U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
NATIONAL OCEAN SERVICE
OFFICE OF RESPONSE & RESTORATION
COASTAL PROTECTION AND RESTORATION DIVISION
c/o U.S. Environmental Protection Agency , Region 4
Waste Management Division
61 Forsyth Street, Atlanta, GA 30303

TECHNICAL MEMORANDUM

SUBJECT: Residue-Effects of Mercury in Fish

BY: Tom Dillon

DATE: March 26, 2001

1. Background

Mercury is a toxic and persistent bioaccumulative chemical found at many hazardous waste sites. Mercury poses risk to fish by two exposure routes; 1) direct exposure to abiotic media (sediment and water) and 2) the ingestion of contaminated prey. Risk posed by the first route is typically evaluated in ecological risk assessments by screening/refining Contaminants of Potential Concern (COPCs) and, if appropriate, toxicity tests with site-specific media. Ecological risk due to the direct exposure to abiotic media is not addressed in this memorandum.

Mercury accumulates in aquatic biota and may biomagnify to higher trophic levels due to the selective retention of methylated mercury. Quantifying this exposure for ecological risk assessments is usually accomplished using food web models. Models directly link abiotic media that may require remediation (e.g., sediments) to the risk estimates. Collecting higher trophic level fish to address this exposure pathway is usually undesirable because; 1) fidelity to the site may be low and/or highly uncertain and 2) links to potential remedial actions with site media are lost.

A mercury food web model for the red drum (*Sciaenops ocellatus*) has been cooperatively developed by natural resource trustees, EPA and responsible parties at an estuarine Superfund site on the Texas Gulf Coast (Evans and Engel, 1994). This model predicts whole body methylmercury concentrations in red drum arising from the exclusive ingestion of contaminated prey items. Red drum exposure to mercury-contaminated water and sediment are ignored in the model.

Interpreting the biological significance of mercury residues in fish requires an examination of residue-effects information published in the scientific literature. Conceptually, residue-effects information is similar to dose-response curves generated in toxicological experiments. At low doses, no effects are observed. At high doses, effects

are severe and frequently encountered. Between the low and high doses, adverse effects may be less severe.

2. **Purpose**

The purpose of this memorandum is to summarize the technical basis for developing a No Observable Adverse Effect Level (NOAEL) and a Lowest Observable Adverse Effect Level (LOAEL) for mercury in fish tissue. The NOAEL represents the very low end of the conceptual dose-response curve while the LOAEL signifies that region of the curve where adverse effects are beginning to emerge. Together, the NOAEL and LOAEL represent the threshold response for assessment endpoints involving fish. As with any ecological assessment endpoint, protective levels emerging from the threshold response may become Remedial Goal Objectives (RGOs) at hazardous waste sites and subsequent clean-up levels.

3. **Approach**

Beckvar et al. (1996) reported a tabular summary of mercury residue-effects information available for fish. Individual papers identified through literature searches and compilations developed by the US EPA (Jarvinen and Ankley, 1999) and the US Army Corps of Engineers Environmental Residue-Effects Database (www.wes.army.mil/el/ered) were obtained and critically reviewed. The tabular summary of Beckvar et al. (1996) has recently been updated (Table 1). Papers included represent a general bias for investigations reporting whole body concentrations, laboratory exposures, and ecologically important endpoints (e.g., survival, growth, reproduction, behavior). Because the purpose of this memorandum is to identify a NOAEL and LOAEL, those papers reporting both effects and no effects in whole body fish tissue were evaluated further (Table 2).

4. **Analysis**

Whole body tissue concentrations associated with no effects range between 0.02 μg/g and 2.7 μg/g with a median highest effect value of 0.15 μg/g (Table 2). Mercury residues associated with the lowest adverse effects ranged between 0.04 μg/g and 19 μg/g with a median value of 0.30 μg/g. Within each set of published results, the difference between the highest no effect and lowest effect level was small. The difference between the two median values is two-fold. This suggests a narrow threshold response in mercury-exposed fish. Lower concentrations are generally observed in investigations involving early life stage exposures (egg, embryo, larvae, fry).

5. **Discussion**

In their review of mercury effects on freshwater fish, Weiner and Spry (1996) suggest whole body concentrations of 5-10 μg/g in adult fish are associated with sublethal or lethal effects. They estimate a slightly lower no-observed-effect concentration of 3 μg/g. However, they note that adverse effects on early life stages of fish occur at much lower tissue concentrations (0.07-0.10 μg/g). This is consistent with the observations of many investigators that early life stages are more sensitive to contaminant exposure than adults. In concluding their review, Weiner and Spry (1996) posit that the greatest risk to fish populations is the maternal transfer of mercury to developing eggs and embryos.

Mercury is a neural toxin that exerts its adverse effects at the molecular level by tightly binding with sulfhydral moieties in proteins (enzymes and cellular membranes). This biochemical effect is often manifested at the organismal level by altered behavior. Two publications have emerged since the Weiner and Spry review (1996) which report concomitant mercury residues and behavioral effects. Fjeld et al. (1998) exposed grayling embryos (*Thymallus thymallus*) to methylmercury for 13 days. Tissue residues in exposed embryos ranged between 0.09 μg/g and 3.80 μg/g, wet weight (Table 2). Exposure was terminated soon after hatching and fish raised to adulthood under normal, controlled conditions. *Three* years after exposure as embryos, adult fish were tested in the laboratory for their ability to compete for and capture live prey. Fish with embryo mercury residues between 0.30 and 3.8 μg/g were significantly impaired in their ability capture and compete for live prey. Embryos in the control and lowest (unaffected) exposure group had mercury residues of 0.01 μg/g and 0.09 μg/g, respectively. This investigation demonstrates that fish feeding behavior in adults can be permanent impaired following short-term mercury exposure as embryos. Embryo mercury residues in the lower exposure treatments reported by Fjeld et al. (1998), 0.09-0.3 μg/g, are very similar to the early life stage effects range suggested by Weiner and Spry (1996); i.e., 0.07-0.10 μg/g.

Altered fish behavior (leading to significant mortalities) was also reported in a recent paper by Matta et al. (2001). *Fundulus heteroclitus* were chronically exposed to methylmercury in food then induced to spawn. Spawning males with tissue concentrations equal to or greater than 0.47 μg/g exhibited altered behavior (extreme aggression or pacificity) which led to significant mortalities in the passive fish. Survival of fish with slightly lower residues, 0.20 μg/g or less, was unaffected. In both the Matta et al. (2001) and Fjeld et al. (1998) investigations, the differences between the highest no effect and lowest effect concentrations were very small (0.09 μg/g vs 0.3 μg/g and 0.20 μg/g vs 0.47 μg/g).

When attempting to identify residue-based NOAELs and LOAELs from laboratory studies, it is appropriate to consider regional or site-specific background information. These data provide independent perspective, especially regarding the appropriateness of a

potential NOAEL. That is, if tissue concentrations in fish collected from "clean" background locations approximate the NOAEL, one's confidence in the proposed NOAEL increases. The NOAEL-to-background comparison is also important from the risk management/remedial action perspective. Actions are rarely taken for risks at or below background.

In a national study of chemical residues in fish, field samples were collected by EPA at hazardous waste sites as well as background locations (EPA 1992). In fish from the EPA Region 4 background locations, whole body mercury was detected in 10 of 11 samples. Concentrations ranged between 0.03 and 0.29 μg/g wet weight with a median value of 0.17 μg/g (Table 3). This median background concentration is almost identical to the median highest no effect value (0.15 μg/g) observed in laboratory toxicity studies (Table 2).

Two independent investigations provide fish tissue reference data for the LCP site. Matta et al. (1998) reported mean mercury concentrations in fish collected at two reference stations in the Crescent River to be 0.02 and 0.04 μg mercury/g dry weight. Using percent solids reported in Matta et al. (1998, Table 3.4), these mean concentrations would be 0.005-0.01 μg mercury/g wet weight. Similar low tissue concentrations (0.023 μg mercury/g wet weight) were reported by Sprenger et al. (1997) for forage fish collected in the Troup Creek and the Little Satilla River reference locations for the LCP project.

6. **Conclusions**

a. Laboratory toxicity studies indicate whole body mercury tissue concentrations associated with no effects approximate a median value of 0.15 μg/g wet weight (Table 2). This concentration is nearly identical to the median concentration (0.17 μg/g wet weight) found in whole fish from background locations throughout EPA Region 4 (Table 3). These toxicologically-based and background concentrations are about an order of magnitude greater than those observed for fish collected at multiple reference stations for the LCP site, Brunswick, GA.

b. Laboratory toxicity studies indicate lower whole body mercury tissue concentrations associated with adverse effects range between 0.04 μg/g and 19 μg/g with a median value of 0.30 μg/g wet weight (Table 2). More frequent, severe adverse effects in fish are likely as tissue concentrations increase.

c. Within individual investigations, the difference between the no effects and lowest effect tissue concentration is small (Table 2) suggesting a sharp threshold response for mercury-contaminated fish.

d. As with most environmental contaminants, early life stages of fish (eggs, embryos, fry) are generally more sensitive to mercury than adults. Short-term mercury exposure to early life stages can have permanent, adverse effects on successful feeding behavior later in life.

e. Maternal transfer of mercury to early life stages represents a viable exposure pathway.

f. Laboratory toxicity studies indicate the lowest whole body mercury tissue concentrations associated with adverse effects in fish embryos approximate 0.07-0.1 μg/g wet weight (Weiner and Spry 1996, Fjeld et al. 1998).

7. **Uncertainties in Selecting a NOAEL and a LOAEL**

Many sources of uncertainty are embedded in any residue-effects analysis (see discussion in Jarvinen and Ankley 1999). Most uncertainties are associated with laboratory toxicity studies generating the biological effects and chemical residue data. Specific sources of uncertainty include interspecific contaminant sensitivity, life stage differences, exposure regimes (e.g., duration, food vs. water), endpoints examined and analytical chemistry. We attempted to reduce some of these uncertainties by focusing on published investigations that employed longer exposures, examined biologically important endpoints such as growth, reproduction, behavior and reported whole body tissue concentrations. Of these sources of uncertainty, differences in life stage appear to have the largest quantitative impact on mercury residue-effects in fish.

In selecting a NOAEL and LOAEL, it is useful to discuss the uncertainties associated with the low, middle and high portions of the conceptual dose-response curve. At the lower, no effects end of the curve, 0.15 μg/g wet weight appears to be a representative concentration (Table 2). Certainty in this value is increased by two independent lines of evidence. One, the 0.15 μg/g value closely mirrors median whole fish concentration at background locations in EPA Region 4 (0.17 μg/g wet weight). Two, mercury in fish collected at LCP reference stations are about an order of magnitude lower (≅0.01-0.02 μg/g wet weight).

Uncertainty at the high end of the dose-response curve is inherently low because adverse effects are more frequent and typically severe (e.g., death). In their review, Weiner and Spry (1996) conclude that adverse effects in fish are consistently observed at whole body tissue concentrations between 5 and 10 μg/g wet weight.

Greater uncertainty is encountered in the middle of the dose-response curve. This is especially true at the lower end of the curve where the LOAEL resides. This difficulty is compounded by the observation in many investigations that the difference between no

effects and effects may be small. If we accept 0.15 μg/g wet weight to be a reasonably certain NOAEL, the median lowest effects level of 0.30 μg/g wet weight (Table 2) can be considered a reasonable, albeit less certain, LOAEL. The 0.30 μg/g wet weight value represents most of the lowest effects tissue concentrations reported in Table 2. Based on its guidance for ecological and human health risk assessments, EPA generally defers to the more conservative, environmentally protective toxicity reference values. A LOAEL of 0.30 μg/g wet weight for mercury effects on fish is consistent with that guidance. It is interesting to note that 0.30 μg/g wet weight is also the tissue concentration EPA recently (January 2001) recommended fish not exceed to be protective of human health (www.epa.gov/waterscience/standards/methylmercury)..

NOAEL and LOAEL are single points on a toxicological dose-response continuum. One can always argue that a specific value could be slightly higher (less protective) or slightly lower (more protective) based on the available data and accompanying uncertainty. However, the values developed above and recommended below appear consistent with information presented in the scientific literature as well as EPA guidance for ecological risk assessments.

8. **Recommended NOAEL and LOAEL for Residue-Effects of Mercury in Fish**

a. NOAEL: 0.15 μg/g wet weight

b. LOAEL: 0.30 μg/g wet weight

c. Use the above values to interpret whole body adult fish residues predicted from higher trophic level food web modeling (Evans and Engel 1994) conducted for the ecological risk assessment at the LCP site.

Table 1. A summary of mercury residue-effects literature for fish.

Fish Species	Life Stage	Exposure	Duration	Endpoint	Tissue Analyzed	No Effect Residue (ug/g, ww)	Effects Residue (ug/g, ww)	Effect Description	Reference
Rainbow trout *Oncorhynchus mykis*	adult through spawning	$HgCl_2$ 0.24 µg/l flow-thru	400-528 days	mortality, teratogenic	egg gonad	0.04 0.09	0.26-3.67 0.49-4.57	significant reduction in alevin survival (4-day post hatch); significant increase in teratogenic effects	Birge et al. 1979
"	eyed eggs to 10-day old fry	$HgCl_2$ 0.18-107 µg/l in sediments + 0.25-6.4 µg/l in overlying water	20 days = 10-day pre- and 10-day post-hatching	mortality	whole body of 10-day old fry	0.02	0.04 0.3 0.9	55% mortality at 10 days 77% mortality at 10 days 100% mortality at 10 days	Birge et al. 1979
"	Developing embryo	$HgCl_2$ 0.1-0.14 µg/l flow-thru	8 days	mortality	eggs	0.02 - 0.04	0.07 0.1	17-21% mortality at 4 days 100% mortality at 8 days	Birge et al. 1979
"	fingerlings	CH_3HgCl in food	84 days	growth, behavior, physiology	whole body		10-30 30-35	diminished growth and appetite; darkened skin and lethargy	Rodgers and Beamish 1982
"	fry-juvenile	total mercury 50 µg/g in food	270 days	growth, behavior	brain liver muscle whole body	 0.2	16-30 26-68 20-28 19	darkened skin; diminished appetite, visual acuity, and growth; loss of equilibrium	Matida et al. 1971
"	fingerlings	CH_3HgCl 4-24 µg/g in food	105 days	growth, histology, biochemistry	muscle	<0.2 12	12-24 19-24	hyperplasia of gill epithelium increased blood packed cell volume, reduced growth	Wobeser 1975
"	subadult	CH_3HgCl 4 µg/l flow-thru	30-98 days	mortality, behavior	brain liver muscle		7-32 32-114 9-52	diminished appetite and activity	Niimi and Kissoon 1994
"	subadult	CH_3HgCl 9 µg/l flow-thru	12-33 days	mortality, behavior	whole body		4-27	diminished appetite and activity	Niimi and Kissoon 1994

Fathead minnow *Pimephales promelas*	Fry fed dry food	$HgCl_2$ 0.31-4.51 µg/l flow-thru	60 days	survival, growth, development	whole body	0.12 - 0.8	1.3 4.2	retarded larval growth retarded larval growth, 50% mortality, spinal curvature	Snarski and Olson 1982
"	Fry fed live food	$HgCl_2$ 0.26-3.7 µg/l flow-thru	60 days	survival, growth, development	whole body	0.22 - 2.64	4.7-7.60	retarded larval growth; control growth better on live food	Snarski and Olson 1982
"	Full life cycle on live food + F1 larvae	$HgCl_2$ 0.26-3.7 µg/l flow-thru	41 weeks	survival, growth, reproduction	whole body F0 adult fish	0.32	1.36 - 2.84 4.47 - 18.8	reduced growth in F0 females and F1 fry spawning ceased; external sex. features absent	Snarski and Olson 1982
Brook trout *Salvelinus fontinalis*	continuous exposure of F0,F1 fish + F2 embryos	CH_3HgCl 0.29 µg/l aqueous	273 days	mortality, growth, reproduction	brain liver gonad F0 whole body	5 8 3 2.7		no apparent effects	McKim et al. 1976
"	continuous exposure of F0,F1 fish + F2 embryos	CH_3HgCl 0.93 µg/l aqueous	273 days	mortality, growth, behavior	brain liver gonad F0 whole body		17 24 12 5-7	increased mortality, decreased growth, lethargy, and deformities in F1 embryos, no spawning	McKim et al. 1976
"	continuous exposure of F0,F1 fish + F2 embryos	CH_3HgCl 0.93 µg/l aqueous	273 days	mortality	F2 embryo		2.2	deformed embryos; 100% mortality 3 weeks after hatching	McKim et al. 1976
"	continuous exposure of F0,F1 fish + F2 embryos	CH_3HgCl 2.9 µg/l aqueous	273 days	mortality	F1 embryo		12.5	Deformed embryos; no hatching observed	McKim et al. 1976
Channel catfish *Ictalurus punctatus*	Embryo to 4-day old larvae	$HgCl_2$ 0.3µg/L flow-thru	10 days	mortality	eggs		0.014-0.34	Survival reduced in dose-dependent manner. Median lethal concentration at 4 days post-hatching corresponds to a tissue concentration of 0.06 ug/g ww.	Birge et al. 1979
Walleye *Stizostedion vitreum vitreum*	1 year old	Methylmercury 5-13 µg/g in food	42-63 days	mortality, behavior, physiology	brain liver muscle	<1	3-6 6-14 5-8	emaciation; loss of locomotion, coordination and appetite.	Scherer et al. 1975
"	1 year old	Methylmercury 5-13 µg/g in food	240-314 days	mortality, behavior, physiology	brain liver muscle	<2.5	15-40 18-50 15-45	88% mortality; emaciation; poor locomotion, coordination and appetite.	Scherer et al. 1975
"	juveniles	methylmercury 0.14-1 µg/g in food	180 days	development, physiology	whole body (minus viscera)	0.06*	0.25 2.37	testicular atrophy and impaired development, impaired immune function testicular atrophy and impaired development, impaired immune function; impaired growth in males	Friedmann et al. 1996

Striped mullet *Mugil cephalus*	juvenile	CH_3HgCl 1 µg/l aqueous CH_3HgCl 10-20 µg/l aqueous	10,13 days 7,10,13 days	physiology	whole body	<0.1	0.3 5.0	fin regeneration inhibited fin regeneration inhibited	Weis and Weis 1978
Grayling *Thymallus* *thymallus*	embryos exposed, 3 yr adults tested	CH_3HgCl 0.16-20 µg/l aqueous	13 days until hatching	behavior, hatching, development	whole body (fry)	0.09	0.27 0.63 3.8	adult foraging efficiency, prey capture reduced adult foraging efficiency, prey capture reduced adult foraging efficiency, prey capture reduced; fry exhibit scoliosis, jaw deformities; embryos hatching reduced	Fjeld et al. 1998
Killifish *Fundulus* *heteroclitus*	adults exposed, 2 generations followed	CH_3HgCl 0.5-54 µg/g in food of F0 Adults	42 days F0 only	mortality, growth, reproduction, sex ratios	whole body F0 adults F1 eggs	0.2 <0.02	0.47 1.0-1.1 11-12 0.01 0.63	Abnormal behavior & reduced survival in F0 males; Reduced F1 fry growth Abnormal behavior & reduced survival in F0 males; Reduced F1 fry growth; altered F1 sex ratios Abnormal behavior & reduced survival in F0 males; Reduced F1 fry growth; altered F1 sex ratios; reduced fertilization success in F1 altered sex ratio in F1 altered sex ratio in F1, reduced fertilization success in F1 adults	Matta et al. 2001

Table 2. Residue-effects literature (from Table 1) reporting both effects and no effects in whole body fish tissue.

Reference	Life Stage Chemically Analyzed	No Effect Concentrations (mg/kg ww)	Effects Concentrations (mg/kg ww)	Types of Effects Measured	Exposure Regime
Birge et al. 1979	10-day old fry	0.02	0.04 - 0.9	survival	8-500 days water, sediment
Fjeld et al. 1998	embryo	0.09	0.3 - 3.8	hatching, fry deformities, 3 year old adult prey capture and foraging efficiency	13 days aqueous
Matida et al. 1971	fry/juvenile	0.2	19	growth, behavior, vision	270 days food
Weis and Weis 1978	juvenile	<0.1	0.3	caudal fin regeneration	7-13 days aqueous
Friedmann et al. 1996	adult (less viscera)	0.06	0.25 - 2.37	growth and gonadal development	180 days food
Matta et al. 2001	adult	0.2	0.4 - 12	survival, behavior, reproduction	42 days food
Snarski and Olson 1982	juvenile/adult	0.3 - 0.8	1.2 - 4.2	growth and reproduction	30-60 days, 41 weeks aqueous
McKim et al. 1976	adult	2.7	5-7	survival, growth, reproduction	147 weeks, 2 generations aqueous
Median Highest NOAEL =		**0.15**	**0.30**	**= Median LOAEL**	

Table 3. Concentrations of mercury (ug/g wet weight) in whole fish collected from background stations in USEPA Region 4.
Information taken from Appendix D of USEPA Report 823-R-92-008 (National Study of Chemical Residues in Fish, 1992).

From Table D-1				From Appendix D-5			From Appendix D-6		
						Hg			
				Fish		**Conc.**	**%**	**Wet Wt.**	**Date**
Episode	**Waterbody**	**Location**	**ST**	**Common Name**		**(ug/g)**	**Lipid**	**(g)**	**Collected**
3169	Inland Lake	Blout Co.	AL	Black Redhorse	WB	0.16	11.3	20.1	8710
3177	Chattahoochee R.	Gainsville	GA	Carp	WB	0.03	6.7	20.16	8709
3178	Chattooga R.	Clayton	GA	North.Hogsucker	WB	0.23	3.03	20.32	8709
3179	Chestatee R.	above Lake Lanier	GA	Golden Redhorse	WB	0.24	8.2	20.08	8709
2139	Cattaloochee Creek	Cattaloochee	NC	Carp	WB	0.08	7.9	19.96	8705
3166	Nanthalia R.	Macon Co.	NC	White sucker	WB	0.29	8.2	20.04	8410
3187	St. Helena Sound		SC	Summer Flounder	WP	0.05*	2.8	20.05	8711
2301	Buffalo R.	Flatwoods	TN	Sm. Bass	WB	0.18	NM	NM	8501
2301	Buffalo R.	Flatwoods	TN	Bluegill	WB	0.20	NM	NM	8501
2301	Buffalo R.	Flatwoods	TN	Black Cappie	WP	0.11	NM	NM	8501
2301	Buffalo R.	Flatwoods	TN	Rock Bass	WP	0.14	2.1	20	8501
				Median Conc.		**0.17**			

WB=Whole Body WP=Whole Predator NM=not measured

* Mercury detection limit of 0.05 ug/g was used for this non-detected sample

Common Fish Name	**Scientific Name**
Black Redhorse	*Moxostoma duquesnei*
North.Hogsucker	*Hypentelium nigricans*
Golden Redhorse	*Moxostoma erythrurum*
Carp	*Cyprinus carpio*
White sucker	*Catostomus commersoni*
Summer Flounder	*Paralichthys dentatus*
Sm. Bass	*Micropterus dolomieui*
Bluegill	*Lepomis macrochirus*
Black Cappie	*Pomoxis nigromaculatus*
Rock Bass	*Ambloplites rupestris*

References

Beckvar, N., J. Field, S. Salazar, R. Hoff. 1996. Contaminants in aquatic habitats at hazardous waste sites: Mercury. NOAA Technical Memorandum NOS ORCA 100. Seattle: Hazardous Materials Response and Assessment Division, National Oceanic and Atmospheric Administration, 74 pp.

Birge, W.J., J.A. Black, A. G. Westerman, and J.E. Hudson. 1979. The effects of mercury on reproduction of fish and amphibians. In: J. O. Nriagu (ed.). *The Biogeochemistry of Mercury in the Environment*. Elsevier/North-Holland Biomedical Press. pp. 629-655.

Evans, D.W. and D.W. Engel. 1994. Mercury Bioaccumulation in Fishfish and Shellfish from Lavaca Bay, Texas: Desscriptive Models and Annotated Bibliography. NOAA Tech. Memo NMFS-SEFSC-348, 89 pp.

Fjeld, E., T.O. Haugen., and L.A. Vollestad. 1998. Permanent impairment in the feeding behavior of grayling (*Thymallus thymallus*) exposed to methylmercury during embryogenesis. Sci. Tot. Environ. 213: 247-254.

Friedmann, A. S., M. C. Watzin, T. Brinck-Johnsen, J. C. Leiter. 1996. Low levels of dietary methylmercury inhibit growth and gonadal development in juvenile walleye (*Stizostedion vitreum*). Aquat. Toxicol. 35: 265-278.

Jarvinen, A.W. and G.T. Ankley. 1999. Linkage of effects to tissue residues: Development of a comprehensive database for aquatic organisms exposued to inorganic and organic chemicals. SETAC Press, Pensacola, FL.

Matida, Y., H. Kumada, S. Kimura, Y. Saiga, T. Nose, M. Yokote, and H. Kawatsu. 1971. Toxicity of mercury compounds to aquatic organisms and accumulation of the compounds by the organisms. Bull. Freshwater Fish. Res. Lab. (Tokyo) 21: 197-227.

Matta, M.B., S. Salazar, L. Mill, G. Gray, J. Linse, P. Peronard, L. Francendese. 1998. LCP Chemical Site Monitoring Study Data Report. August 25, 1998, 128 pp.

Matta, M.B., J. Linse, C. Cairncross, L. Francendese, and R.M. Kocan. 2001. Reproductive and trans-generational effects of methylmercury or Aroclor 1268 on *Fundulus heteroclitus*. Environ. Toxicol. and Chem 20(2): 327-335.

McKim, J.M., G.F. Olson, G.W. Holcombe and E.P. Hunt. 1976. Long-term effects of methylmercuric chloride on three generations of brook trout (*Salvelinus fontinalis*): toxicity, accumulation, distribution and elimination. J. Fish. Res. Bd. Can. 33: 2726-2739.

Niimi, A. J. and G. P. Kissoon. 1994. Evaluation of the critical body burden concept based on inorganic and organic mercury toxicity to Rainbow Trout (*Oncorhynchus mykiss*). Arch. Environ. Contam. Toxicol. 26: 169 - 178.

Rodgers, D.W. and F.W.H. Beamish. 1982. Dynamics of dietary methylmercury in rainbow trout, *Salmo gairdneri*. Aquat. Toxicol. 2: 271-290.

Scherer, E., F.A. J. Armstrong, and S.H. Nowak. 1975. Effects of mercury-contaminated diet upon walleyes *Stizostedion vitreum vitreum* (Mitchell). Fish. Mar. Serv. Tech. Rep. No. 597, 21 pp.

Snarski, V.M. and G.F. Olson. 1982. Chronic toxicity and bioaccumulation of mercuric chloride in the fathead minnow (*Pimephales promelas*). Aquat. Toxicol. 2: 143-156.

Sprenger, M. et al. 1997. Ecological Risk Evaluation of the Salt Marsh and Adjacent Areas of the LCP Superfund Site, Brunswick, GA., April 1997, by Environmentl Response Team, Edison, NJ.

US Environmental Protection Agency. 1992. National Study of Chemical Residues in Fish. EPA 823-R-92-008.

Weiner, J.G. and D.J. Spry. 1996. Toxicological significance of mercury in freshwater fish. In: Environmental Contaminants in Wildlife, Interpreting Tissue Concentration. Edited by W. N. Beyer, G.H. Heinz, A.W. Redmon-Norwood, SETAC Special Publication Series, Lewis Publishers, Boca Raton, FL.

Weis, P., and J.S. Weis. 1978. Methylmercury inhibition of fin regeneration in fishes and its interaction with salinity and cadmium. Estuarine Coastal Mar. Sci. 6: 327-334.

Wobeser, G. 1975. Prolonged oral administration of methyl mercury chloride to rainbow trout (*Salmo gairdneri*) fingerlings. J. Fish. Res. Bd. Can. 32: 2015-2023.