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Anton M. Scheuhammer, Michael W. Meyer, Mark B. Sandheinrich and Michael W. Murray

Effects of Environmental Methylmercury on the Health of Wild Birds, Mammals, and Fish

Wild piscivorous fish, mammals, and birds may be at risk for elevated dietary methylmercury intake and toxicity. In controlled feeding studies, the consumption of diets that contained Hg (as methylmercury) at environmentally realistic concentrations resulted in a range of toxic effects in fish, birds, and mammals, including behavioral, neurochemical, hormonal, and reproductive changes. Limited field-based studies, especially with certain wild piscivorous bird species, e.g., the common loon, corroborated laboratory-based results, demonstrating significant relations between methylmercury exposure and various indicators of methylmercury toxicity, including reproductive impairment. Potential population effects in fish and wildlife resulting from dietary methylmercury exposure are expected to vary as a function of species life history, as well as regional differences in fish-Hg concentrations, which, in turn, are influenced by differences in Hg deposition and environmental methylation rates. However, population modeling suggests that reductions in Hg emissions could have substantial benefits for some common loon populations that are currently experiencing elevated methylmercury exposure. Predicted benefits would be mediated primarily through improved hatching success and development of hatchlings to maturity as Hg concentrations in prey fish decline. Other piscivorous species may also benefit from decreased Hg exposure but have not been as extensively studied as the common loon.

INTRODUCTION

Numerous studies document the toxic effects of methylmercury (MeHg) in individuals of various vertebrate species. However, it is less clear whether current environmental levels of MeHg pose health hazards to free-living fish and wildlife, and especially to populations of animals rather than to individuals. Here we present a brief synthesis of the scientific state of knowledge regarding current levels of MeHg exposure and its toxic effects in fish and wildlife. Because of the paucity of information on the toxicology of MeHg in reptiles and amphibians, our report focuses on fish, birds, and mammals.

Under most conditions, fish and wildlife are exposed primarily to MeHg rather than to other chemical forms of Hg, and the route of exposure is primarily through the diet. Thus, our report focuses on the effects of MeHg at ecologically relevant levels of dietary exposure. Our report is not intended to be a comprehensive critical review of the literature, and we pay particular attention to recent studies that have not been included in previous reviews. For a more detailed discussion of various aspects of Hg exposure, accumulation, and toxicology in fish and wild birds and mammals, the reader is directed to prior reviews (1–6).

EXPOSURE

Species and Habitats at Greatest Risk

Because of biomagnification of MeHg, long-lived piscivorous or other top predatory animals feeding in aquatic food chains are at greatest risk for elevated dietary MeHg exposure, accumulation, and toxicity. These species include large predatory fish, such as walleye (*Sander vitreus*), northern pike (*Esox lucius*), and lake trout (*Salvelinus namaycush*); mammals, such as mink (*Mustela* spp.), otter (*Lutra* spp.), polar bears (*Ursus maritimus*), and seals (*Phocidae* and *Liliaceae spp.*); and piscivorous birds, such as common loons (*Gavia immer*), bald eagles (*Haliaeetus leucocephalus*), osprey (*Pandion haliaetus*), kingfishers (*Alcedo* spp.); and some seabirds, such as albatross (*Diomedeidae*) and certain Arctic species (7). Conversely, terrestrial nonpiscivorous species (e.g., granivorous and insectivorous birds) typically demonstrate relatively low Hg exposure (<0.5 mg kg^{-1} wet weight in blood) (8, 9) and are generally not considered to be at risk for MeHg toxicity (10). High, potentially toxic concentrations of Hg (>5 mg kg^{-1} wet weight in brain, or >20 mg kg^{-1} in liver) have occasionally been reported in a variety of predatory wildlife species (5). Factors influencing Hg exposure in fish and wildlife, and concerns regarding tissue sampling and analysis have recently been reviewed (11, 12)

Piscivorous wildlife living in inland freshwater habitats often experience higher Hg exposure than the same species from nearby estuarine or marine habitats. In Maine, bald eaglets sampled at nests on inland lakes had higher blood-Hg concentrations than did eaglets raised in nests on rivers, estuaries, or marine (coastal) habitats; and a similar trend was found for belted kingfishers (*Ceryle alcyon*) nesting in the same region (8). Higher Hg concentrations were reported in feathers from chicks of both great egrets (*Ardea alba*) and white ibises (*Eudocimus albus*) at a variety of inland freshwater sites throughout peninsular Florida than in feathers of the same species from coastal colonies (13). Tissues of otter from inland habitats in Nova Scotia, Canada, had higher Hg concentrations than those from corresponding marine coastal habitats (14).

Piscivorous fish and wildlife living near local point sources of environmental Hg contamination may experience elevated Hg exposure (15, 16), which may persist long after new inputs of Hg have ceased (17–19). Some formerly important sources of environmental Hg pollution that resulted in toxicity and death of wild birds and mammals in the past (e.g., use of organomercurials as seed dressings and effluents from Hg-cell chloralkali plants) have been eliminated or greatly reduced.

Environments remote from point-source releases of Hg can also contain fish and piscivorous wildlife with elevated Hg concentrations. For example, some common loons and river otters (*Londra canadensis*) from the interior of Nova Scotia, Canada, and from several New England states (US) have among the highest tissue Hg concentrations reported for these species (8, 20, 21). Elevated Hg concentrations have also been reported in some predatory marine mammals and birds, especially from relatively remote northern locations (7, 22–26). In general, regions that receive relatively high atmospheric Hg loadings and are characterized by a high proportion of Hg-sensitive aquatic ecosystems in which Hg methylation rates are relatively high (low-alkalinity, low-pH lakes; surface waters with large upstream or adjoining wetlands; waters with adjoining or upstream terrestrial areas subjected to flooding; and dark-water lakes and streams) pose the greatest risk for

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piscivorous wildlife, because it is in these environments that trophic transfer of Hg is high and fish accumulate the greatest concentrations of Hg (5, 27, 28).

Temporal and Geographic Trends in Exposure

Where they were studied, temporal trends of Hg in wildlife were found to vary by region and by the magnitude of the time period chosen for analysis. Studies of trends in areas affected by industrial activity have sometimes reported declining Hg levels in recent years (21, 29); however, more long-term analyses, especially in remote locations, such as the North Atlantic and the Arctic, where global atmospheric inputs of Hg likely predominate, indicate increasing Hg concentrations in piscivorous wildlife over the past 30–150 years (24, 30, 31). In Florida, Hg in fish-eating birds increased substantially during the 1990s, compared with earlier decades, reflecting trends in local source deposition, which then later declined as local emissions were controlled (13, 32).

Extensive sampling of common loon feathers, blood, and eggs indicates a general west-to-east gradient of increasing Hg exposure in this species across North America, roughly consistent with patterns of atmospheric Hg deposition (33, 34). However, within specific regions, there can be wide variation in Hg exposure in loons, associated with differing land-use patterns, and physical and chemical characteristics of local watersheds—for example, the degree of lake acidification (8).

Demethylation and Interactions with Selenium

Most fish and wildlife are exposed to Hg primarily as MeHg through diet. However, in at least some predatory aquatic wildlife species, after tissue accumulation of MeHg, a portion of the body's MeHg burden may be demethylated. The resulting inorganic Hg often accounts for a significant, yet highly variable, fraction of the total Hg present in certain tissues, especially in liver and kidney (and perhaps brain). Some other tissues, such as skeletal muscle; fur; feathers; and eggs show little or no evidence of demethylation. Methylmercury in liver generally predominates when total Hg concentrations are less than ~10 mg kg^{-1} wet weight; however, with greater Hg accumulation, an increasingly high proportion of the total liver Hg is often present as an inorganic species. Frequently, animals with the highest liver-Hg accumulation have the lowest MeHg fraction, despite being exposed to Hg primarily as MeHg in fish (35, 36). Although there is some uncertainty regarding the toxicity of inorganic Hg derived from demethylation, a detoxification mechanism for MeHg in seabirds and marine mammals has been proposed that entails demethylation by reactive oxygen species and subsequent formation of high molecular weight Hg-selenium (Se)-protein compounds, which subsequently undergo degradation in lysosomes, creating an insoluble Hg-Se compound (35, 37). Ultimately, insoluble mercuric selenide (tiemannite) or other stable, insoluble Hg-Se-protein fragments accumulate in the liver through time (23, 38). The cellular mechanisms by which this process occurs and the energy costs involved are not well understood.

Different species exhibit differences in their apparent ability to demethylate Hg, which may affect their relative sensitivity to MeHg toxicity; however, this has not been explicitly studied. For example, free-living river otters generally accumulate higher concentrations of Hg than mink; however, in otter brains, only ~74% of total Hg was in the organic form (MeHg), compared with ~90% for mink (39, 40), indicating that otters may be better able to metabolize organic Hg into an inorganic form. Also, a significant correlation between Hg and Se was reported in brains of otter but not mink (41). A positive correlation between Hg and Se levels in the brain was also observed in monkeys exposed to MeHg, with no exposure to Se other than through their regular diet (42). In addition, fish and wildlife living in environments with elevated Se levels exhibit lower-than-expected Hg accumulation (43, 44).

Diets supplemented with Se, including organic forms of Se present in biological materials, can protect against or delay the onset of MeHg-induced neurotoxicity (45). However, although selenomethionine supplementation ameliorated the neurotoxic effects of dietary MeHg exposure in adult mallard ducks (*Anas platyrhynchos*), reproductive impairment was more severe in ducks that consumed a diet supplemented with both MeHg and selenomethionine than in birds consuming diets with elevated levels of MeHg or selenomethionine alone (46). Differences in the dietary intake of Se by different wildlife species, or the same species in different environments, may contribute to variability in the expression of MeHg toxicity. The extent to which different wildlife species demethylate MeHg, the consequences of this process on the toxicology of MeHg, and the role of Se in MeHg accumulation and toxicity in wildlife at ecologically relevant doses require further investigation.

EFFECTS

Fish

Compared with humans and mammalian and avian wildlife, relatively little is known of the toxicological significance to fish of environmentally realistic exposures to MeHg (4, 5). Laboratory studies typically exposed fish to aqueous concentrations of MeHg that are 10^4- to 10^5 -fold greater than those in natural waters (4), rather than using realistic dietary exposure conditions. The route of administration of Hg in these studies was also unrealistic, because diet, not water, is the main source of MeHg exposure in wild fish (47, 48).

Overt effects on fish growth and survival occur only at high tissue Hg concentrations (6–20 mg kg^{-1} wet weight in muscle) (4), observed primarily in fish from highly contaminated environments, such as Minamata Bay, Japan (49, 50), and Clay Lake in the English-Wabigoon River system, Ontario, Canada (51). However, several laboratory studies demonstrated MeHg impairment of fish behavior, gonadal development, production of sex hormones, and reproduction at concentrations more typical of those in fish from flooded, low alkalinity, or other Hg-sensitive habitats (52–55). Suppression of gonadal development, egg production, and spawning were reported in juvenile fathead minnows (*Pimephales promelas*) fed MeHg-contaminated diets until sexual maturity (56). Mated pairs of fish with mean carcass concentrations of 0.71 mg kg^{-1} wet weight (males) to 0.86 mg kg^{-1} (females) experienced a 39% reduction in spawning success. Similar carcass concentrations were also associated with disrupted reproductive behavior of male fathead minnows (57), suppressed plasma estradiol and testosterone, and reduced reproductive success (52). Suppressed estradiol was likely due to increased numbers of apoptotic follicular cells caused by MeHg exposure (58). Significant inverse relationships between concentrations of MeHg and estrogen, testosterone, and 11-ketotestorone was also observed in white sturgeon (*Acipenser transmontanus*) from the lower Columbia River (59). Methylmercury concentrations that suppressed sex hormones, altered reproductive behavior, and impaired reproduction in fish were similar to mean concentrations in carcasses of white perch (*Morone americana*; 0.78 mg kg^{-1}), walleye (0.71 mg kg^{-1}), and northern pike (*Esox lucius*; 0.56 mg kg^{-1}) from northeastern North America (60) and less than those measured in axial muscle of fish from many lakes and reservoirs (Fig. 1).

Although maternal transfer of dietary MeHg bioaccumulated during oogenesis is the primary mechanism of MeHg

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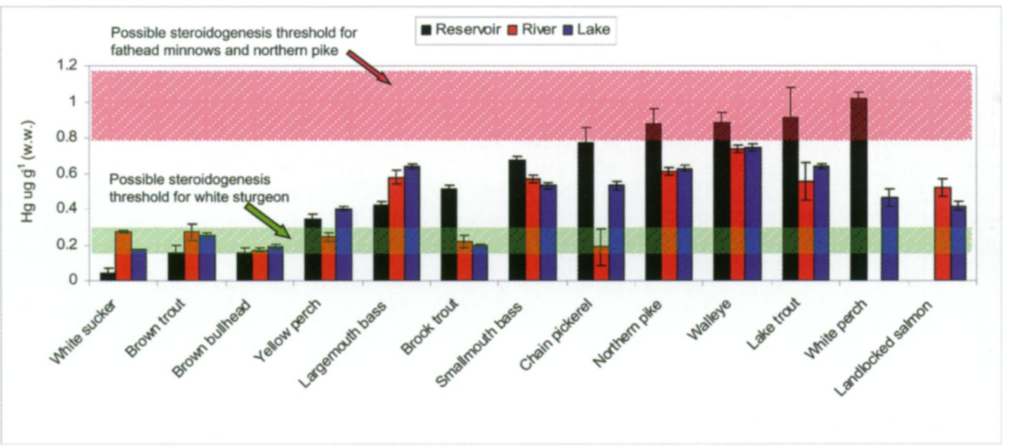

Figure 1. Mean Hg concentrations in fillets of standard-length freshwater fish of various species from across northeastern North America (data adapted from [60]), combined with estimated Hg concentration thresholds associated with steroidogenic effects. Threshold ranges adapted from (52) [fathead minnows]; Sandheinrich, M., Drevnick, P., Wiener, J., Knights, B., and Jerimison, J. unpubl. data [northern pike]; and (59) [white sturgeon].

exposure to fish embryos (61), there is limited information on the effects of maternally transferred MeHg in fish. Aqueous exposure of eggs of rainbow trout (*Oncorhynchus mykiss*) to inorganic Hg resulted in total Hg concentrations of 0.07–0.10 mg kg^{-1} wet weight in eggs and significantly increased embryonic mortality (62). These concentrations are within the range of total Hg measured in eggs of yellow perch (*Perca flavescens*) from semi-remote lakes in northern Wisconsin, US (63). Thus, based on laboratory studies that examined the effects of dietary MeHg in fish, combined with data on concentrations of MeHg in free-living fish, it is plausible that MeHg at environmentally relevant concentrations may affect reproduction in wild populations of fish. Differences among species in their reproductive sensitivity to MeHg, as well as the population-level consequences of impaired reproduction in individual fish remain to be determined.

Mammals: Suggested Toxicity Thresholds

Mink and otter are the mammalian wildlife species for which the greatest amount of information exists regarding Hg exposure and toxicity. Data from several studies indicate that consumption of diets that contained Hg (as MeHg) ≥ 1 mg kg^{-1} wet weight caused neurotoxicity and death in adult mink and otter (5). The US Environmental Protection Agency (USEPA) estimated lowest observable adverse effect level (LOAEL) for mink is currently 0.18 mg kg^{-1} body weight d^{-1}, or 1.1 mg kg^{-1} wet weight (approximately 3–4 mg kg^{-1} dry weight) MeHg in the diet (64). Neurological signs in MeHg-intoxicated mammals typically include lethargy, ataxia, limb paralysis, tremors, convulsions, and ultimately death.

Brain Hg (probably primarily as MeHg) concentrations in otter and mink with overt MeHg poisoning are typically >5 mg kg^{-1} wet weight (5). After exposure to MeHg, brain-Hg concentrations in the range of 12–20 mg kg^{-1} wet weight during postnatal development in a variety of small mammal species used in medical research were associated with blindness, spasticity, and seizures, whereas 3–11 mg kg^{-1} were associated with more subtle effects on behavior and cognition, including increased activity, poorer maze performance, abnormal startle reflex, impaired escape and avoidance behavior, and abnormal visual evoked potentials (65). Numerous individual free-living mink or otter from various locations in North America have brain-Hg concentrations sufficiently high (>3 mg kg^{-1}) to be associated either with clinical MeHg intoxication or with more subtle neurological impairments that could detrimentally affect survival (5, 21).

Recently, various neurochemical changes in brains of free-living otter (66) and mink (67) were correlated with brain-Hg concentrations <5 mg kg^{-1} wet weight; and similar effects were demonstrated in captive mink fed diets that contained ecologically realistic concentrations of MeHg (68) (Fig. 2). It is probable that the current level of MeHg exposure of free-living mink and other piscivorous mammals in a number of Hg-sensitive environments is sufficiently high to have subtle neurotoxic and other consequences (69, 70). Aqueous MeHg concentrations likely exceed the USEPA derived mammalian wildlife criteria for mink (57 pg MeHg L^{-1}) (71) in many aquatic ecosystems; however, it is currently unclear whether documented environmental concentrations and toxic effects on individual animals have population-level impacts in mink or other mammalian species.

Birds: Suggested Toxicity Thresholds

In adult birds, Hg concentrations (as MeHg) >15 mg kg^{-1} wet weight in a variety of tissues, including brain, are associated with overt signs of MeHg intoxication and death (5). Nonlethal effects of lower Hg concentrations have been less well studied, except for effects on reproduction. Egg-Hg concentrations >1 mg kg^{-1} wet weight are associated with impaired hatchability and embryonic mortality in a number of bird species, and brain-Hg concentrations >3 mg kg^{-1} wet weight are associated with mortality in developing bird embryos (5). In free-living common loons, diets that contained >0.3 mg kg^{-1} wet weight Hg (as MeHg) were associated with severely reduced reproductive success, mainly as a result of decreased egg laying and territorial

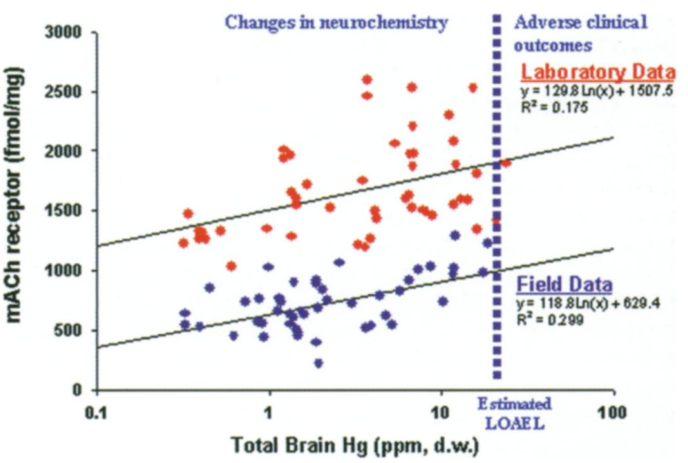

Figure 2. Concentration of muscarinic acetylcholine receptors in brains of free-living wild mink and captive mink fed diets with different levels of MeHg as a function of brain Hg concentration. Changes in receptor concentrations occur at Hg concentrations below those associated with overt toxicity. Data adapted from Ref. 68. Estimated LOEL for overt MeHg intoxication (~20 mg kg^{-1} dry weight or ~5 mg kg^{-1} wet weight Hg in brain) based on (98).

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fidelity by breeding adults (15). Many lakes in North America have fish with Hg concentrations that exceed 0.3 mg kg^{-1} wet weight (60, 72).

The MeHg effect threshold for common loon chicks was established via subchronic dose-response studies (73–75). Loons were dosed daily from hatch through day 105 with fish diets that contained control, 0.08, 0.4, or 1.2 mg kg^{-1} wet weight as MeHg chloride. No overt signs of toxicosis or significant reductions in growth or food-consumption rates were observed in any dose group (75), but there was evidence of reduced immune response and histological changes (central nervous system demyelination) in chicks that received ecologically relevant doses of MeHg (0.4 mg kg^{-1} diet wet weight) (76). A preliminary loon chick LOAEL was estimated at 0.4 mg kg^{-1} wet weight in diet (fish), whereas a preliminary no observable adverse effect level (NOAEL) in diet was estimated at 0.08 mg kg^{-1} wet weight (76).

Birds: Recent Field Studies

Correlations between MeHg exposure, reproductive impairment, and other effects have been examined in common loons breeding in the northern United States and eastern Canada (8, 15, 20, 76–80) and in wading birds nesting in south Florida and the Everglades (13). Although these correlational studies cannot be used to establish rigorous exposure thresholds of effect, they can identify populations where significant statistical relations exist between MeHg exposure and demographic and/or physiological parameters, and can assist in establishing effects thresholds.

Common Loon Reproduction in New Hampshire and Maine, US. Evers et al. (77) measured common loon productivity, behavior, and biochemical markers in relation to Hg exposure in 212 breeding territories and used adult blood-Hg concentrations to assign breeding territories to categories of Hg toxicity risk. Loon territories where adult blood Hg levels exceeded 3.0 mg kg^{-1} produced 40% fewer fledged young than territories where adult blood Hg was less than 1.0 mg kg^{-1}; these territories were categorized as "high risk." Territories where adult loon blood Hg exceeded 4 mg kg^{-1} were classified as "extra-high risk," because these concentrations were associated with impaired productivity, elevated levels of corticosterone in blood, developmental asymmetry in flight feathers, and adverse changes in essential breeding behaviors. On average, circulating corticosterone hormone levels increased 14.6% for every mg kg^{-1} increase in blood Hg ($n = 239$). Paired secondary feathers (one from each wing) from adults on high or extra-high risk territories had greater differences in mass than feathers sampled from territories with low Hg exposure risk ($n = 227$). Adult loons in high-risk territories also left eggs unattended 14% of the time, compared with 1% in lower-risk territories. A significant negative relation was found between adult blood Hg and foraging behavior, and a significant positive relation was observed between adult blood Hg and brooding behavior.

The majority of loon eggs collected at nests in New Hampshire and Maine ($n = 448$) contained elevated Hg concentrations. In Maine, 11% of eggs had Hg concentrations between 1.3 and 2.0 mg kg^{-1} wet weight, and 4% were >2 mg kg^{-1} wet weight (77). Egg Hg concentrations >1 mg kg^{-1} wet weight are associated with impaired hatchability in a number of avian species (3, 5, 20).

Common Loon Reproduction in Kejimkujik National Park, Nova Scotia, Canada. Common loons in some areas of Atlantic Canada have among the highest mean blood Hg concentrations in North America (20, 33). Concentrations were highest at Kejimkujik National Park, Nova Scotia, where adult blood-Hg levels averaged >4 mg kg^{-1} wet weight and where lower-than-normal loon productivity has been observed for many years (81, 82). At Kejimkujik, 92% of adult loons sampled had blood Hg concentrations in the "extra-high risk" category (>4 mg kg^{-1} wet weight) suggested by Evers et al. (77). The Hg exposure was related to impaired loon productivity and altered breeding behavior in loons in Atlantic Canada (83). The majority of common loon eggs collected at Kejimkujik nests had potentially toxic Hg concentrations (>1 mg kg^{-1} wet weight). Mercury concentrations in both loons and fish in Kejimkujik lakes were correlated with lake pH; however, prey fish abundance was not correlated with lake pH, indicating that pH-related prey depletion was not occurring and was not a confounding factor influencing loon productivity at Kejimkujik.

Common Loon Reproduction in Wisconsin, US. Field studies conducted in Wisconsin showed reduced common loon reproductive performance on acidic lakes where fish Hg concentrations and loon Hg exposure levels were elevated (79, 80). Subsequent studies found that, on acidic lakes, loon chick food intake rates and survival were lower, adult foraging behavior was altered, and blood Hg of adults and chicks, as well as prey Hg concentrations were higher than on circumneutral lakes (78). However, several environmental parameters, such as lake pH, co-varied with Hg. Thus, a laboratory approach was deemed necessary to directly test the effects of Hg exposure on common loon reproductive performance and to estimate LOAELs and NOAELs (75, 76).

One-hundred-fifteen adult loons from Wisconsin were assessed for Hg exposure via blood samples during 2002–2004, and 7 (6% of those sampled) had a blood Hg level >3.0 mg kg^{-1} wet weight (76), within the high-risk Hg exposure category of Evers et al. (77). Fifty-four percent of unhatched loon eggs collected from 33 nests in Wisconsin during 1996–2000 were categorized as "background" (0.0–0.6 mg kg^{-1} Hg wet weight) and 46% as "elevated" (0.6–1.3 mg kg^{-1} wet weight); none exceeded 1.3 mg kg^{-1} wet weight. All adult loons and loon eggs with elevated Hg concentrations sampled in Wisconsin were from acidic lakes (pH < 6.3).

A Wisconsin common loon demographic model was developed to evaluate population performance within a 5000 km^2 risk-assessment region of northern Wisconsin (76). The 5-stage deterministic projection matrix model predicts an annual growth rate of 0.9988 by using adult survival, fertility, and juvenile recruitment rates measured in the risk-assessment region during 2002–2004. Controlled dosing studies with loons (chick survival and hatching rates), combined with exposure assessment of the Wisconsin loon population, indicate that 10% of northern Wisconsin loon chicks have exposure levels associated with toxicity in the laboratory (Fig. 3), and 10% of adult female loons have exposure levels associated with a 30% reduction in egg hatching rate (76) (Fig. 4). The benefits of decreasing the Hg content of fish can be simulated by increasing the hatching and survival rates to account for the impairment assumed to be caused by elevated Hg exposure. If all chicks that are at risk for toxicity within the assessment region die before transition to adult stage (a conservative assumption), then reductions in Hg emissions to levels that result in Hg concentrations in loon prey <0.08 mg kg^{-1} wet weight (loon chick NOAEL from dosing study) should increase juvenile survival by 10%; and hatching success could be increased by 30% in 10% of the reproducing females. Inclusion of these adjustments to hatching success and chick survival in the projection matrix model results in a predicted improvement in annual population growth rate from 0.9988 to 1.011. When assuming a 10% improvement in chick survival and breeding success, the population growth rate within the risk-assessment region is predicted to increase by 1.7% annually. When assuming a total adult population in the study area of

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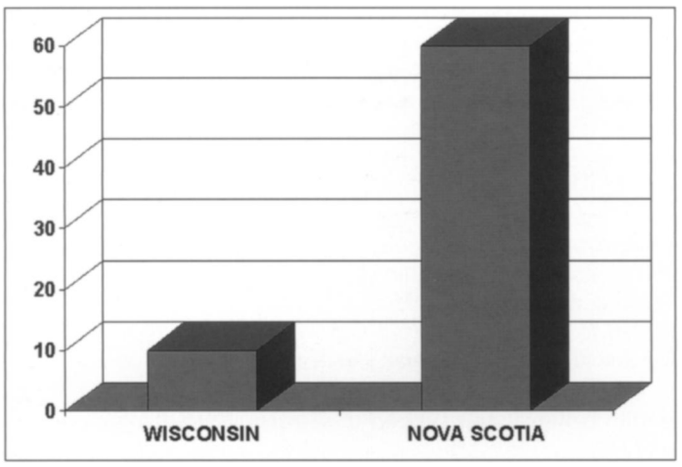

Figure 3. Percentage of loon chicks from Wisconsin, US, and Nova Scotia, Canada, having blood Hg concentrations associated with toxic effects (76) (>0.3 μg/m, as determined in controlled laboratory dosing studies [75]).

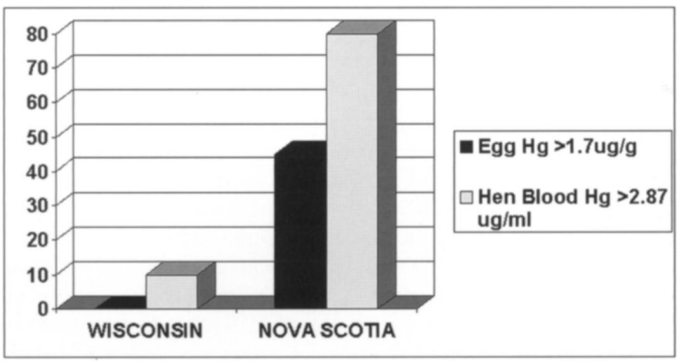

Figure 4. Percentage of regional common loon populations in Wisconsin, USA, and Nova Scotia, Canada, having egg Hg or hen blood Hg greater than thresholds associated with reduced hatching success.

approximately 1200 individuals (estimated population size in 2003), a 1.7% increase in annual growth rate would translate into 20 additional adults being recruited into the breeding pool each year. This K-selected species has an estimated lifespan of 25 years and an estimated annual survival rate of 0.92, thus the increase could plausibly translate into measurable improvement in population performance. Mercury emission reductions are predicted to have a much greater beneficial impact on the annual growth rate of the New England and Nova Scotia common loon populations where much larger proportions of chicks and hens are exposed to Hg levels associated with toxicity (Fig. 3), as determined in controlled dosing studies.

Aquatic Bird Population Decline in South Florida and the Everglades. Water bird populations (primarily wading bird species in the order Ciconiiformes) have declined precipitously in south Florida and the Everglades National Park since the mid 1930s. There was a ≥90% reduction in the numbers of nesting pairs of wood storks (*Mycteria americana*), great egrets (*Ardea albus*), white ibises (*Eudocimus albus*), and snowy egrets (*Egretta thula*) (84, 85). Loss of habitat, changes in hydroperiod, marsh compartmentalization, and salinization of estuarine feeding areas are thought to be primary factors responsible for the decline (84–86). However, MeHg toxicity has been proposed as an additional contributing factor (87) and as a potential impediment to the recovery of these species. Elevated liver Hg concentrations were found in 30–80% of breeding-age birds of various species found dead in the Everglades during 1987–1991 (88). In prebreeding female white ibises in the Everglades, estradiol concentrations were negatively correlated with Hg concentrations, as was the number of nesting attempts, suggesting that Hg exposure may cause fewer birds to nest or more birds to abandon nests because of subacute effects on hormone systems (89). Similar effects of Hg exposure on nesting behavior were previously documented for common loons in northwestern Ontario, Canada (15).

In recent years, dramatic declines in wading bird Hg exposure were documented in the Everglades. By using a previously established predictive relation between Hg consumption in food and feather Hg concentration for great egrets, Frederick et al. (13) estimated that average Hg concentrations in the diet of egrets declined by 67% between 1994 and 2000, and concluded that the Everglades underwent a biologically significant decline in Hg availability during that time period, probably due mainly to decreased local Hg inputs.

Bald Eagles and Osprey. Although both bald eagles and osprey are large piscivorous birds that experience elevated Hg exposure in some environments, these species have not been well studied with respect to potential effects of Hg on reproductive success or other population parameters. Nevertheless, the existing published reports indicate a lack of association between Hg exposure and productivity of free-living eagles or osprey in different locations in North America (19, 90–92).

Immunotoxic Effects

It has long been recognized that exposure to Hg may have autoimmune consequences. In wild birds, there is also circumstantial evidence that environmental MeHg exposure may be associated with a higher potential for infection by disease organisms. Dead loons found in an emaciated condition or with more parasitic infections generally had higher tissue-Hg concentrations than loons found dead in good body condition (93). In Florida, herons that died from chronic disease had significantly higher liver Hg concentrations (9.76 ± 2.40 mg kg^{-1}) than those that died in good body condition (1.77 ± 1.79 mg kg^{-1}) (87). Similarly, a number of immunological parameters were affected in egrets fed fish that contained an environmentally relevant concentration of Hg (0.5 mg kg^{-1}) (94). Low concentrations of both inorganic Hg and MeHg inhibited avian white blood cell phagocytosis *in vitro*; however, similar effects were not observed in blood of birds exposed to elevated MeHg *in vivo* (95). For mammals, harbour porpoises (*Phocoena phocoena*) demonstrated a relation between infections and elevated Hg accumulation (96, 97). The immunotoxicological effects of dietary MeHg exposure require further scientific research.

CONCLUSIONS

There is consistent evidence across a number of species that wild populations of fish, birds, and mammals in some regions of North America consume diets with MeHg concentrations sufficiently high to be toxic to individuals as determined from controlled dosing studies. Some wildlife species that feed at high trophic levels in aquatic food chains, especially species that feed primarily on fish or on other piscivorous species, are at particularly high risk for elevated exposure and potential toxicity. Laboratory dosing studies with fish and with piscivorous birds and mammals, indicate that ecologically relevant MeHg exposures can cause significant behavioral, physiological, immunological, neurochemical, reproductive, and histological changes. For fish and amphibians, field studies are limited and direct evidence of altered reproduction or other population parameters because of MeHg exposure in free-living

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populations are not presently available. However, for some wild piscivorous bird species, field studies demonstrated significant positive relations between MeHg exposure and numerous indicators of MeHg toxicity, including reproductive impairment, at ecologically relevant levels of Hg contamination. The weight of evidence indicates that reproduction is the demographic parameter most affected by exposure to MeHg in birds (and plausibly for fish and mammals as well). Potential population impacts will vary as a function of species life history, as well as regional differences in MeHg exposure because of regional and local variability in fish Hg concentrations. Modeling indicates that some regional common loon populations may benefit significantly from reductions in Hg emissions, which would result in decreasing concentrations of Hg in prey fish, and increased reproductive success mediated primarily through better hatching success and survival of hatchlings. Although the risk-assessment approach used for wildlife often emphasizes sustainability of wildlife "populations" rather than the health of individuals, conserving habitat quality is also a major consideration. Environmental Hg contamination results in degradation of Hg-sensitive aquatic ecosystems, because inorganic Hg is methylated, rapidly enters the food web, and biomagnifies to potentially toxic concentrations in fish and their predators.

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