

Low levels of dietary methylmercury inhibit growth and gonadal development in juvenile walleye (*Stizostedion vitreum*)

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Abstract

Mercury levels in the aquatic environment of North America have been increasing, raising the possibility that this highly toxic heavy metal might alter fish populations. Previous investigations have demonstrated toxic effects of mercury on teleost reproduction, but these findings were observed following unrealistically high exposures. In this study, we used concentrations frequently observed in North American lakes to investigate the effects of dietary methylmercury on growth, gonadal development, and plasma cortisol levels in juvenile walleye (*Stizostedion vitreum*). For a period of 6 months, two groups of walleye were reared on untainted catfish fillets, while two test groups were fed fillets injected with methylmercury, one group receiving $0.1 \mu\text{g Hg g}^{-1}$ food (low-mercury diet) and the other receiving $1.0 \mu\text{g Hg g}^{-1}$ food (high-mercury diet). After the exposure period, fish fed the low- and high-mercury diets had mean body burdens of $0.254 \pm 0.015 \mu\text{g Hg g}^{-1}$ and $2.37 \pm 0.09 \mu\text{g Hg g}^{-1}$, respectively. Dietary mercury significantly impaired both growth and gonadal development in males, which was apparent as reduced fish length, weight, and gonadosomatic index. Testicular atrophy was observed in fish fed the mercury-tainted fillets, but was nonexistent in control animals. Mercury also suppressed plasma cortisol in juveniles (sexes combined). The findings of this study suggest that dietary methylmercury, at levels currently found in the aquatic environment, might reduce juvenile survival by impairing growth and immune function. Furthermore, these results suggest that methylmercury might also affect reproductive potential of teleosts by impairing testicular development in young.

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1. Introduction

The environment of fish in both developed and remote aquatic ecosystems is changing to contain increasing levels of mercury (Slemr and Langer, 1992; Swain et al., 1992). Although release of mercury from point sources has been reduced, mercury from more diffuse atmospheric origins has been rising (Fitzgerald, 1995). Once in aquatic systems, mercury is converted by sediment microbes to the more toxic methylated form that rapidly biomagnifies through the food web (World Health Organization, 1990). Acid rain compounds the environmental impact of anthropogenic mercury because aquatic organisms concentrate more mercury when living in waters of lower alkalinity (Spry and Wiener, 1991). The persistence of mercury in the environment is illustrated by the finding that its concentration, unlike that of cadmium, arsenic, and lead, did not decrease in North American freshwater fish between 1976 and 1984 (Schmitt and Brumbaugh, 1990).

The significance of the accumulation of mercury in the biotic environment is not well understood. In cases of extreme contamination, such as Minimata Bay, fish suffer severe neurological impairment and mortality (Kitamura, 1968); however, levels of mercury in lakes and streams of North America rarely reach these levels (Wiener and Spry, 1996). At lower concentrations, mercury accumulates in the gonads of fish (Lockhart and Uthe, 1972; McKim et al., 1976; Hodson et al., 1994; Pelletier and Audet, 1995) where it inhibits gonadal recrudescence (Dey and Bhattacharya, 1989; Kirubakaran and Joy, 1992) and gametogenesis (Kirubakaran and Joy, 1988; Kirubakaran and Joy, 1992; Wester and Canton, 1992). It also reduces growth (Panigrahi and Misra, 1978; Rodgers and Beamish, 1982; Snarski and Olson, 1982; Weis and Khan, 1990; Niimi and Kissoon, 1994) and immune function (Roales and Perlmutter, 1977), which can affect offspring survival. Therefore, instead of producing large fish kills such as those observed in Minimata Bay, lower concentrations of mercury might affect fish populations indirectly by impairing reproductive performance.

Unfortunately, our knowledge of the effects of mercury on teleost reproductive processes is derived largely from the results of studies that employed exposure scenarios rarely observed in North America. In most cases, exposure concentrations were in the $\mu\text{g Hg l}^{-1}$ range, much higher than the ng Hg l^{-1} levels observed in natural waters (Wiener and Spry, 1996). Investigators also frequently used inorganic mercury even though more than 95% of mercury in fish exists primarily in the more toxic, methylated form (Kamps et al., 1972; Huckabee et al., 1979). Even in waters severely contaminated with dissolved inorganic mercury, methylmercury still constitutes 85% or more of the total mercury content in fish (Southworth et al., 1995). Furthermore, in spite of the fact that dietary uptake characterizes most mercury accumulation in natural waters (Mathers and Johansen, 1985; Spry and Wiener, 1991; Harris and Snodgrass, 1993), the majority of studies examining reproductive effects administered mercury to the water. Although the influence of exposure

method on mercury toxicity has not been well studied, Phillips and Gregory (1979) demonstrated that rainbow trout (*Oncorhynchus mykiss*) assimilated a much greater percentage of methylmercury when the metal was administered through the diet (68%) rather than the water (10%).

Therefore, the objective of our study was to determine whether consumption of a diet containing levels of methylmercury similar to those encountered in the wild would result in impaired growth, gonadal development, and immune function of young fish. Walleye (*Stizostedion vitreum*) was selected as the target species for this study because the effects of environmental mercury will most likely be observed first in upper-level predators. Piscivorous fish, and walleye especially, accumulate the metal to a much greater extent than other teleost species (Bahnick et al., 1994). The dietary levels of mercury chosen for this study (0.1 and 1.0 $\mu\text{g Hg g}^{-1}$ food) bracket those commonly encountered by piscivorous fish in North American lakes and streams (Mathers and Johansen, 1985; Parks et al., 1991; Borgmann and Whittle, 1992).

2. Materials and methods

2.1. Juvenile fish

Six-month-old walleye were obtained in October 1993, from the Vermont Fish and Wildlife Department's Bald Hill Hatchery (Newark, VT). Fish were acclimated in four 180 l aquaria over a period of two and a half months. At the beginning of the experiment in January 1994, fish were grouped together in a holding tank and randomly divided among the four aquaria (two control and two treatment tanks) to yield 22 animals per tank. Fish length (total) and weight were recorded at this time. Animals were allowed to recover from handling stress over an 11 day period. During this time, two fish from each of the control tanks displayed signs of illness (erratic swimming behavior, loss of equilibrium) and were removed from the study.

2.2. Mercury exposure

Fish were maintained on a diet of farm-raised catfish fillets cut initially into 1 g pieces. Three and a half months into the exposure period, the amount of catfish fillet given per fish was increased from 1.0 to 1.5 g. Fillets fed to fish in the treatment tanks were injected with methylmercury (Sigma Chemical Company, St. Louis, MO) dissolved in distilled water. One group received fillets injected with 0.1 $\mu\text{g Hg g}^{-1}$ food (low-mercury diet) and the other received fillets injected with 1.0 $\mu\text{g Hg g}^{-1}$ food (high-mercury diet). Analyses of fillets showed concentrations of less than 0.04 $\mu\text{g Hg g}^{-1}$ in the control diet, 0.137 $\mu\text{g Hg g}^{-1}$ in the low-mercury diet, and 0.987 $\mu\text{g Hg g}^{-1}$ in high-mercury diet. To prevent aquatic exposure, non-assimilated (excreted) methylmercury was removed from the aquaria by pumping water through activated charcoal filters that were changed every 10 days. In addition, a 40% water change was performed on each tank every 7-10 days. At the

conclusion of the 6 month experiment, mercury content of water samples was below the detection limit in all tanks.

Fish in all groups were fed three times each week. Because of the aggressive feeding behavior displayed in all tanks and the time required to swallow the food, consumption was typically limited to one piece of fillet per fish. When walleye were large enough (6 weeks into the exposure period), their diet was supplemented twice monthly with fathead minnows (*Pimephales promelas*), each weighing 1.3–1.5 g. For the mercury-exposed groups, minnows were injected with methylmercury to produce a tissue content similar to that in the catfish filets. Fish in all tanks continued to feed aggressively throughout the experiment, and all food was consumed at each feeding.

All aquaria contained moderately hard reconstituted water ($463 \text{ mg CaCO}_3 \text{ l}^{-1}$) at a temperature of 20°C . The light cycle followed the ambient photoperiod. Aquarium pH was maintained at $7.0 (\pm 0.8)$ throughout the experiment, with the exception of one brief period when it fell below 6.0 in the two control tanks and the tank receiving the low-mercury diet. Because walleye are photophobic and easily stressed by movement outside the aquaria, throughout both the acclimation and experimental periods tanks were completely covered in black plastic except for a 2 in. strip along the front bottom.

At the end of the 6 month exposure period, tanks were processed one at a time between 14:00 and 18:00 h in the following order: first control group, $0.1 \mu\text{g Hg g}^{-1}$ food group, $1.0 \mu\text{g Hg g}^{-1}$ food group, and second control group. Immediately prior to processing each tank, MS222 (Sigma Chemical Company) was added to the aquarium water, rapidly anaesthetizing all fish in the tank. Fish were then removed individually and total length and body weight of each were recorded. A blood sample was collected from the gill sinus venosus and by cardiac puncture. The gonads were removed, weighed, and fixed in formalin.

2.3. Mercury body burden

All animals in the mercury-treatment groups and 12 fish from the control group were analyzed for mercury content (whole fish minus viscera). Fish containing a mercury content below the detection limit were assigned a value of $0.04 \mu\text{g Hg g}^{-1}$ (the detection limit) for purposes of statistical analyses. The amount of mercury accumulated by each of the groups fed a mercury tainted diet was estimated using the following formula:

$$\text{Accumulation} = (\text{Hg}_E - \text{Hg}_{BK} / \text{Hg}_F) \times 100$$

where Hg_E is average total mercury content per exposed fish, Hg_{BK} is average total mercury content per control fish (background) and Hg_F is total amount of mercury fed per fish over the 6 month exposure period.

Average total mercury content per fish was calculated by multiplying mean mercury content of each group by the mean weight of each group. The concentration measured in samples of injected filets ($0.987 \mu\text{g Hg g}^{-1}$ for the high-mercury diet

and $0.137 \mu\text{g Hg g}^{-1}$ for the low-mercury diet) was used to estimate the total mercury fed over the 6 month period.

2.4. Mercury analyses

All mercury analyses (total mercury) were performed by Hazleton Environmental Services, Inc. (Madison, WI) using cold vapor atomic absorption spectrometry (AAS). The detection limit of this assay was $0.2 \mu\text{g l}^{-1}$ for water samples and $0.04 \mu\text{g g}^{-1}$ for tissue samples (wet weight).

2.5. Gonadal development

The sex of each animal was determined by standard histological analysis on sections of paraffin-embedded, formalin-fixed gonads. Males and females were easily distinguishable in histological section. As a crude measure of gonadal development, the gonadosomatic index (GSI) was determined using the following formula

$$\text{GSI} = (\text{gonadal weight}/\text{total body weight}) \times 100$$

To look more closely for developmental abnormalities, sections counterstained with haematoxylin and eosin were examined for lesions.

2.6. Cortisol

Plasma cortisol was determined by the Clinical Assays solid phase radioimmunoassay (INCSTAR Corporation, Stillwater, MN). This assay performed well with walleye plasma samples; a good dilution linearity indicated no adverse matrix effects. The detection limit for this assay, as calculated by the manufacturer, is $0.21 \mu\text{g dl}^{-1}$. The intra-assay precision according to our cumulative quality control data was 7.96% (coefficient of variation, *CV*) for 148 assays of a control sample with a mean of $4.24 \mu\text{g dl}^{-1}$ cortisol and 5.94% (*CV*) for 148 assays of a control sample with a mean of $19.72 \mu\text{g dl}^{-1}$ cortisol.

2.7. Statistical analysis

Differences in response variables (mercury body burden, length, weight, cortisol levels) were first analyzed using two-way analysis of variance (ANOVA) to detect a significant interaction between animal gender and treatment. Where a significant interaction was found, sexes were analyzed separately in a one-way ANOVA. If no interaction was found, sexes were combined in a one-way ANOVA. Male and female GSIs were analyzed separately because of the fundamental difference in tissue types (testes and ovaries). Significant differences indicated by ANOVA ($P < 0.05$) were further analyzed by Fisher's post-hoc tests. Within a tank, simple regression analyses were used with the calculation of correlation coefficients (*r*). Differences between mortality rates were examined using Kaplan–Meier statistics.

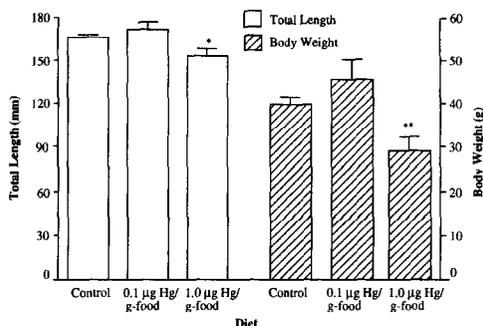


Fig. 1. Mean total body length and weight of male juvenile walleye at the end of the 6 month experimental period. Fish were fed a diet of untreated catfish fillets (control) or fillets injected with 0.1 or 1.0 $\mu\text{g Hg g}^{-1}$ food. Single and double asterisks indicate a significant difference from the control ($P < 0.02$ and $P < 0.004$, respectively). Bars represent one standard error.

Measurements of weight, length, and male GSI from the two control tanks were pooled in all statistical analyses because no significant differences were found between fish in these two tanks using one-way ANOVA. The female GSIs of the two control tanks were not compared because one of these tanks contained only two females. Owing to a laboratory error, cortisol values from one of the control tanks were not available; therefore, the comparison of cortisol levels is based on fish from only one control tank.

3. Results

3.1. Mercury body burden

The average mercury contents and accumulation rates in fish are shown in Table 1. Fish fed the low- and high-mercury diets had significantly higher body burdens of mercury than control fish. There were no significant differences between males and females in either treatment group.

3.2. Growth

At the start of the exposure period, there were no significant differences in the

Table 1
Mean mercury content (± 1 SE) and accumulation rate in juvenile walleye

	Control	0.1 $\mu\text{g Hg g}^{-1}$ food	1.0 $\mu\text{g Hg g}^{-1}$ food
Mercury content ($\mu\text{g g}^{-1}$)	0.06 (± 0.005)	0.25 (± 0.015) ^a	2.37 (± 0.09) ^a
Accumulation rate (%)	NA	68	88

^aSignificantly different from control ($P < 0.0002$).

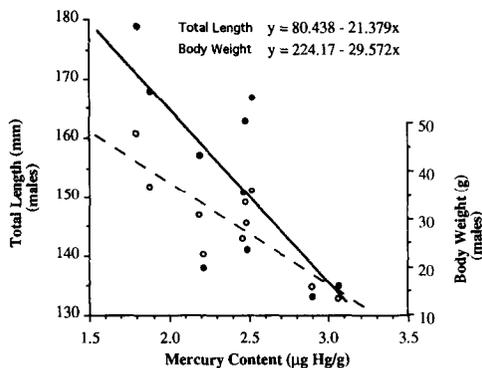


Fig. 2. Total length (●) and body weight (○) versus mercury body burden (wet weight) for male juvenile walleye reared on a diet tainted with $1.0 \mu\text{g Hg g}^{-1}$ food. A significant negative correlation between mercury content and both length (solid line; $r=0.82$; $P<0.004$) and weight (broken line; $r=0.74$; $P<0.02$) was found.

mean lengths and weights of fish among the treatment and control groups. At the end of the exposure period, because a two-way ANOVA revealed a significant interaction between gender and mercury exposure for both length and weight, males and females were analyzed separately. The mean length and weight of males fed the high-mercury diet was significantly lower than that of the control group (Fig. 1). There were no significant differences between males in the control and low-mercury diet groups, or between females in any group. For males fed the high-mercury diet, regression analysis revealed a significant inverse correlation between the body burden of mercury and the length and weight of the fish (Fig. 2).

3.3. Mortality rates

The mortality rate of the fish fed the low and high mercury-tainted diets were 45% and 32%, respectively. The control group had a 28% mortality rate. The relatively high mortality rate of control animals reflects the difficulty of maintaining this species under laboratory conditions. Although the mortality rates in the mercury treatments were slightly higher, when Kaplan–Meier survival statistics were compared, these differences were not significant.

3.4. Gonadal development

Histological examination of the gonads revealed that there were 14 males and nine females in the control tanks, eight males and four females fed the low-mercury diet, and ten males and four females fed the high-mercury diet. The gender of six fish in the control tanks and one fish fed the high-mercury diet could not be determined because their gonads were lost in processing.

The mean gonadosomatic indices of male and female fish reared on the mercury-tainted diets were lower than those in control fish, though these differences were not

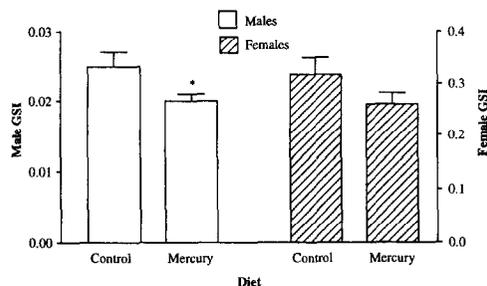


Fig. 3. Gonadosomatic indices in male and female juvenile walleye reared for 6 months on a diet of untreated catfish fillets (control) or fillets injected with 0.1 or 1.0 $\mu\text{g Hg g}^{-1}$ food. Asterisk indicates a significant difference from control ($P < 0.05$). Bars represent one standard error.

significant in the ANOVA. However, with the additional statistical power generated by combining fish from the two mercury treatments, the mean GSI of males fed either mercury-tainted diet was significantly lower than that of males fed the control diet (Fig. 3). Female GSIs were still not significantly different in this combined test.

Histological analyses yielded more striking results. Testes from fish fed the high-mercury diet showed serious disruption of the normal architecture with significant, multifocal cell atrophy (Fig. 4(a)). Adjacent residual cells were swollen. Testicular atrophy was observed to a lesser degree in fish fed the low-mercury diet (Fig. 4(b)) and was non-existent in control fish (Fig. 4(c)). Ovaries from both control and mercury-fed fish did not contain any predominant lesions.

3.5. Cortisol

Levels of cortisol were significantly lower in fish reared on the low-mercury diet than in fish from the control group (Table 2). Although plasma cortisol was also lower in fish reared on the high-mercury diet, this difference from control was not significant. To look for an effect of anaesthesia on cortisol levels, plasma steroid levels and sampling order within each tank were analyzed by regression analysis. No significant correlations were detected.

4. Discussion

The findings of this study suggest that a diet containing relatively low concentra-

Table 2
Mean plasma cortisol levels (± 1 SE) in juvenile walleye following exposure to dietary methylmercury

	Control	0.1 $\mu\text{g Hg g}^{-1}$ food	1.0 $\mu\text{g Hg g}^{-1}$ food
Cortisol ($\mu\text{g dl}^{-1}$)	15.6 (± 1.8)	8.0 (± 1.8) ^a	11.4 (± 2.0)

^aSignificantly different from control ($P < 0.03$).

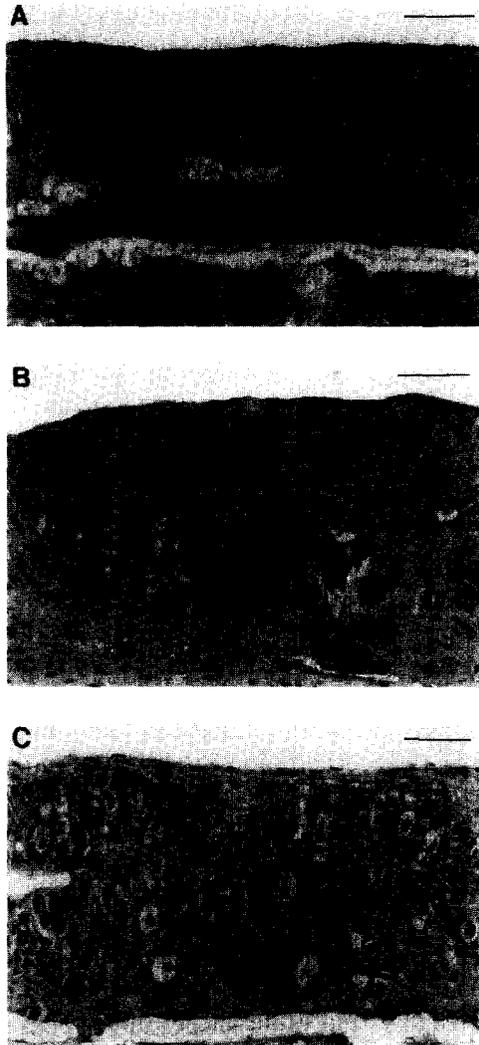


Fig. 4. (a) Extensive focal atrophy (arrows) in the testes of a 1-year-old walleye fed catfish filets tainted with $1.0 \mu\text{g Hg g}^{-1}$ food (bar $50 \mu\text{m}$). (b) A cross-section of testis from a 1-year-old walleye fed a diet of catfish filets containing $0.1 \mu\text{g Hg g}^{-1}$ food for 6 months (bar $50 \mu\text{m}$). Limited focal atrophy is present (arrow). (c) Section of a testis from a 1-year-old walleye maintained for 6 months on untainted catfish filets (bar $50 \mu\text{m}$). The testis is filled with undifferentiated spermatogonia (arrows).

tions of methylmercury, one similar in concentration to that which is available to many piscivorous fish in North America, can inhibit growth, testicular development, and immune function in young teleosts. Mercury ingestion and body burden were associated with lower plasma cortisol levels and reduced body weight and length. Gonadal development in male fish reared on the mercury-tainted diet was also impaired, with testes displaying regions of severe atrophy. To our knowledge,

these are the first data to suggest that environmentally realistic concentrations of mercury in food might impair the reproductive development of young fish.

The exposure regimens employed in this study were designed to reflect mercury exposure in the natural environment. The concentrations of dietary mercury achieved in this study bracket the levels commonly encountered by piscivorous fish in North American lakes and streams. A nationwide study by the US Environmental Protection Agency in 1984 detected concentrations of mercury in yellow perch (*Perca flavescens*), a common prey species for walleye, between 0.03 and 0.22 $\mu\text{g Hg g}^{-1}$ (Schmitt and Brumbaugh, 1990). Relevant to the walleye population targeted in this study, yellow perch taken in 1987 from various sites in Lake Champlain contained levels of mercury between 0.13 and 0.35 $\mu\text{g Hg g}^{-1}$ (Vermont Agency of Natural Resources, 1990). Focusing on the small fish and invertebrates that might reasonably be used as prey by young walleye, mercury content for small smelt ranges from 0.03–0.07 $\mu\text{g Hg g}^{-1}$ in Lake Ontario (Borgmann and Whittle, 1992) to 0.07–0.57 $\mu\text{g Hg g}^{-1}$ in Lake Simcoe (Mathers and Johansen, 1985). Small yellow perch (1–2 years old) from lakes in the Adirondacks in New York (Simonin et al., 1994) and the Winnipeg system in Ontario (Parks et al., 1991) contained 0.08–0.7 $\mu\text{g Hg g}^{-1}$. Levels in crayfish from lakes and rivers in Ontario range from 0.03 to 3.5 $\mu\text{g Hg g}^{-1}$ (Parks et al., 1991).

At the end of the 6 month exposure period, fish in the two mercury exposure groups contained concentrations of mercury similar to those observed in midsize adult walleye taken from Lake Champlain (mean 1.02 $\mu\text{g Hg g}^{-1}$, range 0.46–2.17 $\mu\text{g Hg g}^{-1}$; Vermont Agency of Natural Resources, 1990). Because mercury content in fish increases with age, the concentrations we achieved in the fish exposed to the high-mercury diet were clearly at the upper end of what might typically be expected for young fish in the wild. Nevertheless, the similarity in levels reinforces the general appropriateness of this exposure regimen for assessing the impact of environmental mercury on natural fish populations.

Our finding that dietary mercury inhibits growth in juvenile walleye raises the possibility that this metal impairs teleost reproductive success by affecting offspring survival. Because good growth during the summer lessens the susceptibility of young-of-year fish to predation and winterkill (Forney, 1976), reductions in growth can result in higher offspring mortality.

Other investigators have reported the inhibition of both appetite and growth by mercury, but these effects were demonstrated using concentrations well above what might be expected in the natural environment. For water-borne exposure, levels of mercury in the $\mu\text{g l}^{-1}$ range impair growth, feeding, and/or prey capture (Panigrahi and Misra, 1978; Rodgers and Beamish, 1982; Snarski and Olson, 1982; Weis and Khan, 1990; Niimi and KISSOON, 1994). For dietary exposure, severely contaminated food suppresses appetite and growth. Scherer et al. (1975) described decreased feeding and emaciation in 2-year-old walleye fed a diet of contaminated northern pike (*Esox lucius*) heads that contained an average of 7.9 $\mu\text{g Hg g}^{-1}$ for about 300 days. Other investigators likewise report decreased feeding and growth at even higher concentrations of dietary mercury (Matida et al., 1971; Rodgers and Beamish, 1982).

Our observation that juveniles consumed all the food provided suggests that the appetite-suppressant effect of this metal does not occur at lower levels of exposure. If the reductions in growth we observed are not the result of appetite suppression, then some other mechanism(s) must be involved. One possibility might be through the ability of mercury to reduce circulating levels of cortisol, as suggested by the findings of this and other studies (Kirubakaran and Joy, 1991; Hontela et al., 1992). Another mechanism might involve inhibition of thyroid function. Exposure to water-borne mercury has resulted in atrophy of pituitary thyrotrophs in the freshwater murrel *Channa punctatus* (Joy and Kirubakaran, 1989), and in decreased levels of circulating T_4 in *Channa punctatus* and the catfish *Clarias batrachus* (Bhattacharya et al., 1989; Kirubakaran and Joy, 1994). Both cortisol and thyroid hormone play important roles in teleost metabolism (Soengas et al., 1992; Vijayan et al., 1994).

The current finding that dietary mercury is associated with a decrease in the GSI suggests that this metal can impair gonadal development prior to the onset of sexual maturity. The testicular atrophy observed in fish fed the mercury-tainted diet most likely contributed to this decrease in GSI. The mechanism by which mercury causes this atrophy is unknown, but mercury can alter mitotic activity in teleosts (Wobeser, 1975; Dial, 1978; Wester and Canton, 1992). Other mechanisms might involve the ability of mercury to decrease hypothalamic gonadotroph size and number (Joy and Kirubakaran, 1989). Atrophy in developing testes will likely have a long-term impact on individual reproductive potential. Identifying which aspects of adult gonadal function are affected by mercury accumulation, such as steroidogenesis and/or gametogenesis, awaits further study, but both of these testicular functions might be affected. Catfish (*Clarias batrachus*) exposed to 32–37 $\mu\text{g Hg l}^{-1}$ for 180 days showed retarded gamete maturation and impaired Leydig cell 3β -hydroxy- Δ^5 -steroid dehydrogenase activity (Kirubakaran and Joy, 1988; Kirubakaran and Joy, 1992). Spermatogenesis was inhibited in guppies (*Poecilia reticulata*) following a 3 month exposure to 4.5–8.0 $\mu\text{g Hg l}^{-1}$ (Wester and Canton, 1992).

The finding that walleye reared on 0.1 $\mu\text{g Hg g}^{-1}$ food contained significantly lower plasma levels of cortisol than those of control animals raises the possibility that the immune system of juvenile fish might also be altered by exposure to relatively low levels of this metal. While the change in plasma cortisol could have been the result of the 2 h difference in plasma collection between treatment and control groups (15:00–16:00 h vs. 17:00–18:00 h, respectively), this seems unlikely. Malison and coworkers found that circulating levels of cortisol in walleye do not change during this time period (J.A. Malison, personal communication, 1995).

As in mammals, cortisol plays an important role in the stress response and immune function of teleosts. Chronic exposure to pollutants might exhaust the cortisol-producing endocrine system. Fish from sites polluted with several toxicants, including mercury, exhibit an attenuated cortisol response during capture (Hontela et al., 1992). Interestingly, this effect is most pronounced in juvenile fish (Hontela et al., 1995). Northern pike taken from a lake heavily contaminated with mercury had lower levels of plasma cortisol than fish from a more pristine lake (Lockhart and Uthe, 1972), but the attenuation of this cortisol was partially reversed in fish that

were transplanted to less contaminated waters. Catfish exposed to 32–37 $\mu\text{g Hg l}^{-1}$ for 180 days showed a time-dependent decrease in plasma cortisol levels (Kirubakaran and Joy, 1991). The adrenal cortex in these animals contained areas of necrosis and hyperplasia, and pituitary corticotrophs were hypertrophied and degranulated, suggesting a high level of ACTH secretion.

Taken together, the findings of this study suggest that levels of mercury currently found in the diet of wild piscivorous fish are likely to be harmful to juvenile gonadal development and probably can reduce overall survival. Because decreases in these upper level predators can lead to significant changes throughout aquatic food webs (Carpenter et al., 1985), adverse effects of mercury on piscivore reproductive performance could have implications for populations throughout the lake ecosystem.

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