

Technical Support Document: National-Scale Mercury Risk Assessment Supporting the Appropriate and Necessary Finding for Coal- and Oil-Fired Electric Generating Units - DRAFT

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Technical Support Document: National-scale Mercury Risk Assessment Supporting the Appropriate and Necessary Finding for Coal- and Oil-fired Electric Generating Units - DRAFT

U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
Research Triangle Park, North Carolina

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Executive Summary

The EPA has completed a national-scale risk assessment for mercury to inform the appropriate and necessary determination for 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). See section III of the Preamble to the proposed U.S. EGU Toxics Rule.

This document (the "Mercury Risk TSD") describes this national-scale mercury risk analysis. This executive summary provides an overview of the risk assessment including the design of the risk assessment and the risk estimates that were generated. Additional technical detail on the risk assessment design as well as more in-depth presentation and interpretation of the risk estimates generated are provided in the body of this document.

Scope of the analysis

The national-scale risk assessment for Hg focuses on risk associated with Hg released from U.S. EGUs that deposits to watersheds within the continental U.S., bioaccumulates in fish and then is consumed as MeHg in fish eaten by subsistence fishers and other freshwater self-caught fish consumers. The risk assessment considered the nature and magnitude of the risk to public health posed by current U.S. EGU Hg emissions and the remaining risk posed by U.S. EGU Hg emissions once CAA requirements potentially reducing Hg from U.S. EGUs are in place. In both cases, we also assess the contribution of U.S. EGUs to potential risks from MeHg exposure relative to total MeHg risk associated with Hg deposited by other sources both domestic and international.

The overall design and scope of the risk assessment reflect the following factors related to exposure to air emissions of Hg: (a) the dominant pathway associated with ambient air Hg releases is through the consumption of fish that have bioaccumulated MeHg originally deposited to watersheds following atmospheric release and transport; (b) the primary focus in quantifying risk associated with consumption of fish containing MeHg is risk to children born to mothers who were exposed to MeHg during pregnancy through fish consumption; (c) because U.S. EGU Hg is likely to make a very small contribution to Hg in non-U.S. sourced bought fish consumed in the U.S. and in bought fish sourced from further off the U.S. coast, it is not useful to assess risks due to consumption of those bought fish;[1] and (d) the type of fish consumption likely to experience the greatest contributions from U.S. EGU-sourced Hg is associated with fishing activity at inland freshwater rivers and lakes located in regions with elevated U.S. EGU Hg deposition.

Current conditions with regard to U.S. EGU emissions based on the 2010 ICR show HAP emissions are closer to the 2016 emissions than to the 2005 emissions (due in part to Hg reduction co-benefits of existing state and Federal actions).[2] For this reason, in discussing risk estimates, we focus on the 2016 results.

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[1] Furthermore, although commercial fish sourced closer to the U.S. coast (including estuarine areas) may have greater U.S. EGU impacts in some cases, relative to the average U.S. EGU impact nationally, because of uncertainty in modeling the linkage between U.S. EGU deposition and the apportionment of mercury in these fish, this commercial consumption pathway is not included in the quantitative risk assessment.

[2] A number of air quality modeling runs were completed in support of this rule. For this risk assessment, we modeled risk for a 2005 and 2016 scenario, reflecting emissions of 52.9 and 29 tons of total mercury from U.S. EGUs, respectively. We also developed a current estimate of Hg emissions from EGUs based on the 2010 ICR data and that estimate was 29 tons of mercury from U.S. EGUs.

The risk assessment calculated both the magnitude of the U.S. EGU incremental contribution to total potential exposure and risk and the percent of total Hg exposures and risk contributed by U.S. EGUs (i.e., the fraction of total risk associated with U.S. EGUs) to individual watersheds for which we have fish tissue MeHg data.

Given the goal of determining whether a public health hazard is associated with U.S. EGU emissions, we have focused this analysis on those populations likely to experience the greatest risk when fishing at inland (freshwater) locations including subsistence-fishers.[3] In defining these high-end populations, we have included behavior that places them at greater risk (e.g., focusing on somewhat larger fish in supplementing their diet and focusing their fishing activity at individual watersheds - see Section 1.1 for additional detail). While these attributes define a subset of subsistence fishers, we think that they are reasonable and that fishing populations with these attributes are likely to exist and be active to some extent at the watersheds included in this risk assessment. Because the general recreational angler population is likely to experience individual risk levels well-below these high-consuming fisher populations, we have not quantified risk for this more generalized population, although the consumption rates for subsistence fishers are likely to be similar to consumption rates for high-end recreational anglers. Consumption rates for the high-end fishing populations included in the risk assessment are based on studies in the published literature, and are documented in Section 1.3 and Appendix C.

Although Hg-related risks associated with commercial fish consumption are a public health concern, the relatively low contribution of U.S. EGU Hg to this source of dietary fish (relative to non-U.S. Hg emissions), and the high levels of uncertainty in mapping U.S. EGU Hg emissions to concentrations of MeHg in ocean-going fish, precludes our assessment of this consumption pathway as part of the risk assessment. In the specific case of commercial fish sourced from near the U.S. coast and the Great Lakes, although there is the potential for U.S. EGUs to have a greater role in affecting Hg levels in these fish, 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 it in the risk assessment. However, it is likely that the range of potential exposures to U.S. EGU Hg deposition across inland watersheds captures the types of potential exposures that occur in near-coastal environments.

Risk Characterization Framework

EPA assessed risk from potential exposure to MeHg through fish consumption at a subset of watersheds across the country for which we have measured fish tissue MeHg data. This risk assessment uses estimates of potential exposure for subsistence fisher populations to generate risk metrics based on comparisons of MeHg exposure to the reference dose.[4] Because of

[3] As noted in Section 1.2, subsistence fish consumption is defined by the EPA as individuals who rely on noncommercial fish as a major source of their protein (USEPA, 2006). This definition does reflect a degree of subjectivity in terms of what is meant by "major source of their protein". For this risk assessment, we consider our high-end consumption rates (i.e., a meal every 1-2 days) as clearly subsistence.

[4] We also generated estimates of the loss in intelligence quotient (IQ) points for the same populations, however, we are focusing on the reference dose because it represents a more sensitive risk metric that potentially captures a wider range of neurobehavioral health effects (see Section 1.2 for additional discussion). .

limitations in quantifying the number of high-consumption fishers active across the set of modeled watersheds, it is not possible to generate population-weighted distributions of risk.

For the analysis, we have developed a risk characterization framework for integrating two types of U.S. EGU-attributable risk estimates. This framework estimates the total percent of watersheds with fish tissue MeHg samples (approximately 2,400 out of 88,000 watersheds) that are at risk due to potential exposures to MeHg attributable to U.S. EGU. This total percent of watersheds where populations may be at risk from EGU-attributable Hg includes watersheds that either have deposition of Hg from U.S. EGUs that is sufficient to lead to potential exposures that exceed the reference dose even without considering the contributions from other U.S. and non-U.S. sources, or have deposition of Hg from U.S. EGUs that represents a fraction (e.g., 5, 10, 15, or 20 percent) of total Hg deposition from all sources, in watersheds where potential exposures to MeHg from all sources (U.S. EGU, U.S. non-EGU, and non-U.S.) exceed the RfD.[5]

The results of the analysis include the total percent of watersheds where populations may be at risk from EGU-attributable Hg, as well as the two component estimates. This framework allows us to consider whether U.S. EGUs alone or in combination with other sources of Hg, pose a potential public health hazard. The framework also allows us to evaluate the impacts of the proposed regulation on this potential public health hazard.

Analytical Approach

Watersheds can be defined at varying levels of spatial resolution. For the purposes of this risk analysis, we have selected to use watersheds classified using 12-digit Hydrologic Unit Codes (HUC12) (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 Hg deposition and changes in MeHg levels in aquatic biota.

After estimating total MeHg risk based on modeling consumption of fish at each of these watersheds, the ratio of U.S. EGU to total Hg deposition over each watershed (estimated using Community Multi-scale Air Quality, CMAQ, modeling) is used to estimate the U.S. EGU-attributable fraction of total MeHg risk. This apportionment of total risk between the U.S. EGU fraction and the fraction associated with all other sources of Hg deposition is based on the EPA's Office of Water's Mercury Maps (MMaps) approach that establishes a proportional relationship between Hg deposition over a watershed and resulting fish tissue Hg levels, assuming a number of criteria are met (USEPA, 2001). Each of the steps in the analysis is briefly described below.

Methodology for Assessing MeHg Levels in Fish Tissues

The fish tissue dataset for the risk assessment includes fish tissue Hg samples from years 1995 to 2009, with approximately 50,000 unique samples from 4,115 HUC12s out of approximately 88,000 HUC12s in the continental U.S. The samples are more heavily focused on locations east of the Mississippi River. For this risk assessment, a subset of the data from 2000 and later was selected, with samples distributed across 2,461 HUC12s, which provided samples more representative of current conditions with regard to patterns of mercury deposition.

[5] Any contribution of Hg emissions from 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 EGUs contributed 5 percent or more to deposition to the watershed. EPA believes this is a conservative approach given the increasing risks associated with incremental exposures above the RfD.

The fish tissue samples in the master dataset come primarily from three sources: the National Listing of Fish Advisory (NLFA) database is managed by EPA;[6] the U.S. Geologic Survey (USGS), which manages a compilation of Hg datasets as part of its Environmental Mercury Mapping and Analysis (EMMA) program, and compiles Hg fish tissue sample data from a wide variety of sources (including the NLFA) and posts these data at http://emmma.usgs.gov/datasets.aspx; and EPA's National River and Stream Assessment (NRSA) study data, which includes nearly 600 fish tissue Hg samples collected at randomly selected freshwater sites across the U.S. during the period 2008 to 2009. Additional detail on these fish tissue MeHg data are provided in Section 1.3 and Appendix B.

Data from these three datasets were combined into a single master fish tissue dataset covering the period 1995 to 2009. As noted above, only a fraction (2,461) of the approximately 88,000 watersheds in the continental U.S. had fish tissue concentration data and, therefore, could be included in risk characterization. Most of the watersheds with measured fish tissue MeHg data had multiple measurements (the average number of fish tissue measurements for the period 2000 to 2009 for each of the 2,461 watersheds is 10, although some watersheds had up to 270 measurements). The assessment used the 75th percentile fish tissue value at each watershed as the basis for exposure and risk characterization, based on the assumption that subsistence fishers would favor larger fish which have the potential for higher bioaccumulation (i.e., use of a median or mean value could bias low the likely MeHg levels in typically consumed fish). The 75th percentile represents the upper bound of the interquartile range, which is generally seen as a reasonable limit on the central tendency of a distribution. Selection of the 75th percentile represents a reasonable assumption that acknowledges the median or mean fish may give too much weight to smaller, less likely to be eaten fish, while avoiding assumptions that consumers would always be able to catch and eat the largest fish with the highest MeHg levels.

Air Quality Modeling of Hg Deposition over Watersheds

Deposition of Hg was estimated using the CMAQ model v4.7.1 (www.cmaq-model.org). The CMAQ v4.7.1 is a state of the science three-dimensional Eularian "one-atmosphere" photochemical transport model used to estimate air quality (Byun et al., 2006, Appel et al., 2007, Appel et al., 2008). The CMAQ simulates the formation and fate of photochemical oxidants, ozone, primary and secondary PM concentrations, and air toxics at a 12 km gridded spatial resolution 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 et al., 2002). Because measurements for the dry deposition of Hg do not currently exist, the modeled dry deposition performance could not be evaluated. In EPA's view, CMAQ model wet deposition estimates agree well with the Mercury Deposition Network (MDN) monitoring sites with a minimal seasonal bias. Additional information on the CMAQ modeling is provided in Section 1.3 and Appendix E and Appendix F.

CMAQ modeling at a 12 km resolution was used to estimate total annual Hg deposition from U.S. and non-U.S. anthropogenic and natural sources over each watershed. In addition, CMAQ simulations were conducted where U.S. EGU Hg emissions were set to zero to determine the contribution of U.S. EGU Hg emissions to total Hg deposition. U.S. EGU-related Hg deposition characterized at the watershed-level for the two scenarios assessed (2005 and 2016) is

[6] http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/

summarized in Table ES-1 for the complete set of 88,000 HUC12 watersheds, while Table ES-2 summarizes the percent of total Hg deposition attributable to U.S. EGUs (by percentile).

Table ES-1. Comparison of Total and U.S. EGU-Attributable Hg 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

* Statistics are based on CMAQ results interpolated to the watershed –level and are calculated using all ~88,000 watersheds in the U.S.

Table ES-2. Comparison of Percent of Total Hg 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.

We note the following observations regarding estimated deposition based on information presented in Table ES-2: (a) U.S. EGUs are estimated to contribute up to 30 percent of total Hg deposition for the 2005 scenario and up to 11 percent for the 2016 scenario (99th percentile values – see Table ES-2); (b) on average, U.S. EGUs contribute a substantially smaller fraction of total Hg deposition (2 to 5 percent for the 2016 and 2005 scenarios, respectively – see Table ES-2), this reflecting contributions made by other U.S. air emissions sources and more importantly, by non-U.S. sources; and (c) U.S. EGU-related deposition is predicted to decrease substantially between the 2005 and 2016 scenarios.[7]

[7] The estimated decrease in U.S. EGU Hg emissions between 2005 and 2016 is due, in part, to decreases in SO_2 and other criteria pollutant emissions pursuant to Federal requirements and enforcement actions. If those controls were not maintained, and Hg emissions were to increase from current levels, the U.S. EGU attributable deposition, and fraction of deposition would be somewhere between the 2005 scenario and 2016 scenario.

EPA also evaluated the potential for "hot spot" deposition near U.S. EGU emission sources on a national scale, based on the CMAQ modeled Hg deposition for the 2005 and 2016 scenarios. We calculated the excess deposition within 50 km of U.S. EGU sources by first calculating the average U.S. EGU attributable Hg deposition within a 500 km radius around the U.S. EGU source. This deposition represents the likely regional contribution around the EGU. We then calculated the average U.S. EGU attributable Hg deposition within 50 km of the U.S. EGUs to characterize local deposition plus regional deposition near the EGU. Excess local deposition is then the 50 km radius average deposition minus the 500 km radius average deposition. Summary statistics for the excess local deposition are provided in Table ES-3. Table ES-3 shows both the mean excess deposition around all U.S. EGUs, and the mean excess deposition around just the top 10 percent of Hg emitting U.S. EGUs. Table ES-3 also shows the excess Hg deposition as a percent of the average regional deposition to provide context for the magnitude of the local excess deposition. In 2005, for all U.S. EGU, the excess was around 120 percent of the average deposition, while for the top 10 percent of Hg emitting U.S. EGU, local deposition was around 3.5 times the regional average. By 2016, the absolute excess deposition falls, however, the local excess still remains around 3 times the regional average for the highest 10 percent of Hg emitting U.S. EGUs (note, additional detail on this hot spot assessment is not provided in the main body of the TSD, since it is clearly laid out here).

Table ES-3. Excess Local Deposition of Hg Based on 2005 CMAQ Modeled Hg Deposition

Category of Results	50km-Radius-Average Excess Local Deposition values (µg/m2)	
	Mean Across EGUs (percent of regional average deposition)	
	2005 Scenario	2016 Scenario
All U.S. EGU sites with Hg emissions >0 (672 sites)	1.65 (119%)	0.36 (93%)
Top ten percent U.S. EGU in Hg emissions (67 sites)	4.89 (352%)	1.18 (302%)

This analysis shows that there is excess deposition of Hg in the local areas around EGUs, especially those with high Hg emissions. Although this is not necessarily indicative of higher risk of adverse effects from consumption of MeHg contaminated fish from watersheds around the U.S. EGUs, it does indicate an increased chance that Hg from U.S. EGUs will impact local watersheds.

Estimating the Proportion of Total Hg Exposure Associated with U.S. EGUs and Projecting Changes in Fish Hg concentrations

The MMaps approach specifies that, under certain conditions (e.g., Hg deposition is the primary loading to a watershed and near steady-state conditions have been reached), a fractional change in Hg deposition to a watershed will ultimately be reflected in a matching proportional change in the levels of MeHg in fish. This proportionality assumption between deposition changes and fish tissue MeHg concentrations can be used to both estimate the portion of total Hg exposure that is associated with U.S. EGUs and project changes in fish Hg concentrations (and

consequently total exposure) associated with changes in total Hg deposition in the future.[8] This assumption holds in watersheds where air deposition is the primary source of Hg loadings, and as a result, watersheds where this is not the case are removed from the risk analysis. MMaps is discussed in greater detail in Section 1.3 and in Appendix E and Appendix F.

For the 2005 analysis, CMAQ modeling results for a particular watershed allow U.S. to estimate the proportion of total exposure (estimated for that watershed) that is associated with U.S. EGU deposition (i.e., based on the ratio of U.S. EGU Hg deposition to total Hg deposition over the watershed as specified by the MMaps approach). In the case of the 2016 future simulation, we can first project changes in total fish tissue Hg levels (for that watershed) by comparing estimates of total Hg deposition in 2005 to estimates for 2016 generated by CMAQ and then again, apportion that adjusted total risk between U.S. EGUs and all other sources, based on comparing U.S. EGU Hg deposition to total Hg deposition in 2016.

Patterns of U.S. EGU-attributable fish tissue MeHg concentrations are summarized in Tables ES-4 and ES-5. Table ES-4 compares total and U.S. EGU-attributable fish tissue MeHg concentrations for the 2016 and 2005 scenarios by watershed percentile (including the percent reduction between 2005 and 2016). Table ES-5 summarizes the percent of total fish tissue MeHg concentrations attributable to U.S. EGUs (also by watershed percentile).

Table ES-4. Comparison of Total and U.S. EGU-Attributable Fish Tissue MeHg Concentrations for the 2005 and 2016 Scenarios

Statistic	Fish tissue MeHg concentration (ppm)				% change (2016 versus 2005) in fish tissue MeHg concentration	
	2005 Scenario		2016 Scenario			
	Total	U.S. EGU-attributable	Total	U.S. EGU-attributable	Total	U.S. EGU-attributable
mean	0.31	0.024	0.29	0.008	-6.7%	-65%
50th Percentile	0.23	0.014	0.20	0.005	-10.0%	-61%
75th Percentile	0.39	0.032	0.36	0.011	-6.4%	-67%
90th Percentile	0.67	0.056	0.63	0.019	-5.9%	-66%
95th Percentile	0.91	0.079	0.87	0.026	-4.7%	-67%
99th Percentile	1.34	0.150	1.29	0.047	-3.7%	-68%

Table ES-5. Comparison of U.S. EGU Fraction of Total Fish Tissue MeHg Levels*

Statistic	U.S. EGU-attributable percent of total fish tissue MeHg levels	
	2005 Scenario	2016 Scenario
Mean	9%	4%
50th Percentile	6%	3%
75th Percentile	14%	5%

[8] The risk assessment estimates risk for future points in time once near steady state conditions have been reached following simulated changes in mercury deposition and does not attempt to simulate the temporal profile of that response. As noted in Section 1.3 and Appendix E, the amount of time required for MeHg levels in fish to fully respond following a change in mercury deposition can range from years to decades depending on the nature of the watershed involved (e.g., methylation potential, importance of watershed runoff to load mercury into the waterbody).

Statistic	U.S. EGU-attributable percent of total fish tissue MeHg levels	
	2005 Scenario	2016 Scenario
90th Percentile	20%	7%
95th Percentile	26%	9%
99th Percentile	40%	18%

* These values are specifically for the 2,461 watersheds included in the risk assessment.

We note the following observations regarding fish tissue MeHg levels based on information presented in Tables ES-4 and ES-5: (a) U.S. EGUs can contribute up to 18 percent of total fish tissue MeHg levels for a subset of the watersheds with fish tissue data (99th percentile watershed for the 2016 Scenario – see Table ES-4); (b) on average, U.S. EGUs contribute 4 percent of total fish tissue MeHg levels (for the 2016 Scenario); and (c) reflecting the pattern seen with Hg deposition, the U.S. EGU-attributable fraction of fish tissue MeHg levels is estimated to decrease significantly between the 2005 and 2016 scenarios.

Defining Subsistence Fisher Scenarios

As discussed above, this analysis focuses on higher-consumption self-caught fisher populations active at inland freshwater locations, because these populations are expected to experience the greatest U.S. EGU-attributable risks. Therefore, in reviewing studies of fishing populations, emphasis was placed on identifying surveys of higher consumption fishing populations active at inland freshwater rivers and lakes within the continental U.S. Information on the studies used to develop the high end fish consumption scenarios for the risk analysis is provided in Section 1.3 and in Appendix C.

Based on EPA's review of the fish consumption literature, EPA defined consumption rates for the subsistence fisher populations modeled across the 2,461 watersheds included in the risk assessment. We used the studies referenced above as a guide to characterize high-end consumption behavior for a scenario that could be assessed broadly across the 2,461 watersheds. Generally all of the studies identified high-end percentile consumption rates (90th to 99th percentiles for the populations surveyed) ranging from approximately one fish meal every few days to a larger fish meal very day (i.e., 120 grams per day (g/day) to greater than 500 g/day fish consumption). We used this trend across the studies to support application of a generalized female high-end fish consumption scenario (high-end female consumer scenario) across most of the 2,461 watersheds.[9]

Consumption rates for this high-end female consumer were based on values presented for female fishers in South Carolina. Values from the South Carolina study were used because they specifically covered high-end consumption by women, which is the population of interest in this risk assessment. Furthermore, the consumption rates identified in the South Carolina study (123 g/day to 373 g/day for the 90th and 99th percentiles, respectively) are in the range of values seen across the other studies reviewed in designing this analysis and therefore are considered to be generally representative of subsistence consumption (these consumption rates translate into approximately one 8oz fish meal every other day).

[9] Reflecting the fact that higher levels of self-caught fish consumption (approaching subsistence) have been associated with poorer populations, we only assessed this generalized high-end female consumer scenario at those watersheds located in U.S. Census tracts with at least 25 individuals living below the poverty line (this included the vast majority of the 2,461 watersheds and only a handful were excluded due to this criterion).

In addition to the studies used to define the high-end fish consuming populations modeled in the risk assessment, we also reviewed a large number of additional studies characterizing higher-level self-caught fish consumption in the U.S. While these studies had limitations that prevented their use as the basis for defining high-end fisher scenarios to include in the analysis, in several instances, they did support the levels of self-caught fish consumption modeled in the analysis. These studies are described in Appendix C.

Risk Related to Exposure to MeHg in Fish and Assessment of Contribution of U.S. EGUs to MeHg Exposure and Risk

This section provides an overview of risk estimates generated for the 2,461 watersheds included in the risk assessment.[10] As noted above, we focus on risk estimates generated for the high-end female consumer assessed at the national level, since this population, as defined for this analysis, provides the most comprehensive coverage for watersheds with fish tissue MeHg data across the U.S. and because the consumption rates used to model this population represent subsistence levels that would characterize populations most likely to experience high levels of exposure to MeHg and thus experience higher risk.

We estimated total exposure to MeHg at each of the 2,461 watersheds.[11] Estimates of total Hg exposure were generated by combining 75th percentile fish tissue values with the consumption rates for female subsistence fishers. A cooking loss factor (actually reflecting the fact that the preparation of fish can result in increased Hg concentrations) was also included in exposure calculations (Morgan et al., 2007).

We estimate the fraction of total potential exposure that is associated with U.S. EGU Hg deposition at each watershed using the proportionality assumption supported by MMaps (see above). Once total potential exposure to MeHg has been estimated and the U.S. EGU-attributable portion of that exposure has been estimated, we then estimate risk based on exposures above the RfD, both due to total MeHg, and MeHg attributable to U.S. EGU without consideration of other U.S. and non-U.S. sources.

A summary of risk estimates is presented here and detailed summaries of the risk estimates are presented in Section 2.6. Our estimates of total percent of watersheds where populations may be at risk from EGU-attributable Hg are as high as 28 percent.[12] The upper end estimate of 28 percent of watersheds reflects the 99th percentile fish consumption rate for that population modeled, and a benchmark of 5 percent U.S. EGU contribution to total Hg deposition in the watershed. Any contribution of Hg emissions from EGUs to watersheds where potential

[10] Each fish consumption rate scenario was assessed for a subset of the 2,461 watersheds based on consideration for where "source populations" for each scenario were located (i.e., a watershed was modeled if it fell within a U.S. Census tract containing the source population for a particular fisher scenario). The high-end female consumption rate scenario is the scenario with the broadest spatial coverage since it was applied to all watersheds intersecting U.S. Census tracts with at least 25 poor white individuals (i.e., the "source population" for high-end female self-caught fish consumers). This meant that this scenario was assessed for 2,366 of the 2,461 watersheds with fish tissue MeHg data.

[11] As noted earlier, each high-end fish consuming population included in the analysis was assessed for a subset of these watersheds, depending on which of those watersheds intersected a U.S. Census tract containing a "source population" for that fish consuming population. Of the populations assessed, the high-end female consumer scenario was assessed for the largest portion (2,366) of the 2,461 watersheds.

[12] As noted earlier, the determination of whether U.S. EGUs make a significant contribution to total Hg deposition is only considered for watersheds where total risk is considered to represent a potential hazard to public health (e.g., Potential MeHg exposure from all U.S. and non-U.S. sources exceeds the RfD).

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 EGUs contributed 5 percent or more to deposition to the watershed. EPA believes this is a conservative approach given the increasing risks associated with incremental exposures above the MeHg RfD. Scenario The percent of where populations may be at risk from EGU-attributable Hg based on the 2010 ICR would be somewhat higher due to the greater level of Hg emissions in that case (35 tons in 2010 compared with 29 tons in 2016).

Of the total percent of watersheds where populations may be at risk from EGU-attributable Hg, we estimate that up to 22 percent of watersheds included in this analysis could be potentially at risk based on consideration of the U.S. EGU attributable fraction (e.g., 5, 10, 15, or 20 percent) of total Hg deposition over watersheds with total risk judged to represent a public health hazard (MeHg total exposure greater than the RfD). The 22 percent estimate is also generated for the 2016 scenario and is based on the same assumptions used in the estimate of total risk provided earlier (i.e., 99th percentile fish consumption rate, and a 5 percent U.S. EGU contribution to total Hg deposition over a given watershed). We do note however, that, specifically with regard to the HQ estimates, any contribution of mercury from EGUs to watersheds with exposures exceeding the MeHg RfD represents a potential 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. We think this is a conservative approach given the increasing risks associated with incremental exposures above the MeHg RfD.

Of the total percent of watersheds where populations may be at risk from EGU-attributable Hg, we estimate that up to 12 percent of the watersheds could potentially be at risk based on watersheds where the U.S. EGU incremental contribution to exposure exceed the MeHg RfD, without consideration of contributions to exposures from U.S. non-EGU and non-U.S. sources. The upper end estimate of 12 percent of watersheds is based on the 2016 Scenario and reflects a scenario using the 99th percentile fish consumption rate.

The two component estimates of percent of watersheds where populations may be at risk from EGU-attributable Hg do not sum to the total percent watersheds where populations may be at risk from EGU-attributable Hg of 28 percent due to overlap in the risk estimates – some watersheds where U.S. EGUs contribute greater than 5 percent to total Hg deposition also have U.S. EGU attributable exposures that exceed the RfD without consideration of exposures from other U.S. and non-U.S. Hg sources.

The percentage of watersheds where U.S. EGUs contribute to exposures of concern increases dramatically as we consider higher fish consumption scenarios. Exposures based on the 99th percentile consumption rate represent close to maximum potential individual risk estimates. These consumption rates are based on data reported by fishers in surveys, and, thus, represent actual consumption rates in U.S. populations. However, EPA does not have data on the locations where these high self-caught fish consuming populations reside and fish, and as a result, there is also increased uncertainty about the percent of watersheds that might experience potential exposures at the highest levels.

With regard to the other fisher populations included in the full risk assessment (Vietnamese, Laotians, Hispanics, blacks and whites in the southeast, and tribes in the vicinity of the Great Lakes), our risk estimates suggests that the high-end female consumer assessed at the

national-level generally provides coverage (in terms of magnitude of risk) for all of these fisher populations except blacks and whites in the southeast.[13]

Variability and Uncertainty (Including Discussion of Sensitivity Analyses)

The risk assessment has been designed to reflect consideration for key sources of variability associated with the exposure scenario (e.g., spatial pattern of total and U.S. EGU-related Hg deposition, spatial variation in fish tissue MeHg levels, variation in the location and behavior of high-consuming fisher populations). The degree to which critical sources of variability have been reflected in the design of the analysis is discussed in Appendix F, Table F-1.

Key sources of uncertainty potentially impacting the risk analysis include: (a) uncertainty in predicting Hg deposition over watersheds using CMAQ; (b) uncertainty in predicting which watersheds will be subject to high-end fishing activity an the nature of that activity (e.g., frequency of repeated activity at a given watershed and the types/sizes of fish caught); and (c) uncertainty in using MMaps to apportion exposure and risk between different sources including U.S. EGUs and predict changes in fish tissue MeHg levels for future scenarios. We describe key sources of uncertainty impacting the risk analysis, including their potential impact on the risk estimates and the degree to which their potential impact is characterized as part of the analysis in Appendix F, Table F-2.

As part of the risk assessment, we have also completed a number of sensitivity analyses focused on exploring the impact of uncertainty related to the application of the MMaps approach in apportioning exposure and risk estimates between sources (U.S. EGU and total) and in predicting changes in fish tissue MeHg levels. These sensitivity analyses have explored: (a) concerns over including watersheds that may be disproportionately impacted by non-air Hg sources;[14] and (b) concern that the MMaps approach may be more representative when applied to stationary waterbodies (in the analysis, the MMaps was applied to watersheds including a mixture of flowing and stationary waterbodies). The results of the sensitivity analyses, when considered in aggregate, suggest that uncertainties due to application of MMaps are unlikely to have a substantial effect on the risk estimates discussed here.

Key Observations

The following key observations result from consideration of the risk estimates generated (additional detail on these observations is presented in Section 2.8):

- Reflecting current emissions, U.S. EGUs can contribute up to 11 percent of total Hg deposition (for the 99th percentile watershed in the 2016 Scenario). U.S. EGUs (for the 2016 scenario) contribute on average, about 2 percent of total Hg deposition across the country.

[13] Specifically, upper percentile risk estimates for the high-end female consumer assessed at the national level were notably higher than matching percentile estimates for the Hmong, Vietnamese, Hispanic and Tribal populations. By contrast, risk estimates for whites in the southeast were somewhat higher than the high-end female consumer, while risk estimates for blacks in the southeast were notably higher (see summary of risk estimates in the TSD supporting the A&N Determination (see section 2.6.1 for additional detail on these risk estimates).

[14] In addition to non-air Hg sources of loadings, some regions of concern may also have longer lag period associated with the linkage between Hg deposition such that the fish tissue MeHg levels we are using are actually associated with older historical Hg deposition patterns.

The average U.S. EGU deposition decreased from approximately 5 percent of total to approximately 2 percent of total for the 2005 and 2016 Scenarios, respectively.

- Although U.S. EGUs contribute on average, about 2 percent of total Hg deposition for the 2016 Scenario, they contribute about 4 percent of the fish tissue MeHg levels at watersheds included in this analysis. This discrepancy reflects the fact that fish tissue MeHg sampling is focused in the eastern half of the country which has higher U.S. EGU Hg deposition levels, compared with the national average, and, therefore, the fraction of MeHg in fish tissue attributable to U.S. EGUs will also be larger given that it is driven by estimates of U.S. EGU Hg deposition over these watersheds. U.S. EGUs are estimated to contribute up to 18 percent of fish tissue MeHg levels in the 2016 scenario (for the 99th percentile watershed).
- Comparing the pattern of U.S. EGU-attributable Hg deposition with watersheds containing fish tissue MeHg data results in our concluding that, while we have some degree of coverage for high U.S. EGU impact areas, this coverage is limited. For this reason, we believe that the actual number of where populations may be at risk from EGU-attributable Hg (i.e., watersheds where U.S. EGUs could contribute to a public health hazard) could be substantially larger than estimated.
- We estimate that up to 28 percent of the watersheds included in this risk assessment could have populations potentially at-risk under the 2016 scenario. This total risk estimate reflects a combination of watersheds where the U.S. EGU incremental contribution alone is considered to represent a potential public health hazard or where U.S. EGUs make at least a 5% contribution to total Hg deposition over watersheds where total risk is considered to pose a public health hazard. The 28 percent total risk estimate is also based on application of the 99th percentile consumption rate for the high-end female consumer.
- Reductions in U.S. EGU-attributable Hg will reduce the magnitude of US EGU-attributable risk, although substantial risk from Hg deposition will likely remain as a results of these sources.
- Sensitivity analyses were conducted primarily to examine uncertainty in applying the MMaps approach for linking Hg deposition to fish tissue MeHg levels. These analyses suggest that uncertainty related to the MMaps approach is unlikely to substantially affect our assessment of the public health hazard posed by Hg emissions from U.S. EGUs.

1. Review of Analysis Approach

1.1. Purpose and Scope of Analysis

This document (the "Mercury Risk TSD") describes the national-scale risk assessment for mercury completed to inform the appropriate and necessary determination for 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). See Section III of the preamble to the proposed U.S. EGU Toxics Rule. This risk assessment focuses on risk associated with mercury released from U.S. EGUs that has deposited to freshwater watersheds within the continental U.S., bioaccumulated in fish and is consumed as methylmercury (MeHg) in dietary fish by the public.

The following policy-related questions were developed to help guide the design of the risk assessment: (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 requirements,[15] 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? The last policy-related question reflects the fact that mercury emitted from U.S. EGUs does not result in a distinct and isolated exposure pathway, but rather is combined with mercury 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 MeHg, we determine what share of total exposure is attributable to U.S. EGUs.

In addition to the above policy-related questions, the overall design and scope of the risk assessment reflects consideration of important technical factors related to air-sourced mercury, including, in particular, mercury released from U.S. EGUs (Note, a number of these technical factors are discussed in greater length, including provision of relevant citations, later in Section 1.3):

- While mercury exposure and risk can occur through a variety of pathways, the dominant pathway associated with ambient air releases is through the consumption of fish that have bioaccumulated mercury originally deposited to watersheds following atmospheric release and transport. Deposition of mercury to watersheds includes mercury originating from local/regional sources, combined with mercury that has been transported over greater distances, including mercury released outside of the U.S.. Generally oxidized (divalent) and particle-bound mercury will deposit relatively closer to the release source, while elemental mercury will travel further, often becoming part of the global pool, before being deposited.[16]

[15] For purposes of this analysis, we focus on 2016 as this is the first year after compliance would be required to occur.

[16] 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

- Available information supporting the quantification of mercury-related health effects provides the strongest support for modeling neurological deficits in children who were exposed to MeHg during pregnancy through maternal fish consumption.[17]
- U.S. EGU emitted mercury is likely to make a very small contribution to MeHg in foreign-sourced commercial fish consumed in the U.S. and in commercial fish sourced form further off the U.S. coast. Therefore, the risk assessment, while acknowledging these sources of exposure to U.S. EGU-sourced mercury, does not quantify these risks since the U.S. EGU-attributable portion of these risks is likely to be very small and any quantitative estimates of U.S. EGU-attributable risk would be highly uncertain.[18]
- While areas closer to the US coast (including estuarine areas) and from the Great Lakes may have elevated U.S. EGU impacts in some cases, because of uncertainty in modeling the linkage between U.S. EGU deposition and the apportionment of mercury in fish, we have not included this commercial consumption pathway in the quantitative risk assessment.[19]
- The type of fish consumption likely to experience the greatest contributions from U.S. EGU-sourced mercury is associated with fishing activity at inland freshwater rivers and lakes located in regions experiencing relatively elevated U.S. EGU mercury deposition. While the average U.S. EGU percent contribution to total mercury deposition in the U.S. in 2005 was estimated at ~5% and in 2016 at ~2%, some watersheds had U.S. EGU contributions ranging up to 30% and higher in 2005 (see Section 2.3). Therefore, efforts to identify areas with likely high U.S. EGU attributable MeHg exposures and risk are focused on assessing risk for those areas which have (a) relatively elevated fish tissue MeHg levels and (b) relatively elevated levels of U.S. EGU mercury deposition (with the assumption that the elevated U.S. EGU deposition in these regions translates into a larger

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global scale deposition. Hg(2+) and Hg(p) deposit quickly from the atmosphere impacting local and regional areas in proximity to sources.

[17] 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 during pregnancy) 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 1.3).

[18] While mercury released from U.S. EGUs does contribute to contamination of foreign-sourced commercial fish, the fraction contributed by U.S. EGUs is extremely small. Current estimates of U.S. EGU mercury emissions are ~29 tons per year (see section 2.2), compared with global anthropogenic mercury emissions (for 2005), excluding biomass burning, estimated at approximately 2,120 tons with a range of 1,347 to 3,255 tons/year (Pirrone et al., 2010, UNEP, 2010). Based on these estimates, we would expect U.S. EGUs to contribute less than 1% of the mercury in commercially (foreign) sourced fish. Therefore, particularly in the context of estimating individual risk, U.S. EGU contributions to risk that residents in the US experience through consumption of foreign-sourced commercial fish is expected to be to small to characterize. 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-US anthropogenic emissions which are globally transported.

[19] 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., a number of factors make the simulation of the linkage between mercury deposition and fish tissue MeHg levels in these near-coastal areas and the Great Lakes challenging and uncertain. Specifically, the size of the waterbody involved (i.e., inner coastal waterways, near coastal areas and the Great Lakes) combined with the potential for fish to have larger habitats in these locations, relative to inland lakes and rivers, means we cannot adequately quantify the EGU contribution to fish tissue MeHg levels. Given the greater uncertainty associated with simulating the linkage between near coastal U.S. EGU deposition and fish tissue MeHg levels, we have elected not to simulate this pathway in the risk assessment.

fraction of the MeHg levels being attributed to this source category). Furthermore, while recreational angler activity is likely to occur broadly across the U.S., high self-caught fish consumers (i.e., the subsistence populations) will experience the greatest degree of U.S. EGU-attributable risk if they are active at high U.S. EGU-impact watersheds.

In consideration of the policy questions and technical factors discussed above, the national-scale Hg risk assessment was designed as follows.

- *Evaluate risk for two scenarios - a 2005 Scenario and a 2016 Scenario.* Risk is estimated both for a 2005 Scenario and a 2016 Scenario, the latter reflecting consideration of potential HAP emission reductions from CAA requirements. The latest emissions data (see Section 2.2) suggest that current 2010 U.S. EGU emissions are closer to levels reflected in the 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, as the CAA directs the Agency to consider only Federal CAA requirements in estimating future HAP emissions (and attendant risks) associated with EGUs. Thus, we conclude that if we find there exists a public health hazard from current U.S. EGU mercury emissions based on the 2016 Scenario, we will also find that a public health hazard will continue to exist in 2016.
- *Include estimates of total (all mercury deposition sources) risk 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 mercury 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 before taking into account deposition and exposures resulting from other sources of Hg. These two risk metrics reflect the cumulative burden of mercury exposures and incremental contribution that the U.S. EGU attributable deposition makes to the overall exposures to MeHg.[20]
- *Focus on assessing risk to subsistence fishers active at inland watersheds***.** Given the goal of determining whether a public health hazard is associated with U.S. EGU emissions, we have assessed risk for a set of subsistence populations active at inland (freshwater) watersheds. By focusing on inland watersheds, we are focusing on those locations with the greatest U.S. EGU-attributable mercury deposition and consequently the greatest U.S. EGU-attributable fish tissue MeHg levels. Furthermore, by focusing on subsistence fisher scenarios, we focus on those self-caught fish consumers with the highest intake rates and therefore, those who will experience the greatest MeHg exposures at a given watershed. In defining the high-end fisher populations to include in the analysis, we have used peer-reviewed study data characterizing behavior for a variety of high-end fisher populations

[20] 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 placing it in context with regard to risk associated with total MeHg in the fish being consumed.

active in different regions of the country (e.g., Laotians, Great Lakes Tribal populations, Black and White anglers active in the Southeast - see Section 1.3).

- *Generate watershed-level estimates of risk for a representative fisher for each fisher population (these estimates are not population-weighted)*: In modeling risk for these populations, we generate watershed-level estimates based on the subset of watersheds in the U.S. where we have fish tissue MeHg data and where we believe a given high-end fisher population could be active.[21] Because it is not possible to enumerate these high-end fisher populations, we cannot develop population-weighted risk distributions.[22] Therefore, in modeling risk, we generate a risk estimate for each high-end fisher population for each watershed where we believe that population could be active. We are then able to generate percentile risk estimates, based on the set of watershed-level risk estimates generated for each fisher population (i.e., assuming each watershed gets equal weight in deriving that risk distribution).
- *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 mercury to this source of dietary fish (relative to non-US mercury emissions), leads us to exclude this consumption pathway from the risk assessment. In the specific case of commercial fish sourced from near the US coast (e.g. Chesapeake Bay) and the Great Lakes, while there is the potential for U.S. EGUs to have a greater role in effecting mercury levels in these fish, as noted earlier, uncertainty associated with modeling the linkage between U.S. EGU mercury deposition and mercury exposure and risk for this dietary pathway precludes us from including this pathway in the risk assessment.

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[21] The potential for a high-end fisher population to be active at a given watershed is based on consideration for whether members of the demographic group from which that fishing population originates are located in the US Census tract(s) intersecting that watershed. For example, if we are considering Hispanic high-end fishers, we would only assess that scenario at watersheds located in U.S. Census tracts with at least 25 poor Hispanics (in this case, poor Hispanics represent a "source population" for this category of high-end fisher – see Section 1.3 for additional detail).

[22] 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 consumers of childbearing age who either fish themselves and consume at this level, or are associated with male fishers who fish at this level (with that female in turn consuming at a subsistence rates). However, currently available information does not allow us to estimate each of these subgroups of high-end fishers. Specifically, while we have data on frequency of recreational angling within the U.S., this covers general recreational fishing and not subsistence fishing. Furthermore, there are concerns as to whether surveys of recreational activity would effectively capture subsistence fishers who include poorer individuals who traditionally have lower survey response rates. We do have surveys like the Burger et al., 2002 study which provide fish consumption rates for percentiles of the survey populations, with the upper percentiles (i.e., 95th and 99th percentiles) approaching subsistence levels (we could interpret this as suggesting that 1 to 5% of the surveyed fishing population at these shows consumes at a subsistence level). However, we would still be concerned as to the degree that this type of fishing show population accurately captures rates of subsistence fishing by poorer individuals who may not frequent a show like this in proportion to their prevalence in the general population. And finally, we have focused on a subset of female subsistence consumers that we believe (a) are reasonably likely to exist at a subset of our watersheds and (b) are likely to experience higher risk due to their behavior (i.e., favor larger fish as a dietary source, focus their activity at individual watersheds). While we believe it is reasonable to assume that a subset of high-end fishers would have these attributes, this introduces additional uncertainty into any effort to enumerate this female high-end consuming population.

1.2. Overview of Risk Metrics and the Risk Characterization Framework

The risk assessment uses estimates of exposure for subsistence fisher populations[23] to generate two categories of risk metrics including (a) IQ loss in children born to mothers from these high-consuming fisher populations and (b) hazard quotient (HQ) estimates generated by comparing exposure estimates for these populations to the MeHg RfD. As discussed in greater detail in Section 1.3, risk estimates (for these populations) are generated for the subset of watersheds in the US where we have sampled fish tissue data. Because of limitations in quantifying the number of high-consumption fishers active across the set of modeled watersheds, it is not possible to generate population-weighted distributions of risk (see previous section). However, we use the watershed-level estimates of risk to consider the percentage of watersheds modeled that fall within specific risk ranges.

Both the IQ loss and HQ risk metrics are further stratified to consider both total risk as well as U.S. EGU-attributable risk. In considering U.S. EGU-attributable risk we generate two types of risk estimates:

- *The percent or fraction of total risk at a given watershed that is associated with U.S. EGU's*. We consider the magnitude of total risk (IQ loss or HQ) and then estimate the fraction (or percent) of that total risk estimates that is attributable to U.S. EGUs.
- *Risk when deposition from U.S. EGUs is considered before taking into account deposition and exposures resulting from other sources:* Here, we estimate risk based on the U.S. EGU incremental contribution to total exposure. Specifically, for IQ loss, we are using the U.S. EGU-portion of exposure at each watershed to generate an estimate of U.S. EGU attributable IQ loss and for HQ, we are comparing U.S. EGU-attributable exposure against the MeHg RfD.

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. With regard to HQ estimates based on the MeHg RfD, we considered exposures above the RfD to represent a potential public health hazard.[24] Because HQ estimates, by convention, have been reported to one significant figure, reflecting precision in the underlying

[23] Subsistence fishers are individuals who rely on noncommercial fish as a major source of protein (US 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.

[24] EPA's interpretation for this assessments is that any exposures to MeHg above the RfD are of concern given the nature of the data available for mercury 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 pregnant mothers; therefore, the RfD does not include extrapolations from animals to humans, and from the general population to sensitive subpopulations. In addition, there is no evidence for a threshold observed for critical effect of neurological deficits in children studied in the prinicipal studies of the IRIS assessment for MeHg. This additional confidence in the basis for the RfD suggests that all exposures above the RfD can be interpreted with more confidence as causing a potential hazard to public health.

RfD, mathematically, we considered exposures that were at least 1.5 times the RfD (i.e., an HQ ≥ 1.5), to represent a potential public health hazard since these would round to an HQ of 2.

Risk Characterization Framework

We have developed a 3-stage framework for integrating the risk metrics described above (throughout this document, this will be referred to as the "risk characterization framework"):

Stage 1 *– consider various degrees of U.S. EGU contribution to total risk 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 mercury by: (a) identifying those watersheds where total risk meets or exceeds levels considered to represent a potential public health hazard (i.e., HQ ≥ 2 or IQ loss estimates of 1 to 2 points or greater) 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% [20%?]). We note that, any contribution of mercury emissions from U.S. EGUs to watersheds where potential exposures from total mercury 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 deposition to the watershed. EPA believes this is a conservative approach given the increasing risks associated with incremental exposures above the RfD..

Stage 2 *– identify watersheds where risk based on considering deposition from U.S. EGUs before taking into account deposition and exposures resulting from other sources of Hg represents a potential public health hazard?* Here we identify watersheds with populations potentially at-risk due to U.S. EGU-attributable risk (prior to considering mercury contributed by other sources). Although this stage focuses on U.S. EGU exposure, it is important to keep this incremental exposure in perspective with regard to total MeHg exposure which typically dominates the U.S. EGU increment across watersheds.

Stage 3 – *what is the combined total number of watersheds (and percentage) where populations may be at risk from U.S. EGU-attributable Hg?* Here we combine estimates from Stages 1 and 2 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 alone, represents a potential public health hazard.

This framework allows us to consider whether U.S. EGU-related exposure when considered alone, 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 mercury emissions.

Note, that while we present both MeHg RfD-based HQ and IQ loss-based risk metrics in section 2.6, in discussing risk estimates in the context of determining whether a potential public

health hazard exists due to U.S. EGU mercury emissions, we focus on the HQ estimates. This reflects concerns that the IQ loss endpoint may not capture all of the neurodevelopmental effects associated with MeHg exposure. Specifically, 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 neurdevelopmental 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 therefore could further underestimate overall neurodevelopmental impacts (Axelrad et al., 2007).

1.3. Overview of Analytical Approach

This section describes the analytical approach used in conducting the national-scale mercury risk assessment (note, additional detail on specific modeling elements can be found in the appendices).

Figure 1-1 provides a flow diagram of the risk analysis identifying the major analytical steps and associated modeling elements. The risk assessment is based on estimating a set of subsistence fisher scenarios at watersheds across the U.S. where we have measured fish MeHg concentration data. After we have estimated total MeHg risk based on modeling consumption of fish at each of these watersheds, we use the ratio of U.S. EGU to total Hg deposition over each watershed (estimated using Community Multi-scale Air Quality (CMAQ) modeling) to estimate the U.S. EGU incremental contribution to total Hg risk. This apportionment of total risk between the U.S. EGU fraction and the fraction associated with all other sources of Hg deposition is based on the EPA's Office of Water's Mercury Maps approach (MMaps) that establishes a proportional relationship between Hg deposition over a watershed and resulting fish tissue Hg levels, assuming a number of criteria are met. Each of the steps in the analysis is briefly described below.

Specifying the spatial scale of watersheds

The fist 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. A number of 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 12-digit Hydrologic Unit Code classifications (HUC- 12's) (representing a fairly refined level of watersheds 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 mercury deposition and changes in MeHg levels in aquatic biota could be effectively explored at the level of these more spatially refined watersheds. In addition, use of a more refined spatial scale (i.e., use of HUC12s rather than a coarser scale of watershed) in linking changes in mercury deposition to changes in fish tissue Hg levels 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 MeHg levels. As discussed later in this section, this linkage is central to generating risk estimates and determining the fraction of total risk associated with U.S. EGUs. Note, the term "watershed" when used in this document refers to HUC12s unless otherwise noted (see Appendix A for additional detail on the rationale for selecting HUC12s as the watershed spatial scale to use in the analysis).

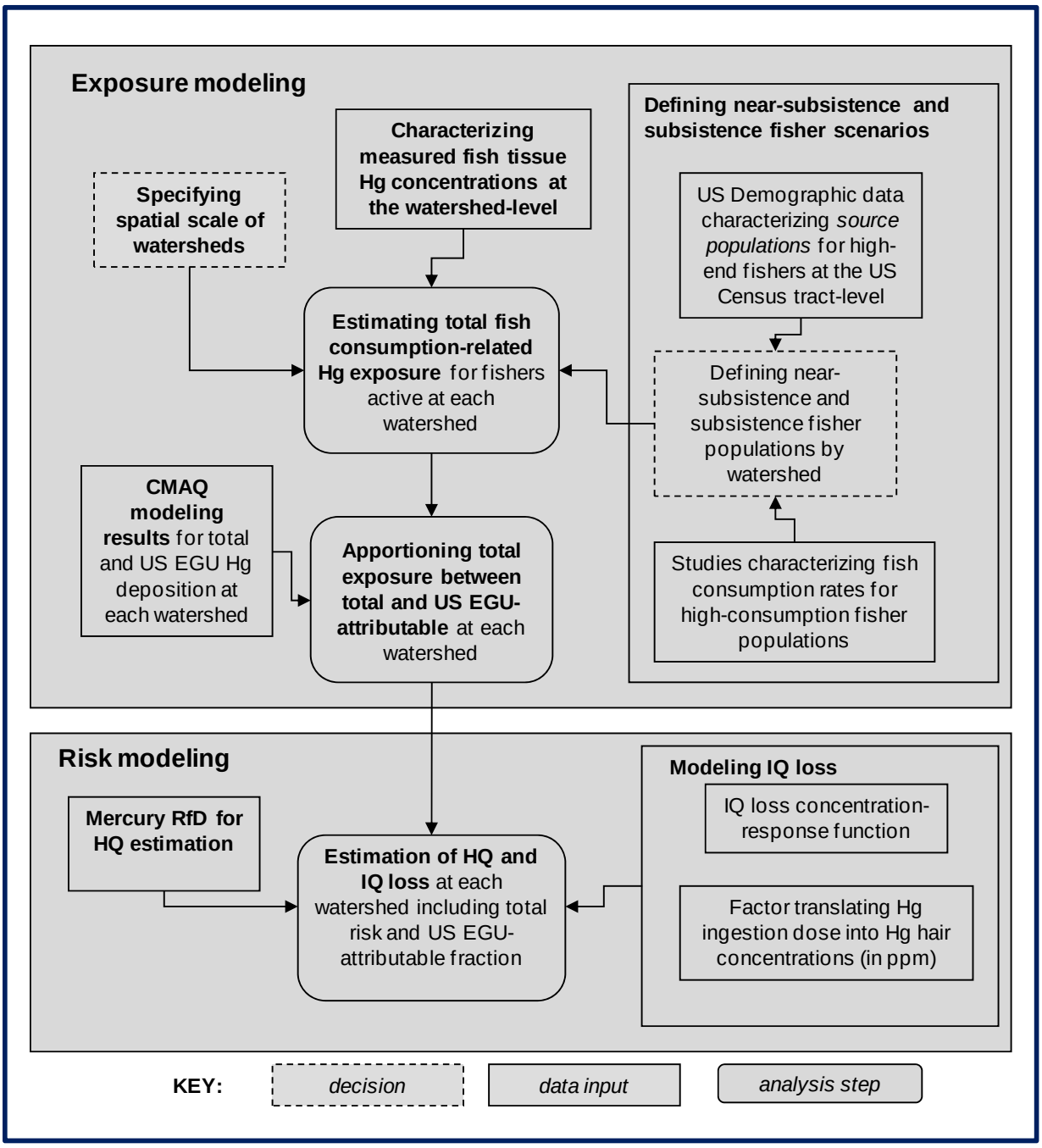


Figure 1-1 Flow Diagram of Risk Analysis Including Major Analytical Steps and Associated Modeling Elements

Characterizing measured fish tissue Hg concentrations

The next step was to identify which of the approximately 88,000 HUC12s in the continental U.S. had fish tissue concentration data and therefore, could be included in the risk characterization. Although we had compiled fish tissue Hg sampling data for the period 1990 to 2009 from a variety of sources (see Appendix B), we decided to use a subset of these data from the period 2000 to 2009 in the risk assessment in order to exclude fish tissue samples that likely reflected Hg deposition levels from the 1990's when anthropogenic emissions in the U.S. were higher than for period after 2000. We recognizes the complex spatial and temporal nature of the response of fish tissue Hg levels 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 (see Appendix B). Inclusion of fish tissue Hg sampling data collected between 2000 and 2009 resulted in the ability to characterize fish tissue Hg levels for 2,461 of the 88,000 HUC12's in the continental U.S. These watersheds were not randomly sampled across the 88,000 HUC12s, and watersheds in the eastern U.S. are more heavily represented. In addition, because the samples were often based on state sampling, the samples are not evenly distributed across states, and some states have very few samples, while others have a large number of samples. Uultimately, the risk assessment includes estimates of risk for up to 2,461 watersheds depending on the fisher population being considered (fisher populations that are less ubiquitous such as the Vietnamese or Great Lakes Tribal populations will not be active across the entire set of 2,461 watersheds and would therefore, only be assessed for a subset of the watersheds– see discussion below).[25]

Most of the watersheds with measured Hg fish tissue data had multiple fish tissue MeHg measurements. This necessitated selecting a fish tissue MeHg statistic to use as the basis for generating risk estimates. This analysis uses the 75th percentile fish tissue value (computed separately for each watershed) to model risk. The selection of this statistic reflected the potential for subsistence-like fishers to favor larger fish, which would likely have relatively higher mercury levels due to greater bioaccumulation (see Appendix B for additional detail).

Defining subsistence fisher scenarios

Next, we identified the suite of subsistence fishing populations to evaluate in the risk assessment. As discussed in the introduction, this analysis focuses on populations with higher consumption rates of self-caught fish who have the potential to fish at inland freshwater locations, since these populations are expected to experience the greatest U.S. EGU-attributable risks. Therefore, in reviewing studies of fishing populations, emphasis was placed on identifying surveys of higher consumption fishing populations active at inland freshwater rivers and lakes within the continental U.S.

A number of studies were identified that characterized activity for a selection of high-end fishing populations that met our criteria. These populations included: (a) white and black populations (including female and poor strata) surveyed in South Carolina (Burger et al., 2002), (b) Hispanic, Vietnamese and Laotian populations surveyed in California (Shilling et al., 2010)

[25] As discussed in Appendix B, in identifying the 2,461 watersheds to include in the risk assessment, we excluded those that contained gold mines or non-EGU sources of mercury emissions meeting specific criteria we identified as potentially representing a significant contribution to mercury loading within a watershed.

and (c) Great Lakes Tribal populations (Chippewa and Ojibwe) active around the Great Lakes (Dellinger et al., 2004). For most of these fisher populations (with the exception of the Tribal populations near the Great Lakes) we assumed that high-end fisher population could be generalized beyond the specific areas covered in a particular study. This type of generalization was necessary to provide greater coverage for the continental U.S. and in particular, the eastern part of the U.S. where U.S. EGU deposition is higher and where we have more measured Hg fish tissue data. In deciding how to extend coverage for each fisher population, we have considered several factors including (a) the degree to which high-end fishing activity might be culturally related and therefore more likely to be followed by populations (e.g., of a given ethnicity) living across the U.S. and (b) the degree to which high-end fishing activity might be driven by economic need (i.e., to truly supplement diet) and therefore linked to groups of individuals living below the poverty line. In each case, we have described our rationale for the regional extension that has been applied for each of the high-end fisher populations included in the analysis (see Appendix C and Table C-1 for additional detail on the regions used in modeling each fisher population). Note, that in identifying the specific subset of the 2,461 watersheds to model for each fisher population, we considered whether there was a "source population" for that fishing population within the US Census tract that intersected each watershed. For example with Vietnamese fishers, we would require that at least 25 Vietnamese were located in a US Census tract intersecting a watershed in order for that watershed to be included in the set modeled for this fisher population (additional detail on "source populations" is presented in Appendix C).

The studies used to characterize high-end fishing behavior for these populations also included either (a) high-end percentile self-caught fish consumption rates (90^{th} to 99^{th} percentile values generally on the order of 100g/day to ~400 g/day or more) or (b) the statistical parameters necessary to calculate those high-end percentiles (e.g., median and standard deviations). These subsistence-level consumption rates were used in modeling risk for these populations (see Appendix C and specifically, Table C-1 for specific consumption rates for each of the fisher populations included in the analysis.

As part of the analysis, in addition to the high-end fisher populations listed above, we also included a high-end female consumer scenario that was applied more broadly to most of the watersheds included in the risk assessment.[26] This fisher scenario was based on fish consumption rate data provided in the study by Burger et al., 2002 (see Appendix C, Table C-1) and the consumption rates involved are generally supported by a number of the studies reviewed (see Appendix C for additional detail). Because this high-end female consumer population (a) covers the population of greatest concern from a MeHg exposure standpoint (women of child-bearing age) and (b) was fairly widely applied (increasing the potential for including high U.S. EGU-impacted watersheds), we have focused the discussion of risk results provided later in Section 2.6 on this fisher population. With regard to this fisher population, as well as the other populations considered in the analysis, we would point out that the high-end fish consumption rates considered, while representing high-end (near bounding) levels, are still reasonable in terms of subsistence consumption. Most of the rates used (see Appendix C, Table C-1), translate into

[26] We applied this high-end fish consuming population to watersheds located in U.S. Census tracts which had at least 25 people living below the poverty line (this results in this scenario being applied to most of the watersheds where we have fish tissue MeHg data, since this is a less stringent criterion for inclusion). This requirement of having at least 25 people below the poverty line reflects the assumption that near-subsistence levels of fishing activity is more likely among individuals who are economically disadvantaged.

between one fish meal every few days to a large fish meal every day. Viewed from the perspective of the type of subsistence fishing activity that is the focus of this analysis, these consumption rates are reasonable (i.e., they represent consumption rates in line with subsistence behavior).

Estimating total fish consumption-related Hg exposure

The next step in completing the analysis was to estimate total exposure to Hg at each of the 2,461 watersheds for the set of high-end fish consumption populations identified for each watershed. Estimates of total mercury exposure were generated by combining 75th percentile fish tissue value with the consumption rates for a particular fisher populations. Note, that a cooking loss factor (actually reflecting the fact that the preparation of fish can result in increased mercury concentrations) was also included in exposure calculations (see Appendix D for additional detail on the calculation).

Apportioning total MeHg exposure between total and U.S. EGU-attributable exposure

Next we needed to estimate the fraction of total exposure that is associated with U.S. EGU Hg deposition at each watershed. U.S. EGU apportionment of total Hg exposure is based on application of the MMaps assumption (see Appendix E). Essentially this approach assumes that under near steady state conditions, a fractional change in mercury deposition to a watershed will be reflected in a matching proportional change in the levels of MeHg in fish. We have extended this proportionality assumption to allow us to apportion MeHg levels in fish between mercury sources based on the associated apportionment of mercury deposition within a given watershed between these sources. Of course, the process of mercury loading and impacts on mercury bioaccumulated in fish is complex and involves varying temporal profiles depending on a variety of factors (e.g., methylation potential of the waterbody, role of watershed sediment erosion and runoff in mercury loading to the watershed etc). However, for purposes of this analysis, we make the assumption that given sufficient time to achieve near steady-state conditions, a given ratio of total Hg deposition to U.S. EGU deposition will ultimately be reflected in the fish tissue MeHg levels. In addition, we note that the MMaps assumption does require that certain criteria be met (e.g., atmospheric deposition is the primary source of mercury loading to the watershed and that factors related to methylation potential in watersheds be held constant for a sufficient time to allow near-steady state conditions to be reached). These criteria and the degree to which they were considered in our analysis are further described in Appendix E. For example, as discussed in greater detail in Appendix E, in identifying the 2,461 watersheds to include in the risk assessment, we excluded those that contained gold mines or had significant non-aerial sources of mercury loading.

CMAQ modeling completed at the 12km grid cell resolution was used to estimate total annual mercury deposition from US and foreign anthropogenic and natural sources over each watershed, including the fraction of deposition contributed by U.S. EGUs. As noted in the discussion of the scope of the analysis presented in Section 1.1, we are modeling two temporal period in the analysis: (a) a 2005 scenario representing 2005 conditions as reflected in the 2005 NEI mercury emissions inventory and (b) an 2016 based case scenario after CAA-related regulations potentially reducing Hg emissions from U.S. EGUs (e.g., the Transport Rule) are in place. In the context of these two temporal scenarios, the CMAQ modeling results together with

the MMaps assumption can be used to both estimate the portion of total Hg exposure that is associated with U.S. EGUs and project changes in fish Hg concentrations (and consequently total exposure) associated with changes in total Hg deposition in the future. Specifically, for the 2005 analysis, CMAQ modeling results for a particular watershed allow us to estimate the proportion of total exposure (estimated for that watershed) that is associated with U.S. EGU deposition (i.e., based on the ratio of U.S. EGU Hg deposition to total Hg deposition over the watershed). In the case of the 2016 simulation, we can first project changes in total fish tissue Hg levels (for that watershed) by comparing estimates of total Hg deposition in 2005 to estimates for 2016 generated by CMAQ and then again, apportion that adjusted total risk between U.S. EGUs and all other sources, based on comparing U.S. EGU Hg deposition to total Hg deposition in 2016. See Appendix E for additional detail on CMAQ modeling.

Estimate risk including HQ and IQ loss

Once both total exposure and the U.S. EGU incremental contribution to that total exposure have been estimated, we can then estimate risk, including both HQ and IQ points lost. The HQ estimate is generated by comparing total Hg exposure (as annual-average bodyweight-adjusted ingested MeHg dose) to the MeHg RfD.[27] Similarly, the U.S. EGU incremental contribution to total HQ is estimated by comparing the U.S. EGU increment of exposure to the MeHg RfD. As noted earlier, an HQ > 1.5 (which rounds to an HQ of 2) is considered to represent a potential public health hazard, since it signifies that exposure has been assessed to exceed the MeHg RfD.[28]

In the case of IQ loss, we first covert 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 conversion is accomplished using a factor based on a one compartment toxicokinetic model used for deriving the MeHg RfD by Swartout and Rice (2000). Then a CR function relating hair mercury levels to IQ points lost in children born to mothers whose exposure is modeled in this analysis is used to predict IQ points lost for those children. 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).[29]

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

[27] The MeHg RfD is 0.0001 ug/kg-day (equivalent to 0.1 ug/kg-day) and was published by EPA in the Integrated Risk Information System in 2001 (http://www.epa.gov/iris/subst/0073.htm)

[28] 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 exposure above the RfD increase the risk.

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

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.

1.4. Discussion of key sources of uncertainty and variability

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. The key sources of variability associated with the analysis include: (a) variation in the pattern of total and U.S. EGU-attributable mercury deposition across watersheds in the US, (b) variation in the patterns of fish tissue MeHg levels across the US, and (c) variation of the types of high-end fishing activity likely to occur in different parts of the country. These sources of variability and the degree to which they are reflected in the design of the analysis are identified and described in Appendix F, Table F-1.

Regarding uncertainty, in Appendix F, Table F-2, we have identified sources of uncertainty impacting the analysis and attempted to 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).

In addition to the sources of uncertainty addressed in Table G-2, which focus on factors related directly to the exposure scenarios modeled in the risk assessment, our decision not to model risk associated with consumption of Great Lakes fish and fish sourced from near U.S. coastal locations (including estuarine areas) also adds additional uncertainty into the analysis. As explained above in Section 1.1, these sources of self-caught and commercial fish were not modeled primarily due to challenges associated with linking specific areas of U.S. EGU mercury deposition to fish in these waterbodies. A related concern is that the greater dilution and potential mobility of fish in these larger waterbodies, could reduce the impact of elevated U.S. EGU mercury deposition of portions of these waterbodies. Despite these considerations, the risk assessment may have overlooked elevated U.S. EGU-attributable risks for high-consuming fisher populations active in these locations. However, we would point out that we still expect the greatest U.S. EGU-attributable risk to occur at inland freshwater bodies with (a) relatively elevated fish tissue Hg levels and (b) relatively elevated levels of U.S. EGU mercury deposition.

1.5. Differences between the 2005 Section 112(n) Revision Rule analysis and the current analysis in support of the Propose U.S. EGU Toxics Rule

In 2005, EPA conducted a set of technical analyses to support revision of the 2000 appropriate and necessary finding.[30] 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:

[30] 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.

Higher spatial resolution through use of CMAQ 12km grid cells*:* We are now using 12 km grid cells in estimating mercury deposition using CMAQ, whereas in the 2005 analysis, we used 36 km grid cell modeling. The more refined grid cells used in the current analysis are more appropriate for representing areas of elevated U.S. EGU deposition (and total Hg deposition in general) compared with the 36 km grid cells used in the 2005 analysis. The 12 km grid cell 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 mercury 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 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 2009. By contrast, the the 2005 analysis used data collected between 1999 and 2003 (in that case to support an analysis completed in 2005).

Subsistence fisher activity better defined and considered more ubiquitous: Based on an extensive review of available literature, we have 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, Tribal populations). Although it was necessary to extrapolate high-end fishing activity to regions beyond those covered in the underlying studies, we do believe that the literature generally supports the plausibility of high-end subsistence-like fishing activity existing across to some extent 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 populations in different regions of the country, adds support to assessing this type of fishing behavior across the modeled watersheds (for additional detail on the fishing populations included in this risk assessment, including consumption rates see Section 1.4 and Appendix C).

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 ended up applying a high-end self-caught percentile values (95^{th} and 99^{th} percentiles) based on Tribal fishing practices in the Northwest to watersheds across the country.[31] The updated literature review we have done for the current analysis, has lead us to

[31] Note, that these NW Tribal fishing estimates are subject to considerable uncertainty when they, in particular, are extrapolated to cover 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 significant uncertainty in extrapolating this type of highly-specific cultural-based fishing activity to other Tribes, let alone 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.[32]

For the current analysis, we are also using the 75th percentile fish tissue MeHg level 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 fish tissue MeHg level across species of fish in a given HUC8, which is 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 have compared total exposure to the MeHg RfD to generate an HQ estimate based on total mercury 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 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 US-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 mercury 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 mercury-related risk associated with the non-U.S. EGU portion of risk.

For the current analysis, we also generated estimates of IQ loss (these were not generated for the the 2005 analysis). Estimates of a specific health endpoint associated with the U.S. EGU-attributable fraction of mercury in fish provides a risk metric that can be more appropriately considered in isolation (i.e., it is more reasonable to consider the U.S. EGU-attributable IQ loss

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other fish consumers in the U.S. By contrast, extrapolation of more generalized high-consumption 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 far less uncertainty.

[32] Note, that particularly in the situation where a study specifically characterized poor high-end fishing populations, as is done in the Burger 2010 study of activity in SC, we considered it reasonable to assume that poor 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).

than to focus on the U.S. EGU-attributable fraction of an HQ). This reflects the fact that IQ loss quantifies a discrete increment of a public health effect, while with the RfD, it is more difficult to characterize what a "fraction" of an HQ value actually represents in terms of potential health significance. However, as discussed in Section 1.2, there is concern that the IQ endpoint may not fully capture all of the neurodevelomental effects associated with MeHg exposure and for this reason, in presenting risk estimates in the context of determining whether there is the potential for a public health hazard associated with exposure to U.S. EGU-sourced mercury, we focus on the MeHg RfD-based HQ estimates and not on the IQ loss estimates, although both are presented.

2. Discussion of Analytical Results

This section provides a discussion of the results from the various analyses completed as part of the risk assessment for the 2005 simulation and the 2016 simulation. Prior to discussing these results, 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). The specific sets of analyses described in this section include: (a) mercury emissions from U.S. EGUs (Section 2.2), (b) mercury deposition from U.S. EGUs as modeled using CMAQ (Section 2.3), (c) fish tissue MeHg concentrations (Section 2.4), (d) relationship between mercury deposition and methlymercury fish tissue concentrations (Section 2.5) and (d) risk assessment results, including MeHg RfD-based HQ estimates and IQ loss estimates (Section 2.6). In discussing each of these category of results, emphasis is placed on identifying key policy-relevant observations. In Section 2.7, 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 2.8, we provide a summary of critical observations from the analysis.

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 discussed in the following sections (note, that this only highlights portions of the design of the analysis – the reader is referenced to Section 1.3 and associated Appendices for a more in-depth discussion of the analysis design):

- 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 Lakes. In comparing any risk profiles generated in this analysis to risks estimated in other contexts, the specific focus on this analysis on these high-end populations needs to be considered (e.g., risks in this analysis will generally be substantially higher than those estimated for recreational fishers).

- The analysis is watershed-focused and risks are generated for subsets of the 2,461 watersheds for which we have measured fish tissue MeHg data. This watershed coverage (which is only about 4% of the watersheds in the U.S.), 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 mercury deposition. Further, we note that the watersheds with fish tissue MeHg data are concentrated in the eastern part of the country

and therefore, this portion of the continental U.S. is more heavily represented in the watershed-level estimates of risk that are generated. Given that U.S. EGU mercury 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 a strength of the analysis.

- The analysis uses the MMaps approach to relate changes in mercury deposition over particular watersheds to resulting changes in mercury fish tissue concentrations. Similarly, this approach allows us to reflect source-apportionment of mercury deposition (i.e., between U.S. EGU and all other sources) in the underlying fish tissue levels. It is then possible to translate those changes in fish tissue levels (or source-apportioned fish tissue levels) into equivalent changes in exposure and risk. This approach assumes that near steady state conditions are met in the fish tissue MeHg concentrations, which may take years to decades at a given watershed following changes in mercury deposition.

- The analysis generates a series of risk metrics based on (a) estimates of MeHg RfD-based HQ and (b) estimates of IQ loss in children born to mothers exposed to MeHg through high-end fish consumption. We do consider the U.S. EGU-related contribution to both types of risk. However, particularly in the context of HQ, U.S. EGU incremental contributions to total risk should always be considered in the context of total HQ which is typically substantially larger than the U.S. EGU incremental contribution when considered in isolation.

- Because it is not feasible to enumerate the subsistence-like fisher populations modeled in this analysis, we could not generate distributions of population-weighted risk for specific fishing populations assessed (e.g., poor Hispanic fishers, or Tribal fishers in the vicinity of the Great Lakes). To reiterate, this reflects the fact that we are focusing on fishers engaging in high fish consumption and it is difficult to count individuals in this subset of each fishing population (and assign them to specific watersheds). While we can not enumerate these populations, we do believe that, based on surveys of their behavior, that this type of subsistence-like activity could reasonably be expected to occur across some fraction of the 2,461 watersheds included in the analysis.[33] Therefore, we have assessed high-end fisher risk for each watershed. We then consider the fraction of watersheds with simulated high-end risk within specific categories of interest. While not a population-representative characterization of risk, this approach does allow us to consider percentiles of watersheds based on a reasonable assumption that this kind of high-end fishing activity could occur across the watersheds modeled.

2.2. Mercury Emissions from U.S. EGUs

The most recent data on U.S. EGU emissions based on information collected from industry through the Information Collection Request (ICR) show total mercury emissions of 29 tons in 2010. This shows a significant reduction in U.S. EGU mercury emissions from 2005, when mercury emissions were estimated to be 52.9 tons. The reductions between 2005 and 2010

[33] As discussed in section 1.3 and in additional detail Appendix C, we only considered specific high-end populations for those watersheds located in US Census tracts with a "source population" greater than 25 for the fishing population being assessed.

are largely due to state mercury regulations and federal enforcement actions that achieve mercury reductions as a co-benefit of controls for NOx and SO2 emissions. The EPA projection of total mercury emissions from U.S. EGUs in the 2016 (once a number of the CAA-related regulations are fully in effect) is 29 tons. Given these estimates of total mercury emissions, characterization of "current conditions" would be better represented by our 2016 Scenario than the 2005 Base Case, since total emission for the former (at 29 tons) is closer to our projection of current 2010 emissions (at 29 tons). By contrast, the 2005 analysis reflects total mercury emissions (52.9 tons) which are significantly higher than our estimate of current emissions in 2010. For this reason, as mentioned earlier, we emphasis risk estimates for the 2016 Scenario in presenting and interpreting risk estimates.

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

This section characterizes patterns of U.S. EGU-related mercury deposition for the two scenarios assessed (2005 and 2016 Scenario) using CMAQ. In presenting and discussing these results, we contrast U.S. EGU-attributable deposition with deposition from all sources combined. This discussion is based around a series of figures and tables conveying relevant information, which are described below. After presenting these figures, a set of bulleted observations is presented 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 mercury deposition (ug/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 mercury deposition (ug/m^2) at the watershed-level, again for the 2005 and 2016 scenarios, respectively.

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

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

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

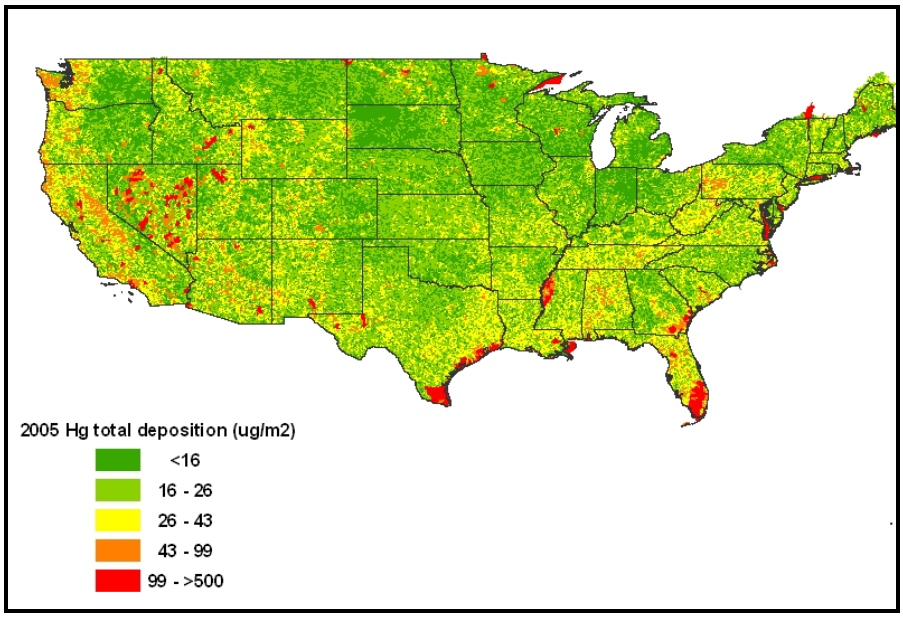


Figure 2-1. Total mercury deposition by watershed (2005)

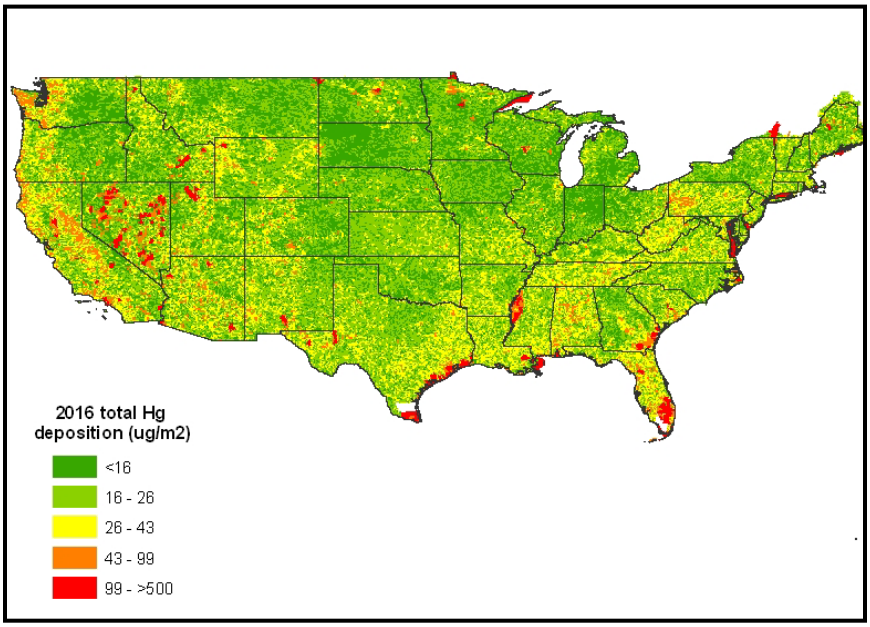


Figure 2-2. Total mercury deposition by watershed (2016)

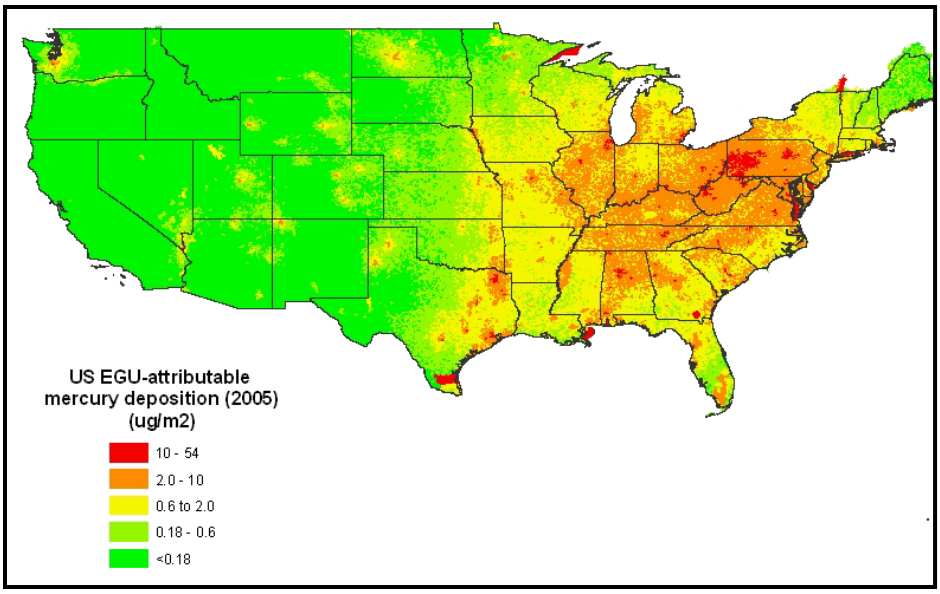


Figure 2-3. U.S. EGU-attributable mercury deposition by watershed (2005)

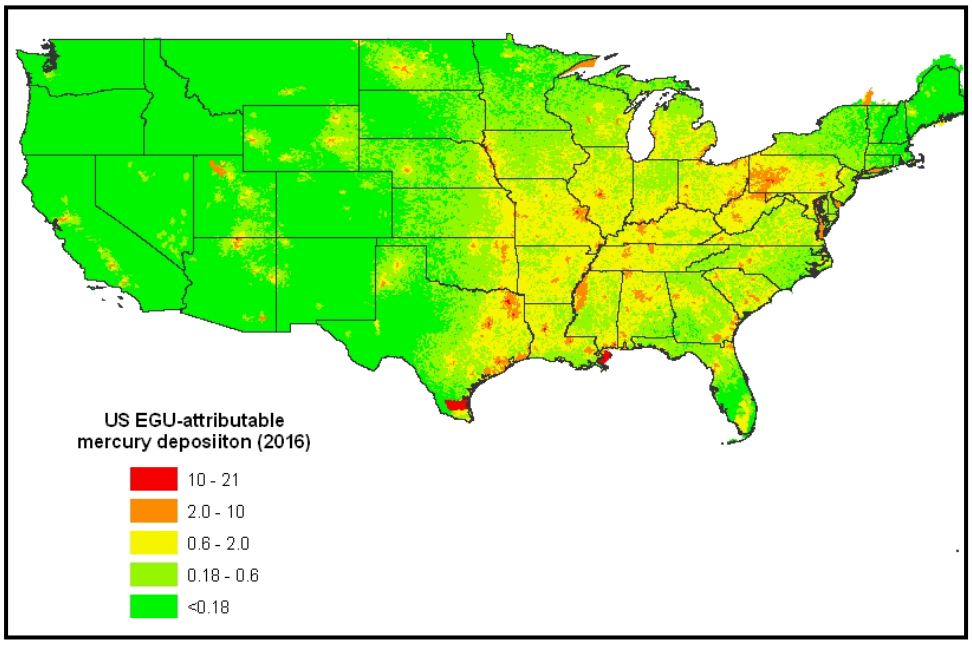


Figure 2-4. U.S. EGU-attributable mercury deposition by watershed (2016)

Table 2-1. Comparison of total and U.S. EGU-attributable mercury deposition (ug/m2) 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 U.S.

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 for this category due to presence of a number of watersheds with very small U.S. EGU-attributable deposition values which skewed this distribution.

Consideration of information presented above in Figures 2-1 through 2-4 and in Tables 2-1 through 2-3 resulted in the following observations regarding estimates of total and U.S. EGU-attributable mercury deposition for the 2005 and 2016 scenarios (note, all observations referencing the U.S. are for the continental U.S.):

- *Patterns of total and U.S. EGU-related Hg deposition differ considerably*: The pattern of total Hg deposition across the country is different from the pattern of U.S. EGU deposition. There are areas of elevated total Hg deposition 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 half of the country with one of the main regions of elevated deposition being 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 do have elevated U.S. EGU mercury deposition, many of the highest areas (and largest expanses) of U.S. EGU deposition occur inland (e.g., Ohio River Valley, areas in northeast Texas and along the Mississippi River).

- *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)*: On average across the U.S., U.S. EGUs contribute 5% of total Hg deposition under the 2005 scenario with this level decreasing to 2% under the 2016 scenario (see Table 2-3). The remaining Hg deposition (i.e., ~95% and ~98%, respectively for the two scenarios) originates from other U.S. sources of mercury emissions and from foreign sources (both anthropogenic and natural). There is a considerably decrease in U.S. EGU Hg deposition between the 2005 and 2016 scenarios, with this resulting primarily from implementation of the Transport Rule, state mercury regulations and Federal enforcement actions.[34] The median reduction in U.S. EGU Hg deposition was 41% with reductions ranging up to 85% for the 95th% watershed (when ranked according to magnitude of reduction in U.S. EGU Hg deposition) (see Tables 2-2 and 2-3).

- *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*: In the 2005 scenario, while on average, U.S. EGUs only represented 5% of total Hg deposition in the U.S., values ranged up to 30% for the 99th% watershed (see Table 2-2). While overall U.S. EGU Hg deposition decreased substantially for the 2016 scenario, still, U.S. EGUs contributed 11% of total Hg deposition for the 99th% watershed (ranked according to U.S. EGU deposition) (see Table 2-2).

2.4. Fish Tissue MeHg Concentrations

[34] Controls on PM precursors, including directly emitted PM and SO_2, can have significant secondary reductions on divalent and particle-bound mercury, both of which produce much of the local and regional deposition.

This section characterizes the set of 2,461 watershed-level fish tissue MeHg samples used in the analysis. As noted earlier in Section 1.3, the 75th percentile of the samples falling within a given watershed is used as the basis for the risk estimates generated for that watershed. Consequently we have used the 75th percentile statistic (at the watershed level) as the basis for summarizing the fish tissue MeHg data presented in this section. Recall also, that as discussed in Section 1.3, the MMaps approach was used to estimate the U.S. EGU-attributable portion of each 75th percentile fish tissue value within each watershed (based on the fraction of total mercury deposition associated with U.S. EGUs for each watershed). Similarly, for the 2016 scenario, baseline fish tissue sampling data used in 2005 was first adjusted to reflect changes in total deposition (between the 2005 and 2016 Base Cases) for a given watershed (also using the MMaps approach), and then the U.S. EGU-attributable fraction was estimated, again based on the fraction of total mercury deposition over that watershed that is associated with U.S. EGUs.

The summary of fish tissue MeHg data is based around a series of figures and tables conveying relevant information. The figures and tables used in summarizing the fish tissue data are described below. Note, that, as discussed in Section 2.3, most of the areas experiencing elevated U.S. EGU-attributable mercury deposition are located in the eastern half of the country. We also have greater coverage with mercury fish tissue data in the eastern part of the country. In addition, because the Transport Rule primarily affects U.S. EGUs in the eastern half of the country, we mainly see reductions in U.S. EGU-attributable risk (in comparing the 2005 to 2016 scenarios) for the eastern portion of the country. For these reasons, in illustrating spatial trends/patterns in fish tissue data in this section through figures, we focus primarily on the eastern half of the country (note, however that data presented in tables are for the whole continental U.S.). After presenting these figures and tables, a set of bulleted observations is presented 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 2-5: Map of 2,461 watersheds with fish tissue sampling data used in the risk assessment. This map not only illustrates general coverage of the fish tissue data for different regions of the country, it also illustrates the relatively small size of the HUC12 watersheds used in the analysis.

- Figure 2-6: Map of the subset (approximately 2,170) of the 2,461 watersheds falling in the eastern half of the U.S.. This map uses color gradients to illustrate spatial variation in the <u>total</u> mercury fish tissue concentrations (for the 2005 Base Case) across the watersheds and as such, clearly illustrates how difficult it is to identify any discernable patterns with this approach given the small size of the watersheds. Because of this, we decided instead, to use graduated circles (with circle size tracking 75th percentile fish tissue concentrations at each watershed) in the remainder of maps presented in this section, since this approach allows spatial patterns to be more readily discerned.

- Figure 2-7 and 2-8: Maps presenting CMAQ modeling results for total mercury deposition (ug/m^2) at the watershed-level in the eastern U.S., for the 2005 and 2016 scenarios respectively.

- Figures 2-9 and 2-10: Maps presenting CMAQ modeling results for U.S. EGU-attributable mercury deposition (ug/m^2) at the watershed-level in the eastern U.S., again for 2005 and 2016 scenarios, respectively.

- Figures 2-11 and 2-12: Maps of the upper 10^{th} percentile of watersheds based on total mercury fish tissue levels for the 2005 and 2016 scenarios, respectively. These maps allow us to consider how spatial patterns (and overall magnitude) of watersheds with the highest fish tissue levels change between the 2005 and 2016 scenarios.

- Figures 2-13 and 2-14: Maps of the upper 10^{th} percentile of watersheds based on U.S. EGU-attributable mercury fish tissue levels for the 2005 and 2016 scenarios, respectively. These maps allow us to consider how spatial patterns (and overall magnitude) of watersheds with the highest U.S. EGU-attributable fish tissue levels change between the 2005 and 2016 Base Cases. Note, that these maps are particular relevant to consideration of changes in the patterns of U.S. EGU-attributable risk between the 2005 and 2016 Base Cases.

- Table 2-4: Summary of statistics (min, max, mean, 50^{th}, 75^{th}, 90^{th}, 95^{th} and 99^{th} 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.

- Table 2-5: Summary of statistics (min, max, mean, 50^{th}, 75^{th}, 90^{th}, 95^{th} and 99^{th} percentiles) for U.S. EGU-attributable fraction of total Hg fish tissue (these results are reflected directly in total and U.S. EGU-attributable risk calculations).

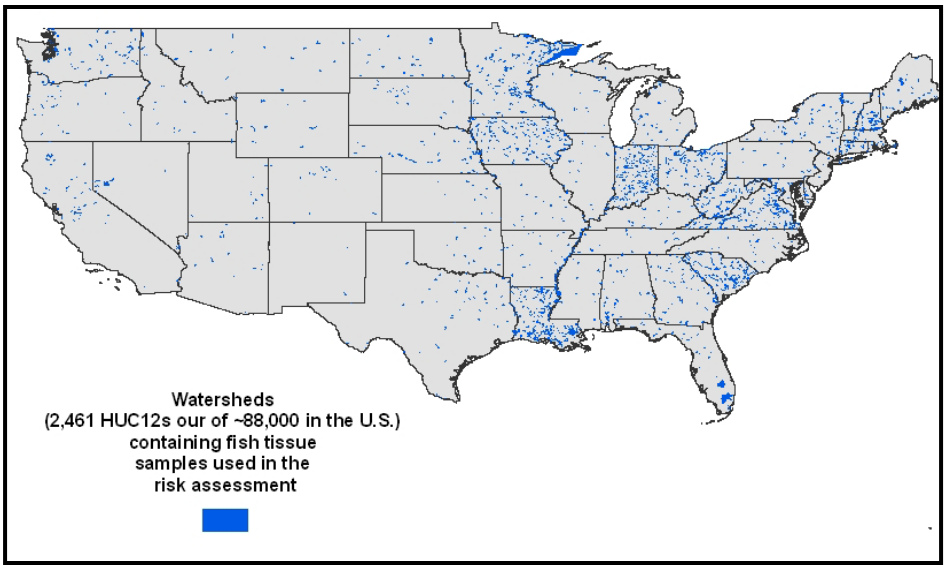


Figure 2-5. Location of 2,461 watersheds with mercury fish tissue data included in the risk assessment

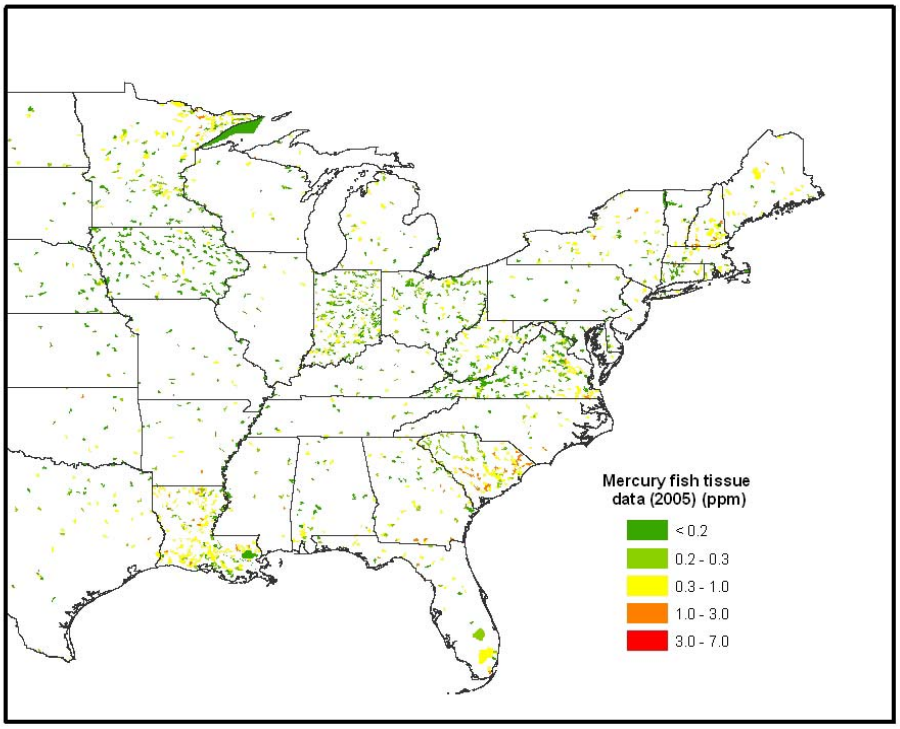


Figure 2-6. Subset of 2,170 watersheds (from the larger set of 2,461 included in the risk assessment) located in the eastern half of the country. (this maps also illustrates limitations with using color-coding at the watershed-level to explore trends in Hg fish tissue concentrations – see text)

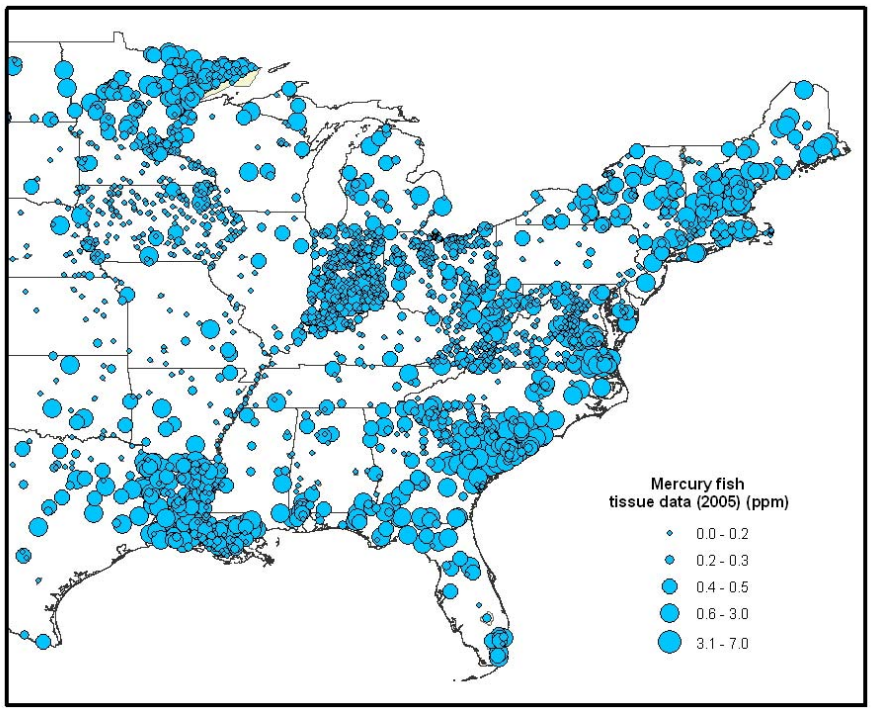


Figure 2-7. Total Hg fish tissue concentrations (for the 2005 Base Case) for the subset of watersheds included in the risk assessment located in the eastern U.S.

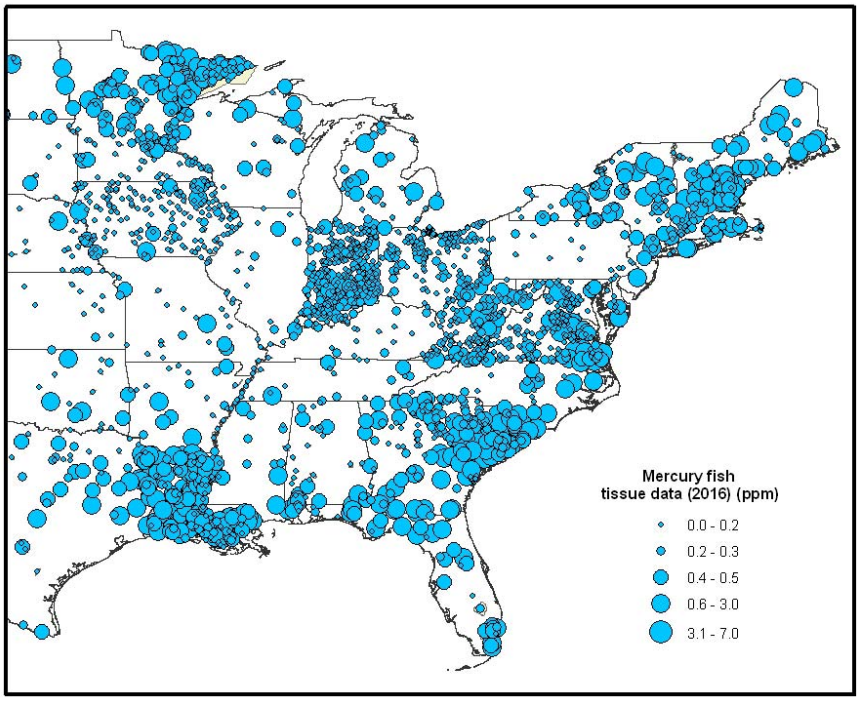


Figure 2-8. Total Hg fish tissue concentrations (for the 2016 Base Case) for the subset of watersheds included in the risk assessment located in the eastern U.S.

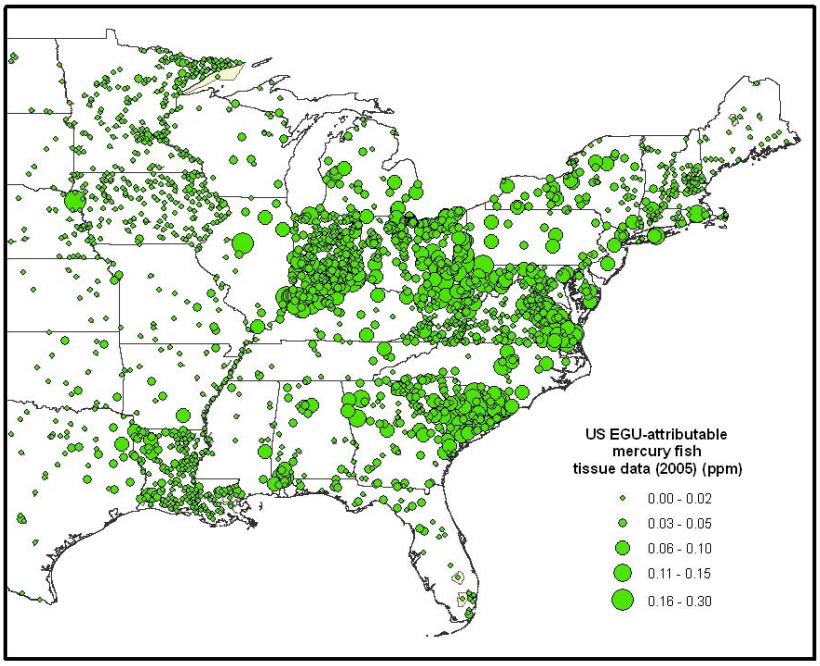


Figure 2-9. U.S. EGU-attributable Hg fish tissue concentrations (for the 2005 Base Case) for the subset of watersheds included in the risk assessment located in the eastern U.S.

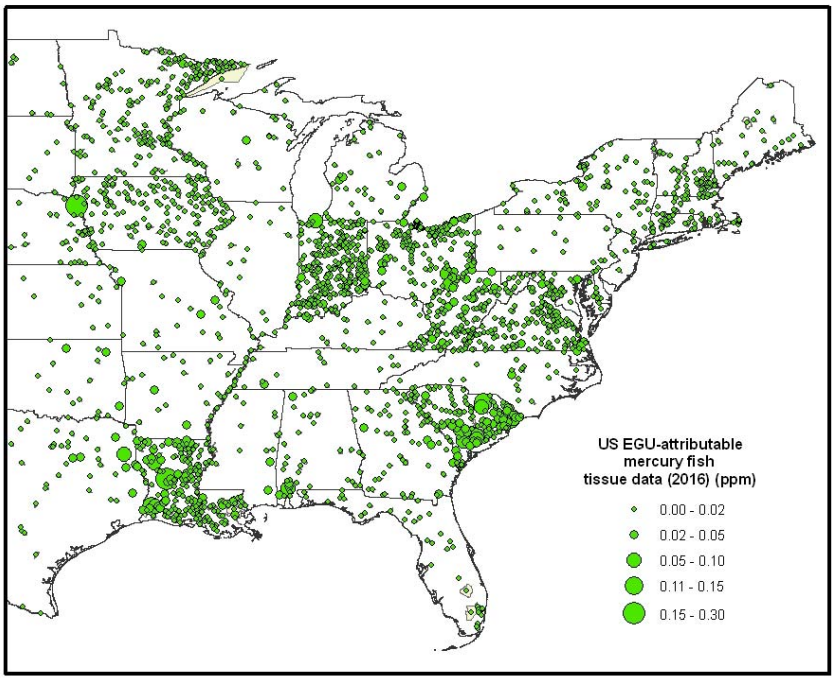


Figure 2-10. U.S. EGU-attributable Hg fish tissue concentrations (for the 2016 Base Case) for the subset of watersheds included in the risk assessment located in the eastern U.S. (Note, this map uses the same scale as Figure 2-7, thereby supporting direct comparison between these two time periods)

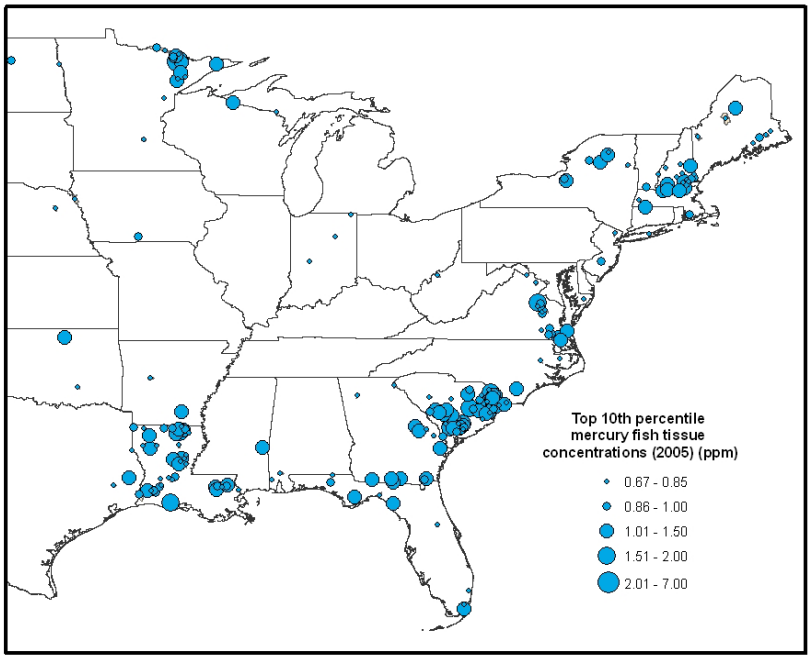


Figure 2-11. Top 10th percentile of watersheds based on total Hg fish tissue concentrations (for the 2005 simulation). (ranking is based on full national set of watersheds included in the risk assessment, but map focuses on locations in the eastern U.S.)

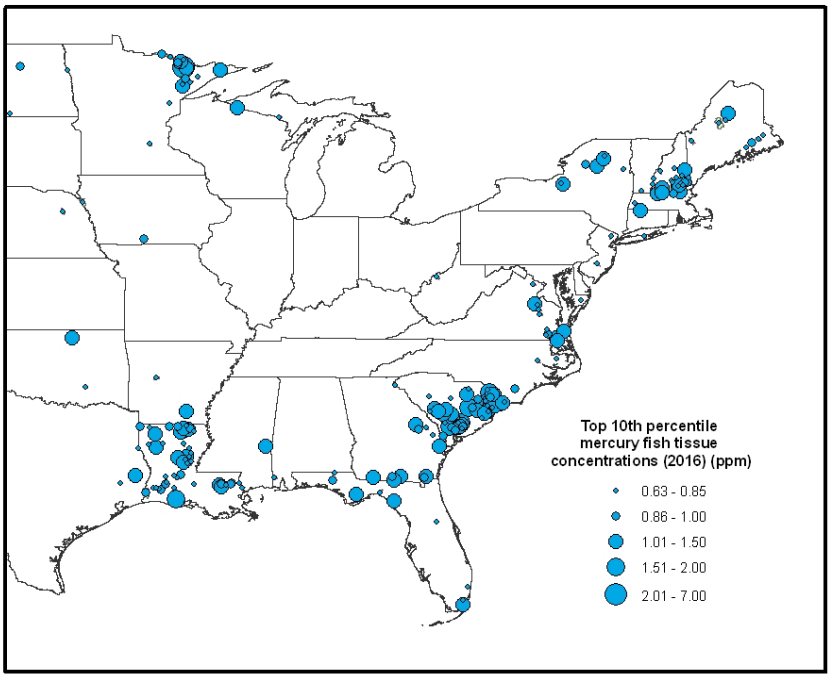


Figure 2-12. Top 10th percentile of watersheds based on total Hg fish tissue concentrations (for the 2016 simulation). (ranking is based on full national set of watersheds included in the risk assessment, but map focuses on locations in the eastern U.S.)

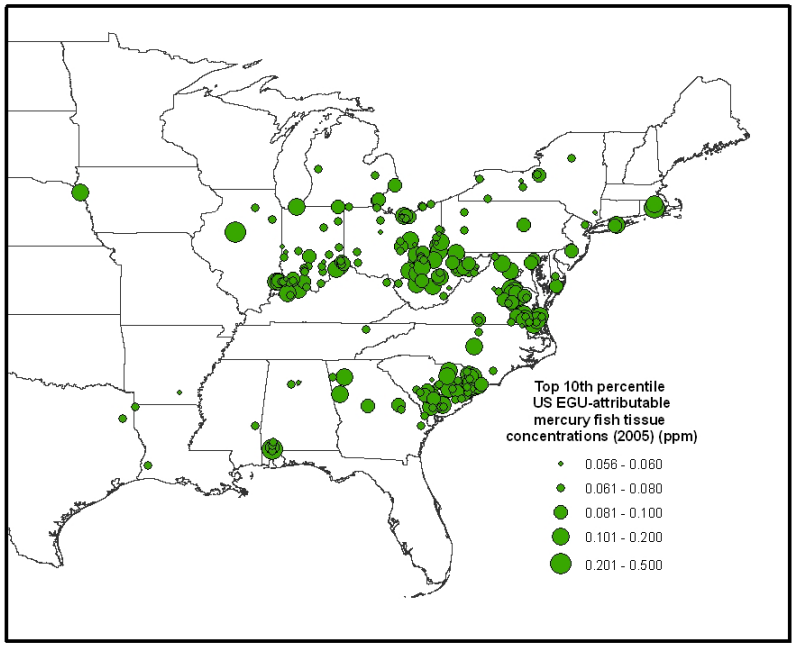


Figure 2-13. Top 10th percentile of watersheds based on U.S. EGU-attributable Hg fish tissue concentrations (for the 2005 simulation). (ranking is based on full national set of watersheds included in the risk assessment, but map focuses on locations in the eastern U.S.)

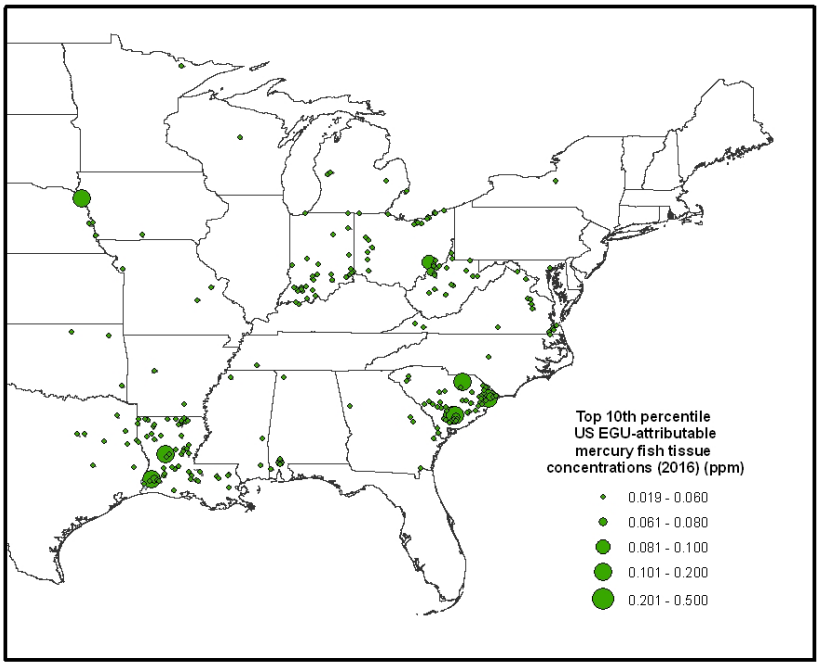


Figure 2-14. Top 10th percentile of watersheds based on U.S. EGU-attributable Hg fish tissue concentrations (for the 2016 simulation). (ranking is based on full national set of watersheds included in the risk assessment, but map focuses on locations in the eastern U.S.)

Table 2-4. Comparison of total and U.S. EGU-attributable Hg fish tissue concentrations (including % change) for the 2005 and 2016 scenarios.

Statistic	Hg fish tissue concentration (ppm)						% change (2016 versus 2005) in Hg fish tissue concentration	
	2005 scenario			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	0.31	0.024	7.7%	0.29	0.008	2.9%	-6.7%	-65%
Median	0.23	0.014	6.2%	0.20	0.005	2.7%	-10.0%	-61%
75th %	0.39	0.032	8.3%	0.36	0.011	2.9%	-6.4%	-67%
90th %	0.67	0.056	8.3%	0.63	0.019	3.0%	-5.9%	-66%
95th %	0.91	0.079	8.7%	0.87	0.026	3.0%	-4.7%	-67%
99th %	1.34	0.150	11.2%	1.29	0.047	3.7%	-3.7%	-68%

Table 2-5. Comparison of *U.S. EGU fraction of total Hg deposition* (used to apportion Hg fish tissue concentrations and risk) between the 2005 and 2016 scenarios. Note, that these values are specifically for the 2,461 watersheds included in the risk assessment.

statistic	U.S. EGU-attributable fraction of total Hg fish tissue levels	
	2005 scenario	2016 scenario
Mean	0.09	0.04
Median	0.06	0.03
75th %	0.14	0.05
90th %	0.20	0.07
95th %	0.26	0.09
99th %	0.40	0.18

Consideration of information presented above in Figures 2-5 through 2-14 and in Tables 2-4 through 2-5 resulted in the following observations regarding estimates of total and U.S. EGU-attributable fish tissue MeHg concentrations across the 2,461 watersheds included in the risk assessment:

- *Focus on U.S. EGU-attributable Hg fish tissue levels is in the eastern half of the U.S.:* Given (a) that the number of watersheds with measured fish tissue MeHg data is substantially greater in the east (see Figure 2-5) and (b) more importantly, that the 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), trends in U.S. EGU-attributable Hg fish tissue levels discussed here are driven by data 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*. 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.[35] 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 MeHg levels***:** 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 ~ 4% 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 2-9 and 2-10 for U.S. EGU-attributable Hg fish tissue levels with the relatively smaller changes seen in Figures 2-7 and 2-8 for total Hg fish tissue 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*: Under the 2005 scenario, U.S. EGUs can range up to 40% of total Hg fish tissue levels (for the 99th% watershed). Under the 2016 Scenario, this pattern is reduced, but U.S. EGUs can still contribute up to 18% of total Hg fis tissue levels (again, for the 99th% watershed) (see Table 2-5).

2.5. Comparing Patterns of Hg Deposition with Hg Fish Tissue Data for the 2,461 Watersheds Included in the Risk Assessment

In addition to the observations provided in the last two sections based on consideration of the CMAQ-based Hg deposition estimates and Hg fish tissue data separately, it is also possible to directly compare spatial patterns between these two sets of data. This comparison provides information that can help in interpreting risk estimates discussed in Section 2.6. Specifically we can consider: (a) whether the watershed-level Hg fish tissue levels are positively correlated with total Hg deposition, (b) how patterns of Hg deposition for the 2,461 watersheds where we have Hg fish tissue data compare with patterns for the full set of 88,000 watersheds in the U.S. and (c) to what extent the watersheds for which we have Hg sampling data provide coverage for areas of elevated U.S. EGU deposition. To address these questions, we have presented a series of figures and tables below including:

[35] As discussed in section 1.3, U.S. EGU-attributable Hg fish tissue levels are directly based on U.S. EGU Hg deposition (at the watershed-level) together with application of the MMaps approach.

- Figures 2-15 and 2-16: For the 2005 and 2016 scenarios respectively, maps showing areas of elevated U.S. EGU-related Hg deposition[36] and the degree to which the 2,461 watersheds with fish tissue sampling data used in the risk assessment provide coverage for these areas.

- Figure 2-17: Provides plot for the 2005 Scenario of Hg fish tissue levels versus total Hg deposition 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: Presents cumulative distribution plots comparing U.S. EGU-attributable deposition for the 2,461 watersheds used in the risk assessment with U.S. EGU-attributable deposition of the entire set of ~88,000 watersheds in the U.S. Separate sets of plots are provided for the 2005 and 2016 Scenarios, allowing trends for each scenario to be compared to each other. These plots allow us to consider whether the watersheds with fish tissue MeHg 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 mercury deposition across the country.

[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.

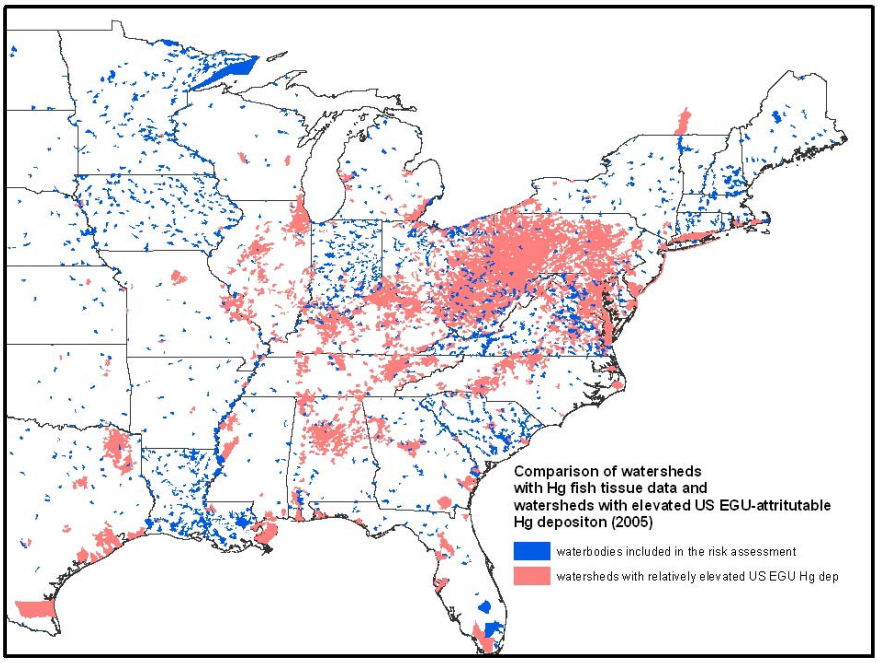


Figure 2-15. For the 2005 scenario, comparison of coverage of watersheds with Hg fish tissue data (used in the risk assessment) for areas in the eastern U.S. with relatively elevated U.S. EGU-attributable Hg deposition.

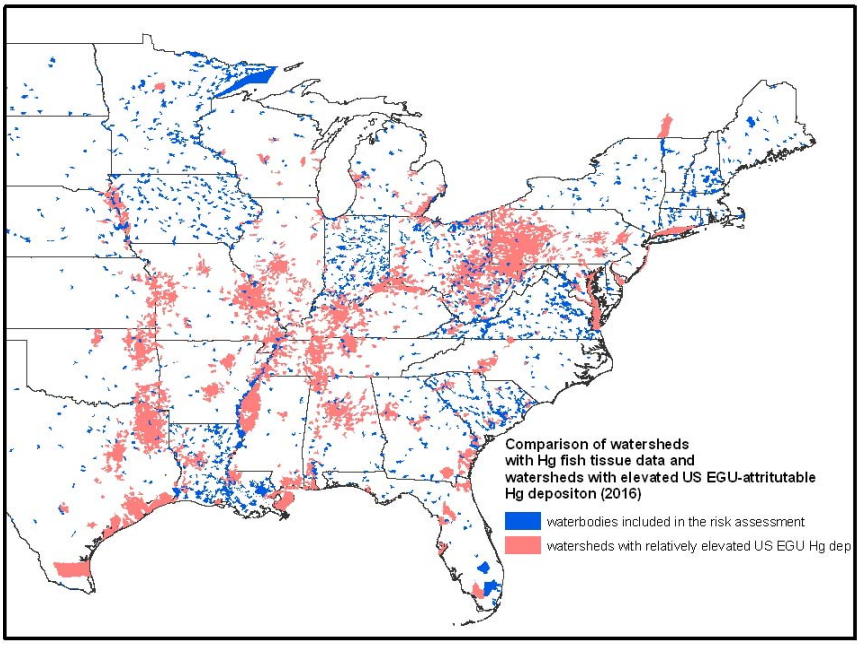


Figure 2-16. For the 2016 Scenario, comparison of coverage of watersheds with Hg fish tissue data (used in the risk assessment) for areas in the eastern U.S. with relatively elevated U.S. EGU-attributable Hg deposition.

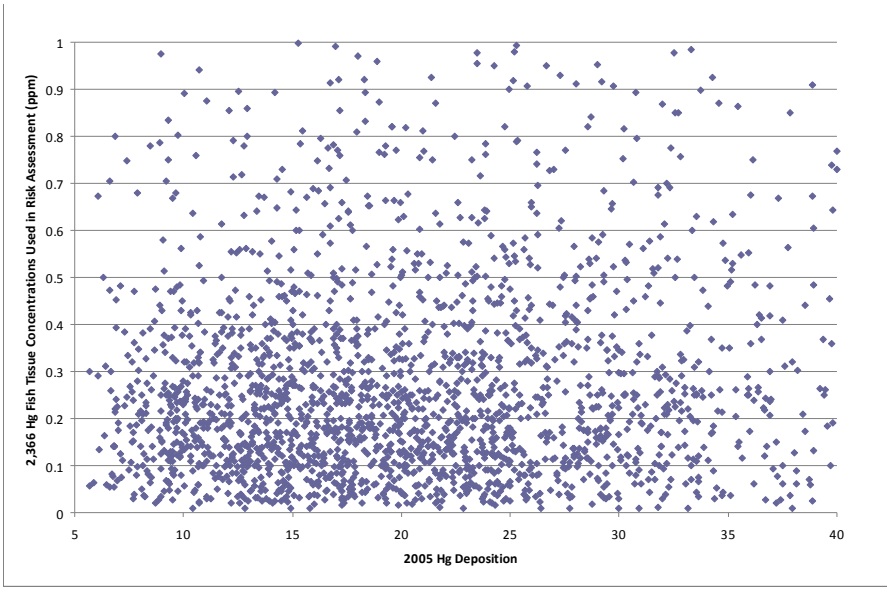


Figure 2-17. For the 2005 scenario, plot of total Hg fish tissue concentrations versus total Hg deposition for the 2,366 watersheds included in the risk assessment for the high-end female consumer population.

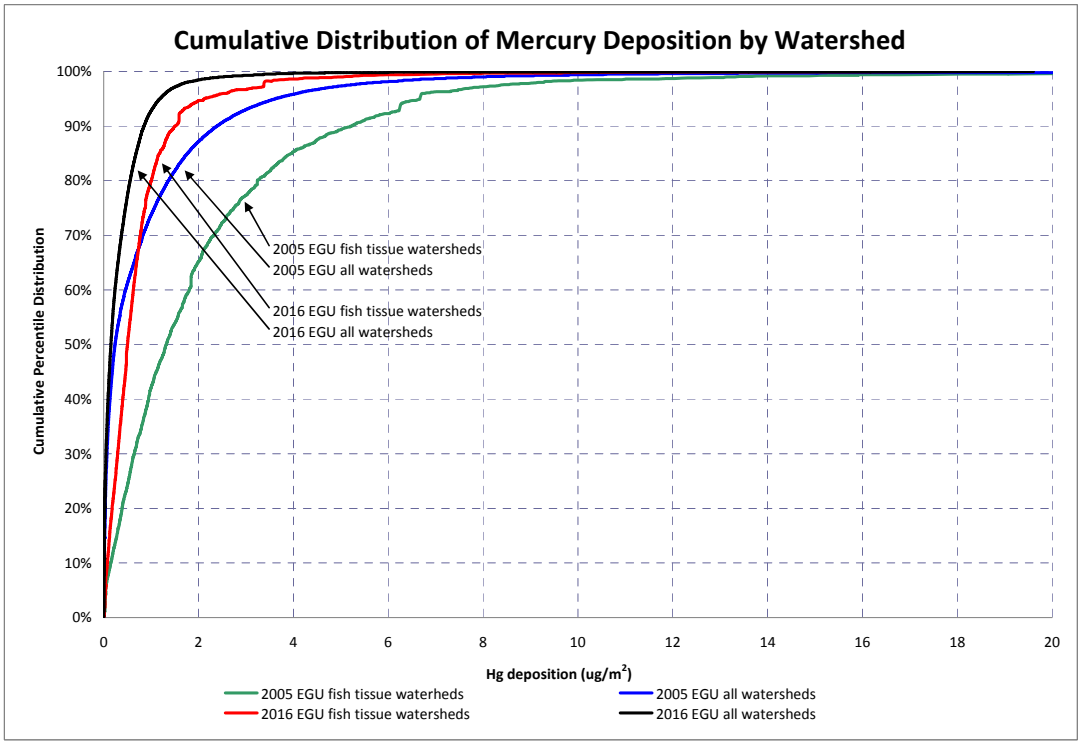


Figure 2-18. Cumulative distribution plots of U.S. EGU-attributable Hg deposition over the 2,366 watersheds used in modeling the high-end female consumer population as contrasted with all 88,000 watersheds (plots provided both for the 2005 and 2016 Scenarios).

Consideration of information presented above in Figures 2-15 through 2-18 resulted in the following observations regarding how estimates of Hg deposition estimates relate to measured fish tissue MeHg levels, when considered at the watershed-level:

- *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:* As depicted in Figures 2-15 and 2-16 (for the 2005 and 2016 Scenarios, respectively), while the 2,461 watersheds used in the risk assessment to fall into regions in the east with elevated U.S. EGU Hg deposition, the degree of coverage is limited. This can be seen by noting in these figures the wide expanses of areas of elevated U.S. EGU Hg deposition (shown in red) and therefore, not covered by watersheds modeled for risk in the analysis.[37]

- *Hg fish tissue levels are not correlated with total Hg deposition (the relationship is highly dependent on methylation potential of individual waterbodies)*: As shown in Figure 2-17, total Hg fish tissue levels (summarized at the watershed-level) are not correlated with levels of total Hg deposition when looking across watersheds (i.e., the highest total mercury deposition watersheds does not always have the highest fish tissue MeHg levels). This is not unexpected given that the relationship between total Hg deposition and total Hg fish tissue levels is highly dependent on the methylation potential at the waterbody-level. As discussed above in Appendix E, a variety of factors that display spatial variability are associated with methylation potential (e.g., pH, sulfate deposition, turbidity etc). Therefore, we would anticipate that there would not be a direct correlation between total Hg deposition and Hg fish tissue levels, again looking across watersheds. The MMaps approach and underlying analyses (see Section 1.3 and Appendix E), support a proportional relationship between mercury deposition and fish tissue MeHg levels within a given watershed, such that changes in deposition will be reflected in changes in fish tissue levels. In other words, the correlation between Hg deposition and fish tissue MeHg concentrations does not appear to hold between watersheds, but is espected tos hold within a given watershed.

- *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

[37] We completed a follow-on assessment to help interpret the significance of the group of watersheds with "elevated U.S. EGU deposition" that were not covered in the risk assessment (i.e., areas shown in red in Figures 2-15 and 2-16). Specifically, we were interested in knowing, for the subset of 2,366 watersheds we did assess for risk for the female high-end consumer, what is the percentage that (a) had Hg deposition above the threshold identified here for "elevated deposition" (i.e., 3.79 and 1.28 ug/m2, respectively for the 2005 and 2016 Scenarios) and (b) had U.S. EGU incremental risk of an HQ =1.5. This percentage lets us know, for the watersheds we modeled, what fraction of watersheds with elevated U.S. EGU Hg deposition also had U.S. EGU-incremental risk representing a potential public health hazard. The results of the assessment are 37% and 9%, respectively for the 2005 and 2016 Scenarios. We can consider use these estimates to help interpret the areas in Figures 2-15 and 2-16 that have elevated U.S. EGU Hg deposition and are NOT included in the risk assessment. If these watersheds tracked the pattern seen in the watersheds we modeled, then we would expect to see ~40% of the red highlighted areas in Figure 2-15 translate into relatively elevated U.S. EGU incremental risk watersheds and ~10% of the red highlighted watersheds in Figure 2-16. Note, however, that there is substantial uncertainty in extrapolating trends seen across our modeled watersheds to the non-modeled watersheds with elevated U.S. EGU Hg deposition..

favored areas with relatively higher total Hg deposition. This can be seen by comparing cumulative plots of modeled watersheds (where we have fish tissue MeHg data) against plots for the entire set of 88,000 watersheds. This comparison suggests that watersheds where fish tissue MeHg data were collected tended to have higher total Hg deposition, then the full set of watersheds. This likely reflects to some extent, the fact that fish tissue sample are focused in the eastern half of the country, which does have elevated total Hg deposition compared to the broad central region (see Figure 2-1 and 2-2).

2.6. Overview of Risk Estimates

This section provides an overview of risk estimates generated for the 2,461 watersheds included in the risk assessment. As noted earlier in Section 1.2, presentation of risk estimates will focus on the high-end female consumer population assessed at the national-level, since this population 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 population represent subsistence levels and are supported by a number of studies (see Section 1.3 and Appendix D). While this fisher population is emphasized in summarizing risk estimates, we do provide risk estimates generated for the other populations covered in the analysis (e.g., blacks in the southeast, Tribal populations near the Great Lakes, Hispanics). In summarizing risk estimates, we will provide estimates for both the 2005 and 2016 Scenarios, while placing the most emphasis on the 2016 estimates. The remainder of this section is organized as follows:

- Overview of percentile risk estimates generated for the different fisher populations evaluated (Section 2.6.1): In this section, we provide percentile risk estimates (for HQ and IQ loss risk) for the high-end female consumer population assessed at the national level. We then summarize percentile risk estimates (HQ only) for the broader set of fisher populations assessed in the analysis. The percentile risk estimates provided in this section allow us to (a) consider Stage 2 of the 3-Stage framework developed to support the interpretation of risk estimates (i.e., consider the U.S. EGU-related increment of total risk – see Section 1.2) and (b) consider the magnitude of risk across the set of fisher populations assessed in the risk assessment.

- Overview of the number (and frequency) of watersheds with populations potentially at risk due to U.S. EGU-sourced mercury (Section 2.6.2): This set of risk estimates provides the main input to the risk characterization framework (see Section 1.2). Specifically, watersheds with populations potentially at risk comprise:

    - Watersheds where total risk is considered to represent a public health concern and where U.S. EGUs contribute to that total risk (in the analysis, we considered various increments of U.S. EGU contribution including 5%, 10%, 15% and 20%, although as noted in Section 1.2, we focus on 5%). This represents Stage 1 of the rick characterization framework AND/OR,

    - Watersheds where risk when considering U.S. EGUs mercury emissions before considering other sources of mercury represents a potential public health hazard (this is Stage 2 in the risk characterization framework)

To support the discussion of risk estimates, a series of tables summarizing those estimates are presented in the subsections below. A list of observations based on consideration for the risk estimates summarized is presented at the end of each subsection.

2.6.1. Overview of percentile risk estimates

In presenting percentile risk estimates in this section, we have sorted risk estimates by U.S. EGU-attributable risk in order to track trends in the magnitude of this category of risk.[38] The percentile estimates themselves, are based on risk bands around the percentile value (rather than being based on the specific watershed at that percentile). Specifically, we have taken the average of the 5% of watershed values surrounding the specific percentile estimate in the table. So, for example, for a 50th% risk value presented in the table, we have actually taken the average of the 47.5th through 52.5th percentile watershed-level risk estimates (after ranking them by U.S. EGU-attributable risk, as mentioned earlier). We used this risk band approach, rather than using the estimates from the singe watershed located specifically at that percentile, because we wanted to capture general trends in the patterns of *U.S. EGU-attributable* and *total risk* for watersheds around that percentile.[39] Note also, that while this risk assessment does focus on subsistence levels of fish consumption, the risk tables summarized here do include risk estimates based on mean fish consumption rates for these higher consuming populations, which are relatively high compared to general recreational angler rates. The following tables are used to summarize risk estimates for the fisher populations included in the analysis.

- Tables 2-6 and 2-7: Presents risk percentiles for both IQ loss and RfD-based HQs for the high-end female consumer assessed at the national-level (for 2,366 watersheds) for the 2005 and 2016 Scenarios, respectively. We do note that overall confidence in IQ loss estimates above approximately 7 points decreases because we begin to apply the underlying IQ loss function at exposure levels (ppm hair levels) above those reflected in epidemiological studies used to derive those functions.[40] We have flagged IQ loss estimates above 7 as subject to greater uncertainty.

- Table 2-8: Provides risk percentiles for RfD-based HQs for the remaining fisher populations assessed in this analysis specifically for the 2005 scenario. We did not evaluate these populations for the 2016 Scenario, since the relative magnitude of risks for these additional populations can be inferred by comparing risks for these populations against the risk generated for the high-end female consumer population (for the 2005

[38] This means that when reviewing the risk estimates presented in tables in this section, the trend in risk across watershed percentiles (i.e., higher percentiles will have higher risk) will be seen for the U.S. EGU-attributable portion of the risk and not necessarily for total risk, since the watersheds were ranked on U.S. EGU-attributable risk and on total risk, prior to generating the percentile summaries provided in the tables.

[39] Note, that watersheds can display considerable variation in the relationship between total and U.S. EGU-attributable risk. Therefore, if we had selected the specific watershed associated with a given percentile, that watershed could misrepresent the general trend (in terms of the relationship between total and U.S. EGU-attributable risk) for watersheds in the vicinity of that percentile. By taking average values for these "risk bands" around each percentile, we get more stable and meaningful results in terms of capturing trends in risk estimates across the percentiles presented in the risk tables.

[40] The 39.1 ppm was the highest measured ppm level in the Faroes Island study, while ~86 was the highest value in the New Zealand study (USEPA, 2005) (a 7 IQ points loss is approximately associated with a 40 ppm hair level given the concentration-response function we are using).

scenario) as summarized in Table 2-8. Similarly, we did not calculate IQ loss estimates, since these are linearly related to HQ loss and can be inferred from the estimates presented in Table 2-8 (based on comparison of magnitude of HQ estimates between different fisher populations).

Table 2-6. Percentile risk estimates for the high-end female consumer population assessed nationally (2005 scenario) (for both total and U.S. EGU incremental risk, including IQ loss and MeHg RfD-based HQ estimates)

Fisher consumption rate percentile and rate (g/day)	Watershed percentile									
	50th percentile		75th percentile		90th percentile		95th percentile		99th percentile	
	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU
IQ loss (points)										
mean (39)	0.8	-	0.7	-	1.0	0.1	1.2	0.2	1.6	0.3
90th (123)	2.5	0.2	2.3	0.3	3.0	0.4	3.9	0.5	5.1	1.0
95th (173)	3.5	0.3	3.2	0.4	4.2	0.5	5.4	0.7	7.2*	1.4
99th (373)	7.6	0.7	6.9	0.9	9.1*	1.1	11.7*	1.6	15.5*	2.9
RfD-based HQ										
mean (39)	3.5	0.3	3.2	0.4	4.2	0.5	5.4	0.7	7.2	1.4
90th (123)	11.1	1.1	10.1	1.3	13.3	1.6	17.1	2.3	22.8	4.3
95th (173)	15.6	1.5	14.2	1.8	18.7	2.3	24.1	3.2	32.0	6.0
99th (373)	33.6	3.3	30.7	3.9	40.3	4.9	51.9	6.9	69.0	13.0

- 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 text)

Table 2-7. Percentile risk estimates for the high-end female consumer population assessed nationally (2016 Scenario) (for both total and U.S. EGU incremental risk, including IQ loss and RfD-based HQ estimates)

Fisher consumption rate percentile and rate (g/day)	Watershed percentile									
	50th percentile		75th percentile		90th percentile		95th percentile		99th percentile	
	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU
IQ loss (points)										
mean (39)	0.6	-	0.6	-	1.0	-	1.3	-	1.8	0.1
90th (123)	1.7	-	2.0	-	3.2	0.1	4.0	0.2	5.6	0.4
95th (173)	2.4	-	2.8	-	4.5	0.2	5.6	0.2	7.9*	0.5
99th (373)	5.3	0.1	6.0	0.2	9.8*	0.4	12.1*	0.5	16.9*	1.1
RfD-based HQ										
mean (39)	2.5	-	2.8	-	4.6	0.2	5.6	0.2	7.9	0.5
90th (123)	7.7	0.2	8.8	0.3	14.4	0.5	17.7	0.8	24.8	1.6

95th (173)	10.9	0.2	12.3	0.4	20.2	0.8	24.9	1.1	34.9	2.2
99th (373)	23.4	0.5	26.6	0.9	43.6	1.7	53.6	2.3	75.3	4.8

- 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 text)

Table 2-8. Percentile risk estimates for the full set of fishing populations included in the analysis (2005 scenario) (for both total and U.S. EGU incremental risk, only for RfD-based HQ estimates)

Fisher consumption rate percentile and rate (g/day)	Watershed percentile									
	50th percentile		75th percentile		90th percentile		95th percentile		99th percentile	
	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU
High-end female consumer assessed nationally										
mean (39)	3.5	0.3	3.2	0.4	4.2	0.5	5.4	0.7	7.2	1.4
90th (123)	11.1	1.1	10.1	1.3	13.3	1.6	17.1	2.3	22.8	4.3
95th (173)	15.6	1.5	14.2	1.8	18.7	2.3	24.1	3.2	32.0	6.0
99th (373)	33.6	3.3	30.7	3.9	40.3	4.9	51.9	6.9	69.0	13.0
Poor white fishers in the Southeast										
mean (39)	5.0	0.5	4.9	0.6	7.3	0.7	7.3	1.0	8.8	1.6
90th (93)	12.0	1.1	11.6	1.4	17.6	1.7	17.5	2.5	21.1	3.9
95th (129)	16.6	1.6	16.1	1.9	24.4	2.4	24.3	3.4	29.2	5.5
99th (286)	36.9	3.5	35.8	4.2	54.1	5.3	53.9	7.5	64.7	12.1
Poor black fishers in the Southeast										
mean (171)	19.0	1.9	24.7	2.3	26.2	2.9	39.6	4.2	40.0	7.2
90th (446)	49.6	4.9	64.4	5.9	68.2	7.6	103.3	10.9	104.3	18.8
95th (557)	61.9	6.1	80.5	7.4	85.2	9.5	129.0	13.6	130.3	23.4
99th	NC*									
Poor Hispanic nationally										
mean (26)	1.5	-	2.3	0.2	3.0	0.3	3.6	0.5	6.2	0.9
90th (98)	5.5	0.3	8.6	0.7	11.6	1.2	13.6	1.8	23.4	3.5
95th (156)	8.8	0.5	13.6	1.1	18.4	2.0	21.7	2.8	37.2	5.5
99th	NC*									
Vietnamese										
mean (27)	1.4	-	3.0	0.2	1.7	0.3	2.8	0.4	4.6	0.9
90th (99)	5.2	0.3	10.9	0.6	6.2	1.0	10.4	1.4	16.8	3.1
95th (152)	8.0	0.5	16.8	1.0	9.5	1.6	15.9	2.2	25.8	4.8
99th	NC*									
Laotians										
mean (47)	2.2	0.1	3.3	0.3	2.0	0.4	3.9	0.5	6.5	1.5
90th (145)	6.6	0.4	10.2	0.9	6.0	1.3	12.0	1.5	20.0	4.6
95th (266)	12.2	0.7	18.8	1.6	11.1	2.4	22.0	2.7	36.7	8.5
99th	NC*									
Tribal (near Great Lakes)										
mean (62)	4.1	0.1	7.4	0.2	9.4	0.4	8.7	0.6	6.9	0.9
90th (136)	8.9	0.2	16.2	0.4	20.6	0.9	19.1	1.3	15.0	2.0

Fisher consumption rate percentile and rate (g/day)	Watershed percentile									
	50th percentile		75th percentile		90th percentile		95th percentile		99th percentile	
	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU
95th (213)	13.9	0.4	25.4	0.6	32.2	1.4	29.9	2.0	23.5	3.2
99th (493)	32.2	0.9	58.6	1.4	74.5	3.2	69.2	4.5	54.4	7.4

- IQ loss or HQ estimates <0.1.

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

Observations regarding the percentile risk estimates presented here reflect our interpretation of the potential health significance associated with both MeHg RfD-based HQ and IQ loss estimates (see Section 1.2). Consideration of the risk estimates summarized above in Tables 2-6 through 2-8 results in the following observations regarding percentile risk estimates generated for the fisher populations assessed:

- *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:* While there are some differences in total IQ loss and HQ estimates generated for the high-end female consumer assessed nationally between the 2005 and 2016 Scenarios, there is no systematic trend between the scenarios (see Tables 2-6 and 2-7). Furthermore, for the high-end female consumer assessed at the national-level, these estimates of total risk often exceed one IQ point loss and an HQ of 1.5 (i.e., levels of potential health concern) across most of the combinations of consumption rates and watersheds (see Tables 2-6 and 2-7). The absence of a substantial change in total risk between the two simulation years is not surprising given the relatively small fraction of total mercury deposition contributed by U.S. EGUs on average across the modeled watersheds. This means that even substantial reductions in U.S. EGU deposition between the simulation years is unlikely to substantially affect total risk (although, as noted elsewhere, it can have a substantial impact on risk at the subset of watersheds where U.S. EGUs do contribute a larger fraction of total deposition).

- *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:* Comparison of the U.S. EGU-attributable risk estimates presented in Tables 2-6 and 2-7, suggest that these categories of risk decrease significantly between the 2005 and 2016 Scenarios. As noted earlier, this reduction largely reflects the implementation of PM controls which have the co-benefit of reducing divalent and particle-bound mercury together with state regulations targeting mercury emissions directly. As noted in Section 2.2, because current (2010) emissions of mercury are likely closer to levels used in modeling the 2016 Scenario (with the 2005 scenario reflecting emission levels that are substantially larger than current conditions), we focus here on presenting observations based on the 2016

Scenario. For the high-end female consumer assessed at the national-level, under the 2016 Scenario (see Table 2-7), U.S. EGU-attributable IQ loss only meets or exceeds one point for the highest consumption range at the upper 1% of the watersheds. And, given the relatively high total exposure associated with this simulation, our calculation of IQ (including the U.S. EGU-attributable portion) for this combination of consumption rate and watershed percentile is subject to increased uncertainty.[41] By contrast, for the 2016 Scenario, estimates of U.S. EGU increment-based HQ for this fisher population exceed 1.5, although these exceedences are still limited to combinations of higher percentiles of watersheds and consumption rates (e.g., 95th and 99th % consumption rates paired with the 95th and 99th % watersheds – see Table 2-7). These HQ-based risk estimates can be used to inform Stage 2 of the 3-Stage risk characterization framework. Specifically, for the 2016 Scenario, a fraction of watersheds (top 5 to 10%) have U.S. EGU increment-based HQ's that exceed 1.5 based on modeling subsistence-level fish consumption for the high-end female consumer assessed at the national-level (see Table 2-7).

- *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:* Risk estimates generated for the 2005 scenario for the set of high-end fisher populations assessed in this analysis suggest that risks (across all combinations of consumption rates and watersheds) are generally higher for the high-end female consumer population assessed at the national-level with the exception of black and white fishers assessed in the southeast (contrast estimates presented in Table 2-8). For example, high-end female consumer risk estimates assessed at the national-level are approximately twice as high (in terms of both total and EGU-attributable) as estimates generated for Hispanics and Vietnamese also assessed nationally (see Table 2-8). Risk estimates for the high-end female consumer are approximately 50% higher than estimates generated for Laotians assessed nationally and Tribal populations in the vicinity of the Great Lakes. However, risks (both total and U.S. EGU-attributable) for white fishers in the southeast are somewhat higher that risk estimates generated for the high-end female consumer at the national-level, while estimates for black populations assessed in the southeast are notably higher. Risk estimates for these two southeastern fisher populations are likely higher due to: (a) the fact that the southeast has relatively higher total and U.S. EGU-attributable fish tissue concentrations compared with the full set of watersheds with fish tissue levels nationally (see Figures 2-1 through 2-4) (this means that upper-end percentiles fish tissue values will be higher in the southeast) and (b) in the case of the black fisher populations, percentile consumption rates are substantially higher than consumption rates for the high-end female consumer population (assessed at the national-level) see Table 2-8, "Fisher consumption rate percentile rate (g/day)" column. While risk estimates are higher for the two populations assessed in the southeast, we decided to focus the discussion of risk estimates in both Section 1.6.1 and 1.6.2, on the high-end female consumer assessed at the national-level. As noted earlier, this reflects several

[41] As noted in section 2.6.1, estimates of total IQ loss above 7 points, involve simulation of mercury hair levels that exceed those used in the epidemiological studies underlying the function used in modeling of IQ loss and therefore are subject to greater uncertainty (not in the potential for IQ loss to occur, but rather in our ability to quantify degrees of loss above 7 points).

factors: (a) focusing risk estimates on national-level analysis allows us to consider a larger number of watersheds reflecting greater regional variability in factors related to fish consumption exposure and risk (e.g., methylation potential, fish species), (b) risk estimates in the southeast are driven in part by watersheds in SC and we have concerns over the potential for these estimates having non-air Hg contributions (from gold mining – see Appendix E), and (c) fish consumption rates for both of the southeastern-focused populations (white and black) are based on smaller sample sizes compared with the estimate for the high-end female consumers and therefore, we have greater confidence in the consumption estimates generated for the high-end female consumer population.[42]

2.6.2. Overview of number (and frequency) of watersheds with populations potentially at-risk due to U.S. EGU mercury emissions

This section discusses risk estimates based on identifying the number of watersheds with populations potentially at risk due to mercury released from U.S. EGUs. As noted in Section 1.2, 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 when deposition from U.S. EGUs is considered before taking into account deposition and exposures resulting from other sources of mercury. The estimates of watersheds with at-risk populations discussed in this section are used in the 3-Stage risk characterization framework described in Section 1.2 for interpreting risk estimates. Specifically, the first category of at risk populations described above maps to Stage 1 of the 3-Stage approach, while the second category maps to Stage 2. The combination (i.e., mathematical union) of these two groups of watersheds with at risk populations comprises the set of watersheds represented in Stage 3 of the framework.

We note that, specifically with regard to the HQ estimates, any contribution of mercury from EGUs to watersheds with exposures exceeding the MeHg RfD represents a potential 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. We think this is a conservative approach given the increasing risks associated with incremental exposures above the MeHg RfD.

The estimates of watersheds with potentially at-risk populations discussed in this section are all based on the underlying risk estimates generated for the high-end female consumer population. The decision to focus on this fisher population in discussing risk estimates is discussed in the previous section. The estimates of watersheds with potentially at-risk populations are summarized in tables described below (note, observations based on consideration of these risk estimates are presented in bullets following the tables).

- Tables 2-9: Identifies watersheds with potentially at-risk populations based on consideration for different degrees of U.S. EGU contribution (i.e., 5, 10, 15 and 20%) at watersheds where total risk is considered to represent a potential public health hazard (i.e., meet or exceed 1 IQ point or an HQ of 1.5 or higher). For reference purposes, the table also identifies the total number of watersheds (out of the 2,366 assessed for the

[42] Sample size used in computing consumption rate percentiles for the female population is 149, as contrasted with 39 and 98 for poor blacks and whites, respectively (Burger et al., 2010).

high-end female consumer population) with total risk exceeding the two thresholds, regardless of the U.S. EGU percent contribution (see the "≥ 0%" row of results in the table). In presenting results, the tables include both the number of watersheds meeting specific criteria as well as the percent (of the 2,366 watersheds assessed) that this represents. (Stage 1 of the 3-Stage framework)

- Tables 2-10: Identifies watersheds with potentially at-risk populations based on consideration for the magnitude of risk when deposition from U.S. EGUs is considered before taking into account deposition and exposures resulting from other sources of mercury. Risks are presented both for U.S. EGU-attributable IQ loss and for U.S. EGU increment-based HQ. (Stage 2 of the 3-Stage framework)

- Table 2-11: 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 2-9 and 2-10). As noted earlier, this analysis focuses on MeHg RfD-based HQ estimates rather than IQ loss estimates, since the HQ-based risk estimates generate a larger percentage of watersheds with populations potentially at-risk compared with the IQ loss estimates. Consequently, Table 2-11 considers the number and percent of watersheds that have (a) U.S. EGUs contributing to total risk of an HQ ≥ 1.5, OR (b) an HQ ≥ 1.5 based on considering U.S. EGU mercury deposition alone, before factoring in other sources of mercury deposition. This represents Stage 3 of the risk characterization framework.

Table 2-9. Watersheds with potentially at-risk populations based on consideration for various degrees of U.S. EGU contribution to total risk (for IQ loss and HQ)

EGU risk threshold	Number and percentage of HUCs meeting risk threshold criteria											
	2005 analysis						2016 analysis					
	90th fish consumption		95th% fish consumption		99th% fish consumption		90th fish consumption		95th% fish consumption		99th% fish consumption	
Total IQ points lost = 1												
≥ 0%	1667	(70%)	1948	(82%)	2224	(94%)	1558	(66%)	1858	(79%)	2202	(93%)
≥ 5%	948	(40%)	1124	(48%)	1287	(54%)	288	(12%)	394	(17%)	495	(21%)
≥ 10%	609	(26%)	726	(31%)	829	(35%)	43	(2%)	69	(3%)	94	(4%)
≥ 15%	345	(15%)	420	(18%)	473	(20%)	15	*	27	*	37	(2%)
≥ 20%	167	(7%)	202	(9%)	225	(10%)	7	*	16	*	20	*
Total RfD-based HQ = 1.5*												
≥ 0%	2191	(93%)	2266	(96%)	2348	(99%)	2162	(91%)	2248	(95%)	2346	(99%)
≥ 5%	1268	(54%)	1305	(55%)	1345	(57%)	482	(20%)	505	(21%)	521	(22%)
≥ 10%	816	(34%)	834	(35%)	853	(36%)	91	(4%)	95	(4%)	97	(4%)
≥ 15%	471	(20%)	475	(20%)	480	(20%)	37	(2%)	38	(2%)	38	(2%)
≥ 20%	223	(9%)	226	(10%)	228	(10%)	20	*	20	*	20	*

* Following convention for reporting HQ estimates to one significant digit, although this query is based on HQ ≥ 1.5, this translates from a science policy standpoint into an HQ ≥ 2.

Table 2-10. Watersheds with potentially at-risk populations based on consideration for risk (both IQ loss and HQ) based on U.S. EGU mercury deposition and resulting exposure before considering other sources of mercury deposition

EGU risk threshold	Number and percentage of 2,366 HUCs meeting risk threshold criteria*											
	2005 analysis						2016 analysis					
	90th fish consumption		95th% fish consumption		99th% fish consumption		90th fish consumption		95th% fish consumption		99th% fish consumption	
IQ loss												
≥ 1 pt	28	*	67	(3%)	353	(15%)	3	*	7	*	27	*
≥ 2 pts	1	*	5	*	69	(3%)	0	*	0	*	7	*
RfD-based HQ												
≥ 1.5**	277	(12%)	495	(21%)	1064	(45%)	17	*	48	(2%)	284	(12%)

* 2,366 watersheds reflected in this summary are those watersheds out of the 2,461 assessed for risk for the high-end female consumer (at the national-level)

** Following convention for reporting HQ estimates to one significant digit, although this query is based on HQ ≥ 1.5, this translates from a science policy standpoint into an HQ ≥ 2.

Table 2-11. 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

EGU risk threshold	Number and percentage of HUCs meeting risk threshold criteria											
	2005 analysis						2016 analysis					
	90th fish consumption		95th% fish consumption		99th% fish consumption		90th fish consumption		95th% fish consumption		99th% fish consumption	
U.S. EGU-attributable risk ≥1.5* HQ OR total risk ≥1.5* HQ and U.S. EGU contribution of:												
≥ 5%	1271	(54%)	1321	(56%)	1477	(62%)	484	(20%)	524	(22%)	672	(28%)
≥ 10%	879	(37%)	946	(40%)	1221	(52%)	96	(4%)	121	(5%)	325	(14%)
≥ 15%	584	(25%)	682	(29%)	1109	(47%)	49	(2%)	76	(3%)	292	(12%)
≥ 20%	391	(17%)	550	(23%)	1073	(45%)	34	*	61	(3%)	286	(12%)

* Following convention for reporting HQ estimates to one significant digit, although this query is based on HQ ≥ 1.5, this translates from a science policy standpoint into an HQ ≥ 2.

Observations regarding watersheds with potentially at-risk populations due to U.S. EGU sourced mercury are presented below. Note, that these observations focus primarily on results generated for the 2016 Scenario, as explained earlier. In addition, this set of observations is oriented around the 3-Stage risk characterization framework.

- *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:* While total IQ loss estimates (as described earlier) can extend above 2 IQ points for a substantial fraction of the watersheds modeled, less than 1% of the

watersheds have U.S. EGU incremental IQ loss estimates extending into the 1 to 2 point range. However, as noted in section 1.2, IQ does not fully cover all of the neurologic domains such as motor skills and attention/behavior associated with MeHg exposure and therefore, there are concerns that risk estimates based on IQ could underestimate overall neurodevelopmental impacts (Axelrad et al., 2007). For this reason, in considering watersheds with potentially at-risk populations due to U.S. EGU-attributable mercury, we focus on MeHg RfD-based estimates of risk rather than IQ. *Between 2 and 22% of those watersheds with total risk HQs ≥ 1.5 have U.S. EGUs contributing at least 5% of total mercury deposition:* 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 risk of an HQs ≥ 1.5). Considering a 5% U.S. EGU contribution at watersheds where total risk is considered a potential public health hazard, we have up to 22% of the watersheds falling into this category (with the 22% value reflecting risk modeled using the 99th percentile fish consumption rate for the high-end female consumer – see Table 2-9). It is important when considering this risk metric to reiterate that any exposure above the MeHg RfD represents a potential public health hazard. .

- *Between 2 and 12% of the watersheds have HQs ≥ 1.5, based on U.S. EGU mercury deposition before factoring in any other sources of mercury:* Our analysis suggests that between 2 and 12% of the 2,366 watersheds modeled in the risk assessment for high-end female consumers could have an HQ ≥ 1.5 when U.S. EGU mercury deposition and resulting exposure are considered before other sources of mercury deposition. This range reflects the range of fish consumption rates considered for the high-end female consumer (i.e., 95th or 99th percentile consumption rate, respectively – see Table 2-10).

- *Combining (mathematical union) the two sets of watersheds with at-risk populations due to U.S. EGU mercury emissions*: 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% - see Table 2-11). Note, that this range also reflects 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 3 of the 3-stage risk characterization framework.

2.7. Sensitivity Analyses

This section discusses several sensitivity analyses conducted to assess the potential impact of key sources of uncertainty (all related to the application of the MMaps approach in our analysis) on risk estimates. We note that, in designing the sensitivity analysis, we focused on application of the MMaps approach because it represents a critical element of the analysis and is acknowledged as representing a potentially important source of uncertainty. The sensitivity analyses address two specific uncertainties related to application of the MMaps approach: (a) concerns over including watersheds that may be disproportionately impacted by non-air mercury sources and (b) application of the MMaps approach to both flowing and stationary freshwater bodies (if, in reality, the approach is better at predicting source-apportioning Hg fish tissue levels for stationary waterbodies).

The sensitivity analyses addressing the first area of uncertainty (potential inclusion of watersheds from regions with substantial Hg contributions from non-air deposition) included two analyses: (a) constraining the risk analysis to only include those watersheds in the upper 25th percentile with regards to total Hg deposition (i.e., watersheds with relatively elevated levels of total Hg deposition so we knew this source of loading played a larger role) and (b) excluding four states where we have concerns over the potential for non-air mercury playing a greater role (ME, MN, SC and LA).[43] The results of both sensitivity analyses are presented in terms of their impact on the "at risk" metrics described in Section 2.6.2. Specifically, the presentation of the sensitivity analysis results parallel the "at risk" summary table layout used in Section 2.6.2 and as such, present risk estimates for the high-end female consumer (2005 scenario) (see Tables 2-12 through 2-14).

The other area of uncertainty (application of the MMaps approach to both stationary and flowing waterbodies) was assessed by running the risk assessment only for watersheds with fish tissue MeHg values taken from stationary watersheds (i.e., ponds and lakes). Specifically, we generated a set of percentile risk estimates, again for the high-end female consumer assessed nationally, for the 2005 scenario (see Table 2-15). In presenting the results, we also include the core risk estimates generated for this scenario using fish tissue MeHg samples taken from both stationary and flowing waterbodies. Observations resulting from considering the results of the sensitivity analyses are presented at the end of this section.

Table 2-12. Sensitivity analysis results based on constraining analysis to (a) watersheds in the top 25th percentile with regard to total Hg deposition and (b) exclude watersheds located in MN, LA, SE or ME) – Results for watersheds with potentially at risk populations based on U.S. EGUs making a specified contribution to total risk (Stage 1 of the 3 stage framework)

EGU risk threshold	Number and percentage of HUCs meeting risk threshold criteria (2005 scenario)											
	2005 analysis						2016 analysis					
	90th fish consumption		95th% fish consumption		99th% fish consumption		90th fish consumption		95th% fish consumption		99th% fish consumption	
Total HQ ≥ 1.5 and U.S. EGU contribution:												
ALL WATERSHEDS (2,366)												
≥ 0%	2191	(93%)	2266	(96%)	2348	(99%)	2162	(91%)	2248	(95%)	2346	(99%)
≥ 5%	1268	(54%)	1305	(55%)	1345	(57%)	482	(20%)	505	(21%)	521	(22%)
≥ 10%	816	(34%)	834	(35%)	853	(36%)	91	(4%)	95	(4%)	97	(4%)
≥ 15%	471	(20%)	475	(20%)	480	(20%)	37	(2%)	38	(2%)	38	(2%)
≥ 20%	223	(9%)	226	(10%)	228	(10%)	20	*	20	*	20	*

[43] The rational for excluding the four states in this sensitivity analysis examining the MMaps approach is as follows. 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 raising the concern that some other factor may be in play (e.g., other non-air sources, or perhaps substantially increased methylation potential). MN was excluded for the same reason as ME with additional concern for taconite mining which could provide non-air Hg loading. SC was also excluded due to higher fish Hg levels and Hg air deposition that (while elevated in some locations) is not uniformly higher than other states in the region (in addition, there is a history of gold mining in SC). Finally LA was excluded as part of the sensitivity analysis due to concerns for the substantial industrial activity which could result in non-air Hg impacts.

EGU risk threshold	Number and percentage of HUCs meeting risk threshold criteria (2005 scenario)											
	2005 analysis						2016 analysis					
	90th fish consumption		95th% fish consumption		99th% fish consumption		90th fish consumption		95th% fish consumption		99th% fish consumption	
SENSITIVITY ANALYSIS 1 - Watersheds in top 25th % with regard to total Hg deposition (591)												
≥ 0%	554	(94%)	570	(96%)	584	(99%)	548	(93%)	564	(95%)	585	(99%)
≥ 5%	323	(55%)	332	(56%)	341	(58%)	89	(15%)	90	(15%)	94	(16%)
≥ 10%	182	(31%)	185	(31%)	192	(32%)	32	(5%)	32	(5%)	33	(6%)
≥ 15%	122	(21%)	124	(21%)	126	(21%)	19	(3%)	19	(3%)	19	(3%)
≥ 20%	64	(11%)	65	(11%)	67	(11%)	10	(2%)	10	(2%)	10	(2%)
SENSITIVITY ANALYSIS 2 - Watersheds excluding those in MN, LA, SC and ME (1,844)												
≥ 0%	1673	(91%)	1745	(95%)	1827	(99%)	1645	(89%)	1727	(94%)	1825	(99%)
≥ 5%	1133	(61%)	1170	(63%)	1210	(66%)	449	(24%)	472	(26%)	488	(26%)
≥ 10%	791	(43%)	809	(44%)	828	(45%)	77	(4%)	81	(4%)	83	(5%)
≥ 15%	460	(25%)	464	(25%)	469	(25%)	33	(2%)	34	(2%)	34	(2%)
≥ 20%	220	(12%)	223	(12%)	225	(12%)	19	*	19	*	19	*

Table 2-13. Sensitivity analysis results based on constraining analysis to (a) watersheds in the top 25th percentile with regard to total Hg deposition and (b) exclude watersheds located in MN, LA, SE or ME) – Results for watersheds with potentially at risk populations based on U.S. EGU-incremental contribution to total risk (Stage 2 of the 3 stage framework)

EGU risk threshold	Number and percentage of HUCs meeting risk threshold criteria (2005 scenario)											
	2005 analysis						2016 analysis					
	90th fish consumption		95th% fish consumption		99th% fish consumption		90th fish consumption		95th% fish consumption		99th% fish consumption	
RfD-based HQ)												
ALL WATERSHEDS – core analysis (2,366)												
HQ ≥ 1.5	277	(12%)	495	(21%)	1064	(45%)	17	*	48	(2%)	284	(12%)
SENSITIVITY ANALYSIS 1 - Watersheds in top 25th % with regard to total Hg deposition (591)												
HQ ≥ 1.5	90	(15%)	140	(24%)	274	(46%)	8	*	25	(4%)	90	(15%)
SENSITIVITY ANALYSIS 2 - Watersheds excluding those in MN, LA, SC and ME (1,844)												
HQ ≥ 1.5	225	(10%)	423	(18%)	879	(37%)	5	*	17	*	167	(7%)

Table 2-14. Sensitivity analysis results based on constraining analysis to (a) watersheds in the top 25th percentile with regard to total Hg deposition and (b) exclude watersheds located in MN, LA, SE or ME) – Results for watersheds with potentially at risk populations based on combining both Stage 1 and Stage 2 results (Stage 3 of the 3 stage framework)

EGU risk threshold	Number and percentage of HUCs meeting risk threshold criteria (2005 scenario)					
	2005 analysis			2016 analysis		
	90th fish consumption	95th% fish consumption	99th% fish consumption	90th fish consumption	95th% fish consumption	99th% fish consumption
U.S. EGU-attributable risk ≥1.5 HQ OR total risk ≥1.5 HQ and U.S. EGU contribution of:						
ALL WATERSHEDS (2,366)						

EGU risk threshold	Number and percentage of HUCs meeting risk threshold criteria (2005 scenario)											
	2005 analysis						2016 analysis					
	90th fish consumption		95th% fish consumption		99th% fish consumption		90th fish consumption		95th% fish consumption		99th% fish consumption	
≥ 5%	1271	(54%)	1321	(56%)	1477	(62%)	484	(20%)	524	(22%)	672	(28%)
≥ 10%	879	(37%)	946	(40%)	1221	(52%)	96	(4%)	121	(5%)	325	(14%)
≥ 15%	584	(25%)	682	(29%)	1109	(47%)	49	(2%)	76	(3%)	292	(12%)
≥ 20%	391	(17%)	550	(23%)	1073	(45%)	34	*	61	(3%)	286	(12%)
SENSITIVITY ANALYSIS 1 - Watersheds in top 25th % with regard to total Hg deposition (591)												
≥ 5%	323	(55%)	334	(56%)	377	(64%)	89	(15%)	99	(17%)	146	(25%)
≥ 10%	202	(34%)	219	(37%)	306	(52%)	34	(6%)	44	(7%)	97	(16%)
≥ 15%	152	(26%)	179	(30%)	286	(48%)	24	(4%)	37	(6%)	92	(16%)
≥ 20%	115	(19%)	153	(26%)	278	(47%)	16	(3%)	30	(5%)	90	(15%)
SENSITIVITY ANALYSIS 2 - Watersheds excluding those in MN, LA, SC and ME (1,844)												
≥ 5%	1135	(62%)	1179	(64%)	1275	(69%)	449	(24%)	476	(26%)	543	(29%)
≥ 10%	811	(44%)	860	(47%)	1035	(56%)	77	(4%)	87	(5%)	208	(11%)
≥ 15%	529	(29%)	609	(33%)	923	(50%)	35	(2%)	44	(2%)	175	(9%)
≥ 20%	339	(18%)	478	(26%)	888	(48%)	22	*	30	(2%)	169	(9%)

Table 2-15. Sensitivity analysis results based on estimating risk for watersheds with Hg fish tissue levels based only on stationary waterbodies (i.e., excluding samples taken from flowing waterbodies) – Results for both total risk and U.S. EGU-attributable risk

Fisher consumption rate percentile and rate (g/day)	Watershed percentile									
	50th percentile		75th percentile		90th percentile		95th percentile		99th percentile	
	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU	total	U.S. EGU
Primary analysis (all watersheds – lakes and rivers - 2005 scenario)										
mean (39)	3.5	0.3	3.2	0.4	4.2	0.5	5.4	0.7	7.2	1.4
90th (123)	11.1	1.1	10.1	1.3	13.3	1.6	17.1	2.3	22.8	4.3
95th (173)	15.6	1.5	14.2	1.8	18.7	2.3	24.1	3.2	32.0	6.0
99th (373)	33.6	3.3	30.7	3.9	40.3	4.9	51.9	6.9	69.0	13.0
SA (lakes only – 2005 scenario)										
mean (39)	2.9	0.1	3.5	0.3	3.8	0.4	4.6	0.6	6.5	1.4
90th (123)	9.1	0.3	11.0	0.8	12.1	1.3	14.5	2.0	20.5	4.4
95th (173)	12.8	0.5	15.5	1.1	17.0	1.8	20.4	2.7	28.8	6.2
99th (373)	27.6	1.0	33.5	2.4	36.6	4.0	44.0	5.9	62.1	13.5

Observations regarding the sensitivity analyses completed for this analysis are presented below.

- *Generating risk estimates including only those watersheds falling in the top 25th percentile with regard to total Hg deposition:* The percent watersheds with potentially at risk populations ranged from just slightly higher to as much as 50% higher compared with the core analysis which did not exclude watersheds based on the magnitude or

ranking with regard to total Hg deposition (see Tables 2-12 through 2-14). These results suggest that focusing on those watersheds with relatively greater total Hg deposition would result in a slightly larger fraction of "at risk" watersheds.

- *Generating risk estimates excluding watersheds located in four states (AL, SC, ME and MN):* This sensitivity analysis resulted in different effects on the Stage 1 and Stage 2 risk estimates. Estimates of the number of watersheds with at risk populations due to U.S. EGUs contributing at least 5% (note, other percentage contributions are also presented) to total risk (Stage 1) demonstrated a mixture of moderate increases and decreases compared with the core analysis, depending on the "percent contribution" category considered (see Table 2-12). However, estimates of the percentage of watersheds with at risk populations when considering U.S. EGU mercury deposition (without other sources – Stage 2) based on dropping out the four states was 7% as compared with 12% when considering all states (for the 99th percentile fish consumption rate - see Table 2-13). While this sensitivity analysis does suggest that excluding these 4 states does reduce the percent of watersheds with potentially at risk populations included in Stage 2, there is still a notable fraction (i.e., 7% under the 99th% consumption rate based on an HQ $\geq$ 1.5 representing a potential health concern – see Table 2-14).

- *Focusing only on stationary waterbodies (lakes and ponds) and excluding flowing waterbodies did result in notably lower U.S. EGU-incremental risk on average for the waterbodies, however risk estimates for upper end watersheds were not substantially effected:* As can be seen in Table 2-15, running the risk model using only fish tissue MeHg estimates from stationary waterbodies did result in lower U.S. EGU incremental risk for the average watershed. However, importantly, high-end estimates - estimates for the 90th percentile watershed and above - of U.S. EGU incremental risk were not significantly different with that difference disappearing and actually reversing at the highest level (i.e., the risk estimates for the 99th percentile watershed actually tended to be higher for the simulation focusing on stationary waterbodies). Given that the primary focus of the risk assessment is on high-end percentile risk, the fact that this sensitivity analysis only showed difference in risk at mean watersheds with this difference diminishing and actually reversing at the highest watersheds, argues that this source of uncertainty (i.e., that the MMaps approach is most appropriate for stationary waterbodies) does not substantially affect the analysis.

2.8. Summary of Key Observations

Key policy-relevant observations drawn from discussions presented in Sections 2.3 through 2.7 (and including Section 1.4 – the discussion of uncertainty and variability) are presented below:

- Estimates of U.S. EGU mercury emissions suggest that the 2016 Scenario is likely closer to current (2010) emissions compared with the 2005 Base Case (which has substantially higher total mercury emissions for this sector). Therefore, risk estimates generated for the

2016 Scenario have received greater emphasis and the 2005 Base Case estimates are de-emphasized (since they are likely substantially higher than current conditions).

- U.S. EGUs can contribute up to 11% of total Hg emissions over a subset of watersheds (for the 2016 Scenario - value cited is for the 99th% watershed). However, in general, other sources besides U.S. EGUs dominate Hg deposition, with U.S. EGUs contributing on average (again for the 2016 Base Case), 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 specific hot spots in a number of areas, including most notably, the Ohio River valley. U.S. EGU-related Hg deposition estimates show a significant reduction between 2005 and 2016 Base Cases, reflecting mainly implementation of PM controls with the average U.S. EGU deposition decreasing from ~5% of total to ~2% for the 2005 and 2016 scenarios, respectively.

- U.S. EGUs can contribute up to 18% of MeHg in fish tissue (99th percentile watershed value for the 2016 Scenario). However, generally, U.S. EGUs contribute a much smaller fraction averaging 4% 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.

- Comparing the pattern of U.S. EGU-attributable Hg deposition with watersheds containing Hg fish tissue data (i.e., the watersheds reflected in the risk assessment) results in our concluding that, while we have some degree of coverage for high U.S. EGU impact areas, this coverage is limited. For this reason, we 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.

- Estimates of total risk from all sources of mercury using the RfD based metrics do not show a substantial reduction between the 2005 and 2016 Scenarios, which is expected given that sources other than U.S. EGUs dominate Hg deposition over the vast majority of watersheds and emissions for these sources remain largely unchanged in the simulation. However, U.S. EGU-attributable risk does demonstrate a notable reduction between the 2005 and 2016 Scenarios, primarily reflecting implementation of PM precursor emissions controls, as note earlier.

- Under the 2016 Base Case, IQ loss when we consider U.S. EGU mercury deposition without including other sources of mercury is below the 1 to 2 IQ point range for over 99% of the watersheds included in the risk assessment (based on the high-end female consumer scenario). The 1-2 IQ point loss range was identified by the SAB as representing a level of clear public health significance, not that IQ loss below that is of no health significance. It is important to note, that for a substantial fraction of these watersheds, <u>total</u> IQ loss (reflecting all mercury sources) does reach or exceed this range of IQ loss. And in those instances, even a relatively small incremental reduction in IQ loss related to reducing U.S. EGU emissions would be considered beneficial from a

public health standpoint. In addition, as noted in Section 1.2, we do not think the IQ endpoint fully captures the neurodevelopmental risk associated with MeHg exposure and for this reason, we have focused on MeHg RfD-based risk estimates in considering the potential public health hazard associated with U.S. EGU-attributable mercury exposure. Risks estimated for the high-end female consumer population at the national-level are generally higher than those assessed for a number of the other populations covered (e.g, Vietnamese, Hispanic, and Great Lakes Tribal fishers) and therefore provide coverage for those additional fisher populations. However, risk estimates for both white and black fishers in the southeast were higher than estimates for the high-end female consumers assessed nationally. It is also important to note the uncertainty and limitations in the data used for the southeast fisher populations (see Section 2.6.1). Given our desire to provide broader coverage for the U.S. and concerns over Hg fish tissue levels in South Carolina (SC) potentially reflecting non-U.S. EGU loading (fish tissue values in SC play an important role in driving risk for the two southeastern fishing populations), we have placed greater emphasis in discussing risk estimates on the female high-end consumers assessed at the national-level.[44]

- Based on application of the 3-stage risk characterization framework described in Section 1.2, we estimate that from 3 to 28% of the watersheds included in this risk assessment could be classified as potentially having at risk populations under the 2016 Scenario. This percent range is based on a 95th to 99th percentile consumption rates for the high-end female consumer assessed at the national-level and assumes further, that an HQ of ≥ 1.5 (i.e., an exposure just above the RfD – see above) represents a potential public health concern. These stage-3 results reflect the aggregation of results from Stages 1 and 2 of the 3-Stage Risk Characterization Framework (i.e., watersheds where the U.S. EGU increment-based HQ ≥ 1.5 and watersheds where total risk is ≥ 1.5 HQ and U.S. EGUs make at least at 5% contribution to that risk, respectively).

- 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 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 MMaps approach for linking Hg deposition to Hg fish tissue levels, suggest that uncertainty related to the MMaps approach is unlikely to substantially effect an assessment of whether Hg emissions from U.S. EGUs constitute a public health concern.

[44] We note that the female high-end consumer population benefits from (a) being assessed at the national-level (which both increases the number of watersheds assessed for risk and more fully reflects spatial patterns of U.S. EGU-attributable mercury impacts and fish tissue MeHg levels across the country) and (b) has consumption rates based on a larger sample size compared with the more focused southeastern black and white populations (see Appendix C, Table C-1). For these reasons, we believe that risk estimates for the female high-end consumer population have higher overall confidence (in addition, they better cover on the population of concern for the endpoints modeled – women of childbearing age who consume relatively large amounts of self-caught fish).

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Appendices: Additional Technical Detail on Modeling Elements

(Note, citations for the appendices are provided in the citation list for the main document – see above)

A. Specifying spatial scale of watersheds

As mentioned earlier in Section 1.3, the specification of the spatial scale for watersheds to be used as the basis for the risk assessment (i.e., HUC12's) was based, in part, on consideration for the size of watersheds included in two studies examining the relationship between mercury deposition and changes in mercury concentrations in aquatic media and biota. The Knights et al., 2009 study considered a number of modeling frameworks for predicting changes in fish mercury levels, following changes in aerial mercury deposition. The study 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. Response times for changes in mercury fish tissue levels following a 50% reduction in aerial mercury deposition were simulated for the different watersheds. The simulations showed that all five locations had a two-phase response in fish tissue mercury concentrations including (a) an initial 1-3 year response linked to immediate reductions in aerial deposition directly to the waterbody itself and (b) a longer-term (decades) timeframe for a full system response that would include such factors as changes in watershed erosion and loading to the waterbody. The study also showed that deeper lakes with larger watersheds could have longer response times. The article reported that the initial "faster" response (taking 1-3 years) could account for 40-60% of the total steady state response in some instances.

The results of this study can be interpreted as suggesting that, if we are considering the more immediate change in fish tissue mercury levels (occurring within a few years following a change in mercury loading), we should focus on assessing the fractional change in mercury deposition to the waterbody itself. However, if we can assume that near steady-state conditions are met and we are interested in a more complete simulation of changes in fish MeHg levels then we would want to consider changes in the level of mercury deposition over the entire watershed (and not just the waterbody). These observations support using the watershed (and not the individual waterbody) as the basis for linking Hg deposition with fish tissue MeHg levels. 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 mercury levels following a change in mercury deposition. In this study, a 3yr loading of labeled mercury to the waterbody and watershed (separate labeled mercury applied to each location) was followed by measurement of mercury in

various media and biota to see how long it took for the loaded mercury to impact different compartments. This analysis showed that impacts on aquatic biota from watershed mercury could be very slow (on the order of decades). While mercury deposited directly to the waterbody could have a more immediate effect on aquatic biota, this effect was buffered by the methylation of older mercury which resulted in a lag in the impact (after 3-4 years, there had been a 30-40% increase in biota mercury concentrations, however the actual change in deposition to the lake over that same time period had been ~120%, suggesting that this buffering was occurring). The study concludes that lakes which receive most of their mercury deposition loading directly from the atmosphere (e.g., catchment lakes) could see an effect on biota within a decade while lakes with more complex watershed loading conditions could see a two-phase temporal change with a longer lag. 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 support use of a more refined spatial scale for watersheds in the risk assessment.

In addition as mentioned earlier in Section 1.3, use of watersheds that are more spatially refined increases the potential for capturing spatial gradients in the deposition of mercury and resulting variation in the impact of those loadings on aquatic freshwater biota. Conversely, use of larger watersheds, while allowing us to model more of the country in the risk assessment, could result in the dilution of areas of elevated mercury deposition from U.S. EGUs (and therefore, by association, the dilution of U.S. EGU-attributable mercury exposure and risk).

Consideration for the information presented above, resulted in us identifying HUC12s as the optimal watershed size for the risk analysis. HUC12's vary based on topography but generally are from 5-10km on a side.

B. Characterizing measured fish tissue concentrations at the watershed-level

In developing the fish tissue dataset for the risk assessment, we began with the master dataset that has been developed by our team to support the Regulatory Impact Analysis (RIA). This master datasets included fish tissue Hg samples from years 1995-2009, with approximately 50,000 unique samples from 4,115 HUC12s across the US, although the samples are more heavily focused on locations east of the Mississippi. Note, that for the risk assessment, we used a subset of the data from 2000 and later, with samples distributed across 2,461 HUC12s. The use of fish tissue samples from this later time frame was intended to focus on samples more representative of current conditions and less likely to reflect Hg deposition levels prior to 2000 when substantial reductions in Hg emissions and hence deposition were taking place. The fish tissue samples in the master dataset come primarily from three 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, it contains data for over 43,000 mercury 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 mercury 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. The compilation includes (1) state-agency collected and reported data (including Delaware, Iowa, Indiana, Louisiana, Minnesota, Ohio, South Carolina, Virginia, Wisconsin, and West Virginia) from over 40,000 fish tissue samples, covering the period 1995 to 2007 and (2) over 10,000 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/).

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

Data from these three datasets were combined into a single master fish tissue dataset covering the period 1995 to 2009. One problem encountered in combining these datasets is the potential duplication of samples in the NLFA and USGS state-collected data. Unfortunately, these two datasets do not contain directly comparable and unique identifiers that allow duplicate samples to be easily identified and removed. Therefore, as an alternative, the samples from these two datasets were subdivided 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 excluded from the master data.

In finalizing the master datasets a number of criteria were used to screen the fish tissue samples (e.g., include only freshwater fish species, exclude estuarine locations, exclude fish less than 7 inches in length). In addition, we assigned "river" and "lake" identifiers to each fish tissue sample (additional detail on the process used to develop the master fish tissue dataset can be found in USEPA, 2011, Section 5.2.2).

As note earlier, for the risk assessment we used a subset of the Master dataset including fish tissue samples collected 2000 and later. This risk assessment dataset comprised a total of 23,878 samples (12,500 from lakes and 11,478 from rivers) covering a total of 2,461 HUC12s. Subsets of this dataset were used in modeling risk for each of the fisher populations included in the analysis (i.e., those HUCs intersecting US Census tracts containing "source populations" associated with a particular fisher scenario – see discussion in Section 1.3).

Each watershed with measured Hg fish tissue data, tended to have multiple values (the average number of fish tissue measurements for the period 2000 to 2009 for the 2,461 watersheds is 10, although some watersheds had up to 270 measurements). Therefore, we also needed to identify a summary statistic to use for each watershed to represent fish tissue levels in estimating exposure and risk. As noted earlier in Section 1.3, we selected the 75th percentile fish tissue value at each watershed as the basis for exposure and risk characterization. Selection of the 75th percentile value was based on the assumption that 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 mercury 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% sample, then risk estimates could be biased accordingly. In deriving the 75th percentile value for a given watershed, we first generated a set of "river" and "lake" fish tissue percentiles (e.g., a "lake" 75th percentile representing the 75th percentile fish tissue Hg level among lake samples located within that HUC12). We then identified the higher of the lake-75th percentile and river-75th percentile, if both existed, and used that higher value in modeling risk.

C. Defining subsistence fisher scenarios to model

A number of criteria had to be met for a study to be used in providing explicit consumption rates for the high-end fisher populations of interest in this analysis. For example, studies had to provide estimates of self-caught fish consumption and not conflate these estimates with consumption of commercially purchased fish. Furthermore, these studies had to focus on freshwater fishing activity, or at least have the potential to reflect significant contributions from that category, such that fish consumption rates provided in a study could be reasonably applied in assessing freshwater fishing activity. As noted earlier, given our interest in higher-end consumption rates, the studies also had to either provide upper percentile estimates, or support the derivation of those estimates (e.g., provide medians and a standard deviations). Studies of activity at specific waterbodies (e.g., creel surveys), while informative in supporting the presence of higher-end consumption rates, could not be used as the basis for defining our high-end consumption rates since there would be uncertainty in extrapolating activity at a specific river or lake more broadly to fishing populations in a region. Therefore, we focused on studies characterizing fishing activity more broadly for specific regions or states. Application of these criteria resulted in the selection of three studies as the basis for characterizing high-end fish consumption rates for the fisher populations included in the analysis. These studies, together with the fisher populations characterized and a description of the regional coverage assumed for each fisher population (see below) are presented in Table C-1.

As noted earlier in Section 1.3, with the exception of the Tribal fisher population assessed for the Great Lakes (which was restricted to activity on lands ceded to the Ojibwa or Chippewa), we did extend coverage for the other fisher populations beyond the specific areas covered in the surveys.[45] For example, while the Vietnamese and Laotian survey data were collected in California, given the ethnic/cultural nature of these high fish consumption rates, we assumed that this type of high-end fish consumption behavior could be associated with members of these ethnic groups living elsewhere in the U.S. Therefore, the high-end consumption rates referenced in the California study for these ethnic groups were used to model risk at watersheds elsewhere in the U.S. In deciding which of the 2,461 watersheds included in the risk assessment might be subject to fishing activity by a given fisher population, we used U.S. Census data to determine if

[45] The decision to restrict activity for the Tribal fishers to the ceded territories reflects the fact that fish consumption rates are particular to Tribal practices and can vary considerably across Tribes, arguing against extrapolation of fish consumption rates across Tribes.

a "source population" for that fisher population was located in the tract(s) intersecting each watershed. For example we modeled the Vietnamese high-end fisher population only at those watersheds associated with U.S. Census tracts containing at least 25 Vietnamese (i.e., a "source population" for that fisher population). This approach was similarly done for each of the other fisher populations.[46]

Looking beyond these specific ethnic groups, we also need to establish a more generalized high-end (subsistence) scenario that could be assessed broadly across the 2,461 watersheds. Generally all of the studies identified high-end percentile consumption rates (90th to 99th percentiles for the populations surveyed) ranging from approximately one fish meal every few days to a fish meal a day (i.e., 120 g/day to greater than 500 g/day fish consumption). We used this general trend across the studies to support application of a generalized high-end female consumer scenario across most of the 2,461 watersheds.[47]

While we believe that the approach of extending coverage for these fisher populations beyond the regions reflected in the underlying surveys is reasonable, we do acknowledge uncertainty associated with this extrapolation (see Section 1.4 and Appendix F for a discussion of key uncertainties in the analysis).

In addition to the studies cited in Table C-1 used to define the high-end fish consuming populations modeled in the risk assessment, we also reviewed a large number of additional studies characterizing higher-level self-caught fish consumption in the U.S. While these studies had limitations that prevented their use as the basis for defining high-end fisher scenarios to include in the analysis, they do generally support the levels of self-caught fish consumption modeled in the analysis. Several of these "supporting" studies are briefly described below:

- A study by Burger et al., 1999, examining 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 determining 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. However for both groups, the study did suggest that upper end percentile consumption rates could be high and certainly 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 does support the presence of high-end consumption rates for both Blacks and Whites that approach or meet subsistence levels in this area of the country.

[46] In the case of black and white fisher populations in the southeast, we further assumed that "source populations" for each of these fisher groups would comprise at least 25 members (of that race) below the poverty line at the tract level. This reflected the potential for greater subsistence fishing activity among economically disadvantaged individuals.

[47] Similar to the white and black fisher populations in the southeast, we only applied this fisher populations at watersheds located in tracts with at least 25 individuals below the poverty line, which meant that 2,366 of the 2,461 watersheds with Hg fish tissue data were assessed for risk for this more generalized female high-consumer population.

- 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 (CT, FL, MN, and ND). The study did provide estimates of high-end self-caught fish consumption for populations in the four states. 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 hold for self-caught fish consumption in particular). 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, we would point out that the study designs used in these surveys may not effectively capture the relatively small fraction of the overall population likely engaging in high-end subsistence levels of self-caught fishing behavior. This study does provide added support for the existence of subsistence fishing populations, at least within FL and for Tribes within MN. However, failure of the surveys reviewed in the study to capture similar behavior in ND and CT does not necessarily suggest that this type of behavior is non-existent, although it may suggest that it is less prevalent than in FL.

Table C-1. Fisher Populations Included in the Analysis for Hg Exposure and Risk

Fish consuming populations covered by study (and reference information)	Overview of study	Self-caught fish consumption rates (mean, 90th, 95th, 99th) g/day	Observations from the study relevant to the risk assessment	Extrapolation of study populations in 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)	- black: 171, 446, 557, NC * - white: 38.8, 93, 129, 286 - female: 39.1, 123, 173, 373 * n 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)	Sample size is variable – out of 458 respondents, 39 are blacks, 149 are female and 98 are poor – 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% is more than 10X greater than the mean or median intake rate for wild-caught fish). Results are also provided for poor (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 US Census tracts containing members of source populations below the poverty line for the white and black populations.	- the black and white fisher populations were extrapolated to cover all watersheds modeled for risk in the Southeastern states.[a] The rationale for this was that fishing activity by these two groups could be generalized in this region of the country. Note, however that these scenarios were only assessed for watersheds in the Southeast located within US 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 risk to pregnant women in particular), we extrapolated the female consumption rates to all watersheds in the continental US with at least 25 individuals below the poverty line (this is the high-end female consumer population referenced in 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.	- Hispanic: 25.8, 98*, 155.9, NC** - Lao: 47.2, 144.8*, 265.8, NA* - Vietnamese: 27.1, 99.1*, 152.4, NA* * 95th percentile values were provided in the study, however 90th percentile values were not provided and 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.	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.	- the Hispanic fishing scenario was extrapolated to cover watersheds located in US Census tracts with at least 25 poor members of the ethnic populations (e.g., the Hispanic consumption rates would be applied to the subset of the 2,461 watersheds located in US Census tracts with at least 25 poor Hispanic individuals). - the Laotian and Vietnamese fishing scenarios were extrapolated to cover watersheds located in US Census tracts with at least 25 members of the underlying ethnic group.

Fish consuming populations covered by study (and reference information)	Overview of study	Self-caught fish consumption rates (mean, 90th, 95th, 99th) g/day	Observations from the study relevant to the risk assessment	Extrapolation of study populations in the risk assessment
High-end self-caught fish consuming Chipewa 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.	- reported value for all Tribal areas (in the study) combined: 62, 136.2, 213.1, 492.8 Note, that all higher percentiles (90th – 99th) were derived using Crytal Ball (based on median and standard deviations and an assumption of log-normally distributed variability in consumption rates)	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.	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.

[a] southeast for purposes of this analysis comprises: Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, South Carolina, Tennessee, Virginia and West Virginia

D. Estimating total mercury-related exposure at the watershed-level

The following equation is used to estimate total mercury exposure at a particular watershed as an annual average of the daily methylmecury intake per kg body weight:

$$IR = \frac{FTC * FIR * CAF}{BW}$$

IR: daily MeHg intake rate (ug/kg-day). This ingestion dose estimate can be directly compared with the merthylmercury RfD to generate a total HQ.

FTC: mercury fish tissue concentration (ug/g or ppm): The 75th% value provided for each of the 2,461 watersheds (see Section 1.3).

FIR: fish ingestion rate (g/day). These values are specific to a given population (see Table 1-1 in Section 1.3)

CAF: cooking adjustment factor (unitless): Because MeHg is not volatile and is contained primarily in the muscle, this translates into a factor increase of 1.5 for concentration of mercury in the cooked fish (Morgan, Berry, and Graves, 1997).

The IR value described above can be directly compared with the MeHg RfD to generate HQ estimates. However, in order to estimates IQ loss, we need to convert this dose estimate into an equivalent maternal hair concentration since the IQ loss function uses hair mercury as the dose measure. To do that, we use a dose-to-hair conversion factor (DHCV) of 12.5 (units ppm per unit ug/kg-day) that converts ingested dose (IR) to hair mercury concentration in ppm. As noted earlier in 1.3, the DHCV factor is based on a one compartment toxicokinetic model used for deriving the MeHg RfD by Swartout and Rice (2000).

E. Establishing the U.S. EGU-attributable fraction of total exposure

As noted earlier in 1.3, establishing the increment of total exposure estimated for a given fishing population active at a given watershed that is attributable to U.S. EGUs requires to key elements: (a) application of the MMaps assumption linking source contribution to Hg deposition over that watershed to source apportionment of fish tissue Hg levels and consequently exposure estimates and (b) use of CMAQ modeling results characterizing the fraction of mercury deposition over a given watershed that is attributable to U.S. EGUs (as contrasted with the fraction attributable to all other US and foreign-sourced anthropogenic and natural mercury emissions). In this section, we provide additional detail on each of these elements.

Mercury maps assumption

To analyze the relationship between Hg deposition and MeHg concentrations in fish across the 2,461 watersheds included in the risk assessment, as discussed in Section 1.3, we applied the EPA's Office of Water's Mercury Maps approach. MMaps implements a simplified form of the IEM-2M model applied in EPA's Mercury Study Report to Congress (USEPA, 1997). By simplifying the assumptions inherent in the freshwater ecosystem models that were described in the Report to Congress, the MMaps 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 (e.g., over the long term fish concentrations are expected to decline proportionally to declines in atmospheric loading to a waterbody). This solution only applies to 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. EPA recognizes that concentrations of MeHg in fish across all ecosystems may not reach steady state and that ecosystem conditions affecting mercury 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 (note, however, that as described below, we have excluded those watersheds containing gold mines or with other non-EGU related anthropogenic mercury releases exceeding specified thresholds). Finally, EPA recognizes that MMaps does not provide for a calculation of the time lag between a reduction in Hg deposition and a reduction in the MeHg concentrations in fish. Despite these limitations, EPA is unaware of any other tool for performing a national-scale assessment of the change in fish MeHg concentrations resulting from reductions in atmospheric deposition of Hg.

Given that the MMaps approach only applies in those situations where aerial deposition is the dominant source of mercury loading to a watershed, in identifying the 2,461 watersheds to include in the risk assessment, we excluded 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 was 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 (online link: <http://tin.er.usgs.gov/mineplant/>. The identification of watersheds with substantial non-EGU anthropogenic emissions was based on a TRI-net query for 2008 or non-EGU mercury sources with total annual on-site Hg emissions (all media) of 39.7 pounds or more. This threshold value corresponds to the 25th percentile annual US-EGU mercury emission value as characterized in the 2005 NATA. The EPA team considered the 25th percentile US-EGU emission level to be a reasonable screen for additional substantial non-U.S. EGU releases to a given watershed.

There are a number of limitations and uncertainties associated with the application of the MMaps approach in the context of this risk assessment. Several of these limitations are briefly discussed here, but a more complete discussion is presented in Section 5.3.2 or the RIA TSD supporting this regulatory review (USEP, 2011). The application of MMaps in apportioning fish tissue mercury levels and consequently exposure and risk between U.S. EGUs and all other sources of mercury at the watershed-level, assumes that the relationship between fish tissue levels and mercury deposition has remained fairly consistent such that near steady-state conditions have been reached. However, in reality, patterns of mercury 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 mercury deposition from earlier time periods (e.g., the 1990s) when mercury emissions from US sources were experiencing substantial decreases. In addition other factors that can impact rates of mercury methylation (e.g., sulfur deposition to waterbodies, pH and eutrification for those watebodies) have also likely 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 does introduce uncertainty into the application of the MMaps approach. However, we believe that the

MMaps approach for apportioning fish tissue mercury levels is still appropriate to use, particularly if we are not attempting to characterize the temporal profile for apportionment and instead, can assume that sufficient time has passed for near steady state conditions to be reached.[48] Uncertainty related to the application of the MMaps model in the context of this risk assessment is further discussed in Appendix F.

CMAQ modeling

The Community Multi-scale Air Quality (CMAQ) model v4.7.1 (www.cmaq-model.org) is a state of the science three-dimensional Eularian "one-atmosphere" photochemical transport model used to estimate air quality (Byun and Schere 2005; Appel, Gilliland et al. 2007; Appel, Bhave et al. 2008). 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). The emissions data used in the base year and future reference and future emissions adjustment case 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 reference case is intended to represent the emissions associated with growth and controls in that year projected from the 2005 simulation year. Other North American emissions of criteria and toxic pollutants (including mercury) are based on a 2005 Canadian inventory and 1999 Mexican inventory. 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).

[48] We recognize that these predictions of U.S. EGU apportionment of fish tissue Hg levels may not be realized for several years or even decades, and, for that reason, the current risk from Hg from EGUs may be considerably higher because EGU mercury emissions were substantially higher prior to 2005. Furthermore, we recognize that these MMaps-based apportionments of fish tissue Hg levels assume that conditions (in terms of patterns of mercury deposition and factors related to methylation such as sulfate deposition) also hold relatively constant for years to decades such that near steady state conditions in the fish tissue mercury concentrations are realized.

F. Variability and Uncertainty

Table F-1. 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
Variation in the patterns of total and U.S. EGU-attributable mercury deposition across the U.S.	Patterns of annual deposition of mercury including total (all source) and estimates of the U.S. EGU fraction (by watershed) displays considerable spatial variability across the U.S. based on the results of 12 km grid cell CMAQ modeling (see Section 2.3)	By extrapolating CMAQ grid cell results at the more spatially refined HUC12 watershed level, we retain the greater degree of spatial resolution in characterizing mercury deposition obtained through the use of the 12k CMAQ grid cell simulations.
Variation in the patterns of mercury fish tissue levels across the U.S.	Mercury fish tissue measurements can display considerable spatial variability across watersheds.	We have fish tissue measurements for roughly 4% of the watersheds in the U.S. (i.e., 2,461 watersheds with measured values out of 88,000 based on data from 2000-2009). While our measured fish tissue levels do generally provide some degree of coverage for areas with elevated mercury 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 mercury deposition for which we do not have fish tissue measurements (see Section 2.5).
Variable response of mercury fish tissue levels to changes in patterns of mercury deposition (MMaps approach)	The impact of changes in mercury deposition on mercury concentrations in fish within a given watershed 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). Not only do these factors contribute to variation in the degree to which fish tissue mercury levels will respond to changes in mercury deposition, they also affect the temporal profile of that response.	Variation in the methylation potential across waterbodies is minimized as a factor in our analysis to a certain extent given our use of the MMaps approach combined with measured mercury fish tissue levels. Specifically, variation in methylation potential should be implicitly reflected in the measured fish tissue levels used in the analysis (i.e., if watershed "a" has a much greater methylation potential than watershed "b", then measured fish tissue levels for "a" should be higher reflecting that difference in methylation potential). In other words, we are applying the MMaps proportionality assumption to measured fish tissue levels that should reflect underlying differences in methylation potential across waterbodies. 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.
Variation in the types of subsistence fisher populations active in different regions	Studies reviewed in developing the approach for this analysis suggests that there can be considerable variation 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	Surveys of near-subsistence and subsistence fishing populations allow us to clearly define this type of activity for specific areas covered by those surveys (e.g., Hispanic, 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

Source of variability	Description	Degree to which source is reflected in design of risk analysis
	driven be socio-economic status (SES) related factors (e.g., poverty, degrees of income). In addition, we would expect that access to freshwater fishing locations would also play a role.	the U.S. Therefore, we did extrapolate coverage from areas reflected in these studies to other portions of the U.S. There is uncertainty associated with this process. As discussed in Section 1.3 a lack of comprehensive survey information on near-subsistence and subsistence fishing activity for all regions in the U.S. is a limitation of the analysis.
For a given fisher population, variability in fishing activity (e.g., species harvested, fishing activity focused at one or more waterbodies)	High-end fisher populations could display considerable variability both in terms of the degree to which they frequent specific waterbodies or watersheds and the degree to which they target specific types of fish (or at least sizes of fish). Both of these factors can impact estimates of exposure. If a fisher population distributes their activity across a range of waterbodies and harvests a variety of fish species (and sizes) than the distribution of exposure and risk across that population will be smaller compared with a population that focuses activity at individual waterbodies and tends to focus on larger fish and/or higher trophic level fish (which will tend to have higher mercury concentrations, other factors equal).	We do not have comprehensive information characterizing the nature of high-end fisher behavior in terms of the factors listed here (i.e., degree to which fishing activity targets individual waterbodies and fish species). Given that this analysis was aimed at assessing whether a public health hazard exists due to U.S. EGU mercury emissions we did consider the situation in which high-end fishers would engage in fishing activity that could result in somewhat higher exposure and risk, other factors equal . Specifically, we modeled exposure and risk assuming that (a) fishing activity is focused within a given watershed (i.e., at waterbodies within that watershed) and (b) that the fishers modeled would tend to favor somewhat larger fish given that they are engaged in subsistence activity and therefore supplementing their diet with fish (recall that we used the 75th percentile fish tissue value in exposure and risk simulations – see Section 1.3). Note, that if a portion of a fisher population actually distributes their activity between watersheds and/or consumes a mixture of fish species and sizes (reflecting a fish tissue level closer to the median or mean for a watershed), then risks would be lower than those estimated here.

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

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 relating to the estimation of mercury deposition over watersheds using the CMAQ model (e.g, estimating	Emissions are quantified from anthropogenic and natural sources, but re-emission of historical emissions (pre-2005) are not well characterized by the modeling system.	Generally all of the sources of uncertainty reflect the fact that mercury deposition estimated over specific watersheds may be over or under-estimated. The one source of uncertainty for which we can differentiate the potential direction of impact (i.e., bias) is mercury wet deposition where we believe that	The analysis did not include any specific quantitative analyses aimed at characterizing uncertainty associated with these three sources of uncertainty, with the exception of qualitative consideration for the potential seasonally-differentiated bias in wet deposition discussed here.

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
mercury emissions from U.S. EGUs, chemistry associated with mercury fate and transport, prediction of wet and dry deposition, and global inflow of mercury into the U.S.)	The complete set of mercury oxidation and reduction reactions has not been identified by the scientific community. Uncertainty in a wide variety of model inputs (e.g., emissions, meteorology, global inflow to the modeling domain, and chemistry) impacts estimates of mercury wet and dry deposition. 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.	estimates may be slightly low in the fall and slightly high in the winter. This assessment of potential bias is based on comparisons of weekly estimates (generated by CMAQ) against measurements of wet mercury deposition collected as part of the Mercury Deposition Network, which operates under the National Atmospheric Deposition Program (http://nadp.sws.uiuc.edu/MDN/).	
Characterizing subsistence fishing activity within areas of high U.S. EGU mercury deposition	There is uncertainty associated with predicting high-end fishing activity at specific watershed and at watersheds located within specific regions. Furthermore, there is uncertainty associated with characterizing the nature of that fishing activity (including the frequency of activity at different waterbodies, types of fish targeted).	If subsistence fishing activity is assumed at a given watershed or at group of watersheds within a region and in reality, factors preclude that type of fishing activity (e.g., lack of ready access, poor fishing stock etc), then those risk estimates are not representative and actual risk (assessed over the set of watersheds modeled) would be reduced, since these point estimates would be removed. Similarly, if high-end fishing activity by a given fisher or fishing family tends to be distributed across watersheds and not focused at a single watershed, as assumed here, then the upper-end risks estimated across the watersheds will be reduced, since watersheds with the highest	We did not explore these sources of uncertainty. 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 would model this behavior in our analysis. If these assumptions are relaxed, then risk will be reduced (specifically for the watersheds with the highest U.S. EGU-attributable risk). However, we did

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
		U.S. EGU-attributable risks will be combined with lesser-impacted watersheds in generating risk estimates (and not assessed independently for risk assuming activity focused specifically on them). Similarly, for the types of fish taken, if high-end fishers do not favor larger fish, then risk could be overstated by our use of the 75th percentile fish.	not explicitly model these alternative behavioral profiles, because of our goal of capturing reasonable estimates of high-end risk.
Application of MMaps assumption in generating estimates of the U.S. EGU-attributable fraction of risk	Uncertainty associated with Applying the MMaps assumption in the context of this analysis results from a number of factors: (a) fish tissue measurement data used in the analysis may still reflect earlier historical patterns of mercury deposition (from the 1990's), (b) watersheds display substantially different methylation potentials, resulting in differences in the impact of a unit change in mercury deposition on fish tissue levels, (c) factors related to methylation in watersheds (e.g., sulfate deposition, pH, eutrafication) have not remained constant over time, resulting in variation in the methylation potential of watersheds over time, (d) despite efforts to exclude watersheds with substantial non-air sources of mercury loading, some watersheds with substantial non-air impacts may have been retained in the analysis, and (e) the potential	If air deposition patterns from the 1990s are reflected in some of the mercury fish tissue measurements we are using 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 we may overstate 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. Differences in methylation potential across watersheds (as discussed in table C-1), are likely to be reflected in the underlying fish tissue levels themselves. Therefore, these methylation differences are likely not to have a substantial impact on our analysis (note, that 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). The potential that we may have failed to exclude watersheds with significant non-air mercury loadings could introduce high-bias into our estimates of U.S.	As part of the sensitivity analyses completed for the analysis, we did consider the issue of potentially having included watersheds with substantial non-air impacts. Although as described in Appendix E, we did exclude watersheds with active gold mines and/or non-EGU anthropogenic sources of Hg release meeting specified criteria, there still is the potential that we may included some watersheds that should have been excluded. Specifically, this concern exists in LA, SC, MN, and ME, where there are either broader concerns over non-air mercury sources (e.g., taconite mining in MN and gold mining in SC) or where there appear to be relatively higher mercury fish tissue levels in the absence of elevated mercury deposition (again raising the concern over non-air deposition sources). To examine the potential impact of locations with elevated non-air mercury sources, we completed two sensitivity analyses including: (a) an analysis of risks when watersheds falling in these four states are excluded and (b) an analysis for risk only for the subset of 2,366 watersheds falling in the upper 25th percentile with regard to total mercury deposition (i.e., those watersheds having relatively elevated mercury deposition) (see Section 2.7 for additional detail on these sensitivity analyses). As part of our sensitivity analyses, we also

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 the MMaps approach is more applicable in the context of stationary waterbodies (lakes and ponds) than flowing waterbodies (streams and rivers).	EGU-attributable risk, since we would overstate the role of U.S. EGUs in contributing to risk, by overlooking the other non-air sources. If the MMaps approach is more effective at linking areal Hg deposition to Hg fish tissue levels for stationary waterbodies, then our application of the MMaps approach to both stationary and flowing could result in a distribution of watershed-level risks that could be incorrect.	examined the issue of applying MMaps to stationary and flowing waterbodies. Specifically, we ran the model for a lakes-only simulation and compared these results with the baseline run including both lakes and rivers (see Section 2.7).
Estimating IQ loss for children born to high-fish consuming mothers modeled in the analysis	There are a number of sources of uncertainty associated with modeling IQ loss in this analysis: (a) IQ may not fully capture the most sensitivity cognitive endpoints associated with mercury exposure , (b) potential confounding from long-chained polyunsaturated fatty acids (LCPFAs) found in fish, and (c) including potential outliers from the epidemiological datasets used in deriving the IQ loss functions.	IQ may not represent the most sensitive cognitive endpoint for mercury exposure (Axelrad et al., 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 mercury exposure (when we focus on modeling the IQ loss endpoint alone). The potentially beneficial effects of LCPFAs (found in fish) on neurological development can confound the effects of mercury by potentially masking those adverse effects. This would result in IQ loss slopes that are biased low, since the IQ loss they are representing is counteracted to some extent by the LCPFAs exposure. 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 mercury, 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.	Because we do not have readily available data to support quantitative analyses of the first 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. We note, that 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. 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).

G. Potential for Deposition "Hotspots" in Areas Near U.S. EGUs

EPA also evaluated the potential for "hot spot" deposition near U.S. EGU emission sources on a national scale, based on the CMAQ modeled Hg deposition for 2005 and 2016. We calculated the excess deposition within 50 km of U.S. EGU sources by first calculating the average U.S. EGU attributable Hg deposition within a 500 km radius around the U.S. EGU source. This deposition represents the likely regional contribution around the EGU. We then calculated the average U.S. EGU attributable Hg deposition within 50 km of the U.S. EGUs to characterize local deposition plus regional deposition near the EGU. Excess local deposition is then the 50 km radius average deposition minus the 500 km radius average deposition. Figure 1 shows a map of the excess local deposition based on the 2005 CMAQ modeling. Figure 2 shows excess local deposition based on the 2016 Base Case.

The maps in Figures 1 and 2 were generated by applying an averaging kernel to the 12km EGU attributable mercury deposition estimates from CMAQ. Averaging kernels assign a mean value to each grid based on the averages of all neighboring grids within a predefined window. In this case, window or kernel sizes were 50 km and 500 km-radiuses. Then 50km-radius average values were subtracted from 500km-radius averages to create the final hot spot image.

Summary statistics for the excess local deposition are provided in Table 1. Table 1 shows both the mean excess deposition around all U.S. EGUs, and the mean excess deposition around just the top 10 percent of Hg emitting U.S. EGUs. Table 1 also shows the excess Hg deposition as a percent of the average regional deposition to provide context for the magnitude of the local excess deposition. In 2005, for all U.S. EGU, the excess was around 120 percent of the average deposition, while for the top 10 percent of Hg emitting U.S. EGU, local deposition was around 3.5 times the regional average. By 2016, the absolute excess deposition falls, however, the local excess still remains around 3 times the regional average for the highest 10 percent of Hg emitting U.S. EGUs.

This analysis shows that there is excess deposition of Hg in the local areas around EGUs, especially those with high Hg emissions. Although this is not necessarily indicative of higher risk of adverse effects from consumption of MeHg contaminated fish from waterbodies around the U.S. EGUs, it does indicate an increased chance that Hg from U.S. EGUs will impact local waterbodies around the EGU sources, and not just impact regional deposition.

Figure G-1. Excess Local Deposition in 2005

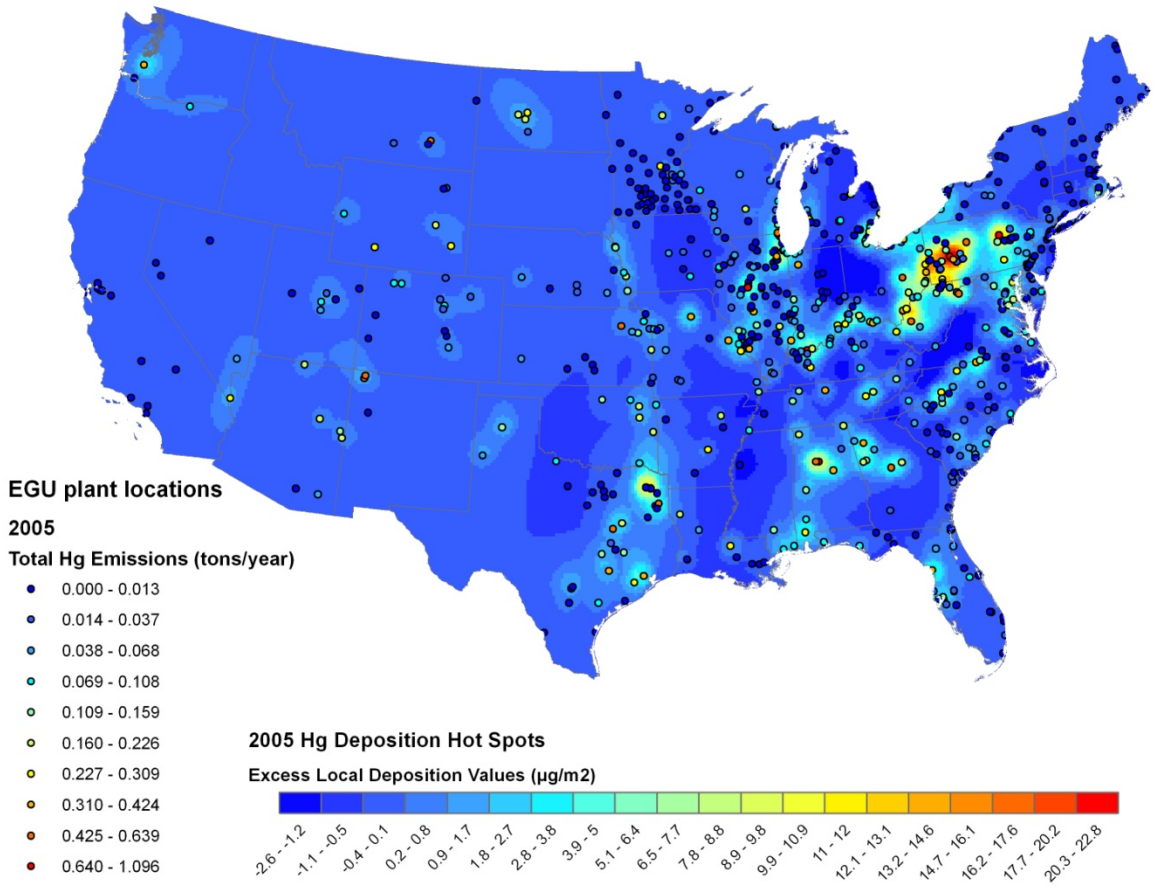


Figure G-2. Excess Local Deposition in 2016 (Base Case)

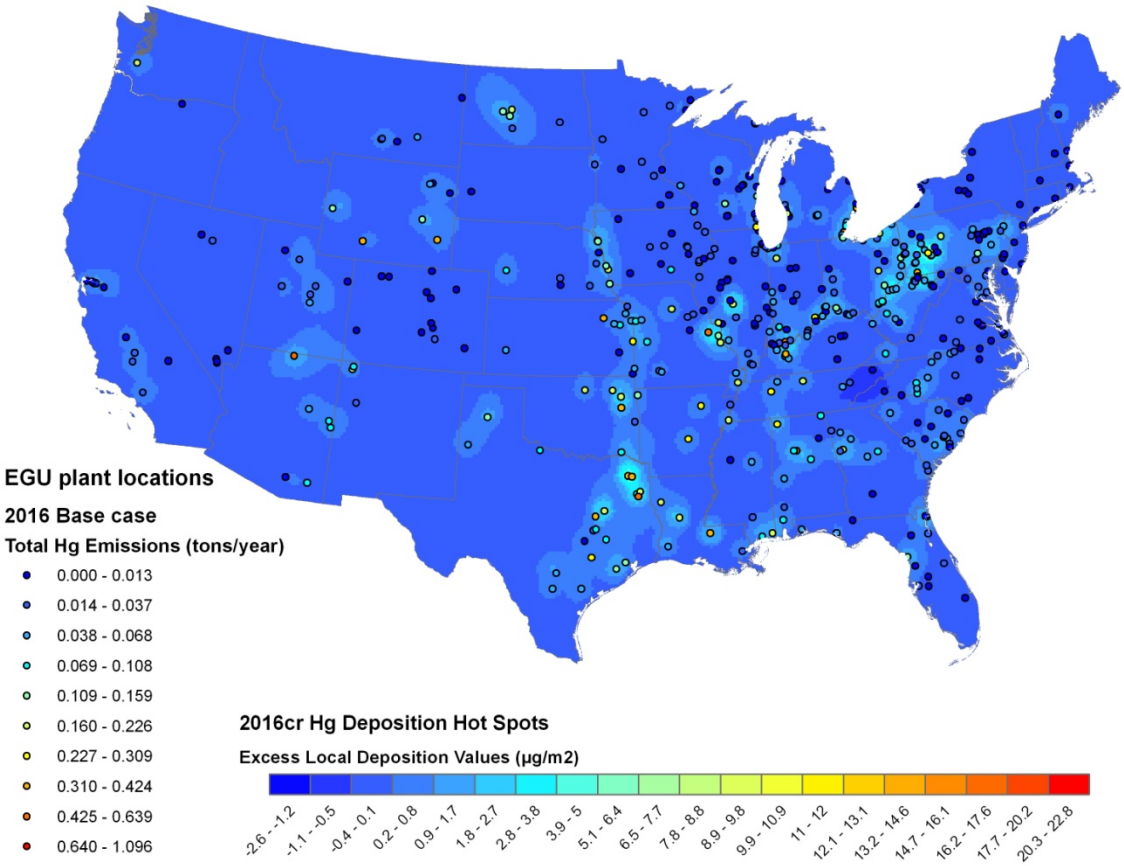


Table G-1. Excess local deposition of hg based on 2005 CMAQ modeled Hg deposition

	50km-Radius-Average Excess Local Deposition values ($\mu g/m^2$)	
	Mean Across EGUs (percent of regional average deposition)	
	2005	2016
All U.S. EGU sites with Hg emissions >0 (672 sites)	1.65 (119%)	0.36 (93%)
Top ten percent U.S. EGU in Hg emissions (67 sites)	4.89 (352%)	1.18 (302%)

United States Environmental Protection Agency	Office of Air Quality Planning and Standards Health and Environmental Impacts Division Research Triangle Park, NC	EPA-452/D-11-002 March 2011