PENOBSCOT RIVER MERCURY STUDY

Chapter 2

Setting mercury remediation targets for surface sediments in the Penobscot estuary

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1 SUMMARY

The intent of this chapter is to summarize recent scientific literature regarding the toxic effects of mercury (Hg) on birds, fish, and mammals and use this information to set targets for the reduction to the exposure of various species of biota to methyl Hg in the contaminated zone of the Penobscot River. The Introduction sets out the various reasons why targets could be required for different species of biota. These reasons include: return to regional background, protection of human health, and protection of biota health. A series of sections following considers these reasons for setting targets individually and reviews available information regarding what safe levels might be. In general, recent studies have tended to lower threshold concentrations that have been shown to cause toxic effects in various biota species.

Later in the chapter, individual species are discussed and targets for Hg reductions are derived for the amount of reduction in Hg concentrations in each species. The individual species (and groups of species) considered are lobster, rock crabs, cormorants, prey fish, eels, songbirds and shorebirds, black ducks and bats. Reduction targets depend on the reason for suggesting reductions and available information. For example, for eels, reduction targets are based on the upstream background concentration, whereas for songbirds, reduction targets are based on toxic effects thresholds. Suggested targets range from no reduction needed in cormorants, about a 10% to 15% reduction in methyl Hg in rock crabs to 75% to 80% reductions in total Hg in songbirds, shorebirds, black ducks and some prey fish. In the upper Penobscot estuary, reductions of 50% or more in Hg in surface sediments are needed to meet targets; whereas, in Mendall Marsh, reductions of 80% will be required.

Based on our observations that methyl Hg in sediments is directly related to total Hg concentrations, and based on information from the scientific literature that demonstrates that Hg in biota responds directly to methyl Hg supply to food chains, it is assumed that if sediment total Hg concentrations can be decreased by remediation actions, then biota Hg concentrations will follow in proportion. It is also assumed that if significant reductions can be made in the Hg concentrations in the most contaminated zone of the Penobscot, then concentrations in biota further south in Penobscot Bay will also take place.

2 INTRODUCTION

In Phase I of the Penobscot River Mercury Study, it was shown that the Penobscot River, from Brewer to Fort Point, is contaminated with mercury (Hg). The geographic extent and pattern of the contamination and the timing of contamination is consistent with the HoltraChem plant at Orrington being the main contributor of mercury to the system. The most contaminated zone is the upper estuary between Brewer and Fort Point. It is in this upper estuary where we propose to concentrate remediation efforts (see Chapters 1 and 21). During Phase I of the Study, it was shown that concentrations of Hg in most surface sediments in the upper estuary exceed guidelines for toxic effects on invertebrates living in those sediments (see Phase I Report). Also, concentrations of Hg in some species are of concern for toxic effects and of concern for human consumption of those species. Since the Phase I Update report was produced, it has also been shown that some prey fish in the contaminated zone of the river are at or near concentrations of Hg that would cause concern for predator species eating those prey fish. It has also been recently found that concentrations of Hg in black ducks living in Mendall Marsh are very high (Chapter 14). These findings raise the question of what level of Hg reduction would be required to reduce or eliminate toxic effects and concern over human consumption.

The purpose of this chapter is to set targets for total Hg concentrations in surface sediments. The focus is total Hg in sediments because we have concluded that methyl Hg concentrations in biota in the upper estuary are largely controlled by total Hg concentrations in surface sediments. Therefore, reducing total Hg in surface sediments to target levels should lead to the protection of wildlife and of human consumers. We set surface sediment targets by first establishing targets (needed reductions) for Hg in biota. Targets for Hg in sediment were then set by reducing present sediment concentrations by the same percentage reduction as is needed for the biota. The sediment Hg targets are being used for the screening of candidate remediation options (see Chapters 1 and 21).

An underlying assumption made for this exercise is that if concentrations of total Hg in surface sediment, where methyl Hg is produced, were reduced by a certain proportion, say by 1/2, then concentrations of methyl Hg in various species of biota would be reduced proportionately, that is, by 1/2. Other studies have shown that methyl Hg in biota responds to both increases and decreases in loading rates of inorganic Hg. These studies include observational studies of contaminated systems, studies of correlations between atmospheric loading of Hg and Hg in biota, and experimental additions of Hg to ecosystems (e.g., Munthe et al. 2007; Hammerschmidt and Fitzgerald 2005; Orihel at al. 2006; Harris et al. 2007). The assumption of direct proportionality is supported by results from the MESOSIM experiment (Orihel et al. 2006, 2007) where isotopic mercury was added to mesocosms in a lake and a directly proportional response of Hg in biota was observed, at least in the short term.

The following objectives were used in setting targets of Hg concentrations in sediments and biota:

1. To reduce Hg concentrations to levels seen in areas of the region that are not contaminated by local sources (regional background).
2. To protect the health of people who eat fish, birds or shellfish.
3. To protect the health of various species of biota, especially birds and fish.
4. To protect wildlife (top predator) health.
5. To protect the health of invertebrates living in surface sediments.

2.1 Targets to Achieve Regional Background Concentrations

It would be unreasonable and unachievable to suggest remediation strategies that had the objective of reducing concentrations of Hg below regional background levels, whether in sediments or biota. We define the regional background as the concentrations of Hg found in sediments and biota in estuaries along the central Maine coast subject only to atmospheric deposition. Because Hg is found everywhere in low to moderate concentrations and even if those concentrations were thought to be causing toxic effects, suggesting reductions to below regional background would not be feasible if the objective is to remediate the impacts of the HoltraChem plant.

We have determined regional background Hg concentrations for sediments in a number of estuaries along the mid-coast of Maine (see Chapter 17). The pre-industrial background level for Hg in sediments, as determined by deep layers in sediment cores in Fort Point Cove, is very low, ranging from 18 to19 nanograms per gram dry weight (ng/g dry wt.) (Figure 2-1). Present concentrations of Hg in surface sediments in areas of the region not affected by point-sources of Hg (St. George estuary, Narraguagus estuary, East Branch of the Penobscot, and Penobscot Bay near Vinalhaven Island) are somewhat higher than the pre-industrial background, ranging from 28 to 50 ng/g dry wt. The National Oceanic and Atmospheric Administration (NOAA) considers concentrations below 51 ng/g dry wt. to be background (Buchman 2008). Areas in the region that appear to be slightly contaminated with Hg, including the Sheepscot estuary and the Penobscot River between Old Town and Veazie, have Hg in surface sediments ranging from 78 to 145 ng/g dry wt. (Figure 2-1). All these concentrations are below NOAA guidelines for any possible impact on invertebrates living in sediments. The NOAA Threshold Effects Level (the concentration below which adverse effects are expected to occur rarely) for freshwater sediments is 174 ng/g dry wt. and the NOAA Probable Effects Level (level above which toxic effects are frequently expected) is 486 ng/g dry wt. (Buchman 2008). The Old Town – Veazie (OV) reach (upstream of the Veazie dam) of the Penobscot, that is upstream of the region most contaminated with Hg, has surface sediments with total Hg concentrations that average about 90 ng/g dry wt. Therefore, 90 ng/g in sediments would appear to be the minimum concentration below which any targets would be impractical.

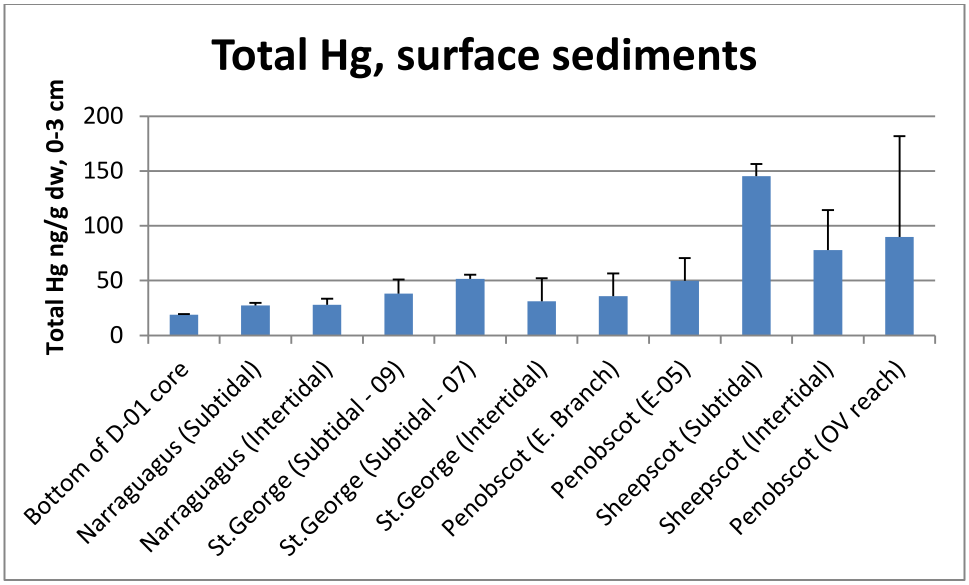

Figure 2-1. Mean concentrations of total Hg in surface (0 - 3 cm) sediments in various relatively uncontaminated locations in the Penobscot basin and other estuaries in Maine. Error bars are one standard deviation.

2.2 Targets to Protect Human Health

Different agencies in different states and countries have decided upon different levels of Hg as the accepted limit to protect the health of people eating fish, shellfish and birds. In Maine, the Department of Environmental Protection (DEP) has set 200 ng/g wet wt. methyl Hg in fish and shellfish as a protective level for human health whereas the United States Environmental Protection Agency (EPA) has set 300 ng/g wet wt. methyl Hg to protect human health. These two concentrations are in agreement when adjustments are made to account for the higher than average rate of fish consumption in Maine relative to average consumption rates for the U.S. population.

2.3 Targets to Protect Fish Health

Methyl Hg can be directly toxic to fish. Sandheinrich and Wiener (2011) have recently reviewed the literature on the toxic effects of Hg on fish. Results from recent studies have identified that harmful effects can occur at Hg concentrations that are lower than previously thought to be harmful. Harmful concentrations are approaching background levels in many areas. Sandheinrich and Wiener (2011) reviewed both laboratory and field studies and found that the results of these two different approaches were generally comparable. They catalogued effects observed by various studies by the biological endpoints that were being studied: biochemical (including the concentrations of blood plasma components and of enzymes indicative of oxidative stress), behavioral, reproductive, histological (cell structure) and growth. The population significance of biochemical effects is less clear than those that directly affect parameters such as growth and spawning behavior. Sandheinrich and Wiener (2011) concluded that adverse effects of Hg in fish are evident at 300 to 700 ng/g wet wt. on a whole body basis, typically equivalent to approximately 400 to 900 ng/g wet wt. in axial muscle.

Sandheinrich et al. (2011) identified a threshold concentration for toxic effects of 300 ng/g wet wt. on a whole body basis. Depew et al. (2012) estimated sensitivity for various biological effects and concluded that fish are most sensitive for reproductive effects, then biochemical, behavioral and growth effects. Little is known about interspecific differences in the sensitivity of fish to Hg; however, there are large differences in the sensitivity of different bird species to Hg (Heinz et al. 2009). Both Sandheinrich and Wiener (2011) and Sandheinrich et al. (2011) noted that there are many piscivorous fish populations with Hg concentrations that exceed the values assumed to cause sublethal toxic effects. Based on the above, it would appear that 500 ng/g wet wt. (0.5 micrograms per gram [µg/g]) in fish muscle tissue is a reasonable target to avoid toxic effects in fish and this will be used to set fish targets (see below).

2.4 Targets to Protect Bird Health

Toxic effects of methyl Hg in birds in four different tissues: eggs, blood, muscle, and feathers, are discussed below.

Many recent studies have examined toxic effects in birds in relation to concentrations of Hg in blood. These have been summarized for the Penobscot River Mercury Study by Evers (2012). Evers used the approach of estimating an effects concentration of methyl Hg at which a 20% reduction in fledging success in a wild bird population would be expected – this is termed the EC20 concentration. For blood concentrations in insectivorous birds, Evers' review indicated that a concentration of approximately 1,200 ng/g wet wt. (1.2 µg/g wet wt.), based on many species, is a reasonable estimate of the EC20. Sandheinrich (2010) concluded that 3 µg/g was Lowest Observed Adverse Effects Level (LOAEL) and that the threshold concentration for birds was probably somewhat lower. Recent studies indicating somewhat lower threshold concentrations of Hg in bird blood include DeSorbo and Evers (2007) who studied bald eagle reproductive success and found a negative correlation between blood Hg in nestlings and reproductive success (young fledged) over the range 100 to 1,200 ng/g wet wt. (0.1 to 1.2 µg/g wet wt.) in blood. The California clapper rail in San Francisco Bay has been shown by Ackerman et al. (2012) to have its body condition negatively affected at blood Hg concentrations starting over the range of about 150 to 1,500 ng/g (0.15 to 1.5 µg/g). However, Ackerman et al. (2008b) found no effect of Hg on the survival of hatchling terms in San Francisco Bay CA at levels of 6.44 µg/g wet wt. in blood (6.44 µg/g fresh wt. in feathers). Franceshini et al. (2009) demonstrated that baseline concentrations of corticosterone, an important metabolic regulating hormone, in tree swallow blood was correlated with blood Hg concentrations over the range of about 100 to 1,000 ng/g (0.1 to 1.0 µg/g) total Hg in blood. There are also a number other studies documenting effects on birds at levels below 2,000 ng/g (2.0 µg/g wet wt.) in blood. Of particular relevance to this review is the paper by McKay and Mayer (2012) which examined the singing behavior of male breeding Nelson's sparrows and found aberrations in birds related to Hg in blood, over the range of approximately 1,000 to over 6,000 ng/g wet wt. Thus, 1,200 ng/g wet wt. (1.2 µg/g wet wt.) would appear to be a reasonable toxic effects threshold for Hg in the blood of insectivorous birds as recommended by Evers (2012).

Effects of methyl Hg on bird eggs have been well studied recently, although the double-crested cormorants, an important species of consideration in the Penobscot System, have received little attention. Heinz (1979) proposed a threshold concentration for effects on mallard eggs of 800 ng/g wet wt (0.8 µg/g wet wt.) and the studies of Heinz and Hoffman (2003) supported this threshold concentration. In a review conducted for the Penobscot River Mercury Study, Sandheinrich (2010) concluded that 1,000 ng/g wet wt. (1.0 µg/g wet wt.) in eggs is the LOAEL for bird eggs and that the threshold concentration is probably somewhat lower than this concentration. Evers (2012), in his review of the recent literature, recommended the use of and EC20 of 650 ng/g wet wt. (0.65 µg/g wet wt.) for piscivorous birds like cormorants. Heinz et al. (2009) conducted egg injection experiments with 26 different species of bird eggs and found quite large inter-specific differences in tolerances to methyl Hg. They found that cormorants have quite low sensitivity to methyl Hg, compared to most of the other bird species tested. This implies that the threshold of 800 ng/g is quite protective for the cormorants found in the Penobscot.

Concerning probable toxic effects threshold concentrations for Hg in the muscle tissue of birds, there have been few studies done and none published in the last 25 years. This is perhaps due to the difficulty of collecting muscle tissue samples from birds. A study of the common loon with a range of Hg concentrations of 760 to 2,320 ng/g wet wt. (0.76 to 2.32 µg/g wet wt.) in muscle found that at the high end of the range there was reduced male adult body condition and reduced reproductive success (Barr 1986). So, setting a toxic effects threshold for Hg in bird muscle tissue is problematic and we have not done so for this report. However, the black duck is the only species of concern for which we have sampled muscle tissue and we do have blood Hg levels for that species as well, so the difficulty is establishing a concern concentration for muscle for birds may not be a problem for the present purposes.

There have been a number of recent studies on toxicity of Hg in birds related to concentrations in feathers that allow for an estimation of toxic effects thresholds. Hg concentrations in bird feathers reflect the concentrations of Hg circulating in the blood at the time of feather formation, which is directly related to Hg exposure through the diet (Johnels et al. 1979; Solonen and Lodenius 1990; Becker et al. 1994; Bearhop et al. 2000; Fournier et al. 2002; Ackerman et al. 2008a). Given a constant environmental exposure, Hg concentrations in individual feathers decline with the order in which they are molted. This reduction in feather concentration is matched by a decline in Hg concentrations in the blood during the molt period (Braune and Gaskin 1987). As a result, for each molt cycle the first feathers molted have the greatest Hg concentrations and the last feathers molted generally have the lowest Hg concentrations. It follows that the Hg concentrations in feathers associated with toxicity to the bird are most relevant for specific feather types (Westermark et al. 1975; Applequist et al. 1984; Wolfe et al. 1998; Jackson et al. 2011).

Heinz (1979) reported significant reproductive effects in a treatment group of mallards (*Anas platyrhynchos*) which had mean Hg concentrations in primary feathers between 9,000 and 11,200 ng/g fresh wt. (9.0 and 11.2 µg/g fresh wt.). Bowerman et al. (1994) reported mean total Hg concentrations in four types of bald eagle (*Haliaeetus* *leucocephalus*) feathers (primary, secondary, tail and body) ranging from 19,000 to 23,000 ng/g fresh wt. (19 to 23 µg/g fresh wt.), with no significant reproductive effects associated with those Hg concentrations. The authors noted that the co-occurrence of elevated organochlorine contaminants, with known reproductive effects, may have masked Hg-related toxicity. Heath and Frederick (2005) reported mean Hg concentrations in breast feathers from female white ibis (*Eudocimus albus*) sampled in the Everglades of 6,440 ± 510 ng/g fresh wt. (6.44 ± 0.51ug/g fresh wt.) and a negative correlation between Hg concentrations in feathers and estradiol concentrations. Brasso and Cristol (2008) reported reproductive effects in tree swallows (*Tachycineta bicolor*) nesting in contaminated areas along the South River, VA, associated with total Hg concentrations in primary feathers (P1) of 13,550 ± 6,940 ng/g fresh wt. (13.55 ± 6.94 (min-max, 5-26) µg/g fresh wt.). Hg concentrations in P1 feathers from the reference area were reported as 2,340 ± 870 ng/g fresh wt. (2.34 ± 0.87 (1-4) µg/g fresh wt.). Ackerman et al. (2008c) found no effect of Hg in stilt and avocet in San Francisco Bay at levels up to 44,300 ng/g (44.3 µg/g fresh wt.) in feathers. Jackson et al. (2011) modeled the probabilities of nest failure in the Carolina wren (*Thryothorus ludovicianus*) associated with estimated increases in blood and feather Hg concentrations. At a 20% reduction in nest success (EC20), the estimated Hg concentration in body feathers was 3,400 ng/g fresh wt. (3.4 µg/g fresh wt.) and the estimated Hg concentration in tail feathers was 4,700 ng/g fresh wt. (4.7 µg/g fresh wt.). Evers (2012) followed Jackson et al. (2011) in recommending these threshold levels. Eisler (2006) proposed a Hg criterion for the protection of birds of 5,000 ng/g fresh wt. (5.0 µg total Hg/g fresh wt.) in feathers, with no distinction made for feather type. Ackerman et al. (2008a) used both 5,000 ng/g fresh wt. (5 µg/g fresh wt.) and 20,000 ng/g fresh wt. (20 µg/g fresh wt.) in feathers as the range of concentrations associated with avian toxicity. Thus, it seems that 5,000 ng/g (5 µg/g) Hg in bird feathers is a reasonable toxicity threshold.

2.5 Targets to Protect Fish Predator Health

One important consideration in setting targets for Hg in prey species is to protect the health of predators. The fish-eating birds and mammals that we sampled in the Penobscot system were generally not at risk from eating contaminated fish, probably mainly because they were not primarily feeding in the river itself (see, however, section on bird health). Therefore, this section will concentrate on the recent literature concerning effects of Hg in prey on fish predators (summarized above) and threshold concentrations of Hg in prey producing toxic effects in those fish predators.

There has been a large amount of recent research on this topic, summarized by Sandheinrich (2010) and Depew et al. (2012). Sandheinrich found a LOAEL concentration of 180 ng/g (0.18 µg/g) (whole body) in prey species and noted that the threshold effect concentration is lower. Depew et al. (2012) provided threshold concentrations for prey of fish for various classes of effects, including lethality, growth, behavior, reproductive and biochemical. The two classes of effects for methyl Hg toxicity for which fish were most sensitive were reproductive (spawning success, reduced fecundity, altered levels of sex steroids and altered spawning behavior) and biochemical (altered blood or plasma biochemistry, altered neurochemistry, changes in gene transcription, changes in cell physiology, pathological damage to organs or tissues and altered behavior). For biochemical effects, Depew et al. (2012) found that the highest no effects level in prey fish was 60 ng/g (0.06 µg/g), the Threshold Effects Level (TEL) was 180 ng/g, the LOAEL was 140 ng/g. They proposed a threshold level of 60 ng/g (0.06 µg/g). For reproductive effects, Depew et al. (2012) found the highest no effects level was 40 ng/g, the LOAEL was 50 ng/g and proposed a threshold level of 40 ng/g. LOAEL is the lowest concentration at which effects have been observed whereas the TEL is calculated from the LOAEL and No Observed Adverse Effect Level (NOAEL), being the square root of the product of the 50th percentile of the NOAEL and the 15th percentile of the LOAEL. Based on these data, a reasonable level to protect predator health might be 50 ng/g (0.05 µg/g) in prey fish, although it is difficult to translate sublethal effects in individuals, such as altered neurochemistry, to population level effects. This level is very low compared to those often seen in natural fish populations.

In the Penobscot, the main predatory fish are eels; however, striped bass are known to enter the river at various times.

2.6 Targets to Protect Mammal Health

There are few relevant studies to determine what might be toxic concentrations of Hg in bats in the Penobscot system. Toxic effects of Hg in humans are thought to start at around 10,000 ng/g wet wt. (10 µg/g wet wt.) in hair (Murata et al. 1999; Burton et al. 1977). Burton et al. (1977) found decreased swimming ability and deviant behavior in mice at fur Hg concentrations of 7,800 to 10,800 ng/g wet wt. (7.8 to 10.8 µg/g wet wt.) and Sleeman et al. (2010) found tissue abnormalities in an otter with fur concentrations of Hg of 183,000 ng/g wet wt. (183 µg/g wet wt.). Effects on adrenocortical levels in big brown bats at a contaminated site in Virginia were not evident at 28,000 ng/g wet wt. (28 µg/g wet wt.) in fur and 110 ng/g wet wt. (0.11 µg/g wet wt.) in blood (Wada et . 2010). Neurochemical changes were also not found in little brown bats sampled at the same contaminated site (mean fur Hg 132 ± 94 µg/g fresh wt.; Nam et al. 2012). It is important to note, however, that just because effects are not found in a particular study, it does not necessarily mean that effects did not exist because effects could be taking place on some response that was not measured. So, a reasonable limit for Hg in bat fur for toxic effects might be 10,000 ng/g wet wt. (10 µg/g wet wt.).

2.7 Target Setting for Individual Species and Compartments

Using the thresholds established above, we will now go through a series of exercises to set targets for a number of species that we studied in the Penobscot estuary.[1]

American lobster – Average total Hg (unadjusted) in lobster tail at five sites in the area of Fort Point Cove and the south end of Verona Island in the Penobscot exceeded the 200 ng/g wet wt. criterion (Table 2-1). From 50% to 96% of lobsters at these sites exceeded 200 ng/g wet wt. total Hg. These concentrations are higher than the range of Hg concentrations found in lobsters at seven reference sites in Maine that are further

[1] Note that Hg means and ranges given in this section are all calculated from raw concentrations, unadjusted for individual size, age or sex, using data from all sampling years (usually 2006 to 2010). Means may not be the same as those presented with the objective of comparing years, sites or areas, where statistical adjustment for factors such as size are used to provide comparability of data.

from the Penobscot and HoltraChem. However, there is not sufficient information to determine toxic effects thresholds for the lobsters themselves (Sandheinrich 2010). A target concentration of 200 ng/g wet wt. methyl Hg or total Hg (we have found that 100% of the total Hg in lobster tails in the Penobscot is methyl Hg) would protect human consumers eating lobster from these sites and would put these animals close to background concentrations at the highest sites in Maine (Sowles 1997). Reductions to 200 ng/g total Hg in lobster tail muscle would require decreases ranging from less than 10% to about 50% or more.

Table 2-1: Target setting for American lobster.

American lobster	Location or source of information	Total Hg ng/g wet wt. in tail (means & % > 200)	Methyl Hg ng/g wet wt. (means)
CONCENTRATIONS IN AREAS OF CONCERN	Penobscot Bay (S. Verona)	485 (96%)	
	Penobscot Bay (Fort Point)	228 (50%)	
	Penobscot Bay (Odom Ledge)	291 (77%)	
	Penobscot Bay (Wilson Point)	338 (80%)	
	Penobscot Bay (Turner Point)	203 (75%)	
CONCENTRATIONS IN REFERENCE AREAS	Seven sites in Maine (Sowles 1997)	82 to 208	
Toxic effects levels	Sandheinrich 2010	Insufficient information	Insufficient information
HUMAN CONSUMPTION TARGETS	Maine DEP action level of methyl Hg	200 (if 100% methyl Hg)	200
TARGET CONCENTRATIONS	Below consumption target	200 (if 100% methyl Hg)	200

<u>Rock crabs</u> – Average methyl Hg concentrations in rock crabs exceeded the 200 ng/g wet wt. limit for human consumption set by Maine DEP at four sites in upper Penobscot Bay and individual crabs exceeded this limit at six additional sites. Unadjusted methyl Hg averaged 319, 308, 204,and 227 ng/g at the sites with means exceeding 200 ng/g in 2006 (the sites were, 1-3, 1-4, 1-10, and 3-1; see Phase I Update report for site locations). The proportion of these samples exceeding 200 ng/g was 25, 33%, 30% and 40%. At uncontaminated reference locations, total Hg in rock crabs and blue crabs (which share similar diets) were noticeably lower than in Penobscot Bay. Mean concentrations at three reference locations in Connecticut and Florida ranged from 60 to

156 ng/g wet wt. (Jop et al. 1997; Karouna-Renier et al. 2007); whereas, total Hg in crab muscle in New York-New Jersey Harbor was 170 ng/g wet wt. (NYSDEC 1996). Therefore, background concentrations are lower than those required to meet targets for human consumption. Reductions of about 10% to 15% of current concentrations would be required for safe human consumption.

American eel – The only species of fish in the contaminated zone of the Penobscot River that would appear to be at direct risk from the toxic effects of mercury is the American eel. It was concluded above that fish with more than 500 ng/g wet wt. in muscle tissue are at risk due to sublethal toxic effects. Over the period 2007 to 2010 the mean total Hg concentrations in eels in the Brewer to Orrington (BO) and Orrington to Bucksport (OB) reaches of the river ranged from 496 to 619 ng/g wet wt. in axial muscle (Table 2-2). The upstream reach of the Penobscot (Old Town to Veazie) had a grand mean concentration of Hg in eels of 333 ng/g wet wt.

There are a large amount of data on background concentrations from uncontaminated sites in Maine, North America and Europe (Table 2-2). Concentrations of Hg at reference sites varied widely (Table 2-2), from 60 to over 1,000 ng/g wet wt. However, it is difficult to compare concentrations from other sites to the Penobscot because of differences in the life stage, size and age of fish analyzed.

On the basis of criteria designed to avoid sublethal toxic effects to the eels themselves, a target of about 500 ng/g wet wt. would be appropriate. To protect the health of people eating eels, the target for eels would be 222 ng/g wet wt. (Table 2-2, assuming that 90% of the mercury in eels is methyl Hg – See Chapter 14 for data from this study on the percentage of methyl Hg in eel muscle from the Penobscot River). However, based on the information we have been able to obtain from the literature and by talking to Maine Department of Marine Resources (Dr. Gail Whippelhauser, Personal Communication, October 2012), there does not appear to be a large amount of eels from the Penobscot consumed by local people. Also, any remediation efforts would not to be able to bring the average concentration of Hg in eel muscle below about 260 ng/g wet wt., the normalized concentration in the Old Town to Veazie reach, directly upstream of the contaminated zone of the river. So, 260 ng/g wet wt. is a reasonable and practical target for eels. From 82% to 91% of the eels sampled in 2007 to 2010 in the BO and OB reaches of the Penobscot exceeded this target (Table 2-2). Reaching a target concentration of 260 ng/g wet wt. total Hg in eels would require reductions of about 50% of present concentrations, and while this would not reach the target concentration of 222 ng/g wet wt. total Hg for human consumption, it would reach the target at what appears to be the regional background concentration.

Table 2-2: Target setting for American eel.			
AMERICAN EEL		**Total Hg in muscle (ng/g wet wt.) (means) and number exceeding target of 260**	**Methyl Hg in muscle (ng/g wet wt.) (means)**
CONCENTRATIONS IN AREAS OF CONCERN	Penobscot BO3	498 (90%)	
	Penobscot BO4	619 (87%)	
	Penobscot OB1	454 (82%)	
	Penobscot OB5	496 (91%)	
CONCENTRATIONS IN REFERENCE AREAS	Penobscot (Old Town – Veazie reach)	333 (60%)	
	European reference sites (Italy, Bosnia) (Mancini et al. 2005; Has-Schön et al. 2008)	60 – 160	
	N American reference sites Nova Scotia (Freeman & Horne 1973) Georgia (Burger et al. 2001) New Brunswick (Zitko et al. 1971) (excludes one sample from an impoundment) St. Lawrence estuary (Hodson et al. 1994)	720 150 50-946 (whole body)	400 70 – 760 (10 sites)
	Maine reference sites Leaman (1999), 3 Maine rivers	330-642	
TOXIC EFFECTS LEVELS	Sublethal effects	500	
HUMAN COSUMPTION TARGETS	USEPA action level	333 (if 90% methyl Hg)	300
	Maine DEP action	222 (if 90% methyl Hg)	200
TARGET CONCENTRATIONS	**BASED ON CONCENTRATIONS IN**	**260**	**234**

Table 2-2: Target setting for American eel.			
AMERICAN EEL		**Total Hg in muscle (ng/g wet wt.) (means) and number exceeding target of 260**	**Methyl Hg in muscle (ng/g wet wt.) (means)**
	OLD TOWN – VEAZIE REACH		

Prey fish – All of the prey fish that have been sampled in the Penobscot are known to be eaten by a wide variety of predators, including eels, striped bass, crabs, and piscivorous birds.

For rainbow smelt over the period 2006 to 2010 in the Penobscot estuary, Hg concentrations in muscle tissue averaged from about 45 to 120 ng/g wet wt. (0.045 to 0.12 µg/g wet wt.), unadjusted for size. This would be approximately 30 to 100 ng/g on a whole body basis. From 25% to 100% of rainbow smelt exceeded the toxic effects threshold to protect predator health of 50 ng/g (0.05 µg/g). For the two upstream sites where rainbow smelt were caught (OB1E and OB1S), 92% to 100% of fish exceeded 50 ng/g in muscle. For sites between Bucksport and the south end of Verona Island, 57% to 97 % of fish exceeded 50 ng/g, whereas for sites further south, 26% to 83% of fish exceeded 50 ng/g. Hg in rainbow smelt from 25 Canadian lakes averaged 60 ng/g (0.06 µg/g), also exceeding the toxic effects threshold (Swanson et al. 2006); however, Hg exposure of lake populations would be expected to be greater than Hg exposure of river populations.

For *Fundulus* (mummichogs), over the period 2006 to 2010, Hg in muscle tissue at various sites in the BO and OB reaches varied from a mean of 131 to 307 ng/g wet wt., which is approximately 110 to 250 ng/g (0.11 to 0.25 µg/g) on a whole body basis. All fish therefore exceeded the threshold of 50 ng/g (0.05 µg/g for toxic effects), by about 2 to 10 times. At two wetland sites (W17 and W21), mean values were 218 and 352 ng/g, and therefore all these fish also exceeded the toxic effects threshold to protect predator health.

For tomcod, unadjusted mean Hg concentrations in muscle tissue ranged from 100 to 290 ng/g (0.10 to 0.29 µg/g) over the 2006 to 2010 sampling period. This translates to approximately 75 to 220 ng/g (0.075 to 0.22 µg/g) on a whole body basis, or about 1½ to 4 times the toxic effects threshold to protect the health of predators. Tomcod at two contaminated sites have been reported in the literature, as follows: 150 ng/gwet wt. (0.15 µg/g wet wt.) (mean from 12 fish) in muscle tissue from St. Lawrence River estuary, Quebec, Canada (Duchesne et al. 2004); 140 ng/g wet wt. (0.14 µg/g wet wt.) (one fish only) in skinned and eviscerated whole fish from the Meadowlands, NJ (Santoro and Koepp 1986). Hg in tomcod in Labrador, Canada was about 130 ng/g (0.13 µg/g), but these fish were much larger than those sampled in the Penobscot (Anderson 2011).

Double-crested cormorants – As noted above, the threshold concentration for toxic effects of Hg on bird eggs is around 800 ng/g wet wt. (0.8 µg/g wet wt.). It was reported in the Phase I report that Hg in cormorant eggs at one site in 2006 averaged 880 ng/g wet wt. (0.88 µg/g wet wt.), which is above the threshold (Table 2-3). However, over the whole sampling period (2006 to 2008), the unadjusted mean total Hg concentrations at the three nesting sites furthest north in the estuary were all below 800 ng/g and few eggs exceeded the this threshold (Table 2-3). Concentrations of Hg in cormorant eggs appear in the contaminated area of Penobscot Bay do appear to be above regional background concentrations (Table 2-3). However, cormorants have been shown to be relatively insensitive to Hg as compared to other species (Heinz et al. 2009). It would therefore appear that no reductions in Hg in this species are required to prevent toxic effects.

Table 2-3: Target setting for Hg in double-crested cormorant eggs.

DOUBLE-CRESTED CORMORANTS		**Mean total Hg (ng/g wet wt.) in eggs at different sites**
CONCENTRATIONS IN AREA OF CONCERN	Luce Cove	670 (0 of 7 (0%) over 800)
	Sandy Point	360 (0 of 87 (0%) over 800)
	Fort Point	790 (2 of 3 (67%) over 800)
CONCENTRATIONS IN REFERENCE AREAS	8 reference sites in Maine (data from BioDiversity Research Institute, Gorham, Maine)	280 (range – 110 – 450)
	2 “clean” reference sites in N. America (Bay of Fundy, Canada and WA; Burgess and Braune 2001; Henny et al. 1989)	260 – 280
	Hg-impacted San Francisco Bay-Delta (Davis et al. 2005)	170 – 1,170
Toxic concentrations	Reproductive effects in other bird species	800
TARGET CONCENTRATIONS	Based on toxic levels and background concentrations	Present concentrations

Nelson’s sparrows – Sampling over the period 2006 to 2010 at six sites in Mendall Marsh and W17 determined that total Hg in blood of adult Nelson’s sparrows ranged from (unadjusted) means of 2,900 to 5,200 ng/g wet wt. (2.9 to 5.2 µg/g wet wt.) (Table 2-4). These levels are much higher than the regional background, as established by us and by Shriver et al. (2006) which ranged from about 400 to 700 ng/g (0.4 to 0.7 µg/g). As established in the above section on threshold concentrations, there is now reliable recent evidence that sublethal toxic effects are probably present as low as 1,200 ng/g (1.2 µg/g) in bird blood. Reducing Hg concentrations in Nelson’s sparrows by up to 75%

will put populations below this threshold level. Even greater reductions would be required to put Nelson's sparrows within the range of regional background concentrations.

Table 2-4: Target setting for Nelson's sparrow in Mendall Marsh.		
NELSON'S SPARROWS		**Total Hg in blood ng/g wet wt. (means)**
CONCENTRATIONS IN AREAS OF CONCERN	Six sites in Mendall Marsh and W17, 2006 - 2010 (after hatch year)	2,900 – 5,200
CONCENTRATIONS IN REFERENCE AREAS	Five reference areas in southern Maine (our sampling)	500 – 700
	Five coastal marshes in Maine (Shriver et al. 2006)	410 +/- 160
TOXIC CONCENTRATIONS	Reproductive effects in other bird species	1,200
TARGET CONCENTRATIONS	Below toxic concentrations	<1,200 (up to 75% reduction)

Other songbirds and shorebirds – There are four other species of songbirds and shorebirds that inhabit Mendall Marsh and other contaminated wetlands adjacent to the Penobscot River that show elevated concentrations of Hg in blood (Table 2-5).

Hg in the blood of song sparrows at contaminated sites in the Penobscot ranged from averages of 110 to 1,920 ng/g wet wt. (0.11 to 1.92 µg/g wet wt.) as compared to a target to eliminate toxic effects of 1,200 ng/g wet wt. (1.2 µg/g wet wt.) and as compared to means of 20 to 350 ng/g wet wt. (0.02 to 0.35 µg/g wet wt.) at reference sites in Massachusetts and Maine (Table 2-5). From 0% to 67% of birds at the 17 contaminated sites sampled exceeded the 1,200 ng/g wet wt. threshold as compared to 0% of birds at reference sites. Reductions of up to about 40% would be required to lower blood Hg concentrations in song sparrows to the toxic threshold but would not reduce them to concentrations found at reference sites.

Hg in the blood of swamp sparrows at contaminated sites ranged from about 400 to over 3,000 ng/g wet wt., or up to about 2.5 times the toxic effects threshold of 1,200 ng/g wet wt. (Table 2-5). Thus, levels in swamp sparrows needed to be reduced up to 3 times to be below the toxic effects threshold. From 33% to 100% of the individual birds at these contaminated sites exceeded the toxic effects threshold. At three reference sites in Maine, mean blood concentrations of Hg in swamp sparrows were 140 to 490 ng/g (0.14 to 0.49 µg/g) and 0% to 20% of these birds exceeded the toxic effects threshold. Average Hg in swamp sparrows at seven sites in Wisconsin ranged from 80 to 220 ng/g (0.08 to 0.22 µg/g). Reductions of up to about 60% would be required to lower blood Hg concentrations in swamp sparrows to the toxic threshold but, as for song sparrows, would not reduce them to concentrations found at reference sites.

In red-winged blackbirds, average Hg in blood at eight contaminated sites in the Penobscot ranged from 1,450 to 6,260 ng/g wet wt. (1.45 to 6.26 µg/g wet wt.) (Table 2-5). From 32% to 100% of individual birds at eight contaminated sites had Hg higher than the toxic effects threshold. At two reference sites in Maine and New Jersey, Hg in blackbird blood was 170 and 230 ng/g (0.17 and 0.23 µg/g), respectively. None of the birds at the Maine reference site exceeded the toxic effects threshold. To reduce red-winged blackbirds to the toxic effects limit, they would have to come down by up to about 80%. Reductions of this amount in blackbirds would not reduce all birds to within the concentrations seen at reference sites.

Virginia rails at 10 sites in Mendall Marsh also exceeded the toxic threshold, by up to about 3 times (Table 3-5). Means at these contaminated sites ranged from 870 to 2,940 ng/g wet wt. (0.87 to 2.94 µg/g wet wt.) and 0% to 100% of individual rails exceeded the toxic effects threshold. Mean Hg at two reference sites in Maine were 160 and 410 ng/g (0.16 and 0.41 µg/g); none of the birds at these sites exceeded the toxic effects threshold. Reductions of up to 70% in Virginia rails will reduce mean concentrations below toxic levels but will not reduce all birds to within the range seen in reference areas (Table 2-5).

Table 3-5: Current levels of Hg in the blood (µg/g wet wt.) of four species of birds that inhabit contaminated wetlands in the Penobscot basin.

SPECIES	Background concentrations (ng/g wet wt. blood)	Total Hg in blood at contaminated Penobscot sites (ng/g wet wt.)	Reduction needed to meet target of 1,200 ng/g (wet wt. in blood)
Song sparrow	210 – 350 (Eastern Massachusetts: Evers et al. 2005)	110 – 1,920 (16 sites)	Up to about 40%
	20 – 33 (our sampling – 8 sites in Maine)		
Swamp sparrow	About 80 to 202 in 7 wetlands in Wisconsin (Strom and Brady 2011)	370 – 3,160 (10 sites)	Up to about 60%
	140 – 490 (3 sites in Maine, our sampling)		
Red-winged blackbird	230 (Meadowlands, New Jersey; Tsipoura et al. 2008)	1,450 – 6,260 (8 sites)	Up to about 80%
	170 (Spurwink Marsh, Maine, our sampling)		
Virginia rail	160 – 410 (2 reference sites, Maine, our sampling)	960 – 3,890 (10 sites)	Up to about 70%

Black guillemots – Average Hg concentrations in black guillemots in 2007 (the only year they were sampled) at two sites were 700 to 1,200 ng/g wet wt. (0.7 to 1.2 µg/g wet wt.) in eggs, and 1,300 ng/g wet wt. (1.3 µg/g wet wt.) at both sites in adult blood. This compares to concentrations in eggs at a reference site of about 500 ng/g (0.5 µg/g). Although no information is available about Hg toxicity thresholds in guillemots specifically, the toxic effects threshold for Hg in bird eggs generally is around 800 ng/g and is about 1,200 ng/g for Hg adult bird blood. Current concentrations exceed these toxic thresholds in many individuals. If concentrations at sites in Penobscot Bay were lowered by about 35%, this would place most individual birds and eggs below toxic thresholds; these reductions would not lower Hg in guillemot eggs below those of a reference site.

Black ducks – As noted above, there are no recent studies of the likely threshold concentrations of Hg in the muscle tissue of birds that would cause harm to the birds themselves. The only study, on loons, conducted 25 years ago, found decreased body condition and reproductive success when concentrations in muscle was more than 2,000 ng/g wet wt. (2 µg/g wet wt.) (Barr 1986). However, our studies on the black duck indicate that muscle concentrations follow blood concentrations very closely. Hg in black duck muscle averaged about 750 ng/g wet wt. (0.75 µg/g wet wt.) from the most recent sampling. Hg in black duck blood averaged about 800 ng/g, compared to the presumed limit of 1,200 ng/g for toxic effects. Hg in the blood and muscle of ducks is essentially all methyl Hg. To protect human consumers of black ducks, Hg in muscle tissue needs to come down from 750 to 200 ng/g (about a 75% reduction of present concentrations). It should be noted that these targets were set based on consumption of fish and shellfish, so the use of the 200 ng/g concentration for black ducks has not been set by state agencies. If blood levels in black ducks also were reduced by about 75% from of present concentrations, this would put all individual ducks below the threshold limit of 1,200 ng/g for sublethal effects and thus the health of the black ducks would also be protected.

Bats – Little brown bats sampled at six contaminated sites in the Penobscot in 2008 averaged 10,900 to 23,600 ng/g wet wt. (10.9 to 23.6 µg/g wet wt.) in fur, as compared to the toxic effects threshold of 10,000 ng/g (10 µg/g). Bats caught near the HoltraChem site were the highest (23,600 ng/g). From 12% to 91% of the bats from the six sampling sites exceeded 10,000 ng/g. Little brown bats at two reference sites sampled by us in Maine averaged 3,900 and 4,200 ng/g; 0% to 7% of these bats exceeded 10,000 ng/g. Fur from little brown bats in Ontario and Quebec, Canada averaged 1,300 to 2,500 ng/g dry wt. (1.3 to 2.5 µg/g dry wt.) (which should be equivalent to wet wt. or fresh wt. for fur) (Hickey et al. 2001) and big brown bats (a different species) at a reference site in Virginia averaged 400 ng/g (0.4 µg/g) wet wt. in blood and 10,900 ng/g fresh wt. (10.9 µg/g fresh wt.) in fur (Wada et al. 2010). A contaminated site in Virginia (South River) had big brown bats with mean Hg concentrations of 28,000 ng/g wet wt. in fur (Wada et al. 2010). Thus, levels observed at HoltraChem are 4 to 5 times those seen at the South River, although it may not be valid to compare different species of bats. Toxic effects of mercury may take effect at about 10,000 ng/g wet wt. in fur (see above). Approximately a 50% reduction in Hg in the fur of bats sampled near the HoltraChem site and at Bald

Hill Cove would be required to reduce mean levels to below the presumed toxic effects threshold.

3 CONCLUSION

Of species living primarily in river habitats (in the upper estuary), most require reductions of about 50% of present concentrations to meet reduction targets, including eels, and prey species such as rainbow smelt, Fundulus, and tomcod (Table 3-6). These reduction targets are based on toxic effects to one species (eels) and predicted toxic effects on fish predators eating prey fish (rainbow smelt, *Fundulus*, tomcod). Thus, if total Hg in surface sediments in the contaminated zone can be reduced from present concentrations of about 800 to 900 ng/g dry wt. to about 400 to 450 ng/g, these reductions can be accomplished. For biota species living in Penobscot Bay, reductions of 1/2 to 2/3 are likewise required to meet target concentrations. Although we are not recommending any active remediation of Penobscot Bay, it is likely that reductions of Hg in the upper estuary would eventually result in similar reductions in Hg in surface sediments in the Bay. Reductions of Hg in surface sediments to about 400 to 450 ng/g will also reduce those sediments to concentrations below the NOAA Probable Effects Level for total Hg in freshwater sediment of 486 ng/g and in marine sediment of 700 ng/g (Buchman 2008).

Reductions needed in Mendall Marsh and other marshes in the contaminated zone to affect the decreases required in biota living in those habitats are greater and therefore are much more challenging (Table 3-6). Nelson's sparrows and black ducks will require up to 75% reductions in exposure to methyl Hg, while other song and shore birds will require up to an 80% reduction. To approach these levels of Hg reduction in Mendall Marsh biota, we will recommend that more than one type of active remediation procedure be used in the marsh (see Chapter 1 and 21).

Table 2-6: Summary of species considered for reduction targets, their primary habitat, and the amount of reduction from present concentrations that would be required to meet targets, as discussed above.

Species	Habitat	Reductions needed
Lobster	Bay	Up to 50%
Rock crabs	Bay	10% to 15 %
Eels	River	50 %
Rainbow smelt	River and Bay	Up to 60%
Fundulus	River and Bay	50% to 80%
Tomcod	River and Bay	35% to 80%
Cormorants	Bay	None
Nelson's sparrows	Wetlands	Up to 75%
Other songbirds and shorebirds	Wetlands	Up to 80%

Table 2-6: Summary of species considered for reduction targets, their primary habitat, and the amount of reduction from present concentrations that would be required to meet targets, as discussed above.		
Species	**Habitat**	**Reductions needed**
Black guillemots	Bay	35%
Black ducks	Wetlands	75%
Bats	River and wetlands	50%

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5 REFERENCES

Ackerman, J.T., C.A. Eagles-Smith, J.Y. Takekawa, J.D. Bluso, T.L. Adelsbach. 2008a. Mercury concentrations in blood and feathers of prebreeding Forester's terns in relation to space use of San Francisco Bay, California, USA, habitats. Environmental Toxicology and Chemistry. 27(4):897-908.

Ackerman, J.T., C.A. Eagles-Smith, J.Y. Takekawa and S.A. Iverson. 2008b. Survival of port fledgling Forster's terns in relation to Hg exposure in San Francisco Bay. Ecotoxicology. 17:789-801.

Ackerman, J.T., J.Y. Takekawa, C.A. Eagles-Smith, S.A. Iverson. 2008c. Mercury contamination and effects on survival of American avocet and black-necked stilt checks in San Francisco Bay. Ecotoxicology. 17:103-116.

Ackerman, J.T., C.T. Overton, M.L. Casazza, J.Y. Takekawa, C.A. Eagles-Smith, R.A. Keister and M.P. Herzog. 2012. Does mercury contamination reduce body condition of endangered California clapper rails? Environmental Pollution. 162:439-448.

Anderson, M.R. 2011. Duration and extent of elevated mercury levels in downstream fish following reservoir creation. River Systems. 19/3:167-176.

Applequist, H., S. Asbirk, I. Draback. 1984. Mercury monitoring: mercury stability in bird feathers. Marine Pollution Bulletin. 15:22-24.

Barr, J.F. 1986. Population dynamics of the common loon (*Gavia immer*) associated with mercury-contaminated waters in northwestern Ontario. Occasional Paper - Canadian Wildlife Service. 56:1-23.

Bearhop, S., G.D. Ruxton, R.W. Furness. 2000. Dynamics of mercury in blood and feathers of great skuas. Environmental Toxicology and Chemistry. 19(6):1638-1643.

Becker, P.H., D. Henning, R.W. Furness. 1994. Differences in mercury contamination and elimination during feather development in gull and tern broods. Archives of Environmental Contamination and Toxicology. 27:162-167.

Bowerman, M.W., E.D. Evans, J.P. Giesy, S. Postupalsky. 1994. Using feathers to assess risk of mercury and selenium to bald eagle reproduction in the Great Lakes region. Archives of Environmental Contamination and Toxicology. 27:294-298.

Brasso, R.L. and D.A. Cristol. 2008. Effects of mercury exposure on the reproductive success of tree swallows (*Tachycineta bicolor*). Ecotoxicology. 17:133-141.

Braune, B.M. and D.E. Gaskin. 1987. Mercury levels in Bonaparte's Gulls (Larus philadelphia) during autumn molt in the Quoddy Region, New Brunswick, Canada. Archives of Environmental Contamination and Toxicology. 16:539-549.

Buchman, M.F. 2008. NOAA Screening Quick Reference Tables, NOAA OR&R Report 08-1. Seattle, WA, Office of Response and Restoration Division, National Oceanic and Atmospheric Administration. 34 p.

Burger, J., K.F. Gaines, C.S. Boring, W.L. Stephens, J. Snodgrass, M. Gochfeld. 2001. Mercury and selenium in fish from the Savannah River: Species, trophic level, and locational differences. Environmental Research. 87: 108-118.

Burgess, N. and B. Braune. 2001. Increasing trends in mercury concentrations in Atlantic and Arctic seabird eggs in Canada. Society of Environmental Toxicology and Chemistry Europe, Abstract, 2001 meeting.

Burton, G.V., R.J. Alley, G.L. Rasmussen, P. Orton, V. Cox, P. Jones, D. Graff. 1977. Mercury and behavior in wild mouse populations. Environmental Research. 14:30-34.

Davis, J., J. Hunt, T. Adelsbach, D. Crane, L. Phillips. 2005. Monitoring legacy and emerging pollutants at the top of the San Francisco Bay food web. Society of Environmental Toxicology and Chemistry, Abstract, Baltimore meeting.

Depew, D.C., N. Basu, N.M. Burgess, L.M. Campbell, E.W. Devlin, P.E. Drivnick, C.R. Hammerschmidt, C.A. Murphy, M.B. Sandheinrich, J.G. Wiener. 2012 Toxicity of dietary methylmercury to fish: Derivation of ecologically meaningful threshold concentrations. Environmental Toxicology and Chemistry. 31:1-12.

DeSorbo, C.R. and D.C. Evers. 2007. Evaluating exposure of Maine’s bald eagle population to mercury: Assessing impacts on productivity and spatial exposure patterns. Report BRI 2007-02. BioDiversity Research Institute, Gorham ME, 36 p.

Duschesne, J.F., B. Levesque, D. Gauvin, B. Braune, S. Gingras, E. Dewailly. 2004. Estimating the mercury exposure dose of a population of migratory bird hunters in the St. Lawrence River region, Quebec, Canada. Environmental Research. 95:207-214.

Eisler, R. 2006. Mercury Hazards to Living Organisms. CRC Press, Taylor and Francis Group. New York.

Evers, D.C. 2012. The effects of methyl mercury on wildlife. A comprehensive review. BioDiversity Research Institute. October 2012, 50 pages, with appendix tables. Produced for the Penobscot River Mercury Study.

Evers, D.C., N.M. Burgess, L. Champoux, B. Hoskins, A. Major, W.M. Goodale, R.J. Taylor, R. Poppenga, T. Daigle. 2005. Patterns and interpretation of mercury exposure in freshwater avian communities in northwestern North America. Ecotoxicology. 14:193-221.

Fournier, F., W.H. Karasov, K.P. Kenow, M.W. Meyer, R.K. Hines. 2002 The oral bioavailability and toxicokinetics of methylmercury in common loon (*Gavia immer*) chicks. Comparative Biochemistry and Physiology Part A. 133:703-714.

Franceshini, M.D., O.P. Lane, D.C. Evers, J.M. Reed, B. Hoskins, L.M. Romero. 2009. The coricosterone stress response and mercury contamination in free-living tree swallows Tachycinata bicolor. Ecotoxicology. 18: 514-521.

Freeman, H.C., D.A. Horne. 1973. Total mercury and methyl mercury content of the American eel (*Anguilla rostrata*). Journal of the Fisheries Research Board of Canada. 30:173-176.

Hammerschmidt, C.R., W.F. Fitzgerald. 2005. Methylmercury in mosquitoes related to atmospheric mercury deposition and contamination. Environmental Science & Technology. 39:3034-3039.

Harris, R.C. and 23 co-authors. 2007. Whole-ecosystem study shows rapid fish-mercury response to changes in mercury deposition. Proceedings of the National Academy of Sciences of the United States. 104:16586-16591.

Has-Schön, E., I. Bogut, V. Rajkovic, S. Bogut, M. Cacic, J. Horvatic. 2008. Heavy metal distribution in tissues of six fish species included in human diet, inhabiting freshwaters of the Nature Park “Hutovo Blato” (Bosnia and Herzegovina). Archives of Environmental Contamination and Toxicology. 54:75-83.

Heath, J.A. and P.C. Frederick. 2005. Relationship among mercury concentrations, hormones, and nesting effort of white ibises (*Eudocimus albus*) in the Florida Everglades. The Auk. 122:255-267.

Heinz, G.H. 1979. Methylmercury: Reproductive and behavioral effects on three generations of mallard ducks. Journal of Wildlife Management. 43:394-401.

Heinz, G.H. and D.J. Hoffman. 2003. Embryotoxic thresholds of mercury: Estimates from individual mallard eggs. Archives of Environmental Contamination and Toxicology. 44:257-264.

Heinz, G.H., D.J. Hoffman, J.D. Klimstra, K.R. Stebbins, S.L. Konrad, C.A. Erwin. 2009. Species differences in the sensitivity of avian embryos to methylmercury. Archives of Environmental Contamination and Toxicology. 56:129-138.

Henny, C.J., L.J. Blus, S.P. Thompson. 1989. Environmental contaminants, human disturbance and nesting of double-crested cormorants in northwestern Washington. Colonial Waterbirds. 12: 198-206.

Hickey, M.B.C., M.B. Fenton, K.C. MacDonald, C. Souliere. 2001. Trace elements in the fur of bats (Chiroperta: Vespertilionidae) from Ontario and Quebec, Canada. Bulletin of Environmental Contamination and Toxicology. 66:699-706.

Hodson, P.V., M. Castonguay, C.M. Couillard, C. Desjardins, E. Pelletier and R. McLeod. 1994. Spatial and temporal variations in chemical contamination of American eels, *Anguilla rostrata*, captured in the estuary of the St. Lawrence River. Canadian Journal of Fisheries and Aquatic Sciences. 51:464 - 478.

Jackson, A.K., D.C. Evers, M.A. Etterson, A.M. Condon, S.B. Folsom, J. Detweiler et al. 2011. Mercury exposure affects the reproductive success of a free-living terrestrial songbird, the Carolina wren (*Thryothorus ludovicianus*). The Auk. 128:759-769.

Johnels, A., G.Tyler, T. Westermark. 1979. A history of mercury levels in Swedish fauna. Ambio. 8(4):160-168.

Jop, K.M., R.C. Biever, J.R. Hoberg, S.P. Shepherd. 1997. Analysis of metals in blue crabs, *Callinectes sapidus*, from two Connecticut estuaries. Bulletin of Environmental Contamination and Toxicology. 58:311-317.

Karouna-Renier, N.K., R.A. Snyder, J.G. Allison, M.G. Wagner, K.R. Rao. 2007. Accumulation of organic and inorganic contaminants in shellfish collected in estuarine waters near Pensacola, Florida: Contamination profiles and risks to human consumers. Environmental Pollution. 145:474-488.

Leaman, N.G.G. 1999. Mercury contamination in the silver stage of American eels, *Anguilla rostrata*, from three rivers in Maine. Zoology, University of Maine, Orono, Maine.

Mancini, L., S. Caimi, S. Ciardulla, M. Zeiner, P. Bottoni, L. Tancini, S. Cautadella, S. Caroli. 2005. A pilot study of the contents of selected pollutants in fish from the Tiber River (Rome). Microchemical Journal. 79:171-175.

McKay, J.L. and C.R. Maher. 2012. Relationship between blood mercury levels and components of male song in Nelson’s sparrows (*Ammodramus nelsoni*). Ecotoxicology. 21:2391-2397.

Munthe, J., R.A. Bodaly, B.A. Branfireun, C.T. Driscoll, C.C. Gilmour, R. Harris, M. Horvat, M. Lucotte, O. Malm. 2007. Recovery of mercury contaminated ecosystems. Ambio. 36:33-44.

Murata, K., P. Weihe, A. Renzoni, F. Debes, R. Vasoncelos, F. Zino, S. Araki, P.J. Jørgensen, R.J. White, P. Grandjean. 1999. Delayed evoked potentials in children exposed to methylmercury from seafood. Neurotoxicology and Teratology. 21:343-348.

Nam, D.H., D. Yates, P Ardapple, D.C. Evers, J. Schmerfeld and N. Basu. 2012. Elevated mercury exposure and neurochemical alterations in little brown bats (*Myotis lucifugs*) from a site with historical mercury contamination. Ecotoxicology. 21:1094-1101.

NYSDEC (New York State Department of Environmental Conservation). 1996. Chemicals in fish, shellfish and crustaceans from the New York – New Jersey Harbor Estuary. PCB, Organochlorine Pesticide and Mercury. Edited by NYSDEC Conservation.

Orihel, D.M., M.J. Paterson, C.C. Gilmour, R.A. Bodaly, P.J. Blanchfield, H. Hintelmann, R.C. Harris, J.W.M. Rudd. 2006. Effect of loading rate on the fate of Hg in littoral mesocosms. Environmental Science & Technology. 40:5992-6000.

Orihel, D.M., M.J. Paterson, P.J. Blanchfield, R.A. Bodaly, H. Hintelmann. 2007. Experimental evidence of a linear relationship between inorganic mercury loading and methylmercury accumulation by aquatic biota. Environmental Science & Technology. 41:4952-4958.

Sandheinrich, M.B. 2010. Review of recent literature on the effects of methylmercury on wildlife, invertebrates, and fish. Report for the Penobscot River Mercury Study, December 2007, revised January 20, 2010. 46 p.

Sandheinrich, M.B., S.P. Bhavsar, R.A. Bodaly, P.E. Drevnick, E.A. Paul. 2011. Ecological risk of methylmercury to piscivorous fish of the Great Lakes region. Ecotoxicology. 20:1577-1587.

Sandheinrich, M.B. and J.G. Wiener. 2011. Methylmercury in freshwater fish: Recent advances in assessing toxicity of environmentally relevant exposures. Pp 169-190 IN WN Beyer and JP Meador (eds.) Environmental contamination in biota: Interpreting tissue concentrations, CRC Press, Boca Raton, FL.

Santoro, E.D. and S.J. Koepp. 1986. Mercury levels in organisms in proximity to an old chemical site. Marine Pollution Bulletin. 17:219-224.

Shriver, W.G., D.C. Evers, T.P. Hodgman, B.J. MacCulloch, R.J. Taylor. 2006. Mercury in sharp-tailed sparrows breeding in coastal wetlands. Environmental Bioindicators. 1:129-135.

Sleeman, J.M., D.A. Cristol, A.E. White, D.C. Evers, R.W. Gerhold and M.K. Keel. 2010. Mercury poisoning in a free-living northern river otter (*Lontra canadensis*). Journal of Wildlife Diseases. 46:1035-1039.

Solonen, T. and M. Lodenius 1990. Feathers of birds of prey as indicators of mercury contamination in southern Finland. Holarctic Ecology. 13(3):229-237.

Sowles, J. 1997. Memo to S. Ladner, Court Deposition Exhibit, U.S. District Court, District of Maine, dated September 29, 1997. 4 p.

Strom, S.E., R.S. Brady. 2011. Mercury in swamp sparrows (*Melospiza geogiana*) from wetland habitats in Wisconsin. Ecotoxicology. 20:1694-1700.

Swanson, H.A., T.A. Johnston, D.W. Schindler, R.A. Bodaly, D.M. Whittle. 2006. Mercury bioaccumulation in forage fish communities invaded by rainbow smelt (*Osmerus mordax*). Environmental Science & Technology. 40:1439-1446.

Tsipoura, N., J. Burger, R. Feltes, J. Yacabucci, D. Mizrahi, C. Jeitner, M. Gochfeld. 2008. Metal concentrations in three species of passerine birds breeding in the Hackensack Meadowlands of New Jersey. Environmental Research. 107:218-228.

Wada, H., D.W. Yates, D.C. Evers, R.J. Taylor, W.A. Hopkins. 2010. Tissue mercury concentrations and adrenocortical responses of female big brown bates (*Eptesicus fuscus*) near a contaminated river. Ecotoxicology. 19:1277-1284.

Westermark, T., T. Odsjö and A.G. Johnels. 1975. Mercury content of bird feathers before and after Swedish ban on alkyl mercury in agriculture. Ambio. 4(2):87-92.

Wolfe, M.F., S. Schwarzbach, R.A. Sulaiman. 1998. Effects of mercury on wildlife: a comprehensive review. Environmental Toxicology and Chemistry. 17(2):146-160.

Zitko, V., B.J. Finlayson, D.J. Wildish, J.M. Anderson, A.C. Kohler. 1971. Methylmercury in freshwater and marine fishes in New Brunswick, in the Bay of Fundy, and on the Nova Scotia Banks. Journal of the Fisheries Research Board of Canada. 28:1285-1291.

APPENDIX 2-1:

Sandheinrich 2010 report on methyl mercury toxicity in biota

APPENDIX 2-2:

Evers 2012 report on methylmercury toxicity to wildlife