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Reducing Exposures to Mercury

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by

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Abstract

Mercury is an environmental contaminant affecting neurological development, the immune system and cardiac health. Air emissions become environmental pollutants impacting air, water and land. Consumption of mercury-contaminated fish can create public health concerns, primarily to pregnant women and their fetuses. New York State's public policy response addresses incidental exposures with annual fish advisories and restrictions on mercury emissions and products sold or used. Additional public policy responses to the mercury problem can include: direct notification of advisories to fish consumers, enhanced public health notification of benefits and risks of consuming fish, labeling requirements on mercury-containing products, and expanded scientific data collection to track mercury interactions in the atmosphere to determine confounding factors contributing to mercury exposures.

PART I – SOURCES OF MERCURY

CHAPTER 1 – DEFINING THE PROBLEM

1.0 Natural Sources of Mercury

Mercury is an insidious environmental pollutant, present in our air, water and soils. As a naturally-occurring element, mercury can be found across all continents and in the oceans. Its unique characteristics, such as its ability to be a liquid metal at standard atmospheric pressures and to vaporize readily, promote its pervasive use in our industrialized society. Mercury is also naturally found at trace levels in coal, in ores as well as in vegetative matter. As part of a natural cycle (see Figure 1 below), mercury gases are released and redeposited interconnecting the lithosphere, atmosphere and biosphere. Circulating air currents carry reactive air pollutants, such as ozone, that can oxidize elemental mercury (Hg^0), influencing where mercury may deposit.

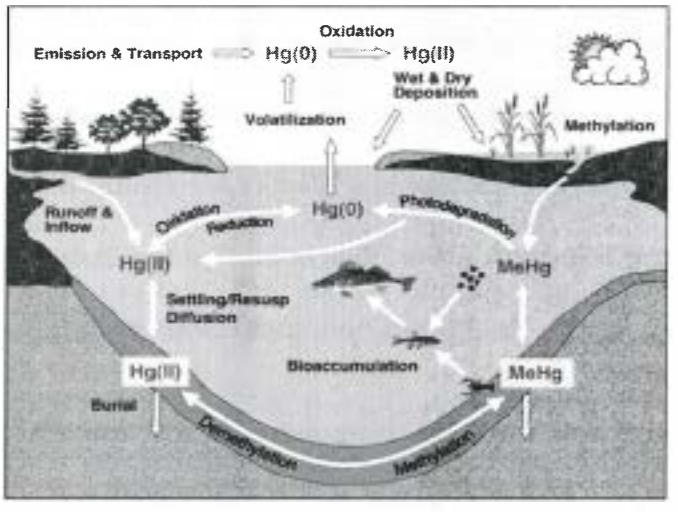

Figure 1 : Natural mercury cycle processes (from Engstrom, 2007)

Mercury is present in three main species, or, forms: elemental, inorganic (as a metallic salt or reactive divalent gas), and organic. Air emissions are primarily inorganic as elemental mercury and reactive gaseous mercury. Elemental mercury gas is significantly less soluble than divalent (Hg^{2+}) mercury, allowing it to persist in the atmosphere for longer periods, thereby travelling further distances. Natural emissions are almost entirely elemental mercury gas (Streets et al, 2005). Elemental mercury is thus difficult to attribute to a particular source, and is also global in extent. Inorganic mercury compounds can be formed naturally, such as HgS and HgCl, but are mainly synthesized for pharmaceutical uses. Mercury has a strong affinity for carbon (Lawson, 1999), thus organic mercury tends to form in the natural environment through bacterial transformation of inorganic Hg^{2+}. Of the three forms of mercury, organic mercury is the most toxic form affecting human and animal systems.

Natural sources of mercury in the atmosphere include volcanic eruptions and lava off-gassing, mercury-containing soil erosion and evasion, as well as re-emission from photolytic transformation of reactive divalent mercury in shallow waters. Approximately 30% of the total mercury load in the atmosphere[1] derives from natural sources. Areas along active geologic subduction belts, such as the "Ring of Fire" in East Asia and Western South America, may experience higher contributions from natural sources. In regions where these geologic zones combine with negligible industrial pollution controls from coal and petroleum combustion, as well as from mining and cement operations, significant impacts

1 Trasande et al (2005) projects 70% of the 5500 metric tons of annual US mercury emissions derive from anthropogenic sources; others, such as Zhang & Wong (2007) estimate natural emissions in the order of 2000 tons per year plus an additional 2000 tons from re-emissions of all deposited mercury into the environment, with 2000-2200 tons from anthropogenic sources.

to the environment may result. Public policy decisions that do not respond to the significant natural contribution of elementary mercury from different regions and air flow patterns, can hinder the intent of improving public health by not addressing all the factors that may contribute to creating highly-concentrated deposition ("hot spot") zones.

1.1 Adding Anthropogenic Emissions to the Natural Cycle

In our industrialized world, the natural and anthropogenic mercury cycles collide, creating severely impacted areas far from known source regions. Reactive mercury, consisting of divalent gaseous mercury and particulate mercury (Hg_P), are often found within 50 miles of an industrial source (IWLA, 2004; Malcolm & Keeler, 2002). Elemental mercury – released both naturally and anthropogenically - can be transformed by reactive oxidants, such as ground level ozone and other air pollutants, into the more soluble[2] divalent mercury form which precipitation cleanses from the atmosphere and which is transformed biologically in the lithosphere into organic mercury. Elemental mercury can persist in the atmosphere up to a year, depending on meteorological conditions, traveling several hundreds of miles from its original source (Malcolm & Keeler, 2002; Hall, 1995). Thus, mercury can be present in varying forms based on type of sources, relative distances from these sources and meteorological conditions of the receiving downwind environment. Public policy assessments will need to evaluate these complex natural and anthropogenic interactions when setting discharge limitations on anthropogenic sources, in order to answer the systemic question regarding the effectiveness of pollution controls in reducing local and regional air pollution.

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2 Divalent mercury gas (Hg2+) is 100,000 times more soluble than elemental mercury (Hg0) (Lindberg et al., 1998). Particulate mercury is adsorbed to cloud droplets, enhancing its removal from the atmosphere and localized deposition (Lawson, 1999)

Even at relatively low temperatures[3], mercury can vaporize into the atmosphere due to its unique characteristics – thus, anthropogenic use of ores can result in mercury pollution where other trace metals may not present concerns. Industrial mercury air emissions can vary significantly in the form of mercury emitted, based on the type of regulatory controls used. Without controls, higher concentrations of reactive mercury are emitted, such as divalent gaseous mercury (Hg^{2+}) and particulate mercury (Hg_P) (Streets et al, 2005). These forms of mercury comprise a significant portion of residential and small-scale industrial emissions resulting from burning coal (Streets et al, 2005). Where more sophisticated pollution abatement controls are used, less particulate mercury is emitted and more reactive divalent mercury is solubilized from the waste stream, which results in a higher percentage of elemental gas emitted relative to total mercury emissions[4]. Pollution controls thus follow the Law of Conservation of Mass; they do not destroy the material. Air pollution controls concentrate the mercury into fly-ash residues used in the cement industry or deposited into landfills. Finding the form that presents the least hazardous exposures to wildlife and humans is the challenge with mercury.

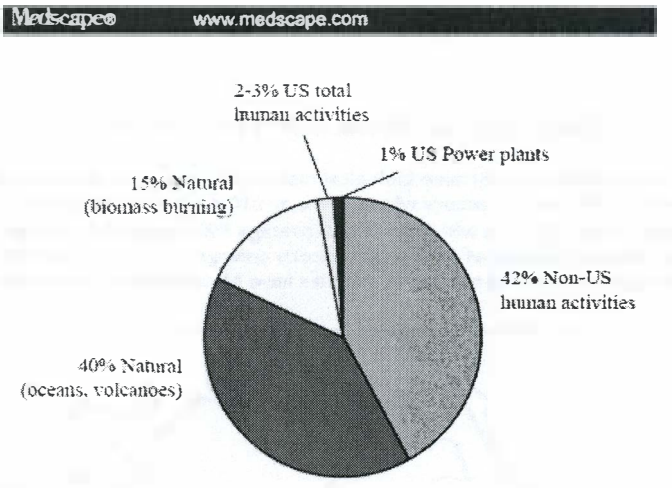
The global contribution of mercury, as graphically presented by Charnley (2006) in Figure 2 below, supports a high percentage originating from natural sources. Considering the volumes from natural sources and global industrial discharges, the differences by which

3 Temperatures above 150 °C can accelerates vaporization of mercury into the atmosphere, thus smelting, coal combustion and many chemical synthesis processes contribute to mercury air pollution (Zhang & Wong, 2007). Elemental mercury at room temperature also volatilizes enough to present inhalation exposure concerns.

4 Where particulate or other pollution controls are in use, such as with coal-generated power plants, approximately 32-35% of divalent mercury and between 10-16% of particulate mercury is released, with up to 46-56% of mercury emissions comprised of elemental mercury gas (Zhang & Wong, 2007; Streets et al, 2005; Shetty et al., 2008)

Figure 2: Percentages of mercury contributions globally to the atmosphere

(from Charnley, 2006)

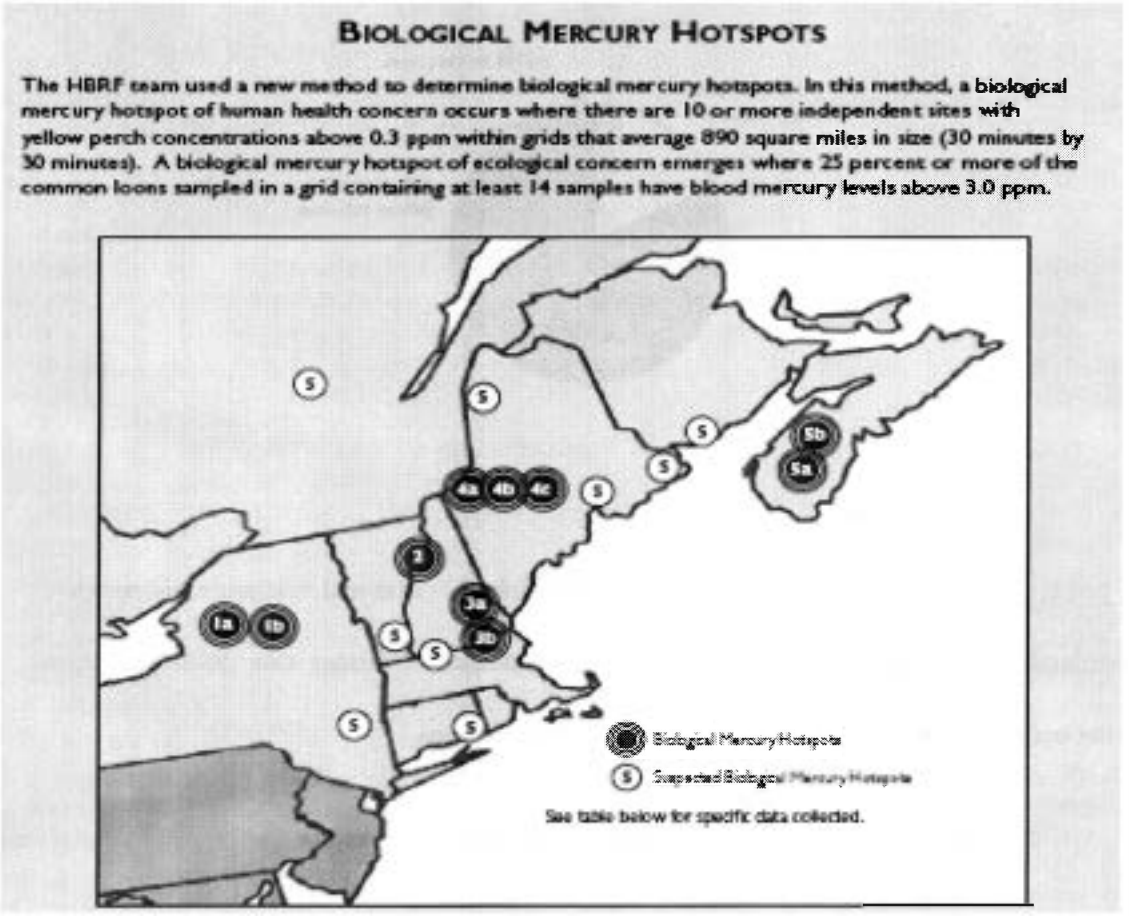


US policy can affect the global response will likely be minimal without international concordances. Yet a national response to reduce emission sources can yield significant benefits on a regional and local basis in minimizing exposures.

1.2 Regional Differences with Natural Mercury Emissions

The percentage of atmospheric mercury from natural sources will vary regionally across the United States, based on geologic conditions. Areas rich in coal or nonferrous ores may contribute more mercury naturally, as would regions where active volcano emissions occur. In New York State, natural sources contribute only an estimated 4 to 16% of the total atmospheric mercury levels (Seigneur et al, 2003). However, the amount of mercury in fish

and piscivorous waterfowl is higher[5] relative in New York than in western regions. Air currents carry natural and anthropogenic mercury emissions eastward, resulting in "hot spot" deposition in the wet, downwind regions of New York, New England and Maritime



(source: HBRF, 2007)

Figure 3: Biological "hot spots" for mercury in northeastern North America.

5 Blood Hg levels in Wisconsin loons ranged up to 5.2 ppm, with New England loons detecting up to 7.4 ppm; feather levels of mercury were up to 46 ppm in New England but only ranged up to 25 ppm in Wisconsin (Mitro et al, 2006). Perch is listed as a low mercury fish species in Wisconsin but is considered a high mercury fish species warranting fish consumption advisories in New York State.

Canada. International cooperation is thus essential in regulating mercury. Sustainability responses are not strictly environmental, but also have significant political and economic costs.

CHAPTER 2: ANTHROPOGENIC SOURCES – INDUSTRY

"If by fire Of sooty coal th' empiric alchymist Can turn,
or holds it possible to turn,
Metals of drossiest ore to perfect gold."
- John Milton [*Paradise Lost*]

2.0 Global Industrial Emissions

In the United States, mercury emissions result primarily from the burning of coal to generate electrical power. In China, the primary anthropogenic source of atmospheric mercury is the nonferrous mining operations, especially zinc smelting (Zhang & Wong, 2007). Significant quantities of mercury vapor are released in smelting, up to 386 tons per year according to Zhang & Wong (2007). Globally, however, the greatest single contributing source is from coal combustion. Thus a focal plan is needed to address coal combustion emission technology in particular; however, all contributing anthropogenic sources need to be assessed in order to reduce controllable emissions. Different sources may require different regulatory and/or technological approaches at controlling emissions.

The type of coal used is a major factor in the forms of mercury emitted, and thus may dictate the preferred pollution control technology employed. As outlined in Table 1 below, the softer the coal (lignite), the higher percentages of non-reactive, elemental mercury are likely to be emitted. Elemental mercury is predicted to deposit significant distances from a source, whereas reactive gaseous mercury species tend to deposit near a source. As such, the type of coal burned at a facility can add to the development of mercury "hot spots" when a softer coal emits mercury that affects a downwind community more than locally. In the United States, western coal (mainly from Wyoming) is primarily

bituminous, or, anthracite; Eastern coal (from Pennsylvania mainly) is lignitic to sub-bituminous and yields more transportable elemental mercury than western coal (Kolker et al, 2006; USGS, 2001). This difference can impact downwind regions, like New York.

Table 1: Percentages of mercury species prevalent in different grades of coal

Mercury Species	Bituminous	Sub-bituminous	Lignite
Hg^0	20%	65%	85%
Hg^{2+}	35 %	20%	10%
Hg_p	45 %	15%	5%

(Table created from data presented in Yang et al, 2007)

The varying levels of mercury present in a raw material, such as coal or ore, contribute to the mercury load in the atmosphere where these materials are burned or processed. In China, where the higher mercury concentration metal ores are mined, more mercury is emitted in these provinces (Zhang & Wong, 2007). Couple the higher mercury-containing raw materials with focused industrialization based on proximity to transport routes, and there can be severe disparities in regional mercury emissions.

Wilson et al. (2006) outline the global distribution of mercury emissions, from both point and non-point sources, based on 2000 emission datasets (see Figure 4). This figure presents the total areal distribution of anthropogenic mercury emissions in terms of kilograms per 0.5° cell, with the darkest hues representing 5,000 to 10,000 kg per cell (Wilson et al, 2006). As demonstrated in the figure, Southeast Asia, in particular China, is a major contributor of mercury emissions, based on its growing industrialized consumption of coal. The Arabian Peninsula may experience higher than average levels from petroleum oil fires, another release mechanism for mercury. Other regions, such as Africa, may exhibit

elevated mercury levels spatially, based on mercury mining activities, such as artisan gold processing which uses mercury in the amalgamation and results in significant mercury vapor

Figure 4: Graphic distribution of mercury emissions from human activities
(From Wilson et al. (2006))

(Source:
http://www.amap.no/Resources/HgEmissions/HgInventoryMaps.html#HgT2000)

off-gassing (Streets et al, 2005)[6] India's growing industrialization may contribute to elevated coal consumption and mercury emissions, whereas Eastern Europe may reflect elevated levels from a combination of aging industrial facilities with little to no pollution controls coupled with mining activities. Some of the world's most highly industrialized regions, such as Europe, Eastern United States and Brazil, reflect lower mercury contributions compared

6 Artisanal gold mining was banned in China in 1996 but may persist in remote areas where gold is mined, according to Streets et al (2005).

to the growing industrialized countries like China and India, perhaps from use of pollution control devices.

Figure 4 presents but a snapshot in time. Protecting our future requires policy makers to consider the future global response of using natural resources containing mercury. China is the largest producer of zinc, a significant contributor to the global mercury load. Zinc ores contain sulfides, which also bind strongly with mercury. As the quality of the zinc ore declines, the amount of mercury present has been increasing, along with its release into the air from the smelting process (Zhang & Wong, 2007). Understanding that the mercury content of ores processed can be on the upswing can assist policy makers in focusing regulations that can address effective industrial controls in the near future. Within the highly industrialized temperate belt of the Northern Hemisphere, downwind regions may receive significant contributions from upwind source areas, emphasizing the need for a global approach to controlling mercury emissions as well as public policy that considers geographic and scientific contributions in the mercury debate.

2.1 North American Industrial Emissions

North American anthropogenic activities contribute approximately 20% of the mercury deposition load to the environment (Selin et al., 2007), indicating natural and global sources are significant influences on mercury deposition in the United States. Streets et al. (2005) estimates that coal-generated power plants are the primary mercury polluters, contributing 1% of the total global mercury emissions (but 41% of anthropogenic mercury). Using 2000 data, China emits an order of magnitude more mercury from coal-generated

power plants than does the United States (200 tons versus 21 tons, Zhang & Wong, 2007). However, most mercury deposition in the United States occurs east of the Mississippi River.

Electricity is generated through steam-driven turbines; coal is a fuel often used to create the steam. Heating coal releases natural gases locked into coal, a geological receptacle of plants that absorbed mercury from the ancient atmosphere. Mercury is but one of these gases released in the flue gas. Concentrated levels of mercury are also contained in the fly-ash waste materials. This fly-ash material is used in the cement industry, which can create in itself significant re-emission problems[7]. The higher the fly-ash mercury content is, the greater the concern for mercury air emissions as a pollutant.

Both power generation and cement production are necessary elements of a growing industrial region, and both sources contribute heavily to atmospheric mercury pollution. China is also the world's largest cement producer (23 tons in 1999), indicating this country's public policies can significantly impact the world's atmosphere.

2.2 Global Public Policy Responses

Mercury emissions are not restricted to the industrialized world, nor specific to the United States economy. Thus, a response that is global will be needed to minimize the influence of anthropogenic emissions on wildlife and human health. A recent concordance to reduce mercury emissions as soon as practicable was reached in February 2009 with the 140 countries participating in the United Nations, including the United States and China (NRDC, 2009c). The United Nations Environmental Program will be constructing a treaty

7 The top 100 cement kilns are to be regulated by the USEPA by September 2009 (Green, 2008).

to be signed by participating members to reduce air, water and land discharges of mercury to the environment. An effective date of 2013 is anticipated, and will address coal-fired power plants – an industry with mercury emissions still not regulated nationally in the United States.

Implementation of this treaty, with China as a participant, will help stem the burgeoning increases expected with mercury emissions with the growth of China's economy and dependency on coal as a fuel source[8]. China is a rapidly-growing economic entity that consumes over a billion tons of coal annually (Zhang & Wong, 2007). In order to reach the goal of reduced global emissions, efficient air pollution control technologies will need to be shared[9]. Cooperative responses can advance environmental benefits without detracting unnecessarily from the economic growth process. Coupling the significant mercury load produced currently by China, with the rising economic production being demonstrated by China, a focused agreement to build environmentally friendly power plants is critical. Without this international cooperation, the end results cannot be significantly achievable. The biggest "players" in this enviro-economic realm will need to have the most effective tools to make the differences sought by all.

2.3 National Public Policy Responses

In 1990, the United States Environmental Protection Agency (USEPA) received a directive from Congress to address mercury as an environmental air pollutant. The Clean

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8 75% of China's power derives from coal-fired power plants: up to 560 new plants are to be built over the next eight years (NRDC, 2009c) . China has higher Hg content coal and poor removal efficiencies on its power plants (Zhang et al, 2008)

9 Advanced technology could become a bartering tool for the United States to reduce its trillion dollar debt to China.

Air Act Amendment of 1990 targeted mercury under a category of hazardous air pollutants designed "persistent, bioaccumative or toxic" compounds, or, PBTs, due to the ability of these pollutants to bioaccumulate, creating additive exposures that contribute to a high body burden over time.

A component of the 1970 Clean Air Act required new or substantially improved power plants to undergo pollution controls, under a continuously evolving New Source Review (NSR) rule (Federal Register, 2009). At the time, operating plants were provided a "waiver" in upgrading their pollution controls to address hazardous air pollutants (HAPs). The waiver's intent was that as plants aged, upgrades would be made. However, this NSR requirement has been a focus of controversy with the electric-generating power industry since it was issued in 1977. The end result is that the idea of the waiver has backfired. According to the National Wildlife Federation's (2006a) Fact Sheet on Mercury Pollution from Power Plants, 68% of power plants today are older than 30 years – a reflection of the industry's desire to avoid meeting New Source Review requirements. The current political and economic climate in the United States is focusing on infrastructure improvements. This is an area where modernization is desperately needed to meet the increasing power demands of our society and also address the environmental and public health hazards of facilities emitting high levels of HAPs.

The Clean Air Mercury Rule (CAMR), issued final by the USEPA in March 2005, was developed as part of the "Clear Skies Initiative" (USEPA, 2009a) in response to the 1990 Clean Air Act Amendments (CAAA 1990) directive to regulate mercury air emissions. The national regulatory approach was to assign each state (plus two tribes) an emissions budget and provide a trading model for which a plan to meet this emission budget (through

either adopting in full, or key components, of the model) was to be submitted to the USEPA (Bergeson, 2006). The heart of this rule was a business model of a "cap-and-trade" response to drive mercury emission reductions. This philosophy allows for a set number of end pollutants (for example, in pounds) to represent a "cap" for a facility's emissions. However, the "trade" component allows the same facility to buy credits if they cannot meet the capped allowance. Widely criticized by environmentalists and health professionals[10], this approach has sparked controversy with CAMR and impacted downwind states, as it doesn't address the heaviest polluting facilities' underlying problems. The position argued by environmentalists is that air pollutants do not respect geopolitical boundaries and thus the ability of a facility to buy credits to offset its emissions, irrespective of its location to downwind receptors, can allow continued impacts to downwind "hot spots" of mercury contamination. Many states, tribes and environmental groups found this approach to be unacceptable in addressing mercury exposure concerns.

Fourteen states, including New York, sued the USEPA to prevent this rule from becoming a national public policy program. Of issue was that the CAMR violated the provisions of the Clean Air Act in regulating mercury emissions, as it specifically exclude sources such as coal and gas power plants from meeting Maximum Achievable Control Technologies (MACT) standards that applied to other regulated industries for air emissions. Instead of MACT, these industries with significant mercury emissions were able to participate in the "cap-and-trade" approach. The March 2005 final mercury rule was diametrically opposite to earlier USEPA determinations in 2000 that coal-generated power plants should meet MACT standards under Section 112 of the CAA (AMA, 2006).

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10 Over 600,000 comments were submitted in opposition to CAMR (AMA, 2006)

Recent court decisions have resulted in uncertainty about the future of regulating emissions, as required under CAAA 1990. The "Mercury Emissions Reduction Act" [H.R. 1087] was proposed to address the gap in requiring coal-fired power plants, to meet MACT standards, in response to the CAMR controversy. In February 2008, the CAMR rule was vacated by the courts, prompting an appeal by the USEPA (Air Pollution Consultant, 2008). In July 2008, the Clean Air Interstate Rule (CAIR) was additionally vacated by the courts. But in December 2008, the court reversed itself, allowing CAIR to remain in effect because "having a flawed rule temporarily in place was better than having no rule at all" (Barringer, 2008b). Finally, by February 2009, the Supreme Court declined to hear the EPA's appeal of the lower court's ruling that the CAMR specifically created a loophole for the coal-burning power plants; thus effectively ending the "cap-and-trade" plan (NRDC, 2009d). This approach was scheduled to be implemented in 2010, but now these facilities will need to meet the MACT standards as do other regulated industries (Pollution Engineering, 2009). The past year has been tumultuous; the courts recognized the "Clear Skies Initiative" of CAIR and CAMR had good intentions of reducing emissions that contribute significantly to public health exposures, but yet also recognized that the rules were flawed and required better controls to achieve the intended end point.

With the December 2008 federal appeals court decision reversing the earlier decision to vacate the Clean Air Interstate Rule (CAIR), some progression forward is expected in addressing the hazardous air pollutants (HAPs) on a national level. This decision was important, as action is necessary for HAPs; a recently-issued USEPA report found the number of non-attainment areas for air quality has doubled for fine-particle pollution (Barringer, 2008b). Regulatory action is needed also for mercury: there are over

450 coal-generating power plants today emitting mercury without federal emission requirements (NRDC, 2009c)

2.4 State Actions on Regulating Mercury Emissions

States have taken the lead in addressing mercury emission requirements. Between 2002 and 2005, twenty states across the nation had enacted legislation that called for reductions of mercury emissions by 75-90% (NWF, 2006b). Regulatory limits on municipal and medical waste incinerators, issued in 1995, have yielded significant (78%) declines in mercury emissions over a short period of time (Han et al, 2008). This supports the benefits that can be realized locally from regulating emissions.

New York State was not on the forefront of this movement, although is one of the twenty that has some plan in action at the legislative proposal level. New York has promulgated rules on emissions caps in lieu of participation in the now-vacated CAMR, with Phase I to begin by 2010 and Phase II, requiring facilities meet specific emission limitations, to begin by 2015 (King et al, 2008). New York State issued a report in 2006 on actions it will take to address the "Mercury Challenge", including regulatory responses to legislation passed in 2004. This report details specific actions New York State is taking to address mercury in our environment: from air emissions, to wastewater containing dental amalgams, to mercury contained in the products we used in our daily lives.

3.0 Natural Capital as an Economic Cost

An argument commonly raised concerns the cost of providing pollution controls and the variability of mercury content with different coal sources. Although the coal quality has an impact on the species emitted (Table 1), implementation of MACT standards is likely to reduce over 90 percent[11] of total mercury emissions for all types of coal (Yang et al, 2008; Charnley, 2006). There are two main pollution control technologies that have been in use for decades: Electrostatic Precipitators (ESPs) and Fabric Filter baghouses (FF), which are often used in conjunction with other technologies such as Flue-gas Desulfurization (FGDs) and Spray Dry Absorbers (Kolker et al, 2006). Of the technologies in use today to meet National Ambient Air Quality Standards, fabric filters are the most efficient, removing over 80% of total mercury from flue gases (Wang et al., 2008; Zhang et al., 2007; Cao et al., 2008). Municipal incinerators are more likely to have fabric filter baghouses installed whereas coal-burning power plants have ESPs preferentially (Scala & Clack, 2008). New technology is under development that may be more effective, such as activated carbon injection using iodine-treated carbon or brominated-carbon injection upstream of the pollution control devices. They have demonstrated removal efficiencies greater than 90% in incinerator use (AMA, 2006). Lawyer Martha Keating states "...this technology has been available and shown to be cost-effective since 2002" (ES&T Online News, 2007). These newer technologies under development to meet air emission standards could provide jobs,

11 Range can be 0-90% for Hg – activated carbon injections can increase removal rates by 25% (Yang et al, 2008)

as well as immense savings in terms of environmental and public health benefits for the population. Most states with emission plans have set pollution control minimum efficiencies of 75 to 90% (NWF, 2006a).

3.1 Toxic Release Inventory (TRI) Program

The initial step of any new program is establishing a baseline upon which improvements can be demonstrated. In the reauthorization of the Superfund law, the Emergency Planning and Community Right-to-Know Act (EPCRA) was born. EPCRA provides baseline information about environmental discharges to air, land and water. Annual reporting of pollutant discharges to the environment for selected chemicals is required under Section 313, to both the USEPA and to the state where the facility is located. Mercury is designated as a PBT compound, thus does not have a *DeMinimus* quantity allowance as do other, non-PBT chemicals. The USEPA is required to provide this reported information in a database to the public, which is posted on their website (www.epa.gov/tri). The most recent dataset, the 2007 Reporting Year, was posted on March 19, 2009. Recent changes under the *2009 Omnibus Appropriations Act*, effective March 11, 2009 require the PBT compounds to be reported on the detailed forms (USEPA, 2009c). These changes will affect the upcoming July 2009 deadline for the 2008 Reporting Year datasets. As a result, the public will be better informed about a company's environmental releases.

Through the TRI database, an evaluation can be made of what types of facilities release mercury to the air and their quantities. Nationally, mining and coal-based power plants account for the top polluters. For New York State, the industry that emits the most

mercury into the air is a cement kiln facility. The leading facility, LaFarge Cement in Ravena, had increased its mercury emissions dramatically over the past five years. This may be a response to economic growth in the Capital District during the same time period or a reflection of different measurement techniques used with the past owner. Based on 2002 TRI data, it emitted approximately 37 pounds of mercury, consistent with past historical releases. The 2006 dataset reported 414 pounds of mercury emitted into the air, but with newer pollution controls under development, economic growth of the facility may be accomplished concurrent with reductions in mercury emissions[12] (USEPA, 200ct).

The TRI database allows regulating authorities, as well as the public, to track emissions and land/water discharges over time. This process provides information on efficiency of pollution controls, the impacts of reducing mercury-containing products in manufacturing, and where more reductions are needed in the future. Although power plants and cement industries are significant sources of mercury pollution in New York State, they are not sole sources of mercury exposure to the public, and like cumulative exposures to people, totality of mercury controls amongst all sources is warranted to reduce mercury emissions.

CHAPTER 4: MERCURY-CONTAINING PRODUCTS

Geology and biology are often interwoven. We use natural resources to enhance the quality of life with products that reduce time or work effort, but are often unaware of the

12 Recent newspaper reports state LaFarge has reduced its emissions to approximately 160 pounds for Reporting Year 2008 (official numbers due July 1, 2009) (Green, 2008).

human health impacts that these products can present. We use mercury, a known developmental toxicant, in a variety of daily activities for the physical benefits this metal can provide yet we do so without realizing the potential for harm these products can create if not used properly or at significant concentrations above safety margins.

4.0 Mercury Use in Lighting

Most fluorescent lamps use some mercury in either powder or liquid metal form, due to its high electrical and thermal conductivity physical properties coupled with a low vapor pressure. The result is more energy-efficient lighting that can last much longer than incandescent or other types of light. Although the mercury short-arc or vapor lamps can contain greater than 1,000 mg of mercury (Hg), most (50%) of fluorescent lamps sold contain between 5-10 mg, with 25% containing between 10-50 mg Hg, according to the National Electrical Manufacturers Association (IMERC, 2008c). Mercury can be found in lights commonly used in many residences, including black lights, bug zappers and tanning lamps, and can be found in most "neon" lights, except red neon which uses only neon gas (IMERC, 2008c). Neon lights can have up to 600 mg Hg (IMERC, 2008c). As lamp bulbs are fragile and have a tendency to break in the home, knowing which lamps present an exposure concern is as important as knowing the proper methods in cleaning up a broken mercury lamp bulb. Mercury vapors are odorless, so exposures can occur without a warning smell or other indicator of a problem.

Recent increased use of mercury-containing compact fluorescent light bulbs (CFLs) presents a more universal, daily exposure scenario for the American public. Using CFLs is considered a "green" response to reducing electricity usage, as they are significantly more efficient than incandescent counterparts. With Americans feeling the need to reduce

wasteful power consumption and overall energy usage, sales of CFLs have increased substantially, with a 70% increase between 2001 and 2004 (IMERC, 2008c). When a CFL bulb replaces an incandescent bulb, the amount of energy saved over the lifetime of the CFL bulb correlates to a savings between 5-13 mg/kg of Hg from the emissions cycle at a coal-burning power plant (IMERC, 2008c). Saving energy is a driving rationale for many people in buying the more expensive CFL – it is a personal action that saves energy costs in economic and environmental terms. However, the "green" benefit can come with a surprisingly personal risk for the consumer, often without awareness of the risk. If the CFL is broken within the home, a comparable amount of mercury that was saved from the atmosphere can be released into the home, resulting in high levels of mercury vapors and personal exposure. Many consumers are unaware of the presence of mercury in these bulbs, thus precautions are not likely to be implemented. Consumer outreach and product labeling are needed to increase the awareness of this commonly used source of mercury in the home. Two-thirds of all CFLs have approximately 4.5 mg Hg, with most containing less than 10 mg Hg (Energy Star, 2008).

Broken CFL bulbs in enclosed air spaces can present a significant inhalation exposure. Stahler et al (2008) detected elevated concentrations of elemental mercury gas following breakage, up to 52,000 micrograms per cubic meter of air ($\mu g/m^3$). Of concern, the air levels were highest near the floor (child breathing zone) than at 5 feet (an adult breathing zone). Both zones exceeded the Maine Ambient Air Guideline of 0.3 $\mu g/m^3$ (Stahler et al, 2008) during the first 15 minutes following bulb breakage. Mercury levels could also rebound or persist over a longer period if the spill was not properly cleaned. Recommendations for cleaning a spill include allowing an initial ventilation period of

approximately 15 minutes and clearing people of the area; then wiping the spill with a paper towel, containing the waste including the shards in a sealed glass jar or in a double-layer sealed plastic bag.

Mercury is also contained in electronic devices that use cold-cathode lamps for liquid crystal displays (LCDs). LCDs are used for backlighting in a multitude of products contained in the home: flat-screen computer monitors and televisions, cell phones, iPods©™, cameras, copiers and even in automobile dashboards (IMERC, 2006c). Most of the electronic products on the market today use LCDs because they are more energy efficient and can last longer due to the properties of metallic mercury. The amount of mercury used in a cold-cathode lamp is comparable to a typical CFL, but the potential for damage and release is less. Awareness of the presence of mercury is more a disposal concern for these common products.

4.1 Medical and Dental Uses of Mercury

In the scientific world, vacuums and pressures are often measured in terms of "inches mercury" to indicate the mercury level of a pressure gauge. Mercury-containing instruments, such as thermometers or sphygmomanometers (for blood pressure monitoring), and mercury dental amalgams were another source presenting incidental exposures to children as determined by the ATSDR (Besser, 2009) report. Children today are unlikely to receive mercury-based dental amalgams; however, they are likely present in adults including pregnant or breastfeeding mothers.

Mercury-based dental amalgams were commonly used to fill cavities in the recent past. There are now alternatives to mercury; however, the costs are higher so the use of

mercury-based amalgams has not significantly declined (Besser, 2009). The amalgams now come prepared in capsule form, reducing the use and storage of elemental mercury in the office. Amalgam capsules can contain up to 1,000 mg Hg, with up to 50% of the metal used in an amalgam being mercury (silver, tin, copper and zinc are other metals used – IMERC, 2008d). The higher percentage of mercury provides a soft malleable metal base that is easy to work into crevices. Up to 3-17 μg/day of mercury vapor (Hg^0) are released from the amalgams, in particular if a person grinds their teeth (Besser, 2009). A Swedish study (Ask et al., 2002) found mercury amalgams in women were a source of inorganic mercury (40%) as expected, yet also discovered methyl mercury was the primary species (60%) detected in the placenta and that the median concentration in cord blood was twice maternal blood levels. This study also found positive correlations with the amount of mercury in the body and the number of amalgams in the mouth. Dental amalgams are considered to be the primary source of inorganic mercury exposure to the general population, through oxidation of Hg^0 to Hg^{2+}. The Swedish study raises questions on further transformation to organic mercury, which can easily cross blood barriers. Inorganic mercury concentrates in the brain (if it can pass the barrier) and in the kidneys.

4.2 Mercury in Vaccines

Up until recently, childhood vaccines could contain an antibacterial preservative (thimerosal) that was approximately 50% mercury by weight. Significant controversy has arisen in the past decade between medical practitioners and families of autistic children as to the health impact from this injected form of mercury. The controversy of significantly

elevated mercury exposure to their children and the concurrent increased numbers of children diagnosed with autism since the late 1980s continues to rage, with CDC and FDA officials claiming there has not been "evidence of harm" based on neurological studies[13,14]. Each pediatric vaccine that contains the maximum thimerosal per dose contributes 25 μg mercury (see Appendix B for a list of thimerosal containing vaccines as of the 1999 recommendations). Most pediatric vaccines are 0.5 mL, although current recommended dose volumes for infants 6 months to less than 3 years old are 0.25 mL. For some children, up to 187.5 μg of mercury could have been injected during the first 6 months from routine vaccinations that contained thimerosal (Halsey, 1999; CDC, 1999; Baker, 2008).

Thimerosal has since been removed from most childhood vaccines, although some contain a trace, defined by the FDA as "detecting 1 μg mercury or less" (US FDA, 2007). Appendix B provides a list of recommended vaccines for children less than 6 years old, as of 2005, and the thimerosal content. Following the Institute of Medicine (Committee, 2000) recommendation to remove thimerosal from childhood vaccines as a means of reducing mercury exposures to the most sensitive populations, most vaccine manufacturers have supplied mercury-free or trace level doses for children. Insert materials, available upon request, contain information on the amount of mercury (if present) in a vaccine.

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13 Many of the autism studies only recognized DSM-IV classified autism, which does not include the more subtle neurological disorders of Pervasive Developmental Disorder nor Asperger's Syndrome. Additionally, children were assessed for autism only in the 0-3 year old ranges; PDD-NOS is often not diagnosed until speech or motor skill delays are pronounced (2-4 years old), and Asperger's Syndrome becomes apparent in the middle school years when social awkwardness is highlighted.

14 Thompson et al's (2007) study detecting slight neurological impairment with thimerosal containing vaccines, potentially attributable to chance, screened out 203 ICD-9 categories that included sensory, motor, neurological, and language disorders as well as pre-term or multiple births – children who would present a sensitive population for receiving high bolus mercury vaccinations.

Single-use vaccines did not require an antibacterial preservative – only multiple-dose vials required a preservative to ward off bacterial contamination from re-injection into the vial, thus not all shots had mercury. Today, children receiving vaccinations with trace levels of mercury are significantly less exposed than children in the 1990s: down to a maximum of 3 μg versus 62.5 μg mercury for a three-shot vaccination visit[15]. The current schedule of 28 recommended vaccines is also presented in Appendix B: Vaccines.

4.3 Other Mercury Sources

According to the Center for Disease Control and Prevention (CDC), the most common source of mercury exposures result primarily from mismanagement of mercury in a home: tracking contamination home from use at a workplace, or through inappropriate cleanup of broken materials containing mercury.

Mercury is used for electrical switches due to its metallic qualities (a good electron conductor) and its unusual covalent bonding that enhances its ability to vaporize (Lawson, 1999). In the United States, common uses of mercury in a home setting include green-initiative compact fluorescent light bulbs, light-up sneakers, dental amalgams, batteries and pressure gauges. Mercury vapors are also a part of several religious or cultural practices among Latin American or Caribbean immigrant communities. A *azogue* (elemental mercury) is carried in an amulet, typically worn around the neck, and used for sprinkling

15 At a RfD of 0.1 μg/kg body weight/day, a 10 pound baby (4.54 kilograms) is allowed a daily intake amount of 0.454 μg/day. Thus, 62.5 μg of ethyl mercury is about 138 times the RfD for methyl mercury. These concentrations represent an acute high dose, drawing the concerns of the AAP, which issued a statement in July 1999 encouraging removal of thimerosal from childhood vaccines as soon as possible. By 2001, thimerosal-free vaccines were required.

around a home (or vehicle) (Besser, 2009). Mercury has been used in thermometers, switches, lighting transformers, and as a bacterial agent in pharmaceuticals (in particular, skin-lightening ointments and eye drops/contact lens solution16).

An older home may present a few unusual sources of mercury, from paints to old natural gas regulators, and older thermostats. Before 1990, indoor latex paints could contain up to 300 parts per million (ppm) [17] phenyl mercury as a fungicide (Meier, 1990). In homes where old paint has been recently used or has been improperly sealed, mercury vapors could result from the paint volatilizing. Former natural gas regulators, which may be disconnected from use but still in the home, contained elemental mercury to create a seal. Natural gas for residential use became increasingly popular in the 1950s across most of the United States and up to 1961, these regulator units were installed inside homes (Besser, 2009). Thermostats are also potential sources of mercury in homes, some containing up to 4,000 mg Hg (IMERC, 2008b).

4.4 Regulating Mercury-Containing Products

When the products we use to improve our daily living no longer provide this service, waste is created. As mercury-containing products are used and discarded, such as

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16 A review of two local Saratoga Springs, NY store brands' (Price Chopper and Hannaford) contact lens solutions indicate they do not contain thimerosal but other preservatives. The Hannaford brand specifically states it does not contain thimerosal, as does Complete® contact lens solution. Bausch & Lomb's saline solution also states it does not use thimerosal, but it is also not a disinfectant.

17 A 4 year old boy was poisoned by mercury vapors emitted from recently used interior latex paint, found to contain 900 ppm Hg, three times the standard. In response, the USEPA banned mercury from use interior latex paints. Exterior paints continued to have mercury added as a fungicide.

fluorescent lamps, thermometers, thermostats, regulator switches, batteries and pressure gauges, the waste stream becomes a significant source of mercury emissions where this material is incinerated. In the United States, control of mercury emissions from these waste streams has been implemented through regulations. Incineration of mercury-containing products has been sharply curtailed through passage of state laws, and environmental effects are being demonstrated locally and regionally (NEIWPCC, 2007). Since incinerators contribute both to air and land (via fly-ash residues) comparable to coal-burning power plants, the global impacts can also be significant for waste incineration.

Recent improvements in technology and the regulations requiring reduced mercury in the environment have coalesced, resulting in manufacturers phasing out products that contain mercury, such as in thermostats (IMERC, 2008b). New York has promulgated regulations (S.1941, A. 8410) to prohibit use of gauges, manometers and fever thermometers that contain mercury (Chemicals Policy, 2009). They are already prohibited from disposal into household trash (IMERC, 2008b). Programmable thermostats are an alternative to using mercury. For residents of states that belong to the Interstate Mercury Education and Reduction Clearinghouse (IMERC)[18] consortium, recent models are less likely to have mercury in the flow regulators. As of 2006, some manufacturers, such as Whirlpool and Maytag, have removed mercury as a component in newer gas ranges. However, other manufactures (such as GE Appliances and Electrolux) have applied to IMERC states for a waiver allowing an extended time period to continue selling these products (IMERC, 2008a).

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18 The following states are part of this consortium for regulating mercury-containing products: California, Connecticut, Rhode Island, New Hampshire, Vermont, Massachusetts, Maine, New York, Louisiana, Minnesota, Washington, Illinois, New Jersey and North Carolina.

Organic mercury's ability to cause cellular damage was useful when it was used as a fungicide; however, increasing health concerns about mercury's similar impact on humans' nervous systems have led to mercury restrictions or bans. Following the Iraqi seed grain poisonings, where over 6,500 people were hospitalized and 459 people died (Baker, 2008; USEPA, 1997a), in 1976 the United States banned use of mercury as a pesticide (Meier, 1990). Phenyl mercury continued to be used as a fungicide in paints. By 1991, mercury use in interior paint was banned, and exterior paints were labeled, following a severe mercury poisoning incident of a child from mercury vapors from paint. Manufacturers voluntarily provided warning labels for unsold store stocks prior to the ban's effective date (Meier, 1990).

In 2006, the USEPA published its "Roadmap for Mercury", outlining its long-term goal of reducing mercury exposures. Mercury use in daily products could lead to exposures through improper use, breakage or improper waste disposal. Many leads in reducing mercury have been undertaken by associations, such as the National Electrical Manufacturers Association or regional groups, such as IMERC. The USEPA has established the "Mercury Challenge" to encourage partnerships with companies nationally to reduce use of mercury-containing products in its National Partnership for Environmental Priorities. The USEPA provides technical assistance in this partnership in understanding how to reduce mercury use. In July 2004, New York State issued its Mercury Products Prohibition law; the USEPA is drafting a similar law this year (NYSDEC, 2006; USEPA, 2009b).

A primary focus that New York State has taken is with outreach activities to educate people on the use of mercury in schools and in hospitals. Nine brochures were published in

2008 and target specific audiences, such as school nurses, superintendents, school boards, and custodial staff. They are distributed via health and environmental work groups, mailings, and outreach educational activities to highlight actions needed to properly address mercury sources in instruments used in these settings. A new manual is being prepared for county and public health departments and will included these brochures.

4.5 Regulating Dental Amalgams and Cosmetic Use of Mercury

The FDA regulates the use of preservatives in health care products under Code of Federal Regulations, Title 21, Part 700: Food, Drug and Cosmetic Act. The FDA did not require efficacy testing of drugs until the 1960s, nor was safety testing a component of approval for new drugs until 1938. Thimerosal, under the name Merthiolate, was patented in 1928, thereby bypassing today's regulatory reviews (Baker, 2008). The FDA Modernization Act of 1997 required an assessment of mercury content in all products regulated by the FDA (Baker, 2008). Recently, the FDA rescinded past opinions determining thimerosal-containing products should not be classified as drugs, such as one provided in 1939 (for topical applications) and another in 1944 (for mercury-containing cosmetics) unless the product meets specific requirements based on mercury content and no or limited non-mercury replacement options exist (eCFR, 2009). These materials are now considered drugs based on the known toxicity of mercury on the body system. Dental amalgams however, are still considered to be a "medical device".

The New England states, through NESCAUM coordination, have implemented state-level restrictions on discharge of dental amalgam wastes to the environment. In 2000,

Massachusetts followed an economic strategy of buying into a program early to save later – comparable to a college-funding operation. They offered dental offices the option of installing amalgam separators early, before regulations went into effect, and they would then be exempt from newer restrictions until 2007 or 2010, depending on when the separators were first installed. This proposal was surprisingly effective: up to 75% of dentists installed the separators and wastewater levels of mercury have significantly declined (King et al., 2008). This program has been a model for other states and even the federal government. As of January 2009, an agreement has been established between the USEPA, the American Dental Association (ADA) and the National Association of Clean Water Agencies to install dental amalgam separator units, as well as recycle amalgam wastes as a means of reducing mercury loading to the environment (USEPA, 2009b). In New York, dentists were required to install separators beginning in 2002 with existing dental offices to have separators by 2008 (NYSDEC, 2006).

Neither the ATSDR nor the FDA recommend people undergo procedures to remove all mercury-based dental amalgams as this removal process is likely to expose a person to more significant levels of mercury (Besser, 2009). The ADA's position is that removing fillings strictly due to the presence of mercury is unwarranted and that "continued use of dental amalgam as a restorative material does not pose a health hazard for the non-allergic patient" (ADA, 2007). This position has not been updated since 1986, although the health consequences of mercury exposure have undergone significant evolution towards concern about low level exposure damage to numerous body systems. In the New York Heavy Metals Registry, dentists are noted as being a commonly-reported occupation associated with mercury detections above guidelines (NYSDOH, 2006)..

In 1986, there were few studies on the effects of low-dosage, continuous exposures of mercury. Recent actions in 2008 on the part of the FDA include reopening a public comment period for a final rule on classifying dental amalgams as a medical device with special controls, based on concerns raised in legal, environmental and health circles on the safety of mercury amalgams (IMERC, 2008d, US FDA, 2008). In 2002, the rule was originally published as a proposed rule, but a joint committee met in September 2006 and found the literature research was sparse and recommendations for action were thus limited by the paucity of research evaluated at that point. Recommendations were made to increase the literature review to countries other than the United States, to study the pharmacokinetics of mercury, as well as recommend labeling changes to restrict use for pregnant women and children, and consider informed consent from patients (US FDA, 2006). The final ruling has not yet been published, but the FDA has invited researchers to provide information on exposure effects to both patients and dental professionals. Hopefully, their evaluation will consider the impact dental amalgams may have on the overall body burden of mercury relative to increased risks of neurological and developmental impacts on a fetus, not just on an adult.

Part II - Exposure Concerns

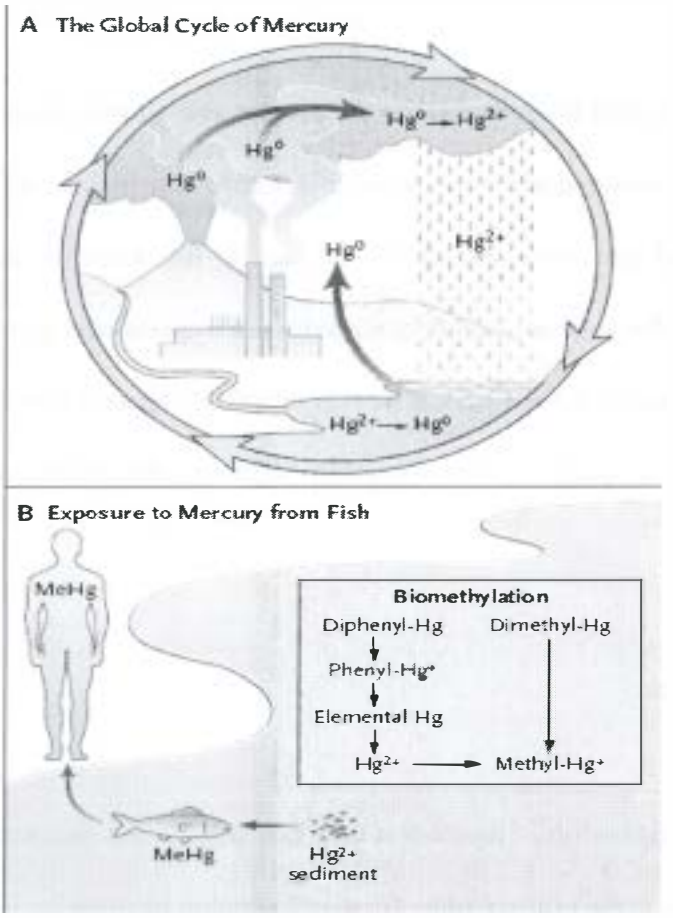
CHAPTER 5: BIOACCUMULATION OF MERCURY IN WILDLIFE

Fish has been heralded for centuries for the protein and nutrient sources it provides. Medical advisories have encouraged people to eat more fish, as a healthy alternative to red meat. At what level and over what period of time, eating fish presents subtle to severe neurological, cardiovascular, immunological or reproductive impairments is still under study. Bioaccumulation is a concern for the person or fish or wildlife species that eats many fish; but biomagnification is a quieter, more dangerous concern that many people do not understand well.

5.0 Pathway of exposure

Environmental deposition of mercury is only one stage in the mercury cycle (Figure 5). Atmospheric mercury is the primary route for contamination of water bodies and wildlife in remote areas, far from industrial mercury sources and even from natural sources. The Adirondack Mountains in Upstate New York are a prime example of a remote pristine area that demonstrates a severe problem with mercury levels in aquatic species and the wildlife that feeds upon them. Mercury levels in the water, even at low concentrations, present an environmental and public health threat by the biomagnification of this compound uptrophic levels in the food chain. End users include all piscivorous consumers, including humans. As a result, fish advisories are in effect across most of the United States. The USEPA's 2005

Figure 5: Mercury transformation in the atmosphere and biosphere
(Clarkson et al, 2003)



National Fish Tissue Study analyzed fish collected from every state in the nation for pollutants. Mercury was found in all fish sampled; it was also the most prevalent and most concentrated pollutant found.

Fish absorb mercury from contaminated water through their gills as well as through consumption of plankton and other smaller fish. Bioaccumulation occurs when a predator consumes numerous smaller species at the lower trophic level. Mercury has a high

bioaccumulation factor[19], thus a portion is retained in the tissues instead of being fully excreted by the different body systems. With consumption of increasing numbers of smaller species over time, mercury bioaccumulates; as these fish are consumed by larger species, the mercury biomagnifies by orders of magnitude up to the next trophic level.

Table 2: Biomagnification of mercury

Trophic level	Biomagnification Factor
Water	1
Phytoplankton	10,000
Zooplankton Plant-Eating Fish	100,000
Piscivorous (Predator) Fish	1,000,000
Piscivorous (Predator) Birds/Game	10,000,000

(data from HBRF, 2007)

The biomagnification pathway demonstrates how critical it is to understand the effects of mercury pollution for assessing environmental and public health responses to this contaminant. Researchers are studying the environment from a multitude of perspectives, from Hall (1995) evaluating the effects of ozone, a pervasive air pollutant, on mercury transformation to more soluble mercury in the atmosphere, to Evers (2005) and the Hubbard Brook Research Foundation consortium of scientists who are evaluating the environmental end point impacts on loons from mercury. A field study conducted by this author found strong correlations of air concentrations of mercury with pollution influences, such as ozone, as well as weather influences such as increased levels on sunny afternoons and reduced Hg^0 levels following rain events (McLelland, 2008). A conclusion from this study was that a paucity of data is available in New York State from air monitoring that can

19 Bioaccumulation factors (BAFs) compare the ambient concentration of a chemical to the levels found in biotic tissues. Monson & Brezonik (1998) reference a logarithmic BAF of 4.90-5.43 for methyl mercury in zooplankton, indicating approximately 10,000 times the level found in water

allow for correlations of Hg^0 levels with concentrations of reactive air pollutants. The majority of data published involves mercury precipitation concentrations, not air concentrations of elemental mercury that can provide additional insight on specific conditions that may oxidize the Hg^0 for eventual deposition as Hg^{2+}-laded rain.

5.1 Biological "Hot Spots"

Mercury levels in the Northeast have been found to be elevated in more than just fish species, indicating the air pollution levels can present more impacts than had been considered only a few years ago. Studies conducted by the Hubbard Brook Research Foundation have assessed biological "hot spots", defined as "areas with two or more species that had mercury levels above known thresholds for adverse effects" (Daley, 2005). Elevated mercury levels have been detected in mink, otter, salamanders, eagles, songbirds and loons, indicating that fish consumption alone is not the only mode of transport (Evers, 2005). Insects must also present a concern for biomagnification of mercury transfer up trophic levels, as non-piscivorous birds are also reflecting elevated mercury levels.

Loons are considered a representative environmental indicator for wildlife impacts, based on their longevity and territorial behaviors, in addition to their preference for similar fish species (perch) as anglers. New England loons, hatching 40% fewer young in loons with higher mercury levels, support concerns about wildlife reproductive impacts (Evers, 2005; Daley, 2005). Researchers are finding that the levels of mercury in the blood and feathers of loons in the Adirondack Mountains and Maritime Province of Canada are high enough to present physiobehavioral impacts.

Loons in "biological hot spots" such as the Adirondacks and Nova Scotia (Figure 2) have significantly higher mercury levels in their blood, feathers and eggs than other regions studied. With high blood Hg levels, researchers are finding idiosyncratic behaviors such as excessive grooming, erratic flapping of wings, weakening wing and legs, and difficulty coordinating muscle movement– all of which can impair the loon's mating ability (Evers, 2005; NWF, 2006c). Reproductive impacts may begin with reduced or impaired mating patterns from idiosyncratic behaviors, followed by egg damage from mercury. The elevated levels of mercury in the eggs may be affecting the viability of these hatchlings.

The patterns observed in wildlife need to be a concern to humans, who also consume the same fish species. Humans excrete mercury through the hair in much similar fashion as loons depurate mercury into their feathers. Of special concern is the potential for generational impacts from maternal consumption to the developing fetus; methyl mercury has been found to transfer into loon eggs, and is known to migrate through the placental barrier to the human fetus. Mercury transfers into eggs, damaging their viability. This raises the question of preferential mercury transfer from mother to fetus, as seen with congenital Minamata Disease - where severe impacts occurred in the fetus than with the actual consumer.

CHAPTER 6: TOXICOLOGY OF MERCURY: HUMAN IMPACTS

6.0 Mad Hatter's Disease

Lewis Carroll memorialized the common phrase "Mad as a Hatter"[20] in his book *Alice in Wonderland.* Hatters were commonly afflicted with symptoms often mistaken for

20 The phrase was used in England in the 1830s. Mercurial poisoning impacts of hatters in Newark, New Jersey was initially described by Dr. J. Addison Freeman in a medical journal published

alcoholism: drooling, tremors, memory lapses, mood swings, visual and auditory hallucinations, distorted vision and slurred speech. Dementia and erethism[21] affected hatmakers in the late 17th and 18th Centuries, due to high exposures to mercuric compounds. Mercury use in the hat industry was prevalent in New Jersey and especially in Danbury, CT -the hat capital of the world in the 19th Century (Sawicki, 2004; Weeden, 1989; Corrosion Doctors, 2008). The terms "hatter's shakes" (more specifically, "Danbury Shakes") were used in describing the severe tremors typical of people who worked in this industry. Mercuric nitrate was used in the felting industry, resulting in high levels of mercury vapor exposure from the generated steam (Hatters at Orange NJ, 2009; Corrosion Doctors, 2009). Eventually, in 1941, the United States Public Health Service banned its use due to its toxic nature and a refocused use for mercury in detonators (Weeden, 1989; Wikipedia, 2009). At that time, exposure to mercury was considered an occupational hazard, not something that was likely to afflict the general public. The first reported exposures from consumption of contaminated fish occurred in Sweden in the early 1950s (Myers, 2004), but widespread concern over contaminated food arose only after W.E.Smith's photographs appeared in Life magazine in 1971 (see Figure 4: Tomoko in her bath).

6.1 Heavy metal poisoning in humans

Awareness of environmental toxins has come at a price: often following crippling disasters like Minamata. Historically, environmental regulations have been reactionary, not proactive. Today's use of reference doses (RfDs) and minimum risk levels (MRLs) are based

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in 1860 in *Transactions of the Medical Society of New Jersey*. An article "Mad as a Hatter" was published in the English magazine, Punch, in 1862; *Alice in Wonderland* was published in 1865 (Restak, 2006).

21 Erethism effects are part of Minamata Disease, Mad Hatter's Syndrome, mercury poisoning, metal fume fever (USEPA, 1997b; Corrosion Doctors, 2009) and reflects bizarre mood swings and behaviors

on lowered values below observed toxic effects, where effects are not expected to occur. They are defined with knowledge that the science may not be exact. With mercury, as with lead, there is still uncertainty of where, and if, a threshold level occurs. Threshold levels define a concentration below which no adverse effects are known to occur. Toxicology is an uncertain science, with adverse effects derived mainly from accidents. An accident helps in revising the toxicological database when scientists and medical doctors assess what happened at what concentrations. Unfortunately, this information can often only be used in establishing nebulous baselines of effects, due to limited exposure information and/or use of different species in assessing biological responses.

Defining lower risk of health effects or No Observed Adverse Effect Levels (NOAELs) can be complex with environmental contaminants. For gross accidental exposures, the impact is typically acute but short termed, yet severe in concentration and effect. The complexity compounds when there are lower levels of exposures over a longer time period, especially for environmental contaminants. Compounding or synergistic effects of mercury with other environmental pollutants may cloud the ability of scientists to assess the levels of a singular pollutant that present symptomatically. In defining Minamata disease initially, effects were confused with possible contribution from other contaminants found. Lower concentration exposures to a single pollutant or mixture of similar pollutants, that are more chronic and long-term in nature present challenges to the toxicologist when assessing where a threshold level for health effects has been reached. Lead, a heavy metal similar in its systemic impacts as mercury, has been extensively researched over decades, with the current understanding assuming a threshold level may not exist. Neurological effects continue to be

demonstrated at very low blood levels and over time, as bioaccumulated lead leaches back into the system. This may be the situation with mercury toxicity.

The toxic impairment of the body system from exposure to lead is comparable to mercury, a similar, pervasively used heavy metal with no biological demand. Thus, these compounds are true pollutants to our system, unlike other trace metals like selenium that in proper concentrations can be beneficial to human health. If public policy fails to address the lessons learned from lead poisoning when assessing human threshold levels for mercury, a repeat of neurological damage to entire populations is likely. Lead levels once considered "safe" for children in the 1960s, 60 ug/dl, today require an adult worker to be removed from the exposure for medical reasons (ATSDR, 2007). Historically, actions occurred only at symptomatic effect levels (>60 ug/dl), but with ensuing research on the subtleties of neurological damage, action levels have been lowered dramatically (six fold or greater) to reflect emerging science. Current CDC guidelines provide for mitigative actions at 10 ug/dl blood lead levels; however, they also acknowledge that sensitive populations may be affected at levels lower than the 10 ug/dl action level. No threshold has been established for lead.

6. 2 Minamata and Niiagata Japan – mercury contamination of fish

Methyl mercury as a neurotoxin was highlighted by birth defects and severe health impacts, including death, that resulted from consumption of mercury-contaminated foods, such as fish in Japan in the 1950s and 1960s and wheat grain in Iraq in the 1970s. Methyl mercury toxicity has been closely studied since the 1959 determination that "Minamata disease" resulted from mercury poisoning from consuming local fish caught in Shiranui Sea. The Chisson Corporation had discharged mercury into Minamata Bay as an operational

waste from the production of acetaldehyde (used in plastic production) where mercury was used as a primary catalyst. Both organic and inorganic mercury wastes were directly discharged. At the time, it was not known that the inorganic mercury wastes could be transformed by the anaerobic bacteria in the sediments into the more toxic organomercury form, (mono)methyl[22] mercury. Cats were the first indication of a problem: feral cats, who fed primarily on fish and fish scraps, developed neurological and psychomotor impairments, often leading to their deaths. The "crazy dancing cats" only preordained the impact on people, primarily families of fishermen. By 1956, people were being hospitalized with severe neurological seizures leading to coma and death. Infants exposed to high prenatal levels of mercury died[23] from congenital mercury poisoning effects or demonstrated severe neurological damage, such as cerebral palsy and mental retardation (Newland et al, 2008). These conditions were significant between the 1956-1959 time period. Neurological and sensorimotor deficits were the most pronounced affects from contaminated fish consumption. Of public health concern was the associated impacts seen with newborns and small children, who did not eat the fish but whose mothers did. As seen in the famous photo taken by W.E. Smith (Figure 6), the most severe damage occurred to the fetus, with significantly less damage to the mother that consumed the contaminated fish.

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22 Monomethylmercury is the species discussed as methyl mercury in this paper; however, dimethyl mercury is a more toxic organic mercury compound. A Dartmouth professor had been working with dimethylmercury, using latex gloves, and spilled some mercury. The gloves were inadequate to prevent dermal absorption and the concentrations were significant enough to cause severe neurological (sensory and motor) impairment and eventual death. The symptoms read as if she had "hatter's shakes", including the auditory and visual disturbances.

23 Five infants that died from mercury poisoning during this period were autopsied, with brain concentrations of mercury ranging from 0.4 to 15.4 parts per million (Newland et al, 2008).

Figure 6: "Tomoko in her bath": W.E.Smith's photodocumentation of the toxic effects of Minamata Disease (Source: http://www.photobookguide.com/review/w-eugene-smith/minamata/)

A second event of mercury-poisoning from fish consumption occurred in the mid-1960s in another waterway, in Niigata. Fishermen working the Agano River in the late 1950s to mid-1960s, downstream of the Showa Denka acetaldehyde plant, developed severe symptoms that doctors recognized as Minamata disease. Sensory impairment was most prevalent in this epidemic, with most victims reflecting vision damage (Urasaki et al, 2004). Early diagnosis lead to exposure testing, which did not occur in Minamata due to the uncertainty of the "source" of the health effects[24]. Coincidentally, in 1963 cerebral palsy was noted as being common in the same areas by researchers at Kumamoto University, investigators of Minamata Disease for the government (Urasaki et al, 2004; Ui, 1992). Professor Kitamura linked this birth defect to methyl mercury poisoning – by 1965, the Niigata poisoning impacts were also linked to mercury fish consumption (Urasaki et al, 2004). The significance of the cerebral palsy "cluster" findings was that prenatal exposures alone could be irreversibly damaging to the fetus. Pregnant women were tested for mercury

24 Elevated levels of other metals, such as arsenic, copper, lead, zinc, manganese and iron were also found in the sediments of Minamata Bay, leading researchers to assess other contaminants prior to determining methyl mercury as the primary toxic agent (Myers, 2004).

in their hair, and were offered termination if levels were greater than 50 ppm[25] (Saito et al, 2004).

Minamata disease, exemplified by acute exposures, presented severe neurological damage: sensory system dysfunction with hypo- and hypersensitivity of extremities (fingers, lips, toes), auditory disturbances and constriction of the visual field (tunnel vision); gross motor dysfunction with tremors and ataxia; as well as fine motor dysfunction with dysarthia. Parathesia was prevalent among all adult victims, yet was not considered an "effect" that was officially recognized[26]. As the populations aged, the numbers of people diagnosed with Minamata disease increased (Hasada, 1995). This long-term development of associated problems heralded more foreboding concerns: the potential for developmental exposure to present effects later in life. Current research is assessing if prenatal exposure can impact the body systems later (Rice & Barone, 2000; Urasaki et al, 2004; Saito et al, 2004, Myers et al, 2004).

6.3 Iraq: mercury contamination of grain

The Niigata poisoning event introduced the concept of significant adverse prenatal impacts to a developing fetus, prior to the thalidomide birth defect devastation in the United States and England in the late 1960s. In Iraq, on the heels of the Niigata and thalidomide poisonings, wheat grain was treated with a mercury fungicide. The treated grain

25 Thirteen babies were born of mothers with more than 50 ppm mercury in hair samples (one had 293 ppm Hg), and were later evaluated for neurological and sensory motor impairment outcomes as adults, with four demonstrating congenital mercury poisoning from prenatal exposure (Saito et al, 2004).

26 The Minamata disease outbreak occurred during the economic recovery period in Japan from World War II, thus the political clout exerted by a major industry created roadblocks in approving "damages". Chisson Corp. Ltd provided compensatory damages to the fishing industry.

was inadvertently used in making bread. In this poisoning event, in 1972, infants and young children were the most impacted, with the adults significantly less impaired, although it was the mothers who ate the contaminated food. Effects did not appear for months after the wheat was consumed, yet over 6500 people eventually were hospitalized and over 450 people died. Even in Niigata, mothers with significant hair concentrations of mercury (>50 ppm) gave birth to children with no observed impacts, even over time, although other children were adversely affected where maternal hair levels were lower (to 20 ppm) (Urasaki et al, 2004). These differences awoke public health officials and scientists to the strong potential for gestational impacts from organomercury contamination and the uncertainty of sensitivity of individuals to pollutants. Prenatal exposures in Iraq resulted in large scale impacts, leading to research on the toxicity of mercury and the transfer of this lipophilic compound across placental and blood-brain barriers. The knowledge that a pollutant can impair the development of a fetus from the mother's consumption habits has since been a pillar of public health awareness campaigns, such as with smoking, drinking alcohol and using drugs.

6.4 Mercury Body Burden

The totality of exposures is needed to answer if mercury causes adverse health outcomes, especially as it has a high bioaccumulation factor. The question of exposure is not just incidental contact, but how much has entered the body, and by what route. Additionally, one needs to assess how long the exposure occurred for – if it was an acute or chronic exposure- and how much was the dose. Bioaccumulation refers to an increasing concentration of a pollutant within a body system over time. When a pollutant, such as

mercury or lead, cannot be excreted fully, but is retained in muscle or brain tissue or within bones, then repeated exposures to that pollutant will result in increasing levels in the body. This is referred to as "body burden". With mercury, even low level exposures that are repeated, can cause harm. It is the cumulative nature of mercury exposure that warrants the most concern in the United States. Societal impacts may be most pronounced because low levels in isolation can pose concerns for long-term effects for individuals, yet it is the subtle impairments that occur broadly with low levels, decreasing cognitive function among an exposed population.

As mercury cycles through the blood system when inhaled or ingested, there are multiple endpoints in the system that can be impaired by accumulating concentrations of mercury. The different forms of mercury that a person can be exposed to will affect the body system differently: elemental (metallic) mercury vapor is accumulated by the kidneys, whereas organic mercury targets the central nervous system. Mercury interacts with (sulfur-bearing) proteins, and the body's response system will produce auto-antibodies in response to these altered proteins – resulting in immune system dysfunction with mercury exposures (AMA, 2004; Duncan, 2009; Clarkson et al., 2003; Rice & Barone, 2000, ATSDR, 2002). The cardiovascular system is another targeted system, with hypertension a primary response to elevated mercury levels. Studies of children conducted years following prenatal exposure to mercury found blood pressure effects, but additional studies were recommended (Thurston et al., 2007). The AMA (2004) reports causal concern for mercury exposures and cardiac health impacts, especially among men (Burros, 2008).

6.5 Impacts to a developing brain

The population most at risk for long-term damage from mercury exposures is the developing fetus. The fetal brain is most susceptible to damage during the development process from chemicals that can alter cellular response or change the critical biochemical organization of an organ system. The fetal brain is 10 times more sensitive than an adult brain to nervous system impairments (Halsey & Goldman, 2001), and the Minamata, Niigata and Iraq poisonings demonstrated the fetus was significantly damaged at levels that did not impair the mothers (Harada, 1995). Disruption of the highly orchestrated neural and cellular development system can lead to significant damage if exposures occur during critical periods. Exposure following system development presents damage differently, affecting the ability to perform normally.

Toxicological studies often use rodents that, in the case of mercury, could be misleading. Human fetal development of the neural system occurs primarily during the pre-natal period, unlike rodents where post-natal development spikes for the mature neurological system (Rice & Barone, 2000). As such, epidemiological studies are important in assessing levels that may present risks and those that could present health effects.

CHAPTER 7: EPIDEMIOLOGICAL STUDIES

Three critical epidemiological studies have assessed methyl mercury exposures: the Seychelles Child Development Program (off Eastern Africa), the Faeroe Islands (near Iceland) and in New Zealand. In each of these three longitudinal studies, methyl mercury exposures occurred through marine fish/mammal consumption and parent-child cohort

studies were evaluated to assess the risks to adult and developing child. In the United States, fish consumption typically is lower than for these populations studied.

7.0 Seychelles versus Faeroe Islands and New Zealand Studies

In determining appropriate regulatory levels in the United States, the three major epidemiological studies conducted in the 1980-1990s were assessed: Faeroe Islands, Seychelles Child Study Program and New Zealand. In all three studies, maternal/child pairs were followed from delivery to age seven in a longitudinal study format. Two of the studies, the Faeroe Island and New Zealand, found adverse neurological effects. The Seychelles study initially did not find neurological effects, but suggested potential cardiovascular effects as the children aged. Cardiovascular health effects have been noted in studies done on fish-eating Finnish men, and current research is assessing the emerging connection between methyl mercury and heart disease (Charnley, 2006; Burros, 2008). Later studies have determined some subtle neurological impairment from the Seychelles cohort (Myers et al., 2003, 2009), supporting the overall response of these studies – neurological impairment is demonstrated in prenatal and postnatal exposures to methyl mercury.

Most exposures today occur from eating mercury-contaminated fish. The source of methyl mercury varied between the three epidemiological studies: in the Seychelles, women ate marine fish containing mercury at levels (0.3 ppm) comparable to the mean freshwater fish levels in the United States (Myers et al, 2003; USEPA, 1997a); in the Faeroe Islands, whale meat was the source - which may not contain high levels of selenium[27] when compared to fish; and, in the New Zealand study, the primary fish was shark. In New

27 Selenium may offer limited counteracting benefits to the neurological system, as recent studies are evaluating (Choi et al, 2008; Myers et al, 2004).

Zealand, at the lowest adverse effect levels, language developmental disorders were noted. A benchmark does of 17 ppm mercury (in maternal hair) was found in this cohort to correspond to no adverse effects (Gearhart et al, 1995). At this NOAEL, Gearhart et al (1995) conducted pharmacokinetic modeling to extrapolate fetal brain levels of methyl mercury levels (up to 50 parts per billion, or ppb) which also corresponded to a maternal mercury consumption level of 0.8-2.5 micrograms per kilogram body weight per day (ug/kg/d). In 1995, the USEPA reference dose (RfD) was 0.3 ug/kg/d. Grandjean et al. (1998) found hair levels of methyl mercury in this "safe" range of 10-20 ppm to yield subtle neurological impairments, supporting a lowered NOAEL. The USEPA has since decreased its RfD to 0.1 ug/kg/d.

With biomagnification of mercury contamination resulting from increasing trophic level consumption, it is the larger species of fish (and marine mammals) that present the greatest concern (Simonin et al, 2008; USDHHS, 2004; USEPA, 2005). Shark is listed in the United States as a commercial fish that should not be consumed by pregnant or breast-feeding women (USDHHS, 2004). Whale meat is not common to sustenance whalers in Alaska. The typical commercial fish diet in the United States is more most Americans, however, can reflect the exposures expected by the Inuit and other closely paralleled with the Seychelles study; although the American-based neurological tests used in assessing impact may have been biased against this non-English speaking population. A biomarker is a screening analytical tool used in collecting data on body burdens for contaminants. Current epidemiological investigations into mercury exposures look to cord blood as the most accurate biomarker of methyl mercury levels in the human system. Earlier investigations in the 1990s, such as the Seychelles Child Study Program, assessed mercury levels in maternal hair to extrapolate the potential levels available to the fetus. It had been presumed that levels

in hair correlated closely with levels found in the infant. Yet, Grandjean (1998) in studying the mother-child cohorts from the Faeroe Islands found the ratio of newborn cord blood to maternal blood to be higher, by 70%, thus a 1.7 multiplier may be more accurate. For testing purposes, urine is often used as a biomarker for contaminant excretion; however, it has not been found to be an accurate evaluation of methyl mercury.

7.1 NHANES – Biomarker Results from US populations

In 1999-2000, the Centers for Disease Control and Prevention (CDC) conducted its first National Health and Nutrition Examination Survey (NHANES), an assessment of hair and blood mercury levels from American women and children (CDC, 2001). These levels established an initial baseline of mercury exposures for the general United States population, with an understanding that some subset populations, such as anglers and people living along the East Coast[28], may consume more fish that could elevate mercury body burdens. The NHANES dataset for 1999-2000 was small in scale (approximately 2400 people) and only evaluated 12 locales, implying the levels may not be truly reflective of a national exposure baseline (CDC, 2001). Blood mercury levels globally were assessed by the World Health Organization as averaging 8 ppb, with hair levels approximately 2 ppm (USEPA, 1997a). The 1999-2000 NHANES dataset found most women did not have elevated blood mercury or hair mercury levels. The mean blood mercury levels for women are 1.02 ppb, four times greater than the mean for children 1-5 years old (0.3 ppb) (CDC, 2001). However, Park & Johnson (2006) found fish-eating women had levels seven-fold

28 A study in 2005 by the Sierra Club and Greenpeace found New York State women of childbearing ages had more than twice the mercury levels of nationwide averages, with 46% of New York City women detecting over 1 ppm of mercury in their hair (average level was 1.4 ppm) (Pearson, 2006)

higher (0.59 μg/g, or ppm) than women who had eaten no fish over a 3 month period (0.08 ppm). Establishing a "safe" level has been difficult as additional research disproves earlier findings of threshold levels. In 1985, the idea of a threshold for mercury was beginning to be questioned, as was the increased sensitivity of the fetus and pregnant mother (Inskip & Piotrowski, 1985).

7.2 Thimerosal exposures to Ethyl Mercury

Ethyl mercury, the mercury metabolite of thimerosal, can be found in eye drop solutions as a preservative. Recent research, using *in vitro* testing, found that many preservatives, such as thimerosal, are toxic - causing ocular damage at the cellular level (Epstein et al, 2009). Ethyl mercury has been found to metabolize to methyl mercury in a body system. Methyl mercury health impacts include vision constriction by damaging the neurons in the brain, and is a common effect of mercury poisoning. Epstein et al.'s (2009) tests of common preservatives, at the levels found in these eye solutions, found thimerosal to be the most toxic and concluded for all tested preservatives that "even at low concentrations, these agents will cause some degree of ocular tissue damage". The toxicity of thimerosal in topical medicines was assessed by the FDA in the 1980s, with restrictions initiated in 1998 (Silbergeld, 2008). Thimerosal additives were restricted in Europe in the 1990s (Halsey, 1999; Baker, 2008), due to concerns about human toxicity.

With the increased number of recommended vaccinations by the late 1980s, the number of shots a child received in one day also increased. Of concern with the multiple vaccinations are the potential spikes of mercury in the body burden from intermittent and bolus injections - a concern raised by the American Academy of Pediatricians (AAP) as the guidelines did not consider these exposures when setting reference dose levels. In 1999, the

AAP and the Public Health Services agencies (FDA, CDC, ATSDR) recommended thimerosal be removed from childhood vaccines, which was shortly followed by a supporting statement by the American Association of Family Physicians(CDC, 1999b). By 2000, the Institute of Medicine (IOM), a private scientific group assessing vaccine safety for the National Academy of Science, supported removing thimerosal from vaccines29 - although did not support a causal connection between thimerosal and adverse neurological or immunological effects (Stratton et al, 2001). Manufacturers were encouraged to increase production of non-mercury containing vaccines, which had been the initial difficulty in providing non-mercury vaccines to the general public following the 1999 and 2000 recommendations. If a non-mercury containing vaccine was not available due to limited supplies, the most sensitive populations were encourage to receive the mercury-containing vaccine rather than risk complications from the influenza virus (AAP, 1999). The final lots manufactured with thimerosal with up to 25 μg ethyl mercury had an expiration date of January 2003, according to the National Network for Immunization Information website (www.immunizationinfo.org).

Methyl mercury is lipophilic and this characteristic allows it to cross the placenta and undeveloped blood-brain barriers, resulting in mercury accumulation in developing brains (EPA, 1997b). For young children with an undeveloped blood-brain barrier, mercury-containing vaccines (primarily the flu vaccine today) can add to the body burden of mercury by allowing all forms of mercury from a multitude of sources, to enter brain tissues. The barrier restricts passage of inorganic mercury into the brain; although organic mercury passes easily. Organic mercury can be oxidized in the brain into inorganic mercury, which

29 The IOM issued a report in 2000, recommending thimerosal be eliminated from vaccines as of 2001; however, not all vaccines were mercury-free as of 2001. The influenza vaccine, recommended for children and pregnant women, can contain up to 25 ug of mercury.

cannot pass beyond this barrier, resulting in accumulation in brain tissues. A developed blood-brain barrier prevents inorganic mercury into the brain, but cannot prevent oxidized organic mercury accumulations, thus even adults are susceptible to mercury bioaccumulation impacts.

For children who received the highest mercury-containing vaccines, up to 187.5 μg of mercury could have been injected into their systems by their 6 month visit, exceeding the USEPA reference dose (AAP, 1999; CDC, 1999b; Baker, 2008). As mercury is slowly cleared from a body system30, vaccination schedules only 60 days apart could create additional overloads of mercury as new mercury is added before the prior exposure has been cleared. For the youngest infants, these exposures could be significant for the potential impact to the neurological system and their developmental progression over time. A concern raised by the AAP (1999) was the guidelines had not been devised considering bolus or intermittent exposures, such as received with vaccines. An emerging area of study associated with mercury exposure includes the long-term impact of early mercury exposure during the critical development periods of neurological growth, up to adolescence. Additionally, some researchers are assessing the aging process impacts from mercury exposures, where damage during development may become more apparent as the body system ages (Weil et al, 2005). Understanding how mercury impacts the developing brain and at what levels can present damage is the focus of many epidemiological studies and animal research. Currently, few studies are available on ethyl mercury and its pharmacokinetics in an infant's system.

As of July 2008, only trace levels of thimerosal are allowed in vaccines given to children under the age of 3 and to pregnant women in New York State. Informed consent

30 Some data indicates ethyl mercury may clear faster in children than adults, with a half-life of 7-10 days versus 20 for adults (Clarkson et al, 2003; Pinchichero et al, 2002).

from the child's guardian or woman receiving the thimerosal-containing vaccine is required if limited supplies of a vaccine impedes this requirement, according to New York State Public Health Law 2112. This follows the "Precautionary Principle" of acting in caution considering the potential harm that could be done if actions were not taken.

CHAPTER 8: FISH CONSUMPTION ADVISORIES

Oh mercy mercy me
Oh things ain't what they used to be
Oil wasted on the oceans and upon our seas
Fish full of mercury.

- Marvin Gaye, "Mercy, Mercy Me (the Ecology)" (1971)

Most fish consumed in the United States are purchased from a commercial fish market, such as the grocery store or in a restaurant; wild freshwater fish comprise only about 10% of average fish consumption (Charnley, 2006). The majority of fish advisories issued address state level concerns with sport fish caught in freshwater environments, although a regional coastal fish advisory is being considered by East Coast states for bluefish.

8.0 Fish Advisories

In 2001, the FDA issued its fish advisory addressing mercury in commercial fish, recommending pregnant women not eat any of four specific fish: shark, tilefish, swordfish and king mackerel. Following issuance of this advisory, Oken et al. (2003) found pregnant women did reduce the amount of fish consumed, demonstrating the effectiveness of a multi-faceted approach of outreach to health professionals and the media[31]. However, as of 2000, the American Medical Association (AMA) has promoted consuming one to two servings of fish a week as a means of reducing the risk of coronary heart disease. These conflicting messages have harmed the effectiveness of either advisory by creating confusion. The ensuing concerns raised by many (health professionals, state regulators, general public) reflected uncertainty in the intended message.

In response, the EPA and FDA issued a joint advisory on March 19, 2004 to clarify consumption recommendations for both recreational and commercial fish species (USDHHS, 2004). However, the target did not deviate from protecting the most sensitive population: the fetus. Breastfeeding women were specifically added from the earlier version to address continuing exposure to infants during the postnatal period. The advisory specified that both recreational sport fish and commercial fish were included in consumption recommendations, and the recommendation was not to eat less fish overall, but to choose fish with lower mercury levels[32] The revised advisory included canned[33] albacore (white)

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31 Decreases in hair mercury levels in Faeroese women also resulted following distribution of an advisory letter to all women between the ages 26-30 years, supporting an effective campaign (Weihe et al., 2005).

32 Mercury has been found at trace levels in all fish tissue sampled by the 2005 USEPA Fish Tissue Study, a response reflected by states individual testing data. It is unlikely there are "no mercury" species.

33 27% of seafood consumed in the US is canned tuna fish (AFS, 2002).

tuna and light tuna. Light tuna is typically made with skipjack, a smaller tuna species that is lower in mercury content (Consumer Reports, 2009).

8.1 What is a "Safe" Exposure Level for Mercury?

"Alle Ding sind Gift und nichts ohn Gift; alein die Dosis macht das ein Ding kein Gift ist"
-Paracelsus, 16th Century Alchemist
["All things are poison and not without poison; only the dose makes a thing not a poison"]

With different federal programs providing different numbers to describe a "safe" level for methyl mercury (see Table 4), the message to the public about what to eat and how much was "safe" was poorly synthesized. Congress requested that the National Academy of Science evaluate the appropriate guidance level. The National Research Council (NRC) formed the Committee on the Toxicological Effects of Methylmercury to make this assessment. In 2000, the NRC Committee determined that the EPA reference dose (0.1 μg/kg/d mercury intake) was most appropriate to minimize risks of neurological impairment from methyl mercury (Stratton et al., 2001; Halsey and Goldman, 2001; AFS, 2002). This level used the Faeroe Island study, which found subtle neurological impairments at much lower levels than the Minamata or Iraq databases, which were mainly driven by symptomatic effects. The Faeroe Island study used blood levels of mercury as the biomarker, thus could be directly correlated to the NHANES dataset recently completed. Blood levels have been found to be a more representative biomarker of recent mercury exposure than hair levels, which could be imprecise and variable among different aged populations (Budtz-Jorgensen et al, 2004; Grandjean, 1998; Weil et al, 2005).The EPA has concluded from the mercury studies conducted that:

> "there is no safe level of methylmercury in the blood within the range of exposures measured in the human studies of health effects of mercury, which were as low as 1 part per billion. About 50 percent of the women of child-bearing age in the United States have at least 1 part per billion of mercury in their blood" (EPA, 2003, p.61).

The initial NHANES 1999-2000 data, limited in scale, found 7.8% of the women studied had blood levels above the RfD equivalent (5.8 μg/L in blood). Significant uncertainty exists relative to prenatal exposure at low levels for long-term health impacts, but new findings indicate adverse effects may be permanent when exposure occurs during development.

Table 3: Mercury concentration guidance values

Mercury Exposure Levels *		
Concentration $_{\text{biomarker}}$	Primary Route of Exposure	Regulatory Agency
58 μg/L $_{\text{blood}}$	Ingestion	USEPA benchmark dose Level (BMDL) – adverse effects noted
50 ppm $_{\text{hair}}$	Ingestion/Inhalation	USEPA benchmark dose Level (BMDL) – adverse effects noted
5.8 μg/L $_{\text{blood}}$ (17 ppm $_{\text{hair}}$) 0.1 μg /kg/day	Ingestion	NRC/USEPA Reference Dose (RfD)
0.3 μg/m^3	Inhalation	USEPA Reference Concentration (RfC) for inorganic mercury

1 μg/g $_{\text{fish tissue}}$	Ingestion	USEPA Fish tissue guidance** FDA Acceptable Daily Intake (ADI) for commercial fish
0.3 mg/L (ppm)	N/a	USEPA water quality criterion
0.2 μg/m^3	Inhalation	ATSDR Minimum Risk Level (MRL) (chronic) for mercury
0.3 μg/kg/d	Ingestion	ATSDR MRL (chronic) for methylmercury

*The most protective levels are noted on this table. The World Health Organization recommends an ingestion limit of 0.47 μg/kg/d, almost five times the EPA reference dose level. The FDA level for ingestion, established in 1978, allows for up to 0.4 μg/kg/d, four times the EPA reference dose.

** Most of the ingestion levels for fish assume only the noted species is eaten and does not reflect consumption of other fish species nor recognizes other contributory levels of mercury from other sources and/or routes of exposures (ie vaccines, school/home exposures, religious exposures).

8.2 At Risk Sub-Populations

Overall, the United States general population does not reflect significant mercury levels in blood or hair compared to populations who have been exposed to high levels of mercury contamination in their food, such as in Japan or Iraq. However, for those sub-populations that eat several fish meals a week, the increase in mercury levels in blood can exceed levels where neurological impairments have been noted (AFS, 2002). Individual physiological and genetic differences between populations and ages can result in particular sub-populations that are more at risk for mercury toxicity than the general population. Genetic susceptibility is an area of recent research in understanding metal toxicity. As some sub-populations' blood levels are significantly higher than the general mean, an assessment of their exposures should be carefully evaluated. Women of child-bearing age who consume both commercial and sport fish present the greatest risk group, as the most sensitive indicator of mercury toxicity is the developmental effects on the neurological system. The

target population for the FDA/EPA Joint advisory (2004) was thus the fetus which is believed to be impacted at levels that do not present health effects for adults (Grandjean et al, 1998; AFS, 2002; Rice et al, 2003).

8.3 Safe Fish Consumption levels

So how much tuna can one safely eat? The answer will vary based on several factors: weight, gender, age and likelihood of becoming pregnant or being pregnant or breastfeeding. This is why obstetricians and pediatricians should be the front line of information for women planning to become pregnant, are pregnant or are raising young children. The National Resources Defense Council (2009b) provides a website calculator program (www.nrdc.org/health/effects/mercury/tuna.asp) using EPA and FDA data. Based on these calculations, eating only tuna fish, a person who weighs 180 pounds or more can eat one can of tuna per week and meet the EPA Reference Dose. Pregnant and breastfeeding women are recommended by the FDA, supported by a recent study by Consumer Reports on mercury in foods, to not eat any tuna. Young children (over 45 pounds) can eat 1/3 a can of white albacore tuna per week or up to one can of light tuna per week (NRDC, 2009b; Consumer Reports, 2009. Consumer Reports (2009) additionally recommends that women of childbearing age who are not pregnant limit their tuna intake to one can of albacore tuna (or up to three cans of light tuna[34]). The 2004 FDA advisory clarifies that an excess of mercury intake based on seafood consumption over a week is not necessarily hazardous to one's health, but should be balanced over the following week(s).

8.4 Consumer Awareness

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34 Consumer Reports' 2006 testing found up to 6% of commercial light tuna fish contains as much or more mercury as does white albacore, if yellowfin tuna species are used; as such, Consumer Reports has questioned the FDA on lack of notification regarding higher levels in light tuna. Bluefin tuna is a concern for high-end sushi consumers.

Consumers' knowledge about fish advisories however, is inconsistent and may be gender-biased or regionally-biased. A random survey conducted in 1999 across 12 states found only 20% of women were aware of fish advisories (Anderson et al, 2004, AMA, 2004). Most women were aware of mercury's toxicity, but few were aware that their state had issued an advisory for mercury in fish. Women who were aware of it were also likely to have an angler in the household. Ashizawa et al (2005) found more men than women were aware of these advisories. This apparent gender-bias may reflect the demographic that reads these advisories: anglers, who are primarily male. However, the underlying basis of the fish advisories is to protect the fetus, the most sensitive population for methyl mercury consumption effects. Some states, including New York State, have issued fish advisories specifically addressing women of childbearing ages (typically 16-49 yrs old), children and pregnant women. In New York State, a regional advisory was recently issued that recommends women of childbearing ages and children not to eat any fish caught in the waters of the Adirondack Park and Catskill Mountain Park. Although women may not be as actively involved in recreational fishing as males, they are often preparing and consuming the recreational fish caught. Thus, the awareness of the recreational fish advisories should be able to reach all the consumers for these fish.

Currently, in New York State, an annual report is published listing the specific water bodies and the contaminants of concern that have triggered the advisory (NYSDOH, 2008a). The 2008-2009 report is available on the NYSDOH website at (www.nyhealth.gov). This effort may not be sufficient to increase the awareness needed of these advisories, especially as there are regions in New York State that provide a "eat none" advisory for both male and females. Unfortunately, these regions of New York are also areas where

internet service is unreliable or unavailable, local libraries are in towns remote from many of the fishing areas under advisories, and are also hosts to high volumes of tourism from other states and countries. The means of distributing the advisories to the targeted populations, both the fishermen and the consumers, should be enhanced so the risks are better understood.

A recent study in Wisconsin (Knobeloch et al, 2007) also found that age and gender were significantly correlated with mercury levels from consuming fish. Both males and females older than 39 ate more fish meals than younger people. The study also found that men consistently had higher levels of mercury when genders were compared. This could be reflective of larger-sized fish meals, although the number of fish meals was comparable between men and women, or reflect the higher consumption of recreational fish by men over women, who ate more canned tuna. It may also be reflective of different metabolism mechanisms. Men tend to have higher iron levels in blood, and mercury initiating binds to the hemoglobin when entering the blood system. Perhaps this mechanism is important and should be studied in more detail.

8.5 Benefits versus Risks from Fish

An important message about fish consumption is that eating fish provides many benefits. The awareness of fish advisories needs to improve in order to deliver the message that fish protein is important, as are many nutrients found in fish, such as selenium and omega-3 essential fatty acids (EFAs), such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Mozaffarian and Rimm's (2006) studies found consumption of 250-500 milligrams per day (mg/d) of EPA and DHA lowered one's risk of coronary

heart disease by 25%, but the 500 mg/d level also acted as a threshold thus above this level, minimal gains were observed.

Table 4 provides EPA and DHA levels of commonly eaten fish species, as well as selenium – a trace metal that provides both neurological benefit to the body, and is suspected as providing a counteracting response to the effects of methyl mercury[35]. The "Eat Fish, Choose Wisely" brochure produced by the NYCDOH (2008) reiterates this message. Understanding which fish species can present a higher mercury exposure and revising one's fish consumption habits will allow one to reap the neurological benefits of selenium and omega-3 EFAs, and minimize potential neurological damage from mercury. Table 4 details common fish and seafood levels, as provided by Mozaffarian and Rimm (2006) who conducted a clinical review for physicians, finding that the benefits of fish consumption were greater than potential risks from mercury.

Table 5: Beneficial vs Harmful Fish Concentrations

Fish Species	Mercury (µg/g, or ppm)	Selenium (µg/g, or ppm)	EPA+DHA (mg/USDA serving size)
Salmon, farmed	<0.05	0.41	4,504 (6 oz)
Salmon, wild	<0.05	0.46	1,774 (6 oz)
Catfish, farmed	<0.05	0.15	253 (5 oz)
Herring, Atlantic ocean	<0.05	0.47	1,712 (3 oz)
Anchovy	<0.05	0.68	1,165 (2 oz)
Clams	<0.05	0.64	241 (3 oz)
Oysters	<0.05	0.77	585 (3 oz)
Shrimp	<0.05	0.40	267 (3 oz)
Fish burger (fast food)	<0.05	0.17	337 (2.2 oz)
Trout	0.07	0.15	581 (2.2 oz)

35 The Seychelles epidemiological studies did not initially find adverse effects (although Myers et al, 2009 recently detected significant adverse neurological impacts in this cohort group). Several researchers have theorized the selenium levels found in fish may counteract mercury's damage.

Golden Bass (tilefish), Atlantic Ocean	0.14	0.52	1,358 (5.3 oz)
Golden Bass (tilefish), Gulf of Mexico	1.45	0.52	1,358 (5.3 oz)
Tuna, white (albacore)	0.35	0.66	733 (3 oz)*
Tuna, light (skipjack)	0.12	0.80	228 (3 oz)
Swordfish	0.98	0.62	868 (3.7 oz)

*The USDA serving size is 3 ounces, but comes in a 6 ounce can (0.70 parts per million in one can)

Fish species at or near the FDA daily fish intake level of 1 part per million (1 µg/g)

(table adapted from data presented in Mozaffarian and Rimm, 2006)

CHAPTER 9: ROUTES OF EXPOSURES & HEALTH CONCERNS

Following comprehensive research on lead, researchers are concluding that there may not be a "threshold" level, below which no effects are expected to occur. It is possible mercury, another heavy metal that results in neurological impairment at low levels, may also not have a "threshold level". A latency period seems to be supported, at high and low exposure doses.

9.0 Routes of Exposure

Mercury exposure health impacts can differ based on which species is causing the exposure, since inorganic mercury and organic mercury affect the body systems differently. Elemental mercury (as Hg^0 vapor) that is inhaled has a half-life in a body system that is significantly shorter than for organic mercury, typically between 40 and 60 days (AMA, 2004; Myers et al., 2004) Organic mercury stays in the blood for about 60-80 days, but up to 10% partitions into the brain, with residence times that can be months (Knobeloch et al, 2007). The 2004 FDA and EPA joint advisory reports that it can take up to a year for methyl

mercury levels to drop significantly. Inorganic mercury in the brain, however, is more persistent. Brain levels of methyl mercury are very slowly demethylated, with a daily reduction of approximately 1% of total body burden, indicating the brain becomes a "mercury sink" for body burden loads of inorganic mercury (USEPA 1997a, AMA 2004, Weil et al. 2005). Inorganic mercury in brain tissue cannot pass the blood-brain barrier and accumulates for years (Newland et al, 2008; Akagi et al, 1998).

Mercury's persistence in a body may be additionally be affected by the route of exposure. There are four main routes for how mercury can enter a body: dermal contact through the skin, injection into the bloodstream or muscle, inhalation of vapors and particulates into the lungs or ingestion of mercury-contaminated food or liquid.

Dermal absorption of metallic mercury into the blood stream is unlikely to be significant – holding elemental mercury (also called "quicksilver") is most likely to result in inhalation of vapors. The skin serves as a primary barrier system for inorganic mercury, minimizing transfer of mercury into the body. However, the most toxic form of organic mercury, dimethyl mercury, is rapidly absorbed through the skin and through many types of protective gloves, as well as inhaled as a vapor (Nierenberg et al, 1998; Endicott, 1998).

Injection is a route of exposure that is primarily reserved for vaccinations. Minimal studies exist on ethyl mercury, the metabolite of thimerosal that presents the mercury exposure. A study by Pichichero et al (2002) found ethyl mercury to have a much faster half-life in blood (approximately 7 days[36]) in a study on infants receiving thimerosal vaccines. Demethylation studies of ethyl mercury have not been conducted, thus the impact of mercury accumulation in the brain as inorganic mercury is currently undefined.

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36 Harry et al (2004) found the brain levels of mercury in newborn mice did not change although the blood levels had decreased by this same time period. Clarkson et al (2003) found ethyl mercury took 20 days to clear an adult's system.

The primary route of exposure for most Americans is ingestion of mercury-contaminated fish. Ingestion is the least problematic exposure route for metallic mercury because it is not well-absorbed by the gastrointestinal (GI) system and is almost fully excreted through the kidneys and into the urine. Inorganic mercury salts are more soluble, thus are slightly absorbed by the GI system, but are believed to only minimally impact the central nervous system of an adult[37]. However, ingestion is a primary route of exposure for methyl mercury, which is absorbed at the gastrointenstinal tract into the blood stream, systemically affecting the immune system, cells, central nervous system and brain.

Inhalation is of significant concern as a route of exposure, as almost 100% of mercury vapors are absorbed into the bloodstream (USEPA, 1997a). Increased temperatures can increase volatilization rates of Hg^0. Elemental mercury vapor is the primary species inhaled; however, dimethyl mercury – rarely used outside a scientific laboratory – has a very low vapor pressure and exists in liquid form comparable to "quicksilver". Dimethyl mercury presents the most significant inhalation exposure in terms of toxicity, primarily to laboratory workers (OSHA, 1991).

9.1 Latency Periods

In 1996, Karen Wetterhahn, a Dartmouth chemistry professor spilled a drop or two of dimethyl mercury on latex gloves, in a chemical hood. No immediate health effects were observed. However, after 150 days, her body system began to unravel the severe damage that had been insidiously destroying her internally, from the cellular to organ level. The health effects' severity coalesced rapidly into a system shutdown and coma, with Dr. Wetterhahn

37 Infantile and fetal central nervous systems are more impacted by soluble inorganic mercury than adult systems (AMA, 2004; NYSDEC, 2006).

quickly succumbing to catastrophic system failure and death. No observable symptoms occurred for months between exposure and initial signs of neurological damage. With the onset of symptoms, significant poisoning was apparent within two weeks (Nierenberg et al., 1998; Endicott, 1998). Although dimethyl mercury was known to be a "supertoxic" compound, the degree of its toxicity was largely unknown until Dr. Wetterhahn's accident (Nierenberg et al, 1998). Unfortunately, in the study of toxicology, the majority of our understanding of health effect impacts derives from accidental poisonings.

The initial question of latency periods arose following the Iraqi seed poisoning. Initial assessments found no deviation from normal development for prenatally or postnatally-exposed infants a year following the exposures. However, later studies of these children, in the 1980s, did find adverse neurological outcomes, primarily in language acquisition (Baker, 2008). The concern for latency effects was thus born, and the association with lead impacts on developing children was exemplified. Researchers like Weiss (2000, 2002), Rice & Barone (2000), Adams et al. (2000), Clarkson & Magos (2006) and Weil (2005) are concerned with the latency response of a body to pollutants, resulting in impairments later in the life of the exposed person. This is of grave concern for public health officials as it questions if the levels set as being "safe" (more properly defined as being "at a level not expected to cause health effects") are protective of the population in the years beyond exposure.

9.2 The Toxicity Pyramid

Recent research has focused on the progression of latency periods from exposure to body system response (Weiss et al, 2002; Weiss 2000; Weil, 2005). Fifteen years following the

last acute exposure poisoning of Minamata Disease, new diagnoses were reported (Weiss et al, 2002). Dr. Wetterhahn had received a lethal dose, but yet did not demonstrate any outward symptom for 150 days, although the body burden far exceeded known "lethal" doses of mercury in her system (Nierenberg et al, 1998; Weiss et al, 2002). The Iraqi grain poisonings did not affect the population of thousands of people, for months following ingestion of the contaminated wheat, then suddenly the poisoning took affect sharply (Weiss, 2000). A review of accidental mercury poisonings shows there can be an initial "clinically-silent exposure" followed by clinical poisoning (and death) (Weiss, 2000). Additionally, on a dose-dependent level, there can be what Weiss (2000) terms "a toxicity pyramid" of exposures at clinical poisoning levels, followed by subclinical poisoning and then "latent toxicity" when doses occur at lower levels.

Reviews of toxicological studies on methyl mercury exposures found that the latency period was typically related to the dose; with longer periods associated with low-level, chronic exposures (Weiss et al, 2002). Weiss et al (2002) surmise that toxic doses result in initial cell death, damaging a system but not fully – the remaining cells compensate for the damaged ones, but this support system eventually fails and health effects become "symptomatic". Parkinson's Disease is believed to follow this progression, as does multiple sclerosis. Latent health damage from mercury exposure is suspected with Alzheimer's Disease as well (Clarkson et al, 2003). As time progresses, the functional impairment accelerates. As Weiss et al (2002) observe about the correlations between Parkinson's Disease and toxic mercury poisoning: "Most observers agree that the appearance of clinical signs is merely the ultimate phase of a neurodegenerative process whose inception might even be traced to events occurring during early development."

9.3 Public Health Concerns of an Aging Population

This latency period is well understood in the development of cancers; yet, how well is it understood in low-dose exposures of heavy metals to a developing child or fetus? How does a continuing chronic low dose exposure to an adult affect that adult as he or she ages? Is there an acceleration of the aging process from the cellular damage that is known to occur with mercury, or does this damage manifest itself as a predisposition for neurological diseases of the aged like Parkinson's or Alzheimer's diseases? As the United States Baby Boomer population ages, the public health agencies should be concerned about their cumulative mercury exposures. This population had been exposed to calomel in teething powders as babies[38] (identified as a source of mercury poisoning only in 1947), they have numerous cavities filled with mercury dental amalgams, and are becoming more "environmentally-friendly" and installing energy-efficient CFLs, prone to breakage, in their homes. What are the public health ramifications of mercury exposures throughout a lifetime if it may be the aging process that reveals this damage over time? Is the route of exposure or bioaccumulation over time the most critical factor? As a society, we will need to answer these questions to improve health outcomes associated with mercury exposures, something that cannot be "eliminated" because of its presence in nature.

9.3 Sensitive Populations

38 Calomel (mercurous chloride) was used in teething powders for babies for decades prior to it being identified as a cause of "Pink Disease" (acrodynia). Up to 25% of babies with Pink Disease died. According to Weiss (2000), when calomel was removed from use, the incidence of Pink Disease "plummeted".

Genetic differences among individuals may be the primary factor in how long mercury is retained in a body. Sensitive populations for health effects associated with exposures to contaminants typically include: older people (due to the functional cellular loss that occurs with aging); those with compromised immune systems; pregnant women (due to the potential severe impacts to a developing fetus); and, children (often under the age of 6 when full body system functions have not yet matured). Sensitive populations may lack the biological mechanisms to excrete this material easily, allowing a longer residence time and thus more time to construct its damage (Duncan, 2009). Or it could be that sensitive populations reveal the damage from earlier exposures.

Genetic susceptibilities are a new area of focus for environmental pollutants. Immature systems of infants may be vulnerable to pollutant damage, but acute and repeated doses may present impacts that will manifest later in the development process (like adolescent when significant brain and physical changes occur). These questions have been recommended for study by the National Research Committee (2000). Researchers like Adams et al (2000) are concerned that the timing of the exposure can affect different end points, or present long-term damage that is not apparent until the aging process evolves.

Considering the latency issues observed by researchers regarding toxic doses of mercury exposures, and the controversial evaluation of developmental impacts of children receiving thimerosal doses during the 1990s, the answers may not be available until this generation of children ages. Well-designed longitudinal studies are important in order to answer unknown neurological toxicity questions with organic mercury.

Part III- Reducing Exposures Through Knowledge

CHAPTER 10: CONCLUSIONS

Life - a culmination of the past,
an awareness of the present,
an indication of the future beyond knowledge...
- Charles A. Lindbergh

Mercury has been prevalent in our personal past, our historical past and our geologic past. It cannot be eliminated from our lives, but should be examined for how it can continue to pose harm as mercury-bearing materials persist in our lives. Its cumulative nature supports its designation of being a "persistent, bioaccumulative and toxic" compound. Controls to limit or eliminate additive mercury loading to our environment are essential as current study into long-term health effects is just developing.

Sensitive populations may be affected by the multitude of exposures to mercury in our daily lives. Women of childbearing ages are of particular susceptibility to mercury exposures, as they serve as a transfer mechanism to the developing fetus. Neurotoxicity to the fetal brain is the most sensitive endpoint for mercury exposures, thus evaluations of all cumulative exposures should be a primary focus in public policy decisions. Evaluating exposure limits on a body weight basis may not be sufficient, as research is showing that the timing of the exposure may be the most critical determining factor in the ensuing health ramifications over time. Minimizing our body burden of mercury is the most prudent action that can be taken while the research assesses a threshold level.

Public policy actions recently are addressing the "Precautionary Principle" of reducing exposures universally while the scientific debate continues on levels of mercury exposures that are "without harm". Anthropogenic use of mercury can be minimized, with

ensuing positive environmental responses. A balance can be created between the need for more energy, energy efficiency and public health protection. A global response is important to address all anthropogenic mercury deposition; however, local responses do warrant action. Mercury impacts will continue for generations to come, based on the mercury cycle; however, government action today can effectively respond to the societal obligation of protecting public health and the environment.

CHAPTER 11: RECOMMENDATIONS FOR FUTURE ACTION

Public policy cannot be improved upon to be protective if the supporting information is outdated or incomplete. In order to improve upon the message that mercury can present health concerns even at low levels, and the ultimate goal of reducing exposures, information essential for determining if exposures are decreasing is needed. Several means this message can be improved would be to expand programs that collect exposure data, increase scientific collection of data, and extend as well as clarify fish consumption advisories.

The current New York State Heavy Metals Registry should be modified to track all mercury tests, as it has been for lead since 1992. Mercury is no longer an occupation exposure primarily, but can impact children significantly. Yet children younger than 16 are not classified in the Heavy Metals Registry, thus their biological monitoring data is not reported to the State Health Department. The Heavy Metals Registry is thus an incomplete database for evaluating heavy metal exposures to the populations most likely to have health effects. Mercury presents significant exposure concerns to children, as the 2009 ATSDR report details. Mercury is present in household materials and in the food we eat. A more thorough understanding of the prevalence of mercury exposures would be gained if the reporting guidelines for mercury were modified, and standard biomonitoring data was collected to evaluate the potential for health concerns among children. These changes were enacted for lead in 1992, yet the other neurotoxin most likely to affect children, mercury, is not being monitored. Without monitoring, specific public health approaches for children cannot be implemented.

The following list provides the two-pronged approach needed to improve the mercury exposure message:

To clarify and improve the exposure message to the public, I recommend New York State:

1. Require labeling for mercury content in all canned tuna, and require advisory postings regarding high mercury commercial fish at food markets/restaurants

2. Target health consultation agencies and physicians for providing mercury exposure brochures to the populations most at risk, based on personal lifestyles

3. Provide nonverbal fish advisory posters for both recreational and commercial fish products

4. Expand delivery of fish advisory message to campgrounds, tourism information, angler license distributors

5. Improve community awareness of mercury-containing products and disposal procedures

To enhance the scientific research database, the backbone of public policy decisions, I recommend New York State support the following action items:

1. Prioritize epidemiological and/or toxicological studies on effects of ethyl mercury and methyl mercury on the brain , cardiovascular and immune systems.
2. Collect mercury gas (Hg0, Hg2+, HgP) data concurrent with ozone and other HAPs at the Division of Air Resource Air Quality Monitoring stations across the state.
3. Monitor for mercury in blood levels as is done with lead, with Heavy Metal Registry reporting for all ages. I recommend age grouping comparable to the USEPA's designations in their Child-Specific Exposure Factors Handbook (2008).

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Appendix A

Guidelines for Cleaning up A Mercury Spill

NEVER USE A VACUUM CLEANER! Vacuum cleaners can disperse the mercury into many small beads, enhancing the vaporization process. Additionally, a vacuum in use heats the mercury, enhancing the vaporization process. The vacuum can become contaminanted, presenting a continuing source of mercury vapor when used in the future. IF YOU ARE PREGNANT – do not clean up the spill yourself.

1. Open a window in room where bulb broke, shut off any heating or cooling systems to the room, and leave, closing the door.
2. Wait 15 minutes before returning to clean spill
3. Put on disposable gloves, pick up pieces of broken glass shards and put into a glass jar with screw top lid (best). If glass jar is not available, double-bag with sealable plastic bags.
4. Use sticky side of duct tape to pat for remaining small shards.
5. Shine a bright light around area to check for glass shards.
6. Add tape to the zip-lock plastic bags or glass jar.
7. Pat the area with damp paper towels, add to the zip-lock plastic bags or glass jar.
8. Place zip-lock plastic bags outside – they can off-gas any stored mercury vapors.
9. Wash your hands and face.
10. Continue to ventilate room for several hours, if possible.

If a spill occurs on a soft surface, such as a sofa, bed or carpet, follow the above steps but consider the additional step of removing the material from the room into the outdoors for several hours (preferably in sunlight to enhance the vaporization process). AFTER A SPILL – continue to open a window and shut off ventilation systems when vacuuming the room after the spill has been cleaned up. Children, pets and pregnant women should refrain from using this room for while.

Information was obtained from a Fact Sheet published by the Connecticut Department of Public Health "Compact Fluorescent Light Bulbs: What to do if a bulb breaks" [Available at: http://www.ct.gov/dph/]

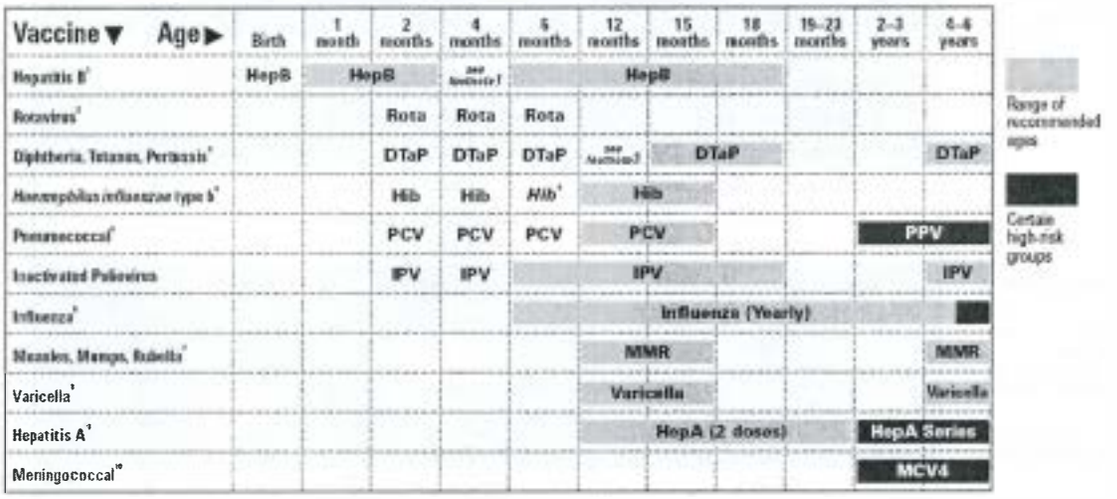
Appendix B

Recommended Vaccine Schedules 2008 and 1999

Table 1: 2008 CDC recommended vaccination schedule

Recommended Immunization Schedule for Persons Aged 0–6 Years—UNITED STATES • 2008

For those who fall behind or start late, see the catch-up schedule

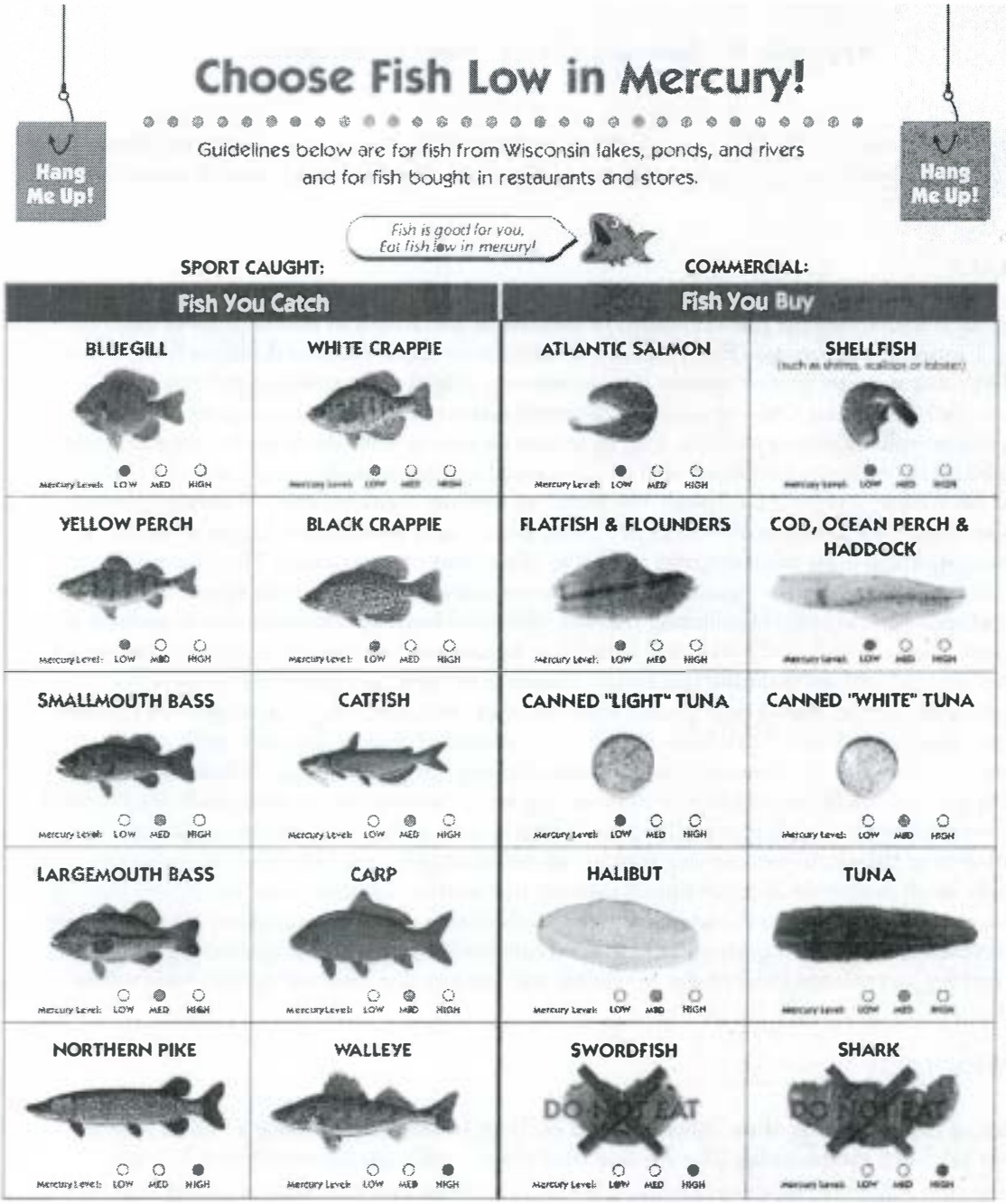


As of December 1, 2007.
Source: http://www.cdc.gov/vaccines/recs/schedules/downloads/child/2008/08_0-6yrs_schedule_pr.pdf

Table 2: 1999 Vaccinations with Thimerosal
Pre-IOM Recommendation to Remove from Childhood Vaccines
(Table data obtained from AAP, 1999)

Vaccine	Manufacturer	Brand	Mercury (ug/0.5 mL)
DtaP	Lederle Laboratories	Acel-Imune	25
	Pasteur Merieux Connaught	Tripedia	25
	North American Vaccine	Certiva	25
	SmithKline Beecham	Infanrix	0
DTwP	All	All	25
DT	All	All	25
Td	All	All	25
TT	All	All	25
DTwP-Hib	Lederle Laboratories	Tetramune	25
Hib	Pasteur Merieux Connaught	ActHIB	0
	Pasteur Merieux Connaught	TriHIBit	25
	Lederle Laborator es	HibTITER (multidose)	25
	Pasteur Merieux Connaught	ProHIBit	25
	SmithKline Beecham	Omni HIB	0
	Merck	PedvaxHIB liquid*	0
	Merck	COMVAX	0
Hepatitis B virus	SmithKline Beecham	Engerix-B	12.5
	Merck	Recombivax HB	12.5
Influenza	All	All	25
Meningococcal	CLI	Menomune A,C, AC	25
	CLI	Menomune A/C/Y/W-135	25
Pneumococcal	Lederle Laboratories	Pnu-Imune 23	25
	Merck	Pneumovax 23	0
Rabies	BioPort Corporation	Rabies Vaccine Adsorbed	25
	Pasteur Merieux Connaught	IMOVAX	0
	Chiron	Rabavert	0

- earlier versions did include up to 12.5 µg/0.5 mL mercury per dose
- Other vaccines not listed, such as the polio virus, varicella (chicken pox), rotavirus and lyme were not manufactured with thimerosal in 1999.



Appendix C: State of Wisconsin Fish Consumption Advisory Poster

From: 2002 American Fisheries Society :Forum on Contaminants in Fish: Proceedings

(Available at: http://www.epa.gov/waterscience/fish/forum/2002/sections1-5.pdf)

Appendix D: Summary of Field Study Investigation

Summary of: McLelland, S.P. 2008 (unpublished research paper). Meteorological Influences on Mercury Air Pollution in the Adirondacks. Independent Study, Geosciences Department, Skidmore College. Fall 2008.

ABSTRACT

Mercury air emissions are the primary source of mercury in watersheds in remote areas of the country. Elemental mercury gas (Hg^0) is known to travel in air masses further distances from source areas than other mercury species, such as divalent mercury (Hg^{2+}) and particulate mercury (Hg_p), which are likely to deposit closer to sources. Elemental mercury comprises the majority of mercury species in atmospheric mercury studies, thus its impact on remote watersheds needs better definition. The Hubbard Brook Research Foundation has sponsored numerous studies to answer why some areas of the continent present biological "hot spots" of mercury contamination. Watersheds on the East Coast and in the Maritime Provinces of Canada consistently demonstrate higher levels of mercury in aquatic species and waterfowl relative to other areas of the country. The Adirondack Mountain region demonstrates significantly high mercury concentrations in both aquatic and waterfowl species. A field study collecting real-time elemental mercury concentrations in ambient air in July and August 2007 found a statistical correlation between elevated levels of mercury during clear days and reduced levels during rain events. Ozone levels were also assessed for this region during the study period, noting high ground level ozone air masses moving from regions of mercury emissions southwest of New York State and into the Adirondack region. Elevated levels of ozone may serve as an oxidant for elemental mercury, transforming $Hg^0(g)$ to $Hg^{2+}(g)$. Divalent mercury is significantly more soluble than elemental mercury and is the mercury species biologically transformed into methylmercury. In a high precipitation environment, such as the Adirondacks, air masses originating from the southwest, carrying mercury air pollutant gases and high levels of ozone can potentially result in transformed, solubilized mercury that washes out of the atmosphere and into the sensitive watersheds of the remote lakes. Elevated levels of mercury transformed into methylmercury in a watershed result in biomagnification of the mercury levels by orders of magnitude up trophic levels, creating an environmental hazard to wildlife and humans that consume the fish within these watersheds.

METHODOLOGY

Ambient air concentrations of elemental mercury gas (Hg^0_g) were analyzed using a Lumex Mercury Analyzer RA-915+ spectrometer. This method used either a multi-gas sensor (Optical III) or a single-gas sensor (Optical II) to read mercury levels. A software program allowed for the RA-915+ data to be downloaded onto a portable laptop computer for continuous real-time data collection and evaluation. A Hoboware relative humidity and temperature sensor was also employed during weekends 2 and 3 to assess correlations of mercury concentrations with varying relative humidity values. Two passive diffusive badges were also used in monitoring for ambient mercury levels at each of the thirteen main sampling stations, to evaluate if these devices could detect mercury at low nanogram levels. The ambient air collection height was approximately 3-5 ft above ground surface, with the exception of the non-canopied sites at Cape Vincent's Tibbett's Point Lighthouse and the Kane Mountain Firetower. Air monitoring occurred at approximately 12 ft above ground surface at Tibbett's Point and approximately 65 ft above ground surface at Kane Mt. Firetower.

DISCUSSION

The WL Gore mercury passive diffusion badges were not sensitive enough to detect elemental mercury gas at the low nanogram per cubic meter levels that the Lumex RA-915+ spectrometer was able to read. The Lumex spectrometer provided real-time data that allowed modifications through the program, such as changing from a multi-gas sensor to the more sensitive single-gas sensor when mercury concentrations hovered around the nanogram to picogram ranges and using additional field instruments such as the Hoboware relative humidity and temperature monitors.

Statistical correlations were supported for clear vs. rainy sampling events, and weaker correlations were found for both mercury relative to ozone levels and for the western region of the Adirondacks detecting elevated levels of mercury in the atmosphere when compared to the eastern stations. Relative humidity values were not supported as being a significant control; however, observed drops in mercury levels were noted when rainfall began and/or when canopy throughfall persisted following a rainfall event. However, additional data is needed to assess this relationship due to the limited number of rainfall event samples during the study period.

CONCLUSIONS

Additional, synoptic mercury monitoring is needed to fully assess the influences of relative humidity and precipitation events on Hg0 transformation into the soluble divalent mercury (Hg2+) for watershed impacts, as well as for re-emission of Hg0 from transformation of Hg2+ to Hg0 biologically. Factors such as biological activity and photolytical reduction in shallow waters may contribute to evasion of mercury gas, and precipitation coupled with anthropogenic air pollutants such as ozone and sulfate could be significant oxidants of elemental mercury into divalent mercury. The migrating pollutant-rich air masses originating from mercury sources upwind are believed to contribute to the designation of the Adirondacks as a biological "hot spot" for aquatic and waterfowl species. Understanding the transformative process of atmospheric mercury into methylmercury affecting living organisms is critical, as biomagnification of this element presents significant physiobehavioral and neurologic impairment to upper tropic level receptors, including humans.

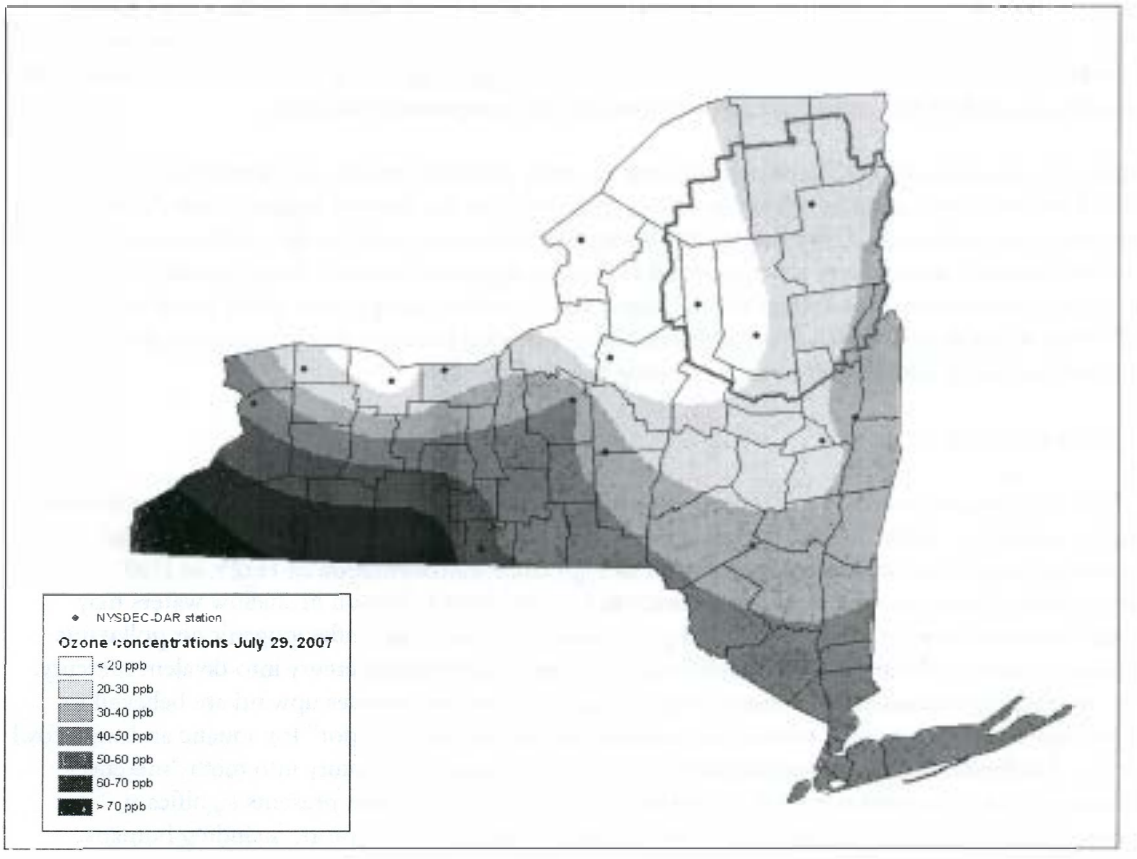


Figure 4: Ozone concentrations (in parts per billion) collected on July 29, 2007 demonstrate low ozone levels across the study region of the Adirondacks and elevated levels originating southwest of New York State. NYSDEC-Division of Air Resource monitoring stations are located on the base map. Low levels of elemental mercury were detected on this date as well within the Adirondack Park. This map does not accurately reflect ozone concentrations south of the Catskills, as data was not evaluated from these stations, but only those DAR locations relative to the study area of concern.

Excerpt from: *Meteorological Influences on Mercury Air Pollution in the Adirondacks. Independent Study, Geosciences Department, Skidmore College. Fall 2008.*