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Sacramento-San Joaquin Delta Regional Ecosystem Restoration Implementation Plan

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Sacramento-San Joaquin Delta
Regional Ecosystem Restoration Implementation Plan

Ecosystem Conceptual Model

Mercury

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PREFACE

This Conceptual Model is part of a suite of conceptual models which collectively articulate the current scientific understanding of important aspects of the Sacramento-San Joaquin River Delta ecosystem. The conceptual models are designed to aid in the identification and evaluation of ecosystem restoration actions in the Delta. These models are designed to structure scientific information such that it can be used to inform sound public policy.

The Delta Conceptual Models include both ecosystem element models (including process, habitat, and stressor models); and species life history models. The models were prepared by teams of experts using common guidance documents developed to promote consistency in the format and terminology of the models http://www.delta.dfg.ca.gov/erpdeltaplan/science_process.asp .

The Delta Conceptual Models are qualitative models which describe current understanding of how the system works. They are designed and intended to be used by experts to identify and evaluate potential restoration actions. They are not quantitative, numeric computer models that can be "run" to determine the effects of actions. Rather they are designed to facilitate informed discussions regarding expected outcomes resulting from restoration actions and the scientific basis for those expectations. The structure of many of the Delta Conceptual Models can serve as the basis for future development of quantitative models.

Each of the Delta Conceptual Models has been, or is currently being subject to a rigorous scientific peer review process. The peer review status of each model is indicated on the title page of the model.

The Delta Conceptual models will be updated and refined over time as new information is developed, and/or as the models are used and the need for further refinements or clarifications are identified.

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Executive Summary

Mercury has been identified as an important contaminant in the Delta, based on elevated concentrations of methylmercury (a toxic, organic form that readily bioaccumulates) in fish and wildlife. There are health risks associated with human exposure to methylmercury by consumption of sport fish, particularly top predators such as bass species. Original mercury sources were upstream tributaries where historical mining of mercury in the Coast Ranges and gold in the Sierra Nevada and Klamath-Trinity Mountains caused contamination of water and sediment on a regional scale. Remediation of abandoned mine sites may reduce local sources in these watersheds, but much of the mercury contamination occurs in sediments stored in the riverbeds, floodplains, and the Bay-Delta, where scouring of Gold-Rush-era sediment represents an ongoing source.

Conversion of inorganic mercury to toxic methylmercury occurs in anaerobic environments including some wetlands. Wetland restoration managers must be cognizant of potential effects on mercury cycling so that the problem is not exacerbated. Recent research suggests that wetting-drying cycles can contribute to mercury methylation. For example, high marshes (inundated only during the highest tides for several days per month) tend to have higher methylmercury concentrations in water, sediment, and biota compared with low marshes, which do not dry out completely during the tidal cycle. Seasonally inundated flood plains are another environment experiencing wetting and drying where methylmercury concentrations are typically elevated. Stream restoration efforts using gravel injection or other reworking of coarse sediment in most watersheds of the Central Valley involve tailings from historical gold mining that are likely to contain elevated mercury in associated fines. Habitat restoration projects, particularly those involving wetlands, may cause increases in methylmercury exposure in the watershed. This possibility should be evaluated.

The DRERIP mercury conceptual model and its four submodels (**1. Methylation, 2. Bioaccumulation, 3. Human Health Effects, and 4. Wildlife Heath Effects**) can be used to understand the general relationships among drivers and outcomes associated with mercury cycling in the Delta. Several linkages between important drivers and outcomes have been identified as important but highly uncertain (*i.e.* poorly understood). For example, there may be significant wildlife health effect of mercury on mammals and reptiles in the Delta, but there is currently very little or no information about it. The characteristics of such linkages are important when prioritizing and funding restoration projects and associated monitoring in the Delta and its tributaries.

1. Overview

Mercury has been identified as a contaminant of concern or chemical stressor in the Sacramento–San Joaquin Delta ecosystem. Sources of mercury include historic mercury and gold mines as well as ongoing atmospheric deposition from regional and global sources. The conceptual model presented here is comprised of the main model and four submodels: **1) Mercury Methylation**, which includes both a) formation of reactive (inorganic) mercury and b) microbial transformation of reactive (inorganic) mercury to (organic) methylmercury; **2) Methylmercury Bioaccumulation**; **3) Human Health Effects**; and **4) Wildlife Health Effects** (Figure 1). Principal intermediate outcomes are methylmercury concentrations in water and sediment (Submodel 1) and methylmercury concentrations in biota (Submodel 2). The final model outcomes are export of

mercury and methylmercury out of the Delta, effects on human health, and effects on wildlife health. In the following narrative, we provide a brief description of each submodel in relation to its drivers, linkages, and outcomes, and the interactions with other conceptual models constructed as part of the Delta Regional Ecosystem Restoration Implementation Plan (DRERIP) for the CALFED Bay-Delta Program. We also discuss the limitations of the overall mercury conceptual model and its submodels, and provide some recommendations for application of the model in resource management.

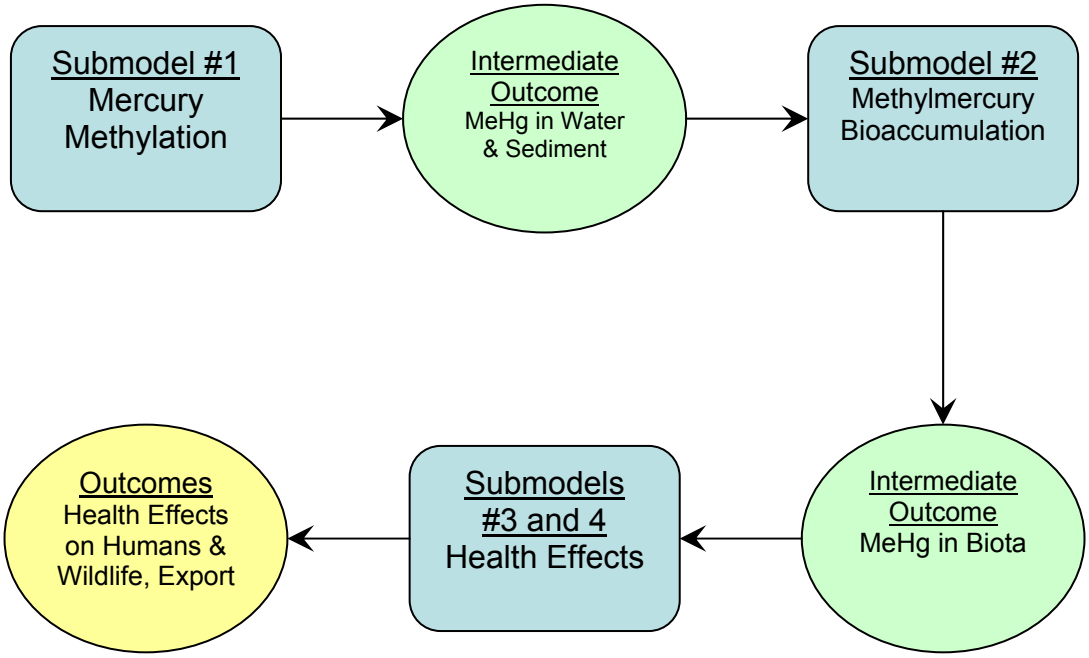

Figure 1. Main compartments of the mercury conceptual model. (after Werner et al. 2008) [MeHg, monomethylmercury]

2. Problem Formulation

The conceptual model described here focuses on the transport, fate, and health effects of mercury and methylmercury on humans and wildlife in the aquatic environment of the Sacramento–San Joaquin Delta, a system comprising many different habitat types and species.

An important consideration is to provide a framework for evaluating how restoration activities might mobilize mercury and/or promote its methylation and bioaccumulation. The mercury conceptual model and its submodels are presented in the DLO (drivers-linkages-outcomes) format, consistent with other DRERIP conceptual models. Where possible, linkages between drivers and outcomes are characterized with regard to three properties: importance, predictability, and level of understanding. Linkages that are highly important but for which there the level of understanding is low or medium may represent high priority topics for future study. Where available, references are given in the text for specific linkages and related processes.

3. Mercury Conceptual Model

Mercury cycling in aquatic environments is complex. The behavior of mercury (Hg) depends on numerous chemical, physical, and biological processes that vary in space as a function of different habitats and their biogeochemical and hydrodynamic environments, and vary in time over different scales (e.g. inter-annual, seasonal, diurnal, tidal). An essential aspect of mercury bioaccumulation and toxicity is the formation of monomethylmercury (MeHg), a toxic form of Hg that readily bioaccumulates. The overall conceptual model for mercury cycling in the Delta (Figure 2) indicates numerous drivers (including links to other DRERIP conceptual models), some intermediate outcomes (MeHg concentrations in water, sediment, and biota) and three final outcomes (export of Hg and MeHg to other environments, health effects on humans, and health effects on wildlife).

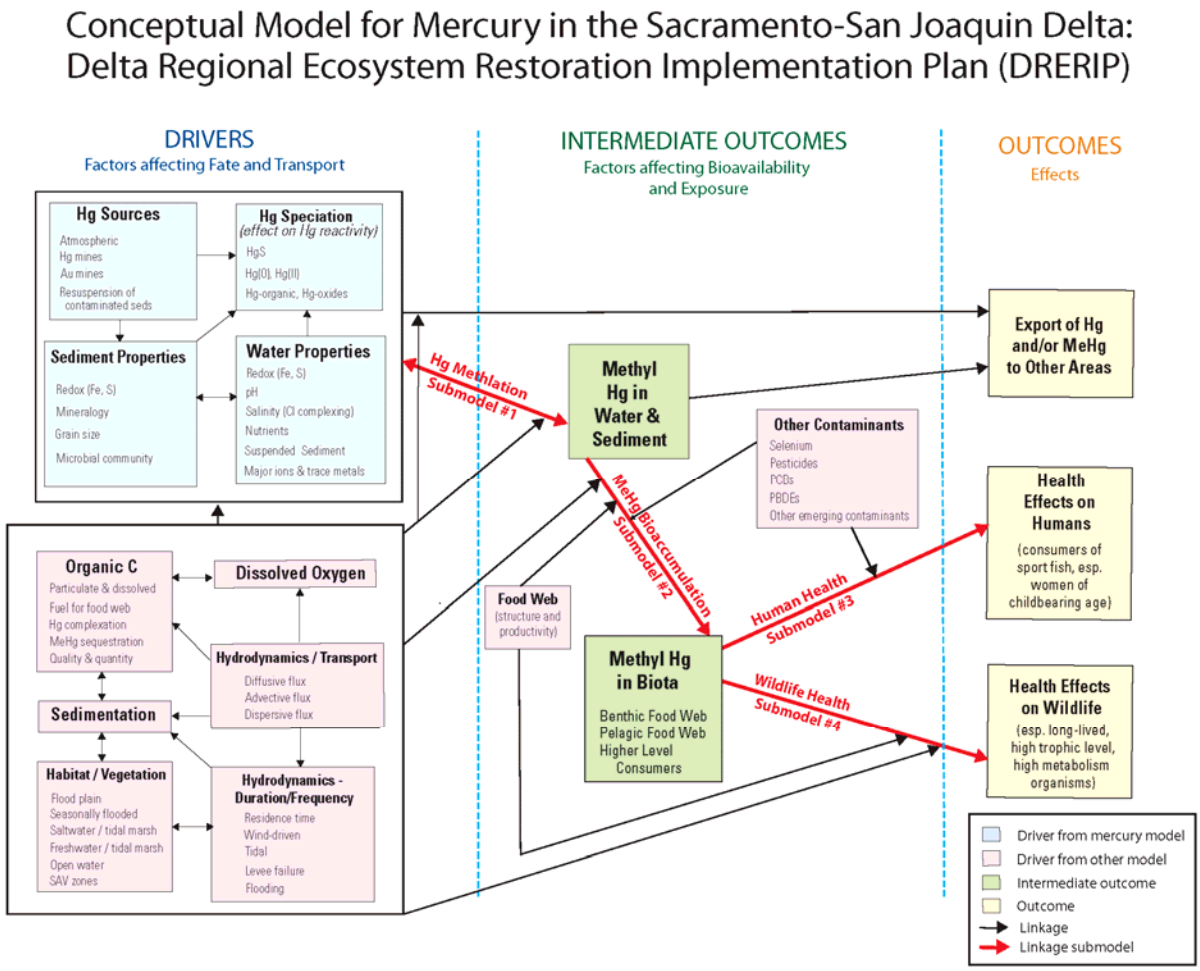

Figure 2. Mercury conceptual model showing four submodels. Drivers from other models (pink boxes) and drivers specific to mercury (blue boxes) represent important factors affecting the fate and transport of mercury. The formation of methylmercury in water and sediment (Submodel 1) is an intermediate outcome that leads to the bioaccumulation of methylmercury (Submodel 2). Methylmercury exposure can lead to adverse health effects in humans (Submodel 3) and wildlife (Submodel 4).

Four of the key linkages on the main mercury model are represented by submodels (Figures 3, 4, 8, and 9) that are described below in subsections 3.1 through 3.4.

3.1 Submodel #1: Mercury Methylation

The conceptual submodel for mercury methylation is illustrated in Figure 3. The overall structure of Submodel 1 starts with inorganic mercury from various sources (Hg mines, gold mines, and atmospheric deposition) and in various forms (sulfide [HgS], elemental [Hg(0)], gold-mercury amalgam [AuHg], Hg-organics, and more soluble forms such as Hg(II)-oxides, -sulfates and -chlorides) and tracks conversion to inorganic Hg(II), some of which is "reactive Hg(II)" or $(Hg(II)_R)$[1] an important intermediate outcome. The $Hg(II)_R$ can be further transformed to monomethylmercury (MeHg) via processes dominated by the activity of microbes (sulfate- and iron-reducing bacteria) in anoxic to suboxic conditions.

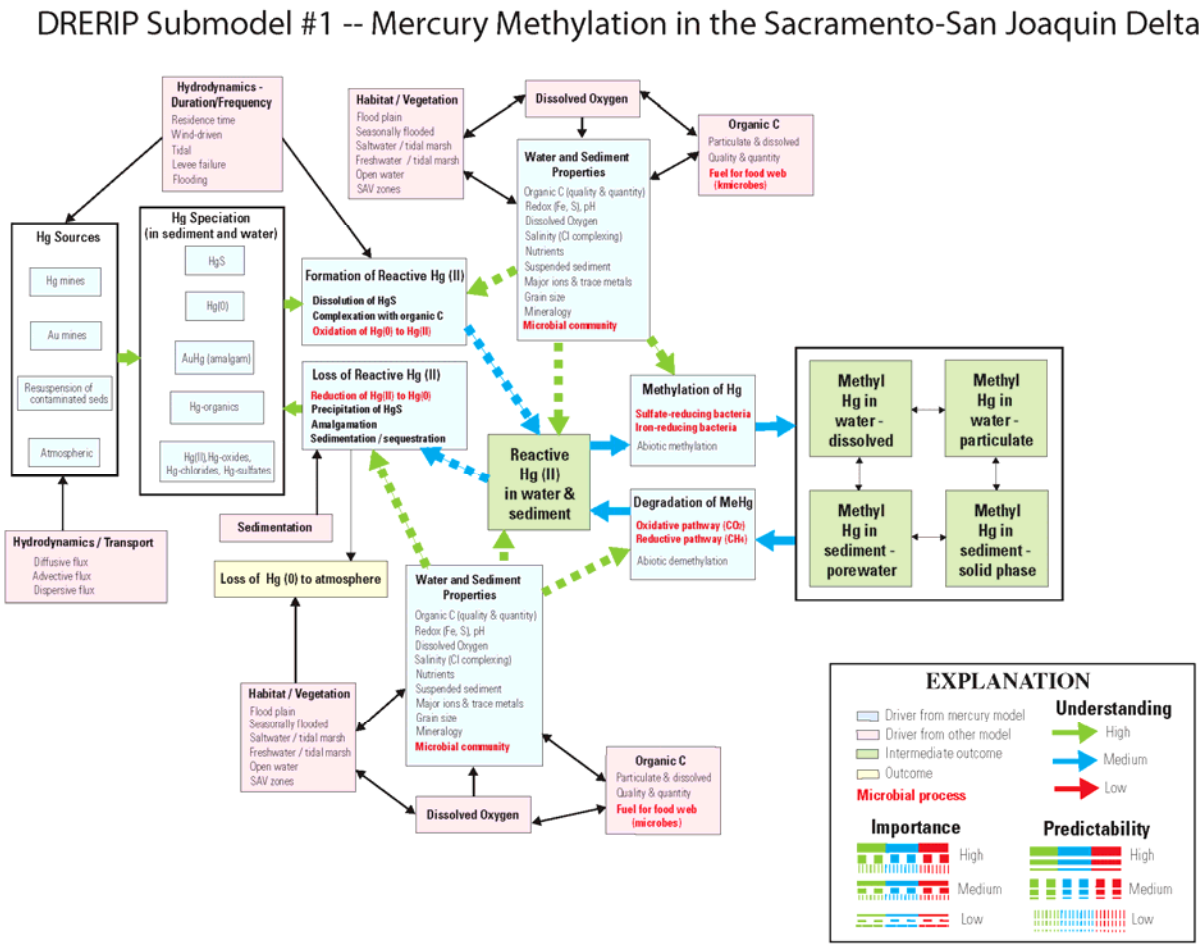

Figure 3. Submodel #1 — Mercury methylation. The source and form (or speciation) of mercury are important factors that help to determine the reactivity of mercury (Hg) with regard to methylation processes. Formation of reactive Hg(II) (the most oxidized form of mercury) is a key intermediate outcome that affects rates of mercury methylation in water and sediment. Degradation of methylmercury (methyl Hg) is also and important process – the difference between rates of Hg(II)-methylation and methyl Hg degradation represents the rate of "net methylation".

[1] Reactive Hg(II) [or $Hg(II)_R$] is operationally defined as the portion of the total inorganic Hg(II) pool that is most readily available for the microbial conversion of Hg(II) to methylmercury (Marvin-DiPasquale et al. 2006).

Once formed, MeHg can degrade (or demethylate) through either microbial or abiotic processes. The difference between the rate of Hg(II) methylation (the forward reaction) and that of MeHg degradation (the back reaction) represents the rate of net MeHg production. Not all inorganic Hg(II) is converted to MeHg. Some Hg(II) may be in a relatively non-reactive form or may not be readily available to microbes because of a physical characteristic such as coarse grain size or a relatively inert coating or mineral intergrowth. $Hg(II)_R$ may become less reactive through physical and biogeochemical processes such as precipitation as Hg-sulfide, microbial reduction to Hg(0), complexation with high-molecular-weight dissolved organic matter, amalgamation with gold or other metals, evasion (from water) or emission (from land) to the atmosphere, or sedimentation and sequestration.

Each of the four key transformations of mercury in Submodel 1 is a chemical reactions thought to be mediated in large part in by microbial activity (as indicated by red lettering in figure 3). Many of the drivers that influence these key transformations are important to the extent that they control the local biogeochemical environment, influencing the composition and productivity of the microbial community in aquatic environments.

3.1.1 Transport and Fate

3.1.1.1 Watershed Hydrology

The Sacramento – San Joaquin watershed represents a large geographic area and a large proportion of California. The Sacramento River watershed is approximately 17,000,000 acres (27,000 square miles) whereas the San Joaquin River watershed is about 9,700,000 acres (15,200 square miles). Because precipitation is more abundant in the north, the Sacramento River Basin (Sacramento River and Yolo Bypass) accounted for about 80% of the inflows to the Delta during the period 1984-2003 (Wood et al. 2006). Based on load calculations, the Sacramento Basin contributed about 83 to 87% of the total Hg input to the Delta (Wood et al. 2006).

There are a wide variety of hydrologic environments in the Sacramento – San Joaquin watershed. The headwaters of most rivers draining the western slope of the Sierra Nevada typically have a snowpack during December – May of each year. The upper watersheds of these rivers are characterized by steep terrain. Many Sierra Nevada, Coast Range, and Klamath Mountain watersheds have extensive water transfer systems consisting of a network of dams, reservoirs, canals, pipelines, and diversions that disrupt natural flows and affect sediment transport. Because of the close association of total Hg with suspended particulate matter, this aspect of the hydrologic system has a large effect on the fate and transport of Hg.

The upper parts of the watersheds tend to have a relatively small percentage of surface water and wetlands relative to total surface area. In a recent national study of large watersheds, a low proportion of wetlands were correlated with relatively low concentration of MeHg as a function of total mercury (Krabbenhoft et al. 1999). In the lower parts of the watersheds, especially on the eastern side of the Central Valley, flood plains consist of extensive gravel deposits that have widely been disturbed by extraction of gold and gravel by dredging and open-pit mining methods. In the lowlands, millions of acres are used for agriculture, In the Sacramento Valley there are about 1,000,000 acres of wetlands, of which about half are planted in rice. Agricultural amendments including various forms of sulfate, may have a profound influence on mercury cycling, but

relatively little research has been done on this topic to date. Large-scale agricultural processes, including of irrigation, wetting/drying, and amendments of sulfate, nitrogen, phosphorus, and other nutrients have been shown to affect mercury cycling in other areas such as the Florida Everglades (Orem 2004).

3.1.1.2 Mining Sources

The primary sources of mercury in the Sacramento-San Joaquin Delta are historical mining operations, including sites of mercury mining in the Coast Range and gold mining in the Sierra Nevada and Klamath Mountains. Because of the widespread distributions of mercury mines in the Coast Range (Rytuba 2003) and gold-mining operations in the Sierra Nevada and Klamath Mountains (Alpers et al. 2005a), mercury contamination from historical mining is present in many Delta tributaries.

Mercury contamination from mining sources is transported to the Delta predominantly in association with suspended particulate matter in surface water (Roth et al. 2001). In mining-contaminated watersheds, the total mercury concentration of suspended sediment (TotHg/TSS) tends to be elevated, at concentrations of 0.3 mg/kg or higher. Wood et al. (2006) compiled available data from 1984 to 2003 and determined the average values of TotHg/TSS for Delta inputs and exports as well as several tributaries to the Sacramento Basin (Sacramento River and Yolo Bypass). Direct inputs to the Delta ranged in values of TotHg/TSS from 0.11 to 0.35 mg/kg; Delta exports ranged from 0.16 and 0.17 mg/kg in the Delta-Mendota Canal and the State Water Project, respectively, to approximately 0.30 mg/kg in exports to San Francisco Bay (the latter number remains uncertain; Wood et al. 2006). As part of the San Francisco Bay mercury control program, the San Francisco Bay RWQCB has developed a target TotHg/TSS concentration of 0.20 mg/kg for Delta exports (Johnson and Looker 2004).

In cores from San Pablo Bay, sediment deposited prior to the onset of the California Gold Rush in 1848 had TotHg concentrations less than 0.1 mg/kg, whereas sediment deposited between 1850 and 1900 had greater than 0.8 mg/kg (Hornberger et al. 1999, Marvin-DiPasquale et al. 2003). The scour and resuspension of Gold-Rush-era sediment within San Francisco Bay may be an important ongoing source of TotHg to the Delta (Wood et al. 2006).

3.1.1.3 Atmospheric Sources and Total Mercury Loads

Mercury is a global pollutant. Natural sources to the atmosphere include volcanic emissions, emission of mercury vapor from soils, rocks, plants, and evasion of mercury vapor from water bodies. Anthropogenic sources to the atmosphere include fossil fuel combustion, especially coal, and other industrial emissions. In many parts of the world, such as the eastern U.S., atmospheric deposition is the dominant source to aquatic systems. However, in northern and central California, various lines of evidence indicate that atmospheric deposition is a relatively minor source of Hg and MeHg to aquatic environments compared with historical mining sources. For example, in rivers draining the northwestern Sierra Nevada, Slotton et al. (1997, 2004b) have shown that MeHg bioaccumulation in invertebrates and fish increases by a factor of 3 to 5 passing from zones upstream of historical gold mining to zones affected by mining, which indicates that mining is the source of most of the bioaccumulated MeHg.

Tsai and Hoenicke (2001) estimated that direct atmospheric deposition contributed approximately 27 kg/yr of total Hg to the San Francisco Bay-Delta Estuary, of which about 5 kg/yr were from

direct wet deposition and 22 kg/yr were from direct dry deposition. Indirect inputs were estimated to contribute another 55 kg/yr of mercury loading to the Estuary. Conaway et al. (2005) determined that combustion of gasoline and diesel in the San Francisco Bay area contributes an average of 5 kg Hg /yr to the environment. Wood et al. (2006) estimated that total atmospheric wet deposition to the Delta during 2000–2003 was 2.26 kg Hg/yr, consisting of 0.85 kg Hg/yr of direct wet deposition to water bodies and 1.41 kg Hg/yr of indirect wet deposition to land surfaces within the Delta.

For comparison, estimated total mercury exports from the Delta to San Francisco Bay during 1934–2003 were 660 ± 233 kg/yr (Wood et al. 2006). An independent estimate of total mercury loads exported from the Delta to San Francisco Bay was 440 kg/yr (Johnson and Looker 2004). Wood et al. (2006) concluded that wet deposition contributes approximately 1% of all Hg entering the Delta. Although this amount may seem insignificant, it is possible that the Hg deposited by wet deposition is more reactive than other sources and therefore may be more easily converted to MeHg and bioaccumulated. The relatively high reactivity of atmospheric mercury is indicated on Table 1 in comparison to other mercury sources to the Delta.

Table 1. Relative magnitude of mercury sources, relative reactivity, and relative importance to methylmercury production to the San Francisco Bay-Delta region.

[L, low; M, moderate; H, high; NA, not applicable]

Source	Hg Speciation	Magnitude of Total Hg Load to Delta	Reactivity (susceptibility to methylation)	Importance to MeHg Production in Delta	Uncertainty	References
PRIMARY SOURCES						
Atmospheric	Hg(II)	L (~1%)	H	M?	M	1
Urban runoff	Various (sorbed?)	L – M?	M? (labile C)	L?	H	1,2
Wastewater-MeHg	MeHg	NA	NA	L – M	L	1,2
Wastewater-Inorganic Hg	Various	L	M?	L	H	1,2
Mercury mines	HgS +	H	L	H	M	1,3
Gold mines	Hg(0), HgAu	H	L – M	H	M	1,3
SECONDARY SOURCES						
Resuspension of sediments	Various	H	L – M	H?	M	1,4
In-place Delta sediments	Various	H	L	L – M?	M	1

References: 1, Stephenson et al. 2007; 2, Wood et al. 2006; 3, Kimball 2006; 4, Domagalski et al. 2004.

3.1.2 Formation of Reactive Mercury(II)

Various experimental studies (e.g. Keiu 2004, Marvin-DiPasquale et al. 2006, Marvin-DiPasquale et al. 2007) have demonstrated that the fully oxidized form of mercury [Hg(II) or the mercuric ion, Hg^{2+}] is the form that is most readily converted to monomethylmercury (MeHg) by microbes. $Hg(II)_R$ is an operationally defined fraction that represents the result of a 15-minute digestion with $SnCl_2$, a strong reducing agent that converts Hg(II) to elemental Hg^0 so that the readily available Hg(II) fraction can be measured (Marvin-DiPasquale et al. 2006, Marvin-DiPasquale and Cox

2007). Experiments with mercury in a variety of model compounds representing a wide range of mercury species indicate that solid phase $Hg(II)_R$ appear to be a good predictor of microbial MeHg production (Bloom et al. 2006).

3.1.2.1 Mercury Speciation

In the Coast Range mercury deposits, the most abundant natural form of mercury is cinnabar, HgS (Rytuba 2003). In mine wastes including calcines derived from roasting mercury ores, the most common forms of mercury are metacinnabar (the high-temperature polymorph of HgS), and more soluble minerals such as mercury chlorides, sulfates, and oxides (Kim et al. 2000). Mercury condenser soot and cyclone dust represent a small proportion of mercury mine waste but contain very high mercury concentrations (Rytuba 2003); forms of mercury in these settings include elemental mercury, metacinnabar, and mercury chlorides and sulfates (Kim et al. 2000).

In the Sierra Nevada, elemental mercury was used extensively for gold recovery in both placer and hard-rock mining operations (Alpers and Hunerlach 2000, Churchill 2000, Alpers et al. 2005a). Elemental mercury and gold-mercury amalgam have been observed in sluices (sites of gold processing) at placer gold mines (Hunerlach et al. 1999; Alpers et al. 2005b), in creeks and rivers draining gold mine sites and in sediments impounded behind dams several miles downstream of gold mines (Hunerlach et al. 2004, Alpers et al. 2006). In the Clear Creek watershed (Shasta County, Klamath Mountains), transformation of elemental mercury in gold dredger tailings to other forms including mercury sulfide was documented (Ashley et al. 2002).

Mercury flux to the atmosphere from soil, rocks, and plants, and water bodies tends to be in the form of elemental mercury vapor [$Hg(0)_{(g)}$]. Dissolved gaseous mercury (DGM), or $Hg(0)_{(aq)}$ is the form of Hg most likely to evade to the atmosphere. In the Florida Everglades, Krabbenhoft et al. (1998) documented relatively low DGM concentrations in surface waters at night, followed by increases of DGM by a factor of 7 to 10 during daylight hours. The variations were attributed to photochemical reactions causing reduction of Hg(II) to Hg(0). In the atmosphere, Hg(0) is (re)oxidized to various oxidized gaseous Hg(II) compounds, or reactive gaseous mercury (RGM) (Lindberg et al. 2007). The mercury in wet and dry deposition consists largely of Hg(II).

The pool of $Hg(II)_R$ in sediment can be strongly influenced by the cycling of other elements, especially sulfur. In a study within the Delta of tidal wetlands at Frank's Tract and the Cosumnes River, $Hg(II)_R$ was inversely correlated with the microbial sulfate reduction rate (SRR), measured with radiolabeled ^{35}S (Marvin-DiPasquale et al. 2007).And in tidal wetland ecosystems of North San Francisco Bay, Yee et al. (2005) noted an inverse correlation between $Hg(II)_R$ and total reduced sulfur (TRS). These relationship indicate that hydrogen sulfide produced by sulfate-reducing bacteria has combined with Hg(II) to form $HgS_{(s)}$, removing (at least temporarily) Hg(II) from the pool of reactive mercury available for methylation and bioaccumulation.

With regard to management implications, Marvin DiPasquale et al. (2005) noted that, "There is clearly a balance between microbial activity and the availability in Hg(II) for methylation, that needs to be recognized when considering the impact of a particular management action on net MeHg production. However, we are only now starting to truly appreciate this balance, on how particular landscape manipulations might impact Hg cycling over the long term."

3.1.2.2 Water and Sediment Properties

Various chemical and physical properties of water and sediment influence the mercury cycle. In sediment, the **grain size distribution** and **mineralogy** affect the surface area and the nature of the species available for chemical reactions such as dissolution/precipitation, oxidation/reduction, and adsorption/desorption. The mineralogy of sediment can affect the speciation of Hg and other constituents that influence geochemical processes. **Porosity** and **hydraulic conductivity**, which depend on grain size distribution and **organic content**, affect the movement of ions in sediment pore waters by diffusive and advective processes.

In the water column, **salinity and pH** are among the major drivers affecting the aqueous speciation of inorganic mercury (Barkay et al. 1997). Mercury-chloride and hydroxide complexes are fairly strong, so the solubility of inorganic Hg is enhanced by higher concentrations of chloride and/or higher pH. In environments with relatively low dissolved organic carbon, some of the most abundant Hg(II) species are: $HgCl_{2(aq)}$, $Hg(OH)Cl_{(aq)}$, and $Hg(OH)_{2(aq)}$ (Powell et al. 2005).

Dissolved oxygen (DO) can affect the **oxidation-reduction (redox) state of mercury** and other elements that are commonly important in mercury cycling such as **sulfur, iron,** and (to a lesser extent) **manganese**. To a large degree, the influence of DO on mercury cycling is manifested by its influence on microbial communities. Environments with low DO (anoxic and suboxic conditions) in the water column or shallow sediment favor the activity of sulfate-reducing and iron-reducing bacteria, which are thought to be primarily responsible for Hg(II) methylation (Ullrich et al. 2001, Munthe et al. 2007).

Reduced sulfur compounds, such as hydrogen sulfide, play an important role in mercury speciation in anoxic environments. The solubility of solid HgS is fairly low, so hydrogen sulfide produced by sulfate reduction in sediments or in the water column can combine with aqueous Hg(II) to produce solid HgS. The neutral aqueous complexes $HgS^0_{(aq)}$ and $Hg(SH)_2{}^0_{(aq)}$ may play an important role in Hg(II) methylation if the hypothesis is correct that Hg(II) methylation is driven primarily by neutral mercury species diffusing through cell walls of sulfate-reducing bacteria (Benoit et al. 1999b, Benoit et al. 2003, Drott et al. 2007).

The effects of **nutrients** (considered here as various forms of nitrogen and phosphorus) on the mercury cycle are indirect in the sense that the nutrients affect biological and microbiological activity which influence mercury cycling. With regard to the formation of $Hg(II)_R$, nutrients play a role in sustaining the activity of Hg-oxidizing microbes. Seasonal supply of limiting nutrients plays a key role in the timing of phytoplankton (algal) blooms, which drive many aspects of biogeochemical cycling in the aquatic environment, including the distribution of DO, redox, and **organic carbon**.

3.1.2.3 Organic Carbon

There are several geochemical interactions that make **organic carbon** an important part of the mercury cycle. Inorganic Hg(II) forms strong aqueous complexes with dissolved organic matter (DOM) (Gill and Bruland 1990, Reddy and Aiken 2000, Haitzer et al. 2002 and 2003, Miller et al. 2007). Mercury has a strong affinity for bonding with reduced sulfur sites on organic matter. These sulfur sites are generally more abundant in the environment that the concentration of mercury in most natural waters (Ravichandran 2004).The kinetic rate of dissolution of cinnabar ($HgS_{(s)}$) is

greatly enhanced by the presence of certain organic acids (Ravichandran 2004, Waples et al. 2005). Organic matter can also inhibit the precipitation and aggregation of Hg sulfides (Ravichandran et al. 1999) which should enhance the pool of $Hg(II)_R$ available for methylation. DOM also plays a key role also in the photochemical reduction of Hg(II) to Hg(0) and the subsequent reoxidation of Hg(0) to Hg(II), affecting loss by volatilization (Ravichandran 2004, O'Driscoll et al. 2006). Overall, dissolved inorganic Hg concentrations are enhanced in aqueous environments with high DOM concentrations, such as **wetland habitats**.

3.1.2.4 Relation of Reactive Hg(II) Formation to Habitat

Measurement of $Hg(II)_R$ concentrations in sediment is a relatively new practice, with a standardized method being recently developed (Marvin-DiPasquale and Cox 2007). Comparison of preliminary data for $Hg(II)_R$ from Bay-Delta habitats indicates that $Hg(II)_R$ concentrations are relatively high in oxidized environments, and relatively low in reduced environments. The latter effect is likely caused by complexation of Hg(II) with $H_2S_{(aq)}$ (or the complexation with solid phase reduced sulfur minerals) formed by sulfate-reducing bacteria to form secondary Hg-sulfides (Marvin-DiPasquale et al. 2007 and unpub. data).

An environmental factor that likely increases the concentration of $Hg(II)_R$ is episodic wetting and drying. During dry periods, exposure to atmospheric O_2 enhances the oxidation rate of Hg-bearing mineral grains and of discrete particles of $Hg(0)_{(l)}$, if present. Periodic wetting and drying at different time scales are characteristics that distinguish several important habitats in the Bay-Delta. As discussed in **section 3.1.1** [Microbial Methylation of Mercury(II)], there appears to be a pattern of relatively low levels of methylmercury in habitats that are perennially flooded (such as "open water" zones), moderate concentrations of methylmercury in habitats that flood frequently and do not fully dry between inundation events (such as low elevation tidal marsh), and relatively high levels of methylmercury in areas that flood at less frequent intervals so that more complete drying may take place between inundation events (e.g. seasonal **floodplains** and **high tidal marsh**). These relationships may be caused in part by the formation of $Hg(II)_R$ by oxidation of Hg(0) and other reduced forms of Hg during the dry periods.

3.1.2.5 Hydrodynamics and Sediment Transport

Transport of particulate material plays an important role in the cycling of inorganic mercury in surface waters (Grigal 2002). In San Francisco Bay, there is generally a good correlation between concentrations of total mercury and suspended sediment in unfiltered water (e.g. Conaway et al. 2003). The Sacramento River is the main contributor of water, suspended sediment, and inorganic mercury to the Delta, comprising about 80% of the annual load of each of these constituents (Wood et al. 2006, Stephenson et al. 2007). In water samples from the Sacramento River, the Delta, and San Francisco Bay, the proportion of inorganic mercury that does not pass through a 0.45 μm filter is variable, but has a median value of about 90% (Domagalski 1998 and 2001, Conaway et al. 2003, Choe et al. 2003). Based on ultrafiltration studies (Choe et al. 2003), of the filter-passing material, about half is "dissolved" (< 1 kiloDalton or kDa) and about half is "colloidal" (1 kDa to 0.45 μm).

3.1.2.6 Loss of Reactive Hg(II)

Once $Hg(II)_R$ has formed, various biogeochemical processes can make it unavailable to Hg(II)-methylating microbes either by transformation to another form of Hg or by physical sequestration. Photoreduction of Hg(II) to Hg(0) has been shown to be a significant process in both saltwater

(Costa and Liss 2000) and freshwater environments (Siciliano et al. 2002). Other abiotic process that can reduce Hg(II) to Hg(0) are reduction by humic substances (Allard and Arsenie 1991, Matthiessen 1998) and reduction by sulfite (Munthe et al. 1991). Microbial reduction of mercury from Hg(II) to Hg(0) is attributed to activity of the mercuric reductase (MerA) enzyme (Chadhain et al. 2006).

Another proecess that can lower available concentrations of $Hg(II)_R$ is reaction with reduced sulfur ($H_2S_{(aq)}$ or $HS^-_{(aq)}$) resulting in the formation of amorphous or poorly crystalline $HgS_{(s)}$, or the formation of aqueous Hg-S complexes that are less susceptible to methylation (Benoit et al. 1999a and 1999b, Drott et al. 2007). In environments with elevated sulfate concentrations and relatively reducing conditions, sulfate-reducing bacteria (SRB) may produce abundant sulfide that combines with Hg(II), resulting in lower concentrations of $Hg(II)_R$ in sediment, as shown in the Central Delta (Frank's Tract) by Marvin-DiPasquale et al. (2007).

In tributaries to the Delta affected by historical gold mining (i.e., most Sierra Nevada rivers), some Hg(0) combines with gold (Au) to form gold-mercury amalgam (AuHg) (e.g. Alpers et al. 2006). The reactivity of AuHg is not well known, but it is probably lower than that of Hg(0)(Bloom 2003).

Another important process that leads to the sequestration of $Hg(II)_R$ is **sedimentation**. Settling of particles in depositional areas or trapping of particles in tidal wetlands may effectively isolate the reactive mercury and prevent it from transforming to MeHg.

3.1.2.7 Mercury-plant interactions and loss of Hg(0) to the atmosphere

Like other metals, mercury concentrations in wetland plant tissues tend to be greatest at uptake sites in plant roots, decreasing from roots to stems to leaf tissues (Cocking et al. 1995, Frescholtz et al. 2003, Windham et al. 2003). Root uptake is enhanced by sulfur-based ligands and humic acids (Moreno et al. 2005) and rhizospheric bacteria (de Souza et al. 1999), but given mercury's low vapor pressure, another route of plant uptake can include leaf-atmosphere exchange (Weis et al. 2002). In soil and water, evasion is positively correlated with UV radiation and moisture (Lindberg et al. 2007), whereas in plants, Hg(0) flux is bidirectional, and is controlled actively by stomatal conductance and passively by concentration gradients between the atmospheric boundary layer and the leaf interior (Leonard et al. 1998a). Species-specific compensation points appear to regulate passive Hg(0) flux based on concentration gradients (Hanson et al. 1995), but recent work has identified active reduction of Hg(II) in plant leaves such that some Hg(0) production may be a byproduct of photosynthetic electron transfer (Battke et al. 2005). Net Hg(0) flux in wetland vegetation tends to be positive (emission; Lindberg et al. 2002, Poissant 2002, Zhang et al. 2006) although net foliar uptake has also been observed (Fay and Gustin 2007). Further, the rates of mercury flux through plant tissues tends to be far greater than uptake and storage alone (Leonard et al. 1998b). Therefore, one effect of vegetation on mercury cycling in terrestrial and aquatic ecosystems is the impact of vegetation on the pool of $Hg(II)_R$ (Windham-Myers and Marvin-DiPasquale 2007, Windham-Myers et al. in review)

In the Delta, a comparative study of mercury-plant interactions in the central Delta region (Frank's Tract) and an eastern tributary region of the Delta (Cosumnes River / McCormack Tract), Marvin-DiPasquale et al. (2007) identified regional differences in submerged aquatic vegetation (SAV) community characteristics and Hg interactions. SAV from the Cosumnes River region had higher Hg levels in their tissue (per unit weight) than those from the Frank's Tract region, however the

overall Hg pools in SAV were about equal on an area basis because of higher vegetation density at Frank's Tract. Total-Hg flux through the plant biomass pool appeared faster in Franks Tract, compared to the Cosumnes River, because of faster plant turnover and decomposition rates and higher ionic strength water conditions.

A limited number of gaseous mercury [$Hg(0)_{(g)}$] flux rates have been measured in the Delta from leaf surfaces of emergent marsh plants and from open water surfaces (Marvin-DiPasquale et al. 2007, Stephen Peters, Lehigh Univ., unpublished data). Net emissions were observed from both water and plant surfaces, and comparatively, maximum Hg(0) flux rates (ng m^{-2} hr) observed from *Scirpus acutus* leaf surfaces (March and July 2005) were approximately 2x higher than maximum rates measured from water surfaces (October 2006). The flux of $Hg(0)_{(g)}$ associated with water surfaces was entirely dependent on incident UV radiation, whereas $Hg(0)_{(g)}$ fluxes from vegetation were strongly correlated with transpiration rates (and CO_2 uptake). Based on these data, Marvin-DiPasquale et al. (2007) concluded that large zones of emergent vegetation could represent relatively significant areas of Hg(0) flux, compared to the flux associated with nearby water-air interfaces.

Mercury release through salt excretion has been observed in halophytes of the eastern U.S. (Kraus 1986, Windham et al. 2001) and in recent studies in the San Francisco Bay (Green 2002, Yee et al. 2007). Salt glands (hydathodes) serve as a mechanism to regulate sodium balance in some salt tolerant plant species (*Avicennia, Spartina, Atriplex, Distichlis*) and can exude other mono- and divalent cations in the process of exuding salt. Hg concentrations salt exuded from *Distichlis spicata* (spikegrass) were correlated with sediment concentrations (Green 2002), and rates of Hg release were 20-fold greater than rates of atmospheric deposition alone (L. Windham-Myers, U.S. Geological Survey, written commun. 2007). The importance of this redistribution of sediment Hg into surficial, reactive Hg(II) salts is unclear, but may represent up to 5% of the static pool of reactive mercury measured in surface soils (0-2 cm depth) (Windham-Myers et al. in review).

3.1.3 Methylation of Mercury(II)

The net formation of monomethylmercury (MeHg) in sediment and/or water is the result of competing microbiological and abiotic reactions, where the net MeHg production rate equals the total gross rate of inorganic divalent Hg(II) methylation minus the total gross rate of MeHg degradation. Multiple biogeochemical processes can convert Hg(II) to MeHg as well as degrade MeHg back to inorganic Hg(II), and while microbiologically mediated processes tend to dominate in natural environments, abiotic processes for both MeHg formation and degradation have been described.

3.1.3.1 Microbial mercury(II) methylation

It is generally accepted (Ullrich et al. 2001, Munthe et al. 2007) that the methylation of Hg(II) in aquatic environments is carried out primarily by **sulfate-reducing bacteria** (SRB; Gilmour et al. 1992, Benoit et al. 2003), although recent reports have confirmed that some **iron-reducing bacteria** (IRB), which reduce iron from ferric [Fe(III)] to ferrous [Fe(II)], also can carry out this process (Fleming et al. 2006, Kerin et al. 2006). Both microbial groups are anaerobes, meaning that they are not active in strongly oxygenated environments. This is one reason that the production of MeHg most commonly occurs in anoxic (oxygen-depleted) surface sediment, and not in the water column,

which generally contains dissolved oxygen (DO) in well mixed aquatic systems (e.g. rivers and the tidally flushed delta). The second reason that MeHg production largely occurs in sediments is that bacteria in general are much more concentrated in sediments than in the water column on a volumetric basis. **Hypoxic zones** in relatively deep water (e.g. the hypolimnion zone of a stratified reservoir or the Stockton Deep Water Ship Channel) are environments where SRB and IRB may be active in the water column.

Two broad classes of factors control microbial Hg(II) methylation: a) those that impact the activity of Hg(II)-methylating bacteria, and b) those that impact the availability of inorganic Hg(II) to those Hg(II)-methylating bacteria. The environmental factors that influence microbial rates of Hg(II) methylation are to a large degree those factors that influence the activity of SRB (King et al. 1999) and IRB (Fleming et al. 2006, Kerin et al. 2006). In general terms, factors that control the activity of microbes in general include temperature, pH, the availability of electron donors (e.g. usable organics) and electron acceptors (e.g. $O_{2(aq)}$, Fe(III), sulfate, and others). The last category, the availability of electron acceptors, generally dictates which types of bacteria are present and active in a given place at a given time. Iron-reducing bacteria and sulfate-reducing bacteria depend on the availability of Fe(III) and sulfate, respectively. Because sulfate is a major constituent of seawater, and is generally in low concentrations in freshwater systems, there is a sulfate gradient that parallels the salinity gradient in estuarine systems like the SF Bay-Delta. The Delta represents the mixing zone of freshwater and higher salinity bay water, and thus is a transition point where the activity of SRB tends to be higher than in the freshwater riverine reaches of the system. Subsequently, the very geophysical, chemical, and hydrological spatial distributions inherent in a complex system such as the SF Bay-Delta dictate the activity and the distribution of major microbial groups that represent a major control on the Hg(II)-methylation process.

The geochemical cycle of **sulfur** is intimately linked to that of mercury, and the balance of sulfate and sulfide is a key control on Hg(II) methylation (Munthe et al. 2007). Addition of sulfate has been shown to stimulate Hg(II) methylation in various laboratory experiments, in sediment and soil amendments, and in field amendments to lakes and wetlands (see Munthe et al. 2007 and references within). The higher concentrations of sulfate in the Central Delta, compared with the incoming rivers, leads to higher rates of microbial sulfate reduction in the Central Delta (Marvin-DiPasquale et al. 2007). However, the major end-product of the sulfate-reduction process is sulfide, and an excess of sulfide has been shown to slow down rates of Hg(II) methylation (Gilmour et al. 1992, Benoit et al. 1999b, Marvin-DiPasquale and Agee, 2003). In part, this may be caused by the sequestration of $Hg(II)_R$ (Marvin-DiPasquale et al., 2007; Yee et al. 2007). Thus, while the Central Delta has generally higher rates of microbial sulfate reduction, compared to river sediments (e.g. the Cosumnes R.) it has on average a lower pool size of $Hg(II)_R$ available to those SRB that can carry out Hg(II) methylation, and subsequently has lower measured rates of MeHg production (Marvin-DiPasquale et al. 2007). Differences in MeHg concentrations and production rates in various Bay-Delta habitats are discussed in **Section 3.1.3.3** (Relation of methylmercury formation to habitat).

Iron is a ubiquitous element with a variable oxidation state (i.e. 0, +2, +3). The distribution of fully oxidized or ferric iron [Fe(III)] is important with regard to sorption of trace elements including mercury, and is the main electron acceptor for IRB. Ferrous iron [Fe(II)], the end product of microbial Fe(III)-reduction, is relatively soluble at neutral pH, and can be abundant in pore water. Fe(II) is susceptible to oxidation to Fe(III) upon exposure to atmospheric oxygen or dissolved oxygen in pore water, which is abundant in the root zones of plants in several Delta and tributary habitats. At neutral pH, Fe(III) is relatively insoluble, and its formation leads to precipitation of amorphous and poorly crystalline hydrous Fe(III) oxides (HFO) which are very fine grained and

have a large reactive surface area that can be important in sorption of trace metals including Hg and MeHg. The activity of IRB will dissolve HFO, releasing sorbed ions to solution. Compared to the numerous studies implicating SRB as the primary microbial group responsible for Hg(II) methylation, much less is know about the role of IRB in this process, which may be locally important in regions of the system that have particularly high iron concentrations (e.g. upstream mining sites).

Oxidation-reduction (or **redox**) state is also an important environmental factor that can influence MeHg projection and concentration. In most low-temperature environments, the 'system Eh" is difficult or impossible to define, as kinetic rates preclude the equilibration of multiple redox couples such as sulfate–sulfide, nitrate–nitrite–ammonia, ferrous–ferric iron, CO_2–methane, and others (Lindberg and Runnells, 1984). Therefore, it is important for each redox-sensitive chemical species to be measured independently, because concentrations of individual redox species cannot be estimated reliably from total elemental concentrations and platinum electrode (Eh) measurements (Nordstrom and Alpers, 1999).

In lake environments, low pH (e.g. 5-6) and increased acidity have been linked to Hg(II) methylation and methyl Hg bioaccumulation (e.g. Wiener et al. 2006). However, pH variation is not considered to be an important factor affecting mercury cycling in the Bay-Delta generally, but may be important in particular sites that are prone to low pH (e.g. those associated with acid mine drainage in some tributaries).

3.1.3.2 Abiotic mercury(II)-methylation

In addition to microbial mediated Hg(II) methylation, a number of abiotic methylation pathways have been demonstrated, including those via Hg(II) reaction with humic matter (Weber 1993) or acetic acid (Gardfeldt et al. 2003), and sunlight mediated Hg(II)-DOM interactions (Siciliano et al. 2005). Ravichandran (2004) indicated that more study of abiotic Hg(II) methylation in wetlands is needed; he hypothesized that "…where organic matter is largely labile and readily biodegradable, it may promote methylation by stimulating microbial growth, and when the organic matter is relatively recalcitrant and consists of high molecular weight humic and fulvic acids, then it may contribute to abiotic methylation." While the relative contribution of abiotic Hg(II) methylation to total net MeHg production is thought be minor in most environments (Ullrich et al. 2001), there still exits a substantial amount of uncertainty regarding the importance of these abiotic processes in specific environments, such as the San Francisco Bay-Delta.

3.1.3.3 Degradation of methylmercury

Once formed, a molecule of MeHg can either bioaccumulate, degrade to an inorganic Hg(II), or be complexed with sediment organic material and be buried (Alpers et al. 2004). There are both biotic and abiotic mechanisms of MeHg degradation (also known as mercury demethylation). Microbial MeHg degradation may proceed by either a reductive (Robinson and Tuovinen 1984, Hobman and Brown 1997, Hobman et al. 2000) or an oxidative (Oremland et al. 1991, 1995; Marvin-DiPasquale and Oremland 1988) pathway. In the reductive pathway the organic methyl (CH_3-) group is cleaved from the MeHg molecule by conversion to methane ($CH_{4(g)}$), and is often followed by a reduction of the remaining Hg(II) to Hg(0). This is indicative of an enzymatic detoxification response of bacteria to potentially toxic MeHg. In the oxidative pathway, the methyl group is oxidized to $CO_{2(g)}$ with no know subsequent conversion of the remaining Hg(II) to Hg(0). Thus, for oxidative demethylation,

the remaining Hg(II) is potentially available for re-methylation. A number of studies have noted that the reductive pathway is more common in extremely Hg contaminated settings, while the oxidative pathway is generally more common in areas with low to moderate mercury levels (Marvin-DiPasquale et al. 2000, Schaefer et al. 2004). Studies in the San Francisco bay suggest that the oxidative pathway generally dominates the microbial MeHg degradation process (Marvin-DiPasquale and Agee 2003, Marvin-DiPasquale et al. 2003).

An important abiotic mechanism of methylmercury MeHg is photodemethylation (Sellers et al. 1996, Gardfeldt et al. 2001). Photodegradation of MeHg has been identified as a significant process affecting the mass balance of MeHg in the Delta (Stephenson et al. 2007). The absolute rates of MeHg photodegradation in the Delta remain uncertain.

3.1.3.4 Relation of methylmercury formation to habitat

The general association of methylmercury with wetlands is well established (e.g. Krabbenhoft et al. 1999, Langer et al. 2001, Lacerda and Fitzgerald, 2001), as is the initial rise in methylmercury concentrations in newly flooded reservoirs (Kelly et al. 1997, St. Louis et al. 2004). In the Delta and its tributaries, a wide variety of habitats are present. Patterns are beginning to emerge with regard to the distribution of MeHg in water, sediment, and biota with regard to habitat in the Bay-Delta region. Table 2 summarizes the present knowledge with regard to MeHg exposure as a function of habitat in the Bay-Delta region. Also included in Table 2 is a summary of flooding characteristics for each habitat. Episodic flooding is a characteristic that may be associated with increased MeHg exposure (Gilmour et al. 2004). There may be also an association between MeHg exposure and time of year that areas that are flooded; those flooded during relatively warm periods of the year may be areas where MeHg will be preferentially produced and bioaccumulated due to enhanced micro- and macro-biological activity during warm periods.

Overall, the habitats with the highest levels of MeHg production, concentration, and exposure to biota are those with periodic flooding events separated by sufficient time to allow complete drying. This may have to do with formation of $Hg(II)_R$ and the re-oxidation of reduced sulfur compounds during dry periods, as discussed in **section 3.1.2.4** [Relation of Reactive Hg(II) Formation to Habitat]. Experimental removal of wetland plants in multiple wetlands also demonstrates the importance of rhizosphere activity on microbial methylation rates (Windham-Myers and Marvin-DiPasquale 2007).The habitats with relatively high of MeHg concentrations in sediments include floodplains which are typically flooded intermittently during later winter and spring, and high-elevation tidal marshes which are flooded only during the highest ("spring" and "perigean") parts of the lunar-tidal cycle. Lower elevation tidal marshes that are wetted on a daily basis tend to have lower MeHg concentrations, based on observations in the Napa-Petaluma area (Yee et al. 2005, 2007). Perennially flooded habitats such as open-water zones with various types of aquatic vegetation (submerged, floating, and emergent) tend to be lower in MeHg in water and sediment than seasonally flooded habitats (Table 1). In the case of submerged and emergent vegetation, the large range in water-column MeHg concentrations is indicative of the influence of tributaries contributing high aqueous MeHg concentrations to the water column in areas such as the Cosumnes River compared with much lower concentrations of aqueous MeHg in the Central Delta (Marvin DiPasquale et al. 2007).

Table 2. Relations between habitats and methylmercury concentrations in sediment and overlying water in the San Francisco Bay-Delta region

[L, low; M, moderate; H, high; NA, not applicable]

Habitat	MeHg	MeHg	Flooding Characteristics			References
	Overlying water	Sediment	Flooding mode	Months typically flooded (wet year)	Conditions during episodic flooding Cool / Warm / Hot (wet year)	
Aquatic vegetation: Submerged	L – H	M	Perennial	All	All	1
Aquatic vegetation: Emergent	L – H	M	Perennial	All	All	1, 9
Aquatic vegetation: Floating	M ?	M ?	Perennial	All	All	
Aquatic perennial (open water)	L	L	Perennial	All	All	1,2
Tidal marsh: High Elevation	M?	H	Episodic (2x/month)	All	All	2,3,4,5
Tidal marsh: Low-Medium Elevation	L – M	L – M	Episodic (2x/day)	All	All	4,5
Riparian (woody)	L – M?	L – M?	Episodic (seasonal)	Jan-Mar (Dec-May)	Cool (to Warm)	
Floodplains	H	H	Episodic (seasonal)	Jan-Mar (Dec-May)	Cool (to Warm)	1
Managed wetlands	?	?	Episodic or perennial	Variable	Variable	
Seasonal wetlands: ag lands, seasonally flooded (rice)	M – H?	M – H?	Seasonal	Apr-Sep	Warm to Hot	6,7
Seasonal wetlands: non-ag, vernal pools	?	?	Seasonal	Jan-Mar (Dec-May)	Cool (to Warm)	
Seasonal wetlands: non-ag, duck clubs	H	H	Seasonal	Oct-Mar	Cool	7
Shaded riverine aquatic	M?	M?	Perennial	All	All	
Inland dune scrub	L – M?	L – M?	?	?	?	
Perennial grassland	L – M?	L – M?	?	?	?	
Agricultural lands, not seasonally flooded	M	M – H	irrigated	?	?	
Natural shorelines	M – H?	M – H?	Episodic (variable)	Variable	All	
Reservoirs: Old	M	L – M	Episodic (seasonal)	Jan-Jun	Cool to Warm	8
Reservoirs: Newly flooded	H	H	Episodic (seasonal)	Jan-Jun	Cool to Warm	

References: 1,Marvin-DiPasquale et al. 2007; 2, Stephenson et al. 2007; 3, Heim, 2003; 4, Yee et al., 2005; 5, Yee et al. 2007; 6, M. Marvin-DiPasquale, U.S. Geological Survey, written commun., 2007; 7, M. Stephenson, California Dept. of Fish and Game, written commun. 2007; 8, Alpers et al. in press.; 9 Windham-Myers and Marvin-DiPasquale 2007

3.1.4 Limitations and Recommendations

Several studies, including those funded by CBDA, have generated high-quality data regarding the concentration of MeHg in water and sediment through the Bay-Delta and its tributaries during that past decade. However, there is no comprehensive synthesis of these data. Some important sources of data that should be compiled and analyzed include: Choe et al. 2003 and 2004, Conaway et al. 2003, Heim, 2003, Wood et al. 2006; Stephenson et al. 2007, Marvin DiPasquale et al. 2007, Yee et al. 2005, 2007.

3.2 Submodel #2: Methylmercury Bioaccumulation

Submodel #2, depicting the bioaccumulation of methylmercury, is shown in Figure 4.

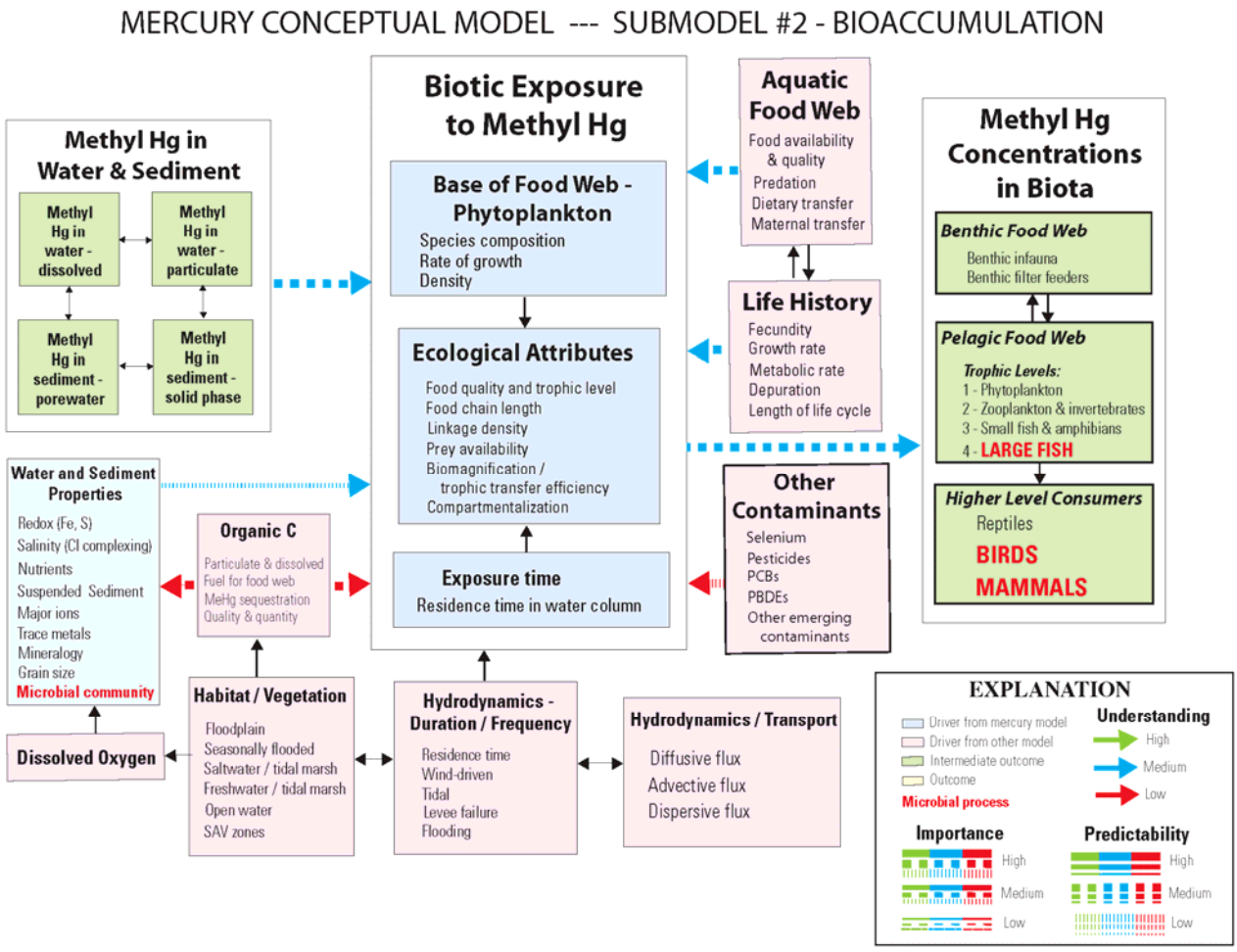

Figure 4. Mercury Submodel #2 — Methylmercury bioaccumulation. This submodel shows the drivers and linkages that relate methyl Hg (MeHg) concentrations in water and sediment with methyl Hg concentrations in biota. Biotic exposure to MeHg at the base of the food web is a critical step in bioaccumulation. Once the MeHg concentration in phytoplankton is established, other ecological attributes and a variety of other drivers determine the MeHg concentration in higher trophic level organisms.

3.2.1 Physical-Chemical Properties of Mercury That Determine Bioavailability

There are two main components to mercury bioavailability. First is the bioavailability of various inorganic mercury compounds to the microbes that produce methylmercury (see section 3.1). Here we discuss the bioavailability of methylmercury once it is produced, in relation to the food web.

Physical-chemical properties of methylmercury that effect its bioavailability to the food web include its solubility in water, its ability to cross cell membranes, and its propensity to form strong chemical bonds with biological proteins (Wiener and Spry 1996). Once dissolved in water, methylmercury may interact directly with the benthic and pelagic food webs. The ability of methylmercury to cross cell membranes is one of the key processes driving bioaccumulation, both through algal uptake at the base of the food web as well as subsequent trophic transfers from prey to predators. The propensity of methylmercury to form strong chemical bonds with biological proteins is the main factor leading to its high retention rate, both within individual organisms and following consumption of one organism by another.

3.2.2 Physical-Chemical Properties of Habitats That Determine Bioavailability

Habitat properties can impact MeHg bioavailability through several mechanisms. Section 3.1. describes the variables that drive bioavailability of inorganic mercury to methylating microbes for transformation to MeHg; however, upon methylation, mercury accumulation into the food web is in part driven by intrinsic physico-chemical properties affecting habitats. These factors include physical properties and processes such as temperature (Odin et al. 1994), sediment resuspension and flow regime (Shanley et al. 2005), and chemical properties such as dissolved oxygen (DO) and dissolved organic carbon (DOC) (Hudson et al. 1994). Temperature has a direct impact on Hg bioavailability in that many biological processes vary with temperature. These processes include the growth of algal cells and their incorporation of methylmercury (Jackson 1988), as well as the feeding rates of heterotrophs in the food web (Post et al. 1996, Harris and Bodaly 1998). Temperature differences in the water column can also result in lake stratification during the warm season, and the subsequent development of anoxic conditions in the bottom waters which causes the production and build up of methylmercury directly in the water column (Watras et al. 1995). This aqueous methylmercury can be highly bioavailable once it is incorporated into the upper waters, as compared to methylmercury produced deep within the sediments (Slotton 1991, Slotton et al. 1995). The flow regime can affect bioavailability by transporting methylmercury from one place to another (Driscoll et al. 2007). For example, following the flooding of normally dry areas by the San Joaquin River in 2006, flow transported high concentrations of newly produced methylmercury many miles downstream, where the elevated exposure resulted in elevated bioaccumulation in small fish (D, Slotton et al., UC Davis, unpublished data, recent CALFED-sponsored studies). Wind-driven resuspension of bottom sediments can transport sediment-bound mercury and can also move methylmercury-adsorbing particles into the water column, rendering aqueous methylmercury less bioavailable to the food web (Kim 2004). Similarly, constituents of the sediments, including many organic compounds, may act as competing binding sites for methylmercury, slowing or stopping its diffusion out of the sediments and into pore water, overlying water, and exposure to the food web (Gagnon et al. 1996, Kim 2004). In the Cache Creek watershed, seasonally-averaged aqueous methylmercury concentrations were strongly linked to biotic methylmercury accumulation at individual sites and among sites with similar characteristics (Slotton et al. 2004a). Correlations between MeHg concentrations in low trophic level organisms and MeHg in co-occurring adult sport fish were found to be robust across diverse sites.

3.2.3 Exposure Time

The effects of time on bioaccumulation include the overall length of exposure time as well as the specific timing of that exposure. Fish tend to accumulate greater burdens and concentrations of methylmercury over time, so older individuals typically have higher body burdens and higher

absolute concentrations of methylmercury than younger ones (Wiener and Spry 1996). However, other taxa such as birds have strong depuration and demethylation mechanisms that allow them to reduce their total body burdens annually (Scheuhammer et al. 2007). In many locales, methylmercury exposure is not steady throughout the year or from one year to the next, instead exhibiting substantial fluctuations in response to environmental stimuli. For example, high flooding years can produce bursts of elevated methylmercury exposure, leading to elevated bioaccumulation in organisms present in those years relative to others (Sorenson et al. 2005 and recent biosentinel data from Slotton et al.). Similarly, rapidly growing organisms, and those with short life-spans-such as many invertebrates and young fish, can exhibit widely varying methylmercury bioaccumulation depending on whether maximum growth occurs in a low or high exposure season (Wiener et al. 2007 and recent biosentinel data from Slotton et al.). Seasonal effects include episodic flooding in the winter and/or spring of some years, a general elevation of biological activity including Hg(II) methylation during the spring-summer (Wiener et al. 2007), and the fall turnover phenomenon in lakes that have seasonally anoxic bottom waters (Slotton 1991, Slotton et al. 1995, Watras et al. 1995). If growth rates are equal, organisms that live and grow at high exposure times will have higher levels of bioaccumulation and higher methylmercury concentrations than similar organisms that live and grow during lower exposure times.

3.2.4 Base of Food web – Phytoplankton

The single largest concentration jump in food web methylmercury bioaccumulation occurs between aqueous methylmercury and algal cells or phytoplankton, with concentration increases typically in the range of 10^5-10^6 (Mason et al. 1995, Driscoll et al. 2007, Wiener et al. 2007). Subsequent bioaccumulation to higher trophic levels typically increases the methylmercury concentration by less than an order of magnitude at each step, though it is these subsequent increases that lead to concentrations that can be harmful. In addition to supplying the largest single concentration increase, algal cells tend to distribute accumulated methylmercury in the easily-absorbed inner cell rather than within the tough, less digestible cell walls (Mason et al. 1996).

The role of phytoplankton in methyl mercury bioaccumulation is complicated because although they act as the direct conduit between aqueous methyl mercury and upper trophic level organisms, the structure of phytoplankton communities and their population dynamics can actually regulate the amount of methyl mercury that is bioaccumulated. Because Hg bioaccumulation ultimately originates at the basal trophic levels of a foodweb, variation in the standing biomass, productivity rate, or taxonomic composition of lower trophic levels may cascade through the food web and result in alterations to the bioaccumulation rate in higher trophic level species (Allen et al. 2005). Algal bloom dilution is the negative correlation between phytoplankton density and mass-specific Hg burdens (Chen and Folt 2005). As algal cell densities increase in the water column, the total amount of Hg per cell decreases (Pickhardt et al. 2002). This reduction in Hg concentration can then be propagated through the food web as demonstrated by Chen and Folt (2005) who showed clear, inverse links between chlorophyll concentrations and Hg concentrations in phytoplankton, zooplankton and fish from 20 lakes in the northeastern U.S.A. In addition, the taxonomic structure of the phytoplankton community may also be an important factor. Some algal species, such as cyanobacteria are less palatable than others to zooplankton. If a phytoplankton community is made up of a high proportion of low quality resources, then zooplankton may selectively feed on certain algal species and much of the mercury bound in the less favorable algae may then fall to the sediment. Although little work has been conducted that explicitly links phytoplankton community structure to Hg bioaccumulation, eutrophic water bodies tend to have less bioaccumulation than

those of lower productivity (Essington and Houser 2003), and many eutrophic lake systems are dominated by taxa such as cyanobacteria. Eagles-Smith et al. (in press-a) also suggest that the high abundance of cyanobacteria in Clear Lake may be related to the surprisingly low Hg concentration found in fish, given the substantial mercury inputs to the lake.

3.2.5 Organism Properties Affecting MeHg Bioaccumulation

Diet is the primary route of methyl mercury exposure in fish and wildlife (Rogers et al. 1987), and is thus the primary driver of bioaccumulation potential within a given organism. Methyl mercury accumulates in biota because of its high binding affinity to sulfhydryl proteins such as those in muscle, and its low depuration rate once bound to those proteins. Thus, in biota mercury tends to increase with age or size (Wiener and Spry 1996) and trophic position (Kidd et al. 1995). However, several other factors have been shown to influence the degree of bioaccumulation in some cases, including: growth rate (Simoneau et al. 2005), foraging habitat (Ackerman et al. 2007a, Eagles-Smith et al. in press-a, Power et al. 2002), and food web structure (Cabana and Rasmussen 1994).

Growth rate has been shown to be a potentially important factor driving mercury concentrations in fish. This is related to growth biodilution which is the result of the biomass accretion increasing relative to Hg uptake (Stafford et al. 2004, Essington and Houser 2003, Simoneau et al. 2005). Fish growth rates may vary because of age, water temperature, and diet. As growth rates change Hg concentrations will also change, even if the Hg burden (total amount of mercury in a fish) remains the same. This is an important consideration for monitoring programs because temporal or spatial changes in fish mercury concentrations may be confounded by these relationships. Without an understanding of growth rates, one may not be able to determine if variability in fish tissue mercury is the result of differences in Hg availability or fish growth rate.

Mercury can vary substantially among habitats (e.g. pelagic, profundal littoral) and the degree of foraging within each habitat may be important in driving biotic mercury concentrations. Habitat-specific foraging can expose various consumers to differential levels of contamination, particularly if one consumer has a strong linkage with prey that are more highly contaminated, resulting in a disparity in Hg concentrations in consumers occupying similar trophic positions (Power et al. 2002). This has been demonstrated with other contaminants (Stewart et al. 2004), and is likely an important mechanism for determining species and habitat-specific accumulation risk. Eagles-Smith et al. (in press-a) showed that foraging habitat was among the most important factors in determining mercury concentrations in several species of fish in Clear Lake, Calif., and was generally more important than trophic position. Similarly, in Eagle Lake, Calif., where mercury contamination is low, foraging habitat was at least as important as trophic position in determining fish mercury concentrations, though neither variable was as important as size (Eagles-Smith 2006).

The effects of both growth rate and foraging habitat are also strongly connected to the effects of food web structure in driving mercury bioaccumulation. Both within and among systems, there may be variation in the transfer of Hg from one trophic level to another. This suggests that there are inherent properties of food webs that affect Hg transfer. Food webs can be extremely complex, with extensive omnivory and a great deal of spatial and temporal variability (Winemiller 1990, Polis 1991, Lawler and Morin 1993, Dunne et al. 2002). They consist of a network of weak and strong trophic interactions (linkages) that may vary both temporally and spatially (Dunne et al. 2002). The strength, density, and habitat specificity of linkages are factors that can potentially confound

interpretations of Hg bioaccumulation because the magnitude of trophic transfer is in part dictated by source Hg concentrations and energetic strength of the trophic interaction. Moreover, the species distribution within a food web can influence Hg uptake by upper trophic level species. For example, the structure of the zooplankton community has been shown to significantly influence Hg bioaccumulation in fish because Hg concentrations vary among zooplankton species (Kainz et al. 2002). Pickhardt et al. (2005) showed in a controlled lab study that even with the same exposure regimes *Daphnia* consistently had mercury concentrations up to three times higher than those of two different copepod species. Consequently, if a system is dominated by Daphnia as opposed to copepods, Hg bioaccumulation may be elevated relative to a system with identical methyl mercury production but a zooplankton community dominated by copepods. Additionally, dietary shifts in response to changes in food web structure may alter bioaccumulation rates in fish if alternate prey sources have different Hg concentrations. For example, inland silversides and young-of-year largemouth bass in Clear Lake, CA changed their diet from primarily pelagic-based to primarily benthic-based in response to a change in food web structure that was induced by the invasion of a planktivore (Eagles-Smith et al. in press-b). Moreover, this change in diet caused silverside and bass mercury concentrations to roughly double (Eagles-Smith et al. in press-b).

All of these factors should be considered when interpreting biological monitoring data for mercury (Wiener et al. 2007). However, across broad areas of the Bay-Delta Estuary, it has been demonstrated that general spatial and seasonal trends in bioaccumulation are primarily a direct reflection of relative methylmercury exposure rather than food web or other related factors (Marvin DiPasquale et al. 2007) and that these trends are consistent across a wide range of co-occurring species (Slotton et al. 2002 and recent biosentinel data). Wiener et al. (2007) conclude that biological monitoring of consistent sentinel organisms such as small, young-of-year fish provide an optimal measure of relative methylmercury exposure, particularly when potentially confounding factors as noted above in this section are also taken into account. Prior CALFED-funded projects have demonstrated a strong linkage between methylmercury in small fish, when carefully averaged over time, and methylmercury in co-occurring adult sport fish in both the Coast Range (Slotton et al. 2004a) and Sierra Nevada (Slotton et al. 2004b). Ackerman et al. (2007a), in the current CBDA bird exposure project, found that mercury in small fish and other prey could be directly linked, with considerable effort, to resulting concentrations in piscivorous birds.

3.2.6 Biological Habitat Properties Affecting MeHg Bioaccumulation

On the regional scale of the Bay-Delta watershed, the most striking spatial pattern for both biosentinel fish (Mississippi silversides; fig. 5; Slotton et al. 2002) and adult sport fish (Davis et al. 2003a, 2008) has proven to be a noted elevation in concentrations in the tributaries as compared to markedly lower levels in the central and southern Delta . Biosentinel data also indicate a secondary increase moving west into the North Bay (fig. 5).

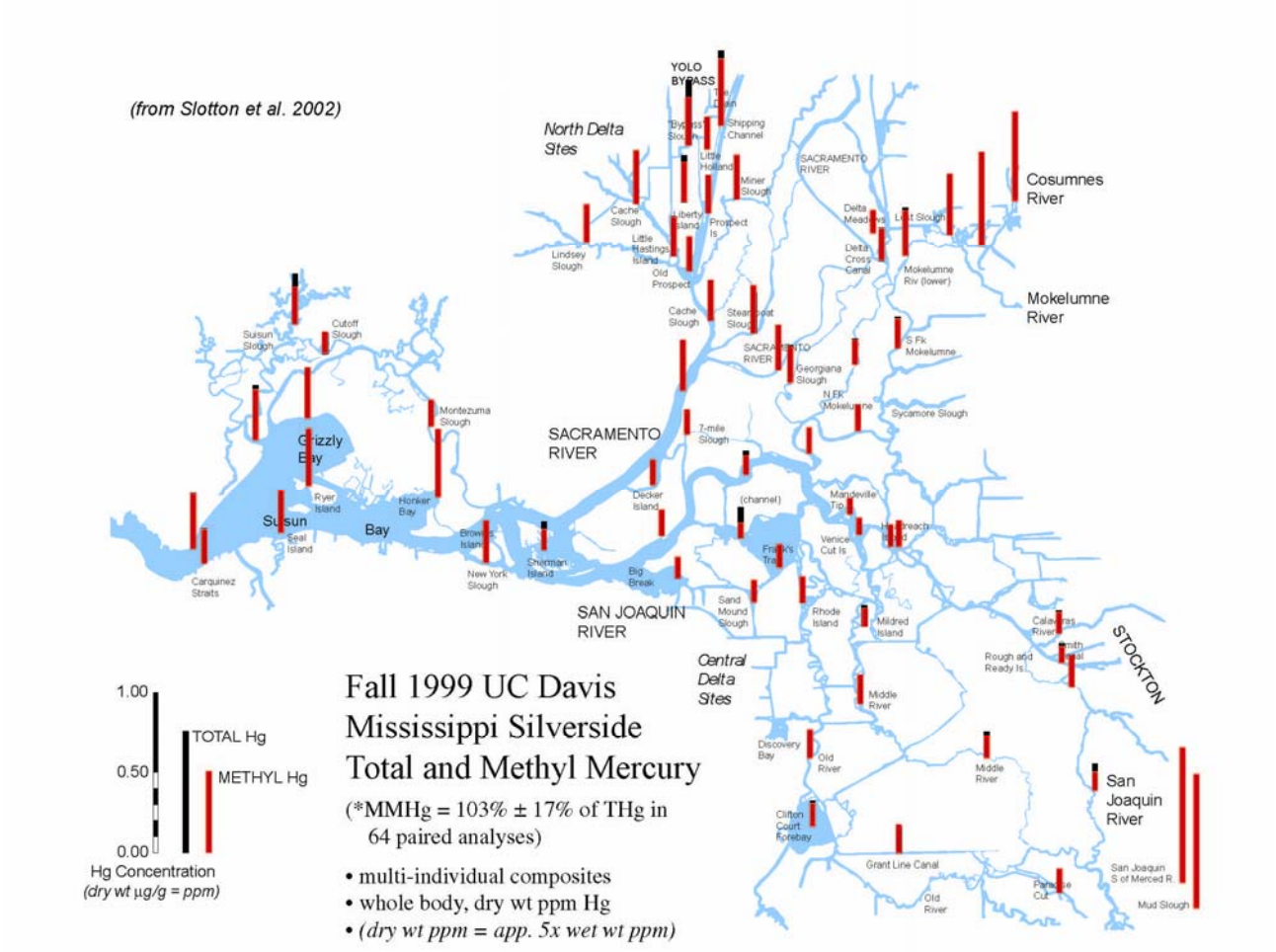

Figure 5. Map showing distribution of total mercury and methylmercury concentrations in Mississippi silversides in the Delta, its main tributaries, and San Pablo Bay (from Slotton et al. 2002).

Melwani et al. (2007) used a GIS approach in an attempt to relate landscape features to adult sport fish mercury concentrations on a large regional scale within the Bay-Delta watershed. They found no obvious correlations between fish mercury concentrations and atmospheric deposition, historic mining districts, or wetlands at the whole watershed scale. At the proximal scale, vegetated wetland and temporarily inundated aquatic habitat appeared to have some relationship with fish mercury concentrations, however their tentative conclusions were based on relatively small sample sizes and contradict the prevailing notion that wetlands generally increase methylmercury accumulation in the food web.

Recent and ongoing biosentinel monitoring, using large sample sizes, indicates some patterns in which the influences of different wetland environments are apparent. One of the most significant factors identified to date in relation to elevated MeHg exposure and bioaccumulation is the episodic flooding of previously non-flooded soils (Fig. 6). This effect was most notable in association with infrequent, large-scale flooding of relatively dry soils, such as occasional floodplain inundation, increasing local as well as downstream exposure. Episodic flooding as an exposure elevating factor was also associated with seasonally flooded managed wetlands and was implicated in the noted elevated exposures associated with high tidal marsh habitats, where tidal flooding is relatively

infrequent compared with lower elevation tidal marshes. Ongoing biosentinel monitoring (D. Slotton et al., UC Davis, unpublished data) indicates that other important factors include the timing of flooding (larger effect in the warm season) and the relative vegetation cover and organic matter of the flooded land. Proximity to mining-related mercury loading sources was also found to be an important factor across all habitats.

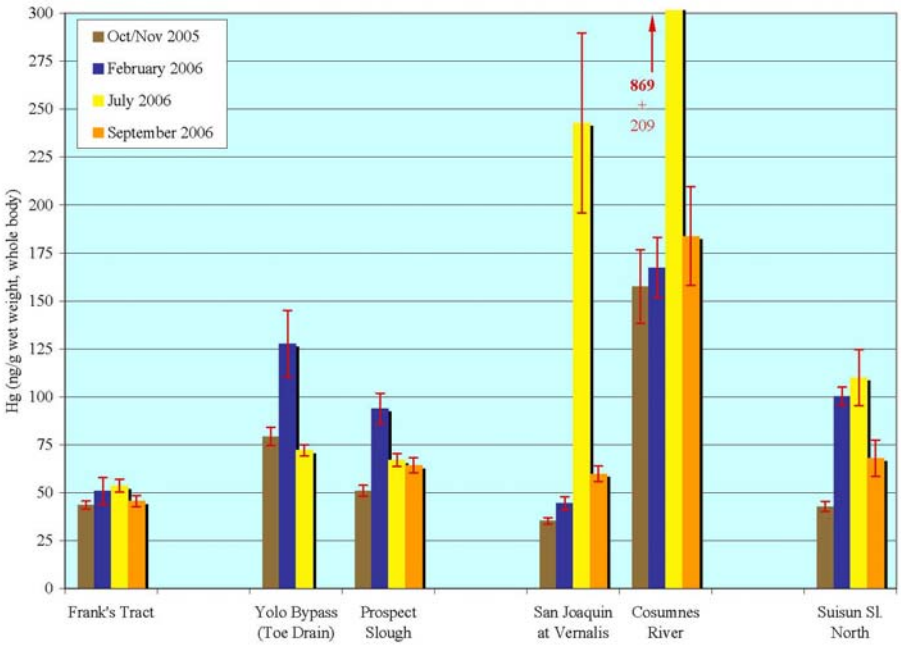

Figure 6. Seasonal patterns of methylmercury in Mississippi silversides at several locations in the Delta (from Davis et al. 2007).

In contrast with episodically flooded habitats, permanently flooded and daily, tidally-flooded, vegetated wetlands tended to have lower MeHg concentrations in biosentinel fish, relative to adjacent non-vegetated aquatic habitat (fig. 7).

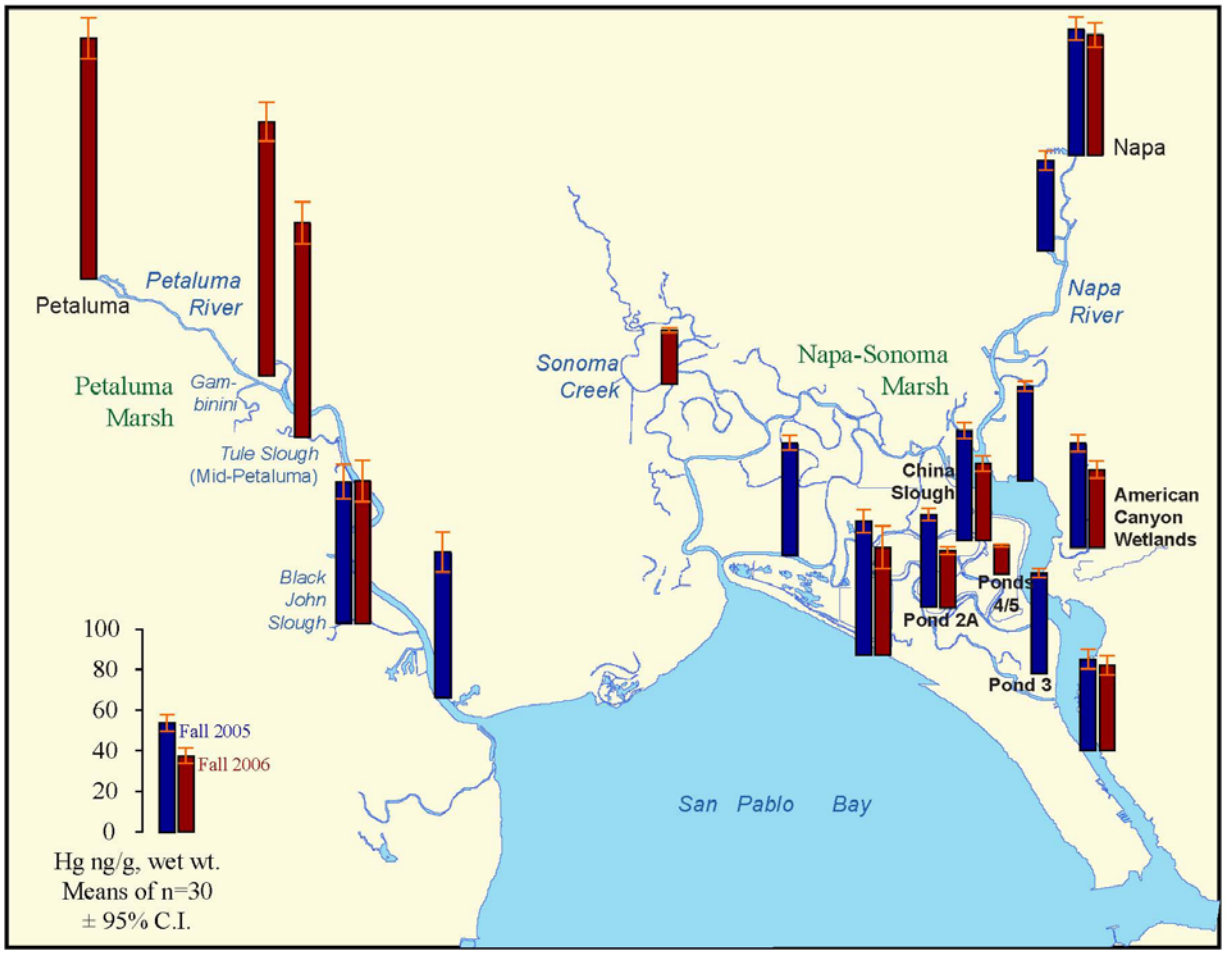

Figure 7. Map showing variations in mercury concentrations of Mississippi silversides in the Petaluma and Napa-Sonoma marshes, fall 2005 and fall 2006 (from Davis et al. 2007).

3.2.7 Intermediate Outcome 2: Concentrations of methylmercury in biota

Previous and ongoing studies in the Bay-Delta watershed indicate a wide range of MeHg concentrations in sport fish (Davis et al. 2003a, 2003b, 2008, and ongoing), small fish and invertebrates (Slotton et al. 1997, 2002, 2004a, 2004b and ongoing), and birds with aquatic-based diets (Elbert and Anderson 1998, Yee et al. 2005, 2007, Ackerman et al. 2007a, in press-a, in press-b, and ongoing). This variation is both spatial and, for the small fish, invertebrates, and birds, seasonal. Concentrations found in prey organisms at some locations and times have been well above threshold levels developed for the protection of wildlife. Similarly, concentrations in some species of sport fish in some locations commonly exceeded health guideline levels by a factor of 2 to 3 (Grenier et al. 2007). Finally, concentrations in aquatic-based birds at some locations and times have been shown to exceed threshold levels developed for their protection by up to 5-fold (Ackerman et al. 2007a), with actual lethal effects associated with Hg and MeHg in some recent data. Clearly, MeHg exposure remains an ongoing challenge for the management of the Bay-Delta watershed.

Table 3. Wildlife exposure considerations.

Exposure Consideration	Relationship to Exposure	Estimate of Importance	Relative Uncertainty	Source
Trophic position	Directly proportional	High	Low	1,2,3
Space use	Varies	High	High	4,5
Foraging habitat	Varies	High	Moderate	2, 3, 6
Age	Varies	Moderate	Low	3, 7
Growth/metabolic rate	Varies	Moderate – High	High	8
Primary productivity	Inversely proportional	Moderate	Moderate	9, 10
Community structure	Varies	Low – High	High	2, 11

[1]Kidd et al. 1995; [2]Eagles-Smith et al. in press-a; [3]Ackerman et al. 2007a; [4]Ackerman et al. 2007b; [5]Ackerman et al. in press-a; [6] Power et al. 2002; [7]Wiener and Spry 1996; [8]Simoneau et al. 2005; [9]Allen et al. 2005; [10]Chen and Folt 2005; [11]Pickhardt et al. 2005.

3.2.8 Limitations and Recommendations

This overview indicates, among other things, our current limited knowledge regarding fish growth rates in the Delta, their effect on mercury (i.e. MeHg) concentrations in fish, and the resultant potential complication in interpreting monitoring data. Although a comparative study of food web dynamics in the Cosumnes River and Central Delta areas indicated that differences in MeHg bioaccumulation were not caused by differences in the food web (Marvin-DiPasquale et al. 2007) additional Delta-specific information is needed on fish diets relative to MeHg exposure to ensure that differences in fish MeHg concentrations among sites, or changes at a particular site through time, are primarily caused by differences in MeHg exposure rather than differences in diet or growth rate. There are also unknowns remaining with regard to the influence of algal bloom dynamics on Delta MeHg bioaccumulation, and zooplankton community structure. This is particularly important in light of the recent Pelagic Organism Decline (POD) which has resulted in a dramatic change to the zooplankton community.

3.3 Submodel #3: Human Health Effects

Submodel #3, showing human health effects, is shown in Figure 8.

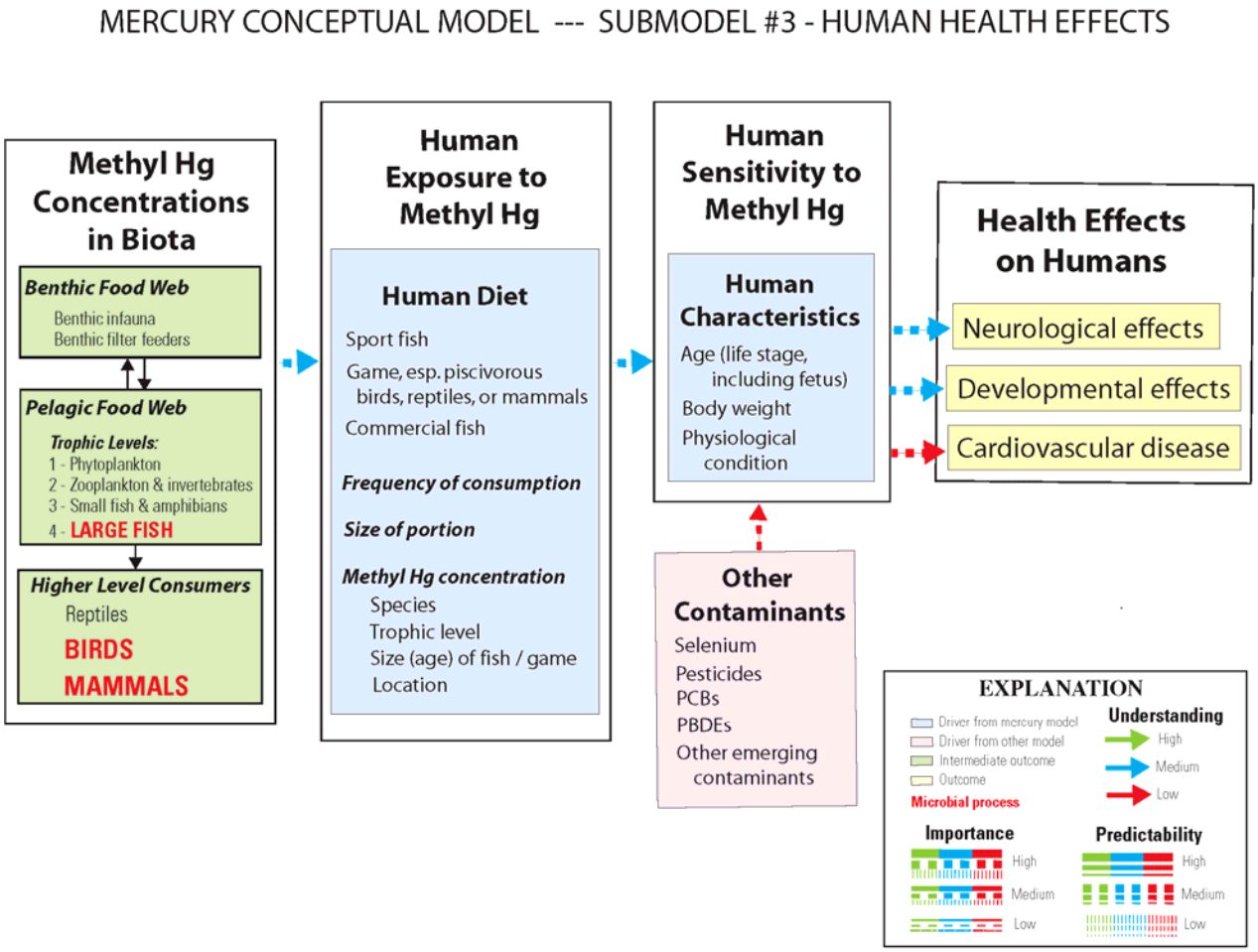

Figure 8. Mercury Submodel #3 — Human health effects. The primary risk to human health from methylmercury exposure is from dietary exposure caused by consumption of fish (sport and commercial) and/or game species such as piscivorous birds, reptiles, or mammals. Neurological and developmental effects in humans from methylmercury exposure are well documented whereas increased cardiovascular disease related to methylmercury exposure is less well established.

3.3.1 Human Exposure

The toxicity of mercury to humans is greatly dependent on its chemical form (elemental, inorganic, or organic) and route of exposure (oral, dermal, or inhalation). Methylmercury (an organic form) is highly toxic and can pose a variety of human health risks (NAS/NRC 2000). It is commonly assumed that methylmercury comprises more than 95 percent of the total amount of mercury found in fish muscle tissue (ATSDR 1999; Bloom 1992). However, several recent studies (Mason et al. 2006 and references therein) indicate that the %MeHg in fish can be lower and more variable,

especially in estuaries. For example, striped bass in Chesapeake Bay had %MeHg of 68 ± 24% in 2003 and 61 ±19% in 2004 (Mason et al. 2006) and spotted bass in Camp Far West Reservoir, California had %MeHg of 87% in 2002-03 (M. Saiki, U.S. Geological Survey, written commun. 2006). Fish consumption is the major route of exposure to methylmercury in the United States (ATSDR 1999).

In general, mercury concentrations in fish and other biota to which humans may be exposed are dependent on the mercury level of the environment, as described in Submodels #1 and #2. Fish at the highest trophic levels (i.e., predatory fish) generally have the highest levels of mercury. Additionally, because of the long biological half-life of methylmercury in fish (approximately 2 years), tissue concentrations in fish increase with increased duration of exposure (Krehl 1972; Stopford and Goldwater 1975; Tollefson and Cordle 1986). As a result, tissue methylmercury concentrations are expected to increase with increasing age and length within a given species, particularly in piscivorous fish. Thus, individuals who consume larger (older), predatory fish taken from a mercury-rich environment will have higher methylmercury exposures than those eating smaller, herbivorous fish taken from a low-mercury environment.

3.3.2 Sensitivity and Toxicity

Human toxicity of methylmercury has been well studied following several epidemics of human poisoning resulting from consumption of highly contaminated fish (Japan) or seed grain (Iraq, Guatemala, and Pakistan) (Elhassani 1982-83). The resulting illness was manifested largely by neurological signs and symptoms such as loss of sensation in the hands and feet and, in extreme cases, loss of gait coordination, slurred speech, sensory deficits including blindness, and mental disturbances (Bakir et al. 1973, Marsh 1987). Review of data collected during and subsequent to the Japan and Iraq outbreaks identified the critical target of methylmercury as the nervous system and the most sensitive subpopulation as the developing organism (U.S. EPA 1997). During critical periods of prenatal and postnatal structural and functional development, the fetus and children are especially susceptible to the toxic effects of methylmercury (ATSDR 1999, IRIS 1995). When maternal methylmercury consumption is very high, as happened in Japan and Iraq, significant methylmercury toxicity can occur to the fetus during pregnancy, with only very mild or even in the absence of symptoms in the mother. In those cases, symptoms in children are often not recognized until development of cerebral palsy and/or mental retardation many months after birth (Harada 1978, Marsh et al. 1980 and 1987, Matsumoto et al. 1964, Snyder 1971). Considerable research has been conducted in recent years to identify the lowest methylmercury exposure level that could result in subtle neurodevelopmental deficits in young children (see NAS/NRC 2000). Given likely exposure scenarios in the United States, more significant effects, as occurred in Japan and Iraq, are not expected.

3.3.3 Transformation and Elimination

Methylmercury, when ingested, is almost completely absorbed from the gastrointestinal tract (Aberg et al. 1969, Myers et al. 2000). Once absorbed, methylmercury is distributed throughout the body, reaching the largest concentration in kidneys. Its ability to cross the placenta as well as the blood-brain barrier allows methylmercury to accumulate in the brain and fetus, which are known to be especially sensitive to the toxic effects of this chemical (ATSDR 1999). In the body, methylmercury is slowly converted to inorganic mercury and excreted predominantly by the fecal

(biliary) pathway. Methylmercury is also excreted in breast milk (ATSDR 1999). The biological half-life of methylmercury is approximately 44-74 days in humans (Aberg et al. 1969, Smith et al. 1994), meaning that it takes approximately 44–74 days for one-half of a single ingested dose of methylmercury to be eliminated from the body.

3.3.4 Limitations and Recommendations

A revised human health advisory for the southern Delta and San Joaquin River was released in draft form (Gassel et al. 2007). A similar update for the northern Delta is scheduled for early 2008 (R. Brodberg, Office of Environmental Health Hazard Assessment, oral commun. 2007). Additional data from the Fish Mercury Project (Davis et al. 2007) will provide additional information that will be important for protection of public health.

3.4 Submodel #4: Wildlife Health Effects

Subsequent to entry and bioaccumulation of methyl mercury through food webs, the primary ecological endpoints of concern relative to exposure are associated with toxicological impacts on fish and wildlife. Individual and population-level effects of methyl mercury on fish and wildlife are difficult to assess because they are often manifested as neurological impairment, which may not be readily detected in the wild. However, recent literature using sophisticated approaches suggests that subtle effects occur at environmentally relevant concentrations, and that these effects may have population-level implications.

A submodel showing the drivers and linkages related to the outcome of wildlife health effects is shown in Figure 9. Below we review the dietary and tissue-level concentrations shown to induce effects, discuss complicating factors to interpreting wildlife mercury concentrations, such as interactions with selenium and other contaminants, and finally make recommendations for wildlife-specific target Hg concentrations while highlighting knowledge gaps needed to better interpret wildlife risk.

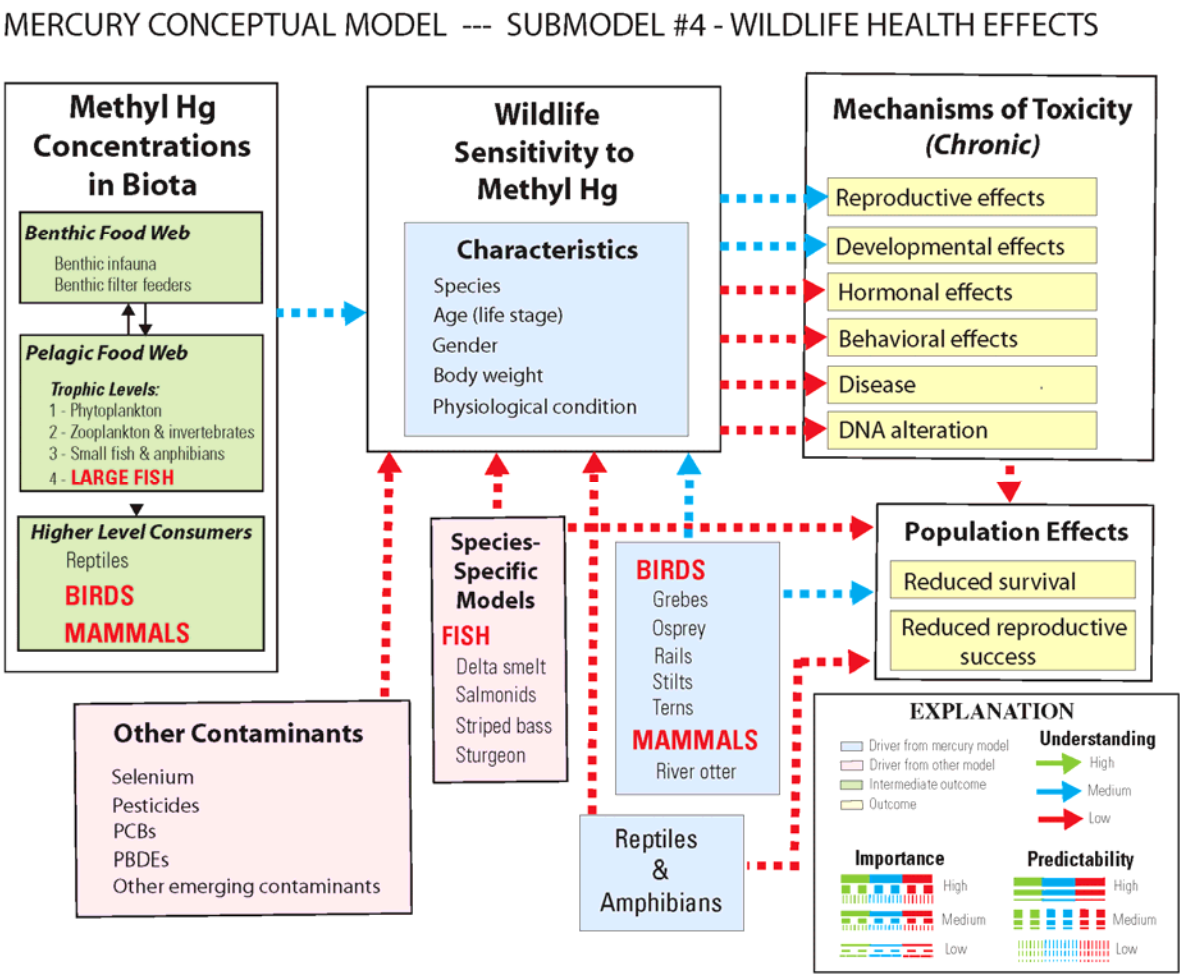

Figure 9. Mercury Submodel #4 – Wildlife health effects. The primary pathway to methylmercury exposure in wildlife is from diet. At particular risk are piscivorous species including fish, birds, reptiles, and mammals. Reproductive and developmental effects from methylmercury exposure are relatively well documented for some species, whereas there is relatively little information on hormonal effects, behavioral effects, disease, and DNA alteration.

3.4.1 Biologically Effective Concentration and Toxic Effects

Interpretations of laboratory benchmarks relative to impairment in the wild are confounded not only by the additional stressors encountered in the wild, but also by the common endpoints used in laboratory-based studies. Additionally, differing mercury sensitivities among taxonomic groups require distinct evaluations. Here, we have chosen to discuss general thresholds levels in three taxonomic classifications: fish, birds, and mammals. Amphibians have been excluded from this assessment because we are aware of no published literature relating diet or tissue concentrations to effects. Table 4 lists dietary and tissue-based threshold values associated with impairment in several species of fish, birds, and mammals.

Table 4. Mercury concentrations (matrix specific) associated with effects in fish, birds and mammals.

	Species	Tissue	Mercury concentration (µg/g, wet weight)	Effect endpoint (footnotes indicate references)
Fish				
	Walleye	muscle	0.25	Hormone response[1]
	grayling (fry)	whole body	0.27	Feeding efficiency[2]
	fathead minnow	whole body	0.39	Spawning[3]
	golden shiner	whole body	0.52	Predator avoidance[4]
	fathead minnow	whole body	0.71	Reproductive behavior[5]
	fathead minnow	whole body	0.86	Hormone response[6]
Birds				
	American kestrel	diet	0.30	Fledgling success[7]
	common loon	diet	0.30	Reproductive success[8]
	common loon (chick)	diet	0.40	Immune response[9*]
	great egret (juv)	diet	0.50	Histology and immune function[10]
	Mallard	diet	0.10	Duckling behavior and survival[11]
	common loon	blood	3.0	Reproduction[8]
	common loon	blood	4.0	Reproduction, hormone response, nesting behavior[8]
	great egret (juv)	blood	12.3	Histology and immune function[10]
	common loon	liver	3 - 14	Egg hatchability[12*]
	black-necked stilt	down feather	5.74	Chick survival[13]
	great egret (juv)	feather	108.0	Histology and immune function[10]
	common tern	egg	3.65	Egg hatchability[12*]
	Mallard	egg	0.86	Duckling behavior and survival[11]
	Mallard	egg	1.0	Embryo deformities[14]
	Mallard	egg	2.3	Duckling neurotoxicity[14]
	Pheasant	egg	0.5 – 1.5	Egg hatchability[15]
Mammals				
	Mink	diet	1.0	Death[16]
	Mink	liver	44.1	Death[16]
	Mink	kidney	28.4	Death[16]
	Mink	brain	5.0	Neurochemistry[9*]
	Mink	brain	15.3	Death[17]
	Otter	diet	2.0	Anorexia, ataxia[17]
	Otter	liver	32.6	Anorexia, ataxia[17]
	Otter	kidney	37.6	Anorexia, ataxia[17]
	Otter	muscle	13.3	Anorexia, ataxia[17]
	Otter	brain	13.3	Anorexia, ataxia[17]

[1]Friedmann et al. 1996; [2]Fjeld et al. 1998; [3]Hammerschmidt et al. 2002; [4]Webber and Haines 2003; [5] Sandheinrich and Miller 2006; [6]Drevnick and Sandheinrich 2003; [7]Albers et al. 2007; [8]Evers et al. 2004; [9]Scheuhammer et al. 2007; [10]Spalding et al. 2000b; [11]Heinz 1979; [12]Wolfe et al. 1998; [13]Ackerman et al. 2007a; [14]Heinz and Hoffman 1998; [15]Fimreite 1971; [16]Wren et al. 1987; [17]O'Connor and Nielsen 1981; [*]Citation is review article.

3.4.1.1 Fish

Nearly all of the methyl mercury accumulated in fish occurs through dietary exposure (see bioaccumulation section), and in general, natural-water methyl mercury concentrations are substantially lower than those shown to induce direct toxicity. Therefore, we have restricted this evaluation to only dietary exposure routes.

Relatively less work has been conducted on fish impairment from dietary methyl mercury than birds and mammals; however, several studies indicate that fish may be at risk from environmentally relevant exposure to methyl mercury. Some of the effects related to mercury in fish include: altered hormone expression (Friedman et al. 2002, Drevnick and Sandheinrich 2003), reduced spawning success and reduced reproductive output (Hammerschmidt et al. 2002), reduced gonadosomatic indices and testicular atrophy (Friedman et al. 1996), liver necrosis (de Oliveira Ribeiro et al. 2002), and altered predator avoidance behavior (Webber and Haines 2003).

Beckvar et al. (2005) developed a fish health risk threshold for fish tissue mercury of 0.20 $\mu g\ g^{-1}$ (wet weight) based largely on the work described above. However, it is possible that more subtle behavioral effects may occur at lower concentrations. These effects are difficult to quantify (especially in the field), but may be critical for determining risk to fish in mercury contaminated water bodies such as the San Francisco Bay-Delta Estuary.

3.4.1.2 Birds

More work has been conducted to assess the effects of methyl mercury on birds, with a recent emphasis on waterbirds which generally have greater exposure than those inhabiting terrestrial habitats. There is some ambiguity surrounding the appropriate tissues used for assessing risk and impairment, but most studies rely on eggs or livers, and to a lesser degree feathers. However, recent studies indicate that blood may be a better matrix due to its integration period, high correlation with internal tissues, and non-lethal collection methods (Evers et al. 2005, C. Eagles-Smith et al., U.S. Geological Survey, unpublished data). Moreover, since dietary Hg concentrations ultimately drive tissue accumulation, other studies only report exposure regimes which do not explicitly identify concentrations in tissues, but rather just diet. Below we evaluate reported toxic endpoints on a tissue-specific basis.

3.4.1.2.1 Liver

Liver has been the most conventionally used tissue for assessing Hg effects on birds, likely because it is the major site of xenobiotic detoxification and because it tends to have higher concentrations than other internal tissues. In their review of mercury effects on wildlife, Wolfe et al. (1998) reported liver concentrations of 3-5 $\mu g\ g^{-1}$ (wet weight) as the lowest published concentrations in avian liver related to deleterious effects, whereas most of the studies associated with effects (ranging from reduced nesting success to death) had liver concentrations of 20-50 $\mu g\ g^{-1}$ (wet weight). However, interpretation of liver mercury concentrations is confounded by (among other things) the potential for demethylation to inorganic mercury. Several studies have shown that the relative proportion of liver methyl Hg declines with increasing THg concentrations (Scheuhammer et al. 1998, Henny et al. 2002), however the toxicity of liver inorganic mercury is relatively unknown (Scheuhammer et al. 2007). Thus, to properly interpret liver burdens mercury must be

analyzed as methyl Hg, and selenium content in the liver should additionally be assessed, both because of its interactive effects and relationship to the demethylation process.

3.4.1.2.2 Eggs

Egg mercury concentrations represent the maternally-derived dose of MeHg available to developing embryos and recently-hatched chicks. Eggs are generally considered a superior matrix for assessing risk to breeding birds because they are indicative of maternal concentrations, and to hatching success and chick survival in a wide range of species. The literature on toxicity of egg-derived Hg is somewhat convoluted, but the most robust studies indicate that a range of 0.5-ca. 4 $\mu g\ g^{-1}$ (fresh wet weight) are associated with decreased hatchability and embryo mortality (Wolfe et al. 1998). However, more current research indicates that there is a great deal of variability in the sensitivity among species (G. Heinz, U.S. Geological Survey, unpublished data), and that current Lowest Observed Adverse Effect Level (LOAEL) range (0.5-0.86 $\mu g\ g^{-1}$) based on mallard studies may be overestimates for other species.

In addition to embryo death and impaired hatchability, maternally derived mercury may also reduce chick survival during the first few days post-hatch. There is currently little information relating egg Hg concentrations to those of chicks; however, current studies indicate that mercury in recently-hatched chick down feathers is strongly correlated to sibling egg mercury (C. Eagles-Smith et al., U.S. Geological Survey, unpublished data), indicating that down concentrations represent *in ovo* exposure to maternally deposited egg Hg. Moreover, Hg in down feathers of recently-hatched black-necked stilt chicks that were found dead were significantly higher than down feather Hg from randomly sampled live chicks of similar age in South San Francisco Bay (Ackerman et al. in press-b). However, threshold levels for such effects are still being developed.

3.4.1.2.3 Feathers

Feathers have been utilized as a monitoring tool for exposure and retrospective studies on numerous taxa throughout the world. Although feathers can be valuable for these purposes, the pharmacodynamics of mercury and physiological differences among species make feathers (except chick down feathers) poor overall indicators for risk at small spatial and temporal scales. In general, a concentration of ~ 20 $\mu g\ g^{-1}$ (fresh weight) in feathers is associated with observable effects; however, because feather Hg only represents exposure (as well as internal tissue Hg redistribution) during a short time frame from the specific location at which the feather was grown (Furness et al. 1986), interpreting concentrations in relation to local exposure conditions can be complicated. Moreover, because feathers represent a sequestration and depuration pathway, linking feather Hg levels to specific effects is often dubious. However, chick down feathers provide an excellent index of egg-mercury concentrations and may be useful in determining *in-ovo* exposure in chicks. Ackerman et al. (in press-b) assessed down feathers in apparently healthy and recently dead, just-hatched American avocet and black-necked stilt chicks in San Francisco Bay. Although there were no differences between groups for avocets, they found that dead chicks had higher down Hg concentrations than those found to be alive.

3.4.1.2.4 Blood

Despite its advantages, the use of blood in assessing effects of mercury to birds has not become widespread until recently. However, several recent field and lab based evaluations of mercury impairment to birds have shown the utility of this matrix (Evers et al. 2004, Evers et al. 2005, Ackerman et al. 2007a). For example, loons from North Eastern United States with blood concentrations exceeding 3.0 μg g^{-1} (wet weight) produced 40% fewer young than those form other areas with blood levels less than 1.0 (wet weight) (Evers et al. 2004). Moreover, the same study showed that blood Hg concentrations in excess of 4.0 μg g^{-1} (wet weight) were associated with elevated stress hormones, impaired productivity, flight feather asymmetry, and reduced nest attendance. Even birds with blood Hg concentrations ranging from 1-3 μg g^{-1} (wet weight) exhibited subtle evidence of Hg-related impairment (Evers et al. 2004). Specific to San Francisco Bay, Ackerman et al. (2007a) found that 6% of avocets, 5% of stilts, and 57% of Forster's terns had blood mercury concentrations exceeding 3.0 μg g^{-1} ww.

3.4.1.2.5 Diet

Because the source of mercury to birds occurs almost entirely through dietary exposure, many studies have focused on Hg dosages in relation to adverse effects rather than tissue concentrations. Unfortunately, many early studies used unrealistic concentrations to examine toxic mechanisms, but are of little environmental relevance. However, over the last few decades controlled dosing studies and field-based investigations of dietary mercury exposure and effects have provided a better understanding of Hg threshold exposure levels in select avian taxa. In common loons from Northeastern United States and Canada, impaired reproduction was documented in areas where prey mercury exceeded 0.3 μg g^{-1} (wet weight) (Evers et al. 2004). However, a laboratory experiment with mallards, found that birds fed a diet containing 0.5 μg g^{-1} (dry weight; ~0.1 μg g^{-1} wet weight) methyl mercury suffered from impaired reproduction and behavior Heinz (1979). In a recent evaluation of regulatory threshold values for mercury exposure to wildlife, the US Fish and Wildlife Service related dietary exposure and trophic transfer to Hg-induced reproductive impairment, and determined that an average dietary concentration of 0.03 μg g^{-1} (wet weight) was protective of threatened and endangered species (U.S. FWS 2003). However, ongoing work in the Estuary is being conducted using site-appropriate conditions and species to evaluate the 2003 determination.

3.4.1.3 Mammals

Like birds, piscivorous marine and aquatic mammals are considered to be at greatest risk to mercury exposure and effects. Among those groups, mink and otter represent a substantial proportion of the information relating to toxicity, likely because of their abundance in the wild and the ability to rear them in the lab. Mammals suffer from similar effects of mercury as birds, with behavioral and reproductive abnormalities occurring at environmentally relevant concentrations. Reviews by Wolfe et al. (1998), Wiener et al. (2003), and Scheuhammer et al. (2007) suggest that dietary mercury exposure of 0.1 μg g^{-1} (wet weight) (corresponding liver concentrations of 0.45 μg g^{-1} (wet weight) had no effect on captive fed mink; however nerve tissue lesions with no associated clinical signs of toxicity were detected in animals fed a diet containing 1.1 μg g^{-1} (wet weight) (corresponding liver concentrations of 22.4 μg g^{-1} (wet weight)), and anorexia, ataxia and death were associated with 50 – 80 days exposure to a diet of 1.8 μg g^{-1} (wet weight) (corresponding liver concentrations of 0.22.3 μg g^{-1} (wet weight). Similar effects were seen in otter fed a diet of 2.0 μg

g^{-1} (wet weight) (corresponding liver concentrations of 35.3 μg g^{-1} (wet weight). Although these studies suggest that realistic dietary Hg doses can be fatal to mammals, comparatively little work has been conducted examining more subtle behavioral effects from lower concentrations. Moreover, methyl mercury readily crosses the maternal placenta and blood-brain barrier, placing developing fetuses at the greatest risk. Unfortunately, there is currently little information relating tissue concentrations or dietary dosages in wild mammals which are associated with impaired reproduction or generational effects on offspring. The river otter is a fish-eating mammal that may be at particular risk from MeHg exposure in the Delta and its tributaries.

3.4.2 Methyl Mercury Effects

3.4.2.1 DNA Alteration

Relatively little information exists on mercury-induced DNA alteration in wildlife or its associated effects. Betti and Nigro (1996) found the *in vitro* exposure to methyl Hg caused DNA strand breaks in bottle-nosed dolphin lymphocytes. However, it is unclear how this relates to tissue, organ, or individual level effects. Cells from rats and hamsters have also been shown to respond to mercury in a genotoxic fashion, with DNA strand breakage, and aneuploidy (a change in the number of chromosomes that can lead to chromosomal disorders) and polyploidy (having more than two homologous sets of chromosomes) occurring in a dose-dependent fashion (Wolfe et al. 1998). Currently, it is not known how tissue or dietary concentrations relate to DNA alteration, or how these alterations manifest themselves within an organism.

3.4.2.2 Tissue and Organ Damage

As noted above, methyl mercury primarily exerts its toxic effect on the central nervous system (Wolfe et al. 1998), resulting in reduced coordination and behavioral abnormalities. These symptoms are accompanied by cellular damage to the brain, including lesions and nerve demyelination. However, inorganic Hg is also a health risk to wildlife because of their ability to demethylate liver methyl mercury. Both birds and mammals show declining proportion of methyl mercury in the liver as a function of increasing total mercury, indicating that a detoxification mechanism (demethylation) may be triggered at elevated MeHg concentrations. It is thought that selenium plays a major role in the demethylation process, though a mechanism has not been identified. Upon conversion from methyl mercury, inorganic mercury can begin to accumulate in both liver and kidney tissues (Scheuhammer et al. 2007) causing nephrotoxicity at elevated concentrations. However, complexation with selenium compounds in the liver and kidney can have a protective effect and reduce the toxicity in inorganic Hg.

3.4.2.3 Abnormal Development

Except at substantially elevated concentrations, methyl mercury is not known to cause gross physical development abnormalities. However, impaired neurological development and learning behaviors have been related to mercury in mammals and birds. It is likely that more subtle impairment in wildlife may occur at lower levels of exposure, but there is currently little data to asses this possibility appropriately.

3.4.2.4 Reproductive Toxicity and Endocrine Disruption

Methyl mercury has been associated with both impaired reproduction and altered hormone expression in wildlife. Mercury-induced reproductive toxicity can take several forms, all of which may ultimately lead to lower productivity. Reduced egg-laying is associated with elevated Hg concentrations in several bird species (Heinz 1979). Breeding behavior has also been shown to vary with blood mercury concentrations in adult common loons, where time spent nest sitting and foraging decreased with increasing Hg concentrations (Evers et al. 2004). Hatchability of eggs also declines with increasing Hg concentrations (Scheuhammer et al. 2007). Although threshold levels vary among taxa, concentrations above 1 $\mu g\ g^{-1}$ wet weight are generally considered embryotoxic (Scheuhammer et al. 2007). Upon hatching, chicks with higher Hg concentrations (or coming from parents fed higher doses) showed impaired behavior such as reduced startle reflex and response to maternal calls (Heinz 1979).

Although the ultimate effect on fitness is unknown, mercury is considered an endocrine disrupting contaminant (Colburn et al. 1993) and has been associated with alterations to both stress and sex hormones. Evers et al. (2004) found that common loon adults with blood Hg concentrations above 1 $\mu g\ g^{-1}$ also had higher corticosterone (stress response) levels relative to birds with lower blood Hg concentrations. Moreover, average corticosterone levels continued to increase in birds with Hg concentrations above 3 $\mu g\ g^{-1}$. Heath and Frederick (2005) compared mercury concentrations to hormone expression in white ibises and found that estradiol concentrations in pre-breeding females declined and testosterone in incubating males increased with elevated Hg concentrations. They also found that white ibis nesting declined with and increasing Hg exposure index. Based on their data, they suggest that lower nest attempts or higher abandonment rates may be related to changes in hormone concentrations caused by Hg exposure. As indicated previously, Hg effects on reproduction are not limited to birds. Hg has also been shown to alter sex hormone concentrations in fish fed relatively low doses of methyl mercury (Hammerschmidt et al. 2002, Drevnick and Sandheinrich 2003), with subsequent reductions in reproductive output.

3.4.2.5 Behavior

As a neurotoxin, behavior appears to be affected by relatively low exposure to mercury in a wide range of taxa. However, the difficulty in quantifying behavioral responses (particularly in the field) likely limits our ability to detect effects. In fish, golden shiner with whole-body Hg concentrations greater than 0.230 $\mu g\ g^{-1}$ wet weight had more dispersed shoals, took longer to settle after exposure to a model predator, and shoaled higher in the water column with than control fish with whole body concentrations of 0.041 $\mu g\ g^{-1}$ wet weight (Webber and Haines 2003).

As previously mentioned, birds such as common loons show impaired reproductive behavior with increasing Hg exposure, and mallard chick behavior was altered in birds from Hg-dosed parents relative to controls. Bouton et al. (1999) also found that juvenile great egrets dosed with dietary methyl mercury at concentrations of 0.5 $\mu g\ g^{-1}$were less likely to hunt fish or seek shade, and had reduced activity relative to control birds. Moreover, wild common loon chicks spent less time brooding and more time preening as their blood Hg concentrations increased (Nocera and Taylor 1998), and they did not balance their increase in activity and energy use with higher feeding rates, suggesting that growth may be impaired.

3.4.2.6 Growth

The effects of mercury on growth are difficult to assess because growth can have such a dramatic effect on Hg concentrations via growth dilution. In fish, Hg concentrations increase with size (age) (see section 3.2) and are often lower in fish with elevated growth rates because biomass accretion is greater than Hg accumulation (see section 3.2). In birds, blood mercury concentrations varied dramatically with age in both Forster's terns and black-necked stilts in the San Francisco Bay (Ackerman et al. 2007a). Blood Hg concentrations were high in newly hatched chicks, due to high *in ovo* Hg exposure, and concentrations declined rapidly after hatching as chicks diluted the Hg in their bodies through growth in size and depuration of Hg into growing feathers. Hg concentrations in chicks began to rapidly increase again just before and during fledging (28-days old) when body growth and feather production slowed and chicks continued to acquire Hg through their diets. This pattern of Hg depuration and accumulation was observed in chicks from both high and low Hg sites (Ackerman et al. 2007a). Using feathers as more discreet measurements of both *in ovo* (down feathers) and dietary (breast feathers) Hg exposure, Ackerman et al. (2007a) used mark-recapture methodology to assess if Hg influenced growth of Forster's tern chicks in San Francisco Bay. They found no relationship between down feather Hg and wing growth rates and a slight negative correlation between breast feather Hg concentrations and wing growth rates. Using laboratory dose-response studies, no relationship between Hg and growth rates were found in either common loon chicks (Kenow et al. 2003) or captive great egrets (Spalding et al. 2000a).

3.4.2.7 Immune-System Effects

Immunotoxic effects have long been postulated as being related to mercury exposure (Scheuhammer et al. 2007); however, the relationship is still poorly understood. Great egret nestlings fed 0.5 $\mu g\ g^{-1}$ dietary methyl mercury had lower packed cell volumes, increased lymphocytic cuffing, increased bonemarrow cellularity, decreased bursal wall thickness, and decreased thymic lobule size than birds fed a control diet (Spalding et al. 2000b). Henny et al. (2002) also found immune impairment in wild black-crowned night heron, snowy egret, and double-crested cormorant chicks from the Carson River, Nevada fed diets containing Hg concentrations of 0.36-1.18 $\mu g\ g^{-1}$ wet weight. Finally, Elbert and Anderson (1998) found a significant positive relationship between kidney Hg and percent eosinophils in the blood of Western grebes from Clear Lake, Tule Lake, and Eagle Lake, California.

3.4.2.8 Population-Level Effects

Population effects due to mercury are notoriously difficult to determine because of the data needed to parameterize population models. To our knowledge there are no studies that have utilized robust demographic population models to asses the effects of Hg exposure and toxicity on wildlife populations. However, population level impacts can be estimated by modeling the effects of mercury on total reproductive output. Evers et al. (2004) found that common loons with blood Hg concentrations below 1.0 $\mu g\ g^{-1}$ wet weight were 37% more successful in fledging young than birds with blood Hg concentrations exceeding 3.0 $\mu g\ g^{-1}$ wet weight. In South Florida and the Everglades the number of nesting waterbirds declined by ~90% (Scheuhammer et al. 2007). Although habitat loss and modifications were deemed the primary causes, Hg contamination may also have been an interacting factor (Scheuhammer et al. 2007).

3.4.3 Factors Affecting Toxicity

With the exception of its chemical form, there is little information on general factors affecting toxicity due to mercury. As has been indicated previously, methyl mercury is substantially more toxic than inorganic mercury, likely because its protein affinity distributes it easily throughout the body and it readily crosses cell membranes and the blood-brain barrier. Although inorganic Hg is fairly toxic in its own right (though not as bioavailable), its main mode of action is through kidney impairment. Also of importance for toxicity may be the physiological state of an organism. Animals which become emaciated due to limited food availability or disease may catabolize internal tissues, thus re-circulating protein-bound Hg and increasing tissue Hg concentrations through biomass attrition. It is also generally thought that toxicity is greater in young individuals relative to adults (Henny et al. 2002). However, it is currently unclear how variable this sensitivity is among species. Moreover, ontogenetic differences in Hg sensitivity quickly become complicated because different endpoints are used to assess sensitivity in each life stage. For example, neurological impairment resulting in mortality is commonly noted as more sensitive in chicks than adult birds, whereas reproductive impairment (which is not applicable to chicks) is thought to be the most sensitive endpoint for adults.

One factor shown to have an important effect on methyl mercury toxicity is selenium (Se) exposure and bioaccumulation (Scheuhammer et al. 2007). As mentioned above, Se interacts with Hg to form less toxic complexes and Se plays an important (though mechanistically unknown) role in the process of demethylation in birds and mammals. Additionally, recent lab dosing studies have shown that when fed in conjunction with methyl Hg, dietary Se has a protective effect on the health of adult mallards (Heinz and Hoffman 1998). However, the Hg and Se combinations had an increased effect on reproductive impairment relative to either Hg or Se alone. For example, the control birds produced 7.6 pair per young, the Se-only birds produced 2.8 pair per young, the Hg-only birds produced 1.1 pair per young, and the Hg plus Se produced 0.2 pair per young (Heinz and Hoffman 1998).

3.4.4 Limitations and Recommendations

The major limitation regarding effects for fish and wildlife is the lack of species-specific toxicity information on those organisms most at risk in the San Francisco Bay-Delta Estuary. Current threshold levels are all based on species such as loons or mallards which may have much different sensitivities (higher or lower) than birds such as Forster's terns, black-necked stilts, least terns, and clapper rails, which have concentrations that may put them at risk to impairment from mercury. There is currently some mercury-related work in progress on birds in the Estuary; however, to fully understand risk to birds additional information is needed. In particular, there is still little information on bird mercury concentrations in the Delta itself and its tributaries. To fully understand the risk to birds, an assessment is needed of those species most likely to be at risk, and the locations (such as the Suisun Marsh, Cosumnes River and Yolo Bypass) where risk may be the highest. Moreover, to our knowledge there is currently no information related to mercury concentrations in aquatic mammals in the Delta. Our toxicity assessment indicates that species found in the Delta, such as otters, may be sufficiently sensitive to mercury that there is substantial risk of impairment. Finally, it is clear that there is currently little if any information on effects of methyl mercury on amphibians and reptiles, and we are aware of little data on exposure of such taxa to methyl mercury in the Delta or possible effects.

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5. References Cited

Aberg, B, Ekman, L, Falk, R, Greitz, U, Persson, G, Snihs, J-O. 1969. Metabolism of methyl mercury (^{203}Hg) compounds in man. Arch Environ Health 19:478-485.

Ackerman, JT, Eagles-Smith, CA, Heinz, GH, Wainwright-De La Cruz, SE, Takekawa, JY, Adelsbach, TL, Miles, AK, Hoffman, DJ, Schwarzbach, SE, Suchanek, TH, Maurer, T. 2007a. Mercury in birds of the San Francisco Bay-Delta: trophic pathways, bioaccumulation and ecotoxicological risk to avian reproduction. 2006 Annual Administrative Report, U.S. Geological Survey, Western Ecological Research Center, Davis, CA, and U.S. Fish and Wildlife Service, Environmental Contaminants Division, Sacramento, CA. 41 p.

Ackerman, JT, Eagles-Smith, CA, Takekawa, JY, Demers, SD, Adelsbach, TL, Bluso, JD, Miles, AK, Warnock, N, Suchanek, TH, Schwarzbach, SE. 2007b. Mercury concentrations and space use of pre-breeding American avocets and black-necked stilts in San Francisco Bay. Sci Total Environ 384:452-466.

Ackerman, JT, Eagles-Smith, CA, Takekawa, JY, Bluso, JD, Adelsbach, TL. in press-a. Mercury concentrations in blood and feathers of pre-breeding Forster's terns in relation to space use of San Francisco Bay Habitats. Environ Toxicol Chem.

Ackerman, JT, Takekawa, JY, Eagles-Smith, CA, Iverson, SA. in press-b. Mercury contamination and effects on survival of American avocet and black-necked stilt chicks in San Francisco Bay. Ecotoxicol.

Albers, PH, Koterba, MT, Rossmann, R, Link, WA, French, JB, Bennett, RS, Bauer, WC. 2007. Methylmercury and reproduction in American kestrels. Environ Toxicol Chem 26(9).

Allard, B, Arsenie, I. 1991. Abiotic reduction of mercury by humic substances in aquatic system - An Important process for the mercury cycle. Water Air Soil Poll 56:457-464.

Allen, EW, Prepas, EE, Gabos, S, Strachan, WMJ, Zhang, W. 2005. Methyl mercury concentrations in macroinvertebrates and fish from burned and undisturbed lakes on the Boreal Plain. Can J Fish Aq Sci 62:1963-1977.

Alpers, CN, Hunerlach, MP. 2000. Mercury contamination from historic gold mining in California. U.S. Geological Survey Fact Sheet FS-061-00, 6 p. http://ca.water.usgs.gov/mercury/fs06100.html

Alpers, CN, Hunerlach, MP, Marvin-DiPasquale, MC, Antweiler, RC, Lasorsa, BK, De Wild, JF, Snyder, NP. 2006. Geochemical data for mercury, methylmercury, and other constituents in sediments from Englebright Lake, California, 2002. U.S. Geological Survey Data Series 151, 95 p. http://pubs.water.usgs.gov/ds151/

Alpers, CN, Hunerlach, MP, Marvin-DiPasquale, M, Snyder, NP, Krabbenhoft, DP. 2004. Mercury and methylmercury in the upper Yuba River watershed: Fluvial transport and reservoir sedimentation. Third Biennial CALFED Bay-Delta Program Science Conference Abstracts; 2004 Oct 4-6; Sacramento (CA). p. 4.

Alpers, CN, Hunerlach, MP, May, JT, Hothem, RL. 2005a. Mercury contamination from historical gold mining in California. U.S. Geological Survey Fact Sheet 2005-3014, 6 p. http://water.usgs.gov/pubs/fs/2005/3014/

Alpers, CN, Hunerlach, MP, May, JT, Hothem, RL, Taylor, HE, Antweiler, RC, De Wild, JF, Lawler, DA. 2005b. Geochemical characterization of water, sediment, and biota affected by mercury contamination and acidic drainage from historical gold mining, Greenhorn Creek, Nevada County, California, 1999–2001. U.S. Geological Survey Scientific Investigations Report 2004-5251, 278 p. http://pubs.usgs.gov/sir/2004/5251/

Alpers, CN, Stewart, AR, Saiki, MK, Marvin-DiPasquale, MC, Topping, BR, Rider, KM, Gallanthine, SK, Kester, CA, Rye, RO, Antweiler, RC, De Wild JF. in press. Environmental factors affecting mercury in Camp Far West Reservoir, 2001–03. U.S. Geological Survey Scientific Investigations Report 2006–5008.

Ashley, RP, Rytuba, JJ, Rogers, R, Kotlyar, BB, Lawler, D. 2002. Preliminary report on mercury geochemistry of placer gold dredge tailings, sediments, bedrock, and waters in the Clear Creek restoration area, Shasta County, California: U.S. Geological Survey Open-File Report 02-401, 43 p. Available at http://geopubs.wr.usgs.gov/open-file/of02-401/

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. Toxicological profile for mercury (update). Prepared by Research Triangle Institute under contract nr 205-93-0606. Public Health Service, U.S. Department of Health and Human Services.

Bakir, F, Damluji, SF, Amin-Zaki, L, Murtadha, M, Khalidi, A Al-Rawi, NY, Tikriti, S, Dhahir, HI, Clarkson, TW, Smith, JC, Doherty, RA. 1973. Methylmercury poisoning in Iraq. Science 181:230–241.

Barkay, T, Gillman, M, Turner, RR. 1997. Effects of dissolved organic carbon and salinity on bioavailability of mercury. Appl Environ Microbiol 63(11):4267-4271.

Battke, F, Ernst, D, Halbach, S. 2005. Ascorbate promotes mercury vapour emission from plants. Plant Cell Environ 28:1487-1495.

Beckvar, N, Dillon, TM, Reed, LB. 2005. Approaches for linking whole-body fish tissue residues of mercury or DDT to biological effects thresholds. Environ Toxicol Chem 24:2094-2105.

Benoit, JM, Gilmour, CC, Mason, RP. 1999a. Estimation of mercury-sulfide speciation in sediment pore waters using octanol-water partitioning and implications for availability to methylating bacteria. Environ Toxicol Chem 18:2138-2141.

Benoit, JM, Gilmour, CC, Mason, RP, Heyes, A. 1999b. Sulfide controls on mercury speciation and bioavailability to methylating bacteria in sediment porewaters. Environ Sci Tech, 33:951-957.

Benoit JM, Gilmour CC, Heyes A, Mason RP, Miller, C. 2003. Geochemical and biological controls over methylmercury production and degradation in aquatic systems. In: Chai, Y, Braids, OC, editors. Biochemistry of Environmentally Important Trace Elements. American Chemical Society, Washington (DC), ACS Symposium Series 835:262-297.

Betti, C, Nigro, M. 1996. The comet assay for the evaluation of the genetic hazard pollutants in Cetaceans: Preliminary results on the genotoxic effects of methyl-mercury on the bottle-nosed dolphin (*Tursiops truncates*) lymphocytes *in vitro*. Marine Poll Bull 32:545-548.

Bloom, NS. 1992. On the chemical form of mercury in edible fish and marine invertebrate tissue. Can J Fish Aquat Sci 49(5):1010-1017.

Bloom, NS. 2003. Solid phase Hg speciation and incubation studies in or related to mine-site runoff in the Cache Creek watershed (CA). Final Report to the California Bay-Delta Authority. 38 p. http://loer.tamug.tamu.edu/calfed/FinalReports.htm

Bloom, N, Bollen, A, Briscoe, M, Hall, G, Horvat, M, Kim, C, Lasorsa, B, Marvin-Dipasquale, M, Parker, J. 2006. International Solid Phase Mercury Speciation Exercise (ISPMSE): Introduction and preliminary results. In: Mercury 2006 Abstracts Book; Eighth International Conference on Mercury as a Global Pollutant; 2006 Aug 6-11; Madison (WI).

Bouton, SN, Frederick PC, Spalding MG, McGill H. 1999. Effects of chronic, low concentrations of dietary methylmercury on the behavior of juvenile great egrets. Environ Toxicol Chem 18:1934-1999.

Cabana, G, Rasmussen, JB. 1994. Modeling food-chain structure and contaminant bioaccumulation using stable nitrogen isotopes. Nature 372:255-257.

Chadhain, SMN, Schaefer, JK, Crane, S, Zylstra, GJ, Barkay, T. 2006. Analysis of mercuric reductase (*merA*) gene diversity in an anaerobic mercury-contaminated sediment enrichment: Environ Microbiol 8:1746-1752.

Chen, CY, Folt, CL. 2005. High plankton densities reduce mercury biomagnification. Environ Sci Technol 39:115-121.

Choe, KY, Gill, GA, Lehman, R. 2003. Distribution of particulate, colloidal, and dissolved mercury in San Francisco Bay estuary: 1. Total mercury. Limnol Oceanogr 48(4):1535-1546.

Choe, KY, Gill, GA, Lehman, RD, Han, S, Heim, WA, Coale, KH. 2004. Sediment-water exchange of total mercury and monomethyl mercury in the San Francisco Bay-Delta. Limnol Oceanogr 49(5): 1512-1527.

Churchill, RK. 2000. Contributions of mercury to California's environment from mercury and gold mining activities; Insights from the historical record. In: Extended abstracts for the U.S. EPA sponsored meeting, Assessing and Managing Mercury from Historic and Current Mining Activities; 2000 Nov 28-30; San Francisco (CA). U.S. Environmental Protection Agency. p 33-36, S35-S48.

Cocking, D, Rohrer, M, Thomas, R, Walker, J, Ward, D. 1995, Effects of root morphology and Hg concentration in the soil on uptake by terrestrial vascular plants. Water Air Soil Pollut 80(1-4):1113-1116.

Colburn, T, vom Saal, F, Soto, AM. 1993. Developmental effects of endocrine-disrupting chemicals on wildlife and humans. Environ Health Perspect 101:378-384.

Conaway, CH, Squire, S, Mason, RP, Flegal, AR. 2003. Mercury speciation in the San Francisco Bay estuary: Marine Chem 80:199-225.

Conaway, CH, Mason, RP, Steding, DJ, Flegal, AR. 2005. Estimate of mercury emission from gasoline and diesel fuel consumption, San Francisco Bay area, California. Atmos Environ 39 101-105.

Costa, M, Liss, P. 2000. Photoreduction and evolution of mercury from seawater. Sci Total Environ 261(1-3):125-135.

Davis, JA, Greenfield, BK, Ichikawa, G, Stephenson, M. 2003a. Mercury in sport fish from the Delta region (Task 2A). Final Report Submitted to the CALFED Bay-Delta Program for the Project: An Assessment of the Ecological and Human Health Impacts of Mercury in the Bay-Delta Watershed SFEI, 88 p. http://loer.tamug.tamu.edu/calfed/FinalReports.htm

Davis, JA, Greenfield, BK, Ichikawa, G, Stephenson, M. 2008. Mercury in sport fish from the Sacramento–San Joaquin Delta region, California. Sci Total Environ 391:66–75.

Davis, J, Hunt, J, Grenier, L, Slotton, D, Ayers, S, Brodberg, R, Gassel, M, Stephenson, M, Ichikawa, G, Ujihara, A, Tan, ML, Kaslow, J, Silver, E. 2007. A Pilot Program for Monitoring, Stakeholder Involvement, and Risk Communication Relating to Mercury in Fish in the Bay-Delta Watershed ("Fish Mercury Project"), CBDA Project # ERP-02D-P67, 17 p.

Davis, JA, Yee, D, Collins, JN, Schwarzbach, SE, Luoma, SN. 2003b. Potential for increased mercury accumulation in the estuary food web: San Fran Estuary Watershed Sci 1(1):1-36 (art 4).

de Oliveira Ribeiro, CA, Belger, L, Pelletier, E, Rouleau, C. 2002. Histopathological evidence of inorganic mercury and methyl mercury toxicity in the arctic charr (*Salvelinus alpinus*). Environ Res 90:217-225.

de Souza, M.P, Huang, CPA, Chee, N, Terry, N. 1999. Rhizosphere bacteria enhance the accumulation of selenium and mercury in wetland plants. Planta 209(2):259-263.

Domagalski, J. 1998. Occurrence and transport of total mercury and methyl mercury in the Sacramento River Basin, California. J Geochem Explor 64:277-291.

Domagalksi, J. 2001. Mercury and methylmercury in water and sediment of the Sacramento River Basin, California. Appl Geochem 16:1677-1691.

Domagalski, JL, Alpers, CN, Slotton, DG, Suchanek, TH, Ayers, SM. 2004. Mercury and methylmercury concentrations and loads in the Cache Creek Watershed, California. Sci Total Environ 327:215-237.

Drevnick, PE, Sandheinrich, MB. 2003. Effects of dietary methylmercury on reproductive endocrinology of fathead minnows. Environ Sci Technol 37:4390-4396.

Driscoll, CT, Han, YJ, Chen, CY, Evers, DC, Lambert, KF, Holsen, TM, Kamman, NC, Munson RK. 2007. Mercury contamination in forest and freshwater ecosystems in the northeastern United States. BioScience 57:17-28.

Drott, A, Lambertsson, L, Björn, E, Skyllberg, U. 2007. Importance of dissolved neutral mercury sulfides for methyl mercury production in contaminated sediments. Environ Sci Technol 41:2270-2276.

Dunne, JA, Williams, RA, Martinez, ND. 2002. Food-web structure and network theory: the role of connectance and size. Proc Nat Acad Sci 99:12917-12922.

Eagles-Smith, CA. 2006. Mercury in fish: food web structure, trophic transfer, and bioaccumulation in two California lakes [dissertation]. Davis (CA): University of California. 145 p.

Eagles-Smith, CA, Suchanek, TH, Colwell, AE, Anderson, NL. in press-a. Mercury trophic transfer in a eutrophic lake: the importance of habitat specific foraging. Ecol Appl.

Eagles-Smith, CA, Suchanek, TH, Colwell, AE, Anderson, NL. in press-b. Changes in fish diets and mercury bioaccumulation in Clear Lake, California: effects of an invasive planktivorous fish. Ecol Appl.

Elbert, RA, Anderson, DW. 1998. Mercury levels, production, and hematology in western grebes from three California lakes, USA. Environ Toxicol Chem 17:210-213.

Elhassani, SB. 1982–83. The many faces of methylmercury poisoning. J Toxicol Clin Toxicol 19(8):875-906.

Essington, TE, Houser, JN. 2003. The effect of whole-lake nutrient enrichment on mercury concentration in age-1 yellow perch. Trans Am Fish Soc 132:57-68.

Evers, DC, Lane, OP, Savoy, L, Goodale, W. 2004. Assessing the impacts of methylmercury on piscivorous wildlife using a wildlife criterion value based on the common loon, 1998-2003. Gorham (ME): BioDiversity Research Institute, rpt BRI 2004-2005; Maine Department of Environmental Protection.

Evers, DC, Burgess, NM, Champoux, L, Hoskins, B, Major, A, Goodale, WM, Taylor, RJ, Poppenga, R, Daigle, T. 2005. Patterns and interpretation of mercury exposure in freshwater avian communities in northeastern North America. Ecotoxicol 14:193–221.

Fay, L, Gustin, MS. 2007. Investigation of mercury accumulation in cattails growing in constructed wetland mesocosms. Wetlands 27(4):1056-1065.

Fimreite, N. 1971. Effects of Dietary Methylmercury on Ring-necked Pheasants: With Special Reference to Reproduction. Canadian Wildlife Service.

Fjeld, E, Haugen, TO, Vøllestad, LA. 1998. Permanent impairment in the feeding behavior of grayling (*Thymallus thymallus*) exposed to methylmercury during embryogenesis. Sci Total Environ 213:247–254.

Fleming, EJ, Mack, EE, Green, PG, Nelson, DC. 2006. Mercury methylation from unexpected sources: Molybdate-inhibited freshwater sediments and an iron-reducing bacterium. Appl Environ Microbiol 72:457–464.

Frescholtz, TF, Gustin, MS, Schorran, DE, Fernandez, GCJ. 2003. Assessing the source of mercury in foliar tissue of quaking aspen. Environ Toxicol Chem 22:2114–2119

Friedmann, AS, Watzin, MC, Brinck-Johnson, T, Leitner, JC. 1996. Low levels of dietary methylmercury inhibit growth and gonadal development in juvenile walleye (*Stizostedion vitreum*). Aquatic Toxicol 35:265-278.

Friedmann, AS, Costain, EK, MacLatchy, DL, Stansley, W, Washuta, EJ. 2002. Effect of mercury on general and reproductive health of largemouth bass (*Micropterus salmoides*) from three lakes in New Jersey. Ecotoxicol Environ Safety 52:117-122.

Furness, RW, Muirhead, SJ, Woodburn, M. 1986. Using bird feathers to measure mercury in the environment: relationships between mercury content and moult. Marine Pollut Bull 17:27-30.

Gagnon, C, Pelletier, E, Mucci, A, Fitzgerald, WF. 1996. Diagenetic behavior of methylmercury in organic-rich coastal sediments. Limnol Oceanogr 41(3):428–434.

Gardfeldt, K, Munthe, J, Stromberg, D, Lindqvist, O. 2003. A kinetic study on the abiotic methylation of divalent mercury in the aqueous phase. Sci Tot Environ 304:127–136.

Gardfeldt, K, Sommar, J, Stromberg, D, Feng, XB. 2001. Oxidation of atomic mercury by hydroxyl radicals and photoinduced decomposition of methylmercury in the aqueous phase. Atmos Environ 35(17):3039-3047.

Gassell, M, Brodberg, RK, Klasing, S, Roberts, S. 2007. Draft safe eating guidelines for fish and shellfish from the San Joaquin River and South Delta (Contra Costa, San Joaquin, Stanislaus, Merced, Madera, and Fresno Counties). California Office of Environmental Health Hazard Assessment. March 2007. http://www.oehha.org/fish/so_cal/sjrsd030907.html

Gill, GA, Bruland, KW. 1990. Mercury speciation in surface freshwater systems in California and other areas: Environ Sci Technol 24:1392-1400.

Gilmour, CC, Henry, EA, Mitchell, R. 1992. Sulfate stimulation of mercury methylation in freshwater sediments: Environ Sci Technol 26:2281-2287.

Gilmour, CC, Krabbenhoft, D, Orem, W, Aiken, G. 2004. Influence of drying and rewetting on mercury and sulfur cycling in Everglades and STA Soils. 2004 Everglades Consolidated Report: Appendix 2B-1 19 p.

Green, P. 2002. Pacific Estuarine Ecosystem Indicator Research Consortium 2002. Annual Report. http://www-bml.ucdavis.edu/peeir/ack.htm

Grenier, L, Melwani, A, Hunt, J, BEzalel, S, Davis, J. 2007. California Bay-Delta Authority Fish Mercury Project Year 1 Annual Report: Sport Fish Sampling and Analysis. Final Technical Report. 29 May 2007. 158 p.

Grigal, DF. 2002. Inputs and outputs of mercury from terrestrial watersheds: a review. Environ Rev 10:1–39.

Haitzer M, Aiken GR, Ryan JN. 2002. Binding of mercury(II) to dissolved organic matter: the role of the mercury-to-DOM concentration ratio. Environ Sci Technol 36:3564-3570.

Haitzer, M, Aiken, GR, Ryan, JN. 2003. Binding of mercury(II) to aquatic humic substances: influence of pH and source of humic substances. Environ Sci Technol 37(11):2436-2441.

Hammerschmidt, CR, Sandheinrich, MB, Wiener, JG, Rada, RG. 2002. Effects of dietary methylmercury on reproduction of fathead minnows. Environ Sci Technol 36:877-883.

Hanson, PJ, Lindberg, SE, Tabberer, TA, Owens, JG, Kim, KH. 1995. Foliar exchange of mercury: Evidence for a compensation point. Water Air Soil Poll 80:373–382.

Harada, M. 1978. Congenital Minamata Disease: intrauterine methylmercury poisoning. Teratol 18:285–288.

Harris, RC, Bodaly, RA. 1998. Temperature, growth and dietary effects on fish mercury dynamics in two Ontario lakes. Biogeochem 40:175–187.

Heath, JA, Frederick, PC. 2005. Relationships among mercury concentrations, hormones, and nesting effort of white ibises (*Eudocimus albus*) in the Florida Everglades.Auk 122:255–267.

Heim, W. 2003. Methyl and total mercury in surficial sediments of the San Francisco Bay-Delta. [M.Sc.thesis], San Jose (CA): California State University San Jose.

Heinz, GH. 1979. Methylmercury: reproductive and behavioral effects on three generations of mallard ducks. J Wildlife Mgmt 43:394-401.

Heinz, GH, Hoffman, DJ. 1998. Methylmercury chloride and selenomethionine interactions on health and reproduction in mallards. Environ Toxicol Chem 17:139-145.

Henny, CJ, Hill, EF, Hoffman, DJ, Spalding, MG, Grove. RA. 2002. Nineteenth century mercury: hazard to wading birds and cormorants of the Carson River, Nevada. Ecotoxicol 11:213-231.

Hobman, JL, Brown, NL. 1997. Bacterial mercury-resistance genes. In: Sigel, A, Sigel, H, editors. Metal ions in biological systems 34: Mercury and its effects on environment and biology. New York: Marcel Dekker, Inc. p 527-568.

Hobman, JL, Wilson, JR, Brown, NL. 2000. Microbial mercury reduction. In: Lovley, DR, editor. Environmental Microbe-Metal Interactions. Washington (DC): American Society of Microbiology. p 177-198.

Hornberger, MI, Luoma, S, Van Geen, A, Fuller, C, Anima, R. 1999. Historical trends of metal in the sediment of San Francisco Bay, CA. Marine Chem 64:39-55.

Hudson, RJM, Gherini, SA, Watras, CJ, Porcella, DB. 1994. Modeling the biogochemical cycle of mercury in lakes: the mercury cycling model (MCM) and its application to the MTL study lakes. In: Watras, CJ, Huckabee, JW, editors. Mercury Pollution: Integration and Synthesis. Boca Raton (FL): Lewis. p 473-523.

Hunerlach, MP, Alpers, CN, Marvin-DiPasquale, M, Taylor, HE, De Wild, JF. 2004. Geochemistry of fluvial sediment impounded behind Daguerre Point Dam, Yuba River, California, U.S. Geol Surv Sci Invest Report 2004-5165 66 p. <http://pubs.usgs.gov/sir/2004/5165/>

Hunerlach, MP, Rytuba, JJ, Alpers, CN. 1999. Mercury contamination from hydraulic placer-gold mining in the Dutch Flat Mining District, California. In: Morganwalp, DW, Buxton, HT, editors. U.S. Geological Survey Toxic Substances Hydrology Program – Proceedings of the Technical Meeting, Charleston, SC, March 8-12, 1999 – Volume 2 of 3 – Contamination of Hydrologic Systems and Related Ecosystems, U.S. Geol Surv Water-Resources Invest Report 99-4018B, p 179-189.
http://toxics.usgs.gov/pubs/wri99-4018/Volume2/sectionB/2304_Hunerlach/

IRIS (Integrated Risk Information System). 1995. Methylmercury (MeHg) (CASRN 22967-92-6). Database maintained by the Office of Health and Environmental Assessment. U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office, Cincinnati (OH). Online at: http://www.epa.gov/iris/subst/0073.htm.

Jackson, TA. 1988. Accumulation of mercury by plankton and benthic invertebrates in riverine lakes of northern Manitoba (Canada): importance of regionally and seasonally varying environmental factors, Can J Fish Aquat Sci 45(10):1744-1757.

Johnson, B, Looker, R. 2004. Mercury in San Francisco Bay: Total Maximum Daily Load (TMDL) proposed basin plan amendment and staff report. Oakland (CA): California Regional Water Quality Control Board, San Francisco Bay Region. Staff Report; 2004 Apr 30.

Kainz, M, Lucotte, M, Parrish, CC. 2002. Methyl mercury in zooplankton: The role of size, habitat, and food quality. Can J Fish Aquat Sci 59:1606-1615.

Kelly, CA, Rudd, JWM, Bodaly, RA, Roulet, NP, St.Louis, VL, Heyes, A, Moore, TR, Schiff, S, Aravena, R, Scott, KJ, Dyck, B, Harris, R, Warner, B, Edwards, G. 1997. Increases in fluxes of greenhouse gases and methyl mercury following flooding of an experimental reservoir. Environ Sci Technol. 31:1334-1344.

Kenow KP, Gutreuter S, Hines RK, Meyer MW, Fournier F, Karasov WH. 2003. Effects of methyl mercury exposure on the growth of juvenile common loons. Ecotoxicol 12:171-182.

Kerin, E, Gilmour, CC, Roden, E, Suzuki, MT, Coates, JD, Mason, RP. 2006. Mercury methylation among the dissimilatory iron-reducing bacteria. Appl Environ Microbiol 72:7919-7921.

Kidd, KA, Hesslein, RH, Fudge, RJP, Hallard, KA. 1995. The influence of trophic level as measured by delta-15N on mercury concentrations in freshwater organisms. Water Air Soil Poll 80:1011-1015.

Kieu, LH. 2004. The seasonal influence of saltmarsh plants (*Salicornia virginica* and *Scirpus robustus*) on methylmercury production and degradation over small spatial scales in South San Francisco Bay, San Francisco [M.Sc. thesis]. San Francisco (CA): San Francisco State University. 72 p.

Kim, CS, Brown, GE Jr, Rytuba, JJ. 2000. Characterization and speciation of mercury-bearing mine wastes using X-ray absorption spectroscopy (XAS). Sci Total Environ 261:157-168.

Kim, EH. 2004. The importance of physical mixing and sediment chemistry in mercury and methylmercury biogeochemical cycling and bioaccumulation within shallow estuaries [dissertation]. College Park (MD): University of Maryland.

Kimball, T. 2006. Mercury methylation in sediments from coastal and sierra watersheds: implications for methyl mercury mitigation in the San Francisco Bay-Delta complex [M.Sc. thesis]. Monterrey (CA): Moss Landing Marine Laboratories and Division of Science and Environmental Policy, California State University Monterrey Bay, 75 p.

King, JK, Saunders, FM, Lee, RF, Jahnke, RA. 1999. Coupling mercury methylation rates to sulfate reduction rates in marine sediments. Environ Toxicol Chem 18(7)1362-1369.

Krabbenhoft, DP, Hurley, JP, Olson, ML, Cleckner, LB. 1998. Diel variability of mercury phase and species distributions in the Florida Everglades. Biogeochem 40(2-3):311-325.

Krabbenhoft DP, Wiener, JG, Brumbaugh, WG, Olson, ML, De Wild JF, and Sabinal, TJ. 1999. A national pilot study of mercury contamination of aquatic ecosystems along multiple gradients. In: Morganwalp, DW, Buxton, HT, editors. U.S. Geological Survey Toxic Substances Hydrology Program – Proceedings of the Technical Meeting; Charleston (SC); 1999 Mar 8-12; Volume 2 of 3 – Contamination of Hydrologic Systems and Related Ecosystems, U.S. Geol Surv Water-Resources Invest Report 99-4018B. p 147-160.
http://toxics.usgs.gov/pubs/wri99-4018/Volume2/sectionB/2301_Krabbenhoft/

Kraus, ML, Weis, P, Crow, JH. 1986. The excretion of heavy metals by the salt marsh cordgrass, *Spartina alterniflora*, and *Spartina's* role in mercury cycling. Marine Environ Res 20:307-316.

Krehl, WA. 1972. Mercury, the slippery metal. Nutr Today 1972(Nov/Dec):90–102.

Lacerda, LD, Fitzgerald, WF. 2001. Biogeochemistry of mercury in wetlands. Wetlands Ecol Mgmt. 9:291-293.

Langer, CS, Fitzgerald, WF, Visscher, PT, Vandal, GM. 2001. Biogeochemical cycling of methylmercury at Barn Island Salt Marsh, Stonington, CT, USA. Wetland Ecol Mgmt 9:295-310.

Lawler, SP, Morin, PJ. 1993. Food web architecture and population dynamics in laboratory microcosms of protists. Am Natural 141:675-686.

Leonard, TL, Taylor, GE, Gustin, MS, Fernandez, GCJ. 1998a. Mercury and plants in contaminated soils: 1. Uptake, partitioning, and emission to the atmosphere. Environ Toxicol Chem 17(10):2063-2071.

Leonard, TL, Taylor, GE, Gustin, MS, Fernandez, GCJ. 1998b. Mercury and plants in contaminated soils: 2. Environmental and physiological factors governing mercury flux to the atmosphere: Environ Toxicol Chem 17(10):2072-2079.

Lindberg, RD, Runnells, DD. 1984. Ground water redox reactions: an analysis of equilibrium state applied to eH measurements and geochemical modeling. Science: 225(4665):925-927.

Lindberg, S, Bullock, R, Ebinghaus, R, Engstrom, D, Feng, X, Fitzgerald, W, Pirrone, N, Prestbo, E, Seigneur, C. 2007. A synthesis of progress and uncertainties in attributing the sources of mercury in deposition. Ambio 36:19-32.

Lindberg, SE, Weijin, D, Meyers, T. 2002. Transpiration of gaseous elemental mercury through vegetation in a subtropical wetland in Florida. Atmos Environ 36:5207-5219.

Marsh, DO. 1987. Dose-response relationships in humans: Methyl mercury epidemics in Japan and Iraq. In: The toxicity of methyl mercury. Eccles, CU, Annau, Z, editors. Baltimore (MD): John Hopkins University Press. p 45-53.

Marsh, DO, Clarkson, TW, Cox, C, Myers, GJ, Amin-Zaki, L, Al-Tikriti, S. 1987. Fetal methylmercury poisoning: Relationship between concentration in single strands of maternal hair and child effects. Arch Neurol 44:1017-1022.

Marsh, DO, Myers, GJ, Clarkson, TW, Amin-Zaki, L, Tikriti, S, Majeed, MA. 1980. Fetal methylmercury poisoning: Clinical and toxicological data on 29 cases. Ann Neurol 7:348-353.

Marvin-DiPasquale, M, Agee, JL. 2003. Microbial mercury cycling in sediments of the San Francisco Bay-Delta. Estuaries 26(6):1517-1528.

Marvin-DiPasquale, M, Agee, J, Bouse, R, Jaffe, B. 2003. Microbial cycling of mercury in contaminated pelagic and wetland sediments of San Pablo Bay, California: Environ Geol 43(3):260-267.

Marvin-DiPasquale, M, Agee, J, McGowan, C, Oremland, RS, Thomas, M, Krabbenhoft, D, Gilmour, C. 2000. Methyl-mercury degradation pathways: a comparison among three mercury-impacted ecosystems: Environ Sci Technol 34(23):4908-4916.

Marvin-DiPasquale, M, Cox, MH. 2007. Legacy mercury in Alviso Slough, South San Francisco Bay, California: Concentration, speciation and mobility. U.S. Geological Survey, Open-File Report 2007-1240, 98 p. <http://pubs.usgs.gov/of/2007/1240/>

Marvin-DiPasquale, M, Hall, BD, Flanders, JR, Ladizinski, N, Agee, JL, Kieu, LH, Windham-Myers, L. 2006. Ecosystem investigations of benthic methylmercury production: a tin-reduction approach for assessing the inorganic mercury pool available for methylation. In: Mercury 2006

Abstracts Book; Eighth International Conference on Mercury as a Global Pollutant; 2006 Aug 6-11; Madison (WI).

Marvin-DiPasquale, MC, Oremland, RS. 1998. Bacterial methylmercury degradation in Florida Everglades peat sediment. Environ Sci Technol 32(17): 2556-2563.

Marvin-DiPasquale, M, Stewart, AR, Fisher, NS, Pickhardt, P, Mason, RP, Heyes, A, Windham-Myers, L., 2005. Evaluation of Mercury Transformations and Trophic Transfer in the San Francisco Bay/Delta: Identifying Critical Processes for the Ecosystem Restoration Program: Annual Report of Progress for Project # ERP-02-P40. Submitted to the California Bay Delta Authority (CBDA) On-line:
http://www.calwater.ca.gov/content/erp_calfed_mercury_2005_project_annual_reports_content.asp

Marvin-DiPasquale, M, Stewart, AR, Fisher, NS, Pickhardt, P, Mason, RP, Heyes, A, Windham-Myers, L. 2007. Evaluation of mercury transformations and trophic transfer in the San Francisco Bay/Delta: Identifying critical processes for the Ecosystem Restoration Program. Menlo Park (CA): U.S. Geological Survey. Final Report for Project # ERP-02-P40. Submitted to the California Bay Delta Authority (CBDA), 40 p.

Mason, RP, Heyes, D. Sveinsdottir, A. 2006. Methylmercury concentrations in fish from tidal waters of the Chesapeake Bay. Arch Environ Contam Toxicol 51:425–437.

Mason, RP, Reinfelder, JR, Morel, FMM. 1995. Bioaccumulation of mercury and methylmercury. Water Air Soil Pollut 80:915-921.

Mason, RP, Reinfelder, JR, Morel, FMM. 1996. Uptake, toxicity, and trophic transfer of mercury in a coastal diatom, Environ Sci Technol 30(6):1835-1845.

Matsumoto, H, Koya, G, Takeuchi, T. 1964. Fetal Minamata Disease: a neuropathological study of two cases of intrauterine intoxication by a methyl mercury compound. J Neuropathol Exp Neurol 24:563-574.

Matthiessen, A. 1998. Reduction of divalent mercury by humic substances: kinetic and quantitative aspects. Sci Total Environ 213(1-3):177-183.

Melwani, AR, Bezalel, SN, Grenier, JL, Hunt, JA, Robinson, AH, Davis, JA. 2007. The relationship between landscape features and sport fish mercury in the Sacramento-San Joaquin Delta Watershed (final draft). Oakland (CA): San Francisco Estuary Institute. Report Submitted to the CALFED Bay-Delta Program, Contract ERP–02D P67, Task 3.1; 2007 Feb 28.

Miller, CL, Mason, RP, Gilmour, CC, Heyes, A. 2007. Influence of dissolved organic matter on the complexation of mercury under sulfidic conditions. Environ Toxicol Chem 26:624-633.

Moreno, FN, Anderson, CWN, Stewart, RB, Robinson, BH, Ghomshei, M, Meech, JA. 2005. Induced plant uptake and transport of mercury in the presence of sulphur-containing ligands and humic acid. New Phytol 166:445-454.

Munthe, J, Bodaly, RA, Branfireun, BA, Driscoll, CT, Gilmour, CC, Harris, R, Horvat, M, Lucotte, M, Malm, O. 2007. Recovery of mercury-contaminated fisheries. Ambio: 36(1):33-44.

Munthe, J, Xiao, ZF, Lindqvist, O. 1991. The aqueous reduction of divalent mercury by sulfite: Water Air Soil Pollut 56:621-630.

Myers, GJ, Davidson, PW, Palumbo, D, Shamlaye, C, Cox, C, Cernichiari, E, Clarkson, TW. 2000. Secondary analysis from the Seychelles Child Development Study: the child behavior checklist. Environ Res A 84:12-19.

NAS/NRC (National Academy of Sciences/National Research Council). 2000. Toxicological effects of methylmercury. Report of the National Research Council, Committee on the toxicological effects of methylmercury. Washington(DC): National Academy Press.

Nocera, JJ, Taylor, PD. 1998. *In situ* behavioral response of common loons associated with elevated mercury (Hg) exposure. Conserv Ecol 2:10.

Nordstrom, DK, Alpers, CN. 1999. Geochemistry of acid mine waters. In: Plumlee, GS, Logsdon, MJ, editors. The environmental geochemistry of mineral deposits. Part A. Processes, methods, and health issues. Society of Economic Geologists, Reviews in Economic Geology 6A:133-160.

O'Connor, DJ, Nielsen, SW. 1981. Environmental survey of methylmercury levels in wild mink *(Mustela vision)* and otter *(Lutra canadensis)* from the northeastern United States and experimental pathology of methylmercurialism in the otter. In Chapman, JA, Pursley, D. (eds) *The Worldwide Furbearer Conference Proceedings.* Maryland: Worldwide Furbearer Conference.

O'Driscoll, NJ, Siciliano, SD, Peak, D, Carignan, R, Lean, DRS. 2006. The influence of forestry activity on the structure of dissolved organic matter in lakes: implications for mercury photoreactions: Sci Total Environ 366:880-893.

Odin, M, Feurtet-Mazel, A, Ribeyre, F, Boudou, A. 1994. Actions and interactions of temperature, pH and photoperiod on mercury bioaccumulation by nymphs of the burrowing mayfly *Hexagenia rigida*, from the sediment contamination source, Environ Toxicol Chem 13(8):1291-1302.

Orem, WH. 2004. Impacts of sulfate contamination on the Florida Everglades ecosystem: U.S. Geological Survey Fact Sheet 109-03, 4 p. <http://pubs.usgs.gov/fs/fs109-03/fs109-03.pdf>

Oremland, RS, Culbertson, CW, Winfrey, MR. 1991. Methylmercury decomposition in sediments and bacterial cultures: Involvement of methanogens and sulfate reducers in oxidative demethylation: Appl Environ Microbiol 57(1):130-137.

Oremland, RS, Miller, LG, Dowdle, P, Connell, T, Barkey, T. 1995. Methylmercury oxidative degradation potentials in contaminated and pristine sediments of the Carson River, Nevada. Appl Environ Microbiol 61:2745-2753.

Pickhardt PC, Folt CL, Chen CY, Klaue B, Blum JD. 2002. Algal blooms reduce the uptake of toxic methylmercury in freshwater food webs. Proc Nat Acad Sci (USA) 99:4419-4423.

Pickhardt, PC, Folt, CL, Chen, CY, Klaue, B, Blum, JD. 2005. Impacts of zooplankton composition and algal enrichment on the accumulation of mercury in an experimental freshwater food web. Sci Total Environ 339:89-101.

Poissant, L. 2002. Mercury surface-atmosphere gas exchange in Lake Ontario/St.Lawrence River ecosystem. Rev Sci Eau 15:229-239.

Polis, GA. 1991. Complex trophic interactions in deserts: An empirical critique of food web theory. Am Natural 147:813-846.

Post, JR, Vandenbos, R, McQueen, DJ. 1996. Uptake rates of food-chain and waterborne mercury by fish: field measurements, a mechanistic model, and an assessment of uncertainties. Can J Fish Aquat Sci 53(2):395-407.

Powell, KJ, Brown, PL, Byrne, RH, Gajda, T, Hefter, G, Sjöberg, S, Wanner, H. 2005. Chemical speciation of environmentally significant heavy metals with inorganic ligands Part 1: The Hg^{2+}– Cl–, OH–, CO_3^{2-}, SO_4^{2-}, and PO_4^{3-} aqueous systems [IUPAC Technical Report]. Pure Appl Chem 77(4):739-800.

Power, M, Klein, GM, Guiguer, KRRA, Kwan, MKH. 2002. Mercury accumulation in the fish community of a sub-arctic lake in relation to trophic position and carbon sources. J Appl Ecol 39:819-830.

Ravichandran, M. 2004. Interactions between mercury and dissolved organic matter: a review. Chemosphere 55(3):319-331.

Ravichandran, M, Aiken, GR, Ryan, JN, Reddy, MM. 1999. Inhibition of precipitation and aggregation of metacinnabar (mercuric sulfide) by dissolved organic matter isolated from the Florida Everglades. Environ Sci Technol 33:1418-1423.

Reddy, MM, Aiken, GR. 2000. Fulvic acid-sulfide ion competition for mercury ion binding in the Florida Everglades. Water Air Soil Pollut 132:89-104.

Robinson, JB, Tuovinen, OH. 1984. Mechanisms of microbial resistance and detoxification of mercury and organomercury compounds: physiological, biochemical, and genetic analyses: Microbiol Rev 48(2):95-124.

Rogers, DW, Watson, TA, Langan, JS, Wheaton, TJ. 1987. Effects of pH and feeding regime on methylmercury accumulation within aquatic microcosms. Environ Pollut 45:261-274.

Roth, DA, Taylor, HE, Domgalaski, J, Dileanis, P, Peart, DB, Antweiler, RC, Alpers, CN. 2001. Distribution of inorganic mercury in Sacramento River water and suspended colloidal sediment material. Arch Environ Contam Toxicol 40(2):161-172.

Rytuba, JJ. 2003. Mercury from mineral deposits and potential environmental impact, Environ Geol 43:326-338.

Sandheinrich, MB, Miller, KM. 2006. Effects of dietary methylmercury on reproductive behavior of fathead minnows (*Pimephales Promelas*). Env Toxicol Chem 25(11):3053–3057.

Schaefer, JK, Yagi, J, Reader, J, Cardona, T, Ellickson, K, Tel-Or, S, Barkay, T. 2004. Role of bacterial organomercury lyase (mer B) in controlling methylmercury accumulation in mercury-contaminated natural waters. Environ Sci Technol 38(16):4304-4311.

Scheuhammer, AM, Meyer, MW, Sandheinrich, MB, Murray, MW. 2007. Effects of environmental methylmercury on the health of wild birds, mammals, and fish. Ambio:36:12-18.

Scheuhammer, AM, Wong, AHK, Bond, B. 1998. Mercury and selenium accumulation in common loons (*Gavia immer*) and common mergansers (*Mergus merganser*) from Eastern Canada. Environ Toxicol Chem 19:197-201.

Sellers, P, Kelly, CA, Rudd, JWM, MacHutchon, AR. 1996. Photodegradation of methylmercury in lakes. Nature 380:694-696.

Shanley, JB, Kamman, NC, Clair, TA, Chalmers, A. 2005. Physical controls on total and methylmercury concentrations in streams and lakes of the northeastern USA. Ecotoxicol 14:125-134.

Siciliano, SD, O'Driscoll, NJ, Lean, DRS. 2002. Microbial reduction and oxidation of mercury in freshwater lakes. Environ Sci Technol 36: 3064-3068

Siciliano, SD, O'Driscoll, N, Tordon, R, Hill, J, Beauchamp, S, Lean, DRS. 2005. Abiotic production of methylmercury by solar radiation. Environ Sci Technol 39:1071-1077.

Simoneau, M, Lucotte, M, Garceau, S, Laliberte, D. 2005. Fish growth rates modulate mercury concentrations in walleye (*Sander vitreus*) from eastern Canadian lakes. Environ Res 98:73-82.

Slotton, DG. 1991. Mercury bioaccumulation in a newly impounded northern California reservoir [dissertation]. Davis(CA):University of California. 363 p.

Slotton, DG, Ayers, SM, Alpers, CN, Goldman, CR. 2004b. Bioaccumulation legacy of Gold Rush mercury in watersheds of the Sierra Nevada of California. Third Biennial CALFED Bay-Delta Program Science Conference Abstracts; 2004 Oct 4-6, Sacramento (CA). p. 388.

Slotton, DG, Ayers, SM, Reuter, JE, Goldman, CR. 1997. Gold mining impacts on food chain mercury in northwestern Sierra Nevada streams. In: Larry Walker Associates, Sacramento River watershed mercury control planning project. Rpt for the Sacramento Regional County Sanitation District, Appendix B. 74 p.

Slotton, DG, Ayers, SM, Suchanek, TH, Weyand, RH, Liston, AM. 2004a. Mercury bioaccumulation and trophic transfer in the Cache Creek watershed of California, in relation to diverse aqueous mercury exposure conditions. Rpt for the CALFED Bay-Delta Authority. 137 p. http://loer.tamug.tamu.edu/calfed/FinalReports.htm

Slotton, DG, Ayers, SM, Suchanek, TH, Weyand, RD, Liston, AM, Asher, C, Nelson, DC. 2002. The effects of wetlands restoration on the production and bioaccumulation of methylmercury in the Sacramento-San Joaquin Delta, California. Rpt for the CALFED Bay-Delta Authority. 76 p. http://loer.tamug.tamu.edu/calfed/FinalReports.htm

Slotton, DG, Reuter, JE, Goldman, CR. 1995. Mercury uptake patterns of biota in a seasonally anoxic northern California reservoir. Water Air Soil Pollut 80:841-850.

Smith, JC, Allen, PV, Turner, MD, Most, B, Fisher, HL, Hall, LL. 1994. The kinetics of intravenously administered methyl mercury in man. Toxicol Appl Pharmacol 128(2):251-256.

Snyder, RD. 1971. Congenital mercury poisoning. New Engl J Med 218:1014-1016.

Sorensen JA, Kallemeyn, LW, Sydor, M. 2005. Relationship between mercury accumulation in young-of-the-year yellow perch and water-level fluctuations. Environ Sci Technol 39:9237-9243.

Spalding MG, Frederick PC, McGill HC, Bouton SN, McDowell LR. 2000a. Methylmercury accumulation in tissues and its effects on growth and appetite in captive great egrets. J Wildlife Diseases 36:411-422.

Spalding MG, Frederick PC, McGill HC, Bouton SN, Richey LJ, Schumacher IM, Blackmore CGM, Harrison J. 2000b. Histologic, neurologic, and immunologic effects of methylmercury in captive great egrets. J Wildlife Diseases 36:423-435.

Stafford, CP, Hanson, B, Stanford, JA. 2004. Mercury in fishes and their diet items from Flathead Lake, Montana. Trans Am Fish Soc 133:349-357.

Stephenson, M, Foe, C, Gill, GA, Coale, KH. 2007. Transport, cycling, and fate of mercury and monomethyl mercury in the San Francisco Delta and tributaries: an integrated mass balance assessment approach. Annual Rpt Project # ERP-02-C06 for California Bay-Delta Authority.

Stopford, W, Goldwater, LJ. 1975. Methylmercury in the environment: a review of current understanding. Environ Health Perspect 12:115-118.

Stewart, AR, Luoma, SN, Schlekat, CE, Doblin, MA, Hieb, KA. 2004. Food web pathway determines how selenium affects aquatic ecosystems: a San Francisco Bay case study. Environ Sci Technol 38:4519-4526.

St. Louis, VL, Rudd, JWM, Kelly, CA, Bodaly, RA, Paterson, MJ, Beaty, KG, Hesslein, RH, Heyes, A, Majewski, A. 2004. The rise and fall of mercury methylation in an experimental reservoir. Environ Sci Technol 38:1348-1358.

Tollefson, L, Cordle, F. 1986. Methyl mercury in fish: a review of residue levels, fish consumption and regulatory action in the United States. Environ Health Perspect 68:203-208.

Tsai, P, Hoenicke, R. 2001. San Francisco Bay atmospheric deposition pilot study, part 1: mercury. Oakland (CA): San Francisco Estuary Institute. http://www.sfei.org/rmp/reports/air_dep/mercury_airdep/ADHg_FinalReport.pdf

Ullrich, SM, Tanton, TW, Abdrashitova, SA. 2001. Mercury in the aquatic environment: a review of factors affecting methylation: Critical Reviews in Environ Sci Technol 31(3):241-293.

U.S. EPA (U.S. Environmental Protection Agency). 1997. Mercury study report to Congress. Volume VII: Characterization of human health and wildlife risks from mercury exposure in the United States. EPA-452/R-97-009. U.S. Environmental Protection Agency, Office of Air Quality Planning & Standards and Office of Research and Development. Washington (DC).

U.S. FWS (U.S. Fish and Wildlife Service). 2003. Evaluation of the Clean Water Act Section 304(a) human health criterion for methylmercury: protectiveness for threatened and endangered wildlife in California. U.S. Fish and Wildlife Service, Sacramento Fish and Wildlife Office, Environmental Contaminants Division. Sacramento (CA). 96 p + appendix.

Waples, JS, Nagy, KL, Aiken, GR, Ryan, JN. 2005. Dissolution of cinnabar (HgS) in the presence of natural organic matter: Geochim Cosmochim Acta 69:1575-1588.

Watras, CJ, Bloom, NS, Claas, SA, Morrison, KA, Gilmour, CC, Craig, SR. 1995. Methylmercury production in the anoxic hypolimnion of a dimictic seepage lake, Water Air Soil Pollut 80:735-745.

Webber, HM, Haines, TA. 2003. Mercury effects on predator avoidance behavior of a forage fish, golden shiner (*Notemigonus crysoleucas*). Environ Toxicol Chem 22**(7)**:1556-1561.

Weber, JH. 1993. Review of possible paths for abiotic methylation of mercury(II) in the aquatic environment: Chemosphere 26(11):2063-2077.

Weis, P, Windham, L, Burke, DJ, Weis, JS. 2002. Release into the environment of metals by two vascular salt marsh plants: Marine Environ Res 54(3):325-329.

Werner, I, Anderson, S, Larsen, K, Oram, J. 2008. Chemical Stressors in the Sacramento-San Joaquin Delta Conceptual Model, Delta Regional Ecosystem Restoration Implementation Plan.

Wiener, JG, Bodaly, RA, Brown, SS, Lucotte, M, Newman, MC, Porcella, DB, Reash, RJ, Swain, EB. 2007. Monitoring and evaluating trends in methylmercury accumulation in aquatic biota. In: Harris, R., Krabbenhoft, DP, Mason, R, Murray, MW, Reash, R, Saltman, T, editors. Ecosystem responses to mercury contamination, indicators of change. Boca Raton, (FL): CRC Press LCC. p 87-122.

Wiener, JG, Knights, BC, Sandheinreich, MB, Jeremiason, JD, Brigham, ME, Engstrom, DR, Woodruff, LG, Cannon, WF, Balogh, SJ. 2006. Mercury in soils, lakes, and fish in Voyageurs National Park (Minnesota): importance of atmospheric deposition and ecosystem factors. Environ Sci Technol 40(20):6261–6268.

Wiener JG, Krabbenhoft DP, Heinz GH, Scheuhammer AM. 2003. Ecotoxicology of mercury. In: Hoffman DJ, Rattner BA, Burton GA Jr, Cairns J Jr, editors. Handbook of Ecotoxicology, 2nd ed. Boca Raton, (FL): CRC Press LCC. p 409-463.

Wiener, JG, Spry, DJ. 1996. Toxicological significance of mercury in freshwater fish. In: Beyer, WN, Heinz, GH, Redmon-Norwood, AW, editors. Environmental contaminants in wildlife: interpreting tissue concentrations. Boca Raton (FL): Lewis Publishers. p 297-339.

Windham-Myers, L, Marvin-DiPasquale, M. 2007. Direct and indirect effects of vegetation on methylmercury production in wetlands as assessed by experimental plant removal. American Geophysical Union, Fall Meeting (abstracts), 10–14 December 2007, San Francisco, CA, USA.

Windham-Myers, L, Marvin-DiPasquale, M, Agee, JL, Jew, A, Ladizinsky, N. in review. Role of salt marsh vegetation in availability of inorganic mercury in tidal marshes of northern San Francisco Bay, California. (to be submitted to Environ Sci Technol).

Windham, L, Weis, J., Weis, P. 2001. Patterns and processes of mercury (Hg) release from leaves of two dominant salt marsh plants: *Spartina alterniflora* (salt cordgrass) and *Phragmites australis* (common reed). Estuaries 24(5):787-799

Windham, L, Weis, J, Weis, P. 2003. Uptake and distribution of metals in two dominant salt marsh macrophytes, *Spartina alterniflora* (cordgrass) and *Phragmites australis* (common reed). Estuarine Coastal Shelf Sci 56:63-72

Winemiller, KO. 1990. Spatial and temporal variation on tropical fish trophic networks. Ecol Monog 60:331-367.

Wolfe, MF, Schwarzbach, S, Sulaiman, RA. 1998. Effects of mercury on wildlife: a comprehensive review. Environ Toxicol Chem 17:146-160.

Wood, ML, Foe, C, Cooke, J. 2006. Sacramento – San Joaquin Delta Estuary TMDL for Methylmercury. Staff Report. draft rpt for scientific peer review, Regional Water Quality Control Board – Central Valley Region. June 2006. (Ch 7 updated July, 2006). http://www.swrcb.ca.gov/rwqcb5/programs/tmdl/deltahg.html#SReports

Wren, CD, Hunter, DB, Leatherland, JE, Stokes, PM. 1987. The effects of polychlorinated biphenyls and methylmercury, singly and in combination, on mink. I: Uptake and toxic responses. Arch Environ Contam Toxicol 16, 441-447.

Yee, D, Collins, J, Grenier, L, Takekawa, J, Schwarzbach, S, Marvin-DiPasquale, M, Krabbenhoft, D, Evens, J. 2005. Mercury and methylmercury processes in North San Francisco Bay tidal wetland ecosystems. Annual rpt project #ERP-02D-P64, California Bay-Delta Authority. On-line: http://www.calwater.ca.gov/content/erp_calfed_mercury_2005_project_annual_reports_content.asp

Yee, D, Collins, J, Grenier, L, Takekawa, J, Schwarzbach, S, Marvin-DiPasquale, M, Krabbenhoft, D, Evens, J. 2007. Mercury and methylmercury processes in North San Francisco Bay tidal wetland ecosystems. Annual rpt project #ERP-02D-P64, California Bay-Delta Authority.

Zhang, HH, Poissant, L, Xu, X, Pilote, M, Beauvais, C, Amyot, M, Garcia, E, Laroulandie, J. 2006. Air-water gas exchange of mercury in the Bay Saint François wetlands: observation and model parameterization. J Geophys Res 111:7582-7589

Lists of Abbreviations, Acronyms, and Chemical Symbols

Abbreviations

C.I., confidence interval
cm, centimeter
kDa, kiloDalton
kg, kilogram
kg yr^{-1} (or kg/yr), kilogram per year
mg, milligram
mg kg^{-1} (or mg/kg), milligram per kilogram (part per million)
n, number of samples
ng m^{-2} hr, nanogram per square meter per hour
ppm, part per million
wt, weight
ww, wet weight
yr, year
پg g^{-1} (or پg/g), microgram per gram (part per million)
µm, micrometer

Acronyms

DGM, dissolved gaseous mercury
DO, dissolved oxygen
DOC, dissolved organic carbon
DOM, dissolved organic matter
DRERIP, Delta Regional Ecosystem Restoration Implementation Plan
GIS, geographic information system
HFO, hydrous ferric oxides
IRB, iron-reducing bacteria
PCB. polychlorinated biphenyls
PBDE, polybrominated diphenyl ethers
RGM, reactive gaseous mercury, $Hg(II)_{(g)}$
SAV, submerged aquatic vegetation
SRB, sulfate-reducing bacteria
SRR, sulfate reduction rate
TSS, total suspended sediment
UV, ultraviolet

Chemical symbols and formulas

Au, gold
AuHg, gold-mercury amalgam
C, carbon
CH_3-, methyl group
$CH_{4(g)}$, methane gas
$CO_{2(g)}$, carbon dioxide gas
$Cl^-_{(aq)}$, chloride ion (aqueous)
Eh, oxidation-reduction potential, relative to the hydrogen electrode
Fe, iron
Fe(II), ferrous iron
Fe(III), ferric iron
$H_2S_{(aq)}$, hydrogen sulfide (aqueous)
$HS^-_{(aq)}$, bisulfide ion (aqueous)
Hg, mercury
$Hg^{2+}_{(aq)}$, mercuric ion (aqueous)
$HgCl_{2(aq)}$, mercuric chloride (aqueous)
$Hg(OH)Cl_{(aq)}$, mercuric hydroxyl-chloride (aqueous)
$Hg(OH)_{2(aq)}$, mercuric hydroxide (aqueous)
$HgS^0_{(aq)}$, mercuric sulfide (aqueous)
$Hg(SH)_2{}^0_{(aq)}$, mercuric di-bisulfide (aqueous)
Hg(II), oxidized (mercuric) mercury
$Hg(II)_{(g)}$, reactive gaseous mercury
$Hg(II)_R$, reactive mercury(II)
Hg(0), elemental mercury
$Hg(0)_{(aq)}$, elemental mercury (aqueous); dissolved gaseous mercury
$Hg(0)_{(g)}$. elemental mercury (gaseous); mercury vapor
$Hg(0)_l$, elemental mercury (liquid)
HgS, mercury sulfide (cinnabar or metacinnabar)
%MeHg, percentage of Hg in the form of MeHg (i.e. MeHg/THg)
MeHg, (mono)methylmercury
Methyl Hg, (mono)methylmercury
MMHg, monomethylmercury
$O_{2(g)}$, oxygen gas
$O_{2(aq)}$, dissolved oxygen (DO)
$OH^-_{(aq),}$ hydroxide ion (aqueous)
S, sulfur
^{35}S, radiolabeled sulfur
Se, selenium
$SnCl_{2,}$ tin chloride
THg, total mercury
TotHg, total mercury

Note regarding web implementation:

Web (html) implementation of the mercury conceptual model is planned. Each box in figures 2, 3, 4, 8, and 9 will be linked to a short text passage that will open in a separate window. Web links to this report and to individual key references will be provided in each window. Additional diagrams and maps showing data from key reports will also be made accessible.