Quantifying the health and economic impacts of mercury: an integrated assessment approach

by

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B.A.Sc., University of Toronto (2011)

Submitted to the Engineering Systems Division
in partial fulfillment of the requirements for the degree of

Master of Science in Technology Policy

at the

MASSACHUSETTS INSTITUTE OF TECHNOLOGY

June 2013

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Abstract

Mercury is a toxic pollutant that endangers human and ecosystem health. Especially potent in the form of methyl mercury, exposure is known to lead to adverse neurological effects, and, a growing body of evidence suggests, cardiovascular ones. Mercury's health impacts have economic consequences, and benefit-cost analyses focusing on these health benefits are used to motivate regulatory action in the United States and elsewhere. However, many existing valuation studies of the health impacts of mercury have substantial limitations, both from a scientific and economic perspective. Because they do not fully model mercury's path from emissions to impacts, they do not fully reflect the spatial and temporal dimensions of the mercury problem. In addition, many do not consider uncertain, but potentially policy-relevant health effects like cardiovascular disease.

This thesis develops an integrated assessment framework that more completely represents mercury's emissions-to-impacts path, and then evaluates its policy relevance. The assessment framework integrates chemical transport modelling, exposure and health impacts modelling, and general equilibrium modelling of the US economy. As a case study, the framework is used to evaluate the benefits of the Mercury and Air Toxics Standards—a recent US regulation that targets emissions from coal-fired power plants—until 2050. I estimate the annual benefit of MATS to be 13 million 2005 USD, compared to a scenario that includes stringent air quality policy, and 414 million 2005 USD when compared to a no policy scenario. I find that the estimate is highly sensitive to uncertainties along the emissions-to-impacts path—in particular, dose-response parameterization, ecosystem lag times, and discount rate. The analysis suggests that given the large ranges of uncertainty involved, more fully representing the emissions-to-impact chain does not lead to substantially different aggregate benefits estimates, compared to those existing in the literature. However, because this approach does provide more insight into the controlling influences behind benefits, it can inform decisions about where policies should be implemented, and of what type, as well as best practices for transparently assessing mercury-related policies.

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Acknowledgments

First and foremost, I would like to thank my advisor, Noelle Selin. Without her guidance, patience, and good humour, this thesis would not have been possible. I have grown and learned so much over the past two years, and much of that has been due to Noelle's dedicated mentorship.

I am also indebted to my friends and colleagues in the Selin group and Joint Program—particularly Rebecca, Leah, Carey, Shaojie, Ellen, Colin, and Tammy—who have held my hand through the trials of programming, and listened to my anxieties and complaints with plenty of good nature. I am so honoured and delighted to have called them my academic family over the course of my Master's degree.

I would like to thank all my dear friends in TPP for sharing some fantastic adventures with me at MIT. Sunny lunch breaks in the grass, beers over soul and bluegrass, endless boardgames, and epic jam sessions have been the highlights of these past two years, and I am so grateful to have met this group of inspiring and incredible individuals.

Many thanks go to my brother, Wayne Giang, for always providing feedback and great discussions. I am sure I would not be studying what I am, and enjoying it so much, without his support and example.

I am so grateful to have found a home at 3 GSP, and would like to say a heartfelt thank you to Nathalie, Brittany, and John for keeping me happy and healthy—even when I am determined not to be while writing a thesis.

It is difficult to express my gratitude for my parents—I know they have always worked their hardest so that I can do what I love, and their love and support has made everything possible.

Finally, I'd like to thank Sean, whose steadfast love, support, and encouragement have kept me afloat throughout. His belief in me has meant the world.

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Chapter 1

Introduction

1.1 Problem Introduction

Even at low levels, chronic exposure to mercury through fish and seafood consumption has been linked to adverse cognitive and—though more uncertain—cardiovascular effects (Mergler et al., 2007; Karagas et al., 2012). In the United States alone, it has been estimated that over 300 000 newborns each year may be at risk of adverse neurodevelopmental effects due to *in utero* exposure to methylmercury (MeHg)—a particularly toxic form of the pollutant (Mahaffey et al., 2004). If there is a causal relationship between mercury exposure and cardiovascular impacts, then it is possible that an even larger population would be at risk for negative cardiovascular outcomes like increased risk of heart attacks (Roman et al., 2011). However, while it is widely acknowledged that these health impacts will have broader socio-economic effects (Swain et al., 2007), assigning a monetary value to these effects remains a critical challenge for environmental policy-makers who are designing and evaluating mercury reduction policies, both in the United States and elsewhere. In fact, this need was highlighted in the recently finalized text of the new international environmental treaty targeting mercury—the Minamata Convention (UNEP, 2013b).

In the US, regulators are required to weigh the benefits of pollution control against the costs of implementing abatement technologies as part of the rule-making and retrospective rule-reviewing process—what is often called benefit-cost analysis (BCA) (Arrow et al., 2012).[1] While there is significant debate over whether or not BCA is an useful, or even ethically appropriate, tool for environmental decision-making (Ackerman & Heinzerling, 2002; Hammitt, 2012), it nevertheless remains a fixture of the US regulatory landscape under executive order (Graham, 2007).[2] As a result, the

[1] Despite this requirement, in many cases, the Environmental Protection Agency is not allowed to take into account the balance of costs and benefits in its rule-making (Portney, 1994). However, even in these cases, results from a BCA can affect the speed at which a proposed rule becomes finalized and implemented. This phenomenon will be further discussed in Chapter 3.

[2] Executive Order 12866 calls for Regulatory Impact Assessments of any proposed regulation, which includes a BCA, and Executive Order 13563 calls for retrospective reviews of existing regulation, using both quantitative and qualitative BCA.

methods by which these cost and benefit valuations are generated can have significant impact on the design and evaluation of public policy, and ultimately, social welfare and equity.

For mercury, as with many pollutants, quantifying the *benefits* of emissions reductions is particularly difficult (Pindyck, 2007).[3] The complexity of mercury's path from emissions to human impacts introduces considerable uncertainties into attributing benefits to policy. For mercury, these complexities are related to the pollutant's multi-spatial, multi-temporal, and multi-media nature (Selin, 2011; Lambert et al., 2012), as well as the subtlety of its potential health effects at low exposures (Karagas et al., 2012). Depending on its chemical form, mercury can act on multiple spatial scales, from local to global (Selin, 2011). In addition, because for most Americans the primary exposure pathway to MeHg is through fish and seafood consumption, global trade of seafood further contributes to mercury's transboundary effects (Sunderland, 2007; Lambert et al., 2012). Mercury also has a complex biogeochemical cycle, and the timescales of its movement between different environmental compartments—atmospheric, aquatic, terrestrial, and biotic—can range from days to millennia (Selin, 2011). Finally, our scientific understanding of MeHg's subtle effects at chronic, low-level exposures remains imperfect (Karagas et al., 2012); however, while these effects may be only subtle at an individual level, over an entire population they can become economically significant (Rice et al., 2010). All of these factors can complicate our understanding of the pathway from emissions to socio-economic impacts, making it particularly difficult to value to benefits of reducing mercury pollution.

Many existing valuation studies of the health impacts of mercury have been limited in how completely they have represented the path from emissions to impacts, and how realistically they have represented its complexities. First, many lack spatial and temporal disaggregation—they do not explicitly consider transport through the environment, or the timescales associated with bioaccumulation through ecosystems (Trasande et al., 2005; Spadaro & Rabl, 2008; Rice et al., 2010). On the economic side, they may offer only static, multiplier-based estimates of economic damages associated with pollution rather than including economy-wide effects (ie. how illness or mortality affects the productivity of the economy as a whole), making it difficult to include compounding effects over time, and to evaluate how the timing of emissions changes benefits (Griffiths et al., 2007; US EPA, 2011d). Finally, as highlighted by Rice et al. (2010), few studies have explicitly included more uncertain, but potentially economically important, health endpoints like cardiovascular effects in their estimates. For instance, US EPA (2011d) focused on only IQ-related effects in their benefits analysis of the recently promulgated Mercury and Air Toxics Standards in the US.

However, is this lack of completeness in how the emissions-to-impacts chain is represented actually problematic? Models are necessarily simplifications of real-world systems, and there are finite limits to how much complexity we can include in our

[3]A key difficulty associated with quantifying the benefits of pollution reduction policy is that there is no direct market for environmental quality (Hanemann, 1994), whereas there are market prices for the costs of policy (eg. cost of abatement technologies).

models, if for only computational reasons (Simon, 1990). What is important then, is to understand when more complete representations are needed, and to answer which classes of policy questions? In the context of benefits assessment for mercury, when is increased model complexity and completeness worthwhile? What is gained (and lost) by disaggregating spatially, temporally, and including uncertain health impacts? With stricter regulations for mercury emissions in the US coming into effect within the next 3 years (US EPA, 2011d), and a global treaty on mercury soon after (IISD Reporting Services, 2013), developing a stronger understanding of how emissions reductions translate into social benefits will be critical for evaluating whether regulation and treaty commitments are effective in achieving their stated goals—and whether these stated goals are sufficient. As a result, there is a need for understanding which tools—and of which complexity—are appropriate for answering this and related policy questions.

1.2 Research questions and goals

This thesis aims to make a methodological, a practical, and a policy contribution. Its methodological objective is to develop a method for quantifying the health and economic impacts of mercury exposure that better integrates environmental and economic realities. Its practical objective is to use this improved method to quantify the health and economic benefits of the most recent US mercury regulations. Its policy objective is to evaluate when such a method would be relevant for answering policy questions, and when investment in additional model completeness and complexity is worthwhile. My work is therefore centered around the following questions:

1. **Methodological:** How can we improve the environmental and economic realism of how we represent mercury's emissions-to-impacts chain in a benefits assessment tool?

2. **Practical:** What are the benefits of the most recent US mercury regulations, using this improved method?

3. **Policy:** For which, if any, policy questions is additional model complexity worthwhile? What are the additional policy-relevant insights can be gained from an assessment that more completely and realistically represents the emissions-to-impacts chain when compared to previous studies?

1.3 Structure and approach

To explore the above questions, I develop a modelling framework that integrates chemical transport modelling (CTM) of the environment with computable general equilibrium (CGE) modelling of the economy, and apply this framework to quantify benefits from a sample policy case—the Mercury and Air Toxics Standards (MATS),

which are the most recent mercury control regulation in the US (US EPA, 2011d). I focus on including global transport, ecosystem time lags, and cardiovascular effects. I compare my results to existing studies in the literature.

The remainder of this thesis is divided into five chapters. Chapter 2 provides an overview of the current state of mercury-related benefits assessment. It begins with a review of the science and policy of mercury pollution: mercury's biogeochemical cycle, human exposure pathways, mercury's health impacts, and regulatory approaches to controlling mercury emissions.

Chapter 3 addresses my first research question. It describes the modelling framework developed for this work, and how it integrates both environmental and economic considerations. It outlines how each link in the emissions-to-impacts chain is represented in the model: emissions to deposition, deposition to exposure, exposure to health effects, and health effects to wider economic effects. Specifically, it addresses the modelling of: a) how mercury emissions travel through, and are transformed in, the environment; b) the dynamics of mercury within aquatic ecosystems; c) how the pattern of global seafood trade affects exposure to mercury in US populations; d) the quantification of health impacts using epidemiological relationships; e) how these health effects can be valued in a CGE model.

Chapter 4 addresses my second research question. It applies the assessment framework developed in Chapter 3 to a sample policy case, the Mercury and Air Toxics Standards. I evaluate how MATS related emissions reductions will change total US welfare. I also explore how key uncertainties in the emissions-to-impact chain may affect these estimates—for instance, future world emissions patterns, ecosystem response times to changes in mercury inputs, and dose-response relationships between mercury exposure and health impacts.

Chapter 5 addresses my third research question, and draws some policy implications from the case study results. It explores the insights that can be gained from a more complete representation of the emissions-to-impacts path for policy design and assessment, and also situates these insights in the broader political context.

Chapter 6 summarizes key findings and recommendations from the preceding chapters, and discusses opportunities for future work.

Chapter 2

Mercury pollution

In this chapter, I review the science and policy of mercury pollution: Where does mercury come from? How do humans become exposed to it? What risks does it pose to human health? And what policy options are available to reduce this risk? Figure 2-1 on page 14 presents a high-level summary of this information. Briefly, inorganic mercury is released, by natural and anthropogenic processes, to the atmosphere, where it can be transported on local to global scales. This mercury is then deposited into surface terrestrial and aquatic ecosystems. In these ecosystems, it can be transformed into methyl-mercury (MeHg), a highly toxic and bioaccumulative organic form. MeHg biomagnifies up aquatic food chains, leading to human exposure through fish and seafood consumption. MeHg is known to have neurologic health effects in humans, though a growing body of evidence suggests that it may have cardiovascular impacts as well. Policy options for reducing the risk of these health effects can be broadly classified as either mitigation approaches—reducing emissions, or adaptation approaches—changing human behaviour, like dietary patterns, to reduce exposure.

Details about mercury's environmental path from emission source to fish and seafood are covered in Section 2.1. Section 2.2 focuses on key determinants of human exposure, and health effects are explored in Section 2.3. Existing policy approaches for controlling the risks of mercury, and their limitations, are described in Section 2.4.

2.1 Biogeochemical cycling of mercury

Although mercury is a naturally occurring substance, it is estimated that human activity like mining and fossil fuel burning has increased its environmental mobilization by three to five times since the pre-industrial period (pre-1850) (Biester et al., 2007; Lindberg et al., 2007), and by seven times when compared to natural levels (Amos et al., 2013). This enrichment has lead to greater human exposure. When mercury is removed from deep mineral reservoirs, either by natural or human-driven processes, it begins to cycle between the atmosphere, water, and land until it is sequestered

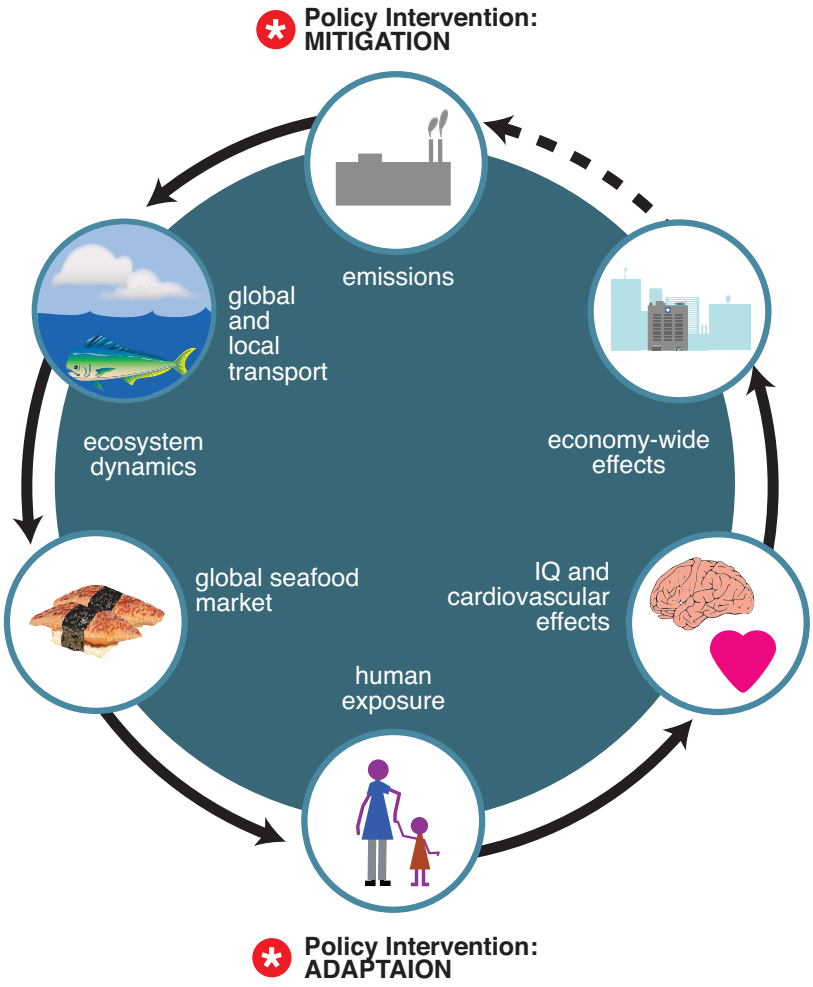


Figure 2-1: Emissions to impact cycle for the general US population.

back in deep mineral sinks, or ocean and lake sediments (UNEP, 2013a). During its time in aquatic systems, mercury can be microbially transformed into methyl-mercury (Benoit et al., 2003). Living organisms—many of which are human food sources—can become easily contaminated with this form of mercury, making it a health risk for both humans and wildlife (UNEP, 2013a). While the time-scale of mercury cycling between atmospheric and surface water and land reservoirs is from years to decades, it can take centuries for mercury to move from these pools into longer-lived reservoirs like the deep ocean and recalcitrant soil (soil from which minerals and nutrients are no longer released in soluble form), and on the order of millennia for mercury to return to its most stable mineral and sediment sinks (Selin, 2009; Amos et al., 2013). These processes are depicted in Figure 2-2, taken from Selin (2009). Therefore, when human activity mobilizes mercury from the lithosphere—or when controls are put in place to reduce mercury pollution—the effects for human exposure may be substantial and long-lasting. This section will provide an overview of the portions of the mercury biogeochemical cycle relevant to human exposure.

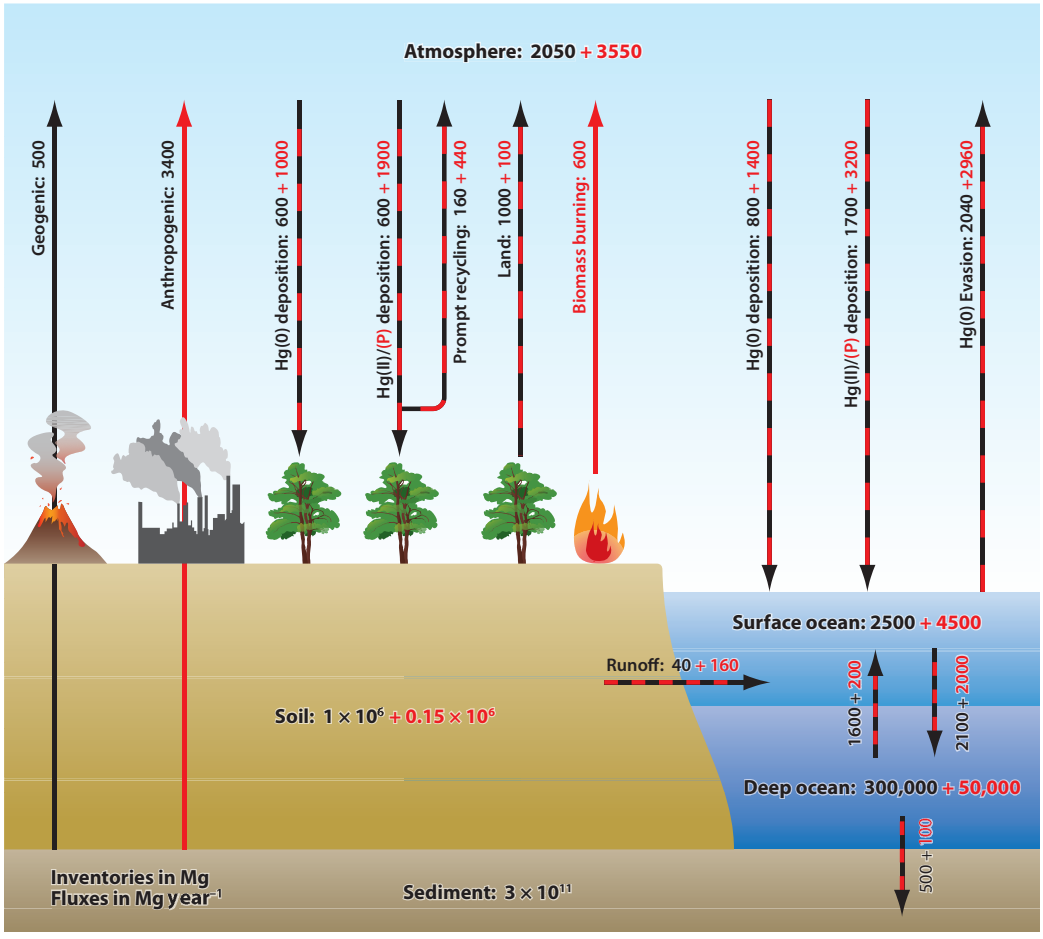


Figure 2-2: Biogeochemical cycle and global budget of mercury, from Selin (2009). Black arrows indicate natural fluxes, while red arrows indicate anthropogenic. All numbers are given in Mg/yr.

2.1.1 Emission Sources

Mercury emissions can be classified as anthropogenic, natural, or re-emissions. Though emission fluxes remain uncertain, as reviewed in Selin (2009), estimates for yearly inputs of mercury to the atmosphere range from 4400 - 11200 Mg, with approximately 30, 10, and 60 % attributed to each category respectively (UNEP, 2013a). How each of these source categories contributes to mercury pollution in the US will be addressed in Section 2.1.2.

Anthropogenic: Anthropogenic sources of mercury pollution include both emissions from intentional uses of mercury, and unintentional, "by-product" emissions (Pacyna et al., 2010a). Humans have used mercury intentionally for over 3500 years for mining/amalgam, medicinal, religious, and decorative purposes (Nriagu, 1979). Today, key intentional uses of mercury include certain products (eg. compact fluorescent lightbulbs)[1] and industrial processes (chlor-alkali, vinyl chloride monomer production), dental amalgam for cavity fillings, and artisanal and small-scale gold mining (ASGM) (Pacyna et al., 2010a). UNEP (2013a) estimates that ASGM is now the largest single source of mercury emissions globally, at 37%—though this estimate is highly uncertain.[2] The second largest source of global mercury emissions is fossil fuel burning, at 25% (UNEP, 2013a). Emissions from fossil fuel burning, as well as mining and smelting of certain ores, are examples of "by-product" emissions: mercury is a trace impurity in the raw fuel or ore, which is released to air upon burning or smelting (Pacyna et al., 2010a). For the US, by-product emissions from coal-fired power plants were the largest single-source of domestic mercury emissions in 2005 (McCarthy, 2005). The likely growth of coal-fired electricity generation capacity in the Global South—in particular in China and India—makes this source category an important one to consider from a global perspective as well (Streets et al., 2009). Overall, direct anthropogenic emissions are estimated to total 2200-4000 Mg/yr (Selin, 2009). The breakdown of these emissions by source category, from UNEP (2013a), is shown in Figure 2-3.

Natural: Mercury is naturally occurring in Earth's crust, so purely natural emission sources include volcanoes, geothermal vents, and rocks and soils that are naturally enriched with mercury (Swartzendruber & Jaffe, 2012). Selin (2009) estimates that these sources account for 500 Mg/yr of emissions.

Re-emissions: A large proportion—60%—of mercury emissions are actually legacy re-emissions, meaning that they are revolatizations of previously deposited mercury

[1] Use of mercury in products can lead to emissions through accidental releases—eg. a broken lightbulb—or from waste incineration.

[2] ASGM accounts for 30% of world gold production, and provides a livelihood for approximately 15 million people (Sippl & Selin, 2012). Artisanal miners use mercury to form amalgams with gold, facilitating the extraction and concentration of gold from ores. The amalgam is then heated, causing the mercury to evaporate as a vapour, leaving behind gold. Because the process is often conducted in the informal sector in developing countries, with minimal personal and environmental protection, it poses a risk to miners, their communities, and the local and global environment. ASGM is an example of how mercury pollution issues can intersect with those of poverty, economic development, and environmental justice.

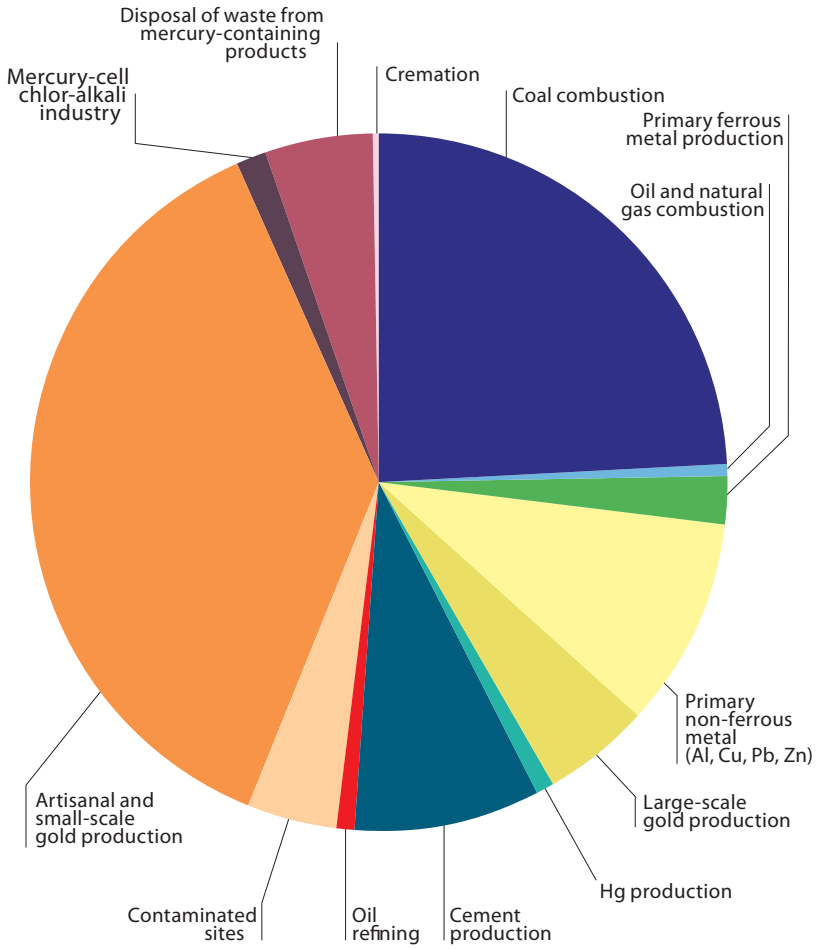


Figure 2-3: Breakdown of anthropogenic emissions, by source category, from UNEP (2013a).

from vegetation, soil, and surface waters back into air (UNEP, 2013a). Re-emissions can happen on short or long-term time scales. For instance, Selin et al. (2008) have used the term "prompt recycling" to describe the phenomenon whereby recently deposited mercury is in fact more likely to return to the air from surface land, water, and snow, within days and months. Based on empirical studies, it has been estimated that 5-60% of deposited Hg(II)—a particular form of mercury that I will describe further in the next section—is recycled in this way (Hintelmann et al., 2002; Amyot et al., 2004; Lalonde et al., 2001; Ferrari et al., 2005). In general, re-emissions contribute to the difficulty of tracking the path from emissions to impact. This points to the potential utility of using computational models to investigate the environmental behaviour of mercury in conjunction with empirical studies. In fact, computational environmental models have become a critical part of how the EPA evaluates the impacts of its policies (US EPA, 2011c,d).

It is also important to make the distinction between re-emissions and natural emissions—though these re-emissions are from "natural" sources, they represent the legacy of prior emissions, which may be anthropogenic or natural. It is assumed that the attribution of re-emissions between natural and anthropogenic sources likely mirrors the ratios in which they were first emitted. Amos et al. (2013) have found, through a modelling study, that only 17% of the mercury currently in surface oceans (and consequently available for re-emission) is natural, and that half of the remaining 83% is legacy mercury from pre-1950 anthropogenic sources. These legacy effects highlight the long timescale of the mercury challenge: persistence in surface reservoirs over time can create a lag-time between emission reductions and observed levels in the environment.

2.1.2 Global and local atmospheric transport

Mercury pollution can act on a range of spatial scales, from local to global (Selin, 2011). This behaviour depends on the chemical form in which mercury exists in the atmosphere: gaseous elemental mercury, Hg(0), or divalent mercury, Hg(II), which can either be in reactive gaseous (RGM), or particulate-bound (PBM) form. Divalent mercury is very water soluble, leading to a short atmospheric life-time (days to weeks) (Selin, 2009). It is as Hg(II) that mercury is predominantly deposited into surface ecosystems, through both wet (ie. rain and snow) and dry (eg. gravitational settling, surface adsorption and absorption) deposition. Mercury emitted as Hg(II) is therefore much more likely to contribute to local and regional pollution. As an example, proximity to Hg(II) emission sources was found to be a contributing factor in the formation of hot spots—areas of elevated mercury concentration in biota—in the US Northeast (Evers et al., 2007). In contrast, in its gaseous elemental form, mercury is relatively stable in the atmosphere, with a lifetime of six months to a year (Selin, 2009). In this form, mercury is capable of long-range transport. In fact, increasing mercury concentrations in Arctic wildlife—an ecosystem remote from sources—can be explained by global transport of Hg(0) (AMAP, 2011). Hg(0) can enter ecosystems either through dry deposition, or through transformation to Hg(II) with subsequent,

rapid wet or dry deposition (Lindberg et al., 2007).

Because mercury deposition patterns depend on speciation (ie. what chemical form mercury is in), understanding the speciation of emissions, and the processes that transform one form of mercury to another is important for benefits assessment. These atmospheric processes are summarized in Figure 2-4. Unfortunately, the exact mechanisms for the oxidation of Hg(0) to Hg(II), and reduction in the opposite direction, are still poorly understood (see Steffen et al. (2008), Hynes et al. (2009), Obrist et al. (2010), and Holmes et al. (2010) for a discussion of potential oxidation mechanisms). In particular, uncertainty about atmospheric reduction processes can intersect with uncertainty about emissions inventories, making it a relevant topic for analyses that evaluate emissions regulations, this thesis included. Edgerton et al. (2006) and Weiss-Penzias et al. (2011) have observed that the ratio of $\frac{Hg(II)}{Hg(II)+Hg(0)}$ downwind of coal-fired power plants (CFPPs), a major source of mercury pollution in the US, is 3 to 5 times lower than that observed in the stack gases at point of emission. Since total gaseous mercury (Hg(II)+Hg(0)) is essentially conserved between the stack and downwind locations, Lohman et al. (2006) have proposed that reduction of Hg(0) to Hg(II) in the plume by SO_2 (another pollutant generated by CFPPs) may explain the discrepancy. Vijayaraghavan et al. (2008) and Zhang et al. (2012) have implemented this "in-plume reduction" (IPR) hypothesis in regional and nested-grid global mercury models, respectively, and have found improved correlations between modelled and observed wet deposition over the contiguous US. Amos et al. (2012) also implemented emissions speciations based on the IPR assumption (along with an empirical gas-particle partitioning relationship), and found improved model performance for RGM and PBM concentrations at five North American surface sites. If the IPR hypothesis is correct, then emissions from CFPPs, and other sources that have SO_2 in their flue gas, like waste incinerators (Zhang et al., 2012), will likely have a much more global impact than previously expected, as emissions partitioning skews towards Hg(0).

The atmospheric processes discussed above make mercury a local, regional, and global policy challenge, the solution to which will require coordination and cooperation at multiple levels of governance (Selin, 2011). Many modelling studies have explored the extent to which domestic action alone could reduce deposition of mercury in the US, and found that primary US sources (ie. current anthropogenic) are responsible for only 17 - 32% of deposition over the contiguous US (Seigneur et al., 2004; Selin & Jacob, 2008; Corbitt et al., 2011). Selin et al. (2008) found that foreign anthropogenic sources contribute 22% to US deposition, legacy anthropogenic sources 16%, and natural sources 32%. Results from Corbitt et al. (2011) are comparable, with foreign anthropogenic sources contributing 23%, and re-emissions (which include legacy anthropogenic and recycled natural sources) 60%. It should be noted that the North American contribution to legacy anthropogenic re-emissions may be significant, given the large historical mining emissions from the gold and silver rush (Pirrone et al., 1998; Streets et al., 2011). Nevertheless, these source attribution findings highlight the need, from a US perspective, for global cooperation to reduce mercury inputs into domestic ecosystems.

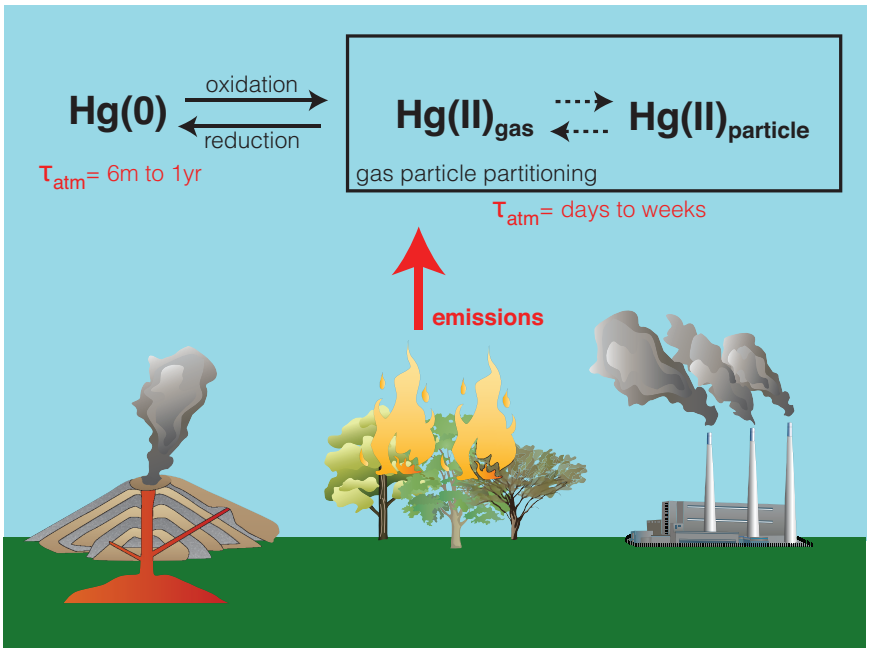


Figure 2-4: Atmospheric transformation processes for mercury.

2.1.3 Ecosystem dynamics

Mercury enters surface ecosystems through wet and dry deposition. In the terrestrial system, this deposition can enrich soil and vegetation directly, and soil indirectly through litterfall of plant debris (Amirbahman & Fernandez, 2012). As mentioned previously, a portion of this deposited mercury is promptly recycled to the atmosphere as Hg(0) (Hintelmann et al., 2002), while the rest is eventually incorporated into the soil pool (Selin, 2009). Soil has a large storage capacity for mercury, and it is estimated that more than 80% of mercury deposited from the atmosphere to terrestrial systems is bound in this medium (see Shanley & Bishop (2012) and Amirbahman & Fernandez (2012) for a review of the relevant studies). Shanley & Bishop (2012) argue that this large soil pool means that aquatic systems are buffered from the full impact of atmospheric inputs in the short-term, but that in the long-term, soil can act as a source. As a result, it may take these ecosystems longer than expected to respond to any decreases in atmospheric emissions.

Aquatic ecosystems, both freshwater and ocean, are particularly important from the perspective of human exposure; it is in these environments that inorganic mercury is transformed to MeHg, and that MeHg bioaccumulates in fish and seafood—important human food sources. For freshwater ecosystems, mercury inputs may occur through atmospheric deposition, transport from terrestrial watersheds (ie. drainage from soil reservoirs), or from point sources like contaminated industrial sites (Driscoll et al., 2007). For oceans, the largest mercury input is atmospheric deposition (Mason & Sheu, 2002; Soerensen et al., 2010), however, inputs from rivers may also be important, depending on the ocean basin, and for coastal ecosystems (Sunderland & Mason, 2007). Other smaller sources include groundwater, benthic sediment flux, and

geothermal vents (Mason et al., 2012).

Once introduced to an aquatic system, a small amount of this inorganic mercury is transformed into MeHg by bacteria, most importantly, sulfate-reducing bacteria (Benoit et al., 2003). These bacteria primarily exist in anaerobic environments, such as freshwater sediment beds and wetlands (Chen et al., 2012b). The production of MeHg in oceans is still poorly understood. MeHg has been observed in ocean water columns but it is unclear whether production is occurring *in situ*, or in coastal zones, with subsequent transport to open oceans—this remains an active area of research (Mason et al., 2012). MeHg is highly toxic, bioavailable, and bioaccumulative (Swartzendruber & Jaffe, 2012). It binds to the proteins and free amino acids within muscle tissue, and therefore biomagnifies at each trophic level of a food chain (Mergler et al., 2007).

A common assumption in regulatory analyses has been that water (and subsequently fish) concentrations of mercury will decrease instantaneously in proportion to changes in mercury input to the water body (US EPA, 2011d); however, current knowledge of ecosystem dynamics suggests that there is a lag time between these linked changes (Knightes et al., 2009; Chen et al., 2012a). A recent study modelling five representative freshwater ecosystems indicates that response to reductions in mercury inputs occurs in two phases: up to 60% of the reductions will be reflected in water and fish concentrations relatively quickly, within a range of years to decades, however the full reductions, taking place in the slower second phase, will not be observed for decades to centuries (Knightes et al., 2009). These time lags are associated with continued mercury loading from soil, even after inputs from atmospheric deposition are reduced (Chan et al., 2012), and other factors controlling transformation (from inorganic mercury to MeHg) and biomagnification up food chains. Knightes et al. (2009) found that larger watersheds, and more complex food chains both contributed to longer lag times.

For oceans, quantitative models linking marine fish tissue concentrations of mercury to changes in inputs are scarce, so the assumption that changes in water mercury concentrations will be reflected proportionally in fish is commonly used. Chen et al. (2012a) provide a synthesis of recent studies on mercury cycling in different ocean systems; these range from open ocean systems, for which atmospheric deposition is the primary mercury input ($\sim 90\%$), to coastal watershed systems, like the San Francisco Bay, that predominantly receive mercury from river inputs. Watershed-fed coastal systems may respond in a range of decades to centuries to reductions in atmospheric deposition, depending on region (and relative importance of watershed inputs) (Chen et al., 2012a), In contrast, surface waters (uppermost 300 m) of open oceans will likely respond within years to decades to decreases in deposition, though it will take on the order of centuries to millennia for these changes to be reflected in the intermediate and deep oceans (Sunderland, 2007; Chen et al., 2012a). However, Mason et al. (2012) argue that MeHg production and biomagnification (through fish foraging) occurs in these surface waters, suggesting that open ocean fish concentrations will likely respond relatively rapidly as well. As a summary, Table 2.1 shows the number of years required for different aquatic systems to reflect different percentages of a reduction in atmospheric deposition. These results assume that surface waters are already at

Table 2.1: Aquatic response times to changes in atmospheric deposition, reported as number of years required to reflect different percentages of a steady-state reduction in deposition. Data for freshwater lakes is from Knightes et al. (2009) and includes farm ponds, seepage lakes, stratified drainage lakes, and bay lakes. Data for surface open oceans is from Sunderland & Mason (2007) and includes the surface Atlantic, Mediterranean, and Pacific/Indian oceans. The approximate range for watershed-fed coastal systems is given in Chen et al. (2012a). Entries marked n/a were not explicitly given in these studies.

	Freshwater Lake (yr)	Surface Open Ocean (yr)	Watershed-fed Coastal
20%	0.3-5	n/a	n/a
40%	0.6-27	n/a	n/a
60%	0.8-76	n/a	n/a
80%	0.8-200	n/a	n/a
95%	n/a	10-50	10-100

steady-state with the atmosphere when reductions in deposition begin, however, for the ocean, this assumption may not be valid; given the current ocean budget of mercury, it is likely that mercury concentrations will actually increase in the near-term, if deposition is held constant (Sunderland & Mason, 2007).

Mercury can have negative health effects at an ecosystem level: toxic effects targeting nervous, immune, and reproductive systems have been documented at current concentrations of mercury in wildlife (Wolfe et al., 1998). Even at levels below the fish tissue concentrated deemed safe by the EPA (Sunderland & Selin, 2013), fish can experience reproductive effects (Depew et al., 2012). In the Arctic, an ecosystem vulnerable to contamination, many biota exceed concentrations of MeHg that are thought to lead to biological effects (including polar bears, and some species of whales, seals, fish, and birds) (Dietz et al., 2013). Loons and songbirds in North America are also thought to be particularly at risk of toxicological effects from MeHg (Evers et al., 2008).

2.2 Determinants of exposure

Humans can be exposed to mercury in three forms: elemental mercury vapour, inorganic compounds containing mercury, and organic mercury compounds (UNEP, 2002). In this chapter (and thesis as a whole), I have focused on methyl-mercury (MeHg), an organic compound, because of its extreme toxicity and its ubiquitous presence in fish and seafood, a staple protein source for US (and other global) populations (McKelvey & Oken, 2012). For the general US population, MeHg through consumption of fish and seafood is the dominant mercury exposure pathway (UNEP, 2002).[3] However, for individuals who are occupationally exposed to elemental mercury

[3]Acute MeHg poisoning—called Minamata disease—incidents have occurred in Minamata, Japan (Harada, 1995), Grassy Narrows and Whitedog, Canada (Wheatley & Paradis, 1995), and throughout

(eg. dentists or artisanal and small-scale gold miners), those who use mercury containing medicinal products, and those exposed to elemental mercury or other mercury compounds through accidental releases from products, these other exposure pathways may be more relevant (Mergler et al., 2007). McKelvey & Oken (2012) and UNEP (2002) provide a comprehensive review of these and other routes of mercury exposure.

In this section, I review the key determinants of exposure for those primarily exposed to MeHg through fish and seafood. Ultimately, exposure depends on an individual's consumption patterns—what kind of fish are eaten and their source (e.g. wild Atlantic salmon), and then how much fish is eaten (e.g. 3 fish meals a week) (Carrington & Bolger, 2002; Mahaffey et al., 2004). Certain populations within the US are more vulnerable to mercury-related harms, and these groups require explicit consideration when regulators try to evaluate and manage risk, as discussed in the Section 2.4.3.

2.2.1 Variability in fish tissue concentrations

Concentrations of tissue mercury in fish can be highly variable due to "source, size, and species" (Dellinger et al., 2012).

Species: Different species may have different food web structures and dietary preferences (Sunderland, 2007); for instance, top-level piscovorious (fish-eating) species are expected to have higher concentrations of mercury than bottom-of-the-chain plankton eaters.

Size: Several studies have found positive correlations between fish size and mercury concentration (for instance Scott & Armsrong (1972); Storelli et al. (1998, 2007)). Within a species, fish size is typically related to age, and as MeHg is persistent in the muscle tissue of fish, the older a specimen is, the more time it has had to bioaccumulate toxins (Storelli et al., 1998).

Source: Recent studies have highlighted the importance of geographic origin as a determinant of mercury concentrations.

Marine (ocean) and estaurine (coastal) fish are sourced from around the world, and demonstrate significant variability, even within a species category. For example, even after normalizing for weight, mercury concentrations in tuna differ in Atlantic, Pacific, and Mediterranean oceans (Rasmussen et al., 2005). This finding is partially demonstrated in Figure 2-5, created with data from Sunderland (2007). Figure 2-5 shows the percentage of total mercury intake from marine and estaurine sources, by species category, for the average US consumer.[4] The category for tuna is broken out into its different source origins.

Source can also be an important determinant of mercury concentration for fresh-

Iraq (Bakir et al., 1973). In all three cases, the exposure pathway differed from that illustrated in Figure 2-1. In the first two cases, mercury-containing industrial effluent was directly discharged into aquatic systems where communities fished. In the third, seeds coated with a mercuric fungicide were baked into bread.

[4]These values do not apply to individuals that primarily eat locally caught fish.

water fish. Though fish from these sources do not represent a large chunk of the commercial market, they are important food sources for many of the vulnerable populations identified previously. Location-specific environmental conditions like pH, acid-neutralizing capability, proximity to wetlands, and watershed size can affect fish mercury concentrations (Evers et al., 2011a).

Aquaculture—fish farming—is an increasingly important source of seafood in the US. In 2010, half of all imported seafood was farmed, and approximately 5% of all fish eaten in the US came from domestic farms (NOAA, 2010). Aquaculture can be marine or freshwater. In both cases, fish are fed controlled diets composed of wild fish from local and global sources (Jardine et al., 2009). Contaminants present in wild fish, including mercury, may therefore be found in farmed fish as well. Studies comparing mercury concentrations in farmed and wild fish have reached varying conclusions, often depending on species—understanding these differences remains an active area of research (Jardine et al., 2009).

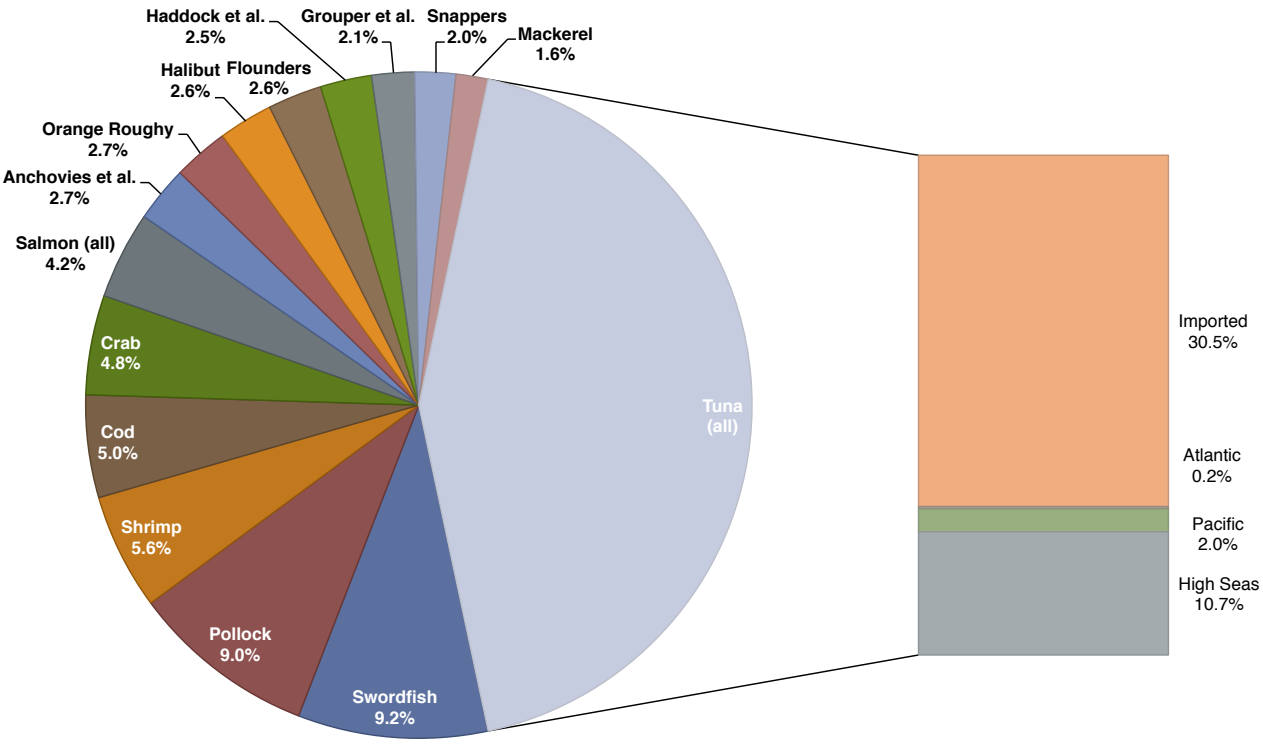


Figure 2-5: Percentage of total mercury intake, by species category, for the average consumer. Data is from Sunderland (2007). These values do not apply to populations that are highly exposed to locally caught fish.

2.2.2 Dietary patterns and vulnerable populations

In the US, Mahaffey et al. (2004) estimate that approximately 9% of women eat fish meals at least once a week, leading to an average daily intake of 1.8 g of fish

and shellfish.[5] More than 90% of fish sold in the commercial US market are from marine or estaurine sources (Sunderland, 2007). Of these marine and estaurine fish, top trophic level predators like shark, swordfish, tilefish, and bluefin tuna (typically used in sashimi), generally have the highest levels of MeHg (Mahaffey et al., 2011). However, for the average US consumer, close to 40% of MeHg intake comes from tuna, and particularly canned tuna (Macintosh et al., 1996; Sunderland, 2007). Other top contributors include swordfish, pollock, shrimp, and cod (Sunderland, 2007).

However, some subpopulations in the US consume fish substantially more frequently than the total population average (Moya, 2004). Recreational anglers, subsistence fishers, some indigenous peoples, and other individuals for whom eating fish is culturally, spiritually, or socioeconmically important, belong to this category, and can be considered vulnerable populations because of their systematically higher exposure to mercury (Dellinger et al., 2012). Moya (2004) found that average fish intake ranged from 5-70 g/day for recreational fishers, and 57-271 g/day for Native American populations. In comparison, the population average (for women of child-bearing age) is 1.8 g/day (Mahaffey et al., 2004). These differences suggest that the US exposure profile is very long-tailed, with some populations experiencing significantly elevated exposure compared to the general population. Mercury can therefore be seen as an environmental justice issue (Nriagu et al., 2012). Many of these groups identified rely more heavily on locally caught freshwater and marine fish than the general population, making pollution in US waters particularly relevant for their exposure (Selin et al., 2010).

Populations can also be vulnerable because they have higher sensitivity to mercury's effects (Dellinger et al., 2012). Pregnant women—because developing fetuses are more sensitive to toxins, and children are more susceptible to mercury's neurologic effects, discussed further in Section 2.3 (Dellinger et al., 2012). As a result, dietary guidelines–which fish to eat, and recommended frequency—is often provided to these groups (Oken et al., 2012).

Though fish may be a primary source of MeHg (and other contaminants), there are also substantial benefits to their consumption, nutritional (Mozaffarian & Rimm, 2006; Oken et al., 2012), and sociocultural (Kuhnlein & Receveur, 1996)—many of which are particularly pronounced for vulnerable populations. Fish are rich in protein and n-3 (omega-3) long-chain polyunsaturated fatty acids (n-3 PUFAs) (Oken et al., 2012). These fatty acids can have positive cardiovascular and cognitive effects, even for pregnant women and their children, so the risks of MeHg must be balanced against these benefits (Mahaffey et al., 2011). Self-caught fish can be an affordable and convenient source of these nutrients. Fishing is also part of many traditional food systems, and therefore cultural heritage. Though it is not always possible, especially for vulnerable populations whose dietary choices are motivated by remote locations, and cultural and socio-economic concerns, choosing fish that are both low in MeHg and high in n-3 PUFAs may be an option for minimizing exposure (Mahaffey et al., 2011).

[5]Many of the population-wide studies of mercury exposure focus on women of childbearing age due to the known adverse effects of MeHg on fetal neurodevelopment.

2.3 Human health effects of mercury

The dangers that MeHg poses to human health became well-recognized in the 1950s when observed on a large scale in communities in the Minamata Bay region of Japan (McAlpine & Araki, 1958). Because industrial effluent containing mercury was directly discharged into the bay, severe MeHg poisoning—now called Minamata disease—occurred in the surrounding fishing communities (Harada, 1995). MeHg easily crosses the blood-brain barrier, and Minamata disease's key target is the brain and nervous system (Mergler et al., 2007). It is characterized by loss of coordination, auditory, visual, and other sensory disturbances, dysarthria (a motor speech disorder), and numbness in limbs (Clarkson, 2002). MeHg also crosses the blood-placenta barrier, and due to the increased sensitivity of the fetal nervous system, children exposed *in utero* also experienced cerebral palsy-like symptoms and mental retardation, even when mothers did not exhibit Minamata disease (Harada, 1995; Mergler et al., 2007). Other major poisoning events occurred in Canada in the 1960s (Wheatley & Paradis, 1995), and in Iraq in the 1970s (Bakir et al., 1973).

Minamata disease remains the "face" of mercury's health effects for many (Clarkson, 2002), however, today, exposures typically experienced by populations worldwide, through the pathway described in Section 2.2, are at much lower levels. In this thesis, I focus on the effects of chronic, low-to-moderate dose exposure to MeHg. I draw heavily from comprehensive reviews by Karagas et al. (2012), Mergler et al. (2007), and the US National Research Council (2000).

2.3.1 Pharmacokinetics

The pharmacokinetics of MeHg are well reviewed in ATSDR (1999) and NRC (2000), so I offer here only a brief summary. It is estimated that 95% of ingested MeHg is absorbed through the gastrointestinal tract, after which it quickly spreads to all parts of the body—including the brain. In pregnant women, MeHg is actively transferred from blood to placenta, and fetal levels of mercury are assumed to be an average of 70% higher than maternal levels (Mergler et al., 2007). In tissues, MeHg is slowly converted to inorganic mercury compounds or cations (Hg^{2+}), and eventually excreted in this form, primarily through feces. In the brain however, conversion to inorganic mercury may trap it there. Overall, the half-time of MeHg in the body is about 45-65 days.

Mercury is typically measured in blood and hair, as the mercury measured in these tissues are primarily MeHg (Karagas et al., 2012). These biomarker concentrations are then used to develop epidemiological dose-response relationships, which relate body concentrations of mercury (dose) to health effects (response). Blood and hair mercury are considered to be more accurate measures of exposure than dietary assessment—because of the variability in fish concentrations (see Sec. 2.2), and because for most individuals, ability to recall past consumption may be unreliable (Karagas et al., 2012). However, when consumption patterns of fish are stable, in frequency and over time, then blood and hair mercury can be linearly related to dietary intake (Mahaffey

et al., 2004; McDowell et al., 2004). In the US, the population average blood and hair mercury concentrations, for women of childbearing age, are 1.02 μg/L blood (Mahaffey et al., 2004), and 0.47 μg/g hair (McDowell et al., 2004), respectively.

2.3.2 Cognitive effects

The events in Minamata suggested that even at low-levels of MeHg, children exposed *in utero* may experience negative neurological effects. This conclusion has been supported by animal studies, including those with non-human primates (NRC, 2000). However, epidemiological studies are typically given the most weight in clarifying links between exposure and population-level health effects. Three large-scale, prospective cohort studies—studies that follow a cohort of similar individuals over time—have been conducted to explore the links between chronic, low-to-moderate maternal MeHg exposure and cognitive and behavioural outcomes in children. These studies evaluated endpoints like attention, fine-motor ability, language and visual-spatial skills, and verbal memory (NRC, 2000). Two studies, one conducted in the Faroe Islands (Grandjean et al., 1997), and the other in New Zealand (Crump et al., 1998), found a negative correlation between prenatal exposure and children's test scores (with one extreme outlier excluded in the New Zealand study). In contrast, a study from the Seychelles Islands (Davidson et al., 1998) found no association. In their review of these studies, NRC (2000) concluded that all three studies were well designed and conducted. (Table 2.2 compares some key characteristics of each study. Table 2.3 shows US hair mercury concentrations to put the exposures observed in the studies into better context.) For regulatory purposes however, the NRC recommended that the EPA use results from the Faroe Islands (for reasons outlined in NRC (2000)). Based on this study, NRC (2000) recommended a reference dose—a level of daily oral exposure at or below which an individual is "likely to be without an appreciable risk of deleterious effects during a lifetime" (qtd. in Rice, 2004)—of 0.1 μg/kg body weight a day. However, some have been critical of the reference dose approach because there is no clear evidence that there is a threshold to mercury-related IQ effects (Rice, 2004). Others have argued that the exposure limit should be reduced by approximately 50% because of imprecision in exposure biomarkers, and failure to account for confounding factors like n-3 PUFAs, which may mask negative effects of mercury (Grandjean & Budtz-Jørgensen, 2007; Budtz-Jørgensen et al., 2007).

Since 2000, new results and analyses have provided additional insight into the nature of the dose-response relationship between maternal MeHg exposure, and child IQ (a global measure of cognitive development). Results from the more recent instalments of the Seychelles Islands study have indicated a potential convergence in results with the New Zealand and Faroe Islands studies (Mergler et al., 2007). Integrative meta-analyses of all three studies by Cohen et al. (2005) and Axelrad et al. (2007) have estimated 0.7 and 0.18 point reductions in IQ per ug mercury/g maternal hair respectively (using weighting and Bayesian approaches, respectively). Both studies assume a linear relationship, and no threshold. Karagas et al. (2012), in their review of the most recent findings, suggest that memory, verbal skills, and visual-motor skills

Table 2.2: Comparison of epidemiological studies evaluating neurologic effects of *in utero* exposure to mercury. Information drawn from NRC (2000). For the New Zealand study, the reported avg. maternal hair mercury concentration excludes the outlier.

	Faroe Islands	Seychelles Islands	New Zealand
Biomarker of Hg Exposure	umbilical cord blood	maternal hair	maternal hair
Neurobehavioural Tests	focused, domain-specific	omnibus, integrative	omnibus, integrative
Pattern of Exposure	3 fish dinners/week and episodic (<1/month) whale consumption	12 fish meals/week	>3 fish meals/week
Average Maternal Hair Hg	3 ug/g	7 ug/g	20 ug/g

may be particularly sensitive to MeHg. They also argue that accounting for confounding factors, like n-3 PUFAs in fish, can have a large effect on results. In summary, researchers and regulators have found the existing evidence sufficiently compelling to conclude that there is likely a relationship between prenatal MeHg exposure and IQ reductions, though there is some uncertainty about the magnitude and shape of this effect. More discussion on the implications of different epidemiological assumptions for benefits accounting (eg. presence of a threshold, shape of the dose-response curve) is provided in Section 3.3.2.

Table 2.3: Hair mercury concentrations in the US, from 1999-2000. Data from Mahaffey et al. (2004).

		Percentile	
	Mean	10th	95th
US females aged 16-49	0.47 ug/g	0.04 ug/g	1.73 ug/g
High fish consumption (<3 meals a week)	0.77 ug/g	0.09 ug/g	2.75 ug/g

2.3.3 Cardiovascular effects

In addition to its neurological effects, there has been increasing concern about MeHg's potential impacts on cardiovascular end points like acute myocardial infarctions (AMIs), hypertension, and changes in heart rate variability (Mergler et al., 2007). While the toxicological mechanisms are not yet fully understood, it is believed that MeHg may reduce the body's anti-oxidative capacity resulting in lipid peroxidation—a believed contributor to cardiovascular stress (Virtanen et al., 2007). As noted by (Karagas et al., 2012), a conclusive pattern has not yet emerged from the limited evidence available; however, the strongest evidence available is for AMIs (heart attacks) (Roman et al., 2011). Existing evidence (which focuses on adult men) suggests that even at relatively low levels of hair mercury, the risk of heart attack may be increased, with approximate doubling of risk between the highest and lowest exposed groups (Guallar et al., 2002; Virtanen et al., 2005).

Though the relationship between MeHg and cardiovascular risk is much less well established than that between MeHg and IQ, the high human and economic cost of cardiovascular disease makes this relationship important to consider from a regulatory standpoint. In fact, in their probabilistic characterization of the benefits of MeHg reduction, Rice et al. (2010) attributed 80% of the benefits to reductions in fatal heart attacks. From an economic accounting perspective, not only do AMIs result in high medical service costs (Wittels et al., 1990; Eisenstein et al., 2001), but they cause lost work and leisure time, as well as foregone future earnings in the case of fatal heart attacks. These issues are further discussed in Section 3.3.3.

2.3.4 Other health effects

There is some evidence from animal studies that MeHg can have autoimmune and immunosuppressive effects (Häggqvist et al., 2005), however, the lack of large-scale epidemiological studies, particularly those that have adequately controlled for confounders, has made it difficult to draw conclusions about these immune system effects in humans (Karagas et al., 2012).

2.4 Policy approaches to risk management

There are two major policy approaches for reducing the risks that mercury poses to human health: mitigation and adaptation. Following Selin (2011), I borrow these terms from the climate change community. By mitigation, I refer to reductions in emissions of mercury to prevent future exposures, and by adaptation, I mean changes in human responses to existing threats—for instance, changing fishing and consumption patterns. In this section, I review existing and forthcoming local, regional, domestic, and international policies, and how they use both of these approaches.

2.4.1 Mitigation: regulating emissions in the US

At the federal level, mercury emissions to air, and releases to water through manufacturing, use, and disposal are regulated—a form of risk prevention and mitigation. I focus on regulations of mercury emissions to air, because for most waterbodies relevant to US exposure, deposition form air is the dominant input source. For a summary of federal action on releases of mercury to water, and mercury in products and processes, consult US EPA (2013c).

The US EPA was given a congressional mandate to regulate mercury emissions to air with the 1990 amendments to the Clean Air Act (42 U.S.C. 7412) (McCarthy, 2005). Under these amendments—specifically, to Sections 112 and 129—mercury was categorized as a Hazardous Air Pollutant (HAP), and the EPA was therefore required to develop emission standards for major sources. Table 2.4, adapted from McCarthy (2005), shows these major sources and dates of first regulation. Emissions standards were to use a Maximum Achievable Control Technology (MACT) approach. MACT essentially requires that newly-built sources put in place best demonstrated control technologies, and that existing sources achieve, at a minimum, the average emissions of the best performing 12% of source units in a given category. At the time of the amendments, coal-fired power plants and waste incineration were the two largest sources. Following stringent MACT regulation of municipal waste combustors and medical waste incinerators in the late 1990s, these sources reduced their emissions by 90% when compared to 1990 levels (O'Neill, 2009). By 2005, coal-fired power plants (CFPPs) were the largest un-regulated source category for mercury, representing 50% of all domestic emissions. Figure 2-6 shows these trends graphically.

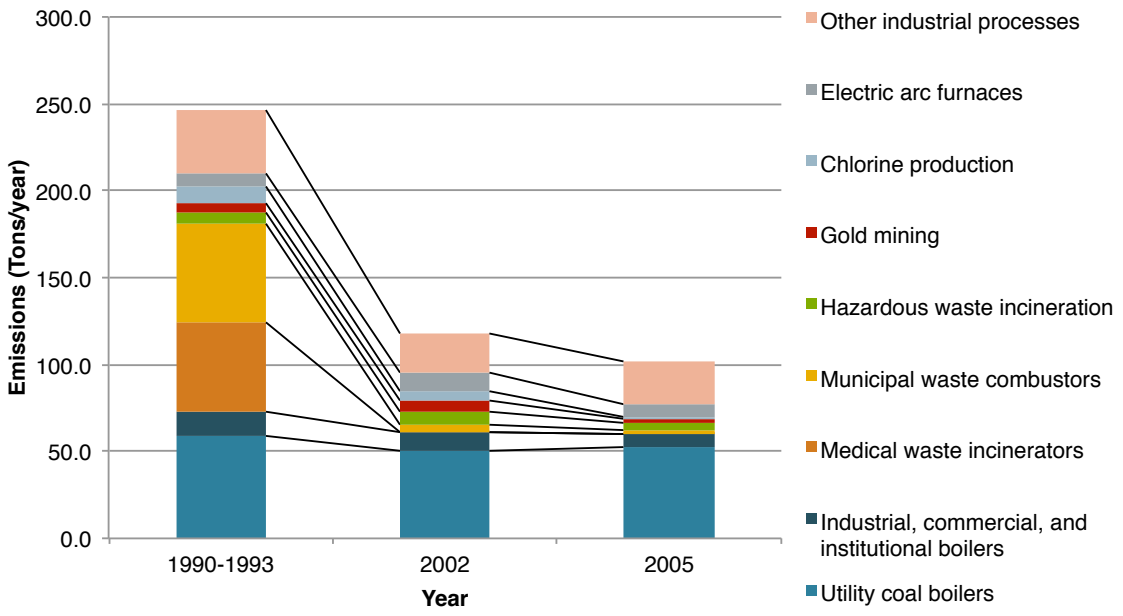


Figure 2-6: US emission trends from 1990-2005. Compiled with data from US EPA (2009). Mobile sources are not shown because they were not included in inventories prior to 2002.

The path to regulating power plant emissions of mercury has been long and difficult. Though regulations for waste incinerators were established in the 1990s, the EPA did not even determine that it was "appropriate and necessary" (in the language of statutory law) to regulate emissions from CFPPs through MACT until 2000 (Milford & Pienciak, 2009). By May 2005, however, when the EPA issued its final Clean Air Mercury Rule (CAMR), it had opted for a cap-and-trade approach to regulating emissions (under Section 111 of the CAA), rather than a MACT standard (under Section 112). These changes in approach demonstrate how regulatory priorities and approaches can change under different presidential administrations—MACT standards for mercury may have been a higher priority under President Clinton (1993-2001)—and subsequently President Obama (2009-present)—whereas President Bush's administration (2001-2008) preferred market based approaches.

Market-based cap-and-trade systems—where a cap is set on total allowed emissions, and allowances/permits to emit are traded amongst polluters—are typically more cost-effective than command-and-control policies, like technology or performance standards. Lowest total costs are achieved when all firms in a regulated industry face the same marginal abatement costs, and command-and-control policies are often at a disadvantage because the government lacks complete information (Goulder & Parry, 2008). In an emissions trading system, the market for allowances implicitly solves this information problem (Goulder & Parry, 2008). Furthermore, an emissions trading program offers economic incentives for the industry itself to innovate towards better control technologies.

However, critics of CAMR were concerned with: a) the possibility that a cap-and-trade approach would create or exacerbate existing mercury hot spots (Beusse, 2006), b) the stringency and timing of the cap (McCarthy, 2005), and c) its legality (Milford & Pienciak, 2009). Cap-and-trade systems assume that a pollutant is well-mixed, so that reductions in one region are equivalent to those in another. For mercury, this is not the case (see Section 2.1.2), leading to a concern about hot spots. Given the time trajectory of the caps, it was possible that emissions reductions similar to those of a MACT policy would not be fully reached until after 2020 (McCarthy, 2005). And finally, from a legal perspective, it was not clear that the EPA had the authority to regulate mercury using a market-based approach rather than with MACT.

In the US, citizens, industry, and even states and municipalities, can exert pressure on regulatory agencies through the legal system. In 2008, the District of Columbia Circuit Court vacated the rule in response to a petition for review by 17 states, the National Congress of American Indians and Treaty Tribes, and several other citizen groups (Milford & Pienciak, 2009). In 2009, the EPA signalled its intention to develop MACT rules for all HAPs from CFPPs, under Section 112 of the CAA (Rallo et al., 2012). In 2011, the EPA promulgated the Mercury and Air Toxics Standards (MATS), with updates for new power plants finalized in 2013 (US EPA, 2013b). This new rule targets mercury, arsenic, chromium, nickel, and acid gases. It calls for 90% mercury removal efficiency from coal, and in achieving these reductions, also offers substantial co-benefits (reductions in pollutants that are not the target of the policy) for pollutants like SO_2 and particulate matter (US EPA, 2013c). For more details

about MATS, consult US EPA (2011d).

It should be noted that substantial progress towards environmental goals can be reached through co-benefits and action on sub-national scales: despite the regulatory ambiguity, emissions from CFPPs decreased substantially between 2005 and 2011, from about 53 to 29 tons/yr (Houyoux & Strum, 2011). This decrease has been attributed to co-benefits from air quality policies like the Clean-Air Interstate Rule (CAIR)[6], and regulations at the state-level (Milford & Pienciak, 2009).

2.4.2 Mitigation: regulating emissions globally

Recognition that mercury is a global problem due to its atmospheric processes, and its global trade (within products and as a commodity) has prompted international action to address this pollutant. For the US specifically, where 23% of domestic deposition can be directly attributed to foreign sources (Corbitt et al., 2011), and whose population consumes fish from global seafood sources (Sunderland, 2007), global action on mercury can have significant implications for domestic health and welfare. I explore this idea further in my analysis in Chapter 4.

International cooperation on mercury began on a regional scale (eg. North America, Arctic countries), but has since expanded to being truly global in scope (Selin, 2011). Mercury has been included in the lists of contaminants regulated by global treaties such as the Basel Convention on the Control of Transboundary Movements of Hazardous Wastes and Their Disposal, the Convention on the Protection of the Marine Environment of the Baltic Sea Area (Helsinki Convention), and the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade (Selin, 2005). However, the push for a dedicated global environmental agreement for mercury began in 2001 when the UN Environment Programme (UNEP) commissioned a scientific and policy assessment that evaluated whether mercury had global effects, and required global action (UNEP, 2002). In 2003, based on the evidence presented in the assessment, the UNEP Governing Council concluded that global action on mercury was warranted—but there was disagreement on what form this action should take. One group of countries, including the European Union, was in favour of a legally binding treaty, while others, including the US, India, and China, felt that this was unnecessary—the compromise solution involved voluntary bilateral relationships for technical assistance, capacity building, and scientific assessment (Selin, 2011).

As mentioned previously however, regulatory priorities can change under different presidential administrations, and in 2009, under President Obama, the US began to endorse a legally-binding global instrument for mercury pollution (Selin, 2011).

[6]The DC Circuit Court actually vacated CAIR in 2008 as well, though subsequently decided to remand it instead so that it would be in place temporarily while the EPA formulated a new rule that would explicitly satisfy the requirements of the CAA (Milford & Pienciak, 2009). Petitioned by industry groups, the DC Circuit Court reviewed this new rule, the Cross-State Air Pollution Rule (CSAPR), and decided that it overstepped the EPA's authority (US EPA, 2013a). CAIR remains in effect in the interim.

Table 2.4: Major sources of mercury in the US and their regulatory status. Adapted from McCarthy (2005) with additional data from Houyoux & Strum (2011).

Source	% of total pre-regulation emissions (1994)	% of total emissions (2005)	Regulatory status
Coal-fired Electricty Generating Units	33%	50%	Regulated 2011
Large (>250 tons/day) Municipal Waste Combustors	19%	2%	Regulated 1995
Coal-fired Commercial/Industrial Boilers	13%	6%	Regulated 2004
Medical Waste Incinerators	10%	<2%	Regulated 1997
Oil-fired Commercial/Industrial Boilers	5%	<2%	No reductions required
Mercury Cell Chlor-alkali Plants	4%	3%	Regulated 2003
Hazardous Waste Combustors	4%	3%	Regulated 1999
Portland Cement Plants	3%	7%	Regulated 1999
Other (Electric Arc Furnaces, Gold Mining etc.)	8%	25%	Generally not regulated except Electric Arc Furnaces, 2004

Negotiations began in 2010, and concluded January of 2013, with finalized treaty text for the Minamata Convention (UNEP, 2013b). Key provisions of the text focus on protecting human and ecosystem health by preventing emissions and releases of mercury. They include (IISD Reporting Services, 2013):

- a ban on any new primary mining of mercury, and an elimination of existing mines by a country fifteen years after becoming a party to the convention
- a phase-out of many mercury-containing products by 2020, and processes by 2025
- national action plans to reduce, and where possible, eliminate, releases and emissions of mercury from ASGM
- the application of Best Available Technologies (BAT) and Best Environmental Practices (BEP) in new sources of mercury emissions to air, and a menu-based approach (goals, emission limits, BAT/BEP) of options for controlling and reducing emissions to air and releases to water from existing sources
- a new financial mechanism to assist developing countries in their implementation of these treaty requirements which includes a fund administered by the Global Environmental Facility (GEF), and a program dedicated to technical assistance and capacity building

2.4.3 Adaptation: fish advisories in the US and their limits

In the US, regulators have used fish consumption advisories as a means of adapting to existing mercury pollution (Dellinger et al., 2012). Fish consumption advisories are voluntary recommendations for how to avoid and minimize exposure. They provide guidance on fish selection (considering source, size, and species), and consumption frequency. These advisories are typically issued for non-commercially caught fish by state, local, and tribal regulators, and compiled by the EPA (Dellinger et al., 2012). In 2010, over 80% of fish advisories for US waterbodies were due to mercury (US EPA, 2011a), representing 35% of US lake acres (not including the Great Lakes) and 25% of river miles (Dellinger et al., 2012). Fish advisories can also be developed for commercially caught fish, however these fall under the purview of the US Food and Drug Administration (FDA). Recently, civil society organizations like the Monterey Bay Aquarium Foundation (2013) have contributed significantly to developing fish advisories for consumers as well.

Many have concerns about the fish advisory approach to risk management, particularly for non-commercially caught fish—some on the grounds of effectiveness, and others on the grounds of environmental justice. Awareness of fish advisories is often low in the public, and is often not significantly higher in high risk groups (e.g. pregnant women, frequent fish consumers)—and even where awareness exists, it is not necessarily a predictor of reduced consumption of high mercury fish (Oken et al.,

2012). In addition, fish advisories can sometimes lead to perverse effects. Fish, while being a source of contaminants like mercury and PCBs, are also a source of nutritionally beneficial n-3 polyunsaturated fatty acids. Many fish are low in contaminants and high in these beneficial fatty acids (Mahaffey et al., 2011). However, fish advisories can discourage the public from eating fish wholesale, rather than from eating high mercury fish in particular (Oken et al., 2012).

Nriagu et al. (2012) has argued that fish consumption advisories may promote environmental injustice. He uses environmental injustice to mean situations where certain communities, particularly ones that are already marginalized, disproportionately experience the harms of environmental degradation. Rather than placing the burden of responsibility on those who release mercury (the polluter-pays principle), fish advisories require action from those most affected by pollution, who are disproportionately "poorly resourced and geographically vulnerable communities of indigenous people, Asia immigrants, and urban poor" (Nriagu et al., 2012). As discussed in Section 2.2.2, these vulnerable, highly-exposed populations have to pay nutritional, and sociocultural costs for these dietary changes. For instance, for indigenous Arctic peoples, substituting store-bought goods for traditional food sources can be very expensive (transport costs) leading to food security concerns, and can involve its own health risks (eg. diabetes) as these foods are often more processed and refined (AMAP, 2011). O'Neill (2004) and Nriagu et al. (2012) have described these advisories as a form of cultural discrimination. They are based on a normative belief about fish consumption—that avoiding certain fish is a small price to pay for "health," as defined by the dominant society.

So how can these legitimate criticisms be addressed, while also achieving timely reductions in risk for exposed populations? As noted by Selin (2011) there are temporal policy challenges associated with mitigation: the long time lags associated with mercury's geochemical cycling mean that emissions reductions may not be completely reflected in fish for decades to centuries, and historical emissions, which cannot be controlled, are a continuing source of contamination. Therefore, even with drastic emissions reductions, adaptive strategies—including even imperfect risk avoidance ones—need to be explored that manage risk in the interim. This will be a critical task for the mercury science and policy research community.

Chapter 3

Model development

In the previous sections, I have outlined some of the environmental and economic complexities—multiple spatial and temporal scales, and potential, costly cardiovascular effects—that are involved in mercury's emissions-to-impacts chain. In this chapter, I describe in detail the development of an assessment framework that captures some of these complexities, addressing my first research question. I also describe how this framework can be applied to evaluate the Mercury and Air Toxics Standards.

Many sophisticated studies have already modelled individual links in mercury's emissions-to-impacts chain, as evidenced by the literature reviewed in Chapter 2. However, much of this work has not been integrated into a comprehensive benefits assessment. My contribution therefore, is integrating these environmental and economic insights into a single assessment tool, to better capture mercury pollution's complexities. My modelling representation of the emissions-to-impact cycle is shown as a schematic in Figure 3-1, on page 38. This figure is an extension of Figure 2-1, from Chapter 2. Modelling components are shown as green arcs, and data inputs are shown as circles. Circles shaded yellow represent external data inputs, and those shaded orange are model-produced data inputs. At a high level, the framework addresses the question: what welfare benefits to the US economy can be expected from emissions reductions? It does so by answering these questions, which correspond to steps in the chain:

1. How might emissions decreases change the amount of mercury entering aquatic ecosystems?
2. How might changing ecosystem inputs affect fish tissue concentrations of mercury?
3. How might changing fish tissue concentrations affect human exposure to mercury in the US?
4. How might changing exposure affect human body loading of mercury?
5. How might changing body loading translate into avoided IQ loss and avoided heart attacks?

6. What might the economy-wide effects of these health changes be?

I address question 1 in Section 3.1, linking emissions to deposition. Questions 2 and 3 are covered in Section 3.2, linking deposition to human exposure. Section 3.3, linking exposure to health impacts, corresponds to questions 4 and 5. Section **??**, connecting health impacts to economy-wide effects, addresses question 6.

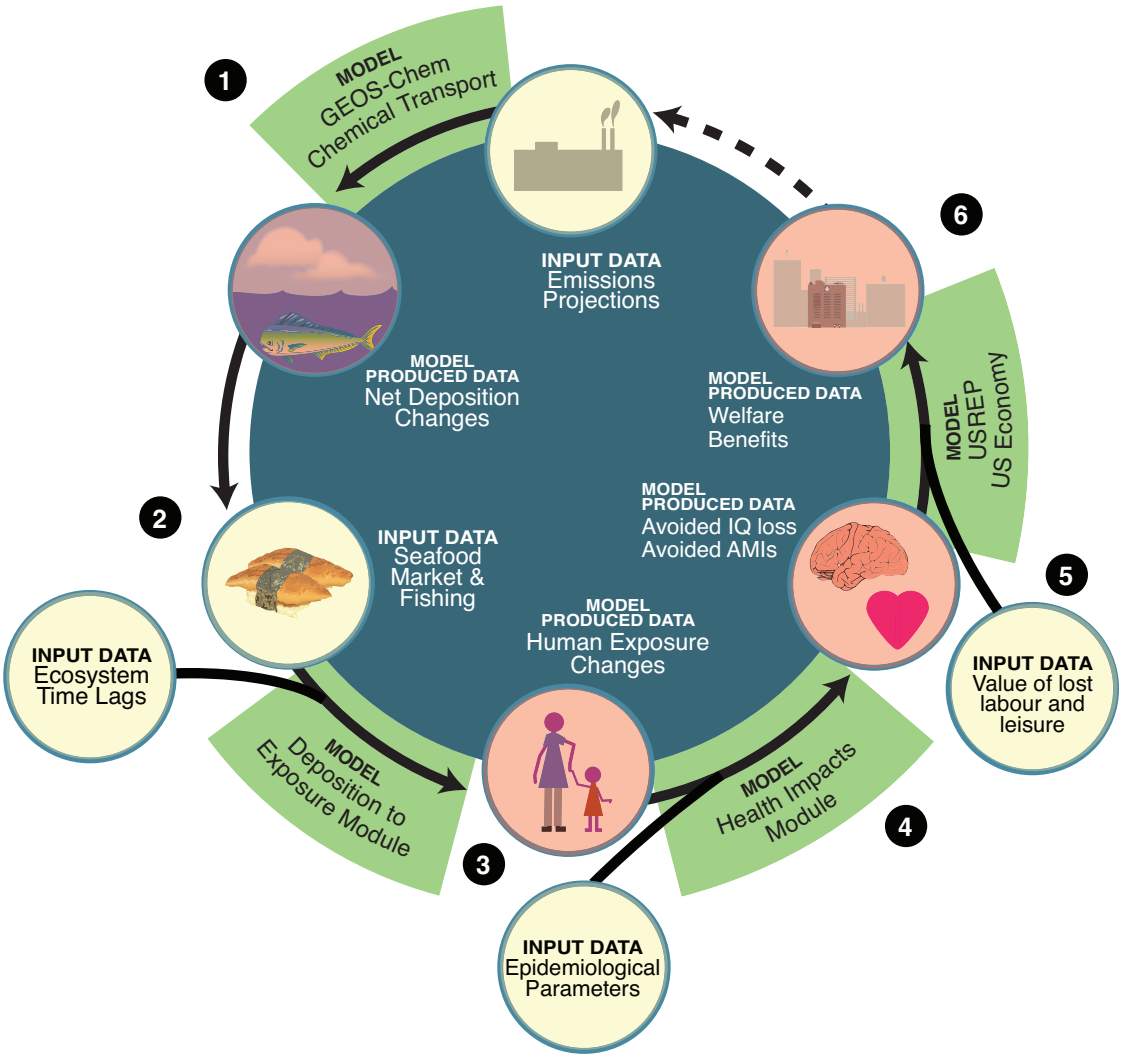
3.1 Emissions to deposition

3.1.1 Emissions projections

Both US and world emissions affect US exposure to mercury. The magnitude of emissions will be affected by the presence of reduction policies, and by different economic growth scenarios. Therefore, to evaluate the potential impact of emissions reductions, I developed a set of future emissions scenarios. These emissions projections integrate work by Zhang et al. (2012), Corbitt et al. (2011), and US EPA (2011b).The scenarios considered are summarized as a matrix in Table 4.3, which shows various US policy, and future world emission trajectories. For each of the scenarios indicated in this matrix, there is an additional third dimension, not shown, for whether IPR (see Section 2.1.2 is assumed for US CFPP sources.

Future (2050) world emissions inventories (excluding the US), at $1° \times 1°$ resolution, were taken directly from Corbitt et al. (2011). Corbitt et al. (2011) prepared these inventories using projections from Streets et al. (2009). For "present-day" world emissions, the inventory from the Global Emission Inventory Activity (GEIA) 2000 (Pacyna et al., 2010a) was scaled to 2006 estimates from Streets et al. (2009); Holmes et al. (2010). The "No Growth" scenario uses these present-day, 2006 emissions. The "Worst Case" and "Best Case" scenarios for future world emissions correspond to the Intergovernmental Panel on Climate Change (IPCC) scenarios A1B and B1, respectively. A1B assumes global increases in the use of coal, with some use of mercury control technologies in developing countries, while B1 assumes global transitions away from coal, with more widespread adoption of higher efficiency mercury control technologies (Streets et al., 2009; Corbitt et al., 2011). A linear interpolation was used to create a trajectory from 2006 to 2050, at 5 year intervals (4 year for the first period).

For the US, I used the spatially distributed inventories prepared by Zhang et al. (2012) based on the US National Emissions Inventory, and scaled the emissions in each US region to projections from the EPA's Integrated Planning Model (IPM) for the "Air Quality Policy", and "Mercury Policy" scenarios (US EPA, 2011b). The IPM model results were used to ensure consistency and facilitate comparison with EPA welfare benefits estimates. The US regions are defined in Figure 3-2, and were selected to match the US regions used in the economic model—described in Section 3.4. For the 'No Policy" scenario, I use the US inventory from Corbitt et al. (2011). The IPM simulates the potential impact of environmental policies on the electric power sector in the contiguous US, and has been a key tool in the EPA's benefit-costs analyses. For



1. How might emissions decreases change the amount of mercury entering aquatic ecosystems?
2. How might changing ecosystem inputs affect fish tissue concentrations of mercury?
3. How might changing fish tissue concentrations affect human exposure to mercury in the US?
4. How might changing exposure affect human body loadings of mercury?
5. How might changing body loadings translate into avoided IQ loss and avoided heart attacks?
6. What might the economy-wide effects of these health changes be?

Figure 3-1: Overview of modelling framework. Model components are shown in green. External input data is shown in yellow, and model produced data is shown in red. Key questions answered by the modelling framework are listed below.

Table 3.1: Emissions scenarios considered. A third dimension considered, not shown here, is whether in-plume reduction is assumed for US CFPP sources.

		Future World Emissions		
		No Growth	Worst Case	Best Case
US Policy	No Policy	x	x	x
	AQ Policy Only	x	x	x
	Hg Policy	x	x	x

MATS, IPM was used to simulate CFPP emissions, at five year intervals until 2030, under air quality policy only (Cross State Air Pollution Rule), and mercury policy (the Mercury and Air Toxics Standards). To calculate total anthropogenic mercury projections for the US, I assumed that the CFPP attributable share of total emissions would drop from 50% to 42% by 2016 in the air quality only case, based on estimates by Houyoux & Strum (2011). I then assume that this fraction is maintained into the future. I assume that the quantity of emissions from non-CFPP sources will be the same in both the air quality and mercury policy scenarios. For the speciation of US emissions, I apply the speciation fractions used by Zhang et al. (2012) to CFPP sources: 43.2% Hg(II) and 56.8% Hg(0) without IPR, and 10.8% Hg(II) and 89.2% Hg(0) with IPR. For non-CFPP sources, I assume that speciation fractions remain constant over time. In terms of the temporal trajectory, the IPM projections reflect the timing requirements of the modelled policies, which require compliance by 2014-2016. The trend from 2020 to 2030, once policies have been fully implemented, is roughly linear, so I extrapolate this linear trend out to 2050.

Many assumptions have been made in creating these emissions trajectory scenarios, including those embedded within the IPM model. The effects of some of these assumptions are captured in the range of scenarios considered (eg. aggressive expansion of coal in the worst case vs. aggressive transition away from coal in the best), however, many have not been included. For instance, it is likely that the spatial distribution of emission sources, in the US and globally, will change between 2006 and 2050. This effect will likely be largest in rapidly developing countries, where a large number of new power plants will be built. In addition, assumptions about future control technology penetration and efficacy may have an effect on total emissions budgets, and emissions speciation. Finally, the IPM is a deterministic linear programming model, with all the attendant limitations of a perfectly optimizing model (Bhattacharyya, 1996). Given that these emissions estimates are being used in an integrated assessment modelling framework, it is important to note that these emissions trajectories are not fully-coupled to the modelled US economy, though it is possible that there would be some pollution feedback effects. Saari et al., in prep. are exploring these feedback loops for ozone and particulate matter pollution.

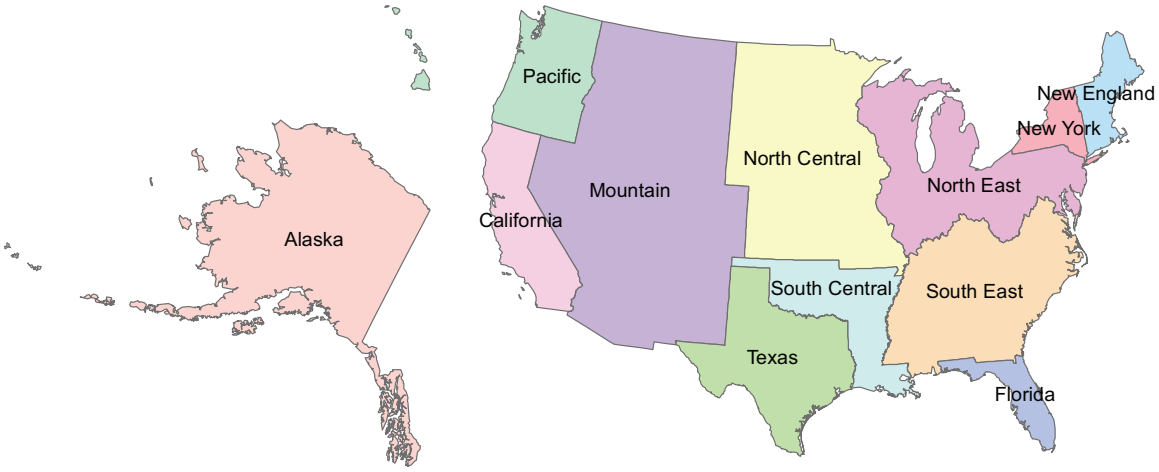


Figure 3-2: US regions modelled. These are based on the regions represented in the US Regional Energy and Environmental Policy (USREP) model (Rausch et al., 2010).

3.1.2 GEOS-Chem Mercury Simulation

The GEOS-Chem global mercury simulation v.9-01-02 (http://acmg.seas.harvard.edu/geos/), is used to model the chemical evolution and transport of mercury through the environment (atmosphere, oceans, land) (Selin & Jacob, 2008; Holmes et al., 2010; Soerensen et al., 2010; Amos et al., 2012). It has been extensively used in the academic literature to address mercury science (Amos et al., 2013; Corbitt et al., 2011; AMAP, 2011), and in regulatory applications—by the EPA, to set boundary conditions for regional air quality models (US EPA, 2005), and by UNEP in their global mercury assessments (UNEP, 2008). The model traces the two forms of mercury—gaseous elemental Hg(0), and divalent Hg(II), the latter of which has equilibrium partitioning between the gas and particulate phases (Amos et al., 2012)—as they cycle through different environmental compartments from their point of emission. It consists of a 3-D atmosphere (Holmes et al., 2010; Amos et al., 2012), and 2-D ocean (Soerensen et al., 2010) and terrestrial (Selin & Jacob, 2008) slabs. I use the reduction-oxidation scheme described in Holmes et al. (2010). Meteorology in the model is driven by assimilated fields from the NASA Goddard Earth Observing System (GEOS-5). Simulations were run at a horizontal resolution of $2° \times 2.5°$, with 47 vertical layers. The model was run from 2004-2009 for initialization. For analysis, simulations were run from 2007-2009. For analysis, simulations results were averaged over these three years to smooth over the effects of interannual variability. I do not consider the effect of future meteorology, to better isolate the effect of emissions. Understanding how future climate, in tandem with future emissions, will affect deposition is a topic for future research.

Simulations were run with all emissions scenarios, as indicated in Table 4.3, with 2050 and present-day emissions inventories. Scenarios in column 1 of the Table ("No Growth" in world emissions) were also modelled with 2010, 2015, and 2020 emissions. Simulation results for the remaining scenarios (column 1, with emissions from 2025-2045, and columns 2 and 3 with emissions from 2010 to 2050) were linearly

interpolated from these existing model runs.

For my purposes, the key data output from GEOS-Chem simulations is net total deposition. Following Corbitt et al. (2011), this is:

$$\sum_{species} dry\ deposition\ +\ wet\ deposition\ -\ rapid\ reemission\ from\ land\ and\ snow$$
$$-\ revolatization\ from\ ocean$$

Not all terms apply, depending on species, and whether the grid box being considered is land, snow, or ocean.

I consider net total deposition to be an appropriate environmental indicator for fish tissue concentrations. Net total deposition is a measure of mercury enrichment to an ecosystem (Corbitt et al., 2011). Recent research suggests that fish concentrations in ocean (Sunderland & Mason, 2007; Mason et al., 2012), and freshwater (Evers et al., 2011b; Drevnick et al., 2012) fish will likely respond proportionally to changes in atmospheric inputs on a scale of years to decades—though the magnitude and timing of a full response may be variable depending on the region (see Section 2.1.3.

3.2 Deposition to exposure

The calculations described below comprise the "Deposition to Exposure" module shown in Figure 3-1. I step through how fish tissue concentrations of mercury will respond to deposition changes (Section 3.2.1), and how this in turn will affect human exposure (Section 3.2.2). Because human exposure in the US is primarily through fish and shellfish consumption, to relate environmental concentrations of mercury to exposure requires incorporating information about the sources of fish that are consumed in the US.

3.2.1 Changes in fish tissue concentration of mercury

To relate changes in net total deposition of mercury to changes in fish tissue concentrations of mercury, I first identify sources of seafood relevant to the US population. For freshwater domestic fishing, I consider deposition to the 12 US regions defined in Figure 3-2. Based on analysis of the National Marine Fisheries Service's (NMFS) fisheries supply data by Sunderland (2007) and Pirrone & Keating (2010), I use 7 marine and estaurine regions, defined in Figure 3-3.

I sum the net total deposition of mercury, over all grid boxes within the source region, for each of the 19 regions. For farmed fish I take an area-weighted average of all source regions, since they are fed controlled diets of wild fish from local and global sources (Jardine et al., 2009). I calculate changes in net total deposition as percent changes over the present-day scenario, rather than as absolute values. I assume that, at steady state, fish will respond proportionally to these percent decreases in atmospheric inputs, but with the time lags shown in Table 3.2. I model the fish response as a linear

Table 3.2: Ecosystem time lags for fish tissue response to changes in deposition. The table shows three possible cases for the time required for fish concentrations to reflect 100% of a steady-state decrease in atmospheric inputs.

	Best Case	Moderate Case	Worst Case
100%	instantaneous	10 yrs	50 yrs

increase to 100% of deposition changes over the course of the time lag. These assumed responses are a simplification of what is reported in the literature (Sunderland, 2007; Knightes et al., 2009; Mason et al., 2012; Chen et al., 2012a); however, I use them as a first approximation of real-world ecosystem dynamics. I use a range of time lag scenarios, from instantaneous to 50 year long responses, to better understand the sensitivity of results to these uncertainties.

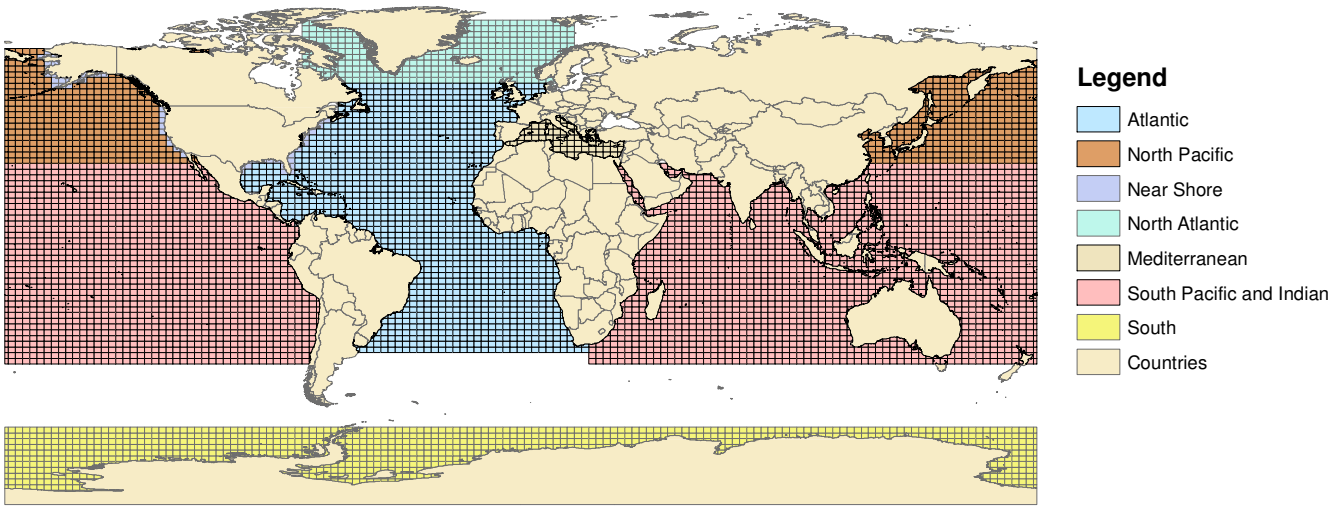


Figure 3-3: Ocean seafood source regions, as defined by Pirrone & Keating (2010). Farmed fish scale to the global average.

3.2.2 Changes in human exposure

To relate changes in fish tissue concentration from different source regions to changes in mercury intake for US populations, I estimate the percent of dietary mercury intake that comes from each source region, for the average individual living in each of the 12 US regions. These estimates—or intake influence coefficients—are shown in Table 3.3 on page 44.

The percentage of intake in each US region attributable to non-commercial, locally-caught freshwater fish was calculated using data from US EPA (2011d). US EPA

(2011d) estimated the average daily mercury intake for recreational anglers using a population centroid approach, described in US EPA (2005). Briefly, the approach models mercury intake as a function of an angler's residential location, distribution of fish mercury concentrations within a potential travel-distance radius, travel frequencies and distances, and fish consumption rate. The first three variables give a weighted average mercury concentration for recreationally caught fish, for anglers living in a certain area. The distribution of fish mercury concentrations was created from three sets of samples: the National Listing of Fish Advisory database, the US Geological Survey fish mercury database, and the EPA's National River and Stream Assessment database. Average mercury concentration for recreationally caught fish, multiplied by the average daily self-caught fish consumption rate, gives the average daily intake of mercury. The EPA used an assumed average daily self-caught fish consumption rate of 8 g/day for anglers, based on their review of the literature. While this value is high compared to the population average (Mahaffey et al., 2004), it is low compared to the average for Native American populations (Moya, 2004).

I take the ratio of total mercury intake from recreational anglers (a subset of the state population) to total mercury intake in each state to find the share of intake attributable to non-commercial, locally caught fish:

$$\%\ local = \frac{\mathbb{E}[intake_{angler\ population}] \times population_{anglers}}{\mathbb{E}[intake_{state\ population}] \times population_{state}}$$

where $\mathbb{E}[intake_{angler\ population}]$ is taken from US EPA (2011d) and $\mathbb{E}[intake_{state\ population}]$ from Mahaffey et al. (2004). These values are then population-weighted to aggregate from state to region-level. In the absence of intake data for recreational marine anglers, I included these individuals in the freshwater angler population.

Non-local intake is assumed to be from commercially caught marine, estaurine, and aquaculture fish. This share is attributed to specific marine and estaurine source regions following Pirrone & Keating (2010) and Sunderland (2007)—see Table 3.3. They provide a breakdown of mercury intake by source region for these fish in the US commercial market by combining NMFS fisheries supply data with an extensively compiled database of fish concentrations, from state, federal, and literature data.

The intake influence coefficients produced by this method are regional averages, which may obscure some of the distributional effects of mercury's health impacts. As discussed in Section 2.2, the distribution of mercury intakes in the US population is very long tailed, with a small number of highly exposed individuals, mostly consuming fish from local, freshwater sources (Moya, 2004). Regional averaging therefore underestimates the contribution of local sources to these vulnerable populations, while overestimating for the general population. To develop more insight into the effects of policy on these vulnerable, high exposure populations, I also consider a case where the entire US population consumes locally caught, freshwater fish, and another where the entire population consumes marine, estaurine, and aquaculture fish.

Table 3.3: Intake influence coefficients for average residents of each US region. Local refers to fish from that US region. Farmed and fresh fish are scaled to an area-weighted average of all other source regions.

US Region	Local	Farmed	Nearshore Marine	North Atlantic	Atlantic	North Pacific	South Pacific and Indian	Med. Sea	Southern
Alaska	36.09%	9.52%	5.05%	4.15%	9.39%	18.85%	16.23%	0.64%	0.06%
California	17.07%	12.36%	6.55%	5.39%	12.19%	24.46%	21.06%	0.83%	0.08%
Florida	28.19%	10.70%	5.67%	4.67%	10.56%	21.18%	18.24%	0.72%	0.07%
Mountain	14.61%	12.72%	6.75%	5.55%	12.55%	25.19%	21.69%	0.85%	0.09%
North Central	59.36%	6.06%	3.21%	2.64%	5.97%	11.99%	10.32%	0.41%	0.04%
North East	17.81%	12.25%	6.49%	5.34%	12.08%	24.25%	20.88%	0.82%	0.08%
New England	14.22%	12.78%	6.78%	5.58%	12.61%	25.31%	21.79%	0.86%	0.09%
New York	9.78%	13.44%	7.13%	5.86%	13.26%	26.61%	22.91%	0.90%	0.09%
Pacific	16.46%	12.45%	6.60%	5.43%	12.28%	24.64%	21.22%	0.84%	0.08%
South Central	41.10%	8.78%	4.65%	3.83%	8.66%	17.38%	14.96%	0.59%	0.06%
South East	31.32%	10.23%	5.43%	4.46%	10.10%	20.26%	17.44%	0.69%	0.07%
Texas	18.65%	12.12%	6.43%	5.29%	11.96%	24.00%	20.66%	0.81%	0.08%

Finally, to calculate exposure changes due to changes in emissions, for each US region:

$$\Delta\ Exposure_{\ US\ region} = \sum_{source\ region} Intake\ Influence_{\ US\ region} \times \Delta\ Deposition$$

For each US region, change in exposure is the sum (over all source regions) of the product of the intake influence coefficient (for that US region and source region, as shown in Table 3.3) and the percent change in deposition over that source region. These exposure changes are expressed as percent change compared to present-day exposures.

3.3 Exposure to Health Impacts

The calculations described below comprise the 'Health Impacts" module shown in Figure 3-1. I focus on mercury's IQ and cardiovascular effects. I discuss how changes in human exposure (through dietary intake) are reflected in concentrations in hair and blood—taking into account existing distributions of these biomarkers in the US population. I then discuss how dose-response relationships, taken from the epidemiological literature, are used to calculate the IQ and cardiovascular effects associated with biomarker concentrations of mercury.

3.3.1 Changes in body loadings of mercury

The first step in evaluating changes in health impacts is understanding how a change in dietary intake of mercury affects body loadings of mercury (see Section 2.3.1). The relationships between intake (I), blood mercury (Hg_{blood}), and hair mercury (Hg_{hair}) can be summarized as a series of linear relationships as follows:

$$Hg_{blood} = \alpha I \tag{3.1}$$

$$Hg_{hair} = \beta Hg_{blood} \tag{3.2}$$

The parameters α and β are transformation coefficients drawn from the literature. These parameters are described below, and a summary of their estimated central tendency, spread, and probability distribution, and of all other parameters in this section, are given in Table 3.7 on page 59.

Dietary intake-to-blood coefficient, α ($\frac{\mu g\ Hg}{L\ blood}\ per\ \frac{\mu g\ Hg}{day}$)

The dietary intake to blood coefficient represents the marginal change in equilibrium blood mercury concentration that would result from a small change in daily dietary Hg intake. Probabilistic estimates of its population-averaged value are based on a

simple pharmacokinetic model that treats the entire body as one compartment, and considers the steady-state blood concentration of a chemical following a pulse-input—in this case, the equilibrium blood Hg concentration following dietary intake (Stern, 2005; Rice et al., 2010). Key assumptions in the calculation of this parameter are blood volume, the half-life of MeHg in the body, the fraction of mercury consumed that is absorbed, and the fraction of mercury absorbed that remains in the blood at equilibrium—maternal blood volume and biological half-time of MeHg being the areas of greatest uncertainty and variation (Stern, 2005; Rice et al., 2010). Blood volume and biological half-time of MeHg are the areas of greatest uncertainty, as there may be considerable variation in body size and metabolism from person-to-person (Rice et al., 2010). Using data from Stern (2005) for the factors mentioned above, Rice et al. (2010) chose a normal distribution for the dietary-Hg-intake to blood-Hg coefficient, centred at 0.6 with a standard deviation of 0.09. I follow their approach.

Blood-to-hair coefficient, β ($\frac{\mu g\ Hg}{g\ hair}\ per\ \frac{\mu g\ Hg}{L\ blood}$)

Hair mercury is another biomarker for an individual's mercury load, and is more commonly reported in dose-response functions relating maternal mercury load to fetal IQ change because hair is more convenient to sample and store (Budtz-Jørgensen et al., 2004). Despite this tendency, blood mercury is often considered a better indicator of the amount of mercury systemically available, as hair mercury concentrations often reflect less recent exposures (Budtz-Jørgensen et al., 2004). The blood-to-hair coefficient is therefore the conversion factor between a marginal change in blood mercury concentration, and the corresponding change in equilibrium hair concentration. Following Rice et al. (2010), I use two values, one derived from pregnant women and the other from non-pregnant women, from a meta-analysis by Allen et al. (2007). I assume a log normal distribution, supported by a meta-analysis by Shipp et al. (2000), with arithmetic mean and standard deviation of 0.21 ± 0.0014 and 0.18 ± 0.0014 for pregnant and non-pregnant women respectively. In the absence of other data, I apply the value for non-pregnant women for the entire adult, non-pregnant population in calculating changes in incidence of MeHg-related heart attacks.

Baseline exposure distributions

Baseline distributions of US exposure to mercury, using blood mercury levels as an indicator, were drawn from Mahaffey et al. (2009). Mahaffey et al. (2009) use data from the 1999-2004 National Health and Nutrition Examination survey (NHANES)—a continuous study carried out by the National Center for Health Statistics that includes both interviews and physical examinations—to assess the distributions of blood mercury concentrations in adult women of childbearing age (16-49) by region and household income. Using reported geometric and arithmetic means, log-normal distributions were parameterized for the four geographic regions defined in Mahaffey et al. (2009)—Northeast, South, Midwest, and West. The choice of a log-normal parameterization was based on statistical analyses from previous studies of hair and

Table 3.4: Parameters for regional log-normal blood mercury distributions

USREP Region	μ	σ	**Arithmetic Mean** (μg/L)	**Geometric Mean** (μg/L)
Alaska	0.166	1.02	1.98	1.18
California	-0.0513	1.03	1.61	0.95
Florida	-0.105	1.1	1.64	0.90
Mountain	-0.0513	1.03	1.61	0.95
New England	0.131	1.15	2.21	1.14
New York	0.131	1.15	2.21	1.14
North Central	-0.416	0.901	0.99	0.66
North East	-0.224	1.03	1.36	0.80
Pacific	-0.041	1.03	1.63	0.96
South Central	-0.105	1.1	1.64	0.90
South East	-0.105	1.1	1.64	0.90
Texas	-0.105	1.1	1.64	0.90

blood mercury distributions (McDowell et al., 2004; Rice et al., 2010). A comparison of the observed cumulative distribution of blood mercury, and the modelled cumulative distribution using a log-normal assumption is shown in Figure 3-4.

Because some of the twelve regions defined in the USREP model spanned multiple regions defined in Mahaffey et al. (2009), exposure distributions for these USREP regions were parameterized by randomly sampling, based on population, from the regional distributions defined in Mahaffey et al. (2009), and then fitting the resulting distribution. The correspondences between states and regions—as defined in Mahaffey et al. (2009) and in the USREP model (Rausch et al., 2010)—are shown in Table A.1 in the Appendix. Blood mercury distributions for each USREP region are parameterized in Table 3.4, and plotted in Figure A-1, in the Appendix.

While the geographic disaggregation of this data set allows us to capture regional differences, there are also limitations associated with its use. Baseline exposure distributions from Mahaffey et al. (2009) were based on adult women of child bearing age. This demographic is appropriate when considering IQ loss due to maternal dietary intake of mercury; however, this demographic may not be representative when considering increased risk of acute myocardial infarctions in the entire adult US population. Differences between men and women in terms of dietary intake patterns, and average blood volume could affect the applicability of a blood mercury distribution based on adult women for the whole adult population. However, because the exposure calculation method (see Section 3.2.2 is highly sensitive to geographic regions, I expect that regional variations lead to greater differences than sex differences, and I use this assumption as a first approximation.

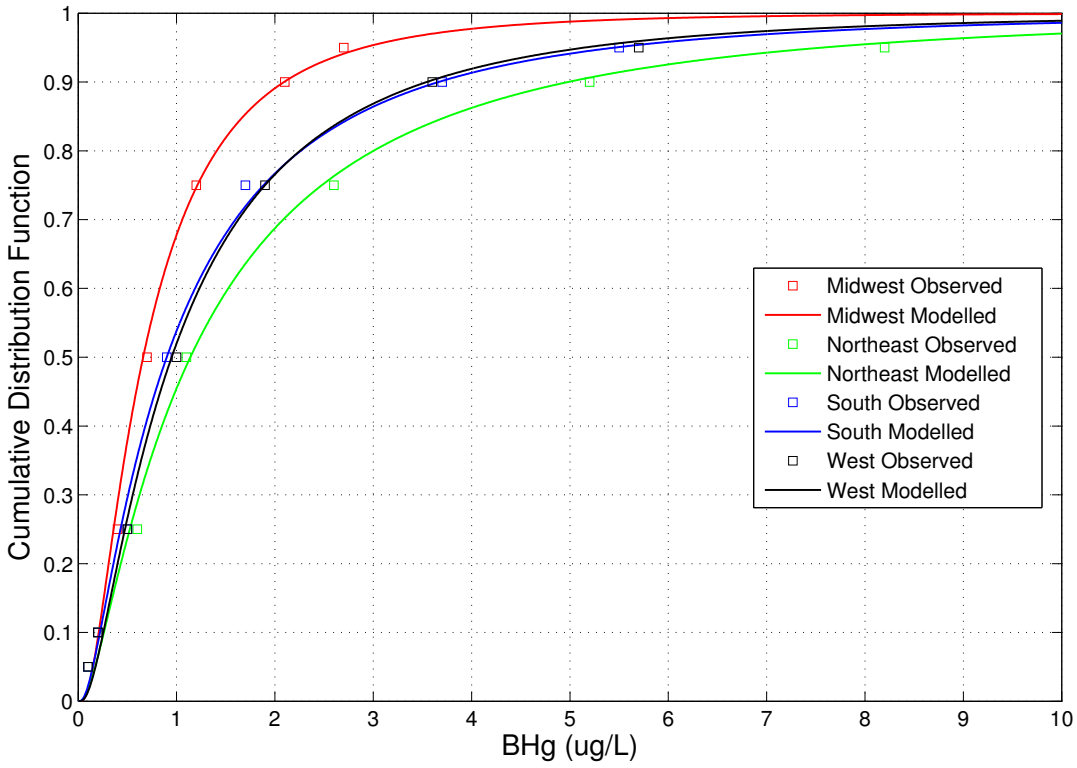


Figure 3-4: Comparison of observed and modelled cumulative distribution, using a log-normal model. Observed data points are taken from Mahaffey et al. (2009).

Future exposure distributions

Changes in mercury intake (due to changes in emissions) are expected to change the central tendency of US blood mercury distributions, though not the log-normal shape or spread. For a reduction in mercury intake, I, by θ percent:

$$\begin{aligned} BHg_{policy} &= \alpha(1-\theta)I \\ &= (1-\theta)BHg_{base} \end{aligned}$$

$$\begin{aligned} \therefore E[BHg_{policy}] &= (1-\theta)E[BHg_{base}] \\ \text{and } Var[BHg_{policy}] &= (1-\theta)^2 Var[BHg_{base}] \end{aligned}$$

Where BHg is a log-normally distributed random variable representing an individual's blood mercury concentration, and α represents the linear conversion factor between dietary intake of mercury and blood mercury. Using the properties of a log-normal distribution, the parameters μ and σ for the post-policy distribution can be calculated

as follows:

$$\begin{aligned}
\mu_{policy} &= ln(E[BHg_{policy}]) - \frac{1}{2}ln\left(1 + \frac{Var[BHg_{policy}]}{E[BHg_{policy}]^2}\right) \\
&= ln((1-\theta)E[BHg_{base}]) - \frac{1}{2}\left(1 + \frac{Var[BHg_{base}]}{E[BHg_{base}]^2}\right) \\
\sigma^2_{policy} &= ln\left(1 + \frac{Var[BHg_{policy}]}{E[BHg_{policy}]^2}\right) \\
&= ln\left(1 + \frac{Var[BHg_{base}]}{E[BHg_{base}]^2}\right) \\
&= \sigma^2_{base}
\end{aligned}$$

3.3.2 IQ effects

To calculate fetal IQ changes associated with a change in maternal body loading of mercury, I assume a linear dose-response relationship between maternal mercury load, assessed through the hair biomarker, and IQ decrements in the child (see Section 2.3.2). The distribution for the random variable Hg_{hair} can be calculated from Hg_{blood} distributions using Equation 3.3.

$$\Delta IQ = \gamma Hg_{hair} \tag{3.3}$$

In the above, ΔIQ represents the change in fetal IQ associated with a given level of maternal hair mercury. The coefficient γ represents the slope of the linear relationship.

Hair to IQ coefficient, γ ($IQ\ points\ per \frac{\mu g\ Hg}{g\ maternal\ hair}$)

The hair to IQ coefficient is the dose-response relationship between maternal Hg exposure, captured by the hair biomarker, and IQ of the child. Data from three large-scale epidemiological studies, taking place in New Zealand, the Seychelles Islands, and the Faroe Islands (Crump et al., 1998; Myers et al., 2003; Grandjean et al., 1997), have been used to characterize this relationship. The New Zealand and Faroe Islands studies indicated statistically significant associations between prenatal mercury exposure and performance on childhood neuropsychological tests (Crump et al., 1998; Grandjean et al., 1997), whereas the Seychelles Islands study did not find a significant correlation (Myers et al., 2003). Axelrad et al. (2007) conducted a meta-analysis of these three studies, using a Bayesian hierarchical model to estimate a dose-response relationship between maternal mercury load and childhood IQ decrements from the integrated data. Their central estimate was -0.18 IQ points per $\frac{\mu g\ Hg}{g\ maternal\ hair}$, with a 95% confidence interval of -0.378 to -0.009, and assumes a linear non-threshold relationship (Axelrad et al., 2007).

However the Axelrad et al. analysis did not control for confounding effects: while

fish and shellfish are the major exposure pathway for MeHg, maternal consumption of these foods also provides key nutrients, like n-3 polyunsaturated fatty acids (PUFAs), which confer neurodevelopmental benefits for offspring (see Mahaffey et al. (2011) for a review of benefits and risks). Budtz-Jørgensen et al. (2007) have estimated that not adjusting for these benefits can downward bias mercury-associated IQ decrements by up to a factor of 2. Following Rice et al. (2010), the central estimate from Axelrad et al. (2007) is adjusted by a factor of 1.5 for this analysis. I do not take the presence of polychlorinated biphenyls (PCBs), another toxic contaminant found in fish and shellfish, into account; while PCBs have also been associated with neurodevelopmental risks (Oken et al., 2012), in an examination of the Faroe Islands data, PCBs were not found to significantly affect neurobehavioural deficits (Budtz-Jørgensen et al., 1999), or to attenuate the mercury effect (Budtz-Jørgensen et al., 2002).

While the NRC has recommended the use of a linear dose-response relationship in the absence of significant evidence for another form, extrapolating the observed relationship to exposure levels that are more relevant for a US population (ie. typically lower exposures) may require caution (Axelrad et al., 2007). If the true relationship is in fact supralinear (steeper at lower doses), which may be supported by evidence from the Faroe Islands study (Budtz-Jørgensen et al., 2000), then this dose response function may underestimate the effect at US exposure levels (Rice, 2004).

3.3.3 Cardiovascular effects

Recently, a workshop assembled by the EPA assessed the strength of evidence for a population-level relationship between MeHg exposure through seafood consumption and adverse cardiovascular health effects (Roman et al., 2011). Their conclusion was that there was now a sufficient body of epidemiological, animal, and *in vitro* evidence to support the development of a dose-response relationship between dietary MeHg exposure and acute myocardial infarctions (AMI), more commonly known as heart attacks (Roman et al., 2011). Following this recommendation, and the work of Rice et al. (2010), I include AMIs in my accounting of mercury-related health effects.

Roman et al. (2011) recommended the use of two epidemiological studies in particular for the development of a dose-response function for mercury exposure and heart attacks—the Kuopio Ischaemic Heart Disease Risk Factor (KIHD) study (Virtanen et al., 2005), and the European Community Multicenter Study of Antioxidants, Myocardial Infarction and Breast Cancer (EURAMIC) (Guallar et al., 2002). Both studies were recommended for their large sample sizes, and because they controlled for negative confounders, the most important being n-3 polyunsaturated fatty acids (PUFAs). PUFAs are an essential nutrient commonly found in oily, cold-water fish, and are believed to reduce risk of cardiovascular disease (Mozaffarian & Rimm, 2006). Failure to control for PUFAs may result in an underestimate of the true effect of mercury (Roman et al., 2011). I use these two studies as the basis for developing a dose-response relationship between mercury and AMIs.

Evidence from the EURAMIC study suggests that the shape of the dose-response function for MeHg related AMIs is log-linear (Guallar et al., 2002; Roman et al.,

2011). The dose-response relationship will therefore take the following form (Rice et al., 2010; Abt Associates, 2010):

$$\begin{aligned}\Delta AMI &= AMI_0 \left(1 - \frac{1}{\exp(\omega \cdot \Delta HHg)}\right) \\ &= AMI_0 \left(1 - \frac{1}{\exp(\omega \cdot \beta \Delta BHg)}\right)\end{aligned}$$

where ΔAMI is the change in incidence of acute myocardial infarctions—both fatal and non-fatal—due to an exposure change, AMI_0 is the baseline incidence of AMIs, ΔHHg and ΔBHg are the change in hair and blood mercury concentration respectively, and ω is the coefficient relating hair mercury concentration to heart attack risk.

I calculate the *total* incidence of AMIs due only to mercury in any given year, t, as follows:

$$\begin{aligned}AMI_{mercury,\,t} &= AMI_{mercury,\,t-1} + \Delta AMI \\ &= AMI_0\,(2 - \exp(-\omega\beta BHg_{t-1}) - \exp(-\omega\beta(BHg_t - BHg_{t-1})) \\ &= AMI_0\,(2 - \exp(-\omega\beta BHg_{t-1}) - \exp(-\omega\beta\Delta BHg))\end{aligned}$$

where BHg and ΔBHg are both random variables, with probability distributions $f(BHg)$ and $g(\Delta BHg)$ respectively. To calculate the number of cases of AMIs at a population-level, I take the expectation of the incidence, $\mathbb{E}[AMI_{mercury,\,t}]$, and multiply by population.

$$\begin{aligned}\mathbb{E}[AMI_{mercury,\,t}] &= \mathbb{E}[AMI_0\,(2 - \exp(-\omega\beta BHg_{t-1}) - \exp(-\omega\beta\Delta BHg)] \\ &= AMI_0\,(2 - \mathbb{E}[\exp(-\omega\beta BHg_{t-1})] - \mathbb{E}[\exp(-\omega\beta\Delta BHg)]) \\ &= AMI_0\,\left(2 - \int_{-\infty}^{+\infty} \exp(-\omega\beta \cdot BHg_{t-1}) \cdot f(BHg_{t-1})\,dBHg \right. \\ &\qquad \left. - \int_{-\infty}^{+\infty} \exp(-\omega\beta \cdot \Delta BHg) \cdot g(\Delta BHg)\,d\Delta BHg\right)\end{aligned}$$

The probability distribution $f(BHg)$ is a log-normal distribution, parameterized by μ and σ, as described previously. As there is no analytic formulation for the difference (ie. cross-correlation) of two log-normal distributions, I use Monte Carlo methods to fit $g(\Delta BHg)$. Ten thousand data points were randomly sampled from each BHg distribution, t and $t-1$. The difference of these two data series was taken, and then refit using the MATLAB distribution fitting tool in the probability and statistics tool box. The resulting density function is a t-location scale distribution, with location parameter μ, scale parameter σ, and shape parameter ν:

$$g(\Delta BHg) = \frac{\Gamma(\frac{\nu+1}{2})}{\sigma\sqrt{\nu\pi}\,\Gamma(\frac{\nu}{2})} \left(\frac{\nu + (\frac{x-\mu}{\sigma})^2}{\nu}\right)^{(\frac{\nu+1}{2})}$$

This is equivalent to a student's t-distribution with $t = \frac{x-\mu}{\sigma}$ with ν degrees of freedom.

I calculate the number of cases of fatal, and non-fatal heart attacks for each region and year as follows:

$$Cases_{fatal} = \mathbb{E}[AMI_{mercury,\,t}] \times Population \times \psi$$
$$Cases_{non\ fatal} = \mathbb{E}[AMI_{mercury,\,t}] \times Population \times (1 - \psi)$$

where ψ is the fraction of AMIs which are fatal. The value for ψ is drawn from the American Heart Association's most recent Heart Disease and Stroke statistics (Roger et al., 2012).

Hair mercury-to-heart attack risk coefficient, ω, ($fractional\ risk\ per\ \frac{\mu g\ Hg}{g\ hair}$)

The hair mercury-to-heart attack risk coefficient is derived from the Virtanen et al. (2005) KIHD study, which followed a cohort of 1871 men for a period of 13.9 years. Virtanen et al. (2005) reported a statistically significant relative risk for acute coronary events of 1.11 per $\frac{\mu g\ Hg}{g\ hair}$ (95% CI = 1.06 to 1.17). This relative risk is equivalent to $\omega = 0.10$. Though this parameter is derived solely from a male cohort, I apply this value to the entire adult US population. This value may therefore be an overestimate as the only analogous study for women did not report a statistically significant dose-response relationship between serum mercury and AMI risk (Ahlqwist et al., 1999). However, it should be noted that the serum mercury biomarker is not highly indicative of MeHg exposures (Rice et al., 2010). Nevertheless, to address this potential overestimation, I follow Rice et al. (2010) in using a triangular distribution with a minimum of 0. The mode and maximum, 0.10 and 0.16, are derived from Virtanen et al. (2005).

3.4 Health impacts to economic impacts

In this section, I describe how mercury-related health impacts have been incorporated into a general equilibrium model of the US economy—the US Regional Energy and Environmental Policy Model (USREP).

3.4.1 US Regional Energy and Environmental Policy Model

USREP is a recursive-dynamic general equilibrium (CGE) model of the US economy developed by Rausch et al. (2011). While USREP is described in detail in Rausch et al. (2010, 2011), here I offer a brief review. USREP represents households, firms, and government as rational economic agents, and finds the optimal, equilibrium condition of the economy given each agent's objective function (what maximizes their utility/happiness), their constraints, and available resources. Market interactions (ie. production and consumption) are based on microeconomic theory, and therefore depend on the relative prices of different goods, services, and availability of production factors like labour and capital. In USREP, the available labour pool results from a

choice between labour and leisure, at the household level. USREP uses 2006 as a base year, and solves recursively for equilibrium economic conditions at 5-year intervals starting in 2010. Production and consumption are modelled as nested constant elasticity of substitution functions. USREP uses the General Algerbraic Modelling System (GAMS), and the Mathematical Programming System for General Equilibrium Analysis (MPSGE).

USREP disaggregates the US into 12 regions, which were first shown in Figure 3-2. It also disaggregates the economy into sectors, which are shown in Table 3.5, which was adapted from Rausch et al. (2010). Energy is the most highly disaggregated sector because USREP was originally intended as a tool to evaluate the distributional impacts of US energy policy. Following the approach outlined by Matus et al. (2008), a pollution health sector was added to USREP by Saari et al., in prep.. I have modified this pollution health sector to include mercury-related health effects.

Following work by Matus et al. (2008), the pollution health sector is a means of accounting for reduced economic productivity due to morbidity effects (non-fatal illness—and in the case of mercury, also IQ loss). Essentially, medical services, and labour and leisure are diverted to this sector to produce good health. Because leisure is included, this can be thought of as an expanded cost-of-illness approach where some non-market effects are considered (though not pain and suffering); however, it should also be noted that the labour supply is determined by a tradeoff between household labour and leisure, so that leisure also has indirect market effects. In the case of IQ loss, I use a strictly cost of illness approach and only consider IQ's effect on total lifetime earnings—the labour input to the pollution health sector. I include non-fatal AMIs through estimated costs of medical care, and the value of lost work and leisure time due to the AMI.

For mortality effects—in the case of mercury, fatal AMIs—I also follow the approach of Matus et al. (2008). A premature fatality is treated as a shock to the pool of labour and leisure available, for the duration of an individual's expected lifetime. The approaches to valuing mercury-related morbidity and mortality effects are described further below.

3.4.2 Morbidity effects: IQ loss

Incremental IQ decrements due to chronic, low-level exposure to mercury may be of little concern on an individual level; however, when spread over a large population, these small decrements may become economically significant (Rice et al., 2010; Trasande et al., 2005). IQ can have a direct impact on an individual's lifetime earnings through hourly wage received, as well as an indirect impact through educational attainment and labour force participation (Schwartz, 1994). It should be noted that IQ is not the only determinant of lifetime earnings, and that factors like household income and parental education attainment have been shown to contribute more strongly to earnings variations (Zax & Rees, 2002). In addition, IQ decrements may have other welfare effects aside from an impact on an individual's lifetime wages, however, because there are no studies (to my knowledge) addressing these non-monetary effects,

Table 3.5: Region and sector disaggregation in the USREP model. Adapted from Rausch et al. (2010). The pollution health sector was added by Saari et al., in prep., and adapted for mercury health impacts in this work.

Regions	**Sectors**
Alaska	**Non-Energy**
California	Agriculture
Florida	Services
New York	Energy-Intensive
New England	Other Industries
South East	Transportation
North East	*Pollution health**
South Central	**Energy**
Texas	Coal
North Central	Convenient Crude Oil
Mountain	Refined Oil
Pacific	Natural Gas
	Electric: Fossil
	Electric: Nuclear
	Electric: Hydro
	Advanced Technologies

I use a cost of illness approach and consider only income effects in our analysis. This valuation of the costs of MeHg-related IQ effects can therefore be considered a lower bound.

To value the cost of IQ effects associated with mercury exposure, I followed the method used by Rice et al. (2010). Rice et al. (2010) calculate a child's lost lifetime earnings due to IQ decrements using a linear relationship.

$$\Delta E = \epsilon IQ \tag{3.4}$$

In the above, ΔIQ represents the change in fetal IQ associated with a given change in maternal hair mercury (see Equation 3.3), and ΔE represents the percent change in lifetime earnings associated with that IQ change. The slope for this linear relationship is represented by the coefficient ϵ. Again, all parameters in this section, and the one that precedes it, are summarized in Table 3.7 on page 59.

IQ to percent earnings coefficient, ϵ (*% per IQ point*)

The IQ to percent earnings coefficient represents the percent change in earnings resulting from a 1 point change in IQ. IQ is thought to affect earnings directly through wage rates, and indirectly through educational attainment and labour force participation (Schwartz, 1994). This valuation method has been used extensively in regulatory analyses of the benefits of reducing lead exposure from lead-based paints (Salkever,

1995; Grosse, 2007).

I use a coefficient value based on two recent economic analyses which implicitly include cognitive ability effects on schooling, and effects of schooling on wage and participation (Zax & Rees, 2002; Heckman et al., 2006). Zax & Rees (2002) estimate a total effect of 0.8% per IQ point, observed at the age of 35, whereas Heckman et al. (2006) estimate 0.9% per IQ point in 30-year old men. Adjusting the Heckman et al. estimate for gender—the effect of 1 IQ point has been observed to be approximately 30 to 40% higher for women than men (Salkever, 1995; Grosse, 2007)—suggests that the total effect of cognitive ability on income is 1.2% for women (Rice et al., 2010). Following Rice et al. (2010), I use a triangular distribution with a minimum at 0.6%, a maximum at 1.2%, and a mode at 0.8%.

Population-level earnings losses due to IQ decrements

To calculate the population-level earnings loss associated with a given level of mercury exposure—either the baseline, or the level associated with a pollution reduction policy—I calculate the expected percent decrease in earnings for an individual, and apply this decrease to each birth cohort in the working population. For random variable BHg, with a log normal probability distribution $f(BHg)$, the expected percent earnings loss associated with mercury exposure, $\mathbb{E}[\Delta E]$, is calculated as follows:

$$\begin{aligned}\mathbb{E}[\Delta E] &= \mathbb{E}[\epsilon\gamma\beta \cdot BHg] \\ &= \epsilon\gamma\beta \cdot \mathbb{E}[BHg] \\ &= \epsilon\gamma\beta \cdot \int_{-\infty}^{+\infty} BHg \cdot f(BHg)\, dBHg\end{aligned}$$

The expectation of the lost earnings for the entire US population in any given year, $\mathbb{E}[LE]$, can therefore be calculated by summing over each region and age cohort, the product of expected percent earnings loss associated with maternal exposure in that cohort's average birth year, average yearly earnings for that age cohort, and the number of US-born individuals of working age in the current year within the age cohort.

$$\mathbb{E}[LE] = \sum_{\substack{region \\ age\ cohort}} \mathbb{E}[\Delta E] \cdot Earnings_{avg} \cdot\ Working\ Population$$

The earnings profile by age cohort, derived from data from the US Department of Labor Bureau of Labor Statistics, is shown in Figure 3-5.

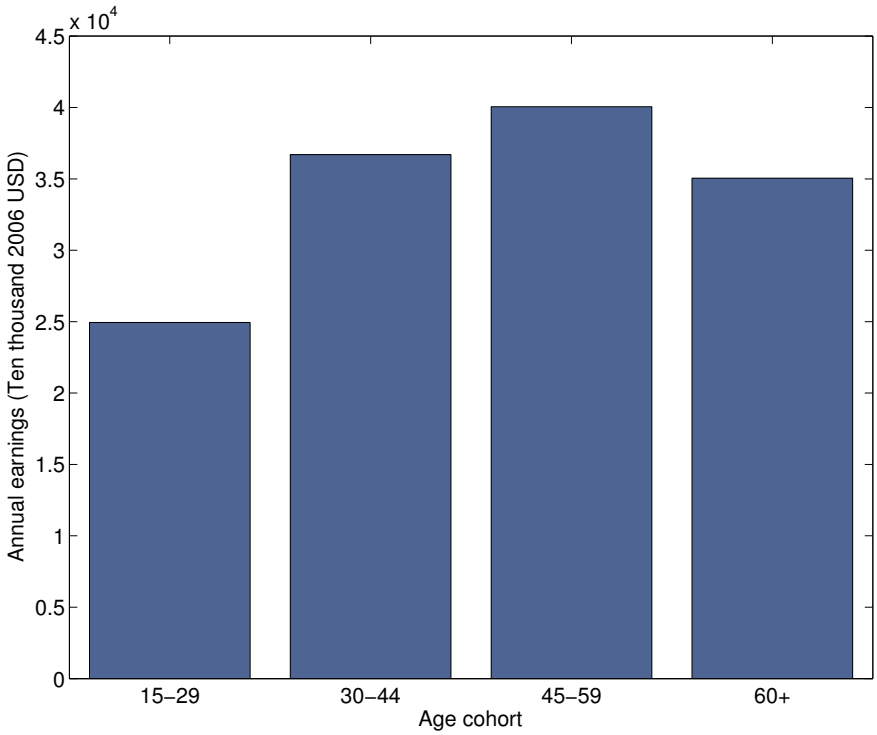


Figure 3-5: US earnings profile by age cohort. Source: US Department of Labor Bureau of Labor Statistics (2012)

3.4.3 Morbidity effects: Non-fatal AMIs

Numerous studies have investigated the large social burden of cardiovascular disease in the US, considering mortalities, medical costs, and lost productivity (Wittels et al., 1990; Cropper & Krupnick, 1990; Russell et al., 1998; Eisenstein et al., 2001). These studies underscore the importance of cardiovascular health endpoints in any economic accounting of the impacts of pollution. In accounting for the cost of an AMI, I consider the following categories: direct medical costs, lost labor, and lost leisure. This is not strictly a cost-of-illness approach, as a value is associated with an individual's leisure time; however, it may underestimate the true economic cost of an AMI as it ignores pain and suffering. For morbidity risks, I use valuation estimates for non-fatal heart attacks employed in the EPA's Regulatory Impact Analysis for the Federal Implementation Plans to Reduce Interstate Transport of Fine Particulate Matter and Ozone (US EPA, 2011c).

Matus et al. (2008) incorporate morbidity health effects for air pollution into a CGE model by allocating a share of the total cost of each health endpoint, elicited through contingent value surveys, to either direct costs of medical services, lost labour, or lost leisure. Based on their judgment, they allocate 50%-80% of total costs to medical services, with the remaining 20%-50% split between labour and leisure. I apply this approach to valuation estimates for non-fatal heart attacks drawn from US

Table 3.6: Valuation of Non-Fatal AMIs by age cohort (US EPA, 2011c), and share of cases by age cohort (Roger et al., 2012)

Age cohort	Lost Earnings	Medical Service Costs	Share of Cases
30-44	11080	88709	0.049
45-54	16331	88709	0.147
55-65	94396	88709	0.301
> 65	0	88709	0.513

EPA (2011c). US EPA (2011c) aggregate data from studies by Cropper & Krupnick (1990), Wittels et al. (1990), and Russell et al. (1998). Cropper & Krupnick (1990) assess the lost earnings associated with a non-fatal AMI over a five year period, while Wittels et al. (1990) and Russell et al. (1998) evaluate the direct medical costs. Table 3.6 shows a breakdown of these estimates by age cohort, as well as each age cohort's share of total cases of AMIs. However, unlike the valuation estimates used by Matus et al. (2008), these valuations are not based on contingent value surveys, and therefore do not implicitly include the value of non-market goods and activities, like leisure. To estimate the total cost of a non-fatal AMI, I inflate the sum of lost earnings and medical costs, according to their assumed share of total costs. For this calculation, I use a weighted average value for lost earnings and medical costs based on the age distribution of AMIs, which is shown in Table 3.6.

3.4.4 Mortality effects: Fatal AMIs

I follow the approach of Matus et al. (2008) to value fatal AMIs. Matus et al. (2008) include mortality effects in a CGE by treating each death as a negative shock to the labour pool that persists for the duration of that individual's expected life. The resulting valuation is not an estimate of the value of a life, but a way of accounting for the market impacts of a premature fatality. They assume the following: an expected lifetime of 75 years, an expected retirement age of 65 years, and that deaths in any cohort occur at the median age for that cohort (Matus et al., 2008). Up until the age of 65, there is both a labour and leisure component to the negative shock, with leisure being valued, at the margin, at the wage rate (Matus et al., 2008). Following expected retirement, only leisure is considered. Based on available statistics from the American Heart Association (Roger et al., 2012), I assume that fatal AMIs occur only in the over 30 population. Each year, I assume that new fatalities occur following the age distribution shown in Table 3.6. I do not include direct medical costs, as I assume that they are greatly outweighed by the cumulative lost earnings associated with a fatality. This assumption may not hold if mortalities occur primarily after retirement. Figure 3-6 shows a more detailed age distribution for share of total AMIs, with data from Roger et al. (2012). This data suggests that fatal AMIs may be fairly evenly distributed around the expected retirement age.

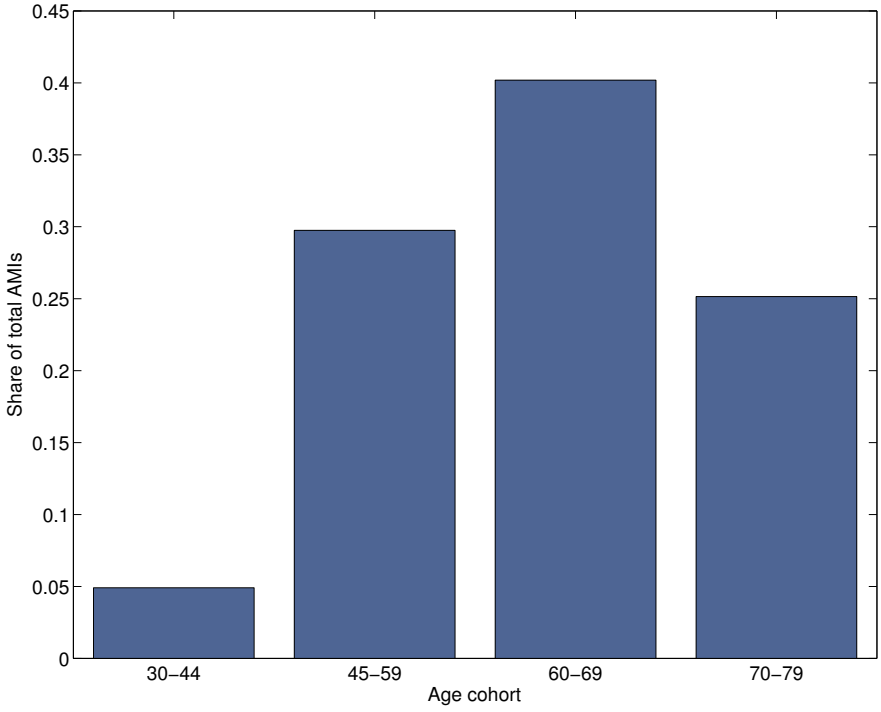


Figure 3-6: Share of total AMIs by age cohort Roger et al. (2012)

Table 3.7: Summary of Parameters

Symbol	Definition	Units	Central Tendency	Spread	Reference
α	dietary intake-to-blood coefficient	$\frac{\mu g\ Hg}{L\ blood}$ per $\frac{\mu g\ Hg}{day}$	mean = 0.6	SD = 0.09	Rice et al. (2010), Stern (2005)
β_p	blood-to-hair coefficient-pregnant	$\frac{\mu g\ Hg}{g\ hair}$ per $\frac{\mu g\ Hg}{L\ blood}$	mean = 0.21	SD = 0.0014	Allen et al. (2007)
β_{np}	blood-to-hair coefficient-not pregnant	$\frac{\mu g\ Hg}{g\ hair}$ per $\frac{\mu g\ Hg}{L\ blood}$	mean = 0.18	SD = 0.0014	Allen et al. (2007)
γ	hair-to-IQ coefficient	IQ points per $\frac{\mu g\ Hg}{g\ maternal\ hair}$	mean = 0.3	95% CI: 0.567 to 0.0135	Axelrad et al. (2007) modified by Rice et al. (2010)
ϵ	IQ to percent earnings	% per IQ point	mode = 0.8	SD = 0.1	Rice et al. (2010); Heckman et al. (2006), Zax & Rees (2002); Salkever (1995)
ω	hair mercury-to-AMI coefficient	risk per $\frac{\mu g\ Hg}{g\ hair}$	mean = 0.10	SD = 0.04	Virtanen et al. (2005)
ψ	fatal AMI fraction	%	mean = 0.14		Roger et al. (2012)
θ	percent exposure reduction	%			

Chapter 4

Case study: Evaluating the Mercury and Air Toxics Standards

The Mercury and Air Toxics Standards (MATS), which were described in Section 2.4, are the most recent US regulations targeting mercury emissions to air. In this chapter, I evaluate the health and economic benefits of these regulations as a case study, using the modeling framework described in Chapter 3. This chapter addresses my second research question (see Chapter 1). I begin by presenting baseline benefits estimates using this new method, and comparing these with others in the literature. I then explore how differing assumptions about uncertain parameters impact estimates, to better understand controlling influences on the estimated values.

4.1 Policy scenarios

I evaluate the benefits of MATS—a dedicated mercury (Hg) policy targeting emissions from CFPPs—against two other policy scenarios: an air quality (AQ) policy only scenario, and a no policy (NP) scenario. Table 4.1 summarizes these three policy cases. MATS, more fully described in Section 2.4, is a Maximum Available Control Technology (MACT) standard, that sets emissions intensity limits (amount. of mercury emitted/unit energy produced) (US EPA, 2011d). These limits are based on the top performing 12% of existing plants for existing sources, and on Best Available Technology (BAT) for new sources. MATS, which was promulgated in 2011, is set to be fully implemented by 2016.

The AQ policy scenario is drawn from the EPA's Regulatory Impact Analysis (RIA) of the benefits of MATS. The EPA considered the benefits of the policy compared to a "base" case that included the Cross-State Air Pollution Rule (CSAPR), and other state and federal air quality policies that were set to come into effect between 2010 and 2016, as of 2010 (US EPA, 2011d). Air quality policies target pollutants like SO_2, NO_x and particulate matter, but have co-benefits for mercury reductions (US EPA, 2011c). Control technologies used to capture SO_2 and NO_x also capture mercury, and in addition, reduced sulfate deposition is thought to reduce the con-

Table 4.1: US policy scenarios used in benefits analysis.

US policy scenario	Description
Air Quality (AQ)	Cross-State Air Pollution Rule (implementation by 2014) and other forthcoming federal and state air quality statutes – sets stringent caps on NO_x, SO_2, and $PM_{2.5}$
Mercury (Hg)	Mercury and Air Toxics Standards (implementation by 2016) – Hg emissions intensity limits on coal-fired power plants
No Policy (NP)	No new air quality or mercury policies (moderate coal growth, and continued use of existing SO_2 control tech. with some mercury co-benefits)

version rate between inorganic mercury, and the more toxic methyl mercury (MeHg) (US EPA, 2011c). Stringent air quality policies may also encourage substitution to other (lower mercury) fuel sources, as they increase the cost of combusting coal.

However, CSAPR—a central part of the AQ scenario—was vacated by the DC Circuit Court of Appeals in 2012, and is not currently in effect. Therefore, in addition to the (now theoretical) air quality policy scenario considered by the EPA, I also consider the benefits of MATS compared to a no policy scenario. The NP scenario assumes that US emissions lie halfway between the IPCC A1B and B1 scenarios created by Streets et al. (2009). This assumption is meant to roughly capture a scenario where no additional air quality or mercury policy is introduced, but existing policies continue until expiration—with moderate coal growth, and most CFPPs employing some form of Flue Gas Desulfurization (FGD) technology to control SO_2 emissions, with minor co-benefits for mercury removal.

4.2 Baseline benefits estimates

In this section, I present the results of the benefits analysis by stepping through the impacts of MATS at each step of the emissions-to-impact path (which was illustrated in Figure 3-1). These steps are: emissions, deposition, exposure, health impacts, and economy-wide welfare benefits.

Table 4.2 identifies the baseline assumptions used in this analysis. These assumptions are associated with uncertainties along the emissions to impact chain (see Chapter 2 for a review). Table 4.2 also shows low and high scenarios for each uncertain parameter, which are used in the uncertainty analysis presented later in this chapter. The numbers to the left of the table link to the number key in Figure 3-1, which illustrates the full chain. The scenarios were drawn from the literature, and are more

Table 4.2: Scenarios used to evaluate sensitivity of exposure and welfare benefits to assumptions.

	Step in the Chain	Uncertainty	Low Case	Base Case	High Case
1	Emissions to Deposition	World Emissions Trajectory	IPCC B1	Average of IPCC A1B and B1	IPCC A1B
		In-Plume Reduction		w/ IPR	w/o IPR
2	Deposition to Exposure	Ecosystem Time Lag	Instantaneous	10 years	50 years
3		Local Diet	All marine/ estaurine	Mix of marine/ estaurine and local freshwater	All local freshwater
4	Exposure to Health Effects	Dose-response Parameters (see Table 3.7)	**Biomarkers**: 2.5th percentile	Central Tendency	97.5th percentile
5			**AMIs**: No causal relationship between Hg and AMIs	Mode	Maximum
			IQ: 2.5th percentile	Central Tendency	97.5th percentile
6	Health Effects to Economy-wide Effects	IQ to Income	**IQ**: 2.5th percentile	**IQ**: Central Tendency of IQ to income coefficient (see Table 3.7)	**IQ**: 97.5th percentile
		Medical Costs	**AMIs**: Medical costs from Russel et al. (1998)	**AMIs**: Average medical costs from US EPA (2011c) literature review	**AMIs**: Medical costs from Wittels et al. (1990)
6	Health Effects to Economy-wide Effects	Discount Rate	No discounting	3%	7%

fully described in Chapter 3.

In this analysis, we consider benefits to the year 2050. This choice was motivated by the availability of emissions projections, but also because an approximately 40 year window reflects the maximum temporal scale of most existing policy efforts at national and international levels (Selin, 2011). However, though a 40 year horizon may be commensurate with the policy process, many of the benefits of mercury reduction will continue to accrue after this point. As a result, the benefits estimates presented in this chapter can be considered a lower bound.

4.2.1 Effects of MATS on emissions

Table 4.3 shows estimates for CFPP emissions in 2005, 2015, and 2050, under the three policy scenarios. Following US EPA (2011d), I show values for 2015 instead of 2016—by when MATS, and the other air quality policies modelled will be implemented—because the model used for emissions projections uses five year intervals. Estimates

for NP–2005, Hg–2015, and AQ–2015 are from US EPA (2011d), while the estimate for NP–2050 is derived from Streets et al. (2009). The remaining estimates (NP–2015, Hg–2050, AQ–2050) are interpolated or extrapolated linearly from these values (see Chapter 3).

For both the Hg and AQ scenario, emissions from CFPPs are at minimum in 2015, at 8 Mg/yr and 26 Mg/yr respectively. However, in all policy cases, emissions increase between then and 2050, in absolute terms, to 8.4 and 27.3 Mg/yr. Increases occur even under Hg policy because MATS sets emissions intensity limits for CFPPs (amt. of mercury/unit energy produced), rather than a strict cap—emissions are therefore expected to grow over time with increasing energy demand.

MATS is expected to substantially reduce mercury emissions from CFPPs, both over time, and when compared to the other policy scenarios. When fully implemented, in 2016, MATS is estimated to lead to a 69% (18 Mg/yr) reduction compared to the AQ policy case, and an 85% (45.6 Mg/yr) reduction when compared to NP—equivalent to 29% and 66% reductions in total US anthropogenic emissions. MATS also leads to a 40 Mg/yr reduction in emissions from 2005. Emissions growth occurs more rapidly in the NP case than in the AQ or Hg cases, with the gap between the Hg and NP case increasing to 64.6 Mg/yr. Emissions in the Hg and AQ case grow at the same rate, so the percentage reduction is maintained to 2050.

There are large regional differences in projected emissions sources, and therefore reductions. Figure 4-1 maps reductions in total anthropogenic (rather than just CFPP) emissions fluxes (μg/m^2/yr) under the three policy scenarios, for 2050. The mercury policy scenario leads to the largest emissions flux reductions in the North East and South East regions relative to the NP case (25-100%), and in the North East and Texas, relative to the AQ case (10-25%).

Table 4.3: Emissions estimates from US coal-fired power plants. Estimates for No Policy (NP)–2005, Mercury Policy (Hg)–2015, and Air Quality Policy (AQ)–2015 are from US EPA (2011d). The estimate for NP–2050 is derived from Streets et al. (2009). The remaining estimates (NP–2015, Hg–2050, AQ–2050) are interpolated or extrapolated linearly from these values.

	2005 (Mg)	2015 (Mg)	2050 (Mg)
Hg	-	8.0	8.4
AQ	-	26.0	27.3
NP	48	53.6	73.0

4.2.2 Effects of MATS on deposition

For each scenario, net deposition is archived from GEOS-Chem—net deposition is the difference between gross wet and dry deposition, of all species, and reemissions (see Section 2.1.1 for an explanation of this concept). It is an indication of mercury enrichment to an ecosystem, and studies suggest that fish in freshwater bodies and the upper oceans will respond (roughly proportionally) to its changes—though on a

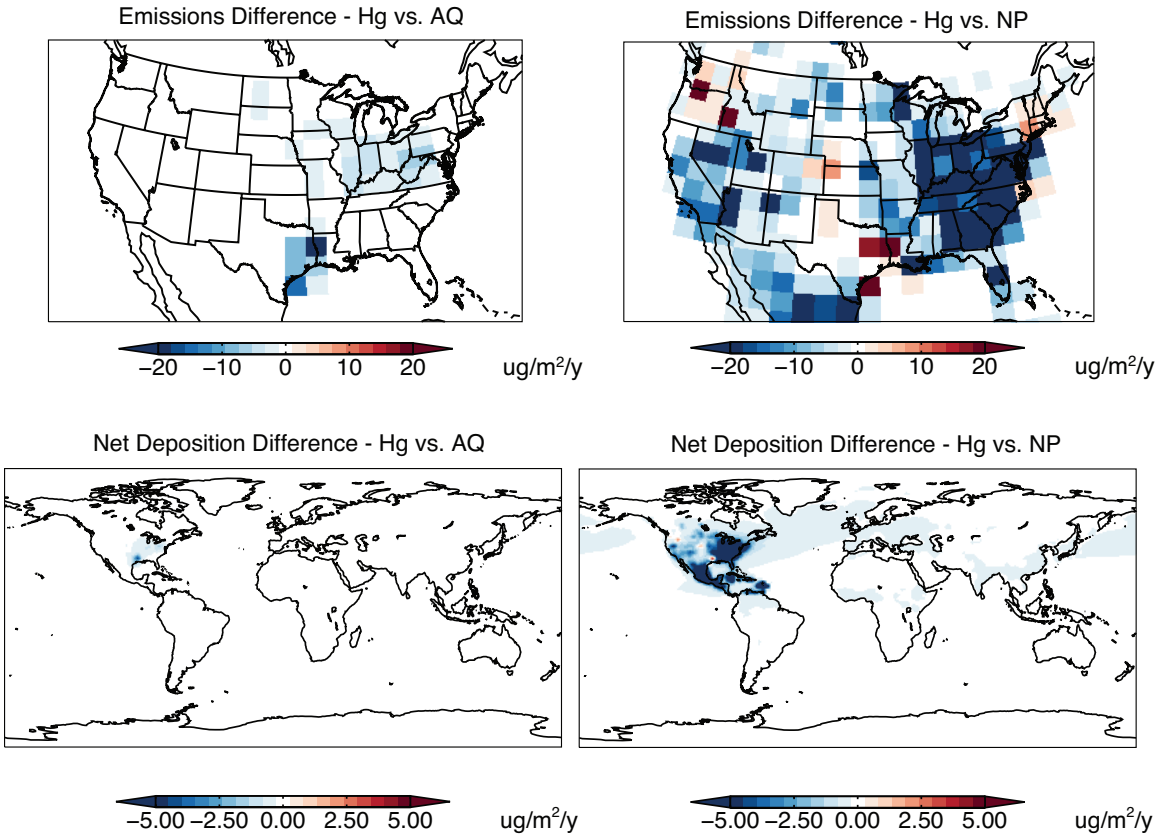


Figure 4-1: Effects of MATS on emissions and deposition, in 2050. Annual emissions and deposition differences between the Hg (MATS) and AQ policy case, and between the Hg and NP case, are shown in $\mu g/m^2$. Emissions are of all mercury species. Net deposition is the difference between gross deposition (wet and dry) and reemissions. Note that the colour scales saturate.

scale of years to decades (Sunderland & Mason, 2007; Mason et al., 2012; Drevnick et al., 2012). This lag is due to continued mercury loadings from non-atmospheric sources that equilibrate more slowly to changes in deposition (like soil) (Chan et al., 2012; Chen et al., 2012b), and the time associated with conversion to MeHg and biomagnification up food chains (Knightes et al., 2009).

The MATS policy scenario reduces simulated net annual deposition to US seafood source regions by approximately 0.4% over the AQ case, and 4% over the NP case (values for 2050). These source regions include the US states, and the Atlantic, Pacific, Indian, and Southern Oceans, as well as the Mediterranean Sea (see Figure 3-3 on page 42 for a map). Over the US alone, the MATS scenario reduces net annual deposition by 14% and 1.3% over the AQ and NP cases, respectively. Figure 4-1 maps reductions in net deposition flux ($\mu g/m^2$), compared to the AQ and NP cases, in 2050. Note that the colour scales saturate. Figure 4-1 shows that the deposition reductions attributable to MATs are concentrated over the contiguous US, though deposition reductions in the North Atlantic and North Pacific are also seen when comparing to the NP case.

The spatial distribution of deposition reductions closely matches that of emissions—with the largest reductions over the North East and South East. For maps of actual deposition, rather than deposition differences, for each policy scenario, see Figures A-2 and A-3, in the Appendix.

The simulated deposition reductions over the US, compared to the AQ case, may be smaller than expected given the size of the emissions reductions, and the estimated percentage contribution of North American sources to US deposition. We may expect that the reduction in deposition would be proportional to the product of the % emissions reduction, and % overall contribution. Selin et al. (2008) and Corbitt et al. (2011) estimate that North American sources contribute 17-32% to deposition over the US. As the projected reductions in total US anthropogenic emissions due to MATS is 29% relative to the AQ case, we may expect that the deposition reduction be slightly lower than the range of 5-9% (taking into account Canadian and Mexican emissions). However, because in-plume reduction was assumed—a chemical phenomenon which is hypothesized to increase the fraction of emissions in the longer-lived gaseous elemental form—these results are less unusual. Zhang et al. (2012) found that including IPR in a nested grid model of the US reduced the percentage contribution of North American sources to US wet and dry deposition to 10-13%. Assuming IPR can therefore dampen the impacts of emissions reductions on local deposition.

4.2.3 Effects of MATS on exposure

Despite decreases in US emissions, US exposure is projected to increase by 2050, regardless of policy case. Figure 4-2 shows exposure changes due to MATS, experienced in 2050. Exposure changes are expressed as % changes relative to 2005, with positive values denoting an increase.[1] By 2050, average US exposure (Panel A of Figure 4-2) increases 30.8%, 19.9%, and 19.2% relative to 2005, in the NP, AQ, and Hg policy scenarios, respectively. MATS therefore avoids a 11.6% increase in exposure compared to the NP case, and 0.7% compared to the AQ case.

The increases in exposure—in spite of US emissions decreases—are due to the dietary patterns of the average US consumer, and growing world emissions. Based on calculations described in Section 3.2.2, 77% of US mercury intake (ie. exposure) comes from marine and estaurine sources–in particular, from the Pacific and Atlantic ocean basins (Sunderland, 2007; Pirrone & Keating, 2010). These ocean basins are strongly influenced by emissions from non-US sources, including East and South Asia, and Russia (Corbitt et al., 2011). Significant emissions growth is expected in these regions by 2050, and so increases in emissions elsewhere in the world offset the exposure benefits of US decreases. In addition, even locally caught freshwater fish are affected by the long-range transport of foreign emissions. Corbitt et al. (2011) and Selin & Jacob (2008) find that 22-23% of US deposition is attributable to primary foreign sources. In this analysis, this fraction is even larger given the IPR assumption. The

[1]The base year for exposure is set as 2005 because this is the most recent year that emissions inventories are available, and because the baseline exposure distributions use data from 1999-2004 (Mahaffey et al., 2009).

influence of these foreign sources is particularly strong in the South East (Corbitt et al., 2011), while domestic sources are more dominant contributors in the North East and Midwest (Selin & Jacob, 2008).

There are strong regional differences in simulated exposure. Panel B of Figure 4-2 highlights three US regions (which are defined in Figure 3-2): North Central, North East, and South East.[2] Overall, exposure increases are lower in the North Central region than in the North and South East because it has lower net deposition (and fewer emissions sources). However, in the North Central region, where local fish consumption contributes close to 60% of mercury intake, the influence of MATS compared to the AQ scenario is larger, with a difference of 1.04% (0.7% is the US average). For the North East, this difference is also slightly larger than the US average (0.80%), however, this is due to the increased influence of domestic emissions on local deposition in this region rather than high local fish consumption rates. In contrast, the South East has a lower than average benefit of Hg policy (0.6%) because its deposition is more heavily influenced by foreign sources.

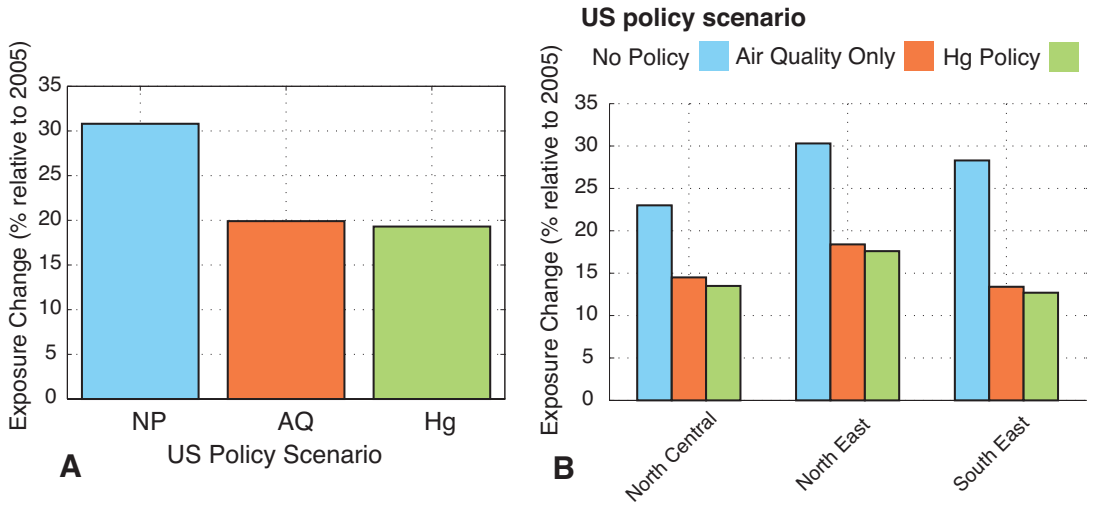


Figure 4-2: Effects of MATS on exposure, in 2050. Exposure changes are expressed as % changes relative to 2005. Positive numbers denote increases. **Panel A** shows the US average, while **Panel B** shows some of the regional variation.

4.2.4 Effects of MATS on health impacts and welfare

Based on model calculations, implementing MATS is projected to lead to 2-40 fewer fatal heart attacks (AMIs) per year, 10-230 non-fatal heart attacks, and 8×10^2-3×10^4

[2]North Central includes North Dakota, Minnesota, South Dakota, Nebraska, Kansas, Missouri, and Iowa. North East includes Ohio, Michigan, Indiana, Illinois, Wisconsin, New Jersey, and Pennsylvania (note, New England has its own region). South East includes Virginia, Kentucky, Tennessee, Mississippi, Alabama, Georgia, South Carolina, and North Carolina.

lost IQ points compared to the AQ only case. It avoids 80-640 fatal AMIs, 500-3900 non-fatal AMIs, and 2×10^4-5×10^5 lost IQ points compared to the NP case. These health impacts are summarized in Table 4.4, and are for the entire US population. The ranges represent the year to year variability, with a general increasing trend in number of avoided health impacts over time.

These health benefits can be translated into economic benefits. Following Matus et al. (2008, 2012) and Nam et al. (2010), I evaluate economy-wide benefits of policy through the welfare metric—the sum of consumption and leisure time, measured as equivalent variation (see Rausch et al. (2010) for details). Briefly, equivalent variation measures of surplus capture the change in income that would bring consumers to the same level of utility, before economic changes. These benefits focus only on avoided costs to the economy, and do not reflect the full value of human life or wellness (they do not include pain and suffering for instance).[3] These estimates are therefore only a lower bound on actual benefits—however, they also offer important insights into the interaction and ripple effects of health impacts on an economy-wide level. Because the model produces an output only at five year periods, following the approach of Matus et al. (2008), I multiply the modelled benefits by five, before discounting, to interpolate for the intervening years.

Welfare benefits due to MATS are estimated at 590 million, or 13 million/yr (2005 USD) compared to the AQ scenario, and 18.2 billion, or 414 million/yr (2005 USD) compared to the NP scenario, for the US population as a whole. These values are present values of the projected future stream of benefits, discounted at 3%. The per year estimates are calculated by dividing the PV benefits by the number of years in the time window considered (44); however this is a simple annualization as benefits vary from year to year. Figure 4-3 plots the present value of the yearly benefit in each modelled period, in constant 2005 USD. Per year benefits are generally increasing over time and range from 2 million to 40 million (2005 USD, PV) over the AQ case, and 90 million-600 million (2005 USD, PV), depending on the year. The increasing trend is due to: population growth, which magnifies the benefits of any exposure reductions; the time lag between avoided IQ loss and and avoided fatalities and their maximum benefit; and compounding effects. In the model, the yearly benefit of avoided IQ loss in an individual is maximized when that individual is in their 40s-50s and at their maximum earning power. Similarly, if mercury exposure reductions lead to an avoided fatality in a 30 year-old individual, then the benefits of that avoided fatality continue to accrue throughout the lifetime of that individual. Finally, welfare benefits to the economy in one year lead to more economic growth in the following—over time, this compounding can contribute to the benefits of policy as well.

The share of benefits attributable to cardiovascular effects ranges from 85-90% depending on the year. This reflects the high economic costs associated with AMIs—particularly with premature fatalities, which shrink the available pool of labor and leisure in the economy. The cardiovascular share increases over time because of the

[3]While leisure is included in the calculation of welfare, in USREP, labour supply involves a choice between labour and leisure at the household level (Rausch et al., 2010)—therefore leisure has market impacts in the model.

Table 4.4: Health and welfare benefits from MATS, compared to the AQ and NP policy scenarios, for entire US population. Ranges given for avoided health impacts represent the year to year variability, starting in 2015. Welfare benefits are the present value stream of future benefits, at a 3% discount rate. Yearly welfare benefits are a simple annualization of the PV benefits (PV/44 yr time window).

	Hg vs. AQ	Hg vs. NP
Avoided Fatal AMIs/yr	2-40	80-640
Avoided Non-fatal AMIs/yr	10-230	500-3900
Avoided IQ Loss (pts)/yr	8×10^2-3×10^4	2×10^4-5×10^5
PV Benefits (2005 billion USD)	$0.590	$18.2
PV Benefits/yr (2005 million USD/yr)	$13	$414

cumulative effects of these premature mortalities—fatalities continue to affect the labour and leisure pool for the duration of their expected lifetime.

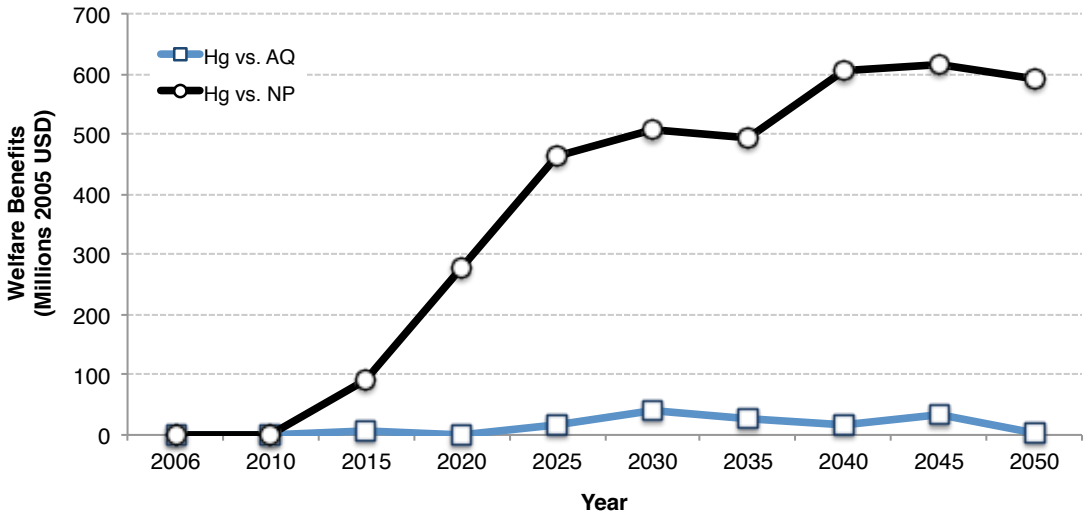


Figure 4-3: Trajectories of future benefits, for the entire US population. The black line represent the benefits of MATS compared to the NP case. The blue line represents the benefits of MATS over the AQ case. Markers indicate modelled years. All values are discounted to present, at 3%.

4.3 Uncertainty analysis

In this section, I conduct an uncertainty analysis to evaluate the sensitivity of benefits estimates to assumptions about the uncertainties along the emissions-to-impact path. This analysis is meant to provide (quantitative) insight into which processes and factors—and at which point in the path—control benefits estimates, and where uncertainties need to be better constrained. For simplicity, this section focuses on the

Table 4.5: Range of benefits from high and low uncertain parameter assumptions, given as percentage changes from the baseline.

Step in the Chain	Uncertainty	Range
Emissions to Deposition	World Emissions Trajectory	-37% to +33%
	In-Plume Reduction	+12%
Deposition to Exposure	Ecosystem Time Lag	-72% to +35%
	Local Diet	-45% to+12%
Exposure to Health Effects	Dose-response Parameters	-79% to +41%
Health Effects to Economy-wide Effects	IQ to Income	-11% to +13%
	Medical Costs	
	Discount Rate	-66% to +155%

benefits of MATS compared to the NP scenario only.

For each uncertainty (rows in Table 4.2), benefits were calculated with high and low case assumptions, holding all other assumptions constant at the base case. These assumptions are specified in Table 4.2. The results of the analysis are summarized in Table 4.5, Figure 4-4, and Figure 4-5. Table 4.5 shows the resulting range of benefits from the high and low cases as percentage changes from the baseline. Figure 4-4 shows these ranges graphically—with all benefits are given as the present value of the future stream, discounted at 3%. Figure 4-5 shows the trajectories of future benefits, again, discounted at 3%. The black line shows the base case trajectory, while the grey area indicates the range of benefits for the scenarios considered. The red, blue, and orange dotted lines mark the bounds of this range and correspond to the high and low cases of the dose-response paramaterizations, ecosystem time lags, and emissions scenarios (only the high case is shown, because the low case does not act as a bound) respectively. Other grey lines show trajectories for all other scenarios. Discount rate—because it leads to the largest range—has been excluded from the figures so that the other uncertainties can be more clearly visualized, however, is included in Table 4.5. The remainder of this section discusses the sensitivity of benefits to uncertainties at each step of the emissions to impact chain, sequentially.

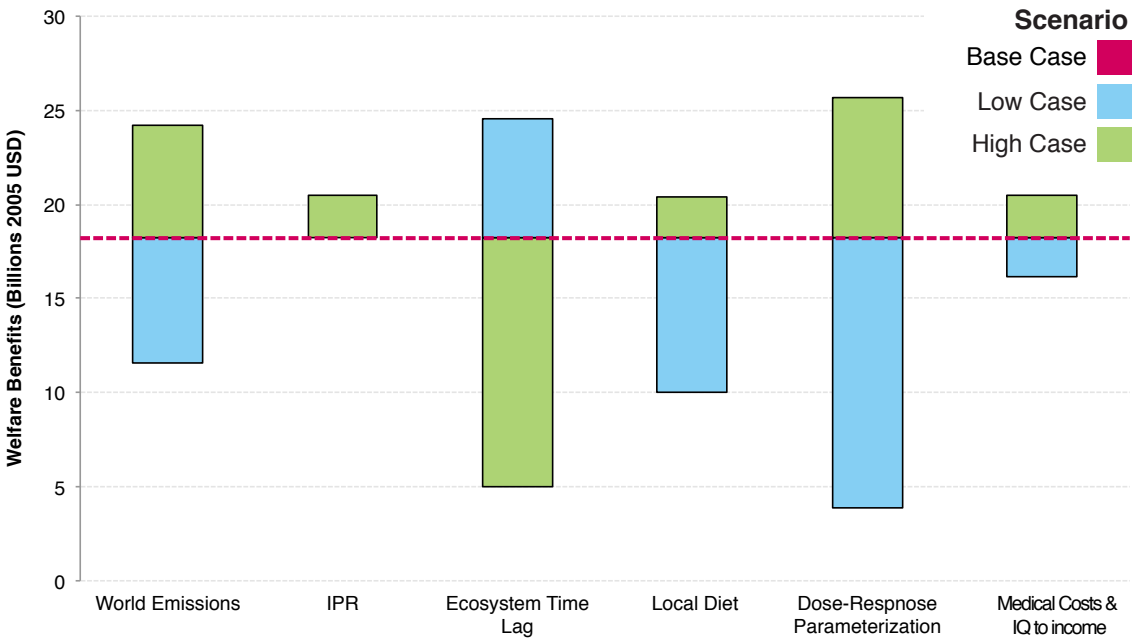


Figure 4-4: Sensitivity of welfare benefits, up to 2050, to assumptions about key uncertainties. Benefits shown are cumulative, to 2050, and discounted to present value at 3%.

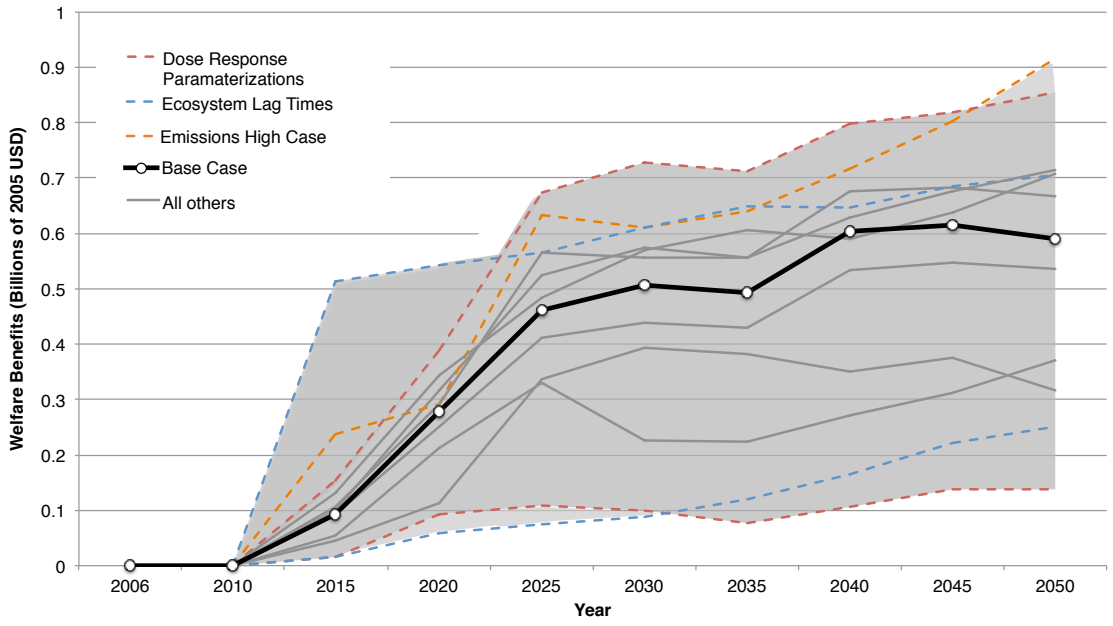


Figure 4-5: Trajectory of future benefits. The black line shows the trajectory for the base case. The grey area indicates the range of benefits for the scenarios considered. Red and blue dotted lines mark the bounds of this range and represent best and worst case dose-response paramaterizations and ecosystem time lags, respectively. Grey lines show trajectories for all other scenarios. All trajectories are shown with 3% discounting.

4.3.1 Emissions to deposition

The high and low world emissions scenarios—based on the IPCC A1B and B1 scenarios (Streets et al., 2009; Corbitt et al., 2011)—lead to a +33% to -37% change in benefits (or 9.19 to 19.38 B 2005 USD, in absolute terms). For IPR, there are only two scenarios rather than 3: with IPR (the baseline assumption) and without. Removing the IPR assumption led to a +12% increase in benefits (20.5 B 2005 USD).

World emissions strongly affect US exposure, and therefore benefits. They lead to the the third largest range of benefits when compared to all factors—and is the only uncertainty, of these three most influential, that is directly under the influence of human control. The range of world emissions scenarios has a larger effect on US exposure to mercury than the range of domestic policies considered. Figure 4-6 compares these two effects in 2050. It plots simulated average US exposure, expressed as % change over 2005, under the high and low case world emissions scenarios, as well as an additional no growth scenario, which assumes that world emissions stay at 2005 levels. For each world emissions scenario, the effect of different US policy scenarios is shown according to the colour scheme. Moving from the high to low world emissions scenario reduces exposure increases over 2005 by 50%, whereas implementing MATS has a maximum reduction of 20%, when compared to a no policy scenario. As mentioned in Section 4.2.3, the strong influence of world emissions on exposure is due to the geographic origin of fish consumed by the general US population, and global transport of emissions. The exposure of the average US consumer is more heavily controlled by marine fish—which are more sensitive to emissions changes elsewhere in the world, than by local freshwater fish—which respond more strongly to domestic US policy (Sunderland, 2007). Even for those who eat primarily local fish (eg. recreational anglers, subsistence fishers, certain indigenous peoples), primary foreign emissions contribute at least a fifth to mercury deposition over the contiguous US (Selin et al., 2008; Corbitt et al., 2011).

The relatively small effect of IPR on benefits can be explained using similar reasoning. IPR is a hypothesized chemical phenomenon whereby the fraction of mercury emissions from CFPPs that are in the atmospherically long-lived form is increased (Lohman et al., 2006; Edgerton et al., 2006; Weiss-Penzias et al., 2011). Removing the IPR assumption is expected to increase the benefits of US policy, because it causes US emission sources to have a larger impact on US deposition. Removing the IPR assumption increases the estimated benefit of MATS against no policy by 12%. However, given that, in the model, removing IPR decreases the Hg(0) fraction of emissions substantially, from 89.2% to 56.8%, this effect may be smaller than expected. This discrepancy can also be explained by the fish sources of the average US consumer. Because most consumers eat marine fish, their exposure is less sensitive to decreases in US deposition.

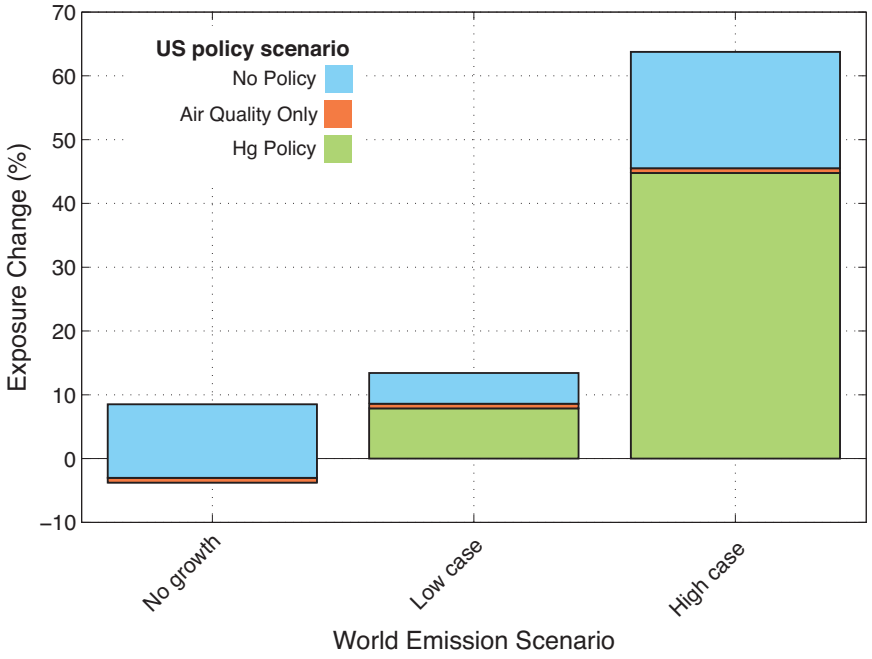


Figure 4-6: Comparing the effects of world emission scenario and US policy scenario on US exposure. Exposure, expressed as % change over 2005 exposure, is shown for 2050, assuming no time lag between deposition reductions and fish response.

4.3.2 Deposition to exposure

The range of ecosystem time lags considered—no lag to 50 years—changes benefits by +35% to -72% against the base case (19.66 to 3.84 B 2005 USD), with the larger benefit associated with no lag. Note that the range is unevenly distributed around the base case in Figure 4-4 because a 10 year lag is assumed in the base case. Variations in time lag led to the second largest range of benefits out of the variables considered. A larger time lag reduces the present value of the stream of future benefits because it pushes many of the benefits beyond the time horizon. For instance, with a 50 year time lag, the benefit of the emissions reductions in 2012 are not fully reflected in human exposure until 2062. In addition, delayed benefits are more heavily discounted. This effect can be seen in Figure 4-5, which shows trajectories of future benefits under the different assumptions. The blue dotted lines represent the trajectories for the 50 year lag (bottom blue line), and no lag (top blue line), while the base case is shown in black. The percent exposure reduction experienced in 2015, with no time lag, is delayed until 2025 in the base case. With discounting though, the benefit associated with this exposure reduction is lower in 2025. Without discounting however, the same exposure reduction, experienced further in the future, will actually result in a larger benefit than if experienced instantaneously—this effect is due to population growth. These competing effects demonstrate how multiple uncertainties (in this case, ecosystem time lag and social discount rate) can interact to affect estimated benefits.

The two extremes for diet considered for this sensitivity analysis were an all local freshwater fish diet, and an all marine and estaurine diet. These scenarios result in a

+12% to -45% change in benefits over the base case (or 10.0 to 20.4 B 2005 USD). The benefits of US policy are expected to be greater for individuals who eat local fish than for those who eat internationally sourced marine and estaurine fish because US sources contribute a greater fraction to US deposition than to deposition in oceans—this expectation is confirmed by the results. This finding suggests that for vulnerable populations who consume large quantities of locally sourced fish (see Section 2.2.2), the benefits of MATS may be much larger than for the general population, the diet of which more likely resembles the all-marine scenario. The distributional implication of MATS, and how these may be incorporated into policy decision-making, are discussed in the next chapter.

4.3.3 Exposure to health effects

Currently, dose-response relationships are considered the most uncertain part of the emissions-to-impacts chain (Axelrad et al., 2007; Roman et al., 2011), and this was reflected in the analysis results. Variability in the parameterization of the dose-response relationships, linking mercury exposure to health impacts, leads to the largest individual range of benefits: -79% to +41% compared to the base case (3.9 to 25.6 B 2005 USD). This is largely driven by uncertainty in cardiovascular impacts: the low end estimates of the dose-response parameters assume that there is no causal relationship between mercury exposure and increased risk of heart attacks, while the high end assumes an elevated risk of 17% per μg/g hair mercury (Virtanen et al., 2005). However, it should be noted that time lag uncertainties lead to a very comparable range, and may be more important when considered in combination with discount rate uncertainty.

Though there is considerable uncertainty in these parameterizations, because an increasingly large body of epidemiological research on this topic exists, probability distributions, rather than just ranges, can be created for each of these parameters. These probability distributions can be used in a more detailed quantitative uncertainty analysis—like that conducted by Rice et al. (2010), which focused on dose response uncertainties. However, this sort of probabilistic analysis is not yet possible for every uncertainty along the emissions-to-impact path, and cannot necessarily be used to compare relative contributions of different steps in the path to total benefits. Considering how multiple kinds of uncertainty analysis—probabilistic, scenario, sensitivity, and qualitative—can be comprehensively represented in an integrated assessment framework may be a topic of future work.

This analysis focused on uncertainty from the magnitude of dose-response parameters rather than the shape of the the dose-response curves themselves. However, the results above demonstrate that benefits can be very sensitive to the shape of the response curve. Some evidence suggests that the dose-response relationship may be supralinear for IQ as well as heart attacks (Rice, 2004; Axelrad et al., 2007). Additional uncertainty characterization that explores the effects of different response shapes for both IQ and cardiovascular effects may provide further insight into the range of potential welfare benefits.

4.3.4 Health effects to economy-wide effects

Changing the valuation of medical costs associated with AMIs, and of a lost IQ point, changes benefits by -11% to +13% (16.2 to 20.5 B 2005 USD). For IQ, the high and low scenarios corresponded to the 97.5^{th} and 2.5^{th} percentile of the IQ-to-percent earnings coefficient. For non-fatal AMIs, the scenarios corresponded to variations in the estimated medical costs of an acute coronary event in the literature (Wittels et al., 1990; Russell et al., 1998; US EPA, 2011c). Because I treat fatal AMIs as a shock to the labour pool, there are no separate valuation assumptions for this endpoint, aside from the assumptions about dose-response relationships (and therefore number of fatal heart attacks). The small range indicates that many of the economic impacts of mercury exposure are attributable to fatal AMIs—which were not affected by the high and low valuation scenarios—and cumulative economic effects.

Increasing or decreasing the discount rate from 3% to 7% and 0% (ie. no discounting) changed the baseline benefit estimate by -66% to +155% (6.2 to 46.6 B 2005 USD). Discount rate has a large effect on estimated benefits because yearly benefits grow over time (see Section 4.2.4)—with the largest yearly benefits of policy occurring several decades after emissions reductions occur (see Figure 4-5). Again, because mercury is long-lived, and its effects are long-acting, choice of discount rate interacts strongly with many other key uncertainties in the emissions to impact path.

4.4 Discussion

4.4.1 Comparing the impact of US mercury policy to results from other studies

Given differences in assumptions, differences between the benefits estimated in this analysis and those reported in similar studies are reasonable. Table 4.6 compares the benefits from this analysis to the most relevant valuation studies in the literature: the Regulatory Impact Assessment (RIA) prepared by US EPA (2011d), comparing MATS to an AQ scenario; and a benefits assessment by Rice et al. (2010), which includes both cognitive and cardiovascular endpoints, and which assumes a similar exposure reduction as that between the MATS and NP scenarios ($\sim$10%). (For a wider comparison, to other valuation studies that focus on the US, see Table A.2 in the Appendix.) To facilitate comparisons, following Rice et al. (2010), I have represented benefits as \$/(μg/day) per capita in addition to annualized gross benefits. For this present analysis, and that by Rice et al. (2010), the population used in the per capita calculation is the entire current US population, while for the estimates from US EPA (2011d), I used only the freshwater angler population ($\sim$10% of the whole), as benefits were only considered in this subpopulation.

Because the studies use fundamentally different approaches, direct comparisons can be difficult. Most existing valuation studies for mercury track the benefits to a single birth cohort that experiences reduced fetal exposure, over the whole lifetime

Table 4.6: Comparison of estimated benefits to relevant studies in the literature. Results from the MATS vs. AQ benefits calculation are compared to estimates from US EPA (2011d), who consider the same scenarios. The MATS vs. NP estimate is compared to those from Rice et al. (2010), who also consider both cardiovascular and cognitive endpoints, and assume a similar exposure reduction ($\sim$10%) as that modelled through MATS vs. NP. The heavy black line in the table separates the pairings.

Analysis	Yearly avoided health endpoint	Population Considered	Annual Benefit	$/(µg/day) per capita pop.
This work: MATS vs. AQ	**Fatal AMIs:** 2-40	All US, > 30 yrs old	13 M 2005 USD	4.60
	Non-Fatal AMIs: 10-230			
	IQ: 800-3x10^4 pts	All US, yearly newborns		
US EPA (2011d): MATS vs. AQ	**IQ**: 510 pts	Freshwater anglers, yearly newborns	3.5-5.2 M 2005 USD	0.58-0.88 (freshwater angler pop)
This work: MATS vs. NP	**Fatal AMIs:** 80-640	All US, > 30 yrs old	414 M 2005 USD	8.90
	Non-Fatal AMIs: 500-3900			
	IQ: 2x10^4-5x10^5 pts	All US, yearly newborns		
Rice (2010): 10% exposure reduction	**Fatal AMIs:** 130	All US, > 30 yrs old	775 M 2005 USD (expected value)	5^{th} percentile: 1.44 50^{th} percentile: 6.58 95^{th} percentile: 105
	IQ: 4x10^4 pts	All US, yearly newborns		

of that cohort—or of a single yearly cohort of AMI patients. In contrast, the analysis presented in this thesis uses a general equilibrium (GE) approach to value benefits: the benefits I report are to the economy as a whole, calculated by taking snapshots of the economy at modelled 5 year periods, until 2050. The GE approach therefore considers multiple cohorts, but not throughout their whole lifetime of benefits. Focusing on benefits to the economy itself can allow for accounting of broader economic effects—which can represent a substantial fraction of total benefits. For instance, Nam et al. (2010) find that, for $PM_{2.5}$, a pollutant with a wide range of cardio-pulmonary health effects, cumulative benefits that occur through investing the welfare gains of previous years back into the economy, account for 12% of the total. However, by focusing on the benefits to individuals, the single cohort approach is better able to account for non-market effects like pain and suffering. For instance, Rice et al. (2010) use the Value of Statistical Life (VSL) method to value premature fatalities. VSL measures the willingness to pay for a small reduction in risk of mortality, normalized to 1 (Cameron, 2010). Willingness to pay measures may better capture effects that do not have market prices—as many health-related effects do not. Nevertheless, in spite of these differences, reasonable comparisons to other estimates, with explainable differences, may be a test of the validity of the new assessment method developed in this thesis.

US EPA (2011d) estimates that the health benefits of MATS over an AQ scenario are an avoided loss of 510 IQ points (per birth cohort), leading to an annual benefit of 3.5-5.2M 2005 USD.[4] These values are smaller than that estimated in my analysis (13 M 2005 USD/yr), however, I consider avoided AMIs in addition to IQ loss, and over a wider population. For assumptions that better match those used by the EPA, we can consider the low scenario for dose-response parameterization used in the uncertainty uncertainty analysis (Section 4.3.3). This scenario assumes that there is no causal relationship between mercury and cardiovascular effects (however also assumes low 2.5^{th} percentile estimates of all other dose-response parameters)—and leads to a benefit estimate of 8 M 2005 USD/yr for the whole US population. US EPA (2011d) projected that MATS would lead to an approximately 6% decrease in exposure for the subpopulation of freshwater anglers (about 10 times as large as the 0.6% US average decrease projected in my analysis[5]). That the US EPA estimate is about a third to half as a large as one which includes more health impacts, and a larger population, suggests that the benefits of MATS (compared to an AQ scenario) may be concentrated in vulnerable populations highly exposed to mercury through freshwater fish. Because I average over regions, these large, subpopulation specific effects are not as well captured in my estimates.

Rice et al. (2010) were the first group (to my knowledge) to include cardiovascular effects in their benefits accounting for mercury reductions. They estimated that a 10% exposure reduction (which is almost directly comparable to the 9.7% reduction projected in the current results) will lead to approximately 130 fewer fatal AMIs,

[4] I have converted their estimates to 2005 real dollars to facilitate comparison.

[5] Note that to facilitate comparison, this % decrease is expressed as % decrease in ug/day intake now, rather than as % change over 2005 exposure.

and $4x10^4$ fewer lost IQ points, annually. Using a probabilistic approach to dose-parameterization uncertainty, they find that the expected value of these reductions is 775 M 2005 USD/yr, with a 5^{th}-95^{th} percentile range of 45 M - 3.2 B.[6] This range is skewed towards the high end, with a long tail, because of the uncertainty associated with mercury-related heart attacks. The estimated number of avoided fatal AMIs and lost IQ points projected in my analysis (80-640, and $2x10^4$-$5x10^5$) are similar to those from Rice et al. (2010), with their estimates falling within my ranges. The low end of this range corresponds to years that fall within the lag time between when deposition reductions occur, and when the 9.7% exposure reduction is fully realized. Rice et al. (2010) did not consider an ecosystem time lag—though they did consider uncertain heart attack cessation lag times (between reduced exposure and reduced risk) ranging from 2 to 10 years. The high end corresponds to later years, by which time the total US population has grown. Because the number of avoided health effects considered is roughly the same, the differences in annualized benefits reflect differences in valuation approaches. The 414 M annual benefit (or 8.90 \$/(μg/day) per capita) is slightly larger than the 50^{th} percentile estimate from Rice et al. (2010), but lower than their expected value estimate—despite the fact that I also include non-fatal AMIs, and broader economic effects in my valuation. Rice et al. (2010) use the VSL approach to quantify the benefits of an avoided fatality—their distribution for this parameter is triangular, with a mode of 5.5 M USD/death, and a minimum and maximum of 1 and 10 M USD/death. Their larger expected value estimate therefore represents additional, non-market benefits, that are not captured in a GE economic model.

Even when normalized to \$/(μg/day) per capita, the benefits of MATS over the AQ scenario are lower than the benefits over the NP scenario. This may be surprising, because we may expect that the benefit, per unit reduction of mercury exposure, is constant. This finding highlights the non-linearities in mercury's potential cardiovascular impacts. Based on existing evidence, the dose-response relationship between mercury exposure and heart attacks is thought to be log-linear—steeper at lower doses (Guallar et al., 2002; Roman et al., 2011). As a result, a large exposure decrease that moves an individual into the steep region of the dose-response curve can have a supralinear effect. Because 85-90% of the calculated benefits are attributable to cardiovascular effects (in this work, and supported by values reported by Rice et al. (2010)), these non-linear effects dominate. The larger exposure reduction of MATS over NP compared to MATS over AQ therefore leads to larger benefits per unit intake reduction. This finding suggests, more broadly, that benefits estimates are sensitive to baseline exposure assumptions. Sensitivity to this parameter may therefore be important to consider in future studies.

Compared to other air pollutants, welfare benefits associated with mercury policy may be relatively small. For instance, the OMB (2003) has estimated that the benefits of the Acid Rain Program, which targeted SO_2 and NO_x emissions from power plants, had annual benefits upwards of 70 B USD. These pollutants, as well as the secondary pollutants they create (especially particulate matter), are known to have a wide range of well-proven cardiovascular and pulmonary effects (Pope & Dockery, 2006). Nam

[6] I have converted their estimates to 2005 real dollars to facilitate comparison.

et al. (2010) estimated that reducing emissions of these pollutants, as proposed in the Clean Air for Europe policies, could lead to benefits of 37-49 B Euro/yr, and Matus et al. (2012) found that China could have achieved annual benefits of 12-47 B USD over the past thirty years had it implemented feasible air quality targets.

However, even though the benefits due to mercury-related health impacts are relatively small, the benefits of implementing MATS as a whole are still likely to be large—far outweighing its costs of implementation. US EPA (2011d) estimated that the costs to industry of implementing MATS would be 9.6 B USD/yr, while the total social benefits of the policy (including both mercury and non-mercury related impacts) ranged from 37-90 B USD/yr. Just as air quality policies can have co-benefits for mercury, mercury policies can have co-benefits for other pollutants. Benefits associated with other pollutants—particularly particulate matter—make up the remainder of the benefits. The results of this analysis indicate that the total benefit of MATS is likely to be even larger than estimated by the EPA, when cardiovascular impacts in the entire US population are included. However, US EPA did not use a GE approach in estimating the costs of implementing MATS, and therefore may have underestimated the economy-wide costs (compared to just industry-specific costs). Exploring the costs of implementing MATS in a GE framework may be a topic of future work. Waugh (2012) has illustrated a method for doing so for non-mercury air quality policies—and the importance of including these economy-wide effects for costs as well.

4.4.2 Summary of uncertainty analysis and extensions

This analysis has made many simplifying assumptions. Nevertheless, several useful insights have been gained that would not have been possible without a full representation of the emissions-to-impacts chain—even if only at a first approximation. In particular, this work has improved our understanding of how different uncertainties along the chain may affect benefits estimates. In this section, I summarize key findings from the uncertainty analysis and discuss limitations and possible extensions.

World emissions. Of the uncertainties that estimated benefits are most sensitive to, future world emissions may be the one over which humans have the most direct control. How optimistic or pessimistic is the range of uncertainties considered? In Section 2.4.2, I described the forthcoming Minamata Convention—an effort by the global community to regulate mercury on an international scale. A key function of this treaty is preventing future exposures to mercury by controlling and reducing emissions of mercury to air (UNEP, 2013b). Selin (2013) has estimated how treaty requirements may translate into actual emissions trajectories. She argues that the provisions for controlling emissions—which rely on a Best Available Technology approach that takes into account socio-economic considerations—will likely result in a total emissions budget towards the low end of the range projected by Streets et al. (2009). The high and low bounds of the Streets et al. (2009) range were used as the high and low emissions trajectories in the uncertainty analysis. If the Minamata Convention does in fact lead to emissions towards the low end of this range, Figure 4-6 suggests

that the benefits for US exposure may be large. However, treaty requirements for emissions may not officially be in effect until 2025 (Selin, 2013)—and the results of the uncertainty analysis indicate that the timing of emissions, and subsequent exposure reductions, have a strong effect on benefits.

Discount rate. Because there is a lag time between emissions reductions and reductions in human exposure—which partially contributes to the increasing yearly benefits of mercury policy over time—the choice of social discount rate can drastically change the magnitude of estimated benefits. Assumptions about discount rate can therefore mask the effects of other uncertainties. As a result, it is critical to recognize that uncertainties related to the timing of exposure reductions (or the timing of health effects) are coupled to uncertainties about discount rate propriety. Keohane (2009) has made a similar argument for particulate matter-related air quality benefits. He argues that it is important not only to elucidate the nature of lag structures themselves (in the case of particulate matter, these are related to lags in health effects), but to clarify, empirically, society's disposition towards discounting future health benefits and risk reductions at all. In Regulatory Impact Analyses, it is standard to include benefits discounted at 3% and 7% rates. The uncertainty analysis carried out in this thesis suggests that including a wider range of rates (including no discounting) may be important for benefits assessments related to mercury—if only to highlight where and how interactions occur between discount rate and other uncertainties.

Dose-response parameterizations. Aside from discount rate, estimated benefits were most sensitive to dose-response parameterization. In particular, the large range of benefits is driven by uncertainty in the relationship between mercury exposure and cardiovascular impacts. When included, cardiovascular health benefits, like avoided AMIs, make up the bulk of welfare benefits (85-90% in the base case), a finding that is supported by Rice et al. (2010). These results indicate that though cardiovascular impacts are more uncertain, they can be highly relevant for policy-making, and efforts should be made to include them in benefits analysis. Better constraining these relationships should be a research and policy priority.

Ecosystem time lag. The range of ecosystem time lags considered (no lag to 50 year lag) led to a range of benefits that was only marginally smaller than that from dose-response parameterizations. These results highlight the importance of ecosystem dynamics for benefits accounting. In the modelling framework developed, these dynamics were simply parameterized as an ecosystem time lag, however, given the importance of this link in the emissions-to-impact chain, coupling to a full ecosystem model may be an useful next step. For instance, Knightes et al. (2009) and Chan et al. (2012) have developed dynamic food web models that explicitly capture processes of methylation and trophic transfer in freshwater lake and stream systems. Unfortunately, scientific understanding of these processes for marine and coastal ecosystems—which are critical fish sources for US consumers—is not yet as advanced as for freshwater systems (Chen et al., 2008). Further research in the dynamics of MeHg accumulation in marine food webs is needed.

In-plume reduction. The effect of IPR on benefits range was found to be relatively small compared to the other variables considered in this analysis; however,

because IPR was limited to US sources, the true effect may be larger. IPR refers to a chemical reaction hypothesized to take place in power plant plumes that changes the speciation of emissions—that is, the fraction of emissions in long-lived or short-lived atmospheric forms (Lohman et al., 2006). The uncertainty analysis found that the effect of speciation changes in US CFPP plumes on benefits is small (12%) because speciation changes in US sources primarily affect deposition over the contiguous US—whereas, the bulk of US mercury intake is scaled to deposition over open oceans. However, due to data constraints, IPR was not implemented in CFPP sources from non-US CFPP sources, nor in plumes from waste incineration sources (in which IPR is also hypothesized to occur (Zhang et al., 2012))—US or foreign. The impact of including IPR in foreign sources may depend on the nature of source-receptor relationships. For instance, modelling studies have found that Asia is the largest contributor to mercury deposition in the oceans (UNEP, 2008; Corbitt et al., 2011)—implementing IPR in Asian emissions may therefore increase deposition to oceans, with resultant increases for US exposure.

Dietary choices. The results suggest that the benefits of domestic mercury policy for US exposure are muted for the general population because the average consumer eats fish from marine, rather than local, freshwater sources (Sunderland, 2007). However, benefits were increased (by 12%) when exposure for the whole US population was scaled to domestic deposition, and in the base case, larger benefits were observed in regions where a larger fraction of the population (eg. recreational and subsistence fishers, and communities that maintain a traditional diet) consumes locally caught freshwater fish. These findings may indicate a need for further disaggregation to better understand effects on vulnerable, highly exposed populations. The methods used for exposure and benefits accounting involved averaging over each of the twelve US regions. While disaggregating the US into twelve regions is already an improvement over some existing studies, further disaggregation is possible—and potentially useful for policy-making. Because exposure distributions are long-tailed (Mahaffey et al., 2004, 2009), and cardiovascular impacts may be non-linear (Guallar et al., 2002; Roman et al., 2011), averaging across a region may overestimate effects for the general population, and grossly underestimate the effects for vulnerable, highly exposed ones. Finer resolution modelling over specific watersheds of interest using a nested-grid simulation embedded in the global simulation (Zhang et al., 2012) may be a topic for future research.

Valuation of medical costs, and lost IQ. In this analysis I conducted only a partial evaluation of the valuation of health endpoints. I considered uncertainty ranges for the value of medical costs associated with AMIs, and the value of a lost IQ point in terms of income, and found that these ranges had a relatively small impact on benefits. However, because of the way premature fatalities were implemented in the general equilibrium economic framework, they could not be included in the uncertainty analysis. Premature fatalities were modelled as losses to the pool of available labour and leisure for the duration of expected life, and therefore, the value of these fatalities was calculated endogenously within the model. In contrast, previous studies have used a VSL approach to value mortality (Rice et al., 2010; US EPA, 2011c), and

found that total benefits are highly sensitive to its estimated value, compared to all other health endpoints.

Uncertainties not considered. This analysis focussed on the effect of future emissions (under US policy, and under different world growth trajectories) on benefits. While it is important to isolate the effect of emissions, understanding the effect of future climate may be equally important. Friedman et al. (2013) explored the effect of both on the atmospheric transport of Polycyclic Aromatic Hydrocarbons, and found that there was a "climate penalty" for some compounds, which reduced the benefit of decreased emissions. Climate change is likely to have an effect on mercury's biogeochemical cycling (through increased wildfires and lost peatlands for instance), increasing mobilization of mercury to air (Selin, 2009). Climate change may also affect fisheries and aquaculture (Brander, 2007), changing dietary patterns and human exposure to MeHg. Quantifying the potential influence of climate, and how it attenuates emissions reductions will be important—particularly as it relates to monitoring the effectiveness of domestic and international policy on reducing ecosystem concentrations of mercury (Selin, 2013).

A second key uncertainty that was not explored relates to the form of the economic model used. General equilibrium models are commonly used in policy analysis because they capture economy-wide effects, and interactions due to price changes in one sector (in the case of this analysis, in household health) (Bandara, 1991). However, GE models have also been criticized for the theoretical assumptions upon which they are based: that agents are perfectly optimizing, and deterministic (Bhattacharyya, 1996). Efforts have been made to improve the realism of these models, particularly with regards to decision-making under uncertainty for modelled agents (Webster et al., 2012). The true economic effects of mercury policy are likely to lie somewhere between those modelled in a perfectly optimizing economy and those estimated through strict, one sector (health) cost-of-illness approaches. However, using a GE analysis—though imperfect—still provides important exploratory insight into the wider economic benefits of mercury emissions reductions, and how they accrue over time. In the future, endogenously coupling changes in economic growth to emissions in the GE framework could also improve its realism.

Chapter 5

Policy Implications

In the last chapter, I evaluated the Mercury and Air Toxics Standards using the new assessment framework developed in this thesis. In this chapter, I attempt to answer my third research question: what additional policy-relevant insights can be gained from an assessment that more completely represents the emissions-to-impacts chain when compared to previous studies?

Results from the case study indicate that the benefit of improved completeness and realism in a benefits assessment tool may not necessarily be in improving aggregate benefits estimates. Though the new method did lead to different median estimates, the uncertainty ranges from most studies (this work included) overlapped (which may also hint at the need to better constrain uncertainties). However, using a full representation of the emissions-to-impacts does allow us to better understand what controls this benefits number. Which factors, and at which point in the chain, affect exposure? Which factors affect the valuation of exposure? How? As a result, the method developed in this thesis may have a comparative advantage not in answering the question of what is the value of policy, but questions about how to design intelligent policy, and how to assess it. I identify three policy questions that the case study results speak to:

1. Where should a mercury policy be implemented to maximize US benefits?
2. What kinds of policy are needed to protect human health?
3. How should mercury policies be assessed?

In the remainder of this chapter, I address each of these in turn. I outline what the results of the analysis suggest for each question, and then situate these findings in the broader policy context.

5.1 Where should a mercury policy be implemented?

5.1.1 Encouraging global action on mercury

The analysis results indicate that for the average US consumer, emissions decreases elsewhere in the world may have a larger impact on exposure than domestic mercury policy. Because there are costs associated with implementing a domestic mercury policy (the costs of MATS are estimated at 9.6 B USD/yr (US EPA, 2011d)), some might argue that it would be more economically efficient if the US used these funds to endorse emissions reductions in other countries that are major emitters—in particular, emitters that contribute significantly to deposition over oceans (East Asian states for instance (UNEP, 2008)). This sort of direct exchange is often called a Coasian bargain (Coase, 1960). To a certain extent, these sorts of direct exchange relationships have been established through the UNEP Global Mercury Partnership program (UNEP, 2013c). However, (since 2009) the US has also been a proponent of developing a global, legally binding treaty for the regulation of mercury (Selin, 2011). The text for this treaty—the Minamata Convention—was finalized in January of 2013 (UNEP, 2013b). For a more in depth review of the Convention, see Section 2.4.2.

A global treaty may be a more effective way of encouraging emissions reductions abroad because it creates shared norms and institutions (Chayes et al., 1995). Establishing norms may in fact be a lower cost method of influencing behaviour, over the long term, than simple exchange because it can lead states to internalize new priorities—reducing the need for inducements (or punishments) at all (Nincic, 2010). Finally, because mercury can act as a global pollutant—and action on mercury is therefore prone to collective action problems [1] (Olson, 1984)—creating selective incentives (like creating global norms, and providing financial and technical assistance upon participation) through the treaty process reduces free-riding problems.

5.1.2 Rationales for domestic action

While the analysis results support endorsing a strong international treaty, there are several reasons why the US may still want to implement a domestic mercury policy—even if it is not the most economically efficient way of reducing average US mercury exposure: agreement upon a strong international treaty may not have been possible if the US had not shown domestic leadership on the issue; policy may be motivated by environmental justice and distributional equity concerns rather than by economic efficiency; and regulating mercury emissions from CFPPs may be an indirect form of climate policy.

[1] Collective action problems occur when group action would lead to the greatest benefit for all, however, because the costs of action are borne privately, but benefits are shared amongst a group, there is a strong incentive to "free-ride."

Improving influence in international negotiations

Like in other environmental regimes, achieving international consensus on mercury has been difficult (IISD Reporting Services, 2013). Multilateral environmental agreements, and the process by which they are created, must be legitimate (fair), salient (relevant), and credible to all parties (Biermann & Pattberg, 2008). These can be challenging requirements given the diversity of needs, world views, and abilities amongst the international community—particularly between countries in the Global North and South. Mercury, because it is long-ranged and long-lived, can introduce its own particular set of international diplomacy challenges. Chief amongst these is the question of responsibility for action. Action on any global, long-ranged pollutant may be hindered by collective action problems, which, as described by Olson (1984), occur when costs of action are borne privately, but benefits are shared equally by the group—or in the case of mercury, regions beyond a country's borders. In these situations, large groups of rational actors may not act in the group interest. On the temporal side, while the bulk of current emissions are from Asia (UNEP, 2013a), countries like the US are chiefly responsible for historical contamination that continues to contribute to the mercury burden cycling through air, water, and land (Streets et al., 2011). In addition to these spatial and temporal concerns, do countries in the Global North have different responsibilities than those in the South because of their financial and technical ability?

Benedick (1998), who was the lead US negotiator during the talks leading up to the Montreal Protocol for ozone depleting substances, has argued that strong leadership by individual nations can play a crucial role in reaching consensus in the face of these challenges. In particular, Benedick (1998) suggests that because the US implemented strong, preemptive domestic policy on ozone-depleting chlorofluorocarbons (CFCs), it demonstrated that change was feasible to other parties, and legitimized the global policy efforts. In addition, preemptive regulatory action may have ensured that technical alternatives were available by the time the global agreement came into force (and therefore the possibility of technology transfer), easing the transition.

In recent years, the US has not taken such a leadership role in global environmental politics. This absence in the global environmental sphere may have just further emphasized the need for the US to "walk the walk" before encouraging others in the global community to regulate mercury. During the negotiations, the US (along with states in the European Union) pushed for a strong article on emissions to air, requiring mandatory controls on new and existing sources, and reductions on mercury emissions from key source categories (rather than the less stringent phrasing "controls") (IISD Reporting Services, 2013). Ultimately however, the final treaty text emphasized flexibility over stringency, and did not require mandatory emissions reductions, opting instead for a best available technique (BAT) approach that accommodates socioeconomic differences (Selin, 2013). Nevertheless, reaching even this agreement represented significant compromise by both sides of the debate—and as noted by IISD Reporting Services (2013), the groundwork has been laid for increases in stringency over time. Though it is difficult to speak of counterfactuals, if the US did not

have relatively strong regulations for emissions to air in place itself, it is possible that its influence would have been weaker.

Environmental justice and equity justifications for policy

There are many justifications for policy. It is often assumed that the utilitarian maximization of net benefits is the only one, however others may include equity, liberty, and security (Stone, 2002). Domestic mercury regulation may be motivated by a desire for environmental equity rather than economic efficiency ones. As Stone (2002) notes though, there is no objective definition for these policy "goals"—what efficiency and equity mean can be fluid, and defining these terms is a political act. In the context of environmental health, I use equity to refer to environmental justice concerns: ensuring that marginalized communities do not disproportionately experience the effects of environmental degradation (Nriagu et al., 2012).

The analysis indicates that estimated benefits are sensitive to dietary choices. The benefits of domestic mercury policy (in terms of exposure and welfare) are larger for those who eat primarily locally-caught, freshwater fish (for recreational, cultural, or socioeconomic reasons). These populations are particularly vulnerable to mercury exposure. Protecting these populations from mercury exposure (an equity argument) may be grounds for domestic policy—even if it may be a less economically efficient way of minimizing exposure reductions for the US population as a whole. In fact, language in Section 112 of the Clean Air Act, from which the EPA derives its authority to regulate mercury emissions, suggests that the Congressional intent behind legislation was to (equitably) protect public health, regardless of cost (O'Neill, 2009).

Regulating air toxics as a proxy for carbon

The US has struggled to develop a comprehensive federal strategy to deal with global climate change (Victor et al., 2005). Climate change has become a politically polarizing issue, and beyond funding research and establishing voluntary programs, little concerted and directed action has occurred (Lutsey & Sperling, 2008). In the face of political gridlock on the issue, the EPA has used its regulatory authority under the Clean Air Act—a statute that focuses on air quality—to regulate greenhouse gas (GHG) emissions from new power plants (Heller, 2013). However, more traditional regulation of air quality, and air toxics can also be seen as part of the "messy but useful" (Heller, 2013) approach to regulating these emissions. Regulating mercury emissions can encourage substitution from coal to other fuel sources, or improvements in plant efficiency (Pacyna et al., 2010b)—approaches that can have climate co-benefits (depending on the alternative fuel). Air toxics policies may therefore be a more politically palatable method to target sources of GHGs.

5.2 What kinds of policies are needed to protect human health?

Humans do not have direct control over many of the uncertainties that affect the benefits of a mercury emissions policy (once that policy is selected). In particular, uncertainties that are related to the atmospheric transport of mercury, its behaviour in ecosystems, and its physical effects on our health are, at present, beyond our control. However, many of these uncertainties also have the largest effect on estimated benefits—the dose-response relationships between mercury exposure and health impacts, and the lag times for ecosystem responses to emissions reductions. Ecosystem lag times in particular (which were only marginally less influential than dose-response parameterizations on estimated benefits) can be a concerning policy challenge. In the long-term, the benefits of an emissions reduction policy are projected to be large—and are increasing over time (Figure 4-3). However, what should be done in the interim, before exposure reductions are experienced in affected populations?

In Section 2.4, I described how policy interventions to reduce the negative impacts of mercury can be either preventative or adaptive. Preventative approaches, like emissions reductions, will have important effects in the long term—especially because mercury has a long legacy in the environment. Given the importance of ecosystem time lags however, adaptive approaches like fish consumption advisories may also be necessary, in tandem with emissions reductions. The uncertainty analysis showed that benefits are sensitive to dietary choices. My analysis focused on choices about sources of fish, however, choices about frequency of fish consumption are also likely to be important. There are numerous problems with fish consumption advisories, many of which were outlined in 2.4.3, however, they may be a necessary, if imperfect, temporary solution. Compensating individuals who must change their diet, or who disproportionately bear the costs of pollution, may help address some of the concerns. Research on other adaptive approaches such as direct ecosystem interventions that reduce methylmercury production (Selin, 2011), or therapeutic treatments that reduce or prevent the physical health effects of mercury exposure should also be encouraged.

5.3 How should mercury policies be assessed?

In the US, benefit-cost analysis (BCA) is a pervasive regulatory tool (Arrow et al., 2012). Though environmental and health policies are not required to pass a benefit-cost test (where benefits must be greater than costs), they must be reviewed under a BCA framework prior to finalization (Graham, 2007). At the EPA, this process is called a Regulatory Impact Assessment (RIA). Even if the results of an RIA do not officially dictate whether or not a policy is promulgated, they can still affect the speed at which it is implemented, and the probability of litigation. In the US, the speed and stringency of regulatory policy can be challenged by citizens (and their interest groups), industry, or subnational governments through the justice system—

this has been seen as a participatory strength of the US system (Petersen et al., 2006). These legal challenges have often occurred based on the contents of RIAs (Wagner, 2009), as the public legitimacy of policy decisions rests on the ability of policy makers to construct a plausible scientific and economic rationale for action within these documents (Jasanoff, 1987).[2] Keohane (2009) has also argued, however, that RIAs serve a critical democratic role as a source of information to subnational regulators and citizens. Information provided in the federal RIA for CAMR may have encouraged states to implement state enforced mercury policies in the time between when CAMR was vacated and MATS instated (see Section 2.4).[3]

Perhaps because BCA has taken on such an important role in the regulatory process, it has many critics and proponents. Proponents argue that BCA is a transparent analysis tool, in that it forces analysts to lay their assumptions bare (Viscusi et al., 2005). Assumptions about how to value a life, and where to draw boundaries, must be made explicitly to value benefits and costs. They also argue that systematically monetizing all costs and benefits allows for comparison on the same scale, and guards against cognitive errors or biases (Sunstein, 2000). However, critics like Ackerman & Heinzerling (2002) argue that in practice, the assumptions that underly BCA become buried. BCA is typically a highly expert driven process, and the public is seldom exposed to the assumptions that underlie the numerical results. Public discussion tends to focus on the numbers themselves, and not the assumptions that are built into them. Amongst other arguments, Ackerman & Heinzerling (2002) also argue that BCA inherently endorses a utilitarian framework that does not acknowledge *who* bears the costs, or experiences the benefits of policy.

Do the results from the case study in this thesis have any implications for how BCA might best be used (or not used) to assess mercury policies? I argue that if BCA must be used, then: uncertain health effects should be included quantitatively to the extent possible; estimated benefits of policy in terms of reduced health impacts should be clearly differentiated from welfare benefits to highlight the effects of discounting and valuation; and, following Keohane (2009), a movement should be made towards thinking in terms of orders of magnitude, rather than fixating on precise numbers.

5.3.1 Quantitatively including uncertain health effects

The case study results suggest that 85-90% of the monetized benefits of reduced mercury exposure may be attributable to reductions in risk of AMIs, when they are included in calculations. In addition, uncertainty in the range of uncertainties in dose-response parameterization led to the largest range of estimated welfare benefits—and this effect was largely driven by uncertainty in cardiovascular effects. In general, cardio-pulmonary effects can have a large effect on benefits accounting, and so their

[2] Jasanoff (1987) would argue that the policy-making process involves first deconstructing scientific (and I would add, economic) knowledge claims, to unearth areas of uncertainty and bias. Decision makers must then reconstruct a plausible rationale—one that has been hardened against such deconstructive attacks.

[3] At least 20 states did so (Milford & Pienciak, 2009).

potential impact should be acknowledged, even when uncertain. Where possible, they should be acknowledged quantitatively. While the EPA does make an effort to conduct a qualitative analysis of uncertain health effects (US EPA, 2011c,d), these nuances often fail to make it into media coverage of policy developments. There are many quantitative methods to both include these effects and highlight their uncertainty—in this thesis, I have used scenario and sensitivity analysis. An alternative method would be to use probability distributions for uncertain parameters and conduct a probabilistic, rather than deterministic benefits assessment (Rice et al., 2010).

5.3.2 Decoupling health impact benefits and welfare benefits

A distinction can be made between the physical impacts of policy (reduced exposure, reduced risk of morbidity or mortality) and the monetized benefits of these impacts (Keohane, 2009). As the uncertainty analysis demonstrated, how a physical impact is monetized—particularly the choice of social discount rate and valuation method—can obscure and interact with effects earlier in the emissions-to-impacts chain. For instance, when are estimated benefits low because projected benefits for health are marginal, and when are they low because the majority of benefits are projected to be experienced in the future? This concern is particularly true for many toxic pollutants, like mercury, that are long-lived in the environment (leading to lag times in exposure responses to policy), and that have health impacts with long-lasting, and often chronic and cumulative effects on the economy. When benefits estimates are presented, it may therefore be useful to both express them in terms of health impacts and monetized values, to better highlight the effects of assumptions about discounting and valuation.

5.3.3 Moving towards order of magnitude estimates

As I mentioned in the opening of this section, the uncertainty ranges for benefits in this work and those in the literature were more or less overlapping—despite differences in assumptions and valuation techniques. More than anything, this highlights the magnitude of uncertainties associated with benefits accounting for mercury. Given these uncertainties, fixating on precise estimates of benefits (and often costs) may be dishonest and it may be more worthwhile and transparent to think in orders of magnitude (Keohane, 2009). This approach may be problematic if the purpose of BCA is primarily to defend against litigation (Wagner, 2009). However, if, as Keohane (2009) argues, the role of benefits assessment is to provide information in a democracy, or even if its role is the positive (which I use, following Hammitt (2012) to mean in contrast to normative) identification of situations where net social benefits outweigh costs, order of magnitude estimates may be equally useful.

Chapter 6

Conclusions

Mercury is an issue of concern because its toxicity endangers human and ecosystem health. Especially potent in the form of methyl mercury (MeHg), exposure is known to lead to adverse neurological effects, and potentially cardiovascular and immunotoxic ones (Mergler et al., 2007). Although mercury is emitted by natural sources, it is estimated that human activity has increased its mobilization by three to five times since the pre-industrial period, leading to greater exposure (Selin, 2009). These attributes have made mercury the target of regulation—both domestically, through the Clean Air Act (O'Neill, 2009), and internationally, in the form of a legally binding global environmental treaty (UNEP, 2013b).

Interest in quantitatively assessing the benefits of mercury reduction policies is motivated by the prominence of benefit cost analysis (BCA) in the US regulatory process (Arrow et al., 2012). Quantifying the benefits of protecting environmental and human health is a difficult task—it requires many assumptions, in particular because there is no direct market for environmental quality (Hanemann, 1994). Moreover, there is no consensus that quantitative BCA is an useful, or ethically appropriate guiding tool in environment and health policy-making (Ackerman & Heinzerling, 2002). Nevertheless, because quantitative benefits analysis is so firmly entrenched in the policy-making process (Graham, 2007), in the near term, attention should be paid to the methods by which these assessments are conducted—and how they can be improved in many dimensions.

Model development

In this thesis, I have argued that many existing benefits assessments of mercury policies lack realism and completeness in how they have represented mercury's path from emissions to wider socioeconomic impacts. In particular, because many do not explicitly model mercury's full path, they do not capture the full spatial and temporal dimensions of the mercury problem. In addition, many do not include uncertain health effects like increased risk of cardiovascular disease, which while uncertain, may be highly policy-relevant. In response to these gaps, I developed an integrated assessment framework, which links a pre-existing chemical transport modelling of mercury in the

environment (GEOS-Chem) to a pre-existing general equilibrium model of the US economy (USREP), to evaluate the health and economic impacts of mercury emissions reductions. I incorporated insights from the rich body of scientific and economic literature to model the links between environmental concentrations of mercury to human mercury exposure in the US and its resultant health effects. I modified the economic model of the US to account for mercury-related morbidity (lost IQ, and non-fatal heart attacks) and mortality (fatal heart attacks) effects. Lost IQ and non-fatal heart attacks are modelled as leading to lower productivity in the economy, by diverting existing resources (services, labour, and leisure) towards a pollution-health sector. Fatal heart attacks are modelled as shocks to the available pool of labour and leisure.

Case study: Mercury and Air Toxics Standards

I evaluated this new method by conducting a case study of the Mercury and Air Toxics Standards (MATS), the most recent US regulations targeting mercury emissions from coal-fired power plants (CFPPs). I assessed the benefits of this policy out to 2050 compared to two counterfactual scenarios: one that includes stringent air quality policies, and a no policy scenario. I projected that MATS will lead to a 29% reduction of US anthropogenic emissions when compared to the air quality scenario, and 70% when compared to the no policy scenario, by 2050. Changes in emissions reduced modelled exposure by 0.7 and 9.7% on average, compared to the air quality and no policy scenarios; however there was strong regional variation in exposure reductions due to patterns of deposition, and dietary patterns (the geographic source and species of fish residents of a given region consume). For the general US population, the reductions in emissions due to MATS did not directly translate to reductions in exposure because the average consumer eats primarily marine and estaurine fish, while MATS leads to the greatest deposition reductions over the contiguous US. However, exposure reductions were greater in regions where a larger share of the population eats locally caught, freshwater fish (subsistence fishers, recreational anglers, certain indigenous peoples).

I found that exposure reductions due to MATS lead to a simulated yearly benefit to the US economy of 13 million 2005 USD, and 414 million 2005 USD under the two scenarios, respectively. The yearly benefits increase over time however, and range from 2-40 million, and 90-600 million. The increasing trend was due to: the time lag between when emissions reductions occur and ecosystems respond fully to these reductions, economic impacts of health effects like lost IQ that come "on-line" and accrue over time, increasing population size, and compounding economic growth effects from reinvesting past welfare gains back into the economy. The share of these benefits attributable to cardiovascular effects ranged from 85-90% depending on the year.

Uncertainty analysis

To better understand the factors that control the value of estimated benefits, I conducted an uncertainty analysis. I explored the impact of different assumptions about key uncertainties in the emissions-to-impacts path, and found that uncertainty in the appropriate social discount rate, the quantitative relationships between exposure to mercury and health impacts, and ecosystem time lags had the largest effects on estimated benefits. Uncertainties related to future world emissions trajectories and dietary choices had smaller but appreciable effects—but are important because they are directly subjected to human control. Because mercury's effects operate over long time scales, choice of social discount rate interacts strongly with uncertainties further up in the chain, and can mask their effects. The uncertainty ranges of the benefits estimated in this work overlap with those from existing studies that consider similar emissions or exposure reduction scenarios—despite differences in valuation approach. This finding highlighted how poorly constrained many of the key uncertainties in the emissions-to-impacts path are.

Policy implications

Does this new assessment of MATS provide any additional policy-relevant insights that existing studies do not? I argued that the comparative advantage of representing the full emissions-to-impact chain, and including uncertain cardiovascular effects, and mercury's full spatial and temporal dimensions was not in reaching a more accurate benefits estimate—the uncertainties involved in assessment may be too large for this—but in developing a better understanding of what controls benefits estimates. I described how the case study results could be used to inform the following questions:

1. *Where should a mercury policy be implemented?*

   For the average consumer, emissions decreases elsewhere in the world (particularly in East Asia) would have a larger impact on exposure than domestic mercury policy. This finding suggests that the US has an interest is supporting global action on mercury—and that this might be a more economically efficient strategy of reducing average US exposure. However, there are still several rationales for implementing a domestic policy: it improves the legitimacy of US demands for global emissions reductions in international negotiation forums, domestic regulations protect vulnerable, highly exposed populations that depend on locally caught freshwater fish, and regulating mercury emissions from CFPPs is an indirect form of climate policy.

2. *What kinds of polices are needed to protect human health?*

   Because humans do not have direct control over the potentially long time lag between emissions reductions and response in fish tissue concentrations—and because these time lags were found to be a large influence on estimated benefits of policy—attempts should be made to couple preventative policy approaches

(emissions reductions) with more adaptive approaches, that address communities that will continue to be negatively affected by mercury in the interim. One adaptive approach is (compensated) changes to dietary patterns (source of fish, frequency of fish meals).

3. *How should mercury policies be assessed?*

   If BCA is going to be used to evaluate mercury-related policies, then the case study results suggest that it would be valuable to do the following:

   - Include, quantitatively, uncertain health effects. Methods that take into account uncertainty, like probabilistic Monte Carlo approaches, can be used.
   - Decouple monetized benefits estimates from health impact benefits to highlight the effect of assumptions about discounting and valuation.
   - Move towards order of magnitude judgments because large uncertainty ranges prevent precise estimates.

Future work

A few areas were identified as opportunities for future work:

- *Further spatial disaggregation through nested-grid modelling over the US*: Finer spatial resolution modelling over the US will make it easier to separate out benefits of policy for vulnerable populations that eat primarily locally-caught freshwater fish, which are partially obscured through regional averaging.

- *Improved integrated assessment coupling*: Closing the link between economic effects of mercury and emissions of mercury is a logical next step for the integrated assessment framework. This would allow for endogenous and internally consistent calculations of mercury emissions (which scale with energy generation, under policy) rather than using external data inputs. In addition, given the importance of ecosystem time lags, coupling the integrated assessment framework to a full ecosystem model could be useful. Ecosystem models better capture conversion from inorganic to methyl mercury, and trophic transfer.

- *Effects of future climate*: Will there be a climate penalty that will reduce any exposure benefits from emissions decreases? The answer to this question should be taken into account when evaluating the efficacy of policy at reducing environmental concentrations of mercury. Effects of future climate can be investigated by coupling the chemical transport model (GEOS-Chem) to a general circulation model with simulated future meteorology.

- *Comprehensive uncertainty management*: I identified key uncertainties at each point in the emissions-to-impacts chain. In the analysis in this thesis, I used a scenario-sensitivity approach to explore these uncertainties. In the future, some

probabilistic analysis can be conducted with uncertainties that have been parameterized as uncertainty distributions (dose-response parameterizations, for instance). However, there are also several uncertainties that cannot be easily quantified as a probability distribution—for instance, uncertainties related to model form (like the shape of the dose-response curve), and model completeness (whether a chemical reaction occurs in the environment). Considering how these differenc kinds of uncertainties can be presented to policy-makers in a comprehensive way may also be a topic for future investigation (Rotmans & van Asselt, 2001).

Appendix A

Additional figures and tables

Table A.1: Correspondence between states and model regions

State	USREP Classification	Mahaffey Classification
Alaska	Alaska	Pacific Coast (subset of West)
California	California	West
Florida	Florida	South
Montana	Mountain	West
Idaho	Mountain	West
Wyoming	Mountain	West
Colorado	Mountain	West
New Mexico	Mountain	West
Utah	Mountain	West
Arizona	Mountain	West
Nevada	Mountain	West
Maine	New England	Northeast
Vermont	New England	Northeast
New Hampshire	New England	Northeast
Massachusetts	New England	Northeast
Rhode Island	New England	Northeast
Conneticut	New England	Northeast
North Dakota	North Central	Midwest
Minnesota	North Central	Midwest
South Dakota	North Central	Midwest
Nebraska	North Central	Midwest
Kansas	North Central	Midwest
Missouri	North Central	Midwest
Iowa	North Central	Midwest
Ohio	North East	Midwest
Michigan	North East	Midwest
Indiana	North East	Midwest

Continued on next page

Table A.1 – *continued from previous page*

State	**USREP Classification**	**Mahaffey Classification**
Illinois	North East	Midwest
Wisconsin	North East	Midwest
New Jersey	North East	Northeast
Pennsylvania	North East	Northeast
New York	New York	Northeast
Delaware	North East	South
Maryland	North East	South
West Virginia	North East	South
Washington	Pacific	West
Oregon	Pacific	West
Hawaii	Pacific	Pacific Coast (subset of West)
Oklahoma	South Central	South
Arkansas	South Central	South
Louisiana	South Central	South
Virginia	South East	South
Kentucky	South East	South
Tennessee	South East	South
Mississippi	South East	South
Alabama	South East	South
Georgia	South East	South
South Carolina	South East	South
North Carolina	South East	South
Texas	Texas	South

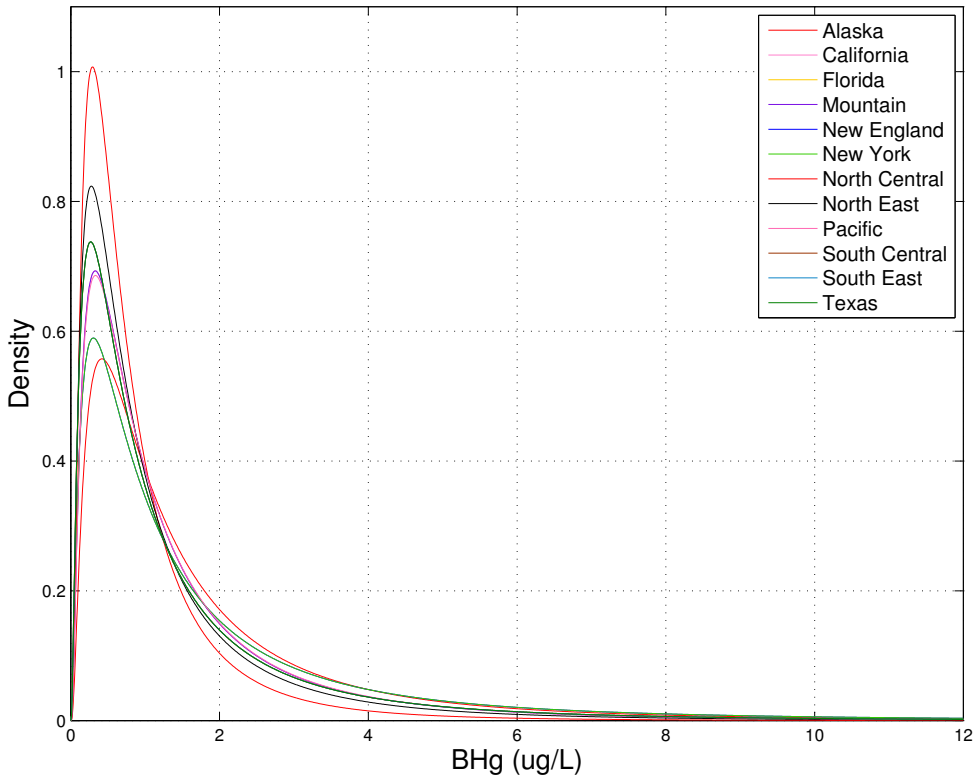


Figure A-1: Regional blood mercury distributions for women aged 16-49 in μg/L

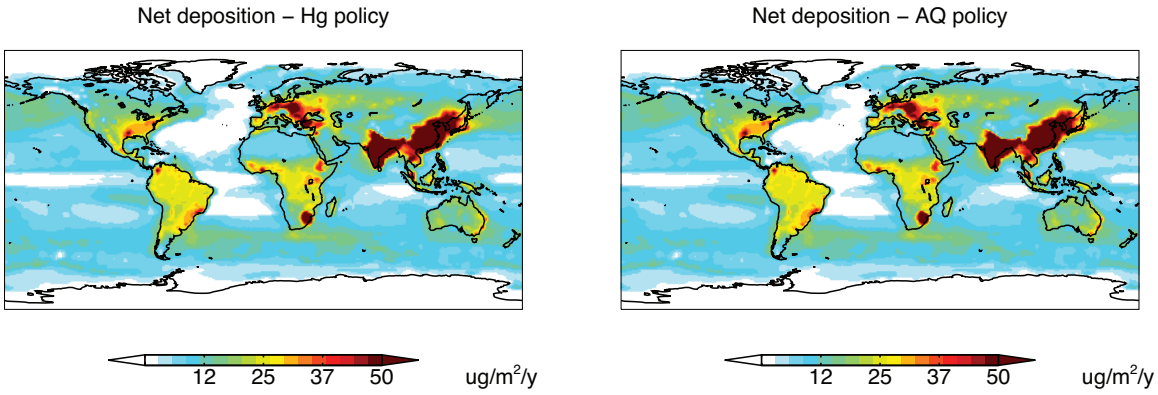


Figure A-2: Projected deposition under Hg policy and AQ policy, in 2050.

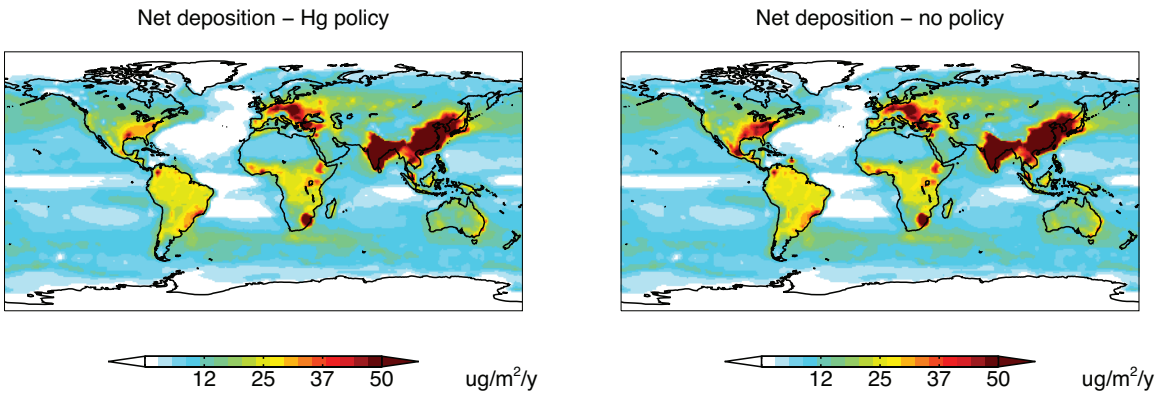


Figure A-3: Projected deposition under Hg policy and NP policy, in 2050.

Table A.2: Comparison of valuation studies

Analysis	**Degree of mercury reduction**	**Considered health endpoints**	**Annual Benefit**	**Range**
Rice et al. (2010)	10% exposure reduction	IQ decrements, fatal AMIs	860 M USD	50 M USD - 3.5 B USD
Trasande et al. (2006)	elimination of all anthropogenic emissions	IQ decrements	8.7 B 2000 USD	700 M - 13.9 B 2000 USD
Trasande et al. (2006)	elimination of emissions from US coal-fired power plants	IQ decrements	1.3 B 2000 USD	51 M - 2 B 2000 USD
Griffiths et al. (2007)	70% emissions reduction from US coal-fired power plants	IQ decrements in recreational fishers		10 M - 210 M 2000 USD
Griffiths et al. (2007)	elimination of US anthropogenic emissions power plants (16% exposure reduction)	IQ decrements in recreational fishers	25 M 2000 USD	
US EPA (2011d)	Mercury and Air Toxics Standards (estimated 90% emissions reduction from US coal-fired power plants)	IQ decrements in recreational fishers		4.2 M - 6.2 M 2007 USD
Present work	9.7% exposure (Hg vs. NP)	IQ decrements, fatal AMIs, non-fatal AMIs	414 M USD	90 - 600 M USD

Bibliography

Abt Associates (2010). BenMAP Environmental Benefits Mapping and Analysis Program: User's Manual Appendices. Tech. rep., Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC.

Ackerman, F., & Heinzerling, L. (2002). Pricing the Priceless: Cost-Benefit Analysis of Environmental Protection. *University of Pennsylvania Law Review*, *150*(5), 1553.

Ahlqwist, M., Bengtsson, C., Lapidus, L., Gergdahl, I. a., & Schütz, a. (1999). Serum mercury concentration in relation to survival, symptoms, and diseases: results from the prospective population study of women in Gothenburg, Sweden. *Acta odontologica Scandinavica*, *57*(3), 168–74.

Allen, B. C., Hack, C. E., & Clewell, H. J. (2007). Use of Markov Chain Monte Carlo analysis with a physiologically-based pharmacokinetic model of methylmercury to estimate exposures in US women of childbearing age. *Risk analysis : an official publication of the Society for Risk Analysis*, *27*(4), 947–59.

AMAP (2011). Arctic Pollution 2011. Tech. rep., Arctic Monitoring and Assessment Programme, Oslo.

Amirbahman, A., & Fernandez, I. J. (2012). The Role of Soils in Storage and Cycling of Mercury. In M. S. Bank (Ed.) *Mercury in the Environment: Pattern and Process*, chap. 8, (pp. 97–118). Berkeley: University of California Press.

Amos, H. M., Jacob, D. J., Holmes, C. D., Fisher, J. a., Wang, Q., Yantosca, R. M., Corbitt, E. S., Galarneau, E., Rutter, a. P., Gustin, M. S., Steffen, a., Schauer, J. J., Graydon, J. a., Louis, V. L. S., Talbot, R. W., Edgerton, E. S., Zhang, Y., & Sunderland, E. M. (2012). Gas-particle partitioning of atmospheric Hg(II) and its effect on global mercury deposition. *Atmospheric Chemistry and Physics*, *12*(1), 591–603.

Amos, H. M., Jacob, D. J., Streets, D. G., & Sunderland, E. M. (2013). Legacy impacts of all-time anthropogenic emissions on the global mercury cycle. *Global Biogeochemical Cycles*, *27*, 1–12.

Amyot, M., Southworth, G., Lindberg, S., Hintelmann, H., Lalonde, J., Ogrinc, N., a.J Poulain, & Sandilands, K. (2004). Formation and evasion of dissolved gaseous

mercury in large enclosures amended with 200HgCl2. *Atmospheric Environment*, *38*(26), 4279–4289.

Arrow, K. J., Cropper, M. L., Eags, G. C., Hahn, R. W., Lave, L. B., Noll, R. G., Portney, P. R., Russell, M., Schmalensee, R., Smith, V. K., & Stavins, R. N. (2012). Is There a Role for Benefit-Cost Analysis in Environmental, Health, and Safety Regulation? In R. N. Stavins (Ed.) *Economics of the Environment*, (pp. 219–224). New York: W. W. Norton & Company, 6th ed.

ATSDR (1999). TOXICOLOGICAL PROFILE FOR MERCURY. Tech. Rep. March, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

Axelrad, D. a., Bellinger, D. C., Ryan, L. M., & Woodruff, T. J. (2007). Dose-response relationship of prenatal mercury exposure and IQ: an integrative analysis of epidemiologic data. *Environmental health perspectives*, *115*(4), 609–15.

Bakir, F., Damluji, S. F., Amin-Zaki, L., Murtadha, M., Khalidi, A., Al-Rawi, N. Y., Tikriti, S., Dahahir, H. I., Clarkson, T. W., Smith, J. C., & Doherty, R. a. (1973). Methylmercury poisoning in Iraq. *Science*, *181*(4096), 230–41.

Bandara, J. S. (1991). Computable General Equilibrium Models for Development Policy Analysis in LDCs. *Journal of Economic Surveys*, *5*(1), 3–69.

Benedick, R. E. (1998). *Ozone Diplomacy: New Directions in Safeguarding the Planet*. Cambridge, MA: Harvard University Press, enlarged ed.

Benoit, J., Gilmour, C. C., Heyes, A., Mason, R. P., & Miller, C. L. (2003). Geochemical and Biological Controls over Methylmercury Production and Degradation in Aquatic Ecosystems. In Y. Chai, & O. C. Braids (Eds.) *Biogeochemistry of environmentally important trace elements. ACS Symposium Series 835*, chap. 19, (pp. 262–297). Washington, D.C.: American Chemical Society.

Beusse, R. (2006). Monitoring Needed to Assess Impact of EPA's Clean Air Mercury Rule on Potential Hotspots. Tech. rep., Environmental Protection Agency, Office of Inspector General, Research Triangle Park, NC.

Bhattacharyya, S. C. (1996). Applied general equilibrium models for energy studies: a survey. *Energy Economics*, *18*(3), 145–164.

Biermann, F., & Pattberg, P. (2008). Global Environmental Governance: Taking Stock, Moving Forward. *Annual Review of Environment and Resources*, *33*(1), 277–294.

Biester, H., Bindler, R., Martinez-Cortizas, A., & Engstrom, D. R. (2007). Critical Review Modeling the Past Atmospheric Deposition of Mercury Using Natural Archives. *Environmental science & technology*, *41*(14), 4851–4860.

Brander, K. M. (2007). Global fish production and climate change. *Proceedings of the National Academy of Sciences of the United States of America*, *104*(50), 19709–14.

Budtz-Jørgensen, E., Grandjean, P., Jø rgensen, P. J., Weihe, P., & Keiding, N. (2004). Association between mercury concentrations in blood and hair in methylmercury-exposed subjects at different ages. *Environmental research*, *95*(3), 385–93.

Budtz-Jørgensen, E., Grandjean, P., Keiding, N., White, R. F., & Weihe, P. (2000). Benchmark dose calculations of methylmercury-associated neurobehavioural deficits. *Toxicology letters*, *112-113*, 193–9.

Budtz-Jørgensen, E., Grandjean, P., & Weihe, P. (2007). Separation of risks and benefits of seafood intake. *Environmental health perspectives*, *115*(3), 323–7.

Budtz-Jørgensen, E., Grandjean, P., & White, R. F. (1999). Methylmercury Neurotoxicity Independent of PCB Exposure. *Environmental health perspectives*, *107*(5), A236–A237.

Budtz-Jørgensen, E., Keiding, N., Grandjean, P., & Weihe, P. (2002). Estimation of health effects of prenatal methylmercury exposure using structural equation models. *Environmental Health*, *1*(1), 1–22.

Cameron, T. a. (2010). Euthanizing the Value of a Statistical Life. *Review of Environmental Economics and Policy*, *4*(2), 161–178.

Carrington, C. D., & Bolger, M. P. (2002). An exposure assessment for methylmercury from seafood for consumers in the United States. *Risk analysis : an official publication of the Society for Risk Analysis*, *22*(4), 689–99.

Chan, C., Heinbokel, J. F., Myers, J. a., & Jacobs, R. R. (2012). A dynamic model using monitoring data and watershed characteristics to project fish tissue mercury concentrations in stream systems. *Integrated environmental assessment and management*, (pp. 1–14).

Chayes, A. H., Chayes, A., & Mitchell, R. B. (1995). Active Compliance Management in Environmental Treaties. In W. Lang (Ed.) *Sustainable Development and International Law*, chap. 6, (pp. 75–89). London: Graham and Trotman Ltd.

Chen, C., Driscoll, C. T., Lambert, K. F., Mason, R. P., Rardin, L. R., Schmitt, C. V., Serrell, N. S., & Sunderland, E. M. (2012a). *Sources to Seafood: Mercury Pollution in the Marine Environment.*. Hanover, NH: Toxic Metals Superfund Research Program, Dartmouth College.

Chen, C. Y., Driscoll, C. T., & Kamman, N. C. (2012b). Mercury Hotspots in Freshwater Ecosystems: Drivers, Processes, and Patterns. In M. S. Bank (Ed.) *Mercury in the Environment: Pattern and Process*, 1, chap. 9, (pp. 143–166). Berkeley: University of California Press.

Chen, C. Y., Serrell, N., Evers, D. C., Fleishman, B. J., Lambert, K. F., Weiss, J., Mason, R. P., & Bank, M. S. (2008). Meeting report: Methylmercury in marine ecosystems–from sources to seafood consumers. *Environmental health perspectives*, *116*(12), 1706–12.

Clarkson, T. W. (2002). The Three Modern Faces of Mercury. *Environmental Health Perspectives*, *110*(Supplement 1), 11–23.

Coase, R. H. (1960). The Problem of Social Cost. *The Journal of Law & Economics*, *3*, 1–44.

Cohen, J. T., Bellinger, D. C., & Shaywitz, B. a. (2005). A quantitative analysis of prenatal methyl mercury exposure and cognitive development. *American journal of preventive medicine*, *29*(4), 353–65.

Corbitt, E. S., Jacob, D. J., Holmes, C. D., Streets, D. G., & Sunderland, E. M. (2011). Global source-receptor relationships for mercury deposition under present-day and 2050 emissions scenarios. *Environmental science & technology*, *45*(24), 10477–84.

Cropper, M. L., & Krupnick, A. J. (1990). The social costs of chronic heart and lung disease.

Crump, K. S., Kjellström, T., Shipp, a. M., Silvers, a., & Stewart, a. (1998). Influence of prenatal mercury exposure upon scholastic and psychological test performance: benchmark analysis of a New Zealand cohort. *Risk analysis : an official publication of the Society for Risk Analysis*, *18*(6), 701–13.

Davidson, P. W., Myers, G. J., Cox, C., Axtell, C., Shamlaye, C., Sloane-reeves, J., Cernichiari, E., Needham, L., Choi, A., Wang, Y., Berlin, M., & Clarkson, T. W. (1998). Effects of Prenatal and Postnatal Methylmercury Exposure From Fish Consumption on Neurodevelopment: Outcomes at 66 Months of Age in the Seychelles Child Development Study. *Journal of the American Medical Association*, *280*(8).

Dellinger, J., Dellinger, M., & Yauck, J. S. (2012). Mercury Exposure in Vulnerable Populations: Guidelines for Fish Consumption. In M. S. Bank (Ed.) *Mercury in the Environment: Pattern and Process*, chap. 14, (pp. 289–300). Berkeley: University of California Press.

Depew, D. C., Basu, N., Burgess, N. M., Campbell, L. M., Devlin, E. W., Drevnick, P. E., Hammerschmidt, C. R., Murphy, C. a., Sandheinrich, M. B., & Wiener, J. G. (2012). Toxicity of dietary methylmercury to fish: derivation of ecologically meaningful threshold concentrations. *Environmental toxicology and chemistry*, *31*(7), 1536–47.

Dietz, R., Sonne, C., Basu, N., Braune, B., O'Hara, T., Letcher, R. J., Scheuhammer, T., Andersen, M., Andreasen, C., Andriashek, D., Asmund, G., Aubail, A., Baagø e, H., Born, E. W., Chan, H. M., Derocher, A. E., Grandjean, P., Knott, K., Kirkegaard, M., Krey, A., Lunn, N., Messier, F., Obbard, M., Olsen, M. T., Ostertag, S., Peacock, E., Renzoni, A., Rigét, F. F., Skaare, J. U., Stern, G., Stirling,

I., Taylor, M., Wiig, O. y., Wilson, S., & Aars, J. (2013). What are the toxicological effects of mercury in Arctic biota? *The Science of the total environment*, *443*, 775–90.

Drevnick, P. E., Engstrom, D. R., Driscoll, C. T., Swain, E. B., Balogh, S. J., Kamman, N. C., Long, D. T., Muir, D. G. C., Parsons, M. J., Rolfhus, K. R., & Rossmann, R. (2012). Spatial and temporal patterns of mercury accumulation in lacustrine sediments across the Laurentian Great Lakes region. *Environmental pollution*, *161*, 252–60.

Driscoll, C. T., Han, Y.-J., Chen, C. Y., Evers, D. C., Lambert, K. F., Holsen, T. M., Kamman, N. C., & Munson, R. K. (2007). Mercury Contamination in Forest and Freshwater Ecosystems in the Northeastern United States. *BioScience*, *57*(1), 17.

Edgerton, E. S., Hartsell, B. E., & Jansen, J. J. (2006). Mercury speciation in coal-fired power plant plumes observed at three surface sites in the southeastern U.S. *Environmental science & technology*, *40*(15), 4563–70.

Eisenstein, E. L., Shaw, L. K., Anstrom, K. J., Nelson, C. L., Hasselblad, V., & Mark, D. B. (2001). Assessing the Clinical and Economic Burden of Coronary Artery Disease: 1986-1998. *Medical Care*, *39*(8), 824–835.

Evers, D. C., Han, Y.-j., Driscoll, C. T., Kamman, N. C., Goodale, W., Lambert, K. F., Holsen, T. M., Chen, C. Y., Clair, T. A., & Butler, T. (2007). Biological Mercury Hotspots in the Northeastern United States and Southeastern Canada. *BioScience*, *57*(1), 29–43.

Evers, D. C., Savoy, L. J., DeSorbo, C. R., Yates, D. E., Hanson, W., Taylor, K. M., Siegel, L. S., Cooley, J. H., Bank, M. S., Major, A., Munney, K., Mower, B. F., Vogel, H. S., Schoch, N., Pokras, M., Goodale, M. W., & Fair, J. (2008). Adverse effects from environmental mercury loads on breeding common loons. *Ecotoxicology (London, England)*, *17*(2), 69–81.

Evers, D. C., Wiener, J. G., Basu, N., Bodaly, R. a., Morrison, H. a., & Williams, K. a. (2011a). Mercury in the Great Lakes region: bioaccumulation, spatiotemporal patterns, ecological risks, and policy. *Ecotoxicology (London, England)*, *20*(7), 1487–99.

Evers, D. C., Wiener, J. G., Driscott, C. T., Gay, D. A., Basu, N., Monson, B. A., Lambert, K. F., Morrison, H. A., Morgan, J. T., Williams, K. A., & Soehl, A. G. (2011b). Great Lakes Mercury Connections: The Extent and Effects of Mercury Pollution in the Great Lakes Region. Tech. rep., Biodiversity Research Institute - BRI 2011-18, Gorhan, ME.

Ferrari, C., Gauchard, P., Aspmo, K., Dommergue, a., Magand, O., Bahlmann, E., Nagorski, S., Temme, C., Ebinghaus, R., & Steffen, a. (2005). Snow-to-air exchanges of mercury in an Arctic seasonal snow pack in Ny-Ålesund, Svalbard. *Atmospheric Environment*, *39*(39), 7633–7645.

Friedman, C., Selin, N. E., & Zhang, Y. (2013). Influence of future emissions and climate on atmospheric PAH transport. In *The 6th International GEOS-Chem Meeting*. Cambridge, MA.

Goulder, L. H., & Parry, I. W. H. (2008). Instrument Choice in Environmental Policy. *Review of Environmental Economics and Policy*, *2*, 152–174.

Graham, J. D. (2007). The Evolving Regulatory Role of the U.S. Office of Management and Budget. *Review of Environmental Economics and Policy*, *1*(2), 171–191.

Grandjean, P., & Budtz-Jørgensen, E. (2007). Total Imprecision of Exposure Biomarkers: Implications for Calculating Exposure Limits. *American Journal of Industrial Medicine*, *50*, 712–719.

Grandjean, P., Weihe, P., White, R. F., Debes, F., Araki, S., Yokoyama, K., Murata, K., Sø rensen, N., Dahl, R., & Jø rgensen, P. J. (1997). Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicology and teratology*, *19*(6), 417–28.

Griffiths, C., McGartland, A., & Miller, M. (2007). A comparison of the monetized impact of IQ decrements from mercury emissions. *Environmental health perspectives*, *115*(6), 841–7.

Grosse, S. D. (2007). How much does IQ raise earnings? *Association of Environmental and Resource Economists Newsletter*, *27*(2), 17–21.

Guallar, E., Sanz-Gallardo, M. I., van't Veer, P., Bode, P., Aro, A., Gómez-Aracena, J., Kark, J. D., Riemersma, R. a., Martín-Moreno, J. M., & Kok, F. J. (2002). Mercury, fish oils, and the risk of myocardial infarction. *The New England journal of medicine*, *347*(22), 1747–54.

Häggqvist, B., Havarinasab, S., Björn, E., & Hultman, P. (2005). The immunosuppressive effect of methylmercury does not preclude development of autoimmunity in genetically susceptible mice. *Toxicology*, *208*(1), 149–64.

Hammitt, J. K. (2012). Positive v. Normtive Justifications for Benefit-Cost Analysis. Tech. Rep. 13, Harvard Kennedy School Regulatory Policy Program Working Papers, Boston, MA.

Hanemann, W. M. (1994). Valuing the Environment through Contingent Valuation. *Journal of Economic Perspectives*, *8*, 19–43.

Harada, M. (1995). Minamata disease: methylmercury poisoning in Japan caused by environmental pollution. *Critical reviews in toxicology*, *25*(1), 1–24.

Heckman, J. J., Stixrud, J., & Urzua, S. (2006). The Effects of Cognitive and Noncognitive Abilities on Labor Market Outcomes and Social Behavior. *Journal of Labor Economics*, *24*(3).

Heller, T. C. (2013). The policy climate. Tech. rep., Climate Policy Initiative.

Hintelmann, H., Harris, R., Heyes, A., Hurley, J. P., Kelly, C. a., Krabbenhoft, D. P., Lindberg, S., Rudd, J. W. M., Scott, K. J., & St.Louis, V. L. (2002). Reactivity and Mobility of New and Old Mercury Deposition in a Boreal Forest Ecosystem during the First Year of the METAALICUS Study. *Environmental Science & Technology*, *36*(23), 5034–5040.

Holmes, C. D., Jacob, D. J., Corbitt, E. S., Mao, J., Yang, X., Talbot, R., & Slemr, F. (2010). Global atmospheric model for mercury including oxidation by bromine atoms. *Atmospheric Chemistry and Physics*, *10*(24), 12037–12057.

Houyoux, M., & Strum, M. (2011). Memorandum: Emissions Overview: Hazardous Air Pollutants in Support of the Final Mercury and Air Toxics Standard. Tech. rep., US EPA: Air Quality Assessment Division, Research Triangle Park, NC.

Hynes, A. J., Donohoue, D. L., Goodsite, M. E., & Hedgecock, I. M. (2009). Our Current Understanding of Major Chemical and Physical Processes Affecting Mercury Dynamics in the Atmosphere and aat the Air-Water/Terrestrial Interfaces. In R. Mason, & N. Pirrone (Eds.) *Mercury Fate and Transport in the Global Atmosphere*, chap. 14, (pp. 427–457). Boston, MA: Springer US. URL `http://www.springerlink.com/index/10.1007/978-0-387-93958-2`

IISD Reporting Services (2013). Summary of the fifth session of the intergovernmental negotiating committee to prepare a global legally binding instrument on mercury: 13-19 January 2013. *Earth Negotiations Bulletin*, *28*(22), 1–26.

Jardine, L. B., Burt, M. D. B., Arp, P. a., & Diamond, a. W. (2009). Mercury comparisons between farmed and wild Atlantic salmon (Salmo salar L.) and Atlantic cod (Gadus morhua L.). *Aquaculture Research*, *40*(10), 1148–1159.

Jasanoff, S. S. (1987). Contested Boundaries in Policy-Relevant Science. *Social Studies of Science*, *17*(2), 195–230.

Karagas, M. R., Choi, A. L., Oken, E., Horvat, M., Schoeny, R., & Kamai, E. (2012). Evidence on the Human Health Effects of Low-Level Methylmercury Exposure. *Environmental Health Perspectives*, *120*(6), 799–806.

Keohane, N. A. (2009). The Technocratic and Democratic Functions of the CAIR Regulatory Analysis. In W. Harrington, L. Heinzerling, & R. D. Morgenstern (Eds.) *Reforming Regulatory Impact Analysis*, chap. 3, (pp. 33–55). Washington, DC: Resources for the Future.

Knightes, C. D., Sunderland, E. M., Barber, M. C., Johnston, J. M., & Ambrose, R. B. J. (2009). APPLICATION OF ECOSYSTEM-SCALE FATE AND BIOACCUMULATION MODELS TO PREDICT FISH MERCURY RESPONSE TIMES TO CHANGES IN ATMOSPHERIC DEPOSITION. *Environmental Toxicology*, *28*(4), 881–893.

Kuhnlein, H. V., & Receveur, O. (1996). Dietary change and traditional food systems of indigenous peoples. *Annual review of nutrition*, *16*, 417–42.

Lalonde, J. D., Amyot, M., Kraepiel, a. M., & Morel, F. M. (2001). Photooxidation of Hg(0) in artificial and natural waters. *Environmental science & technology*, *35*(7), 1367–72.

Lambert, K. F., Evers, D. C., Warner, K. a., King, S. L., & Selin, N. E. (2012). Integrating mercury science and policy in the marine context: challenges and opportunities. *Environmental research*, *119*, 132–42.

Lindberg, S., Bullock, R., Ebinghaus, R., Engstrom, D., Feng, X., Pirrone, N., Prestbo, E., Seigneur, C., & Fitzgerald, W. (2007). Mercury in Deposition A Synthesis of Progress and Uncertainties in Attributing the Sources of Mercury in Deposition. *AMBIO: A Journal of the Human Environment*, *36*(1), 19–33.

Lohman, K., Seigneur, C., Edgerton, E., & Jansen, J. (2006). Modeling mercury in power plant plumes. *Environmental science & technology*, *40*(12), 3848–54.

Lutsey, N., & Sperling, D. (2008). America's bottom-up climate change mitigation policy. *Energy Policy*, *36*(2), 673–685.

Macintosh, D. L., Spengler, J. D., Özkaynak, H., Tsai, L.-h., & Ryan, P. B. (1996). Dietary Exposures to Selected Metals and Pesticides. *Environmental Health Perspectives*, *104*(2), 202–209.

Mahaffey, K. R., Clickner, R. P., & Bodurow, C. C. (2004). Blood Organic Mercury and Dietary Mercury Intake: National Health and Nutrition Examination Survey, 1999 and 2000. *Environmental Health Perspectives*, *112*(5), 562–570.

Mahaffey, K. R., Clickner, R. P., & Jeffries, R. a. (2009). Adult women's blood mercury concentrations vary regionally in the United States: association with patterns of fish consumption (NHANES 1999-2004). *Environmental health perspectives*, *117*(1), 47–53.

Mahaffey, K. R., Sunderland, E. M., Chan, H. M., Choi, A. L., Grandjean, P., Mariën, K., Oken, E., Sakamoto, M., Schoeny, R., Weihe, P., Yan, C.-H., & Yasutake, A. (2011). Balancing the benefits of n-3 polyunsaturated fatty acids and the risks of methylmercury exposure from fish consumption. *Nutrition reviews*, *69*(9), 493–508.

Mason, R. P., Choi, A. L., Fitzgerald, W. F., Hammerschmidt, C. R., Lamborg, C. H., Soerensen, A. L., & Sunderland, E. M. (2012). Mercury biogeochemical cycling in the ocean and policy implications. *Environmental research*, *119*, 101–17.

Mason, R. P., & Sheu, G.-R. (2002). Role of the ocean in the global mercury cycle. *Global Biogeochemical Cycles*, *16*(4), 40–1–40–14.

Matus, K., Nam, K.-M., Selin, N. E., Lamsal, L. N., Reilly, J. M., & Paltsev, S. (2012). Health damages from air pollution in China. *Global Environmental Change*, *22*(1), 55–66.

Matus, K., Yange, T., Paltsev, S., Reilly, J., & Nam, K.-M. (2008). Toward integrated assessment of environmental change: air pollution health effects in the USA. *Climatic Change*, *88*, 59–92.

McAlpine, D., & Araki, S. (1958). Minimata Disease: An unusual neurological disorder caused by contaminated fish. *The Lancet*, *2*(7047), 629–631.

McCarthy, J. E. (2005). Mercury Emissions from Electric Power Plants: An Analysis of EPA's Cap-and-Trade Regulations. Tech. rep., Congressional Research Service.

McDowell, M. a., Dillon, C. F., Osterloh, J., Bolger, P. M., Pellizzari, E., Fernando, R., Montes de Oca, R., Schober, S. E., Sinks, T., Jones, R. L., & Mahaffey, K. R. (2004). Hair Mercury Levels in U.S. Children and Women of Childbearing Age: Reference Range Data from NHANES 1999-2000. *Environmental Health Perspectives*, *112*(11), 1165–1171.

McKelvey, W., & Oken, E. (2012). Mercury and Public Health: An Assessment of Human Exposure. In M. S. Bank (Ed.) *Mercury in the Environment: Pattern and Process*, chap. 13, (pp. 267–288). Berkeley: University of California Press.

Mergler, D., Anderson, H. a., Chan, L. H. M., Mahaffey, K. R., Murray, M., Sakamoto, M., & Stern, A. H. (2007). Methylmercury exposure and health effects in humans: a worldwide concern. *Ambio*, *36*(1), 3–11.

Milford, J. B., & Pienciak, A. (2009). After the clean air mercury rule: prospects for reducing mercury emissions from coal-fired power plants. *Environmental science & technology*, *43*(8), 2669–73.

Monterey Bay Aquarium Foundation (2013). Monterey Bay Aquarium Seafood Watch. URL `http://www.montereybayaquarium.org/cr/seafoodwatch.aspx`

Moya, J. (2004). Overview of Fish Consumption Rates in the United States. *Human and Ecological Risk Assessment: An International Journal*, *10*(6), 1195–1211.

Mozaffarian, D., & Rimm, E. B. (2006). Fish Intake, Contaminants, and Human Health: Evaluating the Risks and Benefits. *The Journal of the American Medical Association*, *296*(15), 1885–1900.

Myers, G. J., Davidson, P. W., Cox, C., Shamlaye, C. F., Palumbo, D., Cernichiari, E., Sloane-Reeves, J., Wilding, G. E., Kost, J., Huang, L.-S., & Clarkson, T. W. (2003). Prenatal methylmercury exposure from ocean fish consumption in the Seychelles child development study. *Lancet*, *361*(9370), 1686–92.

Nam, K.-M., Selin, N. E., Reilly, J. M., & Paltsev, S. (2010). Measuring welfare loss caused by air pollution in Europe: A CGE analysis. *Energy Policy*, *38*(9), 5059–5071.

Nincic, M. (2010). Getting What You Want: Positive Inducements in International Relations. *International Security*, *35*(1), 138–183.

NOAA (2010). NOAA Aquaculture Program Fact Sheet. URL `http://www.nmfs.noaa.gov/aquaculture/docs/aquaculture\_docs/aq\_fact\_sheet\_march\_2010.pdf`

NRC (2000). *Toxicological Effects of Methylmercury*. Washington, DC: National Research Counci, National Academy Press.

Nriagu, J., Basu, N., & Charles, S. (2012). Environmental Justice: The Mercury Connection. In M. S. Bank (Ed.) *Mercury in the Environment: Pattern and Process*, chap. 15, (pp. 301–316). Berkeley: University of California Press.

Nriagu, J. O. (1979). Production and uses of mercury. In J. O. Nriagu (Ed.) *The biogeochemistry of mercury in the environment*, chap. 2, (pp. 23–40). Elsevier/North Holland Biomedical Press.

Obrist, D., Tas, E., Peleg, M., Matveev, V., Faïn, X., Asaf, D., & Luria, M. (2010). Bromine-induced oxidation of mercury in the mid-latitude atmosphere. *Nature Geoscience*, *4*(1), 22–26.

Oken, E., Choi, A. L., Karagas, M. R., Mariën, K., Rheinberger, C. M., Schoeny, R., Sunderland, E., & Korrick, S. (2012). Which Fish Should I Eat? Perspectives Influencing Fish Consumption Choices. *Environmental Health Perspectives*, *120*(6), 790–798.

Olson, M. (1984). *The Rise and Decline of Nations: Economic Growth, Stagflation, and Social Rigidities*. New Haven, NJ: Yale University Press.

OMB (2003). Informing Regulatory Decisions : 2003 Report to Congress on the Costs and Benefits of Federal Regulations and Unfunded Mandates on State, Local, and Tribal Entities. Tech. rep., Office of Management and Budget, Office of Information and Regulatory Affairs, Washington, D.C.

O'Neill, C. A. (2004). Mercury, Risk, and Justice. *Enironmental Law Review*, *34*, 11070–11115.

O'Neill, C. A. (2009). The Mathematics of Mercury. In W. Harrington, L. Heinzerling, & R. D. Morgenstern (Eds.) *Reforming Regulatory Impact Analysis*, chap. 6, (pp. 108–141). Washington, DC: Resources for the Future.

Pacyna, E., Pacyna, J., Sundseth, K., Munthe, J., Kindbom, K., Wilson, S., Steenhuisen, F., & Maxson, P. (2010a). Global emission of mercury to the atmosphere from anthropogenic sources in 2005 and projections to 2020. *Atmospheric Environment*, *44*(20), 2487–2499.

Pacyna, J. M., Sundseth, K., Pacyna, E. G., Jozewicz, W., Munthe, J., Belhaj, M., & Aström, S. (2010b). An Assessment of Costs and Benefits Associated with Mercury Emission Reductions from Major Anthropogenic Sources. *Journal of the Air & Waste Management Association*, *60*(3), 302–315.

Petersen, A., van der Sluijs, J., & Tuinstra, W. (2006). Anticipation and Adaptation in Particulate Matter Policy: The European Union, the Netherlands, and United States. In *Background Papers for TAUC Workshop*, October. Washington, D.C.

Pindyck, R. S. (2007). Uncertainty in Environmental Economics. *Review of Environmental Economics and Policy*, *1*, 45–65.

Pirrone, N., Allegrini, I., Keeler, G. J., Nriagu, J. O., Rossmann, R., & Robbins, J. a. (1998). Historical atmospheric mercury emissions and depositions in North America compared to mercury accumulations in sedimentary records. *Atmospheric Environment*, *32*(5), 929–940.

Pirrone, N., & Keating, T. (Eds.) (2010). *Hemispheric Transport of Air Pollution 2010, Part B. Mercury*. Geneva, Switzerland: United Nations.

Pope, C. A. I., & Dockery, D. W. (2006). Journal of the Air & Waste Management Health Effects of Fine Particulate Air Pollution : Lines that Connect Health Effects of Fine Particulate Air Pollution : Lines that Connect. *Journal of the Air & Waste Management Association*, *56*(6), 709–742.

Portney, P. (1994). The Contingent Valuation Debate: Why Economists Should Care. *Journal of Economic Perspectives*, *8*, 3–17.

Rallo, M., Lopez-Anton, M. A., Contreras, M. L., & Maroto-Valer, M. M. (2012). Mercury policy and regulations for coal-fired power plants. *Environmental science and pollution research international*, *19*(4), 1084–96.

Rasmussen, R. S., Nettleton, J., & Morrissey, M. T. (2005). A Review of Mercury in Seafood : Special Focus on Tuna. *Journal of Aquatic Food Product Technology*, *14*(4), 71–100.

Rausch, S., Metcalf, G. E., & Reilly, J. M. (2010). Distributional Implications of Alternative U.S. Greenhouse Gas Control Measures. Tech. rep., National Bureau of Economic Research Working Paper 16053, Cambridge, MA.

Rausch, S., Metcalf, G. E., Reilly, J. M., & Paltsev, S. (2011). Distributional Impacts of a U.S. Greenhouse Gas Policy: A General Equilibrium Analysis of Carbon Pricing. In G. E. Metcalf (Ed.) *U.S. Energy and Tax Policy*, chap. 3. Cambridge, UK: Cambridge University Press.

Rice, D. C. (2004). The US EPA reference dose for methylmercury: sources of uncertainty. *Environmental research*, *95*(3), 406–13.

Rice, G. E., Hammitt, J. K., & Evans, J. S. (2010). A probabilistic characterization of the health benefits of reducing methyl mercury intake in the United States. *Environmental science & technology*, *44*(13), 5216–24.

Roger, V. L., Go, A. S., Lloyd-Jones, D. M., Benjamin, E. J., Berry, J. D., Borden, W. B., Bravata, D. M., Dai, S., Ford, E. S., Fox, C. S., Fullerton, H. J., Gillespie, C., Hailpern, S. M., Heit, J. a., Howard, V. J., Kissela, B. M., Kittner, S. J., Lackland, D. T., Lichtman, J. H., Lisabeth, L. D., Makuc, D. M., Marcus, G. M., Marelli, A., Matchar, D. B., Moy, C. S., Mozaffarian, D., Mussolino, M. E., Nichol, G., Paynter, N. P., Soliman, E. Z., Sorlie, P. D., Sotoodehnia, N., Turan, T. N., Virani, S. S., Wong, N. D., Woo, D., & Turner, M. B. (2012). Heart disease and stroke statistics–2012 update: a report from the American Heart Association. *Circulation*, *125*(1), e2–e220.

Roman, H. a., Walsh, T. L., Coull, B. a., Dewailly, E., Guallar, E., Hattis, D., Mariën, K., Schwartz, J., Stern, A. H., Virtanen, J. K., & Rice, G. (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), 607–14.

Rotmans, J., & van Asselt, M. B. (2001). Uncertainty management in integrated assessment modeling: towards a pluralistic approach. *Environmental monitoring and assessment*, *69*(2), 101–30.

Russell, M. W., Huse, D. M., Drowns, S., Hamel, E. C., & Hartz, S. C. (1998). Direct medical costs of coronary artery disease in the United States. *The American journal of cardiology*, *81*(9), 1110–5.

Saari, R., Rausch, S., & Selin, N. E. (????). Income, inequality, and the health-related economic impacts of ozone in the United States. *In prep.*.

Salkever, D. S. (1995). Updated Estimates of Earnings Benefits from Reduced Exposure of Children to Environmental Lead. *Environmental Research*, *70*(1), 1–6.

Schwartz, J. (1994). Societal Benefits of Reducing Lead Exposure. *Environmental Research*, *66*, 105–124.

Scott, D. P., & Armsrong, F. A. J. (1972). Mercury concentration in Relation to Size in several species of Freshwater Fishes from Manitoba and Northwestern Ontario. *Journal of Fisheries Research Board of Canada*, *29*, 1685–1690.

Seigneur, C., Vijayaraghavan, K., Lohman, K., Karamchandani, P., & Scott, C. (2004). Global source attribution for mercury deposition in the United States. *Environmental science & technology*, *38*(2), 555–69.

Selin, N. E. (2005). Environment : Science and Policy for Sustainable Development Mercury Rising : Is Global Action Needed to Protect Human Health and the Environment ? *Environment*, *47*(1), 22–35.

Selin, N. E. (2009). Global Biogeochemical Cycling of Mercury: A Review. *The Annual Review of Environment and Resources*, *34*, 43–63.

Selin, N. E. (2011). Science and strategies to reduce mercury risks: a critical review. *Journal of environmental monitoring*, *13*(9), 2389–99.

Selin, N. E. (2013). Global Change and Mercury Cycling: Challenges for Implementing a Global Mercury Treaty. *submitted to Environmental Toxicology and Chemistry*.

Selin, N. E., & Jacob, D. J. (2008). Seasonal and spatial patterns of mercury wet deposition in the United States: Constraints on the contribution from North American anthropogenic sources. *Atmospheric Environment*, *42*(21), 5193–5204.

Selin, N. E., Jacob, D. J., Yantosca, R. M., Strode, S., Jaeglé, L., & Sunderland, E. M. (2008). Global 3-D land-ocean-atmosphere model for mercury: Present-day versus preindustrial cycles and anthropogenic enrichment factors for deposition. *Global Biogeochemical Cycles*, *22*(2), n/a–n/a.

Selin, N. E., Sunderland, E. M., Knightes, C. D., & Mason, R. P. (2010). Sources of mercury exposure for U.S. seafood consumers: implications for policy. *Environmental health perspectives*, *118*(1), 137–43.

Shanley, J. B., & Bishop, K. (2012). Mercury Cycling in Terrestrial Watersheds. In M. S. Bank (Ed.) *Mercury in the Environment: Pattern and Process*, chap. 8, (pp. 119–142). Berkeley: University of California Press.

Shipp, a. M., Gentry, P. R., Lawrence, G., Van Landingham, C., Covington, T., Clewell, H. J., Gribben, K., & Crump, K. (2000). Determination of a site-specific reference dose for methylmercury for fish-eating populations. *Toxicology and industrial health*, *16*(9-10), 335–438.

Simon, H. A. (1990). Prediction and Prescription in Systems Modeling. *Operations Research*, *38*(1), 7–14.

Sippl, K., & Selin, H. (2012). Global Policy for Local Livelihoods: Phasing Out Mercury in Artisanal and Small-Scale Gold Mining. *Environment: Science and Policy for Sustainable Development*, *54*(3), 37–41.

Soerensen, A. L., Sunderland, E. M., Holmes, C. D., Jacob, D. J., Yantosca, R. M., Skov, H., Christensen, J. H., Strode, S. a., & Mason, R. P. (2010). An improved global model for air-sea exchange of mercury: high concentrations over the North Atlantic. *Environmental science & technology*, *44*(22), 8574–80.

Spadaro, J. V., & Rabl, A. (2008). Global health impacts and costs due to mercury emissions. *Risk analysis : an official publication of the Society for Risk Analysis*, *28*(3), 603–13.

Steffen, A., Douglas, T., Amyot, M., Ariya, P., Aspmo, K., Berg, T., Bottenheim, J., Brooks, S., & Cobbett, F. (2008). A synthesis of atmospheric mercury depletion event chemistry in the atmosphere and snow. *Atmospheric Chemistry and Physics*, *8*, 1445–1482.

Stern, A. H. (2005). A Revised Probabilistic Estimate of the Maternal Methyl Mercury Intake Dose Corresponding to a Measured Cord Blood Mercury Concentration. *Environmental Health Perspectives*, *113*(2), 155–163.

Stone, D. (2002). *Policy Paradox: The Art of Political Decision Making*. New York: W. W. Norton & Company, revised ed.

Storelli, M. M., Barone, G., Piscitelli, G., & Marcotrigiano, G. O. (2007). Mercury in fish: concentration vs. fish size and estimates of mercury intake. *Food additives and contaminants*, *24*(12), 1353–7.

Storelli, M. M., Giacominelli Stuffler, R., & Marcotrigiano, G. O. (1998). Total mercury in muscle of benthic and pelagic fish from the South Adriatic Sea (Italy). *Food additives and contaminants*, *15*(8), 876–83.

Streets, D. G., Devane, M. K., Lu, Z., Bond, T. C., Sunderland, E. M., & Jacob, D. J. (2011). All-time releases of mercury to the atmosphere from human activities. *Environmental science & technology*, *45*(24), 10485–91.

Streets, D. G., Zhang, Q., & Wu, Y. (2009). Projections of global mercury emissions in 2050. *Environmental science & technology*, *43*(8), 2983–8.

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), 235–42.

Sunderland, E. M., & Mason, R. P. (2007). Human impacts on open ocean mercury concentrations. *Global Biogeochemical Cycles*, *21*(GB4022).

Sunderland, E. M., & Selin, N. E. (2013). Future trends in environmental mercury concentrations: implications for prevention strategies. *Environmental health : a global access science source*, *12*, 2.

Sunstein, C. R. (2000). Cognition and cost-benefit analysis. *The Journal of Legal Studies*, *29*(S2), 1059–1103.

Swain, E. B., Jakus, P. M., Rice, G., Lupi, F., Maxson, P. A., Jozef, M., Penn, A., Spiegel, S. J., Veiga, M. M., & Pacyna, J. M. (2007). Socioeconomic Consequences of Mercury Use and Pollution. *Ambio: A Journal of the Human Environment*, *36*(1), 45–61.

Swartzendruber, P., & Jaffe, D. (2012). Sources and Transport: A Global Issue. In M. S. Bank (Ed.) *Mercury in the Environment: Pattern and Process*, (pp. 3–18). Berkeley: University of California Press.

Trasande, L., Landrigan, P. J., & Schechter, C. (2005). Public health and economic consequences of methyl mercury toxicity to the developing brain. *Environmental health perspectives*, *113*(5), 590–6.

Trasande, L., Schechter, C., Haynes, K. a., & Landrigan, P. J. (2006). Applying cost analyses to drive policy that protects children: mercury as a case study. *Annals of the New York Academy of Sciences*, *1076*, 911–23.

UNEP (2002). Global Mercury Assessment 2002. Tech. rep., United Nations Environment Programme Chemicals Branch, Geneva.

UNEP (2008). The Global Atmospheric Mercury Assessment: Sources, Emissions and Transport. Tech. rep., UN Environment Programme Chemicals Branch, Geneva, Switzerland.

UNEP (2013a). Global Mercury Assessment 2013: Sources, Emissions, Releases and Environmental Transport. Tech. rep., UNEP Chemicals Branch, Geneva, Switzerland.

UNEP (2013b). Report of the intergovernmental negotiating committee to prepare a global legally binding instrument on mercury on the work of its fifth session. Tech. rep., United Nations Environmentl Programme, Geneva, Switzerland. URL `http://www.unep.org/hazardoussubstances/Portals/9/Mercury/Documents/INC5/5\_7\_REPORT\_ADVANCE.pdf`

UNEP (2013c). UNEP Global Mercury Partnership. URL `http://www.unep.org/hazardoussubstances/Mercury/GlobalMercuryPartnership/tabid/1253/Default.aspx`

US Department of Labor Bureau of Labor Statistics (2012). No Title. URL `http://data.bls.gov/pdq/querytool.jsp?survey=le`

US EPA (2005). Regulatory Impact Analysis of the Final Clean Air Mercury Rule. Tech. rep., Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, NC.

US EPA (2009). Emissions Inventories. URL `http://www.epa.gov/ttn/chief/eiinformation.html`

US EPA (2011a). 2010 Biennial National Listing of Fish Advisories Fact Sheet. URL `http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/upload/technical\_factsheet\_2010.pdf`

US EPA (2011b). IPM Analysis of the Final Mercury and Air Toxics Standards. URL `http://www.epa.gov/airmarkets/progsregs/epa-ipm/toxics.html`

US EPA (2011c). Regulatory Impact Analysis for the Federal Implementation Plans to Reduce Interstate Transport of Fine Particulate Matter and Ozone in 27 States ; Correction of SIP Approvals for 22 States. Tech. Rep. June, U.S. Environmental Protection Agency Office of Air and Radiation.

US EPA (2011d). Regulatory Impact Analysis for the Final Mercury and Air Toxics Standards. Tech. rep., Environmental Protection Agency, Office of Air Quality and Planning Standards, Research Triangle Park, NC.

US EPA (2013a). Cross-State Air Pollution Rule (CSAPR). URL `http://www.epa.gov/crossstaterule/`

US EPA (2013b). Final Mercury and Air Toxics Standards (MATS) for Power Plants. URL `http://www.epa.gov/mats/actions.html`

US EPA (2013c). Mercury: Laws and Regulations. URL `http://www.epa.gov/hg/regs.htm`

Victor, D. G., House, J. C., & Joy, S. (2005). A Madisonian Approach to Climate Policy. *Science*, *309*, 1820–1821.

Vijayaraghavan, K., Karamchandani, P., Seigneur, C., Balmori, R., & Chen, S.-Y. (2008). Plume-in-grid modeling of atmospheric mercury. *Journal of Geophysical Research*, *113*(D24), D24305.

Virtanen, J. K., Rissanen, T. H., Voutilainen, S., & Tuomainen, T.-P. (2007). Mercury as a risk factor for cardiovascular diseases. *The Journal of nutritional biochemistry*, *18*(2), 75–85.

Virtanen, J. K., Voutilainen, S., Rissanen, T. H., Mursu, J., Tuomainen, T.-P., Korhonen, M. J., Valkonen, V.-P., Seppänen, K., Laukkanen, J. a., & Salonen, J. T. (2005). Mercury, fish oils, and risk of acute coronary events and cardiovascular disease, coronary heart disease, and all-cause mortality in men in eastern Finland. *Arteriosclerosis, thrombosis, and vascular biology*, *25*(1), 228–33.

Viscusi, W. K., Harrington Jr., J. E., & Vernon, J. M. (2005). *Economics of Regulation and Antitrust*. Cambridge, MA: MIT Press, 4th ed.

Wagner, W. E. (2009). The CAIR RIA: Advocacy Dressed Up as Policy Analysis. In W. Harrington, L. Heinzerling, & R. D. Morgenstern (Eds.) *Reforming Regulatory Impact Analysis*. Washington, D.C.: Resources for the Future.

Waugh, C. J. (2012). *An Integrated Assessment of Air Pollutant Abatement Opportunities in a Computable General Equilibrium Framework*. Master's thesis, Massachusetts Institute of Technology.

Webster, M., Santen, N., & Parpas, P. (2012). An approximate dynamic programming framework for modeling global climate policy under decision-dependent uncertainty. *Computational Management Science*, *9*(3), 339–362.

Weiss-Penzias, P. S., Gustin, M. S., & Lyman, S. N. (2011). Sources of gaseous oxidized mercury and mercury dry deposition at two southeastern U.S. sites. *Atmospheric Environment*, *45*(27), 4569–4579.

Wheatley, B., & Paradis, S. (1995). Exposure of Canadian Aboriginal Peoples to Methylmercury. *Water, Air, and Soil Pollution*, *80*, 3–11.

Wittels, E. H., Hay, J. W., & Gotto, A. M. (1990). Medical Costs of Coronary Artery in the United States Disease. *Journal of Cardiology*, *65*, 432–440.

Wolfe, M. F., Schwarzbach, S., & Sulaiman, R. A. (1998). EFFECTS OF MERCURY ON WILDLIFE : A COMPREHENSIVE REVIEW. *Environmental Toxicology and Chemistry*, *17*(2), 146–160.

Zax, J. S., & Rees, D. I. (2002). Iq, academic performance, environment, and earnings. *The Review of Economics and Statistics*, *84*(4), 600–616.

Zhang, Y., Jaeglé, L., van Donkelaar, a., Martin, R. V., Holmes, C. D., Amos, H. M., Wang, Q., Talbot, R., Artz, R., Brooks, S., Luke, W., Holsen, T. M., Felton, D., Miller, E. K., Perry, K. D., Schmeltz, D., Steffen, a., Tordon, R., Weiss-Penzias, P., & Zsolway, R. (2012). Nested-grid simulation of mercury over North America. *Atmospheric Chemistry and Physics*, *12*(14), 6095–6111.