United States Environmental Protection Agency

Revised Technical Support Document: National-Scale Assessment of Mercury Risk to Populations with High Consumption of Self-caught Freshwater Fish

In Support of the Appropriate and Necessary Finding for Coal- and Oil-Fired Electric Generating Units

Revised Technical Support Document: National-Scale Assessment of Mercury Risk to Populations with High Consumption of Self-caught Freshwater Fish

In Support of the Appropriate and Necessary Finding for Coal- and Oil-Fired Electric Generating Units

DISCLAIMER

This document has been prepared by staff from the Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency. Any opinions, findings, conclusions, or recommendations are those of the authors and do not necessarily reflect the views of the EPA. Questions related to this document should be addressed to Dr. Zachary Pekar, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, C504-06, Research Triangle Park, North Carolina 27711 (email: pekar.zachary@epa.gov).

Table of Contents:

List of Tables

List of Figures

Executive Summary

The Technical Support Document is a revised version of the TSD for EPA's National-scale Mercury Risk Assessment completed in March 2011. EPA used the previous version of this report as support for the 2011 finding that it is appropriate and necessary to regulate coal and oil-fired electric utility steam generating units in the United States (U.S. EGUs), pursuant to Section 112(n)(1)(A) of the Clean Air Act.

EPA commissioned a formal peer review of this assessment during the public comment period for the regulation through the EPA Science Advisory Board (SAB), which provides independent advice and peer review to EPA's Administrator on the scientific and technical aspects of environmental issues. The SAB established a 22-member panel with representation from academic institutions, industry, federal agencies, and state governments. The SAB supported the overall design of the risk assessment, confirmed EPA's analytical assumptions, and concluded that the risk assessment should provide "an objective, reasonable, and credible determination" of the potential public health hazard. The SAB made many recommendations for improving this TSD, which SAB organized into three general themes: (1) improve clarify of the document regarding methods and presentation of results, (2) expand discussion of sources of variability and uncertainty, and (3) de-emphasize IQ loss as an endpoint. EPA has responded to the peer review by substantially revising this TSD.

Purpose and Scope of Analysis

The goal of this assessment was to determine whether mercury emitted from U.S. EGUs poses a potential public health hazard. Therefore, we have designed this risk assessment as a screening analysis focused on identifying watersheds where there is a public health hazard attributable to U.S. EGU mercury deposition. Mercury emitted from U.S. EGUs, depending on the form of mercury emitted and other factors, can deposit locally and regionally in U.S. waterbodies, as well as contribute to the global pool of mercury, where it can be transported and eventually deposited around the world. This deposited mercury is transformed into methylmercury (MeHg) by microorganisms and then bioaccumulates as MeHg in fish. The primary pathway of concern from a public health standpoint is consumption of mercury-contaminated fish by women of child bearing age (since mercury can stay in the system for some time, both women who are pregnant or about to be pregnant are of concern.) Depending on the level of prenatal exposure, children born to those women may then experience a range of neurodevelopmental effects including decrements on a number of neuropsychological measures.

We focused on consumption from inland freshwater waterbodies in the U.S rather than estuarine or marine waterbodies because the U.S. EGU-attributable mercury deposition is a larger fraction of total mercury deposition, particularly for waterbodies with elevated U.S. EGU-attributable mercury deposition such as the Ohio River Valley. We focused on subsistence fishing populations because they typically have substantially higher consumption rates of self-caught fish than recreational fishers and therefore, experience higher risk. Because we do not have data available on the distribution of subsistence fishing populations in all watersheds in the U.S., we modeled a hypothetical female subsistence consumer at those watersheds where we have fish tissue data and where we believe subsistence fishing activity has the potential to occur.

Risk Assessment Methods

We followed a 10-step process to estimate risk to female subsistence consumers.

1. Model total and U.S. EGU-attributable mercury deposition for the continental U.S.
2. Interpolate mercury deposition to watersheds
3. Estimate 75th percentile fish tissue mercury concentrations at watersheds with fish tissue data
4. Project 75th percentile fish tissue mercury concentrations for the 2016 scenario, which reflects projected Hg deposition without regulation of mercury emissions from U.S. EGUs
5. Define female subsistence consumer scenarios
6. Identify watersheds with subsistence fisher population activity
7. Define self-caught fish consumption rates for the subsistence scenarios
8. Estimate total fish consumption-related MeHg exposure
9. Estimate total MeHg risk
10. Estimate U.S. EGU-attributable risk

We assume a proportional relationship between changes in mercury deposition and changes in fish tissue mercury concentrations. This assumption, supported by the Mercury Maps assessment and other scientific literature, is used to estimate the fraction of U.S. EGU-attributable MeHg in fish and to project fish tissue data to 2016. We use the 75th percentile fish tissue sample because we believe it is likely that subsistence fishers may target larger fish (with somewhat higher MeHg levels) to supplement family meals. Although we can only calculate risk for the 4% (3,100) of the 88,000 watersheds in the continental U.S. for which we have fish tissue mercury data, these data cover 48 states including many states with high levels of mercury deposition and fish tissue mercury concentrations. We excluded watersheds with potentially significant non-air sources of mercury.

In estimating risk for the most comprehensive scenario reflecting exposures for women of childbearing age with subsistence level consumption of freshwater self-caught fish, we apply the scenario to all watersheds with fish tissue mercury data, reflecting the potential for these populations to obtain and consume fish from any watershed. To estimate this risk, we use the EPA's MeHg reference dose (MeHg RfD), which identifies a level of exposure above which there is the risk of adverse health effects. We compare modeled MeHg exposure levels against the RfD to generate a hazard quotient (HQ). An HQ above one represents a potential exposure above the MeHg RfD and EPA, supported by the SAB, considers such an exposure to represent a public health hazard. We use two risk metrics at the watershed level: (a) number and percent of watersheds modeled where the total mercury-based HQ>1 and where U.S. EGU-attributable mercury deposition contributes at least 5% of the risk and (b) number and percent of watersheds modeled where U.S. EGU-sourced mercury considered alone, without taking account deposition from other sources, results in an HQ>1. The combination of these two estimates provides the

total number and percent of watersheds where female subsistence consumers are potentially at-risk.

   We have also modeled an additional set of female subsistence consumer scenarios focused on different socioeconomic (SES) groups (e.g., low income Blacks in the Southeast, low income Hispanics, Vietnamese). Each of these female subsistence consumer scenarios is supported by scientific data on SES specific high-end subsistence-level fish consumption rates for self-caught fish from inland freshwater bodies. We model watersheds for each of these scenarios if there are at least 25 individuals from the SES group in close proximity to the watershed.

Response to SAB Recommendations

   In response to the SAB review, we have substantially modified this TSD. Key modifications include: (a) clarifying that the analysis is watershed-focused and designed to assess risk for female subsistence consumers (and not a representative characterization of risk for recreational anglers), (b) refining and improving the technical presentation in general, including addition of a sample calculation figure clarifying how the watershed-level risk estimates are generated, (c) expanding the discussion of how the fish tissue mercury database was developed, (d) incorporating additional fish tissue mercury data for states with high levels of U.S. EGU mercury deposition, (e) verifying the linkages between type of fish tissue mercury measured, type of fish consumption rates used and application of a cooking/preparation adjustment factor, (f) inclusion of sensitivity analysis involving median watershed-level fish tissue mercury levels, (g) expanded discussion of key sources of variability and uncertainty, and (h) de-emphasized the IQ loss estimates and moved those estimates to an appendix In response to public comments, we have also moved the previously titled "Hotspot Analysis" to a separate TSD titled "Potential for Excess Local Deposition of Mercury in Areas near U.S. EGUs".
   .

Key Observations

- Based on a combination of the two risk metrics, and reflecting consumption rates ranging from the 90th percentile to the 99th percentile, we estimate that 22% to 29% of the watersheds modeled have populations that are potentially at-risk due to U.S. EGU Hg emissions in 2016.

   o Based on U.S. EGU-attributable deposition considered alone, without taking into account other sources of deposition, 10% of the modeled watersheds have populations that are potentially at-risk in 2016 (based on the 99th percentile consumption rate) (2016 is the year when regulations under Section 112 of the Clean Air Act would need to be implemented for EGUs).

   o In 2016, 24% of the watersheds have populations that are potentially at-risk due to total deposition with at least 5% of that total deposition attributable to U.S. EGUs (based on the 99th% percentile consumption rate).

- These risk results reflect U.S. EGU-attributable deposition. In 2016, we estimate that for watersheds modeled in the risk assessment, U.S EGUs contribute up to 16% of total mercury deposition and related fish tissue mercury concentrations. On average, for these

modeled watersheds, U.S. EGUs contribute 3% of total mercury deposition and related fish tissue mercury concentrations.

- Reducing U.S. EGU-attributable mercury will reduce the magnitude of the risk from total mercury exposure. However, a large fraction of modeled watersheds would still have populations at-risk due to deposition from global sources of mercury, although the degree of risk would be diminished.

1   Review of Analysis Approach

1.1   Introduction

The EPA completed a national-scale risk assessment for mercury (Hg) that informed the March 2011 finding that it is appropriate and necessary to regulate electric utility steam generating units in the United States (U.S. EGUs), pursuant to Section 112(n)(1)(A) of the Clean Air Act (CAA). That-risk assessment is documented in the Technical Support Document: National-Scale Mercury Risk Assessment Supporting the Appropriate and Necessary Finding for Coal and Oil-Fired Electric Generating Units (U.S.EPA, 2011a), hereafter referred to as the "Mercury Risk TSD (March publication)" or "March TSD". In making that finding, EPA determined that this assessment should be peer-reviewed, and that the results of the peer review and any EPA response to them would be published before the final rule. EPA conducted a formal peer review through the EPA Science Advisory Board, which provides independent advice and peer review to EPA's Administrator on the scientific and technical aspects of environmental issues. The SAB established a 22-member peer review panel with representation from academic institutions, industry, federal agencies, and state governments. The peer review charge questions and results of the peer review provided in letter form are publically available on the EPA Science Advisory Board website,[1] and are included in this document (see Appendix C).

EPA reviewed the SAB comments and recommendations for strengthening the national-scale risk assessment and developed a revised version of the risk assessment reflecting implementation of a number of the recommendations made by the SAB. This document (hereafter referred to as the "Mercury Risk TSD (revised)" or "Revised TSD" describes the technical approach used in completing the revised version of the risk assessment. While the underlying technical approach used in assessing risk remains essentially the same, some elements of the risk model, including some data inputs, have been modified and the presentation and discussion of methods and risk estimates has been significantly revised in accordance with SAB recommendations.

The remainder of this revised TSD is organized as follows. We begin this section with a summary of the significant recommendations provided by the SAB peer review panel, and an overview of the changes to the technical approach made in response to specific SAB recommendations (section 1.1). We then provide a description of the purpose and scope of the analysis (section 1.2). Next, we provide an overview of the risk metrics generated and the 2-stage risk characterization framework used to help interpret the risk estimates in a policy-relevant context (section 1.3). In section 1.4, we provide an overview of the analytical approach used in the analysis, with subsections addressing specific elements of the analysis. Section 1.5 describes differences between the current risk assessment and the assessment completed in 2005 (the 2005 Section 112(n) Revision Rule analysis). Section 1.6 presents a sample calculation that

---

[1] Link to the SAB letter (which includes the charge questions as an appendix): http://yosemite.epa.gov/sab/sabproduct.nsf/02ad90b136fc21ef85256eba00436459/BCA23C5B7917F5BF8525791A0072CCA1/$File/EPA-SAB-11-017-unsigned.pdf

walks the reader through the process used to generate risk estimates in the analysis. This detailed example calculation should help the reader to see how the various inputs and intermediate modeling calculations come together in generating watershed-level hazard quotient (HQ)-based risk estimates. Section 2 describes analytical results of the risk assessment including both intermediate modeling results and the risk estimates that are generated (a detailed overview of the subsections in section 2 is presented at the beginning of that section). Section 3 presents a summary of key observations from the analysis.

1.1 Summary of Significant SAB Peer Review Panel Recommendations and Revisions Reflected in this Revised Mercury Risk TSD

In response to the comments and recommendations by the SAB as part of their review of the March TSD, EPA revised the technical approach and documentation for the national-scale Hg risk assessment. These enhancements to the technical approach are briefly described below, along with a summary of the original SAB comment in italics.[2]

- *The watershed-focus of the risk assessment should be clearly stated.* We revised the technical approach section to more clearly state that the analysis is focused on assessing risk for subsistence-level consumers of self-caught fish evaluated at the watershed-level.[3] Specifically, we are modeling risk for a set of female subsistence fish consumer scenarios at those waterbodies where (a) we have measured fish tissue mercury (Hg) data and (b) it is reasonable to assume that high-end fishing activity could occur. We emphasize the point that the analysis is not a representative population-weighted assessment of risk. Rather, it is based on evaluating exposure scenarios.
- *Because IQ does not fully capture the range of neurodevelopmental effects associated with Hg exposure, this endpoint should be deemphasized (and covered in an appendix) and primary focus should be placed on the MeHg RfD-based hazard quotient metric.* We modified the structure of the Revised TSD accordingly, including discussion of alternative neurodevelopemental endpoints (besides IQ) reflected in the MeHg RfD (see section 1.3).
- *Clarify the rationale for using an HQ at or above 1.5 as the basis for selecting potentially impacted watersheds.* We revised this discussion accordingly (see section 1.4.5).
- *Additional detail should be provided on the characteristics of the fish tissue Hg dataset, including its derivation and the distribution of specific attributes across the dataset (e.g., number of fish tissue samples and number of different waterbodies reflected in the*

---

[2] We focused this section on those SAB comments that resulted in more significant revisions to the TSD or the risk assessment itself. Those comments that are more editorial in nature, or address less substantial technical elements of the analysis, are not covered here, although we addressed those comments within the Revised TSD. The charge questions for the peer review together with the SAB letter response provided as part of the peer review are included in Appendix C (the SAB letter includes the original EPA charge questions as an attachment).

[3] The assessment focuses on women of child-bearing age who consume subsistence-levels of fish that they catch or are caught by relatives/acquaintances and shared with them (hereafter referred to as *female subsistence fish consumers*). As discussed in section 1.4 and 1.4.3, we model a number of female subsistence fish consumer scenarios that are differentiated to provide coverage for different socio-economic status (SES) differentiated groups. In this context, "subsistence" refers to individuals who rely on noncommercial fish as a major source of protein in their diet (U.S. EPA, 2000). For purposes of this risk assessment, we have interpreted this as representing self-caught fish consumption ranging from a fish meal (8 ounce) every few days to a large fish meal (12 ounces or more) every day.

*percentiles provided for a given hydrologic unit code (HUC)[4], number of species
reflected across HUCs).* We included additional figures and tables describing the
derivation of the HUC-level fish tissue Hg dataset including the filtering steps to derive
the fish tissue dataset and to generate the HUC-level percentile estimates (see section
1.4.2). In addition, we included tables summarizing the distribution of key attributes
within that dataset highlighted by the SAB (e.g., distribution of fish tissue sample size
and number of species across the HUC-level estimates). We also provided a table that
contrasts fish tissue Hg concentrations for fish greater than 7 inches in length with
concentrations based on all fish, without filtering on size (i.e., the dataset used in the risk
assessment, versus all fish combined).

- *Determine whether there is additional (more recent) fish tissue data for key states
  including Pennsylvania, New Jersey, Kentucky and Illinois where U.S. EGUs Hg
  deposition may be more significant.* We expanded the fish tissue dataset by incorporating
  additional (newer) fish tissue data from the National Listing of Fish Advisory (NLFA)
  (which included additional data for four states including MI, NJ, PA, and MN). We also
  obtained additional data for Wisconsin (see section 1.4.2).

- *Regarding the fish tissue dataset used in the risk assessment, clarify which species of Hg
  is reflected in the underlying samples and discuss the implications of differences across
  states in sampling protocols in introducing bias into the analysis.* We clarified that in
  most cases, the fish tissue is measured for total Hg. Furthermore, based on the literature,
  it is reasonable to assume that more than 90% of fish tissue Hg is methylmercury
  (MeHg). Therefore, we incorporated a Hg conversion factor of 0.95 into our exposure
  calculations to account for the fraction of total Hg that is MeHg in fish (see section
  1.4.4). We also expanded the discussion of uncertainty (see Table 2-15 entry B in section
  2.7) to include the potential for different sampling protocols across states to introduce
  bias into the risk assessment.

- *Expand the uncertainty discussion related to the screening out of HUCs from the risk
  assessment to address (a) uncertainty in characterizing significant non-air sources of Hg
  loading reflected in the Toxics Release Inventory (TRI) and (b) failure to consider release
  of Hg from larger urban areas (e.g., sanitary sewer discharges) in screening out HUCs.*
  We expanded the uncertainty discussion presented in Table 2-15 entry D of section 2.7 to
  address these two sources of uncertainty related to the exclusion criteria for HUCs.

- *While there is support for the use of the 75th percentile fish tissue Hg value in the risk
  assessment, there is concern that the low sampling rates reflected across the HUCs may
  low-bias the 75th percentile estimates.* As noted above, we provided additional
  description of the fish tissue dataset including distribution of sample sizes and fish
  species across the HUCs, which includes an improved discussion of uncertainty and
  potential bias related to the derivation and use of the 75th percentile fish tissue levels (see
  Table 2-15 entry C in section 2.7, as well as the discussion in section 1.4.2).

- *Include a sensitivity analysis based on use of the median fish tissue Hg value (as
  contrasted with the 75th percentile value) in generating risk estimates*: We also included a
  sensitivity analysis that used the 50th percentile watershed-level fish tissue Hg

---

[4] For the assessment we have selected Hydrologic Unit Code 12 (HUC12) watersheds as the spatial unit of analysis
(see section 1.4.1). Throughout this revised TSD, unless otherwise stated, the terms "watershed" and "HUC" are
used interchangeably to refer to HUC12's.

concentration, in addition to the 75th percentile value used in the core analysis (see section 2.6)

- *The SAB was generally supportive of the consumption rates used, but recommended that EPA expand its discussion of caveats associated with the fish consumption rates used in the analysis (e.g., high-end consumption rates for South Carolina reflect small sample sizes, the consumption surveys underlying the studies are older and behavior may have changed, consumption rates may reflect seasonality even if they are expressed in annual terms).* We expanded the discussion of uncertainty related to the fish consumption rates to address the caveats identified by the SAB (see Table 2-15 entries F and G in section 2.7).

- *The EPA needs to verify whether the consumption rates are daily values expressed as annual averages and whether they are "as caught" or "as prepared" (these factors have important implications for the exposure calculations).* As noted in section 1.4.4, we carefully reviewed the studies underlying the fish consumption rates used in the risk assessment and verified that the rates are annual-average daily consumption rates and that they represent as prepared estimates. We also expanded the explanation of the exposure calculations to more completely describe the exposure factors and equation used to generate the average daily MeHg intake estimates for the female subsistence fish consumer scenarios (see section 1.4.4).

- *Need to explain exclusion of fish smaller than 7 inches in length from analysis.* We described the rationale for the 7-inch cutoff for edible fish used in the risk analysis (see section 1.4.2).

- *The SAB was generally supportive of the approach used for identifying HUCs with the potential for subsistence activity; however, they did recommend that we identify the number of HUCs excluded from the analysis due to this criterion.* We added a table to illustrate the number of HUCs with fish tissue Hg data used to model risk for each of the female subsistence fish consumer scenarios (see section 1.4.3, Table 1-5).

- *Enhance the discussion of the proportionality assumption linking Hg deposition and fish tissue Hg concentrations, including more recent studies supporting the proportional relationship between changes in Hg deposition and changes in MeHg fish tissue levels.* We expanded our discussion of uncertainty on the proportionality assumption and added citations for the more recent studies supporting the proportionality between changes in Hg deposition and changes in fish tissue Hg concentrations (see Table 2-15 entries I through M in section 2.7 for additional uncertainty discussion and section 1.4.6 for discussion of newer literature supporting the proportionality assumption).

- *Given that there are published studies comparing measurements of wet deposition with CMAQ-based estimates, the presentation of Hg deposition estimates should include maps of wet and dry Hg deposition in addition to the total Hg deposition results used in the risk assessment.:* We expanded the presentation of Hg modeling results to include maps of wet and dry deposition for both the 2005 and 2016 scenarios (see section 2.2).

- *The discussion of the concentration-response (C-R) function used in modeling IQ loss, should be expanded to include coverage for the potential masking effect of MeHg-related IQ loss due to nutrients (polyunsaturated fatty acids - PUFAs) in fish. This is particularly relevant if IQ loss functions derived from saltwater fish consumption are used to model risk associated with freshwater fish consumption.* We expanded the discussion of

uncertainty on modeling IQ loss to address this issue (see Table B-2 entry B in Appendix B).

- *Additional sources of variability should be discussed in terms of the degree to which they are reflected in the design of the risk assessment and the impact that they might have on risk estimates. These include:*
    - o *The geographic patterns of populations of female subsistence fish consumers, including how this factor interacts with the limited coverage we have for watersheds with our fish tissue Hg data.*
    - o *The protocols used by states in collecting fish tissue Hg data.*
    - o *Fisher body weights and the impact that this might have on exposure estimates.*
    - o *Preparation and cooking methods which effects the conversion of fish tissue Hg concentrations (as measured) into "as consumed" values.*

We expanded the discussion of sources of variability presented in Table 2-14 (in section 2.7) to more fully address these sources of variability as requested.

- *Additional sources of uncertainty should be discussed in terms of their potential impact on risk estimates. These include:*
    - o *Emissions inventory (including the non-EGU segment) used in projecting total and U.S. EGU-attributable Hg deposition. This includes the projection of reduction in U.S. EGU emission for the 2016 scenario.*
    - o *Air quality modeling with CMAQ including the prediction of future air quality scenarios.*
    - o *Ability of the Mercury Maps-based approach for relating Hg deposition to MeHg in fish to capture Hg hotspots.*
    - o *The limited coverage that we have with fish tissue Hg data for watersheds in the U.S. and implications for the risk assessment.*
    - o *The preparation factor used to estimate "as consumed" fish tissue Hg concentrations.*
    - o *Proportionality assumption used to relate changes in Hg deposition to changes in fish tissue Hg concentrations at the watershed-level.*
    - o *Characterizing the spatial location of female subsistence fish consumer populations (including degree to which these provide coverage for high-consuming recreational fishers).*
    - o *Application of the RfD to low SES populations and concerns that this could low-bias the risk estimates.*

We expanded the discussion of sources of uncertainty presented in Table 2-15 (in section 2.7) to more fully address these sources of uncertainty and the potential impact on risk estimates.

1.2   Purpose and Scope of Analysis

This document is a revision to the March TSD, and provides a revised description of the national-scale risk assessment for Hg that was completed to inform the finding that it is appropriate and necessary to regulate electric utility steam generating units in the United States (U.S. EGUs), pursuant to Section 112(n)(1)(A) of the Clean Air Act (CAA). The appropriate and necessary finding is based, in part, on an assessment of the potential public health hazard

associated with Hg emitted from U.S. EGUs (see Section III of the preamble to the proposed Mercury and Air Toxics Standard (MATS) for U.S. EGUs).[5] Consequently, the risk assessment was designed to assess whether a potential public health hazard is associated with Hg emitted from U.S. EGUs. Because the focus of the analysis is to determine whether a potential public health hazard exists and not to characterize the full range of risk associated with exposure to Hg emitted from U.S. EGUs, this risk assessment can be viewed as a public health hazard screening analysis. As such, the primary objective is to determine whether individuals exposed to Hg emitted from U.S. EGUs through high-end consumption of freshwater self-caught fish have the potential to experience significant risk. Conversely, the risk assessment is not intended to represent a comprehensive assessment of risk for all segments of the population potentially exposed to Hg emitted from U.S. EGUs. It is beyond the scope of this risk assessment to assess the residual risk remaining after accounting for emissions reductions anticipated from MATS.

Given the purpose for the analysis, the following policy-relevant questions were developed to guide the design of the risk assessment:

(a)   What is the nature and magnitude the potential risk to public health in the United States for individuals experiencing reasonable high-end exposure to Hg in freshwater, self-caught fish that is attributable to current U.S. EGU Hg emissions?

(b)   What is the nature and magnitude of the potential risk for this same group of individuals based on projected U.S. EGU Hg emissions in 2016 considering potential reductions in EGU Hg emissions attributable to CAA requirements?[6] and

(c)   How is total risk from Hg exposure as estimated for both the current and future scenarios apportioned between the U.S. EGUs and other sources of Hg? The last policy-relevant question reflects the fact that Hg emitted from U.S. EGUs does not result in a distinct and isolated exposure pathway, but rather is combined with Hg emitted from other sources (domestic and international) in contaminating fish. Therefore, to consider U.S. EGU contributions to exposure and risk associated with the consumption of fish containing Hg, we determine what share of total Hg exposure is attributable to U.S. EGUs.

In addition to the above policy-relevant questions, the overall design and scope of the risk assessment reflects consideration of a number of important technical factors related to air-sourced Hg, including, in particular, Hg released from U.S. EGUs:

- While Hg exposure and risk can occur through a variety of pathways, the dominant human exposure pathway associated with ambient air emissions is through the consumption of fish that have bioaccumulated Hg originally deposited to watersheds following atmospheric release and transport. Deposition of Hg to watersheds includes Hg originating from local/regional sources, combined with Hg that has been transported

---

[5] 76 FR 24976

[6] For purposes of this analysis, we focus on 2016 as this is the year when compliance with mercury standards would be required to occur.

6

regionally and globally from global anthropogenic and natural sources. Generally oxidized (divalent) and particle-bound Hg will deposit relatively closer to the release source, while elemental Hg will travel much further, often becoming part of the global pool, before being deposited.[7]

- The scientific literature on mercury health effects provides the strongest support for quantifying neurological deficits in children who were exposed to MeHg *in utero* through maternal fish consumption.[8]

- Mercury emitted by U.S. EGUs is likely to make a small contribution to MeHg in foreign-sourced commercial fish consumed in the U.S. and in commercial fish caught further off the U.S. coast. Therefore, this risk assessment, while acknowledging these sources of exposure to U.S. EGU-sourced Hg, does not quantify these risks since the U.S. EGU-attributable portion of these risks is likely to be small and any quantitative estimates of U.S. EGU-attributable risk would be highly uncertain.[9]

- While areas closer to the U.S. coast (including estuarine areas) as well as the Great Lakes may have elevated U.S. EGU deposition in some cases, because of uncertainty in modeling the linkage between U.S. EGU-attributable deposition and Hg in fish, we have not included this commercial consumption pathway in the quantitative risk assessment.[10]

- The type of fish consumption likely to result in exposures to Hg with the greatest contributions from U.S. EGU Hg emissions is associated with fishing activity at inland freshwater rivers and lakes located in regions experiencing relatively elevated U.S. EGU Hg deposition. Some watersheds had U.S. EGU contributions ranging up to 11% and

---

[7] Mercury is a persistent, bioaccumulative toxic metal that is emitted from power plants in three forms: Gaseous elemental Hg (Hg^0), oxidized Hg compounds (Hg^{+2}), and particle-bound Hg (Hg_P). Elemental Hg does not quickly deposit or chemically react in the atmosphere, resulting in residence times that are long enough to contribute to global scale deposition. Hg(2+) and Hg(p) deposit quickly from the atmosphere impacting local and regional areas in proximity to sources.

[8] The EPA's health benchmark for methylmercury exposure (the reference dose or RfD) is based on three epidemiological studies. These studies relate hair mercury levels in mothers (a surrogate for exposure *in utero*) or mercury in cord blood (a direct measure of fetal exposure) to deficits in children's performance on a range of neuro-cognitive tests (see section 1.4.5).

[9] While mercury emitted from U.S. EGUs does contribute to contamination of foreign-sourced commercial fish, the fraction contributed by U.S. EGUs is small. Current estimates of U.S. EGU mercury emissions are ~29 tons per year (see section 2.3), compared with global anthropogenic mercury emissions (for 2005), excluding biomass burning, estimated at approximately 2,320 tons (Pirrone et al., 2010; UNEP, 2010). Since the mercury in commercially caught foreign-sourced fish also includes contributions by natural sources and re-emitted natural and anthropogenic sources, estimated to be as high as 5,207 metric tons/year (Pironne et al, 2010; UNEP, 2010), the fraction contributed by U.S. EGUs is quite small. Therefore, particularly in the context of estimating individual risk, U.S. EGU contributions to risk that residents in the US experience through consumption of commercially caught foreign-sourced fish, at present, is expected to be too small to characterize, given the uncertainties in determining the fraction of mercury that is from U.S. EGUs. This observation would also likely hold for the U.S. EGU contribution to commercial fish sourced from further off the U.S. coast, where total mercury loading is likely to also be dominated by non-U.S. anthropogenic emissions which are globally transported.

[10] While air quality modeling does suggest that some near coastal areas (e.g. the Chesapeake Bay) and portions of the Great Lakes may have elevated U.S. EGU deposition relative to the average levels in the continental U.S., several factors make modeling how changes in mercury deposition affect fish tissue Hg concentrations in these near-coastal areas and the Great Lakes challenging and uncertain. Specifically, the size of these waterbodies relative to inland lakes and rivers and the potential for fish to have larger habitats makes it difficult to quantify the EGU contribution to fish tissue Hg concentrations in these locations. Due to the greater uncertainty associated with modeling near coastal watersheds, we have elected not to simulate this pathway in the risk assessment. Because we have not assessed this pathway, this risk assessment may underestimate the number of at-risk watersheds in the U.S.

higher in 2016 (see Section 2.4). Therefore, efforts to identify areas with high U.S. EGU-attributable MeHg exposures and risk are focused on assessing risk for those areas that have (a) relatively elevated fish tissue Hg concentrations and (b) relatively elevated levels of U.S. EGU Hg deposition.

Reflecting consideration of the policy questions and technical factors discussed above, the scope of the national-scale Hg risk assessment is as follows.

- *Hg deposition was modeled for two years, 2005 and 2016*: The 2005 deposition is used to represent deposition that is reflected in the sampled fish tissue MeHg levels for 2000 – 2009. The projected 2016 deposition reflects emissions after imposition of CAA requirements. The available emissions data (see Section 2.3) suggest that current 2010 U.S. EGU emissions are closer to levels reflected in the projected 2016 Scenario and substantially lower than levels reflected in the 2005 Scenario. As a result, the 2016 Scenario analysis is most relevant for this rulemaking. Further modeling of future emissions indicates that in the absence of binding federal regulations U.S. EGU emissions are not likely to be substantially reduced between 2010 and 2016 (although the CAA requires the Agency to consider only federal CAA requirements in estimating future HAP emissions and attendant risks associated with EGUs). The 2016 Scenario is thus used to estimate risks related to both current emissions and emissions after implementation of the CAA requirements and risks are not generated for the 2005 scenario.[11] The estimated Hg emissions for U.S. EGUs in 2016, used in the deposition modeling for this TSD, is 29 tons.
- *Focus on assessing risk for female subsistence fish consumers of self-caught fish obtained from inland freshwater watersheds:* Given the goal of determining whether a public health hazard is associated with U.S. EGU emissions, we have assessed risk for those individuals likely to experience the greatest exposure and risk from consuming fish impacted by Hg emitted from U.S. EGUs. This translates into a focus on women of child-bearing age who consume subsistence-levels of fish caught from inland freshwater waterbodies.[12] By focusing on inland waterbodies, we are focusing on those locations with the greatest U.S. EGU-attributable Hg deposition (see Figure 2-4) and consequently the greatest U.S. EGU-attributable fish tissue Hg concentrations. By focusing on subsistence-level fish consumption scenarios, we focus on those self-caught fish consumers with the highest intake rates and therefore, those who will experience the greatest Hg exposures at a given watershed. In defining the high-end fisher populations to include in the analysis, we have used peer-reviewed study data to characterize consumption rates for a variety of high-consuming fisher populations that reflect different SES groups and are active in different regions of the country (e.g., Laotians, Great Lakes Tribal populations, Black and White anglers active in the Southeast - see Section 1.4.3). As noted earlier in section 1.1 for the analysis, we focus on women of child-bearing age

---

[11] Deposition estimates for the 2005 scenario are used in scaling fish tissue Hg concentrations to represent future fish tissue levels in 2016, which are then used in turn to model total risk for the 2016 scenario (see section 1.4.6).
[12] While subsistence-level consumption can reflect consumption by individuals whose SES status compels them to supplement their diet with self-caught fish, elevated levels of inland freshwater self-caught fish consumption can also be experienced by recreational anglers who fish often and consume a large amount of the fish they catch.

8

since the MeHg RfD used in assessing risk is based on neurodevelopmental effects in children exposed to mercury *in utero*.

- *Watershed-level assessment based on modeling female subsistence fish consumer risk at watersheds where there is the potential for this type of high fish-consuming activity and where we have fish tissue Hg data*: The risk assessment is conducted at the watershed level, focusing on the subset of inland watersheds in the U.S. where we have fish tissue Hg data and where we believe high fish-consuming subsistence fisher populations could be active.[13] Specifically, we generate a set of risk estimates for each watershed reflecting the type of subsistence fisher activity that could potentially exist at that watershed. Because it is not possible to enumerate these high-end fisher populations, the risk estimates that are generated are not population-weighted and instead are given a uniform weight for each watershed-level risk estimate generated.[14] This focus on subsistence fishing activity and associated consumption reflects the fact that this is a screening-level analysis for risk associated with mercury emitted from U.S. EGUs and is not intended to provide a comprehensive picture of the distribution of risk across all types of fishers potentially active at watersheds where we have Hg fish tissue data.

- *Exclude commercial fish consumption from the quantitative risk analysis.* Although risk associated with commercial fish consumption may be a potential public health concern under certain circumstances, the relatively low contribution of U.S. EGU Hg to this source of dietary fish (relative to non-US Hg emissions), leads us to exclude this consumption pathway from the risk assessment and to focus instead, on high-end self-caught fish consumers. In the specific case of commercial fish sourced from near the U.S. coast (e.g. Chesapeake Bay) and the Great Lakes, while there is the potential for U.S. EGUs to have a greater role in affecting Hg levels in these fish, as noted earlier, uncertainty associated with modeling the linkage between U.S. EGU Hg deposition and Hg exposure and risk for this dietary pathway precludes us from including this pathway in the risk assessment. We do not expect that exclusion of these scenarios will result in significant downward bias in our overall estimates of the proportion of watersheds with populations potentially at-risk due to U.S. EGU emissions of Hg, although the absolute number of watersheds will be understated.

---

[13] With the exception of the typical female subsistence fish consumer scenario (which is assessed across all watersheds with fish tissue Hg data), the potential for the other SES-differentiated female subsistence fish consumer scenarios to be active at a given watersheds is based on determining whether a group of similar SES-differentiated individuals (referred to as a source population) lives in the vicinity of the watershed (see section 1.4.3).

[14] In order to enumerate risk estimates generated for the female high-end consumer scenario used in this risk assessment, we would need to have the following types of specific information: (a) the fraction of anglers who consume at the subsistence-levels modeled for this population specifically at inland freshwater waterbodies, (b) for this population, the fraction that focus their activity at individual watersheds, and target somewhat larger fish to supplement their diet, and (c) for this subgroup, the fraction of female consumers of childbearing age who either fish themselves and consume at this level, or obtain and regularly consume fish provided by fishers who focus their fishing efforts at the individual watershed.. However, currently available information does not allow us to estimate each of these subgroups of high-consuming fishers. Specifically, while we have data on the frequency of recreational angling within the U.S., this covers general recreational fishing and not subsistence fishing. In this analysis, we have focused on a subset of female subsistence-level fish consumers that we believe (a) could potentially exist at a subset of watersheds evaluated in this analysis and (b) are likely to experience higher risk due to their behavior (i.e., favor larger fish as a dietary source, likely to consume fish obtained primarily from individual watersheds, consume larger amounts of self-caught fish). While we believe it is reasonable to assume that a subset of high-end fishers would have these attributes, it is not possible at this point to definitively state at which waterbodies they are active or to enumerate them for purposes of generating population-weighted risk distributions.

- *Include estimates of total risk from all Hg deposition sources, as well as the U.S. EGU incremental contribution to total risk.* As discussed below (Section 1.2), we focus on two aspects of MeHg-related risk: (a) total Hg risk, with an estimate of the percent of that total risk contributed by U.S. EGUs (i.e., the fraction of total risk associated with U.S. EGUs) and (b) risk when deposition from U.S. EGUs is considered alone, before taking into account deposition and exposures resulting from other sources of Hg. These two risk metrics reflect the cumulative burden of Hg exposures and incremental contribution that the U.S. EGU-attributable deposition makes to overall exposures to MeHg.[15]

1.3   Overview of Risk Metrics and the Risk Characterization Framework

The risk assessment generates hazard quotient (HQ) estimates by comparing estimates of modeled potential exposure for subsistence fisher populations to the MeHg RfD. In addition to the HQ estimates, Appendix A provides estimates of IQ loss in children born to mothers from these high fish-consuming subsistence fishing populations. Because of concerns expressed both by the SAB and by EPA staff that IQ loss may not fully capture the full range of neurodevelopmental deficits in children exposed to MeHg *in utero*, we focus on the HQ estimates in making our determination of the public health hazard associated with U.S. EGU Hg emissions.[16]

As mentioned above in section 1.2 and discussed in greater detail in sections 1.4 and 1.4.6, we generate risk estimates at the watershed-level for the subset of watersheds in the U.S. where we have Hg fish tissue data and where we believe the potential exists for high-end fish consumption due to the presence of subsistence fishers. As noted earlier, limitations in quantifying the number of high-consuming fishers active across the set of modeled watersheds prevents us from generating population-weighted risk distributions. However, we do use the watershed-level risk estimates (with uniform weighting across watersheds) to generate risk

---

[15] When exposures are to be compared to the EPA's reference dose (RfD) for MeHg in order to generate a hazard quotient (HQ), we must first consider total MeHg exposure given the definition of the RfD, which is intended to be compared against total exposure to a given hazardous air pollutant. Once an HQ reflecting total exposure is calculated, we can then consider the U.S. EGU incremental contribution to that total risk. However, U.S. EGU incremental risk in the form of an HQ should not be considered in isolation without considering total risk associated with MeHg in consumed fish.

[16] Concerns have been raised in the literature that if mercury affects a set of specific neurological functions, then use of full-scale IQ as the modeled health endpoint, could underestimate the neurodevelopmental impacts on other targeted functions (Axelrad et al., 2007). In addition, two of the most sensitive endpoints in the Faroe Islands study were the Boston Naming Test and California Verbal Learning Test, both of which can represent a significant educational risk depending on severity, and those tests are not directly assessed as part of measuring IQ in children. In addition, IQ does not cover other neurologic domains such as motor skills and attention/behavior and therefore, risk estimates based on IQ will not cover these additional endpoints and could further underestimate overall neurodevelopmental impacts (Axelrad et al., 2007). The wide range of neuropsychological effects potentially associated with Hg exposure has also been highlighted by Grandjean et al., (1997) who described developmental delays in verbal skills, learning and short-term memory, and more recently by Wijngaarden et al., (2006), who provided benchmark dose calculations for 26 endpoints including a number of neuropsychological measures. These studies highlight the range of neurodevelopmental effects in children potentially associated with Hg exposure. In contrast to the IQ loss metric, the HQ metric based on the RfD reflects consideration for a wider array of neurodevelopmental effects in children (e.g., Boston Naming Test, Continuous Performance Test, California Verbal Learning Test, McCarthy Perceived Performance, McCarthy Motor Test, finger tap – U.S. EPA's Integrated Risk Information System in 2001 - http://www.epa.gov/iris/subst/0073.htm).

distributions from which we can identify specific risk percentiles, (e.g. the 95th percentile of risk across watersheds). In this context, the distribution of modeled subsistence fisher risk estimates represents the range of risk across modeled watersheds based on assessing the same subsistence fisher scenario at each watershed (without population-weighting). Percentile risk estimates obtained from this distribution are for the population of watersheds, rather than the population of subsistence fishers.

Reflecting the goals of the analysis presented in Section 1.2, the HQ risk metrics are calculated for both <u>total risk</u> and <u>U.S. EGU-attributable risk</u>. In considering U.S. EGU-attributable risk, we generate two types of risk estimates:

- *The percent or fraction of total risk attributable to U.S. EGUs at watersheds where total risk is considered to pose a potential public health hazard:* We consider the magnitude of total HQ risk, identifying those watersheds with total MeHg exposure exceeding the RfD (i.e., an HQ>1) and then estimate the fraction (or percent) of that total risk that is attributable to U.S. EGUs.
- *Risk focusing on U.S. EGU deposition and excluding other non-U.S. EGU sources:* For this metric, we estimate risk based on the U.S. EGU incremental contribution to total exposure. Specifically, for HQ, we compare U.S. EGU-attributable exposure against the MeHg RfD.

In assessing the potential public health significance of HQ estimates, we considered total MeHg exposures above the RfD to represent a potential public health hazard.[17]

<u>Risk Characterization Framework</u>

We have developed a 2-stage framework for using the risk metrics described above to address the policy-relevant questions outlined in section 1.2. This 2-stage framework is illustrated in Figure 1-1 and each of the stages is also described below (throughout this document, this will be referred to as the "risk characterization framework"):

Stage 1 – *Identify watersheds with populations potentially at-risk due to U.S. EGU Hg based on application of the two risk metrics:*

 a) *The percent or fraction of total risk attributable to U.S. EGUs at watersheds where total risk is considered to pose a potential public health hazard :* Here we identify watersheds with populations potentially at-risk due to U.S. EGU Hg by identifying (a) those watersheds where <u>total</u> risk meets or exceeds levels

---

[17] EPA's interpretation for this assessment is that any exposures to MeHg above the RfD are of concern given the nature of the data available for Hg that is not available for many other chemicals, where exposures have often had to be significantly above the RfD before they might be considered as causing a hazard to public health. The scientific basis for the mercury RfD includes extensive human data and extensive data on sensitive subpopulations including children exposed *in utero* ; therefore, the RfD does not include extrapolations from animals to humans, and for database deficiencies. In addition, there is no evidence for a biological threshold observed for critical effect of neurological deficits in children studied in the principal studies of the IRIS assessment for MeHg. This additional confidence in the basis for the RfD suggests that all potential exposures above the RfD can be interpreted with more confidence as representing a hazard to public health.

considered to represent a potential public health hazard (i.e., HQ > 1); and (b) U.S. EGUs contribute to total risk at this subset of watersheds with elevated risk (we have considered various increments of U.S. EGU contribution ranging from 5 to 15%). Any contribution of Hg emissions from U.S. EGUs to watersheds where potential exposures from total Hg deposition exceed the RfD is a hazard to public health, but for purposes of our analyses we evaluated only those watersheds where we determined U.S. EGUs contributed 5 percent or more to Hg deposition in the watershed. EPA believes this is a conservative approach given the increasing risks associated with incremental exposures above the RfD.

b) *Risk focusing on U.S. EGU deposition and excluding other non-U.S. EGU sources::* Here we identify watersheds with populations potentially at-risk due to U.S. EGU-attributable risk (prior to considering Hg contributed by other sources). Although this metric focuses on U.S. EGU exposure, it is important to keep this incremental exposure in perspective with regard to total MeHg exposure in which non-U.S. EGU sources of deposition typically dominate the U.S. EGU increment across watersheds.

Stage 2 – *calculate the combined (total) number of watersheds and percentage of watersheds where populations may be at-risk from U.S. EGU-attributable Hg:* Here we combine estimates from Stages 1a and 1b to consider watersheds where populations may be at-risk due to (a) U.S. EGUs contributing to exposures at watersheds where total risk potentially poses a potential public health hazard or (b) U.S. EGUs making an incremental contribution to total Hg exposure which, when considered separate from total Hg exposure, represents a potential public health hazard.

This framework allows us to identify watersheds where U.S. EGU-related exposure considered separately, or as a portion of total risk, represents a potential public health hazard. More specifically, it allows us to estimate the number and percentage of watersheds where populations may be at-risk due to U.S. EGU-related Hg emissions.
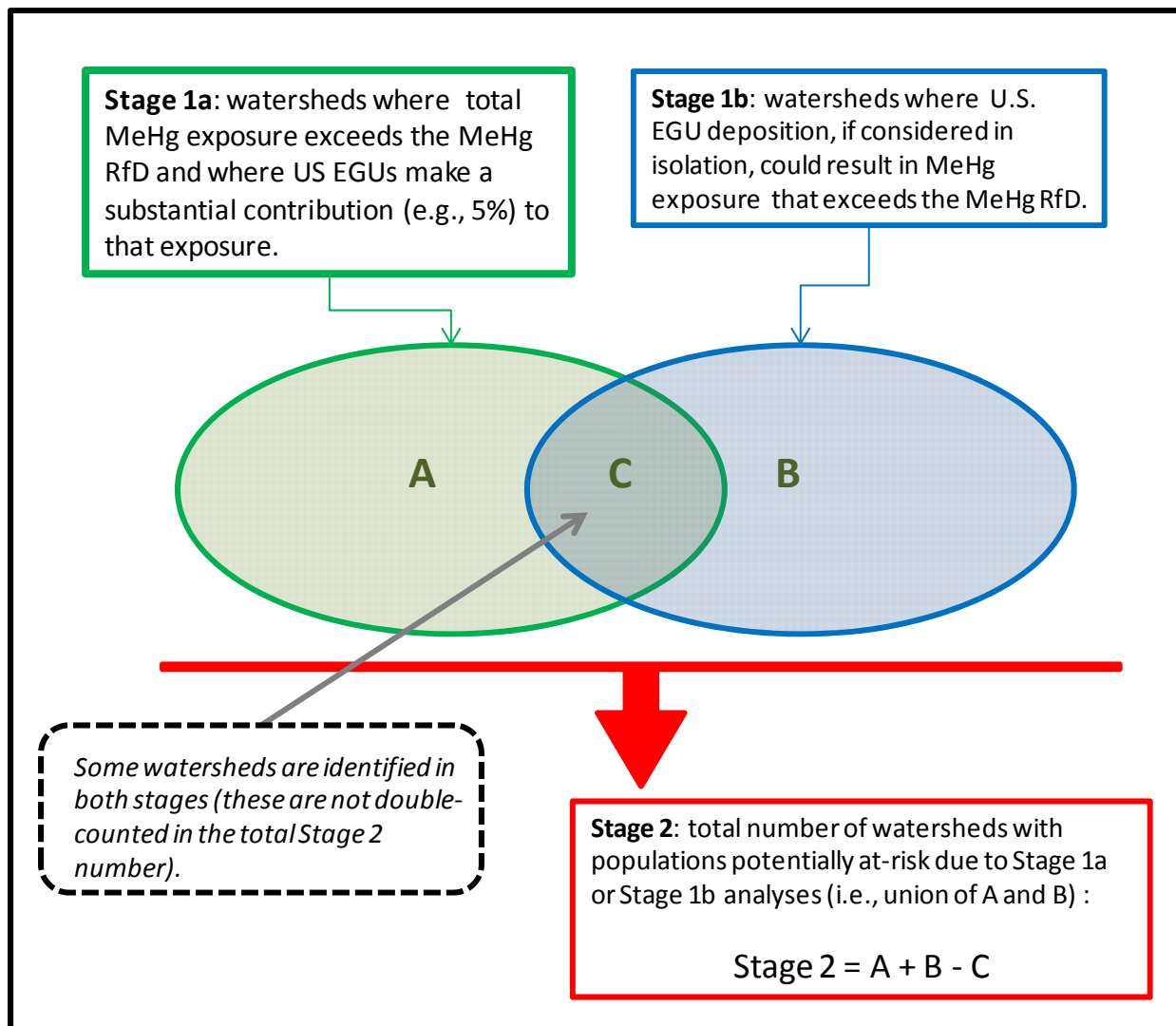
Figure 1-1. 2-Stage Risk Characterization Framework

1.4 Overview of Analytical Approach

This section provides an overview of the analytical approach for this risk assessment, which we illustrate in Figure 1-2. In this figure, we identify the subsections of the TSD with more detailed technical information on each of the analytical steps. We also provide a detailed example calculation of the total HQ and U.S. EGU incremental HQ at the watershed-level in section 1.6.
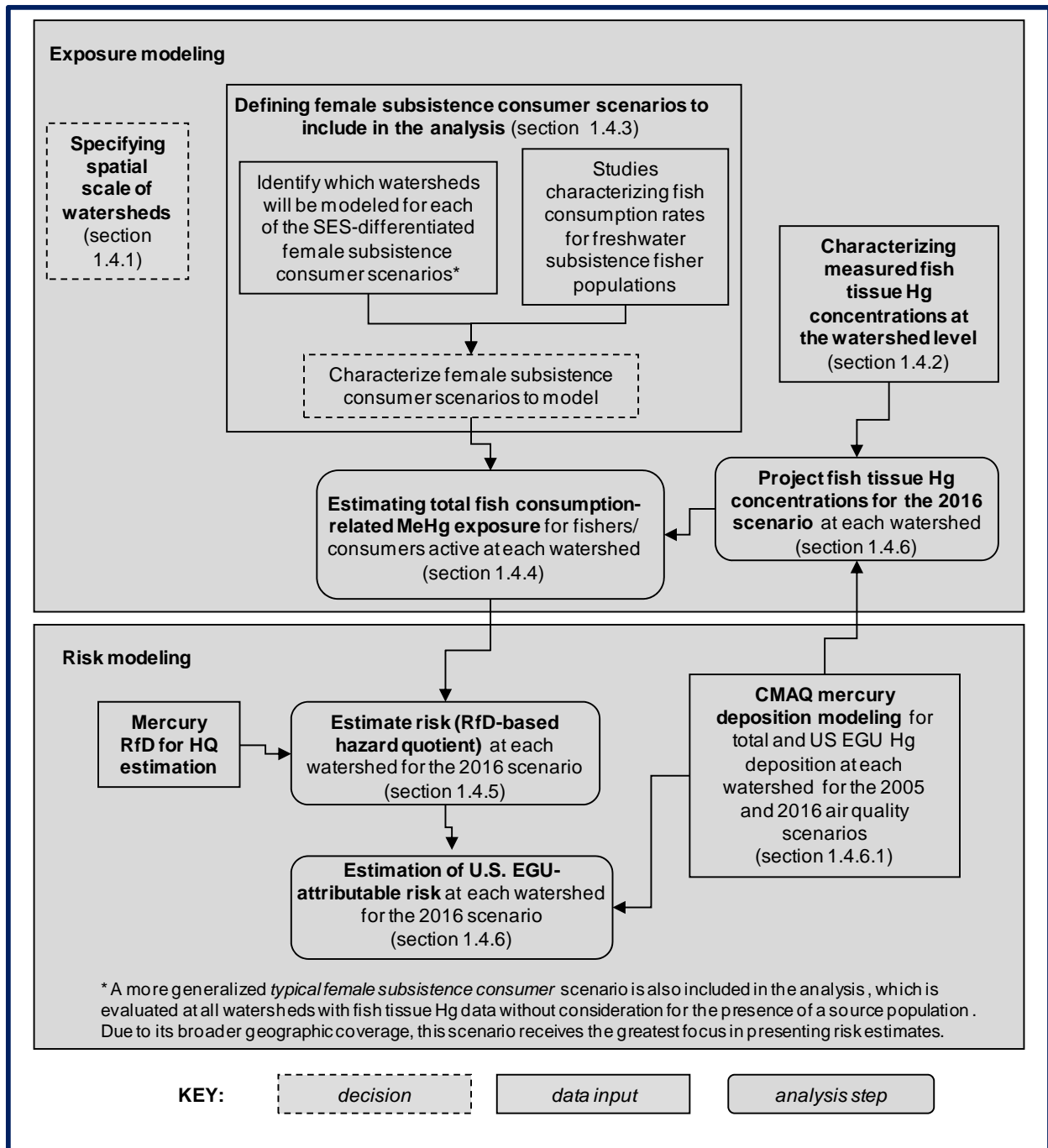
Figure 1-2   Flow Diagram of Risk Analysis (for the 2016 air quality scenario) Including Major Analytical Steps and Associated Modeling Elements

The risk assessment assesses risk to female subsistence fish consumers from potential exposure to MeHg from consuming fish caught in U.S. watersheds where we have measured fish tissue Hg concentration data and where we have determined that subsistence fishing activity could occur. As noted earlier in section 1.2, risks are only estimated for the 2016 scenario since

underlying U.S. EGU emissions used in modeling this scenario are closer to actual 2010 emissions than are emissions estimates used in modeling the 2005 scenario. However, as discussed below, Hg deposition estimates for the 2005 scenario are used in scaling fish tissue Hg levels to represent future levels associated with the 2016 scenario (which are in turn used in modeling risk for the 2016 scenario). We provide a brief description of each analytical step of the risk analysis reflected in Figure 1-2:

11. *Model mercury deposition:* We modeled total and U.S. EGU-attributable mercury deposition for the continental U.S. for 2005 and 2016 using the CMAQ model at 12 km grid resolution. (See section 1.4.6.3).

12. *Specify the spatial scale of the watersheds:* We selected Hydrologic Unit Code (HUC) 12 as the appropriate spatial scale for watersheds, which tend to measure a few kilometers (km) on a side and match up with the spatial resolution of the mercury deposition modeling. We interpolated the gridded mercury deposition estimates to deposition for each watershed in the continental U.S. (See section 1.4.1)

13. *Characterize measured fish tissue Hg concentrations (75th percentile) at each watershed*: We estimate the 75th percentile fish tissue Hg concentrations based on measurement data collected primarily by the states collected from 2000-2010 for inland freshwater fish species larger than 7 inches. We excluded watersheds without fish tissue data from remainder of the analysis. (See section 1.4.2). The fish tissue data did not include saltwater or estuarine fish.

14. *Project 75th percentile fish tissue Hg concentrations for the 2016 scenario*: We used the assumption of a proportional relationship between Hg deposition and fish tissue Hg concentrations together with deposition estimates for the 2005 and 2016 scenarios to project fish tissue Hg concentrations for the 2016 scenario. Specifically, we used the ratio of 2016 deposition to 2005 deposition at a given watershed to adjust the 75th percentile fish tissue Hg concentration discussed in Step 3, to represent a 2016 concentration for that watershed (see section 1.4.2.1).

15. *Define female subsistence fish consumer scenarios:* The analysis focused on females who consume subsistence-levels of fish that were caught at inland freshwater waterbodies. Our literature review identified seven female subsistence fish consumer scenarios that we selected to include in the analysis: (1) typical female subsistence fish consumer, (2) low income White fishers in the southeast, (3) low income Black fishers in the southeast, (4) low income Hispanic fishers, (5) Vietnamese fishers, (6) Laotian fishers, and (7) Tribal fishers (Chippewa active near the Great Lakes). (See section 1.4.3).

16. *Identify watersheds with subsistence fisher population activity:* For the *typical female subsistence fish consumer* scenario, we assume subsistence fisher population activity at all watersheds where we have fish tissue Hg data. For the Tribal fishers, we assume activity within all watersheds with fish tissue Hg data located within territories ceded to these tribes. For the remaining scenarios, we use demographic data to determine if at least 25 individuals with SES attributes matching those of the subsistence consumer population are present in the vicinity of a watershed, and if so, we assume that subsistence fishing activity might occur at that watershed. (See section 1.4.3)

17. *Define self-caught fish consumption rates for the subsistence scenarios:* We used survey data published in the literature to identify self-caught fish consumption rates for each female subsistence fish consumer scenario. In each case, at the high end (e.g., 90th to 99th percentiles), consumption rates reached subsistence levels, ranging from an 8oz self-caught fish meal every few days to an 8oz meal every day and higher in some cases. (See section 1.4.3).

18. *Estimate total fish consumption-related MeHg exposure (2016 scenario)*: We estimated exposure in the form of daily-average MeHg intake at the watershed-level for each female subsistence fish consumer scenario potentially active at a given watershed. These estimates of exposure use the projected 75th percentile fish tissue Hg concentrations for 2016 described in Step 4 above. We were careful to match the fish sampling data with type of sampling (e.g., filet skin on, whole fish), cooking adjustment, and type of consumption rates (e.g., as purchased, as consumed). The exposure estimates represent potential exposure to MeHg from fish caught in the watershed, not population-weighted exposures. (See section 1.4.4)

19. *Estimate of total MeHg risk (RfD-based HQ) at each watershed (2016 scenario)*: We compare the watershed-level exposure estimates modeled for each female subsistence fish consumer scenario (for 2016) to the MeHg RfD to generate HQ estimates for total MeHg exposure. (See section 1.4.5)

20. *Estimate of U.S. EGU-attributable risk (2016 scenario)*: We used the same proportionality assumption discussed in Step 4, together with total and U.S EGU-attributable Hg deposition estimates (generated at the watershed-level) for the 2016 scenario to estimate the fraction of total HQ (for the 2016 scenario) attributable to U.S. EGUs. Specifically, we used the ratio of U.S. EGU-related deposition to total deposition (for the 2016 scenario) at a given watershed calculate the fraction of total risk (generated in Step 9) that is attributable to U.S. EGUs (see section 1.4.6).[18]

1.4.1 Specifying the spatial scale of watersheds

The first step in designing the analysis was to specify the spatial scale of the watersheds to use as the basis for risk characterization. As noted above, this risk assessment is based on estimating risk at watersheds for which we have measured Hg fish tissue data. Two studies (Knights et al., 2009, Harris et al., 2007) examining the response of aquatic freshwater ecosystems to changes in Hg deposition focused on watersheds with dimensions closest to HUC-12. In each of the studies, researchers used watersheds reflecting a fairly refined spatial scale

---

[18] Given the assumption that fish tissue Hg concentrations and exposure and risk are linearly related, the proportionality assumption (together with the ratio of U.S. EGU-related to total Hg deposition for the 2016 scenario) could be applied either to the fish tissue Hg concentrations or to total risk in order to generate U.S. EGU-attributable risk. For the analysis, as reflected in Step 10, we applied the proportionality assumption and deposition ratio once total risk (for the 2016 scenario) had been generated. However, we could have used the proportionality assumption to estimate the fraction of the 75th percentile fish tissue Hg concentration (projected for the 2016 scenario) that was attributable to U.S. EGUs at each watershed and then used that fractional fish tissue value to estimate U.S. EGU-attributable risk for the 2016 scenario. Both approaches would generate the same risk estimate, reflecting the underlying linearities in the exposure and risk models.

(approximately 5-10 km on a side). This suggests that, at least in the context of these studies, researchers believed that the relationship between changes in Hg deposition and changes in MeHg levels in aquatic biota could be effectively explored at the level of these more spatially refined watersheds. Each of the studies is briefly summarized below.

The Knights et al., 2009 study focused on characterizing the temporal pattern of reductions in fish tissue Hg concentrations following reductions in Hg deposition over waterbodies and associated watersheds. This study relied on modeling and included simulation of five different types of waterbodies ranging from a seepage lake (with little watershed loading) in Florida to a stratified drainage lake in NH. The scale of the five watersheds included in the Knights et al., 2009 study range from 20 by 100 km (for the coastal plain river location in GA) to 5 by 10km (for the Lake Waccamaw NC site). Three of the five locations had watersheds in the 10 by 10km range (see Figure 2 in the article). Given that the majority of locations in the study had smaller watersheds (i.e., in the 10 by 10km range), we conclude that this would represent a reasonable watershed spatial scale to use in linking changes in aerial deposition to changes in fish tissue levels (i.e., as the basis for risk characterization in the analysis).

An article by Harris et al., 2007, which is based on the METALLICUS study (specifically lake 658 catchment in northwestern Ontario, Canada), also examined the temporal profile associated with changes in media and biota Hg levels following a change in Hg deposition. In this study, a 3-yr loading of radio-labeled Hg to the waterbody and watershed (separate labeled Hg applied to each location) was followed by measurement of Hg in various media and biota to see how long it took for the loaded Hg to impact different media compartments. The single watershed involved in this study is relatively small (only a few km on a side). Therefore, the spatial scale of the watershed involved in this study also supports use of a more refined spatial scale for watersheds in the risk assessment.

In addition to considering the scale of watershed reflected in these two studies of Hg loading response, use of a more refined spatial scale (i.e., use of HUC12s rather than a coarser scale of watershed) in linking changes in Hg deposition to changes in fish tissue Hg concentrations also reduces the potential for averaging out areas of high Hg deposition. The HUC12 represents the most refined scale of watershed currently available at the national level and therefore was chosen as the basis for linking changes in Hg deposition to changes in fish tissue Hg concentrations. Conversely, use of larger watersheds, while allowing us to model more of the country in the risk assessment, could result in the unwarranted dilution of areas of elevated Hg deposition from U.S. EGUs (and therefore, by association, the dilution of U.S. EGU-attributable Hg exposure and risk). The SAB expressed support for the use of HUC12-scale watersheds.

1.4.2 Characterizing measured fish tissue Hg concentrations at the watershed level

The next step in the analysis was to characterize fish tissue Hg concentrations at the watershed level using measured fish tissue Hg data. This process involved three tasks: (a) develop the database of fish tissue Hg concentrations based primarily on state-level data, using a number of filtering steps that are described below, (b) use the fish tissue database to generate percentile estimates (75th and 50th percentiles) for each watershed containing fish tissue Hg

measurement data and (c) filter the set of watersheds with fish tissue Hg percentile estimates to exclude locations potentially impacted by non-atmospheric Hg sources.

The SAB recommended that EPA evaluate whether there were additional fish tissue data that could be used to update the fish tissue dataset (see Section 1.1). We implemented this recommendation, and as a result, the fish tissue Hg dataset used in the revised national-scale risk assessment is an augmented version of that used in the March assessment. Because the fish tissue Hg dataset is different from the March TSD, this section provides an overview of the 2010 Mercury Fish Tissue (MFT) dataset as well as the augmentation steps of the 2010 MFT dataset based on additional fish tissue data identified for a handful of states. We have also included additional detail on the derivation of the fish tissue Hg dataset in response to an SAB recommendation. We describe each step to develop the dataset ultimately used in this risk assessment, which is illustrated in Figures 1-3 through 1-6. We also provide the number of fish tissue Hg measurements and watershed-level estimates associated with each step in the filtering process.

Development of the 2010 Mercury Fish Tissue (MFT) database used in the March version of the national-scale mercury risk assessment

To develop the 2010 MRT dataset, we began with fish tissue samples from three main sources:

- National Listing of Fish Advisory (NLFA) database. The NLFA, managed by EPA (http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/), collects and compiles fish tissue sample data from all 50 states and from tribes across the United States. In particular, the 2010 version of the NLFA used in this analysis contains data for over 45,000 Hg fish tissue samples collected from 1995 to 2007.

- U.S. Geologic Survey (USGS) compilation of mercury datasets. As part of its Environmental Mercury Mapping and Analysis (EMMA) program, USGS compiled Hg fish tissue sample data from a wide variety of sources (including the NLFA) and has posted these data at http://emmma.usgs.gov/datasets.aspx. To avoid duplication in our analysis, we excluded all of the USGS data originating from the NLFA. As shown in Figure1-3, we included data from the USGS compilation that originated from two main categories of sources:

  (1) state-agency collected and reported data (including Delaware, Iowa, Indiana, Louisiana, Minnesota, Ohio, South Carolina, Virginia, Wisconsin, and West Virginia) from nearly 40,000 fish tissue samples, covering the period 1995 to 2007 (referred to as "USGS States" in Figure 1-3)

  (2) over 6,000 fish tissue samples from several other sources, including the National Fish Tissue Survey, the National Pesticide Monitoring Program (NPMP), the National Contaminant Biomonitoring Program (NCBP), the Biomonitoring of Environmental Status and Trends (BEST) datasets of the USFWS and USGS (http://www.cerc.cr.usgs.gov/data/data.htm), and the Environmental Monitoring and

Analysis Program (EMAP) (http://www.epa.gov/emap/). (referred to as "USGS Other" in Figure 1-3)

- EPA's National River and Stream Assessment (NRSA) study data. These data include nearly 600 fish tissue Hg samples collected at randomly selected freshwater sites across the United States during the period 2008 to 2009.

All of the measurements in the NLFA database were obtained from state fish advisory programs managed by the health department, natural resource department or environmental protection agency in each state. In many cases, these data were not sampled randomly but instead reflect protocols intended to target (1) areas know to support recreational and or commercial fishing and (2) areas believed to have elevated levels of chemical contamination (i.e., MeHg) in fish.[19] Therefore, the state-level data do not provide a representative characterization of the distribution of MeHg concentrations in fish tissue at waterbodies across the state and are instead, likely to be biased towards locations with higher Hg fish tissue concentrations as well as species that are typically consumed by the general public. However, because the goal of this analysis is to determine the potential for a public health hazard from Hg emitted by U.S. EGUs, it is beneficial to have fish tissue measurement data potentially biased towards waterbodies with greater Hg impacts as well as species typically consumed by recreational or commercial fishers because this reduces the likelihood that high risk watersheds would be omitted due to gaps in fish sampling.

The majority of fish tissue Hg measurements in these datasets are for total Hg and not MeHg. However, research published in the literature suggests that 90 to 95 (or greater) percent of total Hg in fish tissue is MeHg (U.S. EPA, 2000). Based on this research, for purposes of assessing exposure and risk, we have assumed that 95% of each fish tissue Hg concentration is MeHg (see section 1.4.4).

Data from the four datasets shown in Figure 1-3 were combined into a single "2010 Master" Hg fish tissue (MFT) sample dataset covering the period 1995 to 2009. One problem encountered in combining these datasets is the potential duplication of samples in the NLFA and

---

[19] We reviewed approaches used by Pennsylvania and Wisconsin for determining where to collect fish tissue Hg measurements. These states were chosen because they either have relatively high fish tissue Hg concentrations (Wisconsin), or relatively elevated U.S. EGU-related Hg deposition (Pennsylvania). Pennsylvania identifies sampling locations based on (a) a need to verify or delist specific locations for fish advisories, (b) provide additional data points for their Water Quality Network and (c) respond to requests from selected State Environmental and Wildlife officials to sample locations of potential concern (Pennsylvania Department of Environmental Protection's "Fish Tissue Sampling and Assessment Protocol" available at: http://www.portal.state.pa.us/portal/server.pt/community/fish_consumption/10560). Wisconsin selects sampling locations based on providing coverage for (a) northern parts of the state where high Hg fish tissue levels have been found, (b) areas were pollutions impacts are suspected, (c) areas with high fishing pressure and (d) locations that are considered "indicators" for specific watersheds (Wisconsin Department of Natural Resources, "A Summary of Mercury Concentrations in Fish (edible portions) from Wisconsin Waters 1990-2005", available at: http://dnr.wi.gov/fish/consumption/1990-2005mercurysummary.pdf). While the details of these two strategies do differ somewhat, generally both states are targeting areas either suspected of having a potential Hg problem, or areas with elevated fishing activity. In both cases, the set of fish tissue Hg concentrations are not going to be generally representative of trends across watersheds in the state and instead, are likely to favor coverage of more heavily impacted watersheds (at least for the fraction of samples collected to cover areas of potential concern with regard to mercury impacts).

USGS-States data. Unfortunately, these two datasets do not contain directly comparable and unique identifiers that allow duplicate samples to be easily identified and removed. In order to identify potentially duplicative samples, we subdivided the samples from these two datasets into data groups according to the year and state in which they were collected. If both datasets contained a data group for the same year and the same state, then the data group with the fewer number of observations was not included in the master data. This process excluded 18,860 potentially duplicate samples from the master dataset.

In finalizing the 2010 MFT sample dataset, the following filters were used to further screen the fish tissue samples:

(a) excluded samples that were not geo-referenced (i.e., did not include latitude and longitude information)

(b) excluded samples with missing date information

(c) excluded samples that were not from freshwater fish species or freshwater locations (i.e., excluded estuarine locations),

(d) excluded samples from fish smaller than 7 inches in length. [20] Additional discussion of uncertainty related to this filtering step is presented later in this section.

These filters excluded a total of 21,674 samples from the dataset (in addition to the 18,860 duplicates referenced earlier). For the majority of measurements in the database, we also have information on the type of waterbody (river/lake) and type of sampling method used (e.g., filet skin on). Additional detail on the process used to develop the master fish tissue dataset can be found in U.S. EPA, 2011a, Section 5.2.2.

Even though we compiled fish tissue Hg sampling data for 1995 to 2009, we decided to restrict data to 2000 to 2009 in the risk assessment (the SAB supported this decision). We excluded fish tissue samples that likely reflected Hg deposition levels from the 1990's when anthropogenic emissions in the U.S. were higher than after 2000. We recognize the complex spatial and temporal nature of the response of fish tissue Hg concentrations to changes in Hg deposition and loading and acknowledge that a portion of the sampling data from 2000 to 2009 could still reflect higher Hg loading rates from earlier periods. Excluding the samples collected before 2000 further reduced the size of the initial dataset by 27,522 observations (the potential impact on the risk assessment related to our decision to focus on data from 2000 and later is discussed in section 2.7, Table 2-15, Entry I).

After completing all of these filtering steps, the 2010 MFT sample dataset contained 23,770 observations (samples) in the U.S.

---

[20] Seven inches represents a minimum size limit for a number of key edible freshwater fish species established at the State-level. For example, Pennsylvania establishes 7 inches as the minimum size limit for both trout and salmon (other edible fish species such as bass, walleye and northern pike have higher minimum size limits (*Summary Book: 2001 Pennsylvania Fishing Laws and Regulations* available at: http://fishandboat.com/fishpub/summary/inland.html).

Original Data Sources (>=1995)

NLFA (45,166)

USGS States (39,972)

USGS Others (6,098)

NRSA (590)

Data Merging

Merged NLFA-USGS States (66,278)

2010 Master (72,966)

Data Screening

Reason Dropped	N
Missing Lat/Long	-9,177
Missing Date	-1,520
Not a Freshwater Fish	-2,638
Not a Freshwater Location	-3,192
Length < 7 inches	-5,147
Year < 2000	-27,522
Total	**-49,196**

2010 MFT Sample Dataset (23,770)

Figure 1-3   Diagram Illustrating Step-wise Procedure Used to Develop 2010 Mercury Fish Tissue (MFT) Dataset Used in the 2010 National-Scale Mercury Risk Assessment

<u>Augmentation of the 2010 MFT database with additional fish tissue mercury data obtained for a subset of states (i.e., MI, NJ, MN, PA, and WI).</u>

Since the March TSD, we augmented the 2010 MFT sample database with additional sampling data from selected states and years. The composition and processing of these "augmentation" data are shown in Figure 1-4. All of these data were provided to EPA by the individual states, but they have not yet been incorporated into the most recent versions of the NLFA. The main criteria for selecting these particular 5 states are: (1) a majority of the sample data they provided to EPA were geo-referenced and included the year in which they were sampled (more recent data for a number of the other states did not have these critical descriptors) and (2) the states are located in areas of the country with relatively high levels of Hg deposition.

To avoid potential duplication of samples with those included in the initial Hg fish tissue database, the augmentation data only included samples for the years 2003 to 2009 for MI, NJ, MN, and PA, and for 2000 to 2010 for WI (Figure 1-4 provides more detail).[21] All of the data were reorganized and converted into Microsoft Access databases, using the NLFA fields and

---

[21] The version of the NLFA used in developing the 2010 MFT dataset did not include samples collected between 2003 and 2009 for MI, NJ, MN or PA. Similarly, that earlier version of the NLFA did not include any WI samples collected between 2000 and 2010. Therefore, addition of samples from the latest version of the NLFA from these years for these 5 states would supplement the MFT without duplication.

formats. They were then combined into the "Augmentation Master" dataset shown in Figure 1-4, which includes a total of 12,864 samples.

Using the same set of screening steps, we ended up with an Augmentation MFT sample dataset containing 11,797 observations in the U.S.[22]

For this analysis, we then combined the 2010 MFT sample dataset with the Augmentation MFT dataset (as shown in Figure 1-5) to create the 2011 MFT sample dataset. This combined dataset includes 35,567 fish tissue Hg samples from years 2000-2010. As with the original 2010 MFT database, samples from the 2011 MFT database are located across the U.S.; however, they are more heavily focused in locations east of the Mississippi River.

Reason Dropped	N
Missing Lat/Long	-643
Missing Date	-0
Not a Freshwater Fish	-13
Not a Freshwater Location	-0
Length < 7 inches	-411
Year < 2000	-0
Total	**-1,067**
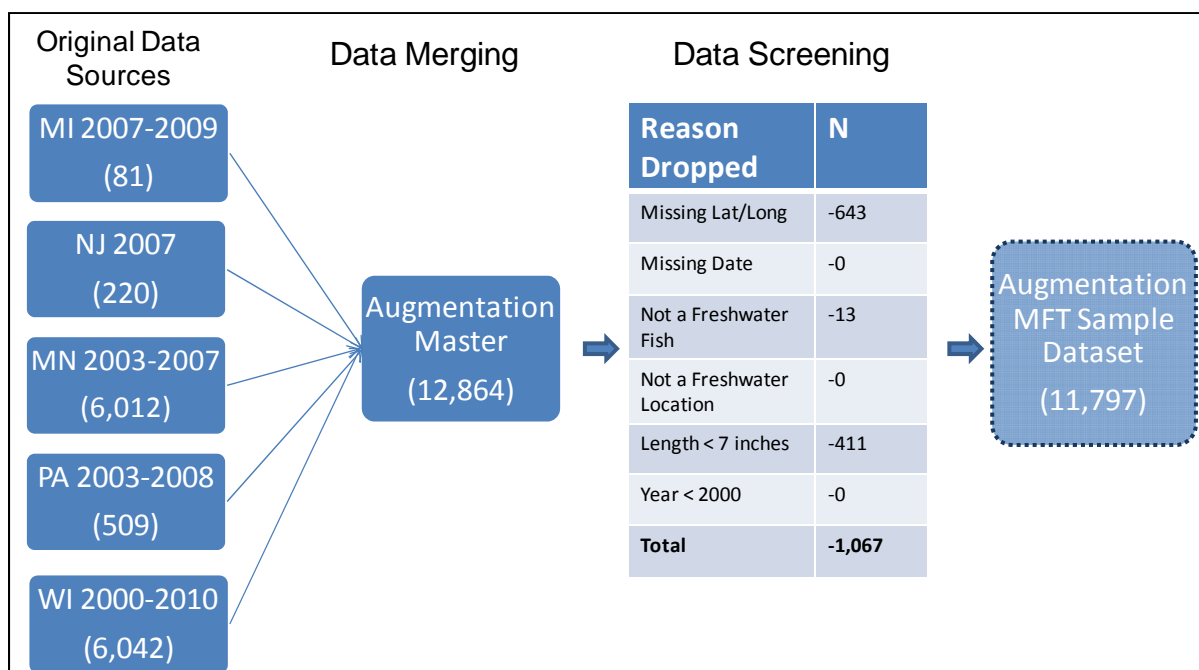
Figure 1-4   Diagram Illustrating Step-wise Procedure Used to Develop the Augmentation Mercury Fish Tissue (MFT) Dataset

---

[22] The data fish tissue sample data from PA did not include a measure of fish length; therefore, all of the samples were retained and assumed to be above the 7 inch threshold. In the March assessment, these samples were excluded, however, in order to provide a more complete spatial coverage in PA, which has high levels of U.S. EGU Hg deposition, we elected for this revised assessment to include these samples.
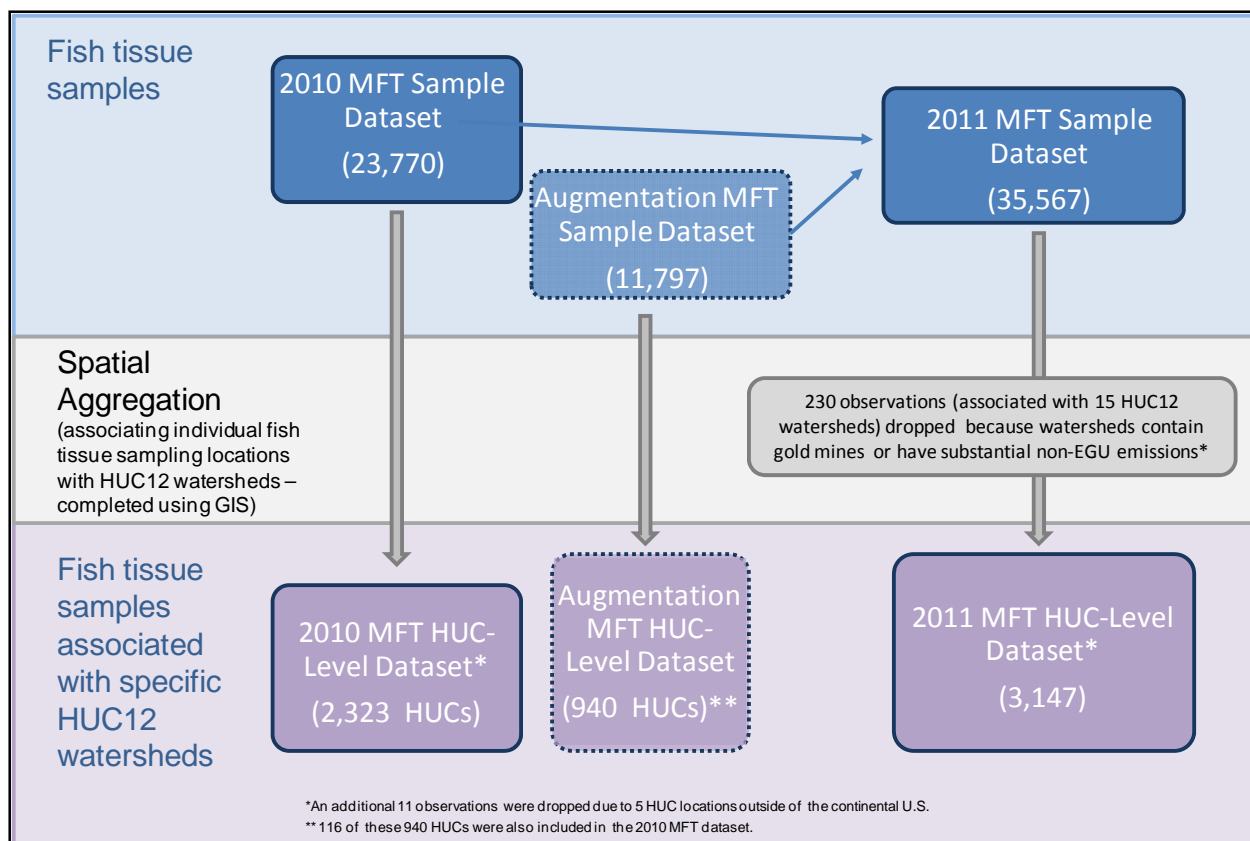
Figure 1-5 Diagram Illustrating Step-wise Procedure Used to Combine the 2010 MFT and Augmentation MFT Datasets

<u>Use the fish tissue database to generate percentile estimates (75th and medians) for each HUC-12 watershed containing fish tissue Hg measurement data</u>

To conduct the risk and exposure analysis, we then spatially aggregated the 2011 MFT sample data to the watershed level. To begin this process, we used the latitude and longitude information from each sampling location to identify the HUC-12 watersheds in which each site was located.

As a final data screening step, we then excluded HUC-12 watersheds (and the samples located within those watersheds) that either contained active gold mines or had other substantial non-U.S. EGU anthropogenic emissions of Hg.[23] These watersheds were excluded because the assumption of linear proportionality between Hg deposition and fish tissue Hg concentrations, supported by the MMaps study is most supportable in those situations where aerial deposition is the dominant source of Hg loading to a watershed. We identified watersheds with gold mines

---

[23] The filtering out of locations with potentially significant non-air mercury loadings was also completed in generating the 2010 MFT database. At this stage, to restrict the analysis to the continental U.S., where we have Hg deposition modeling, we also excluded 11 samples that were located in 5 HUC-12 watersheds in Alaska.

using a USGS data set characterizing mineral and metal operations in the U.S. (USGS, 2005). The data represent commodities monitored by the National Minerals Information Center of the USGS, and the operations included are those considered active in 2003. We identified watersheds with substantial non-EGU anthropogenic emissions using a TRI-net query for 2008 for non-EGU Hg sources with total annual on-site Hg emissions (all media) of 39.7 pounds or more.[24] This threshold value corresponds to the 25th percentile annual U.S.-EGU Hg emission value as characterized in the 2005 NATA.[25] The 25th percentile U.S.-EGU emission level was selected as a reasonable screen for additional substantial non-U.S. EGU emissions to a given watershed. The SAB endorsed this as a sound approach, while noting that the degree of conservatism implied by this approach is unknown. The SAB also commented that while other screening criteria could be applied, such as removing watersheds near urban areas with potential waste runoff, these would be unlikely to substantially change the results. In addition, applying additional screening criteria would have the negative impact of further reducing the geographic scope of the analysis.

Application of the filtering described here (i.e., excluding locations with active gold mines or other substantial non-U.S. EGU anthropogenic emissions of Hg) resulted in 15 HUC12 watersheds, containing a total of 230 observations, being excluded from the 2011 MFT dataset (see Figure 1-5). The final number of HUC12s with fish tissue Hg data included in the risk assessment is 3,141 (containing 35,567 samples), which represents a 33% increase in the number of watersheds assessed since the March TSD, as summarized in Figure 1-5.

As shown in Figure 1-5, this approach yielded 3,141 HUC-level sets of fish tissue Hg estimates for the augmented (full) dataset used in the revised national-scale mercury risk assessment (this compares with 2,317 HUC-level fish tissue Hg estimates used in the March version of the risk assessment).

Because most HUC-12 watersheds with measured fish tissue Hg data have multiple sampled values, often distributed over multiple sampling sites, we needed to identify and calculate summary statistics for each watershed, in order to represent fish tissue Hg levels in estimating exposure and risk. To do this, we generated summary statistics (means and percentiles) for MeHg concentrations in each HUC-12 and compiled these summary data into a separate HUC-level dataset. The following two-step procedure was used to generate HUC-level statistics: (a) calculate mean and percentile (25th, 50th, 75th and 90th) fish tissue Hg values for each sampling site within a HUC and (b) for the mean, or a given percentile, take the average of the applicable values across the sampling sites within a HUC to generate a single HUC-level estimates (for the mean, or specific percentile). For example, if we had two sampling sites (each with multiple samples) and we wanted a 75th percentile fish tissue Hg value for that HUC, we would first compute the 75th percentile values at each sampling site and then take the average of those two 75th percentile values. If there is only one sampling site in a given HUC, then we simply compute the mean or percentile of interest from the measurements at that site and that statistic is used to represent the HUC. This approach reflects a fisher who fairly consistently targets the same size (represented by percentile) fish at each location where they fish within a

---

[24] U.S. EPA Toxics Release Inventory (TRI) accessed at: http://www.epa.gov/tri/ .
[25] U.S. EPA 2005 National Air Toxics Assessment (2005 NATA) accessed at: http://www.epa.gov/ttn/atw/nata2005/..

watershed. We are then constructing a representative fish tissue Hg value for them, by taking the average of the fish they are simulated to catch across their fishing locations (within a given HUC). As noted earlier, our simulation of female subsistence fish consumer exposure and risk reflects the assumption that fishing activity is targeted within a given watershed.[26]

Summary statistics for the 2011 MFT HUC-level datasets are reported in Table 1-1.[27] The average number of fish tissue measurements for the period 2000 to 2009 for the 3,141 watersheds is 11.23, although some watersheds contained up to 360 measurements. Most watersheds also have multiple species (mean of 2.69) and to a lesser extent, multiple sampling sites within the watershed (mean of 1.41). Multiple sampling sites in this context can be different streams and/or lakes located within the same watershed. Distributions of other HUC-level percentiles (and the mean) are also presented for purposes of comparison.

Table 1-1 Summary Statistics for the 2011 MFT HUC-level Data

Distribution of various attributes across the 3,141 HUCs	Mean	Std. Dev.	Min	Max
Number of samples per HUC	11.23	21.05	1	360
Number of species per HUC	2.69	2.19	1	17
Number of sampling sites per HUC	1.41	1.25	1	33
Average of different HUC-level Hg concentration percentiles (including the mean) across the 3,141 HUCs				
Average of location-specific mean Hg concentrations (ppm) in HUC	0.27	0.24	0	3.56
Average of location-specific 25th percentile Hg concentrations (ppm) in HUC	0.19	0.19	0	2.20
Average of location-specific 50th percentile Hg concentrations (ppm) in HUC	0.25	0.23	0	3.56
Average of location-specific 75th percentile Hg concentrations (ppm) in HUC *	**0.32**	**0.31**	**0**	**6.61**
Average of location-specific 90th percentile Hg concentrations (ppm) in HUC	0.40	0.41	0	7.35

* 75th percentile row bolded since this is the statistic used in the risk assessment

---

[26] The SAB recommended that a single fish tissue Hg value not be used to represent a watershed when there are multiple measurement sites (e.g., multiple lakes) within that watershed. This recommendation reflected concern that waterbodies, even when in close proximity within the same watershed, can display different methylation rates such that they are likely to respond differently to a unit change in Hg deposition. The approach outlined here for calculating the 75th percentile fish tissue Hg value does not use a single fish tissue Hg value in instances where there are multiple sites within a watershed. Instead, we calculate the 75th percentile fish tissue Hg for each site (i.e., with each value representing a specific sampling site, which often translates into a distinct waterbody) and then take the average of those values, reflecting fishing activity that is distributed across the sampling sites, or waterbodies within a specific watershed.

[27] In the 2010 MFT dataset used in the March version of the risk assessment, approximately 48% of the fish tissue Hg samples were obtained from rivers (with the rest coming from lakes). For the augmentation dataset, we did not specify river versus lake in developing the fish tissue Hg dataset, since this distinction was downplayed by the SAB as a factor significantly impacting application of the proportionality assumption in the context of estimating the U.S. EGU-attributable portion of exposure and risk.

As noted in Section 1.4, we selected the 75th percentile fish tissue value at each watershed as the main basis for exposure and risk characterization.[28] Selection of the 75th percentile value was based on the assumption that a subset of subsistence fishers would favor larger fish which have the potential for higher bioaccumulation (i.e., use of a median or mean value could low-bias likely catch-related Hg levels). There is uncertainty associated with this assumption and should fishers at a particular watershed favor fish that are either larger or smaller than the type of fish reflected in the 75th percentile sample, risk estimates could be biased accordingly. Uncertainty related to use of the 75th percentile fish tissue Hg value in exposure modeling (specifically, potential bias in the estimation of the 75th percentile value) is discussed in section 2.9, Table 2-15, Entry C. As recommended by the SAB, we also included of a sensitivity analysis using the median fish tissue Hg concentration at each HUC – see section 2.8).
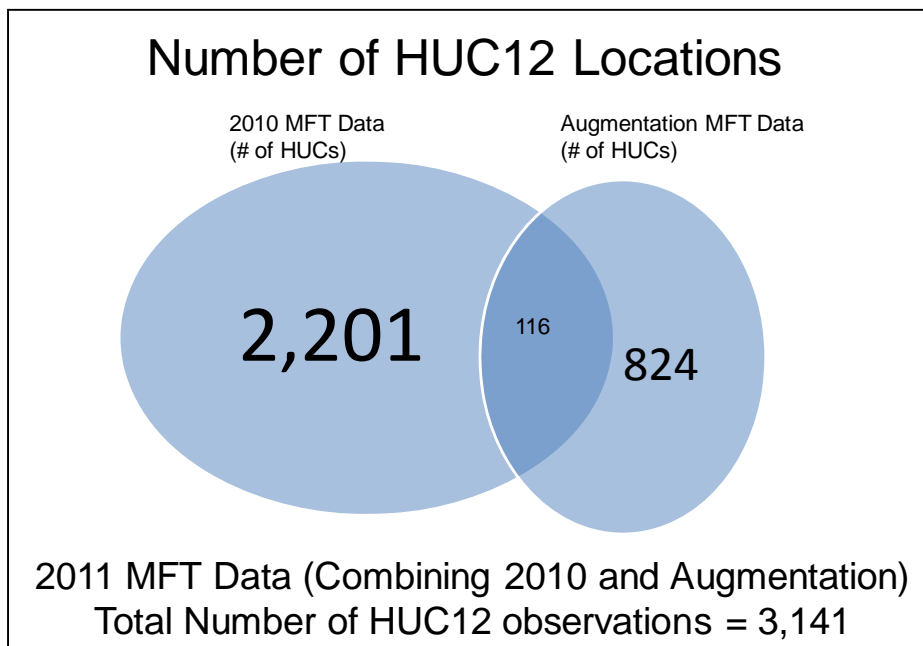
Figure 1-6 Diagram Illustrating Number of HUC12s with Fish Tissue Mercury Data (for 2010 MFT, Augmentation MFT and the Combined 2011 MFT Datasets)

<u>Uncertainty related to low sample size for a substantial fraction of the HUC12s and exclusion of fish <7 inches in length in calculation the HUC-level percentile estimates</u>

While the majority of the 3,141 watersheds included in the risk assessment do have multiple measurements, a substantial fraction (41%) have only 1-2 samples, which potentially biases low a 75th percentile estimate for those watersheds. To examine the potential magnitude of low bias in the 75th percentile estimates, we have summarized the distribution of 75th percentile

---

[28] We note that the 75th percentile value is a constructed statistic that reflects the fish tissue samples available at a given watershed. For some watersheds the number of samples is very low, 1 or 2 in some cases (see Table 1-2), and as a result, estimation of the true 75th percentile of fish tissue mercury concentrations in those watersheds is likely to be biased low. This is discussed later in this section. Depending on the degree to which the underlying fish tissue data are representative of actual fish targeted by subsistence fishers active at that waterbody, this statistic may or may not actually represent fish tissue Hg concentrations associated with fish consumption by those fishers.

fish tissue values (as used in the risk assessment) for various sample-size-based strata of the 3,141 watersheds (see Table 1-2). We have also included two plots (as recommended in the SAB peer review) including (a) a histogram comparing the frequency of different sample sizes across the HUCs included in the risk assessment (Figure 1-7) and (b) a LOESS (locally-weighted scatter plot smoothing)-based fitted curve relating the 75th percentile HUC-level values and the frequency of samples sizes by HUC (Figure 1-8).[29] The number of sampling sites also varies across watersheds included in the risk assessment, with some watersheds having up to 30 or more distinct sampling sites.

While supporting the use of the 75th percentile fish tissue Hg value in the risk assessment, the SAB recommended a sensitivity analysis involving use of a median fish tissue Hg value in place of the 75th percentile value. This sensitivity analysis is discussed in section 2.6. However, we have provided fish tissue summary statistics here for median fish tissue statistics to support comparison with the 75th percentile values (Table 1-3).

Table 1-2 Summary of 75th Percentile Hg Concentrations in Fish Tissue Samples by Number of Observations and Number of Sites per HUC

Number of Observations per HUC	Percentiles (of the 75th percentile HUC-level fish tissue Hg concentration - ppm)					Mean	N
	25th	50th	75th	90th	95th		
1 Observation	0.077	0.1564	0.2887	0.493	0.67834	0.226803	838
2 Observations	0.09995	0.1775	0.315	0.5135	0.765	0.256943	458
3-5 Observations	0.12	0.205625	0.339	0.577	0.8	0.283682	468
6-10 Observations	0.185	0.282	0.464	0.743	0.985	0.374177	506
> 10 Observations	0.25	0.37	0.553	0.85	1.083412	0.444645	871
Total	**0.1294**	**0.245**	**0.422684**	**0.673355**	**0.893**	**0.324052**	**3141**
Number of sampling sites per HUC	**25th**	**50th**	**75th**	**90th**	**95th**	**Mean**	**N**
1 Site	0.12	0.233	0.4	0.672	0.87	0.31308	2371
2 Sites	0.160967	0.276706	0.45044	0.71	1.047125	0.360398	553
3-5 Sites	0.179823	0.299482	0.478662	0.603667	0.738476	0.350198	196
6-10 Sites	0.125972	0.224625	0.479822	0.693108	0.8652	0.325642	19
> 10 Sites	0.2698	0.410415	0.494276	0.847259	0.847259	0.419069	8
Total	**0.1294**	**0.245**	**0.422684**	**0.673355**	**0.893**	**0.324052**	**3147**

Based on the *observations*-related data presented in Table 1-2 and Figure 1-8, we acknowledge the potential for low-bias in the 75th percentile values for HUCs with lower sample sizes. Specifically, in Table 1-2, the mean 75th percentile HUC-level value increases across strata as the sample size increases. This suggests that as the number of samples increases, estimates of the 75th percentile will be more likely to approach the true value, and thus are more likely to represent higher end fish tissue levels. Similarly, in Figure 1-8, the smoothed regression line (for the 75th percentile HUC-level trend) has a positive slope, denoting that higher 75th

---

[29] The LOESS procedure involves fitting of a least-squares curve based on application of a smoothing (or bandwidth) parameter. Following the example provided by the SAB, we used a smoothing parameter of 0.2 in our application of the LOESS procedure. The LOESS-based curve presented in Figure 1-8 reflects exclusion of two outliers.

percentile values are associated with higher HUC-level sample sizes.[30] Interestingly, the mean 50th percentile HUC-level statistic across sample size strata (see Table 1-3) also tends to be higher with more samples, but the trend is not as substantial or consistent as for the 75th percentile estimates. This is expected because the median is a central tendency statistic, and therefore any specific sample is likely to be closer to the central tendency than to other percentiles. As a result, the small sample size issue is not anticipated to introduce significant bias in the context of generating median estimates, but it may underestimate higher-end values such as the 75th percentile. Uncertainty related to sample size and potential bias in the 75th percentile HUC-level fish tissue Hg concentrations is also discussed in section 2.7, Table 2-15.

Interestingly, we do not see a consistent trend across the sampling site strata (i.e., the fish tissue Hg statistic does not increase in a consistent manner with increasing number of sites – see Table 1-2). This is not unexpected since the number of sampling sites is not directly correlated with the number of observations at a given watershed (e.g., there are watersheds that have a few sites each with a large number of observations and there are watersheds with a larger number of sites, but each with only 1-2 observations).
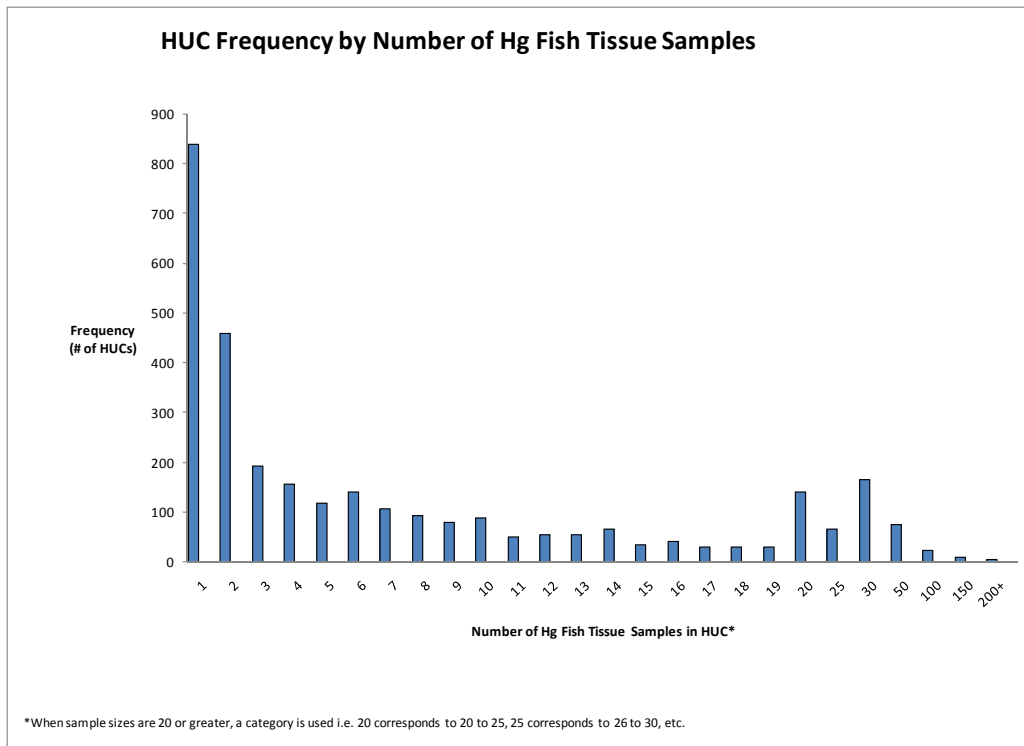
Figure 1-7 Histogram Characterizing Frequency of Sample Sizes Across HUCs Included in the Risk Assessment (illustrates fraction of HUCs with small sample size of 1-2)

---

[30] This trend (higher sampled HUCs having higher 75th percentile fish tissue Hg values) could simply reflect the fact that some states may target sampling at locations believed to have higher mercury impacts based on previous sampling.

28
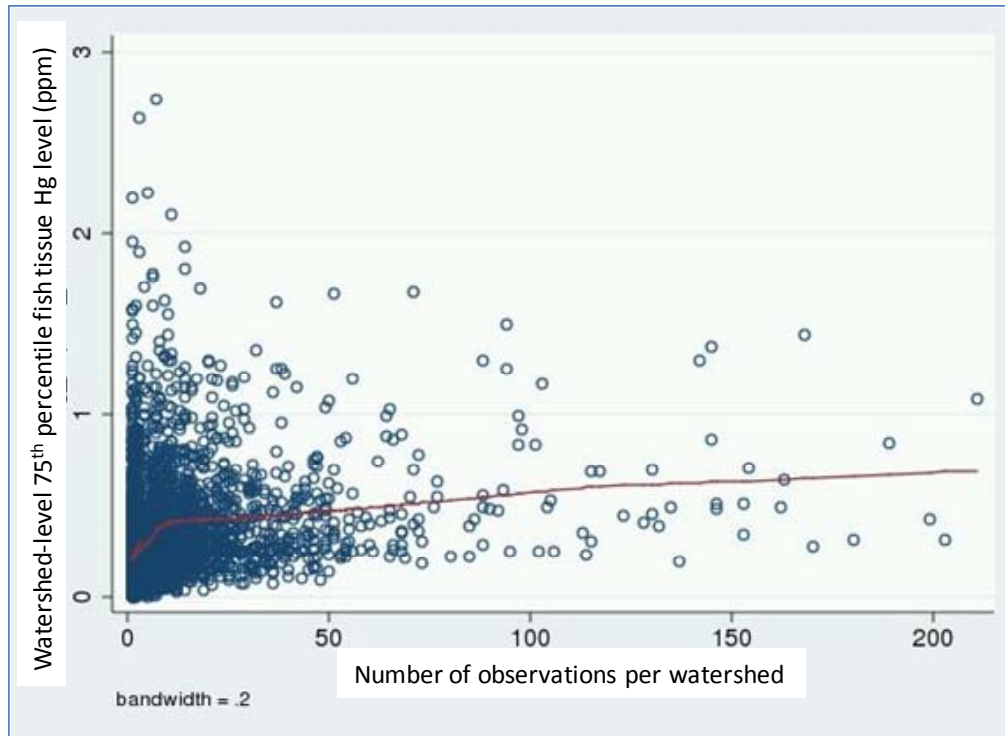
Figure 1-8 LOESS (locally-weighted scatter plot smoothing) Least-Square Regression of 75th Percentile HUC-level Fish tissue Hg Levels Against HUC-level Sample Size

Table 1-3   Summary of 50th Percentile Hg Concentrations in Fish Tissue Samples by Number of Observations and Number of Sites per HUC

Number of Observations per HUC	Percentiles (of the 50th percentile HUC-level fish tissue Hg concentration - ppm)					Mean	N
	25th	50th	75th	90th	95th		
1 Observation	0.077	0.1564	0.2887	0.493	0.67834	0.226803	838
2 Observations	0.08055	0.1362	0.25	0.408	0.5235	0.195935	458
3-5 Observations	0.08403	0.1443	0.235	0.425	0.639	0.205501	468
6-10 Observations	0.1295	0.207913	0.36	0.524	0.72	0.274554	506
> 10 Observations	0.178	0.252444	0.376818	0.59	0.728846	0.310015	877
Total	**0.104**	**0.19**	**0.3203**	**0.5033**	**0.674**	**0.25001**	**3147**
Number of sampling sites per HUC	**25th**	**50th**	**75th**	**90th**	**95th**	**Mean**	**N**
1 Site	0.1	0.1815	0.31	0.505	0.6633	0.245554	2371
2 Sites	0.1165	0.2105	0.347335	0.531	0.7371	0.269281	553
3-5 Sites	0.134761	0.205034	0.32434	0.439024	0.496111	0.247729	196
6-10 Sites	0.100278	0.175125	0.338674	0.491046	0.638407	0.236554	19
> 10 Sites	0.199375	0.314303	0.41785	0.633312	0.633312	0.326412	8
Total	**0.104**	**0.19**	**0.3203**	**0.5033**	**0.674**	**0.25001**	**3147**

There is also uncertainty due to excluding fish samples smaller than 7 inches. Specifically, if subsistence fishers also target fish smaller than 7 inches in (and if those smaller fish have lower MeHg concentrations), then the risk estimates could be high-biased for this

group of subsistence fishers, since we excluded those smaller fish in our modeling. However, it is important to reiterate that this analysis is not intended to generate a representative assessment of the distribution of exposure and risk across the entire set of higher consuming self-caught freshwater fishers. Given the goal of the analysis is to determine whether the potential exists for adverse health impacts linked to self-caught fish consumption, it is reasonable (and indeed a stated element of the scope of the analysis – see section 1.2) to focus on those behaviors by subsistence fishers that would place them at greater exposure and risk. Given this goal, it is reasonable to assume that a subset of subsistence fishers could focus on larger fish (i.e., > 7 inches) as they attempt to supplement their diet. By basing exposure and risk modeling on the 75th percentile fish and excluding fish smaller than 7 inches, we have targeted this subset of subsistence fishers in this risk assessment.

While a reasonable assumption is that a subset of high-consuming subsistence fishers will choose to catch and eat fish larger than 7 inches, reflecting recommendations by SAB, we include a sensitivity analysis to examine the degree to which exclusion of fish smaller than 7 inches affects risk estimates. Table 1-4 compares trends in different HUC-level percentile fish tissue Hg concentrations (and the mean) based on consideration for (a) only fish > 7 inches in length (used in the national-scale mercury risk assessment), and (b) fish of all lengths. Of particular interest are the bolded rows in the table that provide results based on the 75th percentile sample, which is the value used in the risk assessment. The results provided in Table 1-4 suggest that excluding fish smaller 7 inches did not have a large impact on the HUC-level percentiles of fish tissue Hg. This can be seen by comparing the 75th percentile HUC-level estimates using only the fish larger than 7 inches with the 75th percentile estimates based on all fish combined. For example, if we compare the average of the HUC-level 75th percentile values when only fish >7 inches in length were included (0.32 ppm) with the average of the HUC-level 75th percentile values when all lengths are included (0.31 ppm) we see that the two values are similar. This observation holds even if we look at higher end percentiles of the HUC-level 75th percentile values (e.g., the 95th percentile of the HUC-level 75th percentile values are 0.89 ppm and 0.86 ppm, for the >7 inch and all lengths included, datasets, respectively). One likely reason that the exclusion of fish smaller than 7 inches did not have a large impact on the 75th percentile HUC-level estimates is that this subset of smaller fish represents a relatively small fraction of the entire fish tissue sample dataset considered for this analysis (i.e., ~3,400 measurements for <7 inch fish versus 35,600 measurements for > 7inch fish, or roughly ten times more samples for >7 inch fish). Because the impact on fish tissue concentrations is small, we do not carry forward this sensitivity analysis through the calculation of risks, as the impact on risk is also likely to be small.

Table 1-4   Comparison of HUC-Level Fish Tissue Hg Statistics for (a) Fish Tissue Dataset with Fish >7 Inches and (b) Dataset with All fish Lengths Included

HUC-level statistic	N	Fish tissue Hg concentration (ppm) - as relevant						
		Mean	25th %	50th %	75th %	90th %	95th %	99th %
(a) Fish Length > 7 Inches (as used in the national-scale Hg risk assessment)								
mean	3,147	0.27	0.11	0.20	0.35	0.54	0.71	1.09
25th %	3,147	0.19	0.08	0.14	0.25	0.40	0.52	0.89
50th %	3,147	0.25	0.10	0.19	0.32	0.50	0.67	1.06
75th %	**3,147**	**0.32**	**0.13**	**0.25**	**0.42**	**0.67**	**0.89**	**1.36**
90th %	3,147	0.40	0.14	0.29	0.53	0.86	1.13	1.86
Number of samples per HUC	3,147	11.23	1	4	12	28	44	106
Number of species per HUC	3,147	2.69	1	2	4	6	7	10
Number of sample sites per HUC	3,147	1.41	1	1	1	2	3	5
(b) All fish lengths								
mean	3,268	0.25	0.10	0.19	0.33	0.52	0.68	1.05
25th %	3,268	0.18	0.07	0.13	0.24	0.38	0.50	0.85
50th %	3,268	0.24	0.10	0.18	0.30	0.48	0.65	1.02
75th %	**3,268**	**0.31**	**0.12**	**0.23**	**0.40**	**0.65**	**0.86**	**1.34**
90th %	3,268	0.39	0.13	0.27	0.51	0.83	1.10	1.82
Number of samples per HUC	3,268	11.83	1	4	13	29	46	123
Number of species per HUC	3,268	2.85	1	2	4	6	8	11
Number of sampling sites per HUC	3,268	1.41	1	1	1	2	3	5

1.4.2.1   Projecting 75th percentile fish tissue Hg concentrations for the 2016 scenario

Once 75th percentile fish tissue Hg concentrations are generated for each of the watersheds, we then adjust these estimates to represent 75th percentile fish tissue Hg concentrations for the 2016 scenario which are used in modeling risk for this scenario. As mentioned in section 1.4 (Step 4 associated with Figure 1-2), projection of fish tissue Hg concentrations for 2016 is based on application of the proportionality assumption combined with Hg deposition estimates for the 2005 and 2016 scenarios. Specifically, for a given watershed, we multiply the 75th percentile measured fish tissue Hg concentration, by the ratio of total Hg deposition in 2016 to total Hg deposition in 2005. This results in a projection of 2016 fish tissue Hg concentrations. That 2016 projected total fish tissue Hg concentration is then used to model HQ for the 2016 scenario in that watershed. Detail on the proportionality assumption (supported by Mercury Maps simulations and more recent literature) is presented in section 1.4.6.

1.4.3   Defining subsistence fisher scenarios to include in the analysis

As described in section 1.4, defining subsistence fisher scenarios included in this analysis requires three tasks: (a) identify subsistence fisher populations, (b) assess where they might be

active and (c) define fish consumption rates for female subsistence fish consumers associated with those populations. These three tasks are described below.

Identify subsistence fisher populations

As discussed in sections 1.2 and 1.4, this analysis estimates risk for scenarios representing female subsistence fish consumers who have the potential to consume fish caught at inland freshwater locations, since these populations are expected to experience the greatest U.S. EGU-attributable risks. Therefore, in reviewing studies of fishing behavior, we prioritized surveys characterizing fishing activity for populations with these characteristics. In addition, we needed studies that provided statistically rigorous estimates of annual-average daily fish consumption rates. Subsistence levels of fish consumption are likely only experienced by relatively small fractions of the study populations surveyed (e.g., 90th to 99th percentile consumption rates).

Although we reviewed many studies, we ultimately identified three studies that met our criteria for characterizing subsistence-level self-caught freshwater fish consumption. These three studies included several subsistence populations differentiated by ethnicity and SES status including: (a) White and Black populations (including female and low income strata) surveyed in South Carolina (Burger et al., 2002), (b) Hispanic, Vietnamese and Laotian populations surveyed in California (Shilling et al., 2010) and (c) Great Lakes Tribal populations (Chippewa and Ojibwe) active around the Great Lakes (Dellinger et al., 2004).

These studies were used to characterize behavior for female subsistence fish consumers assumed to be associated with the subsistence fisher populations described in the studies.[31] Specifically, based on these studies, we characterized behavior for seven distinct female subsistence fish consumer scenarios which were included in the risk assessment. These scenarios are listed (and briefly described) in Table 1-5.

Table 1-5 Spatial Extent and Number of HUCs Reflected in Risk Modeling for the Female subsistence fish consumer Scenarios Included in the Risk Assessment

Female subsistence fish consumer Scenario	Description	Spatial Extent	Number of watersheds (HUC12s) Assessed For Risk
Typical (non-SES differentiated)	Generalized (non-SES differentiated) female subsistence fish consumers associated with fishing activity that could occur at any of the watersheds across the nation with fish tissue Hg data (i.e., source population construct is not applied here).	National (all HUCs with fish tissue Hg measurements)	3,141
Low income	Low income White female subsistence fish consumers	Southeast	860

---

[31] With the exception of the Burger et al., 2002 study, which did provide consumption rates for females, we assumed that rates provided for specific SES-differentiated populations could apply to pregnant females associated with those fishers (e.g., either as family members or acquaintances). We recognize that there is uncertainty associated with this assumption, but would also point out that the female consumption rates provided in Burger et al., 2002 (particularly for the higher percentiles) are in the range of values seen for males and for non-sex differentiated survey groups both in that study and in the other two studies of high-end self-caught fish consumption cited here.

Female subsistence fish consumer Scenario	Description	Spatial Extent	Number of watersheds (HUC12s) Assessed For Risk
White (southeast)	active in the Southeast; in watersheds with fish tissue Hg data where there are at least 25 White individuals below the poverty line.		
Low income Black (southeast)	Low income Black female subsistence fish consumers active in the Southeast, in watersheds with fish tissue Hg data where there are at least 25 Black individuals below the poverty line.	Southeast	756
Low income Hispanic	Low income Hispanic female subsistence fish consumers active at any watersheds in the continental U.S. with fish tissue Hg data where there are at least 25 Hispanic individuals below the poverty line.	National (where there are low income Hispanics)	1,325
Great Lakes Tribal	Tribal (Chippewa/Ojibwe) female subsistence fish consumers active at any watersheds with fish tissue Hg data within the ceded territories in the vicinity of the Great Lakes (i.e., source population construct not applied here).	Vicinity of Great Lakes (ceded territories)	361
Laotian	Laotian female subsistence fish consumers active at any watersheds in the continental U.S. with fish tissue Hg data where there are at least 25 Laotians.	National (where there are Laotians)	131
Vietnamese	Vietnamese female subsistence fish consumers active at any watersheds in the continental U.S. with fish tissue Hg data where there are at least 25 Vietnamese.	National (where there are Vietnamese)	336

Assess where the subsistence fisher populations might be active

The following approach was used to identify the spatial extent of activity for each of the seven female subsistence fish consumer scenarios. For all of the scenarios (with the exception of the Tribal scenario associated with Great Lakes), we assumed that high-end fishing behavior could be generalized beyond the specific geographic areas covered in a particular study. This type of generalization was necessary to provide sufficient coverage for the continental U.S. and in particular, the eastern part of the U.S. where U.S. EGU-attributable deposition is higher and where we have more measured Hg fish tissue data. In deciding how to extend coverage for each fisher population, we considered several factors including (a) the potential for high-end fishing activity to be culturally-related and therefore more likely to be followed by populations of a given ethnicity living across the U.S. and (b) the potential that subsistence fishing activity (and related consumption) might be driven by economic need. Because we assumed that fishing activity is culturally related, we generalized fishing activity by Hispanics, Laotians and Vietnamese beyond California, where the study providing consumption rates was conducted, to the nation. Similarly, we assumed that surveys of high-end fish consumption by low income Whites and low income Blacks in South Carolina might be generalized to the southeast, reflecting similar cultural practices by these groups within that region.

Once we identified the regions of the country where a particular subsistence fisher population might be active, we then identified the specific subset of watersheds within that region where the high-end fishing activity (and consumption by female subsistence fish consumers associated with those subsistence fishers) might occur. This task was challenging

33

because we do not have data characterizing the number and location of subsistence fishers for any of the fishing populations under consideration. Therefore, we developed and applied a *source population* construct to guide identification of areas (watersheds) where a given subsistence scenario might be active.[32] This approach requires that in order for a subsistence fisher scenario to have the potential to be active at a given watershed, that watershed must be in close proximity to a source population matching the ethnic/SES composition of the subsistence fishers being represented. In this case, we specified that a watershed would have to intersect a U.S. Census tract with at least 25 individuals with the same ethnic/SES composition as the subsistence fisher scenario (i.e., a *source population*) for there to be the potential for that type of fishing at that watershed. So, for example the Laotian subsistence fisher scenario was only assessed at those watersheds intersecting U.S. Census tracts with at least 25 Laotians. In the case of low income White and Black subsistence fishers in the southeast as well as Hispanics assessed nationally, we required that the watershed intersect U.S. Census tracts with at least 25 individuals from that ethnic group who fall below the poverty line. The SAB peer review panel agreed that the criterion of using 25 persons within a source population to identify watersheds with the potential for subsistence fishing activity is a reasonable approach. The specific approach used to extrapolate coverage regionally and then determine the subset of watersheds to include in risk modeling for each subsistence fisher scenario is described both in Table 1-5 and in greater detail in Table 1-6 (in Table 1-6 see columns "C" and "E"). As described in Table 1-6 (columns "C" and "E"), the approach used for the Tribal population and typical female subsistence fish consumer scenarios differs from those used for the other scenarios. In the case of the Tribal subsistence fisher scenario, given the potential for fishing activity to be closely associated with heritage cultural practices, we have not extrapolated fishing activity for the Chippewa/Ojibwe outside of their ceded territories, although we do assume that all watersheds within those territories with fish tissue Hg data have the potential for fishing activity by this group.

The *typical female subsistence fish consumer* scenario differs from the other scenarios in that it is not SES-differentiated and is assessed nationally at all watersheds with fish tissue Hg data (i.e., the source population construct is not used to constrain its spatial extent). The generalized nature of this scenario means that it provides broader coverage for subsistence fisher activity and for this reason we assume that this scenario (together with female subsistence consumption associated with this fishing activity) could potentially occur at any watershed with fish tissue Hg data.[33] As noted in section 1.2 and 1.4, because the typical female subsistence fish consumer is the most generalized of the scenarios and has the greatest spatial extent, we place greater emphasis on this scenario in presenting risk estimates.[34] Application of the typical female

---

[32] As described in section 1.4, a *source population* is a group of individuals with demographic attributes matching those of the subsistence fisher group being evaluated. While we cannot enumerate the subsistence fishers directly, we can use demographic data to determine if the underlying source population is present in the vicinity of a watershed with fish tissue Hg data.

[33] We are not suggesting that we expect the scenario to occur as a certainty at all watersheds, or that we can predict the level of activity (number of fishers or consumers) at each watershed. Rather, for this analysis, we are stating that we believe it reasonable to assume that the typical female subsistence fish consumer scenario (and associated fishing activity) could potentially occur at some subset of the watersheds with fish tissue Hg data.

[34] The typical female subsistence scenario included in this revised risk assessment is similar to the high consuming female scenario included in the March version of the Mercury Risk TSD. However, the typical female subsistence scenario is applied without consideration for a source population, while in the case of the high-consuming female angler; we did consider a source population based on poverty. Because the typical female subsistence scenario does not use a source population (and is applied uniformly to all watersheds with fish tissue Hg data except for those

subsistence fish consumers scenario to all watersheds with fish tissue Hg data also helps to address the concern raised by the SAB that the March version of the national-scale Hg risk assessment may have excluded more remote water bodies that could be fished by subsistence anglers (leading to an underestimation of the percent of watersheds where Hg exposure from U.S. EGU sources is a risk). As mentioned earlier, Table 1-5 lists the seven female subsistence fish consumer scenarios and presents the number of watersheds assessed for risk for each scenario.

<u>Define self-caught fish consumption rates for the subsistence scenarios</u>

After identifying the set of female subsistence fish consumer scenarios to include in the analysis and determining where those scenarios should be applied, next we defined high-end (subsistence) self caught fish consumption rates for those scenarios. The three studies referenced in Table 1-6 provided either subsistence-level consumption rates at the 90th to 99th percentile, or the statistical parameters necessary to calculate those percentiles (e.g., median and standard deviations).[35] In establishing consumption rates for the seven female subsistence fish consumer scenarios, we either used rates from the studies directly (as in the case of the typical female subsistence fish consumer which uses female consumptions rates from the Burger et al., 2002 study) or we used non-sex differentiated consumption rates, assuming that they applied to women of childbearing age. As noted earlier in this section, there is uncertainty associated with the assumption that non-sex differentiated consumption rates could apply to women of child-bearing age (see footnote 33).

The subsistence-level consumption rates used in modeling risk for these scenarios are presented in Table 1-6, along with notes relevant to their interpretation in the context of this risk assessment. Of particular interest was whether the consumption rates represented annual-averages of daily "as consumed/prepared" values. As noted earlier in 1.4, this issue is critical to ensuring the appropriate technical linkages between the types of fish tissue Hg measurements made, application of preparation adjustment factors and ultimately, calculation of exposure and risk. This issue is discussed in greater length in the next section.

The fish consumption rates presented in Table 1-6 clearly represent subsistence behavior since they reflect an individual making significant contributions to their diet through self-caught fish consumption. In most instances, the rates are equivalent to between one 8oz fish meal every few days to a larger fish meal (12oz or more) every day. While these consumption scenarios are identified as "subsistence" due to their magnitude, they could be experienced by individuals purposefully supplementing their diet due to economic need or due to high levels of recreational fishing activity. While these scenarios could represent either subsistence or high-end recreational fishing, the resulting exposure and risk characterization would be similar. For typical female subsistence fish consumers, as mentioned earlier, this scenario is applied uniformly across all watersheds with fish tissue Hg data, and whether we define consumption as subsistence or high-end recreational fishing does not affect the exposure and risk characterization. In either case, the

excluded due to potential impacts from non-air deposition), this scenario does provide greater coverage geographically than did the high-consuming female which was only applied to watersheds with fish tissue Hg data and at least 25 members of the source population (individuals living below the poverty line).

[35] In those instances where a specific percentile was not provided, we estimated that value using the statistical parameters provided together with the assumption that the underlying fish consumption distribution was lognormal.

risk estimates represent risk experienced by an individual residing in the vicinity of a particular watershed who consumes self-caught fish at a subsistence level.[36] In addition to the three studies we used to define subsistence fish consumption rates, we also reviewed additional studies characterizing higher-levels of self-caught fish consumption in the U.S. While these additional studies had limitations that prevented their use in this risk assessment, they generally support the rates of self-caught fish consumption modeled in the analysis.

A subset of these "supporting" studies is briefly described below:

- A study by Burger et al., 1999, examined recreational and subsistence fishing activity along the Savannah river in Georgia to determine the role played by socio-economic status (SES) factors (including race, education and income) and determined levels of self-caught fish consumption in this study area. The study suggested that all three factors are associated with levels of fishing activity. Specifically, in the case of race, the study showed that Blacks tend to have much higher rates of fish consumption than Whites. For both groups, the study suggested that upper-end percentile consumption rates could be high enough to approach subsistence levels. For example, a ~200 g/day fish consumption rate represented the 98th percentile for Whites, but only the 92nd percentile for Blacks. This study supports the presence of high-end consumption rates for both Blacks and Whites that approach or meet subsistence levels in this area of the country. While this study did provide support for high self-caught fish consumption rates and SES differentiation of fish consuming populations, because of its geographically limited scope (i.e., focus on Savannah River), we elected not to use it directly in defining consumption rates for the risk assessment.

- A study by Moya et al., 2008 examined factors associated with regional differences in patterns of fish consumption, including age, ethnicity (including Tribal affiliation), socioeconomic status (e.g., income, education), and type/source of fish consumed (freshwater, marine, and estuarine obtained from commercial sources versus self-caught). The study examined fishing activity in four states (i.e., CT, FL, MN, and ND) and provided estimates of high-end self-caught fish consumption for populations. Higher, subsistence-level consumption rates were identified for fishing populations in FL, MN (specifically for Tribes) and CT (for Asian populations, although it is not clear whether the rates for Asians are for self-caught fish consumption). Higher-end rates reported for ND and for general fishers in CT and MN did not approach the range of subsistence levels of consumption. However, the study designs used in these surveys may not effectively capture the small fraction of the overall population likely engaging in high-end subsistence levels of self-caught fishing behavior. This study supports the existence of subsistence fishing populations in FL and for Tribes in MN. However, failure to capture similar behavior in ND and CT does not necessarily suggest that this type of

---

[36] To generate population-weighted risk estimates, it is important to differentiate between high-end recreational and necessity-based subsistence activity, since the associated populations at any given watershed could differ, leading to different population-weighted risk distributions. However, because we lacked other data needed to calculate population-weighted risks, this differentiation is not relevant.

behavior is non-existent, but it may suggest that subsistence behavior is less prevalent than in FL. We did not elect to use the data for FL to define consumption rates since we already had the Burger et al., 2002 study which covers a region of the Southeast (South Carolina) where we have a larger number of watersheds being modeled for risk.

Table 1-6 Fish consumption rates and additional behavior-related information for subsistence populations included in the analysis

(A) Fish consuming populations covered by study (and reference information)	(B) Overview of study	(C) Assumption regarding where the subsistence fisher scenario (and associated female subsistence fish consumers) might be active	(D) Self-caught fish consumption rates (mean, 90th, 95th, 99th) g/day and notes on type of consumption rate (e.g., temporal averaging period and as purchased versus as consumed)	(E) Notes on consumption rate information relevant to the risk assessment
Higher self-caught fish consuming populations (White, Black and female) surveyed in South Carolina Citation: Daily consumption of wild fish and game: Exposures of high end recreationalists, Burger et al., International Journal of Environmental Health Research, 12:4, p. 343-354, July, 2002	Random survey of participants in the Palmetto Sportsmen's Classic in Columbia SC (1998). Population interested in fishing/hunting (not general population – represents outdoor enthusiasts in SC)	- the Black and White fisher populations were extrapolated to cover watersheds modeled for risk in the Southeastern states. The rationale for this was that fishing activity by these two groups could be generalized in this region of the country. These scenarios were only assessed for watersheds in the Southeast located within U.S. Census tracts with at least 25 individuals from that ethnic group below the poverty line. - given the focus of the risk assessment on consumption by women (in considering exposures for pregnant women in particular), we extrapolated the typical female consumer scenario to all watersheds in the continental U.S. and, given the more generalized nature of this scenario (no ethnic or SES differentiation), we assessed the scenario for all watersheds included in the risk assessment (i.e., we did not apply the source population criterion used for the other scenarios).	- Black: 171, 446, 557, NC * - White: 38.8, 93, 129, 286 - female: 39.1, 123, 173, 373 * the sample size for this population is only 39, reducing overall confidence in a 99th consumption rate (therefore, this high-end percentile was not included in the risk assessment) Consumption rates are annual-average values expressed as meals or portions (as prepared) (median, 75th, 90th, 95th, and 99th). Survey asked respondents by month for number of meals of different type of fish and serving size (here models were used to demonstrate different meal or serving sizes). Authors could then use this to estimate monthly consumption rates and convert these into an annual average.	Sample size is variable – out of 458 respondents, 39 are Blacks, 149 are female and 98 are low income – Black n is relatively smaller than the other groups, which increases uncertainty in higher percentile values provided for this group. The authors point out that these results highlight the considerable spread between high-end consumers and more typical behavior (95th percentile is more than 10X greater than the mean or median intake rate for wild-caught fish). Results are also provided for low income (0-20K$ annual income). These consumption rates are relatively high particularly for the higher percentiles (90th, 95th and 99th rates are: 285, 429 and 590 g/day). This observation forms the basis for our decision to assess a number of the subsistence populations only for watersheds located in U.S. Census tracts containing members of source populations below the poverty line for the White and Black populations.

(A) Fish consuming populations covered by study (and reference information)	(B) Overview of study	(C) Assumption regarding where the subsistence fisher scenario (and associated female subsistence fish consumers) might be active	(D) Self-caught fish consumption rates (mean, 90th, 95th, 99th) g/day and notes on type of consumption rate (e.g., temporal averaging period and as purchased versus as consumed)	(E) Notes on consumption rate information relevant to the risk assessment
Higher self-caught fish consuming ethnic populations including Hispanics, Laotians and Vietnamese surveyed in California Citation: Contaminated fish consumption in California's Central Valley Delta (Shilling et al., Environmental Research 110, p. 334-344 (2010)	Study looks at subsistence fishing activity among ethnic groups associated with more urbanized areas near the Sacramento and San Joaquin rivers in the Central Valley in CA.	- the Hispanic fishing scenario was extrapolated to cover watersheds located in U.S. Census tracts with at least 25 low income members of the ethnic populations (e.g., the Hispanic consumption rates would be applied to the subset of the 3,141 watersheds located in U.S. Census tracts with at least 25 low income Hispanic individuals). - the Laotian and Vietnamese fishing scenarios were extrapolated to cover watersheds located in U.S. Census tracts with at least 25 members of the underlying ethnic group.	- Hispanic: 25.8, 98, 155.9, NC* - Lao: 47.2, 144.8, 265.8, NC* - Vietnamese: 27.1, 99.1, 152.4, NC* * 95th percentile values were provided in the study. 90th percentile values were calculated using Crystal Ball (based on the median and standard deviations provided) assuming a log-normality of the consumption rate distributions. 99th percentile consumption rates were not provided (or derived) for any of these populations due to small sample sizes of the study populations. Consumption rates are annual-average estimates (cooked weight - as filets) based on the fact that they compare their average values against other rates from the literature including rates used by the EPA in the regulatory context - all of which are annual averages. Survey used different sized models of cooked fish filets.	The authors note that many of these ethnic groups relied on fishing in origin countries and bring that practice here (e.g., Cambodian, Vietnamese and Mexican). The authors also note that fish consumption rates reported here for specific ethnic groups (specifically Southeast Asian) are generally in-line with rates seen in WA and OR studies.

(A) Fish consuming populations covered by study (and reference information)	(B) Overview of study	(C) Assumption regarding where the subsistence fisher scenario (and associated female subsistence fish consumers) might be active	(D) Self-caught fish consumption rates (mean, 90th, 95th, 99th) g/day and notes on type of consumption rate (e.g., temporal averaging period and as purchased versus as consumed)	(E) Notes on consumption rate information relevant to the risk assessment
High-end self-caught fish consuming Chippewa and Ojibwa Tribal populations active in the vicinity of the Great Lakes. Citation: Exposure assessment and initial intervention regarding fish consumption of tribal members in the Upper Great Lakes Region in the United States. Dellinger , Environmental Research 95 (2004) p. 325-340	This study contrasted self-reported fish consumption rates by Tribes in the Great Lakes area with "actual" fish consumption rates collected for a subset of the original study population (147 of 822 from 4 Tribal population/location combinations). The study found that actual fish consumption rates were lower than reported values.	Activity only assumed to occur in areas ceded to the Tribes covered in the study (regions in the vicinity of the Great Lakes). Because fishing activity is highly variable across Tribes (and closely associated with heritage cultural practices) we have not extrapolated fishing behavior for these Tribes outside of the specific populations and regions covered.	- reported value for all Tribal areas (in the study) combined: 62, 136.2, 213.1, 492.8 All higher percentiles (90th – 99th) were derived using Crystal Ball (based on median and standard deviations and an assumption of log-normally distributed variability in consumption rates) Consumption rates appear to be annual average values. Study includes reference to querying for the number of fish meals in a year, which suggests that estimates are annual-averages (but it is not explicitly stated). Ingestion rates are in-line with other high-end consumer rates cited in this table. It also appears that estimates are for amount consumed (i.e., meal weight), based on the fact that the "actual" estimates provided in the article focused on this type of consumption rate.	While the "actual" consumption rates collected for a subset of the families were far lower than the reported values (often an order of magnitude smaller), a number of factors resulted in a decision to use the reported values rather than the actual values in the risk assessment. First, and most importantly, the sample size is very small for the "actual" analysis with n's ranging from 12 to 54 individuals (representing a smaller number of associated families) for the different survey groups. These small sampling rates reduce the probability of capturing individuals with higher consumption rates in the broader population. It also appears that the actual values may cover walleye specifically and not include all fish, which could bias these values downward. There is concern that, even if consumption rates have decreased, actual heritage cultural practices could still exist (or there could be a desire to return to those rates), in which case, risks levels associated with those higher historical consumption rates could be important to assess. And finally, the high-end percentile consumption rates derived based on reported mean consumption rates (and standard deviations) are in-line with subsistence consumption rates seen for other populations in the U.S. Therefore, these Tribal high-end fish consumption rates would general comport with subsistence fish consumption activity and therefore are considered reasonable to include in the risk assessment.

1.4.4   Estimating total fish consumption-related MeHg exposure (2016 scenario)

Next, we estimated total exposure to Hg for the set of seven female subsistence fish consumer scenarios being evaluated (for the 2016 scenario). To generate estimates of total Hg exposure, we combined the 75th percentile fish tissue Hg values (projected for 2016) with three exposure-related factors including: (a) the consumption rates for a subsistence fisher scenarios in grams of fish per day, (b) a conversion factor between total Hg measurements in fish and MeHg levels in fish and (c) a food preparation adjustment (cooking fish can increase MeHg concentrations).

In modeling MeHg exposure through fish consumption, it is important to verify that the linkages between factors used in modeling exposure (i.e., the variables in the exposure equation listed above) are conceptually correct. For example, we need to convert the total Hg levels in fish to MeHg levels in order to calculate the HQ, which is based on MeHg intake. Similarly, if fish ingestion rates are specified in terms of "as cooked" or "as consumed" rather than "as purchased" or "as market basket," then we must factor in cooking/preparation in specifying the fish tissue Hg concentrations. Failure to correctly link these exposure factors can result in biased estimates of MeHg intake and consequently biased risk estimates. Below we discuss each exposure factor, including the data used to specify that factor and the linkage of that factor to others in the exposure equation.   Then, we present the exposure equation.

Mercury fish tissue concentration (FTC): We calculated the 75th percentile fish tissue Hg estimate for each watershed and then project those to represent the 2016 scenario (as described in section 1.4.2.1). Generally, measured fish tissue Hg is in terms of total Hg. Because we need MeHg levels in fish, we convert total Hg levels measured in fish into MeHg levels using the mercury conversion factor (MCF).

Mercury conversion factor (MCF): We applied a factor of 0.95 (unitless) to convert total Hg levels in fish tissue samples into MeHg levels. This conversion factor is based on conclusions reached in the Mercury Study Report to Congress (MRTC) (U.S. EPA, 1997) that more than 90% of Hg in fish is MeHg. The conclusion presented in the MRTC is in turn based on two studies (Bloom, 1992 and Morgan et al., 1994). The 0.95 factor represents a median value between an assumption that all of the Hg in fish is MeHg and the lower bound value of 0.90 cited in the MRTC document.

Food preparation/cooking adjustment factor (FPCAF): Because we use fish consumption rates for "as consumed" portions, we need to adjust the fish tissue Hg concentrations to reflect concentrations after food preparation. Cooking fish typically increases MeHg levels per unit fish because Hg concentrates in the muscle while preparation involves removal primarily of non-muscle elements of the fish (e.g., water, fat etc) (Morgan et al., 1997). The FPCAF factor used in the analysis is 1.5 (i.e., an increase of 50% in the MeHg concentration per unit fish due to preparation/cooking). This factor is based on the Morgan et al., 1997 study, which estimated factors of between 1.1 and 1.5 for walleye and 1.5 and 2.0 for lake trout.

The SAB recommended that additional studies be acknowledged as providing information regarding the FPCAF, citing two alternative studies (Farias et al., 2010 and Musaiger et al. 2008). After assessing these studies, we conclude that they do not support a lower

preparation/cooking loss adjustment factor. The Farias study appears to suggest that preparing fish by Manaus residents in the Amazon could decrease Hg concentrations. Although this initially suggests that the 1.5 factor used in the risk assessment is biased high, a closer read of the Farias study suggests that the authors may have measured non-fish components added to dishes (e.g., onions, heavy breading etc) in post-cooking measurements, which could provide the appearance of a cooking loss in Hg while actual fish tissue Hg concentrations could have increased. The Musaiger et al. 2008 study compared Hg levels across different types of fish meals after preparation rather than pre and post-cooking Hg levels. In fact, the authors state that cooking is not a means of reducing Hg because it typically removes fat and water, while Hg is located in the meat and therefore will not be reduced.[37]

Fish consumption rate (FCR): We reviewed the three studies providing fish consumption rates used in the risk assessment to verify that consumption rates (a) are annual-averages and not rates reflecting elevated consumption during shorter seasonal periods and (b) represent "as consumed" rather than "as purchased" values. Based on the review of the three studies we concluded that the consumption rates are annual average daily consumption rates for "as cooked/as prepared." In some instances, a study did not explicitly state whether rates were annual-averages and/or "as cooked", and we had to infer this from other information provided in the article. Details regarding our assessment of each of the three studies is provided in Table 1-6 (column D).

The following equation shows how these factors were combined to generate estimates of annual-average daily MeHg exposure per kg body weight:

$$IR = \frac{FTC(2016) * MCF * FPCAF * FCR}{BW}$$

where,

IR = daily MeHg intake rate (ug/kg-day)

FTC(2016) = Hg fish tissue concentration (ug/g or ppm of total Hg) projected for the 2016 scenario

MCF = Hg conversion factor (unitless)

FPCAF = food preparation/cooking adjustment factor (unitless)

FCR = fish consumption rate (g/day)

BW = body weight (kg)

---

[37] As part of revising the national-scale Hg risk assessment, we completed a literature review focusing on the issue of cooking/preparation of fish and the effect on Hg concentration. This literature review included the two studies recommended by the SAB. That review did not identify any studies that argued against use of an adjustment factor of 1.5 and consequently, we continue to use that factor in modeling exposure and risk.

1.4.5   Estimating risk (RfD-based hazard quotient) (2016 scenario)

Once we estimate the MeHg intake rate for each female subsistence fish consumer scenarios at each watershed (for 2016), we compare these exposure estimates to the MeHg RfD to generate HQ estimates. The MeHg RfD is 0.0001 mg/kg-day (equivalent to 0.1 µg/kg-day) and was published by the EPA in the Integrated Risk Information System in 2001 (US EPA, 2001a) (http://www.epa.gov/iris/subst/0073.htm).

Reflecting precedent in interpreting HQ estimates, we consider exposures above the RfD (i.e., an HQ above one) to represent a potential public health hazard. Consequently, watersheds with female subsistence fish consumer exposures above the RfD are considered to have the potential for consumers of self-caught fish from that watershed to experience a public health hazard due to MeHg exposure. An important factor to consider in interpreting HQ's is the precision underlying these risk estimates. While precision associated with the exposure estimates may be higher, the RfD is only reported to one significant digit, which limits the precision in the HQ estimate. Consequently, we interpret an HQ of 1.5 or greater as representing an exposure that exceeds the RfD (since this value will round to two expressed as a whole number). Conversely, exposures of 1.49999 or less are considered not to exceed the RfD given this rounding convention for HQs.[38]

In response to SAB recommendations that the IQ loss endpoint may not fully capture the range of neurodevelopmental effects associated with Hg exposure, we have deemphasized this category of risk metrics and moved the IQ discussion to the appendices. We discuss the approach used to generate IQ loss estimates including the concentration-response function in Appendix A and we provide the results and approach used to interpret the public health significant of IQ loss in Appendix B.

1.4.6   Estimation of U.S. EGU-attributable risk (2016 scenario)

Next, we estimated the fraction of total risk (HQ) that is attributable to U.S. EGUs (for the 2016 scenario). The estimate of U.S. EGU-attributable risk is at the core of the 2-stage risk characterization framework (see Section 1.3). We estimate the U.S. EGU-attributable fraction of total risk using the linear proportionality assumption linking changes in Hg deposition over watersheds with changes in fish tissue Hg concentrations.[39] As noted in section 1.4.2.1, this proportionality assumption states that, under steady state conditions, a change in Hg deposition over a given watershed will result in a proportional change in fish tissue Hg concentrations and associated exposure and risk. The proportionality assumption is supported by the Mercury Maps

---

[38] Note, that for the U.S. EGU incremental contribution analysis, an HQ of less than 1.5 does not necessarily indicate there is no public health hazard related to U.S. EGU emissions. Rather, it suggests that, for those specific watersheds, we need to also consider whether total risk (i.e., the HQ reflecting total MeHg exposure) exceeds 1.5 and therefore represents a potential public health hazard. If that is the case, then we would consider the degree to which U.S. EGUs contribute to that total exposure because incremental exposures above the RfD increase the risk.

[39] Because risk at the HUC-level is linearly related to the fish tissue Hg concentration (see sections 1.4.4 and 1.4.5), this proportionality assumption can be used to relate changes in mercury deposition to change in total risk at that HUC.

analysis (U.S. EPA, 2001b). In addition, a number of additional studies (some of which were highlighted by the SAB in their review of the March TSD) support a proportional relationship between changes in Hg deposition and changes in fish tissue Hg concentrations, although with important caveats related to the temporal response of aquatic systems to changes in Hg deposition/loading. There are also important criteria that must be met for the proportionality assumption to hold, which we discuss below.

We used the CMAQ air quality model to estimate Hg deposition over U.S. watersheds for all sources of mercury, including U.S. and non-U.S. sources. We also modeled Hg deposition directly from U.S. EGUs by zeroing out Hg emissions from U.S. EGU sources and subtracting the results from total Hg deposition to isolate the U.S. EGU contribution. The CMAQ modeling for this analysis is described in section 1.4.6.3.

Hg deposition estimates at each watershed coupled with the proportionality assumption described above can be used to estimate U.S. EGU-attributable HQ risk for the 2016 scenario. To estimate the U.S. EGU-attributable fraction of that 2016 HQ estimate, we use the ratio of U.S. EGU Hg deposition versus the estimate for total Hg deposition (both for the 2016 scenario in that watershed). The step-wise procedure for completing the estimate of U.S. EGU-attributable risk for 2016 is outlined below:

> **Generate U.S. EGU-attributable HQ risk estimates for the 2016 scenario:** We use the 2016 total and U.S. EGU-attributable CMAQ deposition results with the estimated total HQ risk estimates for 2016 to estimate the U.S. EGU-attributable HQ risk at each watershed as follows:

$$\text{2016 EGU-HQ} = \text{2016 THQ} * (\text{2016 EGU Hg dep} / \text{2016 total Hg dep})$$

Where:

2016 EGU-HQ: U.S. EGU-attributable HQ risk for the 2016 scenario

2016 THQ: HUC-level total HQ risk for 2016 generated as described in section 1.4.5.

2016 EGU Hg dep: 2016 CMAQ-based projections of <u>U.S. EGU-related Hg </u>deposition over a given watershed)

2016 total EGU dep: 2016 CMAQ-based projections of <u>total Hg </u>deposition over a given watershed

1.4.6.1 Mercury Maps analysis

Results of the EPA's Office of Water's Mercury Maps analysis support the proportionality assumption (U.S. EPA, 2001b). The Mercury Maps analysis used a simplified form of the IEM-2M model applied in EPA's Mercury Study Report to Congress (U.S. EPA, 1997). By simplifying the assumptions inherent in the freshwater ecosystem models that were described in the Report to Congress, the Mercury Maps model showed that these models converge at a steady-state solution for MeHg concentrations in fish that are proportional to changes in Hg inputs from atmospheric deposition. This steady-state solution only applies in situations where air deposition is the only significant source of Hg to a water body, and the physical, chemical, and biological characteristics of the ecosystem remain constant over time.

Consequently, the proportionality assumption used to estimate the U.S. EGU-attributable fraction of risk would ideally only be applied to watersheds where these criteria have been met. Application of the proportionality assumption in situations where these criteria have not been met introduces uncertainty in the apportionment of total risk. EPA recognizes that concentrations of MeHg in fish across all ecosystems may not reach steady state and that ecosystem conditions affecting Hg dynamics are unlikely to remain constant over time. EPA further recognizes that many waterbodies, particularly in areas of historic gold and Hg mining in western states, contain significant non-air sources of Hg.[40]   Finally, EPA recognizes that Mercury Maps does not estimate the time lag between a reduction in Hg deposition and a reduction in the MeHg concentrations in fish.

In their peer review, the SAB noted that there are other modeling tools available to link deposition to fish tissue concentrations, but did not consider them to be superior for this analysis, nor did they recommend their use. The SAB specifically noted that the Regional Mercury Cycling Model (R-MCM) could also be used for a national assessment, but they also noted that the R-MCM is more data intensive and the results produced by the two model approaches should be equivalent. We did not have all of the data inputs (water chemistry, methylation potential, etc) that would be required to run the R-MCM, and given the SAB recommendation, and their comment that "it is unlikely that substantial additional insight would be gained with the alternative model framework," we elected to use the proportionality assumption as supported by the Mercury Maps modeling and the peer-reviewed literature. Because the Mercury Maps approach only applies in those watersheds where aerial deposition is the dominant source of Hg loading, we excluded watersheds with substantial non-U.S. EGU anthropogenic emissions of Hg from the risk assessment as described in section 1.4.2.

There are a number of limitations and uncertainties associated with the application of the Mercury Maps approach in the context of this risk assessment. These limitations are discussed here and addressed in Table 2-15. Applying Mercury Maps to apportion fish tissue Hg concentrations and consequently exposure and risk between U.S. EGUs and all other sources of Hg at the watershed-level assumes that the relationship between fish tissue levels and Hg deposition has remained fairly consistent such that near steady-state conditions have been reached. However, in reality, patterns of Hg deposition for the period during which the fish tissue samples were collected (2000 to 2009) have not remained constant. In addition, those fish tissue concentrations may actually reflect patterns of Hg deposition from earlier time periods (e.g., the 1990s) when Hg emissions from U.S. sources were experiencing substantial decreases.[41] In

---

[40] As described below, we have excluded those watersheds containing gold mines or with other non-EGU related anthropogenic Hg emissions exceeding specified thresholds.

[41] The discussion of model uncertainty provided in the Technical Support Document describing the Mercury Maps analysis (MMAPs TSD, U.S. EPA, 2001) also addresses the fact that proportionality between a decrease in Hg deposition and changes in fish tissue Hg concentrations may not be fully realized for some time due to the lagging effect of Hg that has built up in sediment. Simulations discussed in the MMAPs TSD suggested that substantial concentrations of Hg could build up in sediment with these loadings effectively buffering the impact of reductions in Hg. This issue does not invalidate the application of the proportionality assumption in the national-scale mercury risk assessment, but it does suggest that the full effect of predicted reductions in risk may not be seen in the near-term (this issue is discussed further in the context of other studies in section 1.4.6.2). The discussion of model uncertainty in the MMAPs TSD also addresses potential non-linearity in methylation. Citing the DiPasquale, et al, 2000 study, the MMAPs TSD notes the potential for reduced methylation at very high Hg loadings typically experienced in mining areas. However, since our analysis excluded areas likely impacted by active gold mines, we believe that this issue has been largely ameliorated in the context of the national-scale mercury risk assessment.

addition, other factors that can affect rates of Hg methylation (e.g., sulfur deposition to waterbodies, pH of the waterbodies) also have not remained constant over the past 1-2 decades for most watersheds. The fact that many of these factors related to methylation in fish have not remained constant introduces uncertainty into the application of the Mercury Maps based proportionality assumption. However, we believe that the Mercury Maps approach for apportioning fish tissue Hg concentrations is still appropriate to use, particularly if we are not attempting to characterize the temporal response and instead, can assume that sufficient time has passed for near steady state conditions to be reached (see section 1.4.6.2 below). Furthermore, while we have excluded watersheds with substantial non-air loading of Hg from industrial activity and mines, we did not consider municipal sewage emissions of Hg to watersheds. This introduces additional uncertainty into the analysis, since watersheds could have a substantial fraction of Hg loading originating from municipal sewage treatment. In these instances, failure to consider this source of non-air loading could result in a high-bias in the estimates of the U.S. EGU-attributable fraction of deposition (and hence risk), since the contribution from this non-air source would not have been considered.

1.4.6.2 Additional research supporting the proportionality assumption and examining the issue of temporal response

The SAB commented that several recent publications have supported the finding of a linear relationship between Hg loading and accumulation in aquatic biota (Orihel et al., 2007; Orihel et al., 2008; Harris et al., 2007). The SAB noted that these studies suggest that that Hg deposited directly to aquatic ecosystems can become quickly available to biota and accumulated in fish, and reductions in atmospheric Hg deposition should lead to decreases in MeHg concentrations in biota.

EPA has reviewed the studies identified by the SAB, together with a study by Knightes et al., (2009). All of these studies, to varying degrees, suggest that when we are considering reductions in fish tissue Hg concentrations following reductions in Hg deposition to a watershed, we are likely to see a 2-stage response including: (a) an initial more rapid phase of reduction (ranging from months to a few years) reflecting a decrease in direct loading to the waterbody and subsequent reductions in fish tissue Hg concentrations related to decreased water column Hg levels and (b) a second slower phase of reduction (years to decades or more) reflecting longer term changes in the rate of erosion/runoff loading to the waterbody and the potential buffering effect of historical reservoirs of Hg in sediment. These findings suggest that we might not see an equivalent fractional reduction in risk matching the reduction in aerial deposition for near-term reductions (less than one year) due to historical reservoirs of Hg in sediment that continue to load the benthic food web and buffer the Hg response. However, for reductions over a longer period of time (sufficient for steady state, or near steady state conditions to be met in the aquatic system), the proportionality assumption likely holds and we would expect to see a reduction in risk matching the fractional reduction in Hg deposition. Since we have stated that our risk estimates are based on an assumption that steady state, or near steady state conditions are met, regardless of how long that takes, then the temporal response is not a factor and we have increased confidence in applying the proportionality assumption.

1.4.6.3 CMAQ mercury deposition modeling

We modeled total annual Hg deposition from U.S. and foreign anthropogenic and natural sources as well as the fraction of Hg deposition from U.S. EGUs using the CMAQ model. The Community Multi-scale Air Quality (CMAQ) model v4.7.1 (www.cmaq-model.org) is a state of the science three-dimensional Eulerian "one-atmosphere" photochemical transport model used to estimate air quality (Appel et al., 2008; Appel et al., 2007; Byun and Schere, 2006). CMAQ simulates the formation and fate of photochemical oxidants, ozone, primary and secondary PM concentrations, and air toxics over regional and urban spatial scales for given input sets of meteorological conditions and emissions. Mercury oxidation pathways are represented for both the gas and aqueous phases in addition to aqueous phase reduction reactions (Bullock and Brehme, 2002). Mercury estimates from CMAQ have been compared to observations and other mercury modeling systems in several peer reviewed publications (Bullock et al., 2008, 2009; Lin et al., 2007). Additional information about the model, model inputs for this assessment, and model evaluation are available in the Air Quality Modeling TSD (U.S. Environmental Protection Agency, 2011).

The 36km and both 12km modeling domains were modeled for the entire year of 2005. The emissions data used in the 2005 base year and 2016 total Hg emissions and U.S. EGU Hg emissions zero-out cases are based on the 2005 v4.1 platform. Emissions are processed to photochemical model inputs with the SMOKE emissions modeling system (Houyoux et al., 2000). The 2016 total Hg emissions case is intended to represent the emissions associated with growth and controls in that year projected from the 2005 simulation year. Only anthropogenic emissions changed between the 2005 and 2016 simulations, all other model inputs are the same in both simulations. Other North American emissions of criteria and toxic pollutants (including mercury) are based on a 2006 Canadian inventory and 1999 Mexican inventory (U.S. EPA, 2011b).

Global emissions of criteria and toxic pollutants (including mercury) are included in the modeling system through boundary condition inflow. The lateral boundary and initial species concentrations are provided by a three-dimensional global atmospheric chemistry model, the GEOS-CHEM model (standard version 7-04-11). The GEOS-CHEM predictions were used to provide one-way dynamic boundary conditions at three-hour intervals and an initial concentration field for the 36 km CMAQ simulations. The 36 km photochemical model simulation is used to supply initial and hourly boundary concentrations to the 12 km domains. Initial and boundary conditions for the projected future year (2016) 36 km simulations are the same as the 2005 base year. The first 10 days of the 36 km modeling simulation are not used in the analysis, which is beyond the number of days necessary to remove the influence of initial conditions on mercury deposition estimates (Pongprueksa et al., 2008).

The boundary inflow for the CMAQ mercury modeling used in the national-scale mercury risk assessment are based on a global model GEOS-CHEM simulation using a 2000 based global inventory as described in (Selin et al., 2007). A comparison of global mercury emissions by continent for 2000 and 2006 was recently published in (Streets et al., 2009) and show there is no discernable change in mercury emissions from Asia between 2000 and 2006. Given these consistent emissions estimates from Asia, the 2005 boundary inflow to the 36 km CMAQ domain was not adjusted. Mercury boundary conditions are the same for both the 2005

and 2016 simulations based on the consistency in Asian mercury emissions between 2000 and 2006, the declining ambient mercury concentrations in the northern hemisphere since 2000 (Slemr et al, 2011), and the large uncertainties surrounding projected global inventories of mercury emissions.

1.5   Differences between the 2005 Section 112(n) Revision Rule analysis and the current analysis in support of the Appropriate and Necessary Finding

In 2005, EPA conducted a set of technical analyses to support revision of the 2000 appropriate and necessary finding.[42] This section identifies key differences between the watershed-level risk assessment completed in support of the 2005 revision rule and the current risk assessment. These differences include both technical factors related to the design of the assessments, as well as differences in the interpretation of potential public health significance of the risk estimates generated. Key differences between the two analyses include:

Higher spatial resolution through use of CMAQ 12km grid cells: In this analysis, we modeled Hg deposition using CMAQ at a 12 km grid resolution, whereas in the 2005 analysis, we used CMAQ modeling with a 36 km grid cell resolution. The more refined spatial resolution at 12 km is more appropriate for representing areas of elevated U.S. EGU-attributable deposition (and total Hg deposition in general) compared with the 36 km resolution used in the 2005 analysis. The 12 km resolution also matches up with the more refined HUC12 watersheds now being used in the analysis, thereby allowing a more refined treatment of the intersection of aerial Hg deposition and measured fish tissue concentrations at the watershed level.

Application of more refined HUC12 watersheds: The current analysis uses HUC12 watersheds as the basis for risk estimation (these watersheds typically are 5-10 km on a side). By contrast, the 2005 analysis used HUC8s, which are much larger (averaging 40km on a side). The use of more spatially refined watersheds increases the potential for capturing areas of elevated aerial Hg deposition (combined with measured fish tissue levels).

Inclusion of updated fish tissue data: For this analysis, we included measured fish tissue data collected between 2000 and 2010. By contrast, the 2005 analysis used data collected between 1999 and 2003, which was the best available data at the time.

Subsistence fisher activity better defined and considered more ubiquitous: Based on an extensive review of available literature, we identified studies characterizing high-end self-caught fish consumption for a wide variety of source populations (e.g., Hispanic, Vietnamese, Whites and Blacks in the southeast, Great Lakes Tribal populations). Although in many cases, it was necessary to extrapolate high-end fishing activity to regions beyond those covered in the underlying studies, we believe that the literature supports the plausibility of high-end subsistence-like fishing activity across the watersheds included in the analysis. Additionally, the variety of studies identifying self-caught fishing activity at subsistence levels (i.e., a meal every few days to a meal every day) for a variety of diverse SES-differentiated populations in different

---

[42] U.S.EPA. 2005. Technical Support Document: Methodology Used to Generate Deposition, Fish Tissue Methylmercury Concentrations, and Exposure for Determining Effectiveness of Utility Emission Controls.

regions of the country, supports assessing this type subsistence fish consumption behavior across the modeled watersheds.

By contrast, in the 2005 analysis, we concluded that the study data characterizing fishing activity available at that time was limited in its ability to support modeling of subsistence fisher activity for the following reasons: (a) it characterized regional or local activity that could not be readily extrapolated more broadly, (b) fishing activity queried included consumption of saltwater species, or (c) specific high-end percentiles were not identified (or if they were, they only applied during specific harvesting periods - e.g., spearfishing months for Great Lakes Tribes). Therefore, in the 2005 analysis, we applied a high-end self-caught percentile values (95th and 99th percentiles) based on Tribal fishing practices in the Northwest to watersheds across the country.[43] The updated literature review, for the current analysis, led us to revise several of our earlier conclusions regarding high-end fishing activity. Specifically, while many of the studies of subsistence-like activity are regional in nature, when considered together, we now conclude that they support modeling subsistence-like fishing activity more broadly across the entire study area. Additionally, while some of the studies may include saltwater fishing in addition to freshwater (e.g., Burger, 2002), when those studies clearly covered both saltwater and freshwater self-caught fish consumption, we concluded that it was reasonable to assume that subsistence-like fishing activity could occur both at the coast and inland at freshwater bodies.[44]

For the current analysis, we used the 75th percentile fish tissue Hg concentration reflecting the potential for high-end subsistence fishers to target larger fish, which would have greater bioaccumulation potential relative to the average fish. By contrast, in the 2005 risk assessment, we used the maximum of the average fish tissue Hg concentrations across species of fish in a given HUC8, which is potentially a more conservative approach (i.e., resulting in higher risk, other factors equal).

Calculation of RfD-based HQ estimates including total and U.S. EGU-attributable risk and calculation of IQ loss: For this analysis, we compared total exposure to the MeHg RfD to generate an HQ estimate based on total Hg exposure for fishers at a given watershed. Furthermore, to focus on the U.S. EGU component of that total risk, we have generated two related risk metrics: (a) U.S. EGU incremental contribution to total risk which essentially considers the magnitude of the HQ when deposition from U.S. EGUs is considered before taking into account deposition and exposures resulting from other sources of Hg and (b) the percent of

---

[43] These NW Tribal fishing estimates are subject to considerable uncertainty when extrapolated to other areas in the U.S. These specific high-end fish consumption rates were derived for Tribes active in the Northwest who engage in specific cultural practices focused around salmon fishing. There is also significant uncertainty in extrapolating this type of highly-specific cultural-based fishing activity to other Tribes, let alone to other fish consumers in the U.S. By contrast, extrapolation of more generalized high-consuming rates (for ethnic groups and Whites and Blacks) to cover portions of the U.S. as was done in the current analysis is subject to less uncertainty given that these SES-differentiated populations are defined more generally and therefore are likely to demonstrate more consistency in culturally-related practices (such as subsistence fishing) across the country.

[44] In the situation where a study specifically characterized low income high-end fishing populations, as is done in the Burger 2010 study of activity in SC, we considered it reasonable to assume that low income individuals would likely conduct their frequent fishing activity near home. In that case, some of these high-end fishers would likely be located near the coast and some inland. In the case of subsistence-like fishing activity in the southeast, other studies from rivers in that area also showed subsistence-like fish consumption rates when only freshwater rivers were considered (e.g., Burger et al., 1999 focusing on fishing activity on the Savannah river in GA).

total HQ risk attributable to U.S. EGUs. The calculation of U.S. EGU incremental contribution to total HQ is identical to the IDI (index of daily intake) metric used in the 2005 analysis. However an important distinction is that in the current analysis, we highlight the fact that this U.S. EGU-related risk is always associated with a total HQ which is generally substantially larger (i.e., the U.S.-EGU-attributable HQ should not be considered in isolation as was done in the 2005 analysis with the IDI). By contrast, for the 2005 analysis both of the risk metrics used (i.e., the IDI and the comparison of U.S. EGU-related fish tissue concentrations against EPA's water quality criterion expressed as a Hg fish tissue value) essentially considered the U.S. EGU portion of risk in isolation. These risk metrics in the 2005 analysis were not contrasted with the much larger fraction of total Hg-related risk associated with the non-U.S. EGU portion of risk.

1.6   Detailed Example Calculation (watershed-level risk HQ)

This section provides a sample calculation of a watershed-level RfD-based HQ risk estimate generated for the 2016 scenario and walks the reader through each of the calculation steps associated with generating the estimate (see Figure 1-9). We cross-reference to sections of the Revised TSD that cover each of the intermediate calculations and data inputs involved in generating the risk estimate.[45] Each of the calculation steps is summarized below:

- Step 1: Based on the fish tissue Hg measurements available for a given watershed (after filtering), generate the 75th percentile fish tissue Hg concentration. We provide the median, 75th and 90th percentile values in Figure 1-9 to illustrate the spread in percentile values for this watershed). (see section 1.4.2).

- Step 2: Obtain CMAQ Hg deposition estimates for the watershed for the 2005 and 2016 scenarios. Compute the ratio of these two factors (i.e., 2016 deposition/2005 deposition). (see section 1.4.6.3).

- Step 3: Multiply the ratio from Step 2 by the fish tissue Hg concentration to project the fish tissue Hg concentration value to represent the 2016 scenario for this watershed. This step involves application of the proportionality assumption (see section 1.4.2.1).

- Step 4: Calculate intake rate for total Hg for the subsistence fisher scenario being assessed in that watershed using equation with fish tissue Hg concentrations calculated in Step 3(see section 1.4.4 for a discussion of this exposure calculation).

- Step 5: Compare the exposure estimate generated in step 4 to the MeHg RfD to generate a hazard quotient reflecting total Hg exposure for the subsistence fisher at that watershed (see section 1.4.5).

---

[45] The SAB recommended including a cross reference between the calculation steps outlined in Figure 1-9 and the discussion of variability and uncertainty presented in section 2-7. However, the complex interplay of sources of uncertainty and variability with the analytical structure used in the risk assessment precludes presentation of a clean linkage between sources of uncertainty and variability and the analytical framework used in the risk assessment. Most sources of variability and uncertainty impact either directly or indirectly multiple (if not all) of the calculations steps involved in generating risk estimates. Careful consideration of the analytical approach reflected in Figure 2-1 and the flow diagram outlining major analytical steps of the risk assessment presented in Figure 1-2 should allow the reader to determine which elements of the risk model are impacted by specific sources of uncertainty and/or variability.

- Step 6: Obtain CMAQ Hg deposition estimates for the watershed for both total and U.S. EGU-attributable Hg for the 2016 scenario. Compute the ratio of these two factors (i.e., U.S. EGU/total deposition). This ratio will be used to estimate the fraction of total risk attributable to U.S. EGUs (see section 1.4.6).

- Step 7: Estimate the fraction of the total HQ risk that is attributable to U.S. EGUs (for the 2016 scenario). This estimate is based on the proportionality assumption and uses the ratio developed in step 6 (see section 1.4.6).
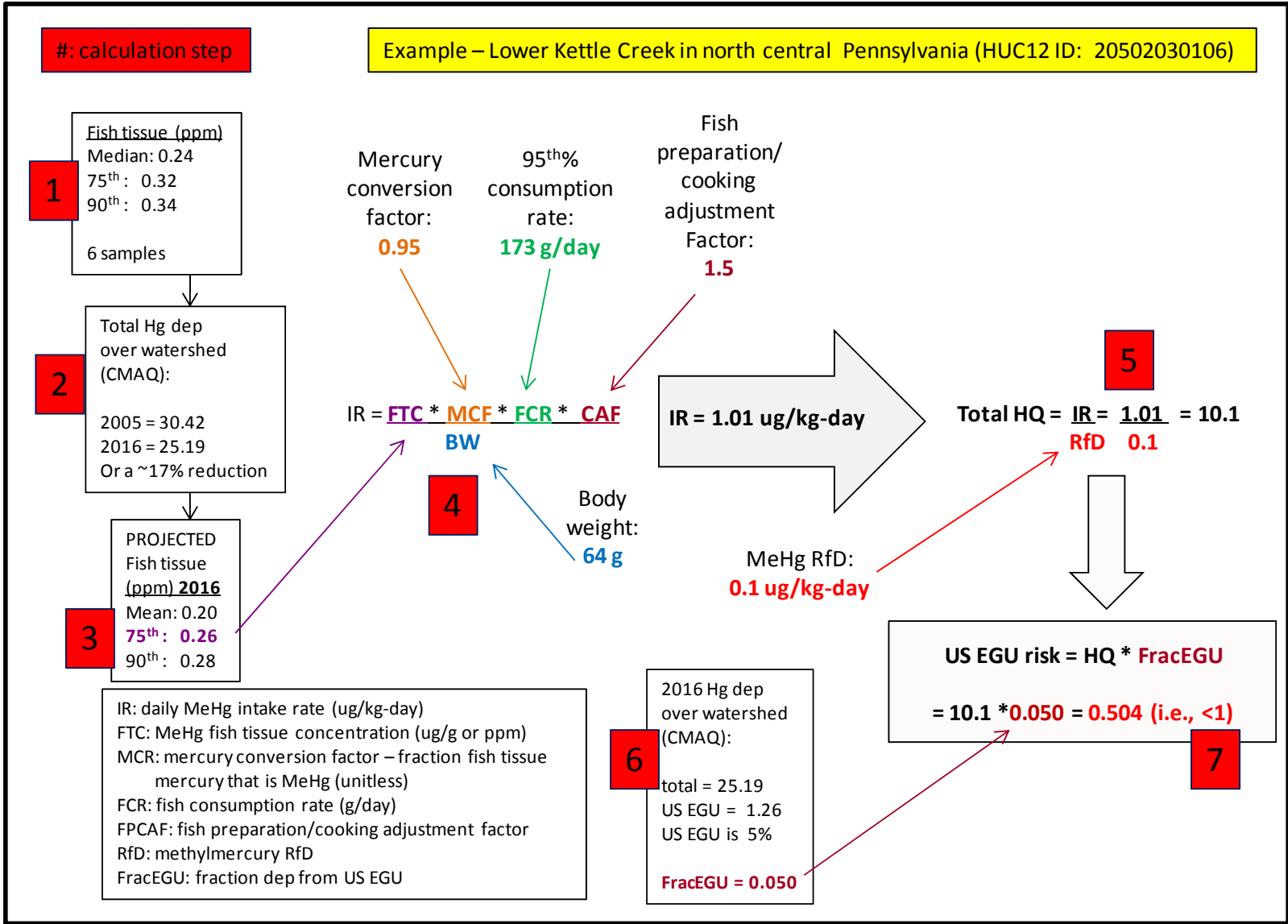
Figure 1-9 Sample Calculation for watershed-level Risk HQ

2   Discussion of Analytical Results

This chapter provides a discussion of the results of the 2016 simulation, including both risk estimates as well as intermediate calculations and inputs associated with generating those risk estimates. EPA's projection of total Hg emissions from U.S. EGUs in 2016 (after other CAA-related regulations are fully implemented) is 29 tons.[46] This national estimate is the same as the estimate of recent U.S. EGU Hg emissions based on information collected from industry through the Information Collection Request (ICR). This shows that significant reduction in U.S. EGU Hg emissions has occurred since 2005, when Hg emissions were estimated to be 52.9 tons, but that few additional reductions are expected to occur without additional regulations to reduce mercury emissions. The reductions between 2005 and 2016 are largely due to regulations and federal enforcement actions that achieve Hg reductions as a co-benefit of controls for NOx and SO_2 emissions. Given these estimates of total Hg emissions, characterization of "current conditions" is better represented by the 2016 Scenario than the 2005 Base Case, since total Hg emissions for the former is equal to our projection of recent emissions. By contrast, the 2005 analysis reflects total Hg emissions (52.9 tons) which are significantly higher than our estimate of recent emissions. For this reason, as mentioned earlier, we only present risk estimates for the 2016 Scenario and have not generated risk estimates for the 2005 scenario (although the 2005 Hg deposition estimates are used in scaling fish tissue Hg concentrations to represent concentrations in 2016).

In this section, we provide a brief overview of critical design elements of the risk analysis that the reader should keep in mind when reviewing the results (section 2.1). We then discuss the intermediate inputs and outputs for individual analytical steps associated with the risk assessment including: (a) Hg deposition from U.S. EGUs as modeled using CMAQ (section 2.2), (b) fish tissue Hg concentrations (section 2.3), (c) relationship between Hg deposition and Hg fish tissue concentrations (section 2.4) and (d) the MeHg RfD-based HQ risk assessment results for the 2016 scenario (section 2.5). In discussing each category of results, emphasis is placed on identifying key policy-relevant observations. In section 2.6, we discuss the results of several sensitivity analyses conducted to characterize the potential impact of specific sources of uncertainty on the risk estimates. In section 2.7, we discuss variability and uncertainty related to the risk assessment. In Section 3, we provide a summary of critical observations from the analysis, which draws on information provided in sections 2.2 through 2.7.

2.1   Key design elements to consider when reviewing the risk assessment results

The following design elements of the analysis should be considered when reviewing the results:

- The analysis focuses on subsistence-like fishing activity at inland freshwater bodies. The analysis is not intended to capture more generalized recreational fishing activity or to reflect self-caught fisher exposure associated with saltwater fishing or fishing in the Great

---

[46] The modeling conducted for the March risk assessment included a representation of the proposed Cross State Air Pollution Rule, which was finalized in June 2011. We have evaluated differences in projected Hg emissions after taking into account differences between the proposed and final CSAPR and determined that the difference is less than 2 tons nationally, which would have little expected impact on our risk estimates.

Lakes. In comparing any risk profiles generated in this analysis to risks estimated in other contexts, the specific focus on these high-end populations shows risks substantially higher than risks for recreational fishers.

- The risk estimates generated are based on a set of female subsistence fish consumer scenarios evaluated at watersheds where (a) we have fish tissue Hg data and (b) this type of subsistence fisher activity could potentially occur. Therefore, the risk assessment is watershed-focused, does not provide population-representative risk estimates for subsistence fishers.

- Risks are estimated for 3,141 watersheds. This watershed coverage (only about 4% of U.S. watersheds) leaves much of the country not covered by the analysis, including a substantial number of watersheds with relatively elevated levels of U.S. EGU-related Hg deposition. Further, the eastern part of the U.S. is more heavily represented in the watershed-level estimates of risk. Given that U.S. EGU Hg deposition is generally higher in the eastern part of the U.S., the fact that the risk assessment is focused on this part of the country is considered to be a strength of the analysis.

- The analysis uses a proportionality assumption to link changes in Hg deposition (over watersheds) to changes in Hg fish tissue levels. This approach assumes that near steady-state conditions are met, which may take years to decades at a given watershed following changes in Hg deposition.

- The analysis estimates risk based on MeHg RfD-based HQ. The U.S. EGU-attributable HQ should always be considered in the context of total HQ which is typically substantially larger than the U.S. EGU-attributable HQ (reflecting the large contribution from non-U.S. EGU sources of Hg). We also assessed IQ loss in children, however, due to concerns that the IQ loss endpoint may not fully capture all of the neurodevelopmental effects associated with MeHg exposure, we have moved discussion of the IQ loss estimates to Appendices A and B.

- Because it is not feasible to enumerate the female subsistence fish consumers modeled in this analysis, we could not generate distributions of population-weighted risk for specific scenarios assessed (e.g., low income Hispanic fishers, or Tribal fishers in the vicinity of the Great Lakes). However, we do believe, based on surveys of their behavior, that this type of subsistence-like activity could reasonably be expected to occur across some fraction of the 3,141 watersheds included in the analysis. Therefore, we have assessed female subsistence fish consumer risk for each watershed where we have fish tissue Hg data. We then consider the fraction of watersheds that meet the risk characterization criteria outlined in the risk characterization framework (see Section 1.3).

2.2 Mercury Deposition from U.S. EGUs as Modeled Using CMAQ

Below we provide the results of the CMAQ modeled Hg deposition for the 2005 and 2016 scenarios for total deposition and U.S. EGU-attributable deposition.[47] For this Revised

---

[47] During the Science Advisory Board (SAB) review of the March TSD held on June 15-17th, Panel members raised questions regarding patterns of mercury deposition reflected in Figures 2-1 though 2-4 of the March version of the

TSD we have also included maps of wet and dry deposition for the 2005 and 2016 scenarios.[48] Unlike the results presented in the subsequent sections that are limited to watersheds with fish tissue data, we modeled Hg deposition in all 88,000 watersheds in the continental U.S. We present a set of bulleted observations at the end of the section that draws on information conveyed in the figures and tables. The set of figures and tables presented include:

- Figure 2-1 and 2-2: Maps presenting CMAQ modeling results for total Hg deposition (μg/m^2) at the watershed-level, for the 2005 and 2016 scenarios respectively.

- Figures 2-3 and 2-4: Maps presenting CMAQ modeling results for U.S. EGU-attributable Hg deposition (μg/m^2) at the watershed-level, for the 2005 and 2016 scenarios, respectively.

- Figures 2-5 through 2-8: Maps presenting CMAQ modeling results for wet and dry Hg deposition (μg/m^2) for the 2005 and 2016 scenarios. These estimates are presented at the original CMAQ 12km grid resolution and have not been interpolated to the watershed-level. Maps presenting wet and dry deposition modeling results have been included to allow readers to consider patterns of deposition over specific locations and potentially compare those with measured data, in those instances where relevant data are (or become) available.

- Table 2-1: Summary of statistics (mean, 50th, 75th, 90th, 95th and 99th percentiles) for total Hg deposition and U.S. EGU-attributable deposition for the 2005 and 2016 scenarios.

- Table 2-2: Summary of statistics (mean, 50th, 75th, 90th, 95th and 99th percentiles) for U.S. EGU-attributable deposition as a percent of total deposition for the 2005 and 2016 scenarios.

- Table 2-3: Summary of statistics (mean, 50th, 75th, 90th, 95th and 99th percentiles) for percent reduction of (a) total Hg deposition, and (b) U.S. EGU-attributable deposition, based on comparison of the 2016 scenario against the 2005 scenario.

Mercury Risk TSD. The figures in the March TSD were intended to show annual total mercury (Hg) deposition per unit area (in units of μg/m^2) by watershed, however they actually displayed intermediate calculations that had not been adjusted by the waterbody-specific surface areas. We corrected the figures and provided them in a memo to the docket (EPA-HQ-OAR-2009-0234-15522). The figures presented here are the corrected figures and match those presented in that memo.

[48] Inclusion of maps presenting wet and dry deposition modeling results reflects suggestions made by SAB Panel members.
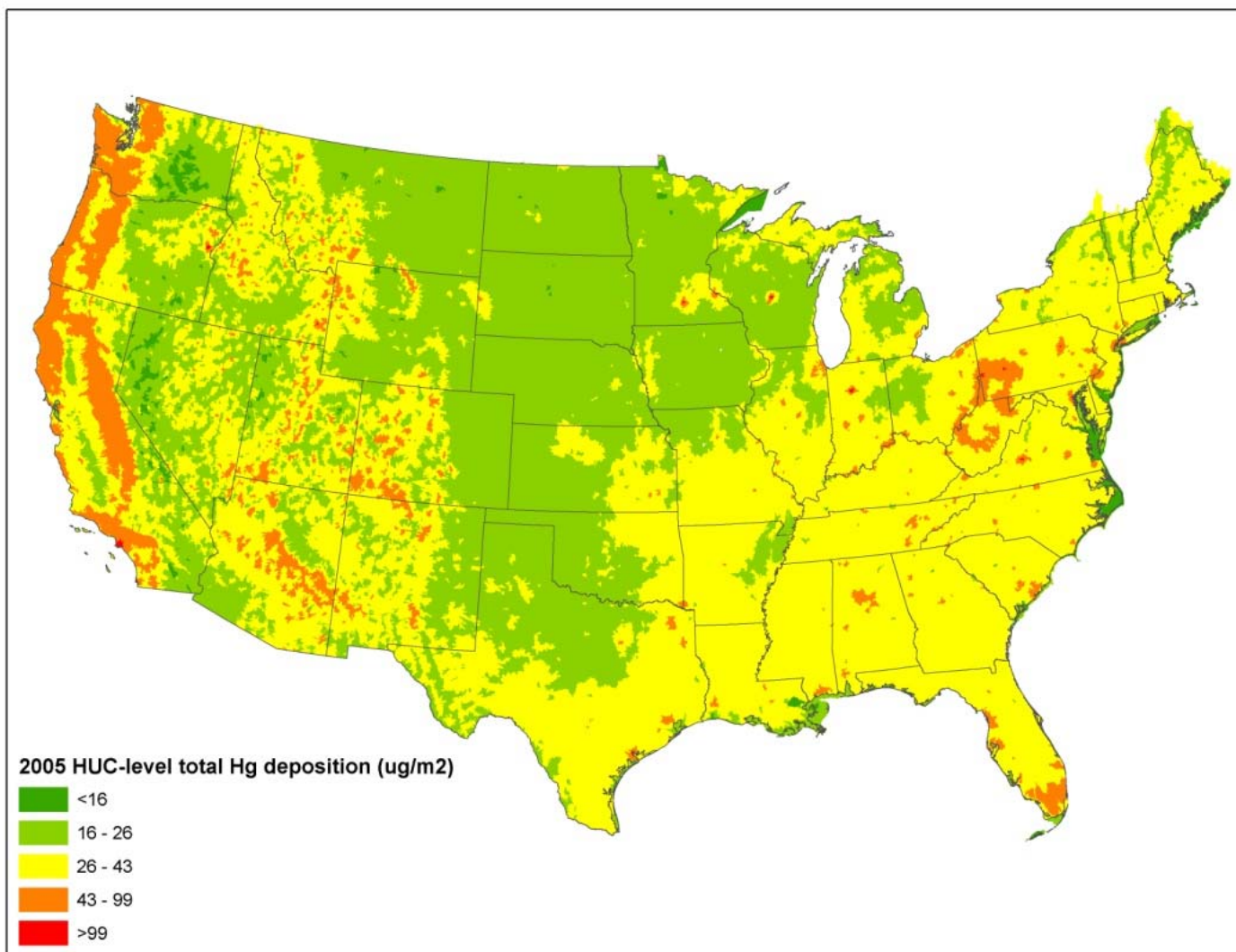
Figure 2-1 Total Mercury Deposition by HUC (μg/m^2) for the 2005 Scenario

56

2016 HUC-level total Hg deposition (ug/m2)

- <16
- 16 - 26
- 26 - 43
- 43 - 99
- >99
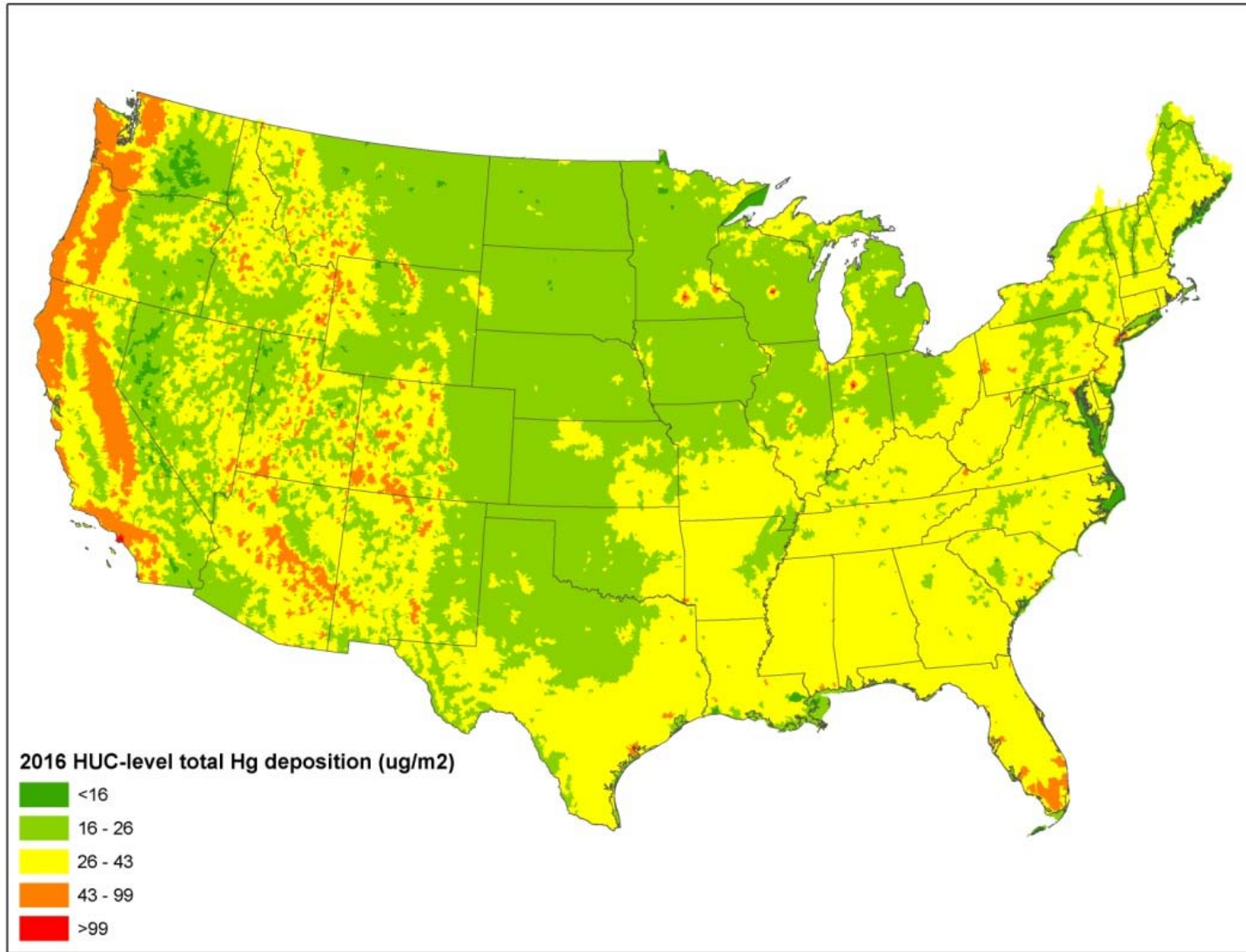
Figure 2-2 Total Mercury Deposition by HUC (μg/m^2) for the 2016 Scenario
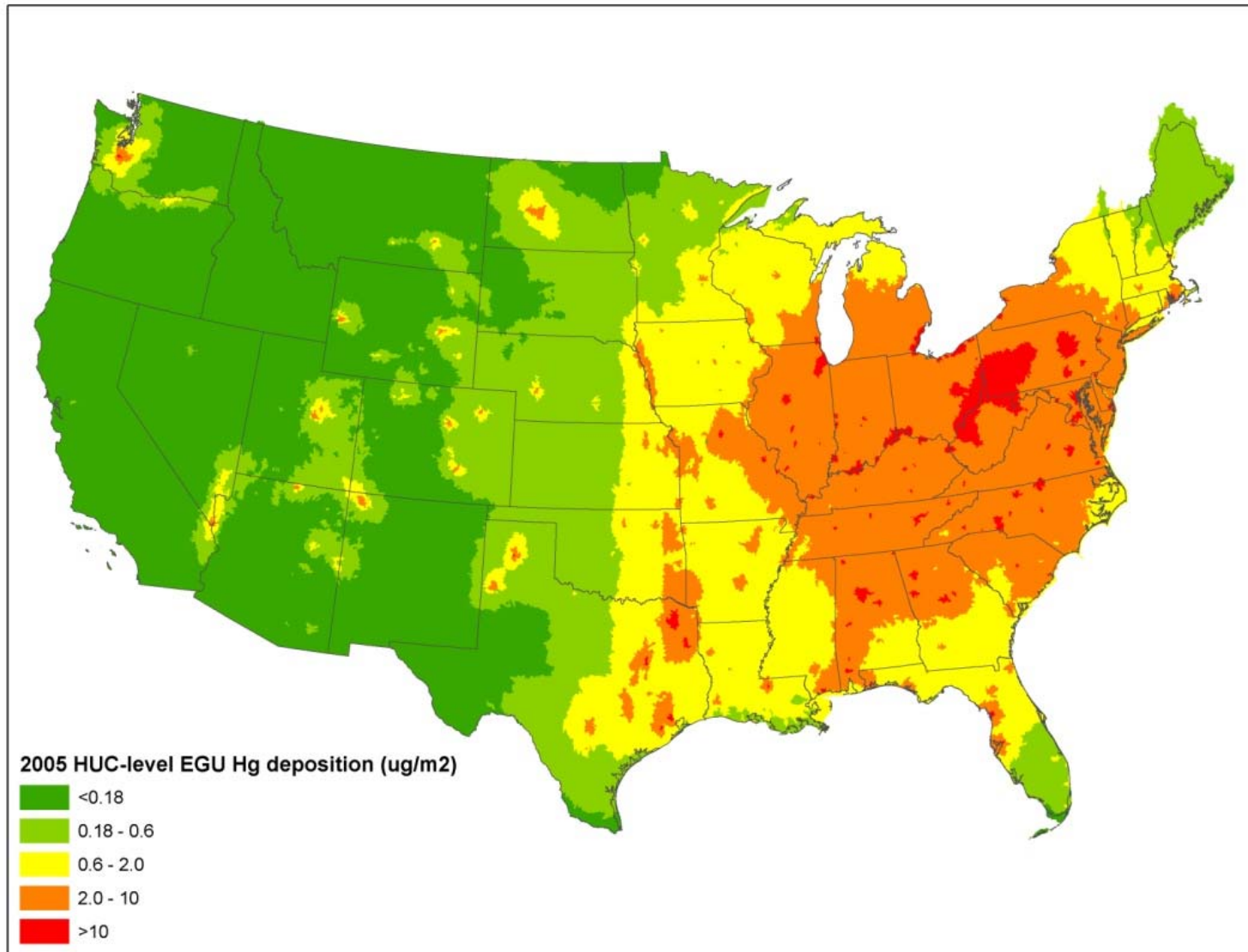
Figure 2-3   U.S EGU-Attributable Mercury Deposition by HUC (µg/m^2) for the 2005 Scenario

2016 HUC-level EGU Hg deposition (ug/m2)

- <0.18
- 0.18 - 0.6
- 0.6 - 2.0
- 2.0 - 10
- >10
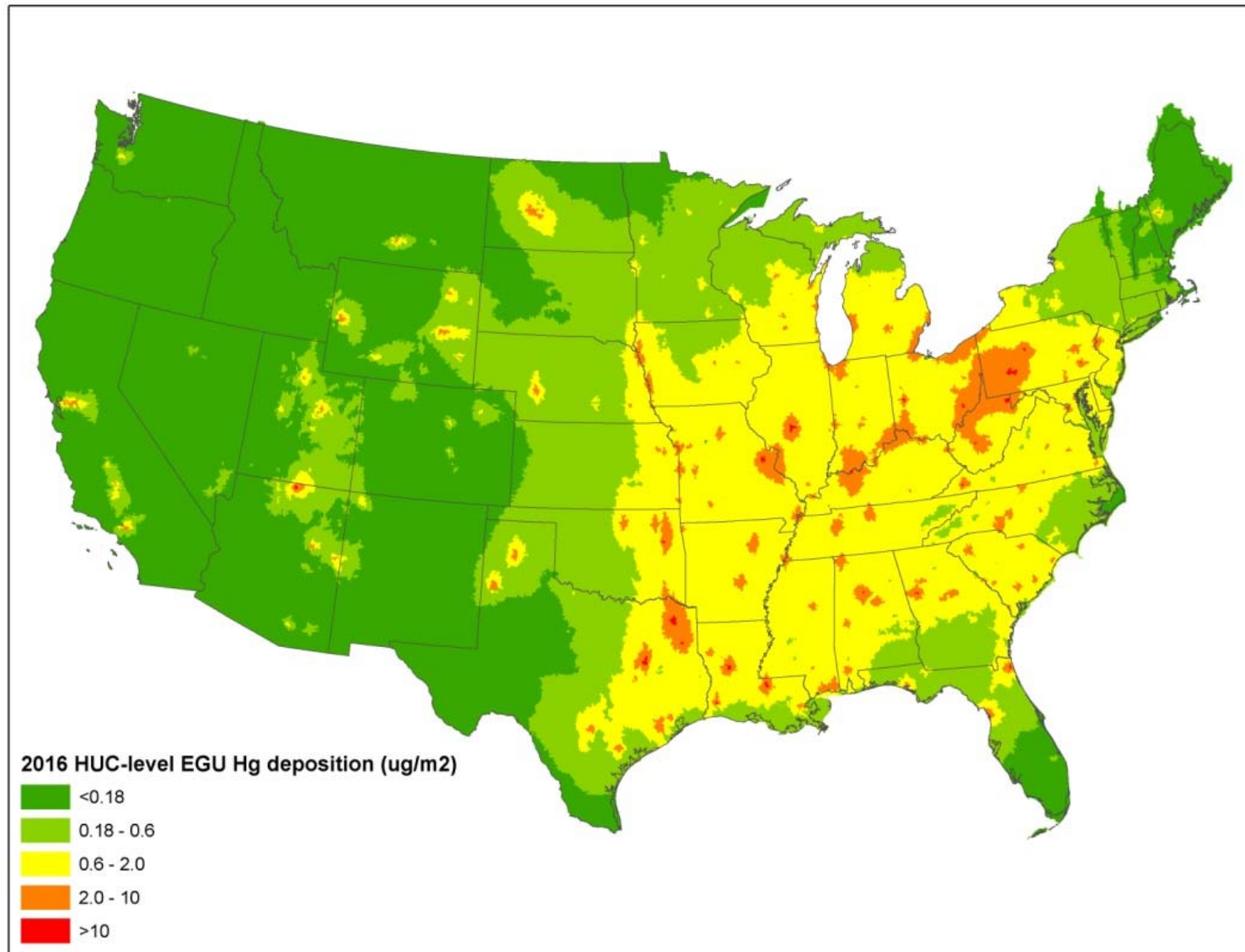
Figure 2-4 U.S EGU-Attributable Mercury Deposition by HUC (µg/m^2) for the 2016 Scenario
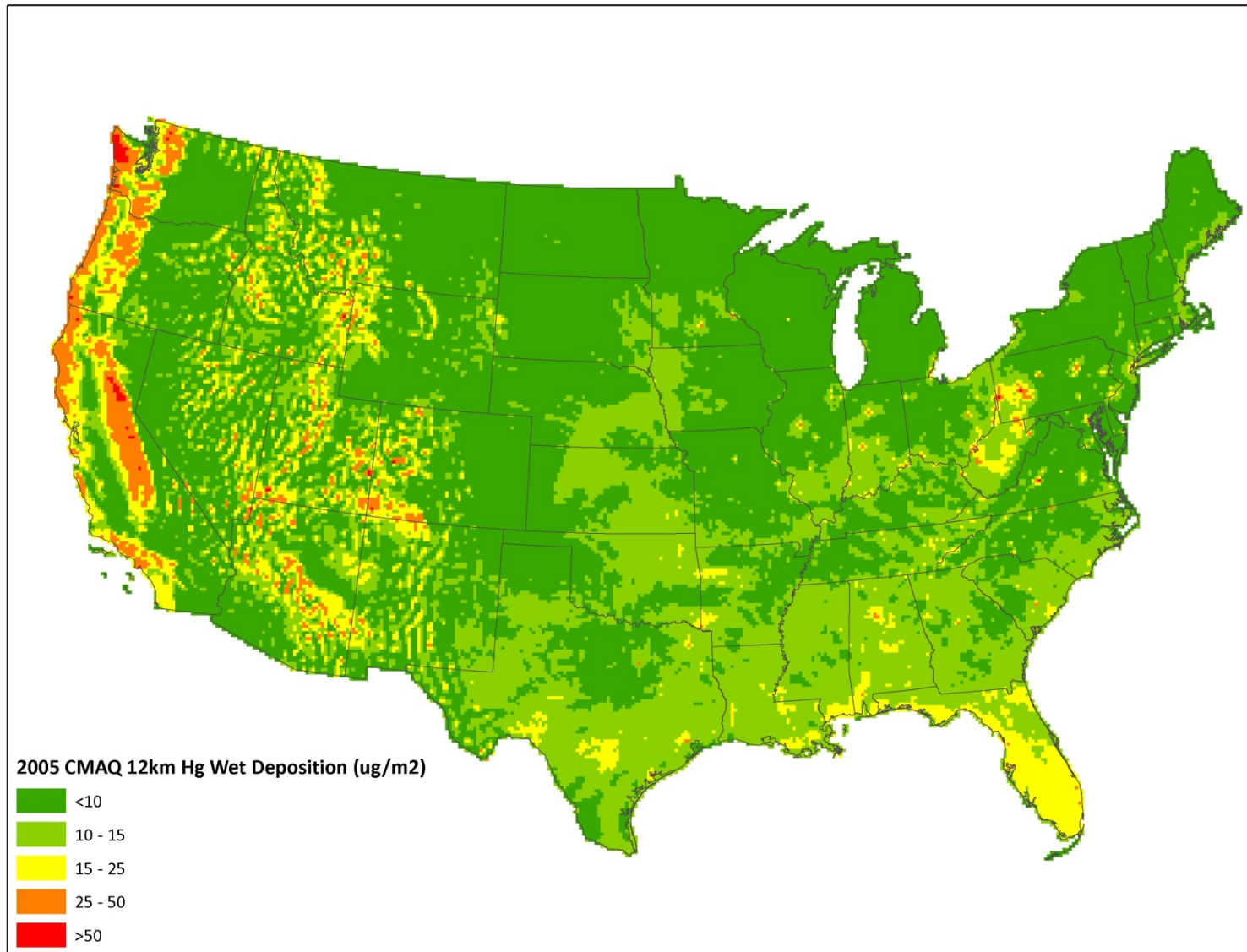
Figure 2-5 Mercury Wet Deposition by 12km Grid Cell (µg/m^2) for the 2005 Scenario
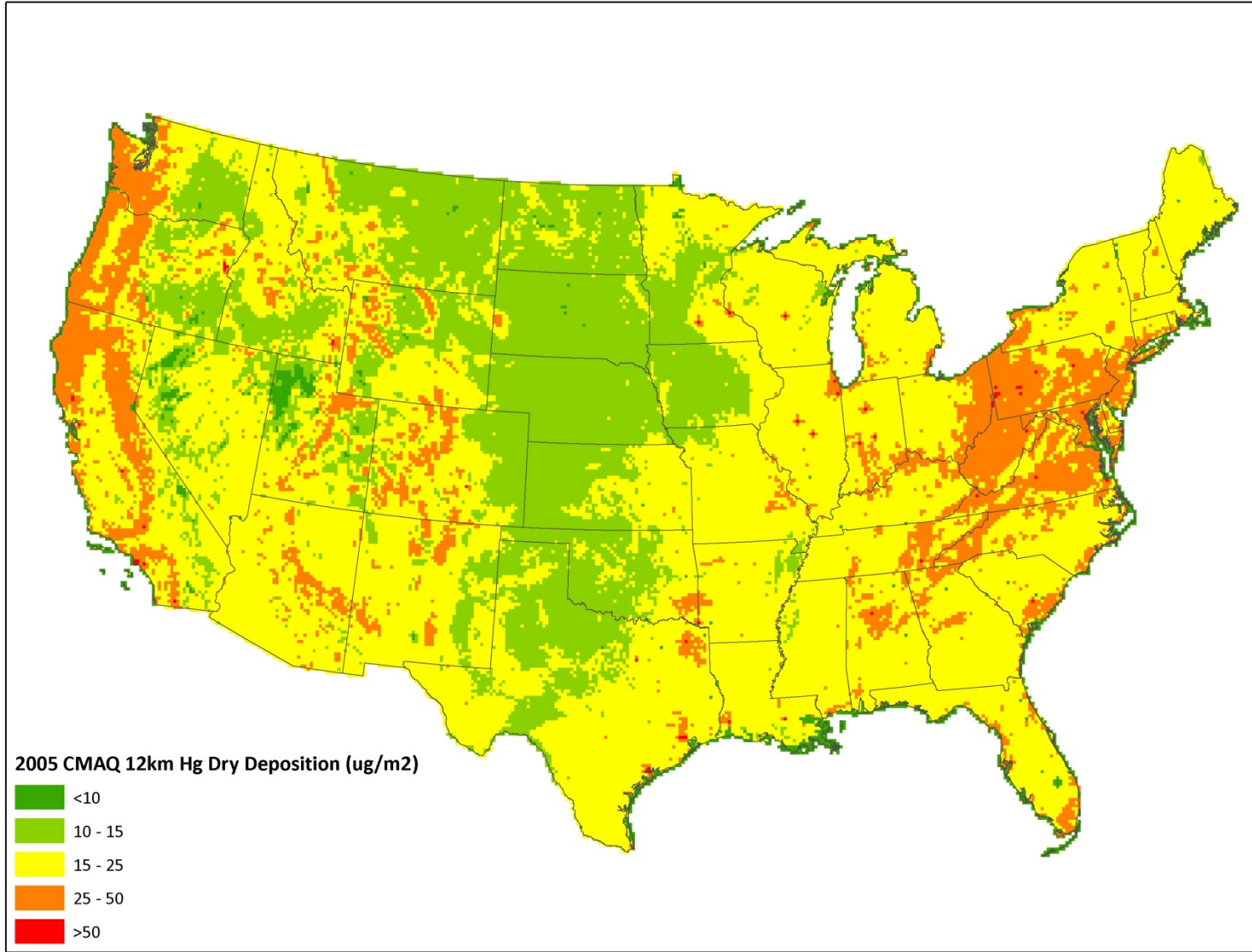
Figure 2-6 Mercury Dry Deposition by 12km Grid Cell (µg/m^2) for the 2005 Scenario

2016 CMAQ 12km Hg Wet Deposition (ug/m2)

- <10
- 10 - 15
- 15 - 25
- 25 - 50
- >50
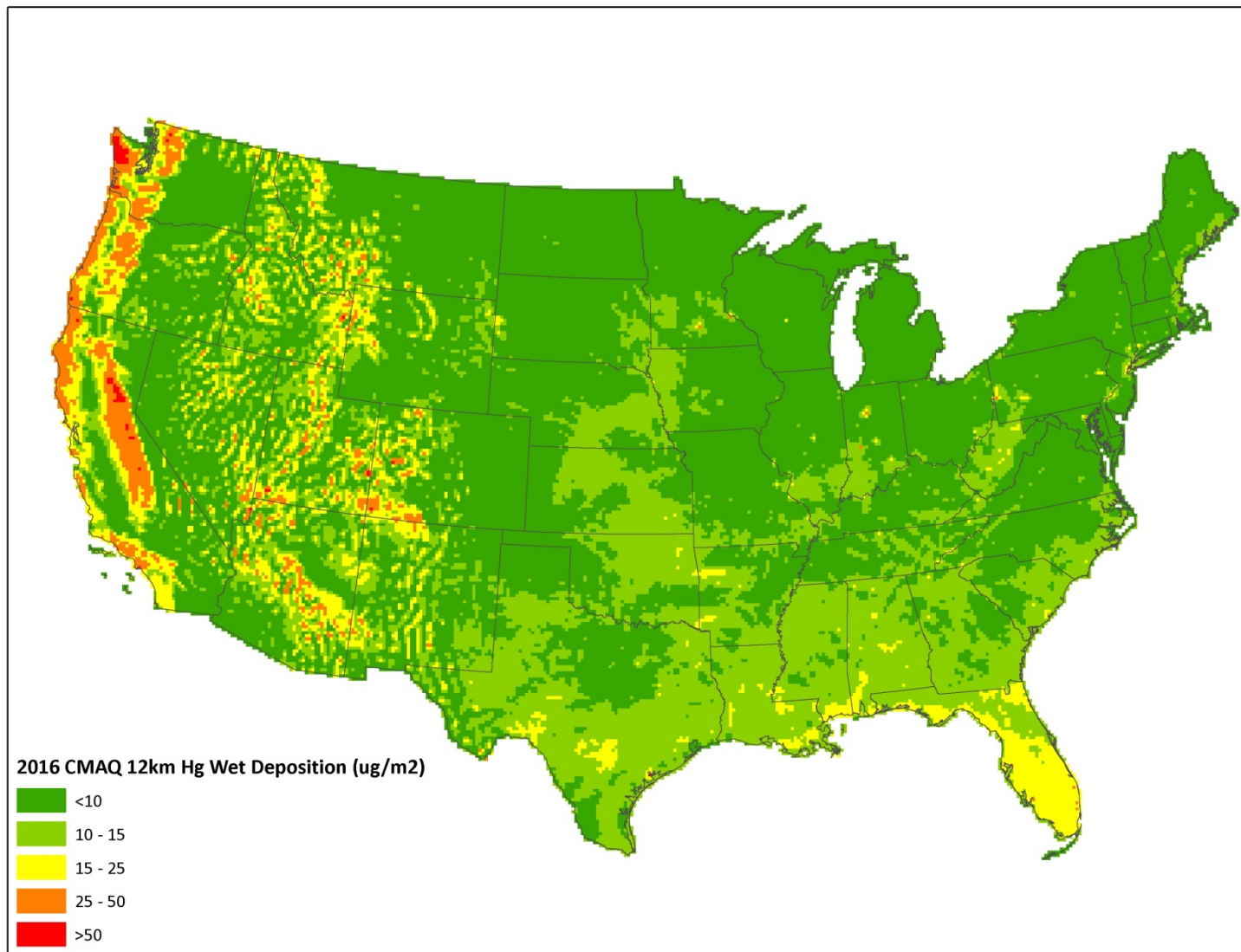
Figure 2-7 Mercury Wet Deposition by 12km Grid Cell (μg/m^2) for the 2016 Scenario

2016 CMAQ 12km Hg Dry Deposition (ug/m2)

- <10
- 10 - 15
- 15 - 25
- 25 - 50
- >50
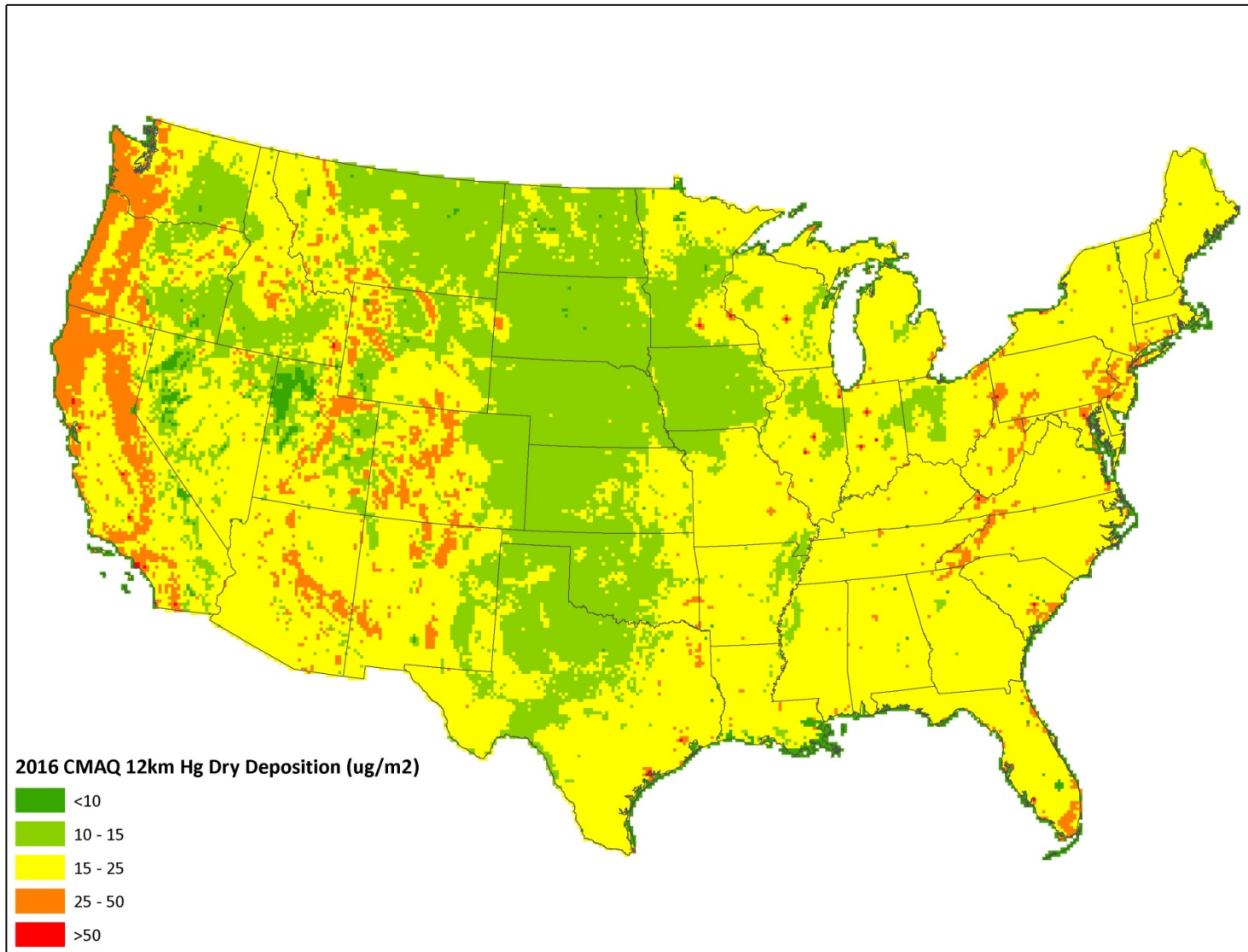
Figure 2-8 Mercury Dry Deposition by 12km Grid Cell (μg/m^2) for the 2016 Scenario

Table 2-1 Comparison of total and U.S. EGU-attributable mercury deposition (µg/m^2) for the 2005 and 2016 scenarios.*

Statistic	2005 scenario		2016 scenario	
	Total Hg Deposition	**U.S. EGU-attributable Hg Deposition**	**Total Hg Deposition**	**U.S. EGU-attributable Hg Deposition**
Mean	19.41	0.89	18.66	0.34
Median	17.25	0.24	16.59	0.15
75th percentile	23.69	1.07	22.83	0.46
90th percentile	30.78	2.38	29.90	0.85
95th percentile	36.85	3.60	35.16	1.18
99th percentile	58.32	7.77	56.23	2.41

* Values are based on CMAQ results interpolated to the watershed –level and reflect trends across all ~88,000 watersheds in the continental U.S. Percentiles for total Hg deposition and U.S. EGU attributable Hg deposition are not matched, e.g. the watershed with the 99th percentile for total Hg deposition will not be the same watershed as the watershed with the 99th percentile for U.S. EGU attributable Hg deposition.

Table 2-2 Comparison of percent of total mercury deposition attributable to U.S. EGUs for 2005 and 2016.*

Statistic	2005 scenario	2016 scenario
Mean	5%	2%
Median	1%	1%
75th percentile	6%	3%
90th percentile	13%	5%
95th percentile	18%	6%
99th percentile	30%	11%

* Values are based on CMAQ results interpolated to the watershed –level and reflect trends across all ~88,000 watersheds in the U.S.

Table 2-3 Comparison of percent reduction of total mercury deposition, and U.S. EGU-attributable deposition, based on comparing the 2016 scenario against the 2005 scenario.*

Statistics	Percent Change in Total Hg Deposition	Percent Change in U.S. EGU-attributable Hg Deposition
Mean	-4%	NC**
Median	-1%	-41%
75th percentile	-5%	-70%
90th percentile	-12%	-80%
95th percentile	-16%	-85%
99th percentile	-27%	-91%

* Values are based on CMAQ results interpolated to the watershed –level and reflect trends across all ~88,000 watersheds in the U.S.

** A mean value was not calculated (NC) for this category due to presence of a number of watersheds with very small U.S. EGU-attributable deposition values which skewed this distribution.

We made the following observations based on the information presented in Figures 2-2 through 2-4 and in Tables 2-1 through 2-3 regarding estimates of total and U.S. EGU-attributable Hg deposition for the 2005 and 2016 scenarios:

- *Patterns of total and U.S. EGU-related Hg deposition differ considerably*: Areas of elevated total Hg deposition are distributed around the country (e.g., west coast, areas in Nevada, southern Mississippi, West Virginia, southeastern Georgia) (see Figures 2-1 and 2-2). By contrast, U.S. EGU Hg deposition is concentrated in the eastern U.S., especially in the Ohio River Valley (see Figures 2-3 and 2-4). Figures 2-3 and 2-4 also illustrate that while some near-coastal areas and portions of the Great Lakes have elevated U.S. EGU-attributable deposition, many of the highest areas (and largest expanses) of U.S. EGU-attributable deposition occur inland (e.g., Ohio River Valley, areas in northeast Texas and along the Mississippi River).

- *U.S. Hg deposition is generally dominated by sources other than U.S. EGUs and the contribution from U.S. EGUs decreases between the 2005 and 2016 scenarios*: On average., U.S. EGUs contribute 5% of total Hg deposition for the 2005 scenario, which decreases to 2% for the 2016 scenario (see Table 2-2). The remaining Hg deposition (i.e., ~95% and ~98%, respectively for the two scenarios) originates from other U.S. sources of Hg emissions and from international sources (both anthropogenic and natural). U.S. EGU-attributable deposition decreases considerably between the 2005 and 2016 scenarios, primarily from implementation of the Cross State Air Pollution Rule (CSAPR), state Hg regulations and Federal enforcement actions.[49] The median reduction in U.S. EGU-attributable deposition was 41% with reductions ranging up to 85% for the 95th percentile watershed (see Tables 2-2 and 2-3).

- *The contribution of U.S. EGU-attributable deposition to total deposition varies across watersheds and can represent a relatively large fraction in some instances*: In the 2005 scenario, while on average, U.S. EGUs only contributed 5% of total Hg deposition in the U.S., this contribution ranged up to 30% for the 99th percentile watershed (see Table 2-2). While overall U.S. EGU –attributable deposition decreased substantially between the 2005 scenario and the 2016 scenario, U.S. EGUs contributed 11% of total Hg deposition for the 99th percentile watershed in 2016 ((see Table 2-2).

2.3   Fish Tissue Mercury Concentrations

This section characterizes the subset of U.S. watersheds with fish tissue Hg data included in the current the risk assessment. This dataset includes (a) the original HUC-level fish tissue dataset used in the March version of the risk assessment (post augmentation data set with 2,317 watersheds) and (b) the additional 940 watersheds with fish tissue data identified as part of our refinement to the March risk assessment (for a total of 3,141 watersheds). In this section, we provide the 75th percentile fish tissue sample for each watershed. As discussed in Sections 1.4.2.1 and 1.4.6, we used the proportionality assumption together with Hg deposition coverages

---

[49] Controls on PM precursors, including directly emitted PM and SO_2, can significantly reduce divalent and particle-bound mercury, both of which primarily deposit locally and regionally. For more information on the emission reductions from CSAPR, see the final Regulatory Impact Analysis, which is available at http://www.epa.gov/airtransport/pdfs/FinalRIA.pdf.

for 2005 (total and U.S. EGU-related) and 2016 (total and U.S. EGU-related) to project fish tissue Hg concentrations for the 2016 scenario and to estimate the U.S. EGU-attributable fraction of total fish Hg.

We provide figures and tables to summarize the fish tissue data. As discussed in Section 2.2, most of the areas with elevated U.S. EGU-attributable Hg deposition are located in the eastern U.S., where we also have more Hg fish tissue data and most of the U.S. EGU emission reductions between 2005 and 2016. We also present a set of bulleted observations based on the figures and tables. The set of figures and tables presented include:

- Figure 2-9: Map of 3,141 watersheds with fish tissue sampling data used in the current version of the risk assessment, including (a) watersheds included in the previous version of the risk assessment (2010 MFT) and (b) additional fish tissue data incorporated to enhance the current version of the risk assessment (augmentation). This map illustrates the uneven coverage of the fish tissue dataset across the continental U.S and highlights which states were included in the additional data incorporated for this version of the risk assessment (i.e., the augmentation HUCs shown in red).

- Figure 2-10: Map of 3,141 watersheds with fish tissue data showing distribution of sampling frequency (number of fish tissue measurements) across watersheds. This map can be used to show the relationship between trends in sampling frequency and fish tissue Hg concentrations.

- Figure 2-11 and 2-12: Maps presenting Total Fish Tissue Mercury Concentrations for the 2005 and 2016 scenarios respectively.

- Figures 2-13 and 2-14: Maps presenting EGU-Attributable Fish Tissue Mercury Concentrations for 2005 and 2016 scenarios, respectively.[50]

- Table 2-4: Comparison of watershed-level fish tissue Hg concentrations (including means and various percentiles) for the March and revised version of the national-scale Hg risk assessment. Table 2-4 includes percentile data for (a) 2010 MFT dataset (the fish tissue data used in the March risk assessment), (b) the augmentation dataset (additional fish tissue data collected to supplement data used in the last risk assessment) and (c) the 2011 MFT dataset (the dataset used in the current revised version of the risk assessment).

- Table 2-5: Summary of statistics (mean and various percentiles) for both total and U.S. EGU-attributable Hg fish tissue levels (for the 2005 and 2016 scenarios). These statistics are based on watershed-level data. In addition, this table also presents the percent reduction (between the 2005 and 2016 scenarios) for both total and U.S. EGU-attributable Hg fish tissue levels.

---

[50] Note, in the March version of the Mercury Risk TSD, we included a series of maps showing fish tissue Hg concentrations at the watershed-level for the upper 10 percent of watersheds with regard to total fish tissue Hg concentrations and EGU-attributable fish tissue Hg concentrations. However, we decided to remove these maps from the revised version of the Mercury Risk TSD given the limited coverage that the fish tissue Hg sampling data provides for watersheds across the U.S.
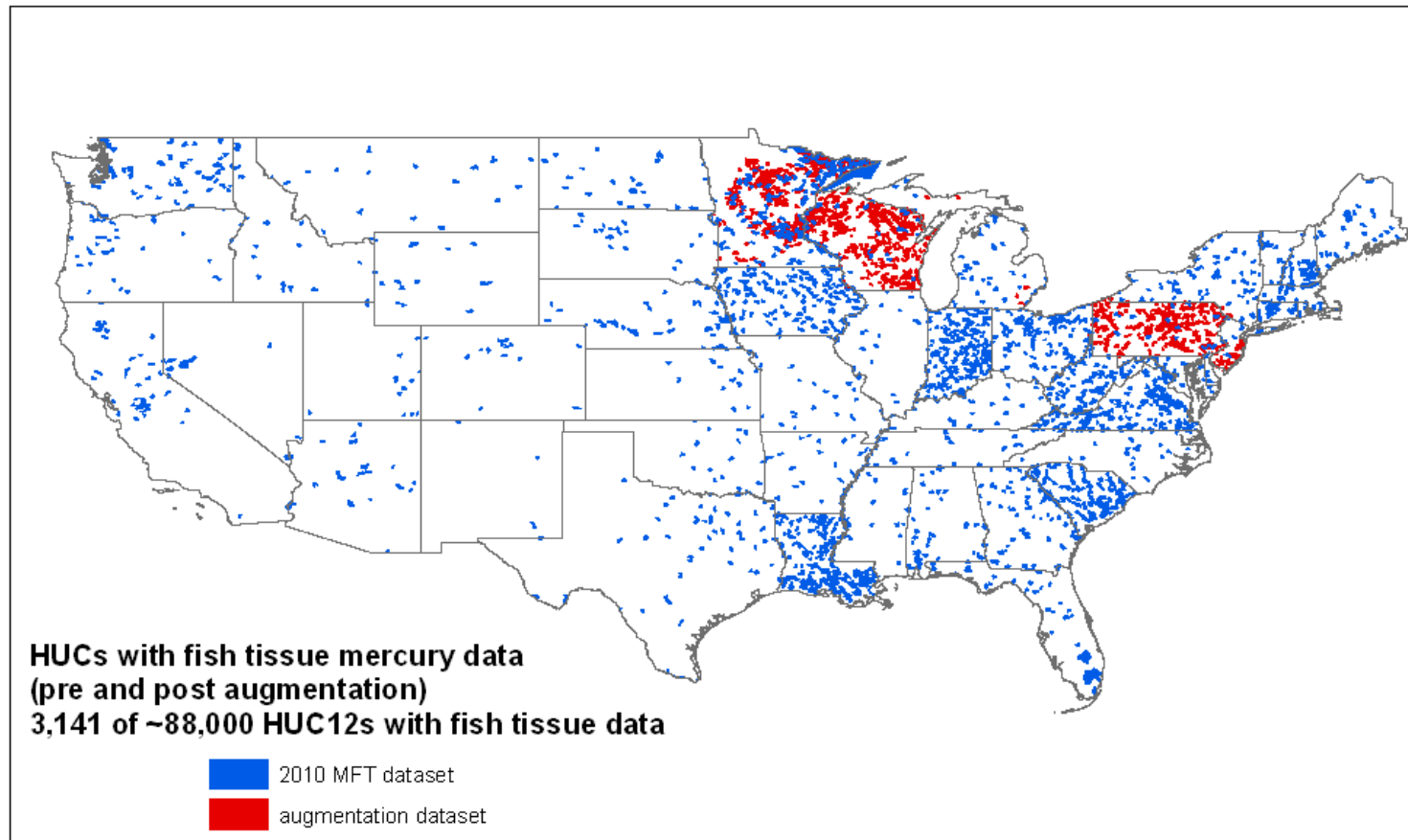
Figure 2-9 Set of 3,141 HUC12s with Fish Tissue Mercury Data Used in the Risk Assessment*

*Includes *2010 Mercury Fish Tissue (MFT)* dataset used in the March version of the risk assessment as well as the *augmentation* dataset incorporated for the current version of the risk assessment.
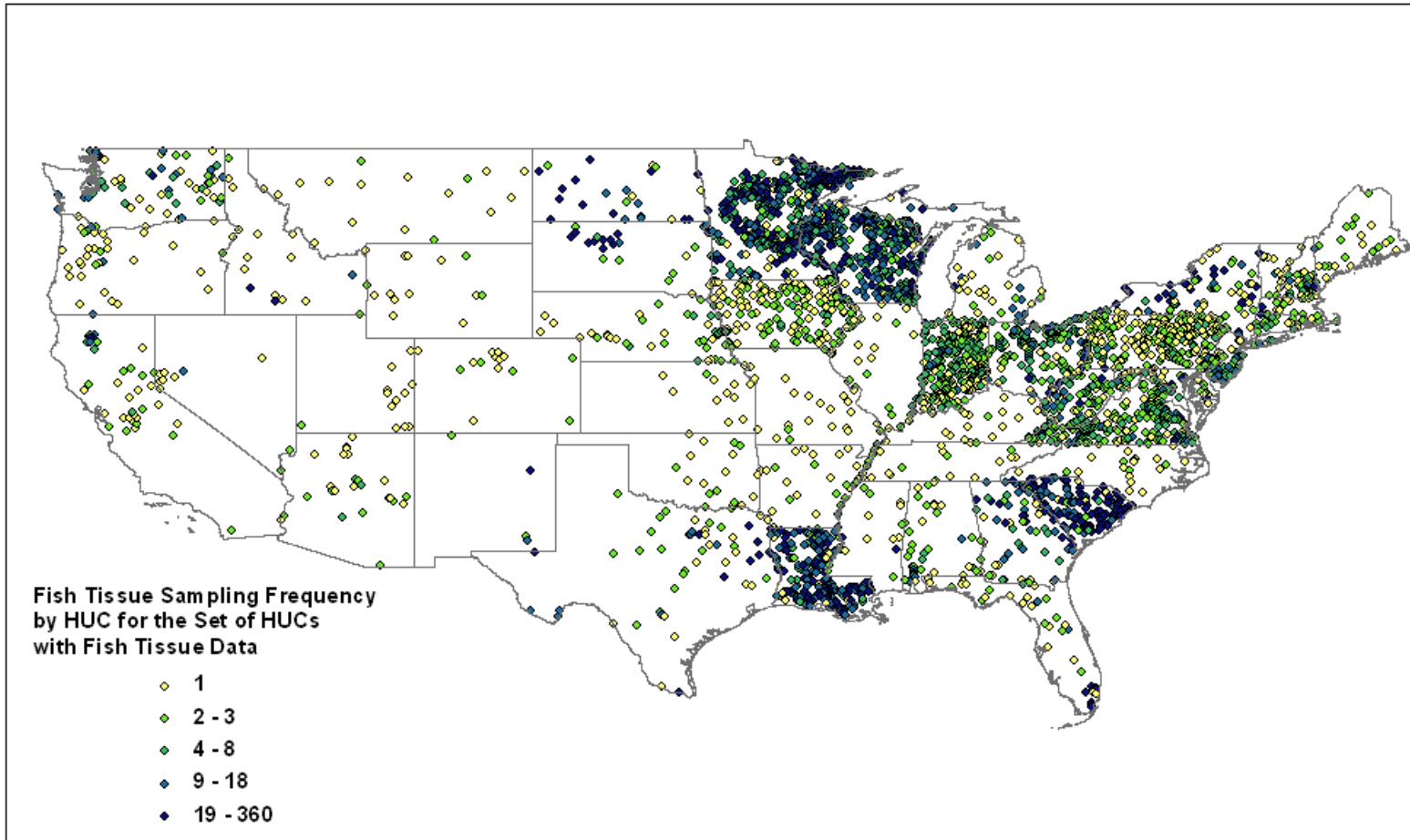
Figure 2-10 Fish Tissue Measurement <u>Sampling Frequency</u> for HUCs with Fish Tissue Data Included in the Risk Assessment

Fish Tissue Mercury Data
(HUC12-level 75th percentile values, ppm)

- 0.000000 - 0.109143
- 0.109144 - 0.195000
- 0.195001 - 0.300000
- 0.300001 - 0.479000
- 0.479001 - 6.605000
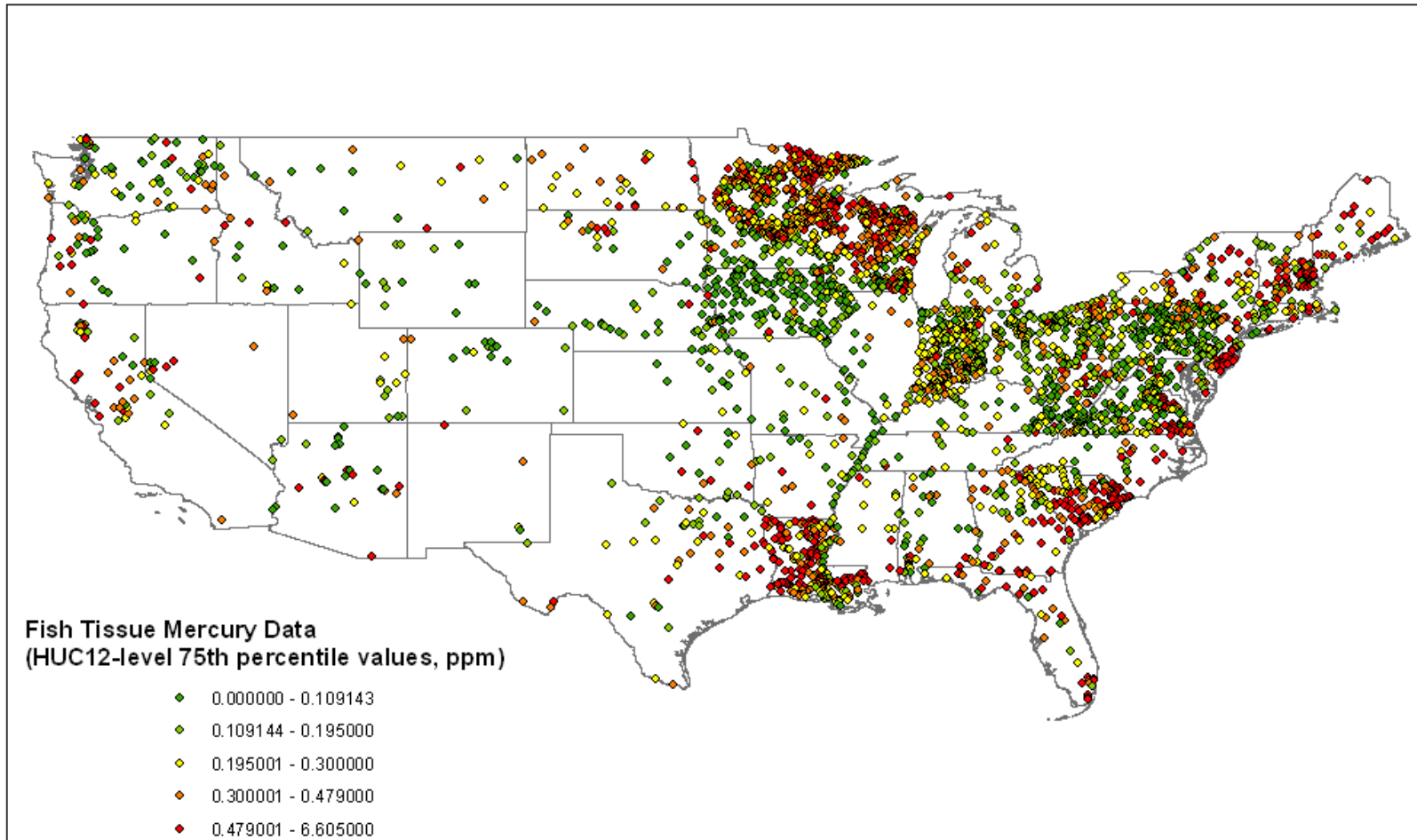
Figure 2-11   Total Fish Tissue Mercury Concentrations for 2005 Scenario (HUC12-level 75th percentile values, ppm)
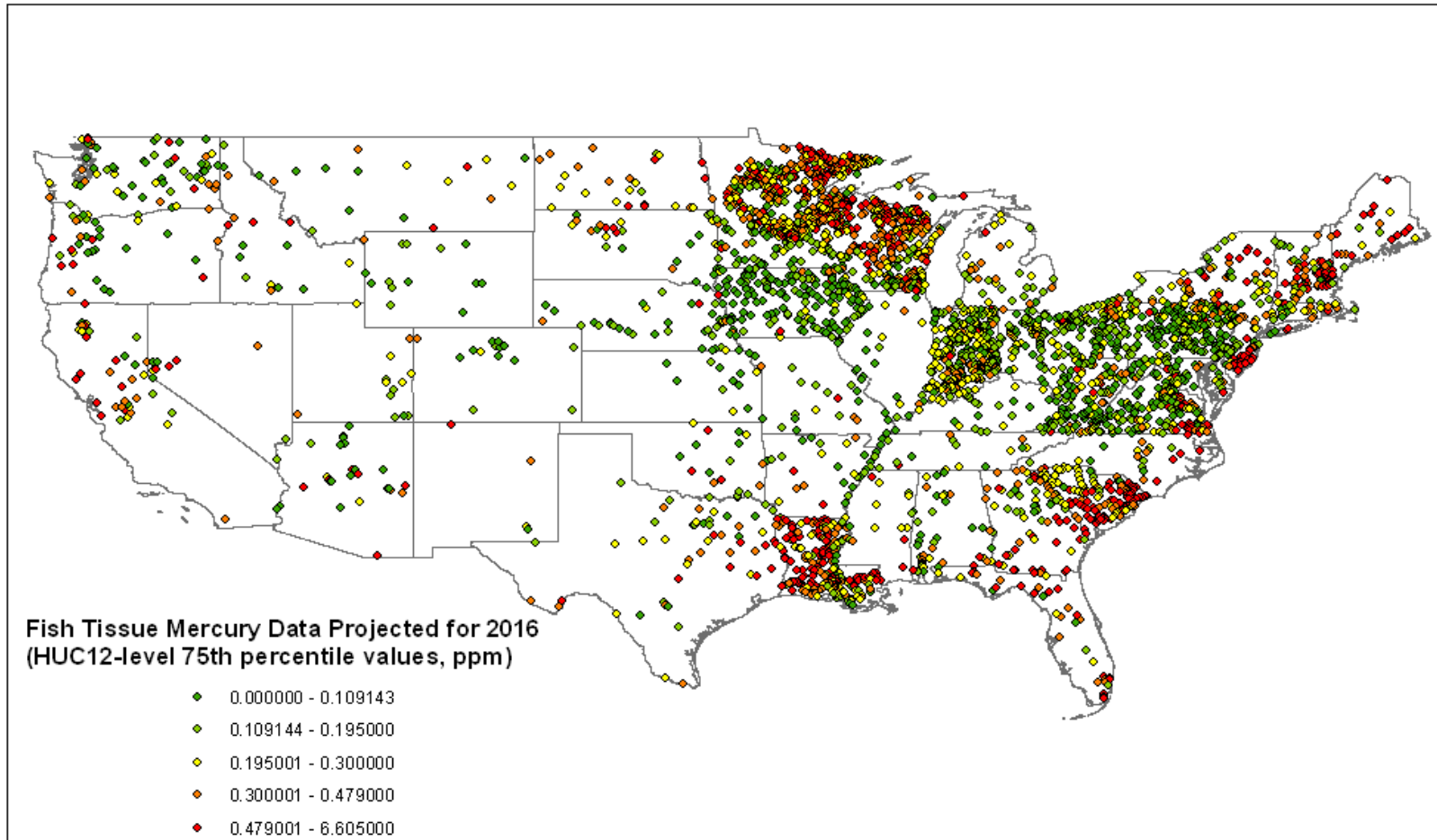
Figure 2-12 Total Fish Tissue Mercury Concentrations Projected for 2016 Scenario (HUC12-level 75th percentile values, ppm)*

* Values estimated by adjusting fish tissue Hg data presented in Figure 2-12 to reflect different patterns of Hg deposition in 2016 (relative to 2005).
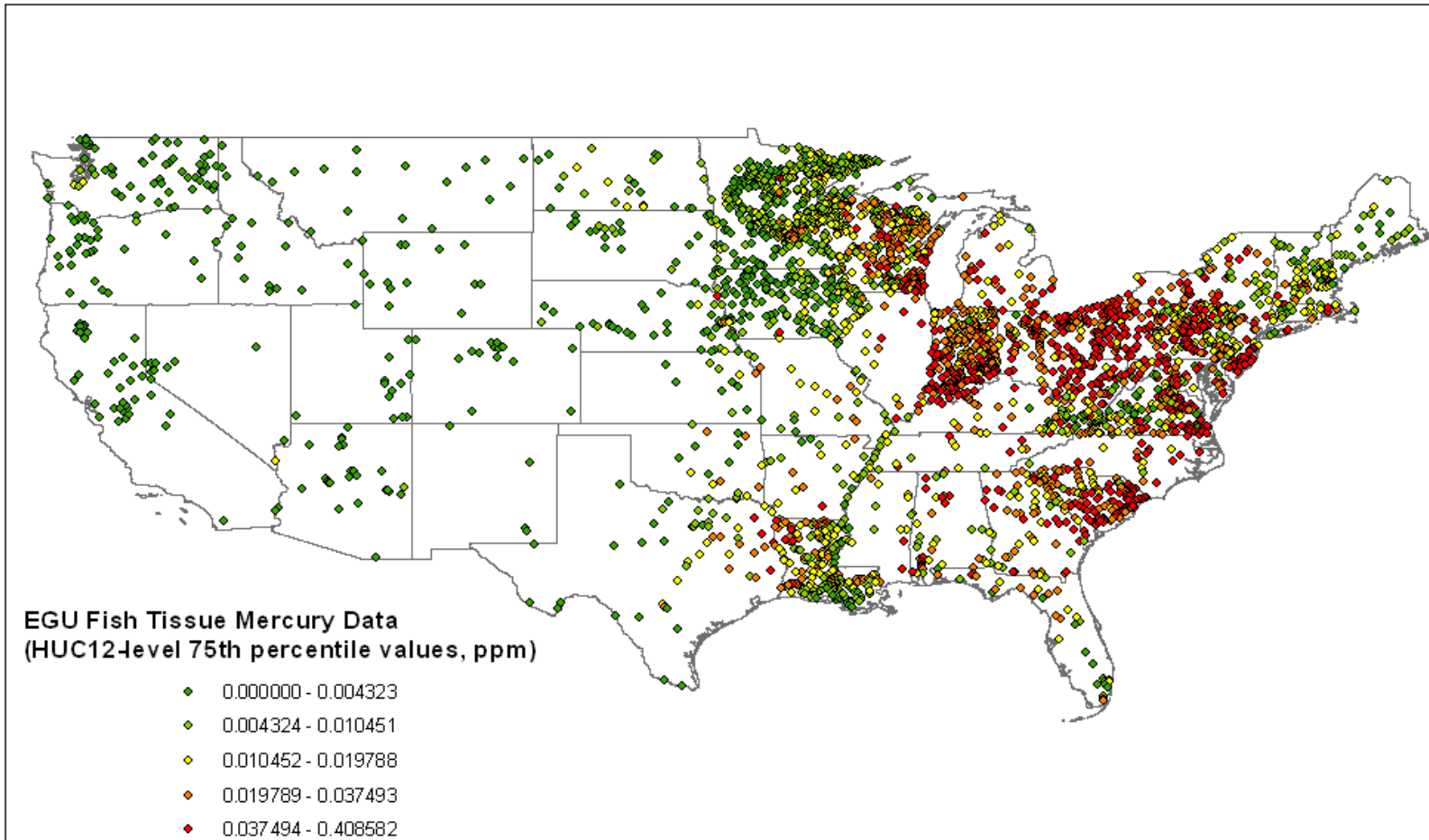
**Figure 2-13 EGU-Attributable Fish Tissue Mercury Concentrations for 2005 Scenario
(HUC12-level 75th percentile values, ppm)***

* Scales are different between the EGU-attributable and total Hg fish tissue concentrations maps.
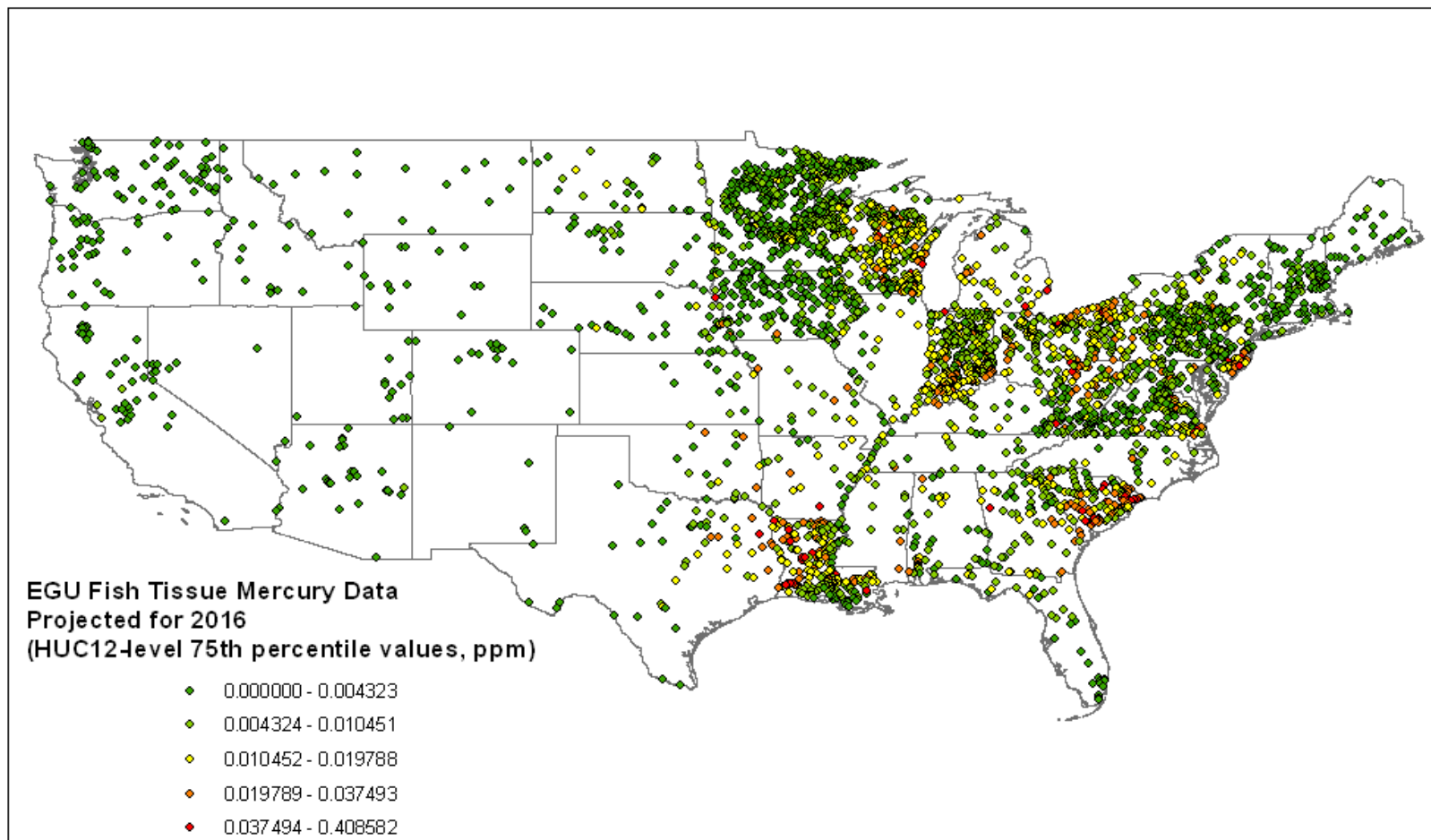
Figure 2-14 EGU-Attributable Fish Tissue Mercury Concentrations Projected for 2016 Scenario (HUC12-level 75th percentile values, ppm)*

* Scales differ between the EGU-attributable and total Hg fish tissue concentrations maps.

Table 2-4 Comparison of HUC-Level Fish Tissue Mercury Concentrations Across Datasets Used in the March Version and Current Version of the Risk Assessment

statistic	Hg fish tissue concentration (ppm) (HUC12-level 75th percentile)		
	2010 MFT dataset (used in the March version of the risk assessment) (2,317)	Augmentation dataset (collected as part of refining analysis) (940)	2011 MFT dataset (used in the current version of the risk assessment) (3,141)
Mean	0.31	0.37	0.32
Median	0.23	0.30	0.25
75th %	0.39	0.47	0.42
90th %	0.67	0.72	0.67
95th %	0.91	0.86	0.89
99th %	1.33	1.44	1.35

Table 2-5 Comparison of total and U.S. EGU-attributable Hg fish tissue concentrations (including % change) for the original fish tissue dataset and 2016 scenarios*

Statistic	Hg fish tissue concentration (ppm)						% change (2016 versus original fish tissue dataset) in Hg fish tissue concentration	
	Used for 2005 scenario			Projected for 2016 scenario				
	Total	U.S. EGU-attributable	U.S. EGU as percent of total	Total	U.S. EGU-attributable	U.S. EGU as percent of total	Total	U.S. EGU-attributable
Mean	0.32	0.024	9.1%	0.30	0.008	3.4%	-8.0%	-43.9%
Median	0.25	0.014	6.0%	0.22	0.005	2.6%	-4.9%	-61.4%
75th %	0.42	0.032	13.5%	0.40	0.011	4.4%	-12.7%	-76.0%
90th %	0.67	0.057	20.4%	0.65	0.018	6.6%	-18.6%	-82.6%
95th %	0.89	0.081	26.2%	0.86	0.026	8.7%	-23.0%	-85.4%
99th %	1.35	0.152	42.9%	1.30	0.045	16.4%	-38.3%	-91.7%

* These percentiles are not for matched HUCs (i.e., the 99th percentile for total Hg fish concentration for the 2005 scenario could occur in a different HUC than the 99th percentile U.S. EGU attributable percent reduction).

We made the following observations based on Figures 2-10 through 2-14 and in Tables 2-4 and 2-5:

- *U.S. EGU-attributable fish tissue Hg levels are higher in the eastern half of the U.S.:* This reflects primarily the fact that levels of U.S. EGU Hg deposition (that largely drives U.S. EGU-attributable Hg fish tissue levels) are much higher in the east (see Figures 2-3 and 2-4).

- *Augmenting the fish tissue Hg dataset significantly improved coverage for Wisconsin, Minnesota, Pennsylvania and New Jersey:* Although the additional fish tissue Hg data

significantly expanded coverage for watersheds in areas with elevated U.S. EGU-attributable Hg deposition (Pennsylvania) and for areas with relatively elevated fish tissue Hg concentrations (Minnesota and Wisconsin), the additional data did not substantially change the overall distribution of fish tissue Hg concentrations. This conclusion is based on a comparison of percentiles across the different datasets (i.e., 2010 MFT, augmentation, and 2011 MFT datasets) as presented in Table 2-4.

- *U.S. EGUs contribute a larger fraction to total Hg fish tissue levels in the U.S. than they do to total Hg deposition (in terms of percent), reflecting the fact that Hg fish tissue samples are focused in the east where U.S. EGU-attributable deposition is greater.* While U.S. EGUs contribute ~5% of total Hg deposition in the U.S. (for the 2005 scenario – see Table 2-2), their contribution to Hg fish tissue levels (summarized at the watershed-level) for the 2005 scenario is larger at ~9% (see Table 2-5). This reflects the fact that Hg fish tissue samples are heavily weighted in the eastern portion of the U.S. where U.S. EGU Hg deposition is typical higher than in the west. By providing greater coverage for the eastern half of the country, the Hg fish tissue sampling data generally provides greater coverage for regions with potentially greater U.S. EGU-attributable risk.

- *Relative to the combined impact of other sources, U.S. EGUs represent a smaller, but still potentially important contributor to total fish tissue Hg concentrations*: U.S. EGUs contribute ~9% of Hg fish tissue levels on average under the 2005 scenario (see Table 2-5). Under the 2016 scenario, the U.S. EGU contribution decreases to ~ 3% on average (see Table 2-5). While U.S. EGU-attributable Hg fish tissue decreases notably between the 2005 and 2016 scenarios, the impact on total Hg fish tissue levels is not that noticeable given that U.S. EGUs contribute a relatively small fraction on total Hg fish tissue levels in general (contrast the pattern of reduction seen in Figures 2-13 and 2-14 for U.S. EGU-attributable Hg fish tissue levels with the relatively smaller changes seen in Figures 2-11 and 2-12 for total Hg fish tissue levels).

- *Despite the relatively small fraction of U.S. EGU-attributable fish tissue Hg on average, for a subset of watersheds, U.S. EGU-attributable deposition has a significant contribution on fish tissue*: Under the 2005 scenario, U.S. EGUs contribute up to 43% of total Hg fish tissue levels (for the 99th percentile watershed). Under the 2016 Scenario, this pattern is reduced, but U.S. EGUs can still contribute up to 16% of total fish tissue Hg concentrations (again, for the 99th percentile watershed) (see Table 2-5).

2.4 Comparing Patterns of Hg Deposition with Hg Fish Tissue Data for the 3,141 Watersheds Included in the Risk Assessment

We also compared spatial patterns of CMAQ modeled Hg deposition and fish tissue Hg concentrations, which can inform interpretation of the risk estimates. Specifically we can consider: (a) whether the watershed-level Hg fish tissue levels are correlated with total Hg deposition, (b) how patterns of Hg deposition for the 3,141 watersheds with Hg fish tissue data compare with patterns for the 88,000 watersheds in the continental U.S. and (c) to what extent the watersheds for which we have fish tissue Hg data correspond to areas with elevated U.S.

EGU-attributable deposition. To address these questions, we developed a series of figures including:

- Figures 2-15 and 2-16: Maps comparing (a) watersheds with fish tissue Hg data with (b) the general pattern of U.S. EGU-attributable Hg deposition across the continental U.S. (for 2005 and 2016). These maps include both the fish tissue dataset used in the March TSD (2010 MFT dataset) and the augmented dataset. These maps show the degree of overlap between fish tissue Hg data and areas of elevated U.S. EGU-attributable Hg deposition (for both 2005 and 2016 scenarios).[51]

- Figure 2-17: Plot comparing the fish tissue Hg concentrations versus total Hg deposition (2005 scenario) by watershed. This plot allows consideration for whether there appears to be a correlation between these two factors at the watershed level.

- Figure 2-18: Cumulative distribution plots comparing U.S. EGU-attributable deposition for the 3,141 watersheds used in the risk assessment with U.S. EGU-attributable deposition of the 88,000 watersheds in the continental U.S. Given emphasis in the risk assessment on the 2016 scenario, we only provided plots for this scenario. These plots allow us to consider whether the watersheds with fish tissue Hg data tended to fall in regions with higher U.S. EGU-attributable Hg deposition and the degree to which this subset of watersheds provided coverage for areas with relatively elevated U.S. EGU Hg deposition across the country.

---

[51] These two maps replace Figures 2-15 and 2-16 from the March TSD. Those earlier maps also compared watersheds with fish tissue Hg data to areas of the country that had *elevated U.S. EGU-attributable Hg deposition*. However, in identifying *areas of elevated U.S. EGU-attributable Hg deposition* for those earlier maps, we attempted to be much more precise. Specifically, we used a deposition threshold for identifying areas of high U.S. EGU-attributable Hg deposition based on the average rate of deposition over watersheds modeled for risk that had U.S. EGU-attributable exposure exceeding the MeHg RfD. In other words, we identified a deposition level associated generally with watersheds having high U.S. EGU-attributable risks and used this bright line as the basis for identifying areas of high U.S. EGU deposition across the U.S.; the implication being, that these could be areas of high U.S. EGU-attributable risk. However, elevated U.S. EGU-attributable risk typically reflects both elevated U.S. EGU-attributable Hg deposition and elevated baseline fish tissue Hg concentrations. Therefore, attempting to consider only U.S. EGU-attributable deposition as a means for identifying areas of potential elevated risk is subject to uncertainty, since it does not also consider the baseline fish tissue Hg concentrations. The current set of figures seeks to support a more general comparison of the watersheds we have modeled for risk against the national pattern of U.S. EGU-attributable Hg deposition, by not attempting to identify a bright line for classifying areas of the country with regard to the magnitude of elevated U.S. EGU-attributable Hg deposition.
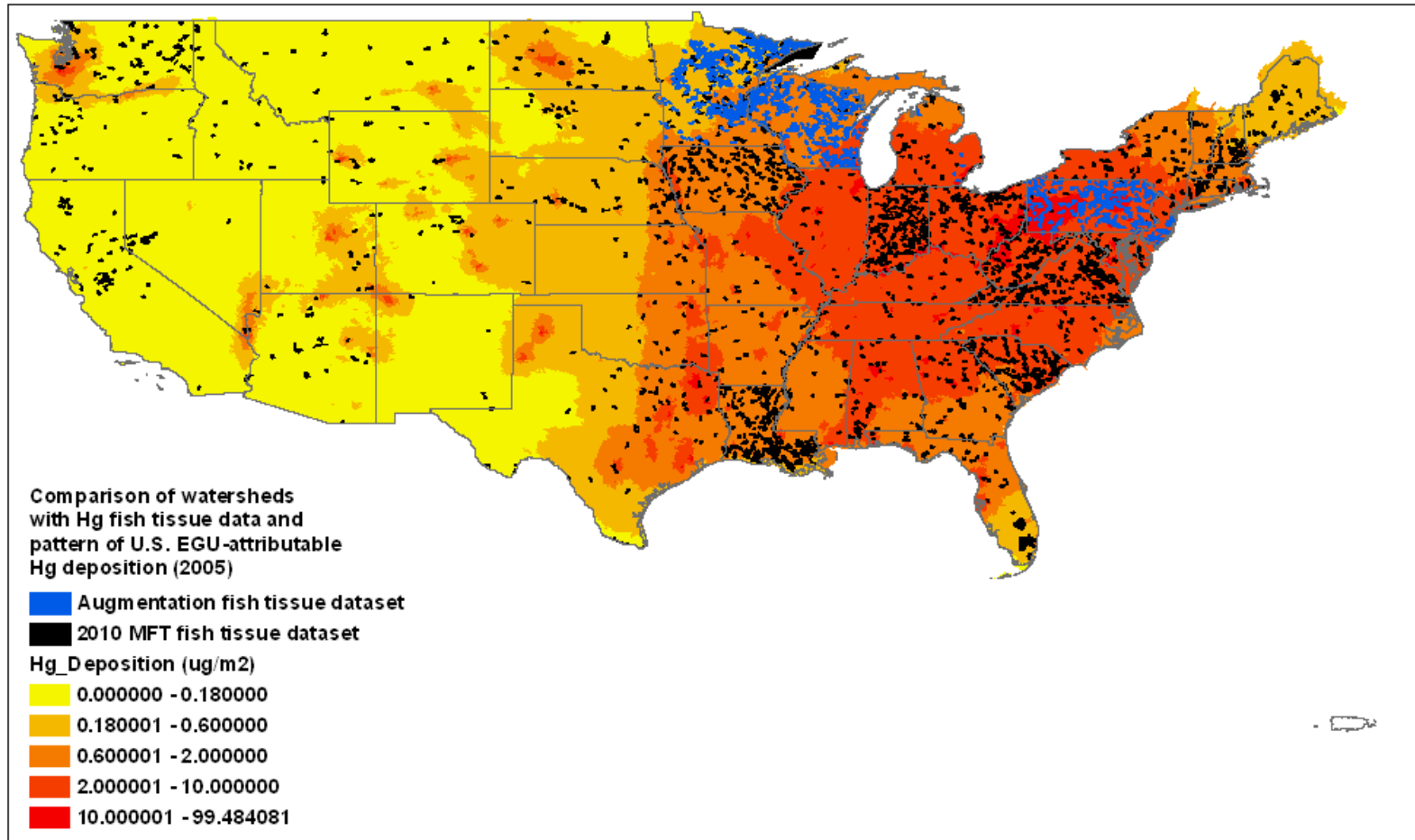
Figure 2-15 Comparison of Locations of Watersheds with Fish Tissue Hg Data* with Pattern of U.S. EGU-attributable Hg Deposition (2005 scenario)

* Fish tissue Hg data includes (a) 2010 MFT data used in March version of the risk assessment and (b) augmentation data added for this revised version.
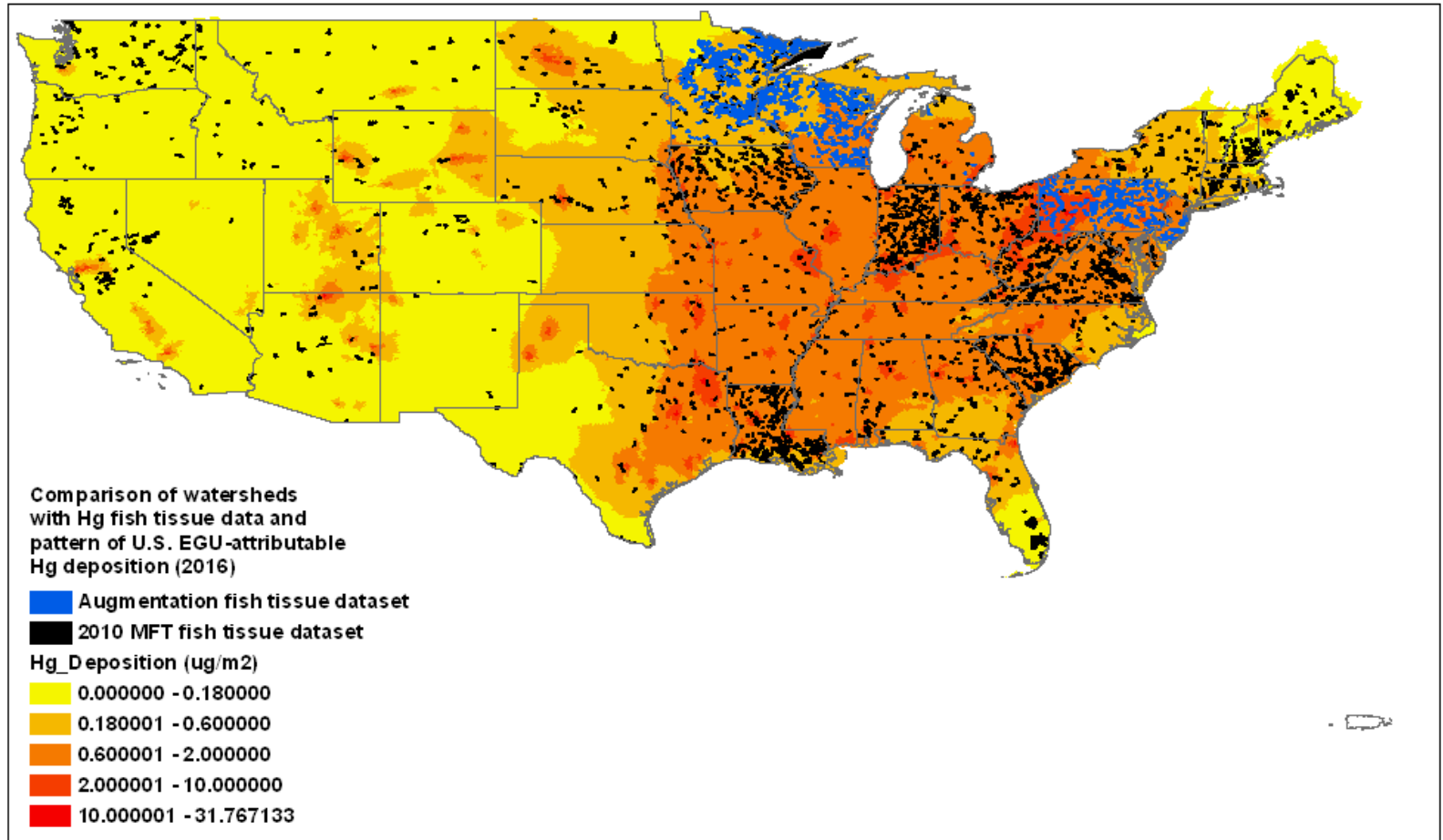
Figure 2-16 Comparison of Locations of Watersheds with Fish Tissue Hg Data* with Pattern of U.S. EGU-attributable Hg Deposition (2016 scenario)

* Fish tissue Hg data includes (a) 2010 MFT data used in March version of the risk assessment and (b) augmentation data added for this revised version.
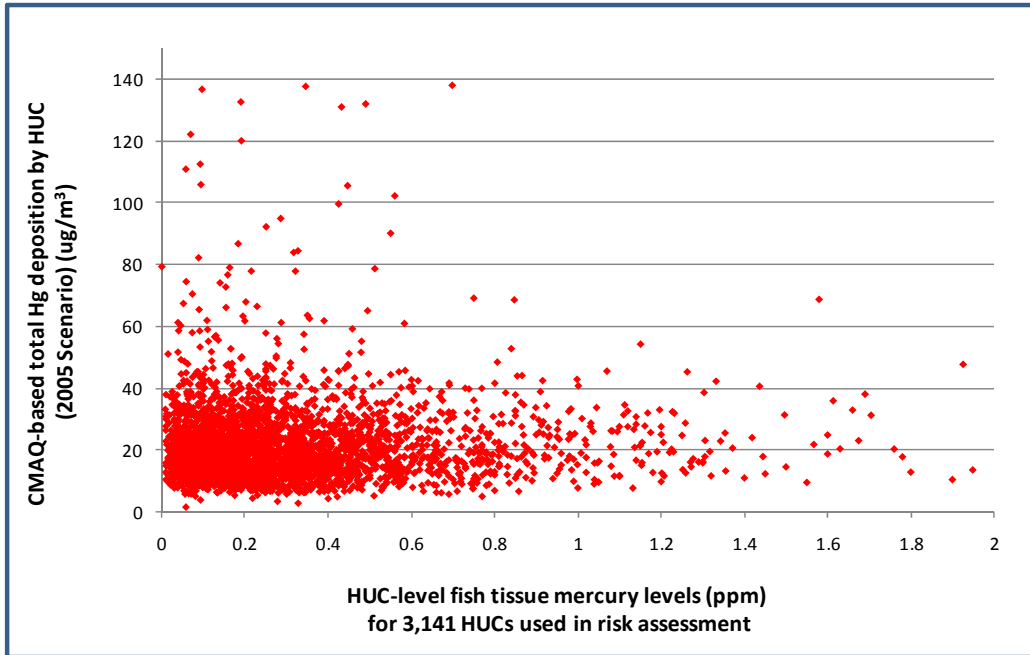
Figure 2-17 For the 2005 Scenario, Scatter Plot of Hg Fish Tissue Concentrations Versus Total Hg Deposition for the 3,141 Watersheds Included in the Risk Assessment
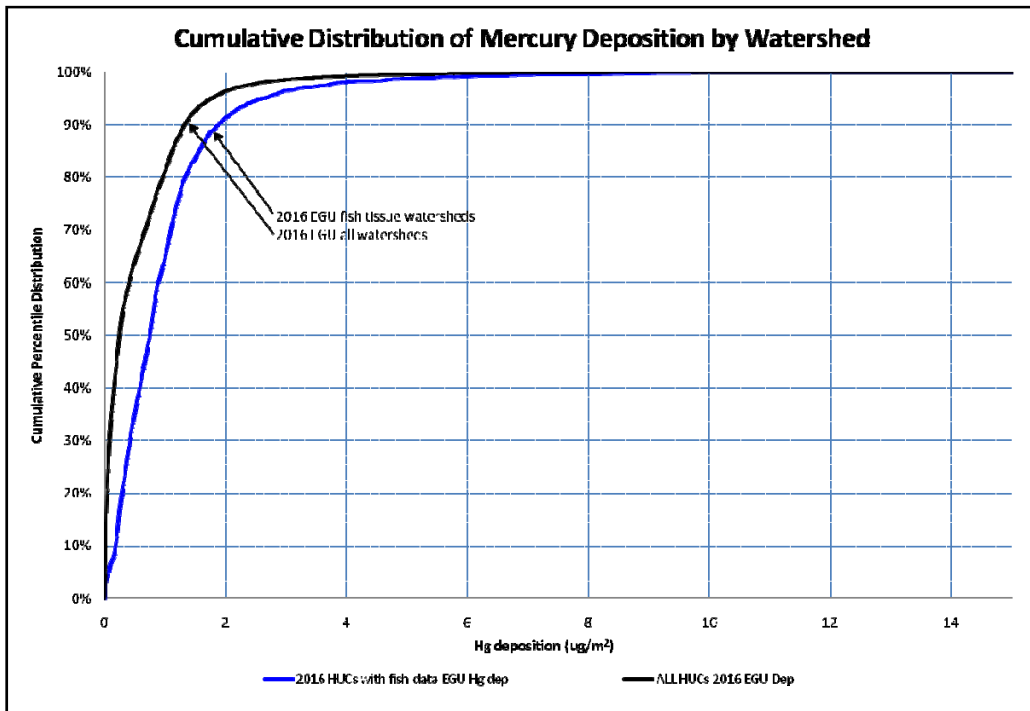
Figure 2-18 Cumulative distribution plots of U.S. EGU-attributable Hg deposition over the 3,141 watersheds used in modeling the high-end female consumer population as contrasted with all 88,000 watersheds (2016 Scenario).

We made the following observations based on the information presented in Figures 2-15 through 2-18 regarding how estimates of Hg deposition estimates relate to measured fish tissue Hg concentrations at the watershed-level:

- *The fish tissue Hg sampling data (watersheds modeled for risk) provide varying coverage for areas with elevated U.S. EGU-attributable Hg deposition. The augmented dataset significantly expanded coverage in key areas. However, the number of "at-risk" watersheds may still be substantially higher than we estimated:* As depicted in Figures 2-15 and 2-16, the 3,141 watersheds used in the risk assessment provide reasonable coverage for some of the regions having the highest U.S. EGU-attributable Hg deposition (i.e., Ohio River Valley), especially with the augmented dataset for Pennsylvania. However, even with the expanded coverage, the majority of areas with elevated U.S. EGU-attributable deposition are not covered in the risk assessment. Therefore, we believe that the number of watersheds with elevated U.S. EGU-attributable exposure and/or risk could be substantially larger (depending on the underlying fish tissue Hg concentrations).[52]

- *Hg fish tissue levels are not correlated with total Hg deposition because the relationship is highly dependent on methylation potential of individual waterbodies*: As shown in Figure 2-17, total Hg fish tissue levels at the watershed-level are not correlated with levels of total Hg deposition across watersheds (i.e., the highest total Hg deposition watersheds do not always have the highest fish tissue Hg concentrations). This is not unexpected because the relationship between total Hg deposition and total Hg fish tissue levels is highly dependent on the methylation potential at the waterbody, which is driven in part by the presence of wetlands, levels of aqueous organic carbon, Ph and sulfate deposition (see section 1.4.6).

- *Hg fish tissue samples were generally collected in regions with elevated total Hg deposition:* As demonstrated in Figure 2-18, Hg fish tissue sampling appears to have favored areas with relatively higher total Hg deposition.[53] This can be seen by comparing cumulative plots of modeled watersheds (where we have fish tissue Hg data) against plots for the 88,000 watersheds. This comparison suggests that watersheds where fish tissue Hg data were collected tended to have higher total Hg deposition than the full set of watersheds. This likely reflects to some extent, the fact that fish tissue sample are focused in the eastern U.S., which has elevated total Hg deposition compared to the broad central region (see Figures 2-2 and 2-3).

---

[52] As noted earlier, in order for a watershed to have elevated U.S. EGU-attributable risk, in the context of this risk assessment, that watershed must have high U.S. EGU-attributable Hg deposition (as a fraction of total Hg deposition) and elevated baseline fish tissue Hg concentrations.

[53] Because the risk estimates for the 2005 scenario are being de-emphasized, we decided to only provide these cumulative plots for the 2016 scenario. However, we note that the same relationship noted here (between U.S. EGU-related Hg deposition for all ~88,000 watersheds and for the subset included in the risk assessment), holds for the 2005 scenario.

2.5   Overview of Risk Estimates

This section provides an overview of risk estimates generated for the 3,141 watersheds included in the revised risk assessment. As noted earlier in Section 1.4.3, presentation of risk estimates focuses on the *typical female subsistence fish consumer* scenario assessed at the national-level, since this scenario provides the most comprehensive coverage for watersheds with Hg fish tissue data across the U.S. and because the consumption rates used to model this scenario represent subsistence levels supported by a number of studies. While this scenario is emphasized in summarizing risk estimates, we also provide risk estimates for the other scenarios (i.e., low income Blacks and Whites in the southeast, Tribal populations near the Great Lakes, Hispanics). In summarizing risk estimates, we focus on the 2016 air quality scenario, since as discussed in section 2.3, U.S. EGU-related Hg emission levels reflected in this scenario are closest to recent conditions. The remainder of this section is organized as follows:

- Overview of percentile risk estimates generated for the fisher scenarios (Section 2.5.1): We provide percentile risk estimates for the typical female subsistence fish consumer scenario at the national level and summarize percentile risk estimates for the other SES-differentiated female subsistence fish consumer scenarios assessed in the analysis. These percentile risk estimates allow us to compare risk (total and U.S. EGU-attributable) across the female subsistence fish consumers scenarios assessed in the risk assessment.

- Overview of the number (and frequency) of watersheds with populations potentially at-risk due to U.S. EGU-attributable Hg deposition (section 2.5.2): This set of risk estimates provides the main input to the risk characterization framework (see section 1.3). Specifically, watersheds with populations potentially at-risk comprise:

  o   Watersheds where total risk is considered to represent a potential public health concern and where U.S. EGUs contribute to that total risk. We considered various increments of U.S. EGU contribution including ≥ 5%, ≥10%, ≥15% and ≥20%, although, we focus on cases where U.S. EGUs contribute ≥5 % as noted in section 1.3.

  o   Watersheds where risk from U.S EGUs alone (focusing on U.S. EGU deposition and excluding other non-U.S. EGU sources) represents a potential public health concern.

To support the discussion of risk estimates, we present a series of tables summarizing those estimates and a list of observations based on this information.

2.5.1   Overview of percentile risk estimates (2016 scenario)

This section compares risk percentiles (for total and U.S. EGU-attributable risk) across the set of female subsistence fish consumer scenarios included in the risk assessment, with an emphasis on the typical female subsistence fish consumer scenario. We also compare and contrast risk for the other female consumer scenarios with the more generalized typical female subsistence fish consumer scenario.

These percentile estimates are not population-weighted, but instead represent the specific watershed-level risk estimates that fall at a specific point within the larger distribution of watershed-level risk estimates for each female subsistence fish consumer scenario. In the following tables, we sorted the total and U.S. EGU-attributable risk estimates separately for each female subsistence fish consumer scenario. This means that the total and U.S. EGU-attributable risk for a particular percentile are not matched by watershed (i.e., the 90th percentile total and U.S. EGU-attributable risk for the Vietnamese subsistence fisher likely occur at different watersheds).[54] While this risk assessment focuses on subsistence levels of fish consumption, the risk tables also include risk estimates based on mean fish consumption rates for these female subsistence fish consumer scenarios, which are relatively high compared to general recreational angler rates but do not represent true subsistence rates.

Table 2-6 provides risk percentiles for RfD-based HQs for all of the female subsistence fish consumer scenarios assessed in the analysis for the 2016 scenario.

Table 2-6 Percentile risk estimates for the full set of female subsistence fish consumer scenarios included in the analysis (2016 scenario) (for both total and U.S. EGU incremental RfD-based HQ)*

Fisher consumption rate (g/day) and percentile**	Watershed percentile risk (RfD-based HQ)*									
	Total					**U.S. EGU**				
	50th	**75th**	**90th**	**95th**	**99th**	**50th**	**75th**	**90th**	**95th**	**99th**
typical female subsistence fish consumer assessed nationally										
39 (mean)	1.9	3.5	5.6	7.4	11.2	-	0.1	0.2	0.2	0.4
123 (90th)	6.1	10.9	17.7	23.4	35.4	0.2	0.3	0.5	0.7	1.2
173 (95th)	8.5	15.3	24.9	32.9	49.7	0.2	0.4	0.7	1	1.7
373 (99th)	18.4	33	53.7	70.9	107.2	0.5	0.9	1.5	2.1	3.7
Low income White female subsistence fish consumer in the Southeast										
39 (mean)	2.1	4.1	6.8	9.3	12.5	0.1	0.1	0.2	0.3	0.6
93 (90th)	4.9	9.8	16.3	22.4	29.9	0.2	0.3	0.5	0.7	1.4
129 (95th)	6.8	13.6	22.7	31	41.5	0.2	0.4	0.8	1	2
286 (99th)	15.2	30.2	50.3	68.8	92	0.5	1	1.7	2.3	4.3
Low income Black female subsistence fish consumer in the Southeast										
171 (mean)	9.4	19.4	32.8	42.2	56.4	0.3	0.6	1	1.4	2.6
446 (90th)	24.6	50.6	85.6	110.1	147.2	0.8	1.6	2.7	3.6	6.8
557 (95th)	30.8	63.2	106.9	137.5	183.8	0.9	1.9	3.4	4.4	8.4
(99th)	NC					NC				
Low income Hispanic female subsistence fish consumer evaluated nationally										

---

[54] Presentation of non-matched percentile risk estimates represents a change from the March TSD. In that version, we used risk bands to match trends in total risk to a specific U.S. EGU-attributable risk percentile. However, given that total and U.S. EGU-attributable risk are not closely correlated, we determined that attempting to present matched percentiles was of little utility. Instead, we have decided for this version of the Mercury Risk TSD to simply provide percentile total and U.S. EGU-attributable risk estimates based on direct queries of the underlying risk distributions.

Fisher consumption rate (g/day) and percentile**	Watershed percentile risk (RfD-based HQ)*									
	Total					U.S. EGU				
	50th	75th	90th	95th	99th	50th	75th	90th	95th	99th
26 (mean)	1.2	2.2	3.5	4.9	8.3	0	0.1	0.1	0.2	0.3
98 (90th)	4.5	8.3	13.3	18.6	31.5	0.1	0.2	0.4	0.6	1.1
156 (95th)	7.2	13.2	21.2	29.6	50.1	0.2	0.4	0.7	0.9	1.7
(99th)	NC					NC				
Vietnamese female subsistence fish consumer										
27 (mean)	1.1	2	3	4.1	6.8	0	0	0.1	0.1	0.3
99 (90th)	4.1	7.2	11.1	15	24.9	0.1	0.2	0.3	0.5	1
152 (95th)	6.3	11	17.1	23	38.3	0.2	0.3	0.5	0.8	1.5
(99th)	NC					NC				
Laotians female subsistence fish consumer										
47 (mean)	1.9	3.7	5.3	5.9	7.7	0.1	0.1	0.2	0.3	0.9
145 (90th)	5.9	11.3	16.1	18	23.7	0.2	0.3	0.6	0.9	2.6
226 (95th)	10.9	20.8	29.6	33	43.4	0.3	0.5	1.2	1.7	4.9
(99th)	NC					NC				
Tribal (near Great Lakes) female subsistence fish consumer										
62 (mean)	5.4	7.7	11	13.2	17.5	0.1	0.1	0.2	0.3	0.4
136 (90th)	11.8	17	24.1	29.1	38.4	0.2	0.3	0.4	0.6	0.9
213 (95th)	18.5	26.6	37.7	45.5	60.2	0.3	0.4	0.7	0.9	1.3
493 (99th)	42.8	61.4	87.3	105.2	139.1	0.6	1	1.5	2.1	3.1

* Percentile risk estimates presented for "total" and "U.S. EGU" are not matched by watershed (e.g., the 90th percentile total and U.S. EGU-attributable risk estimates for a particular female subsistence fish consumer scenario may are unlikely to be from the same watershed).

** Means are provided along with upper-end percentile values.

"-": RfD-based HQ is ≤ 0.1.

NC: It was not possible to derive a 99th percentile consumption rate for this population due to insufficient sample size in the underlying study. Consequently, risk estimates for the 99th percentile consumption rates were not generated.

We made several observations based on the information in Table 2-6 regarding the potential health hazard associated with MeHg RfD-based HQ estimates (see Section 1.4.5).:

- *Risk estimates for the typical female subsistence fish consumer (assessed at the national-level) generally provide coverage for the Hispanic, Vietnamese and Tribal scenarios:* Risk estimates (for both total and U.S. EGU-attributable) generated for the typical female subsistence fish consumer scenario are generally higher than estimates generated for the Hispanic and Vietnamese scenarios. Risk estimates for the typical female subsistence fish consumer scenario are also higher than risks for the Tribal population for U.S. EGU-attributable risk although the Tribal populations tend to have higher total risks. Given emphasis on U.S. EGU-attributable risk in the policy context, these trends would suggest

that the typical female subsistence fish consumer scenario provides general coverage for the other three scenarios (Hispanic, Vietnamese and Tribal), since the typical female subsistence population tends to have higher U.S. EGU-attributable risk estimates.

- *Risk estimates for the typical female subsistence fish consumer (assessed at the national-level) may not provide full coverage for risks experienced by low income Blacks and low income Whites in the Southeast, or for Laotians assessed at the national-level:* U.S. EGU-attributable risk estimates for the Southeastern low income White and low income Black scenarios and for the Laotian scenario are higher than those for the typical female subsistence fish consumer. In the case of low income Whites, this likely reflects the fact that some regions in the Southeast (specifically South Carolina) have relatively elevated total and U.S. EGU-attributable fish tissue Hg concentrations compared with the national distribution (see Figures 2-13 and 2-15). In the case of low income Blacks, higher fish tissue Hg concentrations in South Carolina are combined with fish consumption rates that are substantially higher than those used for the typical female subsistence fish consumer scenario (see Table 1-6). While both the low income White and low income Black subsistence fisher scenarios in the Southeast have higher risk estimates than the typical female subsistence fish consumer scenario (assessed nationally), because the typical female subsistence scenario is assessed at all 3,141 watersheds with fish tissue Hg data, we focus on the typical female subsistence fish consumer scenario to provided policy-relevant risk information to support this rulemaking (i.e., we have greater overall confidence in risk estimates generated for the typical female subsistence fish consumer scenario because it is assessed at a larger number of watersheds with broader spatial coverage). However, the higher risks estimates for these two Southeastern subsistence fisher scenarios should also be considered in the context of interpreting the results of this risk assessment in a science-policy context. In the case of the Laotian scenario, while U.S. EGU-related risks are higher than for the typical female subsistence fish consumer scenario, this scenario was assessed for a small number of watersheds (131 of the 3,141 watersheds with fish tissue Hg data). Specifically, risk estimates generated for the Laotian scenario may reflect relatively rare localized interactions between fish tissue Hg concentrations and U.S. EGU-related Hg deposition, resulting in areas of elevated U.S. EGU-attributable exposure and risk.

2.5.2 Overview of number and percentage of watersheds with populations potentially at-risk due to U.S. EGU mercury emissions (2016 scenario)

This section discusses risk estimates based on identifying the number and percent of watersheds with fish tissue Hg samples with populations potentially at-risk due to Hg emitted from U.S. EGUs (for the 2016 scenario). As noted in Section 1.3, the "at-risk population" classification is based on identifying watersheds where: (a) U.S. EGUs contribute to total risk at watersheds where that total risk is considered to represent a potential public health hazard and/or (b) risk at the watershed-level represents a potential public health hazard from U.S. EGU-attributable deposition when considered alone, without taking into account deposition from other sources. The estimates of watersheds with at-risk populations discussed in this section are used in the 2-Stage risk characterization framework described in Section 1.3 for interpreting risk estimates. Specifically, the first category of at-risk populations described above comprise Stage 1a of the 2-Stage approach, while the second category comprises Stage 1b (see Figure 1-1). The

combination (i.e., mathematical union) of these two groups of watersheds with at-risk populations comprises the set of watersheds represented in Stage 2 of the framework.

In summarizing risk estimates in this section, we focus on estimates generated using the current risk assessment that reflects refinements to the risk model (e.g., inclusion of additional fish tissue data and evaluating the typical female subsistence fish consumer for all watersheds with fish tissue Hg data). However, when we present the Stage 2 risk estimates, we also include a summary of Stage 2 estimates from the March version of the Mercury Risk TSD (then referred to as "stage 3" results) for comparison.

With regard to the HQ estimates, any contribution of Hg from EGUs to watersheds with potential exposures exceeding the MeHg RfD represents a hazard to public health, but for purposes of this analysis, we have focused on those waterbodies where we determined EGUs contributed 5% or more to the hazard. This is a reasonable, conservative approach because risks are associated with all exposures above the MeHg RfD.

The watersheds with potentially at-risk populations are all based on the underlying risk estimates generated for the typical female subsistence fish consumer scenario. The estimates of watersheds with potentially at-risk populations are summarized in tables described below, followed by a set of observations regarding these risk estimates.

- Tables 2-7: Identifies watersheds with potentially at-risk populations based on consideration for different degrees of U.S. EGU contribution (i.e., ≥ 5, ≥ 10, ≥ 15 and $\geq 20\%$) at watersheds where total risk is considered to represent a potential public health hazard (i.e. HQ>1). For reference purposes, the table also identifies the total number of watersheds (out of the 3,141 assessed for the typical female subsistence fish consumer) with total risk exceeding the HQ threshold, regardless of the U.S. EGU percent contribution (see the "$\geq 0\%$" row of results in the table). In presenting results, the tables include both the number and percent of watersheds with fish tissue Hg samples meeting specific criteria that this represents. (Stage 1a of the 2-Stage framework)

- Table 2-8: Identifies watersheds with potentially at-risk populations based on U.S. EGU-attributable deposition considered alone, without taking into account other sources of Hg (Stage 1b of the 2-Stage framework)

- Table 2-9: Presents the union of the two categories of watersheds with potentially at-risk populations (i.e., mathematical union of the Stage 1 and 2 estimates presented in Tables 2-7 and 2-8, see Figure 1-1 for explanation). Table 2-9 considers the number and percent of watersheds that have (a) U.S. EGUs contributing to total risk of an HQ>1, OR (b) an HQ>1 based on considering U.S. EGU Hg deposition alone, without taking into account deposition from other sources (i.e., U.S. EGU-attributable HQ>1). This represents Stage 2 of the risk characterization framework.

- Table 2-10: Presents summary of the same Stage 2 risk metric as Table 2-9 with the March Mercury Risk TSD results.

Table 2-7 Watersheds with potentially at-risk populations based on consideration for various degrees of U.S. EGU contribution to total risk (2016 scenario)

EGU risk threshold	Number and percentage of HUCs meeting risk threshold criteria (RfD-based HQ > 1)*					
	2016 analysis					
	90th percentile fish consumption		95th percentile fish consumption		99th percentile fish consumption	
≥ 0%	2853	(91%)	2983	(95%)	3112	(99%)
≥ 5%	679	(22%)	726	(23%)	754	(24%)
≥ 10%	104	(3%)	114	(4%)	119	(4%)
≥ 15%	37	-	40	-	40	-
≥ 20%	15	-	17	-	17	-

* Following convention for reporting HQ estimates to one significant digit, this requires an HQ ≥ 1.5 (see section 1.4.5). Results presented here for different levels of fish consumption are each rank-ordered separately (i.e., results are not matched for the same watershed across a given row).

Table 2-8 Watersheds with potentially at-risk populations based on consideration for risk based on U.S. EGU mercury deposition and resulting exposure considered alone, without taking into account other sources of mercury deposition (2016 scenario)

EGU risk threshold	Number and percentage of 3,141 HUCs meeting risk threshold criteria (RfD-based HQ risk)*					
	2016 analysis					
	90th fish consumption		95th percentile fish consumption		99th percentile fish consumption	
≥ 1.5*	17	-	52	(2%)	327	(10%)

* Following convention for reporting HQ estimates to one significant digit, this requires an HQ ≥ 1.5 (see section 1.4.5). Results presented here for different levels of fish consumption are each rank-ordered separately (i.e., results are not matched for the same watershed across a the ">1.5" row).

Table 2-9 Combination of watersheds with potentially at-risk populations based on either consideration for (a) U.S. EGU percent contribution to total risk OR (b) risk when U.S. EGU mercury deposition is considered alone, without taking into account deposition from other sources (2016 scenario)

EGU risk threshold	Number and percentage of HUCs meeting risk threshold criteria					
	2016 analysis					
	90th percentile fish consumption		95th percentile fish consumption		99th percentile fish consumption	
U.S. EGU-attributable risk ≥1.5 HQ OR total risk ≥1.5* HQ and U.S. EGU contribution of:*						
≥ 5%	679	(22%)	744	(24%)	917	(29%)
≥ 10%	107	(3%)	141	(4%)	385	(12%)
≥ 15%	47	-	80	(3%)	339	(11%)
≥ 20%	29	-	64	(2%)	331	(11%)

*** Following convention for reporting HQ estimates to one significant digit, this requires an HQ ≥ 1.5 (see section 1.4.5). Results presented here for different levels of fish consumption are each rank-ordered separately (i.e., results are not matched for the same watershed across a given row).

Table 2-10 Reflecting the March version of the risk assessment - combination of watersheds with potentially at-risk populations based on either consideration for (a) U.S. EGU percent contribution to total risk OR (b) risk when U.S. EGU mercury deposition is considered alone, without taking into account deposition from other sources (2016 scenario)

EGU risk threshold	Number and percentage of HUCs meeting risk threshold criteria					
	90th percentile fish consumption		95th percentile fish consumption		99th percentile fish consumption	
U.S. EGU-attributable risk ≥1.5* HQ OR total risk ≥1.5* HQ and U.S. EGU contribution of:						
≥ 5%	484	(20%)	524	(22%)	672	(28%)
≥ 10%	96	(4%)	121	(5%)	325	(14%)
≥ 15%	49	(2%)	76	(3%)	292	(12%)
≥ 20%	34	-	61	(3%)	286	(12%)

* Following convention for reporting HQ estimates to one significant digit, this requires an HQ ≥ 1.5 (see section 1.4.5). Results presented here for different levels of fish consumption are each rank-ordered separately (i.e., results are not matched for the same watershed across a given row).

We made several observations regarding watersheds with potentially at-risk populations due to U.S. EGU–attributable Hg deposition based on the information in Tables 2-7 through 2-10, oriented around the 2-Stage risk characterization framework.

- *Depending on the percentile fish consumption rate, between 3 and 24% of those watersheds with total risk HQs >1 have U.S. EGUs contributing at least 5% of total Hg deposition (2016 scenario):* With this risk metric, we consider the degree to which U.S. EGUs contribute to total risk at watersheds where total risk represents a potential public health hazard (i.e., total exposure leads to an HQ > 1). Considering a 5% U.S. EGU contribution at watersheds where total risk is considered a potential public health hazard, we have up to 24% of the watersheds falling into this category (with the 24% value reflecting risk modeled using the 99th percentile fish consumption rate for the high-end female consumer – see Table 2-7). It is important when considering this risk metric to reiterate that any exposure above the MeHg RfD represents a potential public health hazard.

- *Depending on the percentile fish consumption rate, between 2 and 12% of the watersheds have HQs > 1, based on U.S. EGU Hg deposition before factoring in any other sources of Hg (i.e., an U.S. EGU-attributable HQ>1) (2016 scenario):* Our analysis suggests that between 2 and 12% of the 3,141 watersheds modeled in the risk assessment for high-end female consumers could have an HQ >1 from U.S. EGU-attributable Hg deposition when considered alone, without taking into account other sources of deposition. The low end of the range reflects the 95th percentile consumption rate, and the high end reflects the 99th percentile consumption rate (again for the typical female subsistence fish consumer).

- *Depending on the percentile fish consumption rate, between 22 and 29% of the watersheds are at-risk based on at least one of the risk characterization criteria (2016 scenario)*: Combining the two categories of watersheds with populations at-risk due to U.S. EGU Hg emissions summarized in the last two bullets, we get a total estimate ranging from 22 to 29% of watersheds at-risk. These estimates are sensitive to the

86

specification of the percent U.S. EGU contribution. If a 10 percent contribution threshold is applied, the range of watersheds at-risk ranges from 3% to 12%. The ranges for each percent contribution threshold also reflect the different fish consumption rates considered for the high-end female consumer (i.e., 90th, 95th and 99th percentile fish consumption rates). The results summarized here for total "at-risk" watersheds map to Stage 2 of the 2-stage risk characterization framework.

- *The revised risk assessment and the March Mercury Risk TSD generate very similar Stage 2 risk estimates (2016 scenario)*: Comparison of risk estimates presented in Tables 2-9 and 2-10 suggest that refinements to the risk assessment implemented since the March TSD have not substantially affected overall Stage 2 results (formerly known as "Stage 3" results in the March version of the Mercury Risk TSD).

2.6   Sensitivity Analyses

This section discusses several sensitivity analyses conducted to assess the potential impact of sources of uncertainty related to: (a) the application of the proportionality assumption in linking Hg deposition and fish tissue Hg concentrations (see sections 1.4.2.1 and 1.4.6) and (b) use of the watershed-level 75th percentile fish tissue Hg concentration as the basis for generating watershed-level risk estimates (see section 1.4.2). Given the emphasis placed in the national-scale mercury risk assessment on risk estimates generated for the 2016 scenario, we have also based the sensitivity analyses on simulating risks for the 2016 scenario.

To address uncertainty in applying the proportionality assumption, we conducted two sensitivity analyses focused on concerns that the risk assessment may have included some watersheds that are disproportionately impacted by non-air Hg sources. We included the proportionality assumption in the sensitivity analysis because it represents a critical element of the analysis and is acknowledged as representing a potentially important source of uncertainty (see Table 2-15 in section 2.7).[55] As noted in section 1.4.6, the proportionality assumption linking Hg deposition over watersheds with fish tissue Hg concentrations, only holds for watersheds where aerial Hg deposition is the primary source of loading. The two sensitivity analyses include: (a) excluding four states where we have concerns over the potential for non-air Hg playing a greater role (ME, MN, SC and LA)[56] and (b) constraining the risk analysis to only include those watersheds in the upper 25th percentile of total Hg deposition (i.e., watersheds with

---

[55] In the March TSD, we included a sensitivity analysis on the proportionality assumption for flowing versus stationary waterbodies. However, comments by the SAB suggested that this issue was unlikely to represent a significant source of uncertainty in the analysis and consequently we have not repeated it here.

[56] We excluded ME, MN and LA in this sensitivity analysis for reasons specific to each state. ME was excluded because Hg fish tissue levels there are fairly high, while Hg deposition is not relatively elevated (compared to other eastern states) – this raises the concern that other factor may affected fish tissue Hg (e.g., other non-air sources, or increased methylation potential). MN was excluded because taconite mining could contribute non-air Hg loading. Finally LA was excluded due to substantial industrial activity that could contribute non-air Hg loading. We note that, in the March TSD, we excluded SC from this sensitivity analysis because of higher fish Hg levels and Hg air deposition that (while elevated in some locations) is not uniformly higher than other states. As part of our explanation for the higher fish tissue Hg concentrations in SC we pointed to a history of gold mining in SC (i.e., the potential that significant non-air Hg loading may be a factor). SAB panel members with expertise in this area did point out that the methylation potential of many of the waterbodies in SC (particular central and eastern SC) is relatively high and that this is likely the reason for the higher fish tissue Hg concentrations.

relatively elevated levels of total Hg deposition so we have increased confidence that aerial deposition plays a significant role in loading).

To address uncertainty in use of the watershed-level 75th percentile fish tissue Hg concentration in risk modeling, we conducted a sensitivity analysis using the watershed-level median fish tissue Hg. This sensitivity analysis, which was recommended by SAB, considers the situation in which subsistence fishers do not necessarily target the larger (higher trophic level) fish and consequently could catch fish with lower fish tissue Hg concentrations.

The results of both sensitivity analyses are presented in terms of their impact on the 2-Stage Risk Characterization Framework results (i.e., their impact on estimates of the number and percent of *watersheds with potentially at-risk populations* – section 1.3). Therefore, we provide three tables (Tables 2-11, 2-12 and 2-13) that correspond to each stages of the 2-Stage Risk Characterization Framework. Each table first presents core results, followed by results for each of the three sensitivity analyses (this allows the results of each sensitivity analysis to be readily compared against the core analysis). We present observations from the sensitivity analyses at the end of this section. All results presented in the three tables are based on simulating risk for the typical female subsistence fish consumer scenario.

Table 2-11 Sensitivity analysis results presented as: *watersheds with potentially at-risk populations based on U.S. EGUs making a specified contribution to total risk (2016 scenario)*

EGU risk threshold (percent U.S. EGU contribution to risk)	Number and percentage of watersheds meeting risk threshold criteria (*Total HQ ≥ 1.5 and U.S. EGU percent contribution as dimensioned below*)*					
	90th percentile fish consumption		95th percentile fish consumption		99th percentile fish consumption	
All watersheds (core analysis)						
≥ 0%	2853	(91%)	2983	(95%)	3112	(99%)
≥ 5%	679	(22%)	726	(23%)	754	(24%)
≥ 10%	104	(3%)	114	(4%)	119	(4%)
≥ 15%	37	-	40	-	40	-
≥ 20%	15	-	17	-	17	-
Sensitivity analysis A (exclude watersheds in MN, LA, SC and ME)						
≥ 0%	2104	(89%)	2224	(94%)	2347	(99%)
≥ 5%	638	(27%)	685	(29%)	713	(30%)
≥ 10%	90	(4%)	100	(4%)	105	(4%)
≥ 15%	32	-	35	-	35	-
≥ 20%	14	-	16	-	16	-
Sensitivity analysis B (include watersheds in top 25th percentile with regard to total Hg deposition)						
≥ 0%	712	(91%)	740	(94%)	776	(99%)
≥ 5%	154	(20%)	166	(21%)	178	(23%)
≥ 10%	39	(5%)	42	(5%)	45	(6%)

EGU risk threshold (percent U.S. EGU contribution to risk)	Number and percentage of watersheds meeting risk threshold criteria (*Total HQ ≥ 1.5 and U.S. EGU percent contribution as dimensioned below*)*					
	90th percentile fish consumption		95th percentile fish consumption		99th percentile fish consumption	
≥ 15%	20	(3%)	20	(3%)	20	(3%)
≥ 20%	9	-	9	-	9	-
Sensitivity analysis C (generate risks using median watershed-level fish tissue Hg concentration)						
≥ 0%	2774	(88%)	2932	(93%)	3095	(99%)
≥ 5%	659	(21%)	717	(23%)	751	(24%)
≥ 10%	100	(3%)	111	(4%)	118	(4%)
≥ 15%	37	-	39	-	40	-
≥ 20%	15	-	17	-	17	-

* Following convention for reporting HQ estimates to one significant digit, this requires an HQ ≥ 1.5 (see section 1.4.5).

Table 2-12 Sensitivity analysis results presented as: *watersheds with potentially at-risk populations based on consideration for U.S. EGU-attributable HQ risk (2016 scenario)* *(risk considering U.S. EGU Hg deposition before considering other sources of Hg deposition)*

U.S. EGU-attributable risk threshold	Number and percentage of 3,141 watersheds meeting risk threshold criteria (*U.S. EGU-attributable HQ*)*					
	90th percentile fish consumption		95th percentile fish consumption		99th percentile fish consumption	
All watersheds (core analysis)						
≥ 1.5*	17	-	52	(2%)	327	(10%)
Sensitivity analysis A (exclude watersheds in MN, LA, SC and ME)						
≥ 1.5*	7	-	22	-	209	(9%)
Sensitivity analysis B (include watersheds in top 25th percentile with regard to total Hg deposition)						
≥ 1.5*	9	-	25	(3%)	104	(13%)
Sensitivity analysis C (generate risks using median watershed-level fish tissue Hg concentration)						
≥ 1.5*	11	-	24	-	173	(6%)

* Following convention for reporting HQ estimates to one significant digit, this requires an HQ ≥ 1.5 (see section 1.4.5).

Table 2-13 Sensitivity analysis results presented as: Combination of watersheds with potentially at-risk populations based on either consideration for (a) U.S. EGU percent contribution to total risk OR (b) risk when U.S. EGU mercury deposition is considered alone, without taking into account deposition from other sources (2016 scenario)

EGU risk threshold (percent U.S. EGU contribution to risk)	Number and percentage of watersheds meeting risk threshold criteria *(U.S. EGU-attributable risk ≥1.5* HQ OR total risk ≥1.5* HQ and U.S. EGU: percent contribution as dimensioned below)**					
	90th percentile fish consumption		95th percentile fish consumption		99th percentile fish consumption	
All watersheds (core analysis)						
≥ 5%	679	(22%)	744	(24%)	917	(29%)
≥ 10%	107	(3%)	141	(4%)	385	(12%)
≥ 15%	47	-	80	(3%)	339	(11%)
≥ 20%	29	-	64	(2%)	331	(11%)
Sensitivity analysis A (exclude watersheds in MN, LA, SC and ME)						
≥ 5%	638	(27%)	689	(29%)	782	(33%)
≥ 10%	90	(4%)	108	(5%)	267	(11%)
≥ 15%	34	-	49	(2%)	221	(9%)
≥ 20%	19	-	34	-	213	(9%)
Sensitivity analysis B (include watersheds in top 25th percentile with regard to total Hg deposition)						
≥ 5%	154	(20%)	174	(22%)	232	(30%)
≥ 10%	41	(5%)	54	(7%)	122	(16%)
≥ 15%	26	(3%)	38	(5%)	108	(14%)
≥ 20%	16	(2%)	30	(4%)	105	(13%)
Sensitivity analysis C (generate risks using median watershed-level fish tissue Hg concentration)						
≥ 5%	659	(21%)	721	(23%)	822	(26%)
≥ 10%	101	(3%)	118	(4%)	241	(8%)
≥ 15%	42	-	53	(2%)	188	(6%)
≥ 20%	23	-	36	-	179	(6%)

* Following convention for reporting HQ estimates to one significant digit, this requires an HQ ≥ 1.5 (see section 1.4.5).

Observations regarding the sensitivity analyses include:

- *Generating risk estimates excluding watersheds located in four states (AL, SC, ME and MN):* This sensitivity analysis resulted in different effects on the Stage 1a and Stage 1b risk estimates, although neither was substantially different than the core estimates. Estimates of the number of watersheds with at-risk populations due to U.S. EGUs contributing at least 5% to total risk (Stage 1a) demonstrated a range of moderate increases compared with the core analysis, depending on the "percent contribution" category considered (see Table 2-11). Similarly, estimates of the percentage of

watersheds with at-risk populations when considering U.S. EGU Hg deposition (without other sources – Stage 1b) based on dropping out the four states was 9% as compared with 10% when considering all states (for the 99th percentile fish consumption rate - see Table 2-12). This sensitivity analysis suggests that the risk estimates are relatively robust to exclusion of the four states considered in this sensitivity analysis.

- *Generating risk estimates including only those watersheds falling in the top 25th percentile with regard to total Hg deposition:* These sensitivity analysis results suggest that focusing on those watersheds with higher total deposition results in a mixture of impacts on both Stage 1a and Stage 1b risk metrics. Estimates of the number of watersheds with at-risk populations due to U.S. EGUs contributing between 5% and 10% is lower for the sensitivity analysis than for the core analysis. However, estimates for contributions of ≥ 10% and ≥20% are notably higher than for the core analysis (see Table 2-11). Similarly, estimates of the percentage of watersheds with at-risk populations when considering U.S. EGU Hg deposition alone (without taking into account deposition from other sources – Stage 1b) is 13% as compared with 10% when considering all watersheds (for the 99th percentile fish consumption rate - see Table 2-11). This sensitivity analysis suggests that the risk estimates would be somewhat higher (particularly for the tail of the risk distribution) if we focus on those watersheds receiving the greatest amount of atmospherically deposited Hg. However, the magnitude of risk is not substantially different from the core analysis.

- *Generating risk estimates using the median watershed-level fish tissue Hg concentration (instead of the 75th percentile as used in the core analysis):* These sensitivity analysis results are notably different for the two categories of risk metric considered. Estimates of the number of watersheds with at-risk populations due to U.S. EGUs contributing ≥ 5% are essentially the same as the core risk estimates. However, estimates of the percentage of watersheds with at-risk populations when considering U.S. EGU Hg deposition (without other sources – Stage 1b) is substantially lower than the core analysis (i.e., 5% when the median fish tissue Hg concentration is used as compared with 10% with the core analysis). These results suggest that for some risk metrics, the results of the analysis would be significantly affected were we to use the median fish tissue Hg concentration rather than the 75th percentile. The SAB peer-review panel concluded that "using the 75th percentile of fish tissue values as a reflection of consumption of larger, but not the largest, fish among sport and subsistence fishers is a reasonable approach and is consistent with published and unpublished data on predominant types of fish consumed."

2.7 Discussion of key sources of variability and uncertainty

This section provides a qualitative discussion of variability and uncertainty associated with the risk assessment. Regarding variability, we focus on identifying the key sources of variability associated with modeling risk for the scenarios included in the analysis and then discuss the degree to which those sources are reflected in the design of the risk assessment and implications for the risk estimates generated. The risk assessment has been designed to reflect critical sources of variability to the extent allowed by available methods and data and given the resources and time available. Key sources of variability associated with the analysis include:

91

- The pattern of total and U.S. EGU-attributable Hg deposition across watersheds in the U.S. (including variation in the global inflow of Hg into the U.S.)
- The patterns of fish tissue Hg concentrations across watersheds within the U.S.
- The sampling protocols for measuring fish tissue Hg concentrations used by States which results in varying degrees of spatial coverage of fish tissue Hg concentrations across the states.
- The response of watersheds to Hg loading including variation in the temporal response and degree to which fish tissue Hg concentrations change in response to altered Hg loadings.
- The degree of subsistence fishing activity by different groups across watersheds.
- Subsistence fishing behavior including: consumption rates, species harvested, degree to which activity is focused on one or more waterbodies and differences in cooking/preparation practices. Variation in bodyweight can also impact exposure and risk estimates.

These sources of variability and the degree to which each is reflected in the design of the analysis are identified and described in Table 2-14.

Regarding uncertainty, we focus first on identifying potentially significant sources of uncertainty impacting the analysis. Then we characterize (a) the nature of the impact of each source on risk estimates and (b) the degree to which the potential impact of the source of uncertainty is characterized as part of the analysis (including whether sensitivity analyses completed for the risk assessment address a particular source of uncertainty). The results of this qualitative assessment are presented in Table 2-15.

The SAB commented extensively on the sources of variability and uncertainty that should be addressed in the revised version of this section and those recommendations are reflected in Tables 2-14 and 2-15.

Table 2-14 Key sources of variability associated with the analysis and degree to which they are reflected in the design of the analysis

Source of variability	Description	Degree to which source is reflected in design of risk analysis and implications for the risk estimates generated
(A) 2005 Hg emissions	Emissions approaches have been applied to create a 2005 Hg inventory for separate categories of emissions (e.g., Coal utilities, Portland Cement, Electric Arc Furnaces). Within each category, types of variability for total Hg include (1) annual variability related to product demand, (2) sub-annual (e.g., hourly) temporal variability from production related fluctuations at each facility, (3) variability of emissions control systems over time depending on the control device age and process stages such as startup/shutdown and operating conditions even during standard operations, (4) fuel characteristics including the Hg content of the fuel, and (5) differences in similar pieces of equipment that are relevant when emission factors are applied to a class of process (e.g., not all fluidized bed boilers will have the same emissions potential). Further, the speciation characteristics of the total Hg has variability based on many of the same factors as for total Hg (e.g., production parameters, control systems, fuels, physical properties of individual units)	The analysis accounts for annual variability in that estimates of Hg emissions were developed for the particular 2005 year as the midpoint of the fish tissue data projected to 2016 to estimate risks in the future baseline. The analysis attempted to estimate sub-annual variability through use of (a) hourly EGU demand data that is developed from several years of hourly Continuous Emissions Monitoring (CEM) heat input data and (b) monthly, day-of-week, and diurnal temporal allocation factor for non-EGU sources. The approach for estimating 2005 EGU emissions was tied to 1999 control assumptions and fuels, which creates some uncertainty described in Table 2-15. The variability associated with different pieces of equipment in a class of process was not accounted for explicitly, but the Hg emission factors developed for EGU boilers and other unit types were developed to represent, on average, emissions from a given unit type by averaging emissions test data over a many units at facilities within a given class. The emission factors used to compute Hg for U.S. EGUs were assigned based on boiler type, fuel (coal or oil), and control device configuration. The same characteristics were used to assign speciation fractions.
(B) 2016 Hg emissions	The sources of variability identified for the 2005 emissions also apply to 2016; however, they are addressed differently because of the differences in approach associated with estimating emissions for a future year.	Annual variability is accounted for by the Integrated Planning Model's 2016 estimate of Hg emissions. This model accounts for expected electricity demand, controls in place before 2016, and forecasts of fuel prices resulting in predicted choices of fuels. Further, the model predicts the Hg content of the fuel used in each unit in the future based on forecasts of which coal mine will supply coal to each unit. Thus, IPM does a more complete job than is done in 2005 for including control and fuel information. As in 2005, sub-annual variability uses a) hourly EGU demand data that is developed from several years of hourly Continuous Emissions Monitoring (CEM) heat input data and (b) monthly, day-of-week, and diurnal temporal allocation factor for non-EGU sources. IPM further accounts for differences in equipment using Emissions Modification Factors (EMFs), which adjust the Hg emissions based on the boiler type and control device configuration. The EMFs are not specific to each unit, but rather were developed to represent, on average, emissions changes due to boiler and control device types. Finally, the speciation fractions used in 2005 were also applied in 2016, but the fractions were assigned based on the 2016 unit type, fuel type, and control devices, rather than the base year configuration.
(C) Pattern of total and U.S. EGU-attributable Hg deposition across	Patterns of annual deposition of Hg including total (all source) and estimates of the U.S. EGU fraction (by watershed) displays considerable spatial variability across	By extrapolating CMAQ grid cell results at the more spatially refined HUC12 watershed level, we retain the greater degree of spatial resolution in characterizing Hg deposition obtained through the use of the 12k CMAQ grid cell simulations.

Source of variability	Description	Degree to which source is reflected in design of risk analysis and implications for the risk estimates generated
watersheds in the US (including variation in the global inflow of Hg into the U.S.)	the U.S. based on the results of 12 km grid cell CMAQ modeling (see Section 2.4). This variability includes changes over time in global-scale ambient mercury concentrations due to changes in global emissions and chemistry.	Regarding variation in the global inflow of Hg, the global GEOS-CHEM simulation does provide ambient Hg inflow estimates used in the CMAQ model domain that vary spatially in the horizontal direction and vertically from the surface through the troposphere. The Hg inflow concentrations also vary for each day of the year and within each day. However, the ambient speciated Hg inflow is kept constant between 2005 and 2016 scenarios.
(D) Patterns of fish tissue Hg concentrations across watersheds within the U.S.	Mercury fish tissue measurements can display considerable spatial variability across watersheds (see section 2.3).	We have fish tissue measurements for roughly 4% of the watersheds in the U.S. (i.e., 3,141 watersheds with measured values out of 88,000 based on data from 2000-2010). These measurements are concentrated in the Eastern part of the U.S., although there are some measurements in almost all states. While our measured fish tissue levels do generally provide some degree of coverage for areas with elevated Hg deposition (in terms of both total and U.S. EGU-attributable), this coverage is limited and there are a large number of watersheds with high total and U.S. EGU-attributable Hg deposition for which we do not have fish tissue measurements (see section 2.4).
(E) Sampling protocols for measuring fish tissue Hg concentrations used by States differ, which results in varying degrees of spatial coverage of fish tissue Hg concentrations across the states.	States utilize varied approaches for targeting waterbodies for fish sampling and Hg measurement. In virtually all cases, sampling strategies are not intended to provide a representative characterization of fish tissue Hg concentrations across the state and instead are intended to identify and then target areas suspected of having higher Hg levels, or higher fishing activity.	The sampling protocol used by states in collecting fish tissue Hg measurements is directly reflected in the fish tissue dataset used in the risk assessment. Therefore, any potential bias in the collection of fish tissue data (i.e., tendency to capture more highly impacted waterbodies) is reflected in that dataset. However, having fish tissue Hg data that are potentially biased towards more heavily Hg-impacted waterbodies is not problematic given the focus of the risk assessment. Specifically, given that we are attempting to capture reasonable high-end subsistence fisher risk, having fish tissue data biased towards more highly impacted waterbodies is actually preferable, since it increase the potential that we will identify areas of higher exposure and risk related to the subsistence fisher scenario (it would only be problematic if we were attempting to generate a representative picture of more generalized recreational fisher risk).
(F) Response of watersheds to Hg loading including variation in the temporal response and degree to which fish tissue Hg concentrations change in response to altered Hg loadings.	The response of fish tissue Hg concentrations to mercury deposition can vary greatly depending on a number of factors (e.g., role of watershed in loading to waterbody, methylation potential of the waterbody, rates of sulfate deposition, nature of aquatic biotic foodweb including mix of upper-level trophic fish etc). These factors can also affect the temporal profile of that response.	Variation in methylation potential is reflected directly in the variation in the measured Hg levels in fish across the different sampling locations. This variation is very important, and in fact is a primary determinant of variation in fish tissue Hg concentrations across watersheds. This will have a large impact on the variability in total Hg exposure, and as shown in Figure 2-17, results in a low correlation between total Hg deposition and fish tissue Hg across watersheds. However, this variability will not have an impact on the attribution of exposure at any specific watershed, because the scientific literature supports a linear relationship between changes in deposition of Hg at a watershed and changes in fish tissue Hg at the same watershed. Furthermore, because we are not predicting temporal trends in fish tissue levels and instead consider a future point in time (once near-steady state conditions are reached), variation in the temporal profile of changes in fish tissue levels related to differences in methylation potential of different watersheds is also minimized as a factor in the analysis.
(G) Differences in the spatial distribution and	Studies reviewed in developing the approach for this analysis suggests that there can be considerable variation	Surveys of high- self-caught fish consuming populations allow us to clearly define this type of activity for specific areas covered by those surveys (e.g., Hispanic,

Source of variability	Description	Degree to which source is reflected in design of risk analysis and implications for the risk estimates generated
extent of subsistence fishing activity by different groups across watersheds in the U.S.	in the nature of high-end self-caught fisher populations across regions of the country. This variation reflects ethnic and cultural practices and can also be driven be socio-economic status (SES) related factors (e.g., poverty, levels of income). In addition, we would expect that access to freshwater vs saltwater fishing locations would also play a role.	Vietnamese and Laotian fishing activity in specific regions of California, high-end fishing activity by Blacks and Whites in South Carolina and Tribal activity near the Great Lakes). However, available studies on this type of high-end fishing activity at inland freshwater bodies do not provide comprehensive coverage for all regions in the U.S. Therefore, we extrapolated coverage from areas reflected in these studies to other portions of the U.S. There is uncertainty associated with this process. As discussed in Table 2-15 (entry E) a lack of comprehensive survey information on subsistence fishing activity for all regions in the U.S. is a limitation of the analysis. This also prevents us from generating population-weighted estimates of risk.
(H) Subsistence fishing behavior including: consumption rates, species harvested, degree to which activity is focused on one or more waterbodies and differences in cooking/preparation practices. Variation in bodyweight can also impact exposure and risk estimates.	Subsistence fisher populations can display significant variability in their fishing behavior, which can impact overall exposure and risk. Variation in consumption rates has a direct impact on exposure, as do differences in the types of fish (size and species) potentially targeted. Tendency to focus fishing activity in one spot, versus distributing it across different waterbodies can also impact risk. Specifically, activity distributed across multiple waterbodies will tend to dilute out locations with higher fish tissue Hg concentrations, while focused activity means that one or more fishers may get all of their fish from the more impacted lake. Differences in cooking/preparation can also impact Hg levels on a per unit of fish basis, which, when combined with "as cooked" consumption rates can effected modeled exposure and risk. Body weight will also affect the calculation of exposure, when standardized by body weight (e.g., a heavier individual ingesting the same amount of fish will have a lower body-weighted adjusted exposure than a lighter individual ingesting the same amount of fish).	While we have fairly comprehensive information on consumption rates for the set of female subsistence fish consumer scenarios we included in the analysis, we do not have information (for those fishers) characterizing several of the other behavior-related factors including types of species and size of fish targeted and the degree to which fishers target a single waterbody, versus distribute their activity across multiple waterbodies. However, given the focus of this assessment on capturing reasonable high-end fish consumption by subsistence fishers, we have modeled these scenarios assuming that (a) they tend to favor somewhat larger fish (use of the 75th percentile watershed-level fish tissue Hg concentration) and (b) they focus their fishing activity on a single watershed. While we do not know how representative these types of higher-exposure related behaviors are within the subsistence fisher populations covered in the risk assessment, we do believe it is reasonable to assume that some subset of each population could engage in this type of behavior. With regard to fish cooking/preparation, we have not considered variability in this behavior, although the factors used (1.5 – see section 1.4.4), is a value that reflects the range of adjustment factors seen with different types of fish preparation methods for different types of fish. Similarly for body weight, we have used a central tendency factor and not explicitly modeled variability in this factor.

Table 2-15 Key sources of uncertainty associated with the analysis, the nature of their potential impact on risk estimates, and degree to which they are characterized as part of the analysis

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
Characterizing the pattern of fish tissue Hg concentrations across watersheds in the U.S. (reflects small fraction of total watersheds with fish tissue measurement data, differences in state-level protocols for collection of fish tissue data, substantial number of watersheds with relatively low sample sizes (e.g., 1-2 samples), uncertainty associated with filtering watersheds to exclude locations with potentially significant non-air Hg impacts)			
(A) Relatively small fraction of U.S. watersheds with measured fish tissue Hg data	Fish tissue Hg measurements are available for 3,141 of the ~88,000 watersheds in the U.S. (i.e., ~4% of the watersheds). This relatively low coverage raises concerns that the risk assessment may miss areas of elevated U.S. EGU Hg deposition and/or areas with elevated fish tissue Hg concentrations, both of which could result in a low-biased assessment of U.S. EGU-related risk.	Supplemental data for Pennsylvania that has been incorporated into the fish tissue dataset to enhance the current version of the risk assessment reduces somewhat concerns over missing areas of high U.S. EGU deposition (Pennsylvania has high U.S. EGU deposition and in the March version of the risk assessment had very little coverage by fish tissue Hg data). Similarly, additional data for Wisconsin and Minnesota reduces concerns over missing areas with elevated fish tissue Hg concentrations. However, there is still the potential that areas of high U.S. EGU Hg deposition and/or areas with high fish tissue Hg concentrations have been excluded from the analysis. In addition, there is still an overall low bias in our estimates of the number of watersheds with specified levels of risk since only a fraction of watersheds have fish tissue Hg measurements.	We have semi-quantitatively examined the issue of potentially missing Hg deposition hot spots by presenting Figures 2-15 and 2-16, which show coverage by fish tissue Hg data for areas of the country with elevated U.S. EGU Hg deposition. Examination of these maps suggests, for example, that while Pennsylvania is now fairly well covered by available fish tissue data, Illinois, Kentucky and Ohio are less well covered. It is also important to point out that even while general coverage for high U.S. EGU Hg deposition areas by measured fish tissue data has improved for this version of the risk assessment, the potential still exists that substantial numbers of high-impact watersheds have not been included in the risk assessment due to a lack of fish tissue sampling at those locations.
(B) Differences in state-level protocols for collection of fish tissue data which results in differing degrees of spatial coverage across states and differing degrees of potential bias in the samples collected	States use different strategies for determining where fish tissue samples will be taken geographically as well as the types of fish that will be measured (and the methods of collection). Generally, however, there is likely to be a bias towards targeting waterbodies suspected of having elevated fish tissue Hg concentrations for most states. Differences in the strategies used to collect fish tissue data together with the potential for bias favoring more highly impacted waterbodies, introduces uncertainty into the risk assessment. Specifically, there is	Because the goal of the risk assessment is to capture risk for subsistence fishers that are likely to experience elevated U.S. EGU-attributable risk, having fish tissue Hg concentrations that likely reflect targeting of more highly impacted waterbodies (i.e., biased towards higher risk locations) is not problematic and in fact, is preferable. If it is reasonable to assume that subsistence fisher activity could occur at these sampled watersheds that have relatively higher fish tissue Hg concentrations, then having risk estimates based on these measurements strengthens the analysis from a science policy standpoint. By contrast, if the analysis had been designed to generate a fully representative population-weighted assessment of subsistence fisher risk, then bias in the fish tissue Hg concentrations would be problematic (since it would high-bias an assessment targeted at capturing "typical" risk for these high-consumers).	Given that potential high-bias in the characterization of fish tissue Hg concentrations across the watersheds included in this risk assessment is not problematic and may in fact be preferable, we did not attempt to quantify the impact of this factor on risk estimates.

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
	likely to be varying degrees of high-bias in the characterization of fish tissue Hg concentrations across states.		
(C) Substantial number of watersheds with relatively low sample sizes (e.g., 1-2 samples)	Only 28% of the watersheds with fish tissue Hg data have at least 10 samples (~41% of the watersheds have only 1 or 2 samples). Generating 75th percentile risk estimates for watersheds with a small number of samples (e.g., <10 to 20) is subject to uncertainty and will on average understate the true 75th percentiles across watersheds, although the bias in the estimate of the 75th percentile for any specific watershed is unknown.	The potential that a substantial fraction of the 3,141 watersheds included in the risk assessment could have low bias in the 75th percentile fish tissue Hg concentrations used in modeling risk means that in turn, risk estimates for a substantial number of watersheds could be low-biased. For the very low samples sizes (e.g., 1-2 samples per watershed), it is likely that, as a trend, the estimates of the 75th percentile values are actually more likely representative of the central tendency, e.g. the median.	We examined trends in the watershed-level 75th percentile estimates across different strata of watersheds, where those strata were based on sample size (see Table 1-2). As discussed in section 1.4.2, the average of the 75th percentile estimates increases substantially across these strata (i.e., as the number of samples available to calculated a 75th percentile increases, the trend in 75th percentiles also increases). This suggests that other factors equal (see below), there is a distinct potential for low bias in the 75th percentile fish tissue Hg levels used in risk characterization for those watersheds with lower sample sizes. The important caveat to this is that this observation assumes that there is no correlation between absolute level of fish tissue Hg concentrations and sample size across watersheds. This may not be the case if, for example, waterbodies with higher absolute fish tissue Hg concentrations are also targeted more for sampling (which is likely the case). Therefore, the potential for low-bias in the 75th percentile estimates for watersheds with 1-2 samples probably needs to be softened slightly since there is the potential for these watersheds to have lower "actual" measured fish tissue Hg levels.
(D) Filtering watersheds to exclude locations with potentially significant non-air Hg impacts	As described in section 1.4.2, we filtered watersheds (with fish tissue Hg data) to exclude those potentially impacted by non-air Hg sources including gold mines and any industrial sources meeting a specified Hg release threshold. However, we did not filter out watersheds located near large urban areas which have the potential to release Hg through waste water treatments effluent. Recall that the proportionality assumption used as the basis for generating U.S. EGU-relevant exposure and risk does not hold in	In those instances where risk is modeled for a watershed assuming that air Hg deposition is dominant, when in reality, municipal wastewater discharge may be contributing significantly to Hg loading, then estimates of U.S. EGU-attributable risk for that watershed could be biased high. In this case, we would not have assigned a portion of risk to the municipal discharges and instead would have over-estimated the U.S. EGU-attributable fraction of risk.	Although we did not explicitly consider the issue of urban wastewater Hg loading in our sensitivity analyses, two of the sensitivity analyses did examine the broader issue of uncertainty in filtering our watersheds to exclude areas with substantial non-air Hg loading (se section 2.6). These two sensitivity analyses suggested that the risk estimates generated in the core analysis are relatively robust even when we (a) exclude a set of states that may have substantial non-air Hg loadings across their watersheds (or have higher methylation potentials in their waterbodies) or (b) focus risk modeling on those watershed in the upper quartile with regard total Hg loading (and therefore are more likely to be the dominant source of Hg to a watershed).

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
	instances where non-air Hg loading is substantial.		

Characterizing subsistence fishing activity across areas of high U.S. EGU mercury deposition (includes assessing potential locations for subsistence fishing activity as well as details regarding actual fishing and fish consumption behavior)			
(E) Predicting which watersheds are likely to experience subsistence fishing activity	There is uncertainty associated with estimating which of the watersheds for which we have fish tissue Hg data are likely to experience subsistence fishing activity associated with the female subsistence fish consumer scenarios considered in the analysis. For the typical female subsistence fish consumer scenario, we assumed that activity could occur at any of the watersheds with fish tissue data. However, for the remainder of the female subsistence fish consumer scenarios, we used the concept of "source population" (see section 1.4.3) to guide identification of watersheds with the potential for subsistence fisher activity by these specific SES-differentiated groups of fishers.	Because the goal of the analysis is to determine whether there is the potential for significant risk for female subsistence fish consumers and not to generate representative population-weighted risk distributions for those populations, the importance of rigorously assessing where these populations are active (and the numbers of fishers at each location) is reduced somewhat. However, bias could be introduced into the analysis if we have modeled risk for high-impact watersheds when in reality there is no likelihood of subsistence fishing activity at those locations. The potential impact of this source of uncertainty is difficult to assess. However, as discussed above, because we are not attempting to generate population-weighted risk distributions, it is not expected to substantially impact the risk assessment.	We did not explicitly examine this source of uncertainty related to where specific subsistence fisher populations may be active.
(F) Focus on risks to subsistence fishing populations and associated female subsistence fish consumers (concern that analysis does not cover high-end recreational angler risk and risk by other potentially high-consuming groups)	The SAB raised concerns that by focusing on subsistence fishers, we may have not provided coverage in the risk assessment for other high-consuming populations including the subset of recreational anglers who frequently fish and consume the fish they catch, and thus have near subsistence level consumption rates.	The typical female subsistence fish consumer scenario is actually characterized (in terms of consumption rates) using a survey of recreational anglers conducted in South Carolina (Burger et al., 2002). This population is characterized as "subsistence" for purposes of this analysis due to the magnitude of the upper percentile consumption rates used (i.e., the 90th to 99th percentile values) which are considered subsistence since they represent a substantial contribution to dietary intake of protein. Furthermore, given that in this revised analysis, the typical female subsistence fish consumer scenario was assessed at all watersheds with fish tissue Hg data (excluding those filtered due to high-non air Hg impacts), this scenario would provide complete coverage for high-end recreational anglers, both in terms of consumption rate and potential location of	We did not examine this issue of providing coverage for high-end recreational anglers because, as discussed in the cell to the left, the typical female subsistence fish consumer scenario provides coverage for a high-end recreational angler in the context of this assessment (i.e., given the goal of assessing subsistence-level fish consumption at each watershed without population weighting).

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
		activity. There is no systematic bias introduced into the analysis due to its focus on female subsistence fish consumer scenarios that is consistent with the overall goal of the analysis (i.e., to characterize risk for the most highly impacted fisher populations).	
(G) Characterizing subsistence fisher behavior (including consumption rates, types/size of fish targeted and the degree to which activity is focused at specific waterbodies)	There is uncertainty associated with characterizing subsistence fisher behavior including consumption rates, types and size of fish targeted and the degree to which fishers target specific watersheds rather than distribute their activity across multiple watersheds.	There are relatively comprehensive survey data available for characterizing fish consumption rates for the subsistence scenarios included in the analysis. In fact, the subsistence fisher scenarios included in the analysis were selected, in large part, based on the availability of survey data to characterize consumption rates. In addition, the consumption rates display a reasonable degree of consistency in terms of magnitude across the studies (see Table 1-6). Therefore, we believe that there is relatively low uncertainty associated with characterizing this aspect of subsistence fisher behavior and little concern for the introduction of bias into the analysis.[57] However, the other critical aspects (degree to which fishers target specific sizes and species of fish and the degree to which their activity is focused at one or more watersheds) are not well characterized in the literature and therefore are subject to considerable uncertainty. However, because the analysis is focused on modeling reasonable high-end risk, the importance of uncertainty associated with these additional behavior-related factors is reduced substantially. Specifically, if it is reasonable to assume that a subset of subsistence fishers would	Given that the focus of this analysis is on assessing risk for populations likely to experience the highest reasonable U.S. EGU-attributable risk, we concluded that because it is reasonable to assume that some fraction of high-end fishing populations could focus their activity on a single watershed (and could favor larger fish), we can model this behavior in our analysis without introducing substantial uncertainty. If these assumptions regarding behavior are relaxed (i.e., fishers were assumed to eat smaller fish and distribute their activity across multiple watersheds), then risk will be reduced.* However, we did not explicitly model these alternative behavioral profiles given our focus on capturing reasonable estimates of high-end risk. * The observation that distributing fishing activity between watersheds will reduce risk needs additional clarification. While distributing fishing activity between watersheds may or may not impact central tendency estimates of risk across a group of fishers, high-end risk (risk for individuals at the upper tail of a simulated distribution) will be lower, since instances of having an individual fish at a single high Hg watershed will be removed, with fishing activity at that high Hg watershed now being averaged with activity at other less-impacted watersheds.

[57] Concerns have been raised by the SAB that some of the studies providing the consumption rates are older and that therefore, these studies may not capture current fishing practices. In addition, concerns were raised that sampling frequencies characterizing higher consumption levels for some of the fishing populations were relatively low (e.g., Black fishers in the Burger et al., 2002 study). The sources of uncertainty described here would have a potentially significant impact on the risk assessment if we were attempting to generate a representative picture of the actual distribution of risk across the full body of fishers reflected in each scenario. In that case, errors related to the study being old, or having insufficient survey samples to full characterize specific high-end percentiles of consumption would be a significant concern. However, when the goal of the analysis is to identify a set of high-end subsistence-like fish consumption rates that we believe could exist for a subset of each fishing population (and not to generate a representative population-weighted picture of risk), then the potential impact of these sources of uncertainty is reduced. Concerns were also raised by the SAB that the fish consumption surveys could reflect seasonally-differentiated consumption rates (i.e., higher rates may only occurred for part of the year during increased fishing activity). Regarding this issue of seasonality in the consumption rates, our evaluation of all three studies suggests that they provide annualized consumption rates (see Table 1-6) and therefore, we believe that the likely impact of this source of uncertainty is limited.

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
		target larger fish and focus their fishing activity at a specific watershed, then uncertainty in these two factors is largely ameliorated.	
(H) Estimating unit concentration of MeHg in cooked fish (application of adjustment factors for the fraction of total Hg that is MeHg in fish and cooking/ preparation).	Two adjustment factors are applied to the 75th percentile fish tissue Hg concentration at a given watershed to generate an estimate of MeHg in the cooked fish serving (in ppm). These include a *Hg conversion* factor for estimating the fraction of Hg in fish that is MeHg and a *fish preparation/cooking adjustment* factor for adjusting the MeHg concentration in the fish to reflect preparation of the fish. There is uncertainty associated with both of these factors.	*Mercury conversion factor:* the factor used in this analysis (0.95) reflects consideration for the range of values provided in the Mercury Study Report to Congress (U.S. EPA, 1997) (i.e., ≥0 .90) (see section 1.4.4). It would be possible to include a sensitivity analysis considering 0.90 and 0.99, but given that this factor is linearly related to both exposure and risk, the outcome would be similarly modest: approximately 5% lower and higher risk, respectively for a given watershed-specific estimate. Given available information, we do not believe that any systematic bias is introduced into the analysis due to the use of this parameter value. *Fish preparation/cooking adjustment factor:* The factor used in this analysis (1.5) reflects consideration for the range of preparation factors provided in Morgan et al., 1997) (i.e., 1.1 to 1.5 for walleye and 1.5 to 2.0 for lake trout - see section 1.4.4). Again, as with the Hg conversion factor, we could consider a range of factors as a sensitivity analysis, but again, given that exposure and risk are linearly related to this factor, the outcome would is predictable. In this case, the impact on risk could be more pronounced, since the potential factors range from 1.0 to 2.0. The SAB identified several alternative studies for characterizing this factor, suggesting that those studies would support a smaller (or no) positive adjustment of the MeHg levels in fish reflecting preparation. However, as discussed in section 1.4.4, close analysis of these studies resulted in a conclusion that they in fact, did not support application of alternative (lower) factors. Furthermore, given available information, we do not believe that any systematic bias is introduced into the analysis due to the use of this parameter value.	Uncertainty in these factors was not quantitatively assessed as part of the sensitivity analysis, however, as noted in the cell to the left, the linear relationship between both factors and exposure and risk allows us to readily determine the magnitude of impact that uncertainty in these factors could have on risk at the watershed-level.
Application of proportionality assumption in generating estimates of the U.S. EGU-attributable fraction of risk			
(I) Fish tissue measurement data	Concerns that the proportionality assumption linking Hg deposition	If air deposition patterns from the 1990s are reflected in some of the Hg fish tissue measurements we are using	We did not quantitatively assess this potential source of uncertainty and its impact on risk. However, our decision

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
used in the analysis may still reflect earlier historical patterns of Hg deposition (from the 1990's) more so than deposition from the simulation period (i.e., ~2005)	and fish tissue Hg concentrations may not hold if fish tissue Hg concentrations actually reflect an earlier (higher Hg deposition) period. In this case, bias could be introduced into estimates of reduction in fish tissue Hg concentrations (based on the proportionality assumption) since the effect of changes in current Hg deposition on fish tissue Hg concentrations could be overstated (i.e., there could be an effective buffering of the fish tissue Hg concentrations by the residual effects of earlier higher Hg deposition).	the implications can vary depending on the nature of that difference. If fish tissue levels for a watershed still reflect higher 1990 deposition values, then there may be an upward bias to the estimates of U.S. EGU-attributable risk, since in reality we would expect the underlying fish tissue levels to decrease as the impact of those earlier higher deposition values dissipates. Conversely, if total deposition remains the same, but only the source distribution has changed since the 1990's, then the effect on our risk assessment may not be that significant, since we are making projections based on the current source-mix (or future source-mix) assuming near steady-state assumptions are reached given those specific source mixes. Given currently available information, we cannot evaluate the interplay of these two factors and the way in which they come together to introduce potential bias into estimates of U.S. EGU-attributable risk (we can only discuss each as a source of uncertainty as has been done here).	to use only fish tissue samples from after 1999 is intended to reduce this source of uncertainty.
(J) Watersheds can display substantially different methylation potentials, resulting in differences in the impact of a unit change in Hg deposition on fish tissue Hg concentrations	Differences in methylation potential cause variation in fish tissue Hg concentrations across waterbodies (even when those waterbodies have similar rates of Hg loading). In addition, differences in methylation potential can also translate into different temporal profiles for the response of fish tissue Hg concentrations to changes in Hg loading. Failure to reflect these sources of variability in methylation potential can introduce uncertainty into the analysis.	Differences in methylation potential across watersheds, when present, are likely to be reflected in the underlying fish tissue Hg concentrations themselves. Therefore, these methylation differences will not to have a substantial impact on our analysis and are not expected to introduce systematic bias (the time sequence of changes in fish tissue levels will depend on differences in methylation potentials, but we are not attempting to predict these temporal profiles).	We did not quantitatively assess uncertainty related to this issue. Our determination is that this is not an important source of uncertainty for our risk estimates.
(K) Factors related to methylation in watersheds (e.g., sulfate deposition, pH) have not remained constant	This source of uncertainty is distinct from the issue of methylation potential discussed above (here we talk about factors which can interact with the underlying methylation potential	As with methylation potential discussed above, any changes in factors affecting the underlying methylation potential of a waterbody would eventually be reflected in fish tissue Hg concentrations and therefore not introduce systematic bias into the analysis. For this reason, our concern over this issue is reduced. However,	We did not quantitatively assess uncertainty related to this issue.

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
over time, resulting in variation in the methylation potential of watersheds over time.	to increase or decrease it). If these factors (pH and sulfate deposition for example), have changed for a given watershed, then the methylation rate of that waterbody may also change, thereby impacting the degree to which fish bioconcentrate and ultimately (for higher trophic levels) bioaccumulate Hg. Changes in methylation factors can also impact the temporal profile for changes in fish tissue Hg concentrations.	if changes in methylation factors are continuous and extend into the current simulation period, then there is more concern, since there could be more of a gradual change in methylation across our simulation period (a change that would not be fully reflected in the fish tissue Hg values we have in the dataset). However, it is difficult to characterize the magnitude of the potential impact of this source of uncertainty on risk estimates, including the direction and magnitude of any bias, given the complex interplay of the factors involved (i.e., potential for ongoing changes in pH and sulfate to produce methylation rates that vary over time).	
(L) Effort to exclude watersheds with substantial non-air Hg deposition[58]	Despite efforts to exclude watersheds with substantial non-air sources of Hg loading (see section 1.4.2), some watersheds with substantial non-air impacts may have been retained in the analysis.	The potential that we may have failed to exclude watersheds with significant non-air Hg loadings could introduce high-bias into our estimates of U.S. EGU-attributable risk, since we would overstate the role of U.S. EGUs in contributing to risk, by overlooking the other non-air sources.	We completed two sensitivity analyses exploring this issue and its potential impact on risk including: (a) an analysis of risks when watersheds falling in LA, SC, MN, and ME, are excluded (there is concern that these four states may have significant non-air Hg releases and/or increased methylation potential) and (b) an assessment of risk based only on those watersheds falling in the upper 25th percentile with regard to total Hg deposition (i.e., estimating risk when we focus on those watersheds where we are more confident that aerial Hg deposition plays a dominant role). The sensitivity analysis results suggest that exclusion of the four states where we have concerns over non-air Hg loading does not substantially impact risk. However, when we estimated risk focusing on the watersheds with the highest overall Hg loading (the upper 25th percentile), we did see a moderate increase in risk estimates.
(M) Potential that the modeling framework is not sufficiently refined from a spatial standpoint to capture elevated levels of Hg	If the overall approach for linking Hg deposition (specifically the fraction attributable to U.S. EGUs) is not sufficiently spatially refined, then areas of high U.S. EGU Hg impact and resulting risk	We believe the level of uncertainty associated with precision in modeling exposure and specifically the ability to identify U.S. EGU-related risk "hot spots" (again, using SAB terminology to refer to areas of elevated Hg-related risk) is moderate. CMAQ modeling is provided at the 12km grid resolution which matches	The issue of spatial scale in capturing high-end subsistence fisher risk has not been quantitatively assessed as part of the risk assessment.

[58] For the March version of the Mercury Risk TSD, we also explored the issue of whether the proportionality assumption was more applicable to stationary waterbodies (lakes/ponds) then flowing waterbodies (rivers/streams). However, for this version of the Mercury Risk TSD, reflecting recommendations made by SAB that the proportionality assumption can be readily applied for either category of waterbody, we have not repeated that sensitivity analysis, or discussed this source of uncertainty.

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
deposition ("hot spots" as termed in the SAB comments) (Note, this source of uncertainty also involves CMAQ modeling)	may not be reflected in the risk assessment. This issue speaks to spatial precision in (a) the CMAQ air quality modeling, (b) scale of the watersheds (and degree to which subsistence activity is focused within specific watersheds) and (c) applicability of the proportionality assumption in relating changes in Hg deposition to changes in fish tissue Hg concentrations (or relating fraction of total Hg deposition coming from U.S. EGUs to the fraction of fish tissue Hg attributable to U.S. EGUs).	well the watershed scale used in the analysis (i.e., the HUC12). The HUC12 watersheds, in turn, represent a small watershed which is appropriate if we assume that the subsistence fisher being modeled focuses their activity primarily at the same waterbody (i.e., within a given HUC12 watershed). Therefore, given the focus of this analysis on a reasonable highly-exposed subsistence fisher (i.e., an individual who focuses their activity at a given waterbody), then the modeling framework, including the spatial scale of the various modeling elements (CMAQ modeling, watershed, fish tissue data), would seem appropriate. For this reason, we believe there is little concern for the introduction of bias into the risk assessment due to the precision of the modeling and our ability to capture potential hot spots.	
Factors relating to the estimation of mercury deposition over watersheds using the CMAQ model (e.g., estimating Hg emissions from U.S. EGUs and other sources, chemistry associated with Hg fate and transport, prediction of wet and dry deposition, and global inflow of Hg into the U.S.)				
(N) Hg 2005 U.S. EGU emissions, uncertainties and sources of bias	2005 Hg emissions from EGUs used for this analysis are from the 2005 inventory developed for the National Air Toxics Assessment (NATA). These data were calculated by EPA by scaling Hg data from the 1999 National Emissions Inventory (NEI) using the ratio of 2005 heat input to 1999 heat input. Thus, uncertainties and bias in the 2005 data fall into two classes – those that are due to the 1999 estimates and those resulting from the scaling approach used to estimate 2005 emissions.

The 1999 NEI data uncertainties include uncertainties traditionally associated with emissions estimation, such as uncertainties in the accuracy of test data, the applicability of the test data, the representativeness of emission | The approach taken for the 1999 NEI used estimates developed from the data collected for the ICR done for the Clean Air Mercury Rule. These data were preferentially used over other estimates available from states, local agencies, or tribes. While these approaches include some uncertainties, they used best available practices and data available from the very rich data source of the 1999 ICR. In general, emissions estimates relying on test data will result in the best available emission estimates for sources with such data, and the uncertainties associated with measurement error are considered negligible in comparison to the other sources of uncertainty. Estimates based on emission factors will have greater uncertainty but will not significantly contribute to bias unless the emission factors are not representative. Care was taken to apply the most representative emission factors data to the sources in the cases where this was needed. A great deal was known about throughput at these sources from the ICR data, and so that is not considered a large source of uncertainty. These uncertainties can cause both overestimates and underestimates of emissions, but since there is no way to assess that, it is not possible to quantify the potential impact (including the direction | The impact of this source of uncertainty was not quantitatively evaluated. |

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
	factors for specific units, the oversimplification occurring when emission factors are used rather than continuous monitoring, and the accuracy of throughput estimates. The 2005 approach adds uncertainty as well as bias to emissions estimates because it does not include the impacts of controls added to sources between 1999 and 2005 or changes in fuels, including the Hg content of the fuel. In addition, uncertainties in the throughput information used to scale the emissions can affect the emissions estimates. Lastly, the temporal and speciation representation of emissions in the 2005 model inputs is another source of uncertainty. However, as noted in column to right, the net effect of high-bias in 2005 emissions would be to low-bias risk estimates for the 2016 scenario.	and magnitude of bias) on risk estimates. The 2005 scaling approach will tend to overestimate emissions in 2005 because of additions of control technologies and coal used between 1999 and 2005 that would have reduced emissions. These reductions were not included because only the changes to throughput were considered in the approach. This most likely causes uncertainties that result in a high bias (overestimate) for the emissions in 2005. Since the 2005 deposition results are used in the denominator of the risk scaling approach to predict risk in 2016, overestimates of 2005 emissions are expected to result in an underestimate of risk in 2016. The uncertainties associated with the heat input data in 2005 are very low, since most units included in the mercury estimates had CEM data to record and report heat input for 2005. The speciation approach used test data from the 1999 data collection, which assigned speciation factors based on boiler type, fuel type, and control configuration. While imperfect, these data will tend to minimize inaccuracies in speciation assigned since the dataset used to calculate the fractions was fairly large, and was certainly the most comprehensive Hg speciation data available. Finally, the temporal allocation approach was kept the same between 2005 and 2016 so that any small impacts of these assumptions on deposition results will cancel out using the scaling approach for risk.	
(O) Hg 2016 U.S. EGU emissions, uncertainties and sources of bias	There are three key variables that figure in the modeling of 2016 Hg emissions from U.S. electric generating units (U.S. EGUs): the mercury content of the fuel combusted (essentially various ranks and grades of coal), the extent of coal consumed, and type and performance characteristics of the controls to reduce mercury emissions at a given U.S. EGU. Uncertainty may figure in each of these key variables.	As a result of uncertainty, the 2016 mercury emissions from EGUs may be over or under-estimated. However, the extent of the impact of uncertainty is considerably reduced (as is concern over bias that may impact risk estimates) due to the number of directly measured and quality assured data points that are used to characterize the variables described in the previous column, by the mature nature of the technologies for mercury emission control, and by the commensurate experience and expertise in characterizing their performance as a result of more than a decade of in-field operation of the controls.	The impact of this source of uncertainty was not quantitatively evaluated.

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
(P) Hg Non-EGU emissions, general uncertainties and consideration of potential bias	The 2005 NATA inventory for non-EGU sources is a compilation of state data and data associated with sector-specific rulemaking activities at EPA. Uncertainties include uncertainties traditionally associated with emissions estimation, such as uncertainties in the accuracy of test data, the applicability of the test data, the representativeness of emission factors for specific units, the oversimplification occurring when emission factors are used rather than continuous monitoring, and the accuracy of throughput estimates. In general, the data based on source tests and individual source reporting are of higher quality with less uncertainty. In addition, speciation and temporal allocation considerations add sources of uncertainty. These uncertainties are also applicable to the 2016 emissions, since these emissions are derived by scaling the 2005 emissions based on expected reductions between 2005 and 2016.	Mercury emissions based on facility test data have been used whenever possible for the 2005 NATA inventory, focused on the categories with the highest emissions. For example, emissions sources with 2 or more tons in 2005 include Portland cement, electric arc furnaces, boilers, chemical manufacturing, hazardous waste incineration, chlor-alkali plants, gold mining, and municipal waste combustors. For these sources, which represent 36 tons out of 47 tons non-EGU Hg emissions, 65% of these sources used data developed for rules associated with these sectors. The rule data are in large part based on test data and have otherwise had extensive review by industry experts and the facilities themselves. In combination with the U.S. EGU data, emissions from these higher quality data sources comprises 76% of the total anthropogenic U.S. Hg inventory. Thus, EPA's approach reflects an inventory with much lower uncertainty than if it had been based solely on more generic emission factors. Speciation data used for this analysis are highly uncertain, but reflect best available information. Since the analysis was not quantifying the impact of non-EGU sources, the speciation of non-EGU sources tends to contribute less to the impact on the analysis. Additionally, while temporal allocation uncertainties are also high, the temporal allocation approaches for each sector are used consistently in 2005 and 2016, so their impacts are lessened through the use of the scaling approach.

There are no known sources of bias related to estimation of emissions from non-EGU sources in this analysis. Every attempt was made to use the best available emissions data in 2005 and future year projections to 2016.

However, a possible (but not likely) source of non-EGU emissions underestimates is from natural sources. Public comments on EPA's approach suggest that some natural sources were not considered. However, EPA does include natural elemental Hg emissions from volcanoes, oceans, and land. Further estimates of recycled Hg emissions from oceans and land are | The impact of this source of uncertainty was not quantitatively evaluated. |

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
		included. One source of uncertainty is the treatment of Hg from fires. While EPA did not explicitly include these emissions in the analysis as fires, the model calibration approach used to estimate the recycled land emissions captures such emissions (Seigneur, et. al. 2004). To the extent that there is any underestimation bias, it would reduce the fractional contribution of U.S. EGU sources, however, given the low likelihood of these sources of bias, the impacts on risk are expected to be small.	
(Q)Hg Emissions, changes in uncertainties and bias between 2005 and 2016	The sources of change between 2005 and 2016 emissions are the U.S. EGU emissions and the stationary non-EGU emissions. All other emissions sources (mobile and non-anthropogenic) stayed constant between the two years. For U.S. EGUs, different sources of uncertainties exist in the two different approaches for 2005 and 2016 emissions, as described above. The possible changes in uncertainties from 2005 to 2016 are caused by the changes in methods. For non-EGU sources, uncertainties in 2016 emissions can result from assumptions about emissions reductions associated with other EPA regulations as well as predicting future activity levels associated with economic or other changes.	For EGUs, some types of uncertainties could be greater in 2016 than in 2005 since the emissions are forecast to the future, for example, the future expected electricity demand and coal usage. 2005 emissions estimates have different uncertainties and it is not possible to determine without extensive uncertainty analysis which sources are greater than others. As described above, a bias most likely exists in the 2005 data that does not exist in the 2016 data, resulting in a possible underestimate of risk from the deposition-based scaling approach used for 2016 risk estimates. For non-EGUs, several source categories reduced emissions from 2005 to 2016 as a result of other EPA regulations. While the reductions are estimated, the uncertainties are relatively low given the extensive analyses done to develop the regulations and since compliance with EPA emissions reductions requirements tends to be very high. Uncertainties are introduced because EPA does not include co-benefits of reductions from controls for other pollutants such as $PM_{2.5}$, which might lead to overestimation of the 2016 non-EGU emissions, lessening the impacts of U.S. EGUs, and therefore lowering the risk estimates from EGUs in our analysis. Uncertainty is also introduced in this analysis for non-EGU sources because the possible growth or reduction in emissions associated with economic changes is not included in the 2016 estimates. However, it is not possible to determine the impact of excluding such considerations since non-EGU industry-specific forecasts are highly uncertain themselves, there is a complex relationship between economic changes, regulations on emissions, and new source requirements	The impact of this source of uncertainty was not quantitatively evaluated.

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
		that can reduce emissions.	
(R) Uncertainty in location of 2016 emissions reductions	Uncertainties in the 2016 scenario regarding the specific geographic locations of reductions in EGU-derived mercury deposition as a fraction of total mercury deposition	The upstream U.S. EGU sources of Hg emissions are provided by EPA's power sector model, the Integrated Planning Model (IPM). The uncertainty in the extent of projected Hg emissions from these sources is treated above (see entry "O"). Here the focus is on the question of uncertainty in the location of the 2016 emissions reductions from the standpoint of which EGUs are projected to operate and so serve as sources of Hg emissions. IPM is an extremely detailed bottom-up deterministic linear programming representation of the U.S. power sector. There is no stochastic component in the model that would allow one to characterize the likelihood that the location of EGU sources and the extent of emissions from these sources are shifting. Therefore, we are not able at this time, to evaluate the potential for systematic bias being introduced into risk estimates through this step in our modeling. However, the model's rigorous adherence to economic and engineering fundamentals and detailed representation of all factors affecting power system operation are designed to limit uncertainty and ensure that the model's locational projections are reasonable.	The impact of this source of uncertainty was not quantitatively evaluated.
(S) Global inflow of mercury into the continental United States	There is considerable uncertainty in the global emissions inventory for mercury and given the long residence time of elemental mercury it is possible that inflow into the modeling domain may reflect deficiencies in the global emissions inventory. Some studies suggest global mercury emissions would increase without control implementation (Pacyna et al., 2009; Streets et al., 2009). Recent measurements of ambient mercury at remote locations from 1975 to 2008 suggest global mercury burden is steadily decreasing (Slemr et al., 2011). Given this conflicting information and the uncertainty in projecting	Mercury deposition may be over or under-estimated (i.e., potential bias introduced is not clear in terms of direction or magnitude).	This analysis did not address this source of uncertainty. Mercury inflow was adjusted in another study and was shown to have a larger impact on modeled dry deposition compared to wet deposition. Boundary condition perturbations within realistic bounds resulted in minimally changed distributions of total modeled mercury deposition (Pongprueksa et al., 2008). This study also determined that model spin-up of one week is necessary to remove influence of initial conditions (Pongprueksa et al., 2008). The CMAQ modeling in this analysis had spin-up of more than one week making initial condition influence negligible.

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
	the current global emission inventory, boundary inflow is kept constant between 2005 and 2016.		
(T) Photochemical model prediction of mercury wet deposition estimates	The uncertainty in the wide variety of model inputs such as emissions, meteorology, global inflow to the modeling domain, and chemistry manifest in model estimates of mercury wet deposition.	Mercury wet deposition may be under-estimated during the summer season and over-estimated during the winter (i.e., potential bias introduced is not clear in terms of direction or magnitude). Model performance is described in "Air Quality Modeling Technical Support Document: Point Source Sector Rules" (USEPA, 2011b).	Model estimated weekly mercury wet deposition is compared to observation data to assess model skill simulating this component of mercury deposition. Mercury wet deposition measurements are weekly totals taken at sites that are part of the Mercury Deposition Network (http://nadp.sws.uiuc.edu/MDN/) which operates under the National Atmospheric Deposition Program. This is generally consistent with other published studies that use coarser grid resolution, older versions of CMAQ and older emission inventories (Bullock et al., 2008; Lin et al., 2007).
(U) Photochemical model prediction of mercury dry deposition estimates	The uncertainty in the wide variety of model inputs such as emissions, meteorology, global inflow to the modeling domain, and chemistry manifest in model estimates of mercury dry deposition.	Mercury dry deposition may be over or under-estimated (i.e., potential bias introduced is not clear in terms of direction or magnitude).	This analysis did not address this source of uncertainty. There is a lack of dry deposition observation data that makes a direct quantitative or even qualitative comparison to modeled estimates impossible. Other studies have shown differences in estimated dry deposition based on changes in ambient mercury, reaction rate changes, and changes to the dry deposition scheme (Bullock et al., 2009; Lin et al., 2007; Pongprueksa et al., 2008; Ryaboshapko et al., 2007a; Ryaboshapko et al., 2007b). However, given that these studies are based on earlier versions of CMAQ or other models that are not routinely used for regulatory purposes and that mercury chemistry and dry deposition has changed through model versions, it is not clear that dry deposition estimates in the version of CMAQ used for this analysis would be comparable to earlier studies.
(V) Mercury Chemistry	The complete set of mercury oxidation and reduction reactions has not been identified by the scientific community.	Mercury deposition may be over or under-estimated (i.e., potential bias introduced is not clear in terms of direction or magnitude).	This analysis did not address this source of uncertainty. Other studies have shown differences in estimated total mercury deposition based on changes in reaction rates (Bullock et al., 2009; Lin et al., 2007; Pongprueksa et al., 2008; Ryaboshapko et al., 2007a; Ryaboshapko et al., 2007b). However, given that these studies are based on earlier versions of CMAQ or other models that are not routinely used for regulatory purposes and that mercury chemistry and deposition has changed through model versions it is not clear that total mercury deposition estimates in the version of CMAQ used for this analysis would be comparable to earlier studies.

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
Application of the MeHg RfD in generating hazard quotient (HQ) risk estimates			
(W) Degree to which the RfD as calculated provides coverage for low SES status groups which may be at greater risk for adverse health effects following MeHg exposure	Given a number of factors (e.g., nutritional deficiencies, reduced access to health care and health-related information), there is concern that the MeHg RfD may not provide sufficient coverage for these low SES populations.	The RfD is defined as the amount of the substance of concern that can be consumed without expectation of harm for a lifetime by populations including sensitive subpopulations. The calculated RfD for MeHg includes an uncertainty factor to account for human pharmacokinetic variability (3 fold) and uncertainty and variability in pharmacodynamics. This may be sufficient to account for increased sensitivity to IQ decrements or adverse effects on neurobehavioral functions in low socio-economic status populations. There are no published analyses by EPA or other parties that would permit estimation of uncertainty for this factor and the potential for resulting bias being introduced into the analysis. Two of the human populations on which the RfD was based (the Faroese and the Seychellois) are relatively homogenous for some aspects of SES.	We did not explore this potential source of uncertainty quantitatively. However, as noted in the cell to the left due to the method used in calculating the RfD, concerns that the RfD may not provide coverage for low SES populations is reduced.
(X) The MeHg RfD was derived based on saltwater fish consumption (which can involve higher levels of nutrients that ameliorate the adverse effects of MeHg). However, the RfD is now being applied in the context of freshwater fish consumption (which can involve lower levels of these nutrients).	Recent studies (e.g. Oken et al 2008; Choi et al 2008;) point to the potential for nutrients in fish (particularly marine fish) to ameliorate some of the observed adverse effects of MeHg when co-exposure occurs. However, there was no correction for potential confounding by nutrients in marine fish and mammals in calculation of the benchmark doses used in the RfD derivation. Therefore, there is the potential that these benchmark doses may be underestimates, in which case the HQ estimates based on the RfD could be biased low, particularly in the case of freshwater fish which may have lower levels of these nutrients.	Failure to consider the effects of fish nutrients as a covariate or confounder for nerodevelopmental effects associated with MeHg exposure could result in HQ estimates being biased low. However, currently, available information does not support a rigorous adjustment of the RfD to address potential confounding by fish nutrients.	We did not explore this potential source of uncertainty quantitatively.

3 Summary of Key Observations

This section provides key policy-relevant observations drawn from discussions presented in Sections 2.1 through 2.7. It is important to emphasize, that the risk estimates and additional supporting information summarized here are intended to inform a determination by the Administrator as to whether Hg emitted from U.S. EGUs represents a public health hazard. These observations are not intended to be conclusionary in nature and instead, focus on characterizing the nature and magnitude of risk associated with U.S. EGU Hg emissions.

- Estimates of U.S. EGU Hg emissions suggest that the 2016 Scenario is likely closer to recent (2010) emissions compared with the 2005 scenario (which has substantially higher total Hg emissions for this sector). Therefore, risk estimates have been generated only for the 2016 scenario for the revised risk assessment (CMAQ-based Hg deposition estimates for the 2005 scenario are used in scaling fish tissue concentrations for use in modeling 2016 scenario risk).

- Risk characterization is based on estimates of RfD-based HQ. Due to concerns raised by the SAB peer-review panel that the IQ loss endpoint may not fully capture the range of neurodevelopmental effects associated with MeHg exposure, IQ loss estimates are presented in brief summary form in Appendix B and not used in the risk characterization.

- Based on the 2016 scenario, U.S. EGUs can contribute up to 11% of total Hg emissions for a subset of watersheds. However, in general, other sources besides U.S. EGUs dominate Hg deposition. In 2016 U.S. EGUs contributed on average about 2% of total Hg deposition across the country. U.S. EGU-related Hg deposition is higher in the eastern part of the country with elevated contributions in a number of specific areas, including most notably, the Ohio River valley. U.S. EGU-related Hg deposition estimates show a significant reduction between 2005 and 2016 scenarios, reflecting mainly co-benefits from implementation of criteria pollutant controls with the average U.S. EGU-attributable deposition decreasing from ~5% of total to ~2% for the 2005 and 2016 scenarios, respectively.

- Based on the 2016 scenario, U.S. EGUs can contribute up to 16% of MeHg in fish tissue. However, generally, U.S. EGUs contribute a much smaller fraction averaging 3% for the 2016 Scenario.

- Comparing the magnitude of Hg fish tissue levels with total Hg deposition (as characterized at the watershed-level) suggests that there is not a strong correlation. This is not surprising given the variety of factors which effect methylation potential; factors which can demonstrate substantial spatial variation. However, available evidence supports a steady state linear relationship between changes in Hg deposition and changes in fish tissue Hg concentrations.

- The additional fish tissue Hg data added for the revised risk assessment have significantly improved coverage for watersheds located in areas with elevated U.S. EGU-related Hg deposition such as the Ohio River Valley. However, we still conclude that, generally, our

coverage for high U.S. EGU impact areas remains limited. For this reason, we continue to believe that the actual number of "at-risk" watersheds (i.e., watersheds where U.S. EGUs could contribute to a public health concern) could be substantially larger than estimated.

- Based on application of the 2-stage risk characterization framework described in Section 1.3, we estimate that from 22 to 29% of the watersheds included in this risk assessment could be classified as potentially having at-risk populations under the 2016 Scenario. This estimate is based on risks modeled for the *typical female subsistence fish consumer* and reflects aggregation of results from Stages 1a and 1b of the 2-Stage Risk Characterization Framework (i.e., watersheds where mercury released from U.S. EGUs when considered alone, without taking into account mercury deposition from other sources, would produce an HQ >1 or watersheds where total HQ (reflecting Hg from all sources) is > 1 and U.S. EGUs make at least at 5% contribution to that-risk).

- Comparison of risk estimates generated for the *typical female subsistence fish consumer* scenario with estimates generated for the other six SES-differentiated female subsistence fish consumer scenarios included in this risk assessment results in the following observations: (a) total and U.S. EGU-attributable risks for the Hispanic and Vietnamese scenarios are generally lower than for the *typical female subsistence fish consumer* scenario, (b) U.S. EGU-attributable risks for the Tribal scenario are similar to those for the *typical female subsistence fish consumer* scenario, although total risks are generally higher for the Tribal scenario and (c) U.S. EGU-attributable risks for the Laotian and low income southeastern White and Black scenarios are generally higher than for the *typical female subsistence fish consumer* scenario, although total risks can be higher or lower depending on the scenario. These trends suggest that generally, the *typical female subsistence fish consumer* scenario will provide coverage (in terms of representing risk) for Hispanic, Vietnamese and Tribal scenarios. However, the *typical female subsistence fish consumer* scenario may not provide full coverage for the Laotian and low income southeastern White and Black scenarios, particularly in terms of U.S. EGU-attributable risk.

- If U.S. EGU impacts to watersheds included in the risk assessment were zeroed-out, for a significant majority of those watersheds, total exposure would still exceed (and in most cases, significantly exceed) the RfD. Reductions in U.S. EGU attributable Hg will reduce the magnitude of the risk, although substantial total exposure and risk from Hg deposition will remain.

- Sensitivity analyses conducted primarily to examine uncertainty in applying the proportionality assumption linking Hg deposition to Hg fish tissue levels, suggest that uncertainty related to the proportionality assumption is unlikely to substantially effect an assessment of whether Hg emissions from U.S. EGUs constitute a public health concern. Use of watershed-level 50th percentile fish tissue Hg concentrations (instead of the 75th percentile values used in the core analysis) can result in notable reductions in risk estimates in some instances, but the SAB peer review panel supports use of the 75th percentile estimates.

Citations:

Appel, K. W., A. B. Gilliland, et al. (2007). "Evaluation of the Community Multiscale Air Quality (CMAQ) model version 4.5: Sensitivities impacting model performance Part I - Ozone." Atmospheric Environment 41(40): 9603-9615.

Appel, K. W., P. V. Bhave, et al. (2008). "Evaluation of the community multiscale air quality (CMAQ) model version 4.5: Sensitivities impacting model performance; Part II - particulate matter." Atmospheric Environment 42(24): 6057-6066.

Axelrad, D. A.; Bellinger, D. C.; Ryan, L. M.; Woodruff, T. J. (2007). Dose-response relationship of prenatal mercury exposure and IQ: an integrative analysis of epidemiologic data. Environmental Health Perspectives. 2007, 115, 609–615.

Bloom, N.S. (1992). On the chemical form of mercury in edible fish and marine invertebrate tissue. Can. J. Fish. Aquat. Sci. 49:1010-1017.

Bullock, O. R. and K. A. Brehme (2002). "Atmospheric mercury simulation using the CMAQ model: formulation description and analysis of wet deposition results." Atmospheric Environment 36(13): 2135-2146.

Bullock, O.R., Atkinson, D., Braverman, T., Civerolo, K., Dastoor, A., Davignon, D., Ku, J.Y., Lohman, K., Myers, T.C., Park, R.J., Seigneur, C., Selin, N.E., Sistla, G., Vijayaraghavan, K., (2008). The North American Mercury Model Intercomparison Study (NAMMIS): Study description and model-to-model comparisons. Journal of Geophysical Research-Atmospheres 113.

Bullock, O.R., Atkinson, D., Braverman, T., Civerolo, K., Dastoor, A., Davignon, D., Ku, J.Y., Lohman, K., Myers, T.C., Park, R.J., Seigneur, C., Selin, N.E., Sistla, G., Vijayaraghavan, K., (2009). An analysis of simulated wet deposition of mercury from the North American Mercury Model Intercomparison Study. Journal of Geophysical Research-Atmospheres 114.

Burger, J. (2002). Daily consumption of wild fish and game: Exposures of high end recreationalists, *International Journal of Environmental Health Research*, 12:4, p. 343-354, July, 2010.

Burger, J., Stephens, W. L., Boring, C. S., Kuklinski, M., Gibbons, J. W., Gochfeld M. (1999) Factors in Exposure Assessment: Ethnic and Socioeconomic Differences in Fishing and Soncumption of Fish Caught along the Savannah River. Risk Analysis, Vol. 19, No. 3, p. 427.

Byun, D. and K. L. Schere (2005). "Review of the governing equations, computational algorithms, and other components of the models-3 Community Multiscale Air Quality (CMAQ) modeling system." Applied Mechanics Reviews 59(1-6): 51-77.

Byun, D., Schere, K.L., 2006. Review of the governing equations, computational algorithms, and other components of the models-3 Community Multiscale Air Quality (CMAQ) modeling system. Applied Mechanics Reviews 59, 51-77.

Dellinger, J. A., (2004) Exposure assessment and initial intervention regarding fish consumption of tribal members in the Upper Great Lakes Region in the United States. Dellinger , Environmental Research 95, p. 325-340

Dispasquale, M.M., J. Agee, C. McGowan, R.S. Oremland, M. Thomas, D. Krabbenhoft, and C.C. Gilmour. (2000) Methyl-Mercury Degradation Pathways: A Comparison Among Three Mercury-Impacted Ecosystems. Environ. Sci. Technol., 34, 4908-4916.

Farias L.A., Favaro, D.I., Santos J.O., Vasconcellos M.B., et al., (2010). Cooking Process Evaluation on Mercury Content in Fish,‖ Acta Amazonia, 40 (4), 741–748.

Grandjean, P., P. Weihe, R. White, F. Debes, S. Arak, K. Yokoyama, K. Murata, N. Sorensen, R. Dahl, and P. Jorgensen. (1997). Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. Neurotoxicol. Teratol. 20:1-12.

Harris., R. C., John W. M. Rudd, Marc Amyot, Christopher L. Babiarz, Ken G. Beaty, Paul J. Blanchfield, R. A. Bodaly, Brian A. Branfireun, Cynthia C. Gilmour, Jennifer A. Graydon, Andrew Heyes, Holger Hintelmann, James P. Hurley, Carol A. Kelly, David P. Krabbenhoft, Steve E. Lindberg, Robert P. Mason, Michael J. Paterson, Cheryl L. Podemski, Art Robinson, Ken A. Sandilands, George R. Southworth, Vincent L. St. Louis, and Michael T. TateRudd, J. W. M., Amyot M., et al., (2007). Whole-Ecosystem study Shows Rapid Fish-Mercury Response to Changes in Mercury Deposition. Proceedings of the National Academy of Sciences Early Edition, PNAS 2007 104 (42) pp. 16586-16591; (published ahead of print September 27, 2007).

Knights, D. C, Sunderland, E. M., Barber, M. C., Johnston J. M., and Ambrose, R. B., (2009). Application of Ecosystem-Scale Fate and Bioaccumulation Models to Predict Fish Mercury Response Times to Changes in Atmospheric Deposition. Environmental Toxicology and Chemistry, Vol 28, No. 4., pp. 881-893.

Lin, C.J., Pongprueks, P., Rusell Bulock, O., Lindberg, S.E., Pehkonen, S.O., Jang, C., Braverman, T., Ho, T.C., (2007). Scientific uncertainties in atmospheric mercury models II: Sensitivity analysis in the CONUS domain. Atmospheric Environment 41, 6544-6560.

Morgan, J.N., M.R. Berry, and R.L. Graves. (1997). "Effects of Commonly Used Cooking Practices on Total Mercury Concentration in Fish and Their Impact on Exposure Assessments." Journal of Exposure Analysis and Environmental Epidemiology 7(1):119-133.

Morgan, J.N., M.R. Berry, Jr., and R.L. Graves. (1994). Effects of Native American cooking practices on total mercury concentrations in walleye. Presented at ISEE/ISEA Joint Conference, September 18-21, 1994.

Moya. J., Itkiin, C., Selevan, S.G., Rogers, J.W., Clickner, R. P., (2008). Estimates of fish consumption rates for consumers of bought and self-caught fish in Connecticut, Florida, Minnesota, and North Dacota. Science of the Total Environment (2008), doi:10.1016/j.scitotenv.2008.05.023.

Musaiger, A. O., R. D'Souza, (2008). The effects of different methods of cooking on proximate, mineral and heavy metal composition of fish and shrimps consumed in the Arabian Gulf. Archivos LatinoAmericanos De Nutricion, Organo Oficial de la Sociedad Latinoamericana de Nutricion., Vol 58, No 1, 2008.

Oken e., Radesky, J.S., Wright, R. O., Bellinger D. C., Amarasiriwardena C. UJ., Kleinman, K. P., Hu, H., Gillman, M.W., (2008). Maternal Fish Intake during Pregnancy, Blood

Mercury Levels, and Child Cognition at Age 3 Years in a US Cohort. American Journal of Epmidemiology, Vol. 167, No. 10, p. 1171.

Pacyna, E.G., Pacyna, J.M., Sundseth, K., Munthe, J., Kindbom, K., Wilson, S., Steenhuisen, F., Maxson, P., 2009. Global emission of mercury to the atmosphere from anthropogenic sources in 2005 and projections to 2020. Atmospheric Environment 44, 2487-2499.

Pirrone N. and Keating T, editors. "Hemispheric Transport of Air Pollution (2010), Part B: Mercury. Prepared by the Task Force on Hemispheric Transport of Air Pollution acting within the framework of the Convention on Long –range Transboundary Air Pollution. United Nations New York and Geneva, 2010.

Pirrone N, Cinnirella S, Feng X, Finkelman RB, Friedli HR, Leaner J, et al., (2010). Global mercury emissions to the atmosphere from anthropogenic and natural sources. Atmospheric Chemistry and Physics Discussions, 10(2), 4719-4752.

Pongprueksa, P., Lin, C.J., Lindberg, S.E., Jang, C., Braverman, T., Bullock, O.R., Ho, T.C., Chu, H.W., (2008). Scientific uncertainties in atmospheric mercury models III: Boundary and initial conditions, model grid resolution, and Hg(II) reduction mechanism. Atmospheric Environment 42, 1828-1845.

Rice GE, Hammitt JK, Evans JS. (2010). A probabilistic characterization of the health benefits of reducing methyl mercury intake in the United States. Environmental Science Technology. 2010 Jul 1;44(13):5216-24

Ryaboshapko, A., Bullock, O.R., Christensen, J., Cohen, M., Dastoor, A., Ilyin, I., Petersen, G., Syrakov, D., Artz, R.S., Davignon, D., Draxler, R.R., Munthe, J., (2007a). Intercomparison study of atmospheric mercury models: 1. Comparison of models with short-term measurements. Science of the Total Environment 376, 228-240.

Ryaboshapko, A., Bullock, O.R., Christensen, J., Cohen, M., Dastoor, A., Ilyin, I., Petersen, G., Syrakov, D., Travnikov, O., Artz, R.S., Davignon, D., Draxler, R.R., Munthe, J., Pacyna, J., (2007b). Intercomparison study of atmospheric mercury models: 2. Modelling results vs. long-term observations and comparison of country deposition budgets. Science of the Total Environment 377, 319-333.

Seigneur, C., K . Vigjayaraghavan, K. Lohman, P. Karamchandani, C. Scott. (2004)."Global Source Attribution for Mercury Deposition in the United States." Environ. Sci. Technol. 38, 555-569.

Selin, N.E., Jacob, D.J., Park, R.J., Yantosca, R.M., Strode, S., Jaegle, L., Jaffe, D., (2007). Chemical cycling and deposition of atmospheric mercury: Global constraints from observations. Journal of Geophysical Research-Atmospheres 112.

Shilling, Fraser, Aubrey White, Lucas Lippert, Mark Lubell (2010). Contaminated fish consumption in California's Central Valley Delta. *Environmental Research* 110, p. 334-344.

Slemr, F., Brunke, E.G., Ebinghaus, R., Kuss, J., (2011). Worldwide trend of atmospheric mercury since 1995. Atmospheric Chemistry and Physics 11, 4779-4787.

Streets, D.G., Zhang, Q., Wu, Y., (2009). Projections of Global Mercury Emissions in 2050. Environmental Science & Technology 43, 2983-2988.

Swartout, J., and G. Rice. (2000). "Uncertainty Analysis of the Estimated Ingestion Rates Used to Derive the Methylmercury Reference Dose." Drug and Chemical Toxicology 23(1):293-306. 11-41

U.S. EPA, U.S. Environmental Protection Agency, (1997). Mercury Study Report to Congress. Office of Air Quality Planning and Standards and Office of Research and Development. EPA-452/R-97-003, December 1997.

U.S. EPA, U.S. Environmental Protection Agency, (2000). Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories, Volume 1: Fish Sampling and Analysis and Volume 3: Overview of Risk Management. Office of Science and Technology, Office of Water, U.S. Environmental Protection Agency, Washington, DC. EPA 823-B-00-007.

U.S. EPA, U.S. Environmental Protection Agency, (2001a). Water Quality Criterion for the Protection of Human Health: Methylmercury - Final. U.S. EPA, Office of Science and Technology, Office of Water, EPA-823-R-01-001 January 2001.

U.S. EPA, U.S. Environmental Protection Agency, (2001b). Mercury Maps - A Quantitative Spatial Link Between Air Deposition and Fish Tissue Peer Reviewed Final Report. U.S. EPA, Office of Water, EPA-823-R-01-009, September, 2001.

U.S. EPA, U.S. Environmental Protection Agency, (2005), Neurobehavioral Assessments Conducted in the New Zealand, Faroe Islands, and Seychelles Islands Studies of Methylmercury Neurotoxicity in Children. David C. Bellinger, Professor of Neurology, Harvard Medical School, March 2005. Report to the U.S. Environmental Protection Agency EPA-HQ-OAR-2002-0056-6045[1]

U.S. EPA, U.S. Environmental Protection Agency, (2007a) Review of the National Ambient Air Quality Standards for Lead: Policy Assessment of Scientific and Technical Information OAQPS Staff Paper. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-452/R-07-013 November 2007.

U.S. EPA, U.S. Environmental Protection Agency, (2007b) Lead: Human Exposure and Health Risk Assessments for Selected Case Studies, Volume I. Human Exposure and Health Risk Assessments—Full-Scale and Volume II. Appendices. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA–452/R–07–014a and EPA–452/R– 07–014b.

U.S. EPA, U.S. Environmental Protection Agency, (2011a). Technical Support Document: National-Scale Mercury Risk Assessment Supporting the Appropriate and Necessary Finding for Coal- and Oil-Fired Electric Generating Units. March, 2011. EPA-452/D-11-002.

U.S. EPA, U.S. Environmental Protection Agency, (2011b). Air Quality Modeling Technical Support Document: EGU Mercury Analysis. EPA-454/R-11-008.

UNEP's Division of Technology, Industry and Economics. (2010). Study on mercury sources and emissions and analysis of cost and effectiveness of control measures "UNEP Paragraph 29 study".

USGS, U.S. Geological Survey (USGS). 2005. Active mines and mineral plants in the US. Reston, VA. Available on the Internet at http://tin.er.usgs.gov/mineplant

USGS, U.S. Geological Survey and U.S. Department of Agriculture, Natural Resources Conservation Service, (2009), Federal guidelines, requirements, and procedures for the national Watershed Boundary Dataset: U.S. Geological Survey Techniques and Methods 11–A3, 55 p.

Wijngaarden, Edwin van, Christopher Beck, Conrad F. Shamlaye, Elsa Cernichiari, Philip W. Davidson, Gary J. Myers, Thomas W. Clarkson. (2006). Benchmark concentrations for methyl mercury obtained from the 9-year follow-up of the Seychelles Child Development Study. NeuroToxicology 27 :702–709.

Appendices: Additional Technical Detail on Modeling Elements and Presentation of Supplemental Risk Estimates

(Citations for the appendices are provided in the citation list for the main document – see above)

As noted in section 1.4.5, the IQ loss risk metric has been de-emphasized in presenting risk estimates due to concerns that it may not fully capture the range of adverse neurodevelopmental effects associated with MeHg exposure. For this reason, both the description of the approach used in modeling IQ loss as well as a summary of IQ loss estimates and a discussion of uncertainty related to those risk estimates is presented in appendices, with the technical approach being described in Appendix A and the summary of IQ loss risk estimates and discussion of uncertainty being presented in Appendix B. Appendix C presents the SAB Mercury Panel peer review letter (the attachment to that letter provides the original EPA charge questions).

Appendix A. Technical Approach Used in Modeling IQ Loss

Estimation of IQ loss in children begin with the same exposure estimates used in generating HQ estimates (i.e., estimates of MeHg exposure generated for a given subsistence fisher scenario - see section 1.4.4 for derivation of these exposure estimates). However, these estimates of body weighted-adjusted MeHg exposure need to be converted into an equivalent maternal hair concentration since the IQ loss function uses hair Hg as the dose measure. To do that, we use a dose-to-hair conversion factor (DHCV) of 12.5 (units ppm per unit µg/kg-day) that converts ingested dose (IR) to hair Hg concentration in ppm. The DHCV factor is based on a one compartment toxicokinetic model used for deriving the MeHg RfD by Swartout and Rice (2000).

After generating an estimate of maternal hair Hg level for the subsistence fisher (at the particular watershed being modeled), we then apply a concentration-response (CR) function relating material hair Hg levels to IQ points lost in the child born to that mother. This CR function was published in Axelrad et al., 2007and is based on application of a Bayesian hierarchical model which integrates data from the three key epidemiological studies (Seychelles, New Zealand and Faroe Islands).[59]

Since the CR function was published in the Axelrad et al., 2007 study, a number of authors have raised the possibility that neurological deficits related to Hg exposure through fish consumption could be masked to some degree by the neurologically-beneficial effects of fish oil consumption. Some authors have suggested that the IQ loss factor should be adjusted upward to compensate for this masking effect (see Rice et al., 2010 and Oken, 2008). However, no rigorous basis for a specific adjusted estimate has been provided to-date and therefore, we address this potential for low-bias as part of our qualitative uncertainty discussion (see Table B-2, Appendix B).

---

[59] The IQ loss model uses a linear slope of 0.18 IQ points per ppm hair Hg concentration (Axelrad et al., 2007).

Appendix B. Supplemental Risk Estimates (IQ loss estimates)

This section provides summaries of IQ loss risk estimates generated for the 2016 scenario and discusses key sources of uncertainty associated with the IQ loss estimates. In assessing the potential public health significance of the IQ loss risk estimates, based on recommendations provided by the Clean Air Science Advisory Committee (CASAC) in the context of the last National Ambient Air Quality Standard (NAAQS) review for lead completed in 2008 (US EPA, 2007a), we interpreted IQ loss estimates of 1-2 points as being clearly of public health significance. All of the risk estimates summarized here are based on application of the revised version of the risk assessment model. Specific tables include:

- Table B-1: provides risk percentiles for the IQ loss risk metric for children born to members of the typical female subsistence fish consumer scenario for the 2016 scenario.

- Table B-2: identifies and discusses key sources of uncertainty associated with modeling IQ loss (uncertainty related to modeling exposure, which also impacts HQ estimates, is covered in Table 2-15).

Table B-1. Percentile IQ loss risk estimates for children born to members of the *typical female subsistence fish consumer* **scenario assessed nationally (2016 scenario)** (for both total and U.S. EGU incremental risk, including IQ loss and MeHg RfD-based HQ estimates)

Typical female subsistence fish consumer rate (g/day) and percentile/mean	Watershed percentile									
	Total IQ points lost					U.S. EGU-attributable IQ points lost				
	50th	75th	90th	95th	99th	50th	75th	90th	95th	99th
39 (mean)	0.4	0.8	1.3	1.7	2.5	-	-	-	0.1	0.1
123 (90th)	1.4	2.4	4	5.3	8*	-	0.1	0.1	0.2	0.3
173 (95th)	1.9	3.4	5.6	7.4*	11.2*	-	0.1	0.2	0.2	0.4
373 (99th)	4.1	7.4*	12.1*	16*	24.1*	0.1	0.2	0.3	0.5	0.8

- IQ loss is <0.1 point

* IQ loss estimate subject to greater uncertainty due to application of the underlying concentration-response function for IQ loss at levels of exposure above those in the underlying epidemiological studies (see Appendix A)

Table B-2. Key sources of uncertainty related to modeling IQ loss, the nature of their potential impact on risk estimates, and the degree to which they are characterized as part of the analysis

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
(A) IQ may not fully capture the most	IQ may not represent the most sensitive cognitive endpoint for Hg exposure (Axelrad et al.,	Given this concern, we have focused the risk assessment on the RfD-based HQ risk estimates and	Because we do not have readily available data to support quantitative analyses of the first

Source of uncertainty	Description	Nature of potential impact on the exposure and risk estimates	Degree to which the potential impact of the source of uncertainty is characterized as part of the analysis
sensitive cognitive endpoints associated with Hg exposure	2007 - see Section 1.2). In addition deficits in some categories of cognitive functioning are not captured by IQ. Together, these sources of uncertainty suggest that we could be under-estimating the extent of cognitive impacts associated with Hg exposure (when we focus on modeling the IQ loss endpoint alone).	have deemphasized IQ loss estimates.	two sources of uncertainty (IQ loss not capturing all of the cognitive effects and potential confounding by LCPFAs), we could only address these factors qualitatively. In both cases, the potential effect on the risk assessment would be to potentially down-bias our estimate of cognitive endpoint-related risk for children.
(B) Potential confounding from nutrients, including long-chained polyunsaturated fatty acids (LCPFAs), found in fish	This issue is similar to the nutrient confounding issue discussed above under the RfD-based HQ entry. Specifically, there are concerns that fish nutrients may mask to some extent, the adverse neruodevelopmental effects of MeHg, resulting in a concentration-response function for IQ loss which could be biased low (particularly in the case of freshwater fish consumption which may have lower levels of relevant fish nutrients)	Potential confounding (masking) of the neurodevelopmental effects of MeHg could result in concentration-response functions for IQ loss that are low-biased, which in turn would mean that estimates of IQ loss could be low biased. However, at this point we do not have the information necessary to derive a concentration-response function for IQ loss that would account for this masking effect and therefore, cannot quantitatively evaluate this source of uncertainty.	
(C) How to treat potential outliers from the epidemiological datasets used in deriving the IQ loss functions.	Regarding outliers, when an outlier datapoint from the Seychelles study was included in the integrated derivation of the IQ loss slope factor, the factor was reduced by 25 percent (from -0.18 IQ points per unit ppm hair Hg, to -0.125). If in reality, this outlier actually reflects the true response for a subset of the populations, then risks (as modeled) could be biased high specifically for this subpopulation.	In the case of excluding the outlier from the Seychelles study, we note that the effect (given the linear nature of the IQ loss slope) would be to simply result in a 25% reduction in risk, if we were to include the outlier in derivation of the slope function (i.e., a formal rerun of the model with this alternative slope is not required – we can just consider this magnitude of impact on the primary risk estimates we generate for the analysis).	Although it is possible given the linear relationship of exposure and IQ loss to readily determine the impact of a sensitivity analysis involving application of an IQ loss slope factor that reflects inclusion of the outliers, we did not explicitly complete such an analysis (primarily because the IQ loss risk metric has been de-emphasized with primary focus being placed on the RfD-based HQ metric).

Appendix C. SAB Mercury Panel Peer Review Letter: *Review of EPA's Draft National-Scale Mercury Risk Assessment*

This appendix presents a copy of the SAB's Mercury Panel Peer Review Letter. The original charge questions provided by EPA to the SAB are included at the end of the letter as Appendix A.

September 29, 2011

EPA-SAB-11-017

The Honorable Lisa P. Jackson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Subject: Review of EPA's Draft National-Scale Mercury Risk Assessment

Dear Administrator Jackson:

EPA's Office of Air and Radiation requested that the Science Advisory Board (SAB) review a draft
*Technical Support Document: National-Scale Mercury Risk Assessment Supporting the Appropriate and
Necessary Finding for Coal and Oil-Fired Electric Generating Units - March 2011*. The goal of this
draft document is to characterize human health exposure and risk associated with U.S. electrical
generating unit (EGU) mercury emissions with a focus on a highly exposed subpopulation, subsistence
fishers. The SAB was asked to comment on the risk assessment, including the overall design and
approach, as well as the use of specific models and key assumptions. The SAB was also asked to
comment on the extent to which specific facets of the assessment are well characterized in the Technical
Support Document.

The SAB could not evaluate the risk assessment based solely upon information provided in the
Technical Support Document. Important elements of the methods and findings are missing or poorly
explained. Additional information provided by EPA representatives during an SAB public meeting on
June 15-17, 2011 and a public teleconference on July 12, 2011 allowed the SAB to gain a sufficient
understanding of the risk assessment to conduct this review.

The risk assessment is designed to assess how a reduction in mercury emissions will translate to
reductions in fish tissue methylmercury concentrations, and in turn, to a reduction in potential risk to
subsistence fishers that would result from the consumption of self-caught fish from inland watersheds.
EPA sought advice from the SAB on key components of its analysis. In response, the SAB reviewed
available information and has made the following findings. The SAB considers the spatial resolution of
the modeling of mercury deposition to watersheds to be appropriate for the analysis. There is agreement
that the approach used to identify watersheds to include in the assessment is reasonable. This approach
is based upon the availability of fish tissue methylmercury data and census data on target populations
with potential subsistence fishers. The SAB agrees that EPA's calculation of a hazard quotient for each
watershed is appropriate as the primary means of expressing risk because it is based on an established
reference dose for methylmercury that reflects a range of potential neurobehavioral effects. Intelligence

Quotient (IQ) loss is also used in the assessment to evaluate risk. The SAB considers IQ loss to be an insensitive indicator of methylmercury neurobehavioral effects and is concerned that its use as an endpoint could underestimate risk. The SAB recommends that IQ loss be de-emphasized in the risk assessment and explored as one of several possible secondary public health endpoints as a supplement to the main analysis. Although the SAB considers the number of watersheds included in the assessment adequate, some watersheds in areas with relatively high mercury deposition from U.S. EGUs were under-sampled due to lack of fish tissue methymercury data. The SAB encourages the Agency to contact states with these watersheds to determine if additional fish tissue methylmercury data are available to improve coverage of the assessment.

The SAB identifies additional sources of variability and uncertainty in the risk assessment, as well as limitations imposed by the availability of data. The uncertainties are appropriate for a screening-level public health assessment. The SAB regards the design of the risk assessment as suitable for its intended purpose, to inform decision-making regarding an "appropriate and necessary finding" for regulation of hazardous air pollutants from coal and oil-fired EGUs, provided that our recommendations are fully considered in the revision of the assessment.

In summary, based on its review of the draft Technical Support Document and additional information provided by EPA representatives during the public meetings, the SAB supports the overall design of and approach to the risk assessment and finds that it should provide an objective, reasonable, and credible determination of the potential for a public health hazard from mercury emitted from U.S. EGUs. The SAB finds the current draft document deficient, however, because of its lack of transparency in describing key analytical methods and findings. We urge the Agency to revise the document based on our recommendations.

We appreciate the opportunity to review the draft mercury risk assessment. We look forward to your response.

Sincerely,

/Signed/   */Signed/*

Dr. Deborah L. Swackhamer   Dr. Stephen M. Roberts
Chair   Chair
Science Advisory Board   SAB Mercury Review Panel

NOTICE

This report has been written as part of the activities of the EPA Science Advisory Board (SAB), a public advisory group providing extramural scientific information and advice to the Administrator and other officials of the Environmental Protection Agency. The SAB is structured to provide balanced, expert assessment of scientific matters related to problems facing the Agency. This report has not been reviewed for approval by the Agency, and, hence, the contents of this report do not necessarily represent the views and policies of the Environmental Protection Agency, nor of other agencies in the Executive Branch of the Federal government. Mention of trade names of commercial products does not constitute a recommendation for use. Reports of the SAB are posted on the EPA website at http://www.epa.gov/sab.

Dr. Jana Milford, Professor, Department of Mechanical Engineering, University of Colorado, Boulder, CO

Dr. M. Christopher Newland, Alumni Professor, Department of Psychology, Auburn University, Auburn, AL

Dr. Nicholas Ralston, Human Health Effects Research Group Leader, Energy & Environmental Research Center (EERC), University of North Dakota , Grand Forks, ND

Dr. Stephen L. Rathbun, Professor of Biostatistics, Department of Epidemiology and Biostatistics, University of Georgia, Athens, GA

Dr. Eric P. Smith, Professor, Department of Statistics, 406A Hutcheson Hall , Virginia Polytechnic Institute and State University, Blacksburgh, VA

Dr. Alan Stern, Section Chief-Risk Assessment/ Adjunct Associate Professor, Division of Science, Research & Technology/Dept. of Environmental & Occupational Health, New Jersey Department of Environmental Protection/University of Medicine and Dentistry of New Jersey-Robert Wood Johnson Medical School., Trenton, NJ (Affiliation for identification purposes only)

Dr. Edward Swain, Research Scientist, Minnesota Pollution Control Agency, Saint Paul, MN

Dr. Edwin van Wijngaarden, Associate Professor, Community and Preventive Medicine, Environmental Medicine, and Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, NY

Dr. Robert Wright, Associate Professor, Pediatrics, Division of Environmental Health, Harvard School of Public Health, Boston, MA

SCIENCE ADVISORY BOARD STAFF
Dr. Angela Nugent, Designated Federal Officer, U.S. Environmental Protection Agency, Science Advisory Board (1400R), 1200 Pennsylvania Avenue, NW, Washington, DC, Phone: 202-564-2218, Fax: 202-565-2098, (nugent.angela@epa.gov)

Dr. John P. Giesy, Professor and Canada Research Chair, Veterinary Biomedical Sciences and Toxicology Centre, University of Saskatchewan, Saskatoon, Saskatchewan, Canada

Dr. Jeffrey Griffiths, Associate Professor, Department of Public Health and Community Medicine, School of Medicine, Tufts University, Boston, MA

Dr. James K. Hammitt, Professor, Center for Risk Analysis, Harvard University, Boston, MA

Dr. Bernd Kahn, Professor Emeritus and Associate Director, Environmental Radiation Center, Georgia Institute of Technology, Atlanta, GA

Dr. Agnes Kane, Professor and Chair, Department of Pathology and Laboratory Medicine, Brown University, Providence, RI

Dr. Madhu Khanna, Professor, Department of Agricultural and Consumer Economics, University of Illinois at Urbana-Champaign, Urbana, IL

Dr. Nancy K. Kim, Senior Executive, Health Research, Inc., Troy, NY

Dr. Kai Lee, Program Officer, Conservation and Science Program, David & Lucile Packard Foundation, Los Altos, CA (affiliation listed for identification purposes only)

Dr. Cecil Lue-Hing, President, Cecil Lue-Hing & Assoc. Inc., Burr Ridge, IL

Dr. Floyd Malveaux, Executive Director, Merck Childhood Asthma Network, Inc., Washington, DC

Dr. Lee D. McMullen, Water Resources Practice Leader, Snyder & Associates, Inc., Ankeny, IA

Dr. Judith L. Meyer, Professor Emeritus, Odum School of Ecology, University of Georgia, Lopez Island, WA

Dr. James R. Mihelcic, Professor, Civil and Environmental Engineering, State of Florida 21st Century World Class Scholar, University of South Florida, Tampa, FL

Dr. Jana Milford, Professor, Department of Mechanical Engineering, University of Colorado, Boulder, CO

Dr. Christine Moe, Eugene J. Gangarosa Professor, Hubert Department of Global Health, Rollins School of Public Health, Emory University, Atlanta, GA

Dr. Horace Moo-Young, Dean and Professor, College of Engineering, Computer Science, and Technology, California State University, Los Angeles, CA

Dr. Eileen Murphy, Grants Facilitator, Ernest Mario School of Pharmacy, Rutgers University, Piscataway, NJ

Dr. Duncan Patten, Research Professor, Hydroecology Research Program , Department of Land Resources and Environmental Sciences, Montana State University, Bozeman, MT

Dr. Stephen Polasky, Fesler-Lampert Professor of Ecological/Environmental Economics, Department of Applied Economics, University of Minnesota, St. Paul, MN

Dr. Arden Pope, Professor, Department of Economics, Brigham Young University, Provo, UT

Dr. Stephen M. Roberts, Professor, Department of Physiological Sciences, Director, Center for Environmental and Human Toxicology, University of Florida, Gainesville, FL

Dr. Amanda Rodewald, Professor of Wildlife Ecology, School of Environment and Natural Resources, The Ohio State University, Columbus, OH

Dr. Jonathan M. Samet, Professor and Flora L. Thornton Chair, Department of Preventive Medicine, University of Southern California, Los Angeles, CA

Dr. James Sanders, Director and Professor, Skidaway Institute of Oceanography, Savannah, GA

Dr. Jerald Schnoor, Allen S. Henry Chair Professor, Department of Civil and Environmental Engineering, Co-Director, Center for Global and Regional Environmental Research, University of Iowa, Iowa City, IA

Dr. Kathleen Segerson, Philip E. Austin Professor of Economics , Department of Economics, University of Connecticut, Storrs, CT

Dr. Herman Taylor, Director, Principal Investigator, Jackson Heart Study, University of Mississippi Medical Center, Jackson, MS

Dr. Barton H. (Buzz) Thompson, Jr., Robert E. Paradise Professor of Natural Resources Law at the Stanford Law School and Perry L. McCarty Director, Woods Institute for the Environment, Stanford University, Stanford, CA

Dr. Paige Tolbert, Professor and Chair, Department of Environmental Health, Rollins School of Public Health, Emory University, Atlanta, GA

Dr. John Vena, Professor and Department Head, Department of Epidemiology and Biostatistics, College of Public Health, University of Georgia, Athens, GA

Dr. Thomas S. Wallsten, Professor and Chair, Department of Psychology, University of Maryland, College Park, MD

Dr. Robert Watts, Professor of Mechanical Engineering Emeritus, Tulane University, Annapolis, MD

Dr. R. Thomas Zoeller, Professor, Department of Biology, University of Massachusetts, Amherst, MA

SCIENCE ADVISORY BOARD STAFF
Dr. Angela Nugent, Designated Federal Officer, U.S. Environmental Protection Agency, Science Advisory Board (1400R), 1200 Pennsylvania Avenue, NW, Washington, DC, Phone: 202-564-2218, Fax: 202-565-2098, (nugent.angela@epa.gov)

Table of Contents

ACRONYMS, ABBREVIATIONS AND DEFINITIONS OF TERMS

CASAC	Clean Air Scientific Advisory Committee
CMAQ	Community Multiscale Air Quality Modeling System
EGU	Electrical Generating Unit
EPA	Environmental Protection Agency
GEOS-Chem	A global 3-D chemical transport model (CTM) for atmospheric composition driven by meteorological input from the Goddard Earth Observing System (GEOS) of the NASA Global Modeling and Assimilation Office.
HAP	Hazardous Air Pollutant
Hg	Mercury
HQ	Hazard Quotient
HUC	Hydrologic Unit Codes
IQ	Intelligence Quotient
MDN	Mercury Deposition Network
MeHg	Methylmercury
MMAP	Mercury Maps
PUFA	Polyunsaturated Fatty Acid
RfD	Reference Dose
R-MCM	Regional Mercury Cycling Model
SAB	Science Advisory Board
TRI	Toxic Release Inventory
TSD	Technical Support Document

1. Executive Summary

EPA has proposed National Emission Standards for Hazardous Air Pollutants for coal- and oil-fired Electric Utility Steam Generating Units (EGUs). These proposed standards would require EGUs to decrease emissions of mercury and other hazardous air pollutants (HAP). In order to regulate HAP emissions under the Clean Air Act, Section 112(b), the Agency must make a determination that such regulation is appropriate and necessary based upon a study of the hazards to public health reasonably anticipated from HAP emissions. As part of this determination, hazards to public health from U.S. EGU mercury emissions are evaluated in a draft national-scale risk assessment entitled *Technical Support Document: National-Scale Mercury Risk Assessment Supporting the Appropriate and Necessary Finding for Coal and Oil-Fired Electric Generating Units (March 2011).* This SAB report uses the terms "risk assessment" and "Technical Support Document" interchangeably to refer to EPA's draft document.

The draft risk assessment considers hazards from mercury released from U.S. EGUs and deposited in watersheds within the continental United States. Mercury deposition is estimated using the Community Multi-scale Air Quality (CMAQ) model for watersheds classified using 12-digit Hydrologic Unit Codes (HUC12). The risk assessment focuses on hazard from consumption of methylmercury in self-caught fish, specifically hazard to children born to women who consume local fresh water fish in a subsistence manner. Exposure from fish consumption is estimated for watersheds with data on methylmercury concentrations in fish tissue, and a hazard quotient (HQ) is calculated based upon the current reference dose (RfD) for methylmercury. The contribution of U.S. EGUs to the HQ for each watershed is calculated by comparing U.S. EGU deposition rates with total deposition to the watershed, including other sources, assuming that the contribution of U.S. EGUs to fish tissue concentrations and risk is proportional to their contribution to total deposition. Intelligence Quotient (IQ) loss is also modeled as a health endpoint, with a loss of one or more points from methylmercury exposure considered as a public health concern. Estimated hazards associated with U.S. EGU emissions in 2005 are compared with estimated hazards expected to remain in 2016 "after imposition of the requirements of the Act."

The SAB Mercury Review Panel was asked to comment on the draft risk assessment, including the overall design and approach as well as various technical aspects. The Panel was also asked to comment on the extent to which specific "observations" or conclusions in the risk assessment are supported by the analytical results. During the course of deliberations, the Panel reviewed background materials provided by the Office of Air Quality Planning and Standards, as well as public comments on the topic. The SAB reviewed and approved the report of the Panel. EPA asked the SAB to address fourteen charge questions, many with multiple parts. This Executive Summary highlights the main findings. Detailed responses to the individual charge questions are provided in the body of the report.

The SAB finds the Technical Support Document to lack critical details regarding both the methods used and the results presented. This made the document difficult to review and, in the view of the SAB, unsuitable in its present form to fully support Agency decision-making. Presentations and information provided by Agency representatives helped the SAB understand how the risk assessment was conducted, the rationale for some of the decisions made in approach and the use of data and the translation of the results. With this additional information, the SAB views the risk assessment favorably, concluding that it is able to provide an objective, reasonable, and credible determination of the potential for a public health hazard from mercury emitted from U.S. EGUs. However, the SAB considers the integrity of the risk assessment to be dependent in part on a transparent description of the analysis, and the Technical Support Document needs to be strengthened to provide this description.

This review is based on the text of the report and the additional information provided by the above cited presentations and discussion provided by EPA representatives. Responses to charge questions indicate where improvements need to be made, and a summary of the most critical recommendations is provided in section 4. The SAB's support for the risk assessment is contingent on its recommendations being fully considered in the revision of the assessment.

Overall design

The Panel finds the overall design and general approach used in the risk assessment to be scientifically credible. The Technical Support document, however, needs a more detailed description of the modeling methods and data sources. The report's introduction should make clear from the start that the analysis is a determination of potential exposure at the scale of watersheds.

Critical health endpoints besides IQ loss; use of an IQ loss metric benchmark; and concentration-response function used in modeling IQ loss

The SAB supports the use of the HQ approach in the risk assessment. SAB members agree that because the RfD from which the HQ is calculated is an integrative metric of neurodevelopmental effects of methylmercury, it constitutes a reasonable basis for assessing risk. Other potential health endpoints were also considered by the SAB. The SAB notes that a number of measures of potential neurodevelopmental effects of methylmercury exist, some of which have greater sensitivity to differential mercury exposure than does IQ loss. However, none are viewed by the SAB as suitable for quantitative risk estimation with a reasonable degree of scientific certainty at the present time, and consequently none are recommended for incorporation into the analysis. The SAB does not consider it appropriate for EPA to use IQ loss in the risk assessment and recommends that this aspect of the analysis be de-emphasized, moving it to an appendix where IQ loss is discussed along with other possible endpoints not included in the primary assessment.

While the SAB agrees that the concentration-response function for IQ loss used in the risk assessment has validity, IQ loss is not a sensitive response endpoint for methylmercury and its use likely underestimates the impact of reducing methylmercury in water bodies. The SAB agrees that if IQ loss were retained in the risk assessment despite these reservations, a loss of one or two points on average in a population would be an appropriate benchmark. The SAB agrees that fish nutrients (e.g., omega-3 fatty acids) can potentially ameliorate neurologic effects associated with methylmercury, but there is not sufficient information to recommend a quantitative adjustment in health endpoint measures. However, the SAB agrees that because the RfD from which the HQ is calculated is an integrative metric of risk, it constitutes a reasonable basis for assessing risk. The RfD is an integrative measure because it considers the weight of the evidence and determines a quantitative value that is based on the most sensitive endpoints across multiple studies and endpoints.

Spatial scale of watersheds

The SAB agrees that HUC12 watersheds provide the appropriate level of spatial resolution and offer advantages over previous assessments at lower resolution (e.g., HUC8). The comparability of this scale to CMAQ output makes the transferability and applicability of deposition modeling to the watershed scientifically robust. Further, the finer resolution of HUC12 watersheds is better suited to follow deposition patterns of a single source such as an EGU and increases the likelihood that measured deposition within a watershed is homogeneous. The SAB notes that one disadvantage of smaller

watershed size is that, within a given watershed, the number of fish samples with methylmercury data is diminished. During the public meeting, the SAB questioned some of the figures with maps showing modeled deposition across the United States. Some areas showed intense deposition with no obvious source, leading SAB members to question the accuracy of the modeling or data presentation in the March draft report. EPA provided clarification and updated maps in July 2011 (Pekar 2011). The SAB supports EPA's plans to include these updated mercury deposition maps in the revised report so they correctly reflect total annual mercury deposition per square meter by watershed.

Measured fish tissue mercury concentrations

The SAB agrees that fish tissue methylmercury data are an appropriate basis to estimate the number and percentage of fish-sampled watersheds where populations may be at risk. Although fish data were only available for 2,461 HUC12 watersheds out of 88,000 HUC12 watersheds in the continental United States, this is viewed as sufficient to estimate the number and percentage of fish-sampled watersheds where populations may be at risk. The SAB notes advantages and disadvantages of the Agency decision to limit fish tissue concentration data to the period after 1999 but agrees with this approach, given that older data might not be representative of conditions during the 2005 reference deposition year. The SAB is concerned about the absence of fish tissue data from some watersheds with higher levels of mercury deposition. The EPA is encouraged to contact states with these watersheds to determine whether additional fish tissue data are available to improve coverage of the analysis. The SAB discussed the use of modeling to estimate fish methylmercury concentrations as a means to include more watersheds. With further development, this approach could be used for a national scale assessment such as this in the future but the SAB does not recommend it for the current assessment.

Use of the 75th percentile fish tissue methylmercury value

As a means of selecting methymercury fish concentrations representative of larger, but not the largest, edible fish, the risk assessment uses the 75th percentile fish concentration for watersheds with one or more fish concentration value. The SAB considers this percentile reasonable but is concerned that over half of the watersheds in the assessment have only four or fewer fish samples with methylmercury concentration, and a significant number of these have a single fish sample. The SAB notes that in watersheds where only a few fish samples are available, the 75th percentile concentration and exposure most likely will be underestimated. This should be explained in the report, and a sensitivity analysis should be conducted using the median fish tissue concentration to better represent the distribution of concentrations when the sample size is only one fish. The SAB also recommends that the report describe the sources of fish methylmercury concentration data more fully, including the state sampling programs that provide most of the data. Discussion of sampling programs should include the types and sizes of fish obtained, as well as uncertainties associated with this data set, to improve the transparency of the analysis.

Consumption rates and location for high-end consumers

The SAB finds that the consumption rates and locations for fishing activity for likely highly exposed consumers, i.e., self-caught fish consuming populations modeled in the analysis, are supported by the data presented in the document and are generally reasonable and appropriate given the available data. A diverse range of susceptible populations is represented in the assessment. There are caveats, however, associated with the sources of fish consumption data, the data sets selected for inclusion, and the

suitability of data for inclusion in the risk assessment (e.g., in terms of providing annual average intakes of the edible portion of the fish) that should be acknowledged more fully in the document.

Use of Census data to identify high-end fish consuming populations

The SAB agrees that the criterion of using at least 25 persons per census tract from a given target subsistence fisher population is a reasonable approach to identify watersheds with potentially highly exposed fish consuming populations. While other approaches are possible, none is viewed as being more effective or feasible. The SAB recommends that the document clarify how many watersheds were eliminated due to this inclusion criterion.

Mercury Maps approach

The SAB agrees with the Mercury Maps approach used in the analysis and has cited additional work that supports a linear relationship between mercury loading and accumulation in aquatic biota. These studies suggest that mercury deposited directly to aquatic ecosystems can become quickly available to biota and accumulated in fish, and reductions in atmospheric mercury deposition should lead to decreases in methylmercury concentrations in biota. The SAB notes other modeling tools are available to link deposition to fish concentrations, but does not consider them to be superior for this analysis or recommend their use. The integration of Community Multiscale Air Quality Modeling System (CMAQ) deposition modeling to produce estimates of changes in fish tissue concentrations is considered to be sound. Although the SAB is generally satisfied with the presentation of uncertainties and limitations associated with the application of the Mercury Maps approach in qualitative terms, it recommends that the document include quantitative estimates of uncertainty available in the existing literature.

Exclusion of watersheds with significant non-air loadings

In order to reduce uncertainty associated with the Mercury Maps approach, watersheds with significant non-air loadings of mercury are excluded from the analysis. The SAB agrees with the exclusion criteria used by the Agency. Additional exclusion criteria were discussed, but their application would be unlikely to substantially change the results of the assessment. The SAB also recommends that the EPA provide additional discussion of uncertainties in the mercury emissions from U.S. EGUs and non-EGU sources and the implications of these uncertainties.

Uncertainty and variability and discussion of analytical results

The SAB discussed the characterization of variability and uncertainty in the Technical Support Document in detail. Sources of variability and uncertainty in the assessment are summarized in Appendix F of the draft document. The qualitative nature of this presentation is considered appropriate, but the identification of important sources of variability and uncertainty is considered incomplete. Inclusion of several additional sources of variability and uncertainty is recommended. The SAB notes that the degree of uncertainty associated with the analysis is consistent with a screening level analysis, and despite the various sources of uncertainty inherent in the approach, the analysis is sound and reasonable.

The SAB finds that observations in five areas (mercury deposition from U.S. EGUs, fish tissue methylmercury concentrations, patterns of mercury deposition with mercury fish tissue data, percentile risk estimates, and number and frequency of watersheds with populations potentially at risk due to U.S.

EGU mercury emissions) are generally supported by the analytical results presented in the document. However, there are many examples where results are poorly presented, and in most areas the uncertainties, variability and data limitations are not well characterized. The SAB has numerous specific recommendations to improve presentation of findings and observations.

Responsiveness to the goals of the study

The section of the document on Summary of Key Observations does not encapsulate well the critical issues and significant results of the analysis. The SAB recommends revising this section to link back directly with the goals of the studies as articulated on Page 13 of the document, i.e.: (a) what is the nature and magnitude of the potential risk to public health posed by current U.S. EGU mercury emissions? (b) what is the nature and magnitude of the potential risk posed by U.S. EGU mercury emissions in 2016 considering potential reductions in EGU Hg emissions attributable to CAA (Clean Air Act) requirements? and (c) how is risk estimated for both the current and future scenario apportioned between the incremental contribution from U.S. EGUs and other sources of mercury?

2.   Introduction

EPA's Office of Air and Radiation requested peer review of a *Technical Support Document: National-Scale Mercury Risk Assessment Supporting the Appropriate and Necessary Finding for Coal and Oil-Fired Electric Generating Units - March 2011*, developed to support a proposed rule published in the Federal Register on March 16, 2011 to regulate emissions of hazardous air pollutants from for coal- and oil-fired Electric Utility Steam Generating Units (EGUs). Section 112(n)(1) of the Clean Air Act requires EPA to determine whether it is "appropriate and necessary" to regulate hazardous air pollutants emissions from EGUs under section 112. The "appropriate and necessary" finding requires EPA to perform a study of the hazards to public health reasonably anticipated to occur as a result of hazardous air pollutant emissions, including mercury.

The Science Advisory Board formed an expert *ad hoc* Panel to peer review the draft Technical Document. The Panel addressed fourteen Agency charge questions (see Appendix A) and developed the responses below. The Panel held a public meeting on June 15-17, 2011 to peer review this document and held a public teleconference on July 20, 2011 to discuss the Panel's draft report. The chartered SAB held a quality review to approve the draft report on September 7, 2011.

The SAB had difficulty evaluating the Technical Support Document because it lacked critical details. During the public meeting, presentations and information provided by Agency representatives helped the SAB understand technical aspects of the analysis. With this additional information and clarification, the SAB views the risk assessment positively. However, the SAB considers the integrity of the risk assessment as dependent in part on a transparent description of the methods and findings. The Technical Support Document needs to better explain what was done and why, translate the results into findings that relate to the key goals of the analysis and describe where the uncertainties lie. The SAB's support for the risk assessment is contingent upon a development of a revised document that addresses these issues.

The body of this report is organized to respond to each of EPA's charge questions. Section 3 provides responses and specific suggestions and recommendations for revising the Technical Support Document. Because this SAB report provides many recommendations for strengthening the Technical Support Document, Section 4 provides a list of the specific recommendations made by the SAB.

3. Response to Charge Questions

3.1. Overall design

Question 1: Please comment on the scientific credibility of the overall design of the mercury risk assessment as an approach to characterize human health exposure and risk associated with U.S. EGU mercury emissions (with a focus on those more highly exposed).

Response: The SAB finds that the overall design and general approach used in the assessment are scientifically credible.

The overall approach used in the study is to estimate potential risk at a national scale, attributable to mercury released from U.S. EGUs and deposited to inland waterbodies, for recent (2005) and future (2016) emissions levels. To accomplish this, the analysis links a series of models and data to estimate methylmercury exposure via fish consumption and then compares the exposure with a toxicological benchmark. The series of models allows for the estimation of deposition of mercury emitted by U.S. EGUs into watersheds. The assessment uses measured concentrations of methylmercury in fish tissue samples, as well as estimates of future fish methylmercury concentrations, to estimate the number and percentage of watersheds where populations may be at risk. Human exposure and potential health effects in these at-risk watersheds are then assessed through the pathway of ingestion of self-caught fish from inland water bodies for vulnerable subsistence fisher populations.

Although the overall design and general approach are scientifically credible, the SAB has a number of suggestions and recommendations for enhancing the assessment, based on review of the draft *Technical Support Document* and supplementary presentations and information provided by EPA. The responses to the charge questions below provide those recommendations and suggestions in detail. It will be important for EPA to address these issues. The Technical Support Document would benefit from a more detailed description of the modeling methods and data sources, and results need to be presented more clearly. The Introductory section should make clear, at the earliest possible point, that the analysis is a determination of watershed impact with exposure addressed as a potential outcome. Despite weaknesses in the Technical Support Document and uncertainties inherent in an analysis such as this, the SAB agrees that the risk assessment makes an objective and reasonable determination of the potential for a public health hazard from mercury emitted from U.S. EGUs.

3.2. Critical health endpoints besides IQ loss

Question 2: Are there any additional critical health endpoint(s) besides IQ loss which could be quantitatively estimated with a reasonable degree of confidence to supplement the mercury risk assessment (see section 1.2 of the Mercury Risk TSD for an overview of the risk metrics used in the risk assessment)?

Response: This charge question raises issues about the use of IQ, as well as use of alternative quantitative measures. While several alternative approaches were discussed that might supplement IQ scores, no substitute can be quantitatively estimated with a "reasonable degree of confidence." Moreover, there are doubts that IQ met this standard. The response to this charge question addresses both of these issues below.

<u>Use of IQ.</u> There are significant concerns about the use of IQ for identifying the impact of consuming fish from water bodies with unacceptable levels of methylmercury because IQ will likely result in an underestimation of potential neurobehavioral impacts, compared to analyses using the hazard quotient (HQ). Thus, the SAB considers HQ to be a stronger basis for evaluation of methylmercury hazard. The HQ is based upon the methylmercury reference dose (RfD), which is an integrative measure reflecting a range of neurobehavioral effects, and it incorporates pharmacokinetic variability. The RfD considers the weight of the evidence and determines a quantitative value that is based on the most sensitive endpoints across multiple studies and endpoints. Sensitive endpoints, in this context, are adverse effects that occur at the lowest exposures.

In contrast, the loss of IQ points is likely to underestimate the impact of reducing methyl mercury in water bodies. The reason is that IQ score has not been the most sensitive indicator of methylmercury's neurotoxicity in the populations studied. As noted in the Technical Support Document, in the Faroe Island study the most sensitive indicators were in the domains of language (Boston Naming Test), attention (continuous performance) and memory (California Verbal Learning Test). These two tests are neuropsychological tests that are not subtests of IQ tests and whose relationship with global IQ is not well characterized. In the Seychelles study, the Psychomotor Development Index was the most sensitive measure and, while this index is a component of the Bailey Scales of Infant Development, it is not highly correlated with cognitive measures (Davidson et al. 2008).

Additionally, the use of IQ, or any neuropsychological measure, distracts from the main goal of the document. The analysis in the document emphasizes the number of fish-sampled water bodies from which subsistence fishers would be at risk based on an elevated HQ. As is clear in Tables 2-9 to 2-11 in the Technical Support Document, an analysis based on IQ identifies far fewer water bodies than one based on the HQ. This is because IQ underestimates hazard, as noted above.

The SAB recommends that EPA reframe the document's discussion of IQ. EPA should incorporate IQ and other neuropsychological measures as supplemental information and focus on HQ as the primary critical health endpoint. It is not suggested that the analyses of IQ be removed altogether but rather that the analyses be framed in an appendix to the report as a secondary analyses of impact of reduced exposure on potential health-related outcome. The appendix should discuss the basis for selecting a HQ at or above 1.5 as the criteria for selecting potentially impacted watersheds should be explained. The appendix should also include discussion of potential effects on other measures like developmental delays (Grandjean et al. 1997) or neuropsychological tests (as discussed by van Wijngaarden et al. 2006), presented in the overall context of the weight of evidence.

Alternative quantitative measures. One alternative is developmental delay as described by Grandjean et al. (1997). Here, an estimate of the number of months of delay in verbal skills as tapped by the Boston Naming Test or in learning and short-term memory as tapped by the California Verbal Learning Test was made based on regression coefficients describing the relationship among age, methylmercury exposure, and scores on these tests. The delays were on the order of five to seven months associated with a 10-fold increase in cord blood methylmercury.

A recent analysis by van Wijngaarden et al. (2006) derived Benchmark Dose Level-Lower 95% confidence interval values for 26 endpoints, including IQ and other neuropsychological measures from the nine-year follow up of the Seychelles child development study main cohort. This paper could be cited in a discussion of markers of health impacts of lowering mercury deposition and reducing intake by subsistence fishers.

One SAB member suggests the use of blood markers of selenium-dependent enzyme function, noting that methylmercury irreversibly inhibits selenium-dependent enzymes that are required to support vital-but-vulnerable metabolic pathways in the brain and endocrine system. Impaired selenoenzyme activities would be observed in the blood before they would be observed in brain, but the effect is also expected to be transitory. The use of these measures is a minority view among the SAB members.

The SAB recommends that the Technical Support Document acknowledge and discuss alternative quantitative measures but does not recommend a re-analysis based on these measures.

3.3. **Use of an IQ loss metric benchmark**

Question 3: Please comment on the benchmark used for identifying a potentially significant public health impact in the context of interpreting the IQ loss risk metric (i.e., an IQ loss of 1 to 2 points or more representing a potential public health hazard). Is there any scientifically credible alternate decrement in IQ that should be considered as a benchmark to guide interpretation of the IQ risk estimates (see section 1.2 of the Mercury Risk TSD for additional detail on the benchmark used for interpreting the IQ loss estimates).

Response: The consensus is that if IQ were to be used, then a loss of 1 or 2 points as a population average is a credible decrement to use for this risk assessment. This metric seems to be derived from the lead literature and was peer-reviewed by the Clean Air Scientific Advisory Committee (U.S. EPA CASAC 2007). While its applicability to methylmercury is questionable, the size of the decrement is justified based on the extensive analyses available from the literature reviewed by CASAC. The support for the model of the relationship between IQ and methylmercury exposure comes from Axelrad and Bellinger (2007) and from a whitepaper produced by Bellinger (2005).

The analysis in Table 2-10 showing the effect of using a one- or two -point loss is helpful in evaluating the sensitivity of this measure to the magnitude of the decrement.

3.4. **Spatial scale of watersheds**

Question 4: Please comment on the spatial scale used in defining watersheds that formed the basis for risk estimates generated for the analysis (i.e., use of 12-digit hydrologic unit code classification). To what extent do HUC12 watersheds capture the appropriate level of spatial resolution in the relationship between changes in mercury deposition and changes in MeHg fish tissue levels? (see section 1.3 and Appendix A of the Mercury Risk TSD for additional detail on specifying the spatial scale of watersheds used in the analysis).

Response: The choice of using the HUC12 (Hydrologic Unit Code) watershed delineation of the contiguous 48 United States for this risk assessment is more appropriate and offers at least two distinct advantages over the 2001 Mercury Maps study report that employed the larger-scale HUC8 delineation. First, HUC8s are "cataloguing units" delineation and do not actually represent true watersheds (areas of land where surface water drainage accumulates to an outflow location). Instead, many HUC8 areas have flow lines that cross the unit boundaries, thus making this larger scale delineation not technically correct for any mass accounting procedure like Mercury Maps. The use of HUC12s, which are true watershed delineations, does not violate this mass accounting assumption. A second strength of the use of HUC12's is that they have a similar physical scale to the spatial resolution of the CMAQ output (12 km CMAQ square grid compared to the HUC12 watersheds that are typically about 5-10 km on a side). Comparable scales make the transferability and applicability of deposition modeling to the watershed

more scientifically robust. The use of finer scale watersheds enables modeling and deposition runs that have the detail to follow deposition patterns from a single source, including EGUs. The fine-scale watershed resolution decreases the likelihood that there is a significant deposition gradient within the HUC. Further, the relative biogeochemical and ecological homogeneity of an individual HUC12 watershed allows better validity for ascribing fish concentrations to a specific watershed and that those fish will respond in proportion to changes in atmospheric mercury deposition. The SAB notes, however, that one potential disadvantage of HUC12 is that a number of HUC12 watersheds contain a very limited number of fish samples because of their inherent small size, but other factors described in this response override this concern.

The Technical Support document acknowledges and this SAB agrees that the fish distribution data are highly skewed toward the Eastern United States. That said, the legend of Figure 2-6 in the Technical Support Document indicates that 2,170 out of 2,461 watersheds were from the Eastern United States, leaving approximately 300 samples from Western sites. Given the apparent distribution of high deposition zones in CMAQ modeling runs displayed in Figures 2-1 and 2-2 in the Technical Support Document that are not ground-truthed in Mercury Deposition Network deposition measurement, the SAB is concerned not only about the reality of the identified intense deposition zones (i.e., whether they are truly intense deposition zones, for example, in the state of Nevada), but also whether these watersheds were included in this report's analysis. Fish distribution data appear to overlap with some of these zones of modeled high mercury deposition, and, with 300 fish samples from the Western United States, there is a high probability for overlap.

The SAB is concerned about the possibility that in some watersheds, multiple small lakes may be included within a single HUC12. In some cases, lakes within a small geographic zone have been shown to have quite different chemistry and biological productivity. For instance, within Voyageurs National Park in northern Minnesota, the mercury content of similarly-sized fish of a given species in about 20 lakes varies by a factor of 10 (Wiener et al. 2006), indicating that even lakes near each other can bioaccumulate mercury to greatly differing degrees. In HUCs with multiple lakes, the SAB recommends against using a single fish methylmercury value to describe the HUC. In response to this concern and other charge questions, the SAB recommends that the authors provide a summary table describing the characteristics of the watersheds where fish were collected, including the fraction of fish samples collected from rivers versus lakes, and whether from single or multiple sites.

3.5. **Measured fish tissue mercury concentrations**

Question 5: Please comment on the extent to which the fish tissue data used as the basis for the risk assessment are appropriate and sufficient given the goals of the analysis. Please comment on the extent to which focusing on data from the period after 1999 increases confidence that the fish tissue data used are more likely to reflect more contemporaneous patterns of mercury deposition and less likely to reflect earlier patterns of mercury deposition. Are there any additional sources of fish tissue MeHg data that would be appropriate for inclusion in the risk assessment?

Response: The measured fish tissue data serve as an appropriate basis for the mercury risk assessment because they are widely available and reflect the actual environmental conditions that influence fish methylmercury concentrations and human exposure to methylmercury by the target populations. The SAB notes that the relevant form of mercury in fish tissue for this risk assessment is methylmercury, but there is sometimes ambiguity as to the mercury form actually measured in surveys from which the fish tissue data were taken. Many surveys measure total mercury and assume all mercury present in fish is in

the methyl form. Although empirical data available are largely supportive of this assumption, the Technical Support Document needs to clearly acknowledge this aspect of the fish tissue data.

While it is always desirable to have a larger sample size, the sample size of 2,461 HUC12 watersheds is adequate for the goals of the risk assessment. However, as detailed below, the SAB is concerned about the sources of bias and uncertainty resulting from the state sampling designs used to select watersheds where fish tissue samples were obtained. For purposes of hazard assessment, it is reasonable to have an over-representation of HUC12s in the eastern part of the country given the prevalence of EGUs in the East. However, the description of the character of the data, as well as the selection of analyzable data (e.g., sizes, distribution of fish sizes across watersheds), should be better detailed in the report.

There are advantages and disadvantages to using fish methylmercury data prior to 1999 for the risk assessment. The advantage is that considerable fish data were obtained prior to 1999 and the use of these data could increase the information available for the national risk assessment. The disadvantage is that fish methylmercury concentrations may have changed since 1999 and these older data may not be representative of conditions during the 2005 reference deposition year. Unfortunately, there are few high quality time series data of fish methylmercury concentrations, so it is difficult to quantify the extent to which fish methylmercury concentrations have changed since the 1990s. As a result, the SAB recommends that the EPA utilize fish methylmercury data collected since 1999 for the risk assessment.

Given the spatial distribution of mercury deposition from EGUs and the density of fish methylmercury measurements (Figure 2-15), there are some states that receive what the Technical Support Document terms "relatively elevated" mercury deposition from U.S. EGU emissions and have limited fish methylmercury measurements. These states include Pennsylvania, New Jersey, Kentucky and Illinois. The SAB suggests that the EPA contact these states to investigate if additional recent (since 1999) fish methylmercury data are available to improve the coverage for the mercury risk assessment. For example, the Pennsylvania Department of Environmental Protection, Pennsylvania Fish Monitoring Program has 700 sites for the measurement of the methylmercury content of recreational sport fish, with samples collected from 1979-2007.

EPA's reliance on the National Listing of Fish Advisory and U.S. Geological Survey (USGS) compilation of methylmercury data sets contributes to uncertainty because these data were collected by state agencies with various sampling designs and state protocols. Most of the data are not from probability-based sampling designs, so it is not entirely clear what population the fish tissue samples represent. The direction of impact on the risk assessment of this variation in sampling designs cannot be ascertained. Moreover, some states have greater sampling efforts than others. Particularly strong sampling efforts were observed in South Carolina, Louisiana, Indiana, Iowa, West Virginia and Virginia. As a consequence of this variability in fish-tissue sampling effort, the risk assessment will be strongly influenced by states with high sampling efforts. Moreover, Figure 2-18 suggests that the sample is biased in favor of watersheds with higher mercury deposition and higher EGU-attributable deposition as predicted by the CMAQ model. This bias could in part be due to the over-representation of HUC12s in the East but could also occur if states with high deposition also have high fish-tissue sampling effort. Nevertheless, as per the limitations of the available data, the risk assessment focuses on that portion of the fish-sampled watersheds at risk, rather than attempting to make inferences to the larger population of all 88,000 HUC12 watersheds.

Researchers have developed empirical models for fish methylmercury concentrations using water chemistry and land cover data (Chen et al. 2005; Driscoll et al. 2007; and Watras et al. 1998). These

empirical relationships have been used to estimate methylmercury concentrations for different fish species at state and regional spatial scales. Such an empirical modeling approach could be used to provide more comprehensive estimates of fish methylmercury concentrations across water resources and potentially improve the extent of future mercury risk assessments. However, if this empirical modeling approach was to be used in a risk assessment such as this, it would need to be developed and evaluated at a national scale. Moreover, empirical models would contribute additional uncertainty in the estimation of fish methylmercury concentration. The SAB is not recommending that this approach be used for the current risk assessment. Rather, the EPA might consider use of empirical modeling to improve the information available related to fish methylmercury concentrations in future assessments.

To strengthen the *Technical Support Document* the SAB recommends that it be revised to provide a better description of the character of the data, as well as the selection of analyzable data (e.g., sizes, distribution of fish sizes across watersheds). The SAB also recommends that EPA contact some states that receive what the Technical Support Document terms "relatively elevated" mercury deposition from U.S. EGU emissions and have limited fish methylmercury measurements to investigate if additional recent (since 1999) fish methylmercury data are available to improve the coverage for the mercury risk assessment.

3.6. **Use of the 75th percentile fish tissue methylmercury value**

Question 6: Given the stated goal of estimating potential risks to highly exposed populations, please comment on the use of the 75th percentile fish tissue MeHg value (reflecting targeting of larger but not the largest fish for subsistence consumption) as the basis for estimating risk at each watershed. Are there scientifically credible alternatives to use of the 75th percentile in representing potential population exposures at the watershed level?

Response: Using the 75th percentile of fish tissue values as a reflection of consumption of larger, but not the largest, fish among sport and subsistence fishers is a reasonable approach and is consistent with published and unpublished data on predominant types of fish consumed. While the choice of the 75th percentile is reasonable for the estimation of the methymercury levels of consumed fish, the appropriateness of this approach depends on the data from which the value is derived. The SAB is concerned that around 29% of watersheds sampled have only one fish sample with a fish tissue methylmercury concentration available. Figure 1 below shows a plot of the number of fish tissue samples available for rivers (N= 1551 samples from rivers, 41.5% have one fish measurement) using data provided to the SAB by EPA. There is clear evidence of a very high proportion of samples with only one fish.

12
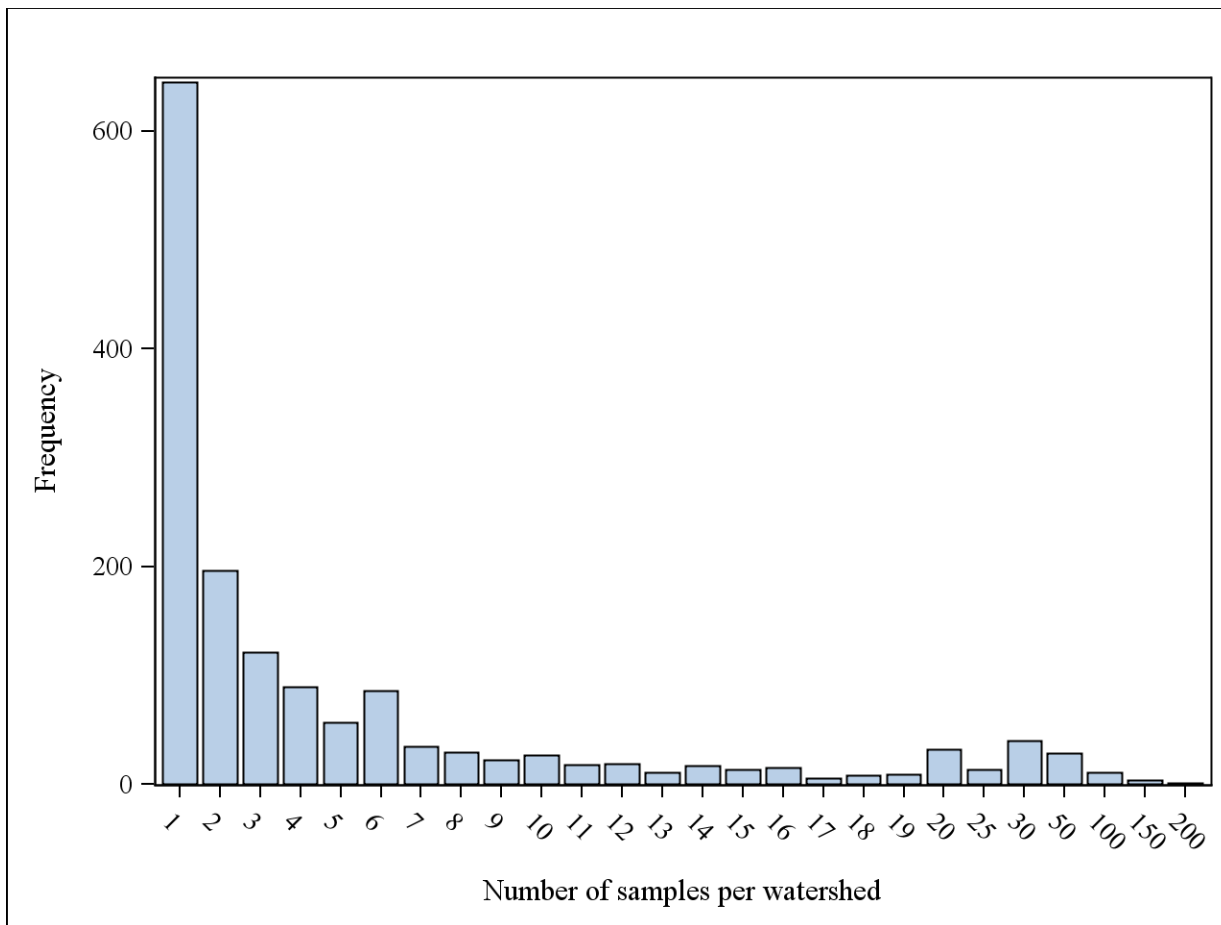
Figure 1. Frequency of fish samples of different sizes for rivers using Excel data provided to the SAB. The x axis corresponds to the number of fish tissue observations per HUC. When sample sizes are 20 or greater, a category is used i.e. 20 corresponds to 20 to 25, 25 corresponds to 26 to 30, etc.

Thus, the estimate of the 75th percentile has considerable uncertainty. The use of only one tissue value for a given watershed is likely to underestimate fish tissue levels if the single fish collected was, on average, smaller than the true 75th percentile, as would occur if the collection were random. Support for this notion is provided by Figure 2 below, which relates the 75th percentile fish tissue methylmercury concentration (on y axis) to the number of fish samples available for any given watershed. The estimate of the 75th percentile appears to increase with increasing sample size, thus suggesting that the 75th percentile fish tissue concentration for watersheds with few fish samples is underestimated.
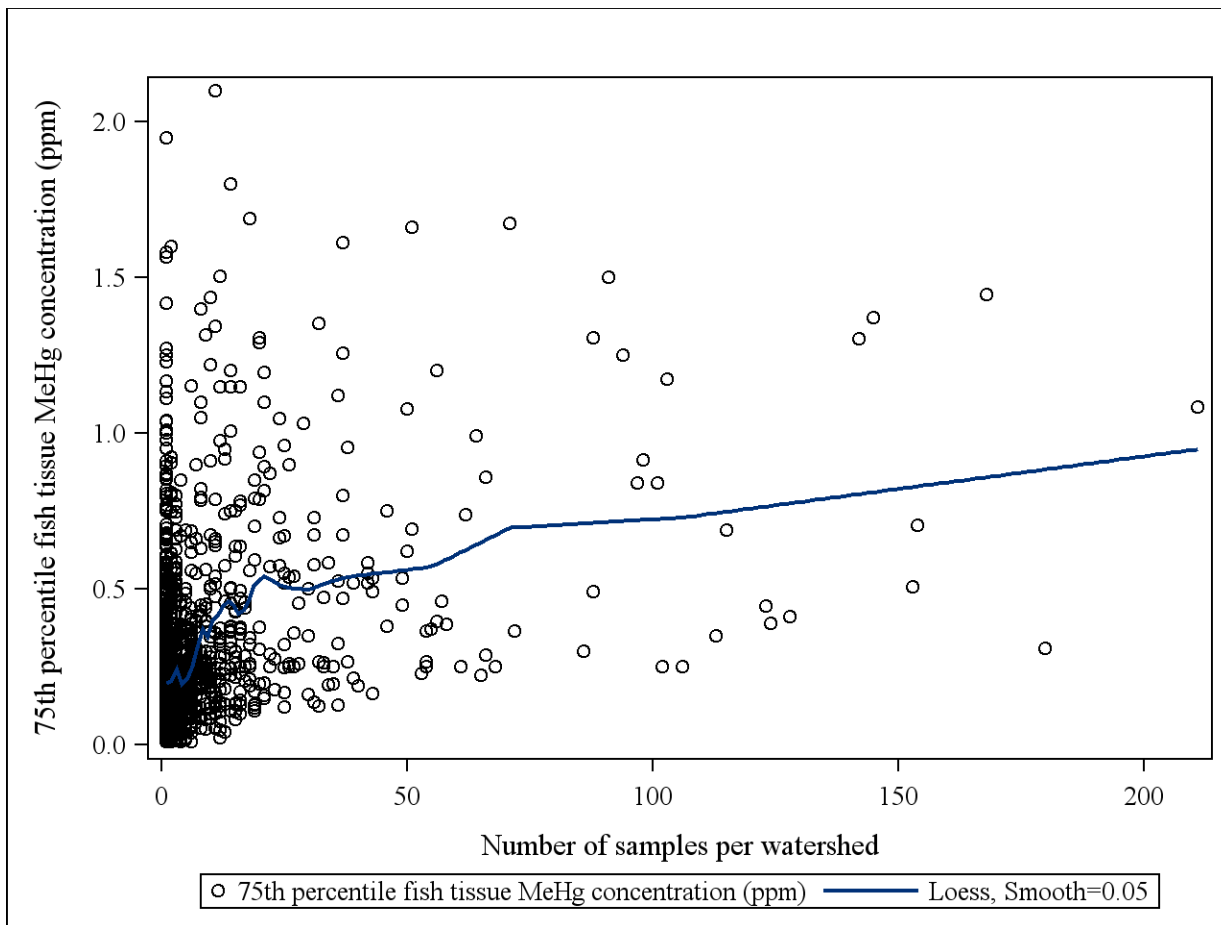
Figure 2. Comparison of 75th percentiles of fish methylmercury concentrations for fish samples across watersheds with different sample sizes, using Excel data on rivers provided to the SAB. The fitted curve is based on a Loess smoother with smoothing parameter set to 0.2.

The SAB recommends inclusion of a graph depicting the number of tissue samples available for analysis by tissue concentration. The SAB also recommends that the document discuss this source of uncertainty, including adding a table with the distribution of the number of available fish samples and the fish size from which they were obtained across watersheds to indicate the extent of the problem. The Technical Support Document should describe in more detail why including fish tissue concentrations from one fish sample is likely to result in an underestimate of the number of watersheds at risk. Furthermore, the SAB recommends that EPA should also conduct a sensitivity analysis using the median fish tissue concentration to better represent the distribution of fish tissue methylmercury levels where the sample size is one and provide a bound on the risk assessment. The use of other percentiles in the sensitivity analysis is not recommended given the limitations of the fish tissue data available.

The SAB acknowledges that fish sampling programs can result in the collection of fish sizes that can be either larger or smaller than the actual ecosystem distribution depending on sample collection methods and objectives (e.g., states may focus on collection of larger predator fish or areas where higher mercury levels tend to be found). The SAB recommends that the document describe more clearly the source of the fish methylmercury data and provide at least a general discussion of how fish sampling programs differ in ways that can contribute variability and uncertainty to the data set, such as fish capture methods and criteria for selecting fish to measure methylmercury concentrations. Given that fish sizes are likely a variable in most datasets, the report should also include information on the sizes of fish that were analyzed. In doing so, the Technical Support Document may be able to quantify the impact, if any, of

14

the size of fish sampled in watersheds with few fish tissue samples available on estimated mercury concentrations. The SAB also recommends that the Technical Support Document clarify that the 75th percentile represents available fish tissue data that may or may not represent the fish in the watershed or the fish consumed.

3.7. Consumption rates and location for high-end consumers

Question 7: Please comment on the extent to which characterization of consumption rates and the potential location for fishing activity for high-end self-caught fish consuming populations modeled in the analysis are supported by the available study data cited in the Mercury Risk TSD. In addition, please comment on the extent to which consumption rates documented in Section 1.3 and in Appendix C of the Mercury Risk TSD provide appropriate representation of high-end fish consumption by the subsistence population scenarios used in modeling exposures and risk. Are there additional data on consumption behavior in subsistence populations active at inland freshwater water bodies within the continental U.S.?

Response: The SAB finds that the consumption rates and locations for fishing activity are supported by data presented in Section 1.3 and in Appendix C of the Technical Support Document. In addition, the targeted locations and fish consumption data used in the analysis are generally appropriate and reasonable given the available data. The risk assessment uses sources that reported daily consumption for populations of low socioeconomic status African- and European-Americans females as the target population for the risk assessment. In addition, consumption rates from a study that targeted Laotian- and Vietnamese-Americans, previously identified in the central valley of California, are included in the assessment, as well as those from a study of Great Lakes tribes. Thus, a diverse range of susceptible populations is represented in the assessment.

The SAB recommends that a few caveats should be acknowledged more fully in the document. The main consumption estimates comes from a relatively small survey of individuals attending a fishing convention in South Carolina, so the consumption estimates reported in the Burger 2002 study may be imprecise, in particular for women. The SAB recommends that the Technical Support Document acknowledge that, while several estimates of fish consumption rates are used in the risk assessment, other estimates reported by Burger could be used. For example, median fish consumption estimates may better represent the distribution of fish consumption data than mean estimates. It should also be acknowledged that the Burger survey was conducted in 1998, and that fish consumption rates even in subsistence populations may have changed.

Another issue raised by the SAB focuses on the seasonality of fish consumption. Data on consumption generated from Southern states (e.g., Burger 2002 data from South Carolina) may reflect year-round consumption, whereas fishers in Northern states may only fish for nine months a year or less. While failure to take seasonality of fishing into consideration could result in overestimation of fish-derived methylmercury exposure for some regions, the SAB notes that some communities preserve fish for consumption outside the fishing season. It is important to be certain that fish consumption rates used in the risk assessment are in the form of annual averages, e.g., consumption rate expressed in terms of grams of fish consumed per week per year. Also, it is unclear whether the risk assessment uses annualized fish consumption rates and whether fish consumption is based on concentrations that are "as caught" or "as prepared." The SAB recommends that this information concerning seasonality be clarified in the Technical Support Document. There is a general agreement that the Technical Support Document adequately utilizes existing data to identify consumption rates and target populations that are representative of the most highly exposed susceptible populations.

Regarding alternative approaches, the SAB notes that population-based fish consumption rates could be applied, although these data tend to show lower fish consumption rates than surveys focusing on subsistence and sport-caught fish (Knobeloch et al. 2005). This would tend to underestimate risks and would not be consistent with the Technical Support Document objective to target sensitive, highly exposed individuals. Therefore, this alternative is not recommended.

In regard to fish consumption generally, the SAB recommends that EPA better explain its rationale for assuming that subsistence consumers eat fish larger than seven inches in length and asks EPA to provide references supporting its assumptions and to discuss uncertainties associated with this assumption.

3.8. Use of Census data to identify high-end fish consuming populations

Question 8: Please comment on the approach used in the risk assessment of assuming that a high-end fish consuming population could be active at a watershed if the "source population" for that fishing population is associated with that watershed (e.g. at least 25 individuals of that population are present in a U.S .Census tract intersecting that watershed). Please identify any additional alternative approaches for identifying the potential for population exposures in watersheds and the strengths and limitations associated with these alternative approaches (additional detail on how EPA assessed where specific high-consuming fisher populations might be active is provided in section 1.3 and Appendix C of the Mercury Risk TSD).

Response: Overall, the SAB agrees that the criterion of using at least 25 persons per census tract from a given target population (Laotian, poor Hispanic, American Indian populations, amongst others) is a reasonable approach. The approach is driven by the necessity of using existing data to identify watersheds with susceptible proximal populations. While the source population selected is somewhat arbitrary, the SAB agrees that it is a reasonable approach, and that other approaches may not be as effective or feasible. Regardless of what number is chosen, the prevalence of subsistence fishing in the target communities remains unknown. EPA indicated that a sample of 25 individuals or greater was selected to be reasonably certain that at least one subsistence fisher is potentially active at the watershed. No major concerns are raised by the SAB concerning this issue. However, the SAB recommends that the Technical Support Document clarify how many census tracts were eliminated due to the use of this cut point. The Technical Support Document should also include information on the relative distribution of the sample size of the susceptible populations in the census tracts that were targeted. That is, an absolute sample of 25 may represent different proportions of the total target population in a given census tract, which may reflect differences in subsistence fishing behavior. The Technical Support Document should also discuss the possibility that more remote waterways are fished by subsistence anglers as well and the potential of this uncertainty for underestimating exposures

3.9. Use of the Mercury Maps approach

Question 9: Please comment on the draft risk assessment's characterization of the limitations and uncertainty associated with application of the Mercury Maps approach (including the assumption of proportionality between changes in mercury deposition over watersheds and associated changes in fish tissue MeHg levels) in the risk assessment. Please comment on how the output of CMAQ modeling has been integrated into the analysis to estimate changes in fish tissue MeHg levels and in the exposures and risks associated with the EGU-related fish tissue MeHg fraction (e.g., matching of spatial and temporal resolution between CMAQ modeling and HUC12 watersheds). Given the national scale of the analysis, are there recommended alternatives to the Mercury Maps approach that could have been used to link

modeled estimates of mercury deposition to monitored MeHg fish tissue levels for all the watersheds evaluated? (additional detail on the Mercury Maps approach and its application in the risk assessment is presented in section 1.3 and Appendix E of the Mercury Risk TSD).

<u>Response:</u> *Limitations/uncertainty associated with Mercury Maps (MMaps) approach and proportionality assumption.* The risk assessment's qualitative characterization of the limitations and uncertainty in the application of Mercury Maps approach is appropriate. The SAB recommends that EPA also summarize quantitative estimates of the uncertainty published in the existing literature in Appendix F of the Technical Support Document. CMAQ is considered to be the appropriate tool for providing the link between EGU emissions and mercury deposition to HUC12 watersheds with methylmercury fish data. There are quite a few comparisons, for example, between mercury wet deposition as modeled by CMAQ and as observed by the Mercury Deposition Network (e.g., Lin et al. 2007, Prongprueksa et al. 2008, and Bullock et al. 2009). A similar search of the literature for other components of this risk assessment would allow at least partial quantification of the variability or uncertainty in this risk assessment, including any literature relating to the time lag in the response of waterbodies to changes in mercury deposition (e.g., Munthe et al. 2007).

The Mercury Maps model states that for steady-state conditions, reductions in fish tissue concentrations are expected to track linearly with reductions in air deposition to a watershed with an intercept of zero for watersheds receiving mercury input exclusively via atmospheric deposition. This proportionality assumption is extended for the Technical Support Document study so that methylmercury levels in fish could be apportioned among mercury sources based on the associated apportionment of mercury deposition within a given watershed. The model is a reduced form of the IEM-2M watershed model used in the *Mercury Study Report to Congress* (U.S. EPA, 1997), whereby the equations of these models are reduced to steady state and consolidated into a single equation relating the ratio of current/future air deposition rates to current/future fish tissue concentrations.

Given these assumptions, Mercury Maps will work only with watersheds in which air deposition is the sole significant source of mercury and steady-state conditions are assumed. This indicates that the extension of the proportionality is valid only when other factors influencing methylation potential and catch profiles (species and trophic levels) remain relatively constant in a given watershed. Watersheds in which mercury input sources other than air deposition, such as mineral recovery operations using mercury, mercury cell chloralkali facilities and geologically high mercury inputs, are present and contribute loads that are significant relative to the air deposition load to that watershed are set aside from analysis in this risk assessment.

Since the Mercury Maps approach was developed, several recent publications have supported the finding of a linear relationship between mercury loading and accumulation in aquatic biota (Orihel et al. 2007; Orihel et al. 2008; Harris et al. 2007). These studies suggested that mercury deposited directly to aquatic ecosystems can become quickly available to biota and accumulated in fish, and that reductions in atmospheric mercury deposition should lead to decreases in methylmercury concentrations in biota. These results substantiate EPA's assumption that proportionality between air deposition changes and fish tissue methylmercury level changes is sufficiently robust for its application in this risk assessment.

Regarding the limitations and uncertainty associated with the application of Mercury Maps, it is acknowledged that the Mercury Maps approach (i.e., the assumption of proportionality between input changes and fish response) represents both a critical element of the analysis and a potentially important source of uncertainty. The sensitivity analyses conducted in the risk assessment addresses two specific

17

uncertainties related to application of Mercury Maps: (1) concerns over including watersheds that may be disproportionately impacted by non-air mercury sources, and (2) application of the Mercury Maps to both flowing and stationary freshwater bodies to verify if the two scenarios would produce different results. The results of these sensitivity analyses suggest that uncertainty related to the Mercury Maps approach is unlikely to substantially alter the assessment result that mercury emissions from U.S. EGUs potentially constitute a public health concern.

Integration of CMAQ data to HUC12 watersheds for estimating changes in fish MeHg, exposures and risks). The use of 12-km spatial resolution in CMAQ modeling is a significant refinement of the previous analysis, which was conducted using 36-km resolution. The integration of CMAQ data at this finer resolution into the analysis for estimating changes in fish tissue methylmercury levels is sound, provided that the proportionality assumption holds true (discussed in the previous response to this charge question).

CMAQ modeling at a 12-km spatial resolution is used to estimate total annual mercury deposition caused by U.S. and non-U.S. anthropogenic and natural sources over each watershed. For the purposes of the risk analysis, watersheds are classified using HUC12 codes (USGS, 2009), representing a fairly refined level of spatial resolution with watersheds generally 5 to 10 km on a side, which is consistent with research on the relationship between changes in mercury deposition and changes in methylmercury levels in aquatic biota. Although interpolating the deposition data from a coarser model grid (CMAQ) to a finer watershed grid (HUC12) will somewhat diffuse the peak deposition near large point sources, the data integration approach is sound.

The CMAQ modeling at 12-km resolution is a considerable (nine-fold) spatial refinement of the modeling conducted to support the Clean Air Mercury Rule (36-km resolution). Modeling results at finer resolution can be used to better resolve deposition patterns near point sources. The confidence in applying the 12-km resolution CMAQ results for estimating fish tissue methylmercury changes and its associated exposure/risk is heavily dependent on the robustness of the proportionality assumption in the Mercury Maps approach. The limitation and uncertainty of this assumption has been elaborated on in the response to the first part of this charge question.

Alternatives to the Mercury Maps approach linking modeled deposition to monitored MeHg fish tissue levels. The SAB agrees with the application of Mercury Maps in this assessment. There are other modeling tools capable of making a national scale assessment, such as the Regional Mercury Cycling Model (R-MCM). However, the R-MCM is more data intensive and the results produced by the two model approaches should be equivalent.

The R-MCM, a steady-state version of the time-dependent Dynamic Mercury Cycling Model, has been publicly available to and used by the EPA (Region 4, Athens, Environmental Research Laboratory) for a number of years. R-MCM requires more detail on water chemistry, methylation potential, etc., and yields more information as well. Substantial data support the Mercury Maps and the R-MCM steady-state results, so that the results of the sensitivity analysis and the outcomes from using the alternative models would be equivalent between the two modeling approaches. Though running an alternative model framework may provide additional reassurance that the Mercury Maps "base case" approach is a valid one, it is unlikely that substantial additional insight would be gained with the alternative model framework.

3.10.   Exclusion of watersheds with significant non-air loadings

Question 10: Please comment on the EPA's approach of excluding watersheds with significant non-air loadings of mercury as a method to reduce uncertainty associated with application of the Mercury Maps approach. Are there additional criteria that should be considered in including or excluding watersheds?

Response: The technique used to exclude watersheds that may have substantial non-air inputs is sound. Although additional criteria could be applied, they are unlikely to substantially change the results.

EPA excludes those watersheds that either contained active gold mines or had other substantial non-U.S. EGU anthropogenic releases of mercury. Identification of watersheds with gold mines is based on a 2005 USGS data set characterizing mineral and metal operations in the United States. The data represent commodities monitored by the National Minerals Information Center of the USGS, and the operations included are those considered active in 2003. The identification of watersheds with substantial non-EGU anthropogenic emissions is based on a Toxic Release Inventory (TRI) net query for 2008 non-EGU mercury sources with total annual on-site mercury emissions (all media) of 39.7 pounds or more. This threshold value corresponds to the 25th percentile annual U.S. EGU mercury emission value as characterized in the 2005 National Air Toxics Assessment. The EPA team considers the 25th percentile U.S. EGU emission level to be a reasonable screen for additional substantial non-U.S. EGU releases to a given watershed.

This appears to be a sound approach. The caveat is that TRI reporting may be biased high or low by the reporting entities, so it is not possible to judge whether the exclusion is reasonably conservative or not. There is no particular step EPA can take to rectify this uncertainty, although sensitivity tests could be run on different reporting thresholds and the number (and area) of excluded watersheds that result. As a minimum, the uncertainty in the TRI should be acknowledged, and the number of watersheds excluded in the base case and the uncertainty analysis should be explicitly stated.

Other criteria that EPA could consider for exclusion of particular watersheds are:
- Watersheds that are near urban areas, since those may have significant mercury inputs from runoff which are not included in the TRI reporting database, and
- Watersheds that are excessively polluted, for example by sanitary sewer discharges or highly anoxic conditions that might deter overall consumer fishing by many users.

3.11.   Concentration-response function used in modeling IQ loss

Question 11: Please comment on the specification of the concentration-response function used in modeling IQ loss. Please comment on whether EPA, as part of uncertainty characterization, should consider alternative concentration-response functions in addition to the model used in the risk assessment. Please comment on the extent to which available data and methods support a quantitative treatment of the potential masking effect of fish nutrients (e.g. omega-3 fatty acids and selenium) on the adverse neurological effects associated with mercury exposure, including IQ loss. Detail on the concentration-response function used in modeling IQ loss can be found in section 1.3 of the Mercury Risk TSD.

Response: As noted in the response to questions 2 and 3, the analyses of IQ should assume a less important role in the final document than in the present one. Question 11 contains three questions pertaining to the concentration-response function describing methylmercury's effect on IQ. The response to the first question is that the rationale for the concentration-response function is appropriate,

but with qualifications noted below. The response to the second question is that there is no alternative concentration response function that should be considered, but the analysis should be tempered, qualitatively, by factors that could influence the shape of the concentration function. The response to the third question is that masking by fish nutrients could influence the shape of the concentration response function, but there is not sufficient information to recommend a quantitative adjustment. These three responses are expanded upon in order below.

The specification of the concentration-response function. The function used comes from a paper by Axelrad and Bellinger (2007) that seeks to define a relationship between methylmercury exposure and IQ. A whitepaper by Bellinger (Bellinger 2005) describes the sequence of steps in relating methylmercury exposure to maternal hair mercury and then hair mercury to IQ. The Technical Support Document furthers notes that IQ has shown utility in describing the health effects of other neurotoxicants. These are appropriate bases for examining a potential impact of reducing methylmercury on IQ, but the SAB does not consider these compelling reasons for using IQ as a primary driver of the risk assessment. Instead, IQ should serve as a secondary measure along with other measures discussed in the responses to questions 2 and 3. The modeling of the impact of IQ should be placed in the appendix and accompanied by the qualifications noted below.

Alternative Concentration Response functions. The concentration-response function derived by Axelrad and Bellinger (2007) is acceptable for use in supplementary analyses in the Technical Support Document. It should be noted, however, that this function is likely to underestimate the effect on IQ of reducing mercury deposition for the reasons itemized here and in the response to charge question 2.

There is another reason that a model based on a linear relationship between exposure and neurobehavioral effect may underestimate the true effect of reducing exposure. It is evident from animal studies conducted under highly controlled conditions that the relationship between daily intake and brain mercury (the most suitable biomarker of exposure) is not linear, but rather is a power function with a power coefficient that is greater than 1.0; the power coefficient was 1.3 in a review of animal studies (Newland et al. 2008). This means that a decrease in intake will produce a greater-than-linear decrease in brain concentration. Thus, the impact of any reductions produced by reducing mercury emissions could be underestimated by the linear model used in the document. This observation is not intended to suggest that a new model be used, only that a qualitative argument should be made that the potential health impact may be underestimated.

A quantitative treatment of the mitigating impact of nutrients. The factors listed in this section could mitigate the concentration-effect relationship and should be mentioned in the Technical Support Document, but there is not enough known about their quantitative impact to support a recommendation of a re-analysis.

There is evidence from the Seychelles study that nutrients can mask effects of prenatal methylmercury exposures. Davidson et al. (2008), Strain et al. (2008) and Stokes-Riner et al. (2011), demonstrated that maternal hair mercury was associated with protein disulfide isomerase only after controlling for the effects of maternal omega-3 polyunsaturated fatty acid (PUFA) status. Controlling for omega-3 PUFAs steepened the slope of the concentration effect relationship (Strain et al. 2008). These nutrients are found in many marine fish species, but less is known about their concentration in freshwater fish and the concentrations may be lower. This issue is important because the concentration-effect relationship used in the Technical Support Document analysis derives from the consumption of marine fish but it is applied in the Technical Support Document to the consumption of freshwater fish. Since the slope might

be steeper with freshwater fish, it is possible that the analysis in the Technical Support Document underestimates the impact of reducing mercury deposition on consumers of freshwater fish.

Not only do omega-3 PUFAs mask methylmercury's neurotoxicity, but they confer benefits of their own that are of direct interest in considering the health impact of fish consumption. The studies by Oken et al. (2005, 2008) directly compared the benefits of fish consumption with the hazards associated with methylmercury exposure. These provide further evidence that the benefits of consuming marine fish may mask methylmercury's effects, a conclusion that is directly relevant to freshwater fish.

One SAB member points out that methylmercury is a potent inhibitor of multiple families of selenium-dependent enzymes that are required by the brain and endocrine system (Carvalho et al. 2008; 2011; Seppanen et al. 2004; Ralston and Raymond 2010). Therefore, the adverse effects of high methylmercury exposures on these enzymes could be accentuated among populations with poor selenium nutritional intakes and diminished among those with rich selenium status. Since the subsistence fish consumers that form the focus of this study are at notable risk of having poor nutrition, mercury exposures may be non-linearly related to toxicity risks. Other SAB members note that effects of selenium on methylmercury toxicity are based primarily on observations in animals, and there is disagreement in the scientific community regarding the significance of these observations to humans.

The same SAB member also suggests that since selenium abundance is largely observed to be inversely related to mercury bioaccumulation (Chen et al. 2001; Paulsson and Lindberg 1989; Belzile et al. 2004), diminishments in fish methylmercury concentrations following reductions in mercury deposition will not be uniform across watersheds. Selenium's inverse relationships to methylmercury bioaccumulation and toxicity may interact to exacerbate mercury exposure risks in watersheds with low selenium availability. This SAB member thinks that special consideration should be given to evaluating potential health risks from consumption of fish with high mercury contents that originate from watersheds in low selenium regions. Other SAB members note that the Mercury Maps (proportional response) approach is not affected by spatial differences in fish methylmercury content, and in fact this is one of the strengths of this approach. Changes in fish methylmercury concentrations may differ among aquatic ecosystems in absolute terms when mercury loading declines, depending upon whether initial fish concentrations are high or low. However, the reductions in fish methylmercury concentrations within these watersheds are nonetheless expected to be proportional to the decreases in loading.

Additional Point. Finally a statement on Page 84, Table F-2 references the Seychelles study instead of the New Zealand study. This should be corrected. The statement is: "Regarding outliers, when an outlier data point from the New Zealand study was included in the integrated derivation of the IQ loss slope factor, the factor was reduced by 25 percent (from -0.18 IQ points per unit ppm hair mercury, to -0.125)." This uncertainty should be acknowledged more explicitly in the body of the document rather than being merely mentioned in detail in a table in the Appendix. No additional analyses in the Technical Support Document are necessary; it could just be mentioned in the section on limitations and uncertainties that risk assessment estimates would be reduced by 25%.

3.12. **Uncertainty and variability**

Question 12: Please comment on the degree to which key sources of uncertainty and variability associated with the risk assessment have been identified and the degree to which they are sufficiently characterized.

Response: To answer this question, the SAB defines variability and uncertainty according to EPA's standard usage, which is consistent with the definitions given by Cullen & Frey, 1999. These definitions are as follows:

"Variability refers to temporal, spatial, or interindividual differences (heterogeneity) in the value of an input. In general, variability cannot be reduced by additional study or measurement."

"Uncertainty may be thought of as a measure of the incompleteness of one's knowledge or information about an unknown quantity whose true value could be established if a perfect measuring device were available."

The Technical Support Document presents a qualitative overview of variability and uncertainty in Appendix F. The qualitative nature of the discussion is appropriate since this is a conditional analysis. However, the SAB recommends an expanded discussion in Appendix F of variability and uncertainty to make explicit the uncertainties associated with the Agency's key analytical choices, which the SAB supports. This discussion could be organized according to the figures depicting sample calculations of high and low EGU impact that were provided at the SAB's public meeting on June 15, 2011 and reproduced below (see Figures 3 and 4, next page). The SAB recommends that these figures be added to the report along with an explanation of how the calculations were conducted.
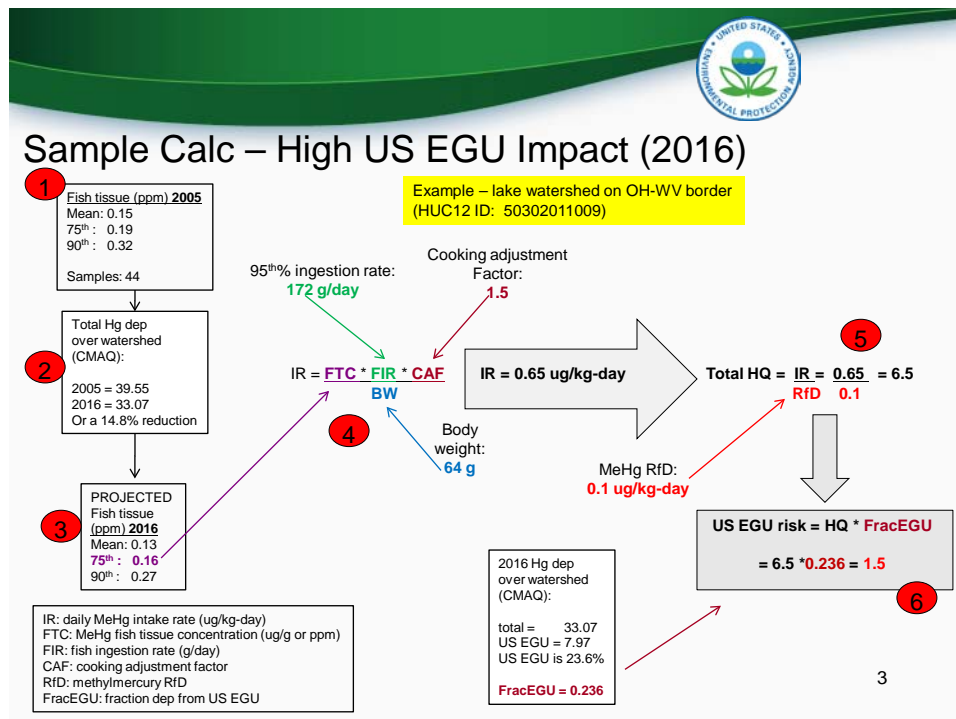
Figure 3: U.S. EPA-provided (June 16, 2011) schematic showing sample calculation – high U.S. EGU impact
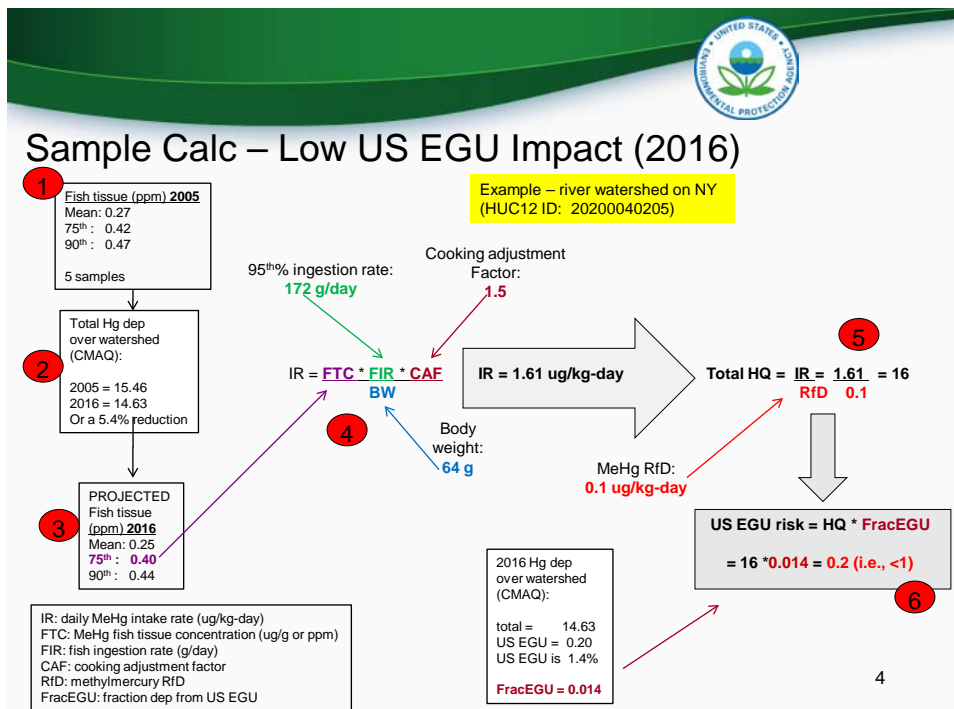
Figure 4: U.S. EPA-provided (June 16, 2011) schematic showing sample calculation – low U.S. EGU impact

In addition to the explicit discussion of variability and uncertainty, the SAB suggests that language be used throughout the Technical Support Document to clarify the scope of the results vis-à-vis variability and uncertainty in data and methods. For example, the Technical Support Document should cite the evaluation of uncertainty in the CMAQ and MMAPs source documents. Notwithstanding the uncertainties in the approach, the SAB considers the approach presented in the Technical Support Document sound and reasonable.

Variability. The SAB notes the topics covered in Appendix F regarding variability. The clarity of the documentation of the impact of individual sources of variability could be improved. Carefully selected maps and additional figures could be particularly helpful in providing this clarity. The SAB recommends that the following sources of variability be included in Appendix F to avoid misinterpretation of study results and outcomes.

- The effect of temporal variability in the following on estimates of mercury deposition
  - Appendix F should describe CMAQ boundary conditions that are necessary to establish in order to run the model for the 2 temporal scenarios
- Variation in geographic patterns of populations of subsistence fishers.
  - Appendix F addresses geographic variability in total and U.S. EGU-attributable mercury deposition and fish tissue concentrations. Appendix F should be expanded to discuss spatial variability in populations of subsistence fishers, noting the limited geographic coverage of watersheds with fish tissue concentrations.
- Variability in nature and protocols of state collection of fish data (see the response to Question 5, also mentioned below).
- Variation in fisher populations; for example, variation in body weights (potentially across race/ethnicities) and fishing and consumption habits.

23

- Variability in the factor used to translate mercury concentration measured at time of collection (i.e., expressed per unit wet weight) in comparison to mercury concentration at point of consumption following cooking.

Uncertainty. Appendix F defines sources of uncertainty for several components of the overall approach and selected parameter characterizations. The level of uncertainty is consistent with a screening level analysis. The SAB advises EPA to strengthen the discussion of each uncertainty presented by identifying at least qualitatively the direction of its effect on the overall risk assessment. For example, the small fish sample sizes results in underestimates of the 75th percentiles, which propagates to conservative underestimates of risk.

The SAB has discussed some sources of uncertainty in responses to other Charge Questions (e.g., Question 9). To summarize, the SAB recommends that Appendix F be expanded to provide a more complete listing and discussion of key uncertainties associated with the assessment. Additional sources of uncertainty that should be considered for expanded discussion include:
- Overall emission inventories, especially the non-EGU inventory derived as a modified version of the National Emissions Inventory (NEI). Appendix F should discuss the uncertainties in inventory components; whether and how the uncertainty changes between the 2005 to 2016 scenarios, including uncertainties in the TRI database; whether there is bias in the EGU and non-EGU components of the inventory; and whether the EGU emission estimates are derived from the best performing facilities or from the complete set of facilities.
- Alternative future scenario forecasts. Appendix F should more clearly describe the variables that are held constant versus factors that are varied between the two scenarios.
- Uncertainty in location of 2016 emissions reductions. Due to EPA's projection methods, there is uncertainty about where emissions reductions will occur between 2005 and 2016, which in turn influences the spatial patterns of deposition from EGUs in the 2016 scenario. Appendix F should address the uncertainties in the 2016 scenario regarding the specific geographic locations of reductions in EGU-derived mercury deposition as a fraction of total mercury deposition.
- Use of CMAQ and performance evaluation of CMAQ. Appendix F should provide more detailed description of uncertainty in CMAQ, including references to existing evaluations of the model.
- "Hot spots"
  - Appendix F should address whether the Mercury Maps approach, as implemented, is adequate to characterize the existence and extent of mercury "hot spots."
- Impacts of excluding watersheds from the analysis.
  - Appendix F should detail the criteria used for excluding watersheds, characterize the watersheds excluded by different criteria, and describe the estimated deposition in these watersheds.
- Representativeness of approximately 2,500 watersheds compared to 88,000 HUC12 nationwide.
  - Appendix F should characterize any bias introduced by looking at this subset of watersheds (e.g., some states are over-represented, such as Indiana and Minnesota, while others are under-represented such as Pennsylvania).
- Fish populations and fish tissue database (see SAB responses to questions 5, 6 and 13 for more detail). Appendix F should include discussion of:
  - Sample size for characterization of Implications of a data set with a low number of fish per watershed. Appendix F should identify the distribution of fish samples per

watershed and the possible implications of this distribution, including the implications of sample size for characterization of 75th percentile fish tissue concentration.

- o Uncertainty in methylmercury fish tissue concentrations from differences in sampling and analytical protocols used by States that contribute data and errors introduced by potential misidentification of locations, etc.

- Adjustment between wet and cooked weight of fish. EPA relies upon a single older study to derive an adjustment factor of 1.5. Alternative and newer peer reviewed studies of cooking effects on mercury in fish should be acknowledged (e.g., Musaiger et al. 2008; Farias et al. 2010) and used to discuss uncertainty associated with this assumed value.
  - o Appendix F should note that this is a constant value applied in the calculation and thus does not bias but could skew the results.
- Uncertainty of the assumption of proportionality and the MMAPs approach (see SAB response to Question 9).
- Characterization of susceptible human populations (see SAB responses to Questions 7 and 8)
  - o Characterizing subsistence fishing activity within high EGU deposition sites.
  - o Implications of choosing subsistence fishers and excluding high-end sport fishers.
  - o Census information that may exclude groups such as students, immigrants).
- Fish consumption rates (see SAB Response to Question 7).
  - o Limitations of the single study used to support the Technical Support Document's fish consumption rate for female subsistence fishers.
  - o Size of fish consumed.
- Derivation of the concentration-response relationship and RfD based on data from marine fish and mammal species, not inland freshwaters.
  - o Appendix F should discuss the uncertainty introduced by not using RfDs derived based on studies of consumption of fish from inland freshwaters. (See SAB response to Question 11).
- Applicability of the concentration-response relationship and RfD for low socio-economic status populations. This relationship has not been examined.
  - o Appendix F should discuss how this relationship may bias the report toward underestimating risk.
- Effect of the nutritional benefits of fish consumption in comparison to risks from mercury. Appendix F should address how the lack of consideration of this factor that may bias the analysis toward underestimating risk (see SAB response to Question 11).

3.13. **Discussion of analytical results**

Question 13: Please comment on the draft Mercury Risk TSD's discussion of analytical results for each component of the analysis. For each of the components below, please comment on the extent to which EPA's observations are supported by the analytical results presented and whether there is a sufficient characterization of uncertainty, variability, and data limitations, taking into account the models and data used.

Mercury deposition from U.S. EGUs
Response: EPA's observations in section 2.3 of the Technical Support Document (p. 35)[1] are generally supported by EPA's observations about mercury deposition as depicted in analytical results provided to

---

[1] Section 2.3 observations:

the SAB by EPA following the SAB meeting in the form of a Memorandum from Zachary Pekar, July 1, 2011, entitled "Clarification and Updating of Mercury Deposition Maps Provided in the Technical Support Document: National-Scale Mercury Risk Assessment." The SAB supports EPA's plan to include updated figures from the memorandum in EPA's Technical Support Document as replacements for Figures 2-1 to 2-4 in the March 2011 draft so that they correctly reflect total annual mercury deposition per square-meter by watershed. The SAB recommends that the spatial patterns of simulated deposition shown in Figure 2-1 to 2-4 be better explained and that EPA should characterize data limitations more effectively.

EPA's observations about mercury deposition as depicted in Technical Support Document Figures 2-1 to 2-4 are supported by analytical results. However the 12-km deposition maps are very different than previously produced maps on the 36-km scale (for example in Texas and Nevada). The SAB recommends that EPA explain these differences and that EPA consider including separate maps of wet and dry deposition and/or aggregating the results into an approximately 36 km grid scale for comparison to earlier maps and to data plots, such as national deposition maps from the Mercury Deposition Network.

In general, the uncertainties associated with these results are not well characterized or adequately quantified. For example, there have been several intercomparison studies among numerical models for long-range transport of mercury and studies on model uncertainty evaluation that are not discussed or referenced. The SAB recommends that EPA summarize these references (Bullock et al. 2009; Pongprueksa et al. 2008; Lin et al. 2007; Ryaboshapko 2007) to help frame the overall uncertainty of the deposition estimates.

In addition, EPA should discuss more completely the inputs that are kept constant for the 2016 scenario and the inputs that are varied (and by how much). This information may merit discussion earlier in the report. In addition, the CMAQ results are very dependent on global boundary conditions that are supplied by the GEOS-Chem model. Uncertainty in those inputs will be carried through to the results. This should be noted.

Fish tissue methyl mercury concentrations
Response: The observations listed in section 2.4 of the Technical Support Document (pp. 43-44)[2] are generally supported by the analytical results. The SAB recommends that EPA clarify the text to improve the description of the analytical results for each bulleted observation as described below.

- Patterns of total and U.S. EGU-related Hg deposition differ considerably.
- US Hg deposition is generally dominated by sources other than U.S. EGUs (with the contribution from U.S. EGUs decreasing between the 2005 and 2016 scenarios).
- The contribution of U.S. EGU deposition to total deposition does vary across watersheds and can represent a relatively large fraction in some (more limited) instances.

[2] Section 2.4 observations:
- Focus on U.S. EGU-attributable Hg fish tissue levels is in the eastern half of the U.S.
- U.S. EGUs contribute a larger fraction to total Hg fish tissue levels in the U.S. than they do to total Hg deposition (in terms of percent), this reflects the fact that Hg fish tissue samples are focused in the east where U.S. EGU deposition is greater.
- Relative to the combined impact of other sources, U.S. EGUs represent a smaller, but still potentially important contributor to total fish tissue MeHg levels.
- Despite the relatively small fraction of total fish tissue MeHg associated with U.S. EGUs on average, for a subset of watersheds, they can make a substantially larger contribution.

Although there is sufficient characterization of variability, EPA should characterize uncertainty and data limitations more fully. Specifically, the small sample sizes of mercury concentrations in fish for the individual watersheds (~29% of watersheds have n=1) will result in lower estimates of mercury concentrations in the 75th percentile as shown earlier in Figures 1 and 2 in this SAB report. This data limitation bias will be propagated to underestimate the hazard in the risk assessment.

The text in the observations should be modified to refer to tissue and mercury "concentrations" rather than "levels" to be more precise. "Level" is a generic term and can refer to any number of different metrics. Finally, where the percentages of EGU-contribution to fish methylmercury are mentioned, EPA should clarify that those values pertain to only fish-sampled watersheds. Given the under-sampling in watersheds where there are high levels of deposition, the percentages indicated could be higher.

Some figures and tables would also benefit from modification or elimination. Figures 2-7 to 2-10 are difficult to interpret because the symbols do not reflect the number of observations for that site. Improved plots should display symbols proportional to sample size and provide color or shading of symbols to represent observed fish concentrations. In addition, the maps shown in Figures 2-7 to 2-14 need to include the western continental United States. These figures unnecessarily cut off the western continental United States. While the SAB understands the reason for this omission (there is minimal expected change in EGU emissions in the western United States), it is important to show the results for the entire United States in the figures of this national assessment. In the absence of national maps, the reader (especially someone with interest in the western United States) many be left wondering about current fish methylmercury concentrations in this region (see Figure 2-6), as well as the model predicted changes in fish methylmercury for the 2016 scenario.

The legend for Figure 2-8 should make it clear that the 2016 mercury tissue concentrations are computed by adjusting the 2005 concentrations to account for lower expected deposition as per the Mercury Maps approach. The third bullet item on page 36 of the Technical Support Document should be corrected to indicate that Figures 2-7 and 2-8 give concentrations of mercury in fish, not total mercury deposition. In addition, the figures showing the top 10th percentile (2-11 to 2-14) should be removed since the pattern of mercury is greatly affected by high sampling effort in South Carolina, Indiana, West Virginia and Louisiana. The current maps could also result in undue public concern in those states. Finally, the text describing Table 2-5 needs to be clarified to state that the relationships are not causal.

Patterns of mercury deposition with mercury fish tissue data
Response: Overall, the SAB agrees that the observations in section 2.5 of the Technical Support Document (pp. 48-49)[3] are supported by the analytical results presented and there is a sufficient characterization of uncertainty, variability, and data limitations. However, a number of changes are needed to better clarify these points. The Technical Support Document should clearly describe the degree to which the non-uniform, state-specific data availability influences this analysis. For example, South Carolina, Louisiana and Indiana all have abundant data availability compared to most states. EPA should discuss how this data availability bias affects the analytical results. The SAB recommends that

---

[3] Section 2.5 observations:
- The fish tissue MeHg sampling data (summarized at the watershed-level) provides limited coverage for areas with elevated U.S. EGU Hg deposition. Therefore, the number of "at risk" watersheds as characterized in this risk assessment may be substantially higher than estimated.
- Hg fish tissue levels are not correlated with total Hg deposition (the relationship is highly dependent on methylation potential of individual waterbodies).
- Hg fish tissue samples were generally collected in regions with elevated total Hg deposition.

this section be substantively rewritten to improve clarity and to highlight the major relevant points. As discussed below, EPA should revise the text in footnote 36, which is critical to the understanding of Figures 2-15 and 2-16, and yet is not clearly enough written for the reader to understand the key information. Also, the figure legends within each of Figures 2-15 and 2-16 need to be changed because the "blue areas" are not "water bodies," but rather "watersheds," which include water bodies that sometimes are more obvious than their watersheds (e.g., the Minnesota portion of Lake Superior, Long Island Sound and perhaps erroneously, the Canadian portion of Lake Champlain). The SAB recommends that these two maps be replotted with a third color that clearly identifies the areas of overlap.

Figure 2-17 is critically important not only to this section, but also to the overall document. The SAB suggests that this figure could be brought into this document much earlier because it adds value to understanding the lack of direct relationships between deposition and mercury in fish. In a sense, it frames the justification for the approach taken in the overall analysis. The SAB recommends that EPA provide a more complete introduction to Figure 2-17 that would state the important premises of the analysis applied in this risk assessment - that spatial variability of deposition rates is only one major driver of spatial variability of fish methylmercury and that variability of ecosystem factors that control methylation potential (especially wetlands, aqueous organic carbon, pH, and sulfate) also play a key role. A question was also raised as to whether Figure 2-17 has been truncated, and if so, did it need to be? That is, are there data above 1.0 ppm fish concentration and 40 ug/m2-yr deposition? The SAB suspects that there are.

Figure 2-18 could similarly be moved to an earlier section of the document because it indicates that the analysis identified watersheds with higher rates of deposition than the national (~88,000 HUC 12 watersheds) trend and that the watersheds with available fish data are in fact, those with higher EGU-derived mercury deposition rates.

The red areas of Figures 2-15 and 2-16 are labeled in each map's legend as "Watersheds with relatively elevated US EGU Hg dep." Footnote 36 explains how the red areas are identified, an explanation that is densely written, as follows:

> Footnote 36. Areas of "elevated U.S. EGU-related Hg deposition" refer to areas that are at or above the average deposition level seen in watersheds with U.S. EGU-attributable exposures above the MeHg RfD. Specifically, we used exposure estimates based on the 95th percentile fish consumption rate (for the female high consumer scenario assessed nation-wide) to identify watersheds with U.S. EGU-attributable exposures above the MeHg RfD and then queried for the average U.S. EGU-related Hg deposition across that subset of watersheds. This average deposition rate differed for the 2005 and 2016 Scenarios (i.e., 3.79 and 1.28 ug/m^2, respectively). These values were used as the basis for identifying watersheds with levels of U.S. EGU-related Hg deposition for the 2005 and 2016 Scenarios presented in Figures 2-13 and 2-14.

It is troublesome that footnote 36 implies that the threshold for what constitutes "relatively elevated U.S. EGU Hg deposition" is different in the two maps. The red area in Figure 2-15 is characterized as an average deposition rate of 3.79 and for Figure 2-16, 1.29 ug/m^2. The next, and last, sentence is confusing, and implies that 3.79 and 1.29 are used as thresholds for identifying the red areas: "These values were used as the basis for identifying watersheds..." This characterization may confuse readers, in that readers probably expect similarly colored geographic areas in adjacent similar maps to be presented as portraying quantitatively similar environmental information, an expectation that these maps

28

apparently do not meet. The SAB suspects that the deposition rate threshold for inclusion in the map is probably relatively constant, and communicating the threshold would be a more useful characterization than describing the average deposition rates, which are different for understandable, but unexplained, reasons. For any given watershed, the threshold is the EGU-attributable deposition rate that produces EGU-attributable exposure "above the MeHg RfD." In practical terms for this risk assessment of subsistence fishers, this threshold is a modeled EGU-attributable increment in fish concentration that is greater than 0.038 ppm methylmercury, a concentration that does not correspond to a constant mercury deposition rate because the concentration varies among watersheds in accordance with the proportionality principle described in the risk assessment. However, the SAB notes that the average mercury deposition rate that produces this incremental methylmercury concentration will be similar between the 2005 and 2016 scenarios. If so, the red areas could then be characterized, for example, as "elevated U.S. EGU-related mercury deposition that refers to areas where deposition from EGU emissions has the potential, even in the absence of mercury from other sources, to cause exposures above the methylmercury RfD." The average threshold EGU-attributable mercury deposition rate for exceeding the threshold could be presented, along with the average deposition in the red area. The revised document should explain why the average deposition rate is lower in the 2016 scenario red area, rather than assume that the reader will immediately know why.

However the red area is dealt with, a more complete and understandable explanation needs to be presented than the current explanation of footnote 36.

Percentile risk estimates
<u>Response</u>. Generally, the percentile risk estimates in 2.6.1 are calculated in a reasonable manner and the observations on pages 53-54 of the Technical Support Document [4] are appropriate. The Technical Support Document especially provides a useful discussion of the uncertainties of high values in Tables 2-5 and 2-7. The SAB has several suggestions to improve the presentation of the material and results for other parts of section 2.6.1.

The Technical Support Document should include an explanation of why the values in Tables 2-6 and 2-7 decrease when going from the 50th to 75th percentile. This is likely because the ranked risk values are not the same as the ranked EGU contributions. This difference should be mentioned. Perhaps the tabled values should be referred to in some way as averaged.

The values in Tables 2-6 and 2-7 are based on averaging the values that are 2.5% below and 2.5% above. EPA should consider whether it is better to use a 2.5% range or use the 10 nearest values. EPA should also describe how the range is selected for the 99th percentile.

---

[4] Section 2.6.1 observations:
- For the high-end female consumer assessed at the national-level, total IQ loss and total HQ estimates do not change in a systematic way between the 2005 and 2016 Scenarios with these levels often being of potential health concern across a wide variety of consumption rates and watershed percentiles.
- By contrast (again focusing on the high-end female consumer assessed nationally), both U.S. EGU-incremental IQ loss and the U.S. EGU increment-based HQ display notable reductions between the 2005 and 2016 Scenarios, but U.S. EGU-attributable risk still exceeds potential levels of concern for a over a quarter of watersheds.
- Estimates of risks generated for the high-end female consumer population (assessed at the national-level) are generally higher than risks estimated for the other high-end fisher populations, with the exception of white and black fisher populations assessed in the southeast.

In section 2 page 54, the paragraph comparing "risks" for high-end females with other populations is oversimplified. Depending on the percentiles considered, "risks" for Laotians, Vietnamese and Tribal fish consumers can also be higher than for high-end females. The highest consumption rates should be summarized in an appendix.

In section 2 page 55, EPA should provide more information on the gold-mining impacted watersheds in the Southeast. For example, it seems that gold mining occurred historically in a relatively small region of South Carolina, and only a few mines have recently been re-activated. Is it really appropriate to discount or question concerns about EGU affected exposure across the whole Southeast on this basis?

In Tables 2-6 and 2-7, EPA should consider reporting consumption rates and putting the percentiles in parentheses rather than reporting the percentiles and having the rates in parentheses.

In Table 2-15 and other places, the mean is included. Since the mean is not a percentile, the table header should be changed or the median used.

Number and frequency of watersheds with populations potentially at risk due to U.S. EGU mercury emissions
Response: The SAB has no significant concerns regarding the observations in section 2.6.2 of the Technical Support Document (pp. 57-58).[5] The SAB recommends that language be added regarding the change in the percentage of watersheds that continue to be above the RfD (or above a change in one to two IQ points, if this aspect of the risk assessment is retained) after EGU emissions are removed. Furthermore, on the SAB recommends that the first bullet point on page 57 to change the language "before taking into account deposition..." to something that does not imply temporality (e.g., "when you factor out other sources of mercury deposition"). The SAB also recommends that if the document discusses loss of IQ points, the revised document should refer to this change in relation to "populations living close to watersheds" rather than "watersheds".

With regard to the target population in a broader context, the size of the potentially impacted population is a key factor to consider in this risk assessment. This issue is outside the scope of the data available for the risk assessment, even though it is very relevant to the objectives of the Technical Support Document and its application to public health policy. The document focuses on subsistence fishing populations as a target population likely to be the most severely impacted by methylmercury consumption in fish. There is scant evidence documenting the prevalence or extent of subsistence fishing in the United States. Some SAB members note similarities in consumption rates among sport fishers and subsistence fishing populations. The inclusion of sport fishers with relatively higher fish consumption rates could expand the size and extent of the targeted susceptible population. Similarly, only limited information on the locations or characteristics of watersheds that are excluded from the analysis is provided (p. 63, bullet 4, Figs 2-15, 2-16). The SAB suggests that more detailed information be included regarding these watersheds and the uncertainties associated with their exclusion. In addition, the document should

---

[5] Section 2.6.2 observations:
- Less than 1% of the watersheds have an IQ loss of 1 point when deposition from U.S. EGUs is considered before taking into account deposition and exposures resulting from other sources of Hg.
- Between 2 and 12% of the watersheds have HQs $\geq$ 1.5, based on U.S. EGU mercury deposition before factoring in any other sources of mercury.
- Combining the two categories of watersheds with populations at-risk due to U.S. EGU mercury emissions summarized in the last two bullets, we get a total estimate ranging from 2 to 28% of watersheds, with this range reflecting in part the U.S. EGU percent contribution that is considered (e.g., 5, 10, 15 or 20%).

address the excluded watersheds within the context of predicted mercury deposition patterns. Some enumeration of the extent to which the target population would be expanded if these factors had been incorporated into the analysis would help provide important additional information on the potential scope and magnitude of the hazards estimated in the assessment. The SAB recognizes that some additional data may be available on the consumption patterns of recreational anglers, but that EPA did not have time or resources to integrate this information into the current analysis.

3.14. **Responsiveness to the goals of the study**

Question 14: Does section 2.8 respond to the goals of the study and does it encapsulate the critical issues and the significant results of the analysis?

Response: Section 2.8 responds to the goals of the study, but the manner in which it highlights the key findings could be improved. The section should be revised to explicitly respond to each of the goals of the study as set out on page 13 of the Technical Support Document:

   (a)   What is the nature and magnitude of the potential risk to public health posed by current U.S. EGU mercury emissions?
   (b)   What is the nature and magnitude of the potential risk posed by U.S. EGU mercury emissions in 2016 considering potential reductions in EGU mercury emissions attributable to Clean Air Act requirements? and
   (c)   How is risk estimated for both the current and future scenario apportioned between the incremental contribution from U.S. EGU's and other sources of mercury?

In response to these goals, the SAB sees that the major finding of the study is that a reduction in mercury emissions will translate to reductions in fish tissue methylmercury concentrations, and in turn, to a reduction in potential risk to subsistence fishers that would result from the consumption of self-caught fish from inland watersheds. While there are numerous unquantified sources of variability and uncertainty that are contained in the numerical estimates of potential risk, the variability and uncertainty do not contradict this basic finding.

3.15. **Confidence in the analysis**

Question 15: Despite the uncertainties identified, is there sufficient confidence in the analysis for it to determine whether mercury emissions from U.S. EGUs represent a potential public health hazard for the group of fish consumers likely to experience the highest risk attributable to U.S. EGU?

[Note: This question was not among the original charge questions. It was formulated by the SAB as an alternative to the second subquestion originally posed by EPA for Charge Question 14, which read as follows: "*In addition, please comment on the degree to which the level of confidence and precision in the overall analysis is sufficient to support use of the risk characterization framework described on page 18.*"]

Response*:* Notwithstanding the uncertainties inherent in this analysis, the Technical Support Document, after incorporation of the recommendations of the SAB, should provide an objective, reasonable and credible determination of the potential for a public health hazard from mercury emitted from U.S. EGUs.

4. Summary List of Recommendations

This SAB report contains many recommendations for improving the Technical Support Document presented in the responses to the charge questions. These recommendations can be organized into three general themes:

1. Improve clarity of the Technical Support Document in terms of the methods used in the risk assessment and presentation of results. The reader should be able to understand how risk calculations were performed, the rationale for key decisions regarding the use of models and sources of input data, and results obtained from the analysis and the conclusions. SAB support for the risk assessment is contingent upon this improvement in clarity being accomplished.

2. Expand the discussion of sources of variability and uncertainty in the risk assessment. Several additional sources of uncertainty should be acknowledged and discussed briefly in the Technical Support document.

3. De-emphasize IQ loss as an endpoint in the risk assessment.

For convenience, specific recommendations have been extracted from the body of the report and are listed below.

Question1: Overall design
- The Introductory section should make clear, at the earliest possible point, that the analysis is a determination of watershed impact with exposure addressed as a potential outcome.

Question 2: Critical health endpoints besides IQ
- The SAB recommends that EPA reframe the document's discussion of IQ. EPA should incorporate IQ and other neuropsychological measures as supplemental information and focus on HQ as the primary critical health endpoint.It is not suggested that the analyses of IQ be removed altogether but rather that the analyses be framed in an appendix to the report as a secondary analysis of impact of reduced exposure on potential health-related outcome. The appendix should discuss the basis for selecting a HQ at or above 1.5 as the criteria for selecting potentially impacted watersheds should be explained. The appendix should also include discussion of potential effects on other measures like developmental delays (Grandjean et al. 1997) or neuropsychological tests (as discussed by van Wijngaarden et al. 2006), presented in the overall context of the weight of evidence.
- The SAB recommends that the Technical Support Document acknowledge and discuss alternative (to HQ) quantitative measures but does not recommend a re-analysis based on these measures.

Question 4: Spatial scale of watersheds
- In HUCs with multiple lakes, the SAB recommends against using a single fish methylmercury value to describe the HUC.
- The SAB recommends that the authors provide a summary table describing the characteristics of the watersheds where fish were collected, including the fraction of fish samples collected from rivers versus lakes, and whether from single or multiple sites.

Question 5: Measured fish tissue mercury concentration
- The SAB recommends that the EPA utilize fish methylmercury data collected since 1999 for the risk assessment.
- The SAB recommends that it be revised to provide a better description of the character of the data, as well as the selection of analyzable data (e.g., sizes, distribution of fish sizes across watersheds), should be better detailed in the report.
- The SAB recommends that EPA contact some states that receive what the Technical Support Document terms "relatively elevated" mercury deposition from U.S. EGU emissions and have limited fish methylmercury measurements to investigate if additional recent (since 1999) fish methylmercury data are available to improve the coverage for the mercury risk assessment.

Question 6: Use of the 75th percentile fish tissue methylmercury value
- The SAB recommends inclusion of a graph depicting the number of tissue samples available for analysis by tissue concentration.
- The SAB also recommends that the document discuss this source of uncertainty, including adding a table with the distribution of number of available fish samples and the fish size from which they were obtained across watersheds to indicate the extent of the problem. The Technical Support Document should describe in more detail why including fish tissue concentrations from one fish sample is likely to result in an underestimate of the number of watersheds at risk.
- The SAB recommends that EPA should also conduct a sensitivity analysis using the median fish tissue concentration to better represent the distribution of fish tissue methylmercury levels where the sample size is one and provide a bound on the risk assessment.
- The use of other percentiles in the sensitivity analysis is not recommended given the limitations of the fish tissue data available.
- The SAB recommends that the document describe more clearly the source of the fish methylmercury data and provide at least a general discussion of how fish sampling programs differ in ways that can contribute variability and uncertainty to the data set, such as fish capture methods and criteria for selecting fish to measure methylmercury concentrations.
- The report should include information on the sizes of fish that were analyzed. In doing so, the Technical Support Document may be able to quantify the impact, if any, of the size of fish sampled in watersheds with few fish tissue samples available on estimated mercury concentrations.
- The SAB also recommends that the Technical Support Document clarify that the 75th percentile represents available fish tissue data that may or may not represent the fish in the watershed or the fish consumed.

Question 7: Consumption rates and location for high-end consumers
- The SAB recommends that a few caveats should be acknowledged more fully in the document. The main consumption estimates came from a relatively small survey of individuals attending a fishing convention in South Carolina, so the consumption estimates reported in the Burger 2002 study may be imprecise, in particular for women. The SAB recommends that the Technical Support Document acknowledge that, while several estimates of fish consumption rates were used in the risk assessment, other estimates reported by Burger could have been used. For example, median fish consumption estimates may better represent the distribution of fish consumption data than mean estimates. It should also be acknowledged that the Burger survey was conducted in 1998, and that fish consumption rates even in subsistence populations may have changed.
- The SAB recommends that this information concerning seasonality be clarified in the Technical Support Document
- The SAB recommends that EPA better explain its rationale for assuming that subsistence consumers eat fish larger than seven inches in length and asks EPA to provide references supporting its assumptions and to discuss uncertainties associated with this assumption.

Question 8: Use of census data to identify high-end fish consuming populations
- The SAB recommends that the Technical Support Document clarify how many census tracts were eliminated due to the use of the 25 individual cut point.
- The Technical Support Document should include information on the relative distribution of the sample size of the susceptible populations in the census tracts that were targeted.
- The Technical Support Document should discuss the possibility that more remote waterways are fished by subsistence anglers as well and the potential of this uncertainty for underestimating exposures.

Question 9: Use of the Mercury Maps approach
- The SAB recommends that the quantitative estimates of the uncertainty associated with use of the Mercury Maps approach published in the existing literature be summarized in Appendix F of the Technical Support Document.

Question 10: Exclusion of watersheds with significant non-air loadings
- The uncertainty in the TRI (screen) should be acknowledged, and the number of watersheds excluded in the base case and the uncertainty analysis should be explicitly stated.

Question 11: Concentration-response function used in modeling IQ loss
- IQ should serve as a secondary measure along with other measures discussed in the responses to questions 2 and 3. The modeling of the impact of IQ should be placed in the appendix and accompanied by the qualifications discussed in section 3.11 of this SAB report.
- A statement on Page 84, Table F-2 references the Seychelles study instead of the New Zealand study. This should be corrected.

Question 12: Uncertainty and variability
- The SAB recommends an expanded discussion in Appendix F of variability and uncertainty to make explicit the uncertainties associated with the Agency's key analytical choices, which the SAB supports. This discussion could be organized according to the figures depicting sample calculations of high and low EGU impact that were provided at the SAB's public meeting on June 15, 2011 and reproduced as Figures 3 and 4. The SAB recommends that these figures be added to the report along with an explanation of how the calculations were conducted.
- The SAB suggests that language be used throughout the Technical Support Document that clarifies the scope of the results vis-à-vis variability and uncertainty in data and methods. For example, the Technical Support Document should cite the evaluation of uncertainty in the CMAQ and MMAPs source documents.
- The SAB recommends that the following sources of variability to be included in Appendix F to avoid misinterpretation of study results and outcomes.
  - The effect of temporal variability in the following on estimates of mercury deposition.
  - Appendix F should describe CMAQ boundary conditions that are necessary to establish in order to run the model for the 2 temporal scenarios.
  - Variation in geographic patterns of populations of subsistence fishers.
  - Appendix F addresses geographic variability in total and U.S. EGU-attributable mercury deposition and fish tissue concentrations. Appendix F should be expanded to discuss spatial variability in populations of subsistence fishers, noting the limited geographic coverage of watersheds with fish tissue concentrations.
  - Variability in nature and protocols of state collection of fish data (see the response to Question 5, also mentioned below).
  - Variation in fisher populations; for example, variation in body weights (potentially across race/ethnicities) and fishing and consumption habits.
  - Variability in the factor used to translate mercury concentration measured at time of collection (i.e., expressed per unit wet weight) in comparison to mercury concentration at point of consumption following cooking.
- The SAB advises EPA to strengthen the discussion of each uncertainty presented by identifying at least qualitatively the direction of its effect on the overall risk assessment. For example, the small fish sample sizes results in underestimates of the 75th percentiles, which propagates to conservative underestimates of risk.
- The SAB recommends that Appendix F be expanded to provide a more complete listing and discussion of key uncertainties associated with the assessment. Additional sources of uncertainty that should be considered for expanded discussion include:
  - Overall emission inventories, especially the non-EGU inventory derived as a modified version of the National Emissions Inventory (NEI). Appendix F should discuss the uncertainties in inventory components; whether and how the uncertainty changes between the 2005 to 2016 scenarios, including uncertainties in the TRI database; whether there is bias in the EGU and non-EGU components of the inventory; and whether the EGU emission estimates were derived from the best performing facilities or from the complete set of facilities.
  - Alternative future scenario forecasts. Appendix F should more clearly describe the variables that were held constant versus factors that were varied between the two scenarios.
  - Regarding uncertainty in location of 2016 emissions reductions. Due to EPA's projection methods, there is uncertainty about where emissions reductions will occur between 2005 and 2016, which in turn influences the spatial patterns of deposition from EGUs in the

2016 scenario. Appendix F should address the uncertainties in the 2016 scenario regarding the specific geographic locations of reductions in EGU-derived mercury deposition as a fraction of total mercury deposition.

o Use of CMAQ and performance evaluation of CMAQ. Appendix F should provide more detailed description of uncertainty in CMAQ, including references to existing evaluations of the model.

o Appendix F should address whether the Mercury Maps approach, as implemented, is adequate to characterize the existence and extent of mercury "hot spots."

o Appendix F should detail the criteria used for excluding watersheds, characterize the watersheds excluded by different criteria, and describe the estimated deposition in these watersheds.

o Regarding representativeness of approximately 2,500 watersheds compared to 88,000 HUC12 nationwide, Appendix F should characterize any bias introduced by looking at this subset of watersheds (e.g., some states are over-represented, such as Indiana and Minnesota, while others are under-represented such as Pennsylvania).

o Fish populations and fish tissue database (see SAB responses to questions 5, 6 and 13 for more detail). Appendix F should include discussion of:
  ▪ Sample size for characterization of Implications of a data set with a low number of fish per watershed. Appendix F should identify the distribution of fish samples per watershed and the possible implications of this distribution, including the implications of sample size for characterization of 75th percentile fish tissue concentration.
  ▪ Uncertainty in methylmercury fish tissue concentrations from differences in sampling and analytical protocols used by States that contribute data and errors introduced by potential misidentification of locations, etc.

o Regarding adjustment between wet and cooked weight of fish: EPA relied upon a single older study to derive an adjustment factor of 1.5. Alternative and newer peer reviewed studies of cooking effects on mercury in fish should be acknowledged (e.g., Musaiger et al. 2008; Farias et al. 2010) and used to discuss uncertainty associated with this assumed value.
  ▪ Appendix F should note that this is a constant value applied in the calculation and thus does not bias but could skew the results.

o Regarding uncertainty of the assumption of proportionality and the MMAPs approach (see SAB response to Question 9 for specifics to be discussed in Appendix F).

o Characterization of susceptible human populations (see SAB responses to Questions 7 and 8)
  ▪ Characterizing subsistence fishing activity within high EGU deposition sites.
  ▪ Implications of choosing subsistence fishers and excluding high-end sport fishers.
  ▪ Census information that may exclude groups such as students, immigrants).

o Fish consumption rates (see SAB Response to Question 7).
  ▪ Limitations of the single study used to support the Technical Support Document's fish consumption rate for female subsistence fishers.
  ▪ Size of fish consumed.

o Derivation of the concentration-response relationship and RfD based on data from marine fish and mammal species, not inland freshwaters.
  ▪ Appendix F should discuss the uncertainty introduced by not using RfDs derived based on studies of consumption of fish from inland freshwaters. (See SAB response to Question 11).

- o Applicability of the concentration-response relationship and RfD for low socio-economic status populations. This relationship has not been examined.
    - ▪ Appendix F should discuss how this relationship may bias the report toward underestimating risk.
- o Effect of the nutritional benefits of fish consumption in comparison to risks from mercury. Appendix F should address how the lack of consideration of this factor that may bias the analysis toward underestimating risk (see SAB response to Question 11).

Question 13: Discussion of analytical results
Mercury deposition from U.S. EGUs
- The SAB recommends that the spatial patterns of simulated deposition shown in Figure 2-1 to 2-4 be better explained and that EPA should characterize data limitations more effectively.
- The 12-km deposition maps are very different than previously produced maps on the 36-km scale (for example in Texas and Nevada). The SAB recommends that EPA explain these differences and that EPA consider including separate maps of wet and dry deposition and/or aggregating the results into an approximately 36 km grid scale for comparison to earlier maps and to data plots, such as national deposition maps from the Mercury Deposition Network.
- There have been several intercomparison studies among numerical models for long-range transport of mercury and studies on model uncertainty evaluation that are not discussed or referenced. The SAB recommends that EPA summarize these references (Bullock, 2009; Pongprueksa et al., 2008; Lin et al, 2007; and Ryaboshapko, 2007) to help frame the overall uncertainty of the deposition estimates.

Fish tissue methyl mercury concentrations
- EPA should characterize uncertainty and data limitations more fully. Specifically, the small sample sizes of mercury concentrations in fish for the individual watersheds (~29% of watersheds have n=1) will result in lower estimates of mercury concentrations in the 75th percentile as shown earlier in Figures 1 and 2 in this document.
- The text in the observations should be modified to refer to tissue and mercury "concentrations" rather than "levels" to be more precise.
- Where the percentages of EGU-contribution to fish methylmercury are mentioned, EPA should clarify that those values pertain to only fish-sampled watersheds. Given the under-sampling in watersheds where there are high levels of deposition, the percentages indicated could be higher.
- EPA should modify or eliminate some figures and tables.
    - o For figures 2-7 to 2-10, improved plots should display symbols proportional to sample size and provide color or shading of symbols to represent observed fish concentrations.
    - o The maps shown in Figures 2-7 to 2-14 need to include the western continental United States.
    - o The legend for Figure 2-8 should make it clear that the 2016 mercury tissue concentrations were computed by adjusting the 2005 concentrations to account for lower expected deposition as per the Mercury Maps approach.
    - o The third bullet item on page 36 of the Technical Support Document should be corrected to indicate that Figures 2-7 and 2-8 give concentrations of mercury in fish, not total mercury deposition.
    - o Figures showing the top 10th percentile (2-11 to 2-14) should be removed since the pattern of mercury is greatly affected by high sampling effort in South Carolina, Indiana, West Virginia, and Louisiana.
    - o The text describing Table 2-5 needs to be clarified to state that the relationships are not causal.

- *Patterns of mercury deposition with mercury fish tissue data.* The Technical Support Document should clearly describe the degree to which the non-uniform, state-specific data availability influences this analysis
- The SAB recommends that this section be substantively rewritten to improve clarity and to highlight the major relevant points.
- EPA should revise the text in footnote 36, which is critical to the understanding of Figures 2-15 and 2-16.
- Also, the figure legends within each of Figures 2-15 and 2-16 need to be changed because the "blue areas" are not "water bodies," but rather "watersheds," which include water bodies that sometimes are more obvious than their watersheds (e.g., the Minnesota portion of Lake Superior, Long Island Sound, and perhaps erroneously, the Canadian portion of Lake Champlain). The SAB recommends that these two maps be replotted with a third color that clearly identifies the areas of overlap.
- The SAB recommends that EPA provide a more complete introduction to Figure 2-17 that would state the important premises of the analysis applied in this risk assessment - that spatial variability of deposition rates is only one major driver of spatial variability of fish methylmercury and that variability of ecosystem factors that control methylation potential (especially wetlands, aqueous organic carbon, pH, and sulfate) also play a key role.
- The revised document should explain why the average deposition rate is lower in the 2016 scenario red area.

Percentile risk estimates
- The Technical Support Document should include an explanation of why the values in Tables 2-6 and 2-7 decrease when going from the 50th to 75th percentile. This is likely because the ranked risk values are not the same as the ranked EGU contributions. This difference should be mentioned. Perhaps the tabled values should be referred to in some way as averaged.
- The values in Tables 2-6 and 2-7 are based on averaging the values that are 2.5% below and 2.5% above. EPA should consider whether it is better to use a 2.5% range or use the 10 nearest values. EPA should also describe how the range is selected for the 99th percentile.
- Section 2 page 54: the paragraph comparing "risks" for high-end females with other populations is oversimplified. Depending on the percentiles considered, "risks" for Laotians, Vietnamese and Tribal fish consumers can also be higher than for high-end females. The highest consumption rates should be summarized in an appendix.
- Section 2 page 55: EPA should provide more information on the gold-mining impacted watersheds in the Southeast. For example, it seems that gold mining occurred historically in a relatively small region of South Carolina, and only a few mines have recently been re-activated. Is it really appropriate to discount or question concerns about EGU affected exposure across the whole Southeast on this basis?
- In Tables 2-6 and 2-7, EPA should consider reporting consumption rates and putting the percentiles in parentheses rather than reporting the percentiles and having the rates in parentheses.
- In Table 2-15 and other places, the mean is included. Since the mean is not a percentile, the table header should be changed or the median used.

Number and frequency of watersheds with populations potentially at risk due to U.S. EGU mercury emissions
- The SAB recommends that language be added regarding the change in the percentage of watersheds that continue to be above the RfD (or above a change in one to two IQ points, if this aspect of the risk assessment is retained) after EGU emissions are removed.

- The SAB recommends that the first bullet point on page 57 to change the language "before taking into account deposition..." to something that does not imply temporality (e.g., "when you factor out other sources of mercury deposition").
- The SAB also recommends that if the document discusses loss of IQ points, that it should refer to this change in relation to "populations living close to watersheds" rather than "watersheds."

Question 14: Responsiveness to the goals of the study
- EPA should revise section 2.8 to explicitly respond to each of the goals of the study as set out on page 13 of the Technical Support Document.

References

Axelrad, D.A., D.C. Bellinger et al. 2007. Dose-response relationship of prenatal mercury exposure and IQ: an integrative analysis of epidemiologic data. *Environmental Health Perspectives* 115(4): 609-615.

Bellinger, D.C. 2005. *Neurobehavioral Assessments Conducted in the New Zealand, Faroe Islands, and Seychelles Islands Studies of Methylmercury Neurotoxicity in Children. Report to the U.S. Environmental Protection Agency*. EPA-HQ-OAR-2002-0056-6045. Available: http://www.regulations.gov [accessed 20 January 2006].

Belzile, N., Y. Chen, J. Tong, J.M. Gunn, Y. Alarie et al. 2004. The antagonistic role of selenium in mercury bioassimilation by living organisms, in: *7th International Conference on Mercury as a Global Pollutant*, Volume 51, Pezdic, J., ed., Ljubljana, RMZ-Materials and Geoenvironment, pp. 803–806.

Bullock, O. R., Jr. et al. 2009. An analysis of simulated wet deposition of mercury from the North American Mercury Model Intercomparison Study. *Journal of Geophysical Research* 114, D08301, doi:10.1029/2008JD011224

Burger, J. 2002. Daily consumption of wild fish and game: Exposures of high end recreationalists. *International Journal of Environmental Health Research* 12:4, p. 343-354.

Carvalho C.M.L., E.H. Chew, S.I. Hashemy, J. Lu and A. Holmgren. 2008. Inhibition of the human thioredoxin system: A molecular mechanism of mercury toxicity. *Journal of Biological Chemistry* 283;18:11913-11923.

Carvalho, C.M.L., J. Lu, X. Zhang, E.S.J. Arnér and A. Holmgren. 2011. Effects of selenite and chelating agents on mammalian thioredoxin reductase inhibited by mercury: Implications for treatment of mercury poisoning. *FASEB Journal* 25 (1), pp. 370-381

Chen, Y.W.. N.Belzile and J.M. Gunn. 2001. Antagonistic effect of selenium on mercury assimilation by fish populations near Sudbury metal smelters. *Limnology and Oceanography* 46 (7) 1814–1818.

Chen, C.Y., R.S. Stemberger, N.C. Kamman, B.M. Mayes and C.L. Folt. 2005. Patterns of Hg bioaccumulation and transfer in aquatic food webs across multi-lake studies in the Northeast US. *Ecotoxicology* 14:135-147.

Cullen, A.C. and H.C. Frey. 1999. *Probabilistic Techniques in Exposure Assessment. Plenum Press*.

Davidson P.W., J.J Strain., G.J.Myers, S.W. Thurston, M.P. Bonham et al.. 2008. Neurodevelopmental effects of maternal nutritional status and exposure to methylmercury from eating fish during pregnancy. *NeuroToxicol* 29: 767-775.

Driscoll, C. T., Y.-J. Han, C. Y. Chen, D. C. Evers, K. F. Lambert, T. M. et al. 2007. Mercury contamination in forest and freshwater ecosystems in the Northeastern United States. *BioScience* 57:17-28.

Farias, L.A., D.I. Fávaro, J.O. Santos, M.B. Vasconcellos, A. Pessôa et al. 2010. Cooking process evaluation on mercury content in fish. *Acta Amazonica* 40(4): 741-8.

Grandjean, P., P. Weihe et al. 1997. Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicology & Teratology* 19(6): 417-428.

Harris, R. et al. 2007. Whole-ecosystem study shows rapid fish-mercury response to changes in mercury deposition. *PNAS* 104 (42) 16586-16591.

Knobeloch L., H.A.Anderson, P. Imm P, D. Peters D and A. Smith. 2005. Fish consumption, advisory awareness, and hair mercury levels among women of childbearing age. *Environmental Research* 97(2):219-226.

Lin C.-J., P. Pongprueksa, O.R.Bullock, S.E.Lindberg S.E., S.O.Pehkonen et al. 2007. Scientific Uncertainties in Atmospheric Mercury Models II: Sensitivity Tests over the Continental United States. *Atmospheric Environment* 41, 6544-6560,.

Munthe, J., R.A. Bodaly, B.A. Branfireun, C.T. Driscoll, C.C. Gilmour et al. 2007. Recovery of Mercury-Contaminated Fisheries. *Ambio* 36 (1), 33-44.

Musaiger A.O. and R. D'Souza. 2008. The Effects of Different Methods of Cooking on Proximate, Mineral and Heavy Metal Composition of Fish and Shrimps Consumed in the Arabian Gulf. *Archivos latinoamericanos de nutrición* 1(58), 103-109.

Newland, M.C., E.M. Paletzet al. (2008). Methylmercury and nutrition: adult effects of fetal exposure in experimental models. *NeuroToxicology* 29(5): 783-801.

Oken, E., J.S. Radesky et al. (2008). Maternal Fish Intake during Pregnancy, Blood Mercury Levels, and Child Cognition at Age 3 Years in a US Cohort. *American Journal of Epidemiology* 167(10): 1171-1181.

Oken, E., R.O.Wright, K.P.Kleinman, D. Bellinger, C.J. Amarasiriwardena et al. (2005). Maternal fish consumption, hair mercury, and infant cognition in a U.S. cohort. *Environmental Health Perspectives* 113 (10), 1376-1380.

Orihel, D.M., M.J. Paterson, P.J. Blanchfield, R.A. Bodaly, and H. Hintelmann. 2007. Experimental evidence of a linear relationship between inorganic mercury loading and methylmercury accumulation by aquatic biota. *Environment, Science & Technology* 41, 4952-4958. http://pubs.acs.org/doi/abs/10.1021/es063061r (accessed 2011).

Orihel D.M., M.J. Paterson, P.J. Blanchfield, R.A. Bodaly, C.C. Gilmour et al.. 2008. Temporal changes in the distribution, methylation, and bioaccumulation of newly deposited mercury in an aquatic ecosystem. *Environ Pollution* 154(1):77-88.

Paulsson, K. and K. Lindbergh. 1989. The selenium method for treatment of lakes for elevated levels of mercury in fish. *Science of the Total Environment* 87–88:495–507.

Pekar, Zakary. 2011. Memorandum from Zachary Pekar, July 1, 2011, Clarification and Updating of Mercury Deposition Maps Provided in the Technical Support Document: National-Scale Mercury Risk Assessment. http://yosemite.epa.gov/sab/sabproduct.nsf/7EEF811B90F741DF852578C00059FC64/$File/MEMO-clarification+and+updating+of+mercury+deposition+maps-July1st.pdf (accessed 9/9/11).

Pongprueksa P., C.-J.Lin, S.E. Lindberg, C. Jang, T.Braverman et al. 2008. Scientific Uncertainties in Atmospheric Mercury Models III: Boundary and Initial Conditions, Model Grid Resolution, and Hg(II) Reduction Mechanism. *Atmospheric Environment* 42, 1828-18451.

Ralston, N.V.C. and L.J.Raymond. 2010. Dietary selenium's protective effects against methylmercury toxicity. *Toxicology* 278:112-123.

Ryaboshapko, A.O., O. R. Bullock Jr., J. Christensen, M. Cohen, A. Dastoor et al. 2007. Intercomparison study of atmospheric mercury models: 1. Comparison of models with short-term measurements and Intercomparison study of atmospheric mercury models: 2. Modelling results vs. long-term observations and comparison of country deposition budgets. *Science of the Total Environment* 376 228–240.

Seppanen, K., P. Soininen, J.T., Salonen, S. Lotjonen and R. Laatikainen. 2004. Does mercury promote lipid peroxidation? An in vitro study concerning mercury, copper, and iron in peroxidation of low-density lipoprotein. *Biological Trace Element Research* 101, 117–32.

Stokes-Riner, A., S. W. Thurston et al. 2011. A longitudinal analysis of prenatal exposure to methylmercury and fatty acids in the Seychelles. *Neurotoxicology and Teratology* 33(2), 325-328.

Strain J., P.W. Davidson, M.P. Bonham, E.M. Duffy, A. Stokes-Riner et al. 2008. Associations of maternal long chain polyunsaturated fatty acids, methyl mercury, and infant development in the Seychelles child development and nutrition study. *Journal of Neurotoxicology*. 5, 776-82.

U.S. EPA. 1997. *Mercury Study Report to Congress*. EPA-452/R-97-003

U.S. Environmental Protection Agency Clean Air Scientific Advisory Committee. 2007. *Clean Air Scientific Advisory Committee's (CASAC) Review of the 1st Draft Lead Staff Paper and Draft Lead Exposure and Risk Assessments* (EPA-CASAC-07-003). p. 6.

U.S. Geological Survey and U.S. Department of Agriculture, Natural Resources Conservation Service. 2009. *Federal guidelines, requirements, and procedures for the national Watershed Boundary Dataset: U.S. Geological Survey Techniques and Methods.* 11–A3, 55 p.

van Wijngaarden, E., C. Beck et al. 2006. Benchmark concentrations for methyl mercury obtained from the 9-year follow-up of the Seychelles Child Development Study. *NeuroToxicology* 27(5): 702-709.

Watras, C. J., R.C. Back, S. Halrvosen, R.J.M. Hudson, K.A. Morrison et al.. 1998. Bioaccumulation of mercury in pelagic freshwater food webs. *Science of the Total Environment* 219:183-208.

Wiener, J.G., B.C. Knights, M.B.Sandheinrich, J.D.Jeremiason, M.E. Brigham et al. 2006. Mercury in soils, lakes, and fish in Voyageurs National Park (Minnesota): importance of atmospheric deposition and ecosystem factors. *Environmental Science & Technology* 40, 6261-6268.

Appendix A: Agency Charge Questions

Background and Charge for the SAB Review of EPA's *Technical Support Document: National-Scale Mercury Risk Assessment Supporting the Appropriate and Necessary Finding for Coal- and Oil-Fired Electric Generating Units (March 2011)*

May 23, 2011

Background

On March 16, 2011, EPA proposed National Emission Standards for Hazardous Air Pollutants (NESHAP) for coal- and oil-fired Electric Utility Steam Generating Units (EGUs). The proposed NESHAP would protect air quality and promote public health by reducing emissions from EGUs of the hazardous air pollutants (HAP) listed in Clean Air Act (CAA) section 112(b), including both mercury and non-mercury HAP. Specifically, the proposed rule would require EGUs to decrease emissions of mercury, other metal HAP, organic HAP, and acid gas HAP. Section 112(n)(1) of the CAA requires EPA to determine whether it is "appropriate and necessary" to regulate HAP emissions from EGUs under section 112. Before the Agency is authorized to make the appropriate and necessary determination, section 112(n)(1) requires EPA to perform a study of the hazards to public health reasonably anticipated to occur as a result of HAP emissions, including mercury, from EGUs after imposition of the requirements of the CAA. EPA completed the required study in 1998. (Utility Air Toxics Study, 1998). Based in part on the results of that study , EPA made a finding in December 2000 that it was appropriate and necessary to regulate HAP emissions from coal- and oil-fired EGUs. In the recently proposed NESHAP, EPA confirmed that finding and concluded that it remains appropriate and necessary to regulate HAP emissions from coal- and oil- fired EGUs. EPA confirmed the finding in part by conducting a new analysis of the human health risks posed by consuming freshwater fish containing mercury that is attributable to U.S. EGU emissions of mercury. EPA is seeking peer review of the data and methods used in the national scale mercury risk assessment as documented in the *Technical Support Document: National-Scale Mercury Risk Assessment Supporting the Appropriate and Necessary Finding for Coal and Oil-Fired Electric Generating Units* (hereafter referred to as the "Mercury Risk TSD").

In determining whether U.S. EGUs pose a hazard to public health, we developed an approach for assessing the nature and magnitude of the risk to public health posed by U.S. EGU mercury emissions (the 2005 scenario). We also estimated the health risks associated with US EGU mercury emissions estimated to remain "after imposition of the requirements of the Act" (the 2016 scenario). Specifically, for the 2016 scenario, we looked at certain regulations, including, for example, the proposed Transport Rule, which have a co-benefit impact on mercury.
Our approach focused on identifying the number of watersheds where the U.S. EGU contribution to total methylmercury (MeHg) risk is considered to represent a potential public health hazard. To do this, we focused on estimating risk associated with human exposures at those watersheds in the U.S. where we have measured data on fish tissue MeHg concentrations (about 4% of the watersheds, or 2,461 out of ~88,000 U.S. watersheds – see section 2.4 and Appendix B of the Mercury Risk TSD). For each of the 2,461 watersheds, we modeled potential risk from high-end (i.e., subsistence-level) self-caught fish consumption. Specifically, we used the fish tissue MeHg data combined with self-caught fish ingestion rates to model exposure, and then we translated that into estimates of total MeHg-related risk (see sections 1.3, 2.1 and Appendices C and D of the Mercury Risk TSD).

In our analyses, we estimated both total risk associated with emissions from all emissions sources, including global emissions, and the incremental contribution to the total risk that was attributable to mercury emissions from U.S. EGUs. We used an assumption of proportionality between mercury deposition over a watershed and the levels of MeHg in fish (and, by association, the levels of exposure and risk). This proportionality assumption is based on the U.S. EPA Office of Water's Mercury Maps assessment (see section 1.3 and Appendix E of the Mercury Risk TSD). Mercury Maps demonstrated that, under certain conditions, a fractional change in mercury deposition will ultimately translate into a similar fractional change in MeHg levels in fish. We note that the time delay between changes in deposition and changes in MeHg levels in fish is not well characterized (there are a range of assumptions and limitations associated with the Mercury Maps approach which we have considered - see below). Application of the Mercury Maps approach allowed us to translate any changes in mercury deposition to changes in MeHg fish tissue levels. It also allowed us to apportion MeHg levels in fish (and, by association, exposure and risk estimates) based on the proportionality assumption. In other words, if the estimated U.S. EGU-related emissions comprise10% of total deposition over a watershed, assuming near steady-state conditions are met, we would assume that eventually 10% of the MeHg in fish (and, therefore, 10% of the total human exposure and risk) would be attributable to U.S. EGUs.

Mercury deposition modeling was completed for two scenarios: 2005 and 2016. The analysis included consideration of mercury emitted from (a) US EGUs, (b) other non-EGU sources in the U.S. (including natural and anthropogenic), and (c) sources outside of the U.S. (both anthropogenic and natural) whose mercury is deposited in the U.S. following long range atmospheric transport. Estimates of mercury deposition within the U.S., both of total deposition and of EGU-related deposition, were completed using the Community Multiscale Air Quality model (CMAQ) version 4.7.1, which generates estimates at the 12 km grid cell-level of resolution.[6,7] CMAQ modeling reflects mercury oxidation pathways for both the gas and aqueous phases in addition to aqueous phase reduction reactions. Mercury "re-emission" is not explicitly modeled in this version of CMAQ; however, approximations of these emissions are included in the CMAQ model and called "recycled" emissions. Speciation of U.S. EGU mercury emissions is based on a factor approach reflecting coal rank, firing type, boiler/burner type, and post-combustion emissions controls. Emissions of mercury from sources in Canada and Mexico are based on the 2006 Canadian inventory and 1999 Mexican inventory, respectively. Estimates of mercury transported into the U.S. from outside North America (i.e., specification of lateral boundary concentrations, pollutant inflow into the photochemical modeling domain, and initial species concentrations) are provided by a three-dimensional global atmospheric chemistry model, the GEOS-CHEM model (standard version 7-04-11). The GEOS-CHEM predictions were used to provide one-way dynamic boundary conditions at three-hour intervals and an initial concentration field for the 36 km CMAQ simulations. The 36 km photochemical model simulation is used to supply initial and hourly boundary concentrations to the 12 km domains.[8] Mercury initial and boundary conditions were based on a GEOS-CHEM simulation using a 2000 based global anthropogenic emissions inventory that includes 1,278 Mg/yr of Hg(0), 720 Mg/yr of Hg(II), and 192 Mg/yr of particle bound mercury.[9] The description

---

[6] Foley, K.M., Roselle, S.J., Appel, K.W., Bhave, P.V., Pleim, J.E., Otte, T.L., Mathur, R., Sarwar, G., Young, J.O., Gilliam, R.C., Nolte, C.G., Kelly, J.T., Gilliland, A.B., Bash, J.O., 2010. Incremental testing of the Community Multiscale Air Quality (CMAQ) modeling system version 4.7. Geoscientific Model Development 3, 205-226.

[7] Byun, D., Schere, K.L., 2006. Review of the governing equations, computational algorithms, and other components of the models-3 Community Multiscale Air Quality (CMAQ) modeling system. Applied Mechanics Reviews 59, 51-77.

[8] USEPA, 2010. Air Quality Modeling Technical Support Document: Point Source Sector Rules (EPA-454/R-11-003), Research Triangle Park, North Carolina.

[9] Selin, N.E., Jacob, D.J., Park, R.J., Yantosca, R.M., Strode, S., Jaegle, L., Jaffe, D., 2007. Chemical cycling and deposition of atmospheric mercury: Global constraints from observations. Journal of Geophysical Research-Atmospheres 112.

of emissions and modeling presented above pertains to the 2005 scenario evaluated in the risk assessment. For the 2016 scenario, EPA projected US EGU emissions based on an Integrated Planning Model (IPM) run.[10] Mercury emissions from other U.S. anthropogenic sources are projected to 2016 based on growth factors and known controls (e.g., boilers, cement kilns). The estimates for non-U.S. global emission sources (i.e., both natural and anthropogenic) were not adjusted for the 2016 scenario.

The risk assessment for mercury focuses on two risk metrics: (a) comparison of estimated exposures to the MeHg Reference Dose (MeHg RfD) to determine the hazard quotient (HQ) for each watershed evaluated, and (b) an estimate of the number of IQ points lost to children born to mothers exposed to MeHg during pregnancy (see 1.2 of the Mercury Risk TSD). The current EPA MeHg RfD reflects the full range of potential neurodevelopmental impacts including effects on IQ, educational development, motor skills and attention. For the risk assessment, we did not estimate the incidence of adverse health effects for health endpoints other than IQ loss, as the literature and available data supporting the modeling of IQ loss is considered to be the strongest and has received the most review by the scientific community.

For each of the risk metrics modeled (RfD-based HQ and IQ loss), we identified a benchmark for a potentially significant public health impact to guide interpretation of the risk estimates. For the RfD-based HQ, we considered any exposure above the RfD (equal to an HQ of 1) to represent a potential public health hazard with recognition, as noted above, that the RfD provides coverage for the full range of neruodevelopmental impacts. In the case of IQ loss, we considered a loss of 1 or more points to represent a clear public health concern. This benchmark was based on advice received from the Clean Air Science Advisory Committee (CASAC) in relation to the Pb NAAQS review. It is important to note that CASAC identified this level of IQ loss in the context of a population-level impact (see 1.2 of the Mercury Risk TSD for additional detail on the benchmarks used to help interpret risk metrics).

For the risk assessment, we focused on high-end (subsistence) fish consumption by women of child-bearing age at inland fresh water bodies; the consumption rates used ranged from the 90th to 99th percentiles and were obtained from peer-reviewed studies of fish consumption by specific populations active within the continental U.S. (see section 1.3 and Appendix C of the Mercury Risk TSD). This overall approach reflects our assumption that U.S. EGUs will have the greatest public health impact on the subset of watersheds in the U.S. that (a) have relatively elevated fish tissue MeHg levels (increasing overall risk levels associated with MeHg exposure through fish consumption at those watersheds), (b) have relatively larger mercury deposition from U.S. EGUs (translating into a greater fractional risk associated with U.S. EGUs), and (c) have subsistence-level fishing activity (resulting in higher self-caught fish intake and higher risk). We have not focused on recreational fishing activity. Recreational fishing may be important from a population risk standpoint; however, these fishers consume less fish overall and will not have the levels of individual-risk likely to be experienced by subsistence fishers. Furthermore, we have not considered U.S. EGU impacts on commercial fish from international or near coastal locations. Although MeHg levels can be relatively high in fish from these locations, the U.S. EGU contribution (as a fraction of overall mercury impacts) is both highly uncertain and likely to be low. The high degree of uncertainty associated with linking U.S. EGU deposition to MeHg levels in fish that are either self-caught or commercially harvested near the U.S. shore led us to exclude consideration of risks linked to consumption of these fish. Specifically, given the greater mobility of these fish and the greater dilution of deposited mercury in the ocean and near coastal waters, application of the Mercury

---

[10] USEPA, 2010. Air Quality Modeling Technical Support Document: Point Source Sector Rules (EPA-454/R-11-003), Research Triangle Park, North Carolina.

Maps approach is subject to significantly greater uncertainty relative to its application to inland fresh water bodies.

The RfD-based risk characterization was done by developing HQs for each watershed. The HQ is defined as the estimate of MeHg exposure divided by the MeHg RfD. Generally (both for methylmercury and for all pollutants) a HQ of 1 or less is considered to represent a level of daily exposure for the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. We developed a 3-stage risk characterization framework to estimate the number of watersheds where the U.S. EGU contribution to total MeHg risk is considered to represent a potential public health hazard based on consideration of the HQ metric:

- Stage 1: estimate the number of watersheds where (a) potential exposure for subsistence level fish consumers exceeds the RfD (e.g., HQ > 1.0), and (b) U.S. EGUs contribute a specific fraction of mercury deposition to those watersheds (and by association, a specific fraction of total exposure and risk). Several fractions of mercury deposition were considered ranging from >5 to >20%.
- Stage 2: estimate the number of watersheds where the deposition from U.S. EGUs would result in exposures to MeHg that exceed the RfD before considering exposures to MeHg attributable to other sources. While we may consider the U.S. EGU increment of exposure, particularly in the context of comparing exposure to the MeHg RfD, it is critical to place the U.S. EGU-incremental exposure in the context of the larger total exposure at a given watershed. This reflects the fact that the MeHg RfD is for total exposure and not increments of exposure considered in isolation.
- Stage 3: estimate the total number of watersheds where populations are at risk from exposures attributable to U.S. EGU mercury emissions by merging the two sets of watersheds identified in stages 1 and 2.

    (see section 1.2 of the Mercury Risk TSD for additional detail on the 3-stage framework)

The second risk characterization was done by modeling potential IQ loss attributable to U.S. EGU emissions resulting in increased MeHg exposure (see section 1.2 of the Mercury Risk TSD). In modeling IQ loss, we first converted annual-average ingested dose estimates for MeHg into equivalent maternal hair mercury levels, since the CR function for IQ loss is based on estimated exposure characterized as maternal hair mercury levels. This was accomplished using a factor based on a one compartment toxicokinetic model used in deriving the methylmercury RfD. Then a CR function relating hair mercury levels to IQ points lost in children born to mothers whose exposure is modeled in this analysis was used to predict IQ points lost for those children. This CR function is based on application of a Bayesian hierarchical model which integrates data from the three key epidemiological studies (Seychelles, New Zealand and Faroe Islands).

As part of the risk assessment, EPA also addressed both variability and uncertainty. Regarding variability, we assessed the degree to which key sources of variability associated with the scenarios being modeled were reflected in the design of the risk model (see sections 1.4, 2.7 and Appendix F, Table F-1 of the Mercury Risk TSD). Regarding uncertainty we included a number of sensitivity analyses intended to consider the potential impact of key sources of uncertainty (with emphasis on application of the Mercury Maps assumption). We also qualitatively discussed additional sources of uncertainty and the nature and magnitude of their potential impact on risk estimates that were generated (see section 2.7 and Appendix F, Table F-2 of the Mercury Risk TSD).

Figure 1 provides a conceptual diagram for the key steps in the risk assessment.

This peer review is intended to focus on the linkages of the key data inputs, and the critical inputs related to fish consumption rates, dose-response information, and fish MeHg levels. Two key inputs to the risk assessment are the MeHg RfD and the estimates of mercury deposition from CMAQ. We believe the MeHg RfD is the appropriate indicator to use because it reflects the full range of potential neurodevelopmental impacts, including effects on IQ, educational development, motor skills, and attention. We are not requesting that this panel review the scientific basis for the MeHg RfD, rather, this review is focused on the estimation of potential exposures to MeHg for comparison against the existing RfD. The current RfD has been subject to extensive peer review and is the EPA reference value for assessing MeHg ingestion risk.[11] In addition, the CMAQ model has been extensively peer reviewed and the mercury fate and transport algorithms are documented in several peer reviewed publications.[12,13,14] Thus, we are not seeking peer review of the mercury components of the CMAQ model. However, as reflected in the charge questions, we are looking for comment on how CMAQ outputs (i.e., mercury deposition estimates) are integrated into the risk assessment to estimate changes in fish tissue MeHg levels and in exposures and risks associated with the EGU-related fish tissue MeHg fraction.

---

[11] U.S. Environmental Protection Agency (U.S. EPA). 2002. Integrated Risk Information System File for Methylmercury. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at: http://www.epa.gov/iris/subst/0073.htm.

[12] Bullock, O. R., Jr., et al. (2008), The North American Mercury Model Intercomparison Study (NAMMIS): Study description and model-to-model comparisons, J. Geophys. Res., 113, D17310, doi:10.1029/2008JD009803.

[13] Bullock, O. R., Jr., et al. (2009), An analysis of simulated wet deposition of mercury from the North American Mercury Model Intercomparison Study, J. Geophys. Res., 114, D08301, doi:10.1029/2008JD011224

[14] Pongprueksa, P., et al (2008), Scientific uncertainties in atmospheric mercury models III: Boundary and initial conditions, model grid resolution, and Hg(II) reduction mechanism, Atmospheric Environment 42: 1828–1845
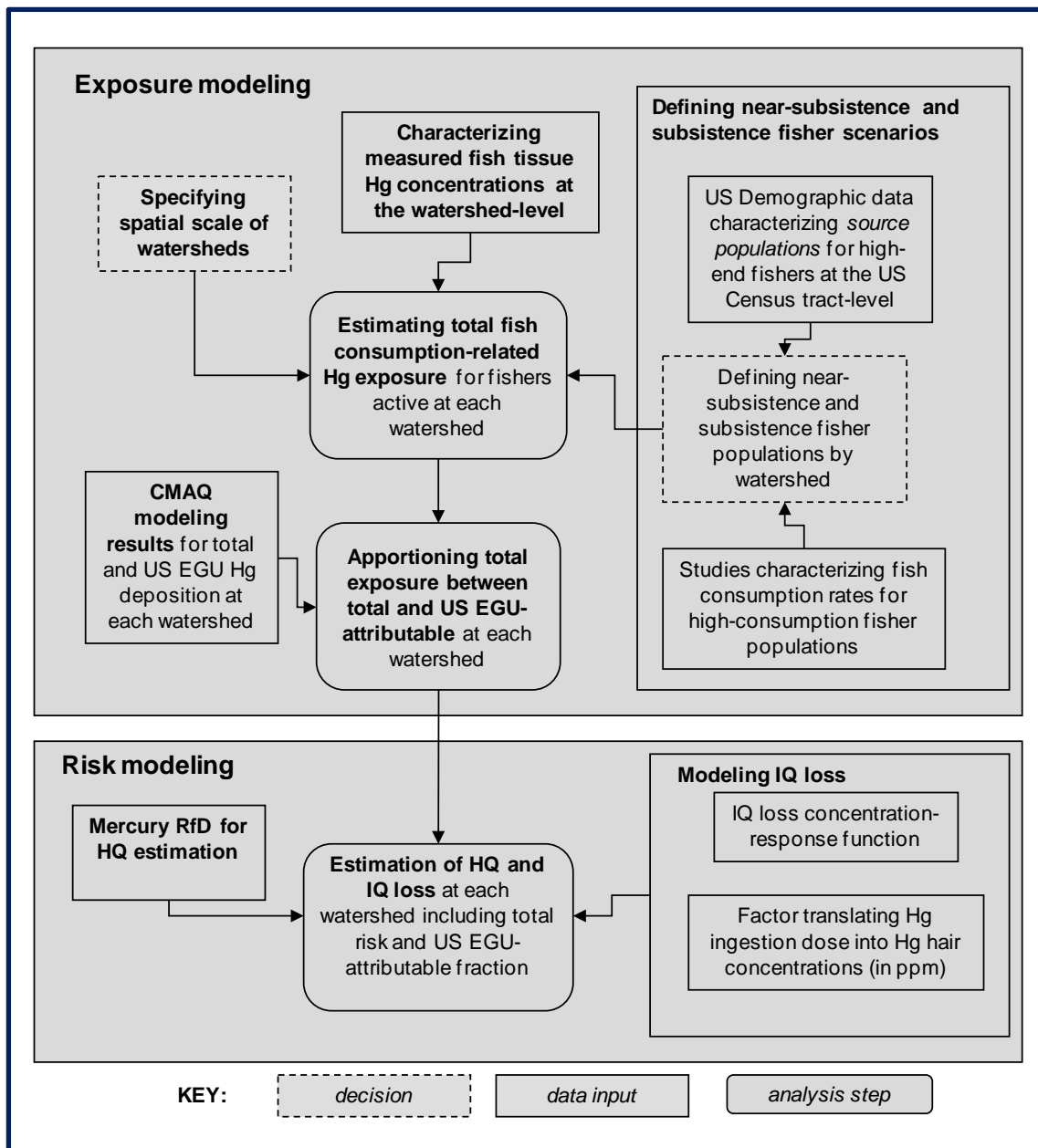
Figure 1. Flow Diagram of Risk Analysis Including Major Analytical Steps and Associated Modeling Elements (Note, GEOS-CHEM results are input into CMAQ modeling box)

Charge Questions

The charge questions presented below are organized by topic and track specific sections within the Mercury Risk TSD beginning with *Purpose and Scope of the Analysis* (section 1.1). We have included brief overviews of the technical focus of each section to help reviewers place each section in context with regard to the overall risk assessment (Note, we did not include any charge questions addressing elements of the *Executive Summary* since all technical content provided in that introductory section is covered in greater detail in the other sections of the TSD for which we have included charge questions).

Purpose and Scope of the Analysis (section 1.1)

This section presents the policy-related questions that were developed to guide the design of the risk assessment. It also highlights some important technical factors related to air-sourced mercury, in particular, mercury released from U.S. EGUs that were considered in designing the risk assessment. And finally, the section provides an overview of key elements of the scope of the risk assessment.

Question 1. Please comment on the scientific credibility of the overall design of the mercury risk assessment as an approach to characterize human health exposure and risk associated with U.S. EGU mercury emissions (with a focus on those more highly exposed).

Overview of Risk Metrics and the Risk Characterization Framework (section 1.2)

This section describes the risk metrics used in the risk assessment (i.e., IQ loss and MeHg RfD-based HQs, including both total risk and U.S. EGU-attributable risk). The section also presents the 3-stage risk characterization framework which uses these risk metrics to estimate the number of watersheds where populations may be at risk due to MeHg exposure with consideration for the U.S. EGU attributable fraction of that exposure. Questions for this section focus on the IQ calculations. As explained above, we are not asking for peer review of the current mercury RfD or its suitability as a benchmark for comparison with mercury exposures.

Question 2. Are there any additional critical health endpoint(s) besides IQ loss which could be quantitatively estimated with a reasonable degree of confidence to supplement the mercury risk assessment (see section 1.2 of the Mercury Risk TSD for an overview of the risk metrics used in the risk assessment)?

Question 3. Please comment on the benchmark used for identifying a potentially significant public health impact in the context of interpreting the IQ loss risk metric (i.e., an IQ loss of 1 to 2 points or more representing a potential public health hazard). Is there any scientifically credible alternate decrement in IQ that should be considered as a benchmark to guide interpretation of the IQ risk estimates (see section 1.2 of the Mercury Risk TSD for additional detail on the benchmark used for interpreting the IQ loss estimates).

Overview of Analytical Approach (section 1.3)

This section of the Mercury Risk TSD (together with the referenced appendices) provides a detailed overview of the technical design and inputs to the risk assessment, with the section being further divided into subsections (unnumbered) that address each of the design elements. Charge questions presented below which address the design of the risk assessment are grouped by each of these design elements.

Specifying the spatial scale of watersheds (presented within section 1.3)

This section describes the spatial unit used as the basis for the risk assessment (the HUC-12 watershed, representing a fairly refined level of watersheds approximately 5-10 km on a side) and provides the rationale for the decision to use that specific spatial scale and spatial unit in the analysis.

> *Question 4: Please comment on the spatial scale used in defining watersheds that formed the basis for risk estimates generated for the analysis (i.e., use of 12-digit hydrologic unit code classification). To what extent do HUC12 watersheds capture the appropriate level of spatial resolution in the relationship between changes in mercury deposition and changes in MeHg fish tissue levels? (see section 1.3 and Appendix A of the Mercury Risk TSD for additional detail on specifying the spatial scale of watersheds used in the analysis).*

Characterizing measured fish tissue Hg concentrations (presented within section 1.3)

This section describes the fish tissue MeHg sampling data used in the risk assessment, including the underlying sources of data used in developing the dataset and factors considered in developing the dataset (e.g., inclusion of data sampled between 2000 and 2009). This section also provides the rationale for using the 75th percentile fish tissue MeHg value (within a given watershed) as the basis for exposure and risk characterization.

> *Question 5: Please comment on the extent to which the fish tissue data used as the basis for the risk assessment are appropriate and sufficient given the goals of the analysis. Please comment on the extent to which focusing on data from the period after 1999 increases confidence that the fish tissue data used are more likely to reflect more contemporaneous patterns of mercury deposition and less likely to reflect earlier patterns of mercury deposition. Are there any additional sources of fish tissue MeHg data that would be appropriate for inclusion in the risk assessment?*

> *Question 6: Given the stated goal of estimating potential risks to highly exposed populations, please comment on the use of the 75th percentile fish tissue MeHg value (reflecting targeting of larger but not the largest fish for subsistence consumption) as the basis for estimating risk at each watershed. Are there scientifically credible alternatives to use of the 75th percentile in representing potential population exposures at the watershed level?*

Defining subsistence fisher scenarios (presented within section 1.3)

This section describes the high-end self-caught freshwater fish consuming populations evaluated for exposure and risk in the risk assessment. The section includes detailed discussion of the self-caught fish consumption rates used in modeling exposure for these study populations.

> *Question 7: Please comment on the extent to which characterization of consumption rates and the potential location for fishing activity for high-end self-caught fish consuming populations modeled in the analysis are supported by the available study data cited in the Mercury Risk TSD. In addition, please comment on the extent to which consumption rates documented in Section 1.3 and in Appendix C of the Mercury Risk TSD provide appropriate representation of high-end fish*

consumption by the subsistence population scenarios used in modeling exposures and risk. Are there additional data on consumption behavior in subsistence populations active at inland freshwater water bodies within the continental U.S.?

Question 8: Please comment on the approach used in the risk assessment of assuming that a high-end fish consuming population could be active at a watershed if the "source population" for that fishing population is associated with that watershed (e.g. at least 25 individuals of that population are present in a U.S .Census tract intersecting that watershed). Please identify any additional alternative approaches for identifying the potential for population exposures in watersheds and the strengths and limitations associated with these alternative approaches (additional detail on how EPA assessed where specific high-consuming fisher populations might be active is provided in section 1.3 and Appendix C of the Mercury Risk TSD).

<u>Apportioning total MeHg exposure between total and U.S. EGU-attributable exposure (presented within section 1.3)</u>

This section describes the application of the Mercury Maps based proportionality assumption to link changes in mercury deposition (over watersheds) to changes in fish tissue MeHg levels. The section also discusses the use of CMAQ modeling output (i.e., gridded mercury deposition estimates for both total mercury and U.S. EGU-attributable mercury) as part of this process of linking changes in U.S. EGU mercury emissions ultimately, to changes in fish tissue MeHg levels in watersheds assessed for risk in the risk assessment.

Question 9: Please comment on the draft risk assessment's characterization of the limitations and uncertainty associated with application of the Mercury Maps approach (including the assumption of proportionality between changes in mercury deposition over watersheds and associated changes in fish tissue MeHg levels) in the risk assessment. Please comment on how the output of CMAQ modeling has been integrated into the analysis to estimate changes in fish tissue MeHg levels and in the exposures and risks associated with the EGU-related fish tissue MeHg fraction (e.g., matching of spatial and temporal resolution between CMAQ modeling and HUC12 watersheds). Given the national scale of the analysis, are there recommended alternatives to the Mercury Maps approach that could have been used to link modeled estimates of mercury deposition to monitored MeHg fish tissue levels for all the watersheds evaluated? (additional detail on the Mercury Maps approach and its application in the risk assessment is presented in section 1.3 and Appendix E of the Mercury Risk TSD).

Question 10: Please comment on the EPA's approach of excluding watersheds with significant non-air loadings of mercury as a method to reduce uncertainty associated with application of the Mercury Maps approach. Are there additional criteria that should be considered in including or excluding watersheds?

<u>Estimating risk including HQ and IQ loss (presented within section 1.3)</u>

This section describes how exposure estimates generated for the high-end fish consuming populations modeled in the analysis are translated into risk estimates for those populations (in the form of both MeHg RfD-based HQs and IQ losses). This section also includes a detailed discussion of the concentration-response function used in modeling IQ loss.

Question 11: Please comment on the specification of the concentration-response function used in modeling IQ loss. Please comment on whether EPA, as part of uncertainty characterization, should consider alternative concentration-response functions in addition to the model used in the risk assessment. Please comment on the extent to which available data and methods support a quantitative treatment of the potential masking effect of fish nutrients (e.g. omega-3 fatty acids and selenium) on the adverse neurological effects associated with mercury exposure, including IQ loss. (detail on the concentration-response function used in modeling IQ loss can be found in section 1.3 of the Mercury Risk TSD).

Discussion of key sources of uncertainty and variability (section 1.4)

This section describes the extent to which the risk assessment design reflects consideration for potentially important sources of variability associated with the type of exposure being modeled. It also discusses sources of uncertainty associated with the analysis, including the nature and potential magnitude of their impact on risk estimates (Note, also that an important part of the analysis – the sensitivity analyses completed primarily to examine the potential impact of uncertainty related to the Mercury Maps approach – are discussed in section 2.7 of the Mercury Risk TSD).

Question 12: Please comment on the degree to which key sources of uncertainty and variability associated with the risk assessment have been identified and the degree to which they are sufficiently characterized.

Discussion of analytical results (section 2)

This section presents estimates generated as part of the risk assessment, including important intermediate calculations as well as the risk estimates themselves – subsections include: (a) estimates of mercury deposition over watersheds (section 2.3), (b) characterization of changes in fish tissue MeHg levels based on modeling the impact of changes in mercury deposition (section 2.4) and (c) presentation of MeHg RfD-based HQ estimates and IQ loss risk estimates (section 2.6). Key observations from the analysis are presented in section 2.8.

Question 13: Please comment on the draft Mercury Risk TSD's discussion of analytical results for each component of the analysis. For each of the components below, please comment on the extent to which EPA's observations are supported by the analytical results presented and whether there is a sufficient characterization of uncertainty, variability, and data limitations, taking into account the models and data used.
- *Mercury deposition from U.S. EGUs*
- *Fish tissue methyl mercury concentrations*
- *Patterns of Hg deposition with HG fish tissue data*
- *Percentile risk estimates*
- *Number and frequency of watersheds with populations potentially at risk due to U.S. EGU mercury emissions*

Question 14: Please comment on the degree to which the final summary of key observations in Section 2.8 is supported by the analytical results presented. In addition, please comment on the degree to which the level of confidence and precision in the overall analysis is sufficient to support use of the risk characterization framework described on page 18.