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Original Contribution

Marine Foraging Birds As Bioindicators of Mercury in the Gulf of Maine

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Abstract: From existing databases, we compiled and evaluated 604 total mercury (Hg) levels in the eggs and blood of 17 species of marine foraging birds from 35 Gulf of Maine islands to provide baseline data and to determine the best tissue, age class, and species for future biomonitoring. While mean Hg levels in most species did not exceed adverse effects thresholds, levels in some individual eggs did; for all species arithmetic mean egg Hg levels ranged from 0.04 to 0.62 (μg/g, wet weight). Piscivorous birds had higher Hg levels than invertivores. Leach's storm-petrel (*Oceanodroma leucorhoa*), razorbill (*Alca torda*), and black guillemot (*Cepphus grylle*) adult blood and egg Hg levels were higher than other species. Our results indicate that adult blood is preferable to chick blood for detecting long-term temporal trends because adult levels are higher and not confounded by metabolic effects. However, since we found that eggs and adult blood are comparable indicators of methylmercury bioavailability, we determined that eggs are the preferred tissue for long-term Hg monitoring because the relative ease in collecting eggs ensures consistent and robust datasets. We suggest specific sampling methods, and based on our results demonstrate that common eider (*Somateria mollissima*), Leach's storm-petrel, double-crested cormorant, and black guillemot are the most effective bioindicators of Hg of the Gulf of Maine.

Keywords: mercury, seabirds, waterbirds, Gulf of Maine, bioindicators

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Introduction

Although mercury (Hg) is a naturally occurring element (Nriagu and Pacyna 1988; Nriagu 1989), anthropogenic mercury levels in the North Atlantic have increased over the last 100 years (Slemr and Langer 1992; Asmund and Nielsen 2000; Mason and Sheu 2002) and in Maine have increased since 1970 (Perry et al. 2005). This increase is attributed generally to anthropogenic input (Lockhart et al. 1998; Mason and Sheu 2002). The historical increase has been reflected in tissues from seabirds in the North Atlantic (Appelquist et al. 1985; Thompson et al. 1992, 1998; Monteiro and Furness 1997), Canadian Arctic (Braune 2007), and within the Gulf of Maine watershed (Evers et al. 1998, 2005). This increase in global Hg levels since the 1900s is of concern because Hg is a persistent toxic heavy metal that bioaccumulates, is biomagnified in wildlife, and has negative neurological and reproductive impacts (Scheuhammer et al. 2007; Wolfe et al. 2007).

Studies on seabirds in many parts of the world have found Hg levels thought to be elevated above background levels, specifically in Antarctica (Norheim et al. 1982), North America (Pearce et al. 1979; Braune et al. 2001), Europe (Furness et al. 1995), Russia (Stout et al. 2002), Asia (Kim et al. 1996), and the North Pacific (Burger and Gochfeld 2000). Moreover, researchers have found these elevated Hg levels in species with diverse foraging strategies (Elliott et al. 1992; Thompson et al. 1992, Burger and Gochfeld 2000).

Birds are used frequently as bioindicators to evaluate where and to what extent Hg is bioavailable (Scheuhammer 1987; Wolfe et al. 1998, 2007; Evers et al. 2005; Scheuhammer et al. 2007). Past studies in eastern Canada found differences in Hg levels in the eggs of several seabird species (Pearce et al. 1979). While there has been a significant effort to characterize Hg levels in seabirds in North America, no studies have sampled marine foraging birds broadly and concurrently at multiple sites in the Gulf of Maine—a region that has been identified to have some of the highest Hg levels in North America (Evers and Clair 2005). Therefore, bird researchers in the Gulf of Maine formed the Gulf of Maine Seabird Contaminant Assessment Network (GOMSCAN) to share existing waterbird Hg data. GOMSCAN is led by BioDiversity Research Institute and is composed of the Canadian Wildlife Service, Kent Island Bowdoin Scientific Station, Maine Coastal Islands National Wildlife Refuge, Maine Department of Inland Fisheries and Wildlife, National Audubon Society, Shoals Marine Laboratory of Cornell University, University of New Brunswick, University of New Hampshire, and U.S. Fish and Wildlife Service. GOMSCAN's goals are to identify species, locations, and trophic levels where Hg is concentrating, and to refine sampling methods for future contaminant studies.

This article presents findings of an initial collaborative screening effort and methods for future coordinated sampling. The main goals of this study were to determine the relationship and patterns of Hg levels in waterbirds within the Gulf of Maine, to evaluate blood and eggs as indicators of methylmercury (MeHg) bioavailability, and to identify species that are the most effective bioindicators of Hg availability in this marine system. We used the following criteria to evaluate if a species was suitable as a bioindicator: Are the birds abundant and widespread in Gulf of Maine, do they represent specific foraging guilds, and/or do they have the potential for Hg levels above estimated effects thresholds. We focused on a 6-year time period (2001–2006) and did not attempt to assess temporal trends in Hg levels.

Methods

From 2001 to 2006 (plus two sites in 1998), GOMSCAN members collected data on Hg levels in individual eggs, egg composites, and blood through multiple concurrent studies of 17 species of aquatic birds breeding on 35 sites in the Gulf of Maine (Figure 1, Appendix A). Viable and non-viable bird eggs were collected and placed in polyethylene bags (15% of the samples were analyzed as composites). During processing, we collected standard egg morphometrics (length, breadth, total egg weight, egg content weight, and volume), determined embryo development, placed the contents into labeled, chemically clean jars, and froze the samples (see detailed methods in Evers et al. 2003).

Juvenile (nest-bound chicks, young of year) and adult birds were captured at their breeding colonies and blood taken. Blood was collected by venipuncture of the coetaneous ulnar (wing) vein. Generally, less than 1.0 cc of blood was collected because most laboratories require only 0.25 cc of blood for Hg analysis. Blood was placed in labeled vials or tubes and frozen. All necessary state and federal permits were in place prior to field collections.

The data utilized in this compilation were generated at multiple laboratories over a period of several years. Differences in sample preparation and analytical methods were considered insignificant, although they were not

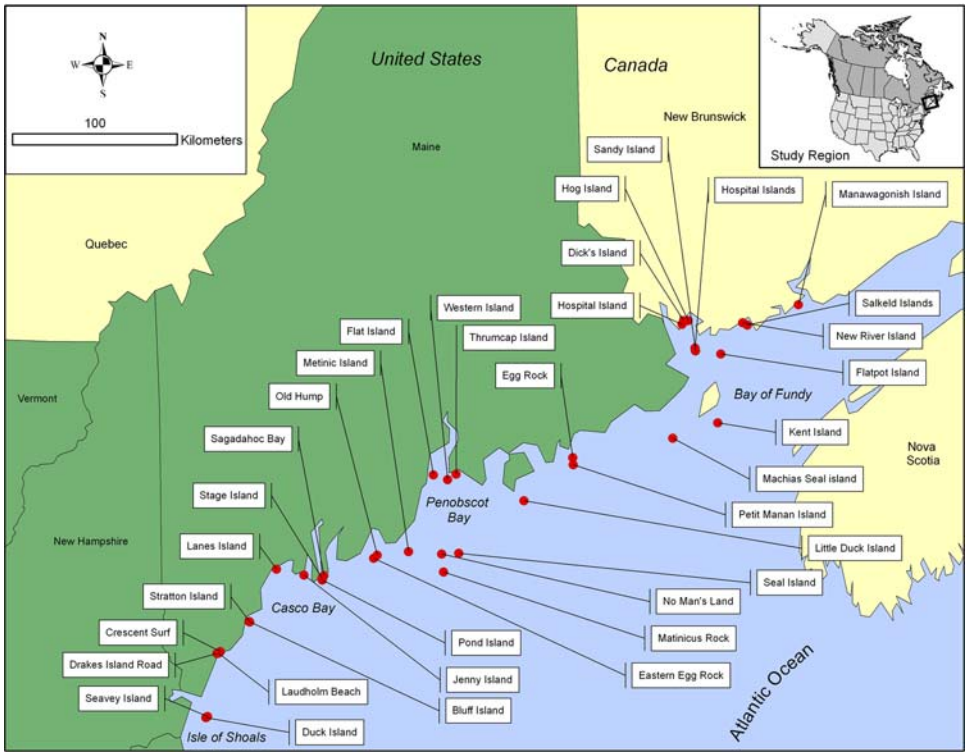

Figure 1. Sampling locations of the Gulf of Maine study area (n = 35 islands).

quantified and split samples were not submitted to all labs to determine inter-lab differences. The quality assurance and quality control procedures—including standard reference material (SRM), blanks, duplicates, and spikes—that each laboratory used did meet federal standards set for the U.S. and Canada (e.g., recoveries of all SRMs were within established certified ranges). Mercury analysis was either by cold vapor atomic absorption spectroscopy (CVAAS) or direct mercury analyzer. Eggs were homogenized, lyophilized, and powdered. Only total Hg was analyzed because > 90% of egg (Scheuhammer et al. 2001; Bond and Diamond 2008) and 95% of blood (Fournier et al. 2002; Rimmer et al. 2005) Hg levels are MeHg. Therefore, we used total Hg levels in these tissues to measure the availability of MeHg. Egg Hg was analyzed as dry weight and converted to wet weight using [(dry weight*(100-% moisture))/100]. We standardized our Hg data to wet weight because it was the common measure in the multiple databases we used for this study. Each laboratory determined percent moisture during the freeze-drying process. Many eggs cracked during the freezing process in the field; therefore, we were able to measure egg volume on only a subset of the eggs. Using this subset, we calculated egg fresh mass (total egg mass/egg volume) and found no significant difference from measured egg mass, demonstrating insignificant loss of moisture (ANOVA, $F_{1,134} = 0.14$, $P = 0.71$). Hg in blood was measured as wet weight.

We performed statistics with JMP (SAS Institute Inc. 2001). Each egg composite (two or more eggs homogenized) was treated as a sample size of one. We $\log_{10}$ transformed the data to improve normality and homoscedasticity. Breaking the Hg data down by bird species and age into 30 groups, the log-transformed Hg data were normally distributed for 76% of the groups as determined using one-sample Kolmogorov–Smirnov tests (SYSTAT 2007). We tested for differences among species in tissue Hg concentrations using analysis of variance (ANOVA), followed by Tukey–Kramer HSD paired comparisons. We pooled data from the same location and species across years (2001–2006). We were not able to test for interaction between island and species because all species were not present at each sampling site. For species sampled at more than four colonies, we tested for Hg differences among islands. We compared the variance of $\log_{10}$-transformed Hg data for individual eggs within double-crested cormorant (*Phalacrocorax auritus*) clutches and among composite samples of five eggs taken from different cormorant nests within several nesting colonies, to assess the relative importance of within-clutch and within-island variation in egg Hg concentrations. To determine the influence of foraging strategy and trophic status on tissue Hg levels among species, we grouped waterbirds into three foraging categories based upon documented diets (Appendix B).

Results

Geometric mean Hg levels in the blood of adult waterbirds differed among seven species measured (Figure 2A; ANOVA, $F_{6,196} = 17.63$, $P < 0.0001$). Blood Hg concentrations in adult razorbills were greater than those in great black-backed (*Larus marinus*) and herring gulls (*L. argentatus*) (Tukey HSD, $P < 0.05$). Arithmetic mean Hg levels in tissues of each species are listed in Appendix B. Juvenile waterbirds also differed in their geometric mean Hg levels in blood (Figure 2B; ANOVA, $F_{11,144} = 15.57$, $P < 0.0001$). Blood Hg concentrations in juvenile black-crowned night-herons (*Nycticorax nyctocorax*) were greater than those in Leach's storm-petrels (*Oceanodroma leucorhoa*) (Tukey HSD, $P < 0.05$). In paired data sets, Hg levels in adult blood were significantly higher than juvenile blood (Table 1; all $P < 0.0001$). The ratio of geometric mean blood Hg levels in adults versus juveniles of Leach's storm-petrel, herring gull, common tern (*Sterna hirundo*), razorbill (*Alca torda*), and Atlantic puffin (*Fratercula arctica*) ranged from 3.8 to 21.61, averaging overall 7.8:1 in the five species (Table 1).

Geometric mean egg Hg levels differed among the 12 species measured (Figure 2C; ANOVA, $F_{11,232} = 37.08$, $P < 0.0001$). Egg Hg concentrations were greater in Leach's storm-petrel, black guillemot (*Cepphus grylle*), and razorbill compared to herring gull and glossy ibis (*Plegadis falcinellus*) (Tukey HSD, $P < 0.05$). Egg Hg levels were not different among islands for common terns (ANOVA, $F_{5,64} = 0.909$, $P = 0.48$) nor for composite samples of double-crested cormorant eggs (ANOVA, $F_{6,39} = 2.09$, $P = 0.07$). However, black guillemot egg Hg levels were different among islands (ANOVA $F_{4,23} = 17.24$, $P < 0.0001$). Geometric mean egg Hg levels were greater in guillemot eggs from Western Island than those from the other four islands sampled (Tukey HSD, $P < 0.05$). There were low within-clutch (Table 2) and within-island differences in Hg levels of cormorant eggs (Table 3). In cormorant clutches where we analyzed each egg, clutches with higher mean Hg levels also had a greater Hg range and standard deviation, despite using $\log_{10}$-transformed Hg data. Based on limited data, this relationship was positive and significant for a polynomial curve fit (2nd degree polynomial, $r^2 = 0.99$, $df = 5$; $P = 0.01$).

Geometric mean Hg concentrations in eggs and adult blood differed among waterbirds classified by foraging

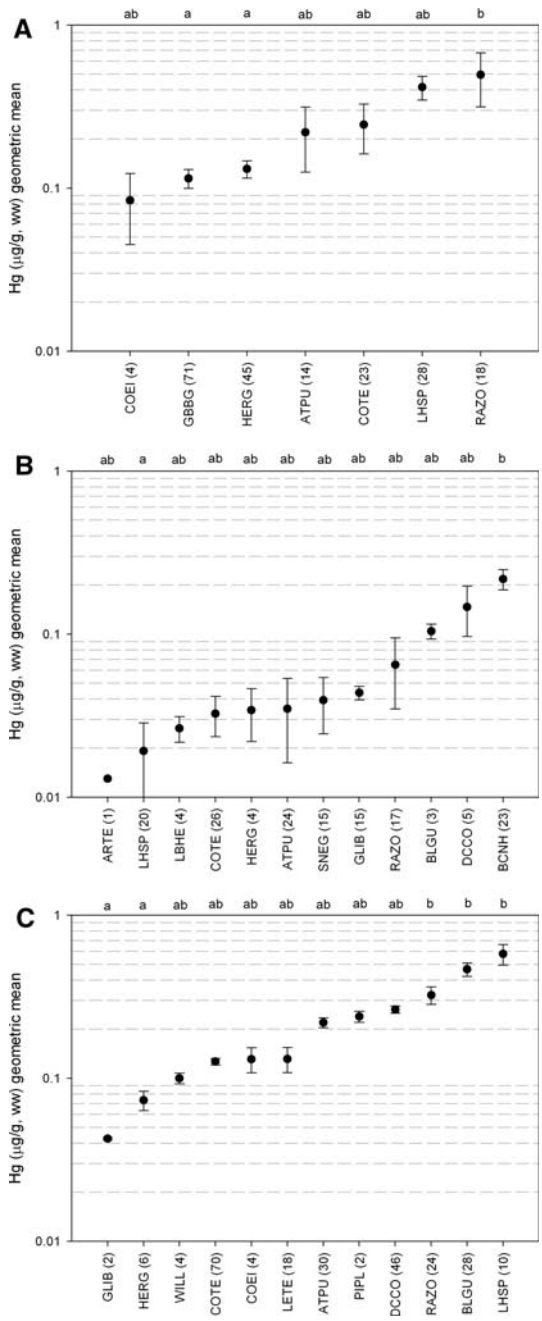

Figure 2. Geometric mean Hg levels (µg/g, ww) ± SE in adult blood **(A),** juvenile blood **(B),** and eggs **(C).** Sample sizes in parentheses. Means not sharing a common letter are significantly different ($P < 0.05$). ARTE, Arctic tern; ATPU, Atlantic puffin; BCNH, black-crowned night-heron; BLGU, black guillemot; COEI, common eider; COTE, common tern; DCCO, double-crested cormorant; GLIB, glossy ibis; GBBG, great black-backed gull; HERG, herring gull; LBHE, little blue heron; LETE, least tern; LHSP, Leach's storm-petrel; PIPL, piping plover; RAZO, razorbill; SNEG, snowy egret; and WILL, willet.

Table 1. Ratio of Hg in Adult and Juvenile Blood

Species	Geometric mean ratio (adult:juvenile)	Statistics (AVOVA)
Leach's storm-petrel	21.6:1	DF 1, 46; $F = 143.00$; $P < 0.0001$
Herring gull	3.8:1	DF 1, 47; $F = 23.96$; $P < 0.0001$
Common tern	7.5:1	DF 1, 47; $F = 81.68$; $P < 0.0001$
Razorbill	7.6:1	DF 1, 33; $F = 67.53$; $P < 0.0001$
Atlantic puffin	6.3:1	DF 1, 36; $F = 44.51$; $P < 0.0001$
Geometric mean of ratios	7.8:1	

Table 2. Within-clutch Total Hg (μg/g, ww) Variation of Double-crested Cormorant Eggs

Island	n	Geometric mean	Min	Max	SE
Bluff Island	4	0.12	0.11	0.13	0.003
Egg Rock	3	0.24	0.23	0.25	0.006
No Man's Land	3	0.30	0.25	0.33	0.024
Sugarloaf Island	4	0.24	0.23	0.26	0.008
Thrumcap Island	3	0.32	0.28	0.40	0.037

Table 3. Within-island Total Hg (μg/g, ww) Variation of Double-crested Cormorant Eggs[a]

Island	n	Geometric mean	Min	Max	SE
Duck Island	3	0.42	0.40	0.45	0.016
Egg Rock	3	0.30	0.26	0.34	0.024
No Man's Land	2	0.23	0.14	0.38	0.118
Stratton Island	3	0.35	0.31	0.40	0.025
Sugarloaf Island	3	0.36	0.32	0.42	0.032
Thrumcap Island	3	0.26	0.21	0.36	0.049

[a]Samples size is the number of five egg composites.

strategy (see Appendix B) (ANOVA, eggs: $F_{2,243} = 56.52$, $P < 0.0001$; adult blood: $F_{2,200} = 11.07$, $P < 0.0001$). Piscivorous birds had greater geometric mean Hg levels in eggs (geometric mean ± SE; 0.30 ± 0.02 μg/g, ww) than species that foraged on both invertebrates and fish (0.14 ± 0.02 μg/g, ww) or on invertebrates alone (0.11 ± 0.02 μg/g, ww; Tukey HSD, $P < 0.05$). Similarly, adult piscivorous birds had greater geometric mean Hg levels in blood (0.35 ± 0.08 μg/g, ww) than adult birds that foraged on both invertebrates and fish (0.16 ± 0.2 μg/g, ww) or on invertebrates alone (0.08 ± 0.03 μg/g, ww; Tukey HSD, $P < 0.05$). We did not observe any differences in juvenile blood Hg levels among foraging groups (ANOVA, $F_{2,153} = 0.18$, $P = 0.84$).

Discussion

Tissue Selection

Blood

Our results indicate that adult blood is more useful than chick blood for detecting long-term temporal trends, because Hg levels in adults are higher and are not confounded by metabolic effects. Blood Hg levels in chicks are lower when Hg is depurated into growing feathers (Spalding et al. 2000) and, consequently, may not represent full dietary exposure. Since the specific relationship between MeHg uptake and feather molt is relatively unknown and may

vary from species to species, interpreting Hg difference is challenging and is therefore confounded by chick age.

In all species with paired data, adult blood Hg levels were significantly higher than those of juveniles (Table 1); adult/juvenile blood Hg ratios by species are valuable in predicting adult Hg levels based on juvenile's or vice versa. This trend has also been observed in herring and Franklin's gulls (*L. pipixcan*; Burger and Gochfeld 1997), common mergansers (*Mergus merganser*), tree swallows (*Tachycineta bicolor*), belted kingfishers (*Megaceryle alcyon*), common loons (*Gavia immer*; Evers et al. 2005), double-crested cormorants, snowy egrets (*Egretta thula*), and black-crowned night-herons (Henny et al. 2002). This difference is attributed to chicks' depurating Hg into their growing feathers thereby reducing blood levels (Spalding et al. 2000; Evers et al. 2005), and to the chicks consuming smaller prey than adults—smaller fish will have bioaccumulated less Hg (Evers et al. 2005).

Leach's storm-petrel had the highest ratio with adults having 21.6 times higher Hg than chicks; this ratio is greater than that observed for other piscivorous birds (Burger and Gochfeld 1997; Burgess et al. 2005; Evers et al. 2005). Trophic position likely does not explain the difference: Hedd and Montevecchi (2006) found no trophic difference between Leach's storm-petrel adults and juveniles; and Antarctic petrel (*Thalassoica antarctica*; Hodum and Hobson 2000) and southern giant petrel (*Macronectes giganteus*; Forero et al. 2005) chicks occupied a higher trophic position than adults. Bond (2007) found no significant relationships (all $r^2 < 0.05$) between δ^{15} N and total mercury in seabird feathers, blood, and yolk, and only a weak relationship in albumen ($r^2 = 0.25$) from seabirds in the Gulf of Maine region. There are at least two possible explanations for this higher ratio in storm-petrels. First, storm-petrel chicks may have different pharmacokinetics of Hg as a result of their slower development (fledging $\pm$ 65 days) compared to other species. This may allow storm-petrel chicks to depurate a higher proportion of their ingested Hg burden to their growing feathers, since their feather growth occurs over a 60–70-day period (Huntington et al. 1996). Second, the observed difference in juvenile and adult blood Hg levels in storm-petrels may result from possible differences in prey Hg levels between the breeding and non-breeding foraging areas. Lower levels of Hg in prey fed to chicks may result in lower blood Hg levels if adults feed on different prey with higher Hg levels prior to the breeding season and bioaccumulate higher levels of Hg in their soft tissues than chicks.

Eggs

Our results indicate that egg Hg levels are as effective as adult blood for biomonitoring. Adult blood and egg Hg levels were within the same order of magnitude, and they both displayed species differences (Appendix A, Figure 2A, C). Although species with paired adult blood and egg data sets did not have identical Hg exposure order, piscivorous birds had significantly higher blood and egg Hg levels than birds feeding on both invertebrates and fish or on invertebrates alone. Therefore, eggs are as effective as blood in detecting difference in Hg exposure between trophic levels.

Eggs are a good tissue for long-term monitoring because they represent recent local dietary uptake (Hobson et al. 1997; Evers et al. 2003; Bond 2007). Egg nutrients are generally allocated from exogenous rather than endogenous sources (Hobson et al. 1997, 2000; Hobson 2006; Bond et al. 2007a) and most species represented in this study are documented income breeders, using exogenous nutrients for egg production (Hobson 2006; Bond 2007). In addition, eggs are relatively easy to collect, and clearly exhibit differences among species and colonies. However, power analysis and research design must take into account the varying levels of Hg within clutches as well as within colonies.

When interpreting Hg levels in eggs, it is important to consider within-clutch variation. In some studies, the first-laid egg exhibits the highest Hg levels, and the last-laid egg the lowest (Becker 1992; Evers et al. 2005). Cormorant egg results in this study show little within-clutch variation (Table 2), suggesting that one egg from a clutch can accurately characterize Hg levels of the laying female. We did find, however, that clutches with higher Hg levels also had higher Hg variation among eggs within the clutch. This indicates that analyzing each egg within a clutch will be necessary at sites where Hg levels are known to be high, such as near a known point source.

Another factor critical to understanding Hg levels is within-colony variation, which ultimately determines the number of samples needed to characterize a colony. We found little within-colony variation in Hg levels in cormorant eggs (Table 3), but, similar to the above findings, we found an increase in within-colony variation with an increase in Hg level. This indicates the importance of conducting a pilot study prior to large-scale sampling.

Several programs that monitor contaminant levels in seabird eggs use parallel data on stable carbon and nitrogen isotope ratios within the same eggs, as an aid in inter-

preting differences in contaminant concentrations between eggs from different nests within the same colony, between different colonies, or between different years at the same colony (Jarman et al. 1996; Hebert et al. 2000; Braune et al. 2002). Using stable isotope data, it is possible to assess if differences in contaminant levels are related to differences in trophic level or source of prey, rather than general changes in contaminant levels in the marine environment.

Species Comparisons

Mean tissue Hg levels in most waterbird species we measured were below suggested toxic thresholds of 0.6–1.3 μg/g (ww) in eggs (Barr 1986; Thompson 1996; Evers et al. 2003), 3.0 μg/g (ww) in adult blood (Evers et al. 2008), and levels reported for juvenile blood, which are age dependent (Kenow et al. 2007, 2008). Recently, findings on the relative sensitivity of various bird species to Hg exposure appear to vary significantly and are related to foraging guilds (Heinz et al. 2008). Therefore, while mean egg Hg levels for Leach's storm-petrel and black guillemot are generally under stated thresholds (Appendix B), evidence from Heinz et al. (2008) indicates reproductive impairment remains plausible and that toxic thresholds for these species need to be developed. Generally the adult and juvenile Hg blood levels were low and consistent with other studies (Kahle and Becker 1999; Thompson and Dowding 1999; Bearhop et al. 2000; Evers et al. 2005; Ikemoto et al. 2005). However, some adult puffins, common terns, storm-petrels, and razorbills had blood and egg Hg levels which exceeded the mean by more than three times (Appendix B). This suggests that certain individuals may have a specialized diet, which results in higher Hg levels in their blood and eggs.

Piscivores in this study had higher Hg levels than invertivores, and birds that forage on both invertebrates and fish. This relationship has been documented in other studies on marine birds (e.g., Burger 2002), waterfowl (Evers et al. 2005), wading birds (Sundlof et al. 1994), and fish (Peterson et al. 2002). This diet difference reflects trophic level differences (Hobson et al. 2002; Evers et al. 2005). Invertivores such as common eider (*Somateria mollissima*), glossy ibis, and willet (*Catoptrophorus semipalmatus*) had lower Hg levels than piscivores such as double-crested cormorant and razorbill. Piscivores also tended to have Hg levels higher than species that feed on both invertebrates and fish. For example, juvenile blood and egg Hg results show that common tern Hg levels are significantly lower than double-crested cormorant tissues. Trophic position and foraging strategies (see Appendix B) may also explain why Hg levels in adult blood of Leach's storm-petrels and razorbills, and eggs of Leach's storm-petrels, guillemots, and razorbills tended to be higher than those of other species, explained further below.

Leach's Storm-petrel

Leach's storm-petrels consistently have some of the highest Hg levels in multi-species studies (Elliott et al. 1992; Elliott and Scheuhammer 1997; Burgess 2006; Bond 2007). The Hg levels of Leach's storm-petrel may be attributed to their mesopelagic foraging strategy. They feed 100–200 km offshore beyond the continental shelf (Huntington et al. 1996) on crustaceans and mesopelagic fish (Watanuki 1985; Hedd and Montevecchi 2006). Monteiro et al. (1996) found that mesopelagic fish have higher Hg levels than surface feeding fish; they suggested that Hg is more readily available to deep-sea fish because the methylation of inorganic Hg mostly occurs in deep water with low oxygen. Myctophids (lanternfish) are important prey for storm-petrels in British Columbia, Canada (Vermeer and Devito 1988); Pearl Island, Nova Scotia, Canada; and Middle Island, Newfoundland, Canada (Linton 1979), accounting for 55% diet mass on Green Island and Gull Island, Newfoundland (Montevecchi et al. 1992), and 77% of the mass of identified fish on Baccalieu Island, Newfoundland (Hedd and Montevecchi 2006). These mesopelagic fish become available to storm-petrels when the fish rise to the surface to feed at night (Hedd and Montevecchi 2006). Myctophids have elevated Hg levels when compared to other fish of a similar size (Monteiro et al. 1996; Lahaye et al. 2006), and have varying Hg levels in different regions in the North Atlantic (Martin et al. 2006). A fourfold increase in Hg levels in band-rumped storm-petrels (*Oceanodroma castro*) over the last 100 years further demonstrates high Hg availability in the mesopelagic zone (Thompson et al. 1998). Therefore, the Hg levels of storm-petrels represent primarily pelagic and mesopelagic zones, which are areas likely removed from point and regional Hg sources, and indicates that storm-petrels may serve as bioindicators of global Hg temporal trends.

Black Guillemots

In contrast to the storm-petrels, guillemots serve as bioindicators of Hg in inshore benthic food webs. Guillemots in the Gulf of Maine feed their chicks primarily rock

gunnels (*Pholis gunnellus*; Butler and Buckley 2002): 68% of the diet of chicks on Kent Island, New Brunswick, Canada (Preston 1968), and 59% on Great Duck Island, Maine, U.S.A. (Hayes 1993). Rock gunnel life history indicates they may bioaccumulate Hg to elevated levels: the fish are long-lived (up to 14 years), live close to sediment in the intertidal zone, and feed on benthic polychaetes, amphipods, mollusks, and crustaceans, which accumulate contaminants (Bigelow and Schroeder 2002; Vallis et al. 2007).

The guillemots' benthic foraging may also explain why their eggs exhibited significant inter-island Hg variation, while common terns and cormorants did not. This difference may reflect prey mobility and foraging range. Common terns feed within 20 km of breeding sites (Nisbet 2002), primarily on white hake (*Urophycis tenuis*) and Atlantic herring (*Clupea harengus*) during the breeding season (Hall et al. 2000). Both of these species are schooling fish that move throughout Gulf of Maine (Scott and Scott 1988). Similarly, cormorants feed within 40 km of breeding sites (Custer and Bunck 1992) and almost exclusively on larger fish (often schooling, up to 40 cm long; Hatch and Weseloh 1999) that are generally highly mobile. In contrast, guillemots feed usually within 4 km of their nesting sites (Butler and Buckley 2002), and their primary prey, rock gunnels, tend to have low mobility during the spring and summer (Vallis et al. 2007); this suggests that Hg levels in guillemots reflect a limited area around their breeding colonies, while Hg levels in terns and cormorants reflect a much broader geographic range. These results are consistent with research on polychlorinated biphenyls (PCBs) in a contaminated site in Labrador, where the high level of PCBs in guillemots were attributed to benthic foraging, small foraging range, and limited dispersal (Kuzyk et al. 2005).

Guillemot egg Hg levels were also among the highest recorded in this study, which corroborates findings from previous studies on Petit Manan Island, Maine (Mierzykowski et al. 2005) and the Faroe Islands (Dam et al. 2004). In our study, mean guillemot egg Hg levels on Western Island (0.76 μg/g, ww) were nearly three times that of the highest levels in cormorant eggs (0.28 μg/g, ww) on nearby Thrumcap Island (6 km away). Similarly, on Eastern Egg Rock and Petit Manan, where tern and guillemot eggs were sampled, guillemot egg Hg levels were 1.8 to 3.8 times higher than terns'.

Other Alcids

Puffins and razorbills are pursuit divers, feeding mainly on local (5–20 km) schooling fish (white hake and Atlantic herring), and marine invertebrates, particularly *Meganyctiphanes norvegica* (Crustacea: Euphausiidae; Northern krill) in the case of puffins (Diamond and Devlin 2003; Bond et al. 2007b). Despite these similarities, razorbills have consistently higher Hg than puffins in feathers (Bond 2007), eggs, and blood (this study). In general, razorbills tend to dive deeper (Piatt and Nettleship 1985) and feed on larger, and therefore older, fish (Bond et al. 2007b), which bioaccumulate contaminants (Wiener and Spry 1996) and could increase the birds' Hg exposure.

Juveniles of Other Species

In our study, blood Hg levels in juvenile black-crowned night-herons, black guillemots, and double-crested cormorants tended to be higher than other species. The higher night-heron levels could be attributed to their feeding at higher trophic levels (we observed them feeding on tern chicks at the sampling site) or in an area with high Hg availability. Cormorant chicks likely also occupy a higher trophic position as they are commonly fed large fish (up to 40 cm; Hatch and Weseloh 1999) and black guillemot levels are likely high for the reasons described above.

Bioindicators

Seabirds are used as bioindicators of persistent bioaccumlamtive toxins (PBTs) around the world (Pearce et al. 1989; Elliott et al. 1992; Furness and Camphuysen 1997; Cifuentes et al. 2003; Braune 2007; Wolfe et al. 2007), and specifically for Hg (Thompson et al. 1990, 1992, 1998; Monteiro and Furness 1995). Since 1972, the Canadian Wildlife Service (CWS) has analyzed Atlantic puffin, double-crested cormorant, and Leach's storm-petrel eggs for Hg in Atlantic Canada (Pearce et al. 1979; Burgess 2006). Currently, there is no such long-term monitoring in the Gulf of Maine. We used the following criteria to select bioindicators: Are the birds abundant and widespread in the Gulf of Maine, do they represent specific foraging guilds, and/or do they have the potential for Hg levels above estimated effects thresholds?

In order to create an informative dataset to monitor long-term trends of Hg and other PBTs in the Gulf of Maine, we propose using an approach modeled after the CWS protocol. From each site, our model calls for the collection of eggs, at least every 4 years (higher frequency will increase the power to detect time trends (Hebert and Weseloh 2003)), from 15 separate nests of the common

eider, Leach's storm-petrel, double-crested cormorant, and black guillemot. The sample size of 15 would detect a 15% difference between sites at a 90% confidence interval (derived from a power analysis using the mean island standard deviation, 0.11, of the $\log_{10}$-transformed data; SAS Institute Inc. 2001). Since there is within-clutch Hg variation, consistently collecting eggs laid in the same sequence (i.e., the first laid egg) would reduce variation within each colony. These eggs should be collected from Isle of Shoals, Casco Bay, Penobscot Bay, and Bay of Fundy—not all sites have all species. If funding limitations prevent a thorough study, sampling could be limited to storm-petrel, cormorant, and guillemot.

Each of these species will serve as indicators of different food webs in the Gulf of Maine. Common eiders provide an inshore, site-specific Hg signal, because they feed primarily on mollusks, crustaceans, and echinoderms (Goudie et al. 2000). Leach's storm-petrels utilize offshore pelagic and mesopelagic food webs that may reflect global Hg levels. Double-crested cormorants are higher trophic-level piscivores that represent the pelagic food web for broad coastal areas because they feed on mobile schooling fish. The black guillemot represents benthic zone near breeding colonies. Guillemots are particularly important bioindicators because benthic feeding birds have higher levels of some contaminants than other species (Braune 1987; Kuzyk et al. 2005) and Hg can concentrate and is methylated in marine sediment (Gagnon and Fisher 1997).

Our results indicate that Hg levels in adult blood and eggs provide a comparable indicator of MeHg bioavailability and are within the same order of magnitude. While both tissues represent recent dietary uptake, sampling eggs is preferred because it is consistent with CWS protocol and researchers can collect suitable sample sizes from nesting colonies over time. The relative ease of collecting eggs is critical to the success of long-term monitoring.

By selecting a diverse suite of indicator species for monitoring Hg in the Gulf of Maine region, future studies will be able to measure changes in Hg in different food webs of the marine environment accurately and across multiple trophic-levels (Evers et al. 2009). This multi-trophic approach would also augment existing programs such as MusselWatch (Kimbrough et al. 2008), and GulfWatch (Chase et al. 2001), which tend to be focused on one component of the food web. Our study provides not only the necessary, broad baseline data to which future studies can be compared, but also the important process for selecting species and tissues, thereby allowing future studies to focus on key questions regarding changes in marine Hg in this important region.

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Appendix

(See Appendix Tables)

Appendix A. Species, Sites, and Sample Sizes for Hg Samples from the Gulf of Maine, 1998–2006

Common name	Scientific name/ Latin name	Site/Island	Nearest town	State/ Province[a]	Latitude	Longitude	Year	Blood		Egg	Total
								Adult	Juvenile		
Common eider	*Somateria mollissima*	Off Lanes Island, Casco Bay	Yarmouth	ME	43.80	−70.12	2000	2			2
		Old Hump, Musconguss Bay	St. George	ME	43.88	−69.36	2000			1	1
		Off Stage Island, Sagadahoc Bay	Georgetown	ME	43.76	−69.76	1998	2			2
		Stratton Island, Casco Bay	Old Orchard Beach	ME	43.51	−70.31	2005			3	3
Leach's storm-petrel	*Oceanodroma leucorhoa*	Machias Seal Island	Cutler	ME	44.50	−67.10	2005–2006	13	18	7	38
		Kent Island	Grand Manan	NB	44.58	−66.76	2004			3	3
		Little Duck Island	Frenchboro	ME	44.17	−68.24	2005	15	2		17
Double-crested cormorant	*Phalacrocorax auritus*	Bluff Island	Old Orchard Beach	ME	43.51	−70.32	2004–2005		3	4	7
		Duck Island, Isle of Shoals	Kittery	ME	42.98	−70.63	2005			3	3
		Egg Rock, Pigeon Hill Bay	Milbridge	ME	44.41	−67.87	2005			6	6
		No Man's Land	Matinicus Isle	ME	43.88	−68.87	2005			5	5
		Stratton Island	Old Orchard Beach	ME	43.51	−70.31	2005			3	3
		Sugarloaf Island	Phippsburg	ME	43.75	−69.77	2004–2005		2	7	9
		Thrumcap Island	Brooksville	ME	44.32	−68.76	2004–2005			15	15
		Manawagonish Island	Saint John	NB	45.21	−66.11	2004			3	3
Snowy egret	*Egretta thula*	Stratton Island	Old Orchard Beach	ME	43.51	−70.31	2001, 2003, 2004		15		15
Little blue heron	*Egretta caerulea*	Stratton Island	Old Orchard Beach	ME	43.51	−70.31	2003–2004		4		4
Black−crowned night−heron	*Nycticorax nycticorax*	Stratton Island	Old Orchard Beach	ME	43.51	−70.31	2001–2004		23		23
Glossy ibis	*Plegadis falcinellus*	Stratton Island	Old Orchard Beach	ME	43.51	−70.31	2001–2004		15	2	17
Piping plover	*Charadrius melodus*	Crescent Surf	Kennebunk	ME	43.34	−70.53	2002			1	1
		Laudholm Beach	Wells	ME	43.33	−70.54	2003			1	1

Appendix A. continued

Common name	Scientific name/ Latin name	Site/Island	Nearest town	State/ Province[a]	Latitude	Longitude	Year	Blood		Egg	Total
								Adult	Juvenile		
Willet	*Catoptrophorus semipalmatus*	Drakes Island Road	Wells	ME	43.33	−70.55	2004			4	4
Herring gull	*Larus argentatus*	Flat Island	Islesboro	ME	44.32	−68.93	2004		4		4
		Hospital Island	St. Andrews	NB	45.12	−67.01	2001–2002	4			4
		New River Island	Pocologan	NB	45.12	−66.54	2002	4			4
		Salkeld Islands	Lepreau	NB	45.11	−66.51	2001–2002	6			6
		Manawagonish Island	Saint John	NB	45.21	−66.11	2002, 2004	3		3	6
		Flatpot Island	Black's Harbor	NB	44.95	−66.72	2002	7			7
		Hospital Islands	Deer Island	NB	44.99	−66.92	2003	21			21
		Kent Island	Grand Manan	NB	44.58	−66.76	2004			3	3
Great black-backed gull	*Larus marinus*	Hospital Island	St. Andrews	NB	45.12	−67.01	2001–2002	11			11
		New River Island	Pocologan	NB	45.12	−66.54	2001–2002	2			2
		Hog Island	St. Andrews	NB	45.14	−66.96	2001–2002	11			11
		Salkeld Islands	Lepreau	NB	45.11	−66.51	2001–2002	6			6
		Manawagonish Island	Saint John	NB	45.21	−66.11	2001–2002	8			8
		Flatpot Island	Black's Harbor	NB	44.95	−66.72	2001–2002	7			7
		Sandy Island	Deer Island	NB	44.97	−66.91	2001	2			2
		Dick's Island	St. Andrews	NB	45.14	−67.00	2002	2			2
		Hospital Islands	Deer Island	NB	44.99	−66.92	2002–2003	22			22
Least tern	*Sternula antillarum*	Crescent Surf	Kennebunk	ME	43.34	−70.53	2002–2003			17	17
		Laudholm Beach	Wells	ME	43.33	−70.54	2003			1	1
Common tern	*Sterna hirundo*	Eastern Egg Rock	St. George	ME	43.86	−69.38	2004–2005			10	10
		Jenny Island	Harpswell	ME	43.77	−69.91	2004–2005			10	10
		Machias Seal Island	Cutler	ME	44.50	−67.10	2005–2006	23	2	5	30
		Petit Manan Island	Milbridge	ME	44.37	−67.87	2001, 2003–2005			21	21
		Pond Island	Phippsburg	ME	43.74	−69.77	2004–2005			10	10
		Seavey Island, Isle of Shoals	Kittery	NH	42.98	−70.62	1998		13		13
		Stratton Island	Old Orchard Beach	ME	43.51	−70.31	2000, 2004–2005		11	14	25

Appendix A. continued

Common name	Scientific name/ Latin name	Site/Island	Nearest town	State/ Province[a]	Latitude	Longitude	Year	Blood		Egg	Total
								Adult	Juvenile		
Arctic tern	*Sterna paradisaea*	Stratton Island	Old Orchard Beach	ME	43.51	−70.31	2000		1		1
Razorbill	*Alca torda*	Machias Seal Island	Cutler	ME	44.50	−67.10	2005–2006	18	17	17	52
		Matinicus Rock	Matinicus Isle	ME	43.79	−68.85	2005			7	7
Black guillemot	*Cepphus grylle*	Eastern Egg Rock	St. George	ME	43.86	−69.38	2006			4	4
		Petit Manan Island	Milbridge	ME	44.37	−67.87	2006			8	8
		Western Island	Deer Isle	ME	44.29	−68.82	2005–2006			9	9
		Seal Island	Matinicus Isle	ME	43.89	−68.74	2006			1	1
		Metinic Island	St. George	ME	43.90	−69.12	2006			6	6
		Little Duck Island	Frenchboro	ME	44.17	−68.24	2005		3		3
Atlantic puffin	*Fratercula arctica*	Machias Seal island	Cutler	ME	44.50	−67.10	2004–2006	14	18	23	55
		Matinicus Rock	Matinicus Isle	ME	43.79	−68.85	2000, 2005			4	4
		Petit Manan Island	Milbridge	ME	44.37	−67.87	2005–2006		6	3	9
Total								203	157	244	604

[a]ME, Maine, U.S.A.; NH, New Hampshire, U.S.A.; NB, New Brunswick, Canada.

Appendix B. Tissue Hg Levels (Arithmetic Mean ± SD, Range, and Sample Size) from Sampling of Seabird Tissues in Gulf of Maine 1998–2006[a]

Species			Blood (μg/g, ww) Mean ± SD (range) ***n***		Egg (μg/g, ww) Mean ± SD (range) ***n*** (t/c)[a]
Common name	Primary foraging category	Foraging habitat/diet	Adult[b]	Juvenile[c]	
Common eider	Invertivore	Nearshore benthic/invertebrates, intertidal mollusks (Goudie et al. 2000)	0.11 ± 0.08 (0.03–0.20) **4**		0.14 ± 0.05 (0.10–0.20) **4/3**
Leach's storm-petrel	Invertivore and piscivore	Mesopelagic, pelagic/plankton and small nekton (Huntington et al. 1996)	0.54 ± 0.37 (0.03 –1.99) **28**	0.03 ± 0.04 (0.01–0.20) **20**	0.62 ± 0.26 (0.29–1.25) **10/3**
Double-crested cormorant	Piscivore	Mid-water, benthic/fish (Hatch and Weseloh 1999)		0.18 ± 0.12 (0.06–0.37) **5**	0.28 ± 0.09 (0.11–0.45) **46/20**
Snowy egret	Invertivore and piscivore	Salt-marsh, intertidal zone/invertebrates, fish (Parsons and Master 2000)		0.07 ± 0.06 (0.02–0.20) **15**	
Little blue heron	Invertivore and piscivore	Estuary/invertebrates, fish (Rodgers and Smith 1995)		0.03 ± 0.01(0.02–0.04) **4**	
Black-crowned night-heron	Invertivore and piscivore	Water edge, marsh/invertebrates, fish, birds, small mammals, garbage (Davis 1993)		0.25 ± 0.15 (0.11–0.72) **23**	
Glossy ibis	Invertivore	Shallow water/invertebrates (Davis and Kricher 2000)		0.04 ± 0.02 (0.00–0.07) **15**	0.04 ± 0.00 (0.04–0.04) **2/0**
Piping plover	Invertivore	Shoreline/invertebrates (Haig 2004)			0.24 ± 0.03 (0.22–0.26) **2/0**
Willet	Invertivore	Mud flats, salt-marsh edge/invertebrates small fish (Lowther et al. 2001)			0.10 ± 0.02 (0.09–0.12) **4/0**
Herring gull	Invertivore and piscivore	Ocean surface, intertidal/berries, invertebrates, lobster bait, fish, small mammals, garbage (Goodale 2000)	0.16 ± 0.11 (0.03–0.53) **45**	0.03 ± 0.02 (0.01–0.06) **4**	0.08 ± 0.02 (0.05–0.10) **6/6**
Great black-backed gull	Invertivore and piscivore	Ocean surface, mudflats, intertidal zone/invertebrates, fish, birds, small mammals, garbage (Good 1998)	0.16 ± 0.13 (0.02–0.73) **71**		
Least tern	Invertivore and piscivore	Shallow water, estuaries, bays/invertebrates, small fish (Thompson et al. 1997)			0.15 ± 0.10 (0.08–0.49) **18/0**
Common tern	Invertivore and piscivore	Open water/invertebrates, small fish (Nisbet 2002)	0.36 ± 0.40 (0.04–1.81) **23**	0.04 ± 0.05 (0.01–0.25) **26**	0.13 ± 0.05 (0.07–0.25) **70/0**
Arctic tern	Invertivore and piscivore	Open water/invertebrates, small fish (Hatch 2002)		0.01 (0.01–0.01) **1**	
Razorbill	Piscivore	Shallow water, nearshore/crustaceans, fish (Hipfner and Chapdelaine 2002)	0.60 ± 0.45 (0.20–2.04) **18**	0.10 ± 0.12 (0.01–0.55) **17**	0.38 ± 0.19 (0.10–0.82) **24/0**

Appendix B. continued

Species			Blood (μg/g, ww) Mean ± SD (range) ***n***		Egg (μg/g, ww) Mean ± SD (range) ***n*** (**t/c**)
Common name	Primary foraging category	Foraging habitat/diet	Adult[b]	Juvenile[c]	
Black guillemot	Piscivore	Shallow inshore waters, benthic/invertebrates, fish (Butler and Buckley 2002)		0.11± 0.02 (0.09–0.13) **3**	0.52 ± 0.23 (0.16–1.01) **28/0**
Atlantic puffin	Piscivore	Shallow water near breeding colonies/fish (Lowther et al. 2002)	0.29 ± 0.35 (0.12–1.51) **14**	0.06 ± 0.09 (0.01–0.43) **24**	0.23 ± 0.08 (0.08–0.45) **30/3**

[a]Sample size for eggs includes the total number of samples and the number that were composites (t/c).
[b]Individuals at least 1 year old.
[c]Young of the year.

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ECOHEALTH

Special Feature: Methylmercury

Original Contribution

Integrated Mercury Monitoring Program for Temperate Estuarine and Marine Ecosystems on the North American Atlantic Coast

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Abstract: During the past century, anthropogenic activities have altered the distribution of mercury (Hg) on the earth's surface. The impacts of such alterations to the natural cycle of Hg can be minimized through coordinated management, policy decisions, and legislative regulations. An ability to quantitatively measure environmental Hg loadings and spatiotemporal trends of their fate in the environment is critical for science-based decision making. Here, we outline a Hg monitoring program for temperate estuarine and marine ecosystems on the Atlantic Coast of North America. This framework follows a similar, previously developed plan for freshwater and terrestrial ecosystems in the U.S. Methylmercury (MeHg) is the toxicologically relevant form of Hg, and its ability to bioaccumulate in organisms and biomagnify in food webs depends on numerous biological and physicochemical factors that affect its production, transport, and fate. Therefore, multiple indicators are needed to fully characterize potential changes of Hg loadings in the environment and MeHg bioaccumulation through the different marine food webs. In addition to a description of how to monitor environmental Hg loads for air, sediment, and water, we outline a species-specific matrix of biotic indicators that include shellfish and other invertebrates, fish, birds and mammals. Such a Hg monitoring template is applicable to coastal areas across the Northern Hemisphere and is transferable to arctic and tropical marine ecosystems. We believe that a comprehensive approach provides an ability to best detect spatiotemporal Hg trends for both human and ecological health, and concurrently identify food webs and species at greatest risk to MeHg toxicity.

Keywords: mercury, marine ecosystems, estuaries, monitoring, birds, Atlantic Ocean

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Introduction

Anthropogenic releases of mercury (Hg) to the environment have impacted the biosphere substantially, and the ocean has received a major environmental insult as a result of increased Hg releases (Mason and Sheu 2002; Sunderland and Mason 2007). Overall, it appears that Hg in the open ocean has increased substantially during the past 200 years, primarily as a result of greater atmospheric Hg deposition (Mason et al. 1994; Selin et al. 2008). Additional local- and regional-scale contamination of the coastal zone has resulted from human activities and associated runoff from the terrestrial environment (USEPA 1997; Balcom et al. 2004). Therefore, while the impact of anthropogenic emissions in North America on the open ocean may be difficult to quantify (Sunderland and Mason 2007), there is growing evidence demonstrating increased levels of Hg in U.S. estuarine and coastal waters and sediments (Mason and Lawrence 1999; Varekamp et al. 2003; Conaway et al. 2007; Fitzgerald et al. 2007). Elevated inputs of Hg are a health concern because inorganic Hg can be converted to methylmercury (MeHg) (Clarkson and Magos 2006), a neurotoxin that bioaccumulates and biomagnifies through aquatic food webs, and in some cases adversely impacting high trophic-level aquatic organisms (Wolfe et al. 1998; Scheuhammer et al. 2007; Wolfe et al. 2007). Humans are exposed to MeHg principally by the consumption of contaminated fish (Fitzgerald and Clarkson 1991; Mahaffey 1998), and elevated MeHg levels in fish have resulted in fish-consumption advisories at state, provincial, and federal levels (Rice et al. 2003; Mahaffey et al. 2004; Environment Canada 2008) because of their proven negative effects to humans (Anderson 2008). Aquatic sediments and low-oxygen waters are the main sites for the conversion of inorganic Hg to MeHg (methylation), which is mediated primarily by sulfate-reducing bacteria (Compeau and Bartha 1985; Gilmour et al. 1992). Of the many potential stressors and contaminants in the coastal environment, the issue of MeHg contamination is one of the most pervasive concerns, as evidenced by the extensive fish-consumption advisories that exist for much of the North American coastline.

The coastal zone plays a crucial role in Hg and MeHg cycles, acting as a site of inorganic Hg entrapment, MeHg formation, and high biological productivity. Sunderland and Mason (2007) estimate that about 8.2 Mmol of Hg yr^{-1} (range, 5.2–11.4 Mmol yr^{-1}) is trapped in the estuarine and coastal zone, which is comparable to the estimated point source Hg inputs to the atmosphere of 11.3–16.9 Mmol yr^{-1}. While the coastal zone is a sink for inorganic Hg, mass balance considerations suggest that estuaries and near-shore marine systems are a net source of MeHg to the coastal ocean (Cossa et al. 1996; Hammerschmidt and Fitzgerald 2004, 2006b).

While a number of field studies have focused on Hg speciation in estuarine and coastal ecosystems (e.g., Baeyens et al. 1998; Benoit et al. 1998; Kannan et al. 1998; Bloom et al. 1999; Hammerschmidt et al. 2004; Heyes et al. 2004; Sunderland et al. 2004; Hollweg et al. 2009), there has been little coordinated monitoring of Hg and MeHg changes in the coastal zone, the effect of these changes on fish MeHg levels, and on humans and wildlife. This is surprising, both because the majority of fish consumed by humans are from coastal and open-ocean marine environments (FAO 2004; Sunderland 2007), and because there are indications that Hg trends have increased over the past decade in wildlife (Rigét et al. 2007).

Structure of Monitoring Program

There is a substantial effort underway to develop a Hg monitoring program across the United States (Mason et al. 2005; Harris et al. 2007), which, while not specifically excluding the coastal zone, has a strong focus on terrestrial and freshwater environs. To augment this effort, the design of a marine monitoring program that quantifies inputs of Hg to coastal and estuarine systems, and associated exposure and impact of MeHg on organisms, is needed. In 2006, the workshop *Fate and Bioavailability of Mercury in Aquatic Ecosystems and Effects on Human Exposure* was convened by the Dartmouth Toxic Metals Research Program, where marine Hg scientists and human health experts were gathered to articulate research and monitoring needs (Chen et al. 2008). From that workshop, we determined that a coordinated and expanded monitoring program is needed to evaluate: (1) spatial and temporal patterns of Hg deposition and transport, (2) MeHg formation and bioaccumulation, (3) wildlife populations at risk from MeHg exposure, and (4) human exposure. These priorities are also embraced by the proposed National Mercury Monitoring Program (Harris et al. 2007). That program identified several monitoring design elements including a national distribution of 10–20 monitoring stations that would include intensive sites bounded by multiple cluster or extensive sites. Intensive sites

would establish cause and effect relationships between Hg deposition and environmental change based on a comprehensive range of measurements, while cluster sites would use fewer measurements to better characterize environmental responses in varying ecosystem types. We suggest following this monitoring design for estuarine and marine ecosystems.

Our region of interest includes the estuarine and coastal ecosystems bounded by Chesapeake Bay north to the Gulf of Maine (Fig. 1). We organized our Hg monitoring program into four major habitat types: estuarine, coastal, semi-pelagic, and pelagic. We define these habitat types using the following criteria: (1) estuarine areas are semi-closed coastal waters that have free access to the ocean and variable, but generally shallow, water depths; (2) coastal areas extend out to a depth of 100 m (and are often within 3 nautical miles [5.6 km] from shore); (3) semi-pelagic areas represent the middle to outer continental shelf and range in water depth between 100 and 200 m (and are often located from 3 to 200 nautical miles [5.6–370.0 km] from shore but occasionally extend further); and (4) pelagic areas are open ocean waters that are >200 m in depth (and are usually >200 nautical miles [>370 km] from shore). Within these habitat types, we recognize different and relatively independent marine food webs exist that should be monitored separately for their ability to transfer MeHg to upper trophic levels. For example, benthic and demersal food webs at the bottom of the ocean need to be monitored separately from pelagic food webs higher in the water column.

We have identified five categories of indicators: abiotic measurements, invertebrates, fish, birds, and mammals. Within the biotic categories, we based taxonomic selection on multiple criteria (Table 1). A description of the target taxa within each of the four major habitat types is followed by sampling strategies that identify best tissues for use and how selenium (Se) interacts with MeHg. The best indicators for evaluating toxicity and spatiotemporal trends of MeHg for protection of human health can differ from those for ecological health. In order to encompass both for fish,

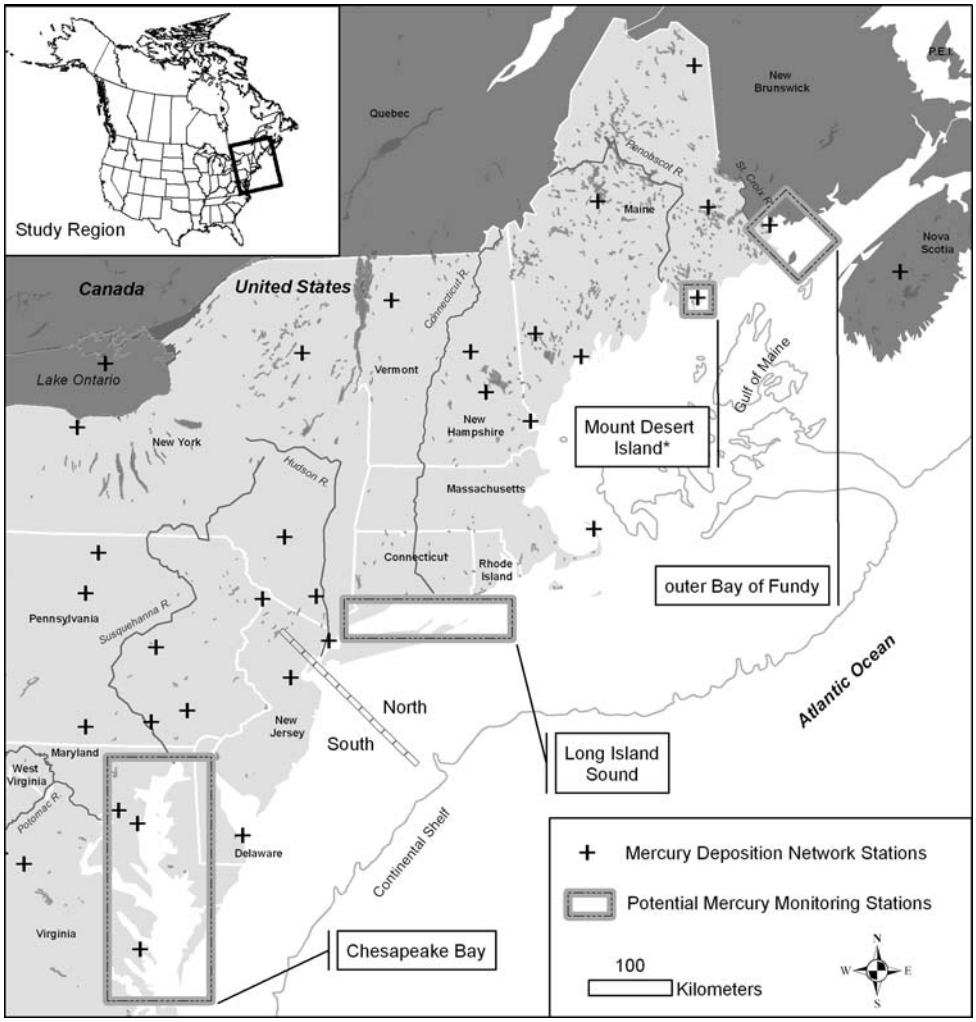

Figure 1. Area for consideration of a standardized Hg monitoring network, including sites of potential Hg monitoring stations and their association with existing Hg deposition network stations. Using the National Atmospheric Deposition Program's (NADP) wet deposition of chloride pattern as a proxy for marine influence on precipitation chemistry, a sharp change in influence is clearly present [David Gay, personal communication]. Marine air greatly influences precipitation chemistry within the first 400–500 km along the Gulf Coast, and less than 250 km along the mid-Atlantic and Northeast coasts of the United States and Canada. See the NADP chloride deposition measurements (http://nadp.sws.uiuc.edu/isopleths/maps2006/cldep.gif).

Table 1. Criteria Used for Each Major Biotic Category of Indicators.

Criteria type	Invertebrates	Fish	Birds	Mammals
Ubiquitous and abundant in study area	×	×	×	×
Ease of sampling	×	×	×	×
Comparability with existing Hg data	×	×	×	×
Understanding of MeHg transfer in food web	×	×	×	×
A distinct point of time is in a major habitat type	×	×	×	×
Used for human consumption	×	×		
Part of existing Hg monitoring programs	×	×	×	
Reflect highest MeHg exposure		×	×	×
Existing information on effects			×	×

we created low and high trophic-level categories that reflect Hg levels for ecological and human health concerns, respectively.

There are several existing, long-term marine Hg monitoring programs. They include the National Atmospheric Deposition Program's Mercury Deposition Network for air (Lamb and Bowersox 2000), the National Oceanographic and Atmospheric Administration's (NOAA) Mussel Watch Program for shellfish (Chase et al. 2001), the U.S. Environmental Protection Agency's (USEPA) National Coastal Assessment Program for shellfish and marine fish (Cunningham et al. 2003), the U.S. Geological Survey's Contaminant Exposure and Effects—Terrestrial Vertebrates program for terrestrial wildlife (Cohen et al. 2003), and the Atlantic, Arctic and Pacific Seabird Egg Contaminant Monitoring Programs of Environment Canada (Pearce et al. 1979; Braune 2007). We use these existing programs as a basis for identifying indicators and recommending Hg monitoring locations. Broad justifications and specific methodologies not detailed in our plan appear in Harris et al. (2007). Associated abiotic measurements and scientific names for the program's indicator organisms are provided in Tables 2 and 3.

Table 2. Suggested Abiotic Compartments for Monitoring Hg from Chesapeake Bay North to the Gulf of Maine

Matrix measured for Hg	Associated measurements
Air (total in wet and dry deposition)	Precipitation
Water (total and methyl)	Dissolved organic carbon
	Particulate organic carbon
	Particulate matter
Sediment (total and methyl)	Organic carbon

Indicator Compartments

Abiotic Indicators

Major sources of inorganic Hg to the coastal zone include direct atmospheric deposition, rivers (including runoff of atmospherically deposited Hg), and water pollution control facilities, with most of the MeHg derived from in situ transformation of inorganic Hg (Mason et al. 1999; Balcom et al. 2004, 2008). Given these sources, it is important to measure both direct atmospheric inputs and to quantify watershed contributions of inorganic Hg to the coastal zone. Collection of sediment cores and determination of total Hg and MeHg in both surficial sediment and water at intensive sites is recommended (Table 2).

At intensive sites, event-based wet deposition collection (Driscoll et al. 2007) and Hg atmospheric speciation and deposition should be measured. While the atmospheric concentration of Hg^0 is reflective of the global atmospheric Hg pool and is not a sensitive local indicator of short-term regional change (Slemr et al. 2003), levels of ionic gaseous and particulate Hg show a higher regional variability that may reflect irregular regional emissions (Schroeder and Munthe 1998; Ryaboshapko et al. 2007a, b). Ionic gaseous and particulate Hg species are, however, relatively difficult to measure (Landis et al. 2002), especially in the coastal zone (Laurier and Mason 2007), and, although there is large uncertainty in the current estimates of Hg dry deposition, new approaches are being developed (Skov et al. 2006). Atmospheric Hg speciation measurements at intensive sites should be coupled with flux estimates from other sources into the coastal zone. Specifically, measurements of groundwater and surface river water fluxes and Hg speciation should provide an ability to examine the role of Hg and

Table 3. Suggested Indicator Taxa for Monitoring Hg from Chesapeake Bay North to the Gulf of Maine[a]

Taxa[b]	Estuary	Coastal	Semi-pelagic	Pelagic
Invertebrates				
Benthic infauna	Amphipods	Polychaetes	–	–
Benthic epifauna and Epipelagic/ Mesopelagic fauna	Periwinkle[A] (*Littorina littorea*) Green crab[A] (*Carcinus maenas*)	Blue mussel[A] (*Mytilus edulis*) Eastern oyster[S] (*Crassostrea virginica*)	Atlantic lobster[N,c] (*Homarus americanus*)	Squid[A] (*Loligo* spp.) Euphausiids[A]
Fish				
Low trophic level	Mummichog[A] (*Fundulus heteroclitus*) American Sand lance[A] (*Ammodytes americanus*)	Striped anchovy[A] (*Anchoa hepsetus*) Butterfish[A] (*Peprilus triacanthus*)	Atlantic herring[A] (*Clupea harengus*)	North Atlantic saury[A] (*Scomberesox saurus saurus*) Lantern fish[N] (Myctophid spp.)
High trophic level	Striped bass[A] (*Morone saxatilis*) Scup[A,c] (*Stenotomus chrysops*)	Bluefish[A] (*Pomatomus saltatrix*) Winter Flounder[A,c] (*Pseudopleuronectes americanus*)	Atlantic cod[N,c] (*Gadus morhua*) Yellowfin tuna[A] (*Thunnus albacaras*)	Swordfish[A] (*Xiphias gladius*) Blue shark[A] (*Prionace glauca*)
Birds				
Invertivores	Nelson's[N] and Saltmarsh[A] sharp-tailed and Seaside[S] sparrows (*Ammodramus nelsoni, A. caudacutus, A. maritimus*)	Common eider[N] (*Somateria mollissima*)	–	–
Piscivores	Black-crowned night heron[A] (*Nycticorax nycticorax*) Belted kingfisher[A] (*Megaceryle alycon*)	Black guillemot[N] (*Cepphus grylle*) Osprey[A] (*Pandion haliaetus*)	Double-crested cormorant[A] (*Phalacrocorax auritus*) Common tern[A] (*Sterna hirundo*)	Leach's storm-petrel[N] (*Oceanodrama leucorhoa*)
Mammals	American mink[A] (*Mustela vison*)	Harbor seal[N] (*Phoca vitulina*) North American river otter[A] (*Lontra canadensis*)	Harbor porpoise[A] (*Phocoena phocoena*) Bottlenosed dolphin[S] (*Tursiops truncatus*)	Pilot whale spp.[A] (*Globicephala* spp.) Beaked whale spp.[A] *Mesoplodon* and *Ziphius* spp.)

[a]Areas where species are principally found are identified as: S, South (Chesapeake Bay to Hudson River mouth); N, North (Long Island Sound to Gulf of Maine; and A, All (Chesapeake Bay to Gulf of Maine).
[b]Species-specific taxa are organized by major habitat type to indicate primary area of residence. Most species are often found in multiple habitat-types, which provides a horizontal trophic relay of Hg, or bioadvection, across habitat types because of species movement patterns and subsequent predator–prey interactions.
[c]Part of the USEPA's National Coastal Assessment Program for the Northeast Region (Acadian and Virginian biogeographic regions).

MeHg transport from associated watersheds to the coastal zone.

Precipitation and other climatic variables also influence export to coastal ecosystems, especially with respect to sporadic and extreme events. Measurements of Hg in wet deposition are accomplished relatively easily and are monitored currently at the national level by the Mercury Deposition Network (MDN), which includes 11 coastal sites within our study area (MDN 2008; Fig. 1).

Changes in atmospheric Hg deposition also are recorded by sediments, and historical records of input have been inferred from the analysis of estuarine and wetland sediment cores (Varekamp et al. 2003; Conaway et al. 2007). There is a large body of experimental and observational evidence for their reliability, and well-established protocols for the collection, processing, and interpretation of these records (Porcella 1996). However, there are confounding effects of watershed input, sediment mixing (physical and bioturbation), and other factors that impact the level of temporal resolution.

While the relationship between biota and sediment Hg and MeHg levels is difficult to construct (Mason 2000), measurements of sediment MeHg provide an integrative measure of the impact of changes in Hg input and other factors on net production of MeHg (Benoit et al. 2003). MeHg in sediment and interstitial waters is available for uptake by benthic organisms and may be an important source to overlying water, either by upward diffusion or bioadvection. It has been shown, for numerous freshwater (Benoit et al. 2003) and some marine (Heyes et al. 2006; Kim et al. 2006) ecosystems, that there is a relationship between short-term Hg methylation rate measured using assays and in situ MeHg concentration or %MeHg in sediments; however, these correlations do not necessarily extend to all coastal marine deposits (Hammerschmidt and Fitzgerald 2004, 2006b; Ogrinc et al. 2007; Hammerschmidt et al. 2008). Assays of Hg methylation are not recommended for the extensive cluster sites. Nonetheless, sediment MeHg concentration and %MeHg may be useful proxies of the rate of change in bulk MeHg concentration and relative production. Measurements of %MeHg in sediment will allow assessment of whether changes of sedimentary MeHg content is related directly or indirectly to changes in atmospheric Hg input, as well as provide an abiotic indicator of potential biological exposure.

Total Hg and MeHg measurements in water, both in bulk and in the dissolved and particulate fractions, have been made in many coastal ecosystems (Kannan et al. 1998; Mason et al. 1999; Whalin et al. 2007; Balcom et al. 2008). Concentrations in water can be influenced by factors unrelated to Hg inputs, such as the variation in particulate matter and dissolved (DOC) and particulate organic carbon (POC), which are needed to improve interpretation (Table 2). Despite spatial variation in these factors, studies have shown a reasonable correlation between MeHg in water and MeHg in freshwater fish, reflecting the influences of bioaccumulation at the base of the pelagic food chain (Brumbaugh 2001). As levels of Hg species in water vary seasonally and with depth within a particular water body, these measurements must be assessed with consideration of anticipated spatial variability. Generally, the relationship between Hg and MeHg concentrations in water and estuarine and marine organisms has not been well documented and is poorly described in the literature.

Marine Invertebrate Indicators

Selection of indicator species at the base of pelagic and benthic food webs in marine systems should include invertebrate species representing different functional feeding groups, such as benthic infauna, benthic epifauna, and epi- and meso-pelagic biota (Tables 1, 3). Inclusion of both benthic and pelagic food webs facilitates an understanding of differing pathways of MeHg transfer into higher trophic levels. Studies in freshwater ecosystems suggest that pelagic feeding organisms have the ability to bioaccumulate greater concentrations of MeHg than benthic feeding fauna (Gorski et al. 2002; Power et al. 2002). However, little is known for marine systems; specifically, some areas may differ from freshwater systems in the manner MeHg is transferred to higher trophic positions.

Benthic infauna that live in the sediments of estuarine and coastal areas are useful taxa for monitoring MeHg bioaccumulation directly from ingestion or absorption from sediment. Benthic amphipods are inhabitants of many sediments and are used frequently in estuarine toxicity testing (e.g., Leptocheirus, Corophium), and which readily accumulate MeHg (Lawrence and Mason 2001). They obtain their food, and likely their MeHg, from ingesting detrital material and sediments. The most abundant sediment infauna are polychaetes.

In estuarine and coastal waters, several nonnative, epifaunal species are common. The periwinkle is a primary consumer that grazes on biofilms and periphyton on sediment and rock surfaces. The green crab also is nonnative to the U.S., but now inhabits coastal waters across the study

area. It is a secondary consumer that feeds on small fish, benthic invertebrates, and detrital material. MeHg concentrations in these two species range greatly across sites and appear to be related modestly with sediment Hg levels [Chen et al., unpublished data]. Lawrence and Mason (2001) showed that the bioaccumulation factor decreased with increasing sediment organic content. Importantly, the fraction of total Hg as MeHg can vary widely in invertebrates (Tremblay et al. 1996a b; Gorski et al. 2002; Mason and Benoit 2003).

Benthic invertebrate indicators for human health risk that inhabit estuary, coastal, and semipelagic areas include a wide variety of shellfish such as crabs, mussels, clams, oysters, and lobsters. Being important commercially harvested species, the monitoring of these taxa for certain contaminants is conducted by governmental regulatory agencies. For example, the NOAA Mussel Watch and Gulfwatch Programs have historically collected contaminant data for blue mussels, eastern oyster, and other bivalves across broad spatial and temporal scales (Chase et al. 2001), although they do not measure MeHg. Several studies have documented elevated muscle Hg concentrations in Atlantic lobster (Greig et al. 1975; Vassiliev et al. 2005; Hammerschmidt and Fitzgerald 2006a), including monitoring efforts by the USEPA (Cunningham et al. 2003). Differences in total Hg concentrations vary less than other metals for synoptically collected, co-located species (Chase et al. 2001), yet there is evidence to suggest that concentrations of MeHg vary in bivalves according to feeding strategy.

Lastly, cephalopods, such as squid are important ecologically and for human health. In the northeastern Atlantic Ocean, benthic cephalopods had significantly higher Hg levels than pelagic species (Bustamante et al. 2006). In the Mediterranean Sea, cephalopods Hg levels can be substantially higher than small fishes of similar trophic position, and appear to account for the highly elevated Hg liver body burdens in marine mammals (Frodello et al. 2000). Euphausiids are also typically relevant epi- and meso-pelagic organisms for monitoring MeHg availability (Braune 1987b; Monteiro et al. 1996).

Fish Indicators

There are numerous fish species that, when routinely sampled for Hg, are useful indicators of human and ecosystem health (Tables 1, 3). For assessing and monitoring trends in ecological health risk, we also emphasize species that are target prey for piscivorous fish and wildlife, and species that maintain sustainable and robust populations. Because there is a growing body of evidence of Hg-related effects to freshwater (Drevnick et al. 2008; LaRose et al. 2008) and estuarine fish species (del Carmen Alvarez et al. 2006), the sustainability of healthy populations of high trophic-level fish species with elevated Hg levels needs to be considered in context with negative impacts from Hg. For assessing and monitoring trends in human health risk, we emphasize commercially and/or recreationally valuable fish that have documented patterns of Hg bioaccumulation.

In estuaries, mummichogs are ubiquitous because they can withstand broad environmental conditions. They are often used for examining environmental Hg loadings (Khan and Weis 1993) and their potential ecological effects (Zhou et al. 1998). Some studies indicate they can build a tolerance to contaminants, including MeHg (Weis 2002), although individuals with elevated MeHg levels are more prone to being preyed on because of impairment to predator-avoidance behaviors (Smith and Weis 1997). While the mummichog inhabits quiet backwaters, the sand lance prefers shallow, sandy brackish waters and is an important prey item for estuarine, coastal, and even semi-pelagic birds. The striped bass is a common, recreationally fished species of estuaries and has been shown to accumulate >0.5 μg/g, ww of Hg (Davis et al. 2002; Mason et al. 2006). However, because it has multiple feeding strategies, migrates among areas of differing Hg sensitivity, and exhibits poor growth–Hg relationships, its use as an indicator species is limited. A species of less importance to the sport fishery industry, but one that has commercial and subsistence interests is the scup or porgy. It is found in estuaries and nearshore areas, and is a primary indicator species used by the USEPA National Contaminant Assessment Program (NCAP) and rarely exceeds 0.10 μg/g (ww) (Cunningham et al. 2003).

In coastal areas, the striped anchovy and butterfish are common and widespread forage fish for piscivorous wildlife. Bluefish are a recreationally important, coastal piscivore that have: (1) well-defined age–growth and growth–Hg relationships, (2) are widely distributed, and (3) been characterized for MeHg contamination in a variety of coastal waters (Ashley and Horwitz 2000; Burger et al. 2005; Hammerschmidt and Fitzgerald 2006a). The winter flounder is a highly valued species for Hg monitoring because of its recreational and commercial interests, extensive use by the NCAP, and prevalence of tumors related to contaminant exposure (Moore et al. 2004)—making it a more sensitive indicator species than the closely related summer flounder (*Paralichthys dentatus*). Both flounder

species are routinely sampled by the NCAP and generally have fillets < 0.10 μg/g (ww) of Hg.

The source of MeHg in semipelagic and pelagic habitats is unknown. MeHg bioaccumulation in these regions may result from (1) deep ocean waters, (2) shelf, slope, and/or deeper ocean sediments, and (3) hydrothermal vents (Kraepiel et al. 2003; Lamborg et al. 2006; Hollweg et al. 2009; Liu et al. 2009), hydrologic advection (Hammerschmidt and Fitzgerald 2006b) and bioadvection from the coastal zone (Fitzgerald et al. 2007), or methylation in waters of high-biological productivity (Topping and Davies 1981; Mason and Sullivan 1999; Chen et al. 2008). Different fish species emerge as suitable indicators of MeHg availability in these offshore habitats. Ecological risk in semipelagic waters can be well documented using Atlantic herring (Braune 1987b). Measurements of Hg in sedentary, demersal species such as the Atlantic cod provide an opportunity to monitor MeHg bioaccumulation in specific locations over time (e.g., Staveland et al. 2005). Other high trophic-level species with commercial value and known Hg concentrations include the yellowfin tuna (Kraepiel et al. 2003) and related species.

For pelagic waters, the North Atlantic saury is one of the most abundant epipelagic species (<200 m depths) in our study area and serves as prey for many high trophic-level species. A more novel fish group to monitor are lanternfish that are found in mesopelagic environments (>300 m in depth) and migrate diurnally over hundreds of meters in depth. They regularly form the prey base for pelagic seabirds such as the Leach's storm-petrel, which forage nocturnally on lanternfish at the ocean surface (Montevecchi et al. 1992). Concentrations of Hg in lanternfish have been used to support the current understanding of deepwater MeHg production in the open ocean, and to identify temporal and spatial patterns in oceanic Hg bioavailability (Monteiro et al. 1996; Martins et al. 2006).

While high trophic-level species such as the blue shark and swordfish are of commercial and recreational interest, because they can bioaccumulate concentrations of Hg that are harmful for human consumption (often >1.0 μg/g, ww; unpublished data from the U.S. Food and Drug Administration [FDA] and USEPA; Branco et al. 2007), low densities and wide-ranging abilities make their use as indicators of specific areas challenging. Although average shark Hg levels have been generically described by the FDA, some species can attain exceedingly high muscle Hg levels depending on their size, prey base, and geographic origin. For example, Garica-Hernandez et al. (2007) found smooth hammerhead sharks (*Sphyrna zygaena*) in the Gulf of California surpassing 21.0 μg/g (ww) in their muscle tissue. Based on δ^{15} N values, less variable prey bases in some shark species, such as the blue shark, make it a more preferable species to monitor Hg levels over spatiotemporal scales of interest than species with highly variable prey bases, such as the shortfin mako shark (*Isurus oxyrinchus*) (Estrada et al. 2003).

Sampling strategies can vary widely and depend on the target habitat and species. Fish are typically analyzed on a whole body basis for ecological health monitoring, while muscle tissue is removed and the fillet is analyzed for assessment of human exposure. Both approaches tend to analyze total Hg on a wet weight (ww) basis. Most of the Hg in fish muscle is in the methyl form (Bloom 1992). Lower trophic-level fish also provide an ability to predict MeHg transfer rates to higher trophic levels. For fish species of commercial and recreational interest, acquisition of tissue samples for Hg analysis using biopsy plugs of muscle directly at the dock is a viable and cost-effective strategy (Bank et al. 2007a). Biopsies and other nonlethal sampling methods are recommended for sharks, which have experienced tremendous declines because of overfishing (Myers et al. 2007).

Since variation in fish Hg concentrations is commonly influenced by the growth characteristics of length, age, or weight, those fish species with well-defined, growth–Hg relationships are the best candidates as indicator species. Monitoring Hg programs using fish should always acquire total length, weight, and ideally other important metrics such as age. There are other associated data that increase interpretation powers, such as Se levels and stable isotope information. The role of bioavailable Se is of growing interest to risks imparted by MeHg from fish to humans (Ralston et al. 2007).

Bird Indicators

Suggested bird species for monitoring Hg trends vary according to foraging guild (i.e., piscivore vs. invertivore) and habitat type (Tables 1, 3). Our selection criteria for birds emphasizes breeding individuals because they are generally territorial (or have small home ranges), are likely consuming prey items that have higher MeHg concentrations than in the winter (Ramlal et al. 1993; Leermakers et al. 1995), and are more reflective of local conditions compared to migrants. Summertime Hg concentrations can also be better linked to meaningful endpoints of adverse effects, such as reproductive success (Burgess and Meyer 2008; Evers et al. 2008). Use of multiple bird species for monitoring Hg provides the most

comprehensive coverage for detecting changes in various habitat types and food webs that may not be predictive from one another (Pearce et al. 1989).

In estuaries, there are often species of high conservation concern (Ackerman et al. 2007) with apparently high sensitivity to Hg input (Heinz et al. 2009), and recently documented adverse reproductive effects from Hg (Schwarzbach et al. 2006). *Ammodramus* sparrows are recommended as indicators for estuarine invertivore food webs. One species, the saltmarsh sharp-tailed sparrow has Hg body burdens that tend to exceed those in associated songbirds (Shriver et al. 2006), and in some estuaries, lowered reproductive success is related to elevated blood Hg levels (Lane et al. 2008). Indicators of the estuarine piscivore food web include a choice of over 10 species of wading birds in our study area. Unfledged wading birds, particularly the black-crowned night-heron, is often used as the indicator age group (Rattner et al. 2000; Henny et al. 2002). Areas without wading bird sampling opportunities can be evaluated for MeHg availability in piscivores through sampling of the belted kingfisher. Kingfishers are a relatively unique indicator, as they are one of the few birds that can be used to compare MeHg availability across marine and freshwater habitats (Evers et al. 2005).

In coastal waters of the Gulf of Maine, the common eider regularly forages on the blue mussel (Wayland et al. 2001), while the piscivorous black guillemot depends on benthic fish (Butler and Buckley 2002). Mean Hg concentrations in eggs of the black guillemot are significantly greater than those in associated seabirds (Goodale et al. this issue), and because their prey occupy relatively small home ranges, guillemots are valuable indicators for characterizing distinct areas of interest. The osprey breeds along our entire study area and is an important indicator species since it is an obligate piscivore, is found across the northern hemisphere, and is commonly monitored for Hg in both freshwater (Hughes et al. 1997) and estuarine and marine ecosystems (Golden and Rattner 2003; Henny et al. 2008; Rattner et al. 2008).

The common tern is another ubiquitous piscivore that regularly forages in coastal and offshore areas and has well-described Hg body burdens (Braune 1987a; Burger et al. 1994; Nisbet et al. 2002). The common tern has Hg body burdens that are comparable to the much larger double-crested cormorant (Goodale et al. this issue). Mercury monitoring efforts with cormorant eggs are an efficient approach because of the cormorant's common status and colonial nesting tendencies.

Describing the availability of MeHg in pelagic waters of our study area using birds is challenging. The Leach's storm-petrel is best. Although this seabird nests on outer coastal islands in the Gulf of Maine, it forages along the continental shelf on mesopelagic organisms such as myctophids, amphipods, and euphausiids (Montevecchi et al. 1992). Atmospheric deposition of Hg on the ocean surface increasingly reflects global airshed Hg concentrations as the distance from mainland increases (Gill and Fitzgerald 1987). Blood Hg levels in the storm-petrel may provide a relatively accessible technique for monitoring changes in food web MeHg availability that is related to either global Hg pools or MeHg sources more distant from the continent.

Bird tissues regularly used for monitoring environmental Hg loads for short-term exposure are blood and eggs (usually as ww) and, for longer-term exposure, feathers (as fresh weight) (Evers et al. 2005). Feathers provide a good measure for examining long-term Hg trends (Thompson et al. 1992; Monteiro and Furness 1997). These three tissues are generally analyzed for total Hg because they are mostly representative of MeHg (Wolfe et al. 2007). Similar to fish, other measurements are typically required to best describe Hg body burdens. While bird age past the first 4–5 years is generally unknown (unless uniquely marked), the size, molt status, sex, and age class (juvenile vs. adult) are important for interpreting Hg levels (Evers et al. 2005). An understanding of Se levels and stable isotopes is also useful. The potential protective role of bioavailable Se in birds appears to be complex (Scheuhammer et al. 2008).

Mammal Indicators

Our suite of mammalian indicators includes a broad mix of terrestrial and marine taxa (Tables 1, 3). Although mustelids are used widely as indicators of MeHg availability in freshwater ecosystems (Strom 2008; Klenavic et al. 2008), two species, the American mink and North American river otter, also forage regularly in estuarine and marine ecosystems where fish are a dominant prey (Yates et al. 2005; Lake et al. 2007).

Pinnipeds are the most accessible marine mammal to be utilized as indicators for coastal habitats. Seals depend on ledges or beaches for pupping and resting, which provides a feasible method for tracking individual MeHg body burdens. Harbor seals are the most common pinnipeds in our study area, have the smallest home range compared to

associated seal species, and are most accessible for sampling purposes. Their importance as a global sentinel species is also well recognized (Ross et al. 1996). Harbor seals selectively forage on small, schooling fish and squid, and their diet changes seasonally (Payne and Selzer 1989).

In semi-pelagic waters, toothed cetaceans forage on prey items such as squid and fish that are higher in the food web than krill and other invertebrates. Their Hg body burdens therefore average higher than baleen cetaceans (Hansen et al. 1990). The harbor porpoise is one of the more common and ubiquitous toothed cetacean in coastal and semi-pelagic waters, feeding on cephalopods and Atlantic herring (Fontaine et al. 1994). In more southern waters of our study area, the bottlenosed dolphin is often used as an indicator of contaminants, including Hg (Kuehl and Haebler 1995; Frodello et al. 2000).

In pelagic and more coastal waters of the northern Atlantic Ocean, pilot whales are useful indicator species because of their dietary importance to many native cultures (Andersen et al. 1987), where consumption has been regulated due to elevated Hg concentrations in muscle tissue (up to 3.3 µg/g, ww) (Weihe et al. 1996). The diet of pilot whales includes fish and cephalopods (Katona et al. 1993). Beaked whales are rarely accessible for sampling purposes unless they are stranded. Their novel value as indicators of MeHg availability in open ocean habitats is their longevity and high trophic-level position, which can result in highly elevated liver Hg concentrations (Bustamante et al. 2003).

Marine mammals are known to bioaccumulate varying levels of MeHg depending on species, diet, age, sex, reproductive status, geographic distribution, and range of ocean habitat (Nagakura et al. 1974; Gaskin et al. 1979; Dietz et al. 1996; Wagemann et al. 1998; Das et al. 2003). Multiple tissues are regularly used to characterize Hg body burdens, including skin, blubber, muscle, kidney, and liver. Muscle is more likely to contain a higher proportion of MeHg (50–100%), while liver contains a lower percentage (O'Hara et al. 2003). While sampling live marine mammals is challenging, samples taken from individuals stranded or by-catch from fish nets does provide a routinely available approach for acquiring tissues. As in fish and birds, the protective or toxic role of associated Se levels should be determined as well. Marine mammals have adapted to elevated levels of dietary MeHg by sequestering it as a nontoxic, inorganic form in the liver, often with a 1:1 ratio with Se (Koeman et al. 1973; Itano et al. 1984; Ikemoto et al. 2004).

Conclusions

A high resolution and comprehensive program for monitoring environmental Hg loads in air, sediment, and water of estuarine and marine environments, and the subsequent ecological response in invertebrates, fish, birds, and mammals, in terms of both human and ecological health concerns is described above. Our intention for developing such a detailed list of indicator compartments is to provide multiple selection options to accommodate existing monitoring efforts, and different site-specific objectives, expertise, and funding. To best detect temporal changes in environmental loading, we recommend a minimum effort to include measurements of wet Hg deposition, MeHg and percent MeHg in estuarine/marine sediment, total Hg and MeHg in young fish occupying small home ranges, high trophic-level fish of greatest local interest for human consumption, and relative breeding birds (Fig. 2).

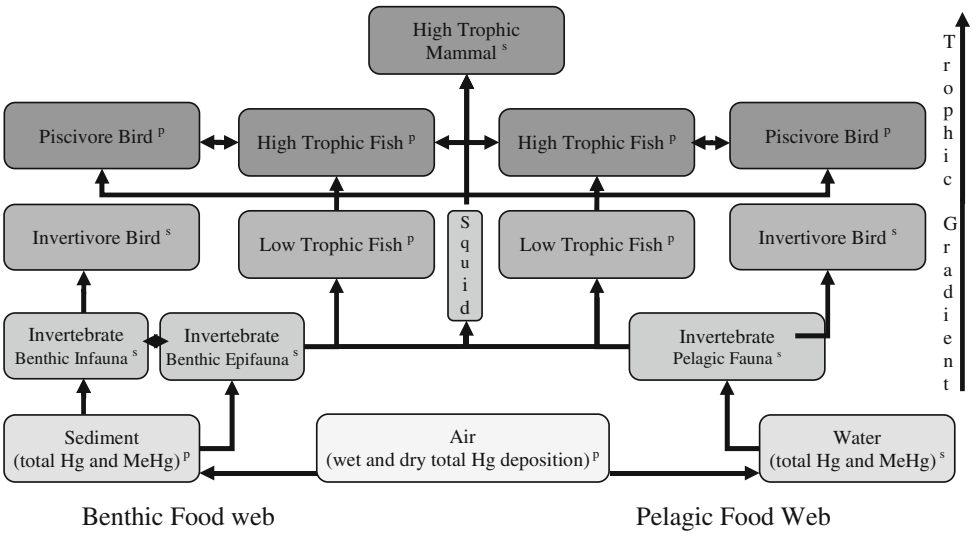

Figure 2. Simplified universal food web components recommended for mercury sampling. Recommended sampling design for intensive sites includes all components. For cluster sites, include at least primary components (p) and secondary components (s) when possible. The influence of squid and other cephalopods as a transfer mechanism for MeHg in the food web may be important for higher trophic levels.

Mercury data have been amassed for several coastal areas and represent multiple compartments measured in parallel with process-level investigations, including the Chesapeake Bay (Mason et al. 1999; Lawson et al. 2001; Hollweg et al. 2009); Long Island Sound (Rolfhus and Fitzgerald 2001; Balcom et al. 2004; Hammerschmidt et al. 2004; Hammerschmidt and Fitzgerald 2006a); Acadia National Park (Bank et al. 2007b; Kahl et al. 2007); and outer Bay of Fundy (Gaskin et al. 1979; Pearce et al. 1979; Braune 1987a,b; Elliot et al. 1992; Sunderland et al. 2004, 2008; Harding et al. 2005; Ritchie et al. 2006) (Fig. 1). These locations are strong candidates for the basis of an organized, standardized monitoring program that tracks estuarine and marine environmental Hg loadings as well as watershed contributions. Within these and other areas, site considerations should encompass protected areas, which commonly serve as important shellfish beds, nursery areas for fish, and areas of wildlife conservation. Examples of such areas include National Wildlife Refuges, National Parks, National Estuarine Reserves, and The Nature Conservancy preserves.

Establishing a standardized, long-term Hg monitoring network in coastal areas is essential for monitoring and demonstrating the impact of domestically produced Hg emissions, as well as the increasing global Hg loads stemming from rapidly growing sources in Asia that, based on current models and estimates, contribute an estimated 54% of the global anthropogenic Hg emissions in 2000 (Pacyna et al. 2006). Today, those sources are projected to contribute at even higher rates, potentially offsetting emission declines in other regions of the northern hemisphere (e.g., eastern United States; Monson et al. 2009). The subsequent uncertainty for increases in the global Hg pool from Asia and elsewhere, combined with effects from climate change (e.g., Faroe Islands; Booth and Zeller 2005) and ocean acidification (Caldeira and Wickett 2003), both of which may exacerbate MeHg availability, are especially serious threats to marine-based human and ecological health. While our template for monitoring environmental Hg loading is most applicable to marine ecoregions identified by Spalding et al. (2007) in the temperate North Atlantic realm, it can be modified for other biogeographic areas by using a simplified universal food web approach (Fig. 2). While arctic realms are of increasing concern for the magnitude of Hg loadings (Lindberg et al. 2002) and related adverse implications to wildlife (Braune et al. 2006) and human health (Jewett et al. 2003), there is also evidence that marine ecoregions encompassing equatorial realms support high trophic marine inhabitants with elevated Hg levels that may reflect habitat sensitivity to environmental Hg loading (Evers et al. 2009). The threats posed by anthropogenically redistributed Hg on marine habitats equire strong scientific underpinnings to understand the considerable complexities in Hg biogeochemistry and MeHg production. This understanding is critical to properly regulating and managing Hg at local, continental, and global scales. Standardized Hg monitoring programs can provide the necessary linkages between those science and policy needs.

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EcoHealth

Editorial

Ecotoxicology of Methylmercury: A Transdisciplinary Challenge

Many of us vividly recall the devastating effects of point source mercury (Hg) pollution in Minimata Bay, Japan, documented by poignant photos of mothers caring for their children afflicted with crippling deformities. From 1952 to 1968, Hg waste from the production of acetaldehyde and vinyl chloride was discharged (200–600 tons) into Minimata Bay. This resulted in Hg bioaccumulation by marine fish and shellfish, biomagnification in the marine food web, and subsequent ingestion by humans. Although this was perhaps the most severe case in history, the processes determining the fate of Hg in the environment are common to all aquatic ecosystems.

The Minimata catastrophe also revealed that the neurotoxicological effects on fetal development from pregnant women ingesting Hg-contaminated fish could be especially severe. This finding, as well as research over subsequent decades on the processes of Hg bioaccumulation and biomagnification in aquatic and marine ecosystems, has led to today's warnings by many public health agencies about pregnant women limiting their consumption of fish like tuna, swordfish, shark, and other species that feed on the top of food chains.

Yet, as with most environmentally mediated health risks involving either chemical or biological agents, the ecological dimensions and health effects of Hg are far more subtle than either the tragic Minimata case or the health warnings suggest. This was illustrated more recently by pioneering transdisciplinary and participatory research carried out in Brazil to address chronic, low-level mercury exposure. These efforts demonstrated the efficacy of an ''ecosystem approach'' in revealing how deforestation and farming practices in the Amazon led to subclinical but nonetheless debilitating neurological manifestations—a ''best practice'' case example in ecohealth (Lebel 2003).

Few environmental health issues demand a transdisciplinary research agenda that spans such a wide range of disciplines—from biogeochemistry and ecosystem ecology to epidemiology and participatory action research—as environmental exposure to methylmercury (MeHg). Also, among the classic environmental health issues involving human and wildlife exposures to persistent and global contaminants (e.g., lead and organochlorides), MeHg remains the least resolved, in terms of both science and policy.

In the summer of 2006, the ''Eighth International Conference on Mercury As a Global Pollutant'' was held in Madison, Wisconsin, to which over 1000 scientists from around the world came to share their understanding of the environmental fate of this pervasive contaminant (Hurley et al. 2007). The global nature of Hg is due to a number of factors: (1) It is generated largely from coal-fired power plants which are increasing worldwide; (2) it is atmospherically transported from these sources to eventual deposition on all land and water surfaces; and (3) it is transferred to humans through fish consumption which knows no international or socioeconomic boundaries.

Since the fate, bioavailability, and toxicity of Hg and MeHg are controlled by physical, biogeochemical, and biological processes, the study of Hg is by necessity an interdisciplinary endeavor. Hg in the environment is found in several forms. In the atmosphere, it is predominantly elemental Hg (Hg^0); in water, it is mostly the inorganic form Hg^{2+}; and in aquatic sediments, it is transformed from Hg^{2+} into the more toxic organic form, MeHg. MeHg is the form preferentially assimilated by living organisms and the dominant form found in fish that humans con-

sume. Each of the environmental transformation steps is controlled by complex chemical and biological processes, as is the bioaccumulation and trophic transfer of Hg in aquatic food webs. As a result, the community of scientists who study Hg fate includes atmospheric scientists, biogeochemists, aquatic ecologists, wildlife ecologists, toxicologists, and epidemiologists.

At the 2006 Hg meeting, there were many discussion and votes were tallied to assess the consensus on certain scientific conclusions. For example, 97% of scientists agreed that although there have been major reductions in Hg emissions in the US and Europe, there has been no change in the total Hg pool—likely due to the increase in emissions from other geographic areas (Anonymous 2007). Less agreed upon were issues related to Hg and human health; among them was the role of selenium in reducing Hg toxicity (69% consensus; Anonymous 2007).

Although there are animal studies showing that selenite reduces inorganic Hg toxicity, there is almost no evidence for protection from MeHg toxicity by the organic forms of selenium found in the human diet. Moreover, there are no human studies for showing a protective role for selenium against Hg neurotoxicity (Mergler et al. 2007). Most agreed that this was an area of Hg research that requires a great deal more investigation.

The wide attendance of the 2006 Hg conference was indeed a reflection of how much research had already been conducted on the fate and effects of Hg. But like all complex environmental problems, there remain many gaps in our knowledge. Since the main vector for human exposure is consumption of marine fish and shellfish, its fate in marine systems is particularly important to understand. Much of the past research has been conducted in freshwater and upland forested ecosystems, and, in many of the discussions by panels of scientists at the meeting, there was a recurring recognition that there needs to be greater focus on understanding Hg fate in marine ecosystems (Mergler et al. 2007; Munthe et al. 2007; Swain et al. 2007). Of the many unanswered questions, examples include: Where are the main sources of Hg methylation in the oceans that supply MeHg to the tuna fish that humans eat? Since MeHg is transferred mostly through food consumption, what are the predator–prey linkages in marine food webs that result in trophic transfer of MeHg? Are coastal populations at greater risk of Hg exposure than inland populations?

These questions and others became the focus of a subsequent workshop sponsored by the National Institute of Environmental Health Science in the autumn of 2006 on Hg fate and bioavailability in marine ecosystems (Chen et al. 2008). Scientists spanning a range of disciplines from biogeochemistry to biomedical sciences were convened and their discussions were the genesis of the MeHg articles found in this issue's Special Feature.

Without a doubt, the Hg problem is far from diminishing. Hg emissions and deposition are driven globally by growing energy demand and the increasing numbers of coal-fired power plants being constructed worldwide. Fisheries in all the world's oceans are impacted by this ubiquitous pollutant and indigenous populations reliant on fish or marine mammal species at the top of the food chain are at particular risk of exposure. Although the human impacts of the Minimata Hg poisonings were extremely acute and particularly disastrous, the more subtle effects of long-term chronic exposure to MeHg are far more common and widespread. The pervasiveness and complexity of this problem and its interaction with accelerating transformations and stresses on ecosystems (remove) worldwide, point to the need for much more research—especially employing transdisciplinary approaches that span marine chemistry, ecosystem ecology, wildlife health, and human physiology and development.

We are pleased to feature this Special Feature on MeHg and hope it will spur increased research interest and intervention to reduce the risk and impacts of MeHg toxicity on ecosystems, including humans.

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ECOHEALTH

Special Feature: Methylmercury

Forum

Mercury Toxicity and the Mitigating Role of Selenium

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Abstract: Mercury is a well-known environmental toxicant, particularly in its most common organic form, methylmercury. Consumption of fish and shellfish that contain methylmercury is a dominant source of mercury exposure in humans and piscivorous wildlife. Considerable efforts have focused on assessment of mercury and its attendant risks in the environment and food sources, including the studies reported in this issue. However, studies of mercury intoxication have frequently failed to consider the protective effects of the essential trace element, selenium. Mercury binds to selenium with extraordinarily high affinity, and high maternal exposures inhibit selenium-dependent enzyme activities in fetal brains. However, increased maternal dietary selenium intakes preserve these enzyme activities, thereby preventing the pathological effects that would otherwise arise in their absence. Recent evidence indicates that assessments of mercury exposure and tissue levels need to consider selenium intakes and tissue distributions in order to provide meaningful risk evaluations.

Keywords: mercury, selenium, toxicity, environment, heavy metals

Mercury is a naturally occurring element that originates from geological materials, but readily distributes into the air, water, soil, and biomass of the environment. The uncharged metallic mercury form (Hg^0) is highly volatile and enters the atmosphere where it can reside for extended periods. Substantial releases of Hg^0 from natural sources such as solar irradiation, combustion (grass and forest fires), and volcanism cannot be controlled. However, there is substantial concern regarding mercury releases caused by human activities. Regardless of the source, atmospheric Hg^0 eventually becomes charged and precipitates as inorganic mercury (Hg^{2+}) deposited on ground and water surfaces. Bacteria convert Hg^{2+} to form methylmercury ($MeHg^+$) that is largely found in association with the sulfur of cysteine and similar thiomolecules in the aquatic biomass. As a result, the most common organic mercury compound found in the environment is MeHg-cysteine. The charge on the Hg moiety is satisfied by complex formation with sulfur, which although thermodynamically stable, is kinetically promiscuous. This is also the predominant form accumulated in the aquatic biomass that eventually became coal. Because many millennia of mercury deposition and bioaccumulation are stored in these organic materials, thousands of years' worth of mercury deposition is released back into the environment when coal is burned. As a result, coal-burning power plants are a dominant source of human-caused mercury emissions to the atmosphere.

Coal-fired power plants in the United States account for ~40% of domestic (~5% of global) human-caused mercury emission (Driscoll et al. 2007); the U.S. Environmental Protection Agency (EPA) estimates that ~25% of

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these domestic emissions are deposited within the conterminous United States, while the remainder enters the global biogeochemical cycle (http://www.epa.gov/mercury/about.htm). Although contributions from the United States continue to diminish as more advanced technologies for mercury capture are developed and employed, environmental mercury deposition may increase in coming years as the result of dramatically increasing inputs from overseas. Mercury releases from coal-fired power plants in Asia are major contributors to the global atmospheric pool (>50%) and are increasingly significant sources of atmospheric mercury deposition. Newly built, large, coal-fired power plants are coming on line in China and India at the rate of approximately one per week. Although these new plants burn coals that often have much higher mercury contents than those burned in North America, they are not being equipped with pollution control technologies that are mandatory in the United States. This is particularly worrisome since China currently burns more coal than the United States, European Union, and Japan combined, and its consumption rate is rapidly growing. Increasing mercury releases from these new power plants in Asia will inevitably result in increasing atmospheric mercury deposition in North America and throughout the Northern Hemisphere.

Atmospheric mercury deposited directly into water, or onto land where it can be washed into bodies of water, is rapidly incorporated into aquatic ecosystems and food webs. There, bacteria in the sediments and water column change it into $MeHg^+$. The transformation of Hg^+ to $MeHg^+$ is a critical step in this process, as $MeHg^+$ is the most readily assimilated form that bioaccumulates in all aquatic organisms and, ultimately, biomagnifies as it is transferred up the food web. Fish and shellfish consumption are the main sources of $MeHg^+$ exposure to wildlife and humans (http://www.epa.gov/mercury/about.htm), and there are particularly acute concerns regarding the effects of prenatal exposure on neurological development in children. Sixty percent of the fish and shellfish consumed by humans are from marine ecosystems, but most of the research on the fate of mercury has been conducted in upland terrestrial and freshwater ecosystems (Mergler et al. 2007; Sunderland 2007; Chen et al. 2008). Far less is known about mercury biogeochemistry in the coastal and open ocean systems.

Although initially difficult to understand, experimental animal and human exposure studies delineate the risks that accompany $MeHg^+$ exposures. In controlled feeding studies in fish, birds, and mammals, the consumption of diets that contained $MeHg^+$ at environmentally realistic concentrations resulted in a range of toxic effects including behavioral, neurochemical, hormonal, and reproductive changes. Limited field-based studies with wild piscivorous bird species demonstrated significant relations between $MeHg^+$ exposure and various indicators of toxicity, including reproductive impairment (Scheuhammer et al. 2007). The most extensive human exposure to $MeHg^+$ was caused by the dumping of mercury compounds into Minamata Bay, Japan. It has been estimated that 27 tons of mercury were dumped into Minamata Bay from 1932 to 1968. Thousands suffered severe mercury poisoning symptoms and many died from what became known as Minamata disease, caused by consumption of fish or shellfish contaminated with up to 50 ppm of mercury (Takeuchi et al. 1962). Of special concern, children that were highly exposed in utero often showed severe neurodevelopmental impairments, even when their mothers exhibited minimal or no clinical signs.

There is growing awareness that the toxicity of $MeHg^+$ is intimately linked with its high binding affinities with selenium. Selenium is a nutritionally essential element with particularly important roles in brain and endocrine tissues. This corresponds remarkably well with the recognition that the target tissues of $MeHg^+$ toxicity are the neuroendocrine and nervous systems. Virtually all forms of animal life that possess nervous systems also possess selenium-dependent enzymes that utilize selenocysteine to perform important antioxidant and redox control functions. These enzyme functions appear to be indispensable, especially in brain tissues where they are required to protect against oxidative damage from reactive oxygen metabolites. Since mercury is uniquely able to inhibit selenium-dependent enzyme activities in brain tissues (Watanabe et al. 1999), the risks of oxidative brain damage as a result of mercury toxicity directly correspond to Hg:Se molar ratios in tissues (Ralston et al. 2007, 2008). Converging evidence from cell culture studies indicates a progressive decrease in the activity of selenium-dependent glutathione peroxidase enzyme activities in cells exposed to mercury (Bulato et al. 2007).

In animal studies where mercury toxicity has been observed, mercury has consistently been present in substantial molar excess of selenium in the affected tissues (Cuvin-Aralar and Furness 1991; Chapman and Chan 2000). Since the binding affinity between mercury and selenium is a million times greater than the affinity between sulfur and mercury (Dyrssen and Wedborg 1991), it is easy to understand why Hg:Se molar ratios in excess of a 1:1

stoichiometry are increasingly toxic. Since fetal supplies of selenium are dependent upon ratios of selenium to mercury in the mother's food sources, inhibition of the biological functions of selenium plays a particularly significant role in the mechanism of prenatal mercury intoxication.

Selenium has been known to play a role in binding toxic metals and potentially reducing toxicity. Accumulation of mercury in tissues of marine mammals or mineworkers following exposure or ingestion is accompanied by increased accumulation or retention of selenium (Koelman et al. 1973; Kosta et al. 1975). The concentrations of mercury in brain tissues of these miners approached, but never exceeded a 1:1 molar ratio (Falnoga et al. 2006). Instead, the amount of "free" selenium in excess of mercury remained essentially constant, even though the amount of mercury rose to concentrations many times higher than the normal brain selenium content. Studies have demonstrated the binding of complexes of mercury–selenium, silver–selenium, and cadmium–selenium by plasma selenoprotein P (Yoneda and Suzuki 1997; Sasakura and Suzuki 1998), leading to the proposal that this protein may function to chelate heavy metals, reducing their toxicity. Binding of zinc (Yan and Barrett 1998), nickel (Mostert et al. 1998), and silver (Sasakura and Suzuki, 1998) by selenoprotein P has also been reported. In miners exposed to high concentrations of mercury, expression of both selenoprotein P protein and glutathione peroxidase activity was increased. These increments were accompanied by elevated selenium concentrations in serum. In addition, selenoprotein P bound more mercury at higher mercury exposure concentrations (Chen et al. 2006).

The ability of selenium compounds to decrease the toxicity of mercury has been established in all species of mammals, birds, and fish investigated (Cuvin-Aralar and Furness 1991; Chapman and Chan 2000; Raymond and Ralston 2004 [and references 16 and 17 therein]). Therefore, it is important to consider the molar relationships between mercury and selenium when investigating neurodevelopmental outcomes of maternal mercury exposure during pregnancy. Since free-ranging marine fish are rich sources of selenium in substantial molar excess of mercury, this may explain why the largest and most recent studies of effects of maternal seafood consumption (and associated $MeHg^{+}$ exposures) on child neurodevelopmental outcomes find substantial benefits ($\sim$5–10 IQ points) instead of harm.

Knowledge of selenium's influence on mercury's fate in aquatic ecosystems and on mercury exposure, bioaccumulation, and toxicity is substantial, but urgently requires increased attention. In order to perform accurate environmental and epidemiological mercury exposure risk assessments, future studies will need to simultaneously assess the amounts and forms of selenium that are also present. Otherwise, important beneficial effects of maternal seafood consumption will continue to be mistakenly associated with risk, while the actual risks of mercury exposure that may accompany consumption of freshwater fish will continue to go unrecognized.

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Special Feature: Methylmercury

Original Contribution

Selenium Health Benefit Values as Seafood Safety Criteria

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Abstract: Selenium (Se) is absolutely required for activity of 25–30 genetically unique enzymes (selenoenzymes). All forms of life that have nervous systems possess selenoenzymes to protect their brains from oxidative damage. Homeostatic mechanisms normally maintain optimal selenoenzyme activities in brain tissues, but high methylmercury (MeHg) exposures sequester Se and irreversibly inhibit selenoenzyme activities. However, nutritionally relevant amounts of Se can replace the Se sequestered by MeHg and maintain normal selenoenzyme activities, thus preventing oxidative brain damage and other adverse consequences of MeHg toxicity. Findings of studies that seem contradictory from MeHg exposure perspectives are entirely consistent from MeHg:Se molar ratio perspectives. Studies that have reported dose-dependent consequences of maternal MeHg exposures on child development uniformly involved seafoods that contained much more Hg than Se. Meanwhile more typical varieties of ocean fish contain much more Se than Hg. This may explain why maternal MeHg exposure from eating ocean fish is associated with major IQ benefits in children instead of harm. Therefore, instead of being avoided, ocean fish consumption should be encouraged during pregnancy. However, the safety of freshwater fish consumption is less certain. In freshwater fish, MeHg bioaccumulation and toxicity are both inversely related to Se bioavailability. Their Se can be far lower than their MeHg contents, potentially making them more dangerous than pilot whale meats. Therefore, to provide accurate and appropriate regulatory advice regarding maternal consumption of seafoods and freshwater fish, Hg:Se molar ratios need to be incorporated in food safety criteria.

Keywords: brain selenium, seafood fish, mercury toxicity

Extremely high methylmercury (MeHg) exposures in Japan (Takeuchi and Eto, 1999; Tsubaki and Irukayama, 1977) and Iraq (Marsh et al., 1987) affected thousands of people and resulted in dose-dependent pathologies that ranged from barely perceptible to lethal. Maternal consumption of heavily contaminated foods in these catastrophes resulted in children being exposed to MeHg in utero. Children who were exposed during fetal development were particularly vulnerable to MeHg, frequently showing signs of severe neurologic damage, although their mothers were asymptomatic (Harada, 1968; Marsh et al., 1987). Based on the severe adverse effects of these disastrous MeHg exposures, a series of prospective human epidemiological studies were performed to evaluate the effects of MeHg exposure from seafood consumption during pregnancy. These studies were designed to test the hypothesis that ''Maternal MeHg exposures are directly associated with adverse child development outcomes.''

Major studies have been conducted to examine the effects of maternal MeHg exposure from fish consumption

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on child developmental outcomes in population groups from New Zealand (Crump et al., 1998), Faroe Islands (Grandjean et al., 1997), Seychelle Islands (Myers et al., 1998, 2000), United Kingdom (Hibbeln et al., 2007), United States (Lederman et al., 2008), and most recently, Denmark (Oken et al., 2008). Evidence from these epidemiological studies have variously reported clinically relevant harmful effects on child health outcomes (New Zealand, Faroes), no harmful effects on child outcomes (Seychelles, United Kingdom, United States, Denmark), or substantial beneficial effects on child neurodevelopment and IQ (United Kingdom, United States, Denmark). Therefore, the results of these studies do not consistently support the conventional hypothesis. Instead, the findings of the largest, most complete, and most appropriate studies directly conflict with the hypothesis and seem to disprove it. However, based on observations in the catastrophic poisoning episodes and generally consistent findings of dose-dependent adverse effects related to maternal MeHg exposures (Clarkson and Magos, 2006; Watanabe et al., 1999a, b) and in animal studies (Ralston et al., 2008), the conventional hypothesis is not likely to be completely wrong, but only seems wrong because it is incomplete.

Misleading and mistaken impressions provided by the media have induced many mothers to avoid or excessively limit their seafood intake during pregnancy in an effort to protect their unborn children. Widespread misunderstandings of regulatory advice have even led some physicians to recommend their pregnant patients completely avoid seafood consumption. Unfortunately, the focus on potential harms from MeHg exposure often has completely overshadowed consideration of the beneficial effects of nutrients present in ocean fish. In response to this unbalanced perspective, the Joint Expert Committee on Food Additives (2003) has recommended that "nutritional benefits be weighed against the possibility of harm when limits on the MeHg concentrations in fish or on fish consumption are being considered." Ocean fish are particularly rich in Se and omega-3 fatty acids (USDA National Nutrient Database, 2009). Therefore, fish consumption improves intakes of these beneficial nutrients, which are particularly important during pregnancy. Similarly, the summary statement of the International Bioindicators Roundtable recommended that Hg:Se molar ratios must be evaluated to interpret effects of Hg exposures and identify susceptibilities (Henshel et al., 2007). Consideration of the balance of relative amounts of Hg and Se in food safety issues seems to clarify many aspects and remove the inconsistencies in effects of MeHg exposure in studies designed to examine the conventional hypothesis.

Metabolism of Dietary Selenium

Selenium (Se) is a nutritionally essential element that is present in all foods, but is particularly abundant in ocean fish. In a survey of 1,100 foods (USDA National Nutrient Database, 2009), seafoods comprised 17 of the top 25 dietary sources of Se. The molecular forms of Se that predominate in foods (Fig. 1) are the amino acids selenocysteine (Sec) and selenomethionine (SeMet). Dietary Sec, SeMet, and other less abundant organic forms of Se in the diet must be degraded to the inorganic selenide form before their Se can be used in protein synthesis (Fig. 1). Selenite often is used in nutrition studies because it is well absorbed and readily forms selenide (Hsieh and Ganther, 1977), the precursor required for Se incorporation into Sec (Fig. 1).

Although inorganic Se, Sec, and SeMet are all readily absorbed in the digestive tract and directly or eventually provide the inorganic Se needed to support intracellular metabolic cycles of Sec synthesis, there are important distinctions between the processes governing the metabolism of Sec that is synthesized in animal tissues and the SeMet that is synthesized in plants. In contrast to other amino acids, Sec is not readily recycled for subsequent incorporation in new proteins, but is instead degraded to release the inorganic Se that is needed for de novo synthesis of Sec just before its insertion into new selenoproteins. Because selenite is already in an inorganic form when it arrives in the cell, it only needs to be reduced to selenide before its incorporation in newly formed Sec.

In contrast to the rapid incorporation of Se from selenite and Sec, SeMet tends to be a "slow release" form of Se for Sec synthesis. Because protein synthesis cycles do not differentiate between SeMet and methionine (Met), SeMet tends to be nonspecifically incorporated into proteins and the rate of SeMet degradation is linked to rates of Met degradation. This results in a relatively slow rate of release of its Se for de novo Sec synthesis in animal cells. As a result, SeMet may engage in many cycles of protein synthesis as a methionine equivalent (Fig. 1) before it is eventually degraded and releases Se for Sec synthesis.

The biochemical and physiological importance of Se is primarily through the activities of Sec, the 21st proteinogenic amino acid that is cotranslationally inserted into proteins at UGA codons in coordination with SECIS (Sec

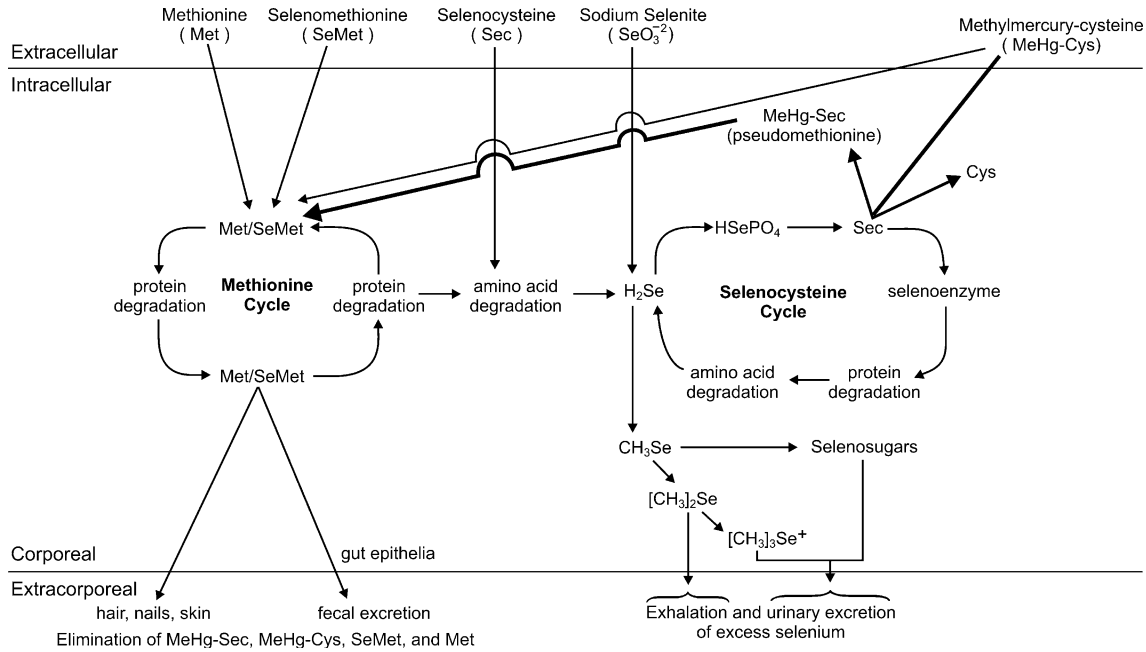

Figure 1. Metabolic cycles of selenomethionine, selenocysteine, inorganic selenium, and the proposed mechanism of MeHg dose-dependent disruption of the Se biosynthetic pathway.

insertion sequence) elements during synthesis of these unique proteins (selenoproteins). Selenoprotein occurrence and distribution vary between tissues, but most known eukaryotic selenoproteins are represented in the mammalian genome and relatively consistently between mammalian orders. Many selenoproteins have cysteine (Cys) orthologues, but the Se of the Sec in selenoenzymes performs biochemical functions that are beyond the capacity of the sulfur of Cys. Whereas the thiol of Cys is protonated at physiological pH, the Se of Sec is ionized, thus more highly interactive in its molecular environment. The redox range of Sec also is more extensive than that of Cys, enabling catalytic functions beyond the reaction potentials of Cys. The enzyme activities of the 25–30 selenoproteins distinguished by genetically directed incorporation of Sec in their primary structure are becoming increasingly well defined. All of the enzyme activities that have been characterized thus far employ the extensive redox potential of the Sec strategically positioned in their active sites to perform their catalytic functions.

Several selenoenzymes, including but not limited to, phospholipid glutathione peroxidase and the thioredoxin reductases, perform important antioxidant functions (Chen and Berry, 2003; Schweizer et al., 2004; Whanger, 2001; Kohrle, 2000) and are expressed with tissue-specific occurrence and distributions throughout the body, but seem to be particularly indispensable in the brain. Selenoprotein P, the most abundant selenoprotein in the plasma, contains 9–10 Sec/molecule, and seems to be a preferentially absorbed vehicle for Se distribution across placental and brain membrane barriers (Kasik and Rice, 1995; Hill et al., 2004). Newly described selenoenzyme functions (Ferguson et al., 2006; Novoselov et al., 2007; Dikiy et al., 2007) include redox control of an abundant class of brain proteins. Essentiality of these functions may explain why forms of animal life that possess nervous systems also selectively express and/or preferentially preserve selenoenzyme activities in brain and neuroendocrine tissues (Behne et al., 2000; Sun et al., 2001; Kohrle et al., 2005).

Mercury Interactions with Selenium

Selenium-dependent protective effects against Hg intoxication have been recognized in all forms of animal life that have been investigated (Cuvin-Aralar and Furness, 1991; Chapman and Chan, 2000). Methylmercury readily crosses placental and blood–brain barriers—in the form of a cysteine adduct (MeHg–Cys) that biochemically resembles methionine (Taylor et al., 1975) and is taken up by the LAT1 amino acid transporter (Simmons-Willis et al., 2002). This may occur because MeHg–Cys is a molecular mimic of Met (Bridges and Zalups, 2005; Aschner and Clarkson, 1989) or because amino acid transport by LAT1 tends to be non-

specific in its transport activities (George, et al., 2008). At low exposures, MeHg–Cys participates in the methionine cycle without pathological consequence; however, at higher exposures MeHg induces toxic effects. At all levels of exposure, MeHg exchanges partners to form covalent associations with chemical species of equal or greater affinities. Because the binding affinities between Se and Hg are approximately a million times greater than those between sulfur and Hg (Dyrssen and Wedborg, 1991), mass action effects support selective sequestration of Se in association with MeHg. When MeHg–Cys encounters the ionized Se of Sec at the active site of a selenoenzyme, its MeHg moiety exchanges its association with the sulfur of Cys for the higher affinity Se group of Sec, resulting in a direct exchange that results in formation of MeHg–Sec.

MeHg exposure cause diminishments to 43% of normal brain Se in weanling rats raised on low-Se diets (Ralston et al., 2008), a value well below the 60% level that may define the minimum for health. High maternal Hg exposures are known to diminish transport of Se across the placenta, diminishing the amount of Se supplied to the fetus by more than 50% (Parizek et al., 1971) and high maternal MeHg diminished fetal brain Se and brain selenoenzyme activities to ~30% (Watanabe et al., 1999a, b). Increasing exposure to MeHg will inevitably lead to increasing sequestration of Sec as MeHg–Sec. Just as MeHg–Cys appears biochemically similar to SeMet and Met, MeHg–Sec would be expected to similarly form a molecular mimic of Met. As a result of this case of mistaken molecular identity, MeHg–Sec distributions would become indistinguishable from those of Met/SeMet. Therefore, the MeHg–Sec adduct has been termed "pseudomethionine" (Ralston et al., 2008) to indicate its expected diversion into the methionine cycle (Fig. 1). The Se of pseudomethionine that is cycled into proteins indiscriminately from Met/SeMet is unavailable for participation in the selenocysteine metabolic cycle (Fig. 1). Because a portion of the Se present in blood and tissues is in the form of MeHg–Sec, the actual amounts of bioavailable Se in these cases is actually much lower than the total amount present.

Because intracellular Se is usually present in substantial excess of Hg, brain cells can normally maintain sufficient free Se to support optimal rates of selenoenzyme synthesis and activity. However, when the concentration of MeHg in the cells exceeds that of Se, the amount of biologically available Se for normal selenoenzyme synthesis diminishes. Therefore, the protective effect of supplemental Se may occur because additional dietary Se is able to offset the Se sequestered by Hg (Watanabe et al., 1999a, b) as pseudomethionine (Ralston et al., 2007, 2008) and maintain selenoenzyme activities.

The biochemical definition of an irreversible inhibitor is a molecule that covalently modifies an enzyme. In the proposed mechanism, MeHg binds with the Sec moiety at the selenoenzyme active site. Because the binding affinity between Hg and Se is extraordinarily high, MeHg is by definition a highly specific irreversible selenoenzyme inhibitor. However, the proposed inhibitor–enzyme complex not only abolishes the activity of the inhibited selenoenzyme, it also restricts Se release from the MeHg–Sec complex, severely limiting or effectively abolishing the bioavailability of the bound Se for participation in future intracellular cycles of Sec synthesis. The proposed mechanism coincides with expectations based on the preponderance of findings regarding Hg/Se interactions at the molecular, cellular, organism, and population levels. Contrary to previous expectations, Hg does not seem to cause oxidative damage directly (Seppanen et al., 2004). Instead, MeHg appears to cause increased oxidative damage as a result of inhibition of selenoenzyme activities that normally detoxify free radicals formed during normal cell metabolism. Selenium's ability to counteract toxic effects of high Hg exposures have been recognized since 1967 (Parizek and Ostadolova, 1967) and subsequent studies increasingly confirm that organic and inorganic forms of Se prevent or ameliorate otherwise toxic consequences of Hg exposure (Iwata et al., 1973; Ohi et al., 1976; Beijer and Jernelov, 1978; El-Begearmi et al., 1982; Whanger, 1992; El-Demerdash, 2001; Ralston et al., 2007, 2008). Maternal Hg exposures compromise redistribution of maternal Se to the fetus (Parizek et al., 1971; Watanabe et al., 1999a, b) and dose-dependently diminish selenoenzyme activities in fetal brain (Watanabe et al., 1999a, b; Stringari et al., 2008). However when maternal Se status is enhanced by feeding rich Se diets, diminishments in total Se and selenoenzyme activities in brains of the exposed offspring were Se dose-dependently prevented and neurological signs of fetal MeHg intoxication were alleviated (Watanabe et al., 1999a, b).

Studies demonstrating Se-dependent amelioration of MeHg toxicity have included Se from yellowfin tuna (Ohi et al., 1976; Ganther et al., 1972), menhaden (Stillings et al., 1974), swordfish (Freidman et al., 1978), and rockfish (Ohi et al., 1980). Therefore, the organic forms of Se present in ocean fish are bioavailable and effective in counteracting MeHg toxicity. It is important to note that the protective effects of Se are less obvious when slow-release forms of Se, such as SeMet, are used (Beyrouty and Chan, 2006) or

when Hg:Se molar ratios remain disproportionate, particularly during fetal exposures. Studies of maternal MeHg exposure need to pay particular attention to blood Hg:Se molar ratios because the developing fetus is entirely dependent on uninterupted delivery of Se across the placenta. In maternal exposures, it seems that Se supplementation cannot counteract adverse outcomes from MeHg exposures if the total dietary Hg:Se ratio is greater than 1:1. As these ratios are approached, the biovailabilities and rate of Se release from food sources become increasingly important aspects. Animal studies demonstrate that dietary Hg:Se ratios will need to be significantly lower than 1:1 to ensure that maternal export of Se to the fetus is unimpaired to prevent adverse neurodevelopmental outcomes in the offspring (Newland et al., 2006; Reed et al., 2006; Reed et al., 2008). High maternal exposures to MeHg causes increased oxidative damage as measured by F_2-isoprostane levels in fetal brain (Stringari et al., 2008), apparently as a consequence of Hg dose-dependent diminishments in brain selenoenzyme activities (Watanabe et al., 1999a, b; Seppanen et al., 2004; Stringari et al., 2008). Prenatal MeHg exposure did not only diminish selenoenzyme activities during fetal development. Although Hg contents in brains of prenatally exposed mice had diminished to near basal levels by postnatal day 21, selenoenzyme activities remained diminished, indicating that prenatal exposure to toxic amounts of MeHg had lasting effects (Stringari et al., 2008).

Although it is clear that MeHg dose-dependently affects selenoenzyme activities and Se dose-dependently counteracts development and reversal of signs of MeHg toxicity, it currently remains unknown whether selenoenzyme inhibition/Se-sequestration are the exclusive causes of MeHg toxicity, or whether other molecular mechanisms also contribute to its pathological syndrome. However, because Se-dependent therapeutic reversal of MeHg-dependent growth inhibition, cessation of declines in motor function, and prevention of lethality were virtually equivalent regardless of whether toxic dietary MeHg was continued (Ralston 2008, unpublished data, 2008), any other effects of MeHg toxicity would be expected to be relatively minor compared with etiologies secondary to selenoenzyme inhibition/Se-sequestration mechanisms.

Selenium Health Benefit Values

To simplify assessment of Se-specific nutritional benefits in relation to potential MeHg exposure risks associated with seafoods, the Se Health Benefit Value (Se-HBV) was proposed by Kaneko and Ralston (2007). The Se-HBV incorporates consideration of both the absolute and the relative amounts of Se and Hg in the diet to provide an index that is easily interpreted. The sign of the calculated Se-HBV indicates the expected health benefits (if positive values are obtained) or health risks (if negative values result) and the magnitude of the obtained values are proportional to the expected benefits or risks. Because it is vitally important to evaluate the Hg:Se stoichiometric relationship, molar concentrations (μmol/kg) are used in the Se-HBV calculation. The Hg:Se and Se:Hg molar ratios are calculated by direct division of the individual molar concentrations, and respectively multiplied times their absolute molar concentrations as follows:

$$\text{Se-HBV} = [(\mu\text{mol Se/kg}) \times (\text{Se/Hg})] - [(\mu\text{mol MeHg/kg}) \times (\text{Hg/Se})]$$

The current iteration of the Se-HBV uses total Hg/Se and Se/Hg molar ratios because total elemental concentrations are more readily available. However, a more sophisticated evaluation can be achieved through use of MeHg instead of total Hg and Sec instead of total Se. In fish fillets, the MeHg concentrations usually tend to represent the bulk of total Hg, but there are instances in which this is not the case. In the case of Se, the total Se in animal proteins includes a significant fraction as SeMet. Although SeMet is well absorbed and readily incorporated into proteins, its far slower rate of release of inorganic Se makes it a long-term Se source, but Se for synthesis of new Sec is more readily available from inorganic forms of Se or Sec in the diet. The degradation rates of SeMet may need to be integrated to enable a time-dependent SeMet Se-release factor to be included in more exacting versions of the equation.

Human Studies

Concurrent maternal exposure to potentially harmful effects of toxicants and beneficial effects of essential nutrients in seafoods is a classic example of statistical confounding. Because both occur together in seafoods, but affect developmental outcomes in opposing directions, it is essential to address potential confounding effects by identifying and differentiating the discrete adverse effects caused by MeHg exposure in distinction from the beneficial effects of the increasing nutrient intakes. Otherwise, these confounding effects will inexorably, although inadvertantly, bias the data

and diminish the statistical robustness and the reliability of the findings. Although the findings of the major epidemiological studies appear to conflict with one another, it is only because the conventional hypothesis, "Maternal MeHg exposures are directly associated with adverse child development outcomes" (Hypothesis 1) does not include consideration of Se. From the more informed perspective that incorporates consideration of Se physiology, the updated version of the hypothesis states, "Maternal MeHg exposures *in excess of Se intakes* are directly associated with adverse child development outcomes" (Hypothesis 2). The current evaluation compares the findings of the human epidemiological studies as tests of the conventional and updated hypothesis.

A number of epidemiological studies of the effects of maternal MeHg exposure on child development outcomes are presented below in their approximate chronological order. Basic descriptions of the study and whether the findings support hypothesis 1 and hypothesis 2 are provided along with characterization of the dietary Se-HBV of the MeHg exposure sources for each study. The results of these descriptions are collected and reported in Table 1 for ease of comparison.

Minamata Bay, Japan

Widespread MeHg poisoning occurred in Minamata, Japan, during the late 1950's. After an industrial plant had discharged many tons of Hg waste directly into the waters of a small bay, MeHg bioaccumulated to extremely high levels in ocean fish that occupied the bay. Because these fish were a major food source for the local population, >2,000 cases of MeHg poisoning developed. Acute MeHg poisoning was characterized by gross disturbance of the central nervous system, which was lethal in some cases, whereas others became comatose and/or developed permanent disabilities with severe symptoms resulting from widespread brain damage. In cases with less severe poisoning, symptoms included motor control and sensation disturbances (Takeuchi and Eto, 1999). Patients who died displayed severe brain atrophy, cerebral and cerebellar lesions, and pathologic changes in cytoarchitecture. Symptoms of MeHg poisoning were characterized by silent latency. In some cases, symptoms did not appear until more than 5 years after high MeHg exposure ceased. Awareness of fetal sensitivity to MeHg neurotoxicity arose when children of asymptomatic mothers displayed disturbances in motor control, mental retardation, and related symptoms. Brain pathology of congenital MeHg poisoning included cortex atrophy/hypoplasia and dysmyelination of the pyramidal tract. In the cerebellum, hypoplasia and degeneration of the granular cell layer and other layers were observed (Harada, 1968). Fish in Minamata Bay were heavily contaminated with MeHg, occurring in concentrations up to ~250 μϖ mol/kg; ~50 mg Hg/kg in the meats of certain fish (Takeuchi and Eto, 1999). Although Se was not measured

Table 1. Hypothesis testing

Study location	Hypothesis 1[a]				Hypothesis 2[b]			
	Development outcomes[c]	Study supports	Does not support	Study conflicts	Calculated ~Se-HBV[d]	Study supports	Does not support	Study conflicts
Minamata	Harmed	Yes	No	No	144 to −5,000	Yes	No	No
Iraq	Harmed	Yes	No	No	−800	Yes	No	No
New Zealand	Harmed	Yes	No	No	144 to −123	Yes	No	No
Faroe Islands	Harmed	Yes	No	No	40 to −83	Yes	No	No
Peru	Not harmed	No	Yes	No	84	Yes	No	No
Seychelle Islands	Not harmed	No	Yes	No	173	Yes	No	No
United Kingdom	Benefitted	No	Yes	Yes	202	Yes	No	No
United States	Benefitted	No	Yes	Yes	144	Yes	No	No
Denmark	Benefitted	No	Yes	Yes	202	Yes	No	No

[a]Hypothesis 1: maternal MeHg exposures are directly associated with adverse child development outcomes.
[b]Hypothesis 2: maternal MeHg exposures *in excess of Se intakes* are directly associated with adverse child development outcomes.
[c]Effects of maternal seafood (MeHg) exposure on child development outcomes.
[d]Approximate Selenium Health Benefit Values of MeHg-containing food sources were calculated using best available data.

in fish from Minamata Bay, it has been established that Se levels in fillet portions from commonly consumed varieties of Pacific Ocean fish range between 5 and 20 μmol Se/kg (Kaneko and Ralston 2007), and in species other than blue marlin, Se contents of fillet do not increase with increasing MeHg levels (Ralston and Kaneko, unpublished data, 2008). The Se content of Pacific Ocean fish other than blue marlin assessed in that study were 9.77 (95% confidence interval (CI), 6.99–12.55) μmol Se/kg. Using this mean value as an approximation of the amount of Se expected to have been present in these fish, the calculated Se-HBV for fish from Minamata Bay would be estimated to have been −5,000 (Table 1). Ocean fish that had only recently entered the bay before being caught would have had much lower MeHg contents and would have retained positive Se-HBVs until they had bioaccumulated significant amounts of MeHg. The intermittent presence of Se-rich fish would have delayed onset of MeHg toxicity and extended the silent latency period before onset of clinical symptoms in this population. Harmful child development outcomes tended to occur in direct association with increasing MeHg exposure. Therefore, the findings of this study support Hypothesis 1. Because the Se-HBVs of the MeHg source were extremely negative, the observation of these severely harmful effects also are consistent with Hypothesis 2.

Iraq Outbreak

In 1971, MeHg-treated seed grain was imported into Iraq during a time of famine. Because there was a food shortage, the seed grain was baked into bread and consumed by malnourished Iraqi villagers. There are no reliable estimates of how many developed toxicity, but within 2 months, there were thousands of hospital admissions and hundreds of hospital deaths from MeHg ingestion. Children of pregnant women exposed in utero manifested severe motor and sensory impairments and delayed mental development (Amin-Zaki et al., 1974). MeHg dose-dependent relationships on adverse effects in offspring exposed in utero are described in a series of reports by Amin-Zaki et al. (1974, 1976, 1979) and Marsh et al. (1980, 1987). Symptoms closely resembled those recorded in Minamata, Japan, but the period of latency was far shorter. Adults developed paresthesia after a latent period of only 16 to 38 days after initiation of exposure. Those who were severely afflicted developed ataxia, motor, and sensory difficulties. Lethality resulted from failure of the central nervous system (Bakir et al., 1973). Concentrations of MeHg in the wheat flour ranged from 4.8–14.6 (mean, 9.1) μg/g (∼45 μmol Hg/kg). The Se content of wheat varies depending on the Se content of the soil in which it is grown, and it was not measured in the wheat consumed in Iraq. However, it is reasonable to assume that it would have been in the medium range of ∼0.2 μg/g (∼2.5 μmol Se/kg) for the sake of the current approximation. Using these values, the calculated Se-HBV of the bread consumed in Iraq at the time of the disaster would have been in the range of −800. The findings of this study support Hypothesis 1, because adverse effects on children generally increased in severity in direct association with increasing maternal MeHg exposure. Because the Se-HBV of the MeHg source was in the harmful range, these increasingly adverse effects also are consistent with Hypothesis 2.

Peru

A study (Marsh et al., 1995) of maternal MeHg exposure on child development outcomes was conducted in Mancora, Peru, between 1981 and 1984. The study site was selected because marine fish were a large source of dietary protein, resulting in high maternal MeHg exposures. Participants consisted of 131 mother-infant pairs analyzed for hair MeHg content. The geometric mean hair level was 7.05 (range, 0.9–28.5) ppm. The peak maternal hair MeHg levels during pregnancy ranged from 1.2 to 30 (geometric mean, 8.3) ppm. This study found no relationship between MeHg exposures and measures of infant development or neurological signs. The authors suggested that because marine fish contain Se, the toxicity of MeHg was reduced and prevented adverse neurological consequences. The mean Se-HBV for ocean fish consumed by the population of Mancora is estimated at approximately 84, using the mean values established by Kaneko and Ralston (2007) for the varieties of Pacific ocean fish expected to have been consumed by this population. Because no harms were noted in association with increasing MeHg exposure, the findings of this study do not support Hypothesis 1. However, because the Se-HBV of the MeHg source was in the beneficial instead of the harmful range, no harms would have been expected. Therefore, the results are consistent with Hypothesis 2.

New Zealand

The effect of MeHg on children exposed in utero from maternal fish consumption was studied between 1982 and

1983. Of ~11,000 mothers who were initially questioned, less than 1,000 had consumed fish more than three times per week during 9 months of pregnancy. Maternal hair Hg concentrations were assessed in these groups and were used as a means of selection. Higher hair Hg during pregnancy was consistently associated with decreased performance in tests of neurodevelopment. The authors concluded that MeHg exposure led to developmental delays and deficits in psychological tests. Crump et al. (1998) performed a reanalysis of the results and no associations between MeHg exposure and children's test scores were identified, unless the results from one child whose mother's hair Hg level was four times higher than for any other mother was omitted. When that outlier was omitted, multiple tests were dose-dependently associated with maternal hair MeHg concentrations. The seafood consumed in this study was primarily "fish and chips" prepared from varieties of fish, including shark. The MeHg contents of shark meats collected from the seas surrounding New Zealand can be very high: average 0.72 (ranging up to 4.4) mg Hg/kg; 3.58 and 22 μmol Hg/kg, respectively (Mitchell et al., 1982). Because Se in shark meats do not increase in the presence of increasing MeHg (Kaneko and Ralston 2007), the ~Se-HBV calculated for a shark with 22 μmol Hg/kg would be –123. Normal varieties of ocean fish with Se-HBVs estimated at approximately 84 (Kaneko and Ralston 2007) would also be expected to have been consumed by this population. It is important to recognize that the New Zealand population had a notoriously poor Se status at the time of this study (Robinson 1988), a factor that would have greatly accentuated their vulnerability to MeHg exposure (Ralston et al., 2008). The findings of this study are consistent with Hypothesis 1, and, because sharks with highly negative Se-HBVs were consumed, the harms noted are also consistent with Hypothesis 2.

Seychelles Islands

The first Seychelles study consisted of a cohort of 779 mother–infant pairs that were selected between 1989 and 1990. This population eats on average ~12 fish meals per week, which is far more than what is typical in the United States. However, no negative effects were found in association with high fish consumption and MeHg exposure (Davidson et al., 1998; Myers et al., 1998, 2000). Average maternal hair Hg levels during the entire pregnancy (range, 0.5–26.7 (median, 5.9) ppm) were used as the marker of fetal Hg exposure. The cohort was evaluated at ages 6.5, 19, 29, and 66 months (Marsh et al., 1995; Myers et al., 1995) and at age 9 years (Myers et al., 2003). Children were repeatedly assessed using multiple sensitive standardized measures of cognitive development and neurological endpoints. Using maternal hair-Hg levels as the independent variable, prenatal MeHg exposure from fish consumption in Seychellois children was not associated with adverse effects. The Seychelles study found that no adverse neurological outcomes in Seychellois children during a 9-year period were associated with prenatal MeHg exposures from maternal consumption of ocean fish (Myers et al., 2000, 2003). A recent report from this population suggests that MeHg effects on psychomotor development are detectable after controlling for polyunsaturated fatty acids (Davidson et al., 2008; Strain et al., 2008) but did not indicate that harmful effects accompanied fish consumption. To the contrary, certain developmental outcomes of prenatally exposed children indicated beneficial effects that correlated with Hg exposures during pregnancy. The authors indicated that the presence of micronutrients in fish provided a plausible explanation for these findings (Clarkson and Strain 2003). The fish consumed in the Seychelles have been assessed for both Hg and Se contents. The average of the observed Hg and Se concentrations in these 16 fish species were 0.34 ± 0.23 μmol Hg/kg and 3.73 ± 1.64 μmol Se/kg (mean ± 95% CI). The mean and 95% CI of the Se-HBVs for these 16 fish species was 173 ± 148 for the various fish types. Because no harms were noted in association with increasing MeHg exposure, the findings of this study do not support Hypothesis 1. However, because the Se-HBV of the MeHg source was in the beneficial instead of the harmful range, benefits instead of harms would have been expected, therefore, the results do support Hypothesis 2.

Faroe Islands

The Faroes Islands studies examined the influence of MeHg exposure from maternal consumption of pilot whale and cod fish on child development outcomes. Seafood constitutes a major part of the diet in the Faroe Islands (Grandjean et al., 1992a, b); >90% of their MeHg exposure arises from consumption of pilot whale, and the majority of dietary Se is from consumption of codfish (Grandjean et al., 1997). Increased Hg in maternal hair and umbilical cord blood were directly related to maternal consumption of pilot whale. At age 12 months, 583 children were evaluated for developmental milestones. Infants who reached milestone criteria early had significantly higher hair Hg than those who did

not. Neurobehavioral tests were used to assess 914 children at age 7 years (Dahl et al., 1996). Higher prenatal MeHg exposure was associated with difficulties on cognitive measures (Grandjean et al., 1997, 1998). The most pronounced difficulties were in the areas of language, attention, and memory, and to a lesser extent sensory and motor functions. The codfish eaten in the Faroes contain small amounts of MeHg, are good sources of Se (Hall et al., 1978), and have a reasonably good Se-HBV of 18 as a result. In contrast, the pilot whale meats consumed in the Faroes are highly contaminated with Hg. Using data from the 1977 collection study (Julshamn et al., 1987) cited by Grandjean et al. (1992a), pilot whale meat contained 3.3 mg/kg, 16.45 μmole Hg/kg, blubber contained 0.7 mg/kg, 3.49 μmole Hg/kg, and kidney contained 18 mg/kg, 89.74 μmole Hg/kg. The average Se concentrations in the meat, blubber, and kidney were 3.17, 1.52, and 16.46 μmole Hg/kg, resulting in Se-HBVs of −85, −7, and −486 respectively, in these tissues. Kidney was being distributed along with meat and blubber throughout the Faroes at the time of the study, but it is not clear whether, or how much, kidney was actually consumed. For this reason, only the cod and whale meat Se-HBVs are listed in Table 1. Pilot whale blubber has unusually high contamination levels with polychlorinated biphenyls (PCBs) resulting in high exposures in the Faroese population, which may present further confounding factors. However, the study authors concluded that they did not recognize effects from the concomitant PCB exposure that indicated any influence on Hg-associated effects.

Most interestingly, the adverse effects that were dose-dependently associated with cord blood Hg were equally proportional to cord blood Hg:Se molar ratios (P. Grandjean personal communication, 2008) in an analysis performed in association with, but not reported in, the article by Choi et al. (2008). Blood Hg:Se molar ratios approached and exceeded 1:1 molar stoichiometry in the cord bloods of children most affected by Hg exposure. Because the association between MeHg toxicity and blood Hg:Se molar ratios has been observed to be more reliable than associations based on blood Hg (Ralston et al., 2008), it seems likely that using cord blood Hg:Se molar ratios will provide a superior basis for analysis of outcomes. This is in sharp contrast to what is expected in populations exposed to MeHg exclusively from ocean fish. It is not expected that cord blood Hg:Se molar ratios will approach 1:1 molar ratios, but should generally be less than 1:3 or 1:5 in most populations (Ralston unpublished data, 2008). The findings of this study support Hypothesis 1 because the adverse effects on child outcomes increased with increasing maternal MeHg. Because the adverse effects also increased in proportion to Hg:Se molar ratios, and the Se-HBV's major source of maternal MeHg exposure was negative, the findings also are completely consistent with Hypothesis 2.

United Kingdom

The Avon Longitudinal Study of Parents and Children (ALSPAC) study examined rates of ocean fish consumption by 11,875 women during pregnancy and assessed effects on developmental, behavioral, and cognitive outcomes in their children from aged 6 months to 8 years (Hibbeln et al., 2007). The study was adjusted for 28 potential confounders to eliminate potential influences of social distinctions. Children of mothers who consumed little or no seafood per week had an increased risk of being in the lowest quartile for verbal intelligence compared with children of mothers who ate more than the EPA recommended amount of seafood. Low maternal seafood consumption was associated with increased risk of suboptimum outcomes for social behavior, fine motor, and communication scores. For each outcome, the lower the mother's intake of seafood was during pregnancy, the greater the risk of suboptimum development in their children. The children of mothers who had not consumed ocean fish were estimated to be at a ∼6 IQ point disadvantage relative to children whose mothers had consumed more than 340 g of fish per week (J. Hibbeln personal communication, 2008). The average Se-HBV for the varieties of white fish (87), oily fish (288), and shell fish (233) are uniformly beneficial, with a group mean of ∼202 for seafoods consumed in the United Kingdom (Ralston unpublished data, 2008). The study's findings that increasing maternal seafood consumption (and increasing MeHg exposure) resulted in improved child outcomes do not support, but directly conflict with, Hypothesis 1. Because seafood have positive Se-HBVs, benefits rather than harm would be expected, and therefore the results are in accord with Hypothesis 2.

World Trade Center (United States)

Prenatal exposure to elemental Hg vapor evolving from debris remaining after the World Trade Center (WTC) disaster was assessed in relation to effects on fetal growth and child development (Lederman et al., 2008). Maternal and umbilical cord blood total Hg of 329 women who delivered at term in lower Manhattan after September 11, 2001 were

assessed. Cord and maternal blood Hg levels were not higher for women residing or working within 1 or 2 miles of the WTC compared with women who lived and worked further away. Cord blood Hg levels were more than twice maternal levels, and blood Hg levels in cord and maternal blood samples were both higher in women who reported eating fish/seafood during pregnancy. Log cord Hg was inversely associated with full IQ scores in the children (b = − 3.8, $p = 0.002$) at age 48 months after controlling for fish/seafood consumption and other confounders. However, fish/seafood consumption during pregnancy was associated with a 5.6-point increase in Verbal and Full IQ scores. The ocean fish consumed in this study would be expected to conform with the Se-HBV of 144 calculated as the average for U.S. fish (Ralston unpublished data, 2008). The study found that maternal seafood consumption (and greater MeHg exposure) was associated with improved child outcomes. Therefore, the findings of this study do not support, but directly conflict with, Hypothesis 1. Because the seafood have positive Se-HBVs, benefits rather than harm were expected and were seen. Therefore, the results of this study are in accord with Hypothesis 2.

Denmark

The Danish National Birth Cohort prospective population-based cohort study examined rates of ocean fish intake by 25,446 women (Oken et al., 2008). Mothers reported child development through a standard interview, which was used to generate developmental scores at aged 6 and 18 months. Higher maternal fish consumption was associated with improved child development (odds ratio, 1.29; 95% CI, 1.2–1.38) for the highest vs. lowest quintile of fish intake) in 18 month old children and similar results were observed at 6 months of age. The authors conclude that maternal fish consumption during pregnancy was associated with improved early child development, rather than harmful effects. The fish consumed in Denmark would be expected to be the same or similar to those consumed in the United Kingdom with a calculated average Se-HBV of 202 (Ralston unpublished data, 2008). The study's findings that increasing maternal seafood consumption (and increasing MeHg exposure) improved child outcomes do not support, but directly conflict with, Hypothesis 1. Because the seafood consumed had positive Se-HBVs, benefits rather than harm were expected and were seen. Therefore, the results of this study are in accord with Hypothesis 2.

Discussion

The most important aspect of the Se-HBV is the positive or negative sign before the value. Foods with a negative sign contain Hg in excess of Se and should be completely avoided during pregnancy. If the Se-HBV of a type of fish has a positive sign, maternal consumption should be encouraged rather than limited. The more positive the value, the more beneficial the expected effects, and the more negative the value, the more harmful. The relationships shown in Table 1 suggest that the Se-HBV is a seafood safety indicator that is consistent with the results of the existing human and animal studies, and along with the preponderance of data available from animal studies, demonstrate the importance of dietary Se in counteracting the potential pathological effects of high MeHg exposures.

Further factors that need to be considered when assessing potential effects of maternal fish consumption include the form and amounts of other important nutrients, such as omega-3 fatty acids in the seafoods consumed. It is conceivable that future iterations of the Se-HBV will incorporate omega-3/omega-6 ratios (as well as other nutrient relationships) and simply become Health Benefit Values. Furthermore, the interactions between Se and other nutrients in the background diet of the exposed population are very significant. Vitamins C and E (May et al., 1998; Beyrouty and Chan 2006) are both involved in Se-dependent redox cycling, and the abundance of these nutrients should be considered when evaluating the importance of dietary Se from fish consumption. The greatest benefits of maternal seafood consumption on child outcomes would be expected to be observed when maternal Se-status is raised from low to normal, but dietary benefits also increase as dietary Se-status increases toward Se-rich (Rayman 2000).

Food safety criteria need to incorporate more sophisticated approaches to evaluating risks related to MeHg exposure from maternal consumption of ocean and fresh water fish. If not the Se-HBV, then some other means of more accurately evaluating risk associated with MeHg exposure from fish consumption needs to be developed to incorporate consideration of Se contents and Hg:Se ratios. Decision makers and agencies responsible for protecting public health need research information that will enable them to provide advice that not only protects the public from harm, but also improves maternal nutrition to optimize child development. Risk evaluations associated with

maternal MeHg exposure need to concurrently determine the relative and absolute amounts of MeHg and Se present in these food sources and employ these as components of their assessments. Until these steps are taken, studies reporting Hg levels in fish and blood of populations that eat fish and without concurrently measuring Se will simply be reporting MeHg exposures, but not providing any meaningful indications of associated risks.

In 2005, Trasande and colleagues suggested that the subtle loss of IQ that they attributed to MeHg exposure from ocean fish consumption (instead of pilot whale consumption) resulted in a cost of $8.7 billion per year for harms due to loss of ~0.1 IQ points per child for ~450,000 exposed children. As we now understand the issue, mothers with elevated blood Hg levels seem to represent the fraction of mothers who actually ate enough seafood to provide IQ benefits to their children. Assuming that the $8.7 billion estimate of Trasande and colleagues was correct, applying their results to the loss of 5.6 IQ points (Lederman et al., 2008) per child for ~3,500,000 children whose mothers did not eat robust amounts of fish, the harms to the U.S. economy from inadequate fish consumption during pregnancy would be expected to result in losses of tens of trillions of dollars to the nation per year.

It is essential to note that MeHg exposures that are unlikely to cause harm in populations that eat Se-rich ocean fish may still be harmful among populations exposed to MeHg without a rich dietary source of Se. Because the Faroes population consumed large amounts of ocean cod, their Se status was very good. However, using this Se-rich population as an indication of risks of MeHg exposure may not provide an accurate estimate of risks of MeHg exposures in populations with a low Se status. The Se levels in freshwater fish are exclusively dependent on the status of their waters of origin. Therefore, the Hg:Se ratios in freshwater fish will be far more variable and instances of disproportionately high MeHg concentrations relative to Se may be characteristic of top predator fish from Se poor regions (Luten et al., 1980; Peterson et al., 2009). Therefore, populations that consume fish from lakes in low Se regions may be at greater risk than is currently anticipated.

The prime directive of professionals in all areas of public responsibility should be, "Primum non nocere" (First, do no harm). Risk assessments based on neurodevelopmental harms from maternal consumption of pilot whale and shark meats with disproportionately high Hg:Se molar ratios (~5:1) and highly negative Se-HBVs seem to have resulted in gross overestimates of the risks associated with MeHg exposure from eating ocean fish. The unbalanced approach of only examining risks resulted in regulatory advisories that emphasized restrictions to minimize MeHg exposure, but overlooked the more substantial positive effects of ocean fish consumption during pregnancy. Maternal consumption of typical seafoods with highly positive Se-HBVs seems not only to be harmless but remarkably beneficial to healthy child development. Exaggerations of the risks of MeHg exposure have caused women to avoid eating ocean fish during pregnancy. Following such misguided advice may have inadvertently caused far more damage to children than the worst possible risks that were supposedly being avoided.

To resolve this issue, it seems that using the Se-HBV or a similarly balanced criterion for assessing seafood safety will alleviate many current misunderstandings of advisories regarding maternal seafood consumption. Using the Se-HBV as an environmental indicator that distinguishes health-promoting from hazardous fish will provide a basis for developing urgently needed regulatory protections against hazardous Hg:Se ratios that may be present in freshwater fish. Seafood safety criteria that include a balanced consideration of nutrients and toxicants will protect and improve public health by properly limiting hazards while encouraging maternal consumption of seafoods that enhance development of healthy children.

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EcoHealth

Special Feature: Methylmercury

Review

Methylmercury in Marine Ecosystems: Spatial Patterns and Processes of Production, Bioaccumulation, and Biomagnification

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Abstract: The spatial variation of MeHg production, bioaccumulation, and biomagnification in marine food webs is poorly characterized but critical to understanding the links between sources and higher trophic levels, such as fish that are ultimately vectors of human and wildlife exposure. This article discusses both large and local scale processes controlling Hg supply, methylation, bioaccumulation, and transfer in marine ecosystems. While global estimates of Hg supply suggest important open ocean reservoirs of MeHg, only coastal processes and food webs are known sources of MeHg production, bioaccumulation, and bioadvection. The patterns observed to date suggest that not all sources and biotic receptors are spatially linked, and that physical and ecological processes are important in transferring MeHg from source regions to bioaccumulation in marine food webs and from lower to higher trophic levels.

Keywords: methylmercury, bioaccumulation, trophic transfer, biomagnification, bioadvection

Introduction

Mercury (Hg) is a pervasive and toxic environmental contaminant (U.S. EPA, 1997). Human exposure to Hg occurs primarily through the consumption of marine fish (Fitzgerald and Clarkson, 1991; U.S. EPA, 2002). The organic form of Hg, methylmercury (MeHg), is of greatest concern because it is a potent neurotoxin (Salonen et al., 1995; Grandjean et al., 1997; Sorensen et al., 1999) and because it accounts for >95% of the Hg in fish (Bloom, 1992; Bank et al., 2007; Sunderland, 2007). Fish are commercially harvested from all over the globe including ecosystems distant from major anthropogenic sources of Hg pollution. This raises important scientific and policy questions regarding these sources of MeHg in the fish and shellfish (Chen et al., 2008).

Biogeochemical, physical, and ecological processes govern the distribution and movement of MeHg from source regions to bioaccumulation in marine food webs. There is great spatial variability in the concentrations of Hg

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across ocean basins (Lamborg et al., 2002; Hammerschmidt and Fitzgerald, 2004; Laurier et al., 2004; Sunderland and Mason, 2007) and in the distribution of biogeochemical conditions favoring Hg methylation. However, the MeHg concentrations found in commercially harvested fish are an integration of feeding in deep and shallow waters across inshore and offshore habitats (Block et al., 2005; Hammerschmidt and Fitzgerald, 2006a). Moreover, commercially harvested fish come from all regions of the world making it particularly difficult to link human exposure to specific fisheries and sources of MeHg (Sunderland, 2007). Given its importance to human and environmental health, it is critical to understand the spatial distribution of MeHg production and fate in marine ecosystems.

The objective of this article is to consider spatial patterns and mechanistic processes of MeHg production, bioaccumulation, and biomagnification across marine ecosystems from estuarine to coastal to open ocean. We discuss: 1) estimates of global sources and sinks of MeHg; 2) processes controlling Hg methylation, bioaccumulation, and transfer from estuaries to the continental shelf; and 2) latitudinal patterns of Hg bioaccumulation and biomagnification in marine food webs.

Scaling Global Sources and Fluxes of MeHg

On a global scale, the majority of methylated Hg found in the ocean is formed within the system from available inorganic Hg(II). Methylated Hg (both mono- and dimethyl- forms) represents a small percentage of total Hg in external sources including precipitation ($<0.2\%$ in coastal/open ocean; Bloom and Watras, 1989; Mason et al., 1992, 1997; Holz et al., 1999; Lamborg et al., 1999) and riverwater (ca. 2%; Coquery et al., 1997; Balogh et al., 1998; Benoit et al., 1998; Hurley et al., 1998; Choe and Gill, 2003; Balcom et al., 2004). When combined with estimates for the global precipitation and riverine flux of total Hg (THg; e.g., Mason et al., 1994; Cossa et al., 1996), these percentages suggest a total external flux of <0.04 Mmol MeHg y^{-1}.

External inputs of MeHg are not enough to account for our estimates of the pool of MeHg in phytoplankton in the world's oceans, thus internal sources of MeHg must dominate. For example, phytoplankton (as represented by oceanic suspended particulate matter) has been measured to contain about 6 ng MeHg g^{-1} C (Topping and Davies, 1981; Mason and Fitzgerald, 1991). As global primary production is 43.5 Pg C y^{-1} (Behrenfeld and Falkowski, 1997), it appears that at least 1.3 Mmoles y^{-1} of MeHg must be supplied to the phytoplankton pool to explain the concentrations observed. This suggests that methylation of Hg within the ocean must exceed external sources by >30-fold and dominate as a source of MeHg in the ocean.

In the rest of this section, we consider internal sources of MeHg and ask whether any one particular biogeochemical process or source region may lead to more MeHg production or bioaccumulation on a global scale than another. The potential sources considered here include: 1) methylation in marine sediments; 2) methylation in or near submarine hydrothermal systems; and 3) methylation in the pelagic water column. A summary of these estimated flux strengths and the approach used to make them is shown in Table 1. The flux of MeHg from sediments is estimated to be 8% of the total Hg load (Fitzgerald et al., 2007), estimated in different ways for the various ocean locations. For example, coastal total Hg loadings were taken to be similar in areal flux as that occurring in Long Island Sound or Chesapeake Bay (~ 0.3 μmole Hg m^{-2} y^{-1}; Mason et al., 1999; Balcom, et al., 2004), and applied to an area equivalent to 1% of total ocean area. The continental shelf/slope region was recently studied by Hammerschmidt and Fitzgerald (2006b) where they determined an average total Hg concentration in shelf sediments to be 0.1 nmole Hg gdw^{-1}, which may then be scaled up by assuming sedimentation rates of 480 g m^{-2} y^{-1} (Bothner et al., 1981) and shelf/slope (<1000 m depth) area of 12% of total ocean area (0.24 Mmole y^{-1}). For deep-sea contributions, the 8% rule was applied to a total Hg load of 2 Mmole y^{-1}, estimated in a global model (Lamborg et al., 2002). The methylation of Hg associated with seawater circulation in submarine hydrothermal systems was estimated by multiplying measured fluid concentrations by estimates of water circulation (Elderfield and Schultz, 1996; Lamborg et al., 2006), suggesting an upper estimate of 0.4 Mmole y^{-1}. This is an upper end estimate because the highest rate of water circulation (1 Sv) was used, and the true flow is likely to be much less. Finally, the rate of methylation within the oceanic water column was estimated by requiring that it balance the other sources of MeHg to the upper ocean (from shallow sediments, primarily) and the loss of MeHg in sinking particle fluxes, estimated by applying the ratio of MeHg to THg in particles to the modeled THg flux of 9 Mmole y^{-1} (Topping and Davies, 1981; Mason and Fitzgerald, 1991; Lamborg, et al., 2002).

Table 1. Estimates for the MeHg production strength of various regions in the ocean

Location	Estimated MeHg Hg flux (Mmole y^{-1})	Estimation method	References
Sediments	0.5[a]	8% of total Hg load	Fitzgerald et al., 2007
Coastal	0.06	~1 Mmole y^{-1} total Hg loading (1% of ocean area, loadings like Long Island Sound and Chesapeake Bay	Mason et al., 1999; Balcom et al., 2004
Shelf/slope	0.2	~2 Mmole y^{-1} total Hg loading (12% of ocean area, loadings according to references at right)	Hammerschmidt and Fitzgerald, 2006a; Fitzgerald et al., 2007
Deep sea	0.2[b]	~2 Mmole y^{-1} total Hg loading	Lamborg et al., 2002
Hydrothermal fluids	<0.4[b]	Measured fluid Hg concentrations multiplied by global fluid flow estimates.	Elderfield and Schultz, 1996; Lamborg et al., 2006
Water column	0.4	Measured particulate Hg concentrations multiplied by global estimates of particle flux	Topping and Davies, 1981; Mason and Fitzgerald, 1991; Lamborg et al., 2002
Bioaccumulation (for comparison)	0.2–1.3	Estimates of fish Hg accumulation as well as phytoplankton accumulation	Topping and Davies, 1981; Mason and Fitzgerald, 1991; Behrenfeld and Falkowski, 1997

[a]This value is the sum of the coastal, shelf, and deep-sea fluxes.
[b]Both of these fluxes are delivered largely to the deep sea, where current data suggest concentrations of methylated Hg species that are too low to be consistent with this input, implying net demethylation in the deep sea if these inputs are accurate.

To the first order, the various estimates suggest that the internal sources of MeHg production are of roughly comparable size, and that combined they provide about the right amount of this species to balance the annual biological uptake rates. It is likely, however, that they are not equivalent in the fraction of the flux that is anthropogenically mobilized, with the shallower water pools containing a larger percentage of "pollution" Hg (e.g., Lamborg et al., 2002; Kraepiel et al., 2003; Sunderland and Mason, 2007). Moreover, each of these sources, although incompletely understood at present, will have different bioaccumulation and food web pathways to various fish species. For example, although deep-sea sources of MeHg to the ocean could be significant (ca. 0.2 Mmole y^{-1}), extremely low biomass and densities of marine organisms in the deep oceans, and limited vertical migration of deep-sea biota and slow rates of vertical water mixing, reduces the likelihood of significant MeHg transfer to shallower food webs (e.g., Fitzgerald et al., 2007). Kraepiel and colleagues (2003) suggested, however, that deep-sea sources of MeHg determined bioaccumulation rates in the surface based on a time trend analysis of Hg content in tuna, but temporal data needed to rigorously test their hypothesis is lacking. It appears more likely that water column and shallow sediment sources supply most of the MeHg accumulating in fisheries.

Processes Controlling MeHg Production, Bioaccumulation, and Trophic Transfer

Hg Transformation in Coastal Sediments

Estuarine and coastal sediments are repositories for Hg where substantial external inputs from direct atmospheric deposition and riverine input are deposited and recycled (Stordal et al., 1996; Turner et al., 2001; Laurier et al., 2003; Balcom et al., 2004). Here, the methylation of Hg is dependent on the physical regime of the estuary and the sediment (e.g., degree of bioturbation, hydrodynamics at the sediment water interface: SWI), sediment chemistry (e.g., organic matter (OM) content, iron content, sulfate and sulfide concentration), and the movement of Hg from sediments to biotic receptors through physiochemical, physiological, and ecological processes. The biogeochemical factors in sediments greatly influence the transformation of inorganic Hg to MeHg that, in turn, determines its

potential for bioaccumulation and biomagnification in food webs.

Estuarine and coastal sediments are areas of high MeHg production due to in situ biogeochemical conditions including high organic matter and sulfate. Microbial processes including sulfate reduction utilize these substrates to produce high concentrations of MeHg if conditions are optimal. Of the factors that are likely to control sediment MeHg and methylation rates are the inorganic Hg loading to the sediment, as well as the geochemical factors that influence the availability of the inorganic Hg, and the supply of OM to sulfate-reducing bacteria. Moreover, the great spatial and temporal variation in estuarine biogeochemistry also results in areas of high MeHg production, release, and bioaccumulation. These coastal sediments are also subject to significant fluctuations in water level and salinity that result in redox transitions in the zone close to the SWI.

MeHg can be transferred from surface sediments to the water column via passive diffusion, advection, and resuspension (Bloom et al., 1999). Efflux into the water column may be controlled by the location of the redoxcline (Merritt and Amirbahman, 2008). For example, oxic or suboxic surface sediments may impede direct MeHg release into the water column (Gagnon et al., 1996; Bloom et al., 1999). This may be a consequence of sorption to certain surface sediments, especially Fe-oxyhydroxides, or increased rates of MeHg demethylation close to the SWI (Merritt and Amirbahman, 2008). The location of the redoxcline, in turn, is controlled by factors such as OM content, sediment physical mixing by the benthic infauna, rate of microbial respiration, and hydrodynamics at the SWI. Lambertsson and Nilsson (2006) observed the highest MeHg concentration in sediments with the highest OM content, and a progressive upward movement of MeHg production maxima with increasing sediment OM.

The abundance of benthic infauna may influence MeHg cycling rates and concentration. At high bioturbation rates, the introduction of oxidants, such as O_2 and Fe(III), deepens the sediment redoxcline (Aller, 1978, 1994; Hines et al., 1999) and this may influence porewater MeHg concentration. Burrowing macrofauna also leads to bioirrigation that may enhance sulfate transport and oxidizing conditions (Furukawa et al., 2000), thereby affecting MeHg production rates. Recently, Benoit et al. (2006) suggested that in the presence of a high burrow density, oxidizing conditions may extend deeper in the sediment and result in a lower sediment methylation rate. They also proposed that intermediate burrow densities ($\sim$500 burrows m^{-2}) may provide optimum conditions for the enhancement of methylation by preventing the buildup of significant dissolved S(-II). In addition to influencing the direct transfer of MeHg to the overlying water, benthic fauna also may be important in biotransfer of MeHg to hyperbenthic organisms and on to pelagic food webs, as discussed later.

Complex interactions among physical, chemical, and biological factors in the coastal zones contribute to the variation in MeHg production from inorganic Hg at the local scale (patchiness). These zones are sites of significant sedimentation of river-borne particulate matter, and as a result, act as storage areas for sediment contaminants including Hg. Areas within the coastal zone that have sediments with small grain size, high OM content, and shallow redoxcline may lead to levels of MeHg production, release, and bioaccumulation. In contrast, areas within coastal zones that are subaerially exposed at times, and in the areas where a relatively high water velocity occurs, the coarse sediment grain size and a low OM content results in a relatively deep oxic/suboxic environment. In these regions, net MeHg production may be diminished and MeHg release and bioaccumulation may be suppressed by enhanced adsorption and demethylation. This local scale spatial heterogeneity in MeHg production and concentration likely affects flux of MeHg to the water column and bioaccumulation by benthic and pelagic organisms.

MeHg Bioaccumulation at the Base of the Food Web

Hg methylation in the pelagic zone is also a potentially important source for MeHg bioaccumulation. In the water column, it is readily concentrated by phytoplankton, the biological conduit for transferring the contaminant to pelagic and benthic food webs (Lindqvist et al., 1991; Watras and Bloom, 1992; Mason et al., 1996). Concentration factors of MeHg from water to aquatic organisms are overwhelmingly largest in phytoplankton, where values can be $>10^5$, compared to subsequent increases to higher trophic levels of only 2–3 times (Baeyens et al., 2003; IAEA, 2004; Hammerschmidt and Fitzgerald 2006a; Pickhardt and Fisher, 2007). Bioconcentration factors in phytoplankton can vary up to six orders of magnitude between different metals but generally less than one order of magnitude for a given metal across taxa (Fisher, 1986; Fisher and Reinfelder, 1995). Temporal or spatial variations in MeHg bioconcentration in phytoplankton could be transferred to higher trophic levels, although relatively few field measurements

have been made, so it is important to examine those factors likely to influence its bioaccumulation in phytoplankton.

It is important to understand the mechanisms underlying MeHg uptake in phytoplankton in order to understand geographic and seasonal variability in contamination of marine food chains. There is evidence for both passive and active uptake of Hg and MeHg by phytoplankton (Moye et al., 2002; Pickhardt and Fisher, 2007). In San Francisco Bay-Delta waters, Hg and MeHg were enriched about 10^4 times out of ambient water by dead cells but 10^5 times in living cells (Pickhardt and Fisher, 2007). Passive uptake of Hg and MeHg sorbed primarily to cell surfaces is not unlike many other particle-reactive metals (Fisher et al., 1984; Fisher, 1985). However the mechanisms for active uptake of MeHg remain unclear. Hg and MeHg could be bound to some organic compounds that are actively acquired by cells (Roditi et al., 2000; Brandt et al., 2008) or act as a surrogate for a compound that is actively taken up. Once taken up, MeHg penetrates more significantly into the cytoplasm of algal cells than inorganic Hg (Mason et al., 1996; Pickhardt and Fisher, 2007) and results in higher herbivore assimilation efficiencies and greater transfer in the food web (Reinfelder and Fisher, 1991).

Variations in dissolved organic matter (DOM) concentration and composition may account for spatial and temporal variations in MeHg bioavailability to phytoplankton. DOM derives from both autochthonous and allochthonous sources (Fenchel et al., 1998), and such variations are more pronounced in estuarine and coastal waters than in the open ocean (Thurman, 1985; Cauwet, 2002), reflecting the more variable biological productivity in coastal systems and input from terrestrial sources. Metals complexed by DOM in natural waters are generally less bioavailable to phytoplankton than free metal ions (Campbell, 1995). Once bound to DOM, ionic mercury can undergo photochemical reduction to elemental mercury, which in turn could influence volatilization and bioavailability (Ravichandran, 2004). Ionic Hg in natural waters is primarily bound to organic complexes, often involving multidentate chelation sites (Lamborg et al., 2003; Han and Gill, 2005). Inorganic and MeHg have strong affinities for thiol groups in DOM (Hintelmann et al., 1997; Benoit et al., 2001; Amirbahman et al., 2002; Haitzer et al., 2003; Han et al., 2006). MeHg also displays a strong affinity for natural colloidal matter (1 kDa−0.45 μm, particularly colloidal material $<$10 kDa) (Choe and Gill, 2001; Bilinski et al., 2000), mainly with thiol-type functional groups (Guentzel et al., 1996), but the influence of colloidal matter on the bioavailability of MeHg in plankton remains largely unknown.

Because the composition and concentration of DOM (including thiol enriched material) varies seasonally and spatially, its influence on the bioavailability of MeHg should also vary spatially and temporally. Further, for low to moderate DOM concentrations, the relationship of DOM concentration and bioavailability of MeHg to phytoplankton appears variable (Gorski et al., 2008). Until more is known about DOM's influence on the uptake kinetics of MeHg in phytoplankton and the rate of production (e.g., from algal blooms) and decomposition of thiol-rich DOM compounds, it will be difficult to predict how regions will vary with regard to MeHg bioconcentration patterns.

From bioconcentration in primary producers, MeHg is potentially transferred to primary and secondary consumers through several physical and biological processes that have important consequences on the fate of the contaminant in pelagic and benthic trophic pathways. The uptake of MeHg by marine consumers in estuarine and coastal ecosystems can occur directly from sediments or via the water column. For benthic organisms, MeHg can be taken up directly from the bulk sediments or from porewater. In addition, organic matter in sediments reduces the bioavailability of MeHg to benthic fauna due to potential effects on feeding rates, changes in K_d, or changes in the solubilization of MeHg in the gut of deposit feeders (Mason et al., 1999; Lawrence and Mason, 2001; Mason, 2002). MeHg bioconcentrated by phytoplankton is incorporated into the pelagic food web by local zooplankton communities grazing on phytoplankton, and subsequently transferred to resident or transient planktivorous biota (Mathews and Fisher, 2008). Alternatively, through pelagic-benthic coupling, suspension-feeding benthic invertebrates transport MeHg from the water column to the sediment-water interface, making it available to benthic food webs. These sediment MeHg repositories are available to benthic infauna and epifauna (e.g., crustaceans, polychaetes) because of those organisms' direct contact with the substrate and their deposit feeding on detrital matter (Lawrence and Mason, 2001). The relative importance of these two pathways is not known, but ongoing studies suggest greater bioaccumulation by pelagic feeding species.

Ecological Transfer of MeHg from Estuaries to the Continental Shelf

Recent investigations suggest that MeHg production in coastal sediments is important to local and potentially

offshore biota (Benoit et al., 2003; Hammerschmidt and Fitzgerald, 2006a), however little information exists on the spatial connectivity between these sources and bioaccumulation by marine food webs that span contiguous ecoscapes, i.e., estuarine, coastal, and open ocean habitats.

Kneib (1997, 2000) defined "nekton trophic relay" as the movement of intertidal carbon production across boundaries within marsh and estuarine ecoscapes. This horizontal bioadvection of energy is mediated by a sequence of predator–prey interactions that actively transport carbon biomass across interconnected habitats. Because of the bioaccumulating properties of MeHg, any trophic transfer of carbon resources likely results in the concomitant bioadvection of MeHg (Fitzgerald et al., 2007). To this end, Kneib's (1997) conceptual "trophic relay" model provides a framework for describing the spatial connectivity between MeHg sources and a continuum of marine trophic assemblages.

Resident nekton complete their entire life history in the estuary or coastal regions and bioaccumulate MeHg through the passive uptake of contaminants from ambient water, and to a larger extent, through dietary uptake of zooplankton or small benthic invertebrates. Resident nekton also undergo daily migrations between estuaries that include movements between intertidal and subtidal habitats. These regular movement patterns result in the spatiotemporal overlap of resident nekton with larger predatory residents and transient nekton, the latter representing species that seasonally utilize estuaries for foraging, refuge, and reproduction. This sequence of predator–prey interactions effectively translocates MeHg from near-shore sediment repositories to estuarine pelagic food webs, and subsequently exposes higher trophic level coastal and oceanic organisms to MeHg contamination.

Consistent with the "trophic relay" concept, the bioadvection of MeHg from estuaries to coastal and open ocean ecoscapes is contingent upon larger-scale movement patterns by transient nekton. Seasonal migrations and ontogenetic shifts in habitat use by these transient species physically translocate MeHg contaminants to coastal environments. Moreover, these species are subjected to periodic predation pressure from oceanic apex fishes. For example, large epipelagic predators (e.g., porpoises, tuna, billfishes, and sharks) have cosmopolitan distributions owing to their highly migratory behavior. Moreover, these species are opportunistic feeders of schooling fish and cephalopods, and will seasonally utilize coastal habitats during brief foraging events (Collette and Klein-MacPhee, 2002 [and references therein]). During these seasonal visits, transient apex predators feed on coastal prey and, in the process, serve as conduits for the bioadvection of MeHg to offshore fisheries. An understanding of these complex migrations, shifts in habitat use, and predator–prey interactions is necessary to determine the relative importance of this ecologically-mediated translocation of MeHg in marine systems.

Spatial Patterns of MeHg Biomagnification in Ocean Food Webs

In studies of aquatic food webs thus far, MeHg bioaccumulated by lower trophic levels biomagnifies with increasing trophic level. However, factors such as geographic differences in Hg emissions, deposition, and the structure of marine food webs in addition to spatial variation in the processes described earlier all influence the fate of MeHg in marine ecosystems. This suggests the hypothesis that there are spatial differences in food web biomagnification of MeHg. To date, inter-comparison between existing food web studies is problematic because of differences in units used (e.g., dry vs. wet weight basis), measurements of whole organisms versus specific tissues, and uneven coverage of ecosystems. Therefore, a limited number of studies are available for comparison of trophic transfer across marine systems.

One way to compare biomagnification across food webs is to plot the linear relationships between Log (μg THg g^{-1} wet weight) and δ^{15} N values: Log [THg] = aδ^{15} N + b (Kidd et al., 1995), and use the regression slope as a measure of biomagnification rate and the intercept as the baseline value for primary producers. A comprehensive study of a marine arctic food web in Lancaster Sound, Canada, at ~74°N, examined a food web of 27 species from particulates to mammals that had a slope of 0.2 (Atwell et al., 1998). Nearby, a study of a northern Baffin Bay food web (ice algae to seal) revealed a similar slope of 0.197 and 0.22 for THg and MeHg, respectively (Campbell et al., 2005). The similarity of the slopes for MeHg and THg regressions and the observed increase in the % of MeHg with trophic level suggests that MeHg is the main form of mercury being bioaccumulated. A Gulf of Maine food web including 10 size categories of plankton up to whales exhibited regression slopes of 0.14 and 0.23 for THg and MeHg, respectively [Harding et al., unpublished]. In contrast, a study focused on upper trophic levels in the Gulf of Farallones, California (krill to sea lion) had a slightly higher

slope of 0.32 (Jarman et al., 1996). Thus, the rates of biomagnification across the above food webs were similar despite the differing food-chain coverage.

While the geographic coverage of Hg food web studies in the world's oceans is not comprehensive, the few available studies permit a limited assessment of spatial variation of bioaccumulated and biomagnified MeHg. For example, in comparing five food chain studies across latitudes between 76°N and 74°S, the concentrations of total Hg in microplankton were within the same order-of-magnitude (Table 2). This was also true for higher trophic categories of mesoplankton, macroplankton, pelagic, plankton feeding fish, and fish-eating mammals. Sea lion Hg burdens in the Gulf of Farallones are an exception, however, as liver concentrations were measured instead of muscle tissue (Campbell et al., 2005). Furthermore, MeHg concentrations in a simplified food chain in Long Island Sound were of the same magnitude as those measured in an arctic food chain (Campbell et al., 2005; Hammerschmidt and Fitzgerald, 2006a).

Based on the comparison of these studies, there is currently no evidence for a latitudinal pattern of Hg bioaccumulation. This is similar to findings in freshwater ecosystems (Campbell et al., 2003). Nor is there a difference between the northern and southern hemispheres, despite the anthropogenic enhancement of atmospheric Hg in the northern hemisphere (Mason et al., 1994). However, fully characterizing these patterns will require more detailed site studies and greater geographic coverage of MeHg in marine food webs.

Conclusions

At the local scale, MeHg production and bioaccumulation in coastal and pelagic systems are both strongly influenced by biogeochemical conditions, including the concentration and nature of organic matter in sediments and in water. In contrast, trophic transfer and bioadvection of MeHg from estuaries to coastal oceans are largely controlled by ecological factors, such as predator–prey interactions and migratory behaviors of different trophic groups. At the global scale, major sources of MeHg production in coastal zones and the deep ocean are not the marine habitats where fish with the highest MeHg concentrations are harvested, nor do food webs in different ocean regions differ greatly in their bioaccumulation and biomagnification despite great spatial variation in Hg emissions and MeHg production. These patterns all suggest that sources and biological receptors, such as pelagic fish species and their human consumers, are not related spatially and that physical and ecological processes are important in transferring MeHg from source regions to bioaccumulation in marine food webs. To fully understand the fate of MeHg in the marine environment, these sources and transfer processes need to be identified, quantified, linked, and evaluated with ecosystem models.

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Table 2. Latitudinal variation of mercury concentrations (μg THg/g wet weight) in various trophic levels of well-studied ecosystems

Geographical location	Lancaster sound[a]	N. Baffin Bay[b]	Gulf of Maine[c]	Gulf of Farallones[d]	Ross Sea, Antarctica[e]
Latitude	~74°N	~76°N	~44°N	~38°N	~74°S
Sampling date	1988–1990	1998	2001–2003	1993–1994	1989–1991
Microplankton	<0.004	0.003	0.002 ± 0.001	–	0.007 ± 0.001
Mesoplankton	0.012 ± 0.002[f]	0.025 ± 0.017	0.003 ± 0.001	–	0.013–0.007 [h]
Macrozooplankton	0.012–0.02 [h]	0.020 ± 0.009	0.006 ± 0.001	0.006 (0.006–0.008)[g]	0.015 ± 0.005
Pelagic fish (muscle)	0.038 ± 0.006	0.04	0.047 ± 0.028	0.02 (0.018–0.022)	0.068 ± 0.066
Seal or porpoise (muscle)	0.214 ± 0.006	0.68 ± 0.29	0.501 ± 0.297	3.8 (0.96–1.46)	0.37

[a]Atwell et al., 1998.
[b]Campbell et al., 2005.
[c]Harding et al. [unpublished].
[d]Jarman et al., 1996.
[e]Bargagli et al., 1998.
[f]Mean ± SD.
[g]Geometric mean (±1 SD).
[h]Range.

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