UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

EPA-SAB-20-xxx

The Honorable Andrew R. Wheeler
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Subject: Science Advisory Board (SAB) Consideration of the Scientific and Technical Basis of EPA's Proposed *Mercury and Air Toxics Standards for Power Plants Residual Risk and Technology Review and Cost Review*

Dear Administrator Wheeler:

As part of its statutory duties, the EPA Science Advisory Board (SAB) may provide advice and comment to you on the scientific and technical basis of certain planned EPA actions. The Environmental Research, Development, and Demonstration Authorization Act of 1978 (ERDDAA) requires the agency to make available to the SAB proposed criteria documents, standards, limitations, or regulations provided to any other federal agency for formal review and comment, together with relevant scientific and technical information on which the proposed action is based. The SAB may then provide advice and comments on the adequacy of the scientific and technical basis of the proposed action.

In April 2019, the SAB Work Group on Planned Actions for SAB Consideration of the Underlying Science evaluated the proposed *Mercury and Air Toxics Standards For Power Plants Residual Risk and Technology Review and Cost Review* and indicated that it ranked "high" on four criteria used by the SAB for determining whether an action merits review: "Addresses areas of substantial uncertainties," Involves major environmental risks," "Relates to emerging environmental issues," and "Exhibits a long-term outlook." The Work Group noted that the proposed action was based on a Residual Risk and Technology Review (RTR) that was conducted using a prescribed methodology, and on consideration of whether the cost of compliance was reasonable when weighed against the health benefits of the proposed rule. The Work Group recommended that the SAB review the cost finding and whether the RTR methodology had been correctly applied in this case. At its public meeting on June 5-6, 2019 the SAB elected to review the scientific and technical basis of the proposed rule. This letter provides the SAB findings and recommendations.

General Comments

The updated methodologies used by EPA to perform the Residual Risk Assessment for the Coal- and Oil-Fired Electric Utility Steam Generating Units (EGU) Source Category are detailed in the report *Screening Methodologies to Support Risk and Technology Reviews (RTR): A Case Study Analysis* (U.S. EPA 2017)[1]. The SAB's review of this case study analysis was published in 2018 (U.S. EPA Science Advisory Board 2018)[2] and is particularly relevant to the Residual Risk Assessment for the Coal- and Oil-Fired EGU Source Category assessment. However, SAB's recommendations do not seem to have been taken into consideration in the published analysis. To ensure conclusions drawn from the Residual Risk Assessment for the Coal- and Oil-Fired EGU Source Category benefit from SAB's most recent technical peer that promotes increased transparency and inclusion of new science, the EPA is encouraged to review the SAB's findings and determine what revisions are warranted.

Assessment of Methylmercury Exposure

EPA's residual risk assessment appears only to include fish consumed from small to mid-size lakes by fishermen and their families. While it is conservative in the assumption of fish consumption by the subsistence fisher (373 g/d, 99th percentile ingestion rate) (Burger, 2002 cited in U.S. EPA 2018),[3,4] this is only a small fraction of fish consumed in the United States. Estimates indicate that estuarine and marine fish and shellfish comprise of over 90% of the market share of commercial fish (Carrington et al. 2004).[5] Even though >80% of fish is imported, there is still an appreciable quantity from the Atlantic and Pacific regions (Karimi et al. 2012).[6] Many of the species of marine fish eaten by Americans do spend large parts of their life in U.S. domestic waters (Sunderland et al. 2016).[7] Further, there are higher levels of mercury in Atlantic than Pacific fish, which may help explain higher mean and 90th percentile blood

[1] U.S. EPA. 2017. *Screening Methodologies to Support Risk and Technology Reviews (RTR): A Case Study Analysis.* Office of Air Quality Planning and Standards, Office of Air and Radiation, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina. [Available at: https://yosemite.epa.gov/sab/sabproduct.nsf//LookupWebProjectsCurrentBOARD/2708C2DBC839301685258060005C87E8/$File/Screening+Methodologies+to+Support+RTRs_A+Case+Study+Analysis.pdf]

[2] U.S. EPA Science Advisory Board. 2018. *SAB Review of EPA's draft technical report entitled Screening Methodologies to Support Risk and Technology Reviews (RTR): A Case Study Analysis*. U.S. EPA Science Advisory Board, Washington, D.C. [Available at: https://yosemite.epa.gov/sab/sabproduct.nsf/0/7A84AADF3F2FE04A85258307005F7D70/$File/EPA-SAB-18-004+.pdf]

[3] Burger, J. 2002. Daily consumption of wild fish and game: Exposures of high end 24 recreationists. *International Journal of Environmental Health Research* 12(4): 343-354

[4] U.S. EPA 2017. Screening Methodologies to Support Risk and Technology Reviews.

[5] Carrington, C.D., B Montwill, and P.M. Bolger. 2004. An intervention analysis for the reduction of exposure to methylmercury from the consumption of seafood by women of child-bearing age. *Regulatory Toxicology and Pharmacology* 4:272-280.

[6] Karimi, R., T.P. Fitzgerald, and N.S. Fisher. 2012, A quantitative synthesis of mercury in commercial seafood and implications for exposure in the United States. *Environmental Health Perspectives* 120:1512-1519

[7] Sunderland, E., C. Driscoll, Jr., C.J. Hammitt,, P. Grandjean, J. Evans, J. Blum, C.Y. Chen, D.C. Evers, D. Jaffe, R. Mason, S. Goho, and W. Jacobs. 2016. Benefits of regulating hazardous air pollutants from coal and oil fired utilities in the United States. *Environmental Science and Technology* 50, 2117−2120. DOI: 10.1021/acs.est.6b00239.

concentrations among Atlantic coastal residents (2.7 and 7.7 µg/l) than values measured in Pacific coastal residents (1.7 and 4.7 µg/l) (Mahaffey 2005 cited in Sunderland 2007).[8,9] However, research suggests recent decreases in mercury emissions have resulted in declines in mercury concentrations in Atlantic coastal fish stocks (Cross et al. 2015).[10] Mercury levels in sediment cores in the Great Lakes have also shown decreases corresponding to reduced emission (Drevnick et al. 2012).[11] Similarly, declines in methylmercury concentrations in freshwater fish in the United States have been found corresponding to domestic mercury emissions reductions (Hutcheson et al. 2014).[12]

The U.S. Food and Drug Administration (FDA) and EPA jointly noted that, in a survey of over 1200 pregnant women in 2005, median fish consumption was just 1.8 ounces per week (79 FR 33559).[13] In that document, both agencies recommended that pregnant and potentially pregnant women *increase* consumption of a variety of fish lower in mercury to 8 to 12 ounces per week, within their calorie needs, because the net effects study showed that this that will facilitate neurological development in children. The report continued to recommend that woman of child-bearing age avoid certain fish with the highest mercury concentrations. However, an FDA presentation (Spiller 2012)[14] showed net benefits associated with consumption of up to 24.5 ounces of fish per week assuming the lower level of toxicity discussed. The calculations in the presentation indicated that there would be adverse effects beginning at this consumption level for several high mercury concentration fish species. For all people who report eating fish, the average reported in the Center for Disease Prevention and Control's National Health and Nutrition Examination Survey (NHANES) from 2005-2010 is about 5 ounces per week, although women typically eat less.[15]

In the 1999-2000 NHANES study, fish consumption was increasing while mercury/hair levels were decreasing.[16] A more recent study found, "On average, U.S. women of reproductive age

[8] Mahaffey, K.R.. 2005. NHANES 1990–2002 Update on Mercury. In: *Proceedings of the 2005 National Forum on Contaminants in Fish*, 18–21 September 2005, Baltimore MD. EPA-823-R-05-006.

[9] Sunderland, E. M. 2007. Mercury exposure from domestic and imported estuarine and marine fish in the U.S. seafood market. *Environmental Health Perspectives* 115 (2), 235−242.

[10] Cross, F. A., D.W. Evans, and R.T. Barber. 2015. Decadal declines of mercury in adult bluefish (1972−2011) from the mid-Atlantic coast of the U.S.A. Environmental Science and Technology 49:9064−9072.

[11] Drevnick, P. E., D.R. Engstrom, C.T. Driscoll, E.B. Swain, S.J. Balogh, N.C. Kamman, D.T. Long, D.G.C. Muir, M.J. Parsons, K.R. Rolfhus, and R. Rossmann. 2012. Spatial and temporal patterns of mercury accumulation in lacustrine sediments across the Great Lakes region. Environmental Pollution 161:252−260.

[12] Hutcheson, M. S., M.C. Smith, J. Rose, C, Batdorf, O. Pancorbo, C.R. West, J. Strube, and C. Francis. 2014. Temporal and spatial trends in freshwater fish tissue mercury concentrations associated with mercury emissions reductions. Environmental Science and Technology 48:2193−2202.

[13] EPA and FDA. 2014. *Environmental Protection Agency and Food and Drug Administration Advice About Eating Fish: Availability of Draft Update*. 79 FR 33559. Available at: https://www.federalregister.gov/documents/2014/06/11/2014-13584/environmental-protection-agency-and-food-and-drug-administration-advice-about-eating-fish

[14] Spiller. P. 2012. *New Advice for Eating Fish During Pregnancy and why We Are Proposing It*. http://jifsan.umd.edu/images/wordpress/2014/11/2014NutritionWebinarDay1_Spiller.pdf

[15] Jahns, Lisa, et. al. Intake of seafood in the U.S. varies by age, income, and education level but not by race-ethnicity. *Nutrients* 6(12) 2014.

[16] Centers for Disease Control and Prevention. 2019. National Health and Nutrition Examination Survey. https://www.cdc.gov/nchs/nhanes/index.htm

were consuming more fish and blood mercury levels were lower in 2009–2010 compared to 1999–2000."[17] The authors also state that "The current study observed that U.S. women of childbearing age who live in coastal regions consumed more fish per month and had higher whole blood Hg concentrations compared to women living in the Midwest after controlling for other confounders. In particular, women who lived in the Atlantic or Pacific coastal regions had the highest fish intake and the highest blood Hg concentrations." In the discussion section, the authors suggest that the decline in women's blood mercury levels may have been driven by changes in fish consumed, specifically, market shares for low-mercury varieties including shrimp, tilapia, salmon and catfish increased, while shares of high-mercury varieties decreased.

Neurodevelopmental Epidemiology and Mercury

Disentangling the neurological benefits of fish consumption from the adverse effects of mercury exposure provides challenges for epidemiology studies. The best studies account for the benefits of fish consumption when evaluating the negative impacts of mercury. Recent epidemiological findings indicate that there are more sensitive neurodevelopmental endpoints than full-scale IQ, as used by the EPA (Sunderland et al. 2016).[18] Further, these impacts have been documented at lower levels than the reference dose established by a National Research Council panel in 2000[19] (Sunderland et al. 2016; Bellanger et al. 2013).[20,21] Other recent reviews have begun to question whether there is evidence for a safe level of mercury exposure (Grandjean et al. 2012; Karagas et al. 2012).[22,23] Karagas et al. (2012)[24] find the strongest effects for multiple neurological impacts, including psychomotor function, memory, and verbal skills cognition at 3-6 years of age with prenatal mercury exposure. These results were found to be consistent among multiple prospective cohort studies that all accounted for fish consumption during pregnancy (Freire et al.

[17] Cusack, L.K., E. Smit, M.L. Kile, and A.K. Harding. 2017. Regional and temporal trends in blood mercury concentrations and fish consumption in women of child bearing age in the united states using NHANES data from 1999-2010. *Environ Health*. 2017;16(1):10. doi:10.1186/s12940-017-0218-4. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5316155/

[18] Sunderland, E., C. Driscoll, Jr., C.J. Hammitt,, P. Grandjean, J. Evans, J. Blum, C.Y. Chen, D.C. Evers, D. Jaffe, R. Mason, S. Goho, and W. Jacobs. 2016. Benefits of regulating hazardous air pollutants from coal and oil fired utilities in the United States. *Environmental Science and Technology* 2016, 50, 2117−2120. DOI: 10.1021/acs.est.6b00239.

[19] National research Council. 2000. *Toxicological Effects of Methylmercury*. National Academy Press, Washington, D.C.

[20] Sunderland et al. 2016. Benefits of regulating hazardous air pollutants from coal and oil fired utilities in the United States.

[21] Bellanger, M., C. Pichery, D. Aerts, M. Berglund, A. Castano, M. Cejchanova, P. Crettaz, F. Davidson, M. Esteban, M.E. Fischer, A.E. Gurzau, K. Halzlova, A. Katsonouri, L.E. Knudsen, M. KolossaGehring, G. Koppen, D. Ligocka, A. Miklavcic, M.F. Reis, P. Rudnai, J.S. Tratnik, P. Weihe, E. Budtz-Jorgensen, and P. Grandjean. 2013. Economic benefits of methylmercury control in Europe: Monetary value of neurotoxicity prevention. *Environmental Health* 12(3)1-10. DOI: 10.1186/1476-069X-12-3.

[22] Grandjean, P., C, Pichery, M. Bellanger, and E. Budtz-Jorgensen. 2012. Calculation of mercury's effect on neurodevelopment. *Environmental Health Perspectives* 120 (12), A452.

[23] Karagas, M. R., A.L. Choi, E. Oken, M. Horvat, R. Schoeny, E. Kamai, W. Cowell, P. Grandjean, and S. Korrick. 2012. Evidence on the human health effects of low-level methylmercury exposure. *Environmental Health Perspectives* 120 (6), 799−806.

[24] Ibid.

2010; Lederman et al. 2008; Oken et al. 2008).[25,26,27] However, it is important to account for fish consumption because the beneficial aspects of fish consumption appear to offset the adverse impacts of mercury (Karagas et al. 2012; Stewart et al. 2003; Jedrychowski et al. 2007).[28,29,30] Although effects at other ages were inconclusive, looking instead by effect at all age groups found many domains to consistently be the most sensitive (Karagas et al. 2012);[31] specifically, memory (Freire et al. 2010; Oken et al. 2005; Weil et al. 2005)[32,33,34] and verbal or language skills (Freire et al. 2010; Lederman et al. 2008; Oken et al. 2008; Surkan et al. 2009).[35,36,37,38]

Despite the fact that it is important to account for the beneficial aspects of fish consumption, in the original Regulatory Impact Analysis for the Mercury and Air Toxics Standards conducted in December, 2011, EPA considered confounders, particularly long chain polyunsaturated fatty acids, but decided there was too much uncertainty in the data to incorporate this into their quantitative estimate of benefits.[39] Later, however, FDA was able to directly incorporate both beneficial effects of fish consumption along with negative health impacts. In 2018, EPA re-proposed this rule and divided benefits into direct and indirect categories. While the indirect benefits were primarily from reducing PM 2.5, the direct benefits were the target of the rule,

[25] Freire C, R. Ramos, M.J. Lopez-Espinosa, S. Díez, J. Vioque, F. Ballester et al. 2010. Hair mercury levels, fish consumption, and cognitive development in preschool children from Granada, Spain. *Environmental Research* 110(1):96–104.

[26] Lederman, S.A., R.L. Jones, K.L. Caldwell, V. Rauh, S.E. Sheets, D. Tang, et al. 2008. Relation between cord blood mercury levels and early child development in a World Trade Center cohort. *Environmental Health Perspectives* 116:1085–1091.

[27] Oken E, J.S. Radesky, R.O. Wright, D.C. Bellinger, C.J. Amarasiriwardena, K.P. Kleinman, H. Hu, and M.W. Gillman. 2008. Maternal fish intake during pregnancy, blood mercury levels, and child cognition at age 3 years in a US cohort. *American Journal of Epidemiology* 167(10):1171-81. doi: 10.1093/aje/kwn034. Epub 2008 Mar 1

[28] Karagas et al. 2012. Evidence on the human health effects of low-level methylmercury exposure.

[29] Stewart, P.W., J. Reihman, E.I. Lonky, T.J. Darvill, and J. Pagano. 2003. Cognitive development in preschool children prenatally exposed to PCBs and MeHg. *Neurotoxicology and Teratology* 25(1):11–22.

[30] Jedrychowski W, F. Perera, V. Rauh, E. Flak, E. Mroz, A. Pac et al. 2007. Fish intake during pregnancy and mercury level in cord and maternal blood at delivery: an environmental study in Poland. *International Journal of Occupational Medicine and Environmental Health* 20(1):31–37.

[31] Karagas et al. 2012. Evidence on the human health effects of low-level methylmercury exposure.

[32] Freire C, et al. 2010. Hair mercury levels, fish consumption, and cognitive development in preschool children from Granada, Spain.

[33] Oken E, R.O. Wright, K.P. Kleinman, D. Bellinger, C.J. Amarasiriwardena, H. Hu et al. 2005. Maternal fish consumption, hair mercury, and infant cognition in a U.S. cohort. *Environmental Health Perspectives* 113:1376–

[34] Weil, M., J. Bressler, P. Parsons, K. Bolla, T. Glass, and B. Schwartz. 2005.Blood mercury levels and neurobehavioral function. *Journal of the American Medical Association* 293 (2005), p. 1875,

[35] Freire C, et al. 2010. Hair mercury levels, fish consumption, and cognitive development in preschool children from Granada, Spain.

[36] Lederman et al. 2008. Relation between cord blood mercury levels and early child development in a World Trade Center cohort.

[37] Oken et al. 2008. Maternal fish intake during pregnancy, blood mercury levels, and child cognition at age 3 years in a U.S. cohort.

[38] Surkan P.J., D. Wypij, F. Trachtenberg, D.B. Daniel, L. Barregard, S. McKinlay et al. 2009. Neuropsychological function in school-age children with low mercury exposures. *Environmental Research* 109(6):728–733

[39] EPA, "Regulatory Impact Analysis for the Final Mercury and Air Toxics Standards, EPA-452/R-11-011, December 2011, p. 4-39.

reducing maternal exposure to methylmercury from recreationally self-caught freshwater fish. EPA mentioned, but did not quantify, other possible human and environmental benefits.

In 2009, FDA released a draft study of the net effects of eating fish and, five years later, in May, 2014, FDA released their final net effects quantitative risk analysis. This analysis examined the net effects of methylmercury and nutrients in fish like omega 3 fatty acids.[40] EPA was invited to comment on the net effects and did so – negatively.

Other Potential Health Endpoints for Mercury

Beyond neurological effects, other potential health endpoints from mercury include cardiovascular disease (Roman et al. 2011),[41] endocrine function (Tan et al. 2009),[42] risk of diabetes (He et al. 2013),[43] and impacts on immune function (Nyland et al. 2011; Karagas 2012).[44,45] The literature is the most developed for cardiovascular endpoints and thus only those are reviewed here.

In 2010, the U.S. EPA held a workshop to review the current science on cardiovascular impacts of methylmercury (MeHg) exposures. The assembled panel of scientists found "the body of evidence exploring the link between MeHg and acute myocardial infarction (MI) to be sufficiently strong to support its inclusion in future benefit analyses, based both on direct epidemiological evidence of an MeHg-MI link and on MeHg's association with intermediary impacts that contribute to MI risk" (Roman et al. 2011).[46] The two mechanisms with the strongest evidence for biological plausibility were oxidation and heart rate variability (HRV). There is consistent evidence in animal studies for MeHg-induced oxidative stress (Roman et al. 2011; Genchi et al. 2017).[47,48] Lipid peroxidation in rats has been shown to increase with MeHg

[40]FDA. 2014. A Quantitative Assessment of the Net Effects on Fetal Neurodevelopment from Eating Commercial Fish (As Measured by IQ and also by Early Age Verbal Development in Children), May 2014. https://www.fda.gov/media/88491/download

[41] Roman, H. A., T.L. Walsh, B.A. Coull, E. Dewailly, E. Guallar, D. Hattis, K. Mariën, J. Schwartz, A.H. Stern, J.K. Virtanen, and G. Rice. 2011. Evaluation of the cardiovascular effects of methylmercury exposures: Current evidence supports development of a dose− response function for regulatory benefits analysis. *Environmental Health Perspectives* 119 (5), 607−614. http://doi.org/10.1289/ehp.1003012.

[42] Tan, S. W. J.C. Meiller, and K.R. Mahaffey. 2009. The endocrine effects of mercury in humans and wildlife. Critical Reviews in Toxicology 39(3):228−269.

[43] He, K., P. Xun, K. Liu, S. Morris, J. Reis, and E. Guallar. 2013. Mercury exposure in young adulthood and incidence of diabetes later in life: the CARDIA trace element study. *Diabetes Care* 36:1584−1589.

[44] Nyland, J. F., M. Fillion, R. Barbosa, Jr., D.L. Shirley, C. Chine, M. Lemire, D. Mergler, E.K. Silbergeld. 2011. Biomarkers of methylmercury exposure and immunotoxicity among fish consumers in the Amazonian Brazil. Environmental Health Perspectives 119(12):1733− 1738.

[45] Kragas et al. 2012. Evidence on the human health effects of low-level methylmercury exposure.

[46] Roman et al. 2011. Evaluation of the cardiovascular effects of methylmercury exposures: Current evidence supports development of a dose− response function for regulatory benefits analysis.

[47] Ibid.

[48] Genchi, G., M.S. Sinicropi, A. Carocci, G. Lauria, and A. Catalano. 2017. Mercury exposure and heart diseases. *International Journal of Environmental Research and Public Health* 14(1). https://doi.org/10.3390/ijerph14010074.

(Huang et al.1996; Lin et al. 1996).[49,50] Increased production of malondialdehyde, a secondary product of lipid peroxidation, was found to increase in rats as a result of long-term, low-dose exposure to MeHg (Grotto et al. 2009).[51] In addition to the animal studies, the EPA panel identified two epidemiological studies that found evidence of MeHg exposure and oxidative stress (Salonen et al. 1995; Grotto et al. 2010).[52,53] The panel also found one study with contradictory findings. The study compared oxidized low density lipoprotein (LDL) among fishermen before and after sport fishing season, which resulted in an increased rate of consumption of both fish and mercury (Belanger et al. 2008).[54]

Decreased HRV is commonly found in those suffering from cardiovascular disease and is a predictor of cardiovascular mortality risk (Hattis 2003; Lahiri et al. 2008).[55,56] The EPA panel found strong evidence of decreased HRV with increased MeHg exposure from the epidemiological evidence (Roman et al. 2011).[57] This relationship was shown in three studies of various populations (Valera et al. 2008; 2009; 2010; Lim et al. 2009).[58,59,60,61] There is also an intervention study in which healthy Japanese adults were either assigned to an experimental group where they ate tuna and swordfish at Japan's provisionally tolerable weekly intake or a

[49] Huang, Y.L., S.L. Cheng, and T.H. Lin. 1996. Lipid peroxidation in rats administered with mercuric chloride. *Biological Trace Element Research* 52:193–206.

[50] Lin T.H., Y.L. Huang, and S.F. Huang. 1996. Lipid Peroxidation in liver of rats administered with methyl mercuric chloride. *Biological Trace Element Research* 54:33–41.

[51] Grotto D., M.M. de Castro, G.R. Barcelos, S.C. Garcia, and F. Barbosa, Jr. 2009. Low level and sub-chronic exposure to methylmercury induces hypertension in rats: nitric oxide depletion and oxidative damage as possible mechanisms. *Archives of Toxicology* 83(7):653–662. .

[52] Salonen, J.T. K. Seppanen, K. Nyyssonen, H. Korpela, J. Kauhanen, M. Kantola et al. 1995. Intake of mercury from fish, lipid peroxidation, and the risk of myocardial infarction and coronary, cardiovascular, and any death in eastern Finnish men. *Circulation* 91(3):645–655.

[53] Grotto D., J. Valentini, M. Fillion, C.J. Passo, S.C. Garcia. D. Mergler, et al. 2010. Mercury exposure and oxidative stress in communities of the Brazilian Amazon. Science of the Total Environment 408(4):806–811.

[54] Béllanger, M.C., M.E. Mirault, E. Dewailly, M. Plante, L. Berthiaume, M. Noël et al. 2008. Seasonal mercury exposure and oxidant-antioxidant status of James Bay sport fishermen*. Metabolism* 57:630–636.

[55] Hattis, D. 2003. The conception of variability in risk analyses— developments since 1980. In: *Risk Analysis and Society in the 21st Century: An Interdisciplinary Characterization of the Field* (McDaniels T, Small MJ, eds). Cambridge, U.K. Cambridge University Press, 15–45.

[56] Lahiri, M.K., P.J. Kannankeril, and J.J. Goldberger. 2008. Assessment of autonomic function in cardiovascular disease: physiological basis and prognostic implications. *Journal of the American College of Cardiology* 51:1725–1733.

[57] Roman et al. 2011. Evaluation of the cardiovascular effects of methylmercury exposures: Current evidence supports development of a dose− response function for regulatory benefits analysis.

[58] Valera B, E. Dewailly, and P. Poirier. 2008. Cardiac autonomic activity and blood pressure among Nunavik Inuit adults exposed to environmental mercury: a cross-sectional study. *Environmental Health* 7:29; doi:10.1186/1476-069X-7-29 [Online 6 June 2008].

[59] Valera B, R. Dewailly, and P, Poirier 2009. Environmental mercury exposure and blood pressure among Nunavik Inuit adults. *Hypertension* 54:981–986.

[60] Valera B, E. Dewailly, and P. Poirier. 2010. Impact of toxic metals on blood pressure, resting heart rate and heart rate variability in an aboriginal population of Quebec (Canada) (Abstract). In: *Proceedings of the Joint Conference—50th Cardiovascular Disease Epidemiology and Prevention– and–Nutrition, Physical Activity and Metabolism Conference*. San Francisco, California. 2–5 March 2010.

[61] Lim S, H-U Chung, and D. Paek. 2009. Low dose mercury and heart rate variability among community residents nearby to an industrial complex in Korea. *Neurotoxicology* 31:10–16.

control group (Yaginuma-Sakurai et al. 2009)[62] and HRV decreased significantly. The Roman et al. paper also summarized evidence for a number of other mechanisms, not summarized here. There was, however, a large study completed with two U.S. cohorts that did not find evidence of cardiovascular risk (Mozaffarian et al. 2011).[63] There were limitations associated with this study. Specifically, it included only low-to-moderate fish consumers and therefore did not include a wide range of exposures, making it difficult to detect any effects. Second, the study suffered from difficulties separating out the positive impact of consumption of long-chain fatty acids in fish (Sunderland et al. 2016).[64] This may suggest that, as with neurological effects, long-chain fatty acids neutralize the negative effects of methylmercury. To consider cardiovascular effects of fish consumption, it is necessary to consider fish as the relevant input so that the net effects of fish can be evaluated. It is also necessary to consider protein alternatives to fish as those alternatives may be worse for cardiovascular health.

Other Benefit-Cost Analyses for Mercury

Scientists have conducted a number of recent analyses of the monetized benefits of mercury reductions. A recent study determined that quantified monetized benefits of reductions in mercury emissions from coal and oil-fired utilities in the 2011 analysis conducted by the EPA understates actual benefits (Sunderland et al. 2016).[65] Three of the reasons were: (1) that the EPA only included mercury exposure through consumption of fish for a small population of recreational fishers, (2) that neurological outcomes actually can occur at a lower concentration than used by the EPA, and (3) that there are potentially other health outcomes that should be quantified by the EPA. A second study quantified cumulative U.S. economy-wide benefits and estimated them to be at least $43 billion (Giang and Selin, 2016).[66] A third study found that including cardiovascular risks from mercury in a cost-benefit assessment is critical, because a probabilistic assessment of the health and economic benefits from a reduction in mercury exposure found that 80% of the monetized health benefits come from reduction in fatal heart attacks, with the remainder coming from IQ gains (Rice et al. 2010).[67] Again, because the net health effects of fish should be the correct measure for a benefits assessment.

Uncertainty

[62] Yaginuma-Sakurai, K., K. Murata, M. Shimada, K. Naka, N. Kurokawa, S. Kameo S, et al. 2009. Intervention study on cardiac autonomic nervous effects of methylmercury from seafood. *Neurotoxicology and Teratology* 32:240–245.

[63] Mozaffarian D, P. Shi, J.S. Morris, D. Spiegelman, P. Grandjean, D.S. Siscovick et al. 2011. Mercury exposure and risk of cardiovascular disease in two U.S. cohorts. *New England Journal of Medicine* 364(12):1116–1125.

[64] Sunderland et al. 2016. Benefits of regulating hazardous air pollutants from coal and oil fired utilities in the United States.

[65] Sunderland et al. 2016. Benefits of regulating hazardous air pollutants from coal and oil fired utilities in the United States.

[66] Giang, A., and N.E. Selin. 2016. Benefits of mercury controls for the United States. *Proceedings of the National Academy of Sciences*. U.S.A. 113:286.

[67] Rice, G.E., J.K. Hammitt, and J.S. Evans. 2010. A probabilistic characterization of the health benefits of reducing methyl mercury intake in the United States. *Environmental Science and Technology* 44:5126–5224.

The SAB notes a number of uncertainties in EPA's analysis. The fisheries model is poor in that the size of the fish is not a variable. Mercury is cumulative. The modeling science is not adequate because so much is not known.

1. Is there is a nonlinear effect for mercury? It is possible that there is a level of exposure below which mercury has no effect. This is not known. It is also possible that there is a hormetic dose which should also be investigated. The usual assumption is that the effect is linear and can be interpolated from known data to levels below detectable levels. This assumption deserves to be tested.
2. The exposure model needs to take into account exposure to mercury from all sources such that reduced exposure to freshwater recreational fish accurately estimates the remaining mercury levels.
3. The exposure models need to account for the fact that methylmercury has been found in prehistoric fish from 2,000 years ago at levels at or above current levels.[68]
4. There are few good physically accurate models of mercury in the human body and even fewer of mercury in fish and land animals. This is a high priority research need.
5. Almost nothing is known about the toxicity of mixtures of pollutants. Emissions from power plants contain potential toxins, and it is not known how they interact with mercury. This is an important question.
6. Given that there appear to be net beneficial neurological effects from eating more, in fact a lot more fish, one key uncertainty is the level of mercury in freshwater, recreationally caught fish compared to fish sold in markets. It is known that there is mercury in large freshwater fish like largemouth bass, pickerel and some catfish, but the data are sparse.
7. The levels of beneficial fatty acids in recreational freshwater fish are also not known, however, given that women would have to increase consumption of fish, all fish, four to six-fold more[69] to even get to 8-12 ounces per week, which is still half of the maximum required for net benefits, additional neurological benefits to neonates from exposure to less fish seems implausible.
8. Cardiovascular benefits to adults from consuming less methylmercury requires attention, as discussed above. This follows from also not knowing the net benefits to adults from consuming different kinds and amounts of recreationally caught freshwater fish.
9. There may be benefits or net costs for children that are not accounted for in the current rule.

Recommendation

For purposes of this or any future mercury regulation, EPA should instigate a new risk assessment, particularly a net effects risk assessment following the FDA model. It should include all relevant health outcomes for neonates, children and adults. It should focus on consumption of recreationally caught freshwater fish, taking into account all other fish

[68] Lockhart, W.L., G.A. Stern, G. Low, M. Hendzel, G. Boila, P. Roach, M.S. Evans, B.N. Billeck, J. DeLaronde, S. Friesen, K. Kidd, S. Atkins, D.C.G. Muir, M. Stoddart, G. Stephens, S. Stephenson, S. Harbicht, N. Snowshoe, B. Grey, S. Thompson, and N. DeGraff. 2005. A history of total mercury in edible muscle of fish from lakes in northern Canada, Science of The Total Environment, 351–352:427-463.

[69] Spiller, P. et.al., "Petition for Reconsideration," FDA-2-17-P-3196.

consumption. This would be useful for both regulating limits on toxic chemicals as well as providing advice to consumers on fish consumption.

Evidence for Benefits of PM Reductions at Low Concentrations

Two recent studies have been conducted to evaluate risks associated with low concentrations of PM 2.5. This is relevant to the evaluation of the mercury standard as a significant portion of the co-benefits were from reductions of PM 2.5 in areas that were already in attainment. The first study constructed a cohort of all Medicare beneficiaries (60,925,443 persons) in the continental United States from the years 2000 through 2012 (Di et al. 2017).[70] Both PM 2.5 and ozone concentrations were estimated using a previously validated air pollution model that used an artificial neural network. Into this network went satellite-based measurements, outputs from a chemical transport model, land-use and meteorologic data. The model was then compared to measured data from the EPA monitoring network. Average annual concentrations were assigned to each individual for PM 2.5 and an annual average ozone concentration was assigned for the warm season. Temperature and relative humidity were assigned based on the zip code. The risks of death from both PM 2.5 and ozone were determined using a two-pollutant Cox proportional hazards model with generalized estimating equations. A second analysis was completed considering only person-years with PM 2.5 exposures less than 12 ug/m^3 and ozone exposures less than 50 ppb. For the entire population, increases of 10 ug/m^3 in PM 2.5 were associated with an increase in all-cause mortality of 7.3% (95% Confidence Interval 7.1-7.5). The value was actually slightly greater when the analysis was restricted to the lower exposures, 13.6% (95% CI 13.1- 14.1).

The second study (Pinault et al. 2016)[71] utilized a prospective cohort study from the Canadian Community Health Survey Cohort, which included 299,500 people and looked at the association of PM2.5 with mortality. Participants were enrolled between 2000 and 2008 and were followed through the end of 2011. The air pollution model used to estimate annual average PM 2.5 concentrations utilized satellite-based measurements, outputs from a chemical transport model, land-use patterns, and measured ground level data. Covariates included income, individual education, marital status, age, sex, immigrant status, minority status, weight, smoking, diet, alcohol consumption, and neighborhood level census data. Cox proportional hazard models were utilized for the analysis. There was no need to conduct a separate analysis for those with low-levels of air pollution as the mean was 6.3 ug/m^3, and the 95th percentile of exposure was 11.3 µg/m^3. Each 10 ug/m3 increase in PM 2.5 was associated with an increase in non-accidental death (HR=1.26; 95% CI: 1.19-1.34), circulatory disease death (HR=1.19; 95% CI: 1.07-1.31), and respiratory disease death (HR=1.52; 95% CI: 1.26-1.84).

[70] Di, Q.., Y. Wang, A. Zanobetti, P. Koutrakis P, C. Choirat, F. Dominici, and J.D. Schwartz. 2017. Air pollution and mortality in the medicare population. *New England Journal of Medicine* 376:2513-2522.

[71] Pinault L., M. Tjepkema, D.L. Crouse, S. Weichenthal, A. van Donkelaar, R.V. Martin, M. Brauer, H. Chen H and R.T. Burnett. 2016. Risk estimates of mortality attributed to low concentrations of ambient fine particulate matter in the Canadian community health survey cohort. *Environmental Health* 15:18.

Alternatively, Cox (2012)[72] has suggested that, not only may there be a threshold, but there may be a hormetic level for PM 2.5. This suggests that there may be a level at which there are actual beneficial effects of PM 2.5 and any regulation that goes below that level could actually be causing harm. This hypothesis was supported by two rat studies that found increased production of antioxidants.

Recommendation

As the vast majority of benefits in this rule are from PM 2.5, the benefits analysis should highlight the fact that co-benefits are from methylmercury reductions and that the primary benefit is related to PM 2.5. If it is decided to include benefits associated with PM 2.5, the evaluation of low level exposures of PM 2.5 should be noted.

Environmental Risk Screening

The environmental risk screening evaluation is detailed in Appendix 9: "Technical Support Document for the Environmental Risk Screen for RTR" of the Residual Risk Assessment for the Coal- and Oil-Fired EGU Source Category for the Risk and Technology Review 2019 Proposed Rule.[73] Technical concerns identified with this section of the report are summarized below.

Selection of Hazardous Air Pollutants (HAPs) to Include in Risk Screening Evaluation

Thirty-one suggested environmental hazardous air pollutants (HAPs) were evaluated for inclusion in the environmental risk screening based on the following four criteria provided in Table 2-1: (1) Persistence and bioaccumulation potential, (2) Inclusion in the TRIM.FaTE multipathway model, (3) Magnitude of emissions, and (4) Relative environmental toxicity – based on toxicity to wildlife, soil communities, and aquatic biota. Based on this evaluation, eight pollutants were included for further evaluation: six persistent bioaccumulative HAP (PB-HAP) – cadmium, dioxins, polycyclic organic matter (POM), mercury (both inorganic mercury and methylmercury), arsenic, and lead; and two acid gases – hydrochloric acid (HCl) and hydrofluoric acid (HF). However, the rationale for excluding selenium and chromium from further risk screening is questioned.

It is unclear why selenium is not designated as a PB-HAP given recognized bioaccumulation concern as reflected in the recent U.S. EPA (2016)[74] water quality criteria guideline for this substance which specifies quality criteria for both fish tissue and water. Further, based on data presented in Table 2-1, this substance has higher absolute emissions (and thus potential for environmental exposure) than any of the other PB-HAPs. In addition, the water quality criterion

[72] Cox, L.A., Jr. 2012. *Hormesis for Fine Particulate Matter (PM 2.5*). International Doses Response Society, October 28, 2011.

[73] U.S. EPA. 2018. Residual Risk Assessment for the Coal- and Oil-Fired EGU Source Category in Support of the 2019 Risk and Technology Review Proposed Rule, EPA Office of Air Quality Planning and Standards, Office of Air and Radiation.

[74] U.S. EPA. 2016. *Aquatic Life Ambient Water Quality Criterion for Selenium – Freshwater* https://www.epa.gov/sites/production/files/2016-07/documents/aquatic_life_awqc_for_selenium_-_freshwater_2016.pdf

(5 ppb) reported for this substance in Table 2-1 is less conservative than U.S. EPA (2016)[75] which specifies a monthly average of 1.5 to 3.1 ppb, depending on whether the receiving water is a lentic or lotic waterbody.

In the case of chromium, 2005 emissions are 10-fold higher while the water quality criterion for aquatic life protection is, depending on speciation assumptions, more than 10-fold lower (Table 2-1) than arsenic. Thus, given relative exposure potential and hazard, chromium would appear to pose a higher risk to aquatic life than arsenic. Therefore, it is not clear why arsenic is included in further risk evaluation while chromium is excluded using the screening criteria identified.

Risk Screening Assumption for Lead

Lead was included in the screen because it is a PB-HAP. While screening quality criteria were identified for soil, wildlife and aquatic life (Table 2-1), multimedia fate modeling to estimate lead exposures to these receptors was not performed since it was stated that this step represents a current limitation of the TRIM.FaTE model. Instead the secondary lead National Ambient Air Quality Standard was assumed to provide a reasonable measure for determining whether an adverse environmental effect occurs. However, the technical basis for assuming that the secondary standard ensures meeting quality criteria for soil and aquatic biota as well as wildlife lacks a sound technical basis. Based on the review by the U.S. EPA Clean Air Scientific Advisory Committee (CASAC) *Review of the EPA's Integrated Science Assessment for Lead*, major concerns are: the inability to relate ecosystem effects to the concentrations of Pb that exist in air, soil, and water; and for ecosystems the importance of atmospheric deposition and transport processes as sources of the Pb in soil and water (U.S. EPA CASAC, 2013).[76] Lead is persistent in the environment and may accumulate in soils and sediments through local deposition from air sources. Ecosystems near point sources of lead have demonstrated a wide range of adverse effects, including losses in biodiversity, changes in community composition, decreased growth and reproductive rates in plants and animals, and neurological effects in vertebrates. Further, ingestion of lead settled onto surfaces is reported to serve as the main route of human exposure to lead originally released into the air (TCEQ 2019).[77] Thus, further analysis appears warranted to confirm the extent to which local ecosystem risks associated with air emissions are potentially under or overestimated.

Recommendation

Consider selection of chromium and selenium for HAPs. Further analysis of lead emissions in air may be warranted to determine exposures in local ecosystems.

Selection of Ecological Benchmarks

[75] Ibid.

[76] U.S. EPA CASAC. 2013. Review of the EPA's Integrated Science Assessment for Lead. U.S. EPA Clean Air Scientific Advisory Committee, Washington, D.C. https://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/39A3C8177D869EA085257B80006C7684/$File/EPA-CASAC-13-004+unsigned.pdf

[77] TCEQ (Texas Commission on Environmental Quality). 2019. *Air Pollution from Lead.* https://www.tceq.texas.gov/airquality/sip/criteria-pollutants/sip-lead , accessed July 24, 2019.

To assess ecological effects, ecological benchmarks were identified for comparison to predicted exposure concentrations. Three general metrics for ecological benchmarks used were: (1) dose-based; (2) concentration-based, e.g., water, soil; and (3) tissue-based. In selecting concentration-based ecological benchmarks two types are used without distinction: causal and associative. Causal endpoints directly link the concentration of a pollutant to adverse effect via toxicity testing and are intended to determine the likelihood that a pollutant will cause adverse effects. This type of benchmarks serves as the basis for ambient water quality criteria. Associative endpoints are often derived for sediments from field studies that examine the co-occurrence of a contaminant with an adverse biological effect. Such ecological benchmarks can help identify sediments that have impaired quality but cannot be used to infer that the specific pollutant for which the benchmark is exceeded is the responsible agent. Thus, not differentiating these types of ecological benchmarks limits the ability to effectively screen true pollutant-specific risks. Causal ecological endpoints should be given preference to associative values, particularly at higher risk tiers, since the later estimates are highly variable and confounded by the presence of other stressors (McGrath et al. 2019).[78] Causal ecological benchmarks for aquatic life protection are available for many of the pollutants investigated this study (Burgess et al. 2013).[79] These water quality criteria can be multiplied by the default sediment-water and soil-water partition coefficient for the pollutant assumed in TRIM.FaTE to provide coherent ecological benchmarks for sediment and soil to support ecorisk screening rather than adopting associative values.
The authors state "Tissue-based benchmarks have little utility for the RTR program because site-specific data for the concentrations of HAPs in animal tissues are not available. Therefore, the identification of benchmarks for the environmental risk screen focused entirely on dose-based (e.g., TRVs) and concentration-based benchmarks."[80] However, site-specific water or soil concentrations are often not typically available as part of risk screening. Thus, if fish concentrations are predicted via multimedia modeling and scientifically-defensible tissue quality criteria are indeed available, as in the case of selenium (see above), and mercury (Fuchsman et al. 2016),[81] such criteria should be applied in risk screening.

EPA should also consider new science in selection ecological benchmarks. To screen risks to wildlife toxicity, reference values (TRVs) were selected from past, often quite dated, literature. However recent critical reviews provide an improved technical basis to select such TRVs for

[78] McGrath, J.A., J. Namita, A.S. Bess, and T.F. Parkerton. 2019. Review of polycyclic aromatic hydrocarbons (PAHs) sediment quality guidelines for the protection of benthic life, *Integrated Assessment and Environmental Management* 15(4):505=518. https://doi.org/10.1002/ieam.4142

[79] Burgess, R.M., W.J. Berry, D.R. Mount, and D.M. Di Toro. 2013. Mechanistic sediment quality guidelines based on contaminant bioavailability: Equilibrium partitioning sediment benchmarks. *Environmental Toxicology and Chemistry*, 32(1):102-114.

[80] U.S. EPA. 2018. *Residual Risk Assessment for the Coal- and Oil-Fired EGU Source Category in Support of the 2019 Risk and Technology Review Proposed Rule*. Appendix 9, page 15.

[81] Fuchsman, P, M.H. Henning, M.T. Sorensen, L.E. Brown, M.J. Bock, C.D. Beals, J.L. Lyndall, and V.S. Magar. 2016. Critical perspectives on mercury toxicity reference values for protection of fish. *Environmental Toxicology and Chemistry* 35(3):529–549.

selected pollutants relevant to this investigation (Beyer & Sample, 2017; Fuchsman et al. 2017).[82,83]

For environmental hazard evaluation of POM, toxicological equivalency factors (ecoTEF) are applied. The technical basis for defining ecoTEF for aquatic and soil/sediment are not transparent and do not reflect the current state of science. This concern was addressed previously in the most recent SAB review of EPA's Screening Methodologies to Support Risk and Technology Reviews (U.S. EPA Science Advisory Board, 2018).[84]

Recommendation

An alternative mechanistic approach which relies on toxic units to assess risks to aquatic, benthic and soil biota from this substance class is recommended (e.g., Burgess et al. 2013).[85]

Bioaccumulation of arsenic from sediment

For arsenic, empirical freshwater fish bioaccumulation factors (BAFs) and biota-sediment accumulation factors (BSAFs) were used to determine tissue concentrations and resulting exposures via the fish ingestion pathway instead of the biokinetic approach. While sufficient data were available to define the BAF (=46 to 95 L/kg wet depending on trophic level), only a single field study was identified to define the BSAF (=0.00018 kg bulk sediment dry / kg wet tissue). However, the arsenic sediment concentration in this field study involved a highly contaminated site with reported surficial sediment concentration of 1,830 mg[As]/ kg[sediment]. Thus, it is unclear if the BSAF derived from this study is representative of lower sediment concentrations that would be characteristic of exposures derived from local air emissions. Based on a cursory

[82] Beyer, W.N., and B.E. Sample, 2017. An evaluation of inorganic toxicity reference values for use in assessing hazards to American robins (Turdus migratorius). *Integrated Assessment and Environmental Management* 13(2):352-359.

[83] Fuchsman, P., L.E. Brown, M.H. Henning, M.J. Bock, and V.S. Magar. 2017. Toxicity reference values for methylmercury effects on avian reproduction: Critical review and analysis. *Environmental Toxicology and Chemistry* 36(2):294-319.

[84] U.S. EPA Science Advisory Board. 2018. *SAB Review of EPA's draft technical report entitled Screening Methodologies to Support Risk and Technology Reviews (RTR): A Case Study Analysis*.

[85] Burgess et al. Mechanistic sediment quality guidelines based on contaminant bioavailability: Equilibrium partitioning sediment benchmarks.

literature review, Cheng et al. (2013)[86] report BSAFs for arsenic in a range of freshwater fish from different trophic levels from freshwater ponds with observed arsenic sediment concentrations that are two orders of magnitude more than the study discussed above. Reported BSAFs for fish ranged from 0.016 to 0.195 depending on trophic level. Therefore, the assumptions invoked by EPA to predict arsenic bioaccumulation in fish from sediment may significantly understate actual tissue concentrations and hence risks to wildlife (and humans) from this exposure pathway.

Summary and Next Steps

In conclusion, the SAB has reviewed the science supporting EPA's proposed *Mercury and Air Toxics Standards for Power Plants Residual Risk and Technology Review and Cost Review* and provides recommendations to strengthen future regulations. The SAB recommends that the EPA review and implement the previous SAB recommendations concerning the agency's RTR Screening Methodology and also consider: conducting net effects risk assessments following the FDA model; highlighting the fact that co-benefits of this action are from methylmercury reductions and that the primary benefit is related to PM 2.5; including additional HAPs in the evaluation; and adopting an alternative mechanistic approach which relies on toxic units to assess risks to aquatic, benthic and soil biota.

Thank you for the opportunity to review the science supporting this proposed action. We look forward to your response to our comments.

Sincerely,

Dr. Michael Honeycutt, Chair
Science Advisory Board

Enclosure

1) Roster, EPA Science Advisory Board

[86] Cheng, Z, K-Ci Chen, K-B Li, X-P Nie, S.C. Wu, C. Kong-Chu, W. Hung, and M. Hung Wong. 2013. Arsenic contamination in the freshwater fish ponds of Pearl River Delta: bioaccumulation and health risk assessment. Environmental Science and Pollution Research 20:4484–4495.

NOTICE

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