UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

April 9, 2020

EPA-SAB-20-004

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 comments 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.

Subsequent to the June meeting, a working group of chartered SAB members was formed to carry out the review. Members of the working group took the lead in SAB deliberations on this

topic at a public teleconference held on January 24, 2020. This report provides the SAB's findings and recommendations.[1]

General Comments

The SAB has reviewed the EPA's "Residual Risk Assessment for the Coal- and Oil-Fired EGU Source Category in Support of the 2019 Risk and Technology Review Proposed Rule" (U.S. EPA 2018)[2] (The Residual Risk Assessment). That document describes the risk assessment that the EPA conducted to assess the human health and environmental risks posed by hazardous air pollutant (HAP) emissions from coal- and oil-fired electric utility steam generating units (EGUs) regulated under the Mercury and Air Toxics Standards (MATS). The SAB provides comments that apply specifically to the Residual Risk Assessment but notes that, due to time constraints, only a cursory technical review of the risk assessment document was conducted. The 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 2017a)[3]. The SAB previously reviewed the methodologies proposed by the EPA and published the findings of that review in 2018 (U.S. EPA Science Advisory Board 2018).[4] While the SAB's prior comments are relevant to conducting the analysis in the Residual Risk Assessment for the Coal- and Oil-Fired EGU Source Category assessment, the SAB's recommendations do not seem to have been taken into consideration. To ensure that conclusions drawn from the Residual Risk Assessment for the Coal- and Oil-Fired EGU Source Category benefit from SAB's earlier technical review that promotes increased transparency and inclusion of new science, the EPA is encouraged to review the findings and recommendations in the 2018 SAB report (U.S. EPA Science Advisory Board 2018)[5] and determine what revisions in the Agency's analysis are warranted.

The SAB notes that the EPA's benefit-cost analysis of the proposed action categorically excludes co-benefits. That departs from the Agency's long-standing practice and is contrary to both the Agency's guidance document on economic analysis (U.S. EPA 2014)[6] and to the recommendations of the Office of Management and Budget (U.S. OMB 2003).[7] As the Agency's

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[1]Two SAB members, Drs. Robert Phalen and Stanley Young, indicated that they did not concur with this report.

[2] 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, U.S. Environmental Protection Agency, Washington, D.C.

[3] U.S. EPA. 2017a. *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, N.C. [Available at: https://yosemite.epa.gov/sab/sabproduct.nsf//LookupWebProjectsCurrentBOARD/2708C2DBC839301685258060005C87E8/$File/Screening+Methodologies+to+Support+RTRs_A+Case+Study+Analysis.pdf]

[4] 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]

[5] Ibid.

[6] U.S. EPA. 2014. *Guidelines for Preparing Economic Analysis. May 2014 Update*. EPA National Center for Environmental Economics, U.S. Environmental Protection Agency, Washington, DC

[7] U.S. OMB (Office of Management and Budget). 2003. *Circular A-4: Regulatory Analysis*. Washington, DC: Executive Office of the President. September 17, 2003.

guidance has been previously reviewed by the SAB, excluding co-benefits is a departure from the Board's recommended practice. Furthermore, a SAB review of a recent update to the Agency's guidance on economic analysis is pending. The Board defers its evaluation of the change in the treatment of co-benefits until the SAB review of the updated guidance on economic analysis.

Human Health Risk Assessment

The SAB has specific concerns regarding the human health risk assessment that was conducted by the EPA for the Residual Risk Assessment (Section 2.5 of the Residual Risk Assessment document). The EPA's residual risk assessment appears to include only fish consumed from small to mid-sized lakes by fishermen and their families. The SAB also notes that the TRIM.FATE model used by the Agency "is not configured to model chemical processes and environmental fate and transport mechanisms in saltwater or brackish waters, nor is it configured to model the very large watersheds and water dynamics of rivers or very large lakes; these types of water bodies are also removed from the screening assessment" (U.S. EPA 2018).[8] While the EPA assessment 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),[9,10] this is only a small fraction of fish consumed in the United States. Much of the exposure to methylmercury comes from ocean fish which, are not included in EPA's estimate. Estimates indicate that estuarine and marine fish and shellfish comprise over 90% of the market share of commercial fish (Carrington et al. 2004).[11] Even though more than 80% of fish consumed in the U.S. is imported, there is still an appreciable quantity harvested from the Atlantic and Pacific regions (Karimi et al. 2012).[12] Many of the species of marine fish eaten by Americans spend large parts of their lives in U.S. domestic waters (Sunderland et al. 2016).[13] Further, there are higher levels of methylmercury in Atlantic than Pacific fish, which may help explain higher mean and 90th percentile blood 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).[14,15] It is estimated that 45% of the methylmercury intake of the U.S. population comes

[8] 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*.

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

[10] 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*.

[11] 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.

[12] 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.

[13] 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.

[14] 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.

[15] 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.

from open ocean marine fish (with 12% coming from the Atlantic) while 37% comes from domestic coastal systems (Sunderland et al. 2018).[16]

It is important to note that only a portion of the methylmercury in ocean fish results from U.S. fossil fuel sources. Mercury in the environment has many sources, including natural sources such as volcanoes and wildfires, and anthropogenic sources, including atmospheric emissions from fossil fuel combustion, metal and cement production, and non-atmospheric sources such as artisanal gold production and run-off from previously contaminated sites. As global mercury cycling needs to be included in any models predicting exposures from a particular source, there are obviously uncertainties associated with model predictions. Globally, Asia emits more mercury into the atmosphere than any other geographic region. North American emissions have been reduced 60% between 1990 and 2010 (Schmeltz et al. 2011),[17] with slight increases in emissions from 2010 – 2015 (Streets et al. 2019).[18] Total global mercury emissions from coal burning are 560 Mg/y.[19] Currently, U.S. atmospheric emissions are estimated to be 43 Mg/y,[20] while global atmospheric emissions are estimated to be 2,390 Mg/year.[21] Therefore, U.S. emissions from fossil fuels are only a small fraction of total mercury atmospheric emissions.

It is important to understand what impact these emissions have on concentrations in fish because, as noted above, fish consumption constitutes the majority of methylmercury exposure. Methylmercury has been found in prehistoric fish, indicating that natural sources have influenced global mercury cycling for over 1,000 years (Wilmsen and Meyers 1972).[22] Total mercury in edible muscle of fish from freshwater lakes in Northern Canada measured at multiple timepoints from between 1970 and 2001 showed fish muscle concentrations increasing in some lakes and decreasing in others over this time period.[23]

More detailed studies have been conducted to look at changes in both atmospheric levels and corresponding fish concentrations following reductions of emissions from specific sites and the overall decreasing trends of U.S. atmospheric emissions. Concentrations of atmospheric mercury have been found to be decreasing (Zhang et al. 2016).[24] Mercury levels in sediment cores in the

[16] Sunderland, E.M., M. Li, and K. Bullard. 2018. Decadal changes in the edible supply of seafood and methylmercury exposure in the United States. *Environmental Health Perspectives* 126:029003. DOI: 10.1289/EHP3460.

[17] Schmeltz, D., D.C. Evers, C.T. Driscoll, R. Artz, M. Cohen, D. Gay. R. Haeber, D.P. Krabbenhoft, R. Mason, K. Morris, and J.G. Wiener. 2011. MercNet: a national monitoring network to assess responses to changing mercury emissions in the United States. *Ecotoxicology* 20:1713-1725.

[18] Streets, D.G., H.M. Horowitz, Z. Lu, L. Levin, C.P. Thackray, and E.M Sunderland. 2019. Global and regional trends in mercury emissions and concentrations 2010-2015. *Atmospheric Management* 201:417-427.

[19] Ibid.

[20] Ibid.

[21] Ibid.

[22] Wilmsen, E.N., and J.T. Meyers. 1972. The mercury content of prehistoric fish. *Ecology of Food and Nutrition* 1:179-186.

[23] 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.

[24] Zhang, Y. D.J. Jacob, H.M. Horowitz, L. Chen, H.M. Amos, D.P. Krabbenhoft, F. Slemr, V.L. St Louis, and E.M. Sunderland. 2016. Observed decrease in atmospheric mercury explained by global decline in anthropogenic emissions. *Proceedings of the National Academy of Sciences of the United States of America* 113(3):526-531.

Great Lakes have shown decreases corresponding to reduced emission (Drevnick et al. 2012).[25] Differences in the relative importance of atmospheric deposition between the five Great Lakes have been observed corresponding in part to changing source profiles (Lepak et al. 2015).[26] Recent studies have evaluated the importance of decreases in local sources on decreases in both atmospheric mercury concentrations and wet deposition (Castro and Sherwell 2015; White et al. 2009).[27, 28] 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).[29] Research suggests recent decreases in mercury emissions have resulted in declines in mercury concentrations in Atlantic coastal fish stocks (Cross et al. 2015).[30] Studies have also shown that concentrations of methylmercury in open ocean fish such as tuna are declining more rapidly than levels in coastal fish as atmospheric levels decline (Lee et al. 2016).[31] Up to 90% of methylmercury concentrations in open ocean fish are estimated to result from atmospheric deposition (Driscoll et al. 2013; Mason et al. 2012; Obrist et al. 2018).[32, 33, 34]

There have been recent advances in global mercury cycling models, in part resulting from advances in mercury stable isotope characterization, allowing for better characterization of transformation processes within the system (Obrist et al. 2018).[35] Ocean water mercury concentrations have been declining in the North Atlantic as U.S. atmospheric sources have decreased, while concentrations in the Pacific have increased (Obrist et al. 2018).[36] Near shore fisheries are impacted less by atmospheric depositions, and more by river runoff that includes mercury-containing effluents (Obrist et al. 2018).[37] A recent paper used a modeling approach to assess costs and benefits of the MATS regulation, considering the impact that decreases in U.S.

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[25] 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.

[26] Lepak, R.F., R. Yin, D.P. Krabbenhoft, J.M. Ogorek, J.F. DeWild, T.M. Holsen, and J.P. Hurley. 2015. Use of stable isotope signatures to determine mercury sources in the Great Lakes. *Environmental Science and Technology Letters* 2(12):335-341.

[27] Castro, M., and J. Sherwell. 2015. Effectiveness of emission controls to reduce the atmospheric concentrations of mercury. *Environmental Science and Technology* 49:14000-14007.

[28] White,, E.M., G.J. Keeler, and M.S. Landis. 2009. Spatial variability of mercury wet deposition in eastern Ohio summertime meteorological case study analysis of local source influences. *Environmental Science and Technology* 1,43(13):4946-4953.

[29] 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.

[30] 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.

[31] Lee, C.S., M.E. Lutcavage, E. Chandler, D.J. Madigan, R.M. Cerato, and N.S. Fisher. 2016. Declining mercury concentrations in bluefin tuna reflect reduced emissions to the North Atlantic Ocean. *Environmental Science and Technology* 50(23):12825-12830.

[32] Driscoll, C.T., R.P. Mason, H.M. Chan, D.J. Jacob. and N. Pirrone. 2013. Mercury as a global pollutant: sources, pathways, and effects. *Environmental Science and Technology* 47(10):4967-4983.

[33] Mason, R.P., A.L. Choi, W.F. Fitzgerald, C.R. Hammerschmidt, C.H. Lamborg, A.L. Soerensen, and E.M. Sunderland. 2012. Mercury biogeochemical cycling in the ocean and policy implications. *Environmental Research* 119:101-117.

[34] Obrist, D., J.L. Kirk, L. Zhang, E.M. Sunderland, M. Jiskra, and N.E. Selin. 2018. A review of global environmental mercury processes in response to human and natural perturbations: Changes of emissions, climate, and land use. *Ambio* 47(2):116-140.

[35] Ibid.

[36] Ibid.

[37] Ibid.

mercury emissions would have both locally and on ocean deposition, and therefore on fish concentrations (Giang and Selin 2016).[38] The authors note that there a number of uncertainties associated with this assessment. The analysis must link policies, emissions, atmospheric chemistry, deposition processes, bioaccumulation, and human exposure, all superimposed on a background of existing reservoirs of mercury in multiple environmental media. The authors also note the importance of assessing mercury on both a local and regional scale. The EPA could find this approach useful when developing its own models.

Recommendation

For this or any future regulation, the EPA should prepare a new exposure estimate that accounts for total exposure. Methylmercury exposure for local populations should consider both exposure from U.S. power plants as well as overall exposure, particularly from ocean fish. In addition, the EPA should consider accounting for the incremental exposure that this sector contributes to overall exposure for the entire population.

Dose-Response Assessment

EPA's dose-response assessment for hazardous air pollutants in the coal- and oil-fired EGU source category is presented in Section 2.7 of the Residual Risk Assessment and, for mercury, specifies reliance on the EPA Integrated Risk Information System (IRIS) assessment. The approach relies on a 2000 National Research Council report discussed below. Additionally, there are several other health endpoints related to methylmercury, also discussed below.

Neurodevelopmental Outcomes **-** The National Research Council reviewed the literature on chronic low-dose prenatal methylmercury exposure and neurobehavioral outcomes on children (NRC 2000)[39] In this report, a reference dose and dose-response relationship was developed based primarily on data from two cohort studies, one from the Faroe Islands and one from New Zealand. Neither of these studies included consumption of fish as a confounder in the analysis of the data. It is difficult to assess the adverse effects of methylmercury on neurodevelopment due to the confounding of the polyunsaturated long-chain fatty acids in fish, and perhaps other components of fish, which have positive effects on neurodevelopment. That means that one needs to account for negative confounding in situations like this that arise when a covariate is a source of exposure, in this case fish consumption (Choi et al., 2008).[40]

The best studies account for the benefits of fish consumption when evaluating the negative impacts of methylmercury, considering there may be health benefits from the matrix (i.e., seafood) in which methylmercury is delivered. Recent epidemiological findings indicate that there are more sensitive neurodevelopmental endpoints than full-scale IQ, as used by the EPA (Sunderland et al. 2016).[41] Further, these impacts have been documented at lower levels than the

[38] Giang, A. and N.E. Selin. 2016. Benefits of mercury controls for the United States. *Proceedings of the National Academy of Sciences of the United States* 113(2):286-291.

[39] NRC (National Research Council). 2000. *Toxicological Effects of Methylmercury.* National Research Council Committee on the Toxicological Effects of Methylmercury, The National Academies Press, Washington, D.C

[40] Choi, A.L., S. Cordier, P. Weihe, and P. Granjean. 2008. Negative confounding in the evaluation of toxicity: the case of methylmercury in fish and seafood. *Critical Reviews in Toxicology* 38(10):877-893.

[41] 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.

reference dose established by a National Research Council panel in 2000[42] (Sunderland et al. 2016; Bellanger et al. 2013).[43,44] Other recent reviews have begun to question whether there is evidence for a safe level of methylmercury exposure (Grandjean et al. 2012; Karagas et al. 2012).[45,46]

Karagas et al. (2012)[47] find the strongest effects for multiple neurological impacts, including psychomotor function, memory, and verbal skills cognition at 3-6 years of age with prenatal methylmercury exposure. These results were found to be consistent among multiple prospective cohort studies that all accounted for fish consumption during pregnancy (Freire et al. 2010; Lederman et al. 2008; Oken et al. 2008).[48,49,50] It is important to account for fish consumption because the beneficial aspects of fish consumption appear to offset the adverse impacts of methylmercury (Karagas et al. 2012; Stewart et al. 2003; Jedrychowski et al. 2007).[51,52,53] Although effects at other ages were inconclusive, looking instead by effect at all age groups found two domains to consistently be the most sensitive (Karagas et al. 2012);[54] specifically, memory (Freire et al. 2010; Oken et al. 2005; Weil et al. 2005)[55,56,57] and verbal or language

[42] NRC (National Research Council). 2000. *Toxicological Effects of Methylmercury*. National Academy Press, Washington, D.C.

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

[44] 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.

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

[46] 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.

[47] Ibid.

[48] 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.

[49] 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.

[50] 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 U.S. cohort. *American Journal of Epidemiology* 167(10):1171-81. doi: 10.1093/aje/kwn034. Epub 2008 Mar 1

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

[52] 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.

[53] 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.

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

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

[56] 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–

[57] 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):1875.

skills (Freire et al. 2010; Lederman et al. 2008; Oken et al. 2008; Surkan et al. 2009).[58,59,60,61] More recently, Orenstein at al. (2014)[62] found that increases in maternal hair concentrations of mercury were associated with decreases in visual memory, learning, and verbal memory in children 8 years of age, on average (range, 7-11 years). These mothers were also exposed to higher levels of polychlorinated biphenyls (PCBs) but outcomes were not associated with PCB exposures.

Other Potential Health Endpoints for Mercury - Beyond neurological effects, other potential health endpoints for methylmercury exposure include cardiovascular disease (Roman et al. 2011),[63] endocrine function (Tan et al. 2009),[64] risk of diabetes (He et al. 2013),[65] hypertension (Hu et al. 2018),[66] and impacts on immune function (Nyland et al. 2011; Karagas 2012).[67,68] The literature is the most developed for cardiovascular endpoints and thus only those endpoints are reviewed here.

In 2010, the EPA held a workshop to review the current science on cardiovascular impacts of methylmercury exposures. The assembled panel of scientists found "the body of evidence exploring the link between methylmercury and acute myocardial infarction (MI) to be sufficiently strong to support its inclusion in future benefit analyses, based both on direct epidemiological evidence of a methylmercury-MI link and on methylmercury's association with intermediary impacts that contribute to MI risk" (Roman et al. 2011).[69] The two mechanisms with the strongest evidence for biological plausibility were oxidative stress and heart rate variability (HRV). There is consistent evidence in animal studies for methylmercury-induced

[58] Freire C, et al. 2010. Hair mercury levels, fish consumption, and cognitive development in preschool children from Granada, Spain.
[59] Lederman et al. 2008. Relation between cord blood mercury levels and early child development in a World Trade Center cohort.
[60] Oken et al. 2008. Maternal fish intake during pregnancy, blood mercury levels, and child cognition at age 3 years in a U.S. cohort.
[61] 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
[62] Orenstein, S.T. S.W. Thurston, D.C. Bellinger, J.D. Schwartz, C.J. Amarasiriwardena, L.M. Altshul, and S.A. Korrick. 2014. Prenatal organochlorine and methylmercury exposure and memory and learning in school-age children in communities near the New Bedford Harbor Superfund site, Massachusetts. *Environmental Health Perspectives* 122(11):1253-1259.
[63] 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.
[64] 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.
[65] 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.
[66] Hu, X.F., K. Singh, and H.M. Chan. 2018. Mercury exposure, blood pressure, and hypertension: A systematic review of dose-response meta-analysis. *Environmental Health Perspectives* 126(7):076002.
[67] 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.
[68] Karagas et al. 2012. Evidence on the human health effects of low-level methylmercury exposure.
[69] 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.

oxidative stress (Roman et al. 2011; Genchi et al. 2017).[70,71] Lipid peroxidation in rats has been shown to increase with methylmercury exposure (Huang et al.1996; Lin et al. 1996).[72,73] 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 methylmercury (Grotto et al. 2009).[74] In addition to the animal studies, the EPA panel identified two epidemiological studies that found evidence of methylmercury exposure and oxidative stress (Salonen et al. 1995; Grotto et al. 2010).[75,76] The panel also found one study with contradictory findings. The study showed decreased oxidized low density lipoprotein (LDL) among fishermen before and after sport fishing season (the fishing season resulted in an increased rate of consumption of both fish and methylmercury) (Belanger et al. 2008).[77]

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).[78,79] The EPA panel found strong evidence of decreased HRV with increased methylmercury exposure based on the epidemiological evidence (Roman et al. 2011).[80] This relationship was shown in three studies of various populations (Valera et al. 2008; 2009; 2010; Lim et al. 2009).[81,82,83,84] There is also an intervention study in which healthy Japanese adults were either assigned to an experimental

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[70] Ibid.

[71] 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.

[72] 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.

[73] 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.

[74] 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.

[75] 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.

[76] 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.

[77] 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.

[78] 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.

[79] 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.

[80] 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.

[81] 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].

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

[83] 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.

[84] 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.

group where they ate tuna and swordfish at Japan's provisionally tolerable weekly intake or a control group (Yaginuma-Sakurai et al. 2009);[85] HRV decreased significantly in the group assigned to eat tuna and swordfish. The Roman et al. paper also provided evidence for a number of other mechanisms not summarized here.

In contrast, a large study including two U.S. cohorts did not find evidence of cardiovascular risk (Mozaffarian et al. 2011).[86] There were, however, 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).[87] This may suggest that, as with neurological effects, long-chain fatty acids neutralize the negative effects of methylmercury on cardiovascular disease outcomes. 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.

A study by Rice et al. (2010)[88] found that including cardiovascular risks from methylmercury in a cost-benefit assessment is critical.. This finding is based on a probabilistic assessment of the health and economic benefits from a reduction in methylmercury exposure showing 80% of the monetized health benefits come from reduction in fatal heart attacks, with the remainder coming from IQ gains (Rice et al. 2010).[89]

Recommendation

For purposes of this or any future mercury regulation, EPA should prepare a new risk assessment. It is recommended that the EPA conduct a risk assessment that accounts for neurological impacts from mercury and includes other health endpoints for methylmercury, particularly cardiovascular endpoints if supported by the available data. To appropriately evaluate the health effects of mercury resulting from exposure to power plants, the EPA also needs to account for exposure from other sources of mercury in order to determine an individual's total mercury exposure.

Public Health Implications of Fish Consumption

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 the

[85] 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.

[86] 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.

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

[88] 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

[89] Ibid.

quantitative estimate of benefits.[90] Later, however, the U.S. Food and Drug Administration (FDA) was able to directly incorporate both beneficial effects of fish consumption along with negative health impacts of mercury exposure. 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, reducing maternal exposure to methylmercury from recreationally self-caught freshwater fish. EPA mentioned, but did not quantify, other possible human and environmental benefits.

Public health officials should provide messaging to encourage consumption of a variety of fish, and, although results of studies are mixed, should concentrate more on consumption of oily fish.[91] In the 1999-2000 Centers for Disease Control and Prevention's National Health and Nutrition Examination Survey (NHANES) study, fish consumption was increasing while mercury levels in hair were decreasing.[92] A more recent study found, "On average, U.S. women of reproductive age were consuming more fish and blood mercury levels were lower in 2009–2010 compared to 1999–2000."[93] 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 mercury 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 mercury 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.

The 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).[94] In that document, both agencies recommended that pregnant and potentially pregnant women *increase* consumption of a variety of fish lower in methylmercury 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 methylmercury concentrations.

In a recently published systematic review of 29 studies in which the amount of fish eaten as a whole food during pregnancy was compared against neurocognitive outcomes in over 100,000 children, beneficial associations were reported in a majority of them while virtually no evidence

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

[91] Hibbeln J.R., J.M. Davis, C. Steer, P. Emmett, I. Rogers, and C. Williams. 2007. Maternal seafood consumption in pregnancy and neurodevelopmental outcomes in childhood (ALSPAC study): an observational cohort study. *Lancet* 2007; 369:578–585.

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

[93] 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/

[94] U.S. 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]

of harm to neurocognition was reported.[95, 96] The review found that consumption of a wide range of amounts and types of commercially available seafood during pregnancy was associated with improved neurocognitive development of offspring as compared to eating no seafood. Overall, benefits to neurocognitive development began to appear at the lowest amounts of seafood consumed (~4 oz/week) and continued into the highest categories of consumption (>100 oz/week). Seafood provided overall benefits to neurocognitive development even when mercury exposures in the same study populations were high by U.S standards. This overall outcome is consistent with previously published assessments of net effects by the FDA and the Food and Agriculture Organization of the United Nations together with the World Health Organization. For all people who report eating fish, the average fish consumption 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.[97]

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.[98]

Fish is widely considered to be cardioprotective and any benefits claimed from reducing methylmercury in fish should account for this (e.g., Mozaffarian et al. 2011).[99] When considering any effect that methylmercury may have on cardiovascular disease, EPA should investigate the direction and extent of the mitigating factors of omega 3 fatty acids on methylmercury, particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), which are widely considered to be cardioprotective (e.g., Nøstbakken et al. 2012).[100] The same may be true for marine selenium. In particular, the EPA should ensure that any findings related to cardiovascular disease should be clinically relevant. For example, hypertension is not by itself a negative health outcome. Just reducing blood pressure is not enough to reduce the risk of death." (Wiysonge et. al.)[101]

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[95] Hibbeln, J.R, P. Spiller, J.T. Brenna, J. Golding, B.J. Holube, W.S. Harris, P. Kris-Etherton, B. Lands, S.L. Connor, G. Myers, J.J. Strain, M.A. Crawford, and S.E. Carlson. 2019. Relationships between seafood consumption during pregnancy and childhood and neurocognitive development: Two systematic reviews. *Prostaglandins, Leukotrienes and Essential Fatty Acids* 151:14-36.

[96] Spiller, P., J.R. Hibbeln, G. Myers, G. Vannice, J. Golding, M.A. Crawford. J.J. Strain, S.L. Connor, J. T. Brenna, P. Kris-Etherton, B.J. Holub, W.S. Harris, B. Lands, R.K. McNamara, M.F. Tlusty, N. Salem, Jr., and. S.E. Carlson. 2019. An abundance of seafood consumption studies presents new opportunities to evaluate effects on neurocognitive development. Prostaglandins, *Leukotrienes and Essential Fatty Acids* 151:8-13.

[97] Jahns, L., S.K. Raatz, L.K. Johnson, S. Kranz, J.T. Silverstein, and M.J. Picklo, Sr. 2014. Intake of seafood in the U.S. varies by age, income, and education level but not by race-ethnicity. *Nutrients* 6(12)6060-6075..

[98] 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

[99] Mozaffarian, D., S. Peilin, J.S. Morris, D. Spiegleman, P. Grandjean, D.S. Siscovick, W.C. Willett, and E.B. Rimm. 2011. Mercury exposure and risk of cardiovascular disease in two U.S. cohorts. *The New England Journal of Medicine* 364:1116-1125, DOI: 10.1056/NEJMoa1006876.

[100] Nostbakken, O.J., I.L. Bredal, P.A. Olsvik, T.S. Huang, and B.E. Torstensen. 2012. Effect of marine omega 3 fatty acids on methylmercury-induced toxicity in fish and mammalian cells in vitro. *Journal of Biomedicine and Biotechnology*2012:417652, dpi:10. 1155/2012/417652.

[101] Wiysonge, C.S., H.A. Bradley, J. Volmink, B.M. Mayosi. and L.H. Opie. 2012. Beta-Blockers for Hypertension. *Cochrane Database of Systematic Reviews* doi.org/10.1002/14651858.CD0023.pub5.

Environmental Risk Screening

The EPA's environmental risk screening assessment is detailed in Appendix 9 of the *Residual Risk Assessment for the Coal- and Oil-Fired EGU Source Category in Support of the 2019 Risk and Technology Review Proposed Rule*.[102] The SAB's concerns regarding EPA's environmental risk screening assessment 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 of Appendix 9: (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, as discussed below, the SAB questions the rationale for excluding selenium and chromium from further risk screening.

It is unclear why selenium is not designated as a PB-HAP given the U.S. EPA (2016)[103] water quality criteria guideline for this substance, which quantitatively incorporates bioaccumulation in the derivation of quality criteria for both fish tissue and water. This criteria document provides a more recent review than outdated information included in the updated Risk and Technology Review (RTR) environmental risk screening methodology.[104] Further, based on data presented in Table 2-1 of Appendix 9 of the RTR residual risk assessment,[105] 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 (5 ppb) reported for selenium in Table 2-1 is less conservative than the U.S. EPA (2016)[106] criterion 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 were 10-fold higher while the water quality criterion for aquatic life protection is, depending on speciation assumptions, more than 10-fold lower than arsenic (Table 2-1). 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 while chromium is excluded in further risk evaluation.

Recommendation

The technical rationale for HAP screening should be re-evaluated. If selenium and chromium are excluded, further justification is required.

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[102] 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.

[103] 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

[104] U.S. EPA. 2017. *Screening Methodologies to Support Risk and Technology Reviews (RTR): A Case Study Analysis*.

[105] 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.

[106] U.S. EPA. 2016. *Aquatic Life Ambient Water Quality Criterion for Selenium – Freshwater.*

Risk Screening Assumption for Lead

Lead was included in the screen because it is a PB-HAP. While screening 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 sufficient technical justification. 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 identified were: the inability to relate ecosystem effects to the concentrations of lead that exist in air, soil, and water; and for ecosystems, the importance of atmospheric deposition and transport processes as sources of the lead in soil and water (U.S. EPA CASAC, 2013).[107] 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 (TCEQ 2019).[108]

Recommendation

Further justification is needed to support the assumption that the secondary air standard for lead is protective of residual risks to local ecosystems.

Selection of Ecological Benchmarks

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 benchmark 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. Further, many of the ecotoxicity hazard studies included in the RTR screening methodology for defining ecological benchmarks are from the 1980's to 1990's and involve compilation of reviews from earlier publications of uncertain reliability.

[107] U.S. EPA CASAC (Clean Air Scientific Advisory Committee). 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

[108] 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.

More up-to-date causal ecological benchmarks for water developed by EPA to support environmental risk assessment (Burgess et al. 2013)[109] 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).[110] Further, these water quality criteria can be multiplied by the default sediment-water and soil-water partition coefficient for the corresponding pollutant modeled in TRIM.FaTE to provide coherent ecological benchmarks for sediment and soil to support risk screening.

In Appendix 9 of the RTR residual risk assessment it is stated that "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., toxicity reference values or TRVs) and concentration-based benchmarks."[111] However, site-specific water or soil concentrations are also 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 is apparent for selenium (see above), and mercury (Fuchsman et al. 2016),[112] such criteria could be applied in risk screening.

EPA should also consider new science in selecting ecological benchmarks for wildlife. 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 TRVs for selected pollutants relevant to the present rule (Beyer and Sample 2017; Fuchsman et al. 2017).[113,114]

For environmental hazard evaluation of POM, toxicological equivalency factors (ecoTEF) are applied. While the use of the TEF concept is widely used in human health risk evaluation, the technical basis for defining ecoTEFs for aquatic and soil/sediment is inconsistent with EPA mechanistic guidance that is available to quantify risks to aquatic and benthic life for this substance class (Burgess et al. 2013; U.S. EPA 2017b).[115, 116]

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[109] 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.

[110] 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

[111] 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.

[112] 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.

[113] 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.

[114] 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.

[115] 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.

[116] U.S. EPA. 2017b. *Developing Sediment Remediation Goals at Superfund Sites Based on Pore Water for the Protection of Benthic Organisms from Direct Toxicity to Non-ionic Organic Contaminants*. EPA/600/R 15/289, EPA

Recommendation

New hazard information that is available for establishing ecological benchmarks for HAPs should be incorporated into the residual risk assessment. An alternative mechanistic approach to assess environmental risks from POM is recommended.

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 are characteristic of exposures derived from local air emissions. Based on a cursory literature review, Cheng et al. (2013)[117] report orders of magnitude higher BSAFs for arsenic ranging from 0.016 to 0.195 in fish collected from freshwater ponds with much lower, likely representative arsenic sediment concentrations than the study discussed above. 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.

Recommendation

Assumptions for quantifying the bioaccumulation of arsenic in fish from sediment should be reassessed and revised based on reliable data that are available.

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 consider including other health effects of methylmercury, such as cardiovascular effects, and addressing technical concerns identified in the SAB's review of the environmental risk screening assessment.

Office of Research and Development, Washington, D.C. [Available at: https://semspub.epa.gov/work/HQ/100000539.pdf]

[117] 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.

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

Sincerely,

/s/

Dr. Michael Honeycutt, Chair
Science Advisory Board

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