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SUBJECT: National-Scale Mercury Risk Estimates for Cardiovascular and Neurodevelopmental Outcomes for the National Emission Standards for Hazardous Air Pollutants: Coal- and Oil-Fired Electric Utility Steam Generating Units – Revocation of the 2020 Reconsideration, and Affirmation of the Appropriate and Necessary Supplemental Finding; Notice of Proposed Rulemaking.

TO: Docket ID No. EPA-HQ-OAR-2018-0794

This technical support document (TSD) describes the technical approach and presents results associated with analyses intended to further characterize the public health burden (risk) associated with mercury (Hg) released from U.S. electric utility steam generating units (EGUs) in the context of the Mercury and Air Toxics Standards (MATS). These additional analyses include:

- Risk of incidence of myocardial infarction (MI) deaths for the general U.S. population associated with U.S. EGU-sourced Hg as accessed primarily through fish consumption (section a).[1]
- Risk of incidence of IQ points lost for children born to mothers within the general U.S. population exposed prenatally to U.S. EGU-sourced Hg primarily through fish consumption (section b).
- Assessment of the potential for increased MI mortality risk for subsistence fishers exposed to U.S. EGU-sourced Hg at freshwater waterbodies within the continental U.S. (represents an extension of analyses for the 2011 risk and exposure assessment (REA) in support of the Appropriate and Necessary (A&N) determination) (section c).

All three analyses are based on the previous projection that U.S. EGU Hg emissions would total 29 tons in 2016 prior to implementation of MATS but after implementation of all other relevant Clean Air Act (CAA) regulations, as described in the 2011 MATS Risk Assessment (MATS REA, U.S. EPA, 2011a). Risks are then presented as annual risks, meaning estimates that would be anticipated in each year subsequent to 2016 assuming a 29 ton annual emissions rate of Hg from U.S. EGUs.[2] Each of the above analyses is described in detail below, including: (a) an overview intended to provide the reader with a conceptual understanding of what is being modeled, (b) a description of the technical approach used including the concentration-response functions (or relevant equations) and the model inputs, (c) a presentation of results and (d) a discussion of uncertainties and characterization of the overall confidence in the analysis.

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[1] While exposure of the general U.S. population to Hg can result from a range of sources including dietary sources other than fish (*e.g.*, rice), for this analysis, we are assuming that the majority of methylmercury (MeHg) exposure results from fish consumption, which is supported in the literature (EPA. 1997. Mercury Study Report to Congress. EPA–452/R–97–003 December 1997). To the extent other food sources cause exposure to MeHg, it is reasonable to attribute at least some of that exposure to Hg emissions from U.S. EGUs, thereby adding to the risk attributable to MeHg in fish tissue.

[2] EPA does recognize that there could be lag-times associated with realizing risks from a particular years' emissions with fish tissue MeHg concentrations and associated health effects (as described later).

Additionally, the EPA has translated the MI-related deaths and IQ points lost estimates presented in sections a and b into dollar (2016) equivalent benefits values. The methods used in those calculations as well as the results and discussion of associated uncertainties are presented in section d.

a. MI mortality risk attributable to U.S. EGU-sourced Hg for the general U.S. population exposed primarily through fish consumption

i. Overview

This analysis estimates the annual incidence of MI deaths in the general U.S. population resulting from consumption of fish containing Hg (as MeHg) originally emitted from U.S. EGUs. This is a pre-MATS analysis that assumes an ongoing emissions rate of 29 tons Hg from U.S. EGUs each year. This is accomplished by first estimating the total burden of MeHg-related MI mortality in the U.S. population and then estimating the fraction of that total increment attributable to U.S. EGUs. The task of modeling this health endpoint can involve complex mechanistic modeling of the multi-step process leading from U.S. EGU Hg emissions to Hg deposition over global/regional fisheries to bioaccumulation of MeHg in fisheries stocks to exposure of U.S. fish consumers through consumption of those commercially-sourced fish (*e.g.*, Giang and Selin, 2016). However, in recognition of the uncertainty associated with attempting to model this complex process, we have instead developed a simpler bounding analysis approach intended to generate a range of risk estimates that reflects the impact of critical sources of uncertainty associated with this exposure scenario including (a) uncertainty in specifying the concentration response (CR) function for MI-mortality including specifically, the potential for masking of the mortality effect by heart-protective agents also found in fish including polyunsaturated fatty acids (PUFAs), and (b) the linkage between U.S. EGU-emitted Hg and concentrations of MeHg in commercially-sourced fish.

It is important to emphasize that this bounding analysis is not intended as a rigorous assessment of this complex exposure scenario aimed at generating a high-confidence best estimate. Rather, this bounding analysis is intended to provide an order-of-magnitude screening estimate for the potential range of MI-mortality associated with U.S. EGU-sourced Hg reflecting the impact of two key sources of uncertainty (identified above and expanded on below) deemed particularly impactful for this exposure scenario.

Regarding the specification of the CR function, with consideration for heart-protective agents found in fish, as described in greater detail in section a.ii below, we have developed a confidence cutpoint approach wherein we specify a degree of MeHg exposure (the confidence cutpoint) above which we have increased confidence in specifying the CR function and below which we are less confident in specifying the CR function. Support for the use of this confidence cutpoint approach includes: (a) recommendations by Roman et al., 2011, that MI mortality risk only be modeled above MeHg exposure levels associated with the KIHD and EURAMIC-based studies they recommend as the basis for deriving risk models for this endpoint,[3] (b) consideration for J-shaped functions reported in several epidemiology studies assessing the MeHg-

[3] The KIHD (Kuopio Ischemic Heart Disease Risk Factor) and EURAMIC (European Community Multicenter Study on Antioxidants, Myocardial Infarction and Breast Cancer) studies are described in detail in Roman et al., 2011 and in the original studies (Virtanen et al., 2005 and Guallar et al., 2002, respectively).

cardiovascular effect (Roman et al., 2011; Hu et al., 2021) wherein initial levels of MeHg exposure are associated with decreasing mortality risk (suggesting possible masking by cardio-protective agents) followed by an inflection point and then a clear trend of increasing risk, and (c) study data from Mozaffarian and Rimm et al., 2006, suggesting that the protective effects of PUFA are associated with an initial (lower) range of intake but that above a specific level of PUFA, protective effects diminish (again pointing to the potential for PUFA masking at lower levels of fish intake and consequently MeHg exposure). In selecting the actual confidence cutpoints for the bounding analysis, ultimately, we used the recommendations from Roman et al., 2011 to consider the mean MeHg exposure levels from the two study datasets (KIHD and EURAMIC) as cutpoints for modeling MI mortality and utilized the other study data cited above as support for those confidence cutpoints, as detailed below.

Regarding the linkage between U.S. EGU Hg and MeHg in commercially sourced fish, the bounding approach assumes that fish sourced from global commercial fisheries are loaded by Hg deposited from the ambient air to those fisheries and that the fraction of that deposited Hg that originates from U.S. EGUs will eventually be reflected as a fraction of MeHg in those fish and subsequently as a fraction of MI mortality risk experienced by the U.S. population. Therefore, the bounding analysis for this modeling step includes: (a) a lower-bound reflecting the assumption that U.S. fish consumption is largely sourced from global fisheries and consequently the U.S. EGU contribution to total global Hg emissions (anthropogenic and natural) defines the U.S. EGU fractional contribution to MI mortality and (b) an upper-bound where we assume that fisheries closer to U.S. EGUs (*i.e.*, within the continental U.S. and/or along the U.S. Atlantic and Pacific coasts) supply most of the U.S. fish consumption and therefore U.S. EGU average deposition over the U.S. (as a fraction of total Hg deposition) is used to define the U.S. EGU fractional contribution to MI mortality. Note, that in both of these cases, our bounding analysis allows us to avoid the complex process of conducting fate/transport to predict specific rates of deposition (and subsequent aquatic food-web modeling) over specific fisheries regions, while still reflecting reasonable bounds for how U.S. EGU Hg emission may impact sources of fish consumed by the U.S. population.

ii. Technical Approach

The presentation of equations, inputs and rationale is divided into two components (a) the modeling of total MeHg-related MI mortality in the general U.S. population (including application of the confidence cutpoint for the MI mortality CR function) and (b) estimation of the fraction of that risk attributable to U.S. EGU Hg emissions (application of the bounding approach referenced in the *Overview* above).

Step 1. Estimation of MeHg-related MI mortality risk to the general U.S. population exposure primarily through fish consumption.

This section describes the approach used to estimate the total MI-mortality associated with MeHg for the general U.S. population. The equation for modeling MeHg-related mortality for this population is presented below. This health impact function, which is log-linear in form (reflecting the underlying epidemiology study supplying the beta coefficient), relates mean hair-Hg for a population to a change in the baseline MI mortality for that population utilizing the beta coefficient as reflected in the exponential model form. In modeling this health endpoint, we make the key assumptions that (a) Centers for Disease Control (CDC) 1999-2000 National Health and Nutrition Examination Survey (NHANES) data for women of child-bearing age can

be extrapolated to represent the general adult population in the U.S. and (b) that the hair-Hg levels reflected in that NHANES dataset result primarily from the consumption of fish containing MeHg. This latter assumption allows us to apply our CR function for MI-mortality including, importantly, the confidence cutpoints intended to reflect potential masking of the MeHg effect by PUFA (this would not be as relevant for non-fish sources of Hg exposure such as rice). Both assumptions are discussed in the uncertainty section.

The specification of the confidence cutpoints integrated into the CR function for MI mortality requires additional explanation. As mentioned earlier, epidemiology studies have suggested uncertainty associated with evaluating the MI effect at lower Hg exposure levels due possibly to masking of the MeHg cardiovascular effect by heart-protective agents contained in fish including, in particular, PUFAs.[4] In an attempt to address that uncertainty, we have incorporated consideration of a confidence cutpoint associated with the MI mortality effect. This confidence cutpoint is intended to represent a level of MeHg exposure (expressed as hair-Hg) above which we have increased confidence in specifying the MI mortality effect and below which we have reduced confidence in specifying that effect.

Support for the application of a confidence cutpoint in modeling MI mortality comes from a number of sources. The EPA's 2010 workshop on the cardiovascular effects of Hg (Roman et al., 2011) recommended that "…consideration could be given to restricting application of the dose–response function derived from these studies [KIHD and EURAMIC] to only those with higher end exposures (*i.e.*, include a possible threshold for MeHg toxicity)." Roman et al., 2011 further notes that the mean hair-Hg levels associated with both studies (1.9 and 0.66 μg/g, respectively), which they recommend as thresholds, correspond to the upper end (75^{th}-95^{th} percentiles) of exposure levels for U.S. women of child-bearing age implying that this range could be used to identify a potential threshold in modeling MeHg-related mortality at the population level, with only a fraction of the U.S. population (5 to 25%) exceeding that level of exposure.

A second source of support for application of these confidence cutpoints are J-shaped functions reported in several studies for the MeHg-cardiovascular mortality effect (Roman et al., 2011; Hu et al., 2021). These J-shaped functions suggest an initial range of MeHg exposure associated with decreasing (or mixed) patterns of risk followed by an inflection point and then a range of increasing (positive sloping) risk. These J-shaped functions could be interpreted as supporting consideration for a confidence cutpoint defined using these inflection points as a means of demarcating the level of MeHg above which there is evidence of a clear adverse MeHg effect and below which there is reduced confidence in specifying the nature of that effect. The Roman et al., 2011 paper presents a plot of MI mortality risk based on the EURAMIC data set (Figure 2, Roman et al., 2011) which clearly displays a J-shaped form with an inflection point around 0.2 mg/kg toenail Hg (this translates into ~0.5 μg/g hair-Hg).[5] More recently, Hu et al., 2021 also suggest a J-shaped relationship between Hg exposure and cardiovascular risk with the

[4] In an assessment of evidence supporting an association between Hg exposure and cardiovascular effects, Roman et al., 2011 (reporting on an EPA workshop focused on the subject) suggested the potential for confounding of the adverse cardiovascular effect of Hg (through fish consumption) by cardioprotective agents including PUFA and selenium. The authors did note inconsistency in the degree to which available studies provide effective control for potential confounding by PUFA and selenium thereby making it difficult to characterize the degree to which these agents interact with the effect of Hg. This point was reiterated in more recent studies (*e.g.*, Hu et al., 2021).

[5] Conversion of toenail-Hg to hair-Hg is based on application of a steady state ratio (2.44 Hg/g hair per μg Hg/g toenail) presented in the supplement to Roman et al., 2011.

inflection point (for fatal/non-fatal IHD) occurring around 1.0 µg/g hair-Hg (see Figure 4, Hu et al., 2021). We note that a relative risk (RR) value of 1.0 for this endpoint was not crossed until an exposure level of 2.0 µg/g hair-Hg was passed (although the risk was clearly increasing after the initial inflection point at 1.0 mg/kg). Evidence for the J-shaped functions referenced here further argues for consideration of a confidence cutpoint in modeling the MeHg MI effect, with that cutpoint being informed by consideration for the range of the inflection points seen in these J-shaped functions (*i.e.*, points of transition in MeHg exposure from a negative to positive slope for risk).

Support for application of a confidence cutpoint for the MeHg cardiovascular effect is also provided by study data suggesting that the beneficial effects of PUFA may be limited to an initial range of PUFA intake with protective qualities diminishing beyond a specific point. This could be interpreted as evidence supporting potential masking of MeHg risk at lower levels of fish consumption where PUFA is heart protective. Evidence for potential threshold-like behavior for the heart protective qualities of PUFA are presented in Mozaffarian and Rimm et al, 2006. That study found that n-3 fatty acid intake up to 250 mg/day (about an ounce of white albacore tuna based on their data) produced clear reductions in cardiovascular mortality but that n-3 fatty acid intake rates above that level did not show further benefit. Assuming that n-3 fatty acid intake tracks with fish consumption (and consequently with MeHg intake depending on the type of fish consumed) then it would be reasonable to posit that PUFA masking of MeHg cardiovascular effects could be limited to this initial level of PUFA intake (up to 250 mg/day). Furthermore, this 250 mg/day PUFA could translate into a specific level of MeHg exposure (depending on the types of fish consumed which determines co-exposure to PUFA and MeHg). For example, based on data presented in Mozaffarian and Rimm et al., 2006, if an individual was to consume one ounce of white (albacore) tuna a day (approximately sufficient to reach the 250 mg/day PUFA threshold for diminution of the cardio-protective effect) they would have a daily MeHg intake of ~1.0 µg/g from that tuna.[6] Interestingly, this value is close to the inflection point seen in the Hu et al., 2021 J-shaped plot for fatal/non-fatal IHD as referenced earlier. As the fish consumption rate continues to increase, at some point PUFA intake passes 250 mg/d at which point there is diminishing PUFA cardiovascular benefit, but a continuing increase in MeHg-mediated risk as demonstrated in both the KIHD and EURAMIC study results (both studies saw clear MeHg-related risk at higher MeHg intake levels as reported in Roman et al., 2011). While the evidence cited here for a potential threshold in PUFA benefit does not definitively show that PUFA masking of the MeHg cardiovascular effect is occurring at lower levels of MeHg exposure, the possibility for a PUFA threshold associated with masking does add support for incorporation of a confidence cutpoint into the modeling of MeHg-related cardiovascular risk.

Based on the information summarized above, in specifying the confidence cutpoints for the MeHg MI-mortality CR function, we decided to use the Roman et al., 2011 recommendations for a potential threshold based on the EURAMIC and KIHD studies (*i.e.*, 0.66 and 1.9 µg/g, respectively) as the confidence cutpoints. In selecting these values, we note that the Roman et al., 2011 paper is reporting the findings of an expert panel assembled by EPA specifically for the purpose of considering cardiovascular effects associated with MeHg exposure. Furthermore, we

[6] The estimate of ~1.0 (actually 1.08) µg/g hair-Hg is based on a one ounce per day intake of white (albacore) tuna which provides ~250 mg/day n-fatty acid intake (see Table 2 in Mozaffarian and Rimm et al., 2006). Table 2 also reports that this form of tuna has average Hg levels of 0.35 µg/g. Using the conversion factors for intake to blood-Hg and blood-Hg to hair-Hg provided in section b below results in the estimate for hair-Hg of ~1.08 µg/g.

note that the two studies were cited by the panel as the basis for modeling MI mortality and that the panel explicitly identified these two hair-Hg values (mean exposure levels for both study populations) as potential thresholds for modeling this endpoint. In addition, we note that these two values (0.66 and 1.9 μg/g hair-Hg) are supported by the inflection points associated with J-shaped functions also reported in the literature and discussed above (*i.e.*, ~0.5 μg/g based on the KIHD plot presented in Roman et al., 2011 and 1.0 μg/g for fatal/non-fatal IHD in Hu et al., 2021). We also note evidence from Mozaffarian and Rimm et al., 2006 suggesting a potential threshold for protective effects associated with PUFA (around 250 mg/d) which based on our hypothetical scenario of a fish diet based on sufficient white (albacore) tuna to approximately attain that degree of PUFA (one ounce) would result in a steady-state hair-Hg level of ~1.0 μg/g, again supporting these confidence cutpoints.

In applying these confidence cutpoints (as part of our bounding analysis for MI-mortality), we include not only the two cutpoints (0.66 and 1.9 μg/g hair-Hg) but also a "no cutpoint" assumption wherein we model risk across the full extent of MeHg exposure essentially assuming no masking by PUFA.

Prior to presentation of the function and inputs below for modeling MI-mortality associated with MeHg exposure in the general fish-consuming U.S. population we provide additional explanation for how the confidence cutpoints are integrated into the calculation of population-level risk since there is some complexity associated with this process. Taking the cutpoint of 0.66 μg/g hair-Hg, conceptually what we are doing is using this value to (a) identify the fraction of the U.S. population that will be included in risk modeling (*i.e.*, the percentile of the NHANES distribution of hair-Hg falling above this exposure level) which sets what fraction of the IHD mortality baseline rate that will be in play and (b) utilizing this cutpoint value (0.66 μg/g) as the representative exposure level for that sub-population being modeled for MI-mortality risk. For example, as described below under the *inputs* discussion, the 0.66 μg/g confidence cutpoint corresponds roughly to the upper 25th percentile of the NHANES hair-Hg distribution and consequently, we will be modeling risk for the upper 25th percentile of the U.S. population. That means that the baseline IHD mortality rate will be multiplied by 0.25 to reflect the fact that only 25% of the U.S. population is included as a subgroup in this incarnation of the risk modeling (since only the upper 25th percentile of the NHANES hair-Hg levels exceed the confidence cutpoint of 0.66 μg/g).

We do note that there are complex temporal aspects to the estimation of MI mortality incidence including, in particular, the timeframe for changes in Hg emissions to be reflected in the bioaccumulation of MeHg in commercial fishing stocks, which would dictate the appropriate year to use for baseline health incidence rates for MI mortality across the U.S. population. However, giving the bounding nature of these estimates, we have adopted the simplifying approach of not attempting to incorporate this temporal element into the generation of quantitative estimates and instead will address it as a potential source of uncertainty discussed qualitatively.

Function (total MI mortality):

MI_mortality_US-pop = Fatal_MI ((1-e$^{(-EE_HgMI*Hair_Hg_pop)}$)*F_Uspop_atrisk

Functional form: log-linear as suggested by Roman 2011 based on the results of the EURAMIC and KIHD studies (Roman et al., 2011)

Inputs:

(Note: the variables *Hair_Hg_pop* and *F_Uspop_atrisk* are themselves potentially dependent on the confidence cutpoint value for MI mortality referenced earlier. For that reason, the confidence cutpoint value(s) are also described below.)

MI_mortality_US-pop: estimates of total Hg-related MI mortality for the general U.S. population associated primarily with fish consumption – ***CALCULATED***.

Fatal_MI: annual incidence of fatal MI in the U.S. adult population that are 45+ years old (CDC Wonder, Number of circulatory system deaths-ischemic heart disease-acute MI, 2016). ***Value: 110,000***

EE_HgMI: effect estimates relating hair-Hg to MI mortality (with 95th percentile confidence intervals).[7] This effect estimate was obtained from the KIHD study (Virtanen et al., 2005) and covers a broad category of acute cardiovascular events. It was assumed that that effect would hold across all contributing categories including fatal MIs and therefore could be used without modification to estimate MI mortality incidence. The Relative Risk value of 1.11 (converted to an effect estimate of 0.1) was selected because of emphasis placed in Roman 2011 on the Hg-MI relationship since it covered acute MI (relative to the other endpoints evaluated in Virtanen et al., 2005). We do acknowledge a number of uncertainties associated with this effect estimate including: a) this is a non-U.S. cohort (*i.e.*, cross-country differences in access to healthcare, exposure to other air pollutants, diet, etc.), b) the cohort is comprised of older men who have not previously suffered a cardiovascular event (we are applying it to a broader age-range of adults, which introduces uncertainty) and c) the baseline death rate used in the study does not distinguish between first onset and repeat onset of cardiovascular event (*i.e.*, it may over-count the number of Hg-attributable deaths by an unknown amount). ***Value: 0.1 (95% CI: 0.06 to 0.16)***

Confidence cutpoint for MI mortality: The derivation of this factor (which impacts input parameters as referenced below) has been discussed at length earlier in this section. However, as a summary we note that these confidence cutpoints are intended to define ranges of MeHg exposure above which we have increased confidence in specifying the nature of the relationship between MeHg and MI-mortality and below which we have reduced confidence, due potentially to masking by heart-protective agents including PUFA. In specifying the confidence cutpoints we used recommendations from Roman et al., 2011 that mean hair-Hg levels from the two study populations be used as prospective thresholds in modeling MI-mortality (0.66 and 1.9 µg/g hair-Hg). We also note, that as discussed earlier, these confidence cutpoints are supported by consideration for other evidence found in the literature including the presence of J-shaped functions (for MeHg-cardiovascular effects) with inflection points within the 0.66 to 1.9 µg/g hair-Hg range and evidence that the PUFA cardiovascular benefit is only seen at lower levels of PUFA

[7] Note, the 95th percentile confidence intervals here refer to the statistical fit of the effect estimate. These are distinct from the confidence cutpoints described earlier that are used to address potential masking of the MeHg effect by heart-protective agents including PUFA.

intake, suggesting that masking of the MeHg effect could be limited to those lower levels of exposure. As noted above, the confidence cutpoints are used to inform two input parameters used in the estimation of MI-mortality for the general U.S. population including: (a) to identify the fraction of the U.S. population that will be modeled for the endpoint (*i.e.*, F_USpop_atrisk – see below) and (b) to specify the representative exposure level for each modeled population (Hair_Hg_pop – see below). As noted earlier, as part of the bounding analysis, we also included a scenario assuming there was no confidence-cutpoint and the MI mortality effect could be estimated across the full range of exposure (*i.e.*, for the full U.S. population of fish consumers).

F_Uspop_atrisk: Fraction of the U.S. population that are at risk for MI mortality due to Hg exposure through fish consumption. If we are not applying the confidence cutpoint for MI mortality, then this value is 1.0. However, if we are applying one of the confidence cutpoints (0.66 or 1.9 µg/g hair-Hg) then, as described earlier, we use these values to query the NHANES dataset characterizing hair-Hg levels in women of childbearing age (1999-2000) (McDowell et al., 2004 Table 2) to identify the closest reported percentile of the NHANES distribution corresponding to that cutpoint. MI mortality would then only be modeled for the percent of the population above that cutpoint (*i.e.*, for that fraction of MI baseline mortality). For example, with the confidence cutpoint of 1.9 µg/g, we see in Table 2 of McDowell et al., 2004 that this corresponds roughly to ~95th percentile of the NHANES distribution. Therefore, for the 1.9 cutpoint, we would set F_USpop_atrisk at 5% or 0.05 (*i.e.*, approximately the upper % of the U.S. population is being modeled for MI mortality). With the 0.66 µg/g confidence cutpoint we would estimate that the upper 25% or 0.25 would be modeled for MI mortality. Note that this approach assumes that we can use this NHANES dataset for women of childbearing age to represent hair-Hg levels for all U.S. adults 45+ years of age which does introduce uncertainty into the analysis, as discussed below. **Values: 1.0, 0.25. 0.05 (for no-cutpoint, an 0.66, or 1.9 µg/g hair-Hg confidence cutpoints).**

Hair_Hg_pop: Hair-Hg exposure level (mean or representative metric) for the population being modeled. This variable is also based on the nature of the confidence cutpoint being applied. If we are not applying a confidence cutpoint, then we use the mean hair-Hg level from the 1999-2000 CDC NHANES data for women of childbearing age as reported in McDowell et al., 2004 (Table 2) (0.47 µg/g hair-Hg). However, if we are using confidence cutpoints then we simply use that cutpoint value as the Hair_Hg_pop value (*i.e.*, 0.66 µg/g for the 0.66 cutpoint and 1.9 µg/g for the 1.9 cutpoint). Theoretically we would want to use the mean hair-Hg value for the segment of the population associated with the upper percentile being modeled (*e.g.*, for the 0.66 µg/g confidence cutpoint, since this maps to the upper 25th percentile of the NHANES distribution then we would ideally want to get the mean for that upper 25th percentile subgroup of women). However, we do not have the underlying distributional data for NHANES and so we simply used the confidence cutpoints themselves to represent the exposure levels for each group (this will likely bias the risk estimates low since these bounding values are by definition lower than the means of the population percentiles they specify). **Values: 0.47, 0.66, 1.9 (µg/g hair-Hg for no, 0.66 and 1.9 confidence cutpoints)**.

Step 2. Estimation fraction of MeHg-related MI mortality risk/benefit attributable to U.S. EGUs

Now that we have an estimate of the total Hg-related MI mortality for the general U.S. population, reflecting primarily fish consumption (Step 1 above), next we estimate the total fraction of that MI mortality attributable to U.S. EGU Hg. As noted earlier, rather than utilizing a complex mechanistic approach to link U.S. EGU-sourced Hg through to individual regional fisheries and then to consumer market-basket fish MeHg and U.S. population exposure, we have adopted a simpler bounding analysis approach. This bounding analysis involves generating two MI mortality estimates including (a) lower-bound estimates based on the assumption that U.S. EGU-sourced Hg becomes part of a global Hg pool which eventually determines the fraction of MeHg in commercial fish and consequently the impact to U.S. populations through commercial fish consumption (*i.e.*, fraction U.S. EGU Hg emissions relative to total global emissions determines fraction of total Hg-related MI mortality attributable to U.S. EGU emissions) and (b) upper-bound estimates based on the assumption that nearer-field U.S. EGU Hg impacts (continental U.S. and near-coastal fisheries) drive U.S. commercial fish exposure and consequently average U.S. EGU deposition over the continental U.S. (as a fraction of total Hg deposition over the same area) determines the fraction of total Hg-related MI mortality attributable to U.S. EGU-emissions. These bounding approaches are described in greater detail below.

Function (U.S. EGU-attributable MI mortality):

US EGU-MI_mortality_US-pop = MI_mortality_US-pop*F_fishHg_US EGU

Inputs:

MI_mortality_US-pop: generated in Step 1 above (**Calculated**)

F_fishHg_US EGU: Fraction of MeHg in consumed fish that is sourced from U.S. EGUs (this translates into the fraction of total Hg-related MI mortality attributable to U.S. EGUs). Two values were developed for this input—an upper and lower bound:

- *Lower-bound*: reflects the assumption that U.S. EGU-emitted Hg becomes part of a global pool that impacts global and regional fisheries equally (*i.e.*, F_fishHg_US EGU equals ratio of total U.S. EGU emitted Hg (annual) relative to total global annual Hg emissions including natural and anthropogenic). This approach makes a number of simplifying assumptions including (a) all forms of Hg emissions (including natural, legacy and anthropogenic) will have similar unit effects on MeHg concentrations in the fish that the U.S. population consumes and (b) that emissions from all sources will be evenly mixed and distributed globally such that they have a uniform (undifferentiated) impact on all sources of fish that the U.S. population consumes including self-caught and purchased (to the extent that those sources of fish drive the hair-Hg levels seen in the U.S. population as characterized using the NHANES data referenced earlier). Total U.S. EGU annual emissions for the U.S. EGU sector following CAA regulations but prior to implementation of MATS is ~29 tons (MATS REA, 2011). The 2015 update to the 2013 AMAP/UNEP Hg Modeling

Document (AMAP/UNEP, 2015, Table A1) provides three estimates for total world-wide Hg emissions (natural & legacy and anthropogenic): 5,535 tons (Chemical Institution Environment Canada) 6,945 (Massachusetts Institute of Technology) and 5,870 (Meteorological Synthesizing Center) – the average of these is ~6,100. Given the 29 tons for U.S. EGUs, this results in a **F_fishHg_US EGU of 29/6,100 = 0.48% or 0.0048**.

- *Upper-bound*: reflects the assumption that fish consumption by the general U.S. population is driven by domestic sources of fish impacted by nearer-field U.S. EGU impacts (*i.e.*, sources of fish within the continental U.S. and along the Atlantic and Pacific coastal seaboards) and that fish sourced from more distant fisheries (*e.g.*, northern Atlantic, Asian-Pacific) have a minimal impact on hair-Hg levels in the general U.S. population. For that reason, we will consider the average of U.S. EGU Hg deposition over the continental U.S. relative to total all-source Hg deposition (for that same U.S. continental area) as defining F_fishHg_US EGU. As for the lower-bound estimate, this estimate of deposition (within the U.S.) reflects modeling of 29 tons for U.S. EGUs. The data necessary to calculate this value are presented in the REA supporting the A&N determination (U.S. EPA, 2011a Table 2-1). Mean U.S. EGU Hg deposition is 0.34 $\mu g/m^2$ while total Hg deposition is 18.7 $\mu g/m^2$ which results in a **F_fishHg_US EGU of 0.34/18.7 = 1.8% or 0.018**.

iii. Results

Table 1 presents the results of the analysis for MI-mortality associated with U.S. EGUs. Those parameters that were varied in generating the range of estimates forming the bounding assessment are also identified (*i.e.*, assumptions made regarding the linkage of U.S. EGU Hg to fish MeHg and consideration for a confidence cutpoint in modeling MI mortality). The confidence interval for each bounding estimate reflects application of the 95^{th} percentile confidence interval for the effect estimate used in modeling MI-mortality risk. To reiterate, these estimates are annual risk estimates based on U.S. EGU Hg emissions prior to implementation of MATS (*i.e.*, 29 tons) with the assumption that that level of Hg release would continue in subsequent years. Furthermore, these estimates are bounding in nature and intended to provide an order-of-magnitude estimate for potential MI-mortality in the general U.S. population associated with U.S. EGU-sourced Hg resulting primarily from fish consumption.

Table 1. Range of U.S. EGU-attributable annual MI-mortality risk (deaths) estimated for the general U.S. population exposed primarily through fish consumption

Field	Range of bounding estimates (with 95th percentile confidence intervals)*					
	LOW			HIGH		
U.S. EGU linkage to fish MeHg	Assuming consumption from all (global) fisheries			Assuming consumption solely from U.S. continental and near coastal fisheries		
MI confidence cutpoint	1.9 µg/g hair-Hg Cutpoint	0.66 µg/g hair-Hg Cutpoint	None**	1.9 µg/g hair-Hg Cutpoint	0.66 µg/g hair-Hg Cutpoint	None**
U.S. EGU-attributable excess MI mortality risk (annual deaths)	5 (3 to 7)	8 (5 to 13)	24 (15 to 38)	17 (11 to 26)	32 (19 to 50)	91 (55 to 143)

*95th percentile confidence intervals for each bounding estimate reflect application of the 95th percentile confidence interval for the MI-mortality effect estimate obtained from Virtanen et al., 2005 (for additional detail see Step 1 above).
** No cutpoint applied – MI mortality modeled for the total general U.S. population.

iv. Discussion of Uncertainty and Overall Confidence

The assessment of Hg-related MI mortality for the general U.S. population is subject to a number of uncertainties including:

- *Support for modeling the MI mortality endpoint*: while we have not attempted to make a definitive determination regarding the causal association between MeHg exposure (through fish consumption) and MI-related mortality, we have considered the literature addressing this health endpoint and in particular, the results of EPA's 2010 workshop on the subject as reported in Roman, et al., 2011 as well as studies published subsequently. Recommendations from that workshop included the development of a CR function relating MeHg exposures with MIs for use in the regulatory context. That recommendation was based on consensus that there was a plausible link between MeHg exposure and MI with the support for that linkage being "moderate to strong", although it was noted that ambiguity regarding the causal mechanism does introduce uncertainty in this assessment of causality. Consideration for these observations together with studies published subsequently (*e.g.*, Hu et al., 2021) have resulted in our concluding that there is sufficient confidence in the linkage between MeHg and MI mortality to conduct a risk assessment. While we have concluded that there is reasonable support for this health endpoint, we do acknowledge uncertainty associated with the specification of its CR function as noted below, resulting in the development and application of our confidence cutpoint approach.
- *Specifying CR function (accounting for the cardioprotective agent PUFA):* Potential uncertainty associated with specifying the CR function for MI-mortality related to MeHg exposure has been addressed in this analysis through (a) incorporation of the 95th percentile for the effect estimate (reflecting statistical fit of the function) into the generation of bounding estimates and (b) application of confidence cutpoints to address potential masking of the MeHg effect by the heart-protective agent PUFA. The latter approach involving confidence cutpoints has been extensively discussed above and consequently will not be expanded on here. We do recognize additional sources of uncertainty associated with applying the KIHD-based effect estimate in modeling risk for the general U.S. population, as noted earlier, including: a) this is a non-U.S. cohort (*i.e.*, cross-country differences in access to healthcare, exposure to other air pollutants, diet,

etc.), b) the cohort is comprised of older men who have not previously suffered a cardiovascular event (we are applying it to a broader age-range of adults, which introduces uncertainty) and c) the baseline death rate used in the study does not distinguish between first onset and repeat onset of cardiovascular event (*i.e.*, it may over-count the number of Hg-attributable deaths by an unknown amount).

- *Utilizing NHANES data for women of childbearing age (1999-2000) to represent hair-Hg levels for the general U.S. population in 2016*: We recognize several sources of uncertainty associated with extrapolating the NHANES dataset to represent the general U.S. population including age extrapolation (women 16-49 years used for 45+), gender extrapolation (women used for total population coverage) and difference in time period (1999-2000 for 2016 simulation period). While NHANES has not continued to collect hair-Hg data in subsequent years since the NHANES dataset referenced here (otherwise we would have preferentially utilized those data), they have continued with blood-Hg monitoring and consequently those more recent data can be used to get a sense for potential bias associated with using the NHANES data for women of childbearing age (1999-2000) to cover 45+ adults in 2016. Specifically, we reviewed summary data for 2011-2016 from the CDC's Fourth National Report on Human Exposure to Environmental Chemicals (tables for blood-total Hg 2003-2016 - CDC, 2019) which provide time trend data and also allow question regarding gender extrapolation to be considered (note, readily available trend data did not provide results for different adult age ranges, limiting our ability to address potential bias in extrapolating from younger age (female) adults to an older total population). Review of the trend data in these tables suggest that: (a) overall Hg levels in blood appear to be decreasing over time (suggesting that we may have biased our risk estimate high since as MeHg levels are reduced in the general population, the confidence cutpoints will identify smaller percentiles of the population for risk modeling above those cutpoints), and (b) men tend to have higher Hg levels in blood relative to women suggesting that we have may biased our analysis low when using NHANES data for women of childbearing age to represent men. Overall, while we recognize these sources of uncertainty, it is not possible at this time to readily characterize the overall impact on our analysis. However, in our estimation, these sources of uncertainty linked to the use of the NHANES data for women of childbearing age would likely not compromise the policy-relevant utility of these risk estimates given their goal of generating an order-of-magnitude MI mortality estimate for U.S. EGU-source MeHg.
- *Linking U.S. EGU-sourced Hg to MeHg in commercial/recreational fish and resulting exposure to general U.S. population*: We acknowledge uncertainty (and the technical challenges) associated with modeling the multi-step process linking U.S. EGU Hg emissions to levels of MeHg in fish consumed by the general U.S. population. For that reason, we have attempted to develop a bounding strategy for tackling this issue that is intended to capture the range of potential linkages (in terms of quantitative magnitude) between U.S. EGU Hg emissions and impacts on MeHg within the fish consumed by the general U.S. population (sourced from both nearer-field domestic and international sources of fish).
- *Temporal aspects related to analysis*: the temporal dimension associated with estimating changes in MI mortality in the general U.S. population of fish consumers resulting from

changes in U.S. EGU Hg emissions is complex. Not only does this reflect the temporal dimensions of atmospheric transport, deposition, methylation and biomagnification (which could take up to years – Giang and Selin, 2016) but also the temporal dimensions associated with the link between changes in MeHg exposure and MI mortality risk. However, given the bounding nature of this analysis, we have elected not to explicitly account for the temporal dimensions in our modeling of risk. We recognize that this introduces uncertainty into the risk assessment since the longer the temporal response time (between changes in U.S. EGU Hg emissions and related changes in MI mortality mediated through general population fish consumption) the greater the degree of uncertainty in specifying input factors such as baseline incidence for MI mortality (for the general U.S. population).[8] Furthermore, as noted earlier, we acknowledge the fact that our characterization of baseline hair-Hg levels using NHANES reflects a 1999-2000 survey date which may not be fully representative of conditions in 2016, which have likely come down since then (*i.e.*, may have high-biased our risk estimates).

Acknowledging uncertainty associated with attempting to generate a single high confidence estimate for this health endpoint, we have instead, developed a range of risk estimates including reasonable high and low bounds. This range reflects consideration for uncertainty in two key inputs including (a) the linkage between U.S. EGU Hg emissions and changes in exposure to MeHg for the general U.S. population primarily associated with fish consumption and (b) characterization of the relationship between population-level exposure to MeHg and the incidence of MI-related mortality (including complexities resulting from co-exposure to heart-protective agents including PUFA). While we acknowledge a range of uncertainties including those documented above, we do believe that the range of risk estimates generated provides reasonable order-of-magnitude estimates for MI mortality in the general U.S. population associated with U.S. EGU Hg emissions (reflecting the 29 tons Hg emitted prior to implementation of MATS).

b. Risk of IQ loss for children associated with the general U.S. population and exposed to U.S. EGU Hg through maternal consumption

i. Overview

This analysis estimates the incidence of IQ loss in children associated with the general U.S. population resulting from maternal consumption of fish containing MeHg originally emitted from U.S. EGUs (with subsequent prenatal exposure to that MeHg). The approach used in modeling this health endpoint shares several elements with the approach described above for modeling MI mortality in the general U.S. population, including in particular, the method used to apportion the total MeHg-related health burden to the fraction associated with U.S. EGU Hg emissions (*i.e.*, the application of the bounding analysis based on consideration for the U.S. EGU fraction of total Hg deposition over the continental U.S. and the fraction of total global Hg emissions attributable to U.S. EGUs). Other elements of the modeling approach including specification of the number of children born annually within the U.S., the specification of baseline hair-Hg levels (utilizing NHANES data) and the characterization of the linkage between

[8] Note, that while we have not considered ecological- or health-related lags in modeling MI mortality risk, as discussed further in section d below, when converting these MI-mortality risk estimates into equivalent (2016) dollar benefits, we have incorporated a 10-year lag (reflecting the ecological response of MeHg in fish) for purposes of discounting those future monetary benefits.

MeHg exposure (prenatal) and IQ loss, are based on methods used to estimate the incidence of IQ loss from freshwater fish consumption by recreational anglers in the original 2011 benefit-cost analysis completed for MATS (U.S.EPA, 2011b).[9]

ii. Technical Approach

The presentation of equations, inputs and rationale is divided into two components (a) the modeling of total MeHg-related IQ loss in children associated with the general U.S. population and (b) estimation of the fraction of that risk attributable to U.S. EGU Hg emissions.

Step 1. Estimation of MeHg-related IQ loss for children associated with the general U.S. population exposed primarily through fish consumption

The approach used here is to (a) identify the numbers of children born annually within the U.S., (b) characterize the mean Hg-hair level for women of child-bearing age within the U.S. utilizing CDC NHANES data (this representing the maternal hair-Hg exposure associated with the mothers of children in "a" above) and (c) apply a CR function linking the magnitude of hair-Hg in mothers to IQ loss in children to generate a total estimate of IQ loss in U.S. children associated with MeHg exposure. There is greater support in the literature for neurodevelopmental effects related to MeHg exposure relative to cardiovascular effects (Roman et al., 2011) and for that reason, we have concluded that the estimates of IQ loss described here are associated with a greater degree of confidence than the MI mortality estimates presented in the previous section. Furthermore, while there is the potential for PUFA and selenium to confound the impact of MeHg on IQ loss, we have elected not to attempt to address these confounders given overall uncertainty associated with that effort, as discussed at length in the 2011 regulatory impact analysis, RIA, (section 4.6.5) and will instead address that source of uncertainty qualitatively.

Function (total IQ loss):

IQ_loss_children_US-pop = Total_US_children*Hair_Hg_maternal*EE_IQ_loss

Functional form: linear (Rice et al., 2010, U.S.EPA, 2011b)

Inputs:

IQ_loss_children_US-pop: estimates of total IQ loss in children associated with the general U.S. population as a result of prenatal exposure to MeHg- ***CALCULATED***.

[9] Regarding the focus of the 2011 MATS benefit-cost analysis on freshwater fish consumption (by recreational anglers), the reader is referred to the 2011 MATS RIA for a full explanation, but in summary, EPA had access to a large amount of measured Hg fish tissue data, which, when combined with the ability to estimate recreational fishing activity at waterbodies associated with those measured fish tissue data, supported the generation of risk (and hence dollar benefits) with a relatively greater overall degree of confidence. By contrast, at the time, EPA concluded that efforts to model other exposure scenarios of potential interest (*e.g.*, commercial fish consumption by the general U.S. population), would be subject to substantial uncertainty, although as noted earlier, recent developments have now allowed us to generate screening-level bounding estimates for these general consumption scenarios, covering both MI mortality in adults and IQ loss in infants exposed in-utero to MeHg. In addition, we recognize the potential for other neurodevelopmental effects potentially associated with pre-natal exposure to Hg besides IQ, however we believe that IQ represents an endpoint with clear public health significance and an endpoint that can also be modeled for Hg with a reasonable degree of confidence. For that reason, we have focused exclusively on this endpoint in modeling neurodevelopmental effects for children born to mothers within the general U.S. population.

Total_US_children: number of U.S. births (2016) U.S. Census (CDC, 2017)). ***Value: 3,941,109***

Hair_Hg_maternal: Hair-Hg exposure level (mean or representative metric) for women of childbearing age in the U.S. Utilized the mean hair-Hg level from the 1999-2000 CDC NHANES data women of childbearing age as reported in McDowell et al., 2004 (Table 2) (0.47 μg/g hair-Hg). **Values: 0.47 (μg/g hair-Hg)**.

EE_IQ_loss: effect estimates relating maternal hair-Hg to IQ loss in children resulting from prenatal exposure. We have utilized the same Axelrad 2007 value as employed in the 2011 MATS benefit-cost analysis (U.S.EPA, 2011b), the Rice et al., 2010 analysis and more recently in the analysis completed by Giang and Selin (2016) (see original documents for additional discussion). We note the potential for co-exposure to PUFA and selenium and that some published studies have attempted to adjust for the potential masking of the MeHg-related IQ impact (*e.g.*, application of a 1.5 adjustment factor by Rice et al., 2010 and Giang and Selin, 2016). However, given the subjective nature associated with specifying this type of adjustment factor, rather than attempting that form of quantitative adjustment, we have instead addressed this potential masking of the IQ loss impact qualitatively in our discussion of uncertainty below. ***Value: 0.18 IQ points/Hair-Hg (ppm) (95%CI: 0.009 to 0.378).***

Step 2. Estimation fraction of MeHg-related IQ loss (in children) attributable to U.S. EGUs

As noted above, the approach utilized to estimate the fraction of total IQ loss (for children associated with the general U.S. population) attributable to U.S. EGUs is the same as that employed above for MI-mortality, including the application of two bound estimates for fractional apportionment.

Function:

US EGU_IQ_loss_children_US-pop = IQ_loss_children_US-pop*F_fishHg_US EGU

Inputs:

IQ_loss_children_US-pop: generated in Step 1 above (**Calculated**)

F_fishHg_US EGU: Described above under MI-mortality (Step 2).

iii. Results

Table 2 presents the results of the analysis for annual IQ loss in children associated with U.S. EGUs. Those parameters that were varied in generating the range of estimates forming the bounding assessment are also identified (*i.e.*, assumptions made regarding the linkage of U.S. EGU Hg emissions to fish MeHg). To reiterate, these estimates are based on the estimated Hg emissions in 2016 from U.S. EGUs (*i.e.*, 29 tons), which is prior to implementation of MATS with the assumption that that level of emission would then continue producing the level of annual IQ loss reported here. As noted below, there is uncertainty in these annual estimates, since there would be a lag period before they would be fully realized for any given year's Hg

emissions. For comparison purposes, the 2011 MATS RIA (Table 4-7, U.S. EPA, 2011b) estimated a total of 697 IQ points for children associated with recreational freshwater anglers for the 2016 zero out of U.S. EGUs (also reflecting ~29 tons Hg emissions).[10]

Table 2. U.S. EGU-attributable IQ points lost annually for children associated with the general U.S. population and exposed prenatally to MeHg through U.S. fish consumption

Field	Range of bounding estimates (including 95th percentile confidence intervals)*	
	LOW	**HIGH**
U.S. EGU linkage to fish MeHg	Assuming consumption from all (global) fisheries	Assuming consumption solely from U.S. continental and near coastal fisheries
U.S. EGU-attributable annual IQ loss for children from maternal fish consumption	1,600 (80 to 3,400)	6,000 (300 to 12,600)

* Parenthetical range reflects 95th percentile confidence interval for the CR function (effect estimate) utilized in modeling IQ loss as applied to each of the bounding scenarios.

iv. Discussion of Uncertainty and Overall Confidence

The assessment of Hg-related IQ loss in children associated with the general U.S. population is subject to a number of uncertainties including:

- *Support for modeling the IQ loss endpoint*: the 2011 MATS RIA includes an extensive discussion of the use of the IQ loss endpoint in risk (and benefits) analysis, including a summary of comments provided by the SAB in their review of the 2011 draft MATS risk assessment (MATS RIA section 4.6.1, U.S.EPA, 2011b). While recognizing that there are a number of neurodevelopmental endpoints associated with MeHg exposure that are more sensitive than IQ loss, the SAB supported the use of IQ loss in conducting quantitative risk (and benefits) analysis because of the degree of overall support for this endpoint. Furthermore, previous analyses conducted by EPA (U.S.EPA, 2011b) and studies in the published literature (Giang and Selin, 2016) have noted the considerable degree of support for the IQ loss endpoint.

[10] The bounding analysis described here generates estimates of total U.S. EGU-sourced IQ loss for children associated with the general U.S. population. In reality, this IQ loss is distributed across the population of children covered by the analysis in some fashion. However, given variation in key factors related to this exposure scenario including (a) spatial patterns of U.S. EGU Hg fate and transport (including deposition and methylation) and (b) variations in fish consumption by mothers (including differences in daily intake, types of fish consumed and geographical origins of that fish), it is likely that modeled IQ loss may not be uniformly distributed across the population of exposed children and may display considerable heterogeneity. However, without conducting more sophisticated probabilistic exposure modeling, it is not possible to characterize potential variation in degree of IQ loss per child associated with U.S. EGU-sourced Hg and for that reason, EPA feels that the estimates of total IQ loss (as presented) is appropriate for informing consideration of potential public health hazard.

- *Specifying CR function (accounting for PUFA and selenium):* While there have been a number of effect estimates advanced for characterizing the relationship between MeHg exposure and IQ loss in children, we have elected to use the same value utilized in the 2011 MATS RIA (*i.e.*, the Axelrad, 2007 value). After review and discussion, that same value was used in more recent published studies (Giang and Selin, 2016). And while efforts have been made to address concerns for the masking of the IQ loss impact by PUFA and/or selenium through the use of subject adjustment factors (Rice, 2010, Giang and Selin, 2016) we have elected to not attempt that form of adjustment, instead recognizing the potential that our IQ loss estimates may be biased low due to this masking phenomenon.
- *Linking U.S. EGU-sourced Hg to MeHg in commercial/recreational fish and resulting exposure to general U.S. population*: This source of uncertainty has been addressed above as part of the discussion of the MI-mortality endpoint (those observations apply for the IQ-loss endpoint discussed here).
- *Temporal aspects related to analysis*: the estimation of IQ loss in children (for the general U.S. population) resulting from U.S. EGU-sourced Hg emissions is subject to the same temporal complexities associated with linking those facility changes in emissions to down-stream changes in concentrations of MeHg in commercially-sourced fish as encountered in modeling MI mortality risk. However, given the more clearly delineated nature of the prenatal pathway associated with IQ loss relative to the more temporally complex nature of the MI-mortality pathway, we believe there is less uncertainty associated with characterizing the temporal aspects of the IQ loss pathway. Given the bounding nature of this risk analysis, we have not attempted to incorporate consideration of temporality into the modeling of this pathway. However, as with the MI mortality pathway, we acknowledge the fact that our characterization of baseline hair-Hg levels (in this case for women of child-bearing age) using NHANES reflects a 1999-2000 survey date may not be fully representative of conditions in 2016.[11]

As with the MI-mortality endpoint, rather than attempting to generate a single high-confidence risk estimate, we have developed bounding risk estimates, reflecting in this case, consideration for uncertainty in linking changes in U.S. EGU-sourced Hg to changes in population-level exposure to Hg through fish consumption. However, unlike the bounding analysis for MI-mortality, we have not attempted to address potential neurological protective agent (*e.g.*, PUFA, selenium) confounding of the MeHg effect through the use of confidence cutpoints, for the reasons given earlier.

c. Assessment of the potential for increased MI mortality risk for subsistence fishers exposed to U.S. EGU-sourced Hg at freshwater waterbodies within the continental U.S.

i. Overview

This analysis of increased MI-mortality risk is an extension of the subsistence-fisher based at-risk watershed analysis originally completed as part of the 2011 risk assessment

[11] Note, however that, as with MI-mortality, when we convert these IQ loss estimates into dollar benefits (in section d below) we do incorporate a 10-year lag for the response of fish tissue MeHg levels to allow for discounting of future monetary benefits.

supporting the appropriate and necessary determination (U.S.EPA, 2011a – see that technical support document for additional detail regarding risk modeling). In that original analysis, a series of subsistence fisher risk scenarios was evaluated for a subset of 3,141 freshwater watersheds (lakes, streams and rivers) within the continental U.S. for which there were sampled MeHg fish tissue data (that fish tissue data allowed for a higher-confidence empirically-based assessment of MeHg risk to be generated for those watersheds). For each watershed, we used those fish tissue MeHg data to characterize total Hg-related risk[12] and then we estimated the portion of that total risk attributable to U.S. EGUs (based on the fraction of total Hg deposition to each of those modeled watersheds associated with U.S. EGU emissions). Watersheds were assessed to be "at risk" if the U.S. EGU attributable exposure alone resulted in the subsistence fisher scenario having a hazard quotient greater than 1.0 (*i.e.*, U.S. EGU-attributable MeHg exposure exceeding the MeHg reference dose (RfD)).[13] Those RfD-based risk estimates continue to provide valuable risk information informing the appropriate and necessary determination but are now supplemented by the MI-mortality based analysis discussed here.

We have now extended the at-risk watershed analysis completed in 2011 for the subsistence fisher scenarios to include an assessment of the potential for increased MI mortality risk. Specifically, we have utilized the U.S. EGU-attributable MeHg exposure estimates (ug/kg-day MeHg intake) generated for each watershed (for various subsistence fisher ingestion rates as discussed earlier) to generate equivalent hair-Hg exposure estimates for each watershed. We then compare those hair-Hg levels to the confidence cutpoints developed for the MI mortality risk assessment described in Section I above. If the hair-Hg level for a particular watershed is above either the EURAMIC or KIHD confidence cutpoint (*i.e.*, above 0.66 and 1.9 μg/g hair-Hg, respectively), then we consider that watershed to be at increased risk for MI mortality related to that U.S. EGU-attributable MeHg exposure. Note, that this is not to suggest that exposures at watersheds where U.S. EGU-attributable hair-Hg is below these confidence cutpoints are without risk, but rather that when exposure levels exceed these cutpoints, we have increased confidence in characterizing risk of MI mortality were we to do so.[14] It is also important to note that in most cases, total MeHg exposure is likely to exceed these confidence cutpoints such that in reality (due again to total Hg exposure) subsistence fishers active at those watersheds would likely exceed these confidence cutpoints for modeling MI mortality when total Hg exposure is considered.

In order to summarize these estimates of increased MI mortality risk, we have used the same percentile approach as originally used in the 2011 REA summarizing HQs for the

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[12] Risk was evaluated by first estimating a daily intake of MeHg (ug/kg-day) by combining the watershed-level MeHg fish tissue value with a specific percentile consumption rate for the subsistence fisher scenario (*i.e.*, mean, 90th, 95th, or 99th percentile). That MeHg intake range was then compared against the RfD for methylmercury thereby generating a hazard quotient (HQ). An HQ greater than 1.0 suggests that exposure exceeds the RfD, which is intended to represent a level of exposure without appreciable harm. Regarding the subsistence fisher fish consumption rate percentiles – these were selected to provide coverage for upper-end (*i.e.*, elevated) exposure given the health hazard (screening) nature of this analysis.

[13] The at-risk determination could also result from a watershed having a total risk (from all global anthropogenic and natural Hg deposition) that exceeded an HQ of 1.0 combined with a significant (*e.g.*, >5%) contribution of U.S. EGUs to that total risk. However, in assessing the potential for increased MI-mortality risk, we have elected to only focus on the U.S. EGU-attributable exposure in order to focus on instances where U.S. EGU-sourced Hg alone pushes MeHg exposure above the confidence cutpoints.

[14] Note that because we are not able to enumerate subsistence fishers at each watershed, it is not possible to model MI mortality risk (incidence).

subsistence fisher scenarios evaluated (see Table 2-6, U.S.EPA, 2011a). In other words, we have identified specific percentile watershed fish tissue MeHg concentration levels corresponding to specific percentile self-caught fish consumption rates (for each subsistence fisher scenario). So, for example, the highest U.S. EGU-attributable hair-Hg estimate for each subsistence fisher type in Table 3 would correspond to the 99th percentile watershed fish tissue value associated with the 99th percentile fish consumption rate (this representing the watershed with the highest MeHg fish tissue concentration combined with the highest subsistence fisher consumption rate).

The 2011 MATS REA analysis estimates HQ values for subsistence fisher scenarios for both a general subsistence fisher scenario and for a number of additional subsistence fisher scenarios dimensioned on race/cultural practice, geographic location and socioeconomic status (poverty). These additional scenarios provide coverage for subsistence fisher populations that are of concern from an environmental justice standpoint given that their increased self-caught fish consumption rates reflecting race/cultural practices and/or poverty could result in increased exposure and risk relative to the general population. Details regarding the modeling of these subsistence fisher populations including the consumption rates utilized are presented in section 1.4.3 of the 2011 REA (U.S.EPA, 2011a). In generating estimates of increased MI mortality risk associated with U.S. EGU-sourced Hg we have included not only the original subsistence fisher scenario, but also the additional subsistence populations included in the original 2011 MATS REA.

ii. Technical Approach

The conversion of exposure estimates generated for each subsistence fisher (in ug MeHg/kg body weight per day or ug/kg-day) into equivalent hair-Hg levels involves a straightforward calculation utilizing two ratios obtained from the literature including Hg blood-intake coefficient and a Hg hair-blood coefficient (it is also necessary to include a body weight adjustment using 64 kg to get total MeHg intake which is needed for application of the first coefficient listed above).

Function (conversion of daily intake into equivalent hair-Hg):

Hair-Hg = Daily MeHg intake*body weight*Hg_blood-intake coefficient*Hg_hair-blood coefficient

Functional form: linear (Rice, 2010, U.S.EPA, 2011a)

Inputs:

Hair-Hg: estimate of hair-Hg level associated with U.S. EGU contributions to MeHg daily intake - ***CALCULATED***.

Daily_MeHg_intake: generated as part of the original watershed-level subsistence fisher risk assessment (we utilize the U.S. EGU-attributable component of total MeHg exposure as reflected in the watershed-level RfD-based HQ estimates presented in Table 2-6 of the 2011 REA (U.S.EPA, 2011a). **Value: variable (calculated by watershed in 2011 REA) µg/kg-day.**

Body weight: Representative adult body weight for women of child-bearing age used in generating watershed-level HQ risk estimates for the 2011 REA (U.S.EPA, 2011a).[15] **Value: 64 kg**.

Hg_blood-intake coefficient: ratio of Hg in blood (µg/L) to total Hg intake (µg/day). Derived by parameterizing a simple one-compartment physiologically-based pharmacokinetic model that estimates an equilibrium blood Hg concentration given a specified Hg intake. Of the four parameters utilized by the model, the most uncertain value is time, or the biological half-life of MeHg (Rice, et al., 2010). **Value: 0.6 µg/L per µg/day.**

Hg_hair-blood coefficient: ratio of Hg in hair (µg/g) to Hg in blood (µg/L). Specification of this parameter is based on an assessment of empirical data from 10 studies and is considered to be fairly robust (Rice et al., 2010). **Value: 0.18 µg/g hair to µg/L blood**.

iii. Results

Table 3 presents the results of the analysis of increased risk for MI-mortality for the subsistence fisher scenarios. As noted earlier, these results are dimensioned on two key parameters (self-caught fish consumption rate and watershed-level hair-Hg exposure). Those watershed percentile hair-Hg values that exceed the EURAMIC-based MI mortality confidence cutpoint (0.66 µg/g hair-Hg) are shaded in the table and those cells that also exceed the KIHD-based MI mortality confidence cutpoint (1.9 µg/g hair-Hg) are bolded. Once again, these cutpoints identify levels of MeHg exposure (hair-Hg) associated with a clear association with MI-related health effects (*i.e.*, increased risk).[16] As with the other risk estimates presented for MeHg, these estimates reflect the baseline for U.S. EGUs prior to implementation of MATS (*i.e.*, 29 tons). In addition, these results only reflect U.S. EGU-sourced Hg (*i.e.*, whether U.S. EGU-attributable hair-Hg alone for the modeled subsistence fisher populations exceeds the confidence thresholds). We recognize that in many cases, total MeHg exposure (*i.e.*, EGU contribution plus contributions from other sources) may exceed these confidence cutpoints such that subsistence fishers active at those watersheds would be at increased risk of MI mortality at least in part due to EGU emissions.

The pattern of hair-Hg exposure estimates (and hence increased MI-mortality risk) across subsistence fisher scenarios presented in Table 3 deserves additional discussion. Hair-Hg levels vary considerably across subsistence fisher scenarios with results for low-income Black subsistence fishers in the southeast being notably higher than the other six subsistence fisher scenarios (matched on watershed percentile and fish consumption rate percentile). For instance, we see that the 99th percentile watershed hair-Hg level for low-income Black subsistence fishers in the southeast based on the 95th percentile fish consumption rate (yielding a hair-Hg estimate of 5.81 µg/g) is more than three times larger than similar estimates for most of the other scenarios,

[15] Because the original 2011 MATS risk assessment calculation focused on women of child-bearing age in assessing risk, exposure estimates for MeHg for the 2011 analysis (as daily intake rates adjusted for bodyweight) utilized the average bodyweight for women of childbearing age (64 kg) in that calculation (EPA Exposure Factors Handbook, U.S. EPA, 1997). Therefore, in generating equivalent hair-Hg levels for use in this screening analysis, we needed to covert these daily (body-weight adjusted) intake rates into total daily intake by multiplying the earlier 2011 estimates by 64 kg (the body weight used in the original calculation.

[16] More specifically regarding these confidence cutpoints, they identify a degree of exposure above which we have increased confidence in specifying the nature of the relationship between exposure and adverse MI-related response. While we believe there is support for MI-related effects at exposures below these confidence cutpoints, there is greater uncertainty in attempting to characterize the nature and magnitude of that risk.

the exception being Laotian-American subsistence fishers where the low-income Black subsistence fishers in the southeast are still estimated to have an exposure level more than double the Laotian level.

Differences in exposures presented in Table 3 across subsistence fisher scenarios reflect two factors: (a) the self-caught fish consumption rates which are presented in the first column of Table 3 and can be seen to reflect considerable variation and (b) spatial variation in the pattern of U.S. EGU Hg-impacted watershed-level fish tissue concentrations used in generating these exposure estimates and in particular, the intersection of regions of activity for the various subsistence fisher scenarios and areas of elevated (or reduced) U.S. EGU Hg-impacted fish tissue concentrations. Regarding "a" (the self-caught fish consumption rates), as discussed in detail in Section 1.4.3 of the 2011 MATS REA (U.S. EPA, 2011a), while the same survey of individuals attending the Palmetto Sportsmen's Classic in Columbia South Carolina in 1998 was used to characterize fish consumption rates for the first three subsistence fisher scenarios in Table 3 (typical subsistence fish consumer, low-income Black fishers in the southeast and low-income white fishers in the southeast), there are concerns over low sample size with this survey which introduces uncertainty into the upper percentile consumption rates for these three populations. However, if viewed more as a characterization of high-end consumption patterns (rather than a formal characterization of population percentiles) implemented in the context of a screening-level risk assessment which is the context here, then this concern over the stability of upper percentile consumption rates is ameliorated to some extent. It is worth noting that the surveys utilized in characterizing fish consumption rates for the other subsistence fisher scenarios are likely of higher quality, although still subject to specific uncertainties (as discussed in section 1.4.3 of the 2011 MATS REA, U.S. EPA, 2011a).

Potentially more important in the context of understanding the pattern of exposure levels presented in Table 3 across subsistence fisher scenarios (and in particular the high values for low-income Black subsistence fishers active in the southeast) is the fact that U.S. EGU-sourced Hg fish tissue concentrations in the southeast and in particular South Carolina are substantially higher than in regions associated with other subsistence scenarios (*e.g.*, California for the Laotian and Vietnamese and the northern Great Lakes for the Tribal scenario). This can be seen by consulting a number of figures in the 2011 MATS REA (U.S. EPA 2011a) depicting patterns in U.S. EGU-Hg deposition (Figure 2-4) and associated patterns in U.S. EGU-Hg impacted fish tissue concentrations (Figure 2-14). The figures referenced here in the 2011 MATS REA also illustrate variation in the coverage of areas of the U.S. (including different areas of the southeast) by measured fish tissue concentration data which can also impact modeling of subsistence fisher exposure and risk, with the potential for data-rich areas (such as South Carolina) driving risk profiles for specific subsistence fisher scenarios. While we recognize the uncertainties related to both fish consumption surveys and the fish tissue Hg data utilized in this risk assessment, we feel that given the screening-nature of this analysis, these sources of uncertainty do not compromise its potential utility in helping to characterize the potential for health hazards associated with U.S. EGU-sourced Hg in the subsistence fisher populations considered here.

Table 3. Identification of watersheds (and associated subsistence fisher scenarios) potentially at increased risk of MI-mortality due to U.S. EGU-sourced Hg in self-caught fish

Subsistence fisher scenario (self-caught fish consumption rate in g/day)	**Watershed percentile exposure level (U.S. EGU-attributable hair-Hg µg/g) with values exceeding MI-mortality confidence cutpoints flagged**				
	50th	**75th**	**90th**	**95th**	**99th**
Typical subsistence fish consumer assessed nationally					
39 (mean)	NA	0.07	0.14	0.14	0.28
123 (90th)	0.14	0.21	0.35	0.48	0.83
173 (95th)	0.14	0.28	0.48	0.69	1.18
373 (99th)	0.35	0.62	1.04	1.45	**2.56**
Low-income White subsistence fish consumer in the southeast					
39 (mean)	0.07	0.07	0.14	0.21	0.41
93 (90th)	0.14	0.21	0.35	0.48	0.97
129 (95th)	0.14	0.28	0.55	0.69	1.38
286 (99th)	0.35	0.69	1.18	1.59	**2.97**
Low-income Black subsistence fish consumer in the southeast					
171 (mean)	0.21	0.41	0.69	0.97	1.80
446 (90th)	0.55	1.11	**1.87**	**2.49**	**4.70**
557 (95th)	0.62	1.31	**2.35**	**3.04**	**5.81**
Low-income Hispanic subsistence fish consumer evaluated nationally					
26 (mean)	<0.01	0.07	0.07	0.14	0.21
98 (90th)	0.07	0.14	0.28	0.41	0.76
156 (95th)	0.14	0.28	0.48	0.62	1.18
Vietnamese-American subsistence fish consumer					
27 (mean)	<0.01	<0.01	0.07	0.07	0.21
99 (90th)	0.07	0.14	0.21	0.35	0.69
152 (95th)	0.14	0.21	0.35	0.55	1.04
Laotian-American subsistence fish consumer					
47 (mean)	0.07	0.07	0.14	0.21	0.62
145 (90th)	0.14	0.21	0.41	0.62	1.80
226 (95th)	0.21	0.35	0.83	1.18	**3.39**
Tribal (near Great Lakes) subsistence fish consumer					
62 (mean)	0.07	0.07	0.14	0.21	0.28
136 (90th)	0.14	0.21	0.28	0.41	0.62
213 (95th)	0.21	0.28	0.48	0.62	0.90
493 (99th)	0.41	0.69	1.04	1.45	**2.14**

iv. Discussion of Uncertainty and Overall Confidence

The assessment of the potential for increased risk of MI-mortality for subsistence fishers related to U.S. EGU-sourced Hg exposure is subject to a number of uncertainties. Many of these sources of uncertainty relate to the method used in modeling exposure for these subsistence scenarios at the watershed-level and have been extensively discussed in the 2011 MATS REA (see section 2.7 and in particular Table 2-15 of that document, U.S.EPA, 2011a). Key sources of uncertainty identified in that earlier discussion include:

- *Characterizing the pattern of fish tissue Hg concentrations across watersheds in the U.S.*: reflects small fraction of total watersheds with fish tissue measurement data, differences in state-level protocols for collection of fish tissue data, substantial number of watersheds with relatively low sample sizes (*e.g.*, 1-2 samples), uncertainty associated with filtering watersheds to exclude locations with potentially significant non-air Hg impacts.
- *Characterizing subsistence fishing activity across areas of high U.S. EGU Hg deposition:* includes assessing potential locations for subsistence fishing activity as well as details regarding actual fishing and fish consumption behavior (note, as discussed in the 2011 REA, demographic data were used to support an assessment of the potential for activity by specific subsistence fisher populations at specific watersheds, even if it was not possible to enumerate those populations). One important caveat is that we did not complete a rigorous literature review to determine whether subsistence fisher self-caught consumption rates had change since development of the original 2011 REA. However, as part of internal EPA peer review, an EPA ORD 2013 report on fish consumption in Connecticut, Florida, Minnesota and North Dakota was brought to our attention (EPA 2013). While the volume of summary fish consumption data presented in the report (specifically tables in section E.12. Additional Details for Targeted Populations) is extensive, our assessment of general trends in the 2013 document suggests that it is generally supportive of the high-end percentile subsistence fisher consumption data we used in the 2011 REA and in the current updated to that analysis. Specifically, in the 2011 REA, we see that the 90th to 99th percentile consumption rates across the subsistence fisher groups range roughly from 100 to 500 g/day which is similar to the ranges seen in the upper percentiles for the 2013 EPA document. Specifically, regarding Tribal populations, we do recognize challenges in obtaining high-end consumption rates for Tribal populations active in areas of high U.S. EGU impact (*e.g.*, Ohio River valley, areas of the central southeast such as northern Georgia, northern South Carolina, North Carolina and Tennessee – see Figure 2-3, 2011 REA. U.S. EPA 2011a) there is the potential for our analysis of Tribal-associated risk to have missed areas of elevated U.S. EGU-source Hg exposure and risk. In that case, estimates simulated for other subsistence populations active in those areas (*e.g.*, poor whites and Blacks in the southeast as reported here in Table 3) could be representative of the ranges of risk experienced by Tribal populations to the extent that their cultural practices result in similar levels of increased fish consumption.
- *Application of the proportionality assumption in linking U.S. EGU-sourced Hg deposition (as a fraction of total Hg deposition) to U.S. EGU-attributable fish tissue data for a given watershed*: reflects the application of the Mercury-maps assumption that fractional changes in Hg deposition will ultimately be reflected as fractional changes in fish tissue MeHg. A number of sources of uncertainty can impact this assumption

including differences in methylation rates and in particular temporal factors related to methylation.

- *Factors relating to the estimation of Hg deposition over watersheds using the Community Multiscale Air Quality, CMAQ, model:* including estimating Hg emissions from U.S. EGUs and other sources, chemistry associated with Hg fate and transport, prediction of wet and dry deposition, and global inflow of Hg into the U.S.

In addition to the above factors related to uncertainty in the original analysis of risk for the subsistence fisher scenarios as presented in the 2011 MATS REA, there are several additional sources of uncertainty related in particular to the assessment of increased risk for MI-related mortality. These include in particular, uncertainty associated with the hair-Hg to blood-Hg coefficient for which the specification of the half-life of MeHg in the body has already been called out as subject to greater uncertainty (see above). Derivation and use of the two confidence cutpoints for MI-mortality (based on the EURAMIC and KIHD studies) has been discussed earlier in Section I above.

While there are a number of sources of uncertainty associated with generating these estimates of increased MI mortality risk at the watershed level, given that the analyses are based on measured fish tissue data linked to detailed CMAQ results and given that we are assessing the potential for increased risk (rather than explicitly attempting to model that MI-mortality risk), we think overall confidence associated with this assessment is reasonable.

d. Estimated economic value of avoided cases of fatal Acute Myocardial Infarctions and additional IQ Points among infants in the general U.S. population attributable to U.S. EGU-sourced Hg through fish consumption

This section presents the estimated screening-level economic value of two benefit categories experienced by the general U.S. population exposed to U.S. EGU-sourced Hg primarily through fish consumption: (a) avoided Acute Myocardial Infarction AMI-related mortality in adults (described in section "a" above) and (b) additional IQ points among infants via reduced maternal exposure (described in section "b" above). The approach used to value these two health endpoints draws heavily on methods provided in supporting documents including the Technical Support Document (TSD) for the Final Revised Cross-State Air Pollution Rule Update for the 2008 Ozone Season NAAQS (U.S. EPA, 2021) for MI mortality and the 2011 MATS RIA (U.S. EPA, 2011b) for IQ points lost.

When quantifying each benefit category, we assume a lag between the ecological response of commercial fish to changes in U.S. EGU-sourced Hg deposition over fisheries. This lag is subject to considerable uncertainty with research suggesting that response times can range from years to decades (Giang and Selin, 2016). The lag time is particularly important when estimating the economic value of each endpoint, as it affects the future year in which estimated benefits will begin to accrue; the present value of benefits falls as they are experienced further into the future. Here we use a ten-year ecological response lag when estimating the value of each benefit category, matching the core value used in Giang and Selin, 2016.[17] We estimate each

[17] Note that the MI-mortality endpoint could experience an additional lag associated with the health effect itself (*i.e.*, time required for changes in methylmercury intake to be experienced as changes in the risk of fatal MI events). However, given the uncertainty associated with mechanisms associated with Hg-mediated MIs (Roman et al., 2011),

benefits category for a 2016 analysis year, since this is the point at which MATS was to be fully implemented. We estimate benefits for a future year of 2026 to account for the ten-year ecological lag, discounting these values back to 2016 using a 3 and 7% discount rate. The benefits estimates presented here reflect the 29-ton increment utilized in generating the underlying risk estimates and are annual.

i. Estimated value of avoided cases of Myocardial Infarction

We estimate the economic value of avoided cases of MI using a Value of Statistical Life (VSL) employed elsewhere by EPA (U.S. EPA, 2021). Specifically, we apply an estimated VSL for the projected year of 2026. That VSL of $10.7 million is adjusted for an inflation year of 2016 and projected income growth to the year 2026. We next multiplied the VSL by the estimated count of avoided premature deaths to estimate the economic value of the avoided cases in 2026. Finally, we discounted this value back to the year 2016 using a 3% and 7% discount rate, consistent with OMB Circular A-4 (Table 4). Note that benefits estimates for the core bounding values (5 and 91 deaths) are bolded, while estimates for the lower 5th percentile and upper 95th percentile MI mortality estimates (3 and 143 deaths) are unbolded.

Table 4. Benefit estimates for reduction in MI-mortality for the general U.S. population due to U.S. EGU-sourced Hg exposed primarily through fish consumption (2016$) *

Estimated MI-Deaths (2026)	Estimated Economic Value of Avoided Cases Discounted from 2026 to 2016 (2016$)	
	7%	3%
3	16,000,000	24,000,000
5	**27,000,000**	**40,000,000**
91	**490,000,000**	**720,000,000**
143	780,000,000	1,100,000,000

* Values rounded to two significant figures

These estimated benefits are subject to a number of uncertainties including those associated with quantifying avoided MI-attributable deaths (as described in section "a" above), including:

- *The lag associated with the response of MI-mortality risk to changes in MeHg accessed through fish consumption.* A non-zero lag would imply that MI-related benefits would be deferred further into the future and thus the discounted value would be smaller than those reported in Table 4.
- *The ecosystem lag.* The ecosystem lag associated with the response of commercial fish to changes in Hg deposition is subject to uncertainty.

we have not attempted to specify a lag for this element of the risk calculation and instead, treat it qualitatively as a source of uncertainty. IQ-loss in infants impacted through maternal Hg exposure is not subject to the same uncertainty regarding a health-effect lag.

- *Uncertainties related to the estimated VSL*. The VSL is subject to uncertainty described in detail elsewhere (U.S. EPA, 2021).

While there are a range of potential uncertainties impacting the benefit estimates for MI-mortality, given the sensitivity (bounding) nature of these estimates, they are considered sufficiently robust to provide an illustrative order-of-magnitude range of the economic value of potential impacts.

ii. Value of IQ points gained

Our approach for valuing additional IQ points among children exposed in-utero to MeHg utilizes the same approach employed in 2011 MATS RIA (U.S. EPA, 2011b). Specifically, additional IQ is translated into a monetary gain reflecting additional future income, net of the cost of additional years of education associated with the cognitive gain using evidence from two studies (Schwartz, 1994 and Salkever, 1995) (see section 4.7.3, MATS RIA, U.S. EPA, 2011b). The estimated future income increases over the course of a lifetime and is discounted (using both a 3 and 7% discounting rate). Combining the two study-related values for this input with the discounting of future income streams with both a 3 and 7% discounting rate, results in four values for use in translating IQ loss into reductions in future income streams including: $11,859 and $1,958 per IQ point (Salkever, 3% and 7% discount rates, respectively) and $8,013 and $893 per IQ point (Schwartz, 3% and 7% discount rates, respectively) (for additional detail on the derivation of these four values see section 4.7.3 of the 2011 MATS RIA (U.S. EPA, 2011b).

However, as with the MI-mortality benefit estimates, we assume a 10 year ecological lag for the commercial fish MeHg levels to respond fully to changes in U.S. EGU Hg emissions. Consequently, the IQ loss benefit estimates calculated using these valuation factors must in turn be discounted again using both 3% and 7% discount rates to adjust the 2026 benefit estimate to represent the 2016 analysis year. The resulting benefits estimates for IQ loss (for the 2016 analysis year) are presented below in Table 5.

Table 5. Estimated benefit estimates for IQ points gained among children associated with the general U.S. population due to U.S. EGU-sourced Hg exposed through maternal fish consumption (2026$) [A]

Estimated IQ points lost (2026)	Schwartz (7% discount on earning stream: $893/IQ point)		Salkever (7% discount on earnings stream: $1,958/IQ point)		Schwartz (3% discount on earning stream: $8,013/IQ point)		Salkever (3% discount on earnings stream: $11,859/IQ point)	
	Value Discounted from 2026 to 2016							
	7%	3%	7%	3%	7%	3%	7%	3%
80	36,000	53,000	80,000	120,000	330,000	480,000	480,000	710,000
1600	**730,000**	**1,100,000**	**1,600,000**	**2,300,000**	**6,500,000**	**9,500,000**	**9,600,000**	**14,000,000**
6000	**2,700,000**	**4,000,000**	**6,000,000**	**8,700,000**	**24,000,000**	**36,000,000**	**36,000,000**	**53,000,000**
12600	5,700,000	8,400,000	13,000,000	18,000,000	51,000,000	75,000,000	76,000,000	111,000,000

[A] Values rounded to two significant figures

The benefits estimates presented in Table 5 are subject to a number of sources of uncertainty including those associated with the estimation of IQ points lost (as described in section "b" above), including:

- *Projecting the long-term effect of IQ loss on lifetime earnings*. To remain consistent with methods employed in the 2011 MATS RIA, we use evidence reported in two studies to

quantify the effect of changes in IQ on lifetime earnings (Schwartz et al. 1994; Salkever et al. 1995). Newer literature may more reliably characterize the relationship between IQ changes in lifetime earnings. Additional sources of uncertainty associated with modeling IQ loss impacts on lifetime earnings are discussed in section 4.8.5.5 of the 2011 MATS RIA, US EPA 2011b.

- *Estimating the ecosystem lag*. The estimated lag describing the response of commercial fish to changes in Hg deposition is also subject to uncertainty.

Similar to the MI-mortality benefit estimate, while there are a range of potential uncertainties impacting the benefit estimates for IQ points lost, given the sensitivity (bounding) nature of these estimates, they are considered sufficiently robust to provide a reasonable order-of-magnitude range.

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