouth bass, pan fish, bullhead
Genesee river	Monroe	8	smallmouth bass, rainbow and brown trout, pan fish, bullhead
Genesee river	Wyoming	9	bass, walleye, panfish
Hudson river	Warren	5	bass, pike
Lower Genesee river	Monroe	8	smallmouth bass, walleye, salmon, rainbow trout, steelhead
Mohawk river/barge canal	Herkimer	6	largemouth and smallmouth bass, tiger muskellunge, walleye
Mohawk river/barge canal	Montgomery	4	largemouth and smallmouth bass, tiger muskellunge, walleye
Mohawk river/barge canal	Oneida	6	largemouth and smallmouth bass, tiger muskellunge, walleye
Mohawk river/barge canal	Saratoga	5	largemouth and smallmouth bass, tiger muskellunge, walleye
Mohawk river/barge canal	Schenectady	4	largemouth and smallmouth bass, tiger muskellunge, walleye
Neversink river	Orange	3	smallmouth bass, brown trout
Oak Orchard creek	Genesee	8	bass, pike, salmon
Oswego river	Onondaga	7	walleye, catfish, carp, bass, sunfish

[a] Panfish include

Table 2. New York State Warm Rivers and Streams Similar to the Upper Hudson (cont'd)

Name	County	Region	Potential Species Present
Ramapo river	Orange	3	walleye, panfish, brown trout, rainbow trout
Raquette river	Franklin	5	smallmouth bass, northern pike, walleye, trout, panfish
Raquette river	St. Lawrence	6	smallmouth bass, northern pike, walleye, pan fish
Sandy creek - 1	Jefferson	6	smallmouth bass, northern pike, trout
Schoharie creek	Montgomery	4	smallmouth bass, walleye, pan fish
Schoharie creek	Schenectady	4	smallmouth bass, walleye, pan fish
Schoharie creek	Schoharie	4	smallmouth bass, walleye, pan fish
Schroon river	Warren	5	largemouth and smallmouth bass, northern pike
Seneca river	Seneca	8	largemouth and smallmouth bass, northern pike, walleye, pan fish, bullhead
Seneca river	Cayuga	7	largemouth and smallmouth bass, northern pike, walleye, pan fish, bullhead
Seneca river	Onondaga	7	largemouth and smallmouth bass, northern pike, walleye, pan fish, bullhead
Susquehanna river	Delaware	4	largemouth and smallmouth bass, northern pike, tiger musky, pickerel, pan fish, catfish
Susquehanna river	Otsego	4	largemouth and smallmouth bass, northern pike, tiger musky, pickerel, walleye, pan fish, catfish
Susquehanna river	Broome	7	largemouth and smallmouth bass, northern pike, walleye, pickerel, pan fish
Susquehanna river	Chenango	7	largemouth and smallmouth bass, northern pike, walleye, pickerel, pan fish
Susquehanna river	Tioga	7	largemouth and smallmouth bass, northern pike, walleye, pickerel, pan fish
Tonawanda creek	Genesee	8	largemouth and smallmouth bass, northern pike, walleye
Tonawanda creek	Erie	9	largemouth and smallmouth bass, northern pike, walleye
Tonawanda creek	Niagara	9	largemouth and smallmouth bass, northern pike, walleye, pan fish, bullhead
Tonawanda creek	Wyoming	9	largemouth and smallmouth bass, northern pike, walleye, pan fish, bullhead
Wallkill river	Orange	3	smallmouth bass, bullhead, pan fish
Wallkill river	Ulster	3	large and smallmouth bass, bullhead, pan fish, chain pickerel
West Branch Delaware river	Delaware	4	smallmouth bass, pickerel, walleye, yellow perch, bullhead, pan fish
West Branch Delaware river	Broome	7	smallmouth bass, pickerel, walleye, yellow perch, bullhead, pan fish

Table 3. Fish Species Distribution for Hudson - Like Rivers and Streams[a]

Species	Percent Meals Eaten
American Eel	0.9
Bass	17.4
Brown Bullhead	9.2
Brown Trout	27.8
Carp	0
Channel Catfish	0.5
Chinook Salmon	1.4
Coho Salmon	1.8
Lake Trout	0
Rainbow Trout	9.8
Walleye	7.5
White Perch	4.5
Other	19.1

a. Connelly et al. (1992)

species appear in Table 3. Their appearance may be due to either erroneous information provided by the survey respondents or the inclusion of rivers classified as Hudson-like that contain limited cold water sections. These cold water sections could contained salmonid species, however, the Hudson River contains no cold water sections and therefore will not contain salmonids. Data in NYSDEC (1990) indicate that the fishing effort in the Upper Hudson is primarily directed toward bass. Based upon this information, it is reasonable to assume that in the absence of good fishing opportunities for chinook and coho salmon, rainbow trout, and brown trout, anglers would instead fish for bass. Therefore, the percent meals eaten for these four species have been included in the percent meals eaten for bass (Table 4).

Table 3 also indicates that a significant percentage of fish meals eaten by New York anglers fall into an "other" category. Connelly et al. (1992) did not provide a method for respondents to identify species caught or eaten, but not specifically listed on the survey. Consequently, those species were attributed to the "other" category. Based on information contained in the Phase I document (EPA, 1991), NYSDEC (1990), and sampling data collected by NYSDEC, bluegill, rock bass, pumpkinseed, black crappie, northern pike, chain pickerel, and yellow perch are the most likely species that would fit within the "other" category for the Hudson. The "other" category percentage derived from Connelly et al. (1992) was divided evenly among these seven species.

Using this approach, a distribution of species preferences based on meals eaten can be identified that represents the distribution of species that would be eaten from the Upper Hudson River (Table 4). Although a distribution of species caught is also available (Connelly et al., 1992), a distribution of species eaten is more appropriately applied to an evaluation of exposure because many species caught by anglers are not eaten. For example, although a small number of carp were caught by anglers surveyed by Connelly et al., the carp were not eaten and are not included as desirable fish species for the Upper Hudson River. Instead, most anglers prefer bass, as indicated by the high percent consumption value of 58%. In addition, it is likely that anglers will consume a small number of bullhead, walleye, white perch, and other sunfish. EPA has reported PCB levels for most of the species listed in Table 4. However, in the absence of species-specific PCB data, concentration data for a similar species could be substituted. For example, PCB concentrations collected for bullhead are an appropriate surrogate for channel catfish, for which there is no PCB concentration data.

Table 4. Fish Species Distribution for Hudson River[a]

Species	Percent Meals Eaten
American Eel	0.9
Bass	58.2
Bullhead	9.7
Walleye	7.5
White Perch	4.5
Bluegill	2.7
Rock bass	2.7
Pumpkinseed	2.7
Black Crappie	2.7
Northern Pike	2.7
Chain Pickerel	2.7
Yellow Perch	2.7

a. Based on Connelly et al. (1992) and NYSDEC (1990).

4.0 CONCLUSIONS

Surveys conducted by NYSDEC (1990) and Connelly et al. (1992) to characterize the fishing behavior of New York State anglers clearly indicate that contrary to statements made by EPA (1991) in the Phase I Reassessment, fisherman do not eat all fish in equal amounts. Instead, anglers preferentially select for species in both catch and consumption. Sampling data collected to characterize PCB levels in fish also indicate that all fish do not contain the similar PCB levels. Therefore, the intake of PCBs is highly dependent on the species selected, and an accurate estimate of the risks to anglers from fish consumption should include species preferences.

The most appropriate method to incorporate the species selection of anglers is through the use of a probabilistic exposure assessment using synthetic life history or Microexposure Monte Carlo analysis. This type of analysis can account for species selection as well as the variations in PCB levels between fish species. Specifically, a Microexposure Monte Carlo analysis can identify a fish species and an associated PCB level, for each meal, based on the percent consumption identified in Connelly et al. (1992).

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ESTIMATING EXPOSURE DURATION FOR UPPER HUDSON RIVER RISK ASSESSMENT

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ESTIMATING EXPOSURE DURATION FOR UPPER HUDSON RIVER RISK ASSESSMENT

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Estimating Exposure Duration for Upper Hudson River Risk Assessment

1.0 INTRODUCTION

In 1991, EPA issued a Phase I Report for the Reassessment RI/FS in which the Agency evaluated the potential human health risks for the Hudson River Superfund site. In this report, EPA (1991) determined that any risks to human health from PCBs in sediment occur through indirect exposure through the fish consumption pathway. Under Superfund Guidance, (EPA, 1989) evaluation of such exposures are specifically required not to consider the impact of any fishing regulations. However, fishing restrictions have been imposed by the State of New York, and thus the estimates of PCB exposure developed for the Upper Hudson River are hypothetical and an overestimation of actual exposures.

In estimating potential risks to anglers who consume fish from the Hudson River (in the absence of fishing restrictions which are currently in place), the Agency assumed that the exposure (fish consumption) would occur for a period of 30 years (i.e., exposure duration). This information was used with other point estimates of exposure and chemical toxicity to estimate potential risk from consumption of fish containing PCBs. As described in GE's comments on the Phase I Report (EPA, 1991), the use of the "point-estimate" approach for estimating risk is extremely conservative and should be replaced with a more sophisticated probabilistic approach (i.e., Microexposure Monte Carlo Analysis). Furthermore, the assumed exposure duration of 30 years needs to be reevaluated. This paper presents a discussion of the technical and regulatory policy issues associated with determining the length of time during which individuals may potentially catch and consume fish from the Upper Hudson River.

Past EPA guidance dictated that an individual's potential exposure to any type of environmental chemical contamination could be assumed to occur over an average lifetime of 70 years (55 FR 8292). This conservative default value was used for most EPA risk assessments conducted during the 1980s, until EPA (1989) developed an alternative estimate of 30 years based on a 1983 survey by the US Bureau of Census on household occupancy times. EPA has stated that this current default value for exposure duration represents the 90th percentile for the number of years an individual is likely to reside at the same residence.

Residential mobility is an accurate predictor of exposure duration for many sources of contamination that occur in or near the home. An individual's potential exposure to indoor air

pollution or contaminated soil, air, and groundwater near their residence is a function of the amount of time spent at home. This exposure may conceivably continue throughout the individual's lifetime unless the person changes their residence.

However, the duration of time an individual remains in one residence may not be a reasonable predictor of the duration of angling from a particular waterbody. An individual may give up angling and not change their residence or may move to a nearby residence and keep fishing the same waterbody. Unlike other types of exposures which often result from proximity to the source, potential exposure from fishing must be actively sought and is only partially dependent on the location of an angler's current residence. Exposure from consuming recreationally caught fish will be most significant for those individuals who continue to fish the waterbody of concern regardless of their current residence. As a result other factors in addition to residential mobility must be considered when predicting the duration of exposure from fish consumption.

2.0 HUDSON RIVER EXPOSURE DURATION

A critical component of any risk assessment is estimating how long or how often an individual may be exposed to the chemical of potential concern. In the case of the Upper Hudson, GE proposes that the exposure duration be defined as the time an angler begins fishing (a practice having been prohibited in the past) and continuing until the angler no longer catches and consumes fish from the Hudson River. The point at which an angler stops fishing varies with the individual angler. Three factors influence the time when an angler stops fishing: (1) the probability that an individual will relocate from his/her current residence (mobility); (2) the probability that an individual will decide to no longer participate in the sport of fishing (angling cessation); and (3) the probability that an individual will die (mortality). The duration of exposure can only be properly estimated when these three factors are considered.

Mobility

When evaluating the influence of the mobility factor on exposure duration for fish consumption, it is necessary to go beyond a strict consideration of residential mobility because, as described above, changes in household location may not lead to changes in fishing behavior. Only when an

individual moves a sufficient distance will a change likely be made in preferred fishing locations. While interstate or U.S. regional mobility data could be used to estimate the number of individuals who give up fishing at a preferred fishing location (due to a significant move in distance), interstate moves (within state) that would also result in a change in angling practices also need to be considered. It is likely that the actual number of anglers who stop fishing at a specific location would be underestimated by relying on interstate or regional mobility data. County mobility, however, may be an appropriate surrogate for representing the probability that an individual gives up angling because he/she moves sufficiently far enough away. These data are available from the U.S. Bureau of Census (1988, 1991) which publishes information on the number of individuals who move out of a given county, but still remain within the same state.

It is recognized that an angler who merely moves from one county adjacent to the Hudson to another adjacent county along the River, may still fish in the same location. In this case, the use of intercounty mobility as a measure of the probability that an angler will discontinue fishing the Upper Hudson may underestimate that individual's exposure duration. However, this underestimate may be balanced by the intracounty moves that actually result in a change in fishing location. In addition, because all areas of the Hudson River do not offer equal access, a move North or South along the river boundary may still move an individual away from their preferred fishing location (Hudson River Access Forum, 1989). This increase in distance from the preferred fishing spot may cause some anglers to choose another waterbody and not continue to fish the Hudson River.

Factors such as age, gender, and race can influence mobility. For example, the frequency of moving is highly dependent on age. Individuals between the ages of 20 to 29 have a greater probability of moving than individuals over 30. Gender also has an impact on mobility. Due to gender-specific tendencies, men are somewhat more likely to move than are women (U.S. Bureau of the Census, 1991). To account for these patterns and to identify the range of variability found in the angler population, it is necessary to identify a distribution of intercounty mobility rates for males and females of each age. Specifically, data on county mobility by age group and gender in the Northeast region are appropriate. Racial considerations do not need to be incorporated into the analysis because the race distribution in the counties along the Hudson as well as the race distribution of anglers in this area are similar to the distribution of races for the general population

Table 1 presents a distribution of the probability of an intercounty move that is age- and gender-specific.

Angling Cessation

In addition to moving, an angler may give up fishing due to lack of interest, bad weather, increasing age or a number of other reasons. In fact, at every age there is a certain probability that an individual will permanently give up the sport. However, due to the difficulty of collecting these data, no study has specifically evaluated this phenomenon. Not only is it difficult for individuals to predict whether they will give up fishing, individuals who report giving up fishing one year may only temporarily withdraw from the sport. These same individuals may start and stop fishing many times over the course of their lifetimes.

A survey in the State of Maine determined that 72 percent of all licensed anglers fish every year once they start fishing (Boyle et al., 1990). This study supports the fact that the majority of anglers are extremely dedicated to their sport, indicating that the number of anglers in the total state population should be relatively constant between years. This type of information can be used to determine the age-specific probability that an individual will permanently cease angling. A similar comparison of the number of anglers in New York to the total state population will identify the relative number of anglers at each age. The change in the number of anglers with increasing age can then be used to estimate the probability that an individual will give up angling.

As an example, an initial analysis using data collected in ChemRisk (1992) indicates that the percentage of anglers in the population increases from age 18 until the mid-20s, where it remains relatively constant for about 20 years. In the mid-40s until the late 60s angling begins to decline significantly. Finally, after about the age of 67, the number of anglers is again roughly stable until age 81, the oldest age recorded in the survey (Figure 1). A similar type of analysis could be performed using New York State data if available.

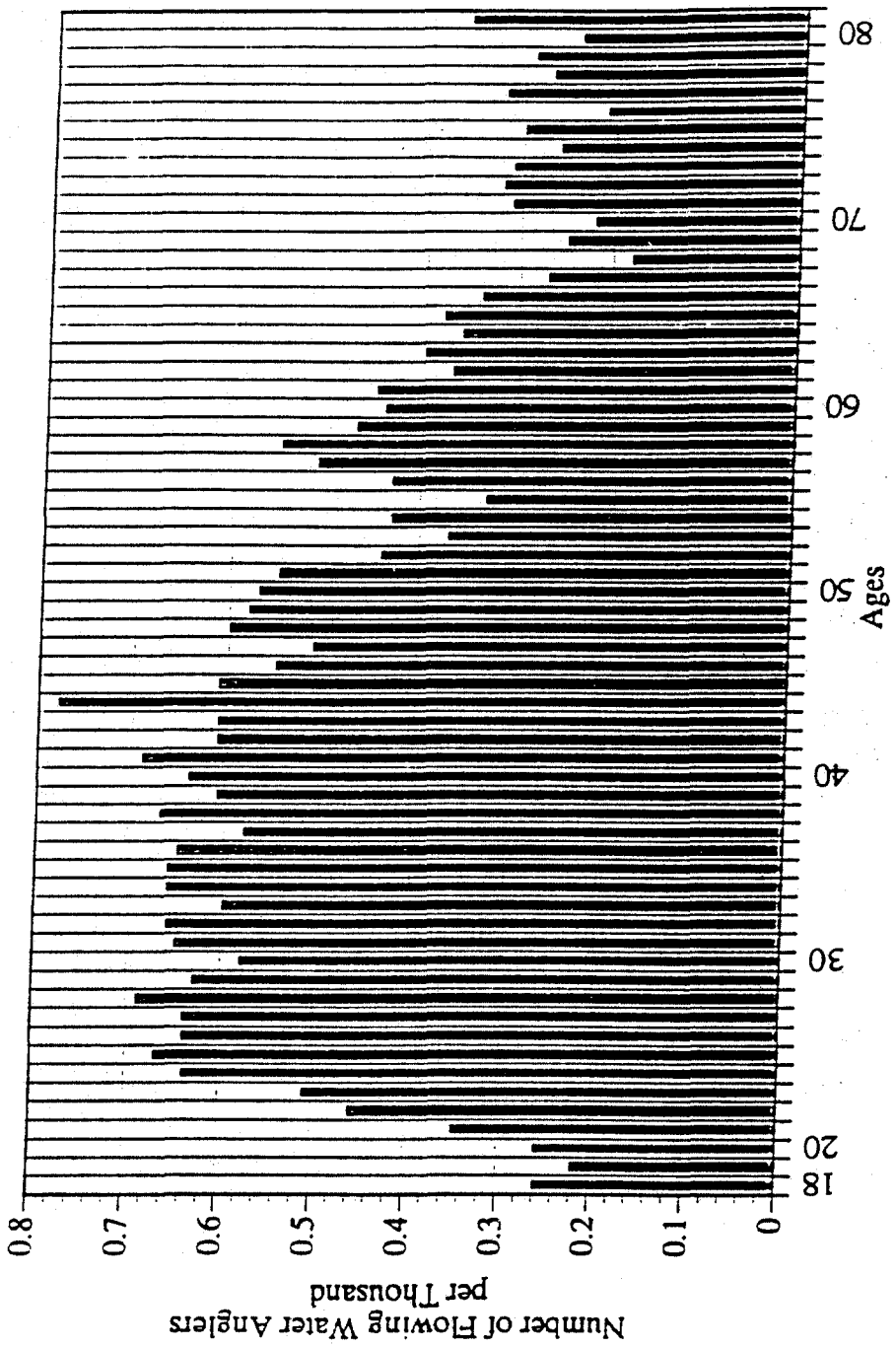

Figure 1. Number of Maine Anglers per Thousand that Eat Self-Caught Fish from Flowing Waters

Table 1. County Mobility Distribution

Age	Probability of an intercounty move
1-4	.039
5-9	.032
10-14	.023
15-17	.026
18-19	.038
20-24	.077
25-29	.070
30-34	.045
35-39	.033
40-44	.028
45-49	.026
50-54	.019
55-59	.015
60-64	.017
65-69	.017
70-74	.011
75-79	.014
80-84	.011
85+	.021

Source: U.S. Census Bureau, 1991

Mortality

Mortality also determines how long an individual potentially catches and consumes fish from the Hudson River. Standard actuarial mortality tables can be used to predict the life expectancy of a given angler and whether that individual would likely remain a member of the population of living anglers. Age- and gender-specific data on mortality are available from the New York State Department of Health (1991) and the National Center for Health Statistics (1990) and can be used to create a complete distribution of the probability of dying at each age (Table 2).

3.0 CONCLUSION

Based on data on the number of years an individual is likely to reside in the same residence, EPA (1991) estimated that all Hudson River anglers catch and consume fish from the Upper Hudson River for 30 years. While changing residences may influence an individual's exposure to fish from the Hudson River, there are other factors that also affect an angler's fishing habits and location. In order to accurately estimate the length of time anglers may fish the Hudson River, it is important to look at mortality rates, age-specific angling probabilities, and mobility. Only by considering these three factors can the true variation in angler exposure be realistically evaluated. The most appropriate method to evaluate these factors is through the use of a probabilistic exposure assessment using a synthetic life history or Microexposure Monte Carlo analysis. The type of analysis can account for the individual variations in mortality, mobility, and angling cessation within the total angler population.

Table 2. Mortality Distribution

	Probability of Dying	
Age	Male	Female
10	0.00016	0.00012
15	0.00082	0.00037
20	0.00148	0.00051
25	0.00156	0.00053
30	0.00167	0.00062
35	0.00201	0.00084
40	0.00251	0.00120
45	0.00364	0.00198
50	0.00596	0.00335
55	0.00976	0.00534
60	0.01597	0.00864
65	0.02378	0.01326
70	0.03693	0.02060
75	0.05671	0.03233
80	0.08621	0.05250
85	0.14309	0.10189
90	1.0	1.0

Source: Johnson & Capel, 1992.

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ESTIMATING FISH CONSUMPTION RATES FOR THE UPPER HUDSON RIVER

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Estimating Fish Consumption Rates for the Upper Hudson River

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ATTACHMENTS:

Estimating Consumption of Freshwater Fish Among Maine Anglers

Selection of Fish Consumption Estimates for Use in the Regulatory Process

The Effect of Sampling Bias on Estimates of Angler Consumption Rates in Creel Surveys


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1.0 INTRODUCTION AND PURPOSE

In 1991, EPA issued a Phase I Report for the Reassessment RI/FS in which the Agency evaluated the potential human health risks for the Hudson River Superfund site. In this report, EPA (1991) determined that any risks to human health from PCBs in sediment occur through indirect exposure through the fish consumption pathway. Under Superfund Guidance (EPA, 1989a), evaluation of such exposures are specifically required not to consider the impact of any fishing regulations. However, fishing restrictions have been imposed by the State of New York, and thus the estimates of PCB exposure developed for the Upper Hudson River are hypothetical and an overestimation of actual exposures.

In estimating potential risks to anglers who consume fish from the Hudson River, EPA (1991) assumed 30 g/day as the average consumption rate for anglers. This rate was used with other point estimates of exposure and toxicity to estimate risks from PCB intake. As described in GE's comments on the Phase I Report, the use of the "point-estimate" approach is overly conservative and should be replaced by a more sophisticated probabilistic approach (i.e., Microexposure Monte Carlo analysis). Furthermore, the assumed fish consumption rate was inappropriate for the Upper Hudson River.

This paper presents a discussion of the technical and regulatory issues related to the selection of fish consumption rates for anglers who would fish the Upper Hudson River in the absence of a ban. The goal is to present a summary of the technical information currently available on fish consumption and to develop the best possible estimates of fish consumption that would apply to the Upper Hudson River area and could be used in a probabilistic risk analysis. Specifically, this paper presents the results of three recent studies of fish consumption rates that have been published in the peer-reviewed literature, *Estimating Consumption of Freshwater Fish Among Maine Anglers* (Ebert et al., 1993), *The Effect of Sampling Bias on Estimates of Angler Consumption Rates in Creel Surveys* (Price et al., 1994), and *Selection of Fish Consumption Estimates for Use in the Regulatory Process* (Ebert et al., 1994). The first of these three papers was published in the North American Journal of Fisheries Management. The other two were published in the Journal of Exposure Analysis and Environmental Epidemiology.

2.0 FISH CONSUMPTION AND THE HUDSON RIVER SITE

The determination of fish consumption rates is an important issue in the evaluation of human health risks from the presence of persistent lipophilic chemicals in waterbodies. The amount of fish consumed by a population of anglers varies depending on the numbers and types of waterbodies fished and the characteristics of the angler population. Fish consumption also depends on factors such as climate, fish species present, fish productivity, river access, and the size of the angler population.

The population at risk from the consumption of fish contaminated with PCBs represents a small fraction of the general population. Only a limited number of individuals living near the Upper Hudson River are freshwater anglers. In recent years freshwater anglers have adopted catch and release programs as a way of maintaining the number of trophy-level fish in North American fishing waters. In fact, a recent survey of freshwater anglers found that two-thirds of anglers who fished rivers and streams did not consume the fish they caught (ChemRisk, 1992). The proposed distribution of intakes developed in this paper is specific to that fraction of anglers who catch and consume fish and is not meant to reflect fish consumption rate for the general population or even for all freshwater anglers.

For the Upper Hudson River the rate of fish consumption should be based on the intake of sport-caught fish and not consumption of fish obtained from restaurants, markets, or other, non-angling sources. Unfortunately, no survey has adequately evaluated the fish consumption rates of anglers using the Upper Hudson River, due to the existence of a State ordered and enforced fishing ban since 1976 on the affected portion of the river. Although two mail surveys have been performed on New York anglers (NYSDEC, 1990; Connelly et al., 1992) and a creel survey (Barclay, 1993) was performed on Upper Hudson river anglers, none of these surveys focused on fish consumption from the river. NYSDEC (1990) evaluated fish consumption from all recreational and commercial sources including self-caught fish from the Hudson. Connelly et al. (1992) evaluated self-caught fish consumption, but the survey included information on consumption of fish from Lake Ontario and other large lakes, the inclusion of which may make the survey results inappropriate for the Upper Hudson River. Barclay (1993) focused on evaluating the effectiveness of fish consumption bans and advisories on the Hudson River and did not develop

quantitative estimates of fish consumption rates. Clearly, even if these surveys were used to develop estimates of fish consumption rates specific to the Upper Hudson River, the results would be of limited use in characterizing actual risks to anglers because of the effect current fishing restrictions have on fish consumption rates.

As stated in the Final Phase 2 Work Plan and Sampling Plan for the Hudson River, EPA (1992) acknowledged that "the Phase 2 baseline assessment will evaluate whether there are adequate data to justify a site-specific or region-specific value for fish consumption that would apply in the Hudson River area in the absence of a fishing ban." In this paper, a surrogate region-specific study is identified that can be used to estimate a fish consumption rate for the Hudson River. An evaluation of EPA's current default estimate that was used in the Phase I Report (EPA, 1991) is presented, as is a brief review of the existing literature on fish consumption rates. This paper also provides a specific recommendation for estimating an appropriate distribution of fish consumption rates for use in the human health risk assessment for the Upper Hudson River.

3.0 EPA ESTIMATES IN THE 1991 PHASE 1 REPORT

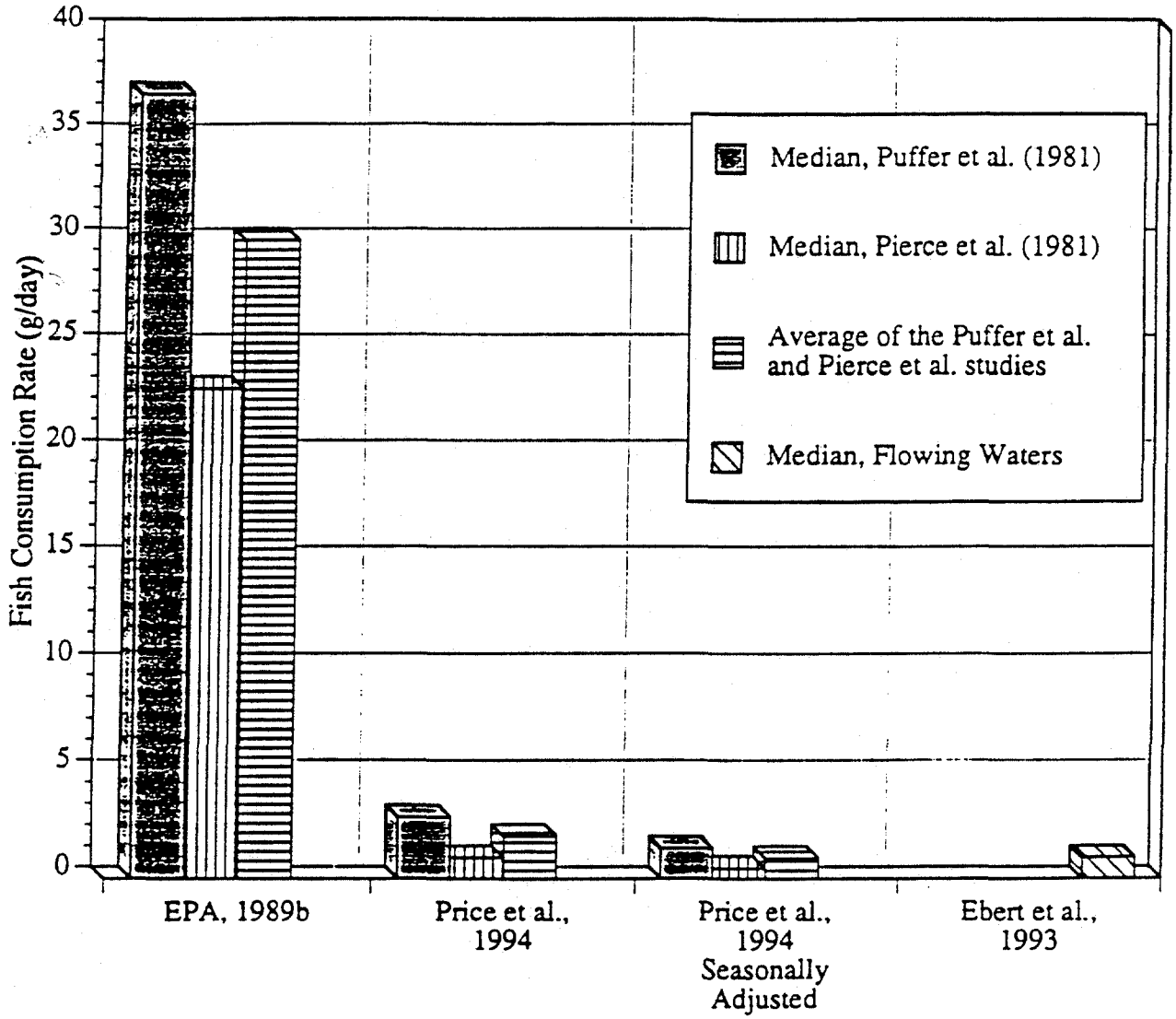
In its Phase 1 Report, EPA (1991) recommended that 30 g/day be used to estimate fish consumption for the typical Hudson River angler. This value is based on the average of the median consumption rates reported by two studies of recreational anglers, Puffer et al. (1981) and Pierce et al. (1981). Pierce et al. (1981) interviewed fishermen on Commencement Bay, a marine/estuarine fishery in Puget Sound near Tacoma, Washington. Based on data provided by Pierce et al. (1981), EPA (1989b) estimated that the median consumption rate for those anglers included in the survey was 23 g/day. The Puffer et al. (1981) study, which was a creel survey of Los Angeles Harbor anglers, reported a median consumption rate of 37 g/day. EPA (1989b) averaged these two median rates to derive their estimate of 30 g/day to represent the average recreational angler. EPA justified the use of this estimate in the Phase I document based on two arguments: first, that the value of 30 g/day is recommended in the *Exposure Factors Handbook* as a default value for large bodies of water (EPA, 1989b); and second, that a study of New York anglers reported a similar estimate of intake (NYSDEC, 1990).

There are a number of reasons why it is inappropriate to apply this value in estimating the fish intake rate for the Hudson River Reassessment. First, the 30 g/day number is based on consumption rates derived from short-term studies of marine fishermen. As discussed in greater detail in Section 4.0, the most appropriate surveys for characterizing fish intake rates for a particular waterbody are long-term surveys performed on similar bodies of water. The use of fish consumption rates from West Coast marine anglers is not appropriate for estimating intake from Eastern freshwater rivers. Further, as discussed in Section 4.0, a number of additional studies have been published since the *Exposure Factors Handbook* (EPA, 1989b) was completed. Several of these studies provide a much better basis for estimating hypothetical consumption rates for the Upper Hudson River (Connelly et al., 1992 ; Ebert et al., 1993).

Second, the estimate of 30 g/day is an unreasonable estimate of fish consumption for the "typical" Hudson River angler since the results of the Puffer et al. (1981) and Pierce et al. (1981) creel surveys significantly overestimate the distribution of intakes for anglers using the surveyed bodies of water. The attached manuscript *The Effect of Sampling Bias on Estimates of Angler Consumption Rates in Creel Surveys* demonstrates that the results of the two creel surveys are strongly biased towards frequent anglers. This bias is inherent to all creel surveys because frequent anglers are more likely to be present when interviewing occurs than infrequent anglers. Due to this bias, the median fish intake for the survey population is substantially higher than the consumption rate for the total population of anglers using the body of water. When this bias is corrected (Price et al., 1994), the median intakes for the total population of anglers in the Puffer et al. (1981) and Pierce et al. (1981) surveys become 2.9 and 1.0 g/day, respectively (Figure 1). Thus, in the distribution of intakes for the total population of anglers, the EPA value of 30 g/day corresponds to approximately the 95th percentile; not the 50th percentile of fish consumption rates, as claimed.

Third, the value of 30 g/day is unlikely to adequately characterize fish consumption from the Upper Hudson due to the potentially limited fishing season for that area. The estimates of angler intake from the Puffer et al. (1981) survey and to a lesser extent the Pierce et al. (1981) survey are based on data from fisheries that are open year round. Independent of the current fishing ban, fishing on the Upper Hudson is likely to be restricted between late fall and early spring due to species

Figure 1. Comparison of Fish Consumption Rates



restrictions and unfavorable weather and fishing conditions. Ice fishing may also be limited on the river due to rapid currents that prevent the build-up of a safe thickness of ice.

In the Puffer et al. (1981) and Pierce et al. (1981) surveys, many anglers reported fishing on a daily basis and more than half of the surveyed anglers reported fishing more than once a week. Those anglers who reported their fishing frequency (e.g. twice a week, once a month) were assumed by the investigators (Puffer et al., 1981 and Pierce et al., 1981) to fish at that frequency throughout the year. Such an assumption implied that an angler who fished once a week made 52 fish trips each year. In contrast, if a limited New York season is assumed, anglers who fish the Hudson River on a weekly basis might fish only 20 to 30 times per year. As a result of the limited season, the median fish intakes by Hudson River anglers would be expected to be approximately one half of the 2.0 g/day calculated for the reanalyzed (Price et al., 1994) Puffer et al. (1981) and Pierce et al. (1981) surveys (Figure 1).

EPA (1991) also cited the results of the New York Statewide Angler Survey (NYSDEC, 1990) as a basis for the 30 g/day estimate of intake. The survey reported that New York anglers consumed an average of 45.2 fish meals per year. Assuming a meal size of 227 g/meal (1/2 lb of fish per meal) (Cox et al., 1987, 1989; West et al., 1989; NYSDEC, 1990), 45.2 fish meals correspond to 28 g/day averaged over an entire year. While this finding suggests that anglers may consume fish at a rate approaching 30 g/day, this intake rate cannot be used as an estimate of the consumption rate for sport-caught fish from the Upper Hudson River because the 45.2 fish meals per year represents consumption of fish from all sources, including purchased fish, gift fish, and fish consumed in restaurants. Since the majority of fish consumed by anglers are purchased and not self-caught (West et al., 1989), the NYSDEC estimate significantly overestimates the typical angler's intake of self-caught fish. Additional evidence of this overestimation can be seen in the reported results of Connelly et al. (1992) who reported 11 self-caught fish meals by New York anglers. Using the same meal size of 227 g/meal, an average intake rate of 7 g/day is derived.

For the reasons stated above, it is clear that the median consumption rate for the typical angler that might fish the Upper Hudson will be much less than 30 g/day, even if the ban was lifted. Therefore, GE recommends that EPA not rely on the default consumption rate given in the

Exposure Factors Handbook (EPA, 1989b) either as a point estimate or as the basis for a distribution of fish intakes.

4.0 RECOMMENDED APPROACH

To adequately characterize potential exposures associated with human health risks from PCBs found in the Hudson River, it is necessary to identify a fish consumption rate that is appropriate for that waterbody. As stated previously, this estimate ideally would be based on surveys of anglers currently fishing the Upper Hudson River; however, the existing ban prevents the collection of any meaningful data. Given this situation, the most accurate information must be taken from studies that characterize recreational consumption from regional freshwater rivers and streams whose productivity and accessibility are similar to the Hudson River. This section presents a review of the available angler surveys that may be applicable to the potential users of the Upper Hudson.

4.1 Published Studies

Numerous estimates of fish consumption rates have been made for both the general population of the U.S. (Javitz, 1980; Rupp et al., 1980; USDA, 1980) and for recreational anglers (Soldat, 1970; Honstead et al., 1971; Pierce et al., 1981; Puffer et al., 1981; Turcotte, 1983; Landolt et al., 1985, 1987; Cox et al., 1985, 1987, 1990; Fiore et al., 1989; West et al. 1989; NYSDEC, 1990; ChemRisk, 1991a,b; Connelly et al., 1992; Richardson and Currie, 1993; Ebert et al., 1993). These studies have reported a wide range of fish consumption values and have examined consumption rates of fish taken from various types of waterbodies ranging from all waters to single bodies of water. A summary of the published surveys is provided in the attached manuscript *Selection of Fish Consumption Rates for Use in the Regulatory Process* (Ebert et al., 1994).

Ebert et al. (1994) provide an in-depth analysis of the studies used to estimate the range of fish consumption rates available for the general population of the United States and recreational anglers. Studies that provide estimates of per capita consumption for the general population are appropriate when evaluating the effects of background contamination levels on the population as a whole from

all fish species found in the marketplace. Examples of these types of consumption estimates can be found in Javitz (1980), Rupp et al. (1980), and USDA (1980).

The studies that evaluate consumption by recreational anglers may be divided into the following categories: those that evaluate consumption of fish from (1) all commercial and recreational sources; (2) self-caught marine sources; (3) multiple freshwater bodies; (4) multiple flowing waterbodies; (5) multiple lakes and ponds; and (6) specific waterbodies (Table 1). Studies by Fiore et al. (1989), West et al. (1989), and Connelly et al. (1990) provide information on total fish consumption by anglers. Pierce et al. (1981), Puffer et al., (1981) and Landolt et al. (1985, 1987) evaluated fish consumption from marine waterbodies. Fiore et al. (1989), West et al. (1989), and Cox et al. (1985, 1987, 1990) looked at consumption from multiple fresh waterbodies. Due to these various methods of estimating fish consumption rates, these studies provide a wide range of consumption estimates. The consumption rates reported for multiple freshwater systems can be further refined to consider only flowing waters or only lakes and ponds. Data for these specific fresh water systems are available in ChemRisk (1991a), Ebert et al. (1993), and Richardson and Currie (1993). Finally, data on consumption from specific waterbodies are available in Soldat (1970), Honstead et al. (1971), Turcotte (1983), and ChemRisk (1991b). The fish consumption rate values reported in these studies vary greatly. Intake for the typical recreational angler ranged from less than 1 g/day to 37 g/day, while the intake rates for the high-end angler ranged from 11 g/day to more than 300 g/day.

4.2 Study Selection

Given this wide range of angler studies and consumption rates, the study and rate of consumption for the assessment of risk to anglers at the Upper Hudson River site should be selected carefully so that the fish consumption rate most appropriate to the Upper Hudson River can be identified. Selecting the appropriate value requires the identification of specific criteria that must be met to ensure that the most appropriate study and data are selected. (Table 2). For example, as primary criteria, General Electric believes that it is critical that the study evaluate self-caught, freshwater fish over a long-term. These primary criteria must be met to ensure that the fish consumption rate closely approximates consumption from the Upper Hudson River. Only when these criteria are met can the secondary criteria be considered to further refine the fish consumption estimate.

Table 1. Fish Consumption Estimates for Recreational Anglers

Study	Consumption Rates (g/d)		
	Mean	Median	"High End"
All Commercial and Recreational Sources			
Fiore et al. (1989)	26	--	63[a]
NYSDEC (1990)	28	--	--
West et al. (1989)	18.3	--	--
Marine - Self-Caught			
Landolt et al. (1985; 1987)	--	15[b]	--
Pierce et al. (1981)	--	23	>54[a]
Puffer et al. (1981)	--	37	339[a]
Multiple Fresh Waterbodies			
Connelly et al. (1992)	6.8	--	32[c]
Cox et al. (1985)	21.8	--	--
Cox et al. (1987)	10.4	7.5	--
Cox et al. (1990)	--	7.5	--
Ebert et al. (1993)	6.4	2.0	26[a]
Fiore et al. (1989)	12.3	--	37.3[a]
West et al. (1989)	7	--	--
Multiple Flowing Waterbodies			
Ebert et al. (1993)	3.7	0.99	12[a]
Multiple Lakes and Ponds			
ChemRisk (1991a)	4.2	1.7	15[a]
Richardson and Currie (1993)	16.2	--	--
Specific Waterbodies			
ChemRisk (1991b)	3.0	0.49	11[a]
Soldat (1970)	1.8	--	--
Honstead et al. (1971)	7.7	--	--
Turcotte (1983)	7.4[d]	--	--

a. 95th percentile.
b. Calculated using a Monte Carlo simulation based on frequency distributions provided by authors.
c. 92nd percentile.
d. Calculated based on 2.5 consumers per angler.

Table 2. Existing Surveys of Fish Consumption Rates in Recreational Anglers

	Primary Criteria			Secondary Criteria		
	Self Caught	Freshwater	Long-term Survey	Flowing Water	Single Waterbody	Regionally Appropriate
ChemRisk, 1991a	X	X			X	X
ChemRisk, 1991b	X	X				X
Connelly et al., 1992	X	X	X			X
Cox et al., 1985, 1987, 1990	X	X				
Ebert et al., 1993	X	X	X	X		X
Fiore et al., 1989	X	X	X			
Honstead et al., 1971	X	X	X		X	
Landolt et al., 1985;1987	X					
NYSDEC, 1990			X			X
Pierce et al., 1981	X				X	
Puffer et al., 1981	X				X	
Soldat, 1970	X	X			X	
Turcotte, 1983	X	X			X	
West et al., 1989		X				

As indicated above, one of the most important aspects that must be included in a selected study is the evaluation of self-caught fish only. Fish consumption from this single source is likely to be much less than consumption from the range of commercial, restaurant, and gift fish that may be available to the recreational fisherman (Ebert et al., 1994). A second primary criteria should require that the study selected evaluate consumption from a freshwater, riverine system. Due to differences in the types and numbers of species found in freshwater and marine systems, consumption of self-caught marine fish is generally higher than fresh waterbodies (Ebert et al., 1994). This may be due to longer fishing seasons, availability of preferred species, or higher productivity rates at marine fisheries. Finally, it is critical that the selected surrogate study is conducted over a long-term. Extrapolation of annual or other long-term intake rates, based on short-term recall surveys, results in additional uncertainty particularly for the upper and lower ends of the distribution (Finley et al., 1994; Wallace et al., 1994). This occurs because activity and consumption by individual anglers are highly variable through the season due to weather, fishing regulations, differences in species availability, and fluctuations in success rates. Although much of this variability tends to average out in longer-term estimates, extrapolation from single-day or short-term measurements will result in an overestimation of the interindividual variation of annual intake.

Evaluation of secondary criteria is also necessary when selecting a consumption rate study. As noted for marine and fresh waterbodies, the rate of consumption from standing waters (lakes and ponds) is higher than the consumption rate from rivers and streams (Ebert et al., 1994). Under ideal conditions it would be favorable to use a study that evaluated consumption from a single flowing system that was like the Hudson. However, if a specific waterbody with appropriate characteristics cannot be identified, it may be more appropriate to use estimates generated for flowing waters only. Finally, the selected study should have collected data from a regionally appropriate waterbody. As recommended by the EPA (1989a), it is best to use site- or region-specific consumption data when conducting a risk assessment. To date, there are a limited number of studies available in the New York/New England area that provide information on consumption of sport-caught fish from freshwater rivers and streams.

Table 2 presents a comparison of the existing surveys and how well they meet the above criteria. As the table indicates, the Ebert et al. (1993) and Connelly et al. (1992) studies most closely approximate consumption from rivers similar to the Upper Hudson River. Connelly et al. (1992) evaluated recreational consumption from New York State freshwaters using a mail recall survey and reported that the average New York angler consumes 11 meals per year of self-caught fish from New York's freshwater fisheries. If it is assumed that each meal is 227 grams in size (1/2 pound), it can be estimated that the average New York angler consumes self-caught freshwater fish, on an annual basis, at a rate of 7 g/day. Ebert et al. (1993) also conducted a mail recall survey of recreational anglers. Specifically, this study evaluated rates of freshwater fish consumption by Maine's anglers. Ebert et al. (1993) reported a mean consumption rate of 6.4 g/day and a median rate of 2.0 g/day for anglers consuming fish from all freshwaters. Lower values of 3.7 and 0.99 g/day were reported for the mean and median from flowing waters only.

Although the Connelly et al. (1992) survey is specific to New York State, there are several factors which limit its usefulness in the assessment of intake for the Upper Hudson River. First, Connelly et al. only present a single point estimate value for fish consumption. The use of a distribution of consumption rates, however, is much more preferable in order to characterize interindividual variability and realistically assess the potential risks to recreational anglers. With only an average consumption rate value, it is not possible to accurately represent the range of recreational anglers, including those who ingest higher amounts of fish.

Second, the mean fish consumption rate determined by Connelly et al. (1992) represents fish eaten from all freshwaters in the state including Lake Ontario and other large lakes. As pointed out by Ebert et al. (1993), intake from rivers and streams is much smaller than intake from lakes and ponds. In addition, the rate of intake from multiple waterbodies is higher than that from a single water system (Ebert et al., 1994). Given these factors, it is highly likely that the fish consumption rate in Connelly et al. (1992) overestimates the actual fish consumption rate on a single portion of the Upper Hudson River.

Third, an additional problem with the Connelly et al. (1992) was the format used to collect data on consumption. Survey respondents were asked to provide waterbody specific data on the type and amount of fish meals eaten over a one-year period using a complex matrix format. A substantial

sample of the survey respondents did not complete this matrix. Since nonrespondents are known to have, on average, lower fishing frequencies and consumption rates (West et al., 1989), the complex matrix format may have resulted in a hidden bias towards the high end in the survey results.

Finally, it is important to note that the original purpose of the Connelly et al. (1992) study was not to identify a consumption rate for New York anglers. Instead, the objectives of this study were to: (1) identify the level of knowledge of New York State anglers about health advisories and contaminants in fish; (2) determine fishing behaviors and consumption patterns in response to these advisories; (3) compare the results of this survey with data collected in 1988; and (4) evaluate the impacts of the New York advisory and make recommendations for future improvements in risk communication. Although questions were asked in the survey regarding fish consumption behaviors, those questions were aimed at estimating how the health advisories altered the consumption behavior of recreational anglers.

While the data from Ebert et al. (1993) are not specific to New York State, the anglers surveyed are a reasonable surrogate. As Table 3 indicates, angler demographics are similar in Maine and New York, suggesting that similar angler characteristics may be found in the two states. In addition, Ebert et al. (1993) present data on rates of consumption from rivers and streams in the New England area. Due to similarities in climate that limit fishing in New York and Maine, it is likely that these states have similar fishing opportunities and similar catch and consumption rates. And finally, the mean fish consumption rate for self-caught fish reported in Connelly et al. (1992) is similar to the mean fish consumption rate value reported in Ebert et al. (1993) (Figure 2).

Given all of these factors discussed above, the Ebert et al. (1993) study performed to evaluate freshwater fish consumption in the State of Maine is believed to provide the best characterization of a distribution of fish consumption rates for the angler who would fish the Upper Hudson River in the absence of a ban. Table 4 presents the distribution of fish consumption rates reported by Ebert et al. (1993) for moving waters.

Figure 2. Key Findings of Ebert et al., 1993 and Connelly et al., 1992

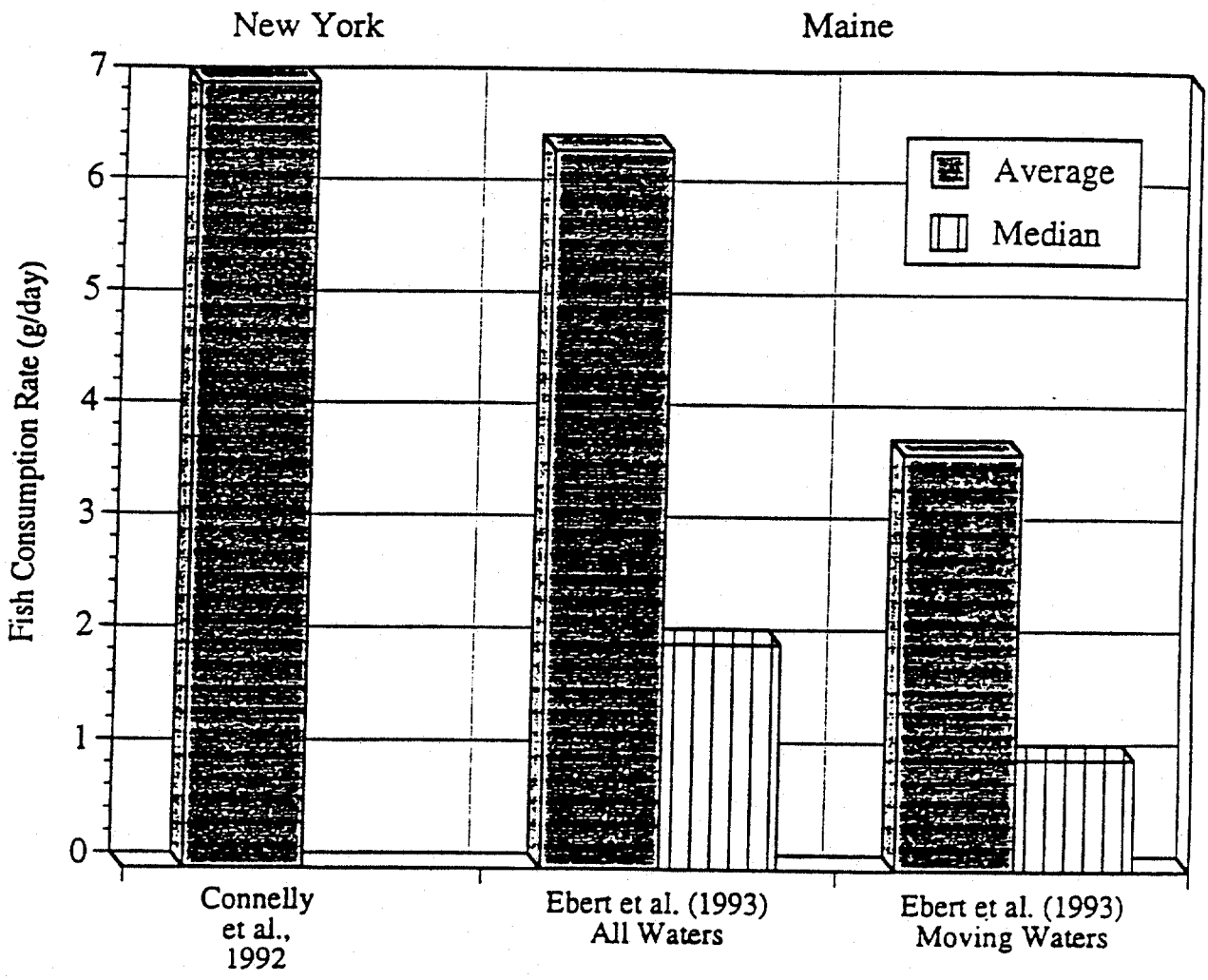


Table 3. Maine and NY Angler Demographics[a]

Demographic Characteristic	New York[b]	Maine[c]
Mean Age	43	44
Sex (percent of respondents)		
Male	84.8	80.7
Female	15.2	19.3
Income Level (percent of respondents)		
<$20,000	23.3	33.3
$20,000 - $49,999	49.9	51.9
>$50,000	26.8	14.8
Education Level (percent of respondents)		
Grades 1 to 11	10.5	16.2
Graduated High School	32.3	32.5
Some College or Trade School	31.3	26.8
Graduate College of Trade School	13.1	18.6
Some Postgraduate	13.0	5.9

a. New York statistics include nonresident and resident anglers. Maine survey includes only resident anglers.
b. NYSDEC, 1990.
c. ChemRisk, 1991a.

Table 4. Distribution of Fish Consumption Rates for Moving Waters (Rivers and Streams)

Percentiles	Fish Consumption Rate
Minimum	0.001
5	0.11
10	0.17
15	0.23
20	0.28
25	0.35
30	0.46
35	0.59
40	0.71
45	0.83
50	0.99
55	1.2
60	1.4
65	1.7
70	2.1
75	2.5
80	3.2
85	4.3
90	6.1
95	12
Maximum	118
Median	0.99
Mean	3.7
StDev	12

Source: Ebert et al., 1993

5.0 CONCLUSIONS

In the Phase I document, EPA (1991) estimated that the typical recreational angler will consume 30 g/day of self-caught fish from the Hudson River. This value is likely to greatly overestimate the potential fish consumption for typical anglers in the absence of fishing restrictions. Instead, Hudson River anglers are more likely to consume (in the absence of fishing restrictions) at rates corresponding to other waterbodies with similar characteristics to the Hudson. An evaluation of the relevant criteria indicates that Connelly et al. (1992) and Ebert et al. (1993) are the most appropriate studies upon which to base Hudson River fish consumption rates. While the two studies have similar estimates of angler intake, GE believes the results of the Maine angler study (Ebert et al., 1993) provide the superior basis for characterizing the distribution of angler fish consumption rates for the Upper Hudson River. It should also be noted that the results of the Maine angler survey are not inconsistent with the results of the Puffer et al. (1981) and Pierce et al. (1981) surveys when the effects of sampling bias and seasonality are considered (Price et al., 1994).

The evidence presented in this issue paper clearly indicates that the typical levels of fish consumption by recreational anglers are approximately 1/30 of the rate used by EPA (1991). This reduced estimate is supported by new data analyses conducted since the development of the Phase I document. Recent studies that evaluated fish consumption on waterbodies similar to the Hudson River clearly demonstrate that differences in waterbody and population characteristics must be considered if fish consumption is to be properly quantified. Continued use of default values (EPA, 1991) will only exaggerate potential human health risks to recreational anglers.

The most appropriate method to evaluate fish consumption is through the use of a probabilistic exposure assessment using synthetic life history or Microexposure Monte Carlo analysis. This type of analysis can account for the variations in fish consumption of individual anglers. GE believes EPA needs to adopt the approach. Furthermore, GE believes that the distribution of fish consumption rates used in the probabilistic risk assessment should be that developed by Ebert et al. (1993).

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ATTACHMENTS:

Estimating Consumption of Freshwater Fish Among Maine Anglers

Selection of Fish Consumption Estimates for Use in the Regulatory Process

The Effect of Sampling Bias on Estimates of Angler Consumption Rates in Creel Surveys

North American Journal of Fisheries Management 13:737–745, 1993

Estimating Consumption of Freshwater Fish among Maine Anglers

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Abstract.—In deriving water quality standards and appropriate restoration levels for contaminated surface waters, the potential for human exposure is often the most important factor to be considered. For certain persistent compounds, like 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) or mixtures of polychlorinated biphenyls, a primary pathway of human exposure is through ingestion of fish obtained from affected waters. Pending water quality regulation for TCDD in Maine required that estimates be made of the rate of consumption of freshwater fish obtained from rivers that receive TCDD discharges. Because commercial freshwater fishers do not exist on Maine rivers, any freshwater fish that are eaten have been caught by anglers. A statewide mail survey of Maine's licensed anglers was undertaken to characterize rates of fish consumption from rivers and streams in Maine. The survey was mailed to 2,500 licensed resident anglers who were randomly selected from state license files. The response rate of 70% (based on deliverable surveys) resulted in a usable sample of 1,612 anglers. Results of this study indicated that, if fish are shared with other fish eaters in the household, the annual average consumption of freshwater river fish per consuming angler in Maine is 3.7 g/d. Comparisons of findings of this study and of studies in other regions of the United States show considerable variations in fish consumption rates, supporting the use of state- or region-specific estimates of fish consumption in establishing water quality regulations for persistent, biologically accumulative compounds.

As society attempts to reduce the amounts of contaminants released into surface water resources, and to determine appropriate restoration levels for contaminated waters, a critical consideration is the quantity of fish that the public consumes from those waters. Ingestion of freshwater fish is potentially the most common pathway of human exposure to certain chemical contaminants in surface waters (Rifkin and LaKind 1991). Recognizing that a relationship may exist between the presence of contaminants in surface waters and uptake by humans through fish ingestion is only the first step in developing water quality regulations. It is also necessary to determine the quantities of fish consumed, the levels of chemical contaminants in the fish tissues consumed, and the potential toxicity to humans who consume those fish (Sherman et al. 1992). While the health effects of certain compounds have been studied extensively, and levels in fish are frequently monitored, estimates of fish consumption from specific water bodies are not readily available (EPA 1992). This lack of data is due largely to the fact that fishery managers and natural resource agencies are primarily concerned with controlling harvest and not with the final disposition of the harvest. Monitoring the consumption of freshwater fish often does not come under the direct purview of any public agency.

An example of this limitation is the recent rule-making process to set an ambient water quality standard for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in Maine's rivers. Because there are no commercial freshwater fisheries in the state, only those individuals who consume sport-caught fish have the potential to be exposed to TCDD in the fish from Maine's impacted rivers. Thus, estimation of angler consumption of freshwater fish from affected rivers was critical to the rule-making process in Maine.

TABLE 1.—Existing fish consumption estimates (mean g/d per person). Numbers in parentheses are median values. Consumption estimates from studies on the U.S. population are per capita.

Reference	Consumers studied	All types of fish, all sources[a]	Marine–estuarine fish		Freshwater fish	
			All sources[a]	Sport-caught[b]	All sources[a]	Sport-caught[b]
Fiore et al. (1989)	Wisconsin anglers			26	12	
Honstead et al. (1971)	Columbia River anglers					7.7
Javitz (1980)	U.S. population	14[c]				
Landolt et al. (1985)	Washington anglers			(15)[c]		
NYSDEC (1990)	New York anglers	28				
Pao et al. (1982)	U.S. population	(37)				
Pierce et al. (1981)	Washington anglers			(23)[d]		
Puffer et al. (1981)	California anglers			(37)		
Rupp et al. (1980)	U.S. population	16	14		1.5	
Soldat (1970)	Columbia River anglers					1.8
Turcotte (1983)	Savannah River anglers					31[e]
West et al. (1989)	Michigan anglers				18	7[f]

[a] All sources includes fish purchased in stores and restaurants as well as recreationally caught fish.
[b] Sport-caught includes only fish that have been obtained by angling.
[c] Estimate based on Monte Carlo simulation using frequency distributions for edible weight of fish, fish per trip, trips per year, and household size.
[d] EPA (1989b) estimate.
[e] Based on harvest estimates; no correction for sharing of harvest.
[f] Estimated value based on data presented in Table 19 in West et al. (1989).

There are several reasons why the existing fish consumption estimates derived elsewhere could not be used to infer freshwater fish consumption in Maine. First, fish consumption studies by Javitz (1980), Rupp et al. (1980), Pao et al. (1982), and NYSDEC (1990) did not distinguish between the consumption of commercially harvested and recreationally harvested fish (Table 1). Thus, the fish consumption estimates from these studies include purchased and sport-caught freshwater and saltwater fish. Consumption of saltwater species was not relevant to the TCDD risk assessment for Maine's rivers, and there are no commercial freshwater fisheries on Maine's rivers.

Second, studies by Pierce et al. (1981), Puffer et al. (1981), and Landolt et al. (1985), although focused on consumption of sport-caught fish, gave consumption estimates for marine or estuarine fishes. There are no data available to evaluate the comparability of consumption of recreationally caught saltwater fish with consumption of recreationally caught freshwater fish.

Third, only six studies specifically estimated consumption of freshwater fish (Soldat 1970; Honstead et al. 1971; Rupp et al. 1980; Turcotte 1983; Fiore et al. 1989; West et al. 1989). Of these studies, only four reported consumption rates for sport-caught fish, and only three estimated consumption of sport-caught fish from riverine fisheries. The river studies were conducted in the Pacific Northwest (Soldat 1970; Honstead et al. 1971) and the southeastern United States (Turcotte 1983). These studies demonstrated considerable variation in estimated consumption; mean rates ranged from 2 to 31 g/d per person.

Therefore, to estimate consumption rates of recreationally caught freshwater species in Maine, we conducted a statewide mail survey of licensed resident anglers. We have identified potential issues in developing fish consumption estimates that we hope will stimulate research to enhance the validity and reliability of future fish consumption estimates. It is also our intent to raise fishery biologists' awareness of the need for estimating fish consumption rates so that future studies of fishing effort, when possible, will include estimates of harvest and consumption.

Methods

Sample Selection

Freshwater fish consumption was estimated for adult anglers who held a Maine resident, inland fishing license.[1] Nonresident anglers were not included in the sample because prior research in-

[1] All adult anglers (≥16 years) are required to obtain a fishing license to fish Maine's inland waters, except members of the Penobscot Indian Nation, who can fish riverine waters adjacent to selected portions of their land without a license. The Penobscots must obtain a complimentary license to fish all other riverine and standing waters in the state. Holders of these complimentary licenses were represented in the sample.

dicated that there is substantially more effort each year by resident anglers, and resident anglers are more likely to fish in Maine every year (Boyle et al. 1989). By sampling only licensed resident anglers, consumption data were collected for the subset of licensed anglers who, as a group, were believed to have the greatest potential opportunity for exposure to TCDD.

A sample of 2,500 licensed resident anglers was randomly selected from Maine's license files. Prior research indicated that participation in warmwater fishing is substantially lower than participation in coldwater fishing in Maine, and that the warmwater species with the lowest participation rates were yellow perch *Perca flavescens* and white perch *Morone americana* (Phillips et al. 1990). Multiplying the inverse of the combined rate for participation in yellow perch and white perch fishing by the desired number of consumption observations for perch (100) led us to conclude that we needed to receive 1,363 completed surveys. To determine the sample size necessary to ensure this number of responses, we assumed that 90% of the mailed surveys would be deliverable, that 90% of the 1989 license holders fished in 1990, and that the survey response rate would be 75%. This resulted in a required sample size of approximately 2,000. An additional 500 anglers were added to the sample to compensate for an unknown percentage of Maine anglers who practice catch-and-release fishing or do not consume fish. This procedure ensured that the number of consumption observations for all other fish species of interest would exceed those for yellow perch and white perch.

Because inland fishing licenses are valid for one calendar year, and recording of license sales is not completed by Maine's Department of Inland Fisheries and Wildlife (IF&W) until March of the following year, the sample was selected from among all anglers who held a 1989 fishing license. This process resulted in a sample of anglers who held licenses in both 1989 and 1990. Boyle et al. (1990) surveyed resident anglers licensed in 1987 regarding their open-water fishing effort during 1988 and found this sampling method to be valid.

The mail survey was pretested with 50 randomly selected anglers. Telephone interviews were conducted with 40% of the pretest participants to learn if they had difficulty in answering or understanding any of the questions. Final revisions were made to the survey, based on responses to the telephone interviews and reviews of returned pretest mail surveys.

All open-water fishing in Maine closes on October 31. However, because open-water fishing for most Maine waters (all but one river) closes on September 30, the survey was implemented in mid-October 1990. Postcards were sent 1 week later, thanking those who had already returned the survey, and asking those who had not yet returned the survey to do so. Three weeks later, on November 7, 1990, a follow-up survey packet was mailed to 1,111 anglers who had not yet responded, and the recipients were asked to complete and return the survey by December 3, 1990.

Survey Design

The design of the survey focused on asking anglers to report the disposition, particularly consumption, of freshwater fish they caught in Maine. This strategy differed from some of the previous fish consumption studies where survey respondents were asked to report the number of fish meals they ate each week (Javitz 1980; Rupp et al. 1980; Pao et al. 1982; West et al. 1989; NYSDEC 1990). To address the TCDD issue, it was important to know where the fish were caught and to exclude fish consumption from sources other than Maine's freshwater (i.e., saltwater species or freshwater species purchased at the market). Only 320 km of Maine's rivers, less than 1% of all riverine environments in Maine, were potentially contaminated by TCDD. Therefore, to obtain a usable sample and to provide an appropriate context, anglers were asked about their fish consumption from flowing (rivers, streams, and brooks) and standing (lakes and ponds) water bodies.

Each respondent was asked to report how many trips had been made to ice fish, open-water fish in standing waters, and open-water fish in flowing waters during the last completed season. Anglers were also asked to report the number of each species of fish caught during the 1990 open-water season and the 1989–1990 ice-fishing season. For fish caught during open-water season, anglers were asked to report the number of fish consumed for each of 15 groups of species, and to identify the number taken from flowing or standing water bodies. Anglers were also asked to estimate the average length for each species of fish that was eventually consumed. In addition to those fish caught by the responding angler, the respondents were asked to describe the number, species, and average length of each sport-caught fish they had consumed that had either been obtained from other members of their households or from individuals outside of their households.

TABLE 2.—Regression parameters for weight–length equations and edible portion (E_i) of fish species harvested by freshwater anglers in Maine. NR = not reported.

Species	Regression coefficients: Intercept	Regression coefficients: Slope	Length range[a] (mm)	Water body and location	Source[b]	E_i[c]
Landlocked salmon (lacustrine Atlantic salmon *Salmo salar*)	−5.145	3.035	270–750	Rivers and lakes, Maine	IF&W	0.40[d]
Atlantic salmon	−5.038	3.00	NR	Unspecified, Scotland	Carlander (1969)	0.40[d]
Lake trout *Salvelinus namaycush*	−5.879	3.306	290–840	Rivers and lakes, Maine	IF&W	0.30
Brook trout *Salvelinus fontinalis*	−5.054	3.022	150–750	Rivers and lakes, Maine	IF&W	0.30
Brown trout *Salmo trutta*	−5.096	3.037	167–936	Rivers and lakes, Maine	IF&W	0.30
Yellow perch *Perca flavescens*	−3.519	2.390	127–320	Rivers and lakes, Maine	IF&W	0.30
White perch *Morone americana*	−5.273	3.177	100–457	Rivers and lakes, Maine	IF&W	0.30
Largemouth bass *Micropterus salmoides*	−3.844	2.606	209–686	Rivers and lakes, Maine	IF&W	0.30[d]
Chain pickerel *Esox niger*	−5.491	3.098	229–566	Unspecified, Florida	Carlander (1969)	0.30
Lake whitefish *Coregonus clupeaformis*	−5.677	3.241	NR	Lake Superior, USA–Canada	Carlander (1969)	0.30
Brown bullhead *Ameiurus nebulosus*	−5.061	3.065	152–192	Lake Butte des Morts, Wisconsin	Carlander (1969)	0.30
White sucker *Catostomus commersoni*	−5.395	3.223	NR	Shadow Mt. Lake, Colorado	Carlander (1969)	0.30
Creek chub *Semotilus atromaculatus*	−3.972	2.98	NR	Des Moines River, Iowa	Carlander (1969)	0.30
Rainbow smelt *Osmerus mordax*	−6.2	3.40	80–220	5 lakes in the Sebago region, Maine	IF&W	0.78[d]
Redbreast sunfish *Lepomis auritus*	−4.69	3.01	NR	Unspecified, Alabama	Carlander (1977)	0.30

[a] Represents the range of lengths of fish used for the regression analysis.
[b] IF&W = Maine's Department of Inland Fisheries and Wildlife (unpublished data).
[c] Portion of whole fish that is edible, based on EPA (1989b), except where noted.
[d] Based on Maine-specific data collected by ChemRisk (unpublished data).

Estimating Fish Consumption Rates

The total weight of freshwater fish from each source that was consumed within each respondent's household was estimated from respondent-provided data on quantity and average length of each fish species eaten that was obtained as a result of the respondent's, other household members', and nonhousehold members' fishing activities. The weight of fish consumed for each species group was estimated as follows:

$$C_i = Q_i \times W_i \times E_i; \quad (1)$$

C_i = total weight (g) of species group i consumed within the angler's household;
Q_i = number of fish of species group i consumed within the angler's household;
W_i = weight (g) per fish of species group i, based on reported average length (lengths were reported in inches but converted to millimeters);
E_i = portion of fish weight that is edible for species group i.

Data on the number of fish consumed were directly obtained from survey responses. The weight was predicted by using the reported average lengths from the survey and length–weight regression equations estimated by IF&W based on several years of length and weight measurements from rivers and lakes in Maine (Table 2). For those species for which Maine-specific equations were not available, the appropriate relationships were obtained from Carlander (1969, 1977).

Because not all of a fish is edible, it was necessary to characterize the edible portion of a whole fish (E_i). Stansby and Olcott (1963) reported that commercial filleting of finfish yields between 20 and 40% edible tissue and that actual yield depends upon the species. The EPA (1989a) has recommended that 30% be used to characterize the edible portion of finfish.

To explore the range and variability of the edible portion, studies were undertaken to estimate the edible portions (fillets) of smallmouth bass *Micropterus dolomieu* and landlocked salmon in

Maine. Twenty-two smallmouth bass were collected from two Maine rivers and 12 landlocked salmon were collected from one river. The whole fish were weighed and then carefully filleted to remove as much flesh from the bones as possible. Fillets from each fish were then weighed, and the fillet weight was compared with the whole-body weight for that fish to determine the edible portion. For smallmouth bass, the mean edible portion was 30%, with a 90% confidence interval ranging from 27 to 30%. The mean edible portion for landlocked salmon was 37% with a 90% confidence interval ranging from 36 to 39%. For the current analysis, the results of the landlocked salmon analysis were used to assume edible portions of 40% for landlocked salmon and Atlantic salmon. The EPA (1989a) recommendation, confirmed by the smallmouth bass analysis, was used to assume an edible portion of 30% for all species in Table 2 except rainbow smelt. For this species, we assumed that half of those consumed were eaten without the head or viscera, and half were eaten with the viscera but without the head. Rainbow smelt data were not available, but for landlocked salmon, the body without the head and viscera represented 68% of the whole fish weight and the body without the head represented 87%, giving an average edible portion of 78%. This average value was used for rainbow smelt.

The total freshwater fish weight consumed from Maine rivers and streams by the angler and other people in the household was then calculated as the sum of C_i for the 15 groups of species. Daily freshwater fish consumption for each individual respondent was estimated by summing the source-specific rates (e.g., open-water fishing, ice fishing), and then dividing by the number of fish consumers residing in the respondent's household and the number of days in a year. To estimate rates of consumption from rivers and streams, equation (1) was used but Q_i and W_i were based only on fish that had been reportedly harvested from rivers or streams during the season.

Our initial analysis of consumption rates was based on the assumption that all freshwater fish obtained for consumption by the angler were shared equally with other household members who consume fish. This assumption was also used by Puffer et al. (1981) and is the approach supported by EPA (1989a). Some researchers have divided total fish consumed by the total number of persons in the household to obtain per-capita fish consumption estimates (Pierce et al. 1981; Landolt et al. 1985). Whereas this approach may be reasonable for estimating consumption of marine species, it is questionable for estimating consumption of freshwater fish because the percentage of the population that eats freshwater species is generally lower than the percentage that consumes marine fish (Rupp et al. 1980). We also conducted a sensitivity analysis to consider the impacts of different assumptions about sharing on consumption rate estimates. Three scenarios were considered: (1) all household fish consumers eat an equal share of consumed fish; (2) only adults in the household consume fish; and (3) the angler alone consumes all of the fish reported.

Statistical analyses were conducted without assuming a distributional model. Because of certain physical limitations (e.g., the high number of zero consumers and limited number of high consumers), fish consumption data do not fit a standard distribution model. To force a fit of these data to a standard model would obscure the true nature of the distribution.

Results

In total, 1,612 surveys were completed and returned, representing 70% of the deliverable surveys. Of these, 1,251 (78%) of the respondents reported having fished during the 1990 open-water season or the 1989–1990 ice-fishing season. Also, 118 individuals did not fish but consumed freshwater fish caught by other anglers, either within or outside of their households. These 118 respondents, with the 1,251 who fished, constituted the 1,369 angler observations (85% of total responses) used in data analyses.

In total, 599 (44%) of the respondents indicated that they ice fished, and 1,127 (82%) of the respondents participated in open-water fishing during the period of interest. Of the individuals who open-water fished, 93% reported having fished in ponds or lakes and 66% reported having fished in streams and rivers.

Twenty-three percent of all anglers surveyed reported that they consumed no freshwater fish caught in 1990. Forty-three percent of the river anglers indicated that they did not consume fish from rivers or streams during the 1990 season, and 19% of river anglers consumed no freshwater fish from any source during that period.

The median fish consumption per angler for those who had eaten fish was 2.0 g/d based on catch from all waters and 0.99 g/d based on fish taken from flowing waters (Table 3). The arithmetic mean consumption by consuming anglers was 6.4 g/d (all waters) and 3.7 g/d (flowing waters). These arithmetic means represented the 77th

TABLE 3.—Estimates of fish consumption (g/d per person) by anglers licensed to fish in Maine's lakes, ponds, streams, and rivers during the 1989–1990 ice-fishing or 1990 open-water seasons. Estimates are based on rank except for those of arithmetic means.

Percentile	All waters		Rivers and streams	
	All anglers[a] (N = 1,369)	Consuming anglers[b] (N = 1,053)	River anglers[c] (N = 741)	Consuming anglers[b] (N = 464)
50th (median)	1.1	2.0	0.19	0.99
66th	2.6	4.0	0.71	1.8
75th	4.2	5.8	1.3	2.5
90th	11	13	3.7	6.1
95th	21	26	6.2	12
Arithmetic mean[d]	5.0 (79)	6.4 (77)	1.9 (82)	3.7 (81)

[a] Licensed anglers who fished during the seasons studied and did or did not consume freshwater fish, and licensed anglers who did not fish but ate freshwater fish caught in Maine during those seasons.

[b] Licensed anglers who ate freshwater fish caught in Maine during the seasons studied.

[c] Those of the "all anglers" category who fished on rivers or streams.

[d] Values in parentheses are percentiles at the mean consumption rates.

and 81st percentiles of the consumption distributions, respectively.

Consumption estimates varied depending on how fish were shared among household members (Table 4). If we assumed that only the angler ate all of the fish consumed, then median rates increased by roughly a factor of 2.5 relative to the scenario in which fish are shared by all household fish consumers. If we assumed that fish were shared by adults in the household, median consumption estimates increased by approximately a factor of 1.2.

Discussion

The EPA (1989b) has recommended that when data on local consumption are not available, a default value of 30 g/d per person "be used to represent consumption rates for recreational fishermen in any area where there is a large water body present and widespread contamination is evident." This rate is the average of the median consumption rates derived in two studies of marine anglers (Pierce et al. 1981; Puffer et al. 1981). Application of this rate to TCDD rule-making for Maine's rivers is inappropriate because it is based on the consumption of marine species. Furthermore, TCDD discharges are not widespread in Maine, but rather affect only 320 (0.5%) of the 59,500 km of rivers and streams in the state. In its recently proposed document entitled "Estimating Exposures to Dioxin-Like Compounds," EPA (1992) has revised its approach to estimating fish consumption from a single small water body and has indicated that a consumption estimate ranging from 1 to 4 g/d may be more appropriate under these circumstances.

The results of the Maine angler survey demonstrate a median consumption per consuming resident sport angler of 2.0 g/d for all freshwater finfish and 0.99 g/d for fish from flowing bodies of water. Both of these estimates are considerably lower than the median value of 30 g/d previously recommended by the EPA, but fall within the revised EPA recommendation of 1–4 g/d.

These consumption estimates fall at the low end of the range of reported consumption estimates for freshwater fish in other geographic locations (Table 1). Although differences could be due to survey methodology, average lengths of fish and harvest rates reported by survey respondents were consistent with IF&W data. Thus, we believe that these differences are likely due to differences in

TABLE 4.—Sensitivity analyses of the effects of assumptions about sharing of fish among household members on estimated consumption rates (g/d per person).

Percentile	All household consumers share		Only adults share		Anglers are only consumers, no sharing	
	All waters	Rivers and streams	All waters	Rivers and streams	All waters	Rivers and streams
50th (median)	2.0	0.99	2.3	1.2	5.0	2.5
66th	4.0	1.8	4.4	2.0	9.1	4.1
75th	5.8	2.5	6.6	3.0	13	6.1
90th	13	6.1	16	6.5	32	14
95th	26	12	28	20	57	27
Arithmetic mean[a]	6.4 (77)	3.7 (81)	7.5 (78)	4.5 (83)	15 (78)	8.9 (83)

[a] Values in parentheses are percentiles at the mean consumption rates.

catch rates, fish size, and length of fishing seasons in Maine relative to other geographic locations. The magnitude of variation of fish consumption estimates reported in Table 1 demonstrates that fish consumption does vary geographically and underscores the need to develop more extensive data on fish consumption so that regional variations can be considered.

It is important to recognize that consumption is likely overestimated in the current study for the purpose of TCDD rule-making in Maine. First, the study was designed to collect data on consumption from all flowing bodies of water, and not just the 320 km of contaminated water. Thus, although individuals may fish in affected river reaches some of the time, it is highly unlikely that all fishing effort is focused on these waters, particularly because there are numerous alternative fisheries in close proximity to each river. Over 80% of Maine's resident anglers fish two or more bodies of water each year, approximately 60% fish three or more, nearly 40% fish four or more, and most riverine fishing in Maine occurs in headwaters and small streams and brooks, not in main stems of larger rivers where TCDD may be present (K. J. Boyle, unpublished data). Consequently, whereas the estimates for rivers and streams include all consumed fish from rivers and streams during the season, it is likely that only a portion of the consumption can be attributed to a single water body.

Second, in a study done for the U.S. Fish and Wildlife Service, Westat (1989) reported that 6-month or 1-year recall periods produce "substantial overestimates" of fishing participation (see also Chu et al. 1992). If participation estimates are overstated in a 6-month to 1-year recall study, it may also be reasonable to assume that consumption is overestimated due to recall bias. To date, there have been no studies specifically conducted for the purpose of evaluating recall bias in fish consumption surveys. This issue needs to be addressed in future studies of fish consumption.

Although fish consumption may be estimated by equating it to harvest, this approach inappropriately assumes that all harvested fish are consumed by the angler. In fact, we found that approximately 30% of the harvested fish were either thrown away, given away, used as bait, or fed to pets. Furthermore, anglers may share catch with friends or family members. Thus, equating the amount of fish harvested with consumption, even if adjustments are made for the edible portion, will overestimate fish consumption.

As noted earlier, some researchers have asked respondents to recall the total number of fish meals consumed over a period of time and to estimate the average size of those meals (West et al. 1989; NYSDEC 1990). This approach was not used in the current study because it was critical to collect information on the sources of the fish consumed. Anglers were surveyed, rather than other household members, because it was believed that they would be best able to accurately report where the fish had been caught. This is an important issue for future research in that anglers may be able to accurately report catch location, a critical issue in contamination studies, but may not accurately report consumption by all household members. Alternatively, household members may be able to report their consumption habits but may not be able to identify the locations from which the fish have been obtained.

Other issues that require further investigation when assessing exposure to chemical contaminants in fish are the sizes of fish consumed, the number of individuals who share in consumption, and the species consumed. Consideration should be given to the household member who consumes the largest quantity of fish, and the sex and age composition of fish consumers. Estimates of exposure must also consider the differences among species in their potentials to accumulate chemical contaminants in their tissues. Anadromous species such as Atlantic salmon and rainbow smelt are likely to have low body burdens of chemical contaminants, whereas other species indigenous to riverine environments, such as white perch, yellow perch, brown bullhead, creek chub, and white sucker, may have larger body burdens of chemical contaminants. All of these factors, although not necessary in estimating total fish consumption, may be crucially important in assessing exposures due to fish consumption.

The need to develop fish consumption estimates is not motivated solely by a single contaminant like TCDD but also arises for numerous other contaminants in aquatic ecosystems. If fish consumption levels for particular types of water bodies in specific regions of the country are known, it will be possible to assess human exposure to any contaminant once the concentration in edible fish tissue has been determined. The specific contaminant being addressed will, however, define the location and extent of fish consumption data required. Therefore, regular collection of fish consumption data as a part of the fishery management process will enhance future assessments of poten-

tial contamination and the ultimate restoration of contaminated waters.

Regulators are often faced with multiple factors that need to be considered in rule making, including public health risks, the size of the potentially affected population, and social factors. Unnecessarily stringent water quality standards could result in substantial economic and social costs. The methodology used in this study allows estimates of consumption to be derived for each respondent. It provides regulators with a full distribution of consumption estimates to be used in the decision-making process. The selection of the most appropriate consumption percentile to be used can then rightfully be made as part of the risk management or policy decision.

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SELECTION OF FISH CONSUMPTION ESTIMATES FOR USE IN THE REGULATORY PROCESS

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The rate of fish consumption is a critical parameter in the assessment of human exposure to persistent chemicals in surface waters. Ideally, exposure assessors should use site-specific information concerning fish consumption rates from a contaminated area; however, this information is not readily available for most bodies of water, and time and economic constraints often do not permit its collection. In such situations, it is necessary to derive a fish consumption rate for the exposed population, based on data presented in existing studies. However, because of differences in the types of waterbodies evaluated, the types of fish consumers surveyed, and the types of survey methods used, the fish consumption estimates available in the scientific literature range widely, making selection of a specific rate a complex task. In the absence of clear understanding of the differences in the studies underlying these fish consumption estimates, exposure assessors have often arbitrarily selected the results of studies that report high rates of intake in order to ensure that public health is being adequately protected. This paper presents a framework to evaluate the applicability of existing studies to different exposure scenarios. It discusses the strengths and limitations of the various survey methods used to estimate fish consumption rates. Its intent is to provide a framework for exposure assessors to assist them in their selection of the most applicable and relevant fish consumption estimates for use in the regulatory situation being considered.

INTRODUCTION

The most significant pathway of potential human exposure to persistent and bioaccumulatable chemicals in aquatic environments is through the ingestion of fish (Rifkin and LaKind, 1991). In an effort to assess whether the presence of these chemicals in surface waters may adversely affect public health, it is often necessary to characterize the potential for human exposure

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2. Abbreviations: cm, centimeter; EPA, United States Environmental Protection Agency; g, grams; g/d, grams per day; kg, kilogram; km, kilometer; NMFS, National Marine Fisheries Service. NPD, National Purchase Diary; NYSDEC, New York State Department of Environmental Conservation; USDA, United States Department of Agriculture.

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consumption, based on the populations of concern and the number, types, and sizes of fisheries being considered. It also provides insights into the differences and limitations of the survey methodologies and the inherent biases of each, thereby providing exposure assessors with information that will assist them in their interpretation of the applicability of specific survey results. Its intent is to provide guidance for exposure assessors in their selection of the most applicable and relevant fish consumption estimates for the specific situations being evaluated.

SOURCES OF VARIATION IN FISH CONSUMPTION ESTIMATES

There are a number of factors responsible for the large variations in rates of fish consumption found in the scientific literature. Generally, these variations are attributable to the survey methodology used, the type of waterbody studied, and the characteristics of the populations evaluated. Some of these sources of variation are discussed below.

Targeted Populations

A major difference among studies of fish consumption is attributable to the population being surveyed. Some studies have investigated fish consumption rates in the general population (Javitz, 1980; Rupp et al., 1980; USDA, 1980; Pao et al., 1982), while other studies have reported rates of consumption by recreational anglers (Soldat, 1970; Honstead et al., 1971; Pierce et al., 1981; Puffer et al., 1981; Turcotte, 1983; Landolt et al., 1985, 1987; Cox et al., 1985, 1987, 1990; Fiore et al., 1989; West et al., 1989; NYSDEC, 1990; ChemRisk, 1991a,b; Connelly et al., 1992; Ebert et al., 1993; Richardson and Currie, 1993). Rates of fish ingestion are likely to differ between the general population and the population of anglers (EPA, 1991). Even within the angling group, rates are likely to be variable due to the fact that some anglers consume no sport-caught fish, some consume only sport-caught fish, and others consume both sport-caught fish and fish from other commercial sources. This is apparent in evaluating the fact that some studies have investigated anglers' intakes of fish from all sources, including purchased, gift, sport-caught, and that consumed at restaurants (West et al., 1989; NYSDEC, 1990), while other studies have reported on the rate of sport-caught fish consumption (Honstead et al., 1971; Soldat, 1970; Pierce et al., 1981; Puffer et al., 1981; Turcotte, 1983; Cox et al., 1985, 1987, 1990; Landolt et al., 1985, 1987; Connelly et al., 1992; Ebert et al., 1993). In addition, some differences in the literature can be attributed to the fact that certain researchers have focused on consumption by subpopulations known to have higher than average intakes (Humphrey, 1987; Richardson and Currie, 1993).

Targeted Waterbodies

In some studies, the rate of sport-caught fish consumption reported by anglers may include marine and estuarine fish (Pierce et al., 1981; Puffer et al., 1981; Landolt et al., 1985, 1987). Other studies specifically evaluate consumption of freshwater fish but include fish obtained from multiple freshwater locations (Cox et al., 1985, 1987, 1989; Fiore et al., 1989; Connelly et al., 1992; Ebert et al., 1993). Still other surveys have only considered consumption of sport-caught fish from a single body of water (Soldat, 1970; Honstead, 1971,

Turcotte, 1983. ChemRisk, 1991a). Surveys conducted for individual waterbodies are greatly affected by the productivity of those waters and the availability of access for fishing. Consequently, there is substantial variation in the resulting estimates of intake.

Regional Considerations

In evaluating the reported estimates of fish consumption for anglers, a further complication is introduced by the existence of regional differences in climate, fishing regulations (e.g., length of season, bag limits, etc.), accessibility to good fisheries, availability of desirable target species, and ethnic or cultural backgrounds. These factors may contribute to variations in reported fish consumption rates. Individuals living in coastal areas are more likely to consume higher quantities of marine fish and lower quantities of freshwater fish while individuals living in inland regions of the country may consume more freshwater fish (Rupp et al., 1980). Due to the migratory patterns of fish, certain species may be available commercially and recreationally year-round in certain regions of the country, but only for limited periods of time in others. Additionally, in some states or on certain bodies of water, fishing may be permitted on a year-round basis, while in other cases, the fishing season is restricted. Finally, fisheries may have catch and release restrictions or limits on the numbers, species, and sizes of fish that may be harvested during the season. All of these factors can significantly effect the rate at which anglers may consume sport-caught fish.

Biases in Consumption Survey Methodologies

Numerous survey types and methods, each with its own inherent biases, have been used to estimate fish consumption rates. These biases can contribute substantially to the variations observed in consumption estimates. The most common methodologies include diary studies, on-site creel surveys, short-term recall surveys, long-term recall surveys, and biological monitoring techniques. Each of these survey methodologies offers distinct advantages and limitations that must be considered when evaluating the fish consumption rates that are derived from them (EPA, 1991).

Diary Studies. Many of the most commonly cited estimates of fish consumption have been based on diary studies. In the 1973/1974 National Purchase Diary (NPD) Study, which underlies the rates reported by Javitz (1980) and Rupp et al. (1980), heads of households were asked to complete a diary of fish purchases each month over a 12-month period. Similarly, the data reported by Pao et al. (1982) were based on a 3-day study conducted by the USDA which included one day of recall and two days of diary entries. Long-term diary studies, like the NPD study, are a useful way of determining per capita rates of fish consumption by the general population. If study participants are diligent in recording the numbers, types, and sizes of fish meals consumed, excellent estimates of annual per capita fish consumption can be derived.

Short-Term Recall Surveys. Short-term recall surveys are the best possible means of gathering accurate information on fishing and consumption activity for a specific period of time. Like long-term surveys, they are generally used to provide information on total consumption over the recall period. However, the extrapolation of annual or other long-term

intake rates results in additional uncertainty when based on short-term recall surveys, particularly for the upper and lower ends of the intake distribution.

The reason for this is as follows. Although an individual may consume fish at a rate in the upper 5th percentile of the distribution during a specific brief period of time (such as a few days or weeks), it is not necessarily true that the same individual will be an upper 5th percentile consumer for each of the brief periods that make up an entire season. Rather, that individual may only consume fish occasionally, may only be interested in consuming certain species when they are available, and if the individual is an angler, is not likely to be equally successful on every trip. The same uncertainty exists for anglers who have had no activity or success during a single two-week period but may, in fact, have different behavior at other times. It is likely that activity and consumption by individual anglers are highly variable through the season due to weather, fishing regulations, differences in species availability, and fluctuations in success rates for the individual angler. Although much of this variability tends to be averaged out in longer-term estimates, extrapolation from single-day or short-term measurements can result in an overestimation in the inter-individual variation of annual intake in a population (EPA, 1992b). Thus, short-term surveys may be useful for characterizing the central tendency in consumption rates but not the variance within the population.

Long-Term Recall Surveys. Long-term recall surveys provide an opportunity for individuals to summarize their activities throughout a fishing season or calendar year. Thus, developing estimates of annual intake from such surveys does not require that the data be extrapolated, and the impact short-term variability in activity patterns is minimized. However, long-term recall studies have potential for recall bias resulting from the tendency of an individual to systematically over- or underestimate his or her activities due to a difficulty in recalling detail over a long period. Westat (1989) reported that recall bias in 6-month or year-long fishing and hunting surveys results in overestimations of angler participation. By analogy, long recall periods can be expected to lead to overestimated rates of fish ingestion.

Creel Surveys. Creel surveys can provide very accurate, waterbody-specific data on the species and sizes of fish consumed but are limited as a basis for deriving longer term consumption rates. As with the short-term recall survey, data collected in a creel survey only represent a snapshot in time for each angler interviewed. Because each angler is only interviewed once during the course of the survey, extrapolation to annualized rates requires that assumptions be made concerning the angler's behavior during the remainder of the year.

In addition, creel surveys tend to over sample the most highly active anglers and under sample the less active individuals. This occurs because the probability of participating in a survey is much greater for frequent anglers who spend more time at a particular fishery (Puffer et al., 1981; Price et al., 1994). Due to this sampling bias, consumption estimates based on creel surveys are likely to be representative only of more frequent anglers and are not representative of the total population of anglers using the surveyed waterbody. Pierce et al. (1981) demonstrated this phenomenon when they showed that approximately 60% of the anglers

interviewed indicated that they fished at least once per week. However, when the total population of anglers using the body of water was determined, anglers who fished at least once per week represented only 6.8% of all anglers.

Biomonitoring. A final method of estimating fish consumption rates is the use of biomonitoring data (Richardson and Currie, 1993). Under this approach, samples of hair, nails, tissue, or bodily fluids are taken from individuals known to consume fish from contaminated waterbodies. The samples are analyzed for the contaminants known to occur in fish. Pharmacokinetic models are then used to determine the dose rate of the contaminant necessary to produce the measured levels (or body burden). This dose rate is then converted to a fish consumption rate based on the average level of contamination in fish tissue.

Biomonitoring offers a number of advantages in estimating fish consumption rates. There is no potential for bias in the self-reporting of consumption rates since the effect of an individual's intake is directly measured. In addition, the measurement of contaminant intake also incorporates the individual's fish preparation and cooking practices. Finally, biomonitoring results reflect the individual's consumption over a long period of time (several months or years).

Despite these advantages, the method also suffers from a number of limitations. The variation in individual measurements of body burden across the population may reflect variations in human metabolism of the contaminant or different chemical concentrations in the fish consumed, rather than a variation in the rate of fish intake. In addition, there may be other sources of exposure to the chemicals of interest that could compound the problem. Because of the multiple sources of variation, biomonitoring can only successfully provide estimates of the average intake rate and cannot be used to accurately characterize the range or "high end" of intake rate in an exposed population. The methodology is also limited to populations whose only source of exposure to a contaminant is from the consumption of contaminated fish. Finally, the approach requires the availability of a reliable, chemical-specific pharmacokinetic model that can quantitatively predict intake from the measurements of an individual's body burden.

SELECTION OF CONSUMPTION RATES

When selecting a fish consumption rate for regulatory decision-making, it is essential that risk assessors carefully evaluate the population that is potentially affected and select a fish consumption rate that is relevant and applicable to that population. It is important to recognize that total fish consumption by an individual is likely to include fish from a combination of sources (Figure 1). An individual may buy marine, estuarine or freshwater fish and shellfish from a local grocer or fish market. In addition, certain individuals may consume marine, freshwater or estuarine fish or shellfish they have caught personally. Finally, individuals may consume fish that have been sport-caught by someone else and given to

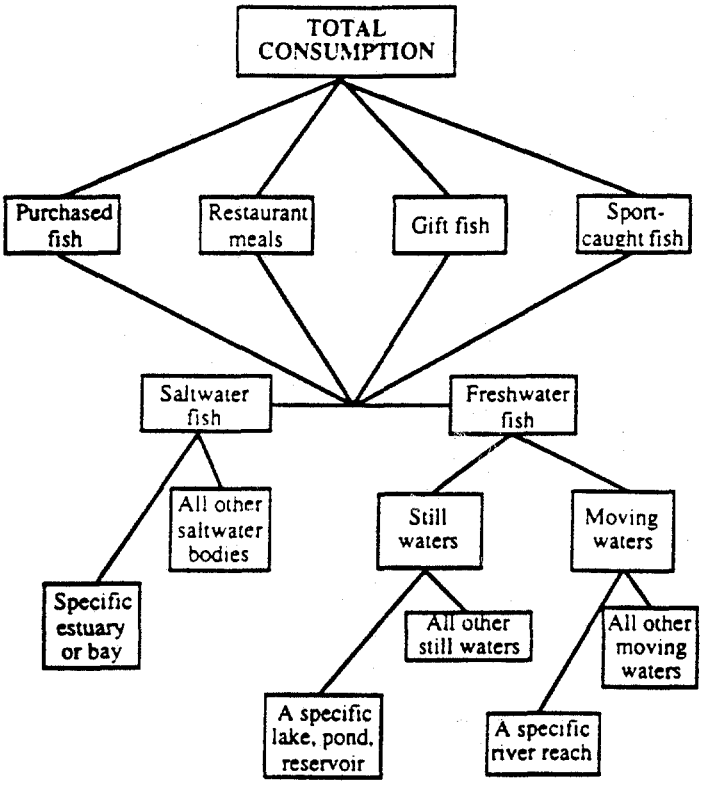

FIGURE 1. Total consumption of fish.

them. These fish may have been obtained from one or more bodies of water. Because total consumption by an individual is comprised of the sum of the rates of consumption for each of these components, estimates may vary substantially, depending upon which components have been evaluated.

In light of this discussion, it is not surprising that a number of different consumption estimates have been derived and are commonly cited in the literature or used as the basis for regulatory decisions. To clarify the bases for these differences and to assist exposure assessors in their selection of the most applicable estimates for their particular situations, the following studies have been grouped according to the types of situations to which they are most relevant.

General Population - Per Capita Estimates

If setting chemical residue levels for fish found in the marketplace is of primary interest, then per capita ingestion estimates for the general population of the United States may be

appropriate. It is important to note, however, that these per capita estimates include nonconsumers of fish. Their inclusion may result in estimates that are not representative of consumers.

These per capita estimates consider the population as a whole, for whom some fraction of the consumed fish may be affected by chemical contamination. They include all types of fish available to the general population: marine, estuarine, freshwater, fresh, frozen, and processed fish from a number of geographic locations. Examples of these types of consumption estimates include the following studies, which are summarized in Table 1.

TABLE 1. Fish Consumption Estimates for the General Population of the United States

Study	Consumption Rates (g/d)		
	Mean	Median	"High End"
Per Capita Estimates — All Types of Fish			
Javitz (1980)	14	—	42[a]
Rupp et al. (1980)	13	—	—
USDA (1980)	21	—	—
Per Capita Estimates — Specific Types of Fish			
Rupp et al. (1980) marine fish	11[b]	7.3[b]	24[b,c]
Rupp et al. (1980) shellfish	3.6[b]	0[b]	11[b,c]
Rupp et al. (1980) freshwater fish	1.5[b]	0[b]	5.1[b,c]
Consumers Only — All Types of Fish			
Pao et al. (1982)	54	37	128[a]

[a] 95th percentile.
[b] Adults only.
[c] 90th percentile.

Javitz, 1980. In 1973-1974, the National Marine Fisheries Service (NMFS) funded a study by NPD Research, Inc. (Javitz, 1980). Each month, individuals participating in this year-long household diary study were asked to record all types of marine and freshwater fish and shellfish meals consumed. Based on these data, Javitz (1980) estimated a per capita rate of consumption that included individuals who did not consume fish, as well as consumers. No distinction was made between the consumption of commercially-harvested and sport-caught fish.

Rupp et al., 1980. Rupp et al. (1980) used the data generated from the NMFS diary survey to estimate consumption of marine fish, freshwater fish, and shellfish for three different age groups within the general population of the United States. Separate estimates of consumption were derived on a regional basis. Although these estimates identified the specific types of fish being consumed (marine, freshwater, etc.), they did not differentiate between commercial and sport-caught fish. There was substantial variation among the region-specific consumption estimates.

USDA, 1980. From 1977 to 1978, the United States Department of Agriculture (USDA, 1980) conducted a survey of 37,874 individuals. This survey included one day of recall and two days of diary records for each survey participant. Based on these survey data, USDA reported a mean consumption rate of fish and shellfish. Because this survey did not target anglers and did not differentiate between types of fish consumed, this estimate includes consumption of all types of fresh, frozen, and processed, freshwater and marine, fish and shellfish.

General Population - Fish Consumers Only

Because per capita estimates of consumption for the general population of the United States are averaged across all individuals, including those who do not consume fish, they may underestimate rates for that portion of the population that eats fish. Thus, when setting chemical tolerances or establishing a generic standard, it may be preferable to use estimates of consumption that are based on fish consumers only, to ensure that levels are adequately protective of the population most likely to be affected.

Pao et al., 1982. Pao et al. (1982) used the data collected in the 1977-1978 USDA survey to derive frequency distributions for the rates of consumption of different foods. Based on their analysis of these data, Pao et al. reported median, mean, and 95th percentile consumption rates for all types of fish and shellfish. These rates were based on data collected from individuals who had eaten fish at least once during the 3-day study period. EPA (1989a) has indicated that data from 3-day dietary records should not be used to estimate annual rates of consumption because many individuals eat fish less frequently than once in three days.

Anglers - Fish from All Commercial and Recreational Sources

Because anglers may consume sport-caught fish in addition to commercially available fish, they are generally assumed to have a higher rate of fish consumption than the general population. As a result, many regulatory programs identify anglers as a subpopulation of concern. Use of an angler's total sport-caught and commercial fish consumption rate is appropriate when evaluating areas where contamination is widespread and where a number of commercial and recreational fisheries are affected, because angler's total fish consumption is likely to include fish from both sources. Examples of studies focusing on total consumption by anglers are discussed below and are summarized in Table 2.

NYSDEC, 1990. Connelly et al. (NYSDEC, 1990) conducted a long-term recall mail survey of New York State anglers in which anglers were asked to recall the number of fish meals consumed over a one-year period. The authors reported that the average New York angler consumed 45 fish meals annually. Assuming an average fish meal size of 227 g (1/2 pound), the average New York angler would consume approximately 28 g of fish daily. Even though anglers were the population targeted for the survey, this estimate included sport-caught fish as well as freshwater, marine, and estuarine fish obtained from markets, restaurants, and as gifts.

TABLE 2. Fish Consumption Estimates for Recreational Anglers

Study	Consumption Rates (g/d)		
	Mean	Median	"High End"
All Commercial and Recreational Sources			
Fiore et al. (1989)	26	—	63[a]
NYSDEC (1990)	28	—	—
West et al. (1989)	18.3	—	—
Marine - Self-Caught			
Landolt et al. (1985; 1987)	—	15[b]	—
Pierce et al. (1981)	—	23	>54[a]
Puffer et al. (1981)	—	37	339[a]
Multiple Fresh Water bodies			
Connelly et al. (1992)	6.8	—	32[c]
Cox et al. (1985)	21.8	—	—
Cox et al. (1987)	19.4	7.5	—
Cox et al. (1990)	—	7.5	—
Ebert et al. (1993)	6.4	2.0	26[a]
Fiore et al. (1989)	12.3	—	37.3[a]
West et al. (1989)	7	—	—
Multiple Flowing Waterbodies			
Ebert et al. (1993)	3.7	0.99	12[a]
Multiple Lakes and Ponds			
ChemRisk (1991b)	4.2	1.7	15[a]
Richardson and Currie (1993)	16.2	—	—
Specific Waterbodies			
ChemRisk (1991a)	3.0	0.49	11[a]
Soldat (1970)	1.8	—	—
Honstead et al. (1971)	7.7	—	—
Turcotte (1983)	7.4[d]	—	—

[a] 95th percentile.
[b] Calculated using a Monte Carlo simulation based on frequency distributions provided by authors.
[c] 92nd percentile.
[d] Calculated based on 2.5 consumers per angler.

West et al., 1989. West et al. (1989) conducted a stratified mail survey of Michigan's anglers and asked them to report their consumption of all types of freshwater fish meals for the previous two-week period. The average consumption rate reported by West et al. (1989) included sport-caught, purchased, gift, and restaurant-purchased freshwater fish.

Fiore et al., 1989. Fiore et al (1989) used a long-term recall mail survey to evaluate consumption of fish by Wisconsin's anglers. In this survey, the authors differentiated between sport-caught and commercially obtained meals. Average daily intakes were reported.

Anglers - Sport-caught Marine Fish

When the affected surface water is a marine waterbody that is frequented by recreational anglers, it is advisable to use estimates of consumption that have been derived from surveys of marine anglers.

Pierce et al., 1981. Pierce et al. (1981) interviewed anglers fishing Commencement Bay in Puget Sound near Tacoma, Washington. Estimated rates were based on the consumption of sport-caught marine finfish and shellfish. Using the Pierce et al. (1981) data, the EPA (1989a) estimated the median rate of consumption by these fishermen to be 23 g/d. A reanalysis of the original raw data, which corrected for oversampling of frequent anglers, resulted in an estimated median rate of 1.0 g/d (Price et al., 1994).

Puffer et al., 1981. Puffer et al. (1981) conducted a creel survey of the consumption of marine fish by anglers who fished Los Angeles Bay. Although all of the fishermen observed in the study were counted, only those fishermen who had creeled fish were subsequently interviewed. The authors reported that the median consumption rate for those successful anglers was 37 g/d. This consumption rate represented consumption of sport-caught marine species from a large marine fishery. Because it oversampled the most frequent Los Angeles Bay anglers (Puffer et al., 1981), it likely overstates consumption for the majority of anglers using that fishery. Price et al. (1994) report that when a correction is made for the oversampling of frequent anglers in the Puffer et al. (1981) study, the resulting median consumption rate is less than 2.9 g/d.

Landolt et al., 1985, 1987. Landolt et al. (1985; 1987) conducted a two-year creel survey of Puget Sound anglers. Based on data collected during interviews with over 2,000 anglers, Landolt et al. reported distributions for the number of trips per year, number of fish caught per trip, numbers of individuals sharing the catch, and the edible weight of each fish caught. Landolt et al. (1985; 1987) calculated average, species-specific consumption rates that ranged from 11 to 40 g/d. However, because angler effort and availability of those species were highly variable through the season, these species-specific estimates cannot be combined to produce estimates of total annual consumption rates.

Anglers - Sport-caught Freshwater Fish from Multiple Waterbodies

In some situations, contamination may affect numerous freshwater recreational fisheries within a given region, but does not impact commercial fisheries. In this situation, it is recommended that exposure assessors select estimates of total sport-caught fish consumption for use in their analyses.

West et al., 1989. As discussed previously, West et al. (1989) reported an average consumption rate for freshwater fish of 18.3 g/d. Although the authors did not specifically derive an estimate of consumption of sport-caught fish, they did indicate that 39% of the freshwater fish consumed by Michigan anglers were sport-caught. Thus, applying this percentage to their mean consumption estimate, an estimate of 7 g/d can be derived for the amount of sport-caught fish eaten by Michigan anglers. This estimate includes fish caught from all fresh waterbodies in Michigan.

Fiore et al., 1989. In the Fiore et al. (1989) analysis, consumption of fish by Wisconsin's anglers was evaluated. Average and 95th percentile rates of consumption of sport-caught freshwater fish were reported from all sources in Wisconsin.

Ebert et al., 1993. A long-term mail recall study of Maine's anglers was conducted by Ebert et al. (1993). In this survey, anglers were asked to recall numbers and sizes of fish harvested for consumption during ice fishing and open water fishing trips in Maine. A distribution of percentiles of fish consumption rates for those respondents who indicated that they had consumed some fish during the year was provided. These estimates included sport-caught freshwater fish harvested from all fresh waterbodies in Maine.

Connelly et al., 1992. A long-term recall mail survey was used by Connelly et al. (1992) to determine rates of sport-caught freshwater fish consumption by licensed New York anglers. The authors reported that mean consumption was 11 meals per year. Using a conservative estimated meal size of 227 g results in an estimated annualized consumption rate of 6.8 g/d. From the data provided by Connelly et al. (1992) the 92nd percentile can be estimated at 32 g/d.

Cox et al., 1985, 1987, 1990. Cox et al. have reported results of a number of surveys conducted of Ontario anglers. These surveys were in the form of questionnaires included in the "Guide to Eating Ontario Sport Fish", which gives consumption advice and is updated annually. Based on responses received from the 1983 questionnaire, Cox et al. (1985) reported a mean freshwater fish consumption rate of 21.8 g/d. A similar mean of 19.4 g/d was reported by Cox et al. for their 1986 survey (Cox et al., 1987). Although the raw data from the 1983 Ontario survey are no longer available, Cox et al.[1] have reported that the median consumption rates from both the 1986 and the most recent Ontario study (Cox et al., 1990) were both 7.5 g/d.

Anglers — Sport-caught Fish from Multiple Rivers/Streams

Ebert et al. (1993) and ChemRisk (1991b) established that consumption rates for fish taken from moving waters (rivers and streams) differ from consumption rates for still waters (ponds and lakes). When contamination affects multiple rivers and streams that are recreational fisheries in a given region, but does not affect standing waters, it is most appropriate to use

[1] Cox — Personal Communication

estimates of consumption of river/stream fish by anglers. To our knowledge, this is the only published study of the consumption of fish from multiple flowing waters.

Ebert et al., 1993. As discussed previously, Ebert et al. (1993) conducted a recall survey of Maine's resident freshwater anglers. Although responding anglers were not asked to recall exact locations where individual fish were harvested, they were asked to report numbers of fish harvested for consumption that were obtained from standing waters (lakes and ponds) and from flowing waters (rivers and streams). Using these data, the authors evaluated consumption from individual types of waterbodies by considering only those fish reported by anglers to have been harvested from the particular type of waterbody. Thus, it was possible to estimate a full distribution of consumption rates for those anglers who reported that they ate fish from rivers or streams. These estimates were not waterbody-specific, but rather were estimates of total consumption of freshwater river/stream fish by Maine's consuming resident anglers.

Anglers — Sport-caught Fish from Multiple Lakes/Ponds

When contamination affects multiple lakes and ponds that are recreational fisheries in a given region, but does not affect flowing waters, it is preferable to estimate ingestion of lake/pond fish by anglers.

ChemRisk, 1991b[2]. In an additional, unpublished analysis of data obtained from their Maine angler survey (Ebert et al., 1993), ChemRisk (1991b) reported the rates of consumption of fish recreationally obtained from lakes and ponds in Maine. These estimates were not waterbody-specific but rather were estimates of total consumption of lake/pond fish by Maine's consuming resident anglers.

Richardson and Currie, 1993. Richardson and Currie (1993) used measured concentrations of total mercury in the hair of Ontario Amerindians as a means of estimating rates of fish consumption by this population. An average concentration of mercury in fish tissues (regardless of species) from multiple lakes within a 100 km radius of each reserve was assumed to be the concentration in consumed fish. To derive estimates of consumption, it was assumed that all measured mercury in fish was methyl mercury, that 100% of the mercury was absorbed, that the half-life in the body is 70 days, and that hair grows at a rate of 1 cm per month. Actual sources of fish consumed, species consumed, and number of meals consumed were unknown. Using the levels of mercury measured in the hair of study participants, the authors reported geometric mean consumption rates of 19 and 14 g/d for male and female Amerindians, respectively.

Anglers — Sport-caught Fish from Specific Waterbodies

Often regulatory actions, like effluent permitting or the selection of remedial options, are targeted to a specific waterbody. When contamination is limited to a single waterbody, the proportion of total consumption resulting from that waterbody is the relevant estimate of

[2] Unpublished data.

interest. If possible, waterbody-specific estimates should be based on local data collected for the site (EPA, 1989b). If it is not possible to collect information on potential consumption from the waterbody in question, then the next step is to evaluate whether estimates of waterbody-specific consumption from other similar waterbodies can be substituted and used as reasonably representative of the waterbody being studied. While a number of surveys have been conducted over the years to determine fishing participation and harvest rates, only a few have specifically evaluated rates of consumption of fish harvested from a specific waterbody.

Soldat, 1970. Soldat (1970) conducted a creel survey of the Upper Columbia River in the Hanford area and reported that the average angler surveyed took 4.7 trips per year and harvested 0.7 meals per trip from the Upper Columbia River annually. Soldat (1970) reported that 45,000 meals were caught, representing 20,000 pounds of edible fish (202 grams per meal). Using this reported 202 g fish meal size, the resulting estimate of consumption from the Soldat study is 1.8 g/d.

Honstead et al., 1971. As reported by Rupp et al. (1980), Honstead et al. (1971) conducted a recall survey and reported that Upper Columbia River anglers consumed an average of 14 meals of sport-caught fish per year and that the average meal size was 200 grams. Based on this, it can be estimated that anglers consumed 2.8 kg per year or approximately 7.7 g/d on average.

Turcotte, 1983. Through data collected in a creel survey, Turcotte (1983) evaluated harvest of freshwater species from non-tidal reaches of the Savannah River and estimated that the average angler harvested 22.6 kg of fish per year. Using an EPA (1989b) estimate that 30% of the harvested fish is edible, results in an edible harvest of 6.8 kg/year or 19 g/day. However, this estimate does not account for sharing of fish with other individuals. In addition, it is based on the assumption that all harvested fish were consumed and did not consider that some fish were likely to have been given away, discarded, or used as bait. If it is assumed that all harvested fish are eaten and that an average of 2.5 individuals shared in the consumption, a value that has been reported in several studies (Puffer et al., 1981; Landolt et al., 1985; Ebert et al., 1993), the resulting estimate is 7.4 g/d.

ChemRisk, 1991a[3]. ChemRisk (1991a) conducted a creel survey of the West Branch of the Penobscot River. In estimating an upper-bound annual consumption rate based on data collected from single interviews of successful anglers, ChemRisk conservatively assumed that each angler was successful on every trip and that the frequency of fishing trips taken up to the time of the interview continued throughout the remainder of the season. Using this methodology for the consuming angling population, a full distribution of consumption rates, with a mean of 5.1 g/d, was reported. However, because it was believed that these assumptions were likely to result in overestimates of consumption by the interviewed anglers, ChemRisk conducted an additional analysis, using fisheries management data simultaneously

[3] Unpublished data.

collected from the West Branch, in which the trends in participation and harvest rates over the season were identified. These trends were used to calculate monthly adjustment factors for fishing frequency and harvest rates which were then incorporated into a Monte Carlo analysis to derive a distribution of consumption rates for the West Branch that considered seasonal fluctuations. This analysis indicated that consumption rates were lower than originally estimated with a mean of 3.0 g/d and a median of 0.49 g/d.

DISCUSSION

While the wide range of consumption values that have been reported in the scientific literature would seem to indicate that rates of fish consumption are highly variable, this variability can be attributed primarily to differences in the types of fish being eaten, the source or sources of those fish, the characteristics of the population being evaluated, and the methods used to collect consumption data. As demonstrated in Table 3, the sources (recreational vs. commercial, marine vs. freshwater, etc.) from which fish have been obtained appear to have a substantial effect on the estimated rates of consumption. Surveys that have considered all sources of fish tend to have the highest estimates of average intakes, while surveys that have focused on a single fresh waterbody tend to have the lowest. When surveys involving similar sources of fish are compared, estimates of consumption are similar.

Based on the data presented in Table 3, the following conclusions can be reached:

- Rates of intake from individual bodies of water are lower than rates of intake from multiple bodies of water;
- Rates of consumption of sport-caught marine fish are generally higher than rates of consumption of sport-caught freshwater fish; and,
- Rates of intake from moving waters are lower than rates from still waters.

Although it appears that rates of consumption of marine fish may be higher than rates of consumption of freshwater fish when comparing studies of marine anglers with those of freshwater anglers, the recent Price et al. (1994) reanalysis of the Puffer et al. and Pierce et al. studies indicates that consumption of marine fish by anglers may be comparable to consumption of freshwater fish, when survey biases are minimized. However, this conclusion cannot be reached with certainty and is an area for future research.

An important additional observation is that the estimate of the "high end" angler intake (the top 10% of anglers) is greatly affected by the duration of the survey. Table 4 presents intake rates of sport-caught fish at the 95th percentile, according to the survey method used. Available intake estimates for the 95th percentile consumer are less than 40 g/d for all long-term (greater than 30- day recall period) surveys. Much higher estimates are found in surveys of shorter duration, likely due to short-term variability biasing the results upward. Because the

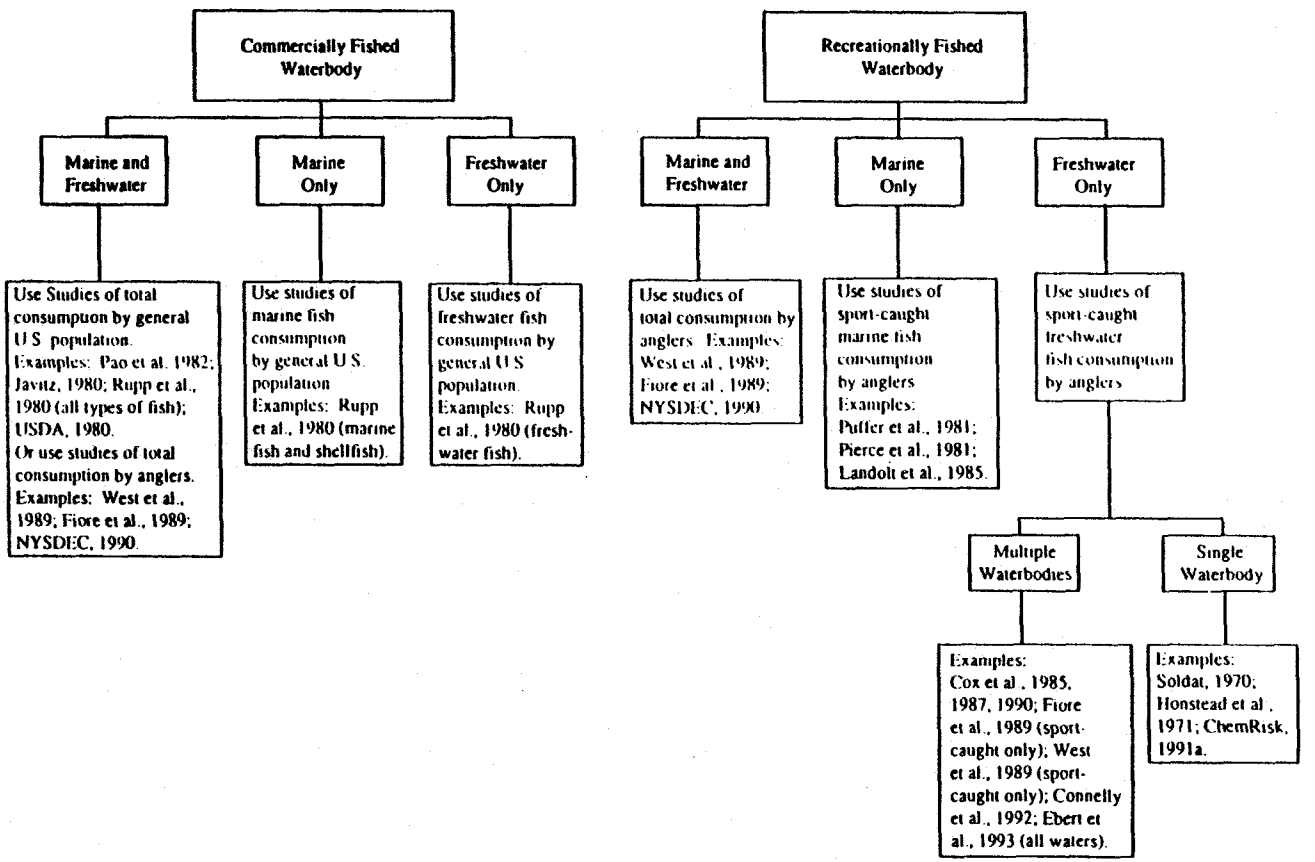

FIGURE 2. Selection of fish consumption rates based on type of waterbody and potentially exposed population.

TABLE 3. Estimates of Average Fish Consumption Rates Per Sources of Consumed Fish(g/d)

Source and Waterbody Type	Range of Average Rates	Reference
General Population Surveys[a]		
Marine, freshwater, and estuarine	12.7 to 54	Javitz et al., 1980 Rupp et al. 1980 USDA, 1980 Pao et al., 1982
Marine only	8.8	Rupp et al., 1980
Freshwater only	1.2	Rupp et el., 1980
Angler Surveys[b]		
Marine, freshwater, and estuarine	18.3 to 28	West et al., 1989 Fiore et al., 1989 NYSDEC, 1990
Marine only	15 to 37[c]	Pierce et al., 1981 Puffer et al., 1981 Landolt et al., 1985
Freshwater-multiple waterbodies	6.4 to 21.8	Cox et al., 1985, 1987, 1990 Fiore et al., 1989 West et al., 1989 Connelly et al., 1992 Ebert et al., 1993
Freshwater-multiple standing waters	4.2 to 16	Richardson and Currie, 1993 ChemRisk, 1991b
Freshwater-multiple flowing waters	3.7	Ebert et al., 1993
Freshwater-single waterbody	1.8 to 7.7[c]	Soldat, 1970 Honstead et al., 1971 Turcotte, 1983 ChemRisk, 1991a

[a] Estimates of consumption by the general population of the United States, including anglers and non-anglers.
[b] Estimates of consumption by anglers only.
[c] These rates are likely to be overestimated due to the oversampling of more frequent anglers during creel surveys.

estimates from the long-term surveys are not subject to short-term variability, they are preferred for estimating average annual consumption rates by risk assessors. This analysis suggests that consumption rates for the general angler population rarely reach the levels of between 140 and 180 g/d frequently recommended for evaluating "high-end" intake (EPA,

1989a,b). Although Puffer et al. (1981) reported a 95th percentile value in exceedance of 180 g/d, Price et al. (1994) have recently demonstrated that this high estimate is not representative of the 95th percentile of the total angler population using the fishery. Reanalysis of the Puffer et al. (1981) data to correct for sampling bias has resulted in an estimated 95th percentile of approximately 35 g/d.

TABLE 4. A Comparison of Estimated Rates of Self-Caught Fish Consumption Per Duration of Recall Period

Recall Period	Range of "High-End" Intakes (g/d)	Reference
1 day	54 to 339	Pierce et al., 1981[b] Puffer et al., 1981[b]
3 day	128	Pao et al., 1982
30 days	42	Javitz, 1980
365 days	26 to 37	Fiore et al., 1989 Connelly et al., 1992 Ebert et al., 1993

[a] All values are reported 95th percentile except Connelly et al. (1992) for which the reported value represents the 92nd percentile.
[b] Reanalyses of these data by Price et al. (1994) have resulted in substantially lower estimates of "high-end" intakes.

The EPA (1989b) has acknowledged that there are substantial regional- and site-specific variations in consumption rates and, as a result, has recommended that site- or region-specific consumption estimates be used wherever possible. Clearly this is preferable due to the variability that can occur among fisheries because of differences in lengths of fishing seasons, the availability of fisheries, the availability of target species, fishing regulations, and the cultural or ethnic backgrounds of the fish consumers.

Unfortunately, due to time constraints or resource limitations, it is not always possible to collect site-specific information or to have the complete distribution. In lieu of these, it becomes necessary to select the most representative consumption estimate based on the population, region, waterbody type, and fishery type of interest.

In risk assessments performed for regulatory purposes, it is important that the fish consumption rate selected be derived from studies that are consistent with the type of waterbody and target population being evaluated. Freshwater fish consumption estimates should not be based on studies of marine fisheries because there are likely to be differences in the species present, the relative productivities of the waters, and the preferences of the anglers. If fish ingestion from a single waterbody is being evaluated, it is best that the rate of intake be based upon a valid intake study from a similar, individual waterbody. It is particularly important to consider whether there are any commercial fisheries on the waterbody of interest.

If there are none, then the rates of intake used should be based on studies which have considered only the intake of sport-caught fish and should not include consumption of fish that have been obtained from restaurants, markets, or other, non-angling sources. General guidance on the selection of appropriate fish consumption estimates is provided in Figure 2.

It is also important to consider the species and size of fish available in the waterbody of interest. Because the species targeted vary among fisheries and among regions, and because different species vary in their propensity to bioaccumulate persistent compounds, exposure potentials may differ substantially. Thus, for risk assessment purposes, it would be ideal to derive species-specific rates of consumption for individual anglers and to combine the intake rates with species-specific fish tissue levels to more accurately define exposures.

It is important to note that a discussion of the selection of consumption rates for subpopulations that may consume more fish than recreational anglers is beyond the scope of this paper. In conducting an exposure assessment, careful consideration must be given to whether such a sensitive subpopulation exists due to income level or ethnic background. If it does, it may be appropriate to select consumption rates that are based on either site-specific studies or studies of similar populations.

In the absence of site-specific information, the selection of a fish consumption rate to be used in the assessment of risks from a contaminated area involves three critical factors. First, the population most likely to be affected must be identified. Second, if possible, the selection of a fish consumption rate for a particular geographic area should be based on a study that has evaluated similar areas with similar resources. Differences in climate, target species, length of fishing season, availability of marine and freshwater fisheries, and cultural/ethnic background can substantially influence rates of consumption. Lastly, waterbody and fishery types are important considerations. Often the population that is most likely to be affected includes anglers who fish the contaminated waters. If contamination is widespread throughout an area, then it may be appropriate to select a consumption estimate from a study that has evaluated total consumption of sport-caught fish by anglers (Fiore et al., 1989; Ebert et al., 1993). If the area affected is a marine area, then estimates of marine fish consumption are most appropriate. Conversely, if the area affected is an inland area, then estimates of freshwater fish consumption should be used. Finally, if only a single waterbody is affected by contamination, the fish consumption rate selected for the evaluation should, if possible, be a rate that has been derived from a study of a waterbody that is similar in nature to the one of interest. If it is not possible to identify a single waterbody within a given region that is directly comparable with the waterbody being evaluated, then a more general estimate of consumption, based on the most comparable study, may serve as a useful surrogate.

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THE EFFECT OF SAMPLING BIAS ON ESTIMATES OF ANGLER CONSUMPTION RATES IN CREEL SURVEYS

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EPA guidance recommends that 30 grams per day be used to represent the consumption rate of fish caught from large bodies of water by a typical angler (EPA, 1989a). This estimate is based on the combined results of the Pierce et al. (1981) and Puffer et al. (1981) surveys of marine and estuarine anglers. An examination of these surveys demonstrates that the method used in both studies — creel survey — oversamples frequent anglers and produces a distribution of consumption rates that overestimates intake rates of the total angler population using the surveyed waterbodies. Weighting the individual survey responses by the inverse of the angler self-reported fishing frequency corrects this bias and produces a more accurate characterization of the total population of anglers using the surveyed waterbodies. This approach is an extension of the methodology used by both Puffer et al. (1981) and Pierce et al. (1981) to estimate the size of the total angler populations. The results of the reanalysis of the Pierce et al. (1981) survey indicate that the median consumption rate for the total angler population is 1.0 g/d. The results of the Puffer et al. (1981) reanalysis indicate a median consumption rate for total angler population of 2.9 g/d. The recalculated distributions of consumption rates were found to be consistent with the results of other angler surveys that use survey methods that do not oversample frequent anglers. The angler intake rate of 30 g/d corresponds to roughly the 90th and 95th percentiles of the total angler populations in the Pierce et al. (1981) and Puffer et al. (1981) surveys, respectively. The results of this paper indicate that the current estimate of 30 g/d significantly overestimates consumption for typical marine and estuarine anglers.

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2. Abbreviations: EPA, United States Environmental Protection Agency; g, grams; g/d, grams per day; NYSDEC, New York State Department of Environmental Conservation; y, year; trips, one day fishing trips

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INTRODUCTION

The rate of consumption of self-caught fish is a critical parameter for many environmental risk assessments. Because persistent lipophilic compounds that are released to surface waterbodies may bioaccumulate in fish, often the most important route of human exposure to these chemicals is through fish consumption (Humphrey, 1983; EPA, 1984; Rifkin et al., 1991; Sherman et al., 1992). Because many surface waterbodies, and in particular most freshwaters, are not commercially fished, consumption of fish is limited to recreational anglers. While such individuals may only represent a fraction of the total population living near an affected body of water, they may represent the majority of risks posed by surface water contamination. Therefore, it is critical to accurately characterize the rate of fish consumption for recreational anglers. Currently, EPA guidance recommends that a rate of 30 grams per day be used to represent the ingestion rate of fish caught from large bodies of water by a typical angler (EPA, 1989a,b). This estimate is based on the combined results of the Pierce et al. (1981) and Puffer et al. (1981,1982) creel surveys (hereafter referred to as the Pierce and Puffer surveys) of marine and estuarine anglers.

Creel surveys are typically used by fisheries managers to evaluate angler participation, effort, and catch/harvest rates from an individual waterbody. Such surveys generally count and interview anglers observed fishing a specified body of water at a specified time. During these surveys, data are collected specific to the individual angler's fishing experience, such as the length of the trip, and the number, size, and species of fish targeted, caught, and harvested by the angler on the day of the interview (EPA, 1991). More recently, creel surveys have been expanded to collect details on the anticipated disposition and/or consumption of the harvested fish (ChemRisk, 1991; Ebert et al., 1993).

A key characteristic of creel surveys is that the probability of an angler being interviewed during the survey is a function of his or her frequency of fishing (Puffer et al., 1981). Anglers who fish frequently have a higher probability of being interviewed than anglers who fish infrequently. As a result, creel surveys tend to oversample the frequent anglers. In addition, the distribution of consumption rates in the anglers interviewed during a creel survey are likely to overestimate the distribution of consumption rates in the entire population of anglers that fish the surveyed waterbody.

In this paper, we investigate the effect of this bias on the estimates of fish consumption that are derived from the Puffer and Pierce surveys. First, we used the inverse of each angler's self-reported annual frequency of fishing to reweight the estimated fish intake rate of each of the surveyed anglers (hereafter referred to as the survey population). This was done to calculate the distribution of consumption rates in the entire angler population that fishes the surveyed body of water (hereafter referred to as the total angler population). This approach is an extension of the methodology used by both Puffer et al. (1981) and Pierce et al. (1981) to estimate the size of the total populations of anglers using the waterbodies they surveyed.

Background

Pierce et al. (1981) surveyed anglers during the months of July through September (summer season) and September through November (fall season) of 1980. More than 500 interviews with individual anglers and fishing parties were conducted at five locations on Commencement Bay in Puget Sound, Washington. For each angler interviewed, the survey collected information on the number of fish caught on the day of the interview, the average weight of each fish caught, the number of people in the angler's family/living group, and the angler's annual fishing frequency. Pierce et al. (1981) presented summary statistics on the number and total weight of each fish species caught, number of anglers, family/living group size, and angling frequency.

Puffer et al. (1981) investigated rates of fish consumption by Los Angeles Harbor anglers. The survey included interviews of more than 1,000 anglers as they fished at 12 locations along the harbor during the summer and fall of 1980. The survey clerks collected information on the number of fish the anglers caught on the day of the interview, the average weight of the fish harvested, the number of fish eaters in the angler's family/living group, and the angler's annual fishing frequency.

Neither the Puffer nor Pierce creel surveys asked the individuals for direct estimates of the amount of fish they consumed. Rather, the surveys collected data on the size of catch, the angler's frequency of fishing, and number of individuals sharing in the catch. These data, along with information on the number and size of fish caught, were used to estimate a typical fish consumption rate for the angler. Puffer et al. (1981) estimated consumption rates of the individual anglers interviewed using the following equation:

$$C = (K * N * W * F/ 365) / E \quad (1)$$

Where C is the estimated daily fish consumption rate (g/person-day); K is the average edible fraction of the fish caught by a surveyed angler; N is the number of fish caught on the day of the survey; W is the average weight of the fish caught on the day of the survey (grams); F is frequency of fishing during the year; and E is the number of fish eaters in the anglers family or living group. Table 1 presents the distribution of fish consumption rates in the Puffer survey population published in Puffer et al. (1981).

Pierce et al. (1981) did not attempt to develop estimates of the consumption rates for the individual anglers. However, in the 1989 *Exposure Factors Handbook* (EPA 1989b), EPA developed an estimate of the distribution of fish consumption rates based on the information provided in the final report. Because Pierce and co-workers did not include the raw data for each of the anglers surveyed and only reported the distribution of angler responses to survey questions, the Agency could not calculate the individual angler's consumption rate using the approach developed by Puffer (Eq. 1). EPA was forced to estimate the distribution of consumption rates based on an alternative approach that used the estimate of the average

TABLE 1. Distribution of Fish Consumption Rates as Reported by Puffer et al. (1981)

Percentile	Consumption Rate (g/d)
5	2.3
10	4
20	8.3
30	15.5
40	23.9
50	36.9
60	53.2
70	79.8
80	120.8
90	224.8
95	338.8

amount of fish consumed by the surveyed anglers per fishing trip and the distribution of fishing frequencies given in the final study report. EPA estimated that the fish consumed by an average angler in the survey population was approximately 380 g/person per angling trip. The estimated distribution of annual consumption rates in the survey population was calculated using the equation:

$$C_F = 380 * F / 365 \quad (2)$$

Where, C_F is the daily fish consumption rate (g/d) of all anglers with a fishing frequency of F (trip/y). The distribution of fish consumption rates calculated by EPA (1989b) using this method is given in Table 2.

TABLE 2. Distribution of Fish Consumption Rates for the Pierce Survey as Estimated in the EPA Exposure Factors Handbook (1989b)

Percentile[a]	Consumption Rate (g/d)
0-<11	1.04
11-<16	2.09
16- <22	6.27
22-<40	12.53
40-<91	54.31
91-100	381.19

[a] Approximate

To derive its recommended rates for anglers, EPA (1989b) used the distributions from the two surveys to derive fish consumption rates for a typical and a worst-case angler (Table 3). The recommended rate of 30 g/d for the typical anglers was based on the arithmetic average of the

median consumption rates from the two surveys. 140 g/d was recommended as the "worst-case" consumption rate based on the arithmetic average of the 90th percentiles of the distributions of consumption rates in the two surveys.

TABLE 3. Rate Percentiles from Puffer and Pierce Surveys Used by EPA (1989b) to Derive Recommended Rates (g/d)

Survey	50th Percentile	90th Percentile
Puffer	37	225
Pierce	23[a]	54[a]
Average	30	140

[a] Estimated by EPA by interpolation

METHODS

Methodology

To calculate the distribution of consumption rates for the total angler populations represented by the two surveys, the estimated consumption rate of each individual angler surveyed was weighted by the inverse of the angler's self-reported fishing frequency. This approach is an extension of the methodology used by both Puffer et al. (1981) and Pierce et al. (1981) to estimate the relative sizes of the survey and total angler populations.

Both Puffer and Pierce recognized that their sample populations were only a fraction of the actual number of anglers using the surveyed waterbodies (total angler population). Both authors used the self-reported frequency of fishing to estimate the total angler population. The equation used was:

$$TAP = \sum_{i=1}^{k} N_F * 365 / F \qquad (3)$$

Where, TAP is the total angler population; and N_F is the number of anglers who reported a fishing frequency of F (trips/y), and k is the number of fishing frequencies reported. Under this approach, each of the anglers surveyed is assumed to be a member of a population of anglers who fish the surveyed body of water at the same frequency as the surveyed individual but most of whom are not fishing on the day the creel survey was performed. The size of this population will on average be equal to 365/F. The sum of these populations is taken as an estimate of the number of anglers in the total angler population for the surveyed waterbody. Table 4 presents the sizes of the survey and total angler populations for the Puffer and Pierce studies as reported by their respective authors.

TABLE 4. Population Size for the Sampled and Total Angler Population in the Puffer and Pierce Surveys

Survey	Sample Population	Total Angler Population
Puffer	1,059	91,606
Pierce	508	3,391

In this analysis, the distribution of consumption rates in the total angler population is calculated in a similar manner. Each of the surveyed anglers is assumed to represent 365/F anglers with similar consumption rates who fish the surveyed body of water. The equation used is:

$$TN_A = N_{AF} * 365 / F \quad (4)$$

where, TN_A is the total number of anglers with a consumption rate of A; and N_{AF} is the number of anglers with a consumption rate of A and a fishing frequency of F. The distribution of consumption rates in the combined populations, obtained by applying Equation 4 to all surveyed anglers, is taken as the distribution of consumption rates for the total angler population.

By a similar argument, the distribution of fishing frequencies in the total angler population can be estimated using the equation:

$$TN_F = N_F * 365 / F \quad (5)$$

where, TN_F is the total number of anglers with a fishing frequency of F; and N_F is the number of surveyed anglers with a fishing frequency of F. The distribution of fishing frequency in the total angler population is thus the distribution of fishing frequency in the combined population obtained by applying Equation 5 to all frequency categories in the survey.

To calculate the distributions of consumption rate and fishing frequency in the total angler population, it is necessary to know the values of N_{AF} and N_F for each of the two surveys. The values of N_{AF} can be developed from the data on individual anglers. The values of N_F used in this paper are taken from the original papers (Puffer et al., 1981; Pierce et al., 1981) wherever possible.

Analysis of the Two Creel Surveys

In order to obtain information on N_{AF} values for the two surveys, we contacted the original authors of the two studies and requested copies of the raw data. The raw data for the Pierce et al. survey were available from the Tacoma-Pierce County Health Department, Tacoma, Washington, in the form of paper copies of the original, completed survey forms.

Copies of a total of 687 interviews were received from the Tacoma-Pierce County Health Department. This number exceeds the number of anglers (508) reported to have been surveyed by Pierce et al. (1981). Many of the survey forms obtained were not usable due to missing data and other problems. This suggests that Pierce et al. (1981) performed some screening of the completed forms before they performed their analyses. Unfortunately, there was no indication of which survey forms had been included by Pierce et al. in their analysis and no information on the criteria used by Pierce et al. to select forms for inclusion in the analysis. Attempts to contact the original authors were unsuccessful. Therefore, we developed and used the following criteria for including survey responses in this analysis:

- All forms that contained incomplete data (with the exception of the fish weight and length data discussed below) were excluded;
- all forms that reported the catch for groups (rather than individual anglers) were excluded;
- all anglers that reported practicing catch and release (fish were not consumed) were excluded; and
- anglers who only consumed shellfish were excluded.

Using these criteria, we identified a total of 451 anglers appropriate for our analysis.

Data on fish consumption rates and other relevant parameters were extracted from the Pierce survey forms and entered into a database. Data taken from the survey forms included: interview number; number of individuals in the angler's living group; use of fish caught; frequency of fishing; fish species caught; number of fish caught; and species-specific average fish weights. Because the present analysis focuses on consumption of fish only, the consumption of crustaceans (crabs) was not considered in this analysis.

Approximately 3.5% of the survey forms included one or more fish without weight data. In addition, a few fish with missing weight data were also missing length data. We developed estimates of mass for these fish based on simple regression models of the relationship between species-specific fish mass measurements and lengths. These regression models were fitted to the fish in the survey that did report lengths or weights. In the few instances where the lengths of fish were also missing, the lengths reported for the same species in the same creel were used in the length-mass regression estimates.

Based upon the data extracted from the survey forms, we estimated a consumption rate for each angler using Equation 1. In developing these estimates, we used the same assumption of edible fraction of fish as reported by Pierce et al. (1981). These consumption rates and the reported fishing frequencies were used to determine the N_{AF} and N_F. The values of N_{AF} and N_F were in turn used to estimate the distribution of consumption rates and fishing frequencies in the total angler population, using Equations 4 and 5.

Unlike the Pierce survey, the raw data for the Puffer study have not been preserved (personal communication with Dr. Harold Puffer). No electronic or paper copies of extracted "raw" data from the survey forms were preserved, and only 350 of the "completed survey" forms (of the more than 1,000 original forms) are still available. Upon a review of the available forms, we determined that a meaningful analysis of the Puffer et al. raw data was not possible, given that less than one-third of the forms were preserved and the remaining forms could not be assumed to be a random sample of the original survey forms. Therefore, the only data available on the study are contained in the summary of the survey results in Puffer et al. (1981). This report on the Puffer survey does contain the distribution of angler frequencies from the N_F. Based on these data, we estimated the distribution of angling frequencies in the total angler population using Equation 5.

As demonstrated by EPA's *Exposure Factors Handbook* (EPA, 1989b), it is possible to obtain an estimate of the distribution of fish consumption rates in a population based on the average amount of fish consumed per fishing trip and the distribution of fishing frequencies (see Equation 2). We calculated the average amount of fish consumed per angler trip in the Puffer survey based on the mean consumption rate and fishing frequency of the anglers in the Puffer survey. The means of these parameters were estimated based on the reported distribution of consumption rates and frequencies (Puffer et al., 1981). The mean consumption rate was estimated to be 91 g/d. The average frequency was 63 trips per year. The average consumption rate per trip is therefore 522 g/person-trip. Using this estimate and the distribution of fishing frequencies in the total angler population, we developed a distribution of consumption rates for the Puffer survey.

Because different approaches were used to estimate the distributions of total angler population fish consumption rates for the Puffer and Pierce surveys, it is important to determine if the two different methods produce different estimates of the fish consumption rates. This was determined by applying both approaches to the Pierce survey results. The two resultant distributions of total angler population fish consumption rates were then evaluated for consistency.

RESULTS

Pierce et al.

The results of our reanalysis of the Pierce survey data are presented in Table 5 along with the results of Pierce's original analysis as reported in Pierce et al. (1981). In general, our estimate of the size of the survey population was smaller, and the surveyed anglers were estimated to consume more fish than the Pierce estimates. Another distinction between our reanalysis and the original analysis is that our study used all 15 angling frequency responses in the completed Pierce survey forms, while Pierce grouped the anglers into six frequency categories (see Table 5).

TABLE 5. **Comparison of Survey Populations as Estimated from the Reanalysis of the Pierce Survey with the Results for the Survey Population Reported in Pierce et al. (1981)**

Parameters	Pierce et al. (1981)	Reanalysis (Consuming Anglers)
Number of Anglers		
Summer	304	225
Fall	204	226
Total	508	451
Total Mass of Fish Caught	2,700 kg	3,300 kg
Average Family Size	3.74	3.65
Number of Trips/Year	Percent of Anglers	
1	10.85	9.76
2	5.40	5.99
3		0.22
4		0.22
6	5.25	3.77
8		0.22
12	18.45	19.07
24		1.11
36		0.44
52	51.30	44.79
93		0.22
104		3.99
156		3.33
208		1.33
365	9.40	5.45

The cumulative distributions of the angling frequencies and daily fish consumption rates for the survey population and total angler population in our reanalysis of the Pierce survey data are presented in Figures 1 and 2. Because the exposure frequencies for individual anglers were evaluated using rough categories of frequency, the distributions can only be specified for a limited number of points on the distribution. The distributions of angling frequencies (Figure 1) show a disproportionate number of anglers at the higher frequencies, e.g., more than 50% of respondents fish more than once a week in the survey population, while less than 6% of the total angler population fish this often. The distribution of angler consumption rates in the survey and total angler population show a similar shift. In the survey population, the median consumption rate is 19 g/d. In the total angler population, less than 6% of the population has a consumption rate of 19 g/d or more.

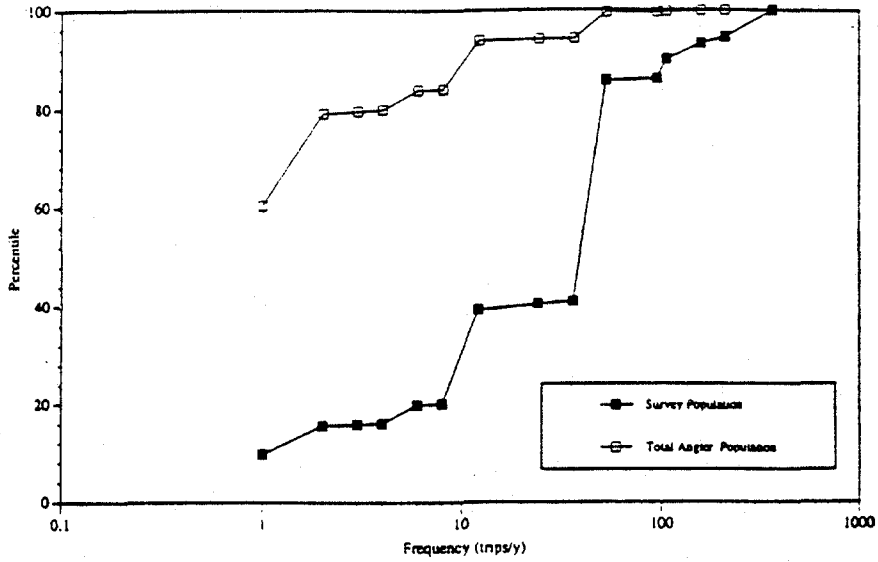

FIGURE 1. Comparison of the estimated cumulative distribution of angling frequencies for the survey population with the total angler population from the reanalysis of the Pierce survey.

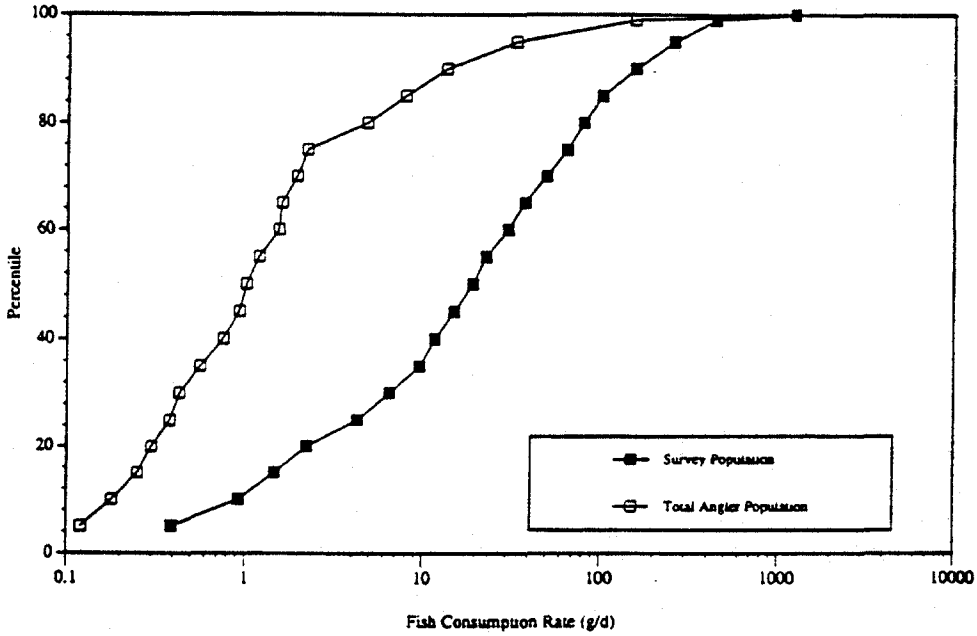

FIGURE 2. Comparison of the estimated cumulative distribution of fish consumption rates for the survey population with the total angler population from the reanalysis of the Pierce survey.

The cumulative distribution of angling frequencies for the survey and total angler populations in the Puffer et al. (1981) survey are presented in Figure 3. The distribution of angling frequencies in the survey population show a disproportionate number of anglers at the higher frequencies with approximately 50% fishing more than once a week. However, in the total angler population, less than 1% of the population fishes with this or greater frequency.

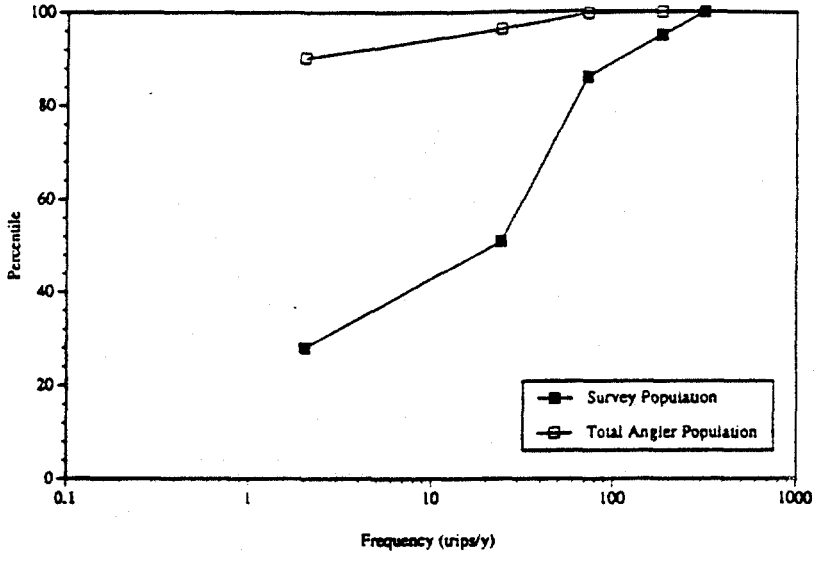

FIGURE 3. Comparison of estimated cumulative distributions of angling frequency in the survey population with the total angler population from the reanalysis of the Puffer survey.

The cumulative distribution of angler consumption rates for the total angler and survey populations derived from the Puffer et al. (1981) data is given in Figure 4. The distribution of survey population rates are taken directly from Puffer et al. (1981). The distribution of consumption rates in the total angler population is derived from the distribution of fishing frequencies for the total angler population. Because the fish consumption rate estimates in the total angler population of the Puffer survey (Figure 4) are based on angling frequency data, the consumption rate curve does not appear to be as smooth as the curve for the Pierce data (Figure 2).

The differences in the consumption rate distributions for the two populations show the same pattern as the Pierce survey results. The median consumption rate in the survey population is 36.9 g/d. In the total angler population, less than 5% of the population has a consumption rate that is greater than or equal to this value.

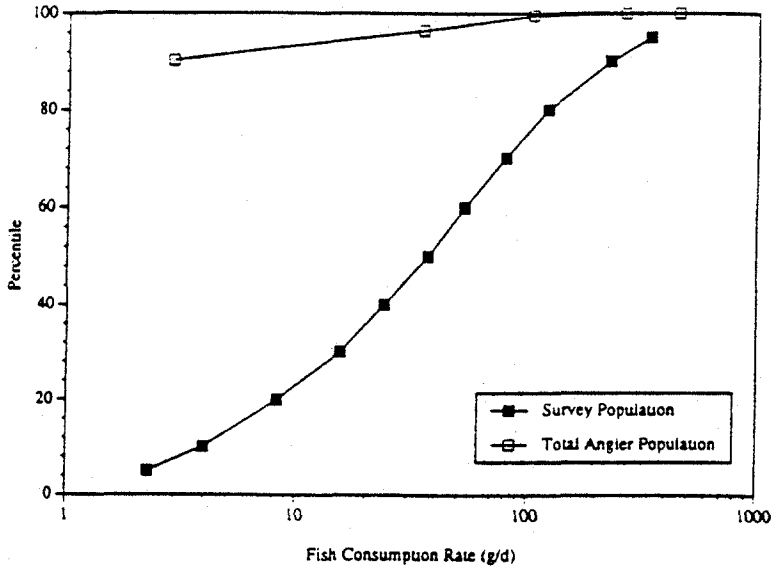

FIGURE 4. Cumulative distributions of fish consumption rates for the survey population with the total angler population from the reanalysis of the Puffer survey.

Table 6 presents the estimated consumption rates for the median and 90th percentiles for the survey and total angler populations derived from the reanalyses of the Pierce and Puffer survey data. In the Puffer survey, the values for the median are taken from the lowest frequency group

TABLE 6. Selected Percentile Consumption Estimates (g/d) for the Survey and Total Angler Populations Based on the Reanalysis of the Puffer and Pierce Data

	50th Percentile	90th Percentile
Survey Population		
Puffer	37	225
Pierce	19	155
Average	29	190
Total Angler Population		
Puffer	2.9[a]	35[b]
Pierce	1.0	13
Average	2.0	24

[a] Estimated based on the average intake for the 0-90th percentile anglers.
[b] Estimated based on the average intake for the 91st-96th percentile anglers.

(less than once a month) that comprises 90% of the total angler population. The 90th percentile is conservatively estimated based on the estimated consumption rate for the next lowest frequency (1-3 times per month) that represents the 91st to the 96th percentiles of the total angler population. In both surveys, the medians and the 90th percentiles of the total angler population are one to two orders of magnitude lower than the equivalent values for the survey populations.

Figure 5 presents two estimates of the distribution of consumption rates for the total angler population in the Pierce et al. (1981) survey. As the figure indicates, the cumulative distribution produced by using the average fish consumption per fishing trip approach overestimates consumption rates for anglers with the lowest consumption rates. However, the two approaches produce similar estimates for the upper portion of the consumption distribution.

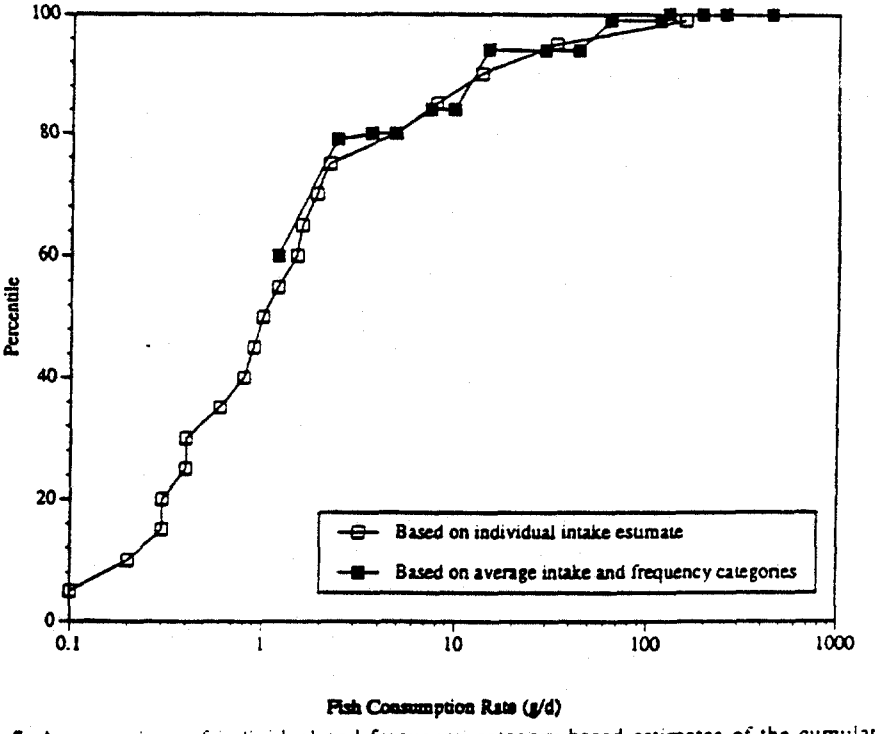

FIGURE 5. A comparison of individual and frequency category-based estimates of the cumulative distribution of fish consumption rates in the total angler population from the Pierce survey.

DISCUSSION

The primary finding of the analysis presented in this paper is that the distribution of consumption rates derived for a survey population differ substantially from the distribution of consumption rates derived for the total population of anglers using a given waterbody. Total

angler populations have much lower fishing and consumption rates than survey populations. This difference occurs because creel surveys oversample more frequent anglers and the intakes of the surveyed anglers provide a biased estimate of the total angler population's intake.

This finding of sharply lower intakes is not due to our reanalysis of the Pierce survey results. Our reanalysis of the Pierce survey data resulted in slightly higher estimates of intake for the survey population than EPA's analysis (EPA, 1989b) (see Tables 3 and 6), due to higher estimates of total catch and lower estimates of the number of anglers. It is not clear why we derived higher estimates of fish caught. The smaller number of anglers included in our analysis is probably due to the elimination of anglers practiced catch and release or who only consumed shellfish.

This paper used two different methods of estimating the distribution of consumption rates for the total angler population. The first approach (Equation 4) includes information on the inter-individual variation in the number of fish caught, the size of the fish, and the number of individuals sharing the fish. This approach was used to reevaluate the Pierce survey results. The second approach (Equation 5) is frequency-based and does not consider these sources of variation. It uses an estimate of the average fish consumption rate per angler trip. This second approach was used to analyze the Puffer survey. The second approach would be expected to underestimate the variation in the distribution of consumption rates because it would not include the variations in size of catch and the number of individuals sharing the catch.

In order to investigate the impact of using the two different approaches, we applied both approaches to the Pierce survey results. Figure 5 presents the resulting distributions of angler consumption rates in the total angler population from the two approaches. As the Figure indicates, the second approach's inability to fully characterize the extremes in consumption rates is clearly apparent in the lower end of the consumption rate distribution. However, the difference between the two estimates appears to be minimal for the upper end of the distribution. This suggests that the use of the second approach, while theoretically less desirable, provides a reasonable estimate of consumption rates for the "typical" and "high end" anglers.

The information on N_F for the two surveys is somewhat limited by the relatively coarse measurement of self-reported fishing frequency. As Table 7 indicates, both surveys asked for the frequency of fishing in terms of once a day, once a week, once a month, etc. As a result, the estimates of consumption rate and frequency developed using Equations 4 and 5 do not appear as smooth distributions. In addition, it is difficult to estimate the average frequency of angling for some categories. For example, there is considerable uncertainty in the actual frequency for individuals in the Puffer survey who reported that they fish less than once a month. In this analysis, we used the average frequency proposed for each of the frequency categories by the original authors wherever possible (see Table 5). This problem is exacerbated in the estimates of frequency for the total angler population. In both the Puffer and Pierce surveys, more than 66% of the total angler populations fall into the lowest frequency

category and only a single estimate of fishing frequencies can be made for these large portions of the populations. This absence of data on the infrequent angler is directly related to the bias in the creel survey methodology toward the frequent angler.

TABLE 7. Estimates of Average Angling Frequencies (trips/y) for Angling Frequency Categories Reported in Puffer et al. (1981) and Pierce et al. (1981)

Category	Average Angling Frequency
Puffer	
Infrequnct (<1/mo)	2
1-3 times/month	24
1-2 times/week	72
3-4 times/week	182
5-7 times/week	312
Pierce	
Yearly	1
2 times/year	2
3 times/year[a]	3[b]
Every 3 months[a]	4[b]
Bimonthly	6
8 times/year[a]	8[b]
Monthly	12
2 times/month[a]	24[b]
3 times/month[a]	36[b]
Weekly	52
Daily during summer[a]	93[b]
2 times.week[a]	104[b]
3 times/week[a]	156[b]
4 times/week	208[b]
Daily	365

[a] Not included in the original Pierce et al. (1981) report but reported by respondents on original survey intake forms.

[b] Estimated by the current authors.

There are two major implications for the findings in this paper. First, current EPA policy on exposure assessment calls for the evaluation of the dose rates received by a population in terms of the "typical" and "high end" exposure rates (EPA, 1991). These rates are to be established for the total angler population exposed to the contaminant. Use of point estimates of consumption by "typical" and "high end" anglers in a survey population to characterize the consumption rates in the total angler population will result in a significant overestimation of consumption rates. A comparison of Table 3 and Table 6 indicates that the estimates of the typical angler derived by EPA (1989b) may be high by one to two orders of magnitude. In fact, the estimate of typical angler consumption, 30 g/d, roughly corresponds to the 95th and 90th percentiles of the total angler populations in the Pierce and Puffer surveys, respectively.

Second, the resulting distribution of consumption rates for the total angler population is expected to more closely agree with the results of other angler survey methods that randomly select individuals from a defined population of anglers (e.g., all individuals with fishing licenses). Examples of such surveys include Ebert et al. (1993), West et al. (1989), and Connelly et al. (1992). These surveys do not have the bias toward oversampling frequent anglers that occurs with creel surveys. As a result, the distribution of consumption rates from these types of surveys are expected to be comparable to the distribution for the total angler population and not the survey population of a creel survey. Figure 6 presents a comparison of the distribution of consumption rates from Ebert et al. (1993) with the estimated distributions for the survey and total angler populations of the Pierce survey. As the figure demonstrates, the consumption rate distribution for the total angler population agrees much more closely with the Ebert et al. (1993) distribution than does the distribution for the survey population. The Connelly et al. (1992) and West et al. (1989) surveys reported intake data that are similar to Ebert et al. (1993). The consistency between the Pierce total angler results and the results of the Ebert et al. (1993), Connelly et al. (1992), and West et al. (1989) is in spite of significant differences, in the types of water surveyed (salt water versus freshwater) and the region of the country (west coast versus upper midwest and the northeast), which would lead one to predict significant differences in consumption behavior. It appears, however, that performing the evaluation on the same total angling population basis eliminates much of the reported variation in the results of angler surveys.

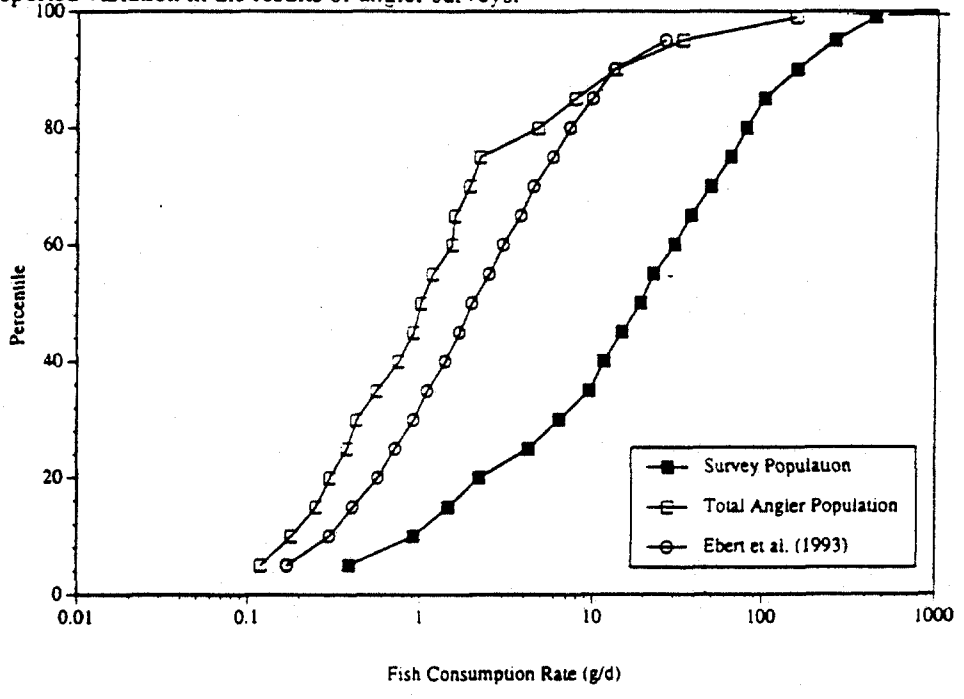

FIGURE 6. Comparison of the estimated cumulative distributions of fish consumption rates for the survey and total angler populations from the reanalysis of Pierce survey (1981) and the results of Ebert et al. (1993).

In summary, evaluation of creel surveys must take into consideration the inherent bias towards oversampling the frequent angler. Estimates of fish consumption rates derived from data collected from creel surveys should be adjusted before they are used to estimate fish consumption rates for total populations of anglers using a given fishery. The results of creel surveys must also be adjusted before they can be directly compared to the results of other types of angler surveys.

ACKNOWLEDGEMENTS

To Todd Abel for his diligence in obtaining the raw data and to Russell Keenan and Ellen Ebert for their thoughtful reviews. This work was supported by General Electric Corporation.

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INTRODUCTION

The rate of consumption of self-caught fish is a critical parameter for many environmental risk assessments. Because persistent lipophilic compounds that are released to surface waterbodies may bioaccumulate in fish, often the most important route of human exposure to these chemicals is through fish consumption (Humphrey, 1983; EPA, 1984; Rifkin et al., 1991; Sherman et al., 1992). Because many surface waterbodies, and in particular most freshwaters, are not commercially fished, consumption of fish is limited to recreational anglers. While such individuals may only represent a fraction of the total population living near an affected body of water, they may represent the majority of risks posed by surface water contamination. Therefore, it is critical to accurately characterize the rate of fish consumption for recreational anglers. Currently, EPA guidance recommends that a rate of 30 grams per day be used to represent the ingestion rate of fish caught from large bodies of water by a typical angler (EPA, 1989a,b). This estimate is based on the combined results of the Pierce et al. (1981) and Puffer et al. (1981,1982) creel surveys (hereafter referred to as the Pierce and Puffer surveys) of marine and estuarine anglers.

Creel surveys are typically used by fisheries managers to evaluate angler participation, effort, and catch/harvest rates from an individual waterbody. Such surveys generally count and interview anglers observed fishing a specified body of water at a specified time. During these surveys, data are collected specific to the individual angler's fishing experience, such as the length of the trip, and the number, size, and species of fish targeted, caught, and harvested by the angler on the day of the interview (EPA, 1991). More recently, creel surveys have been expanded to collect details on the anticipated disposition and/or consumption of the harvested fish (ChemRisk, 1991; Ebert et al., 1993).

A key characteristic of creel surveys is that the probability of an angler being interviewed during the survey is a function of his or her frequency of fishing (Puffer et al., 1981). Anglers who fish frequently have a higher probability of being interviewed than anglers who fish infrequently. As a result, creel surveys tend to oversample the frequent anglers. In addition, the distribution of consumption rates in the anglers interviewed during a creel survey are likely to overestimate the distribution of consumption rates in the entire population of anglers that fish the surveyed waterbody.

In this paper, we investigate the effect of this bias on the estimates of fish consumption that are derived from the Puffer and Pierce surveys. First, we used the inverse of each angler's self-reported annual frequency of fishing to reweight the estimated fish intake rate of each of the surveyed anglers (hereafter referred to as the survey population). This was done to calculate the distribution of consumption rates in the entire angler population that fishes the surveyed body of water (hereafter referred to as the total angler population). This approach is an extension of the methodology used by both Puffer et al. (1981) and Pierce et al. (1981) to estimate the size of the total populations of anglers using the waterbodies they surveyed.

THE EFFECT OF SAMPLING BIAS ON ESTIMATES OF ANGLER CONSUMPTION RATES IN CREEL SURVEYS

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EPA guidance recommends that 30 grams per day be used to represent the consumption rate of fish caught from large bodies of water by a typical angler (EPA, 1989a). This estimate is based on the combined results of the Pierce et al. (1981) and Puffer et al. (1981) surveys of marine and estuarine anglers. An examination of these surveys demonstrates that the method used in both studies — creel survey — oversamples frequent anglers and produces a distribution of consumption rates that overestimates intake rates of the total angler population using the surveyed waterbodies. Weighting the individual survey responses by the inverse of the angler self-reported fishing frequency corrects this bias and produces a more accurate characterization of the total population of anglers using the surveyed waterbodies. This approach is an extension of the methodology used by both Puffer et al. (1981) and Pierce et al. (1981) to estimate the size of the total angler populations. The results of the reanalysis of the Pierce et al. (1981) survey indicate that the median consumption rate for the total angler population is 1.0 g/d. The results of the Puffer et al. (1981) reanalysis indicate a median consumption rate for total angler population of 2.9 g/d. The recalculated distributions of consumption rates were found to be consistent with the results of other angler surveys that use survey methods that do not oversample frequent anglers. The angler intake rate of 30 g/d corresponds to roughly the 90th and 95th percentiles of the total angler populations in the Pierce et al. (1981) and Puffer et al. (1981) surveys, respectively. The results of this paper indicate that the current estimate of 30 g/d significantly overestimates consumption for typical marine and estuarine anglers.

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2. Abbreviations: EPA, United States Environmental Protection Agency; g, grams; g/d, grams per day; NYSDEC, New York State Department of Environmental Conservation; y, year; trips, one day fishing trips

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EVALUATING THE IMPACT OF COOKING PROCESSES ON THE LEVEL OF PCBS IN FISH

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Evaluating the Impact of Cooking Processes
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1.0 INTRODUCTION

In 1991, EPA issued a Phase I Report for the Reassessment RI/FS in which the Agency evaluated the potential human health risks for the Hudson River Superfund site. In this report, EPA (1991) determined that any risks to human health from PCBs in sediment occur through indirect exposure through the fish consumption pathway. Under Superfund Guidance (EPA, 1989), evaluation of such exposures are specifically required not to consider the impact of any fishing regulations. However, fishing restrictions have been imposed by the State of New York, and thus the estimates of PCB exposure developed for the Upper Hudson River are hypothetical and an overestimation of actual exposures.

In the Phase I document, EPA (1991) concluded that the effects of cooking should not be considered in the determination of exposures to PCBs from the consumption of contaminated fish. EPA based this decision on the variability of results in the published literature including some findings of PCB increases. In its comments on the Phase I report, GE advocated that in the risk assessment, the PCB levels consumed by recreational anglers should be reduced due to the loss of PCBs in fish tissue with cooking. GE made this recommendation based on an evaluation of the research that showed that cooking can lead to a significant amount of PCB loss. GE's conclusions were further supported by New York State (NYSDEC, 1991). In the New York State fishing regulations, the New York State Department of Environmental Conservation (NYSDEC) strongly recommended that anglers use a cooking method that does not reuse the fish oils, thereby acknowledging that cooking can lead to a reduction in fish PCB levels (Appendix A). In response to the Phase I comments, EPA (1992) agreed to review the available literature and determine if an adjustment for cooking loss is appropriate.

GE believes that the published data support the conclusion that PCB levels in fish are reduced via different cooking methods. Because the actual dose of PCBs received by an angler is determined by the amount of PCBs in each fish meal the angler consumes, any reduction in the amount of PCBs in fish that occurs during the cooking process will result in a reduction in the angler's dose. In this paper an analysis of the available data and an approach for evaluating the impact of cooking loss on PCB exposure from fish consumption are presented. The analysis is largely drawn from the attached published manuscript entitled *The Effect of Cooking Processes on PCB Levels in Edible Fish Tissue* (Sherer and Price, 1993).

2.0 REDUCTIONS IN PCB LEVELS AS A RESULT OF COOKING

Results reported in the literature

Numerous studies have been conducted that evaluate the effectiveness of different cooking methods to reduce PCB levels in fish (Table 1). Although most studies report some amount of PCB loss with cooking, reported reductions have varied over an extremely large range. In addition, results have not been reported in a consistent manner. Authors have reported reductions as the amount of PCBs lost per gram of fat, per gram of fish wet weight, per gram of fish dry weight, or in total mass of PCBs lost. This inconsistency in methods has hampered comparisons and compilations of results to date. Specifically, the variability in reporting has increased the uncertainty associated with the determination of a single cooking loss value or a percentage loss of PCBs from each of the different cooking methods.

To address this issue, Sherer and Price (1993) analyzed the available literature to determine if a pattern could be identified. The authors converted the results of each study to a percent loss of PCBs on a total mass basis. Conversion of all study results to the same units allowed the authors to compare and compile the results to determine an average PCB loss for each cooking method.

Results of Sherer and Price (1993) analysis

The results of Sherer and Price (1993) clearly demonstrate that cooking leads to a reduction of PCB levels in fish. The investigators determined the average percent reduction of PCBs for different cooking processes, including frying, broiling, baking, and microwave cooking. Although the reductions from individual studies ranged from 0 to 74 percent, the investigators accounted for this variability by analyzing the studies in a consistent manner and by grouping the reductions made by each cooking method (Sherer and Price, 1993).

Volatilization of PCBs and separation of the contaminated lipid from the fish tissue are two mechanisms that have been proposed to explain how cooking removes PCBs from fish (Zabik et al., 1979; Trotter et al., 1989; Shubat, 1992). In volatilization, the high heat of cooking causes

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APPENDIX A

Table 1. Identification of Research Conducted on PCB Losses from Cooking Fish

Study	Method	Fish Species
Ambruster et al. (1987)	poaching baking pan frying broiling	Striped Bass
Ambruster et al. (1989)	poaching baking pan frying broiling	Bluefish
Cichy et al. (1979)	broiling irradiation	Lake Trout
Puffer and Gossett (1983)	pan frying	White Croaker
Skea et al. (1981)	baking deep frying broiling	Brown Trout Smallmouth Bass
Smith et al. (1973)	poaching baking baking in nylon bag	Chenook Salmon Coho Salmon
Trotter et al. (1989)	baking	Bluefish
Zabik et al. (1979)	broiling roasting microwave cooking	Lake Trout
Zabik et al. (1982)	poaching deep-fat frying charbroiling roasting microwave cooking	Carp
Zabik et al. (1993)	baking deep frying charbroiling	Chinook Salmon Walleye Lake Trout
Daubenmire et al. (1993)	baking pan frying charbroiling	White Bass

PCBs to be released from the fish into the air. Loss of lipids is also a function of the temperature and cooking duration, with higher temperatures and longer cooking times causing a greater loss of fat from the edible tissue. As a result, cooking methods such as frying, baking, or broiling are more effective at removing PCBs. These cooking methods are also effective because they do not reuse the removed fat. In comparison, cooking methods such as making fish soup or fish casseroles where the fat is reused, are not effective means of reducing PCB levels.

In addition, fish with higher lipid contents tend to lose a greater amount of PCBs compared to fish with lower lipid contents. Since PCBs accumulate in lipid tissues, those fish with greater amounts of fat will lose greater amounts of PCBs during effective cooking processes. Frying may be particularly effective at removing PCBs because this method may actually extract lipids into the volume of cooking oil used in this type of preparation.

As Sherer and Price (1993) reported, not all studies showed losses from cooking. For example, when expressed on a wet weight basis, the PCB concentrations following cooking often appear to be greater than those present before cooking (Smith et al., 1973; Skea et al., 1981; Trotter et al., 1989). These increases in PCB levels have been attributed to the substantial loss of moisture in comparison to the loss of contaminant. However, when these same data are expressed on a dry mass basis, they consistently show a reduction in PCB concentration. Other increases in PCB levels have been attributed to tthe methods employed during the actual measurement of pre- and post-cooking tissues. For example, the extraction of phospholipid-associated PCBs is more efficient from cooked fish as compared to extractions from raw tissue (Paul and Palmer, 1972). This effect is most often seen in fish with lower fat concentrations, since the small amount of PCBs lost during cooking are offset by the greater amounts of PCBs extracted from cooked tissue during the laboratory analysis (Sherer and Price, 1993).

3.0 APPLYING THE RESULTS OF COOKING LOSS TO THE HUDSON RIVER EXPOSURE ASSESSMENT

The results of Sherer and Price (1993) indicate that cooking leads to substantial reductions in PCB levels in fish. These findings are significant for the Hudson River reassessment because they demonstrate how cooking methods that are commonly used for New York freshwater fish can

remove PCBs and lower anglers' exposures. It is likely that the uncertainties that prevented EPA from including cooking losses in the Phase 1 document can be attributed to the reporting method or extraction technique used. Although some variability in individual cooking losses will still exist between fish meals, this variability in the short-term will be insignificant in any long-term analysis of exposure.

Research has shown that freshwater anglers in the northeastern United States typically use cooking methods that reduce PCB levels in self-caught fish. Studies on the cooking methods used by recreational anglers have been conducted by ChemRisk (1992) and Connelly et al. (1992). Connelly et al. (1992) surveyed anglers in the State of New York on a variety of topics including fish preparation and cooking practices. However, the survey was not designed to determine the specific frequency for each cooking practice (Table 2). ChemRisk surveyed anglers in the State of Maine (ChemRisk, 1992; Ebert et al., 1993). This survey did ask what cooking methods were typically used by freshwater anglers. Table 2 presents the distribution of cooking methods favored by anglers in the ChemRisk study.

While the ChemRisk study was performed in Maine, the findings are believed to be appropriate for the Hudson River anglers for the following reasons. First, the ChemRisk and Connelly et al. studies generally agreed that anglers tend to favor cooking methods that reduce PCB concentrations such as frying or broiling (Table 2). Second, the population of anglers in the ChemRisk (1992) survey and the survey of New York anglers conducted by Connelly et al. (1992) are similar in age, income, and other demographic criteria. Fish consumption rates are also similar for the two surveyed populations, and the species of fish found in the Hudson River are similar to the fish commonly harvested in Maine (e.g., bass, trout, and bullhead).

The results of Connelly et al. (1992) further indicate that most anglers trim their fish in addition to cooking. Although the loss of PCBs associated with trimming has not been specifically evaluated as part of this discussion, studies indicate that trimming can lead to substantial reductions in PCBs (Skea et al., 1981; Shubat, 1992; Armbruster et al., 1989). In addition, NYSDEC recommends that trimming fish is an appropriate method to reduce PCB levels. These findings indicate that the losses associated with cooking, reported by Sherer and Price (1993), are likely to underestimate the actual amount of PCBs lost during the preparation of fish meals.

Table 2. Distribution of Cooking Preferences

	ChemRisk (1992)	Connelly et al. (1992)
Baking	0.179	0.24[a]/0.37[b]
Boiling	0.002	
Broiling	0.164	
Frying	0.621	0.514/0.31
Poaching	0.009	0.24/0.37
Microwave	0.009	
Raw	0.006	
Soup	0.02	0.017/0.13

a. Always/usually use cooking method
b. Sometimes use cooking method

4.0 CONCLUSION

The recent summary of PCB literature has demonstrated that cooking processes are very effective at removing PCBs from fish (Sherer and Price, 1993). The actual amounts of PCBs consumed by recreational anglers are likely to be much less than the levels of PCBs collected from uncooked fillets. Studies by Connelly et al. (1992) and Ebert et. al. (1993) indicate that most anglers do use some type of cooking method that has a high likelihood of reducing PCBs before consumption. NYSDEC supports these findings and has recommended that all recreational anglers cook and trim their fish before consumption. At a minimum, General Electric recommends the incorporation of a cooking loss factor into the analysis. A more realistic evaluation would also include an adjustment factor for trimming. Incorporation of both these factors will assure that the adjustment selected for cooking loss will be an accurate estimate of the true reduction in PCBs that may be experienced by the recreational angler.

The most appropriate method to incorporate the reduction in PCBs with cooking is through the use of a probabilistic exposure assessment using synthetic life history or Microexposure Monte Carlo analysis. This type of analysis can account for the variations in cooking methods that an individual angler may use over the course of a lifetime. Specifically, a Microexposure Monte Carlo analysis selects a different cooking method and PCB reduction value for each meal eaten by a recreational angler and appropriately adjusts the original PCB concentration of the species selected.

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Appendix A
REDUCING CHEMICAL EXPOSURES[1]

Everyone can benefit from eating fish they catch and can minimize their chemical contaminant intake by following these general recommendations:

- Choose fish from water bodies which are not listed in the DOH advisory.
- If you choose to eat fish from water bodies with a DOH advisory, choose fish species not listed in the advisory.
- Choose smaller fish within a species consistent with DEC regulations, since they may have lower contaminant levels. Older (larger) fish within a species may be more contaminated because they have had more time to accumulate contaminants in their bodies.
- Levels of PCBs, mirex, DDT and other contaminants of concern (except mercury) can be reduced by removing the skin and fatty portions along the back, sides and belly of fish. Most contaminants are associated with the fats in fish.
- Cooking methods such as broiling, poaching, boiling and baking, which allow fats to drain out, are preferable. Pan frying is not recommended.[2] The cooking liquids of fish from contaminated waters should be avoided since these liquids may retain contaminants (NYSDEC).

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1. Source: New York State 1993-1994 Fishing Regulations Guide, p. 72.

2. While the oils removed during frying still remain in the pan, many researchers have shown that pan frying can effectively reduce PCBs in fish (Skea et al., 1981; Puffer and Gossett, 1983).

ATTACHMENT:

The Effects of Cooking Processes on PCB Levels in Edible Fish Tissue

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The Effect of Cooking Processes on PCB Levels in Edible Fish Tissue

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A significant factor in estimating human intake of polychlorinated biphenyls (PCBs) from fish consumption is the loss of PCBs during cooking. The total amount of PCBs actually consumed in the cooked fish may be significantly lower than the PCB level present before cooking because lipids and lipophilic compounds like PCBs tend to be removed from the fish during cooking. Several studies investigating the extent of loss of PCB compounds during the cooking process have been published in the peer-reviewed literature. However, because of what is perceived as inconsistent and inadequate data on the removal of these compounds, federal and state regulators typically do not assume that cooking reduces contaminant levels (EPA, 1990; 1991). In this paper, an attempt was made to reduce the uncertainty in the findings of these studies on PCB losses during the cooking process. This was accomplished by (1) eliminating studies that lacked statistical power to determine the degree of reduction, (2) reporting all of the results in a common format, and (3) characterizing studies by cooking method. In addition, the studies that reported increases in PCB concentration after cooking were carefully reviewed to provide a possible explanation of this occurrence. Based upon this analysis, it was concluded that cooking processes such as baking, broiling, microwave cooking, poaching, and roasting remove approximately 20 to 30% of the PCBs. Frying appears to remove more than 50%. PCB cooking losses also appears to be a function of the initial lipid concentration in the fish. Based upon this analysis, it is clear that the information from these studies do provide a reasonable basis for federal and state regulators to permit a quantitative adjust of PCB intakes.

INTRODUCTION

A significant issue in estimating human intake of PCBs from fish consumption is the loss of PCBs that occurs during cooking. Because PCBs are concentrated in body lipids of fish (Reinert *et al.*, 1972; Skea *et al.*, 1981; Armbruster *et al.*, 1987), and lipids tend to be removed from fish during cooking, it then seems reasonable to assume that this loss of lipids can result in a reduction of PCBs in the fish tissue. In addition, PCBs may also be lost by direct volatilization during cooking. As a result of these processes, the total amount of PCBs actually consumed in the cooked fish may be significantly lower than the amount occurring in the raw fish.

Several studies investigating the extent of loss of PCBs during the cooking process have been published in the peer-reviewed literature. Although most of these studies have documented significant reductions in total PCB levels after the cooking process.

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the degree of reduction reported in each of the studies has varied greatly. In addition, certain studies have reported increases in the concentrations of PCB after cooking. Because of what is perceived as inconsistent and inadequate data regarding the effects of cooking on PCB levels in fish, federal and state regulators have been hesitant to assume that cooking reduces PCB levels (EPA, 1990, 1991).

In this paper the authors examine the available literature with the goal of developing specific recommendations for incorporating cooking reductions into quantitative exposure assessments. Based upon this analyses, there does appear to be a reasonable basis for quantitatively adjusting estimates of PCB intake from the consumption of fish based on cooking practices. This paper reviews the currently available studies that address changes in PCB levels as a result of cooking. Estimates of cooking-method-specific alterations in PCB levels are developed based on this literature review.

REVIEW OF LITERATURE

The authors began their study by performing a literature search for peer-reviewed articles that dealt with PCB cooking losses on fish tissue. This search identified nine studies. Two other studies were identified but are not discussed in this paper because they investigated PCB cooking losses in crabs (Zabik *et al.*, 1991) and turkey (Zabik *et al.*, 1990).

The nine articles identified from the literature search contain information on a variety of fish species and cooking methods. Species investigated in the various studies include chinook and coho salmon (Smith *et al.*, 1973), lake trout (Cichy *et al.*, 1979; Zabik *et al.*, 1979), brown trout (Skea *et al.*, 1981), smallmouth bass (Skea *et al.*, 1981), carp (Zabik *et al.*, 1982), white croaker (Puffer and Gossett, 1983), striped bass (Armbruster *et al.*, 1987), and bluefish (Armbruster *et al.*, 1989; Trotter *et al.*, 1989). Cooking methods include boiling, poaching, microwave cooking, broiling, baking, roasting, pan frying, and deep frying.

The analytical methods used in all of the studies are variations of the method developed by Yadrick *et al.* (1972). This process consists of a Soxhlet hexane–acetone extraction of the freeze-dried tissue, acetonitrile partitioning, and florisil–celite column cleanup. Characterization and quantification of PCBs were conducted using gas chromatographic analyses.

A major difficulty, however, in comparing the results of the studies is that PCB losses are not reported in a consistent manner. Reductions in PCBs have been expressed in terms of the amount of PCBs lost per gram of fat, per gram of fish (wet weight), per gram of fish (dry weight), or in total mass of PCB lost. These different reporting methods confound the comparison of the results of the studies and obscure the significance of the literature. It is, therefore, critical to present the results in a consistent manner. In this study, the effect of cooking on the amount of PCBs in the fish is evaluated on a mass basis as follows:

$$\text{Percentage of total PCB mass lost during cooking} = \frac{\text{Total PCB mass in uncooked fillet} - \text{Total PCB mass in cooked fillet}}{\text{Total PCB mass in uncooked fillet}} \times 100.$$

The advantage of presenting data on a total mass basis is that the loss of PCB can be used to directly estimate the impact of cooking losses on the intake of PCBs.

Discussion of Individual Studies

While most of the nine studies reported evidence of cooking losses, only five of the studies were deemed usable in quantifying the PCB cooking losses. Some of the studies were not usable because of experimental methodologies that are inconsistent with the objectives of this study (refer to subsequent discussions regarding Armbruster *et al.* (1989) and Cichy *et al.* (1979)). Other studies were not included because the results lacked statistical significance. These studies typically reported reduction in PCB levels; however, the results were not statistically significant due to small sample sizes and high variability in initial PCB levels in the fish tissue samples. In addition, some studies also lacked sufficient data in order to determine total mass loss of PCBs. Table 1 lists the nine studies under consideration and whether they were included in the final quantification estimates of PCB cooking loss. The following paragraphs briefly discuss the studies and their usefulness in quantifying PCB cooking losses.

Armbruster *et al.* (1987) studied the effects of six different cooking methods on PCB concentrations in striped bass. The authors reported that, although declines occurred with most methods, the declines were not statistically significant due to the high variability in PCB levels in the fish tested and the small sample sizes.

Armbruster *et al.* (1989) reported the combined effects of trimming and cooking on the concentrations of PCBs in bluefish from Long Island Sound. Forty raw bluefish fillets were trimmed and then 10 randomly selected fillets were baked, broiled, fried, or poached. The study found that a combination of trimming and cooking resulted in PCB reductions of 60% by poaching, 68% by baking, 68% by pan frying, and 71% by broiling. Data were reported on a dry weight basis. No data were presented for fat content of the raw fillets. While the study results suggest that cooking processes did reduce PCB levels in fish, it is not possible to clearly determine the fraction of the decline that was due to cooking versus that resulting from trimming.

Cichy *et al.* (1979) studied the combined effects of irradiation and broiling on the levels of PCBs in lake trout fillets. Significant reductions in PCB concentrations were observed during the broiling of irradiated fillets. Because of the study design, which focused on the effects of irradiation and did not investigate the effects of cooking on

TABLE 1

Summary Evaluation of Studies

Study	General findings	Was method appropriate?	Were results statistically significant?	Was a quantitative estimate of mass loss possible?
Armbruster *et al.*, 1987	Small reduction	Yes	No	No
Armbruster *et al.*, 1989	Large reduction	No	Yes	No
Cichy *et al.*, 1979	Small reduction	No	No	No
Puffer and Gossett, 1983	Large reduction	Yes	Yes	Yes
Skea *et al.*, 1981	Large reduction	Yes	Yes	Yes
Smith *et al.*, 1973; Smith, 1972	Small reduction	Yes	Yes	Yes
Trotter *et al.*, 1989	Large reduction	Yes	Yes	Yes
Zabik *et al.*, 1979	Large reduction	Yes	Yes	Yes
Zabik *et al.*, 1982	Slight increase	No	No	No

fish that had not been irradiated, this study was not used to quantitatively estimate PCB losses due to cooking processes.

Puffer and Gossett (1983) studied the effects of pan frying on PCB and DDT concentrations in fillets of white croaker from two locations in California. Five composites from each location were tested. Mean fat contents of the raw fillets were 1.2% for Santa Monica Bay samples and 0.9% for Orange County samples. The results of the analyses were presented from a wet weight and a total PCB and DDT mass basis. PCB losses were 65% for Santa Monica Bay samples and 28% for Orange County samples on a mass basis. The authors attributed the greater losses in Santa Monica Bay samples to the fact that PCB concentrations from that location were 11 times higher than concentrations in Orange County samples.

Skea *et al.* (1981) reported the combined effects of trimming and cooking in reducing the levels of PCBs (Aroclor 1254), Mirex, and DDE compounds in brown trout and smallmouth bass. For smallmouth bass, baking of 20 untrimmed, unskinned fillets (mean fat content of 2.8%) reduced total PCB mass levels by 16% and deep frying of 20 trimmed fillets (mean fat content of 1.3%) in corn oil reduced total PCB mass levels by 74%. For brown trout, smoking of 30 untrimmed fillets (mean fat content of 16.5%) reduced total PCB levels by 27%, and broiling of 30 skinned, fat-trimmed fillets (mean fat content of 8.8%) showed no reduction of PCBs. However, the apparent lack of PCB reduction by broiling brown trout fillets may have been an analytical error since significant reductions of other lipophilic compounds, Mirex and DDE, 26 and 20% respectively, were observed after broiling.

Smith *et al.* (1973) analyzed PCB concentrations in 10 raw samples and 20 cooked samples of chinook salmon. PCB levels were expressed as micrograms of PCB per gram of fat in the fish samples. Also, two raw samples and four cooked samples of coho salmon were analyzed. The average percentage of fat content was 2.65% in the raw chinook steaks and 3.59% in raw coho steaks. Samples were poached, baked, or baked in a nylon bag. The authors reported both small reductions and increases in average concentrations of Aroclor 1248 and Aroclor 1254 during cooking. Statistical analysis performed by the authors indicated that the reductions were not statistically significant. This lack of a clear trend could have been due to small numbers of samples, large variability in PCB content between individual samples, or low body-fat content of the fish.

Additional information for this study is contained in the thesis of Smith (1972), on which Smith *et al.* (1973) is based. The thesis and Smith *et al.* (1973) contained sufficient data to estimate an overall percentage of PCB loss on a mass basis during baking for the Chinook salmon (see discussion in the following section). The number of coho salmon samples was sparse and judged to be insufficient to warrant inclusion into this paper.

Trotter *et al.* (1989) studied the effects of baking on PCBs and lipophilic pesticides in 20 bluefish fillets. The authors initially reported increases in PCB levels on a wet weight basis. Estimates of PCB reduction on a total mass PCB basis were then calculated based upon information provided in the study relative to PCB concentrations and fillet weights before and after cooking. Expressed on a mass basis, the study found a reduction of 27% due to the baking process. Average fat content of the raw fillets in this study was 11.8%.

Zabik *et al.* (1979) assessed the changes in Aroclor 1254 levels in lake trout fillets which resulted from broiling, roasting, and microwave cooking the fish. Duplicate samples from head, middle, and tail portions of the fillets were analyzed for each

cooking method. The total masses of PCBs were reduced by an average of 53% by broiling, 34% by roasting (baking), and 26% by microwave cooking. Mean fat content of the raw fillets was approximately 25% for samples used in the roasting experiment, 26% for fillets used in microwave cooking, and 29% for fillets that were broiled.

Zabik *et al.* (1982) reported the effects of several cooking methods on PCB and DDT levels in carp fillets from Saginaw Bay, Michigan. Mean fat content of the raw fillets was approximately 8%. These authors reported that PCB concentrations were reduced 25% by deep-fat frying, 27% by poaching, 25% by charbroiling, 33% by microwave cooking, and 20% by roasting, when data were expressed on a fat basis. However, when they expressed their results on a total mass basis, data for all cooking methods, except microwave cooking, indicated an increase in PCBs. Zabik *et al.* (1982) attributed these increases to more efficient extraction of phospholipid-associated PCBs during laboratory analyses of cooked tissue as compared with raw tissue.

Development of Quantitative Estimates of PCB Reduction

Of the nine studies identified, five studies contained sufficient data to allow the quantification of PCB loss during cooking on a mass basis. Other than Zabik *et al.* (1982), all of the 10 studies present evidence of loss of PCBs or similar lipophilic compounds during cooking. The subsequent paragraphs present a brief review of how the data in the five studies were used to quantitatively estimate cooking losses.

Zabik *et al.* (1979) reported changes in PCB content of fish fillets on a whole tissue (wet weight) basis (Zabik *et al.*, 1979, p. 139), a fat basis (Zabik *et al.*, 1979, p. 140), and a total mass of PCB basis (Zabik *et al.*, 1979, p. 141). Similarly, Skea *et al.* (1981) reported data for changes in PCB content during baking (Skea *et al.*, 1981, p. 17), broiling (Skea *et al.*, 1981, p. 16), or frying (Skea *et al.*, 1981, p. 18) on a whole tissue (wet weight) basis as well as a total mass of PCB basis. The total mass basis values from each of these studies were used without modification in this paper.

Puffer and Gossett (1983) initially reported changes in PCB content of white croaker samples on a wet weight basis. However, by employing a conversion factor ("weight loss factor") to account for weight loss from cooking, the authors subsequently determined PCB losses on a mass basis (Puffer and Gossett, 1983, p. 69). These estimates were used in this paper.

Trotter *et al.* (1989) initially reported changes in PCB content of bluefish fillets on a whole tissue (wet weight) basis (Trotter *et al.*, 1989, p. 502). Using data on PCB concentrations and weights of individual raw fillets versus cooked fillets, the authors calculated average changes in PCB content on a total mass basis (Trotter *et al.*, 1989, p. 502). The mass of PCBs in the individual raw fillets was calculated by multiplying the reported concentration of PCB in the fillet by its respective raw weight. Comparable calculations were conducted for these fillets in their cooked state. The percentage of change in the mass of PCBs for individual fillets in their raw state versus cooked state was determined, and an average of these percentages was calculated to estimate overall PCB loss during baking of the fillets.

Smith *et al.* (1973) reported the results on a mass per gram of fat basis (micrograms of PCB per gram of fat). As discussed in the previous section, Smith *et al.* (1973) then reported cooking loss by comparing PCB levels, expressed on a mean basis, in raw and cooked fillets. That is,

Fraction of PCB remaining after cooking

$$= \frac{\text{Mean concentration } \mu\text{g of PCB/gram of fat in cooked fillet}}{\text{Mean concentration } \mu\text{g of PCB/gram of fat in raw fillet}}.$$

Because of the high variability of PCBs in individual samples and the relatively small differences between the cooked and raw fillets, cooking loss estimates by this method were not statistically significant.

Based on data provided in Smith (1972), PCB losses during cooking can be estimated by an alternative method. In Smith (1972) detailed information was provided on the levels of PCBs in the baked fillets and in the drippings collected in the pan below. Thus, it is possible to make a conservative estimate of the loss of PCBs by comparing the mass of PCBs in the drippings to the mass of the PCBs in the cooked fillets. The percentage of PCBs removed during cooking is estimated as follows:

Percentage of total PCB mass lost during cooking

$$= \frac{\text{Mass of PCBs in drippings}}{\text{Mass of PCB in cooked fillet} + \text{Mass of PCB in drippings}} \times 100.$$

The mass of the PCBs in the cooked fillet and the dripping from the fillet can be estimated as

$$M_{PCB} = C_{PCB} \times F \times M_f,$$

where M_{PCB} is the mass of PCBs in a fillet or dripping, C_{PCB} is the concentration of total PCBs in micrograms per gram of fat in a fillet or dripping, F is the percentage of fat in the fillet or dripping, and M_f is the mass of the fillet or dripping. Data on the concentration of PCBs (fat basis) and percentage of fat for the individual fillets and their drippings are given in Smith (1972). Data on the average mass of the fillets and drippings are given in Smith *et al.* (1973).

Based on this approach, it was estimated that the average cooking loss was 10% for baking. The calculated 10% loss during baking is a conservative estimate of total PCB loss because the estimate does not reflect the PCBs lost by volatilization during cooking. Had this component of cooking loss been included, the estimate of total loss during cooking would have been larger. This analytical approach was also applied to the results (Smith *et al.*, 1973) of poaching of chinook steaks. However, no meaningful estimates of the percentage of loss could be made due to the extremely low content of fat in the drippings resulting from the poaching process.

DISCUSSION OF REPORTED INCREASES OF PCBs AFTER COOKING

While most studies have reported declines in PCB levels after cooking (Table 2), some studies actually reported increases (Smith *et al.*, 1973; Skea *et al.*, 1981; Zabik *et al.*, 1982; Trotter *et al.*, 1989). The results of the studies that reported increases were generally expressed as a concentration on either a wet weight or fat basis (Smith *et al.*, 1973; Skea *et al.*, 1981; Trotter *et al.*, 1989). In these cases, the PCBs appeared to become concentrated due to a greater percentage of moisture loss than contaminant loss during the cooking process (Skea *et al.*, 1981). Trotter *et al.* (1989) specifically commented on this issue stating, "the relatively large loss of moisture during cooking compensated for the PCB and oil loss and resulted in similar ppm PCB and percent

TABLE 2

Changes in PCB Levels in Fish Samples Resulting from Various Cooking Methods

Method	Study	Fish species	Percentage of change on a PCB mass basis
Bake or roast	Smith *et al.*, 1973; Smith, 1972	Chinook salmon	−10
	Zabik *et al.*, 1979	Lake trout	−34
	Skea *et al.*, 1981	Smallmouth bass	−16
	Trotter *et al.*, 1989	Bluefish	−27
			Average −22
Broil	Zabik *et al.*, 1979	Lake trout	−53
	Skea *et al.*, 1981	Brown trout	0
			Average −27
Fry	Skea *et al.*, 1981	Smallmouth bass	−74
	Puffer and Gossett, 1983	White croaker (Santa Monica Bay)	−65
	Puffer and Gossett, 1983	White croaker (Orange County)	−28
			Average −56
Microwave or poach	Zabik *et al.*, 1979	Lake trout	−26
			Average −26

fat levels in the uncooked and cooked fillets." When the data from these studies are expressed on a mass basis instead of a concentration basis, they consistently show a reduction in PCBs after the cooking process. The data expressed on a mass basis are presented in Table 2.

The one exception to the decrease in PCB mass after cooking was reported by Zabik *et al.* (1982), who reported that PCB mass levels were increased by the cooking process. Zabik *et al.* (1982) suggested that the PCB mass increases could be due to more efficient extraction of phospholipid-associated PCBs during laboratory analyses of cooked fish tissue compared with raw tissue. The analytical method used to extract PCBs from fish tissue (Yadrick *et al.*, 1972) is not necessarily completely effective in extracting

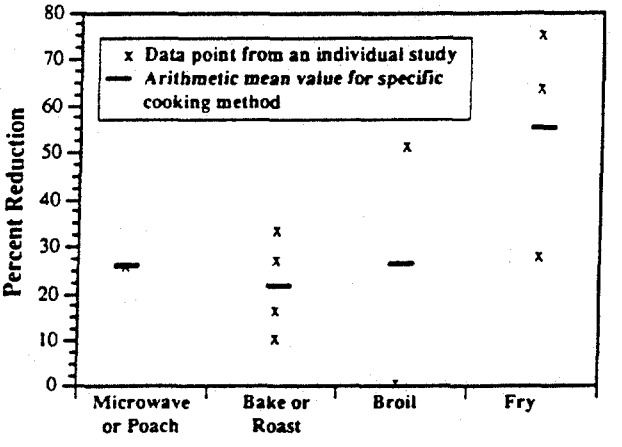

FIG. 1. Percentage of reduction of PCBs in fish fillets relative to cooking method.

TABLE 3

Average Lipid Content of Raw Fish Samples Used in Cooking Loss Studies

Method	Study	Fish species	Percentage of lipid content of raw fillets
Bake or roast	Smith *et al.*, 1973	Chinook salmon	2.7
	Zabik *et al.*, 1979	Lake trout	25.0
	Skea *et al.*, 1981	Smallmouth bass	2.8
	Trotter *et al.*, 1989	Bluefish	11.8
Broil	Zabik *et al.*, 1979	Lake trout	29.1
	Skea *et al.*, 1981	Brown trout	8.8
Fry	Skea *et al.*, 1981	Smallmouth bass	1.3
	Puffer and Gossett, 1983	White croaker (Santa Monica Bay)	1.2
	Puffer and Gossett, 1983	White croaker (Orange County)	0.9
Microwave or poach	Zabik *et al.*, 1979	Lake trout	26.4

intermuscular phospholipids in raw fish tissue. Thermal decomposition of the protein–lipid microstructures may facilitate a more complete extraction of these lipids and associated PCBs. Support for this conclusion is presented by Paul and Palmer (1972), as cited in Zabik *et al.* (1982), who reported, "cooking often causes an increase in the amount of ether extractable material in the lean portion of meat over that found in raw meat, even when the lipid extract is expressed on a dry basis."

The effect described by Paul and Palmer (1972) may occur in all cooking processes; however, the effect may be most conspicuous when total PCB losses are small. As discussed below, several authors have suggested that the degree of PCB removed will be higher in fish with high fat content. In fish with high fat content and high PCB removal rates the small increase in apparent PCB concentration caused by increased PCB extractability is overwhelmed by the larger reduction in PCB from volatilization and fat loss. In fish with low fat levels (carp used in Zabik *et al.* (1982) contained 8% fat) the effect is not overwhelmed by a large loss from fat rendering and is thus observed as an apparent increase.

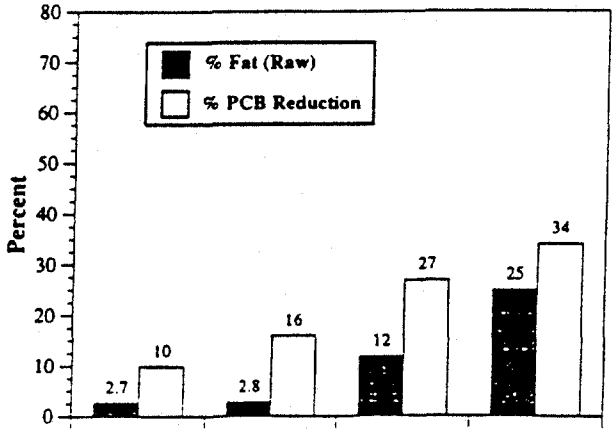

FIG. 2. Extent of PCB reduction relative to percentage of fat content by baking or roasting.

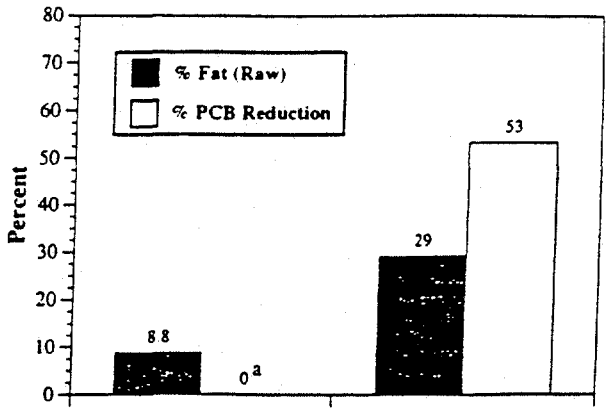

FIG. 3. Extent of PCB reduction relative to percentage of fat content by broiling. [a] Value of 0 is believed to be anomalous because significant reductions were reported for specific lipophilic pesticide residues.

If the Zabik hypothesis is correct, then all reported cooking loss measurements will tend to underestimate the true degree of removal. This will occur since the PCB levels in the raw fish will appear to be smaller due to the decreased extractability. This phenomenon may explain the apparent contradiction in Smith *et al.* (1973) where PCB levels in cooked fish appeared to be unchanged, while approximately 10% of the PCBs were measured in the drippings of the cooked fish.

The authors of this paper believe that it is highly unlikely that PCBs are actually formed during the cooking process. PCBs are commercially produced by the direct chlorination of biphenyl in nonpolar solvents (ATSDR, 1991). Such chemical processes are not likely to occur in fish tissue due to the absence of free chlorine, the presence of polar compounds (water, proteins, carbohydrates, etc.), and the unlikely occurrence of biphenyl or other suitable precursors. Thus, the generation of PCBs during the cooking process is highly implausible. Because of the absence of a plausible mechanism for the formation of PCBs, and the consistent measurements of reductions in PCB on a total mass basis in the majority of published studies, it can be concluded that PCBs are reduced to varying degrees by different cooking methods.

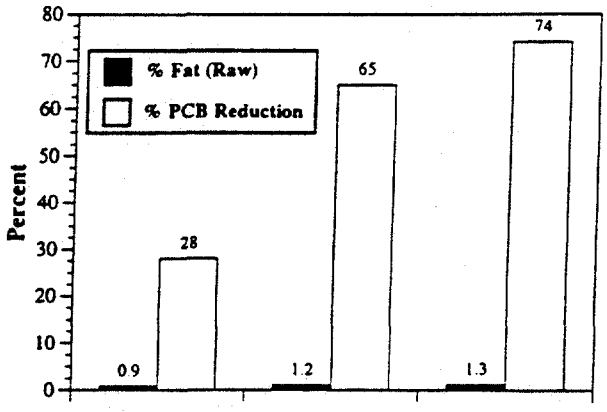

FIG. 4. Extent of PCB reduction relative to percentage of fat content by frying.

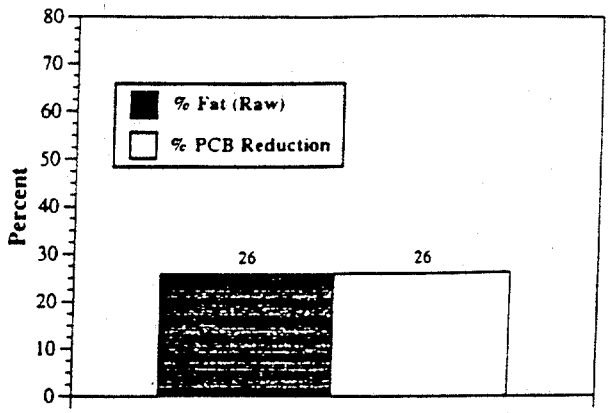

FIG. 5. Extent of PCB reduction relative to percentage of fat content by microwaving or poaching.

REDUCTION IN PCB MASS BY VARIOUS COOKING METHODS

The degree at which PCB mass is reduced during cooking varies with the cooking method. Certain cooking methods, such as microwave cooking or steaming, may be relatively ineffective in removing lipids from the fish due to the low cooking temperatures and/or short cooking times. Certain methodologies, such as stewing or using fish in casseroles, result in minimal reduction in PCB levels since volatilization is believed to be minimal, and the lipids lost during cooking are still consumed. Methods such as broiling or baking are more effective in reducing the amount of PCBs consumed because lipids containing these compounds are separated from the fish and not consumed, and because PCBs are also believed to be volatilized somewhat during these types of cooking processes. Finally, processes such as deep frying may also reduce the PCB concentration in the fish by an actual lipid extraction. In this process, it is hypothesized that PCBs may partition into the large volume of cooking oils and fats.

While the studies discussed in this paper clearly indicate that cooking reduces the PCB mass levels in edible fish tissues, an examination of the results (Table 2) indicates that there is a wide variation in the degree of reduction between the various cooking methods and also within the same method. The hypothesis that PCB loss is predominantly due to fat loss and volatilization suggests that PCB loss should increase for cooking methods that are more severe (i.e., higher temperatures and longer cooking times). To test this hypothesis, the cooking loss data for the various cooking methods

TABLE 4

Average Reduction of PCBs in Fish by Various Cooking Methods

Method	Percentage of reduction[a]
Bake or roast	22
Broil	27
Fry	56
Microwave or poach	26

[a] Mean percentage of reductions as reported in Table 2.

were sorted according to normal cooking temperature. The ranking of method from least to the greatest temperature was microwave cooking, baking (or roasting), broiling, and frying. The percentage of reduction results are presented in Fig. 1. Reduction was greatest for frying; broiling and baking were lower; and data on poaching and microwave cooking were too limited to reach a definitive conclusion as to whether it would yield the lowest reduction. These results qualitatively support the hypothesis that the increased temperature and severity of the cooking method is correlated with the degree of PCB reduction.

It has also been suggested by several authors (Cordle *et al.*, 1982; Zabik *et al.*, 1982) that the degree of cooking losses for lipophilic chemicals should increase with the percentage of total fat content of the fish. Table 3 indicates the percentage of fat content of raw fillets used in specific studies, and Figs. 2 through 5 present the degree of PCB loss as a function of the percentage of fat for the different cooking methods. Based on this limited information, it appears that there may indeed be a correlation of reduction for baking and possibly frying but for other cooking methods the information is far too lacking to determine if a correlation between cooking loss and fat content occurs.

SUMMARY AND CONCLUSIONS

An examination of the literature indicates that cooking of fish fillets reduces the amount of PCBs in the fillet. The degree of reduction of PCBs can vary depending upon the specific cooking method employed and characteristics of the fillet being cooked. Because authors have presented their research data in various manners, a casual review of the literature suggests considerable variability in the results. When the degree of loss is expressed on a mass basis, however, the variability in the reported data is greatly reduced. Evaluation of the reported reductions resulting from each cooking method appear to demonstrate that PCBs are preferentially removed by cooking processes that involve higher cooking temperatures and longer cooking times, and which allow the separation of the rendered fat from the cooked fish.

Based on the available data, typical reduction rates as illustrated in Table 4 can be estimated for different cooking methods. These estimates are probably inaccurate for estimating PCB lost in individual meals, as actual losses in meals will be affected by fillet size, cooking time and temperature, and other factors. However, long-term exposure to PCBs is a function of exposures from many meals. Since the estimate of the average PCB loss by cooking method reflects the results of multiple fish tests in several studies, it provides reasonable guidance for general reductions that are likely to occur over long periods of time. It is, therefore, recommended that the average cooking-method-specific levels derived in this paper be used to evaluate actual exposure to PCBs found in fish.

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HUDSON RIVER SUPERFUND PROJECT

APPROACH FOR PERFORMING HUMAN HEALTH RISK ASSESSMENT: ESTIMATING POTENTIAL PCB EXPOSURE FROM FISH CONSUMPTION

May, 1995

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May, 1995

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HUDSON RIVER SUPERFUND PROJECT: APPROACH FOR PERFORMING HUMAN HEALTH RISK ASSESSMENT: ESTIMATING POTENTIAL PCB EXPOSURE FROM FISH CONSUMPTION

1.0 INTRODUCTION

In 1990 the U.S. Environmental Protection Agency (EPA) began a reassessment remedial investigation and feasibility study (RRI/FS) for the Hudson River PCB-contaminated sediment Superfund site. Subsequently, EPA issued a summary report (Phase 1 Report), project plans that described data collection and analysis activities, and a data collection program was initiated. As part of the data analysis activities, EPA began the development of a computer model for simulating PCB fate and transport in the Upper Hudson River.

In the EPA Phase 1 Report, a preliminary human health risk assessment was prepared. This preliminary risk assessment determined that consumption of PCB-contaminated fish presented the primary source of risk from potential exposure to PCBs at the site and that the final site risk assessment would focus on the fish consumption exposure pathway (EPA Phase 1 Report, B.6-46). EPA is now completing the remedial investigation portion of the RRI/FS process and is entering the phase in which the final baseline human health risk analysis is conducted, remedial action objectives are defined, and remedial alternatives are evaluated.

Since the RRI/FS was initiated, a number of significant changes have occurred in site conditions and national policy related to exposure and risk assessment. The original project plans that describe the procedures to be used for the risk assessment and the feasibility study should be updated to reflect these changes and provide important details on how risk assessment and feasibility studies will be performed. As a result, General Electric (GE) believes it is necessary to highlight a number of important issues related to these aspects of the project and specifically describe how to integrate the risk assessment, the PCB-fate and transport model being developed by EPA, and the feasibility study. Since the risk assessment is central to integrating these issues, the focus of this paper is on issues related to conducting the risk assessment for the Hudson River Superfund site.

Specifically, the purpose of this paper is to:

1. Describe how the risk assessment, the EPA PCB-fate and transport model, and feasibility study should be integrated (Section 2).
2. Describe the risk estimating methodology that should be employed (Section 3).
3. Provide the input parameters for use in the risk assessment (Section 4).

This paper focuses on issues relating to estimates of exposures to PCBs (and associated risks) by anglers fishing the Hudson River in the absence of a ban. PCB toxicological properties will be discussed in other papers. A key point that should be kept in mind is that the existing ban on all fishing in the Upper Hudson River below Bakers Falls almost certainly precludes actual exposure through the fish ingestion pathway. Consequently, the estimates of exposure that are produced by the methodology proposed in this paper will greatly overestimate current exposures and actual hazards to Hudson River anglers. Indeed, the actual exposures and hazards may be zero.

2.0 ROLE OF RISK ASSESSMENT IN THE HUDSON RIVER REASSESSMENT

At Superfund sites, risk assessments are performed in accordance with the National Contingency Plan (NCP) and relevant Agency guidance such as the Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part A) (RAGS) (EPA, 1989). Risk assessments under Superfund provide the key information for deciding if remedial actions are necessary and, if so, for evaluating each action in terms of its potential to reduce risks to acceptable levels. The first component of the risk assessment process is the "baseline" risk assessment that defines and quantifies potential risks to human health and the environment from contaminant sources if no remedial actions are undertaken. If unacceptable risks are identified, the "baseline" risk assessment in combination with an analysis of the Applicable, or Relevant, and Appropriate Requirements (ARARs) is used to define Remedial Action Objectives (RAOs). RAOs describe the relationships between contaminant sources and receptors. By evaluating these linkages, the source of risks can be identified and a range of remedial alternatives can be developed to either control the sources, break the linkages between the sources and the receptors, or control the receptors so exposure does not occur. The remedial action objectives form the basis for developing and evaluating remedial alternatives during the feasibility study.

The second major component of the risk assessment is a "comparative" risk analysis performed during the FS. This evaluation assesses the absolute risk reduction potential, the comparative risk reduction potential, and the short- and long-term effectiveness of each remedial alternative. Each of these measures is required in the feasibility study where alternatives are compared to the nine selection criteria (EPA, 1990). The absolute risk reduction measure is used to assess the protectiveness of each alternative to human health. The comparative analysis compares the risk reduction capabilities of each remedial alternative. Additionally, the short- and long-term effectiveness of each remedial alternative are evaluated to ensure that potential negative short-term impacts are considered as well as the ability of each remedial alternative to achieve remedial action objectives in the long-term. This interrelationship between the risk assessment, feasibility study and the selection of the appropriate remedial alternative is illustrated in Figure 1.

It is anticipated that EPA will follow this process for the Hudson River RRI/FS. The "baseline" risk assessment component of the process has not yet been prepared. Additionally, EPA has not provided much detail as to how the risk assessment will be performed, how the PCB fate and transport model will be used in the risk assessment ("baseline" and "comparative"), or how the model, risk assessment, and feasibility study will be integrated. In order to ensure that risks will be appropriately considered in the remedy selection process, it is essential for EPA to articulate it's approach on these issues before the process proceeds any further.

Figure 2 illustrates the interrelationships between the risk assessment, models and feasibility study for the Hudson River. EPA is in the early stages of the RRI/FS, with the final validation of the data collected still underway and the model development and calibration not scheduled for completion until at least June 1995. The most important and potentially difficult portion of the RRI/FS is yet to come. Due to this and the lack of detail provided on the risk assessment and the feasibility study, there are many issues that are still unresolved. These unresolved issues need to be discussed before EPA irreversibly embarks on the final project direction. In order for the RRI/FS to proceed in a scientifically sound and expeditious manner, EPA must address the following issues that are discussed in more detail below.

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Figure 1. Relationship Between Risk Assessment, Feasibility Study, and Remedial Alternative Selection

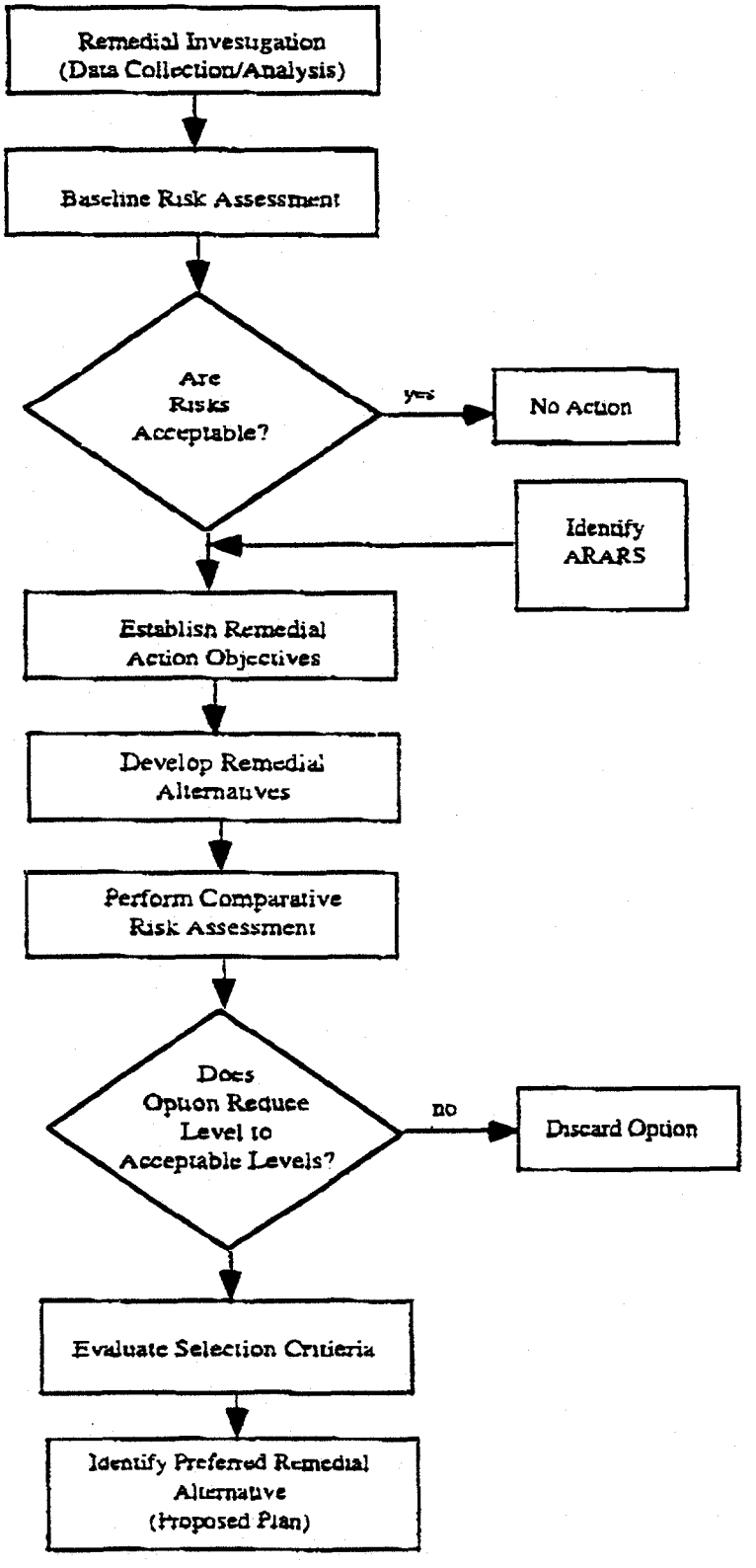
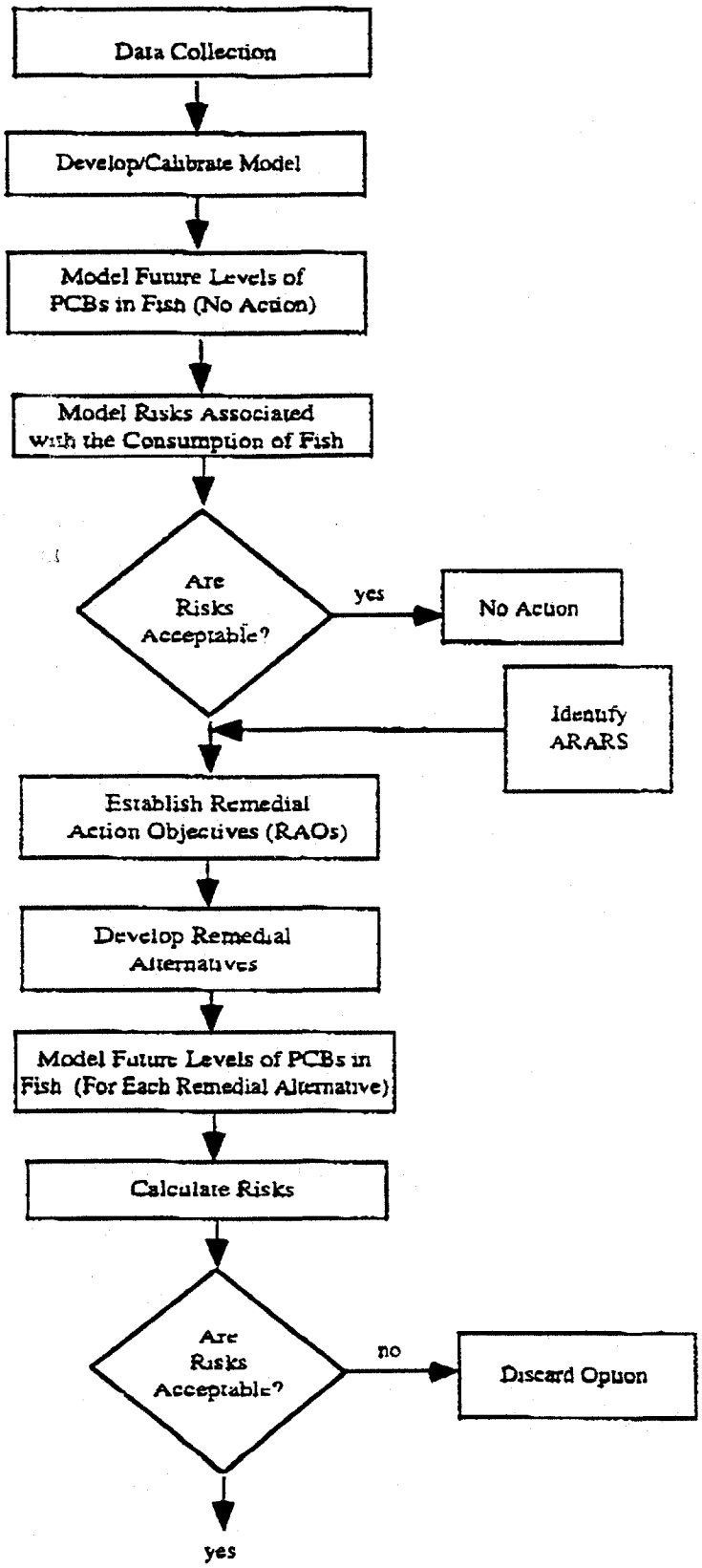


Figure 2. Relationship Between Risk Assessment, Feasibility Study, and Model



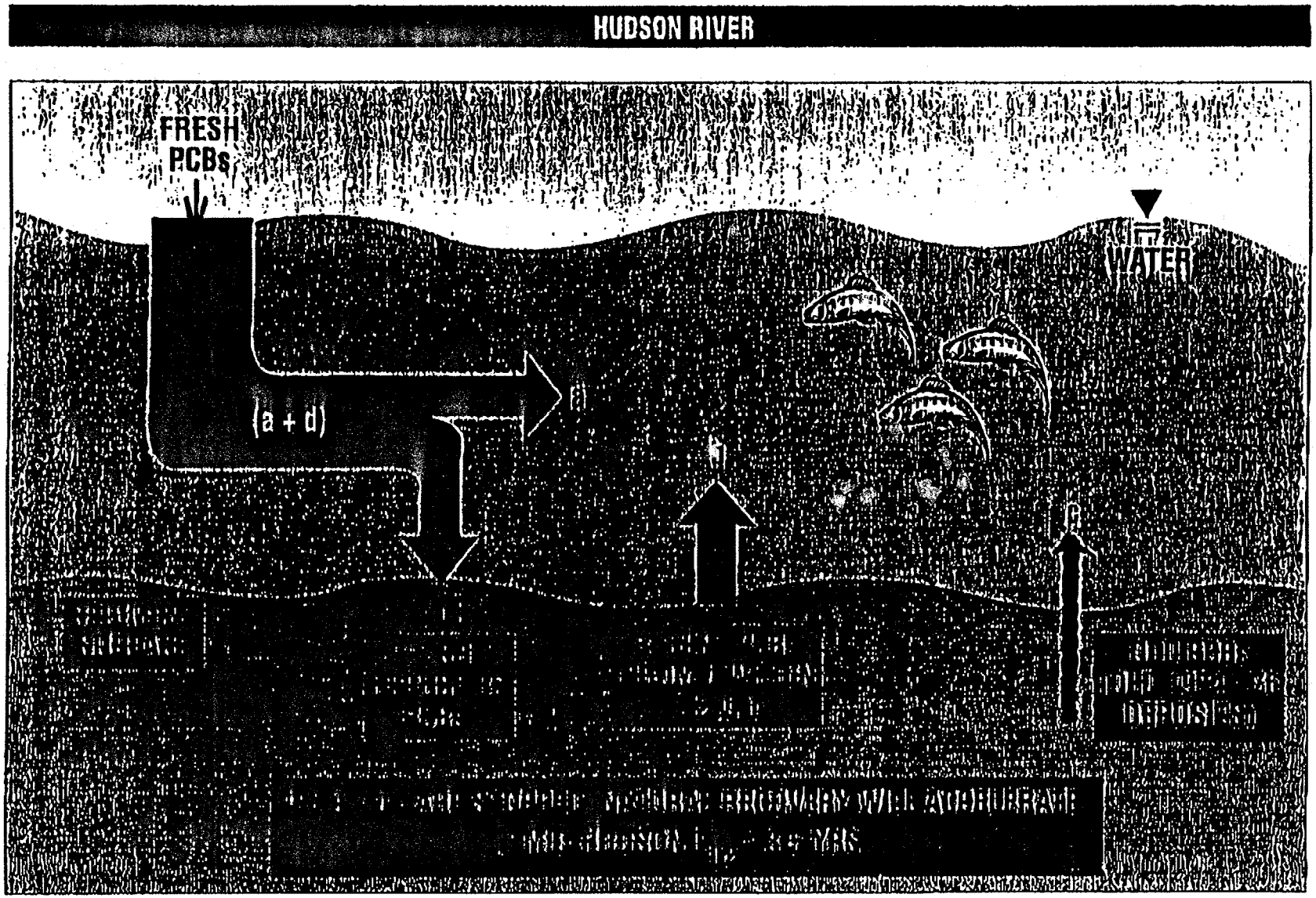
2.1 Differentiation of Source Impacts

U.S. EPA has repeatedly stated that the focus of this RRI/FS is on PCB-contaminated sediments in the Hudson River and not on other PCB sources, such as the old dredge spoil sites (See EPA Phase 1 Report, Page I-2). In the Phase I Report, EPA assumed that other PCB sources did not exist. Since the EPA began the RRI/FS, however, significant new information indicates that sources of PCBs are currently present in the Upper Hudson River upstream of Roger's Island ("upstream source") and have been present historically. This upstream source of PCBs appears to have at least two separate components. One component has been traced to an area near Bakers Falls and is composed of PCB oils and PCB-contaminated groundwater entering the river through fractures in the bedrock. The second component is related to contaminated sediments near the outfall (Number 004) near the Ft. Edward facility. This appears to be a small remnant deposit. The PCB contributions of these two sources have not yet been quantified. Contributions from other potential PCB sources in the Upper River may also occur. Current monitoring programs are not sufficient to identify the presence of specific sources. Since the upstream source is an important contributor to the PCBs in fish in the Hudson River, the risk assessment must be able to differentiate the impact of this source(s) from the contribution from old sediments. This is the only way EPA can properly assess the risk reduction potential from controlling upstream source(s) contrasted with the risk reduction potential of the sediment remediation in the FS. If source impact differentiation is not done, the analysis of remedial alternatives will not be defensible.

As described in various presentations to EPA, the upstream source(s) is composed primarily of unaltered Aroclor 1242. Unaltered Aroclor 1242 has a higher chlorination level and has a significantly higher potential to bioaccumulate in fish than the bioaltered, dechlorinated PCBs present in the sediment. Additionally, the upstream source PCBs are highly bioavailable to the fish through their presence in the water column and in the surficial sediment. By contrast, the majority of the historically-released PCBs are in buried sediments (old sediments) and are not available to the fish for uptake. These concepts are illustrated in Figure 3. The ability of the upstream source to affect PCB levels in fish was clearly demonstrated by monitoring data taken after 1991 when increased loading from the source resulted in immediate increases in PCB fish levels in the Upper Hudson River.

Figure 3.

IDENTIFIED AND CHEMICALLY-DISTINGUISHABLE SOURCES OF UPPER HUDSON FISH PCBs



Another important feature of the upstream PCB source is its apparent longevity. In September 1991 increased activity of the upstream source, apparently due to a failure of a gate in an abandoned paper mill (Allen Mills) adjacent to the river, led to the identification of that source. As a result of this source discovery, the existing historical data were reevaluated to see if the source may have been present historically. The historical data are sparse and are also of limited use due to the relatively high laboratory detection limits for PCBs (for the water matrix). Additionally, most of the historical data were collected downstream of the remnant deposits, which makes it difficult to determine if the PCB levels were from a source upstream of the remnant deposits or were due to the remnant deposits. However, given our current knowledge on the activity of the upstream source (i.e., PCB oil in fractured bedrock), it is probable that the oil seepage from the bedrock had been occurring for some time. The limited data collected above the remnant deposits by NYSDEC in the early 1980s and by HARZA engineering in the late 1980s also are consistent with the upstream source being active during that period of time. As the PCBs in the sediments became buried with new sediment and the PCBs in the buried sediments underwent biologically-mediated dechlorination, the old PCB-contaminated sediments became a less significant source of PCBs to the water and fish. Since the mid to late 1980s, the Bakers Falls source has been key in controlling PCB levels in both the fish and the water column. As a result, the upstream source has reduced the rate of decline in PCB fish levels and has inhibited the natural recovery of the river system.

Given the importance of the upstream source in controlling PCB fish levels, EPA must determine the relative contribution of the upstream and old sediment sources to the levels in the fish. Once EPA determines which, if any, of the PCBs in the fish are derived from the old sediments, the risk assessment for the PCBs in the old sediments can proceed. The EPA Phase 1 risk assessment failed to do this and, as a result, greatly overestimated the risks posed by the PCBs in the old sediments. PCB source impact apportionment will be critical to identifying the remedial action objectives and determining which, if any, of the remedial alternatives can achieve these objectives. This approach allows a rational prioritization of remedial actions.

2.2 PCB Decline Rates

In the risk assessment, the period over which exposure occurs and the concentration to which a hypothetical receptor is exposed are critical parameters. In its Phase I Report, EPA suggested a

default value of a 30-year period of exposure (EPA, 1989). If the Hudson River RRI/FS risk assessment assumes potential exposure to occur over a 30-year period, the next step in the process is to estimate concentrations in fish anticipated over this 30-year period (for both action and no-action scenarios). To do this, EPA might elect to use current values and assume no changes in fish PCB concentrations in the future. Alternatively, EPA may assume that PCB levels in fish will decline due to natural recovery processes. Assuming a constant PCB level is not supportable based on the existing river data which show significant PCB reductions over time. In fact, in its Phase I Report, EPA did assume an annual rate of decline of approximately 26 percent in order to estimate PCB levels in fish 30 years into the future. The decline of PCB fish levels over time greatly reduces exposure intake over the 30-year period when compared to a constant PCB level over the same period. Reduced intake equates to reduced risk. If this is not considered, the risk estimates will be exaggerated and scientifically not supportable.

For the final risk assessment in the RRI/FS, the issue of decline rates is even more complicated because the PCB decline rate for each of the sources must be determined. For the upstream source, the future activity of the source is not clear at this time. GE is aggressively pursuing timely remedial solutions and is hopeful that remediation efforts will be successful. However, the technical challenges presented by the upstream source are significant. PCB oil in fractured bedrock is one of the most difficult types of contamination to control. Since it is possible that the success of remedial efforts will not be known by the time EPA's risk assessments are completed, EPA will need to make various assumptions concerning the future activity of the upstream source. GE recommends that the Agency consider the complete spectrum of possibilities, from an assumption that the upstream source is controlled completely in the near term to an assumption that remediation is unsuccessful in the long-term.

2.3 Assumed Starting Dates for Action and No-Action Alternatives

As described above, an estimate of the PCB levels in fish (by source) over a 30-year period is a necessary input into the risk assessment. Further, a decision must be made regarding the point in time at which potential exposure is assumed to begin. For the "baseline" assessment, GE believes that the appropriate starting point for assuming exposure is no sooner than the date that the Record of Decision (ROD) will be issued for the Hudson River Superfund Site. The "baseline" risk

assessment is intended to reflect the potential risks if no-action is undertaken. Therefore, the soonest a no-action decision could be made is the date on which the ROD is issued.

For the comparative risk assessment of potential remedial alternatives, the starting point should be no sooner than the date on which remediation would start. As an example, if EPA issues an action ROD for sediments in 1997, any such action could not begin until at least 1999 due to the need for PRP negotiations, design, and facilities construction. Based on a 1999 start date, EPA will need to estimate PCB levels in fish from 1999-2029 for the "comparative" risk assessment for each remedial alternative. The risks for no-action must then be compared to the potential risk reductions offered by each remedial alternative during the same time period. This analysis will also need to assess the impact on fish and other adverse effects of implementing each potential remedy.

2.4 Use of Models in the Risk Assessment

The description of the above issues makes it clear that a critical input into the risk assessment will be PCB levels in fish attributable to each source in future years. This information is required for no-action and for each action scenario. The tool used to obtain this information is a technically defensible PCB-fate and transport model ("model") for the Hudson River. This model, if appropriately constructed and calibrated, will be able to apportion PCBs in fish according to the source of the PCBs. The model also provides a mechanism to simulate the effects of each remedial alternative on PCB levels in fish over time. Although EPA to date has not described how the model will be used in the RRI/FS, the description given above is an appropriate way to incorporate the model into the RRI/FS and the associated risk assessments. GE believes that the following information from the model is required for EPA to complete the risk assessment:

1. Fish PCB levels from the date the ROD is issued to 30 years in the future for the "baseline" risk assessment, including total PCB levels in fish, and an apportionment of the PCBs in fish between the old sediments and the upstream source (s).

2. Fish PCB levels from the date remediation would commence to 30 years in the future for the "comparative" risk assessment (no-action and for each remedial alternative identified). Since some combination of sediment and upstream source remediation must be evaluated, the fish PCB levels will also need to be apportioned by source.

3.0 APPROACHES FOR ESTIMATING EXPOSURES TO PCBS IN THE HUDSON RIVER RISK ASSESSMENT

Once PCB levels in fish are determined over time and apportioned by source, the next step is to estimate the potential exposure to PCBs in fish. The method used to estimate PCB intake from potential fish consumption in the Hudson River risk assessment can greatly effect the estimated risks. In the EPA Phase I Report, EPA combined "reasonable" worst case estimates of exposure in a traditional point estimate method to yield an estimate of lifetime average daily dose. As discussed below, this approach greatly exaggerates potential exposure and associated risk and is not justified because more scientifically defensible methods have been developed and accepted by the scientific and regulatory communities. This section of the paper describes a more defensible risk-estimating approach that allows site-specific conditions to be considered.

3.1 Possible Approaches for Characterizing Exposures

There are three approaches that can be used to evaluate exposure to PCBs from consumption of Hudson River fish: a point estimate approach, Monte Carlo analysis, and Microexposure Monte Carlo analysis. The point estimate approach, used by EPA in the Phase 1 Report, is the simplest of the three. This approach assigns point estimates to each of the parameters in a dose rate equation (EPA, 1989). The parameters are a mixture of typical and "reasonable" worst case estimates that are intended to result in a "reasonable upper-bound estimate of exposure" (EPA, 1989). The second approach is the use of a Monte Carlo analysis, where the same equation is used but the point estimate for each parameter is replaced by a distribution of values. The distribution expresses the probability that a specific parameter value will occur for an angler in the exposed population. A distribution of exposures is produced that reflects the possible combinations of different values from the distributions and the probability with which they occur. The third option for exposure assessment is the Microexposure Monte Carlo analysis. This method also uses Monte Carlo simulation, but modifies the exposure equation to better integrate spatial and temporal variations in exposure parameters and to explicitly address correlations between parameters.

Limitations of the Point Estimate Approach

In developing exposure estimates, there are uncertainties associated with estimates of intensity, frequency, and duration of exposure (NAS, 1983; Paustenbach, 1989). Traditionally, regulatory agencies have sought to account for these uncertainties by favoring the use of conservative or "reasonable" worst-case estimates for exposure parameters. This approach overestimates exposures (McKone and Bogen, 1991; Cullen, 1994; Slob, 1994), and this overestimation can occur even when each parameter value itself is "reasonable" (McKone and Bogen, 1991; EPA, 1992a).

The potential for overestimation can be readily demonstrated in the following example. Let us assume that the dose rate for an individual in an exposed population is determined by three parameters A,B, and C. The values for the three parameters are assumed to vary in the exposed population according to three distributions. Finally let the RME for the exposed population be estimated based on the 95th percentile of each of the parameter distribution (a reasonable worst case estimate of the parameter values). Using a simple rate of probability, the likelihood of an individual receiving a dose equal to or greater than the RME is given by the following equation.

$$p = 1 - (1-0.95)^3$$

or

$$p = 0.000125$$

This low probability is equivalent to stating that the RME falls on the 99.9878th percentile of the distribution of doses (Figure 4). Such a high percentile may not be a reasonable estimate for the exposed population. In addition, the potential to overestimate exposure increases with the number of parameters in the exposure model.

As explained by Thompson et al. (1992), and discussed extensively in the risk assessment literature (Finkel, 1990; McKone and Bogen, 1991; EPA, 1992a; Keenan et al., 1994), there are a number of other limitations to the point estimate approach, including:

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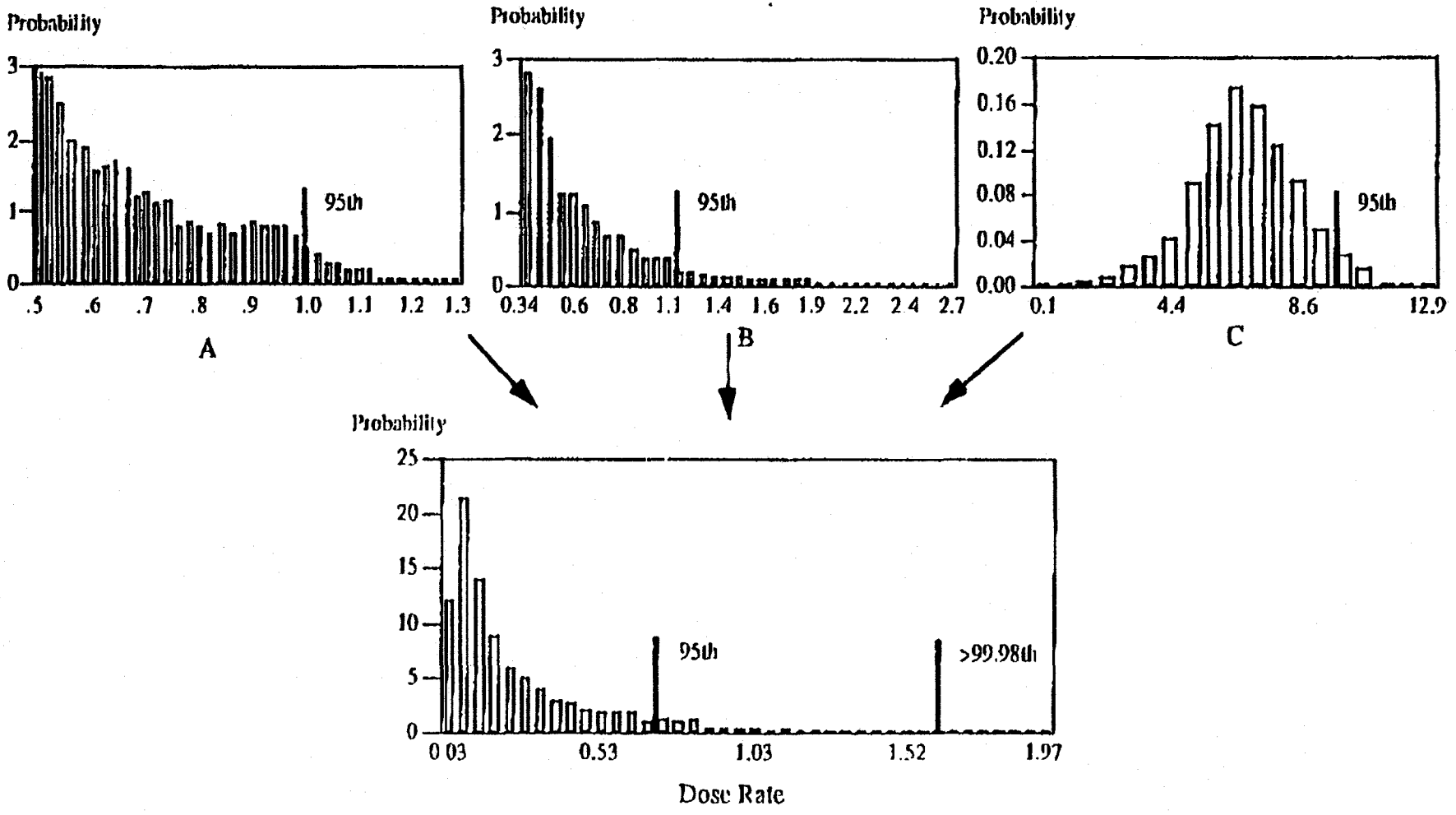
Figure 4. Use of Conservative (but realistic) Parameter Values in Exposure Assessment Can Result in Unrealistic Estimates of Dose Rates

Using the 95th percentile of each parameter results in 99.9+ percentile of actual distribution of intakes



- the current method of selecting conservative values for exposure parameters is a poorly diagnosed process of incorporating value judgement into the scientific stage of the risk assessment/management process;
- risk assessments may consider scenarios that will rarely (if ever) happen;
- the use of point estimates provides disincentives for regulatory agencies as well as risk assessors to generate a better data base for characterizing human exposure; and
- uncertainties in the final point estimates cannot be precisely quantified or even roughly estimated since many of the input parameters are at or near their maxima.

3.2 EPA Policy on Exposure Assessments

Recent changes in EPA's policies and guidelines have focused on improving risk management by presenting decision-makers with the entire range of possible risks rather than a single point estimate (EPA, 1992a, 1992b). The new policy states that numerical risk assessments should always be accompanied by a full characterization of the uncertainties, limitations and assumptions in the risk assessment. The new guidance also replaces the concept of the "maximum exposed individual" and the "reasonable maximum exposure" with a series of exposure descriptors, including individual, population and subpopulation estimates of exposure. The guidelines require that two types of individual exposure be calculated: the typical and the high-end exposure (HEE). The HEE is intended to reflect the doses received by the small but definable "high end" of the population. The primary objective in estimating HEE is that it is a realistic estimate of a potential high-end exposure and is not the result of a theoretical worst-case analysis.

The use of point estimates can only provide "subjective" estimates of the exposure descriptors such as the HEE. In contrast, Monte Carlo analysis is favored by the new approach to exposure and risk assessment. As explained by Hattis and Burmaster (1994), Monte Carlo analysis is not a new technique; it was developed by physicists 50 years ago and has been used in the fields of nuclear engineering, health physics and environmental chemistry. Monte Carlo analysis can be applied to any equation where the distributions of the parameters can be specified. Monte Carlo analysis can effectively characterize the impact of variability or uncertainty in input parameters on the estimates of dose rates in an exposed population. Such a probability distribution function provides risk managers with information necessary for regulatory decision-making. Specifically, the probability

distribution function of the Lifetime Average Daily Dose (LADD) can be used to estimate the dose rates and associated risks for the typical (50th percentile) and the high-end exposures (e.g., 90th to 95th percentile) or can characterize the distribution of the individual risks in the exposed population.

In the Final Phase 2 Work Plan for the Hudson River Superfund site, EPA (1992c) endorsed Monte Carlo methods to the extent that data are available to define parameters. GE agrees with this statement and believes that a probabilistic approach is feasible for this site and will provide the most realistic and reasonable estimates of risk. In fact, GE believes that the current EPA policy on Monte Carlo risk assessment, as set forth in the new *Guidelines for Exposure Assessment* (EPA, 1992a,b), essentially requires the use of Monte Carlo analysis for sites where high quality site-specific data are available. More recently, in its draft document entitled *Estimating Exposure to Dioxin-Like Compounds*, EPA (1994) stated that "Monte Carlo techniques can be a powerful tool for expressing variability and evaluating scenarios in exposure assessments."

In the case of the Hudson River risk assessment, GE believes that the unusually large amount of information on the variation in key exposure parameters is more than adequate to allow the use of probabilistic techniques. As discussed in Section 4.0, considerable data are available that allow the characterization of the variation in the key parameters in the fish consumption pathway for the Hudson River.

3.3 Microexposure Monte Carlo Analysis

GE proposes that a state-of-the-art assessment of potential exposures to PCBs at the Hudson River site be performed using a Microexposure Monte Carlo technique. This approach is a modification of the synthetic life history approach developed by Price et al. (1991; 1992). It has been used by EPA and independent researchers to investigate residential exposure duration (Johnson and Capel, 1992; Sielken, 1994). It also has been used to evaluate childhood lead exposure (Goodrum et al., 1994) and to evaluate exposure to dioxins from the consumption of freshwater fish (Keenan et al., 1993a,b).

Microexposure Monte Carlo, a refinement of traditional Monte Carlo analysis, provides greater flexibility in modeling exposures that vary over time, that are the sum of independent exposure events, and when the age of the angler affects the dose rate. All of these advantages are useful in the assessment of the Upper Hudson River. For example, fish concentrations vary over time. The body weight of an angler and the duration of angling are influenced by the age of an angler. Finally, long-term exposure to PCBs is a function of the consumption of many fish. Microexposure Monte Carlo simulation uses the available information on the distribution of fish concentrations to characterize the distribution of long-term exposures to PCBs.

Microexposure Monte Carlo analysis is a technique in which an individual's total exposure to a contaminant is calculated by summing the doses received by many individual exposure events. Each event is simulated using information specific to the time and location of the event. The number of events and sequence in which they occur in the individual's life can be simulated based upon information on an individual's short- and long-term behavior. Modeling long-term exposures as a summary of separate events is not new; in fact, this approach is recommended by EPA (1992d) for evaluating exposures which occur primarily during childhood, when body weights change rapidly.

The difference between traditional Monte Carlo analysis and Microexposure analysis can be illustrated by comparing how the two techniques are used to estimate the dose used in carcinogenic risk estimates. Traditional Monte Carlo uses the same equations as the point estimate approach but replaces the point estimates with distributions. In traditional Monte Carlo, the dose rate is calculated using the following equation:

$$LADD = \frac{C \times IR \times ED}{BW \times LT}$$

where,

LADD	=	the lifetime average daily dose rate
C	=	the distribution of the average concentrations of the chemical in the medium experienced by an individual over his or her life
IR	=	the distribution of intake rates of the medium in the exposed population
ED	=	the distribution of durations of the individual's exposures

BW	=	the distribution of body weights in the exposed population at the time of exposure
LT	=	the distribution of lifetimes (converted to days) over which the dose is averaged

In contrast, Microexposure Monte Carlo analysis defines lifetime exposure as the sum of potential short-term (e.g., annual, daily) exposures represented by the following equation:

$$LADD = \frac{1}{LT} \sum_{j=1}^{Duration} \frac{1}{BW_j} \sum_{i=1}^{Exposure\ Events_j} C_{ij} \times IR_{ij}$$

where,

C_{ij}	=	the concentration of the contaminant in the environment that an individual is exposed to during the ith exposure event in the jth year of his or her life
IR_{ij}	=	the rate the contaminant enters the body of the individual during the ith exposure event in the jth year of his or her life
$Exposure\ Events_j$	=	the number of exposure events that occur during the jth year of the individual's life
BW_j	=	the average weight of the individual during the jth year of individual's life
Duration	=	the number of years that the individual is exposed
LT	=	a standard lifetime for humans

The Microexposure Monte Carlo technique is very useful in assessing exposures from fish consumption. In a fish consumption scenario, an angler's lifetime intake can be considered the sum of the intakes that he or she receives during each year that he or she fishes from the site. Each year of fish consumption can, in turn, be expressed as the sum of fish meals consumed during that year.

In the case of the Hudson River, the Microexposure equation would be defined as follows:

$$LADD = \frac{1}{LT} \sum_{j=1}^{Angling\ Duration} \frac{1}{BW_j} \sum_{i=1}^{Fish\ Consumed} Fish\ Concentration_{ij} \times Fish\ Size_{ij} \times (1 - Cooking\ Loss_{ij})$$

where,

angling duration	=	the period of time in years that an angler may fish the Upper Hudson
fish consumed	=	the number of fish consumed in the ith year
fish concentration$_{ij}$	=	the concentration of PCBs in the ith fish caught in the jth year
fish size$_{ij}$	=	the size of the edible portion of the ith fish caught in the jth year
cooking loss$_{ij}$	=	the fraction of PCBs lost during the cooking of the ith fish caught in the jth year.

The major advantage of Microexposure Monte Carlo analysis is its flexibility in incorporating information on temporal changes in exposure parameters. For example, PCB concentrations in Hudson River fish have varied significantly over time. Microexposure Monte Carlo modeling is able to explicitly incorporate this temporal variation into estimates of long-term dose rates. As another example, individuals change throughout their lives. Behaviors that are reasonable for one age are not reasonable for another. For young individuals, there are significant changes in body size as the individuals mature. In addition, exposure-related behaviors such as fish consumption and residential mobility also change with age. Microexposure Monte Carlo analysis allows the incorporation of age-related exposure factors into the estimates of long-term dose rates by adjusting the number of fish meals or the individual's body weight by the age of the angler for each year of exposure. As a third example, traditional Monte Carlo analysis of fish consumption has a shortcoming in that it estimates each angler's lifetime exposure by assuming that every fish consumed contains a single uniform concentration (Anderson et al., 1992). In reality fish concentrations vary from one fish to another. By modeling each fish meal separately, Microexposure Monte Carlo analysis considers the varying concentrations in individual fish.

In the assessment proposed for the Upper Hudson River site, an angler's total consumption of fish may be estimated as a series of separate exposure events (individual fish meals). The doses received from these events can be calculated independently and summed to provide estimates of chronic and lifetime exposures. In addition, the duration of an individual angler's exposure is characterized not by adoption of a distribution of durations, but is assessed using information on the angler's age at the time the exposure begins, together with age-specific rates of mobility, mortality, and angling cessation. Finally, exposure parameters such as body weight and fish consumption are also determined based on the age of the angler.

4.0 DEVELOPMENT OF DISTRIBUTIONS FOR EXPOSURE PARAMETERS

The application of Microexposure Monte Carlo analysis to the exposure assessment for the Hudson River risk assessment requires distributions of the interindividual variation in the parameters in the dose rate equation used to evaluate the exposure. The goal, as described above, is to determine the distribution of LADDs in a hypothetical population of anglers who would fish the Upper Hudson in the absence of fishing restrictions. This section presents a summary of key parameters that should be employed for the Hudson River fish consumption Microexposure Monte Carlo risk analysis.

4.1 Fish Consumption Rates

The amount of fish that anglers consume is a key parameter in the estimate of exposure to PCBs from Upper Hudson River sediments. In its Phase 1 Report, EPA (1991) recommended 30 g/day as an estimate of fish consumption by Hudson River anglers. This estimate was based on the median consumption rate reported by marine anglers in surveys by Puffer et al. (1981) and Pierce et al. (1981). The amount of fish consumed by a population of anglers varies depending on the numbers and types of waterbodies fished and the characteristics of the angler population. Fish consumption also depends on factors such as climate, fish species present, productivity, access, and the size of the angler population. In the Final Phase 2 Work Plan, EPA (1992c) expressed a willingness to develop site- or region-specific consumption rates for Hudson River anglers if appropriate data are available. The 30 g/day rate likely overestimates the intake for Hudson anglers, since consumption of self-caught marine fish is typically higher than consumption of freshwater fish. Moreover, the results from Puffer and Pierce are biased toward the frequent angler which overestimates fish consumption (Price et al., 1994).

Unfortunately, no historical survey on the fish consumption rates of anglers using the Upper Hudson River has been performed. In addition, no such survey can be performed, due to the existence of a State ordered and enforced fishing ban on the affected portion of the river. Because of the current fishing restrictions, any survey performed on the Hudson River anglers will not

provide an appropriate baseline for the river. In the baseline risk assessment, the goal is to assess the risks that would occur in the absence of any regulatory controls.

There are three surveys of angler behavior that involve anglers on the Hudson River. Two mail surveys have been performed on New York anglers (NYSDEC, 1990; Connelly et al., 1992) and a creel survey (Barclay, 1993) was performed on Hudson River anglers. However, none of these surveys focused on fish consumption from the Hudson River. NYSDEC (1990) evaluated fish consumption from all recreational and commercial sources, including self-caught fish from the Hudson. Connelly et al. (1992) evaluated self-caught fish consumption, but did not estimate consumption from individual waterbodies. Barclay (1993) collected data on the frequency of self-caught fish meals, but did not calculate a fish consumption rate. In addition, the survey does not contain sufficient information to allow the calculation of a meaningful fish consumption rate for the Upper Hudson River.

Because site-specific data on fish consumption are unavailable, GE recommends that EPA use estimates for the Hudson River based on data from a surrogate study or from regional data. Numerous estimates of consumption rates have been made for both the general population of the U.S. (Javitz, 1980; Rupp et al., 1980; USDA, 1980) and for recreational anglers (Soldat, 1970; Honstead et al., 1971; Pierce et al., 1981; Puffer et al., 1981; Turcotte, 1983; Landolt et al., 1985, 1987; Cox et al., 1985, 1987, 1990; Fiore et al., 1989; West et al., 1989; NYSDEC, 1990; ChemRisk, 1991a,b; Connelly et al., 1992; Richardson and Currie, 1993; Ebert et al., 1993). These studies have reported a wide range of fish consumption values and have examined consumption rates of fish taken from various types of waterbodies ranging from all waters to single bodies of water (Ebert et al., 1994).

Given this wide range of angler studies and consumption rates, the study and rate of consumption for the assessment of risk to anglers at the Upper Hudson River site should be selected carefully. The selection of a surrogate study depends on the characteristics of the population who have the potential to be exposed and the type of waterbody being evaluated. Specifically, it is critical that the study evaluate self-caught, freshwater fish over a long period of time. These criteria must be met to ensure that the fish consumption rate closely approximates consumption from the Upper Hudson. In addition, it would be preferable to use a study that evaluated consumption from a

single flowing system that was similar to the Hudson. However, if a specific waterbody with appropriate characteristics cannot be identified, it may be more appropriate to use estimates generated for flowing waters only. Finally, the selected study should have collected data from a regionally appropriate waterbody.

There are a limited number of studies available in the New York/New England area that provide information on consumption of sport-caught fish from freshwater rivers and streams. The Ebert et al. (1993) and Connelly et al. (1992) studies most closely approximate hypothetical consumption from the Hudson River. Both of these studies evaluated consumption of self-caught freshwater fish by recreational anglers using a mail recall survey. Given these similarities, it is not surprising that both studies reported very similar fish consumption rates. The results of Connelly et al. (1992) indicated that the average New York angler consumes 11 meals per year of self-caught fish from New York's freshwater fisheries. If it is assumed that each meal is 227 grams in size (1/2 pound) (West et al., 1989; NYSDEC, 1990), it can be estimated that the average New York angler consumes self-caught freshwater fish at a rate of 7 g/day. This estimate is very similar to the mean rate of freshwater fish consumption by Maine anglers of 6.4 g/day from all waters reported by Ebert et al. (1993).

Although the Connelly et al. (1992) study is specific to New York State, there are several factors which limit its usefulness in the Upper Hudson River assessment. First, Connelly et al. (1992) only present a single point estimate value for fish consumption. The use of a distribution of consumption rates, however, is necessary in order to characterize interindividual variability and realistically assess the potential risks to recreational anglers. With only an average consumption rate value, it is not possible to accurately represent the range of recreational anglers, including those who ingest higher amounts of fish.

Second, the mean fish consumption rate determined by Connelly et al. (1992) represents fish eaten from all freshwaters in the State (i.e., lakes, ponds, rivers, and streams). As pointed out in Ebert et al. (1993), intake from rivers and streams is only a fraction of the intake from all freshwaters. In addition, the rate of intake from multiple waterbodies is higher than that from a single water system (Ebert et al., 1994). Given these factors, it is highly likely that the fish consumption rate in Connelly et al. (1992) overestimates the actual fish consumption rate on a single portion of the

Upper Hudson River. Finally, it is important to note that the purpose of the Connelly et al. (1992) study was not to identify a consumption rate for New York anglers. Although questions were asked in the survey regarding fish consumption behaviors, those questions were aimed at estimating how the effect of health advisories altered the consumption behavior of recreational anglers.

While the data from Ebert et al. (1993) are not specific to New York State, these data provide a more appropriate surrogate for Hudson River anglers than the Connelly et al. (1992) data. Angler demographics and fishing opportunities are similar in Maine and New York, and the mean fish consumption rates are similar for both studies (NYSDEC, 1990; Connelly et al., 1992; Ebert et al., 1993). In addition, Ebert et al. (1993) provide a complete distribution of fish intake rates for flowing waters, i.e., streams and rivers. Thus, the best region-specific data on fish consumption rates are available from Ebert et al. (1993) and should be used in the Hudson River risk assessment.

The selection of the most appropriate fish consumption rate is discussed more fully in the paper entitled *Estimating Fish Consumption Rates for the Upper Hudson River* and in the manuscripts, *The Effect of Sampling Bias on Estimates of Angler Consumption Rates in Creel Surveys* (Price et al., 1994), *Selection of Fish Consumption Estimates for Use in the Regulatory Process* (Ebert et al., 1994), and *Estimating Consumption of Freshwater Fish among Maine Anglers* (Ebert et al., 1993). EPA should use the distribution of fish consumption rates for flowing waters as developed by Ebert et al. (1993).

4.2 Species Preference

Anglers typically seek to catch certain desirable species and to reject others. Since PCB levels in fish vary by species, it is important to capture this angler preference in the estimates of exposure to PCBs. For example, if anglers tend to favor species which happen to have lower PCB concentrations, their potential exposures will be lower than the average of all the species or the upper-end species.

In the Phase 1 Report, EPA (1991) acknowledged that New York anglers do not spend equal time fishing for all species and that PCB concentrations vary from one species to another. Nevertheless, EPA (1991) chose to average the PCB concentrations from all species sampled to determine a single point estimate (95th upper confidence limit of the mean) of PCB concentration in fish tissue. In the Final Phase 2 Work Plan and Sampling Plan, EPA (1992c) expressed a willingness to refine its estimate if appropriate data were available on species preference.

Recent studies by the New York State Department of Environmental Conservation indicate that New York anglers preferentially select for certain species in both fishing effort and consumption (NYSDEC, 1990; Connelly et al., 1992). In many cases, the species selected were those that accumulate lower levels of PCBs, often because these most desirable species have relatively low lipid contents as compared to other species present in the Upper Hudson. Since the species of fish sampled by EPA for PCB tissue analysis are not necessarily consumed by recreational anglers in amounts proportional to their sampling frequencies, the risk assessment for the Upper Hudson should consider both the tissue levels of PCBs in various fish species, and angler preferences.

Information on species preference specific to the Upper Hudson River is unavailable. However, data on angler preference in freshwater rivers in New York similar to the Upper Hudson River are available from Connelly et al. (1992). Based on these data, it is possible to identify species preferences among New York anglers that can be used as a surrogate for Hudson River anglers. Connelly et al. (1992) collected information on fishing behaviors (e.g., species caught, waterways fished) and fish consuming behaviors (e.g., species eaten, preparation techniques used) of licensed anglers. In order to use these data for the Upper Hudson, it is necessary to identify rivers and streams with characteristics and species similar to the Upper Hudson. Such an analysis results in a list of fish species likely caught in the Upper Hudson and the probability of how often these species are eaten. By taking this approach, a probability distribution that accurately reflects species consumption preferences of Hudson River anglers can be developed. The paper entitled *Determining the Intake of Hudson River Fish by Species* provides a complete discussion of this issue and recommends the appropriate input parameters for the Microexposure Monte Carlo analysis.

4.3 Reduction of PCBs in Fish Tissue from Cooking Processes

Exposure to PCBs from fish consumption depends on the PCB concentration in the fish as they are consumed in a meal. If the cooking process reduces the amount of PCBs in a fish or fish fillet, then the dose the angler receives is reduced. In its Phase 1 Report, EPA (1991) estimated PCB levels in fish tissue from raw samples. EPA (1991) dismissed the impact on PCB levels from various cooking processes, citing an absence of a consensus in the published literature that cooking reduces PCB concentrations in fish. In the Final Phase 2 Work Plan, EPA (1992c) agreed to revisit this issue.

Although EPA acknowledges studies that report PCB reduction from cooking, the variability in these data has led EPA to conclude that the effects of cooking do not warrant consideration. Reported reductions have varied over an extremely large range and have not been reported in a consistent manner. This inconsistency has hampered comparisons and compilations of results to date. To address this issue, Sherer and Price (1993) analyzed the available literature to determine if a pattern could be identified. The authors converted the results of each study to a percent loss of PCBs on a total mass basis, which allowed them to determine an average PCB loss for each cooking method. This analysis indicates that the current literature justifies a reduction in PCB concentrations with cooking practices. The amount of PCB loss depends on the percent lipid in the fish and the specific cooking method used. Fish fillets with high concentrations of lipids tend to lose more PCBs during the cooking process, and cooking methods that remove fat (e.g., frying) tend to be more effective in reducing PCB tissue levels.

The recent summary of PCB literature demonstrates cooking processes likely used are effective at removing PCBs from fish (Sherer and Price, 1993). In addition, research shows that freshwater anglers in the region typically use cooking methods that reduce PCB levels in self-caught fish (Connelly et al., 1992; ChemRisk, 1991a). Anglers are more likely to select preparation methods that lead to greater reductions in PCBs (e.g., frying) than methods that do not substantially reduce PCB concentrations, such as eating raw fish or making fish soup. Thus, the amount of PCBs per fish consumed by Hudson anglers is substantially less than reported in analyses of uncooked fillets

and should be incorporated into the Hudson River risk assessment. In fact, the New York State Department of Environmental Conservation (NYSDEC, 1991) recommends certain cooking practices as a means of reducing exposure to organochlorines in fish. Information on the frequency that freshwater anglers use various cooking methods is available from Ebert et al. (1993) and Connelly et al. (1992).

The paper entitled *Evaluating the Impact of Cooking Processes on the Levels of PCBs in Fish* and the manuscript *The Effect of Cooking Processes on PCB Levels in Edible Fish Tissue* (Sherer and Price, 1993) provide a full discussion of this issue and give the recommended values to use in the Microexposure Monte Carlo analysis.

4.4 Duration of Exposure

EPA estimates exposure in terms of the lifetime average daily dose or LADD. The LADD received by an angler is influenced by the number of years he or she fishes the Upper Hudson River, i.e., the longer the duration, the higher the LADD.

In its Phase 1 Report, EPA (1991) estimated a duration of 30 years as the time an angler may consume fish from the Hudson River. This conservative estimate is based on a 1983 survey by the U.S. Bureau of Census on household occupancy times and represents the 90th percentile for the number of years an individual is likely to reside at the same residence. Although residential mobility is an accurate predictor of exposure duration for many sources of contamination that occur in or near the home, the duration of time an individual remains in one residence may not be a reasonable predictor of the duration of angling from a particular waterbody. Exposure from consuming recreationally caught fish will only affect those individuals who continue to fish the waterbody of concern regardless of their current residence. Therefore, residential mobility alone is not a reliable surrogate for the prediction of exposure duration for fish consumption.

For the Upper Hudson, GE proposes to define the exposure duration as the time an angler begins fishing and continuing until the angler no longer catches and consumes fish from the Hudson River. The point at which an angler stops fishing varies with the individual angler and is

influenced by three factors: (1) mobility; (2) mortality; and (3) the decision to give up fishing. The duration of exposure can only be properly estimated when all of these factors are considered.

When evaluating mobility, it is necessary to go beyond a strict consideration of residential mobility because changes in household location may not lead to changes in fishing behavior. Only when an individual moves a sufficient distance will a change be made in preferred fishing locations. Although interstate or U.S. regional mobility data could be used to estimate the number of individuals who give up fishing each year, interstate moves would not account for intrastate moves that would result in a change in angling practices. County mobility may be the most appropriate scale at which to measure the gain or loss of potential Hudson River anglers due to distance. These data are available by age, gender, and race from the U.S. Bureau of Census and can be used to develop a distribution of the probability of moving at each age.

Mortality also determines how long an individual angler catches and consumes fish. Anglers tend to be individuals who are older than the general population (ChemRisk, 1991a). Standard actuarial mortality tables predict the life expectancy of a given angler and whether that individual remains a member of the population of living anglers. Age- and gender-specific data on mortality are available from the New York State Department of Health and the National Center for Health Statistics and can be used to develop a distribution of the probability of dying at each year of an angler's life.

An angler may lose interest in the sport of fishing and give it up for a number of reasons. In fact, at every age there is a certain probability that an individual will permanently give up the sport. However, due to the difficulty of collecting these data, no study has evaluated this phenomenon directly. Information on the age structure of recreational anglers has been reported by ChemRisk (1991a) and can be used to indirectly gain insight on the fraction of anglers who permanently give up fishing at different ages. The age-structure data indicate that a sizable fraction of anglers give up fishing between the ages of 30 and 60.

A probabilistic analysis of angler behavior can characterize the date and age that an angler gives up fishing based on age-specific data on mortality, mobility, and angler practices. This type of information can be used to determine the age-specific probability that an individual will

permanently stop fishing. The paper entitled *Estimating Exposure Duration for the Hudson River Risk Assessment* provides a full description of this issue and recommends the values to be used in the Microexposure Monte Carlo analysis.

5.0 SUMMARY AND RECOMMENDATIONS

Given the magnitude of the physical, chemical and biological impacts of the potential remedial decision on the Hudson River and the associated costs, it is necessary for EPA to clearly demonstrate the benefits and risks of any proposed action. In doing so, EPA must fully utilize the vast amount of information available concerning the river and use analysis tools that will help reduce the uncertainty in the risk/benefit analysis. When faced with uncertainty, regulatory agencies, such as EPA can adopt very conservative assumptions that often tend to grossly overestimate baseline risks and potential benefits of remedial actions. Alternatively, EPA can thoroughly analyze the data in hand and embrace the refined and proven tools of exposure assessment that are now readily available. The approach for the development and utilization of risk assessments for the Hudson River RRI/FS project as outlined in this paper, will still yield results that are conservative (i.e., do not underestimate potential exposure). However, given the state of development of risk management science, the outlined approach will provide a more accurate estimate of the risk/benefit of any remedial action for the Hudson River than more traditional approaches adopted to analyze this type of problem.

To complete the Hudson River Risk assessment, the EPA should do the following:

1. Use the PCB-fate and transport model to estimate PCB fish levels 30 years into the future for action and no-action scenarios. This is a key input into the risk assessment.
2. Clearly identify all sources of PCBs entering the Upper Hudson River.
3. Determine separately the risk associated with PCBs in fish originating from the upstream source and the old sediments (Baseline Risk Assessment).
4. Evaluate the risk reduction potential of each remedial alternative compared to the no-action alternative. This analysis must include an assessment of the relative contribution of PCBs to the fish from the upstream source(s) and the PCBs in the old sediments (Comparative Risk Assessment).
5. Employ the Microexposure Monte Carlo technique as the technique for estimating risk.

6. Use reasonable exposure values and distributions (i.e., those relevant to the Hudson River site) in the risk assessment as described in Section 4.0.

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March 1, 1996

Mr. Kevin Garrahan
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National Center for Environmental Assessment
USEPA
401 M Street S.W.
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RE: RESULTS OF ADDITIONAL MAINE ANGLER SURVEY ANALYSES

Dear Mr. Garrahan:

This letter summarizes the results of analyses conducted on the 1990 Maine Angler Survey data in response to questions raised during our meeting in Portland last fall.

Information on the Top Ten Percent Consumers of Fish in the Maine Angler Survey

At our meeting, you and your colleagues raised several questions related to whether the anglers with the highest consumption rates had characteristics identifying them as a cohesive subpopulation. To address these questions, we compared angling behavior, demographic characteristics, and advisory awareness for the top ten percent consumers (hereafter referred to as the high consumers) to the remaining 90 percent (hereafter referred to as the remaining consumers). This comparison was performed for both the *all waters* and *rivers/streams* fish consumption rates. We also examined the ratings of site characteristics important to fishing location choice for the high consumers and compared them to the ratings of remaining anglers.

Tables 1 and 2 compare angling behavior between high consumers of fish from all waters and from rivers/streams, respectively. Although the high consumers in both cases generally took more fishing trips, distances traveled to preferred fishing locations were similar (i.e., approximately 30 miles on average) between consumer groups. The high consumers from all waters tended to be much more avid ice anglers than any of the other consumer groups.

Tables 3 and 4 summarize demographic characteristics for high consumers and remaining consumers for both all waters and rivers/streams, respectively. The information presented regarding employment status, educational attainment, ethnicity, and income level is focused on those characteristics that might be relevant to identifying a subpopulation dependent on freshwater fishing for food. No substantial differences were noted between high consumer and remaining consumer groups. While high

rivers/streams consumers were nearly three times as likely to be only seasonally employed, and there was a larger fraction of Native Americans among the high consumers, consumption rates for these groups were not clustered separately from other employment or ethnic groups within the high consumer group. Similarly, although there was a greater representation of households with incomes less than $10,000 per year in the high consumer groups than in the remaining consumer groups, we observed no relationship between income and consumption rates within the groups.

Analysis of responses to questions about fish consumption advisories suggest that the high consumers are better informed than the remaining consumers, as shown in Tables 5 and 6 for all waters and rivers/streams, respectively. Awareness of advisories was greater among high consumers, and a larger fraction of high consumers fished from advisory locations than did remaining consumers, but a majority of all consumers modify their consumption behavior for fish from advisory waters. However, it must be remembered that of the approximately 37,000 miles of rivers, streams, and brooks in Maine, only 200 miles of mainstream, warmwater rivers had any history of pollution or advisories issued at the time of the survey. Not only was just a small portion of available bodies of water affected, but also, the availability of nearby alternative fishing locations makes it unlikely that the survey was biased by angling suppression. Figures 1 and 2 for high consumers from all waters and rivers/streams, respectively, present flow diagrams of awareness of, and behavioral responses to, advisories for these consumer groups. Figures 1 and 2 further support the results in Tables 5 and 6.

A review of the high consumers' responses concerning their preferred fishing locations did not reveal any clumping of preferred locations and revealed only infrequent mention of locations potentially covered under advisories applicable in 1990. For example, the 35 high anglers indicating preferred fishing locations reported 33 different first choices, and only one of these may have been covered by an advisory ("Kennebec River").

To further investigate high consumer's choice of fishing locations, we examined their ratings of important site characteristics. Figures 3 and 4 present the results for all waters and rivers/streams high consumers, respectively. While high consumers from both rivers/streams and all waters ranked the factors related to the productivity of the body of water as being important, they also highly ranked factors which suggest fishing is a pleasurable experience. For example, the highest rankings were given to factors such as presence of desirable fish species, beauty of the surrounding area, the type of waterbody, and the presence of few other anglers. These factors suggest that the high consumers select bodies of water based on recreational objectives. Conversely, factors that one might anecdotally associate with angling as a subsistence activity were consistently among the factors rated as not important. Examples of these factors include proximity to camp, proximity to home, ease of fishing from shore, and easy access from a road.

Overall, we found little evidence that the anglers with consumption rates at or above the 90th percentile are distinguishable from other consumers by factors other than consumption rates. These results suggest that the high consumers from the 1990 Maine Angler Survey do not constitute a cohesive, identifiable subpopulation. Furthermore, the high consumers tended to identify favorable recreational factors as more important influences upon their choice of angling location than were those factors related to reliable provision of food.

Further Information on Effects of Fish Consumption Advisories

The effect of fish consumption advisories on consumption rates was also raised at our meeting. As mentioned earlier in this discussion, only 200 of 37,000 possible miles of Maine rivers, streams, and brooks have a history of industrial pollution that have led to advisories. Out of a total of 748 fishing locations identified by respondents, only 27 were at potentially impacted waters. In addition, no individual angler identified only potentially impacted locations as his or her top five preferred fishing locations.

Figure 5 presents a flow diagram analogous to Figures 1 and 2 that charts the awareness of 1990 Maine Angler Survey respondents to the presence of fish consumption advisories as well as the respondents' behavioral responses to these advisories. Of the 35 percent who were aware of advisories, 27 percent fished at an advisory location. Seventy-four percent of those who fished advisory waters modified their behavior with respect to consuming fish from these locations as a result of the advisory. Only 18 percent of those aware of advisories would have fished additional waters in absence of advisories. Together, these responses suggest that advisories are largely effective in Maine among those who are aware of them. However, the presence of advisories does not substantially limit fishing effort, due to the very limited stretches of advisory waters compared to fishable waters and the observation that no anglers preferred only advisory locations as preferred fishing spots.

Potential for Nonresponse and Complexity-Related Biases

As we discussed during our meeting, the 1990 Maine Angler Survey did not have a component to follow-up with nonrespondents. Our survey enjoyed a high (64 percent) response rate and thus the potential for nonresponse bias is less of a concern than for surveys with lower response rates. It is our belief that if nonresponse follow-up had been conducted, then our final consumption rate estimates would have been adjusted downward if at all. Research has demonstrated that response rates tend to be positively correlated with the salience of an issue to respondents (Haberlein and Baumgartner, 1978). Other recreational surveys indicate that nonrespondents have lower participation rates than respondents (e.g., Brown and Wilkins, 1978; Connelly et al., 1990, 1992; West et al., 1989, West, P.C., 1991; Tarrant and Manfredo, 1993). This evidence suggests that if nonrespondents to the 1990 Maine Angler Survey were different than respondents, then it would have been due to their lesser interest and/or participation in angling and fish consumption.

Having conducted an in-depth consideration of these issues, we are convinced that the anglers who responded to the 1990 Maine Angler Survey are representative of Maine anglers in general. Prior to our 1990 survey, two other mail surveys of Maine anglers were conducted in a largely similar manner. Table 2 of the July 1992 survey report compares respondent characteristics among these three surveys. Each survey was based on random samples and, as illustrated by Table 2, each shared similar respondent characteristics. As described in the July 1992 report, a survey pretest was conducted to assess survey difficulty and complexity among potential respondents, and the final survey instrument was refined following the pretest effort. Based on these facts, we believe that the respondents to the 1990 Maine Angler Survey were representative of Maine anglers characterized in previous angler surveys, despite the added length and complexity of the 1990 survey as compared to the previous angler surveys.

Paul White raised the question of whether the format of detailed questions about numbers and length of fish caught and consumed (e.g., Questions 11, 24, 29, and 31) might have proved too difficult or

challenging for respondents and, as a result, whether anglers might begin but not complete answering these questions. Were this to occur, then fish consumption rates might be underestimated using our analysis procedure. Based on the results of the survey pretest and the results presented in Table 4 of the July 1992 report, we do not believe that this form of complexity-related underreporting or associated bias is present in survey results. The species listing order in Table 4 corresponds to the species order in the relevant survey questions. Were there a systematic effect due to respondents only partially completing the questions, than the consumed quantities by species in Table 4 might show a decreasing trend moving down the species list. No such trend is noted. Furthermore, the species identified and the relative numbers consumed across species and fishing modes correspond to expectations for Maine anglers (e.g., smelt, white perch, and brook trout being the most-consumed species from ice fishing, lakes/ponds, and rivers/streams, respectively).

Use of Average Fish Length to Calculate Consumable Mass

Paul White also raised the question of whether our use of *average* fish length data might cause us to underestimate consumable mass due to the nonlinearity in length-weight relationships for fish species. Although we cannot know whether an underestimate of this nature could have occurred, we can investigate its potential for having a significant affect on consumption estimates.

For such an underestimate to occur, the sizes of consumed fish would have to vary about the reported average, and thus consumption of more than one fish per species would have to have been reported. The majority of anglers reported consumption of 10 fish or less of any particular species. For such an underestimate to be significant, variance in fish size would have to be relatively large. In general, for the species included in the survey, an increase of length on the order of 25 percent is required to generate an increase in mass of 100 percent. Considering that, to maintain the same average, an increase in length evaluated for some fish would be compensated partially by the decreases in length for remaining fish, then the effective increase in mass would be more on the order of 40 percent if actual consumed fish lengths varied over a range of 50 percent of the reported length. We would be pleased to share the details of this analysis with Paul White if requested.

It is also possible that respondents provided lengths in the form of modes rather than averages, i.e., the reported "average" lengths are actually the most commonly eaten length rather than a true average of lengths. If this were the case, then the number of fish consumed that were of different lengths than the reported "average" would be relatively small.

Potential for Suppression of Freshwater Angling and Consumption Due to Marine Alternative

Paul White also raised the question of whether freshwater fish consumption rates were low in Maine due to the presence of the marine angling and consumption alternative. Because the 1990 Maine Angler Survey did not ask questions regarding marine angling practices, this question is difficult to answer directly. However, freshwater angling is extremely popular in Maine. In 1990, Maine issued freshwater fishing licenses to 203,160 residents. Assuming that 75 percent of those resident licensees are male (per 1990 Maine Angler Survey results), then approximately half of Maine's 297,387 males over 18 (1990 data) are licensed anglers. Furthermore, because there are bountiful suitable locations statewide, consumption of freshwater fish would likely be limited only by angler skill, angler avidity, and presence of desirable species.

Certainly, marine fish species are widely available in markets across the state, but no data are available on consumption rates for self-caught fish. It is also not the case that marine fish are readily caught at shore-based locations in Maine's major population centers. While the Maine Department of Marine Resources does not have data for participation rates in marine angling, anecdotally it is believed that in the more densely populated southern part of the state, marine anglers are attracted to charter boat fishing, while most areas north of Rockland are popular dock-fishing sites (personal communication, Lt. LaHaye, Maine Department of Marine Resources).

We sincerely trust that these additional analyses and our discussion of the points you and your colleagues raised concerning the Maine Angler Survey have addressed your questions. Please contact us if additional clarification is needed.

Sincerely,

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Vice President
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cc: Jackie Moya
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John Haggard
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Angus Macbeth
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Dorothy Canter
Douglas Tomchuk
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Table 1

Comparison of Angling Behavior Between Consumer Groups: All Waters

	Consumed Fish at <90th Percentile Rate	Consumed Fish at ≥90th Percentile Rate
Number in Group	940	113
Median Number of Ice Fishing Trips	0	10
Median Number of Rivers/Streams Fishing Trips	2	7
Median Number of Ponds/Lakes Fishing Trips	6	15
Median of Average Days Spent Fishing at Preferred Locations	5	7
Median of Average Distance Traveled to Preferred Fishing Locations (miles)	30	30

Table 2

Comparison of Angling Behavior Between Consumer Groups: Rivers/Streams

	Consumed Fish at <90th Percentile Rate	Consumed Fish at ≥90th Percentile Rate
Number in Group	418	46
Median Number of Ice Fishing Trips	1	0
Median Number of Rivers/Streams Fishing Trips	6	12
Median Number of Ponds/Lakes Fishing Trips	10	22
Median of Average Days Spent Fishing at Preferred Locations	5	8
Median of Average Distance Traveled to Preferred Fishing Locations (miles)	29	27

Table 3

Demographic Comparison Between Consumer Groups: All Waters

	Consumed Fish at <90th Percentile Rate	Consumed Fish at ≥90th Percentile Rate
Number in Group	940	113
Percent Male	78	83
Median Age	41	38
Modal[a] Employment Status	Employed Full-Time (62%)	Employed Full-Time (60%)
Percent Seasonally Employed	4	6
Percent Unemployed	3	3
Percent Retired	15	15
Modal[b] Educational Attainment	High School Graduate (32%)	High School Graduate (39%)
Percent White	89	86
Percent Native American	9	13
Percent with Household Income Less than $10,000 per year	8	15
Median Annual Household Income	$30,000-$39,999	$20,000-$29,999

a. Of eight possible response categories for this variable, the modal group is the group with the largest representation among the response categories. The percentage refers to the relative size of this group.

b. Of nine possible response categories for this variable, the modal group is the group with the largest representation among the response categories. The percentage refers to the relative size of this group.

Table 4

Demographic Comparison Between Consumer Groups: Rivers/Streams

	Consumed Fish at <90th Percentile Rate	Consumed Fish at ≥90th Percentile Rate
Number in Group	418	46
Percent Male	86	76
Median Age	38	37
Modal[a] Employment Status	Employed Full-Time (69%)	Employed Full-Time (63%)
Percent Seasonally Employed	4	11
Percent Unemployed	2	2
Percent Retired	11	13
Modal[b] Educational Attainment	High School Graduate (32%)	High School Graduate (35%)
Percent White	89	85
Percent Native American	9	15
Percent with Household Income Less than $10,000 per year	6	17
Median Annual Household Income	$20,000-$29,999	$20,000-$29,999

a. Of eight possible response categories for this variable, the modal group is the group with the largest representation among the response categories. The percentage refers to the relative size of this group.

b. Of nine possible response categories for this variable, the modal group is the group with the largest representation among the response categories. The percentage refers to the relative size of this group.

Table 5

Knowledge About and Reactions to Fish Consumption Advisories Between Consumer Groups: All Waters

	Consumed Fish Rate <90th Percentile	Consumed Fish Rate ≥90th Percentile
Number in Group	940	113
Percent Aware of Advisories	38	46
Percent of Those Aware Who Fished at an Advisory Location	27	40
Percent of Those Aware for Whom Advisories Affect Whether they Eat Fish from Advisory Locations	82	81
Percent of Those Aware Who Would Fish Additional Waters in Absence of Advisories	20	30

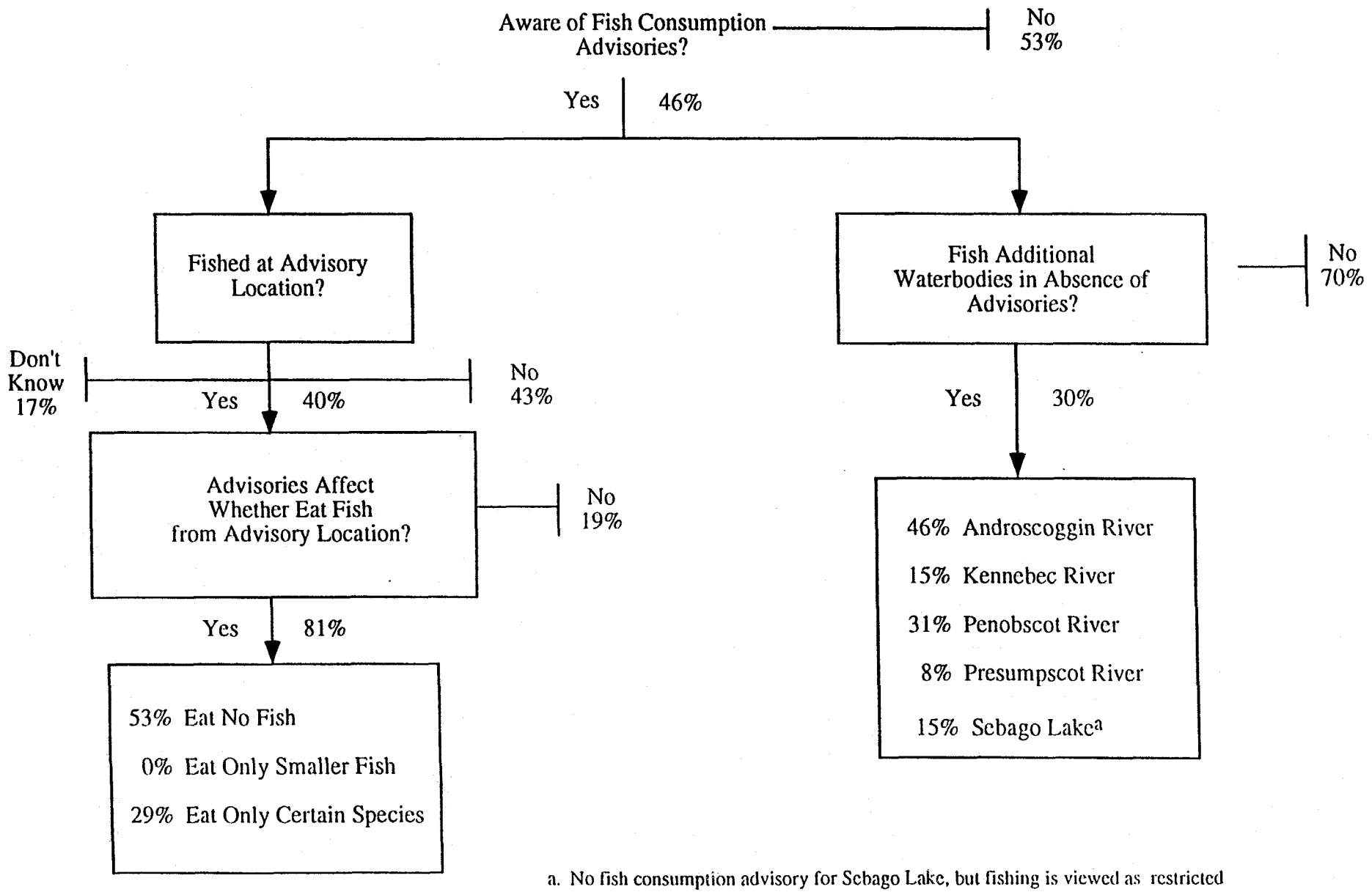
Table 6

Knowledge About and Reactions to Fish Consumption Advisories Between Consumer Groups: Rivers/Streams

	Consumed Fish Rate <90th Percentile	Consumed Fish Rate ≥90th Percentile
Number in Group	418	46
Percent Aware of Advisories	42	52
Percent of Those Aware Who Fished at an Advisory Location	30	50
Percent of Those Aware for Whom Advisories Affect Whether they Eat Fish from Advisory Locations	78	100
Percent of Those Aware Who Would Fish Additional Waters in Absence of Advisories	22	32

Figure 1

Awareness of and Behavior Responses to Fish Consumption Advisories
High Consumers from All Waters

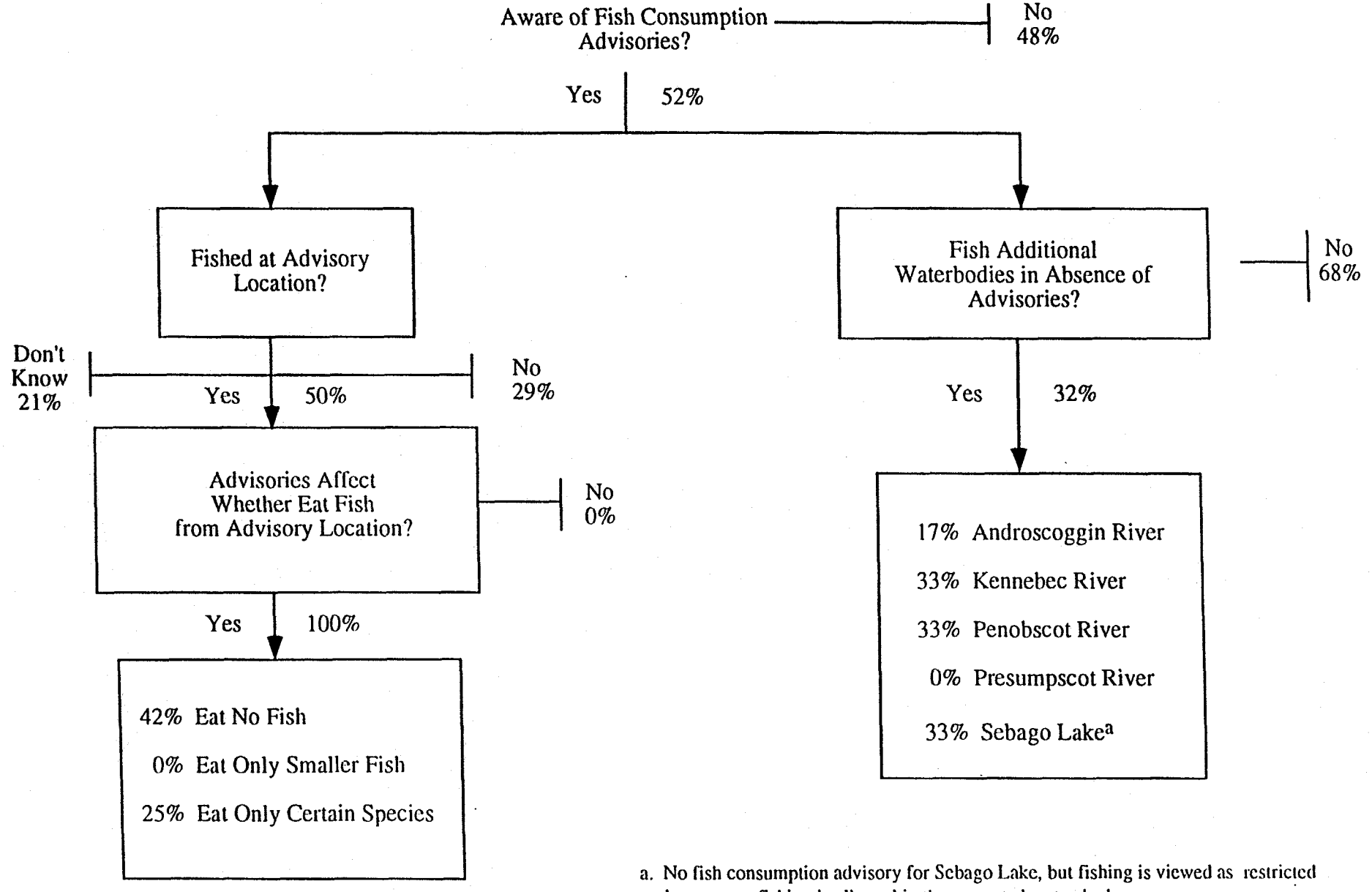


a. No fish consumption advisory for Sebago Lake, but fishing is viewed as restricted because no fishing is allowed in the protected watershed area.

10.2080

Figure 2

Awareness of and Behavior Responses to Fish Consumption Advisories
High Consumers from Rivers/Streams



a. No fish consumption advisory for Sebago Lake, but fishing is viewed as restricted because no fishing is allowed in the protected watershed area.

10.2081

Figure 3

Rating of Site Characteristics Important to Fishing Location Choice Among High Consumers of All Waters Fish Consumers

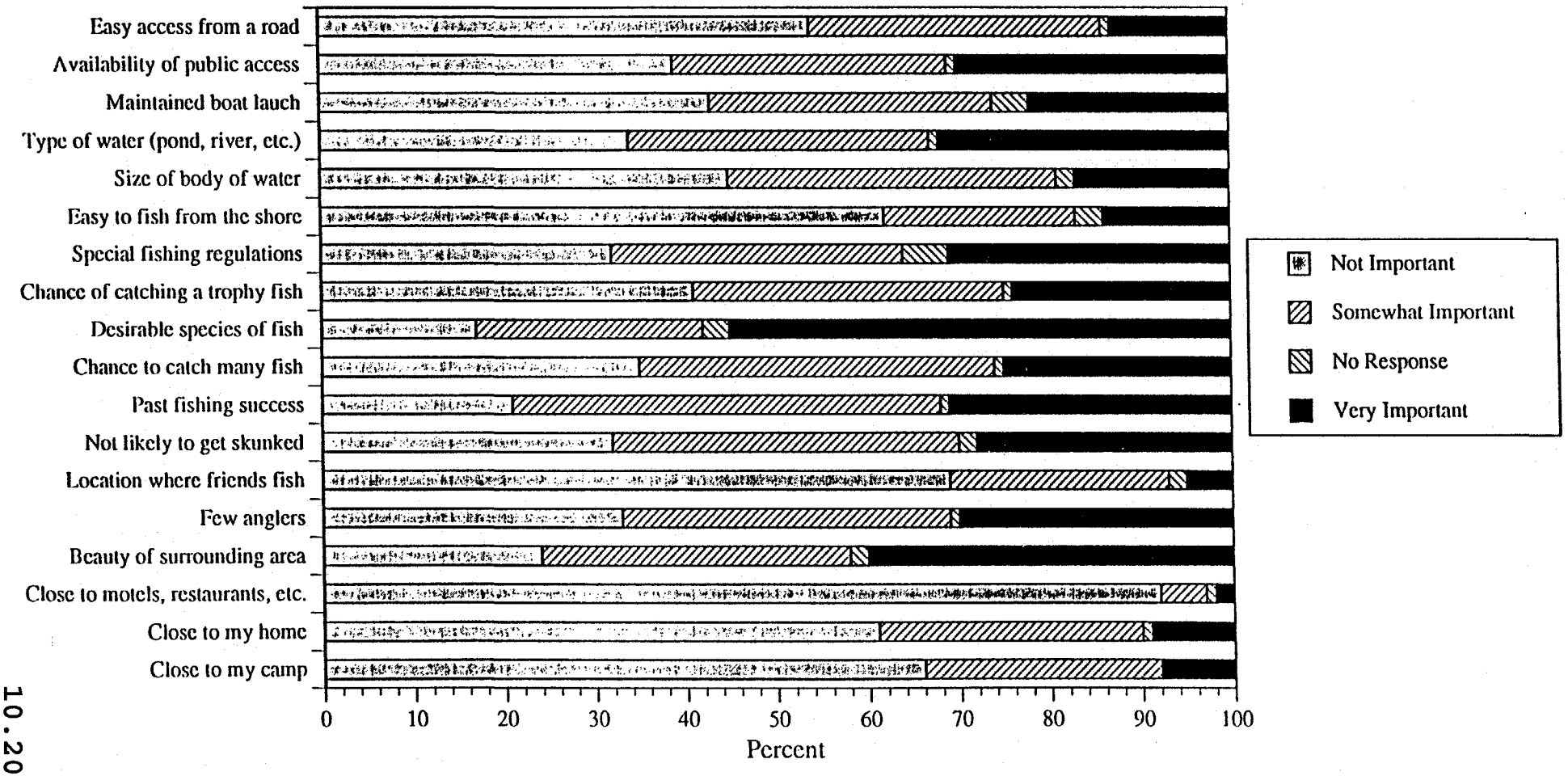


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Figure 4

Rating of Site Characteristics Important to Fishing Location Choice Among High Consumers of Rivers/Streams Fish Consumers

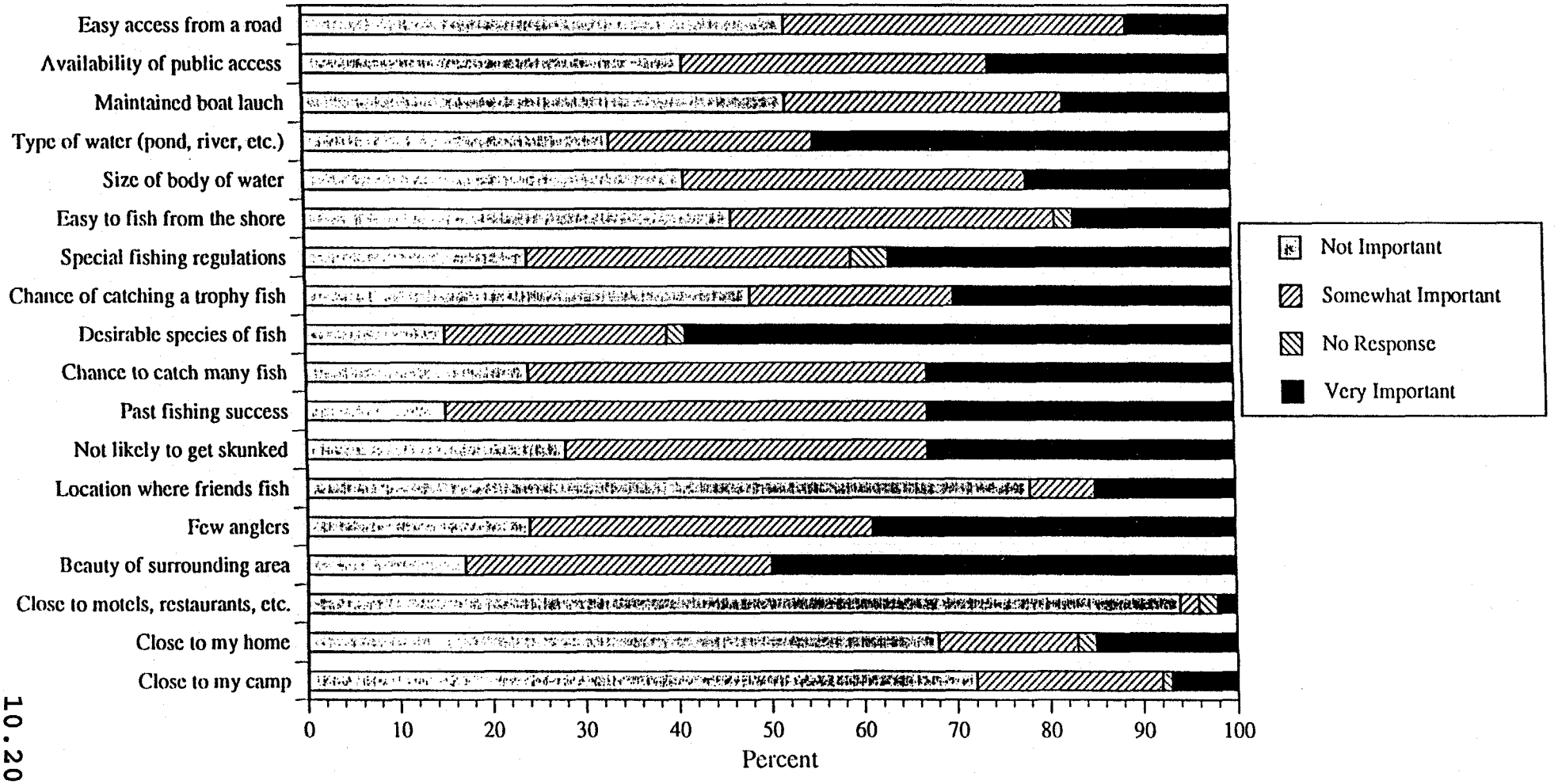

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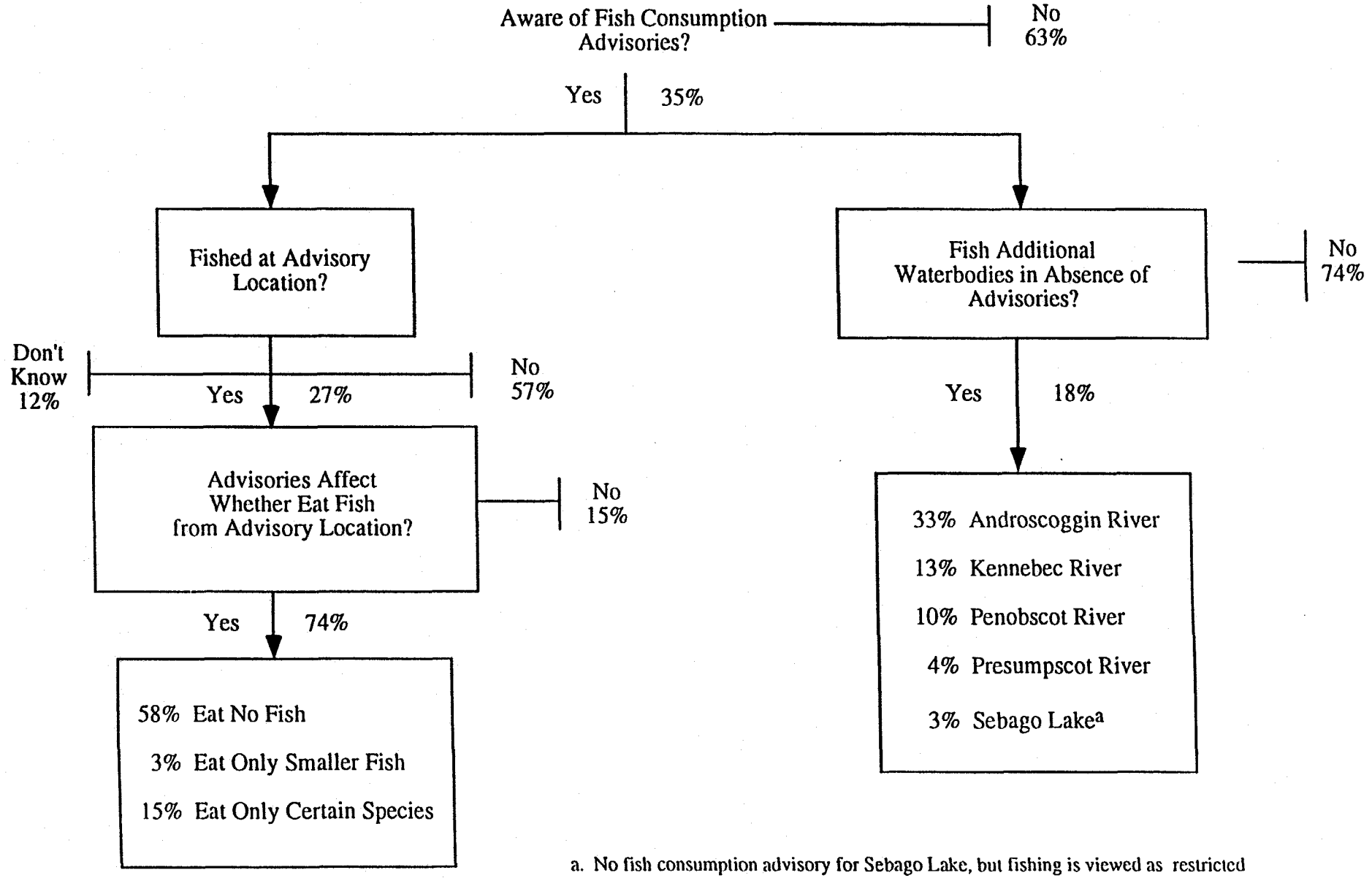
Figure 5

Awareness of and Behavior Responses to Fish Consumption Advisories
All Survey Respondents



a. No fish consumption advisory for Sebago Lake, but fishing is viewed as restricted because no fishing is allowed in the protected watershed area.

10.2084

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APPLICATION OF QUANTITATIVE INFORMATION ON THE UNCERTAINTY IN THE RFD TO NONCARCINOGENIC RISK ASSESSMENTS

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FOR SUBMISSION TO

HUMAN AND ECOLOGICAL RISK ASSESSMENT

Accepted for Publication

ABSTRACT

Recent efforts to improve risk assessment methodologies have sought to provide a fuller representation of the variability and uncertainty in risk estimates in order to provide risk managers with a more complete description of risks. Recently, we and others (Swartout *et al.*, 1998; Price *et al.*, 1997; Slob and Pieters, 1997; Baird *et al.*, 1996) have proposed approaches to characterize the uncertainty in the reference dose, (RfD) a key component of the non-carcinogenic risk estimation process. The operational definition of the RfD as the "lower-bound" estimate of the NOAEL in a sensitive human subpopulation ($NOAEL_{HS}$) is used along with information on the inter-chemical variation in ratios associated with the uncertainty factors used in setting the RfD to characterize the uncertainty in the $NOAEL_{HS}$ (Swartout *et al.*, 1998). This paper presents a description of how information on the uncertainty in the $NOAEL_{HS}$ can be used to characterize the uncertainty and variability in estimates of Hazard Quotients (HQ) and Hazard Indices (HI) for a population. The paper also explores the impact of using alternative approaches for defining inter-chemical variation in the ratios. The benefits of characterizing the uncertainty in noncancer toxicity estimates as well as limitations of the proposed approach are discussed. The analysis suggests four findings. First, the current method of estimating risks from mixtures of chemicals may overestimate the true HI when two or more compounds contribute significantly to the index. Second, the probability of a dose in excess of the RfD exceeding the $NOAEL_{HS}$ depends upon the number of UFs used in deriving the reference dose. Third, jointly assessing both the uncertainty and variability in exposure and the uncertainty in the estimate of the $NOAEL_{HS}$ can have a significant impact on the characterization of

noncarcinogenic risks. Finally, the findings remain generally consistent when various estimates of inter-chemical variation in ratios used.

KEY WORDS

Uncertainty, noncancer risk assessment, Monte Carlo, variability, reference dose, exposure

1.0 INTRODUCTION AND BACKGROUND

Environmental risk assessment is a field of pervasive uncertainty. Over the last ten years, risk analysts have begun to investigate the sources of uncertainty in risk assessment and their effect on risk estimates, especially uncertainties relating to exposure estimates (McKone and Bogen, 1992; Finley and Paustenbach, 1994; Thompson *et al.*, 1992; Price *et al.*, 1996; Baird, *et al.*, 1996). Recently, Swartout *et al.* (1998) have proposed a framework for evaluating non-carcinogenic risks. Under this framework, the RfD is defined as the lower confidence limit in an estimate of a minimum risk level. This minimum risk level is conceptually defined as the NOAEL in a sensitive human subpopulation ($NOAEL_{HS}$). The value of the $NOAEL_{HS}$ for a substance is estimated based on the application of a series of ratios of toxicological endpoints that convert or scale the reported NOAEL or LOAEL in the data for a compound to the $NOAEL_{HS}$. These ratios are associated with the uncertainty factors historically used in setting the RfD (Swartout *et al.*, 1998). This paper examines how quantitative representations of the uncertainty in a noncancer $NOAEL_{HS}$ (Swartout *et al.*, 1998; Price *et al.*, 1997) can be used, alone or in conjunction with uncertainty in exposure estimates, to quantitatively characterize the uncertainty in estimates of noncancer risks[1]. Two hypothetical case studies are presented in which quantitative estimates of the uncertainty in $NOAEL_{HS}$s are used to generate information on the uncertainty in noncancer risk characterizations. The results of these case studies are compared to results obtained using the RfD.

[1] Although the HQ is not really an estimate of risk (i.e., probability of effect), the term risk will be used in this paper as it is consistent with USEPA terminology.

The current system of evaluating noncarcinogenic risk is essentially a comparison of the estimated dose to the RfD (USEPA, 1989)[2]. Such a comparison is used by the risk manager to ascertain whether the exposure is above a dose which is unlikely to result in "adverse" or "deleterious" effects (a dose less than the RfD) or one judged to have some potential to cause an adverse effect (a dose greater than the RfD).

The comparison of the dose and the RfD is expressed in terms of a Hazard Quotient (HQ) (Stara, *et al.*, 1987). The HQ is defined as the ratio of the dose resulting from exposure to a single chemical to the RfD[3].

$$HQ_i = D_i/RfD_i \qquad \text{Eq. 1}$$

where D_i is the dose of chemical *i* and RfD_i is the RfD for chemical *i*. Under this system, an HQ that exceeds a value of 1.0 indicates that the estimated dose is greater than the RfD. The HQ ratio is designed to provide a common measure of relative risk across chemicals and exposure scenarios that is independent of the specific value of the RfD. This approach is intended to provide consistency for risk managers faced with evaluating exposures involving different chemicals with different toxicities. The approach also provides a useful basis for evaluating risks from exposure to mixtures of chemicals or from simultaneous exposure to multiple chemicals (USEPA, 1989; USEPA, 1986). Cumulative

[2] This paper will use the term dose to refer to dose rate (mg/kg-day).

[3] The RfD is expressed in terms of milligrams of chemical per kilogram body weight per day (mg/kg-day).

risks from exposure to multiple chemicals that elicit the same adverse effects and share a common mode of action are calculated using the Hazard Index (HI). The HI is calculated by Equation 2.

$$HI = \sum HQ_i = \sum D_i/RfD_i \qquad \text{Eq. 2}$$

where D_i is the dose of the ith chemical in the mixture and RfD_i is the RfD for the ith chemical. The advantage of this approach is that a single risk metric is developed for exposures to multiple chemicals. For example, the USEPA (1990) and several state agencies (MDEP, 1996; 58 N.J. Rev. Stat., 1997) have stated that all values of HI and HQ greater than 1.0 represent unacceptable levels of risk.

A number of researchers have identified limitations with the current system. These limitations include:

- the values of HQ and HI cannot be converted to quantitative estimates of the probability of adverse effects (Renwick and Walker, 1993; USEPA, 1993);

- there is no assurance that the risks associated with HI or HQ estimates exceeding a value of one will be the same for different chemicals (Renwick and Walker, 1993; USEPA, 1993);

- the combination of upper-bound estimates of risk (or, here, lower confidence limits of the estimate of NOAELs in sensitive populations (Swartout *et al.*, 1998) across multiple

chemicals) misrepresents the uncertainty in the resulting combination (Putzrath and Ginevan, 1991; Putzrath and Ginevan, 1994; Gaylor and Chen, 1996).

In fact, in its Guidelines for the Health Risk Assessment of Chemical Mixtures (USEPA, 1996), U.S. EPA recommended that the uncertainty in the toxicity assessment should be carried through to the risk characterization, stating:

> Nonetheless, if sufficient data are available to derive individual acceptable levels for a spectrum of effects ..., or variabilities of the acceptable levels are known, or if the acceptable levels are given as ranges ..., then the hazard index should be presented with corresponding estimates of variation or range.

As discussed above, the RfD can be viewed as the lower confidence limit on the estimate of the $NOAEL_{HS}$ for a chemical (Swartout *et al.*, 1998). The uncertainty in the $NOAEL_{HS}$ can be quantified using the current equation for RfD derivation and replacing the point estimates of the uncertainty factors with distributions. Probabilistic techniques are used to simulate the resulting distributions of $NOAEL_{HS}$s. These distributions of the $NOAEL_{HS}$s reflect the uncertainty that stems from the lack of complete knowledge as to the true (but unknown) value of the $NOAEL_{HS}$ for a chemical.

When distributions of $NOAEL_{HS}$s are used in the place of the RfD in equations 1 and 2, the results are estimates of the probability that a dose of one chemical or a mixture of chemicals will be greater

than the true $NOAEL_{HS}$ for the chemical or mixture of chemicals. The estimates of dose in equations 1 and 2 can also be replaced with distributions that reflect the variability and uncertainty in the dose estimates (McKone and Bogen, 1992; Finley and Paustenbach, 1994; Thompson *et al.*, 1992; Price, *et al.*, 1992). Using Monte Carlo models of such equations, risk assessors can characterize the probability that portions of an exposed population are exposed at doses above the true but unknown $NOAEL_{HS}$ given the uncertainty in both the toxicity and dose components of the equation.

2.0 APPROACH

The impact of quantitative measures of dose-response uncertainty on the assessment of noncancer hazards was investigated using a series of case studies. These case studies were designed to assess the effect of applying probabilistic $NOAEL_{HS}$s on the following issues. First, how does the number of compounds, and the uncertainty in their individual HQs affect the uncertainty in the HI for a chemical mixture? Second, how is the uncertainty in an HI for an exposure to a mixture of chemicals affected by the number of uncertainty factors used in setting the RfDs for the compounds of the mixture RfD? And finally, how can information of the uncertainty in $NOAEL_{HS}$s be combined with information on variability and uncertainty in dose to characterize the uncertainty and variability in HQs?

The input variables for the case studies are displayed in Table I. A single "reference" distribution was used to represent each uncertainty, as proposed by Swartout et al. (1998). This three-parameter

lognormal distribution has a mean of 0.335, standard deviation of 0.3765 (both expressed as the logarithm to the base 10), and offset value of one. Further discussion of the basis for the reference distribution is provided by Swartout *et al.* (1998). The simulations were run in a Microsoft Excel spreadsheet (v 5.0, Microsoft™ Corporation, 1994) with the @RISK™ add-in (v 3.0, Palisade Corporation, 1994). The number of iterations was selected to achieve stability of ± 3% in the 97.5th percentile of the input distributions using Latin Hypercube sampling. For all cases except case 1.3, stability was reached at 10,000 iterations, while case 1.3 required 15,000 iterations.

3.0 CASE STUDIES

The uncertainty in the estimate of the $NOAEL_{HS}$ is a function of the number of uncertainty factors used in its derivation (Swartout *et al.*, 1998). As a result, compounds with the same RfDs but different numbers of uncertainty factors will have different uncertainty distributions for the $NOAEL_{HS}$. To explore this issue, Case Study 1 considers mixtures of chemicals with RfDs derived using varying numbers of uncertainty factors. In this paper an RfD established with a smaller number of uncertainty factor will be referred to as more "certain" than an RfD with a larger number of uncertainty factors.

Recent trends in risk assessment have been to move toward the quantitative assessment of uncertainty and variability (Price *et al.*, 1996; USEPA, 1992; USEPA, 1995; Frey, 1993; Bogen, 1995; Hoffman and Hammond, 1994). The availability of probabilistic $NOAEL_{HS}$s allows the joint analysis of variability in dose and uncertainty in dose and toxicity. Case 2 investigates the application of

probabilistic $NOAEL_{HS}$s to a distribution of HQs when confidence limits are specified for the distribution of doses.

Uncertainty in the HQs was estimated by substituting distributions for the UFs used in deriving each RfD; the resulting quantity is the uncertainty distribution for the $NOAEL_{HS}$ and was calculated as:

$$NOAEL_{HSi} = \frac{NOAEL_i}{\Pi UF_j} \qquad \text{Eq. 3}$$

where $NOAEL_i$ is the NOAEL for the ith chemical each UF_j is represented by a distribution.

Case 1. Calculation of HIs for Chemical Mixtures Using Uncertainty Distributions of $NOAEL_{HS}$ and Point Estimates of Dose

In this case study a series of examples, Cases 1.1 to 1.3, examine the effect of probabilistic $NOAEL_{HS}$s on estimates of noncancer risk from exposure to multiple chemicals presumed to act by the same mechanism. Case 1.1 consists of mixtures in which the individual constituents, each with RfDs derived using the same number of UFs, contribute equally to the overall HI (i.e., HQs for all constituents are equal). Case 1.2 consists of a mixture in which a single constituent dominates the HI (the HQ for one chemical exceeds the HQs for the remaining constituents), but where the RfDs are again derived using the same number of UFs. Finally, Case 1.3 consists of mixtures in which different numbers of UFs are used in the derivation of RfDs in the mixture (*i.e.*, the RfD for the first constituent uses one UF while the RfD for another uses more than one). These three cases show how

the information on the uncertainty in the $NOAEL_{HS}$ can provide insight to risk managers on which constituents are of primary concern.

Case 1.1

Case 1.1 examines two mixtures (1 and 2) composed of two and five chemicals, respectively. The values of D_i and RfD_i (Eq. 2) components were defined such that summing across the HQs for each of the mixtures results in an HI of 1.0. In all three mixtures, the HQs for the individual constituents are equal. For example, in Mixture 1, each of 2 constituents has an HQ of 0.5; in Mixture 2, each of 5 constituents has an HQ of 0.2.

Figure 1 shows the distributions of uncertainty in the HIs for the three mixtures, using a box-and-whiskers style representation. This graph presents the mean and 2.5th, 5^{th}, 25^{th}, 50^{th}, 75^{th}, 95^{th}, and 97.5th percentiles. The graph depicts the uncertainty in the HQs for each of the chemicals. This uncertainty can be thought of as the probability of the dose D_i exceeding the $NOAEL_{HS}$. A distribution for the HQ for a single chemical is also provided for comparison. For all three of the distributions, there is less than 2.5% probability that the HI exceeds 1.0 (see Figure 1) with the given number of model iterations. Summing HQs across greater numbers of chemicals (all with equivalent point estimates of HQs) results in greater disparity between the point estimate HI and the distribution of HIs. In Mixture 1, for example, the 97.5th percentile HI resulting from the stochastic combination of HQs is 0.5. For Mixture 2, the 97.5th percentile HI is only 0.4. As the number of constituents in

the mixture increases, the upper end HI continues to decrease, since the joint probability of selecting upper-bound HQs (from the tails of the distributions) for all chemicals is smaller.

Case 1.2

Case 1.2 examines a mixture where several constituents contribute to a total HI, but the contribution of one constituent is dominant. In Mixture 3, the HQ for Chemical A is 0.5 and the HQ for each of the remaining chemicals (B - E) is 0.125. As shown in Figure 1, the uncertainty distribution for this mixture has a 97.5th percentile of 0.5. Thus, where one chemical dominated the HI, the upper confidence limit of the estimate of the HI was increased.

Case 1.3

The results given in Case 1.2 are based on constituents with RfDs that are derived using two UFs. When RfDs for mixture constituents have varying levels of certainty (that is, different numbers of uncertainty factors), there can be a change in both location and shape of the distributions of HIs.

Case 1.3 demonstrates that the chemical with fewer uncertainty factors will contribute more to the HI when the mixture constituents have RfDs of varying certainty. Mixture 4 is composed of three chemicals, each having HQs of one. The RfD for Chemical A is derived using only one UF. The RfD for Chemical B is derived using two UFs. Figure 3 shows the distributions of HQs that result from

a Monte Carlo simulation of the uncertainty in the $NOAEL_{HS}$s for each compound. As the figure shows, the HI is dominated by the contribution from Chemical A. Although the point estimate HQ for each chemical is 1.0, the distribution of HQs for Chemical A is greater than that for Chemical B. In fact, the 95th percentile of the distribution for Chemical A is 1.0, while the distribution for Chemical B does not reach 1.0 until above the 97.5th percentile.

For chemicals whose RfDs include more than two UFs, the disparity can be even greater. Figure 2 also shows the distributions of HQs of two chemicals for (Mixture 5). The RfD for Chemical A incorporates only one UF, while the RfD for Chemical B is based on three UFs. This figure illustrates how the addition of more factors reduces the importance of the less certain chemical in the determination of the total HI for the mixture. Table II summarizes the results of Case 1.

Case 2. Incorporation of Uncertainty in $NOAEL_{HS}$ and Uncertainty in Doses into Noncancer Risk Estimates

Case 2.1

Case Study 2.1 examines the impact of using a probabilistic $NOAEL_{HS}$ in the noncancer risk characterization associated with a distribution of doses representing the uncertainty in the dose to a randomly selected individual from an exposed population. The distribution of HQs for the randomly selected individual is first calculated by applying the RfD to the distribution of dose rates. The result,

shown in Figure 3, is that there is 5% probability that the randomly selected individual has an HQ greater than 1.0. A second distribution of HQs is calculated from a Monte Carlo model of equation 1, where the distribution of doses and $NOAEL_{HSS}$ were based on the uncertainty in the dose to an individual and the uncertainty in the $NOAEL_{HS}$. In this case, the fraction of the model runs showing HQs greater than 1.0 is less than 2.5%.

This finding, however, must be interpreted carefully. The result of this case study is an expression of the probability that a *randomly selected* individual has an HQ greater than one. This should not be interpreted to mean that an individual at the 97.5th percentile of the dose distribution has an HQ of 1.0. What the analysis demonstrates is that the consideration of the uncertainty in the $NOAEL_{HS}$ results in a reduction in the estimate of the HQ for randomly selected individuals from an exposed population.

Case 2.2

In Case 2.2 the uncertainty associated with the $NOAEL_{HS}$ is combined with the uncertainty in dose by means of a Monte Carlo model of the total uncertainty in the HQ for various percentiles of a dose distribution for an exposed population. Unlike Case 3.1, the distribution of doses received by a population is expressed in terms of both variability and uncertainty. In this example both the dose and the uncertainty distributions were assumed to be lognormal, with geometric mean, geometric

standard deviation of 0.007, 2.3 (dose) and 1.0, 5.0 (uncertainty in dose). A hypothetical chemical was postulated with animal NOAEL of 2.7 mg/kg-day and RfD derived with two uncertainty factors.

The uncertainty and variability associated with the HQ distribution is assessed in two ways. First, the analysis is conducted using the point-estimate RfD, resulting in a two-dimensional distribution representing both variability and uncertainty in the dose component of the HQ[4]. The distribution of interindividual variability in exposure in this example is the same as the uncertainty in the dose to a randomly selected individual used for Case 3.1; thus, an HQ of one occurs at the median estimate of the uncertainty of the dose to individuals in the 95th percentile of exposure (variability). The second assessment takes into consideration the uncertainty in the $NOAEL_{HS}$, resulting in a two-dimensional distribution representing variability in exposure and uncertainty in both the exposure and toxicity components of the HQ.

The probabilistic $NOAEL_{HS}$ was applied in the following manner. It was assumed that the total uncertainty in the estimate of the HI for each of the percentiles is a function of the uncertainty in the $NOAEL_{HS}$ and the uncertainty in the dose for that percentile. A Monte Carlo analysis was performed that calculated the total uncertainty in the HQs for each percentile of the exposed population using

[4] In order to simplify the analysis, we have assumed that the degree of uncertainty is constant across all percentiles of the exposed population. A more thorough analysis could address the uncertainty in the parameters of the dose distribution through the use of a nested-loop Monte Carlo analysis (Hoffman and Hammond, 1994) however, this approach was not necessary for the purposes of this analysis.

the uncertainty distributions for the $NOAEL_{HS}$ and the uncertainty distribution for the dose of each percentile of the exposed populations. This resulted in the generation of a two-dimensional model of HQs for the exposed population.

Figure 4 presents these two characterizations of the distributions of HQs. The first is the estimate of the HQs that result from the application of the point-estimate RfD to the two-dimensional (variability and uncertainty) model of doses. The second reflects the combined uncertainty in both the $NOAEL_{HS}$ and the variable dose-rate estimates. In both cases the outer two curves can be considered to represent upper and lower 95% confidence limits (UCL and LCL) of the distribution of HQs for the exposed population. The middle curves represent the median estimates, that is, estimates that have an equal probability of under-estimating or over-estimating the true value of the HQs for the population. In the first example, where the RfD is used, the median distribution indicates that 5% of the population has HQs equal to or greater than 1.0. However, the UCL on this distribution suggests that 95% of the population could have an HQ of 1.0 or greater.

The second example gives a different result. As Figure 4 shows, the curves for the second example are shifted downward, and, as expected, the uncertainty bands are expanded. In this analysis, the 95th percentile of the median distribution is much less than one (0.1). Further, the UCL on the distribution indicates that 30% of the population has an HQ equal to or greater than 1.0.

4.0 DISCUSSION

The use of probabilistic $NOAEL_{HS}$s provides a number of insights into the assessment of noncarcinogenic risks. The example in Case 1.1 (Fig 1.) demonstrates that the current methodologies used to evaluate mixtures have an inherent conservative bias. When the HQs for two or more chemicals in a mixture make important contributions to the HI for the mixture, there is a potential to overestimate risk by a factor of two or more. This occurs because there is a very low probability that the true $NOAEL_{HS}$ for each of the compounds will be as low as the estimates of the RfD. The potential for overestimation increases with the number of compounds in the mixture.

Case 1.2 (Fig. 1) demonstrates two points. First, it is evident that the point-estimate approach to characterizing the HI of a mixture may provide a reasonable measure of hazard for mixtures where one constituent dominates the point-estimate HI. Second, the HI distribution will likely reflect the distribution for the dominant constituent *unless the RfD for one constituent is more certain than the others.*

As shown in Case 1.3 (Fig. 2), where RfDs may vary in the certainty of their derivation and all other factors are equal, the chemical with the more certain RfD will dominate the HI. This result implies that giving equal weight to HQs for chemicals with less uncertain RfDs and more uncertain RfDs can bias risk management decisions. (Finkel, 1990) noted that comparisons between outputs subject to hidden levels of conservatism can be precarious when "some real cases are less like the hypothetical

'worst cases' than others are". Such is the case in this example. The point estimate HQ for Chemical A and B indicates that they are equally hazardous. The probabilistic HQ assessment, however, suggests that Chemical B contributes far less to the combined hazard in both mixtures (Mixtures 5-6) than does Chemical A. While current U.S. EPA guidance recommends that the uncertainty in toxicity values be discussed qualitatively in risk assessment (Renwick and Walker, 1993), that guidance does not give risk managers sufficient information to evaluate the magnitude of the uncertainty or to acknowledge the uncertainty in making decisions regarding remediation. With the approach presented in Case 1.3, a risk manager is given additional information suggesting that in both mixtures, Chemical A, whose RfD is most certain, poses a greater hazard than Chemical B at the doses modeled.

Case 2 shows how information on the uncertainty in the $NOAEL_{HS}$ can be directly incorporated into a two-dimensional uncertainty analysis. Further, this example (Fig. 4) provides a visual perspective on both sources of uncertainty in the HQ (exposure and toxicity) as well as the magnitude and direction of uncertainty in the $NOAEL_{HS}$. This analysis is perhaps the most significant in this paper. The criterion for concern for non-carcinogenic effects is the probability that an individual at a site will receive a dose that has some potential for causing adverse effects. Traditionally this has been defined as doses that are more than the RfDs for the relevant compounds ($HQ \geq 1$). In Swartout et al. (1998), the RfD was defined as a lower bound estimate of the $NOAEL_{HS}$ associated with any given chemical exposure. This suggests that a more useful measure of the potential for noncarcinogenic risk is the probability that an individual will receive a dose of a chemical (or mixture of chemicals) that is greater

than the actual $NOAEL_{HS}$ for the compound (or mixture) in the sensitive population. This probability is determined by both the uncertainty in the individual's dose and the uncertainty in the $NOAEL_{HS}$. The characterization of the uncertainty in the $NOAEL_{HS}$, and the combination of this uncertainty with uncertainty and variation in dose estimates presented in this study are steps in characterizing that probability. Using the approaches outlined in this paper, risk assessors can provide managers with estimates of the probability that exposed individuals or fractions of exposed populations will have doses more than the $NOAEL_{HS}$s.

The foregoing results were derived using an uncertainty distribution for UF that is largely based on a specific interpretation of the probabilistic nature of uncertainty factors and not on empirical or mechanistic relationships (Swartout *et al.*, 1998). As a result, the above are relevant only in the context of probabilistic inferences arising from the application of the existing RfD methodology and do not necessarily have biological significance. Furthermore, the conclusions apply only in those situations where the full 10-fold default uncertainty factors are used in the derivation of the RfD. Situations of reduced uncertainty, in which uncertainty factors less than the 10-fold default are used, require the use of modified reference distributions (Swartout *et al.*, 1998).

Recently, Baird *et al.*, 1996; Swartout *et al.* (1997), Schmidt, *et al.*, (1997); and Slob and Pieters (1997) have proposed alternative uncertainty distributions for one or more uncertainty factors. Certain of these factors are based on empirical data. In order to evaluate the effect of alternative distributions on the analysis presented herein, these preliminary uncertainty distributions were used

in Case 1.3, Mixture 5 and the results compared with those observed with the reference uncertainty distribution. Uncertainty factors for interindividual variability, interspecies extrapolation, and subchronic-to-chronic extrapolation were included in the comparison. Since both (Baird *et al.*, 1996 and Schmidt *et al.*, 1997) presented species-specific interspecies distributions, the rat was selected as the test species for the hypothetical compounds.

Table III shows a comparison of the median and 95th percentile HQs and HIs resulting from the use of the reference and alternative uncertainty distributions. As the table demonstrates, where several uncertainty distributions combine (e.g., Chemical B), the results can vary depending upon the uncertainty distribution used. For example, the 95th percentile HQ for Chemical B is 0.15 using the Slob and Pieters (1997) distributions, but is estimated to be 0.75 using the Baird et al. (1996) distributions. Despite this difference, the qualitative results remain consistent within a given set of distributions; Chemical A presents a greater hazard than Chemical B despite their nominally equal point estimate HQ. These results, based on a limited set of alternative distributions, suggest that different quantitative and qualitative interpretations can arise from a alternative uncertainty distributions. There is no clear indication, however, of the eventual impact of data-derived distributions on the interpretation of HQ distributions, either in magnitude or direction.

5.0 CONCLUSIONS

This paper demonstrates how the uncertainty in the $NOAEL_{HS}$ can be incorporated into noncancer risk assessment. The example analyses presented herein show that quantitative uncertainty analysis can lead to risk management decisions that differ from decisions based on point estimates of hazard. In addition, the analysis shows that the uncertainty in the $NOAEL_{HS}$ can be quantitatively incorporated into a two-dimensional analysis of variability and uncertainty to provide information on the significant sources of uncertainty in noncancer hazard estimates.

The approach to noncancer risk assessment presented here is limited in that it is not designed to address the probability of effects at doses exceeding the RfD or $NOAEL_{HS}$. Unlike cancer risk assessment, current noncancer risk assessment is centered around an evaluation of whether an estimated exposure exceeds a "bright line" criterion (HI or HQ > 1.0). Thus, the approach presented here does not differ from current methods of assessing noncancer risks in this regard, but rather, provides a means of characterizing the probability of exceeding the" bright line" test.

This analysis shows that the quantitative assessment of uncertainty in RfDs can provide additional information which may be of use in risk management decision making. One example is the finding in Case 1 that an HI in excess of 1.0 for certain mixtures may be associated with lower potential for risk than a finding of an HQ of less than 1.0 for single compounds. An additional example is the potential to use information on the uncertainty in the HI or HQ in risk-risk comparisons. For

example, if a risk manager was comparing risks of radiation and the noncancer risks from chemicals it may be appropriate to use the most likely estimate of HQ or HI in comparison since the estimates of radiation risks are best estimates and not upper bounds. The proposed approach relies upon the current system of UFs and thus does not require any additional toxicological information. As a result, we believe that the approach can aid risk assessors in achieving the goal of the Guidance for Risk Characterization of "... explaining confidence in each assessment by clearly delineating strengths, uncertainties, and assumptions, along with the impacts of these factors" (USEPA, 1995).

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Figure 1. Uncertainty in Hazard Quotients or Hazard Indices
Case Studies 1.1 and 1.2

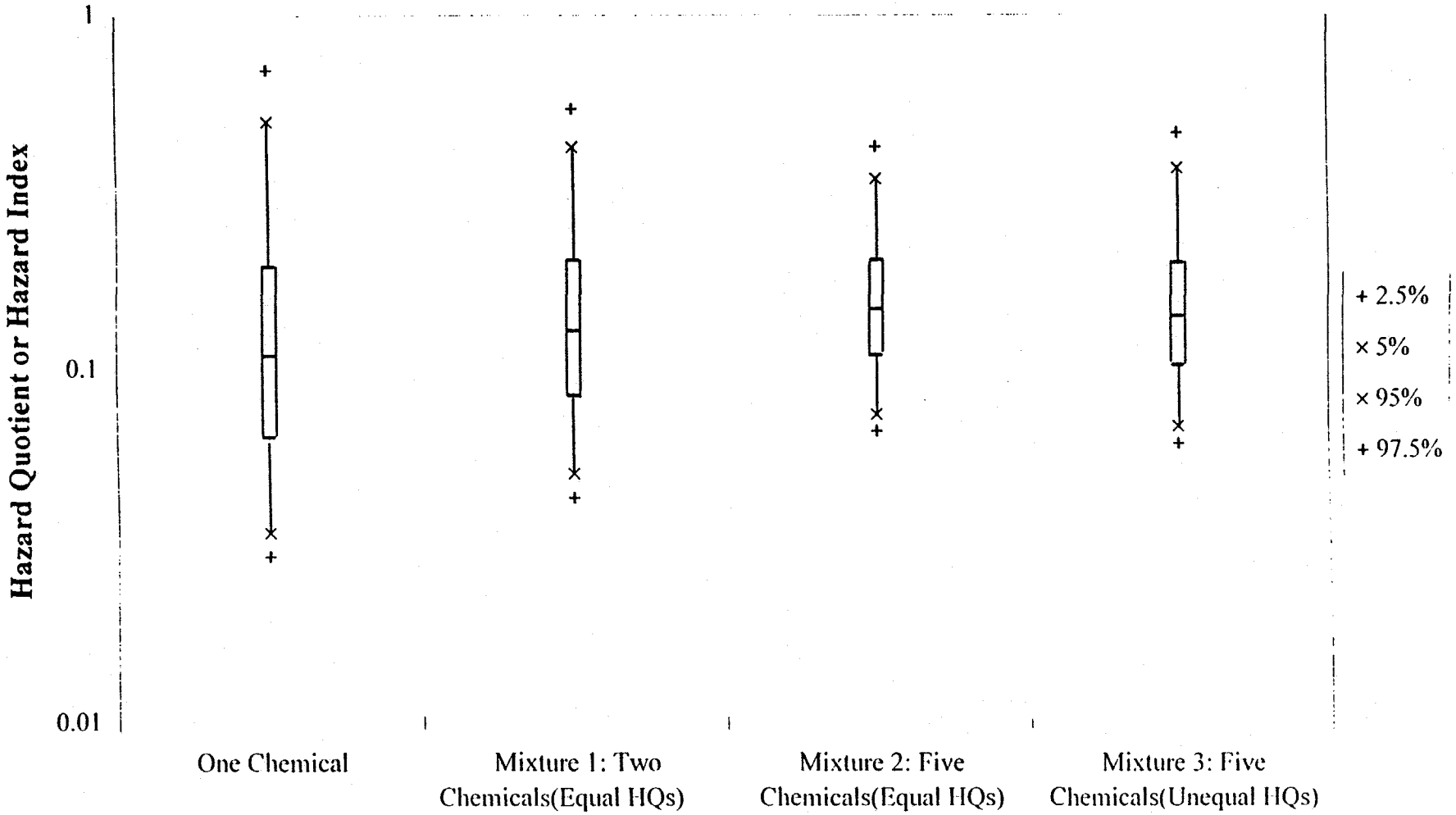

Figure 2. Uncertainty in Hazard Quotients and Hazard Indices
Case Study 1.3

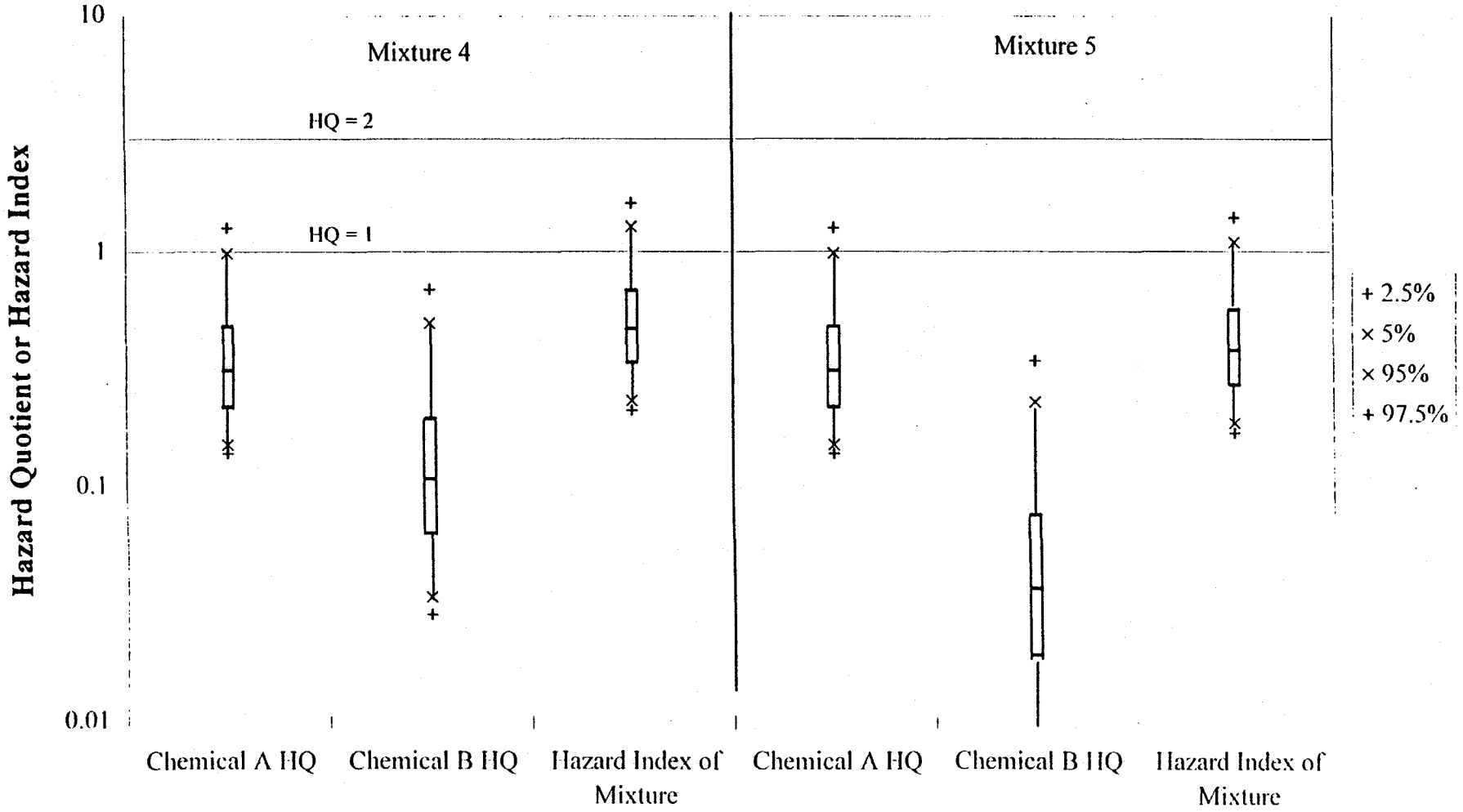

Figure 3. Uncertainty in Hazard Quotients for Randomly Selected Individual Case Study 2.1

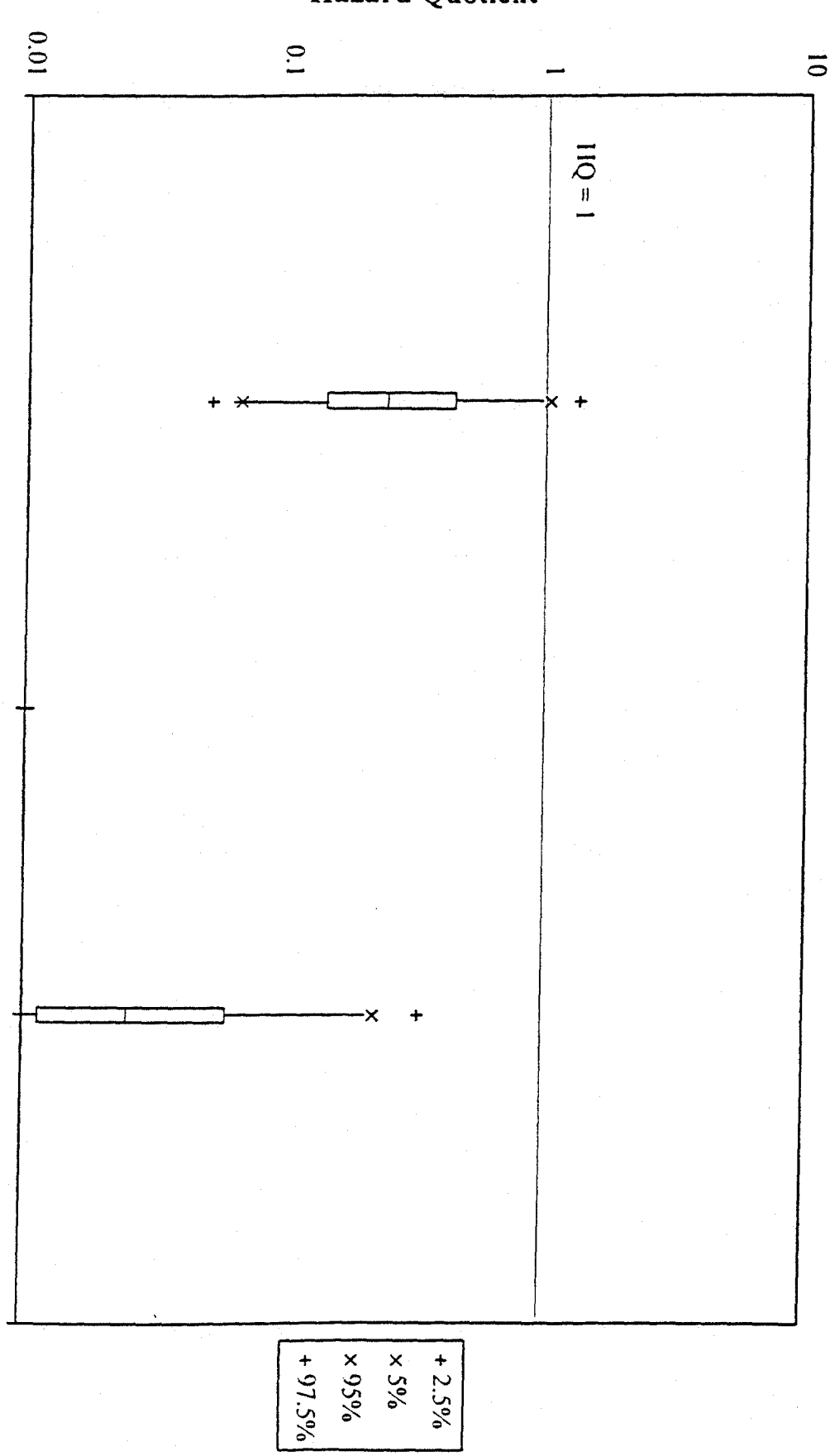

Figure 4. Two-Dimensional Model of Variability and Uncertainty in Hazard Quotients
Case Study 2.2

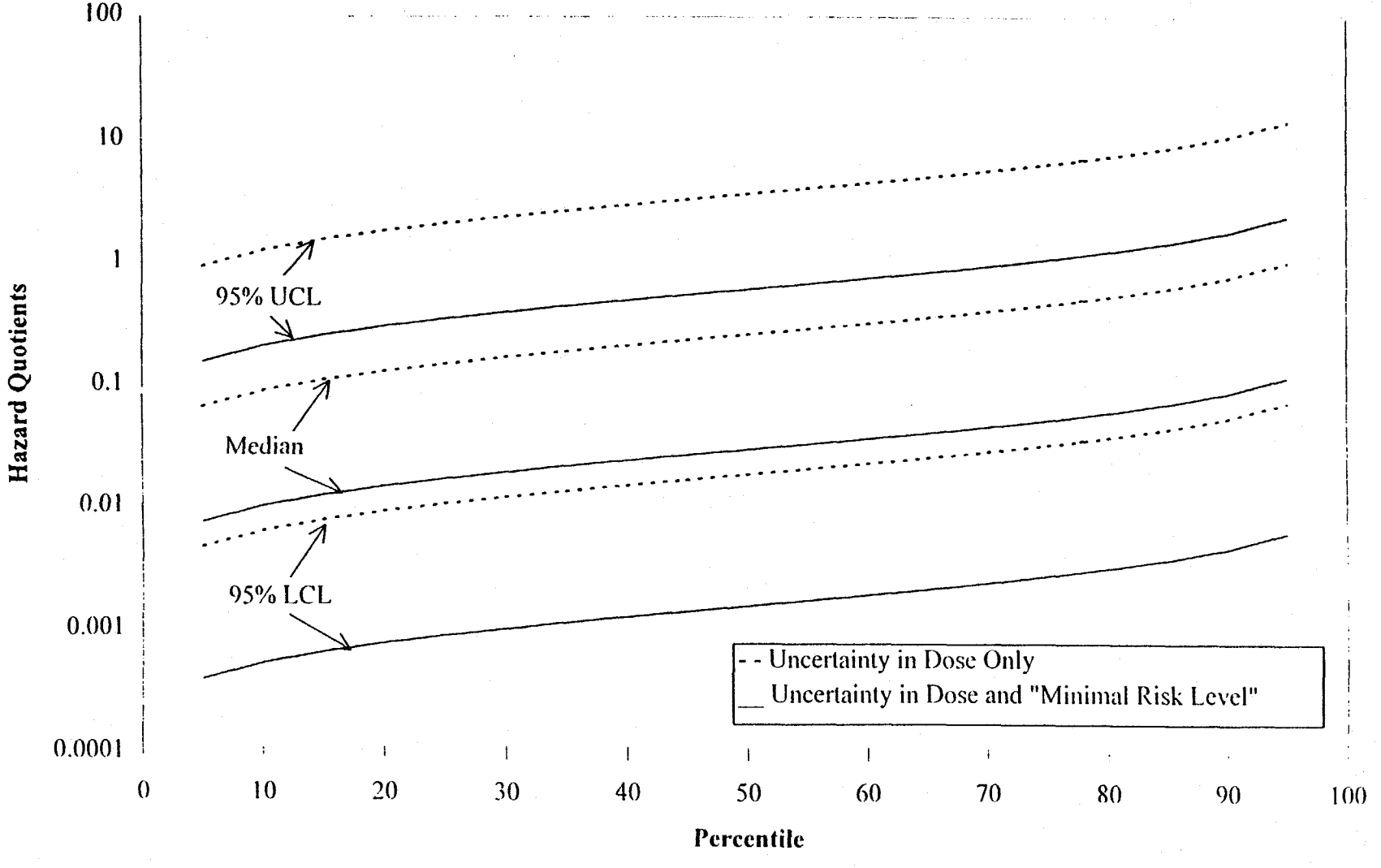

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Table I
Input Variables for Case Studies[a,b]

Example	Chemical	Total UF(s) used	Point Estimate RfD (mg/kg-d)	Exposure (mg/kg-d)	Nominal HQ
		Case 1.1: Mixtures of Compounds with Equal Contribution to HI			
Mixture 1	A	2	1	0.5	0.5
	B	2	1	0.5	0.5
Mixture 2	A	2	1	0.2	0.2
	B	2	1	0.2	0.2
	C	2	1	0.2	0.2
	D	2	1	0.2	0.2
	E	2	1	0.2	0.2
		Case 1.2: Mixtures Where One Compound Dominates the HI			
Mixture 3	A	2	1	0.5	0.5
	B	2	1	0.1	0.125
	C	2	1	0.1	0.125
	D	2	1	0.1	0.125
	E	2	1	0.1	0.125
		Case 1.3: Mixture Components with Different UFs and Same HQs			
Mixture 4	A	1	10	10	1
	B	2	1	1	1
Mixture 5	A	1	10	10	1
	B	3	0.1	0.1	1

a. Additivity of HQs to calculate HI implies that all contributing constituents share a common mode of action or elicit the same adverse effect.

b. NOAEL for each hypothetical chemical is equal to 100 mg/kg-day.

Table II
Comparison of Point Estimate with Median and 95th Percentile Values Derived from RfD Distributions

Substance	Total UF(s) used	Nominal Value	Median	95th Percentile
Case 1.1: Mixtures of Compounds with Equal Contribution to HI				
Mixture 1 HI	2	1	0.1	0.4
Mixture 2 HI	2	1	0.1	0.3
Case 1.2: Mixture Where One Compound Dominates the HI				
Mixture 3 HI	2	1	0.1	0.4
Case 1.3: Multiple Compounds with Different UFs and Same HQs				
Chemical A HQ	1	1	0.3	1
Chemical B HQ	2	1	0.1	0.5
Mixture 4 HI		2	0.5	1
Chemical A HQ	1	1	0.3	1
Chemical B HQ	3	1	0.04	0.2
Mixture 5 HI		2	0.4	1

Table III
Comparison of Case 1.3 Results Using Reference and Empirical Uncertainty Factor Distributions

	Median	95th Percentile
Chemical A HQ, Mixture 5		
Reference[a]	0.32	1
Empirical I[b]	0.27	1.1
Empirical II[c]	0.27	1.1
Empirical III[d]	0.40	0.8
Chemical B HQ, Mixture 5		
Reference	0.04	0.2
Empirical I	0.03	0.8
Empirical II	0.02	0.4
Empirical III	0.03	0.2
HI, Mixture 5		
Reference	0.38	1.1
Empirical I	0.37	1.6
Empirical II	0.34	1.3
Empirical III	0.45	0.83

a. Swartout et al., 1998

b. Baird et al., 1996

c. Swartout et al., 1997 Schmidt et al., 1997 and Baird et al., 1996

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A Case Study and Presentation Of Relevant Issues on Aggregate Exposure

For the ILSI Aggregate Exposure Workshop Program

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I. INTRODUCTION

As scientists and environmental managers, we are called upon to address advancements in the practice of risk assessment. Over the past fifteen years we have improved our understanding of the principles in exposure and risk assessment. We have developed powerful computing capabilities, increased precision in analytical chemistry, evolved statistical applications for exploring the unmeasurable, and enhanced our use of empirical data to better describe exposure scenarios and characterize potential risks. The Food Quality Protection Act of 1996 (FQPA) requires that we characterize risk from all sources of exposure to families of chemicals that may be related in their mechanisms of action. This act applies directly to pesticides, but this concept is gaining favor and will increasingly be applied to other regulatory venues as well.

A. THE CHARGE FROM INTERNATIONAL LIFE SCIENCES INSTITUTE (ILSI)

The ILSI aggregate exposure assessment project identified consultants with expertise in exposure assessment and requested assistance in identifying methodologies, concepts, and software for use in performing aggregate exposure assessments. This monograph presents our response to this project. In this report, we present a discussion of the critical concepts in aggregate exposure and provide recommendations on how they can be addressed by regulatory agencies. We further present a new approach for characterizing aggregate exposure in the form of a software program, "LifeLine™". Finally, we use the LifeLine™ model to characterize the aggregate exposures of the general US population based on the sample data set of pesticide residues in the home, tapwater, and diet provided by ILSI.

B. THE CASE STUDY DEVELOPMENT TEAM

This presentation reflects a joint effort of Hampshire Research Institute (HRI), ChemRisk (a Service of McLaren/Hart, Inc.), and TAS-ENVIRON. These organizations have entered into a formal collaborative arrangement to make tools for the assessment of aggregate and cumulative exposures and risks broadly available to the private and public sectors and to the general public.

HRI was established in 1987 as a 501 (c) (3) scientific and educational organization. Its goal is to promote environmental protection by providing useful and accurate information to the public. As part of this effort, HRI developed the first set of risk assessment tools for the personal computer, RISK*ASSISTANT, and currently supports a thousand users in government, industry, universities, and public interest organizations. HRI also develops publications of environmental analyses, and provides technical expertise to a wide variety of public interest organizations.

ChemRisk is a leading firm in the development of multipathway exposure models and a pioneer in the application of probabilistic techniques (including Monte Carlo analyses) in the evaluation of

exposure, chemical toxicity, and risk. For more than eight years, ChemRisk has used Monte Carlo techniques within multipathway models to provide analyses for clients faced with a broad array of environmental contamination problems. ChemRisk has pioneered the use of microexposure event analysis (Price et al., 1996) as a means of characterizing time-varying exposures to environmental contaminants.

TAS-ENVIRON has been the leading firm in the analysis of dietary exposures to pesticides. In addition, TAS-ENVIRON has led the development of software systems to support these analyses, developing programs and databases such as those used in Exposure 1™ and Exposure 4™. TAS-ENVIRON has developed a unique suite of databases on food composition and on diets for different populations, as well as the knowledge to use these data appropriately. TAS-ENVIRON scientists have been leaders in the risk evaluation approaches, especially in interpretation of the toxicology, statistical and exposure complexities and integrating the information in unique and constructive ways. They have applied these innovations to meeting regulatory standards in the US and internationally.

II. BACKGROUND

Aggregate exposure assessment must be looked at in the context of historical pesticide programs, the requirements of the FQPA, and the larger context of the development of federal policy for risk management. This section presents a review of technical and policy issues that are relevant to the development of aggregate exposure estimates.

A. LIMITATION OF APPROACHES HISTORICALLY USED TO EVALUATE EXPOSURE TO PESTICIDES

Risk assessment approaches presently in use at the Environmental Protection Agency (EPA) reflect the technical limitations that existed when those approaches were instituted in the 1970s and early 1980s. The risk assessments for pesticides were constrained by legislative perspective, scientific tradition and technical limitations. The law required that risk be assessed for each pesticide independently and each use be considered separately. Scientific tradition in this area led to deterministic approaches, often employing "upper limit" or "worst case" approximations of the anticipated exposure. Creating, managing, and manipulating large databases and performing complex iterative calculations was not plausible before the age of fast low-cost computers. Thus the approaches to characterizing exposure were confined to the use of simple exposure and risk algorithms. These approaches introduced conservative bias into estimates of exposure and risks that were poorly understood and often hidden from decision makers. The consequences of such aberrations were tolerable when the assessment was for only one use of one chemical at a time; however, the failings of such approaches will make reasonable assessments of risk from multiple pathways of more than one chemical difficult if not impossible.

With the advancements in the science of exposure assessment and computer technology, we are offered an opportunity to step back and reconsider the question-what are the exposures to pesticides that are relevant to the assessment of risks and how can we determine these exposures.

1. Traditional Exposure Assessments Force the Determination of Exposure into Only Two Categories: Acute and Chronic

Exposures to pesticides from diet, tapwater, residential, and other sources occurs at varying frequencies and intensities. Doses of pesticides received from sources such as tapwater consumption may be relatively constant and continuous in an individual's life, while other sources of exposure may be highly variable. For example, use of a pesticide on a rarely consumed food item or a pesticide which has a limited market share may result in a dietary exposure that is not repeated for weeks or months. Finally, exposure may have temporal structure that affects the doses received by individuals. The use of a pesticide in a home can result in a peak exposure on the day of application and lower exposures on the days that immediately follow application.

In the past, exposure was artificially characterized as either "acute" or "chronic." Based on this categorization, estimates of dose were determined in radically different ways. The high end of the range of residues (e.g., 95th percentile) was used for acute exposures and average residue concentrations were used for chronic exposures. Acute assessments only considered users (those persons who actually ate the commodity in question), while a "per capita" approach was used for chronic assessments that included both users and non-users.

In an acute assessment, the analyst is forced to view exposures in terms of a single day. A user can only be defined as someone using a product or consuming a foodstuff that is actually contaminated on a specific day. This is not a problem in the analysis of isolated sources of exposure; however, in the case of aggregate exposure it requires that all uses occur on a single day (i.e., a stacking of sources). As a result, it is impossible to take into account information on frequency of episodic exposures, day-to-day variation in intensity, or variation in the intensity of the exposure from one episode to another. In a chronic exposure assessment, the analyst is forced to average the doses received from both episodic and continuous sources over either a year or a lifetime. As a result, exposures that are elevated for periods of time of up to several months are ignored in the assessment. Under the current system in place at EPA, the analyst has no discretion to consider any intermediate time-period between one day and one year.

2. Neither Exposure Approach Allows a Proper Consideration of Time

Traditionally, EPA has focused on the intensity of exposures as determined by the level of residue in foods and the amount of food consumed. Little or no attention has been given to the frequency at which these exposures occur or the role of such temporal information.

Both the sources of exposure and the individuals exposed change over time. As a result, doses received by individuals vary at various times in their lives. This variation can be thought of as an intra-individual variation in dose that occurs over an individual's lifetime. Neither the acute nor chronic exposure assessment properly acknowledges this temporal dimension to exposure. Acute assessments focus on a single day and as a result cannot consider day-to-day variation. Chronic exposures ignore day-to-day variation by averaging exposure over long periods of time.

In the past, Monte Carlo models of dietary and aggregate exposure have sought to consider some information on temporal variation in exposure. However, these efforts have persisted in characterizing acute exposures in terms of a single day's dose for an individual. Therefore, the distributions of doses produced by these analyses are a combination of inter-individual and intra-individual variation[1]. It is difficult to separate these components and to evaluate the meaning of such analyses. In addition, such analyses cannot provide any information on the distribution of longer durations of dose (2-day or 30-day average doses).

3. Characterizing Intra-individual Variation in Short-Term Doses

Because an individual's doses vary over time, an individual has more than one short-term dose. For example, a 70-year old individual will have more than 25,000 daily doses over his or her lifetime. Individuals also have multiple longer-term exposures. An individual has thousands of different overlapping annual doses depending on the dates that the dose begins and ends. These doses can vary by orders of magnitude depending on the individual's behavior and temporal variation in the level of contamination. Exposure assessment tools should reveal this intra-individual variation to the decision maker.

4. Frequency and Dose Can Have Complex Inter-relationships

EPA currently allows preferential treatment to "safer" pesticides. These pesticides are defined as pesticides that require lower application rates or are less toxic than others. However, certain pesticides may also warrant consideration because of increased efficacy. If a pesticide is more effective and is not used frequently, the potential for exposure is less than for a pesticide with slightly lower use rates but which is used more frequently. Currently, there is no mechanism for consideration of frequency in the determination of the relative "safer" pesticides.

[1] This estimate of exposure can be best viewed as the dose that occurs on a random day taken from a randomly selected individual.

5. Pesticides Have Complex Temporal and Spatial Correlations

Individuals are constantly exposed to ever changing mixtures of pesticides that occur from multiple sources and routes. Exposures to many of these pesticides are highly correlated. Where there is a choice of pesticides for a particular use, the selection of one pesticide results in the exclusion of other pesticides. This results in strong negative correlations between residue levels of certain pesticides. In contrast, certain pest problems call for the use of multiple pesticide products or products containing multiple active ingredients. In these cases certain pesticide residues will have a strong positive correlation. It is important to have the ability to model these temporal correlations of residues.

6. Aggregate Exposure Assessments and Routes of Exposure

Aggregate exposure assessments will by definition include doses occurring by multiple routes of exposure. In instances where a systemic toxicant is under evaluation, the total dose that is received by all routes may be the relevant dose metric. In other instances, doses by specific routes may have differing toxicological implications. As a result, the risks posed by a pesticide may change as the proportion of dose that is received by different routes changes even when the total dose remains fixed.

In addition, the uncertainty in dose estimates also varies by route. Current methods of estimating dose for various routes of exposure vary in their accuracy and bias. Doses received by oral routes are usually known with better precision than doses received by dermal routes. Because of the greater reliance on default assumptions concerning contact times and absorption, dermal routes are more often over-estimated, and over-estimated to a greater degree, by current methodologies than doses from oral routes.

7. Determination of Toxicologically Relevant Doses Over Time

Many toxicological effects require some duration of exposure in order to occur in humans or animal models. Certain compounds take time to accumulate in the relevant compartments of the organism. Other compounds require time to exhaust an organism's capacity or short-term mechanisms of compensation (tolerance mechanisms). Still other compounds exert their effects by disrupting cyclical processes in the organism in a way that requires persistent exposures (endocrine effects may fall into this category). In addition, some toxicological consequences may be related to the progression of exposure (first to an initiator, then to a promoter). As a result, certain toxicological endpoints do not occur in 1-day studies but are observed in 14-, 30-, or 90-day studies.

Under the current framework, toxicologists are forced to either ignore these longer term effects or to assume that they will occur as the result of a single day's exposure. While this assumption is protective, it results in a significant potential for over-regulation of pesticides. Under current policy, registrants do not have the option of developing exposure durations longer than one-day and as a result cannot make this determination before the Agency.

8. Subpopulations are Currently Not Well-Defined

Subpopulations are evaluated in the risk assessment process in order to determine if risks are unequally distributed across the general population. Currently, subpopulations are defined based on tradition or the intuition of the toxicologist. One basis for the definition of subpopulations that has special consideration under the FQPA is age. Children and infants are a specific concern with Congress and many stakeholders. However, other age-related subpopulations may be of concern as a result of unique sensitivity and/or exposure patterns. These include adolescents (unique dietary patterns), the elderly (dietary patterns and toxicological sensitivity), and women of child-bearing years. Age-related exposure patterns will likely increase in importance with aggregation. Institutional exposures such as those possible in nursing homes, schools, and workplaces will require exposure assessors to focus on other age groups with unique routes or sources of exposure.

The current system of exposure assessment often does not clearly define age-related subpopulations. Children are often lumped together into broad age classes (e.g., ages 1-6), and other groups are not investigated separately. Ideally, exposure assessment should determine whether any age group is correlated with elevated doses and should evaluate and quantify the doses specific to age groups suspected of being uniquely sensitive to a pesticide (see additional discussion on page 21).

B. Risk Management Needs and the Development of Aggregate Exposure Software

The risk assessment process finds itself having to incorporated new technology, increase efficiency (tiered approaches), support risk communication, and achieve a level of constancy and transparency. The discipline of exposure and risk assessment is changing radically. These changes concern not only technical issues, but also the risk management context of the analyses. In the past, many risk management decisions have been made in a closed process dominated by professionals. Thus, a regulatory agency and a company interested in introducing a new chemical into commercial use would discuss available data and analyses of potential risks and benefits with only occasional and limited participation of outside parties.

This process has led to the feeling of disenfranchisement by the public and an endless series of rancorous debates over the meaning and adequacy of the Agency's exposure and risk

assessments. As a result, there have been changes in public policy regarding the use and dissemination of the results of exposure and risk analyses. Early examples include EPA's decision to use "community acceptance" as one of nine criteria for acceptance of a Superfund decision, and Congress' inclusion in the Superfund Amendments and Reauthorization Act of a program of grants to communities to receive independent technical consultation.

This trend has received its two strongest impetus to date in two presidential reports: *Reinventing Environmental Regulation* (1995) and the report of the *President's Commission on Risk Assessment and Risk Management* (1997). In the former, President Clinton and Vice President Gore explicitly called for promoting risk-based decision making in communities, by providing training and easy-to-use risk assessment tools. The latter document explicitly elaborated a new framework for risk management decisions, in which stakeholder participation is central to all aspects of the assessment and management effort. If the scientific and professional communities are to avoid perpetuating or exacerbating this situation, a much greater degree of "transparency" is called for than has been reflected in historic practices.

These concerns have major implications for the implementation of software systems. As HRI has learned from supporting hundreds of RISK*ASSISTANT users, it is not enough to provide help on system operation. It is also necessary to incorporate a vast amount of explanatory and reference information. For every algorithm, variable, and default data point, the user must be able to determine:

- What it is,
- Where and how it fits into the overall analysis,
- Whether it has been validated,
- Where it was obtained, and
- What plausible alternatives are known?

The closed risk management process of previous years was tolerant of "black box" answers and the use of proprietary systems. This will not survive in the redefined, stakeholder-driven risk management system.

The new generation of risk assessment tools is sufficiently complex and powerful that making them available to all stakeholders in a meaningful way will not be simple. Merely publishing reports on underlying concepts, making databases "available", or even distributing source code on the Internet, does not support effective public understanding of these tools.

1. User-Friendly Systems

For most of the public, risk analyses will affect a small, if crucial, element of their lives. While residents near a waste site or a planned facility with high emissions are highly motivated, and learn to address the critical scientific concerns quite rapidly, they no more have the luxury to spend weeks learning a software system than they do to critically review thousand-page reports.

This is also true for a large fraction of the technical user community. In an era when few technical professionals have the ability to concentrate on the relatively narrow perspective on a decision provided by any discipline, but must be able to integrate demographic, hydrologic, meteorologic, and toxicologic perspectives in making decisions, programming and interface styles of a decade ago are simply not viable.

Systems that support stakeholder-based decisions must be, to the maximum extent possible, entirely self-explanatory and intuitive. Perhaps the best model for the next generation of such tools can be gleaned from tax-preparation software. These systems can address the immense complexity of the tax codes, but do so by asking questions and providing answers in terms that are familiar to the average person.

2. Effective Availability of Software

It is possible for a system to be available to the public in a technical sense, without being available in any meaningful sense. Perhaps the most obvious barrier is price. There are software systems for which anyone can purchase a license for fifty thousand dollars, but few would argue that these are meaningfully available to the public. The average user of a personal computer is unlikely to spend more than a few hundred dollars on any software package; those with general utility (e.g. spreadsheets) generally cost far less.

At the other end of the system, there are thousands of software systems (including a number of systems developed by the federal government) that are available for free, but are not of useful to any but the most dedicated potential user. This generally reflects two interacting factors:

- An absence of affordable technical support, and
- A failure to address the non-professional user in system design.

For an academician considering a software system in her/his area of specialization, these considerations may not represent a major barrier. Time (or graduate students) may be available to identify the meaning of obscure variable names, translate data files, and interpret reams of tabular output. For most of the public, however, such systems are not really available.

3. Responsiveness to User Concerns

A key failing of many risk assessments over the past fifteen years has been a lack of sensitivity to the concerns of participants in risk management. At the most obvious level are the hundreds of assessments that have applied default parameters when very different, and clearly more relevant, situation-specific data were available. In many cases, risks to particular populations are driven by location- and situation-specific information that is best known to local communities. A risk assessment approach that is not inherently designed to capture this situation-specific information has little if any value.

The need for system responsiveness is not, however, limited to variables such as the amount of fish consumed in a day. Rather, analytical systems must have sufficient structural flexibility to be able to readily incorporate new analytical techniques for particular user communities. This can range from simple re-arrangements (such as using the same algorithms to calculate risks from concentrations, or to derive target concentrations from predefined risk levels) to the need to incorporate entirely new analyses (such as adding in algorithms to address the efficacy of water treatment systems, or to calculate cumulative risks from chemicals with synergistic modes of toxicity).

Responsiveness to "reality"-incorporation of data which better quantify real biology, reality in chemistry and the time dimensions of the event-move us away from the oppression of conservative default assumptions and rule by the absurd. Little is gained by accrual of exquisite toxicology and chemistry data if meaningless "conservative assumptions" or default parameters dwarf it. These factors, occupying powerful positions in the risk formulae, can drive the calculation and create illusions of risk. The user must be able to easily differentiate between a derived risk, thus created, and a probable risk, inferred from real data and faithful incorporation of information. Risk assessment systems can no longer mask these contributors to the answer. Visibility-transparency-is required in the design. This means the answer (while derived from the most state-of-the-art sciences) must be clear and graphically presented so any reasonable person understands it.

Without modular architecture to address these concerns, any methodology for risk assessment will rapidly become obsolete, resulting in wasted and duplicative efforts by both the scientific and stakeholder communities.

The system used to address the case study problem, LifeLine™, is part of an ongoing effort at developing risk assessment methods that can be incorporated into tools made broadly available to the public. This effort benefits from a decade of collaborative work with the EPA in developing and distributing software to a broad public base, including four versions of RISK*ASSISTANT (and a

separate Russian-language version) as well as RISK*WORKS, a program for occupational risk assessments developed with the University of Liège and the government of Belgium.[2]

III. MODELING APPROACH

This section describes the conceptual modeling approach behind LifeLine™ and the actual mechanisms of the software.

A. CONCEPTUAL MODEL

Our project team believes that aggregate exposure assessment calls for a break with previous dietary and residential exposure assessment approaches. As outlined in the previous section, existing approaches for exposure assessment suffer from a number of limitations which are exacerbated by the requirements of aggregate and cumulative models of exposure. Previous approaches for exposure assessment were in part adopted because of mechanical limitations facing the analyst. Many of those obstacles are surmountable today. For example, better databases exist on the occurrence of residues in foods and water, and an expanded understanding exists on the parameters which contribute to the accrual and/or degradation of residues under different conditions. In addition, vastly expanded computer capabilities provide an opportunity for new calculation techniques and use of large databases.

These new advantages permit a total re-evaluation of the science of exposure assessment and its approaches. The two greatest objectives for any new system to meet must be: (1) to have the foundation for the exposure assessment driven by the characteristics of the toxicology which describe the hazard; and (2) to introduce a time dimension into the assessment methodology.

Thus, in developing an entirely new approach for aggregate risk assessment, the project team sought the following characteristics:

- The approach should be responsive to the toxicology in terms of preserving exposure contributions from different routes for any toxicologically relevant time period.
- It should use all the information available to describe the conditions and activities of the potentially exposed. It should work from the "bottom up," beginning with information on each day's exposure event(s) and how the events occur across the lifetime of the individual. The approach should not take overall generalizations and break up these generalizations into fractions to arrive at descriptions of the individual being exposed.

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[2] At present, approximately 1,000 users (outside of EPA) are being supported in more than a dozen nations, and more than 90 academic licenses have been issued. The team has also gained experience from training EPA users, state and local government officials, and local and regional environmental groups. A set of lesson plans, keyed into the ACS textbook *Chemistry in Context*, is currently being field-tested.

- It should have the capacity to accomplish the assessment of exposure from multiple chemicals (cumulative exposure) so that an approach for aggregation does not have to be abandoned or contorted to accommodate cumulative exposures.
- It should be flexible enough to accept new algorithms and data in the future. This process will be evolutionary in nature. No one perfect exposure system can be developed at a given moment in time.
- It should provide universality so that the approaches are applicable to risk assessments for non-pesticide programs such as drinking water, air emissions, foods, consumer products, etc.

To accomplish this, we have applied Microexposure event analysis (Price et al., 1996), a Monte Carlo-based technique that views an individual's exposures as a collection of separate events. Microexposure event analysis models each exposure event separately and then accrues the doses to produce an estimate of the average dose across any time period of interest. By modeling separate exposure events, information on specific circumstances relevant to exposure can be used to more accurately quantify the doses received from that exposure.

Central to this approach is the shift in emphasis from modeling the exposure to modeling the individual receiving the exposure. The model thus begins by developing a coherent model of the life of an individual. Once this is done, the software can determine how, when, and at what intensity exposure to a given source, or group of sources, occurs in the individual's life. This process is then repeated for additional individuals. By varying the individual's characteristics and exposures, the model creates realistic models of exposures across the actual population.

This approach requires a larger commitment in terms of computer resources but is perfectly manageable using today's desktop personal computers. Central to the concept is the creation of an account for each day's exposure over an individual's lifetime (Figures 1 and 2). All individuals in a defined population are assessed in this way and their histories accrued to create distributions of inter-individual variation in doses. This is The LifeLine™ Exposure Assessment System.

Each "page" in the LifeLine™ is a distribution of an individual's exposure by various routes and the total exposure for that day. There is a "page" for each day in the individual's entire life. This analysis is repeated for all persons in the population as defined. Then, all of the exposures (total as well as the individual routes) for all persons on one day may be accrued into a distribution for that day. This is repeated for all of the days in the LifeLine™, creating a pattern of exposure for the population. This can be viewed across time, revealing those periods of time which have high exposures, or where patterns of the contributions from individual routes of exposure may be of particular interest. The assessor can extract those "pages" over the time period of interest and examine the details of the events and the calculations that led to those exposure profiles. The assessor can evaluate the importance of the underlying data, default values and algorithms that

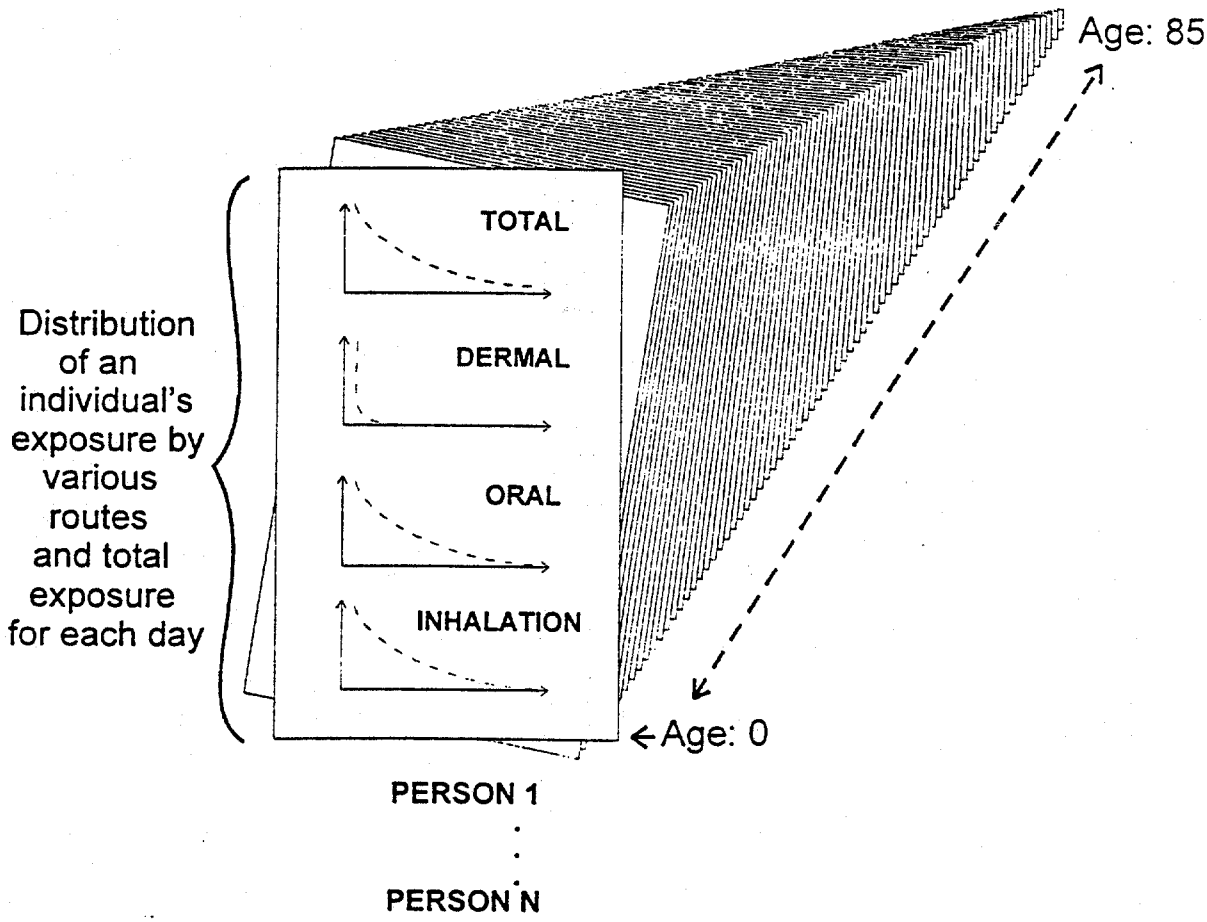

Figure 1. Characterizing an individual's exposure history.

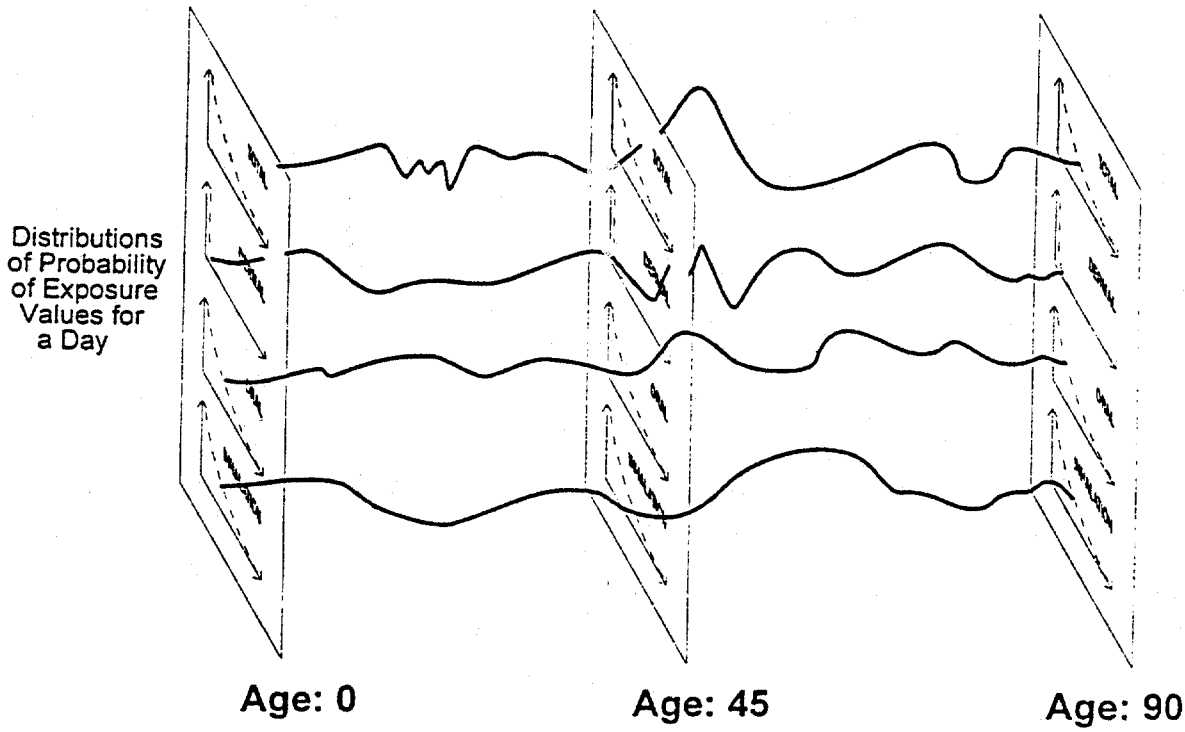

Figure 2. Dose histories for oral, dermal, and inhalation routes and total exposure

contributed to those profiles. Important subgroups may emerge as interesting for defining another analysis, or key defaults may play a dominant role in the assessment.

LifeLine™ is not a specified method of determining exposure that is linked to a specific model or that requires specific types of data. Rather, LifeLine™ is a framework for combining information on exposure from any source into a coherent framework for an individual's total exposure.

LifeLine™ provides a number of advantages. First, by modeling each day in an individual's life, it provides a high level of detail on the occurrence of exposure. Second, it allows the identification of any age group having elevated total or route-specific doses. Therefore, it is not necessary for the assessor to define an age group prior to the analysis. The age groups defined are age-specific and do not require the averaging of individuals into artificial age groups. Third, by completely integrating doses from all sources and routes, LifeLine™ allows the assessor to determine the impact of default assumptions. The assessor can consider:

- Relative contribution from the dermal, inhalation and oral doses
- Relative contribution in any route source from data, default values or models and algorithms within the calculation.
- Relative contribution and changes in contribution by the construct of the boundaries on the Monte Carlo iterations and any associations/disassociations defined in the analysis. (This is an examination of the assumptions dictated by the assessor or intrinsic to the LifeLine™ pre-programmed mode.)
- Relative importance of default factors such as dermal penetration, use rates, seasonality and other event alterations.

Fourth, the assessor will want to understand the characteristics of precision and statistical integrity of the answers from this assessment. Error, bias and uncertainty accompany all data and may be amplified by accompanying default factors or algorithms directing the way those data are used.

The overall error, bias and uncertainty of the answer will be a function of how much each element of the calculation weighed into the answer. For example, the relative contribution to overall error from a distribution of data on surface residues will be very great if dermal and oral routes of exposure were the significant routes and if that distribution was matched with an exaggerated default value for dermal uptake. Such errors and uncertainties can be calculated or approximated for all analyses.

Fifth, the approach can incorporate a wide range of dose information. Any information that defines an exposure opportunity or the circumstances of the person being exposed can be used in

microexposure event modeling. Information about the use profiles, frequency of treatment (efficacy data), pest pressures, seasonality, residue information, residue degradation rates, associations between uses, exclusions on uses or situations, and demographics or other information defining the environment can be included. This information can include descriptions of use, and associations or competitions between different uses of a pesticide or between the use of multiple pesticides. Thus, at this level, information regarding exposure to multiple chemicals can be incorporated.

Because LifeLine™ does not require specific types of data, the model can be used at various steps in a tiered assessment process. Initially LifeLine™ can be run using conservative defaults in the absence of data. At higher tiers of assessment the software can be run using with actual monitoring data. In addition, the software's ability to characterize the important routes of exposure monitoring, for different ages can be used to determine which default assumption(s) are responsible for the estimation of high levels of exposure. The data gaps that underlie these defaults can then be targeted for research or data acquisition activities (monitoring programs) that will allow the replacement of the defaults (higher tiered assessments). In this way, the software will allow the efficient use of resources.

B. How LifeLine™ Works

LifeLine™ is a software approach for characterizing interindividual variation in doses of substances received from multiple sources and multiple routes using Monte Carlo analysis. In this case study, the model is used to evaluate aggregate exposure to a pesticide (Figure 3). However, the approach can be applied to other food additives, environmental contaminants, or other substances of interest. This section presents a description of the key components of the software and its configuration for evaluating aggregate exposure for a single pesticide.

1. Characterizing Individuals in an Exposed Population

As discussed above, the LifeLine™ approach begins by focusing on the exposed individuals. Under typical exposure models, an individual is poorly characterized and the focus is placed on the magnitude of the source of exposure and the size of the dose that occurs under specified conditions. However, the characteristics of the person being exposed (the individual's age, gender, immediate circumstances, and other sources of exposure) is not well defined. This limitation is not a problem when a single source of exposure is considered. However, when multiple sources occur at different times in an individual's life it becomes critical to clearly and consistently define the exposed individual.

One of the strengths of LifeLine™ is its ability to define the characteristics of an individual's life relevant to assessing exposure and selected socioeconomic factors that influence these characteristics. Examples of such factors include:

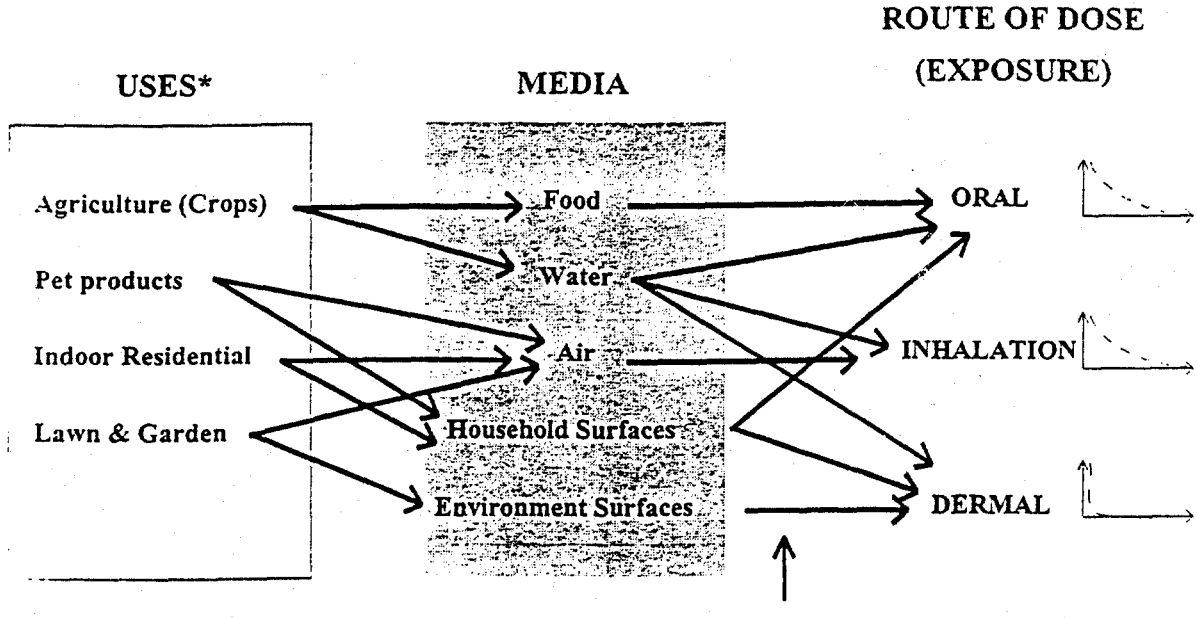

Figure 3. Sources, affected media, and routes of exposure for pesticides and on crops and in homes

- the individual's body weight, inhalation rate, and surface area,
- the diet of the individual,
- the types of housing the individual lives in at various points in his or her life,
- the location of those homes in the US,
- the frequency of pesticide use in the homes, and
- the potential for pesticide contamination of the tap water supplies of the homes.

Traditional Monte Carlo models of variation and uncertainty in dose define the individual only in terms of a few factors such as duration of exposure, intake rates, and body weight. These factors are defined in terms of a single value. However, an individual's body weight varies over his or her lifetime, the frequency of exposure can vary by age, and the rate of intake can vary from day-to-day. As a result, it is necessary to select an array of values for these exposure factors.

This process can be likened to the simulation of a hypothetical individual's life. Values for each of the factors used in the dose estimation equations have to be assigned for each day in the life. This process raises a number of technical challenges to the assessor.

First, many factors are strongly age-dependent. Therefore, age-specific distributions of values must be used in assigning attributes to an individual. Second, while varying over time, exposure factors for an individual are auto correlated. For example, an individual's body weight at any given age could be estimated by randomly selecting a value from distributions of age-specific body weights. While this will aid in the development of realistic body weights, it is unlikely that an individual would have a body weight that falls in the 5th percentile one year and the 95th percentile the next. In order to address this auto correlation, it is necessary to link estimates of an individual's body weights for various years.

Third, certain factors, such as whether a pesticide is used on a given day, are random in the sense that a pesticide is equally likely to be used on a Tuesday or a Wednesday. However, the probability of the occurrence is influenced by a large number of factors such as the season of the year, region of the country, type of home, frequency of use, and time since last use. Therefore, the probability of use is defined in terms of a series of conditional probabilities. Fourth, behaviors on one day can influence exposure on subsequent days. For example, use of a termiticide can result in inhalation exposures to individuals in a home over several months following application. As a result, the concentration in environmental media is a function of the current and prior use of pesticides.

Figure 4 presents an example of selected years of a constructed exposure history for an individual. The exposure histories define the relevant factors for each year of the person's life. Data on these factors are taken from age-specific data in the revised Exposure Factors Handbook (EPA, 1997), publications by the US Bureau of the Census, USDA dietary surveys as

Age/Year	Body Weight (kg)	Inhalation Rate (m^3/min)	Tap Water Intake (g/kg-day)	Frequency of Hand-Mouth Events (events/min)	Surface Area of Hands in Mouth (cm^2/event)	Surface Area of Body (cm^2)
0	10.49	0.0037	11.03	0.020	330.424	5129.61
1	12.10	0.0027	4.86	0.021	310.211	5916.90
2	13.60	0.0038	6.05	0.030	309.353	6650.40
3	16.65	0.0051	6.50	0.023	40.390	8141.85
5	18.41	0.0072	3.16	0.019	45.067	9002.49
8	38.25	0.0085	3.75	0.004	6.002	18704.25
10	40.37	0.0091	3.37	0.006	4.485	19740.93
12	43.03	0.0090	2.63	0.002	4.925	21041.67
15	49.95	0.0093	2.44	0.002	0.916	24425.55
20	45.98	0.0097	2.29	0.008	0.893	22484.22
25	59.25	0.0099	2.15	0.003	0.879	28973.25
30	63.79	0.0098	3.74	0.003	0.908	31193.31
35	65.84	0.0100	2.81	0.003	0.953	32195.76
40	62.02	0.0103	2.84	0.010	0.852	30327.78
45	68.42	0.0105	3.10	0.003	0.889	33457.38
50	74.59	0.0101	2.82	0.003	0.934	36474.51
55	74.88	0.0099	2.37	0.016	0.921	36616.32
60	74.89	0.0098	2.55	0.002	0.945	36621.21
65	70.32	0.0102	3.28	0.003	0.916	34386.48
70	71.14	0.0102	2.47	0.002	0.780	34787.46

Figure 4. A sample output of an individual's personal characteristics over his or her lifetime (only selected years presented)

appropriate, and other sources. In addition, the correlation between body weight over time in an individual was modeled by assuming that an individual will remain at the same percentile throughout his or her life. Finally, body weight and surface area were also correlated (Phillips et al., 1993). This version of the software currently estimates typical inhalation rates; future versions may also consider the level of activity of the individual for certain portions of the day and develop activity-specific inhalation rates. All distributions can be modified by the user to reflect the interpersonal variation in specific populations of interest.

2. Defining the Sources of Exposure

Once the individual and his or her behaviors are defined, it is possible to define whether the individual will be exposed to a pesticide source on each day of his or her life and what the resulting dose from exposure will be. The age of the individual, region of the US, type of home, and gender can be used to define the likelihood of exposure to a source.

Much of a person's exposure opportunity is defined by his or her residence. The type of residence and its location will influence when, if, and how a pesticide is used and when, if, and how a person will be exposed. Individuals reside in different homes for varying blocks of time during their life. During these blocks of time, key exposure-related factors remain constant. For example, the size of various rooms and air exchange rates remain the same for a given residence, as does the source of the individual's tapwater. However, when a person changes residence these factors also change. Therefore it is critical to be able to characterize how and when an individual changes his or her residence. Fortunately, data have been published on the mobility of the US population (USDC,1988; USDC and USD HUD, 1989). This data can readily allow the modeling of moving from one house to another. In the LifeLine™ software, age-specific mobility rates are used to determine the probability that an individual will change houses. As Figure 5 indicates, modeling the mobility (Column 2, where 0=no move, 1=move) can be linked to whether or not residential factors change.

This module also assigns a level of pest problems to each home. This level is used to determine the frequency of the use of pesticides.

The databases used to construct this platform include the US Census and the USDA Food Consumption Survey. Both databases are rich in details about household descriptions, including definition of income, location and source of the drinking water into the household. Assumptions and defaults for the residential environment are taken from the EPA Exposure Factors Handbook (EPA, 1997) and its underlying data sources where possible. Other sources can be used in the model when available.

Age/Year	Mobility	Frequency of Product Use	Single family home?	Lawn and User?	Tap-water Source	House Type	Tap water conc. (ppb)
0	1	0.009589035	0	0	1	2	5.2
1	0	0.011418198	0	0	1	2	5.2
2	0	0.000274109	0	0	1	2	5.2
3	1	0	1	0	0	2	7.9
5	0	0	1	0	0	2	7.9
8	0	0	1	0	0	2	7.9
10	0	0	1	0	0	2	7.9
12	1	0	0	0	2	3	2.3
15	0	0	0	0	2	3	2.3
20	0	0	0	0	2	3	2.3
25	0	0	0	0	2	3	2.3
30	0	0	0	0	2	3	2.3
35	1	0	1	0	1	4	1.1
40	1	0.008989181	1	0	1	3	0
45	0	0.013740027	0	0	1	3	0
50	0	0.009692798	0	0	1	3	0
60	0	0.001323494	0	0	1	3	0
70	0	0.013011659	0	0	1	3	0

Figure 5. A sample output of one individual's residences over his/her lifetime (only selected years presented)

In the application of LifeLine™ used in this assessment, we defined a residence as consisting of a bedroom, kitchen, bathroom, and family/recreational/television room. The model defines the room sizes, air exchange rates, and the floor coverings (e.g., carpet, tile).

3. The Challenge of Characterizing Lifetime Dietary Intakes Based on Short-term Studies

Characterizing doses from pesticide residues in food has long posed a major problem to exposure assessors because of the short duration of dietary surveys. However, we believe that it is possible to characterize longer term estimates of food intakes based on currently available data.

In developing a methodology for characterizing long-term intakes, it is important to recognize that day-to-day variation in food-related exposures is a function of both the variation in residue levels and the variation in diet (Figure 6).

i. Variation in Residue Levels in Food

The pesticide residue levels in the food that an individual consumes on a given day are a function of many factors such as which raw agricultural commodities (RACs) were used in the foods consumed, the origin of the RACs, and the season the RACs were harvested. However, residue levels can be directly monitored at various stages during the process in which food moves from the field to the table. There are three different sources of residue data: market basket data (commodities sampled at retail), field residue trial data, and tolerance levels. These different types of residue information represent the range of data typically available for estimating the concentrations of the chemical in the foods.

The nature and concentration of the residues from pesticide applications in agriculture will change dramatically during food processing. Therefore, sampling at the retail level will provide the closest approximation of the residues expected to be experienced by the eater. It accounts for changes in residue values due to the degradation and commercial processing effects. The key to assessing the overall utility of this market basket data lies in the monitoring survey design. The design should result in representative sampling of the food supply for the population in question and account for the seasonal use of the chemical and for the food market trade dynamics. Without careful design, the survey may yield data that are precise, but not representative of the population for whom the risk assessment is conducted.

Information about field residues can be used along with degradation rate information and processing effects data to estimate the residues in foods as they are eaten. This estimate can take the form of a point value or a distribution of possible residues, derived by applying the distribution of measured field values to a distribution of degradation rates derived for processing effects.

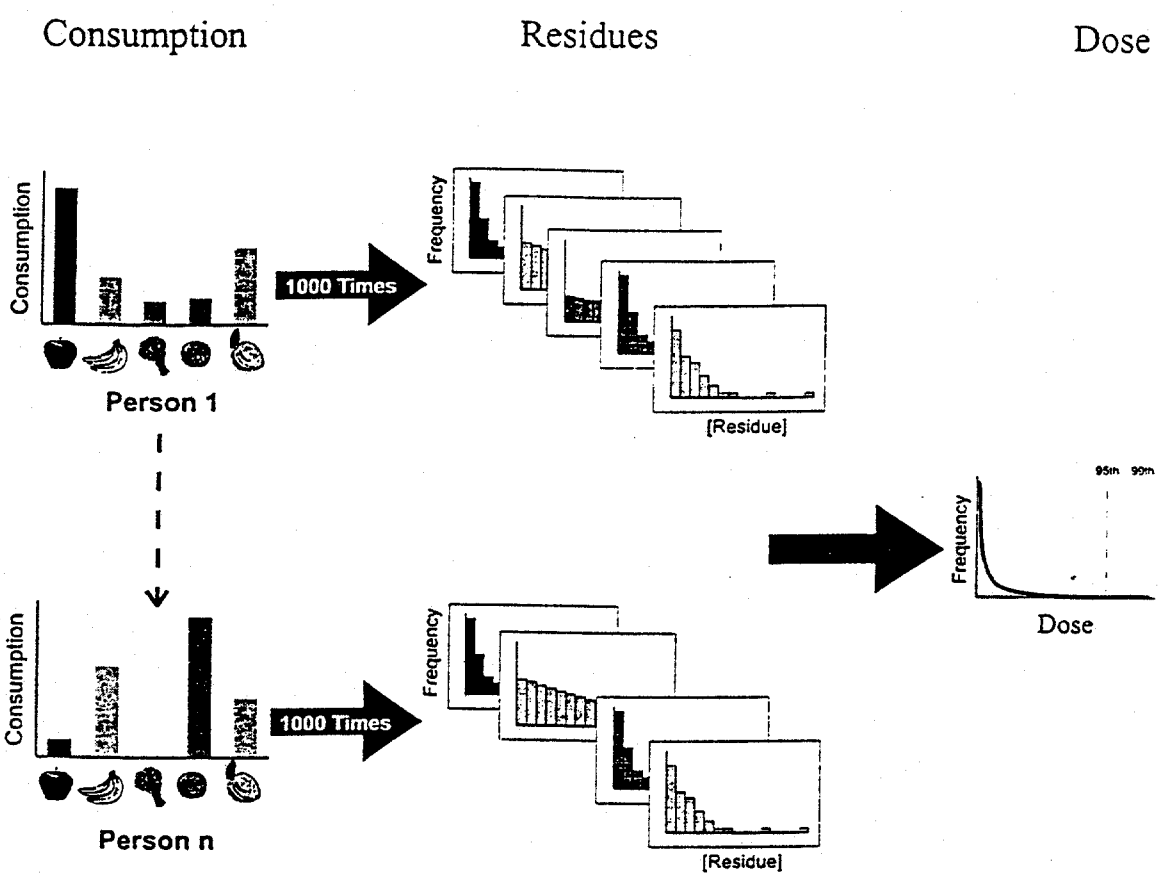

Figure 6. Doses from exposure to dietary residues.

Tolerance levels are probably the least realistic for such risk estimations. Tolerances are based on studies conducted at the highest labeled application rate, using the shortest interval between application and harvest. The tolerance is assigned based on a level that would not be exceeded if these maximum label conditions were followed. Tolerance levels serve well as an enforcement tool, but because of the multiple sources of bias in their derivation, they are poor values to use in an exposure assessment.

Another difficulty with the measurement of residues includes the inability to distinguish between those foods which truly have no residue (the true zero), and those foods which have a residue too low to be detected. There are several conventions for dealing with this dilemma. One convention is to use one-half of the level of quantification for the residence value. Another is to assume that the true zeros can be imputed by market share data. Other conventions are available and can be utilized carefully, with a consideration of the design of the monitoring study and the level of detection afforded by the analytical techniques used.

The interpretation of field trial presents another dilemma. The typical field trial data set is actually a collection of studies conducted in different geographical locations under different climatic conditions. Thus, they are a collection of distributions which may reflect seasonal differences given the probable growing opportunities for some commodities. These residues also reflect the exclusive use of one pesticide at maximum use rates. In the real world, it is more likely that residues will reflect use of several pesticides—precluding the possibility that any one would be used to its maximum frequency.

Despite these factors, the data on the residues in the RACs that comprise an individual's diet can be determined from monitoring studies. Once data on the distribution of residues that could occur on a RAC are established, there is no technical reason why the residue component of an individual's long-term dietary exposure estimates cannot be determined. Estimates of an individual's long-term exposure can assume that the residue levels that occur on a given day in a given food item are a random selection from the distribution of residue levels in the relevant RACs for the individual's location and the season when the food is consumed.

ii. Variations in Diet

As discussed above, the United States Department of Agriculture (USDA) food consumption surveys have been designed to include three-day records for each respondent in the survey. Obviously, there is no one database that includes a record following individuals throughout their lifetime, nor would such a data set necessarily be useful. The dynamics of the food industry are key to the relevance of consumption surveys. The foods available in our marketplaces are changing both in terms of availability, variety, constituents (e.g., source of the sweetener or oil), serving size and other key parameters. Therefore, the ideal situation is to characterize the probabilities of associations among our food choices. For example:

- What foods are frequently eaten together (burgers and fries)?
- What foods are frequently eaten for several consecutive meals (turkey, cake)?
- What foods are confined by ethnic or socioeconomic or regional factors?
- What do we know about food frequency, and related factors?

There exist several studies on these issues, created by marketing managers of food industries and by nutritionists, whose professional missions are to characterize the nutritional status of our population's subgroups. We have only begun to identify and utilize these data and information, but their importance will grow as the future USDA surveys have no time dimension to their survey design.[3] Thus, the time sequencing relationships will be forged using associations created from other databases. The USDA surveys will still have excellent information about the demographics of the people in the survey. From these data, we can create the linkages to the distribution of probabilities for repeat eating behavior and menu selections.

In this case study, LifeLine™ has utilized the USDA dietary survey data to create the dietary profiles over a lifetime by electing one of several possible approaches. This approach maximizes the use of the three-day associations and makes full use of the demographic data collected in the survey. The approach is as follows:

First, LifeLine™ sorted all USDA survey records for all people and all days with full demographics information into age and season categories. Their full three-day histories were kept associated (Figure 7). Second, each set of three records (sorted by year and season) were characterized in terms of their potential for causing exposure to the pesticide. This was done by calculating the three-day average dose associated with each record if pesticide residue occurred at their mean levels. Based upon this calculation, the three-day records were ranked and divided into quartiles. Third, each individual was randomly assigned to a quartile. Fourth, an individual's food consumption was defined by randomly selecting from the appropriate set of three-day records as defined by age, season, and quartile. One three-day record was selected for each season of the individual's life. The individual was assumed to repeat this three-day record approximately 30 times to complete each of the 90-odd days of a season.

In this way, a dietary record was assigned to each day of the individual's life. Once this was done, the food items in these records were used along with the recipes for the foods to

[3] At the request of the users of the USDA survey (those of us doing pesticide exposure assessment), the USDA redesigned their sampling plans to discontinue the use of three-day records. Only one-day records are available from the 1994 surveys onward.

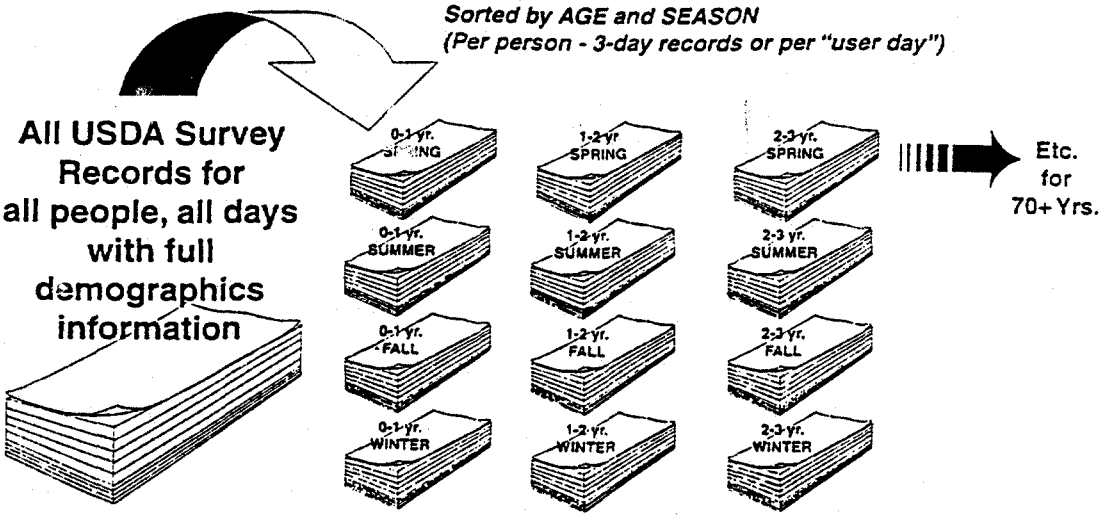

Figure 7. Creating a LifeLine ™ expsoure history with 3-day USDA survey data.

determine the intake of the RACs. The residues were randomly selected from the appropriate distributions of residue values or from the mean residue concentrations as appropriate.

This approach also has the advantage of retaining the demographic information in the USDA surveys. The data provides a means of linking the personal and household modules in the LifeLine™ model to the residential and environmental source information (Figure 8). This allows the exposure information to be applied correctly to an individual in a fixed situation. For example, a person residing in the northwest of modest means in an urban dwelling cannot suddenly get exposed to twelve-month exposures of lawn-care products for fleas.

While this approach was used in the LifeLine™ model for this project, other approaches can also be used. For example, the three-day records may be averaged or data on long-term averages from studies such as the National Human Exposure Assessment Surveys (NHEXAS) could be used to guide assumptions concerning long-term dietary patterns.

4. Using Additional Dietary Information

The USDA surveys also record "where and when" food items were consumed, or purchased in one location and eaten in another. This affords the opportunity to include more information about associations about eating behavior as it relates to institutions (schools, workplace, hospitals, etc.) and food chosen in commercial establishments (lunch delis near work, etc). Such patterns of eating become more important when the time dimension is introduced to the exposure profile. In addition to the possibility that the residues in these institutional foods may be different than those in the foods eaten at home, the possibility for repeated behavior (menu selection) may be conditional on an individual's activity pattern (e.g., time away from home).

5. Characterizing Tapwater Sources, Intakes, and Related Exposures

Dietary sources of exposure also include water in the diet and food. Water in the diet comes from:

- Water intrinsic to the foods that are consumed

  For example, the water in a piece of fruit contributes to an individual's overall intake of fluids. The pesticide residue in this water source comes from the residue information about the food itself.

- Water in foods, added to the food item during processing

  This water source is not usually the local household water source. It is the water added to a can of soda, the water in a can of soup, etc. In current models, this source of water is not

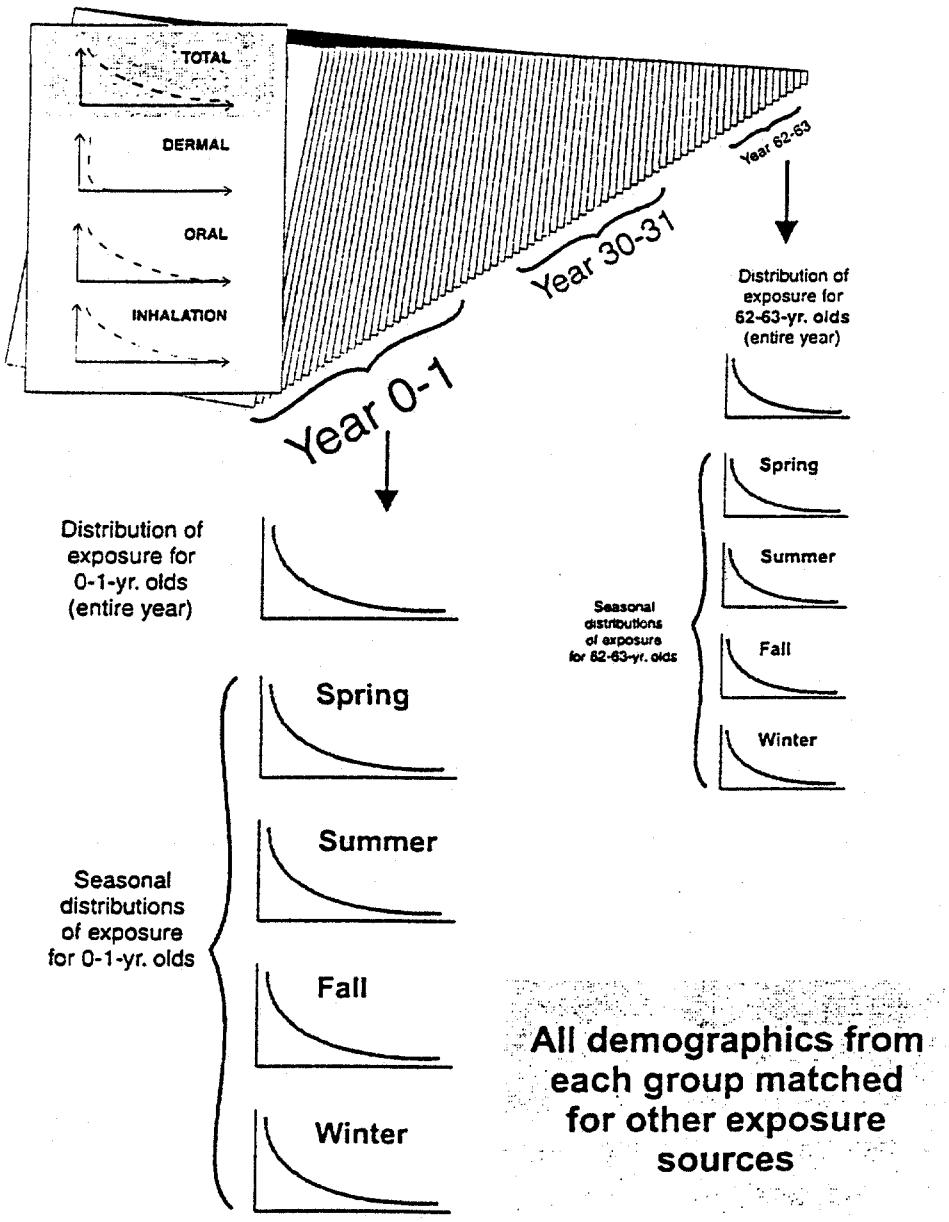
TOTAL
DERMAL
ORAL
INHALATION
Year 0-1
Year 30-31
Year 62-63
Distribution of exposure for 0-1-yr. olds (entire year)
Seasonal distributions of exposure for 0-1-yr. olds
Spring
Summer
Fall
Winter
Distribution of exposure for 62-63-yr. olds (entire year)
Seasonal distributions of exposure for 62-63-yr. olds
Spring
Summer
Fall
Winter
All demographics from each group matched for other exposure sources

Figure 8

addressed independently as a contributor to the overall pesticide load. The residues of the food ingredients in these food items are addressed.

- Water from the home tap

  Tap water contributes to two dietary elements. First there is the beverage component-water as a beverage or water added to beverage concentrates (the water added to concentrated juices or infant formula). This water may be directly from the tap, or heated as part of the preparation of the beverage (coffee, tea). Second, there is the incorporation of the tap water into foods during preparation-the water that is absorbed into spaghetti, or into Jell-O, for example. Usually this entails some type of processing such as heating. If these processing steps alter the nature or amount of the residue, this can be addressed as part of LifeLine™ since the processing steps are related to the form of the food eaten and the components of that food.

The source of tapwater is determined from data in the USDA database. Records of intake include the identification of the source of the tapwater to that household. As discussed above, tapwater concentrations change based on residential mobility. Seasonal variations in the region around the household may also dictate changes in the residues in a water source.

Note that tapwater sources also provide the opportunity for dermal and inhalation exposure via bathing and the off gassing of the chemical from the shower. These exposures are dependent on other factors such as the volatility of the chemical and the dermal penetration potential of the chemical. Presently, such calculations may be driven by one of two extremes:

- EPA policies to date do not require the assessment of dermal and inhalation exposures from this source, even though the calculations are relatively easy,
- When non-dietary exposures are calculated, EPA requires the assumption that there is 100% dermal absorption of the impinging chemical. This infers a large amount of the material in the shower water actually becomes a body burden - an assumption that may be hard to defend and one that may overwhelm the assessment.

These factors contribute to the exposure estimate and must be visible when considering the integrity of the final estimate of dose.

6. Results of the Application of the LifeLine™ Approach

i. Use of Exposure Histories

The result of the LifeLine™ approach is a set of exposure histories for a population. Each day in the population's history can be expressed as a distribution of exposures (total as well as from each source) from the individuals within that population on that day. The individual histories were created from all of the information available, utilized in the Microexposure event model using distributions from Monte Carlo applications which were random or truncated or bounded to reflect the associations or disassociations known from the data or required by default.

Thus the dose received on each day of an individual's life is characterized, without distortion from contrived analytical conventions such as "acute only" or "annualized only," as with previous models. The effect of temporal patterns of pesticide use can be readily seen in the histories, revealing inferences of frequency, seasonality and variation across time. The life history, now set up, is available to the toxicologist to explore for relationships which are meaningful to the queries posed by the toxicology tests and understanding of the mechanisms of action.

The display of an individual's exposure history is extremely useful for distinguishing between important and less important exposure sources. The LifeLine™ display identifies sources that determine the exposures and each source displays a distinctive temporal pattern. In some cases, the total exposure may not change significantly over time, but the toxicologist may be interested if the relative contributors from inhalation, dermal and oral exposures change significantly. The assessor can select a period of interest in the LifeLine™ and explore the details of that section. This defines the age groups of interest and may define other subpopulation characteristics of interest also.

Figure 9 displays a one-year period of an individual's life history where the usual exposure is dominated by the oral dose. For a brief period of time, however, the pattern of exposure changes dramatically. Dermal and inhalation exposures peak with a lesser, but significant, rise in oral exposure. This reflects the use of an indoor pesticide treatment. Activity patterns of the 4-year old provided ample opportunity for day-long inhalation and dermal exposure. Hand-to-mouth activity transferred surface residues to the child's mouth.

The toxicologist is now provided with a profile that displays patterns of acute and chronic exposure and provides choices for considering how risk will be assessed that are a function of the characteristics of the chemical's toxicological activity.

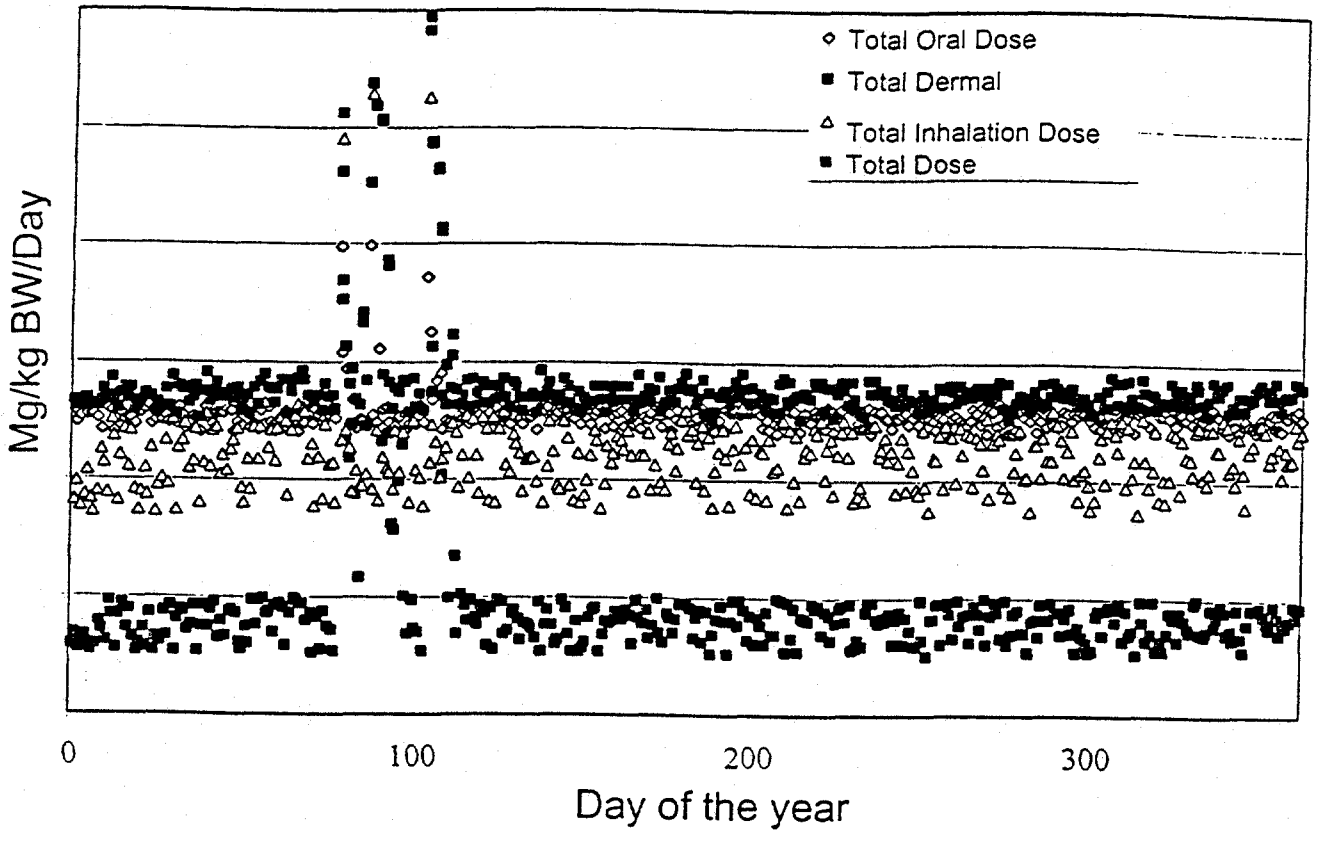

Figure 9. Total Dose and its oral, dermal, and inhalation components for the 4th year of an individual's life

ii. Characterizing Inter-Individual Variation in Exposure

Once the exposure histories are constructed, the assessor has several options for using them to characterize risk and explore the contributions to that risk. The most powerful of these options is the ability to view the exposure in a time dimension, matching it to the toxicological profile of interest. Briefly, the following are some of the available options:

(a.) Considering the Impact of Frequency and Duration of Exposures

Using the exposure history, the toxicologist can dictate the assessments most appropriate to the biological information available regarding the mechanism of action. If the toxic response could result from a brief excursion of exposure above a threshold, a pulse of exposure evident on a daily record could be appropriate to consider. In this situation, the peak exposures for individuals would be used in the final risk assessment.

If the toxicology suggests that the adverse effect results from exposures maintained above a given level over some duration of time, the toxicologist can direct LifeLine™ to conduct a running average for whatever time period is appropriate (3 days? 7 days? 30 specific estimates of dose? etc.) throughout the lifeline of each individual. These durations are portrayed for each individual and accrued into a distribution for the subpopulation, as was done for the one-day dose estimate. The toxicology defines the time period of inquiry. For cancer assessments, a one-year, multiple year or lifetime can be chosen for assessment. In addition, where the toxicological effect is limited to a particular age, the peak or appropriate time averaged dose can be determined for the age of concern.

Even if the toxicological effect is most appropriately based on a 1-day exposure profile, the frequency of an elevated exposure event can be inferred using the running average tool. The exposure profile of a 1-day, 7-day, 14-day, 30-day, 90-day and annual running averages can be compared. The differences between these assessments infer frequency. If there is little difference between the 1-day, 14-day and 30-day profiles, the occurrence of a peak of exposure is relatively frequent. If there is great difference between 30 and 90-day and seasonal profiles, there may be significant seasonal differences in frequency. Such associations are helpful to the toxicologist who might like to know how often the perceived risk may occur, and what factors contribute to that occurrence.

(b.) Consideration of Route of Exposure

The total dose for an individual or for a population is comprised of three routes - oral, dermal and inhalation. The toxicological significance of exposures by these different routes may not

be completely understood in most cases, and results from studies employing any one route may be used to infer toxicological significance by the other routes as well.

Relative contributions to the total exposure by the different routes of exposure may change during the lifetime. Oral exposures may be the major pathway during one period of life, only to be supplanted by inhalation or dermal exposure later in life. Total exposure and relative contributions to exposure may be an important factor to the toxicologist. Therefore, these assessments make that relationship visible, and the exposure profile can be based on the total exposure, with its contributing three routes, or it can be constructed exclusively using only one route.

The results of this profile can be used directly in the risk equation, or can be used to define additional data needs. If the major contributor of exposure is a route for which there is little direct toxicological information, a new study may be in order. If, on the other hand, the major exposure route is one that contributes little toxicological significance, the assessor can take this into account in the risk management process.

The key is the visibility of all routes of exposure, and the ability to examine the sources of which created that exposure opportunity.

(c.) Consideration of the Appropriate Population Subgroups for Closer Inspection

By creating the exposure histories of the individuals in an overall population, the lifelong exposure profile is automatically described. This obviates the need to choose population subgroups by age for specific consideration prior to the assessment. Upon examination of the initial profile, particular sections of the LifeLine™ can be extracted for further examination. This selection may be driven by an interest in the total exposure calculated for that time period, or because of the pattern of contributions by the different routes. This time period can be examined to determine what factors were important in the construction of the estimates of exposure.

The assessor can examine any period on the overall profile by extracting dose estimates for that time frame. This examination may infer significant contributions by particular sources or seasonal variation. If particular product use, frequency assumptions, or database was critical in the calculation, further examination of the population subgroup that may be impacted can be selected. Non-traditional descriptions of subgroups can be assessed. For example, the assessments may be defined for socioeconomic groups, urban or rural households, ethnic groups or regions of the country. As with any subdivision of total databases, the statistical "cost" of such subdivisions must be considered. Subdivisions that enhance error or bias must be identified. Therefore, LifeLine™ will present reports on the

size of the data bases on which the analyses were conducted, and guide the assessor as to the statistical issues raised by such subdivisions.

IV. APPLICATION OF LIFELINE™ TO THE ILSI DATA SET

A. DESCRIPTION OF THE ILSI DATA SET

The data provided by ILSI for this workshop test case represented an idealized composite data set, for a data-rich chemical with multiple potential uses yielding multiple routes of exposure. It is unlikely that any one chemical would accrue such a wealth of information, but these data afforded the opportunity to display the analysis concepts of aggregate exposure. Data were provided for a variety of exposure sources.

1. Food Residue Data

Residue data were provided on a variety of commodities representing three different sources of data: market basket data (commodities sampled at retail), field residue trial data, and tolerance levels. These different types of residue information represent the range of data typically available for estimating the concentrations of the chemical in the foods. These data were used in the Dietary Exposure/Tapwater-Intake Module.

2. Residential Use Data

ILSI provided information for a variety of uses of the chemical: termiticide, granular and liquid turf, indoor crack and crevice, broadcast carpet, and indoor total release fogger. Data include measurements of surface residues, dislodgeable residues, transferable residues, and air concentrations. These data are based on a variety of studies that measured actual residue levels and also simulated activity patterns in treated areas to estimate exposures to individuals wearing various clothing. Some scenarios even include information on the resulting residue levels on toys which were rolled across treated surfaces. These data are used in person and residence modules to ascribe activity levels, event modeling and associations of exposure opportunities across time. This data set was unusually robust; in most cases, the default factors would typically be utilized more frequently in the characterization of residential exposure.

While the data set was unusually rich in information on the levels of residues found in air and on surfaces following applications, it did not include information on the use patterns for the pesticide. Therefore, we developed a number of assumptions for residential use of pesticide. These include:

- Termiticides are used in single dwelling homes once every 10 years;

- One half of all homes do not use a pesticide (fogger, broadcast, or crack and crevice) in any given year;
- The remaining half of all homes use such product 1 to 6 time a year;
- Pesticides are used more frequently in the kitchen and bathroom than in other rooms;
- Turf insecticides are used only in the spring and only on one third of all lawns; and
- The market share of the pesticide was 10%.

3. Drinking Water Data

ILSI provided data from monitoring studies of both private wells and community water supplies. Data from private wells represent a worst case estimate of residues in drinking water: these data were collected only in areas where the product was used and the ground water was vulnerable (i.e., due to soil type and depth of the aquifer, there was a high likelihood of contamination). Data were collected both on raw and finished (processed) water samples from community water supplies. The community water was from three types of sources: ground water, surface water, and blends of both ground and surface water. In addition, for each of the various sources of drinking water, information on the location of the water supply and the number of people served by each type of community water supply was provided. The data provided by ILSI were used to estimate pesticide concentrations in tap water for various classes of households. Data for community water supply systems were provided for 21 states that account for more than 90 percent of the pesticide of interest; the pesticide was assumed to be absent from the tap water of households in other states. The data on concentrations in finished community water supplies were used to generate a distribution of tap water concentrations for households that use community systems in the 21 states. Similarly, the potable well water monitoring data were used to generate a distribution of tap water concentrations for households that use private wells in the states from which data were provided.

B. Use of the ILSI Data Set to Characterize Aggregate Exposure

LifeLine™ was used in three ways to evaluate the aggregate exposure defined by the ILSI data set. First, the model was used to characterize exposure histories that occur from the various sources of pesticide exposure. These histories are powerful tools for evaluating the relative significance of each of the routes of exposure and the uses that contribute to that exposure. They also provide insight to the frequency and temporal patterns of exposures that occur from residential and turf uses of the pesticide. Finally, the histories demonstrate how exposures varied with the age of the individual. Second, LifeLine™ was used to characterize the distribution of a variety of different measures of exposure across the general population. These include 1-, 14-, 30-day annual and lifetime average daily doses (LADDs). Third, the model

was used to explore the sensitivity of the dose distributions to different assumptions concerning market share or the fraction of the nation's wells that were contaminated.

1. Using Exposure Histories to Determine the Relative Importance of Different Sources of Exposure

The following two figures (Figures 10 and 11) present selected years from the exposure histories of two people. These figures provide a picture of the temporal patterns of aggregate exposures by route. When the pesticide is not used in an individual's residence in a given year, and when it does not occur in the individual's tapwater, food is responsible for the individual's total exposure (Figure 10, 0-1 years). This source of exposure is highly variable. In our analysis, the exposures differ by orders of magnitude on a day-to-day basis and can exceed 1 mg/kg/day. When tapwater is contaminated, exposures occur by all three routes. Unlike food-related exposures, tapwater exposures are relatively constant from day-to-day. This is because tapwater intake rates and tapwater concentrations are relatively constant. In the current data set the doses received from direct ingestion and indirect dermal and inhalation pathways are orders of magnitude lower than the dose from food exposure.

Usage of pesticides in residences or on turf results in oral, dermal and inhalation exposures (Figure 10, Years 3 and 12) on the day of application and for subsequent days. When pesticide residues in air and on surfaces decline rapidly (greater than a 20% decline per day), the applications result in significant exposure for only a few days. Consequently, the exposures from broadcast, crack and crevice, foggers and turf applications appear as brief spikes. However, the use of a termiticide differs from other pesticide uses in this example. Because termiticide slowly off-gases into indoor air, it results in prolonged inhalation exposures and appears as a prolonged elevation of inhalation exposure (Figure 10, Year 12 and Figure 11, Year 3).

On certain days, the turf and residential uses can make a significant contribution to the individual's total exposure and may even be the dominant source. However, residential and turf uses only dominate when the food exposure is relatively low. When food exposures are high, they dominate total exposure whether or not there is a concurrent exposure from residential or turf pesticide use. Therefore, when evaluating peak daily exposures that occur over an individual's life, residential use of pesticides has little impact in this case study.

Figure 12 presents a characterization of the variation of aggregate exposure with the age of the individual. As the figure indicates, the range of daily doses is elevated for children. This occurs because of age-related changes in the diet, and to a lesser extent, elevated dermal and oral intake of pesticides from surfaces by children.

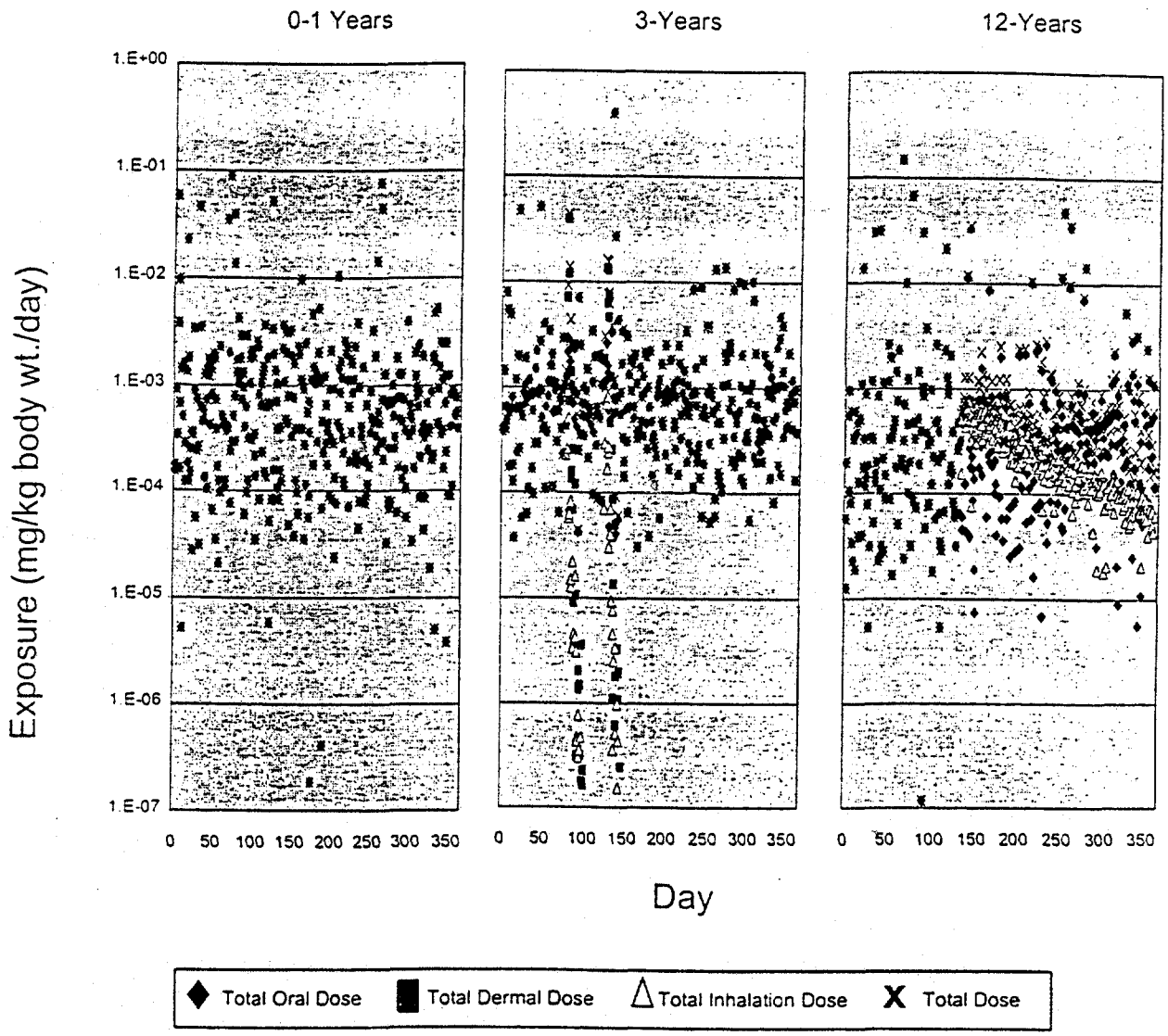

Figure 10. Total oral, dermal, and inhalation daily doses for a typical individual at birth, 3, and 12 year

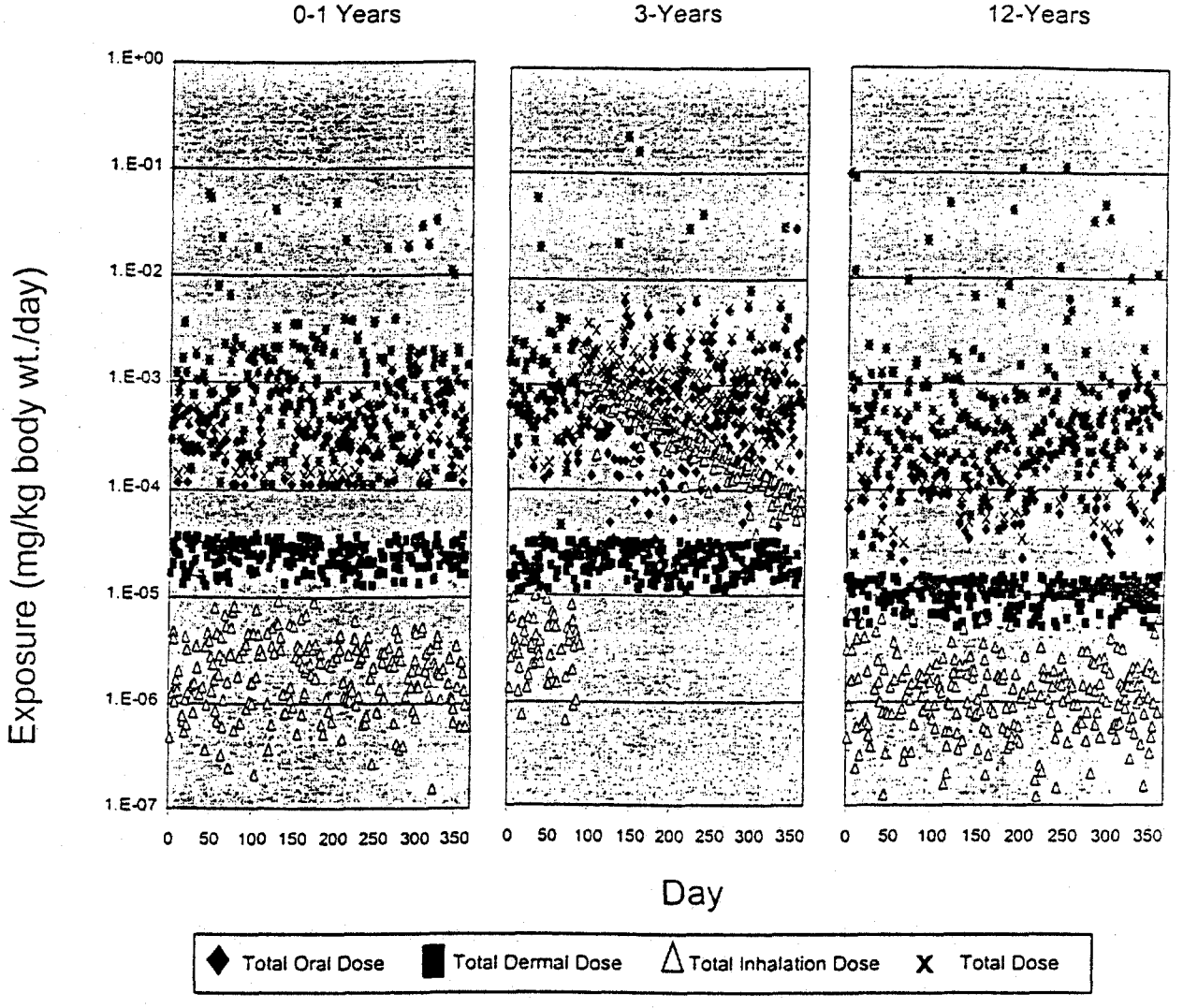

Figure 11. Total oral, dermal, and inhalation daily doses for a typical individual at birth, 3, and 12 years

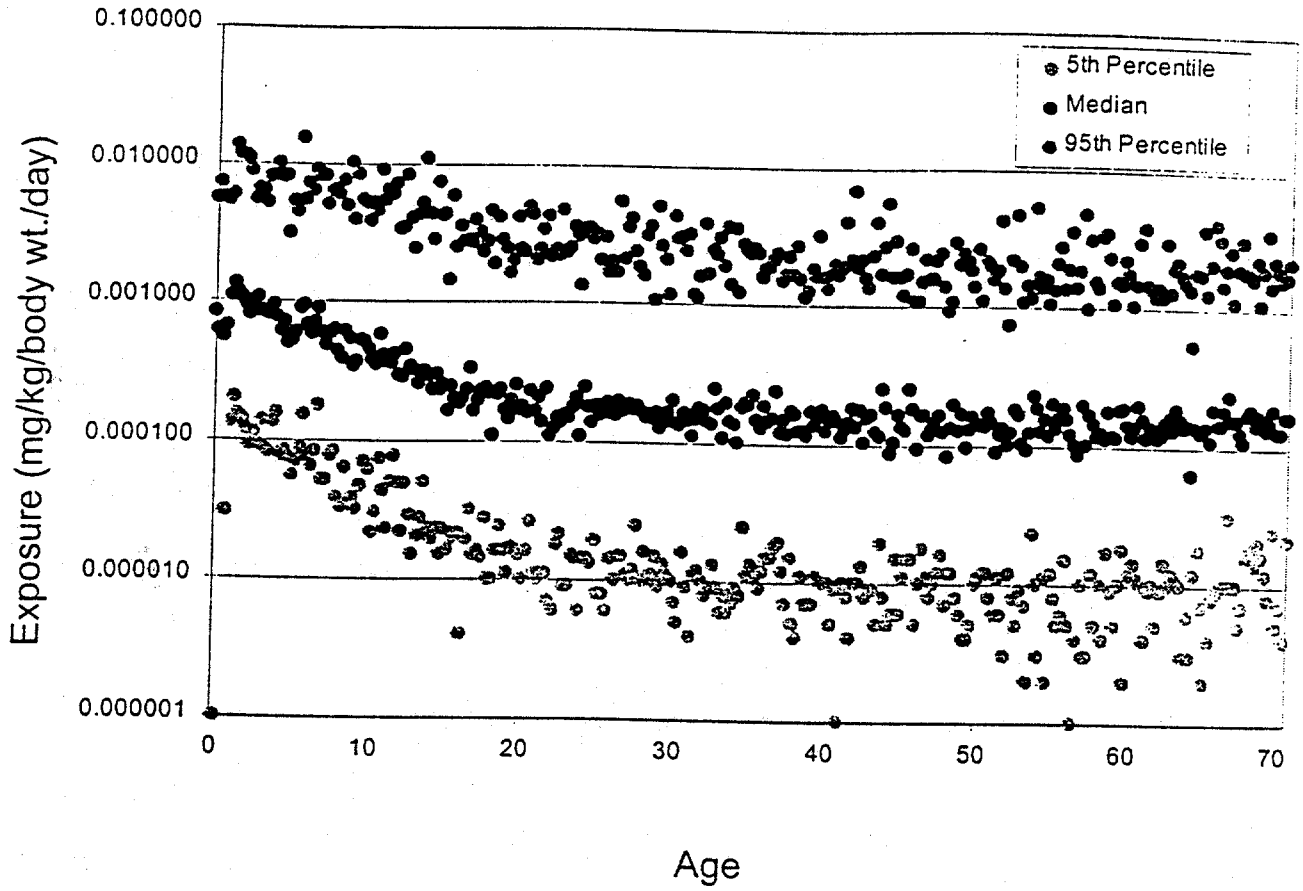

Figure 12. Range of daily doses by age

2. Determining Distributions of Doses from Different Durations of Exposure

The second type of output from LifeLine™ is the distributions of the average doses associated with different durations of exposure across the population under evaluation. We have chosen to assess 1-day, 14-day, 30-day, annual and lifetime average daily doses (LADDs).

A lifetime exposure is really an assemblage of an individual's short-term exposures. (For example, the individual will have over 25,000 separate daily doses). In order to evaluate inter-individual variation it is necessary to define which of the many short-term doses from a person's exposure history will be evaluated. In this analysis, we have reported the highest of the predicted short-term exposures that happens at any point in an individual's exposure history. However, there is no reason why other values such as the median, 95th or 99th percentile, or randomly selected short-term exposure could not be used. Because the highest exposure from each exposure history is used, the distribution of 1-day exposures should be viewed as the distribution of the highest aggregate exposures that occur on any day in the lives of each member of the subject population.

Next, running averages of exposures in consecutive 14-day periods were computed for each individual and all individuals of the population. The highest 14-day exposure that happens on any consecutive 14-day period in the individuals' lives were selected. Similarly, the 30-day and annual exposures refer to the highest exposures that happen over these longer periods. Consequently, the distributions reflect the peak doses that occur in children, as desired in the spirit of FQPA.

The highest daily exposure experienced by the 99th percentile of the population on any day of their lives was 2 mg/kg/day. However, as seen in Figure 13, increasing the averaging time from 1 day to 14 days reduces the exposure received by this high end of the exposed populations by a factor of 10. This difference indicates that high exposures were infrequent. Increasing the duration of the exposure to an annual exposure reduces exposure by two orders of magnitude. Finally, the LADDs for the high-end of the exposed population are three orders of magnitude below the highest 1-day exposures. The lifeline portrays a profile of long term, low-level exposures with infrequent episodes of exposure peaks. The time profile most relevant to the toxicological issues can be chosen to represent the actual risk for that population.

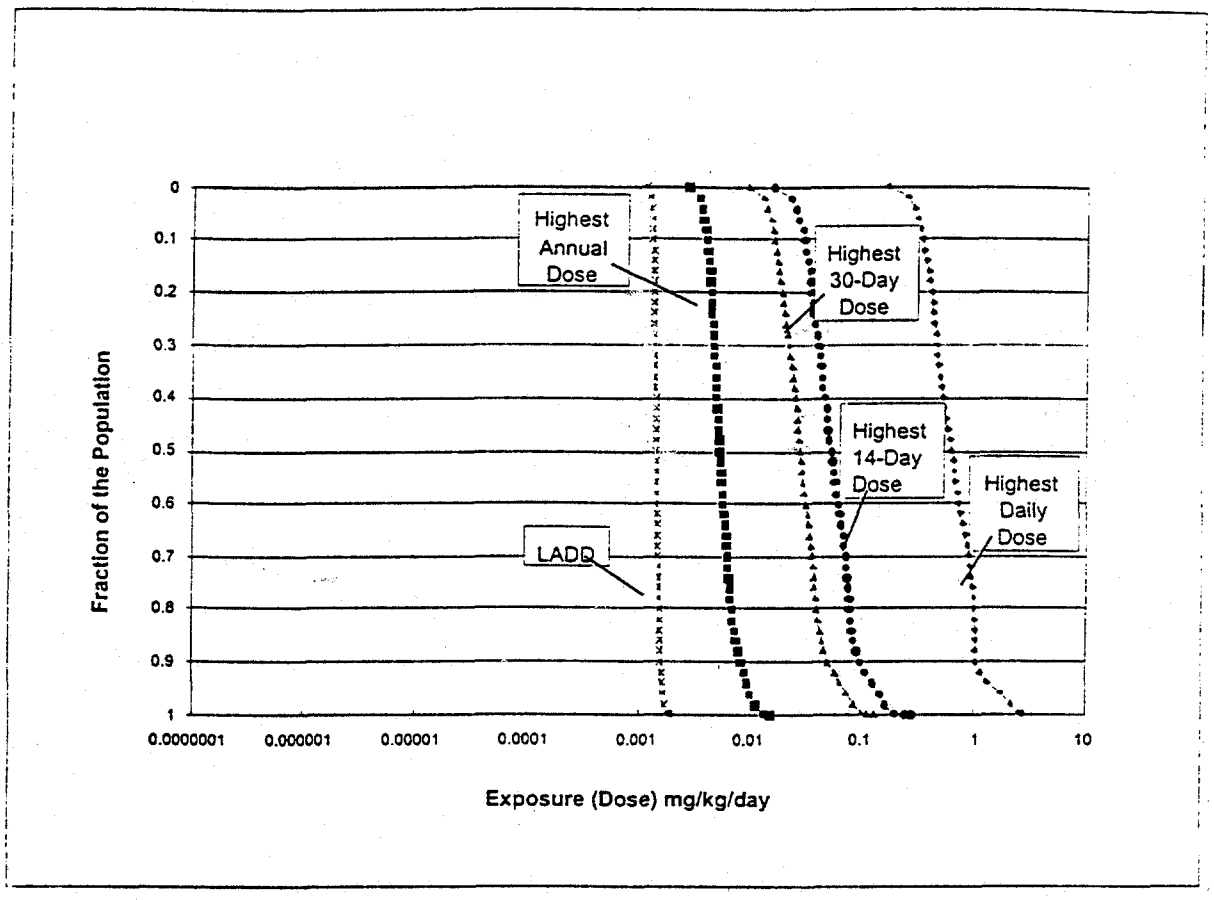

Figure 13. Distribution of aggregate exposures in the general population

3. Examining the Contributions to the Total Exposure

The remaining Figures 14, 15 and 16 examine the impact of the non-dietary sources of exposure and the sensitivity of the results to important factors that determine the frequency of exposure. Because dietary exposures dominate aggregate exposures for the ILSI data set, we have set dietary exposures to zero in this sensitivity analysis for non-dietary sources. As Figure 14 indicates, non-dietary sources result in exposures that are approximately an order of magnitude lower than the dietary exposure for the upper percentiles of the population. In addition, with a 10% market share and only 0.7% of households tapwater supplies affected, LifeLine™ shows that approximately half of the U.S. population are not exposed to non-dietary sources of the pesticides at any time during their lives (Figure 14).

In this assessment we have assumed that the pesticide represents a 10% share of the market. Figure 15 demonstrates the impact of changing the market share to 100%. Under this assumption, the entire population is exposed to the pesticide at some point in their lives, but the increase in exposure is not directly linear. At the high end of the population the exposures are elevated by a factor of 1.5 for the 1- and 7-day exposures and by a factor of 3 for the annual exposures and LADDs.

In comparison to Figure 14, Figure 16 presents the impact of changing the fraction of households with tapwater contamination to 100%. Since tapwater related exposures result in low doses, changing the assumption that all houses will have contaminated water sources has little or no effect on the 1- and 7-day aggregate exposures of people who use pesticides at their residences or on turf grass (bottom half of figure). However, the LADDs were elevated by a factor of 3. While the exposures are low in comparison to residential and turf uses, they are much more frequent and make a significant contribution to lifetime exposures. Finally, individuals who do not use home or turf pesticides (top half of figure) are projected to see very little difference between one day and lifetime exposures. The small differences between these analyses of different exposure periods demonstrate the exposure is constructed from relatively frequent exposure with little intra-person variation (few episodic peaks).

C. Summary

Use of LifeLine™ to characterize aggregate exposure gives insight into the temporal patterns of exposures in the lives of individual people and the distribution of various measures of exposure across individuals in an exposed population. LifeLine™ allows the use of alternative assumptions to identify those factors that are critical for the evaluation of aggregate exposures. In the ILSI data set, the highest 1-day exposures were 2 mg/kg/day for the 99th percentile of the general population. In contrast, the highest 14-day dose was 10 fold lower and the LADD was 1,000 fold lower.

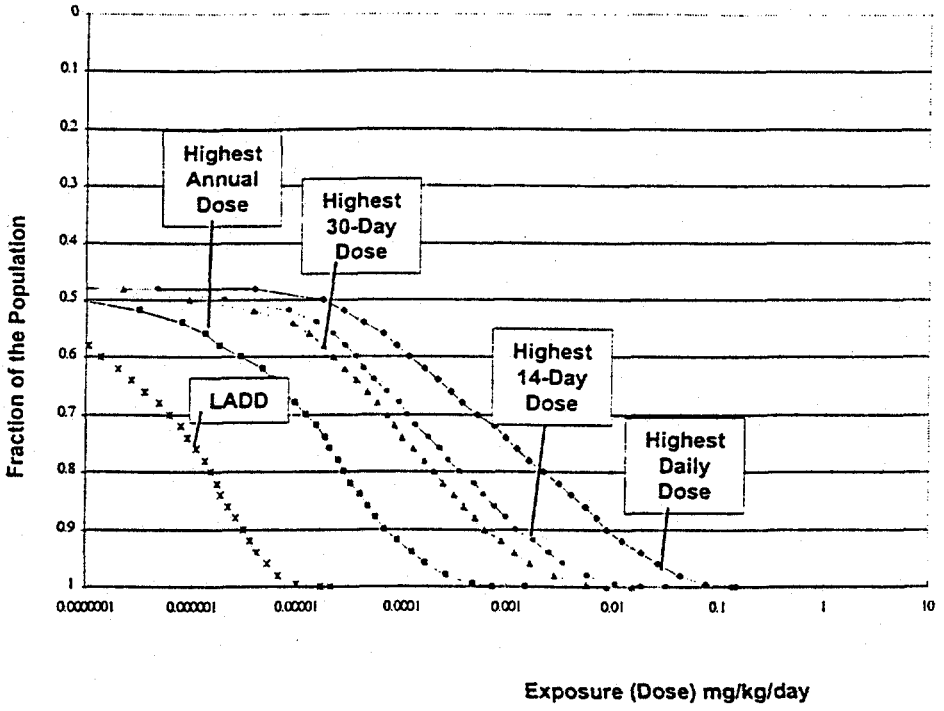

Figure 14. Distribution of non-dietary exposures in the general population

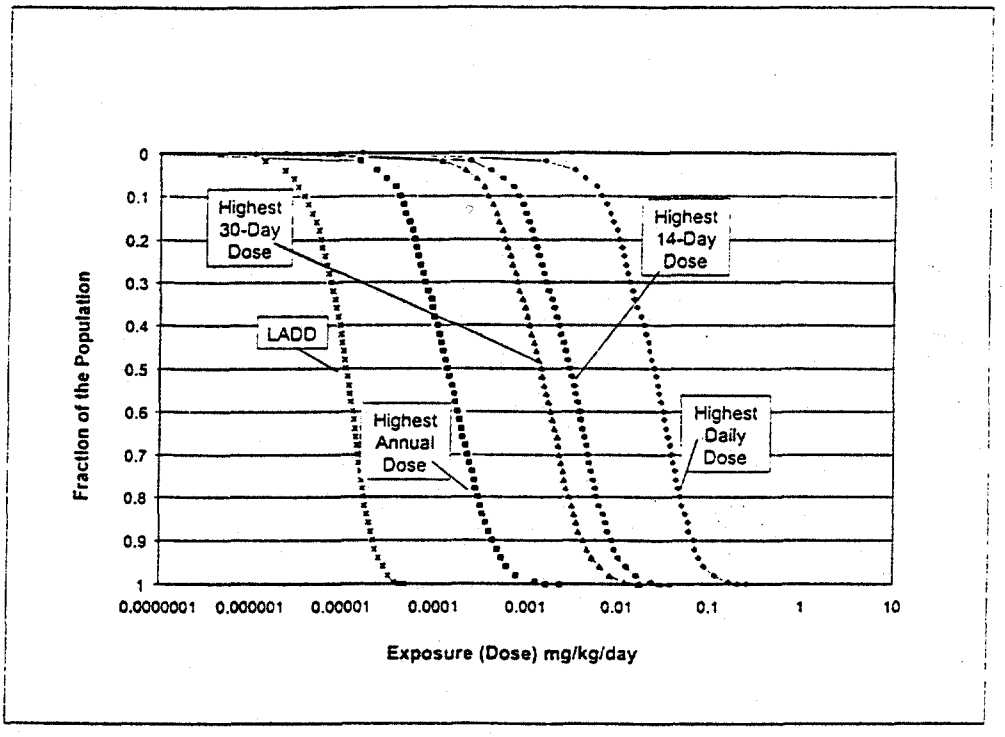

Figure 15. Distribution of non-dietary exposures for 100 market share in residential pesticides

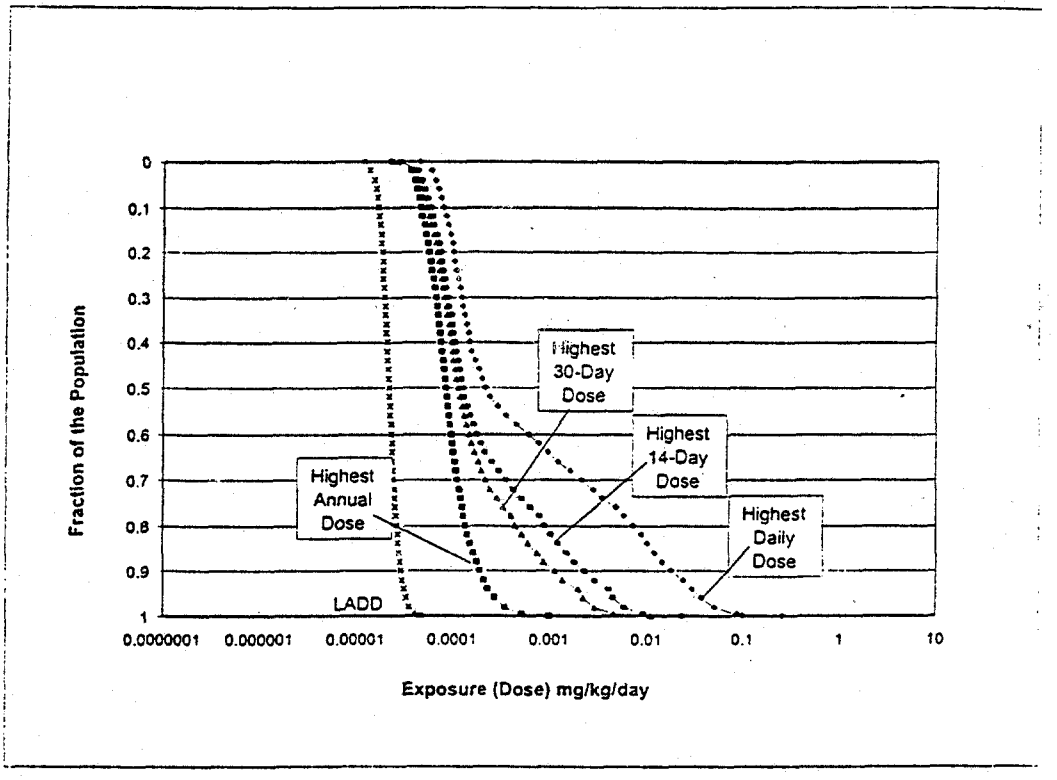

Figure 16. Distribution of non-dietary exposures of all tapwater supplies contaminated

V. DISCUSSION

There are a number of issues that are raised by the aggregate exposure assessment and the use of LifeLine™.

A. How Does the Approach Deal with Variability and Uncertainty in Exposure Estimates?

The LifeLine™ approach is focused on characterizing variability in dose. This is done by constructing exposure histories for individuals whose variation is an accurate model of the interpersonal variation in the population being assessed. This approach provides an objective means of characterizing the upper end of the exposure distribution that can not be achieved by merely adding worst case assumptions (EPA, 1992).

While the models focus on variation, they also provide powerful tools for evaluating uncertainty. By using alternative assumptions, the model can demonstrate the sensitivity of the predicted exposures to alternative values. For example, we have used microexposure event models to investigate the uncertainty in estimates of long term (greater than one year) fish consumption rates (Price et al., 1996).

However, it is possible to use the modeling approach in LifeLine™ to explicitly model uncertainty in dose estimates. This could be done by selecting an individual and using distributions of exposure factors that reflect the uncertainty in the values of those factors for the individual. Model runs of this individual's history would generate a distribution of equally plausible exposure histories. This distribution would provide a powerful description of the uncertainty in the individual's exposures.

B. Model Uncertainty and Bias

Models of exposure introduce uncertainty in exposure assessments. Modeling uncertainty can be evaluated in two ways, sensitivity analysis and model verification. As discussed above the LifeLine™ approach can be subjected to sensitivity analysis that demonstrates which factors are critical to the prediction of the relevant estimates of exposure. These factors can then be the subject of additional investigations. In addition, the approach is sufficiently flexible that alternative modeling assumptions can be investigated and the impact on the relevant exposure estimates determined.

LifeLine™ offers unique advantages in the area of model verification. Because the modeling approach places a large emphasis on defining the characteristics of the exposed individuals, the output of LifeLine™ makes predictions concerning the correlation of exposure with population characteristics that are readily verifiable. For example, it is possible to predict the

distribution of average weekly aggregate exposures for children aged 5 and 12 in a specific population. These estimates can be directly compared to measurement of pesticides or pesticide metabolites in biomonitoring surveys. In fact, it is possible to use LifeLine™ to mirror the population in a survey and generate estimates that are directly comparable to survey results.

C. Communicating to the Public

Over the last 15 years, risk communication has been identified as a critical component in the successful management of risk. LifeLine™ has several characteristics that greatly assist in risk communication.

First, LifeLine™ is an available software system. The software is available to the general public and can be used by local and public groups. In addition, the system is open and verifiable. All inputs to the model and model assumptions are disclosed and can be modified by the user. Third, the model incorporates many of the characteristics of "real life". The individuals in the model are born, age, and move from home to home in an understandable way. They are not locked into performing the same "hypothetical" or "abstract" exposure scenario every day for 30 years. Fourth, the model produces estimates of exposure (Figures 10 and 11) that can be useful in risk communication. These figures convey information on the intensity and route of exposure, the relative importance of the routes and sources of exposure, and how exposure change on a day to day basis.

D. Identifying and Dealing with "Outliers"

Distributions may have values that describe a "long tail" and are distant from the mean of the distribution. There are at least three reasons for this profile, each having its own different implication for the exposure and risk assessment.

1. The "tail" is actually a different distribution a unique population

The values in the tail may share something in common with the body of the distribution, but it could be considered a unique distribution unto itself. For example, if exposure to a chemical in water were being evaluated, the exposure from dermal exposure would be relevant. We might consider bath water, shower water and pool water. The time spent in the shower or in the bath (consider those long relaxing baths) or in a pool would describe a distribution which has a tail at four hours per day. This tail may actually be describing the activity of competitive swimmers in their daily training. While these people, too, are important to consider in a safety evaluation, it may be constructive to do two assessments' one for the general population without them included, and one uniquely for the swimmers.

Where there is opportunity for seasonal, ethnic, regional or other segmentation, the assessments may be more meaningful if they are subdivided to examine the contributing elements of each assessment.

2. The tail is defined by exaggerations or clear bias and error

Some data will contain errors or exaggerations that may be further amplified by weighting factors and default factors, creating a high value in theory where it is unlikely that such values truly exist. In such cases, a "reality check" with other confirmatory information is in order to give the assessor the opportunity to discard these elements of the distribution with the mandate that such actions be documented and defended.

3. The "tail" is truly a distant element of the valid distribution

In this case, the distribution is valid, and without cause to disconnect or dismiss the high values, they must remain as part of the distribution.

Characterization and dealing with the distributions in this way present a valid array of options to the risk manager. The model should not be constrained by arbitrary limitations on dealing with the distributions and defining the scientifically appropriate array of answers to the risk manager. Valid statistical practices can be required here, and such practices are well described and available to the assessors in many statistical references.

E. Issues Related to the Food Residue Data and Assumptions

1. Use of "Percent Crop Treated"

- The tradition of using an estimate of what percentage of the crop was treated is a relatively crude technique for "correcting" the exposure assessments done in previous models. It was generally acknowledged that not all foods are treated with all pesticides. Assessments were done for one chemical at a time, considering only one source of exposure at a time. Crude corrections were useful, especially in the "chronic" assessment models. This information could still be useful in an assessment built with microexposure event modeling, but it is only one piece of information which describes the opportunity for a food item being eaten in a given time frame to have residues of one or more pesticides. Other information can contribute to this assessment also:

- What is the use profile of this chemical and what influences its use-- pest pressures, climate, season, region? Do we have field residue data that is matched to these key influences of use? Should sub-trials data be used individually rather than one complete field residue distribution?

- What chemicals compete? Are these chemicals frequently found on the same commodity?

- What is the efficacy of the chemical under the different influences of use? Based on this information, should one assume maximum label use and maximum frequency under all circumstances (the most conservative assumption), or use efficacy data to correct the assumption to reflect actual use, especially in comparison to its competitors on the market. In this case, the efficacy data translates directly to a quantitative element of the exposure assessment--the frequency of exposure.

- What is the magnitude of the residues when the chemical is used alone or in combination with other pesticides, simultaneously or sequentially? These residue distributions may be quite different than one presented by the field residue trials (for only one chemical at time), and wherever possible, the interrelationships of these multiple-chemical residue distributions should be understood.

Percent-crop-treated is a crude tool when multiple chemicals are considered. The percentages for the different chemicals may overlap, or may compete. One will not know how to apply these directly in such circumstances without guidance from auxiliary information. All data should be used in the best way possible, without blind dictates. The rule, which should guide the assessor, should be "*which data or defaults are most likely to be least wrong?*" The assessor's choice when applying this rule is subject to debate, so the decisions should be documented and defended. But all information should be captured and considered. If data are rejected and defaults used instead, that decision should be scientifically defensible also (i.e., the assessor cannot settle for "the government made me do it" or the traditional "we've always done it this way"). Sometimes surrogate data will be better than the options of "no data at all" or default values.

2. Tapwater Contamination

The most important factor in determining the exposure to tapwater from oral, dermal and inhalation pathways is the presence (or absence) and concentration of pesticide residues in the household water supply (HHWS). Because of this, aggregate exposure assessments should address exposures resulting from household (or domestic) use of tap water, rather than focusing on drinking water alone. In this case study report, the term "drinking water" refers to water used for drinking and other dietary purposes (including food preparation), which is expected to account for the great majority of the exposure associated with residues in the HHWS for most pesticides.

Pesticides can migrate from the point of application to ground water or surface water bodies by a variety of pathways, and may eventually reach waters used as sources of HHWS. Generally, the

most significant pathways include percolation downward through the unsaturated soil zone to the ground water table (generally in dissolved form), and overland transport by surface runoff. In some cases, direct application to surface waters (generally inadvertent, as in the case of aerial spray drift) may also be important. Pesticides in surface runoff may be transported in either a dissolved or particulate form (usually sorbed onto small particles of soil or organic material). Pesticides found in HHWS are generally present in dissolved form, but in some cases may be sorbed onto very small (colloidal) particles. The distinction between these two states can be important in estimating the absorbed dose of a pesticide.

The relative importance of the various migration pathways depends on such factors as the physical and chemical nature of the pesticide; the method of application and conditions of use; and the characteristics of the area in which the pesticide is applied, such as soils, hydrology, and climate. The important characteristics of the pesticide include its solubility, sorptivity, and persistence under various conditions encountered in the environment; these characteristics are generally estimated in laboratory experiments that are conducted before the pesticide is proposed for registration. The method of application (e.g., sprayed from an airplane versus broadcast and disked in to soil by a tractor) may determine the degree to which the pesticide is incorporated into the soil at the time of application. The form of the pesticide as applied (e.g., as granules, wettable powder, or an aqueous solution) may affect uptake by plants and migration from the area of application. The rate of application (i.e., the amount of pesticide applied per unit area) and timing of the pesticide application relative to the growth stage of the crop and the season of the year may also affect uptake and migration.

Both natural and human characteristics of the area in which the pesticide is applied may be important in determining the presence/absence and concentration of pesticides in sources of HHWS. The important natural characteristics include soil types, geomorphology and hydrogeology, and climate; in concert with human activities, these factors affect the rates of ground water recharge and surface water runoff. Important human characteristics include agricultural practices (such as crop selection, pesticide use, and soil amendment and tilling practices) as well as such general characteristics as population density and sources of water supply. While agricultural practices may vary significantly from one field or farm to another, most of the other important characteristics (e.g., climate and population density) are regional in nature. These variables define regions similar to the Land Resource Regions and Land Resource Areas defined by the USDA, which do not generally coincide with state boundaries.

In order to conduct a complete exposure assessment, it is necessary to characterize the extent to which pesticide residues will appear in HHWS as a result of the uses for which registration is sought. In the case of a pesticide that has been in use for many years, data (i.e., actual measurements of the concentration in water supplies) may be available for this purpose. The ILSI case study is atypical in that very little data are available for characterizing concentrations in HHWS for most of the pesticides for which an aggregate exposure assessment is required by

the FQPA. Even in this case, however, the data represent the results of past use and may not be representative of the results of future use patterns. In the more general case (i.e., for new pesticides and new uses of existing pesticides), direct measurements of the concentration of the pesticide residues of interest in HHWS will not be available. In this case, the extent to which pesticide residues will appear in water supplies may be estimated using models. Ideally, these models should be capable of reflecting differences in the characteristics of the pesticide and the proposed conditions of use.

Two general approaches based on modeling are available; these approaches are not mutually exclusive, and elements of both approaches may be applied to specific cases. One approach involves environmental fate and transport modeling; one or more critical scenarios are defined, and chemical fate and transport models are used to forecast the concentrations of pesticide residues in HHWS that might eventually result from a specified pattern of use. The second approach depends on the availability of data (preferably, direct concentration measurements in HHWS) for pesticides with physical and chemical characteristics similar to those of the pesticide for which registration is sought. In any specific case, these two approaches may be used in complementary fashion to characterize the extent to which pesticide residues will appear in HHWS as a result of the uses for which registration is sought.

VI. RECOMMENDATIONS

Based upon the ILSI aggregate assessment project, the team has developed the following recommendations:

- Aggregate and cumulative exposure assessment should employ a model that is based on a dose and time dimension display for total and all contributing exposure sources. It should be relevant to the toxicology and its structure should be transparent, permitting an understanding of the influence of data, defaults, assumptions and algorithms on the answers.

- The risk assessment models should be available, accessible and useable to all interested parties. Then, the logic and construction of the assessment should be visible to all when the answers are presented.

- All default values and traditions should be re-examined to quantify or characterize the bias, error, uncertainty, relevance and representativeness of the factor. Whenever data or any information is available, it should replace the default value if it can be shown that the data are less biased or contain less error or uncertainty, or are more relevant or representative. This policy should guide the use of information even less-than-perfect information.

- Better approaches must be found to anticipate the opportunity for drinking water contamination and the magnitude of the contamination that could occur in these drinking water sources. The focus must be on better fate and transport models and better definition of water sources to the household or commercial food preparation establishment. Source of water could be considered a population subgroup for the risk analysis. Water should be treated as part of the diet in the assessment well as a contribution to the residential exposure.

- Time-linked dietary patterns should be studied, first using the available information on frequency and patterns of menu selection at home, in institutions and in commercial settings. The options for using present and future consumption surveys should be examined. The options should then be utilized in the evolving exposure assessment methodologies.

VII. FINAL THOUGHTS

The LifeLine™ approach is intended to provide both EPA and registrants with a flexible and powerful tool for assessing exposure to pesticides and other substances in food, water, and environmental media. In developing LifeLine™, the team had the following goals. First, design a system that takes advantage of all of the available information relevant to exposure assessment, including; information on product use, household or personal factors, residue levels and degradation kinetics, etc. Second, develop a system that can provide the toxicologist with the information on durations of exposure that match the durations in animal studies. Third, define the population (or individual) exposure history in terms of total dose and dose by the dermal, oral, and inhalation routes. Fourth, provide the toxicologist with estimates of exposure for all age groups. Finally, provide the basis to allow the ready identification of the data, default assumptions, and algorithms that are critical when assessing individuals with the highest rates of exposure. We believe that the modeling approach in the LifeLine™ software meets these goals.

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Use of MicroExposure Event and Toxicokinetic Modeling to Estimate Polychlorinated Biphenyl (PCB) Concentrations in the Blood of Anglers Who Consume Contaminated Fish

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ABSTRACT

Human health risk assessors have traditionally estimated risk by comparing long-term dose rates received from exposure to environmental contaminants to toxicity criteria, such as risk-specific doses and reference doses. A refinement of this method evaluates the potential for environmental exposure to increase contaminant levels in blood and then compares the elevated levels to those associated with toxic effects in animals.

In this study, the distribution of PCB concentrations in the blood of anglers who consume contaminated fish was estimated. PCB concentrations in the blood are dependent on exposure and toxicokinetic considerations. To this end, we used a MicroExposure Event (MEE) model to characterize an individual's long-term dose rate (exposure) as the sum of doses received from separate exposure events. A major advantage of this approach is that it allows the modeling of time-dependent factors, such as short-term variation in exposure and temporal variations in the behavior and characteristics of the exposed individual. The toxicokinetics of PCBs were described using a simple compartmental model that included estimates of PCB half-life. This analysis demonstrates the utility of merging a basic toxicokinetic (TK) model with a MEE model to describe the long-term concentrations of highly lipophilic and persistent compounds in blood as a measure of internal dose.

INTRODUCTION

MEE models of exposure estimate long-term dose rates by viewing exposures as a series of events that vary over time. These models have been used to evaluate human exposure to chlorinated solvents in contaminated groundwater (7) and PCBs and dioxin in fish (9). In this study, a toxicokinetic model is linked to a MEE model to estimate PCB blood levels in anglers consuming contaminated fish. The result of this model is a life history of daily PCB blood concentrations for each angler. These predicted PCB blood concentration histories can be compared to internal dose measures (peak, lifetime average concentration, lifetime area under the curve [AUC]) known to cause toxicological effects in animals.

A hypothetical population of adult recreational anglers was evaluated in this study. The MEE model was constructed based on published information on angler behavior and the toxicokinetic properties of PCBs.

OBJECTIVES

The model was used to investigate the following issues:

- ☐ Does fishing one season versus year round affect PCB body burdens in anglers?
- ☐ Does eating PCB contaminated fish result in PCB blood concentrations above background levels of PCBs in the general population?
- ☐ What effect does averaging time have on the distributions of PCB blood levels in the population?

MODELING APPROACH

MEE models view an individual's long-term exposures as a series of discrete events. In this study, an exposure event is defined as a single meal of fish caught from a contaminated fishery. The MEE model was used to simulate two thousand angler "life histories" to develop distributions of lifetime concentrations of blood PCBs (internal dose measures) and total cumulative doses (exposures) for the assessment of noncancer risks. The model structure is summarized in the following two sections: Characterization of Exposure and Characterization of Toxicokinetics.

Characterization of Exposure

The model input distributions are presented in Table 1 and the model structure is outlined in Figure 1. PCBs from fish consumption and background sources were considered. Simulation of each angler begins by assigning the angler lifetime characteristics (13). The model then determines how many fish meals an angler consumes in a season and which days the meals are consumed. The model simulates every day of an angler's life. On days when fish are consumed, intake of PCBs from both a fish meal and from background sources are inputs to the model. On days when no fish are consumed, only doses of PCBs from background sources are considered. The distribution of PCB concentrations in fish was arbitrarily selected so that 15 percent of the anglers would receive long-term doses of PCBs from fish consumption at or above the reference dose (RfD) for Aroclor 1254 (8).

Characterization of Toxicokinetics

The accumulation of PCBs in an angler is described using a single compartment toxicokinetic model (see Figure 2). The following were assumed to account for PCB exposure (dose), distribution and elimination:

Dose

- Fish consumption (discrete daily fish meal events), and
- Background intake rate of PCBs.

Distribution

- PCBs distribute only within the lipid fraction of the body.

Elimination

- First-order elimination kinetics.
- A single value of half-life used for all congeners.

Table 1. Input Distributions Used in the MEE Model of Anglers

Model Inputs	Distribution Type	Description	Source
Fish consumption rate (kg/day)	Cumulative	min = 0.000023; mean = 0.0064; max = 0.22	(2)
Body Weight[a] (kg)	Cumulative	min = 58.4; mean = 77.2; max = 101.7	(5)
PCB concentration in fish (mg/kg)	Lognormal	mean = 0.1; stdev = 0.04[b]	
Gender	Discrete	male(0.85); female(0.15)	(11)
Background PCB concentration in blood (ug/kg)	Lognormal	min = 2; mean = 4.9; max = 30; stdev = 3.5	(4)
Fraction of lipid in the body			
female	Custom	0.063 + 0.0032*body weight + 0.002*age	(10)
male	Custom	0.088 + 0.0032*body weight + 0.0023*age	(10)
Start age (years)	Truncated normal	min = 5; mean = 33; max = 80; stdev = 13	(14)
Fish meal serving size (grams)	Point estimate	129	(6)
PCB half-life (years)	Point estimate	4.8	(12)
Probability of moving or ceasing to fish[a]	Discrete	move/cease(0.057);no move/no cease(0.943)	(3)
Probability of dying[a]	Discrete	die (0.002); no die (0.998)	(3)
Reference dose (mg/kg-day)	Point estimate	2×10^{-5}	(8)

a. Age dependent function. Values provided are for a 30 year old male.

b. Concentrations in fish were selected such that the majority of anglers received doses in excess of the RfD.

Figure 1. Angler Model

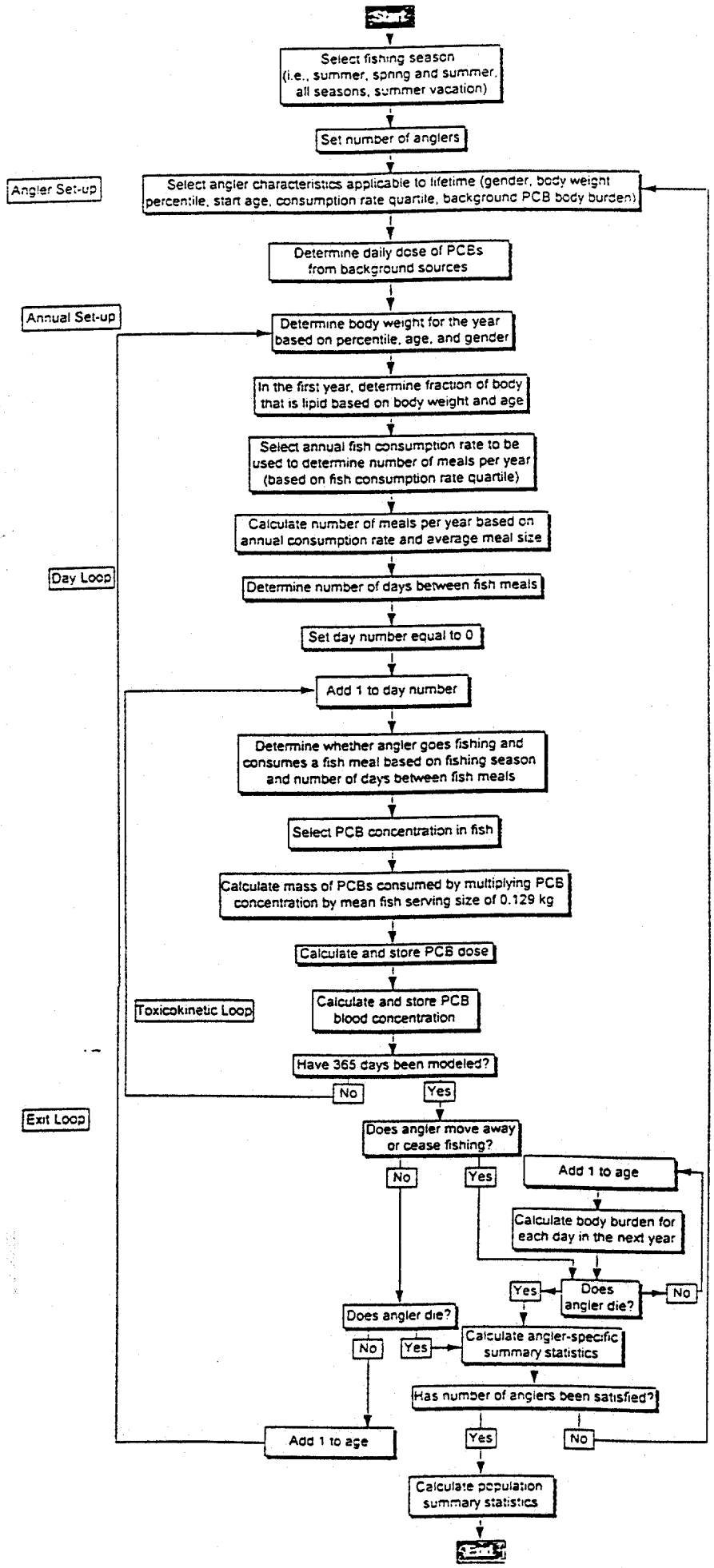
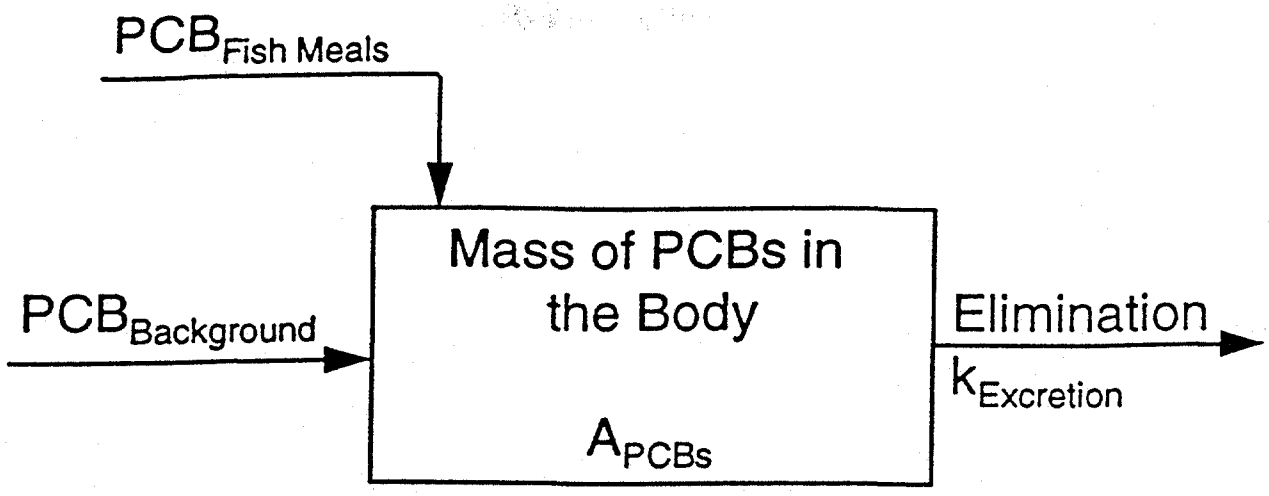


Figure 2. Summary of the Toxicokinetic Model



- $\frac{dA_{PCBs}}{dt} = PCB_{Fish\ Meals} + PCB_{Background} - (k_{Excretion} * A_{PCBs})$

- $C_{PCBs} = A_{PCBs} / V_{Lipid}$

- $k_{Excretion} = \frac{0.693}{t½\ (days)}$

- where:

  $t½ = 1{,}752$ days (12)

  $PCB_{Fish\ Meals}$ = PCBs from fish meals (ng/day);

  $PCB_{Background}$ = PCBs from background sources (ng /day);

  A_{PCBs} = Mass of PCBs in body (ng);

  C_{PCBs} = Concentration of PCBs in lipid (ng /kg lipid);

  $k_{Excretion}$ = First-order elimination rate constant (1/day); and

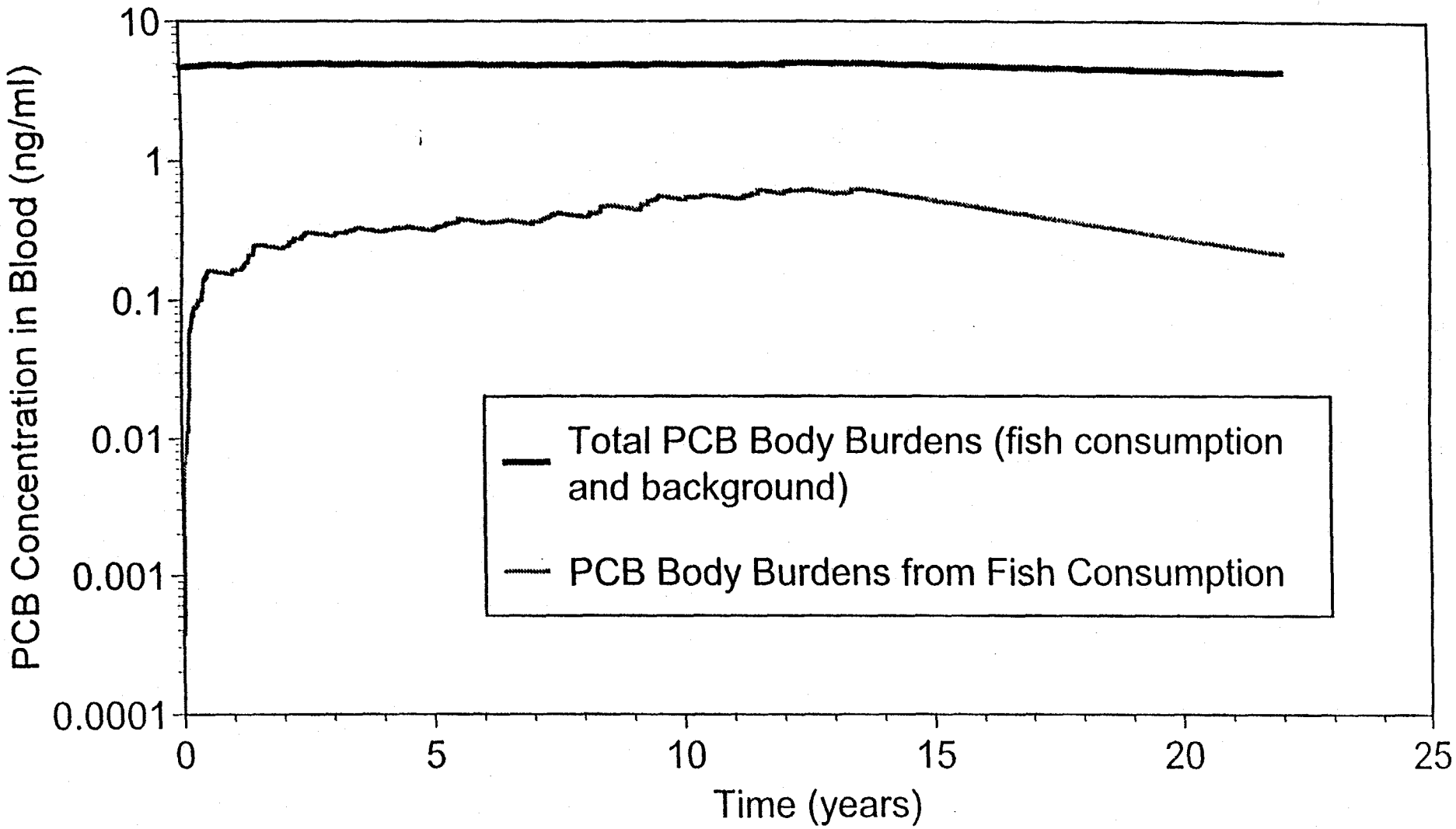
  V_{Lipid} = Total volume of lipid in body (kg).

RESULTS

The following are significant results predicted using a MEE/TK model:

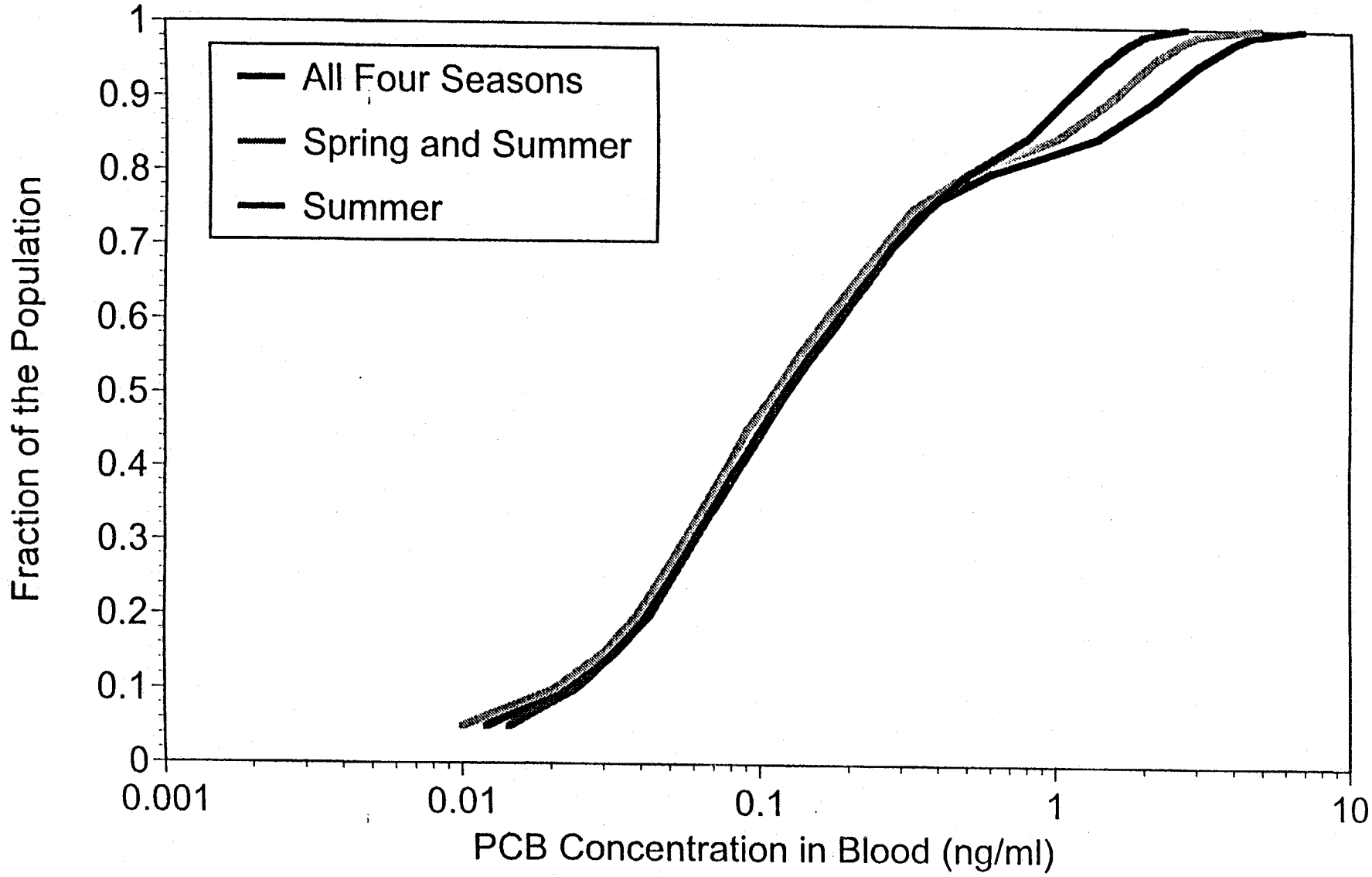
- The long in-vivo half-life of PCBs causes the predicted concentration profile of PCBs in blood to be relatively smooth (Figure 3).
- Fishing season durations had little impact on chronic and subchronic PCB body burdens (Figure 4);
- The peak PCB body burdens did not significantly differ from long-term averages (Figure 5); while the estimates of PCB intake rates for the various averaging periods differed by more than one order of magnitude.
- Overall, the seven-year maximum average PCB body burdens range from 0.4 to 20 ng/ml when both background sources and fish consumption are considered (Figure 6).
- When fish consumption is included as the only PCB source, the blood concentrations range from less than 0.01 to 6 ng/ml (Figure 6).
- The PCB body burdens from fish consumption are small compared to body burdens associated with background sources (Figure 6).
- PCB doses from fish consumption that were greater than the RfD did not affect the total PCB body burdens (Figure 6).

Figure 3. PCB Concentrations in Blood from Beginning of Exposure to End of the Life[a]



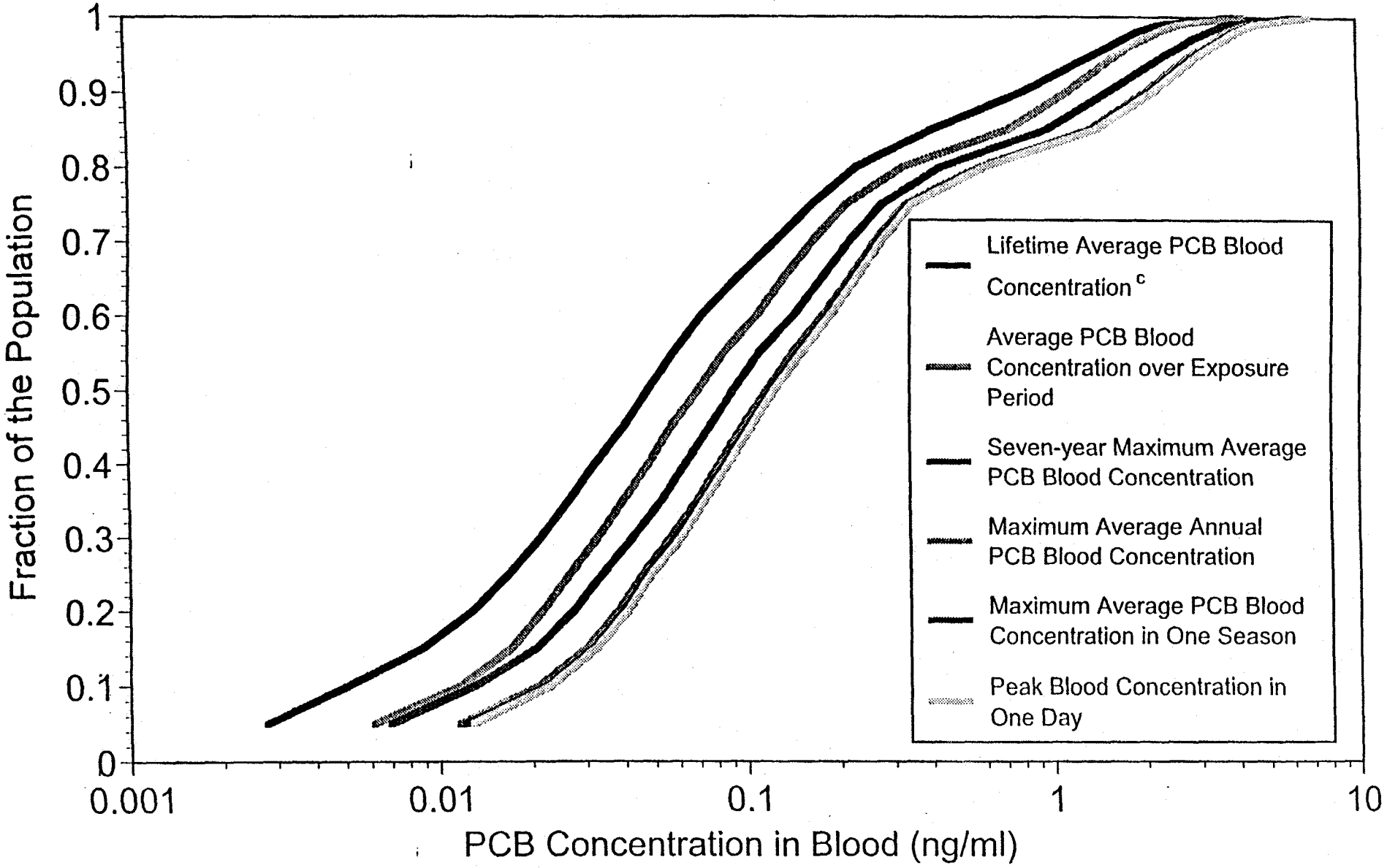
a. Angler fished during the spring and summer for 14 years and consumed 28 fish meals per year. Lifetime extended 9 years after exposure from contaminated fish ceased.

Figure 4. Effect of Fishing Season Duration on Maximum Average PCB Blood Concentration in One Season[a]



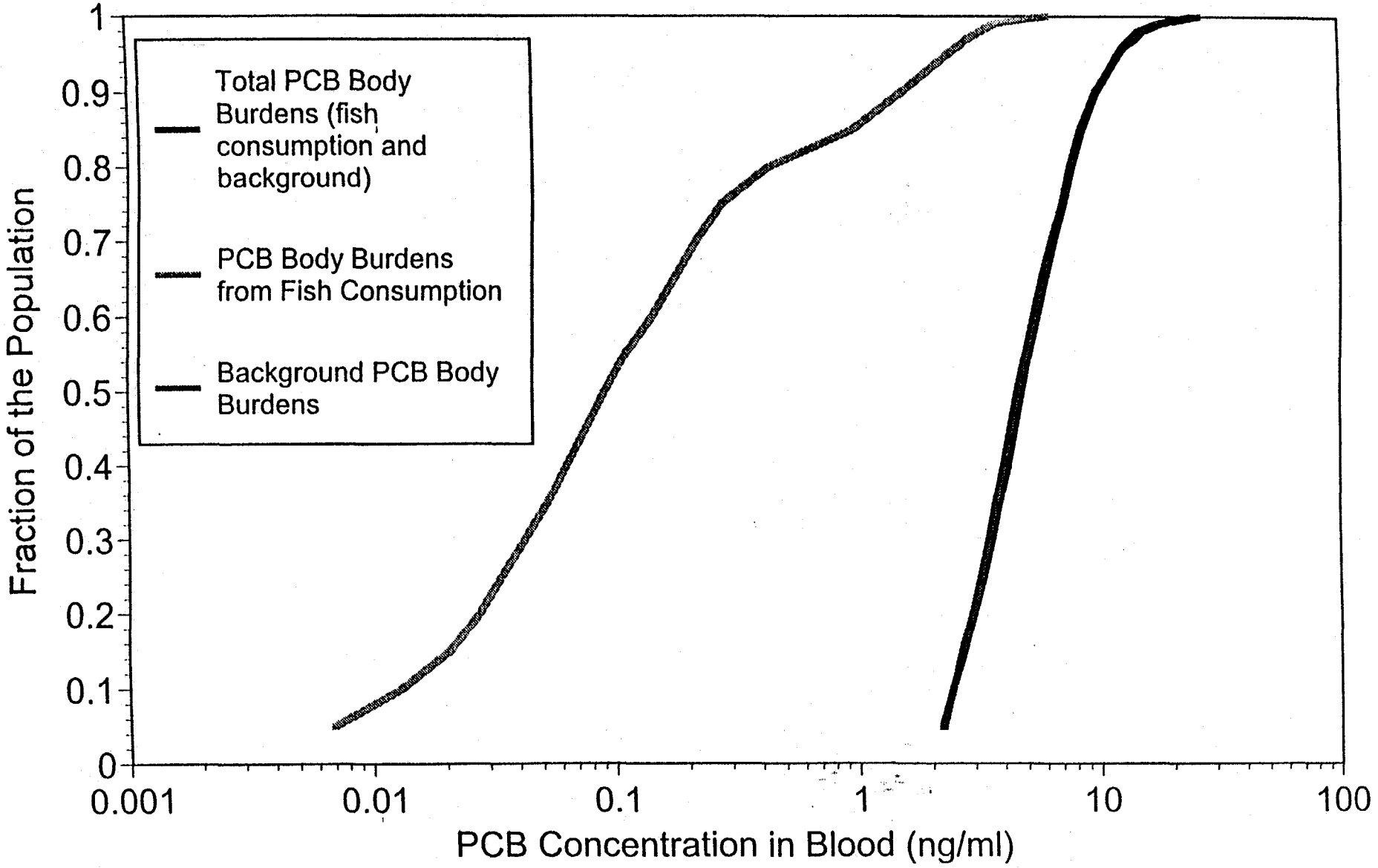
a. PCB body burdens are from fish consumption only.

Figure 5. Distribution of Maximum PCB Body Burdens as a Function of Averaging Time[a,b]



a. PCB body burdens are from fish consumption only.
b. Values reflect a year round fishing season.
c. Lifetime defined as time between start of fishing career and death.

Figure 6. Effect of Background PCB Body Burdens on the Seven-Year Maximum Average PCB Blood Concentration[a]



[a] Year round fishing.

LIMITATIONS

The results of this study should be viewed as preliminary. PCB mixtures found in fish are composed of a number of PCB congeners which have widely varying elimination rates. Consideration of this variation may impact these findings. However, this analysis demonstrates the feasibility of linking complex exposure and toxicokinetic models.

CONCLUSION

The MEE and toxicokinetic methodologies provide the ability to develop coherent models of long-term exposures that can be used to generate estimates of daily PCB body burdens. These estimates can be compared to internal dose measures known to cause toxic effects in animals to evaluate risks. The models also allow the investigation of factors such as magnitude and timing of exposure and the impact of various sources of PCBs (i.e., background sources and fish consumption) on body burdens. Future work should include further consideration of PCB excretion rates.

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Application of Probabilistic Methods to Noncarcinogenic Risk Assessment: A Case Study of Hexachloroethane and Paraquat

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INTRODUCTION

The USEPA has established RfDs for hexachloroethane and paraquat based on studies in laboratory animals (IRIS, 1996). The RfD for hexachloroethane is based on a subchronic rat study (Gorzinski et al., 1985) in which an NOAEL of 1 mg/kg-day was established. Uncertainty factors of 10 each were applied in the derivation of the RfD to account for interspecies extrapolation uncertainty, interindividual variability, and for extrapolation from a subchronic to a chronic toxicological endpoint. The resulting RfD was calculated as 0.001 mg/kg-day. The RfD for paraquat is based on a chronic dog study (Chevron Chemical Company, 1983) which identified a NOAEL of 0.45 mg/kg-day. Two uncertainty factors of 10 each were applied to the NOAEL to account for interspecies extrapolation and interindividual variability, resulting in an RfD of 0.0045 mg/kg-day.

The purpose of this analysis was to explore the impact of the uncertainty in the RfDs on hazard estimates involving the two compounds. Hazard was characterized using both the traditional hazard quotient methodology (USEPA, 1989) and the dose-response methodology proposed by Price et al. (1997). Both the reference uncertainty distribution (Swartout et al., 1997a) and empirical distributions (Gillis et al, 1997; Schmidt et al., 1997; Swartout et al., 1997b) were used in the analyses to investigate the effect of preliminary information on distributions derived from empirical data.

ABSTRACT

Examples are developed as to how a probabilistic representation of the uncertainty in the Reference Dose (RfD) can be applied in a comparative risk analysis for a hypothetical population exposed to two compounds - hexachloroethane and paraquat. The primary noncancer risk assessment tool in the current USEPA Risk Assessment Guidance for Superfund is the hazard quotient (HQ), in which the estimated exposure dose is divided by the RfD. The risk analysis tools used in the examples are the HQ and a model for estimating risk above the RfD (Price et al., 1997). The approach utilizes a distributional characterization of the uncertainty factors (UFs) and of the exposures. The examples are presented with both the default ("reference") UF distributions and empirical UF distributions. Distributions of dose rates used in this assessment were chosen so that the point-estimate hazard quotient for the high-end exposed individual is the same for each compound. The two chemicals, however, differ in total RfD uncertainty and in the steepness of their dose-response curves. The RfD for paraquat includes two areas of uncertainty while the hexachloroethane RfD has three. Experimental data show a 50% response in test animals at twice the NOAEL for paraquat and at 10 times the NOAEL for hexachloroethane, indicating a steeper slope for paraquat. The probabilistic analysis estimates that the 95th percentile HQ for the high end exposure to paraquat is 2 times higher than for hexachloroethane when the reference UF distributions are used, but equivalent to hexachloroethane when the empirical distributions are used. The analysis further indicates that the relative population risk at the 95th percentiles (for both exposure and RfD uncertainty) for paraquat is more than 10-fold greater than for hexachloroethane using the reference UF distributions, while it is only 2-fold greater using the empirical distributions. This analysis demonstrates that the use of empirical distributions can significantly affect risk management decisions. Thus, the pursuit of additional data with which to define empirical distributions is an important effort.

DESCRIPTION OF ANALYSES

Four risk characterization analyses were conducted. Each analysis was conducted twice; first with the reference distribution (Swartout et al., 1997a) and then with empirical uncertainty distributions (Gillis et al., 1997; Schmidt et al., 1997; Swartout et al., 1997b). First, the uncertainty in the hazard quotient for the high-end exposed individual, defined as the individual at or above the 90th percentile exposure, was calculated using a dose corresponding to the 95th percentile of the distribution of doses in a population and the uncertainty distribution for the sensitive population NOAEL (Carlson-Lynch et al., 1997). The distributional RfD was calculated by dividing the NOAEL in animals by the product of the relevant uncertainty distributions.

Second, distributions of hazard quotients representing population variability in exposures were estimated as the ratios of the uncertainty distributions for an individual's exposure and the distributional RfDs. Upper and lower 90% confidence intervals on the distributions were calculated using the dose distribution and the 5th and 95th percentile RfDs from the distributional RfD.

Third, the uncertainty in the response rate for the high end exposed individual was calculated using the dose-response model presented by Price et al. (1997). Fourth, the response rates in the population were calculated, and upper and lower 90% confidence intervals on the response rates were estimated using the 5th and 95th percentile dose-response relationships.

A description of the input data is presented in Table 1.

Table 1. Inputs for Hexachloroethane and Paraquat Reference Dose and Dose-Response Calculations

Variable	Input	Reference
Chemical-Specific Inputs		
Hexachloroethane		
Critical Endpoint	atrophy and regeneration of renal tubules	Gorzinski et al., 1985
NOAELa	1 mg/kg-day	Gorzinski et al., 1985
ED50a	8.63 mg/kg-day	MLE from benchmark dose model
Species	rat	
Uncertainty Factors	UFh, UFa, UFs	USEPA, 1997
Modifying Factors	none	USEPA, 1997
Reference Dose	0.001 mg/kg-day	USEPA, 1997
Dose Distribution	lognormal (base e), μ = -5.8, s = 0.75 (mg/kg-day)	Assumed
95th Percentile Dose (HEE)	0.01 mg/kg-day	Assumed
Paraquat		
Critical Endpoint	chronic pneumonitis	Chevron Chemical Company, 1983
NOAELa	0.45 mg/kg-day	Chevron Chemical Company, 1983
ED50a	1.1 mg/kg-day	MLE from benchmark dose model
Species	dog	
Uncertainty Factors	UFh, UFa	USEPA, 1997
Modifying Factors	none	USEPA, 1997
Reference Dose	0.0045 mg/kg-day	USEPA, 1997
Dose Distribution	lognormal(base e), μ = -4.3, s = 0.75	Assumed
95th Percentile Dose (HEE)	0.045 mg/kg-day	Assumed
Uncertainty Distributions		
Reference Uncertainty Distribution	lognormal (base 10), μ = 0.3349, s = 0.3765	Swartout et al., 1997a
Empirical Uncertainty Distributions		
UFh	empirical distribution of effective dose ratios	Gillis et al., 1997
UFa-rat	empirical distribution of dog/man MTD ratios	Schmidt et al., 1997
UFa-dog	empirical distribution of rat/man MTD ratios	Schmidt et al., 1997
UFs	lognormal (base e), μ = 0.7743, s = 1.152	Swartout et al., 1997b

NOAELa = Animal No-Observed-Adverse-Effect-Level
$ED_{50}a$ = Animal Effective $Dose_{50}$
MLE = Maximum Likelihood Estimate
UFh = Interindividual Uncertainty Factor
UFa = Interspecies Uncertainty Factor
UFs = Subchronic to Chronic Extrapolation Uncertainty Factor
HEE = High End Exposure (USEPA, 1992)

RESULTS

Figures 1 and 2 show the probability distributions for the sensitive human population NOAELs (NOAELh) resulting from the reference and empirical distributions, respectively, compared with the current RfD for each chemical. Figures 3 and 4 compare the HQ uncertainty distributions for hexachloroethane and paraquat that result from the reference and empirical UF distributions, respectively. Using the reference distributions, paraquat is shown to exhibit approximately a 2-fold greater hazard quotient at the 95th percentile than hexachloroethane. The HQs for both compounds at the 97.5th percentile are less than the point estimate HQ of 10. Using the empirical distributions, the HQ uncertainty distributions appear comparable for the two compounds, and the 97.5th percentile for each compound exceeds the point estimate value of 10.

Population distributions of hazard quotients (incorporating exposure variability) for hexachloroethane and paraquat, with 90% confidence intervals, are shown in Figures 5 and 6 for the reference and empirical distributions, respectively. Figure 5 shows that the upper confidence interval distribution for paraquat reaches a hazard quotient of 1 at about the 30th percentile, while upper confidence interval distribution for hexachloroethane does not reach 1 until the 67th percentile. By contrast, the use of the empirical distributions indicates that the upper confidence interval distributions for both compounds reach 1 between the 10th and the 20th percentiles (Figure 6). Neither the reference nor empirical distributions show hazard quotients reaching 10 at the upper 90% confidence interval on the 95th percentile.

Figures 7 and 8 show uncertainty in the response rate for doses 10 times higher than the RfD. Again, using the reference distributions (Figure 7), greater hazard is predicted for paraquat (the 95th percentile response exceeds 10%) than for hexachloroethane (the 95th percentile response is less than 2%). The empirical distributions suggest more similar responses (Figure 8; 10% for paraquat and 5% for hexachloroethane at the 95th percentile).

Finally, Figures 9 and 10 show the upper 90% confidence intervals on population response rates above the RfD. Use of the reference distributions indicates that the 95th percentile response for

paraquat is more than 10-fold greater than the 95th percentile response for hexachloroethane; use of the empirical distributions indicates that the difference is only about 2-fold. Median response distributions for both chemicals and both uncertainty distribution types were zero through the 95th percentiles except for paraquat using the reference distributions, where the dose received by the 95th percentile of the population was estimated to be associated with 1% response.

Figure 1. Uncertainty in Reference Dose Using the Reference UF Distribution

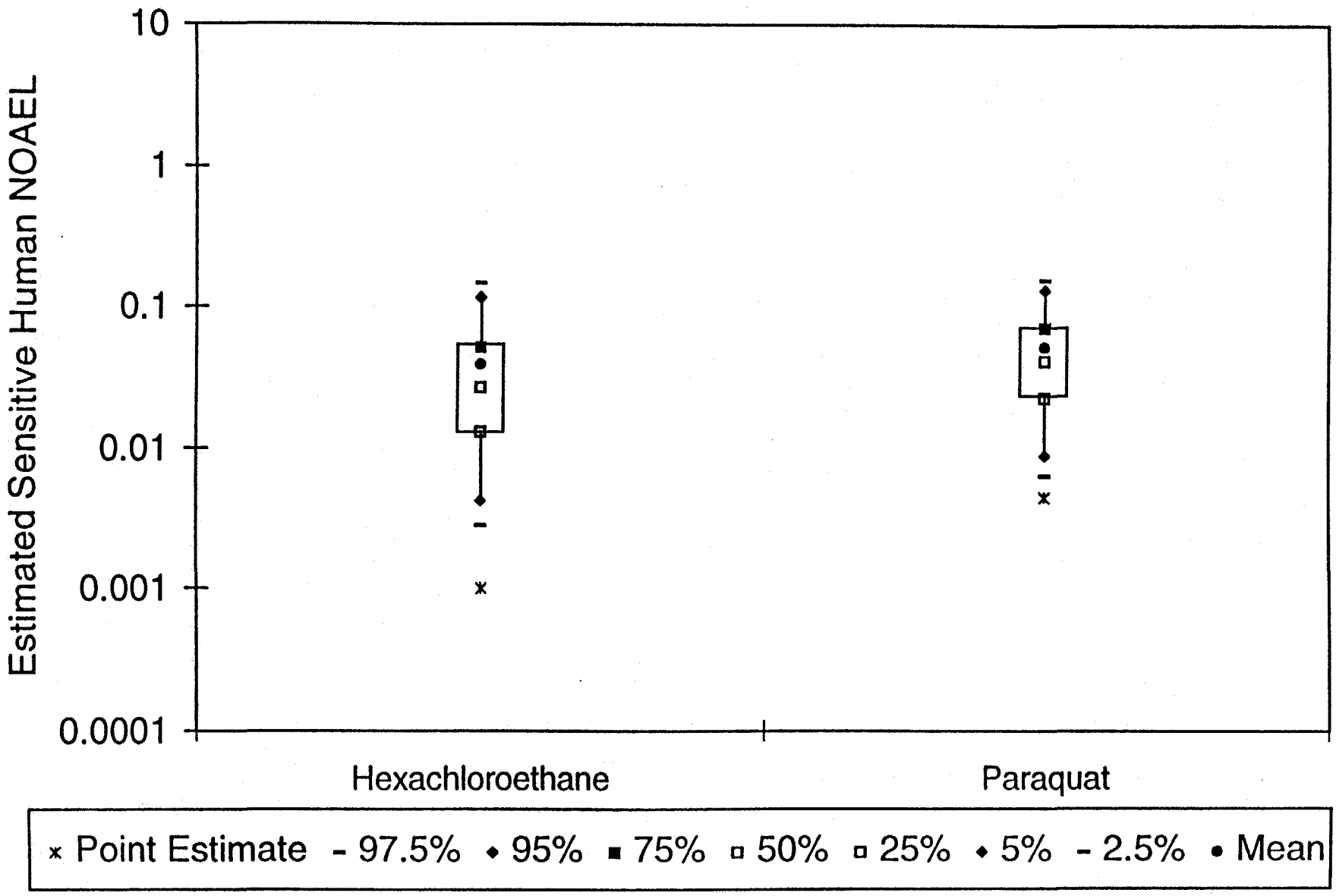

Figure 2. Uncertainty in Reference Dose Using the Empirical UF Distributions

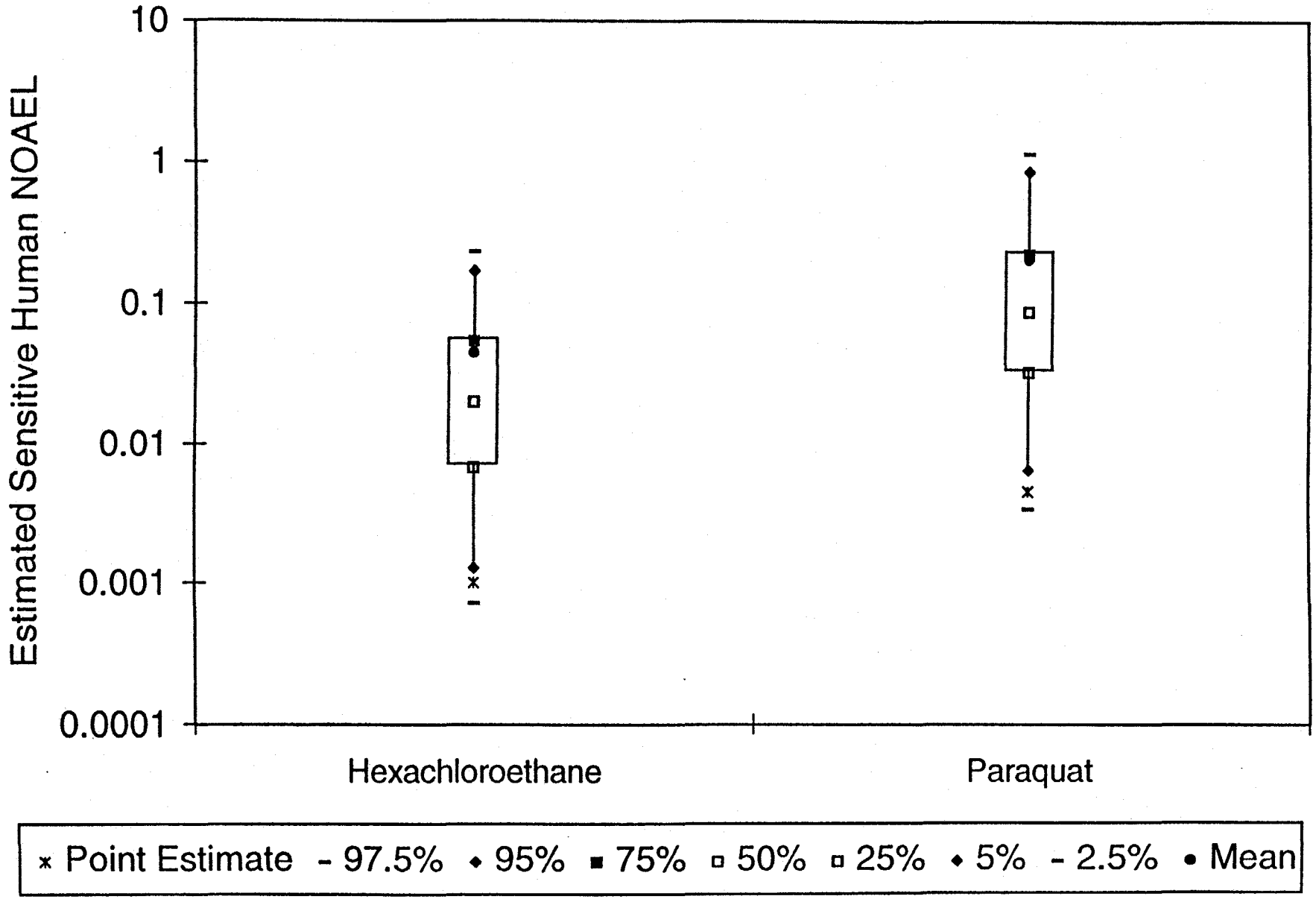

Figure 3. Uncertainty in Hazard Quotients Using the Reference UF Distribution

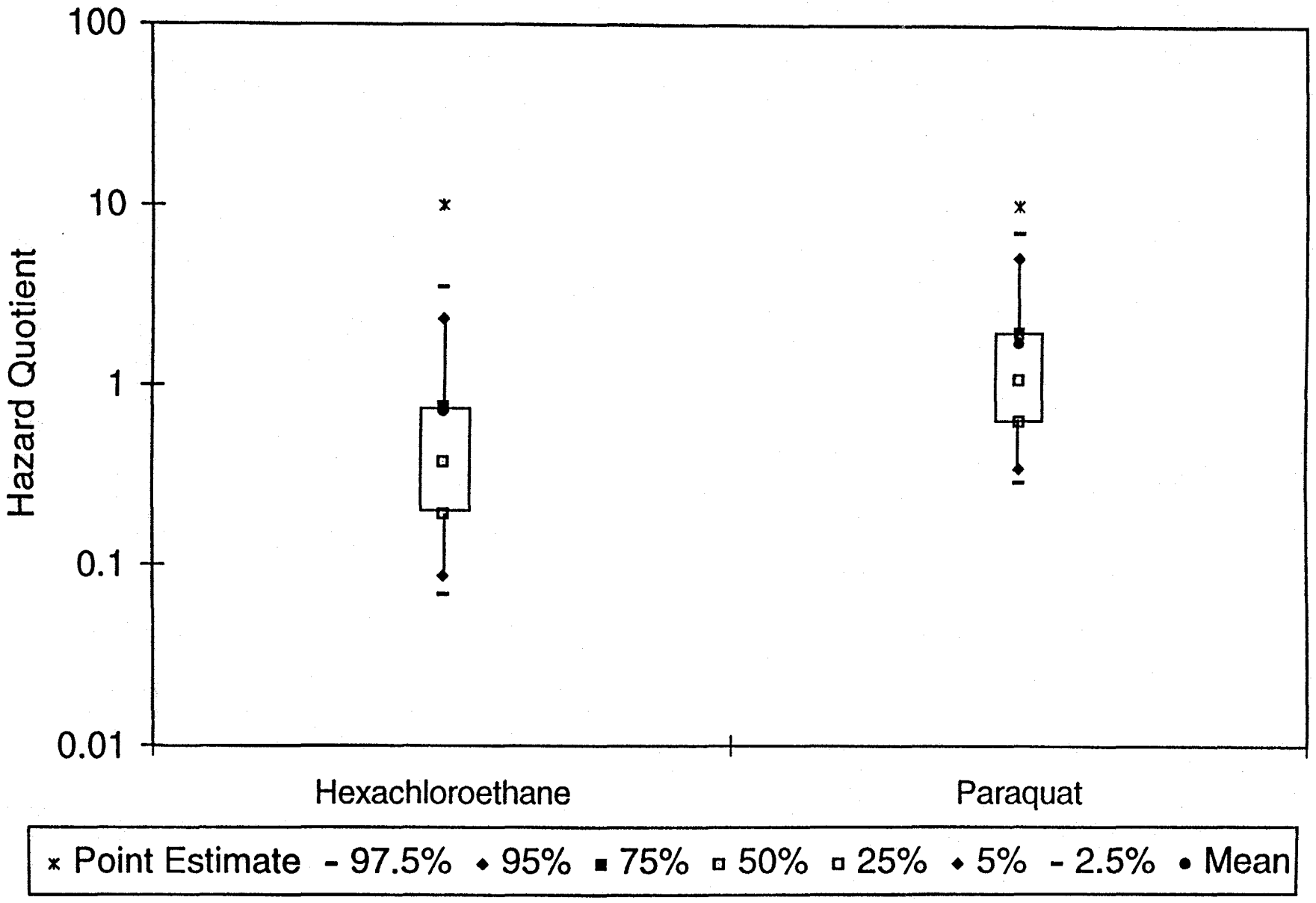

Figure 4. Uncertainty in Hazard Quotients Using the Empirical UF Distributions

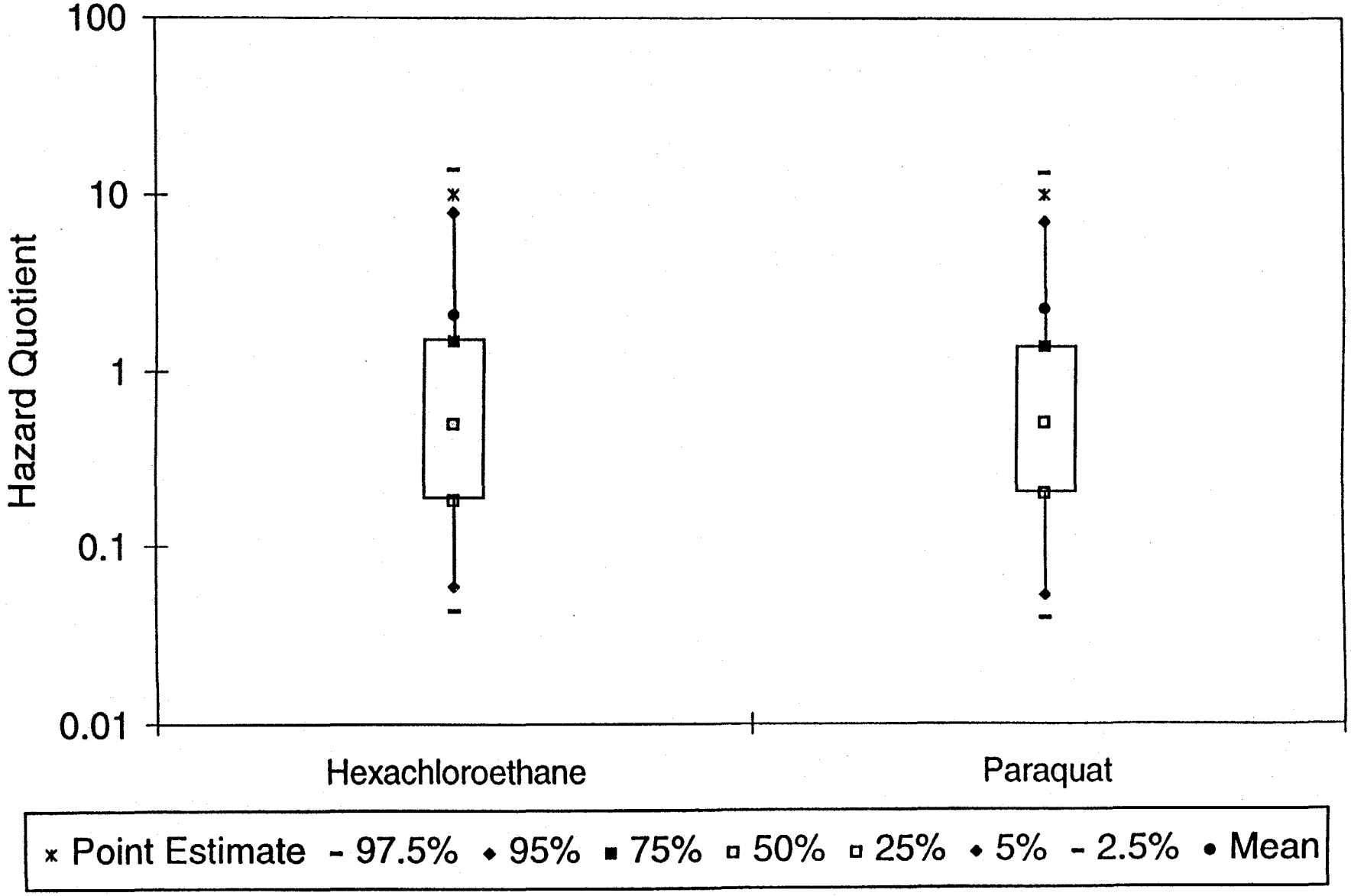

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Figure 5. Population Hazard Quotients with 90% Confidence Intervals Using Reference UF Distribution

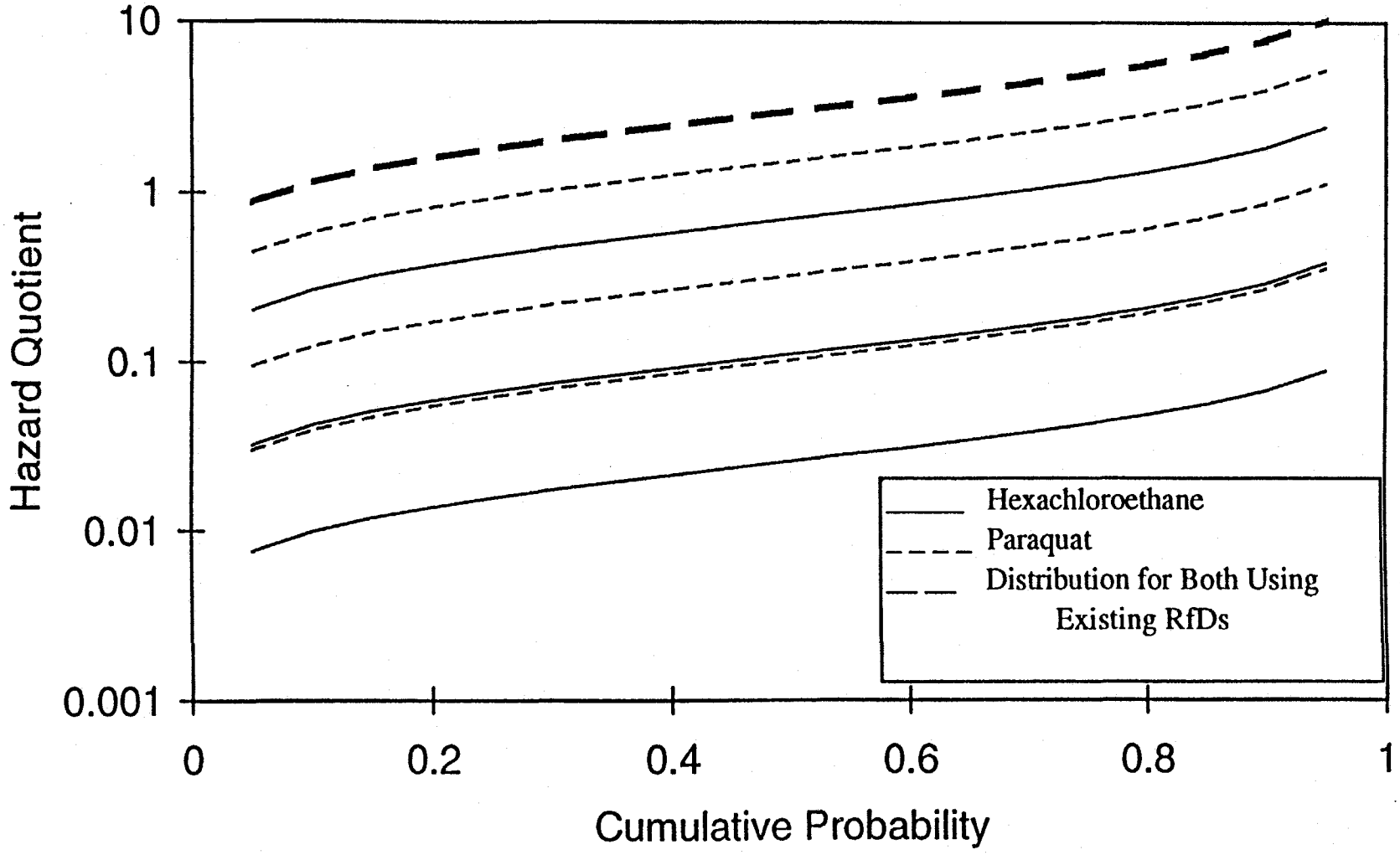

Figure 6. Population Hazard Quotients with 90% Confidence Intervals Using the Empirical UF Distributions

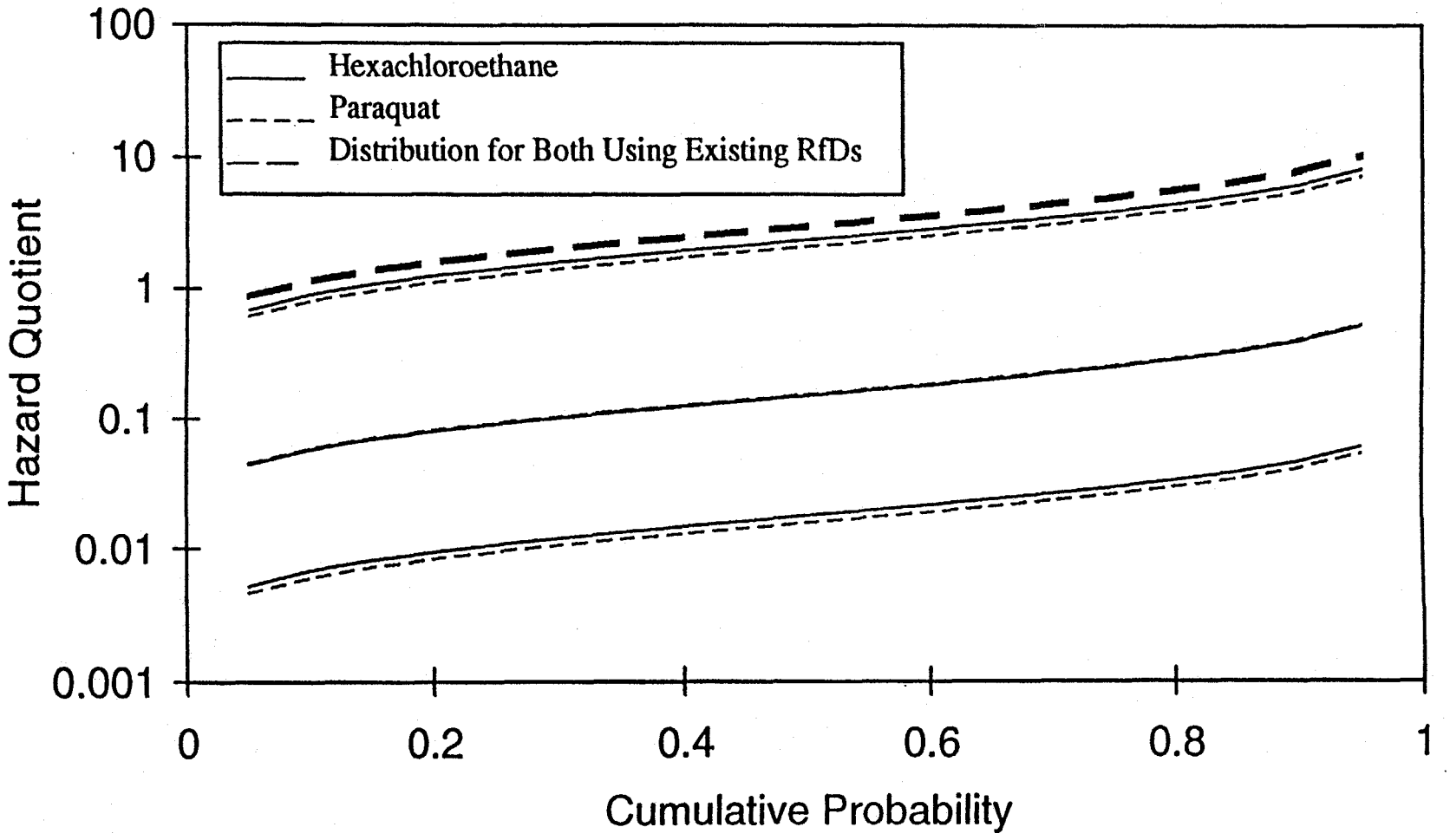

Figure 7. Uncertainty in Response at the High End Exposure Using the Reference UF Distribution

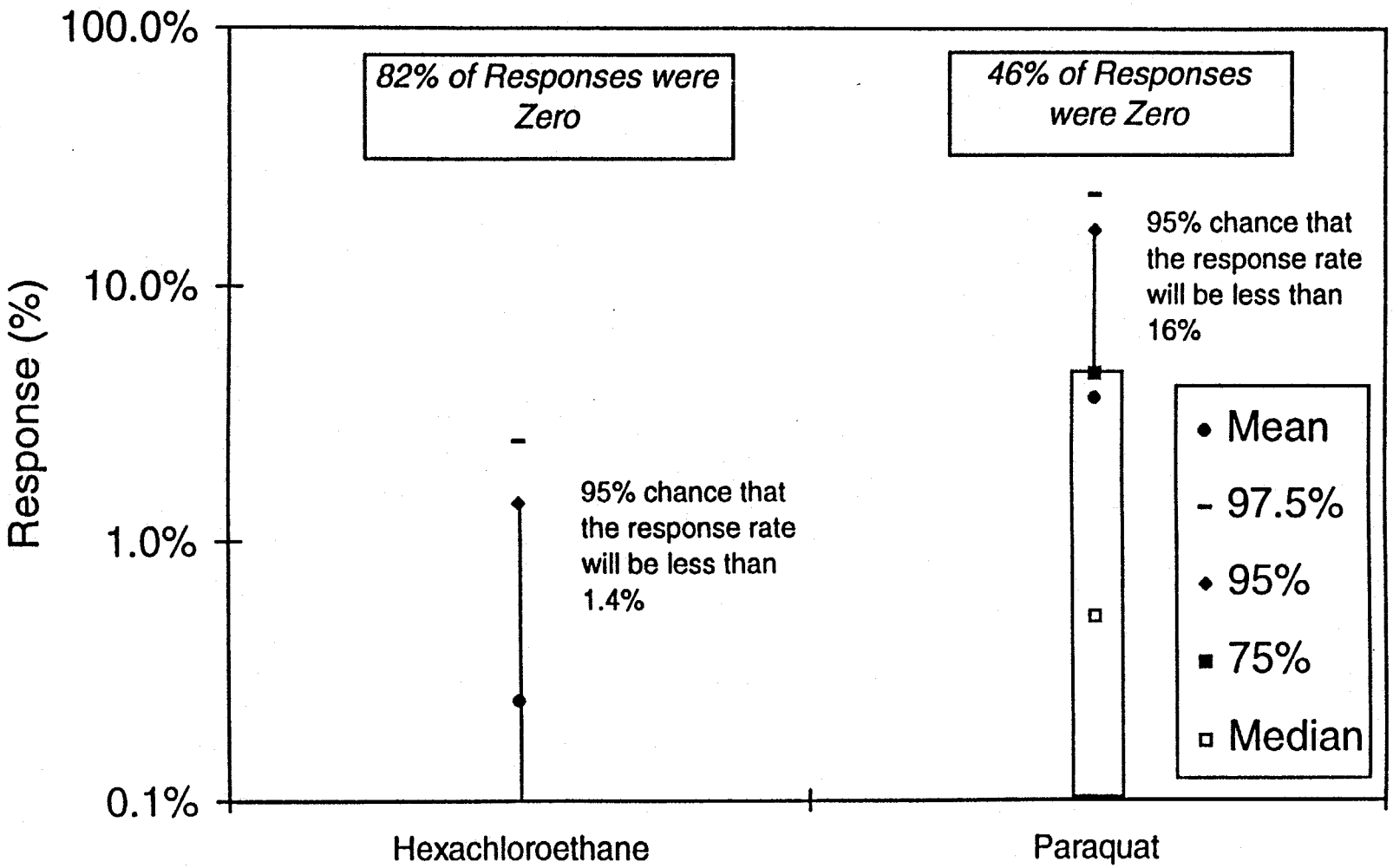

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Figure 8. Uncertainty in Response at the High End Exposure Using the Empirical UF Distributions

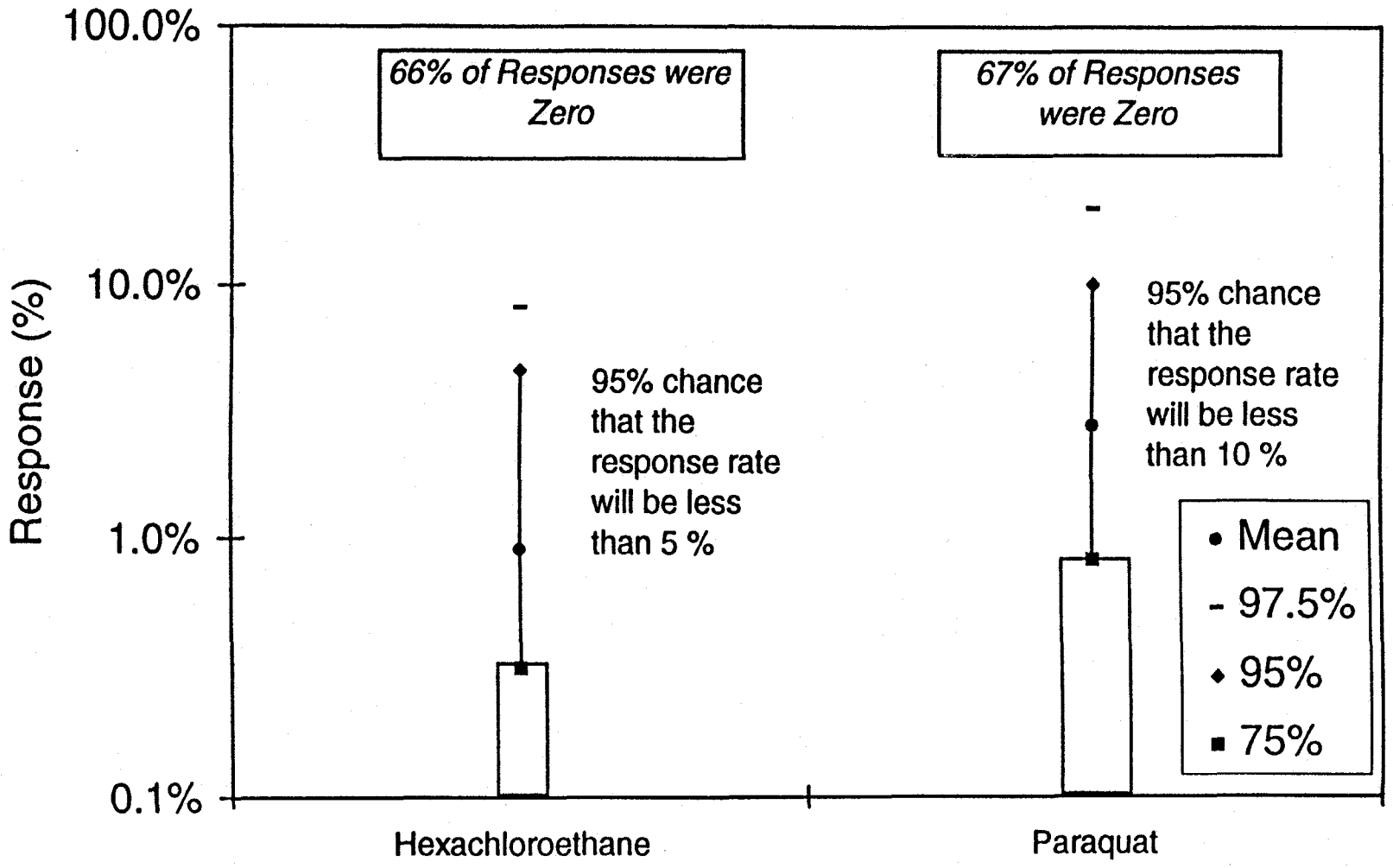


Figure 9. Population Response Based on Noncancer Dose-Response Model Using the Reference UF Distribution

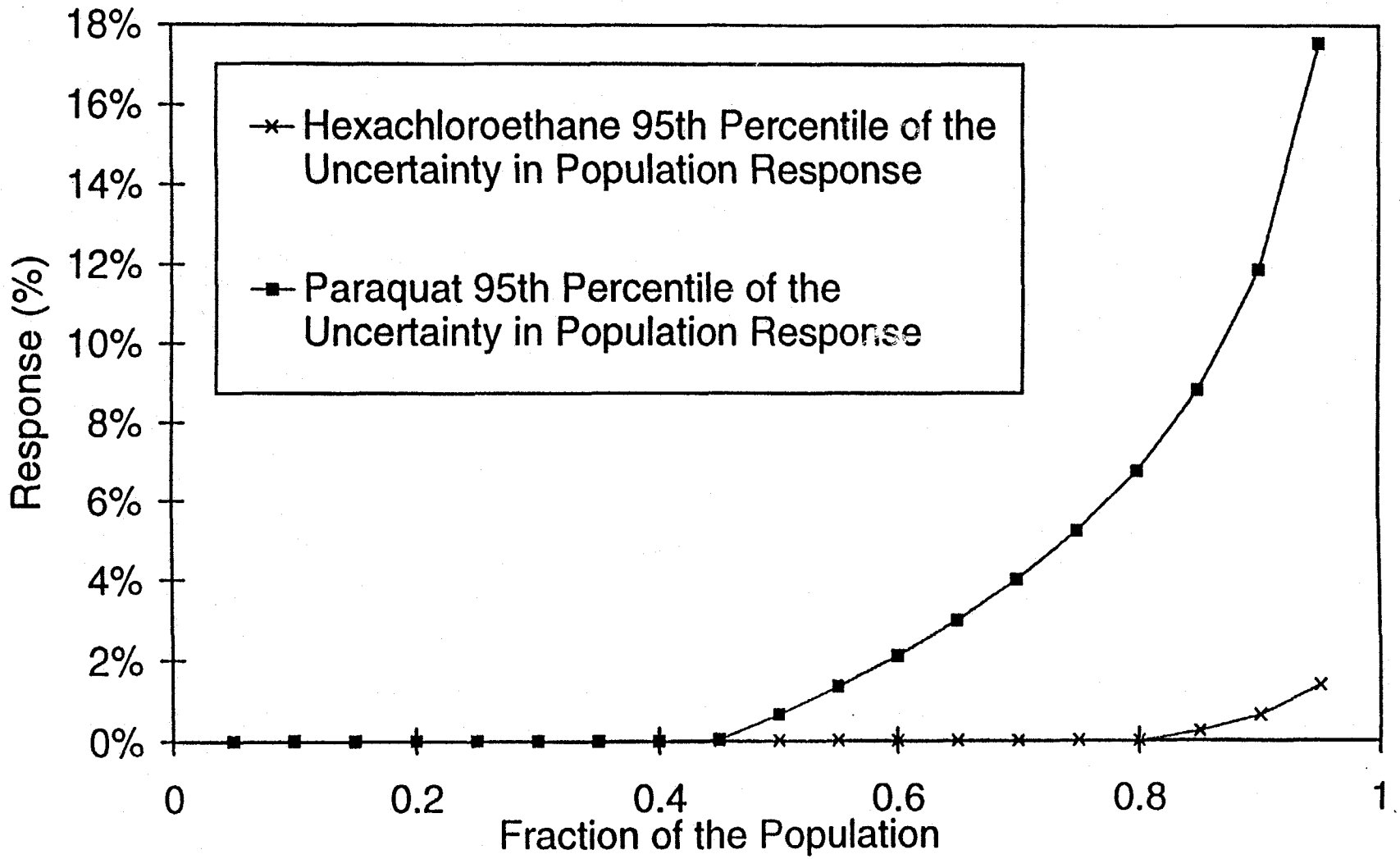
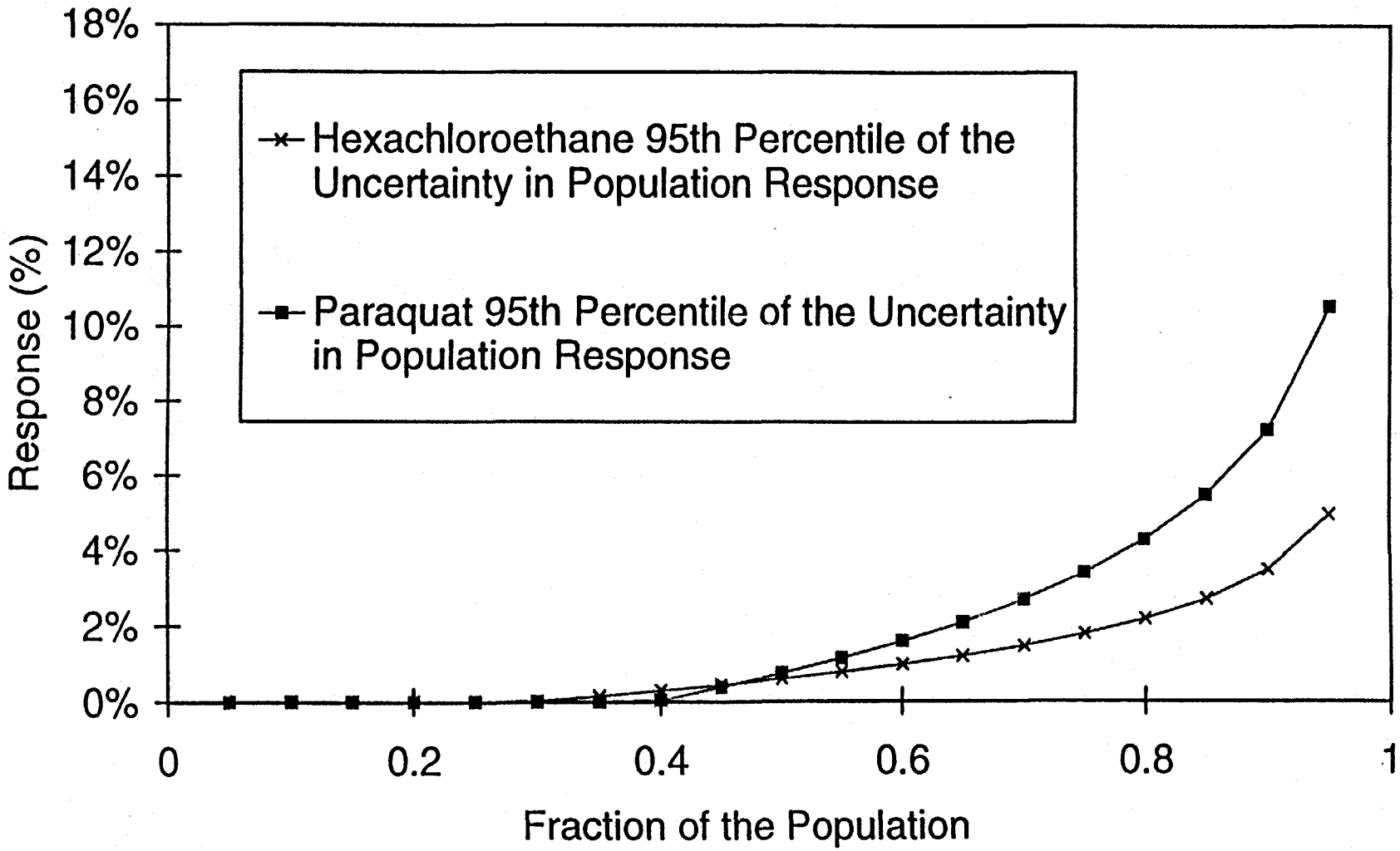


Figure 10. Population Response Based on Noncancer Dose-Response Model Using the Empirical UF Distributions



CONCLUSIONS

- A probabilistic-based approach to RfD uncertainty conveys more information to risk managers on noncancer risk measures than does current guidance.

- Nominally equivalent hazard quotients can differ significantly with a probabilistic assessment of RfD uncertainty.

- Empirically-derived UF distributions can result in qualitatively different conclusions than those based on default UF distributions.

- Relative risks for doses above the RfD are much higher for paraquat than for hexachloroethane using the reference UF distribution, but are virtually the same using the empirical UF distributions.

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